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# SUPERFUND PUBLIC HEALTH EVALUATION MANUAL

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EPA

# Superfund Public Health Evaluation Manual

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# N O T I C E

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16. ABSTRACT

The Superfund Public Health Evaluation Manual has been developed for use by a diverse audience, including EPA regional staff, state Superfund program staff, federal and state remedial contractors, and potentially responsible parties. Individuals having different levels of scientific training and experience are likely to use the manual in designing, conducting, and reviewing public health evaluations. Because assumptions and judgments are required in many parts of the analysis, the individuals conducting the evaluation are key elements in the process. The manual is not intended for use by non-technical personnel to perform technical evaluations, nor to allow professionals trained in one discipline to perform the work of another. Rather, it is the responsibility of remedial project managers, using the manual as a guide, to match the scientific support they deem necessary with the appropriate resources at their disposal.

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# SUPERFUND PUBLIC HEALTH EVALUATION MANUAL

Office of Emergency and Remedial Response
Office of Solid Waste and Emergency Response
U.S. Environmental Protection Agency
Washington, D.C. 20460

## *NOTICE*

This manual provides guidance on methods for public health evaluations that are conducted as part of EPA's feasibility study process at Superfund remedial sites. The manual specifically supports Chapter 5 of the Guidance for Feasibility Studies (U.S. EPA, Office of Emergency and Remedial Response, April, 1985), which briefly describes public health evaluation procedures. This manual does not contain procedures for health assessments, which are separate analyses conducted by the Agency for Toxic Substances and Disease Registry (ATSDR). The procedures and data given in this manual supersede information previously released by the Office of Emergency and Remedial Response on public health evaluation at Superfund sites.

## *ACKNOWLEDGMENTS*

This document was developed by EPA's Office of Emergency and Remedial Response (OERR). Dr. Craig Zamuda and Mr. Bruce Clemens of OERR's Policy Analysis Staff (PAS) were the EPA Project Officers, under the direction of Mr. James Lounsbury, Director of PAS. Additional guidance was provided by Ms. Stacey Katz of the Office of Policy, Planning, and Evaluation (OPPE).

Assistance also was provided by the EPA Work Group, whose members included:

| | |
|---|---|
| Harry Allen | OERR/Hazardous Response Support Division |
| Doug Ammon | ORD (Office of Research and Development) |
| James Baker | Region 8 |
| Judy Bellin | Office of Solid Waste |
| Paul Bitter | Region 5 |
| Brint Bixler | OERR/Hazardous Site Control Division |
| Bonnie Casper | ORD |
| Margaret Chu | ORD |
| Chris DeRosa | ORD |
| Terry Eby | OERR/Emergency Response Division |
| Sally Edwards | Region 1 |
| Dick Hill | Office of Pesticides and Toxic Substances (OPTS) |
| Josephine Huang | ORD |
| Phil Jalbert | OERR/Policy Analysis Staff |
| Meg Kelly | Office of Solid Waste and Emergency Response |
| Jack Kooyoomjian | OERR/Emergency Response Division |
| Arnie Kuzmack | Office of Drinking Water |
| John Mateo | Region 2 |
| Abe Mittelman | Office of Waste Programs Enforcement |
| Esther Rinde | OPTS |
| John Schaum | ORD |
| Anita Schmidt | OPTS |
| Paul Schumann | OERR/Hazardous Site Control Division |
| Ed Schoener | Region 3 |
| Ellen Siegler | Office of General Counsel |
| Jim Spatarella | OERR/Hazardous Site Control Division |
| George Sugiyama | Office of Air and Radiation |

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## *TABLE OF CONTENTS*


**PREFACE** . . . . . 1

**1. OVERVIEW OF THE SUPERFUND PUBLIC HEALTH EVALUATION PROCESS** . . . . . 3

1.1 Description of Process Components . . . . . 4

1.1.1 Baseline Public Health Evaluation . . . . . 4

1.1.2 Analysis of Remedial Alternatives and Development of Performance Goals . . . . . 5

*1.2 Applicability of Process Components to Various Sites* . . . . . 5

**2. BACKGROUND: AGENCY RULES, POLICIES, AND GUIDELINES** . . . . . 9

*2.1 The National Oil and Hazardous Substances Pollution Contingency Plan (NCP)* . . . . . 9

*2.2 Guidance for Remedial Investigations and Feasibility Studies* . . . . . 9

*2.3 CERCLA Compliance with Other Environmental Statutes* . . . . . 10

*2.4 Agency Policy for Planning and Implementing Off-Site Response Actions* . . . . . 11

*2.5 Agency Guidelines on Risk Assessment* . . . . . 12

*2.6 Memorandum of Understanding Between EPA and the Agency for Toxic Substances and Disease Registry* . . . . . 12

**3. STEP 1: SELECTION OF INDICATOR CHEMICALS** . . . . . 14

*3.1 Develop Initial List of Indicator Chemicals* . . . . . 14

*3.2 Select Final Indicator Chemicals* . . . . . 18

**4. STEP 2: ESTIMATION OF EXPOSURE POINT CONCENTRATIONS OF INDICATOR CHEMICALS** . . . . . 27

*4.1 Identify Exposure Pathways* . . . . . 29

4.1.1 Determine Possible Chemical Release Sources and Release Media . . . . . 31

4.1.2 Identify and Characterize Possible Human Exposure Points . . . . . 31

4.1.3 Integrate Release Sources, Environmental Transport Media, Exposure Points, and Exposure Routes into Exposure Pathways . . . . . 34

4.1.4 Determine Presence of Sensitive Human Populations . . . . . 36

4.2 Estimate Exposure Point Concentrations . . . . . 36

4.2.1 Quantify Chemical Releases . . . . . 38

4.2.2 Predict Environmental Fate and Transport . . . . . 39

4.3 Compare to Requirements, Standards, and Criteria . . . . . 42

4.3.1 Compare to Applicable or Relevant andAppropriate Requirements . . . . . 42

4.3.1.1 Maximum Contaminant Levels (MCLs) and Maximum Contaminant Level Goals (MCLGs) . . . . . 50

4.3.1.2 National Ambient Air Quality Standards (NAAQS) . . . . . 50

4.3.1.3 Federal Ambient Water Quality Criteria . . . . . 52

4.3.1.4 State Environmental Standards . . . . . 52

4.3.2 Compare to Other Criteria, Advisories, and Guidance . . . . . 53

4.3.2.1 Proposed MCLs and MCLGs . . . . . 53

## CONTENTS (continued)


4.3.2.2 Drinking Water Health Advisories . . . . . 60

5. **STEP 3: ESTIMATION OF CHEMICAL INTAKES** . . . . . 61

5.1 Calculate Air Intakes . . . . . 61

5.2 Calculate Ground-Water Intakes . . . . . 62

5.3 Calculate Surface Water Intakes . . . . . 66

5.4 Calculate Intakes From Other Exposure Pathways . . . . . 66

5.5 Combine Pathway-Specific Intakes to Yield Total Oral and Total Inhalation Intakes . . . . . 69

6. **STEP 4: TOXICITY ASSESSMENT** . . . . . 73

7. **STEP 5: RISK CHARACTERIZATION** . . . . . 76

7.1 Noncarcinogenic Effects . . . . . 76

7.2 Potential Carcinogenic Effects . . . . . 77

7.3 Uncertainties . . . . . 80

8. **DEVELOPMENT OF PERFORMANCE GOALS AND ANALYSIS OF RISKS FOR REMEDIAL ALTERNATIVES** . . . . . 83

8.1 Reevaluate Indicator Chemicals . . . . . 84

8.2 Identify Potential Exposure Pathways . . . . . 84

8.2.1 Determine Possible Sources of Chemical Release . . . . . 84

8.2.2 Determine Human Exposure Points . . . . . 86

8.2.3 Integrate Release Sources, Transport Media, Exposure Points, and Exposure Routes into Exposure Pathways . . . . . 86

8.2.4 Identify All Exposure Pathways for Each Exposure Point . . . . . 86

8.3 Determine Target Concentrations at Human Exposure Points . . . . . 86

8.3.1 Target Concentrations for Chemicals With Applicable or Relevant and Appropriate Requirements . . . . . 86

8.3.2 Target Concentrations for Chemicals Without Applicable or Relevant and Appropriate Requirements . . . . . 91

8.3.2.1 Apportion Total Potential Carcinogenic Risk Among Multiple Carcinogens . . . . . 93

8.3.2.2 Calculate Target Air Concentrations . . . . . 93

8.3.2.3 Calculate Target Drinking Water Concentrations. . . . . 95

8.3.3 Summarize Data . . . . . 95

8.4 Estimate Target Release Rates . . . . . 95

8.4.1 Predict Environmental Fate and Transport . . . . . 95

8.4.2 Summarize Data . . . . . 102

8.5 Assess Chronic Risk For Noncarcinogens . . . . . 102

8.6 Assess Potential Short-Term Health Effects of Remedial Alternatives . . . . . 102

## *CONTENTS (continued)*


**9. SUMMARIZING THE PUBLIC HEALTH EVALUATION** ..... **110**

9.1 Summarize the Baseline Public Health Evaluation ..... 110

9.2 Summarize Analysis of Remedial Alternatives ..... 114

**10 REFERENCES** ..... **115**

**APPENDICES**

Appendix A - Summary Tables for Chemical-Specific Data ..... 117

Appendix B - Detailed Procedures for Determining Toxicity Constants for Indicator Chemical Selection ..... 123

Appendix C - Memorandum of Understanding Between the Agency for Toxic Substances and Disease Registry and the United States Environmental Protection Agency ..... 164

**GLOSSARY** ..... **174**

## *LIST OF EXHIBITS*


| | | |
|---|---|---|
| 1-1 | Flowchart of the Superfund Public Health Evaluation Process | 6 |
| 1-2 | Continuum of Analytical Complexity for Superfund Public Health Evaluations | 7 |
| 3-1 | Overview of Step 1: Selecting Indicator Chemicals | 15 |
| 3-2 | Concentration and Toxicity Constant Units | 17 |
| 4-1 | Overview of Step 2: Estimating Exposure Point Concentrations | 29 |
| 4-2 | Illustration of Exposure Pathways | 30 |
| 4-3 | Common Chemical Release Sources At Sites in the Absence of Remedial Action | 32 |
| 4-4 | Typical Exposure Points for Chemical Releases from Hazardous Waste Sites | 35 |
| 4-5 | Selected Applicable or Relevant and Appropriate Ambient Requirements | 44 |
| 4-6 | EPA Ambient Water Quality Criteria (WQC) for Protection of Human Health | 46 |
| 4-7 | EPA Proposed MCLs and MCLGs | 54 |
| 4-8 | EPA Drinking Water Health Advisories | 56 |
| 5-1 | Overview of Step 3: Estimating Human Intakes | 62 |
| 5-2 | Standard Values Used in Daily Intake Calculations | 63 |
| 6-1 | Overview of Step 4: Assessing Toxicity | 74 |
| 7-1 | Overview of Step 5: Characterizing Risks | 77 |
| 8-1 | Flowchart of Performance Goals Process | 85 |
| 8-2 | Possible Chemical Release Sources Following Remedial Actions | 87 |
| 8-3 | Common Temporary Chemical Release Sources During Implementation of a Remedial Alternative | 107 |
| 9-1 | Worksheets that Should be Included in a Public Health Evaluation Summary | 111 |
| 9-2 | Summary of the Baseline Public Health Evaluation | 112 |
| 9-3 | Summary of the Public Health Evaluation of Remedial Alternatives | 113 |
| A-1 | Physical, Chemical, and Fate Data | 121 |
| A-2 | Half-Lives in Various Media | 127 |
| A-3 | Toxicity Data for Potential Carcinogenic Effects -- Selection of Indicator Chemicals Only | 133 |
| A-4 | Toxicity Data for Potential Carcinogenic Effects -- Risk Characterization | 137 |
| A-5 | Toxicity Data for Noncarcinogenic Effects -- Selection of Indicator Chemicals Only | 141 |
| A-6 | Toxicity Data for Noncarcinogenic Effects -- Risk Characterization | 149 |
| A-7 | Chemicals and Chemical Groups Having EPA Health Effects Assessment (HEA) Documents | 157 |
| B-1 | Rating Constants (RVe) for Noncarcinogens | 161 |
| B-2 | EPA Weight-of-Evidence Categories for Potential Carcinogens | 163 |

## *LIST OF WORKSHEETS*


| | | |
|---|---|---|
| 3-1* | Scoring for Indicator Chemical Selection: Concentrations and Koc Values in Various Environmental Media | 19 |
| 3-2 | Scoring for Indicator Chemical Selection: Toxicity Information | 20 |
| 3-3 | Scoring for Indicator Chemical Selection: Calculation of CT and IS Values for Carcinogenic Effects | 21 |
| 3-4 | Scoring for Indicator Chemical Selection: Calculation of CT and IS Values for Noncarcinogenic Effects | 22 |
| 3-5* | Scoring for Indicator Chemical Selection: Evaluation of Exposure Factors and Final Chemical Selection | 23 |
| 4-1 | Preliminary Release Source Analysis for Baseline Site Conditions | 33 |
| 4-2* | Matrix of Potential Exposure Pathways | 37 |
| 4-3 | Results of Release Quantification | 40 |
| 4-4* | Contaminant Concentrations at Exposure Points | 43 |
| 4-5* | Comparison of Applicable or Relevant and Appropriate Requirements to Estimated Exposure Point Concentrations | 51 |
| 4-6* | Comparison of Other Federal and State Criteria to Estimated Exposure Point Concentrations | 59 |
| 5-1 | Calculate Air Intakes | 64 |
| 5-2 | Calculate Ground-Water Intakes | 65 |
| 5-3 | Calculate Surface Water Intakes | 67 |
| 5-4 | Calculate Intakes from Ingestion of Contaminated Fish | 68 |
| 5-5* | Pathways Contributing to Total Exposure | 70 |
| 5-6* | Total Subchronic Daily Intake (SDI) Calculation | 71 |
| 5-7* | Total Chronic Daily Intake (CDI) Calculation | 72 |
| 6-1 | Critical Toxicity Values | 75 |
| 7-1* | Calculation of Subchronic Hazard Index | 78 |
| 7-2* | Calculation of Chronic Hazard Index | 79 |
| 7-3* | Calculation of Risk from Potential Carcinogens | 81 |
| 7-4 | Site-Specific Factors Increasing Uncertainty | 82 |
| 8-1 | Release Source Analysis | 88 |
| 8-2* | Matrix of Potential Exposure Pathways for Remedial Alternatives | 89 |
| 8-3 | Identify All Pathways for Exposure Points | 90 |
| 8-4 | Target Concentrations for Chemicals with Ambient Requirements | 92 |
| 8-5 | Apportioning Total Target Risk Among Multiple Potential Carcinogens | 94 |
| 8-6 | Calculation of Target Air Concentrations | 96 |
| 8-7 | Calculation of Target Drinking Water Concentrations | 97 |
| 8-8 | Apportionment of Target Oral Intake via Drinking Water and Fish Consumption | 98 |
| 8-9 | Calculation of Target Surface Water Concentrations Based on Fish Consumption | 99 |
| 8-10 | Final Target Concentrations of Potential Carcinogens | 100 |
| 8-11* | Summary of Exposure Pathways, Exposure Points, and Target Concentrations | 101 |
| 8-12 | Long-Term Target Releases | 103 |
| 8-13 | Summary Table: Exposure to Noncarcinogens | 104 |
| 8-14* | Summary Table: Chronic Intakes and Risks from Noncarcinogens | 105 |
| 8-15 | Matrix of Potential Short-Term Exposure Pathways | 108 |
| 8-16* | Summary Table: Subchronic Intakes and Risks | 109 |


* Designated for inclusion with public health evaluation summary.

# PREFACE

This manual establishes a framework for public health evaluation at Superfund sites and for development of health-based performance goals for remedial alternatives that are based on applicable or relevant and appropriate requirements of other laws, where available, or risk analysis techniques where those requirements are not available. These procedures were developed by EPA's Office of Emergency and Remedial Response (OERR) in conjunction with an Agency-wide Task Force, which included representatives from the program offices, the Office of Waste Programs Enforcement, the Office of Research and Development, the Office of Policy, Planning and Evaluation, and several Regional offices. The procedures provided in the manual are designed to conform to EPA's risk assessment guidelines (51 *Federal Register* 33992-34054, September 24, 1986). In addition, guidance developed by EPA's Office of Waste Programs Enforcement for endangerment assessments at enforcement-lead sites incorporates the procedures in this manual.

Public health evaluation is an important component of the remedial investigation (RI) and feasibility study (FS) phase of cleanup at Superfund sites. This procedures manual was developed to supplement Chapter 5 of the Guidance on Feasibility Studies Under CERCLA. That guidance describes **what** the public health evaluation process is, but not **how** to conduct it. In contrast, this manual provides detailed guidance on how to conduct the evaluation.

The Superfund Public Health Evaluation Manual has been developed for use by a diverse audience, including EPA regional staff, state Superfund program staff, federal and state remedial contractors, and potentially responsible parties. Individuals having different levels of scientific training and experience are likely to use the manual in designing, conducting, and reviewing public health evaluations. Because assumptions and judgments are required in many parts of the analysis, the individuals conducting the evaluation are key elements in the process. The manual is not intended for use by non-technical personnel to perform technical evaluations, nor to allow professionals trained in one discipline to perform the work of another. Rather, it is the responsibility of remedial project managers, using the manual as a guide, to match the scientific support they deem necessary with the appropriate resources at their disposal.

**Public health evaluation cannot be reduced to simple, "cookbook" procedures.** If all judgment could be removed from the process, undoubtedly the results from various sites would be far more consistent. In addition, state-of-the-art public health evaluation techniques have not been fully accepted by all scientists, and important chemical data are frequently unavailable. For instance, toxicity testing has not kept pace with the need for information on many chemicals, and procedures used in exposure assessment often require many assumptions. The universe of uncontrolled hazardous waste sites is both variable and complex, with each site posing a unique set of circumstances. It would be unrealistic to expect that all data necessary to determine precisely the health risks associated with every site will be available. Where data gaps necessitate making assumptions to conduct the public health evaluation for a site, the manual instructs that all such assumptions be clearly documented. The manual is designed to be flexible, allowing the use of professional judgment. It is not a "cookbook". Instead, it provides a systematic process for evaluating potential public health impacts at a site and for documenting and supporting the assessment, its assumptions, and its conclusions.

The manual provides a range of analytical procedures that may be needed at a particular site. It is up to the remedial project manager to determine the level of analysis required by using criteria discussed in this manual. In addition, the manual contains a series of worksheets to assist in performing the public health evaluation. The worksheets are not intended to drive the evaluation, but to provide a consistent format for reporting results. **The results of the public health evaluation should be presented within the appropriate section of the RI/FS report.**

Information gathered for the public health evaluation can be organized in the appropriate worksheets provided in the manual or in a comparable format. The information for the evaluation is important and

the worksheets are only a suggested format. Not all worksheets will be applicable to all sites; site-specific characteristics will determine which worksheets are relevant. Worksheets in this manual are filled in with illustrative examples to help explain the various procedures given in the text. These sample worksheets are for instructional purposes only; indicated values should not be construed as representing actual conditions.

The Superfund Public Health Evaluation Manual is divided into nine chapters. Some of the chapters are applicable to all sites, while some are applicable to a subset of sites. Chapter 1 is an overview of the entire Superfund public health evaluation framework. The second chapter provides background on Agency rules, policies, and guidance relevant to the public health evaluation process. Chapters 3 through 7 give procedures for the baseline public health evaluation, and Chapter 8 presents methods to formulate health-based performance goals for remedial alternatives. The final chapter provides guidance on how to summarize and present the results of the evaluation. Additional information related to the public health evaluation process is included in several appendices to the manual.

Two necessary supplements to this manual are: (1) a set of Health Effects Assessments (HEAs) for toxic chemicals typically found at uncontrolled hazardous waste sites, and (2) the Superfund Exposure Assessment Manual, which provides detailed methods for analyzing chemical releases from waste sites and assessing fate and transport in environmental media. The 58 available HEAs provide a rapid index of up-to-date toxicological information and should be used by EPA personnel and contractors to avoid inconsistency and duplication of effort. Other parties may also find the assessments useful and time-saving. The Agency is planning to develop additional HEAs for many commonly occurring chemicals found at Superfund sites. Copies of HEAs for specific chemicals are available through the National Technical Information Service (NTIS). Appendix A of this manual provides a list of chemicals with HEAs along with their NTIS publication numbers (Exhibit A-7) and also summarizes data from the HEAs necessary for the public health evaluation process (Exhibits A-4 and A-6).

Because toxicity data will change as new information becomes available, OERR will distribute updated summary tables on a regular basis. OERR has compiled the toxicity data from Appendix A along with values for key standards and criteria into a personal computer data base, PHRED--Public Health Risk Evaluation Database. PHRED has been designed to allow the user to both store and print selected fields of chemical data. The software package can be used on an IBM PC/XT/AT or compatible PC/XT/AT. The software consists of two disks: a program disk and a data disk. OERR plans to periodically update the data disk as new information becomes available. OERR also is developing a comprehensive document, the Superfund Risk Assessment Information Directory, to supplement the Superfund Public Health Evaluation Manual and other risk assessment guidance prepared by EPA. The directory will assist in decision-making by providing EPA officials with ready access to the most current risk assessment information. Such a compilation of sources, models, data bases, and individuals will make it possible to rapidly evaluate state-of-the-art risk assessment information, allow quick response to inquiries, reduce possible duplications of effort, and maximize consistency among sources of information.

At the time this manual was prepared for final publication, Congress had just passed a CERCLA reauthorization bill. Throughout this manual, where reauthorization is likely to affect the procedures for conducting public health evaluations, footnotes to the text have been included to describe the changes likely to result. Users should also be aware that citations in this manual to specific sections of CERCLA refer to CERCLA of 1980 (P.L. 96-510) and may not be valid for the reauthorization statute.

For further information concerning the Superfund Public Health Evaluation Manual and process contact the Director, Policy Analysis Staff, Office of Emergency and Remedial Response, U.S. EPA, 401 M Street, S.W., Washington, D.C. 20460.

# CHAPTER 1

## OVERVIEW OF THE SUPERFUND PUBLIC HEALTH EVALUATION PROCESS

The Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA) establishes a national program for responding to releases of hazardous substances into the environment. In addition, the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) establishes the process for determining appropriate remedial actions at Superfund sites[1]. Together, CERCLA and the NCP require that a remedial action selected for a Superfund site be cost-effective and that it be adequate to protect public health. The NCP, Guidance on Remedial Investigations under CERCLA (EPA, 1985b), and Guidance on Feasibility Studies under CERCLA (EPA, 1985a) require that selection of a cost-effective remedy be based on a comparison of alternatives that examines public health impacts, environmental impacts, technological and engineering feasibility, cost, and institutional factors. As a general rule, EPA will pursue remedies that attain or exceed[2] the requirements of applicable or relevant and appropriate federal public health or environmental laws. However, because of unique circumstances at particular sites, there may be alternatives that do not meet the standards of other laws, but that still provide protection of public health, welfare, and the environment. The Agency's most current toxicity data, documented in Health Effects Assessments (HEAs), along with other criteria, advisories and guidance will also be considered and may be used in fashioning remedies.

This manual supplements Chapter 5 of the feasibility study guidance, which provides interim guidance on conducting an evaluation of potential public health impacts at Superfund sites. The manual provides an approach that may be followed for analyzing public health impacts of remedial alternatives. EPA recognizes that other approaches may be equally valid. This manual covers the two key elements of a public health evaluation that should be addressed in any feasibility study, regardless of the approach that is used: (1) the baseline public health evaluation, and (2) the public health analysis of remedial alternatives. Section 104 of CERCLA authorizes taking a removal or remedial action to protect public health, welfare, or the environment when there is a release or substantial threat of release of any hazardous substance or when there is a release or substantial threat of release of any pollutant or contaminant that may present an imminent and substantial danger to the public health or welfare. A baseline public health evaluation is an analysis of site conditions in the absence of remedial action. It provides the remedial project manager with an understanding of the nature of chemical releases from the site, the pathways of human exposure, the degree to which such releases violate applicable or relevant and appropriate requirements and, in the absence of these requirements, a measure of the threat to public health as a result of releases. The information developed in the baseline evaluation provides input for developing and evaluating remedial alternatives. In addition, the baseline evaluation satisfies the NCP requirement to complete a detailed analysis of the no-action alternative, including an evaluation of public health impacts.

The baseline evaluation may also be applied in enforcement situations. Although the level of effort may be more rigorous in an enforcement-lead situation, the basic process is the same. For administrative and judicial enforcement actions under Section 106 of CERCLA, an endangerment assessment must be performed to justify the enforcement action. The endangerment assessment is the risk assessment process the Office of Waste Programs Enforcement (OWPE) uses to determine the magnitude and probability of actual or potential harm to public health, welfare, or the environment by the threatened or actual release of a hazardous substance. The endangerment assessment process is described in the Endangerment Assessment Guidance document signed by the Assistant Administrator of OSWER in the fall of 1985 and explained in the Endangerment Assessment Handbook released by OWPE in October, 1985. The Superfund Public Health Evaluation Manual provides methods employed in the endangerment assessment process and therefore has been made compatible with the requirement for conducting endangerment assessments for Superfund enforcement sites.

Development of performance goals for remedial alternatives is the second key phase of the public

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[1]CERCLA was reauthorized just before this manual was prepared for final publication. Several provisions of the reauthorization measure will affect the procedures described in this manual. In addition, the NCP will be revised as a result of reauthorization.

[2]For instance, the Agency might choose incineration as an alternative that exceeds what would be required by applicable standards because it is a more permanent and reliable solution than RCRA closure standards for land disposal facilities

health evaluation. The manual describes specific procedures for comparing health risks and developing performance goals for remedial measures. The process builds on information collected and evaluated in the baseline evaluation and closely follows the guidelines in the NCP and EPA's policy on CERCLA compliance with the requirements of other environmental statutes.[3]

**THE ANALYTICAL FRAMEWORK PROVIDED IN THE MANUAL IS A FLEXIBLE ONE.** While the manual provides a logical series of analytical procedures, these procedures are not intended to substitute for a well-reasoned thought process or scientific judgment. The manual recognizes that there is a minimum level of analysis and documentation that is necessary in any feasibility study, regardless of the particular approach used. The manual also recognizes that, depending on the number and type of substances present, the amount and adequacy of chemical, physical, and toxicological information known about the substances, the proximity of receptors, the effectiveness of available technology, and the characteristics of the exposure pathways, the remedial project manager will need to carefully consider the level of effort and amount of quantification needed to conduct an evaluation. The remainder of Chapter 1 explains these factors in more detail; however, judgment by the remedial project manager ultimately will determine the appropriate level of analysis.

It is also important to realize that not all of the components of the manual are appropriate to use at all sites. For example, an evaluation of the baseline situation must be conducted at all sites. However, the approach presented in Chapter 3 for selecting indicator chemicals is useful only at sites with a wide array of chemicals. Similarly, part of the performance goal development approach in Chapter 8 is useful only at sites where applicable or relevant and appropriate ambient concentration requirements are not available for all chemicals of interest.

## 1.1 DESCRIPTION OF PROCESS COMPONENTS

The public health evaluation framework presented in this manual has two major components:

- baseline public health evaluation,and
- development of performance goals for remedial alternatives.

As previously mentioned, an analysis of the baseline is a requirement for all remedial sites. Baseline public health evaluations can range from straightforward and uncomplicated to very detailed and complex. In addition to a baseline analysis, the remedial project manager should develop health-based performance goals, which will assist in development and refinement of appropriate remedial alternatives.

### *1.1.1 Baseline Public Health Evaluation*

The baseline public health evaluation covers a wide range of complexity, quantification, and level of effort, depending on numerous site factors. **The evaluation can be viewed as spanning a continuum of complexity and resource requirements.** The appropriate level of detail for a public health evaluation is a site-specific decision.

The baseline evaluation, as described in this manual, involves five steps. They are not a required set of procedures to be followed at all sites because some of the steps (or parts of steps) do not necessarily apply to some sites. As a first step in the process, indicator chemicals are selected, if needed, from among the list of contaminants known to be at the site. The procedure for selecting indicator chemicals, discussed in Chapter 3, incorporates chemical toxicity information, physical/chemical factors, and measured concentrations at the site. The second step in the evaluation, an assessment of exposure concentrations of the indicator chemicals is described in Chapter 4. Chemical releases are estimated and environmental fate and transport may be modeled to project exposure levels via air, ground water, surface water, or other pathways. Following the estimation of exposure concentrations, comparison to applicable or relevant and appropriate requirements (e.g., Federal drinking water standards) is made. The next step involves estimating human intakes. Standard assumptions for daily water and air intake, fish consumption, and other relevant factors are provided in Chapter 5 for use if site-specific information is unavailable. The fourth step of the process, presented in Chapter 6, involves an in-depth review of the toxicity of the indicator chemicals. Appendix A, which contains a listing of critical toxicity values for chemicals commonly occurring at uncontrolled hazardous waste sites, and EPA's Health Effects Assessment documents are important companions to

[3]EPA's CERCLA compliance with other environmental statutes policy is published as an appendix to the preamble of the NCP (50 *Federal Register* 47946-47950, November 20, 1985). The CERCLA reauthorization bill elevates the CERCLA compliance policy requirements to a statutory requirement.

Chapter 6. Finally, in Step 5 (Chapter 7), human health risks are characterized for potential carcinogens and for noncarcinogenic effects by combining the exposure and toxicity information developed in Steps 1 through 4.

### *1.1.2 Analysis of Remedial Alternatives and Development of Performance Goals*

The second component of the Superfund public health evaluation process is analysis and development of health-based performance goals for proposed remedial alternatives. This component is described in Chapter 8. Performance goals for source control[4] remedies will be based on applicable or relevant and appropriate design and operating requirements and best engineering judgment. Where soil removal is part of the remedial action, a risk-based approach can be used to determine the extent of removal. Performance goals for management of migration[5] alternatives will be based on applicable or relevant and appropriate ambient chemical concentration requirements, if available. Otherwise, a target carcinogenic risk range will be used to develop numerical performance goals. The emphasis of the performance goal procedure is to use techniques of risk analysis to assist in setting target levels of contaminant concentrations at exposure points (and for some remedial technologies, such as a waste treatment plant, to set target levels of contaminant discharge or emission). The public health evaluation for remedial alternatives is closely linked with other components of the feasibility study, especially the detailed technical evaluation.

EPA is developing additional guidance to aid in the development of remedial alternatives for certain specific situations (including guidance documents for cleanup of surface tank and drum sites and surface impoundments and for provision of alternate water supplies). These manuals will assist in the development of performance goals in many circumstances.

Exhibit 1-1 is a flowchart illustrating the major components of the Superfund public health evaluation process. The flowchart shows a possible sequence of activities but does not indicate which activities are applicable to which sites, an important topic that is discussed in the next section.

## 1.2 APPLICABILITY OF PROCESS COMPONENTS TO VARIOUS SITES

It should be apparent that not all of the components of the Superfund public health evaluation process described in Section 1.1 apply to all remedial sites. This manual establishes a generic framework that is broadly applicable across sites. As a consequence of attempting to cover a wide variety of sites, many of the process components, steps, and techniques described in the manual do not apply to some sites. In addition most of the components can vary greatly in level of detail. Obviously, determining which elements of the process are necessary, which are desirable, and which are extraneous is a key decision for each site. All components should not be forced into the assessment of a site, and the evaluation should be limited to the complexity and level of detail necessary to adequately assess risks. **It cannot be overemphasized that the manual is not a "cookbook" of procedures that must be followed without exception for each and every site. Rather, the manual establishes a public health framework that must be adapted to individual sites.** Although professional judgment and common sense are the ultimate inputs to deciding applicability and level of detail, the following paragraphs provide some guidance in this area.

Public health evaluation can be thought of as spanning a continuum of complexity, detail, and level of effort, just as sites vary in conditions and complexity. Exhibit 1-2 illustrates the concept of an analytical continuum and identifies some of the site-specific factors affecting level of effort that the remedial project manager must consider. These factors include:

- number and identity of chemicals present;
- availability of appropriate standards and/or toxicity data;
- number and complexity of exposure pathways (including complexity of release sources and transport media);
- necessity for precision of the results, which in turn depends on site conditions such as the extent of contaminant migration, proximity, characteristics and size of potentially exposed populations, and enforcement considerations (additional

[4]Source control remedies are those that remove or control the source of contamination at a site.

[5]Management of migration remedies are those that address substances that have already migrated away from the source.

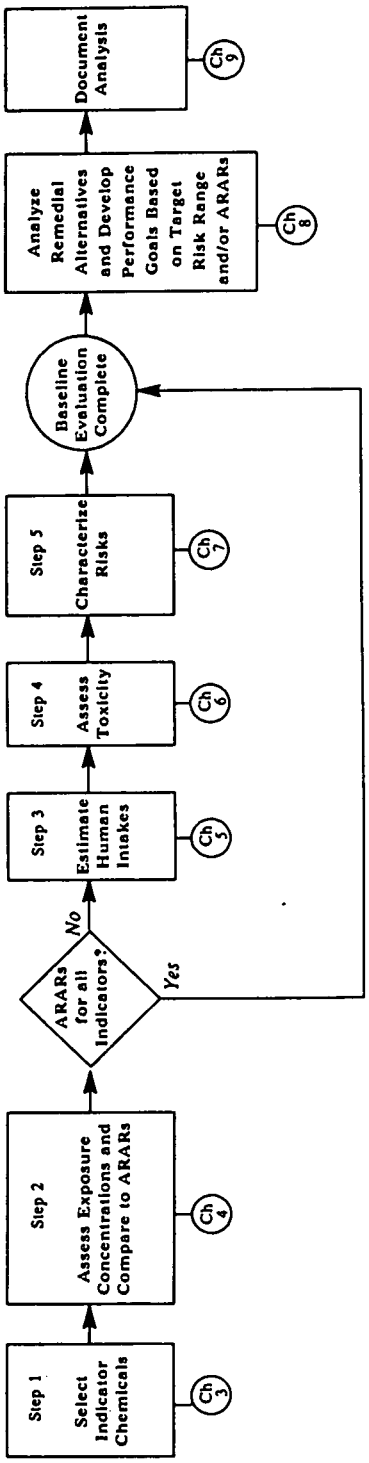


EXHIBIT 1-1
FLOWCHART OF THE SUPERFUND PUBLIC HEALTH EVALUATION PROCESS

EXHIBIT 1-2
CONTINUUM OF ANALYTICAL COMPLEXITY FOR SUPERFUND PUBLIC HEALTH EVALUATIONS

*Increasing Complexity/Level of Effort*

| | | |
|---|---|---|
| 1 or 2 chemicals | 10–15 chemicals | Many chemicals |
| Standards/toxicity data available | Standards/toxicity data mostly available | Standards/toxicity data missing for key chemicals |
| 1 significant exposure pathway | < 3 significant exposure pathways | > 3 significant exposure pathways |
| No ground water problem, or simple hydrogeology | Complex hydrogeology | Highly complex hydrogeology |
| 1 simple source | Complex sources | Multiple complex sources |
| Limited need for precision | Precision needed | Considerable precision needed |
| Substantial monitoring data available | Some monitoring data available; limited extrapolation required | Inadequate monitoring data; modeling required |

quantification may be warranted for some enforcement sites); and

- quality and quantity of available monitoring data[6].

Sites best represented by the descriptions toward the left of the continuum on Exhibit 1-2 correspond to a relatively low level of effort and analytical complexity, while sites corresponding to the descriptions toward the right are more complex and generally will require a greater level of effort. It is important to understand that the factors given on the continuum are largely independent. Thus, one factor may correspond to the need for a complex analysis while others correspond to a simple analysis (e.g., a site may have two chemicals with available standards and only one exposure pathway, via ground water, but may have a complex subsurface and need considerable precision). Although it is clearly a simplification, Exhibit 1-2 should assist in defining the appropriate level of quantitative analysis for a site.

[6]All site monitoring data must be subjected to appropriate quality control-quality assurance programs. Lack of acceptable data may by necessity limit the amount of data available for the public health evaluation, and therefore may limit the scope of the evaluation.

# CHAPTER 2

# BACKGROUND: AGENCY RULES, POLICIES, AND GUIDELINES

To understand the context of the public health evaluation process, it is important to be familiar with EPA rules, policies, and guidelines relevant to remedial actions at Superfund sites. In this chapter, the most important related rules, policies, and guidelines are summarized and references for further information are provided.

## 2.1 The National Oil and Hazardous Substances Pollution Contingency Plan (NCP)

The NCP[7] is a regulation that provides a framework for implementing the response powers and responsibilities established under CERCLA. Subpart F of the NCP outlines the hazardous substance response process and includes provisions for both removal and remedial actions. Federal and state agencies and private parties responsible for preparing feasibility studies for Superfund remedial sites should be familiar with the NCP. The most recent version of the NCP was published on November 20, 1985 (EPA, 1985c)[8]. A copy can be obtained from EPA's Office of Emergency and Remedial Response (OERR), U.S. EPA CERCLA Docket Clerk, 401 M Street, SW, Washington, DC 20460.

The NCP sets forth a five-step remedial response process:

- *Site discovery or notification*: Releases of hazardous substances, pollutants, or contaminants identified by federal, state, local government agencies, or private parties are reported to the National Response Center. Upon discovery, such potential sites are screened to identify release situations warranting further remedial response consideration. These sites are entered into the Emergency and Remedial Response Inventory System (ERRIS). This computerized system serves as a data base of site information and tracks the change in status of a site through the response process.
- *Preliminary assessment and site inspection (PA/SI)*: The preliminary assessment involves collection and review of all available information and may include off-site reconnaissance to evaluate the source and nature of hazardous substances present and to identify the responsible party(s). Depending on the results of the preliminary assessment, a site may be referred for further action. Site inspections routinely include the collection of samples and are conducted to determine the extent of the problem and to obtain information needed to determine whether a removal action is needed at the site or whether the site should be considered for inclusion on the National Priorities List (NPL).
- *Establishing priorities for remedial action*: Sites are scored using the Hazard Ranking System and data from the PA/SI. This scoring process is the primary mechanism for determining the sites to be included on the NPL, which identifies sites eligible for Superfund-financed remedial action.
- *Remedial investigation/feasibility study (RI/FS)*: The RI/FS process is the framework for determining appropriate remedial actions at sites on the NPL. Remedial investigations are conducted to obtain information needed to identify, evaluate, and select cleanup alternatives. The feasibility study is the actual analysis of alternatives based on technological, public health, institutional, cost, and environmental factors. The RI/FS process was developed to identify the most appropriate, cost-effective remedy for a site.
- *Remedial action design and construction*: The detailed design of the selected remedial action is developed and then implemented.

The Superfund Public Health Evaluation Manual provides detailed guidance for the public health analysis that is part of the RI/FS process.

## 2.2 Guidance for Remedial Investigations and Feasibility Studies

As noted in Section 2.1, the NCP requires that a remedial investigation and feasibility study be conducted for sites listed on the National Priorities List. EPA has developed and published guidance for both the remedial investigation (EPA, 1985b) and feasibility study (EPA, 1985a). The RI/FS guidance provides the context into which the public health evaluation fits. The remedial investigation and feasibility study are described briefly below. For more details, refer to the guidance documents referenced above.

The Guidance for Remedial Investigations Under CERCLA is intended to provide a detailed structure for field studies to support remedial decisions under

[7]Part 300, Chapter 40 of the Code of Federal Regulations (40 CFR 300).

[8]Reauthorization of CERCLA will result in revision of the NCP.

CERCLA. The remedial investigation emphasizes data collection and site characterization and is conducted concurrently with the feasibility study. The remedial investigation also supports remedial alternative evaluation and design through bench and pilot studies.

The initial activity in the remedial investigation is scoping. The scoping effort includes the collection and evaluation of existing data, identification of remedial investigation objectives, and identification of general response actions for the feasibility study. A preliminary determination of which federal environmental and public health requirements are applicable or relevant and appropriate to the site is also made as a part of the scoping effort.

Several activities supporting the remedial investigation may require preparation of specific plans or implementation of specific procedures. These include preparing a sampling plan; identifying data management procedures; planning for worker health and safety needs; and identifying and reviewing institutional issues arising from federal, state, and local regulations, policies, and guidelines.

Site characterization is the focal point of the remedial investigation and involves collection and analysis of data needed for various types of assessments in the feasibility study. Because site data and complexity vary, a multilevel approach to data collection is recommended, including problem identification and scoping, followed by problem quantification, followed if necessary by further problem quantification and detailed investigation. The focus, data needs, and data evaluations conducted at each level of the investigation are described in the guidance document.

The Guidance for Feasibility Studies Under CERCLA is intended to provide a detailed structure for identifying, evaluating, and selecting remedial action alternatives under CERCLA. The feasibility study process begins with development of specific alternatives, based on the general response actions identified in the remedial investigation. Remedial technologies are screened for their applicability to the site. Technologies considered appropriate are then combined to form alternatives, which are screened on the basis of public health and environmental concerns and order-of-magnitude costs.

Alternatives that pass the screening process undergo detailed analyses to provide site decision-makers with information for selecting an alternative that is cost-effective. The guidance document describes methods for engineering, institutional, public health, environmental, and cost analyses. The engineering analysis evaluates constructability and reliability to ensure the technical feasibility of alternatives. The institutional analysis examines alternatives in terms of the federal, state, or local requirements, advisories, or guidance. The public health evaluation, for which this manual provides more detailed guidance, assesses potential health risks if no action is taken and for remedial alternatives that are developed. The environmental analysis includes assessment of adverse environmental impacts if no action is taken and the short- and long-term effects of the alternatives. The cost analysis examines capital and operating costs of each alternative.

Once the detailed analyses are conducted, the information is organized to compare findings of the evaluations for each alternative. The objective of this summary is to ensure that important information is presented in a concise format so that the decision-maker can choose the remedy that provides the best balance of human health and environmental protection, engineering reliability, and cost.

Although there are separate guidance documents, the remedial investigation and the feasibility study are interdependent. The activities comprising the remedial investigation and feasibility study are generally performed concurrently rather than sequentially. The remedial investigation emphasizes data collection and site characterization, whereas the feasibility study emphasizes data analysis and evaluation of alternatives.

## 2.3 CERCLA Compliance with Other Environmental Statutes

Section 104 of CERCLA requires that wastes taken off-site during a remedial action be disposed in a facility approved under Subtitle C of the Resource Conservation and Recovery Act (RCRA). CERCLA, however, does not address the requirements of other federal environmental and public health laws (e.g., Clean Water Act, Toxic Substances Control Act) in conducting on-site response actions[9].

The NCP requires that remedies selected for on-site CERCLA response actions attain or exceed

[9]The CERCLA reauthorization bill specifically requires compliance with other federal and state environmental laws; some details of EPA's current compliance policy will likely be changed as a result of reauthorization.

applicable or relevant and appropriate environmental and public health requirements unless one of five specific situations exists[10]. Other federal criteria, advisories, guidances, and state standards should also be considered in fashioning CERCLA remedies and, if pertinent, should be used. For on-site actions (i.e., where wastes are treated, stored, or disposed on-site), permits (e.g., federal/state RCRA or NPDES) are not required for CERCLA response actions; however, all appropriate permits are required for off-site action.

The CERCLA compliance with other environmental statutes policy is critical to an evaluation of remedial alternatives and therefore must be reviewed before remedial options are developed. A copy of the policy is published as an appendix to the preamble of the NCP (50 *Federal Register* 47946-47950, November 20, 1985). To the extent that it is both possible and appropriate, at least one remedial alternative should be developed as part of the feasibility study in each of the following categories:

- alternatives for off-site treatment or disposal;
- alternatives that *attain* applicable or relevant and appropriate Federal public health or environmental requirements;
- alternatives that *exceed* applicable or relevant and appropriate Federal public health or environmental requirements;
- alternatives that *do not attain* applicable or relevant and appropriate Federal public health or environmental requirements, but that will reduce the likelihood of present or future threat from the hazardous substances and that provide significant protection to public health, welfare, and the environment; and
- the no-action alternative.

The CERCLA compliance policy provides a list of requirements that are potentially applicable or relevant and appropriate (i.e., must be used in the development of alternatives) and other federal criteria, advisories, guidances, and state standards that are to be considered and may be used if pertinent. In cases where requirements are deemed applicable or relevant and appropriate to remedial actions developed and considered during the feasibility study process, they should be applied carefully in the public health evaluation, with consideration given to the economic and technical factors used to establish the requirement that may be significantly different from circumstances at a specific Superfund site. For instance, drinking water maximum contaminant levels (MCLs) are developed using certain economic considerations that may not be appropriate to some Superfund sites. In addition, various requirements may be applicable at different points in the exposure pathway.

This manual provides guidance for incorporating applicable or relevant and appropriate requirements into the public health evaluation process. Although RCRA design and operating standards are clearly important requirements to consider in remedial design at Superfund sites, they are not discussed at length in this manual because they do not provide ambient concentration levels for chemicals. This manual focuses on ambient chemical concentration standards and criteria that can be used for comparison to baseline conditions and to set quantitative performance goals. The Office of Emergency and Remedial Response is also preparing further guidance for implementing the compliance policy. That guidance, the Manual on CERCLA Compliance with Other Environmental Statutes, will explain specifically how applicable or relevant and appropriate requirements under other laws should be identified and used in the design of remedial alternatives and will also include case studies to illustrate different situations. The manual is currently in draft form. For further information contact the U.S. EPA CERCLA Docket Clerk, 401 M Street, SW, Washington, DC 20460.

## 2.4 Agency Policy for Planning and Implementing Off-Site Response Actions

In 1985 EPA adopted a policy for Superfund response actions involving off-site storage, treatment, or disposal of CERCLA hazardous substances[11].

The policy requires that certain criteria must be met in selecting a hazardous waste management facility to receive CERCLA hazardous substances. The facility must have either a permit or interim status

[10]The five exceptions are fund balancing, technical impracticality, unacceptable environmental impacts, interim measures, and enforcement actions when strong public interest calls for expedited cleanup and litigation probably would not result in a desired response (see 40 CFR 300.68(i)(5)).

[11]"Procedures for Planning and Implementing Off-Site Response Actions," Memorandum from Jack W. McGraw, Acting Assistant Administrator for Solid Waste and Emergency Response to EPA Regional Administrators, May 6, 1985.

under RCRA. A RCRA compliance inspection must have been performed within six months prior to receiving the hazardous substances. No Superfund hazardous substances may be taken off-site to a RCRA facility if the facility has significant RCRA violations or other environmental conditions that affect the satisfactory operation of the facility *unless* the owner or operator commits to correct the problem and disposal occurs within the facility only at a new or existing unit in compliance with RCRA requirements[12]. In addition, that new or existing unit must not contribute in any significant way to adverse conditions at the facility. The policy also establishes a preference for response actions that use treatment, reuse, or recycling rather than land disposal.

Copies of the procedures and further information are available from the U.S. EPA CERCLA Docket Clerk, 401 M Street, SW, Washington, DC 20460.

## 2.5 Agency Guidelines on Risk Assessment

EPA has adopted guidelines to improve consistency in Agency risk assessments. The guidelines address five areas: carcinogenicity, mutagenicity, reproductive effects, exposure assessments, and assessment of chemical mixtures (EPA, 1986a,b,c,d, and e). Guidelines for assessment of other systemic effects are currently in preparation. The risk assessment guidelines were used in development of the procedures described in this manual and of the supporting toxicity data provided in the Health Effects Assessment Documents. For further background scientific information, users should obtain and review these guidelines and their support documents. Copies are available from EPA's Office of Health and Environmental Assessment, Technical Information Staff, 410 M Street, SW, Washington, DC 20460.

## 2.6 Memorandum of Understanding Between EPA and The Agency for Toxic Substances and Disease Registry

EPA and the Agency for Toxic Substances and Disease Registry (ATSDR) have developed a Memorandum of Understanding (MOU) to define and coordinate joint and respective responsibilities under CERCLA, Executive Order 12316[13], and

the NCP. The MOU establishes policies for conducting response and non-response health activities related to releases of hazardous substances. A copy of the MOU is provided in Appendix E[14].

Under the current MOU, ATSDR's major responsibilities include assessment of populations with current or potential exposure to waste sites, development of health advisories, and follow-up investigation of populations to evaluate future health effects. As defined by the MOU, EPA's major health-related responsibilities are risk assessment and risk management. Risk assessment is defined as a qualitative/quantitative process conducted to characterize the nature and magnitude of potential risks to public health from exposure to hazardous substances, pollutants, or contaminants released from specific Superfund sites. The framework for such EPA public health evaluations is given in this procedures manual.

Where ATSDR is involved, EPA and ATSDR are to coordinate any health-related activities during the remedial process. Health assessments, health advisories, and other information developed by ATSDR should be considered by the public health evaluation team at Superfund sites, and appropriate data and conclusions should be incorporated into the public health evaluation process and reports. Likewise, EPA public health evaluations should be made available to ATSDR for consideration during their analyses. It is EPA's responsibility to incorporate both the results of risk assessments developed as part of the public health evaluation process and any ATSDR analyses into risk management determinations of extent of remedy.

At sites where ATSDR is involved, its staff should be consulted for assistance in interpretation of human health data, such as clinical or epidemiologic survey information. The MOU clearly states that if human subjects testing is necessary, ATSDR will be responsible for such testing and will coordinate it with EPA.

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[12]Under the reauthorization bill, CERCLA wastes transported off-site may only be disposed in a non-leaking waste disposal unit of a permitted RCRA facility. In addition the facility must be in compliance with RCRA corrective action requirements for any other units that are found to be releasing wastes into the environment.

[13]E.O. 12316 delegates to EPA the primary response authority under CERCLA section 104 relating to release of hazardous substances, pollutants, or contaminants. E.O. 12316 delegates to the Department of Health and Human Services authorities for co

[14]Under reauthorization, ATSDR's health-related responsibilities will be expanded significantly. As a result, a new agreement between EPA and ATSDR will be developed.

Under reauthorization ATSDR will be required to conduct health assessments for all sites on or proposed for addition to the NPL, according to a statutorily mandated schedule. The purpose of these ATSDR health assessments is to assist in determining whether actions should be taken to reduce human exposure to hazardous substances and whether additional information (e.g., epidemiologic studies, disease registries, health surveillance programs) on human exposure and associated health risk is needed. Although both EPA and ATSDR are responsible for developing independent analyses related to public health, EPA is solely responsible for making risk management decisions based on these analyses. Currently, EPA and ATSDR are working together to define the roles and responsibilities of the two agencies under reauthorization and the relationship between EPA public health evaluations and ATSDR health assessments. In addition, a procedures document to better integrate ATSDR health assessments in the RI/FS process is being developed.

# CHAPTER 3
# STEP 1: SELECTION OF INDICATOR CHEMICALS

The baseline public health evaluation process consists of five steps, which are shown in the flowchart given earlier in Exhibit 1-1. These steps are discussed individually in Chapters 3 through 7. As emphasized in Chapter 1, not all steps will be needed at all sites because of variability in site conditions.

Prior to initiating these five steps, available site data relevant to detailed public health evaluation should be gathered, organized, and reviewed. Among the types of information to be collected are site background data, disposal history (and records, if available), types of remedial actions being considered, on-site and off-site chemical analysis data, site characterization data necessary for exposure assessment (e.g., topography, hydrogeology), information on local human populations, and any human body burden and health effects data (unlikely to be available at many sites). Data sources will include preliminary assessments and reports, site inspection reports, Field Investigation Team (FIT) reports, remedial investigation scoping documentation, analytical data and reports available from ongoing site characterization (RI) and alternatives screening (FS) activities, and ATSDR health assessments.

The next task of the public health evaluation is to determine whether indicator chemicals need to be selected for the site. The indicator chemical selection procedure described here is designed to identify the "highest risk" chemicals at a site so that the public health evaluation is focused on the chemicals of greatest concern. In general, if less than 10 to 15 chemicals are actually identified at a site, this indicator selection step is not necessary. In such cases, proceed to Chapter 4 and evaluate all of the chemicals at the site. This "shortcut" will be especially useful when only a very few chemicals are present at a site and a simple quantitative analysis is appropriate. However, remedial investigation sampling at hazardous waste sites often demonstrates the presence of a large number of chemical substances. In such instances, conducting a public health evaluation that includes all the identified chemicals may be unnecessarily time-consuming. To avoid unnecessary effort, the Superfund process is based on selected indicator chemicals that pose the greatest potential public health risk at a site. Such indicator chemicals must be chosen carefully so that they represent the most toxic, mobile, and persistent chemicals at the site, as well as those present in the largest amounts (i.e., the "highest risk" chemicals).

Step 1 of the baseline analysis (i.e., analysis of a site under an assumption of no remedial action) is selection, if necessary, of a subset of the chemicals present at a site as indicator chemicals. An outline of this step is presented in Exhibit 3-1, and procedures for the selection are given in the remainder of this chapter. The toxicity data required to complete the selection procedure for many commonly found chemicals are listed in Exhibits A-3 and A-5 in Appendix A. Appendix B documents the methods used to derive the toxicity data given in Exhibits A-3 and A-5.

Two important factors for ranking chemicals in the indicator chemical selection process are their measured concentrations at the site and their toxicity. Additional factors to be considered include physical and chemical parameters related to environmental mobility and persistence. The indicator chemicals selected for the baseline public health evaluation by following the procedures in this chapter will be reviewed later for applicability to the remedial alternatives. Because of concerns related to treatability and additional exposure pathways, more chemicals may need to be assessed in the analysis of remedial alternatives (see Section 8.1).

It is emphasized that the indicator chemical selection process presented here is not supposed to contravene professional judgment. If, after completing the procedures given in this chapter, certain chemicals considered to be potentially significant are not selected, do not hesitate to include them. Simply amend Worksheet 3-5 with an explanation of the reasoning and why this process did not identify them. It is not intended that the indicator chemical selection process exclude any chemical that may cause significant human or environmental harm. Rather, the intent of the process is to ensure that all chemicals posing a significant risk to human health are addressed and to focus the public health evaluation on the primary chemicals of concern.

## 3.1 Develop Initial List of Indicator Chemicals

The first task in the indicator chemical selection process is development of an initial indicator chemical list, which is based principally on chemical toxicity information, site concentration data, and

**Identify Chemicals Present at a Site**

**Determine Representative Concentrations from Site Monitoring Data**

**Calculate Indicator Scores Based on Maximum and Representative Concentrations and Route-Specific Toxicity Data**

**Select Indicator Chemicals Based on Indicator Scores and Physical/Chemical Property Data**

EXHIBIT 3-1
OVERVIEW OF STEP 1: SELECTING INDICATOR CHEMICALS

environmental mobility as reflected in $K_{OC}$[15] (the organic carbon partition coefficient) values. $K_{OC}$ is considered to account for the possibility of substances leaching out of the soil and being introduced into surface and ground water. The initial list will eventually be pared down using additional factors to develop a final indicator list. The indicator chemical selection process is designed for sites with large numbers of chemicals where consideration of all physical, chemical, and concentration information at one time is too cumbersome. If only a moderate number of chemicals are present at a site, all toxicity, chemical, and physical factors may be considered simultaneously.

Each chemical detected at the site above local background levels is scored. If, based on recent monitoring data in the site vicinity, it is clear that levels of certain chemicals do not exceed local background concentrations, and there is no known source (e.g., intact drums, waste pile) at the site, these chemicals may be excluded from the evaluation. However, determining background may be

[15]A chemical's $K_{oc}$ is being used as an estimator of environmental mobility. In general, chemicals with high values have correspondingly high bioconcentration factors, whereas chemicals with low values will tend to be leachable from soil and mobile in ground water. A more detailed discussion of Koc is presented later in the text of this chapter.

difficult. If there is a question about what background is or the relation of a chemical concentration to background, report these doubts but do not exclude the chemical from the evaluation.

The following algorithm is used to score each chemical measured at the site:

$$IS_i = \sum_{j=1}^{3} (C_{ij} \bullet T_{ij})$$

where

$IS_i$ = indicator score for chemical $i$ (unitless)

$C_{ij}$ = concentration of chemical $i$ in medium $j$ at the site based onmonitoring data (units must be mg/l in water, mg/kg in soil, or mg/m$^3$ in air)

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

Concentration values used in this equation for a given chemical should be representative of all available site monitoring data that have been QA/QC validated. Toxicity constants (T values) are derived for each environmental medium and two types of toxic effects (carcinogenicity and other chronic effects). Exhibit 3-2 lists for each medium of concern the units of concentration that should be used to express exposure levels, the exposure route (e.g., ingestion or inhalation), and the corresponding toxicity constants and their units. In all cases, toxicity constant units are the inverse of their respective concentration units so that indicator scores (C×T) will always be unitless. Essentially, the indicator score is a ratio between measured concentration and a toxicity-based concentration benchmark that is used to rank the site chemicals.

Toxicity constants for noncarcinogens (Tn) are derived from the minimum effective dose (MED) for chronic effects, a severity of effect factor, and standard factors for body weight and oral or inhalation intake (e.g., 70 kg body weight, 2 liters/day of drinking water, 20 cubic meters/day of air). Toxicity constants for potential carcinogens (Tc) are based on the dose at which a 10 percent incremental carcinogenic response is observed ($ED_{10}$) and the same standard intake and body weight factors. The intake factor for soil toxicity constants is based on an assumption of 100 milligrams of soil consumed per day for 2- to 6-year-olds (EPA, 1984).

Toxicity constants, T, are medium-specific. The toxicity constant for use with drinking water concentrations is referred to as $^{w}T$, whereas one for concentrations in air is $^{a}T$, and one for concentrations in soil is $^{s}T$. Values for toxicity constants ($^{w}T$, $^{a}T$, and $^{s}T$) for a number of compounds are given in Appendix A. Appendix B describes in detail the methods used for calculating the toxicity constants in Appendix A. The data base for this procedure is adopted from the supporting documentation for the Superfund Reportable Quantities rulemaking. Its use for selection of indicator chemicals at Superfund sites will be reconsidered if another more appropriate data base becomes available for ranking the toxicity of a large number of chemicals.

Because of probable differences in dose-response mechanisms (non-threshold vs. threshold), potential carcinogens (PCs) and noncarcinogens (NCs) are scored and selected independently. Indicator scores for carcinogens and noncarcinogens Indicator scores for carcinogens and noncarcinogens are **not on comparable scales and should never be compared.**

To begin the indicator selection process, use Worksheet 3-1 to list all compounds found at the site. For each compound record its Chemical Abstract Service (CAS) number and $K_{oc}$ value from Appendix A. Record the maximum and minimum observed concentrations as well as a "representative" concentration for each compound. Determination of the representative concentration should be based on an analysis of all the site monitoring data, with the goal being to represent long range trends at potential human exposure points. It may be appropriate to use a geometric or arithmetic mean of some or all of the samples as the most representative concentration, or it may be more appropriate to choose a concentration that reflects a time trend occurring at the site. **Use the monitoring data most relevant to a public health evaluation at the site.** For example, simply averaging upgradient and downgradient well results would usually be inappropriate. To get a concentration that represents the concen- tration of chemicals in a ground-water plume, the mean should generally be calculated based on samples where the chemical has been detected, not including samples below detection limits. Focus on data from locations nearest to expo- sure points. Also, consider detection frequency in determining a representa- tive concentration, giving relatively less weight to chemicals detected infrequently. Be sure to be consistent for all chemicals within each medium so that the selection process is not biased (i.e., do not choose a geometric mean concentration for one chemical and an arithmetic mean for a second).

| Environmental Medium | Environmental Concentration Units | Exposure Route | Toxicity Constant | Toxicity Constant Units |
|---|---|---|---|---|
| Water | mg/l a/ | ingestion | $^{w}T$ | $(mg/l)^{-1}$ |
| Soil | mg/kg b/ | ingestion | $^{s}T$ | $(mg/kg)^{-1}$ |
| Air | $mg/m^3$ c/ | inhalation | $^{a}T$ | $(mg/m^3)^{-1}$ |

a/ Milligrams per liter of drinking water.

b/ Milligrams per kilogram of soil.

c/ Milligrams per cubic meter of air.

EXHIBIT 3-2
CONCENTRATION AND TOXICITY CONSTANT UNITS

Indicate on the worksheet the basis for the representative concentration chosen and note any assumptions or additional information required to use this information. If there are concerns about use of these concentrations, note them. For example, even if the concentrations adequately represent the quantitative monitoring information available, they may not seem to reflect the reality of a 450,000-gallon lined lagoon whose liner may fail at any time. Another concern related to representativeness of monitoring data is detectability. If there is reason to believe that a chemical is present but is not being detected by the sampling and analytical protocols used, be sure to note this also. If a chemical is considered sufficiently important, it may be chosen as an indicator chemical regardless of its concentration. Also note any chemicals that were identified analytically but for which no quantitative data are available.

After completing Worksheet 3-1, refer to Appendix A to determine each compound's toxicologic class (potential carcinogen (PC) and/or noncarcinogen (NC)), severity rating value (noncarcinogens) or weight-of-evidence rating (carcinogens), and appropriate toxicity constants ($^{w}T$, $^{s}T$, and $^{a}T$). Enter this information on Worksheet 3-2. If a chemical is designated as both a PC and NC, complete the indicator scoring procedure for it in both toxicologic classes. Generally, compounds not listed in Appendix A or with insufficient data for indicator scoring should be classified as unknown under toxicologic class.[16] These substances should be listed in the final report to provide an indication of the uncertainty associated with omitted chemicals and to assist headquarters

[16]Users should be aware that a few chemicals (e.g., dichloromethane) have the necessary toxicity values for risk characterization (Exhibits A-4 and A-6) but not for indicator selection (Exhibits A-3 and A-5). This results from the use of different toxicity data bases for deriving indicator selection parameters and risk characterization parameters. Therefore, be sure not to exclude chemicals simply because they lack the toxicity constants necessary for indicator selection.

personnel in identifying data gaps. If you have reason to believe that these compounds may be significant at your site, contact the Environmental Criteria and Assessment Office (ECAO), U.S. EPA, 26 W. St. Clair Street, Cincinnati, Ohio 45268, for guidance in estimating the necessary toxicity constants.

The next task is to calculate IS values for each chemical. List all potential carcinogens on Worksheet 3-3 and all noncarcinogens on Worksheet 3-4.Calculate C times T (C•T) for each medium for each chemical, using both the peak and the representative concentrations. To develop an indicator score (IS), sum the C•T values across media. If a compound is present in both ground and surface water use only the higher C•T value for these two media (i.e., do not include both in the IS score). This approach for water makes the conservative assumption that all drinking water is obtained from the source giving the higher C•T value. Rank the compounds on these two worksheets separately on the basis of the indicator scores.

Record on Worksheet 3-5, in rank order based on IS values, the top-scoring 10 to 15 compounds from both Worksheet 3-3 (potential carcinogenic effects) and Worksheet 3-4 (noncarcinogenic effects). Compare the list of chemicals on Worksheet 3-5 to the chemicals identified with either an H or an L on Worksheet 3-1 (H indicates one of 10 chemicals with highest $K_{OC}$ values, L indicates one of 10 with lowest). If an important exposure scenario at the site involves consumption of contaminated fish and none of the 10 chemicals designated with an H made it onto the initial list, consider placing one or more of them onto that list. Also, if exposure via ground-water contamination is a concern and none of the 10 chemicals designated with an L made it onto the initial indicator list, consider enlarging the list to include one or more of these chemicals.

The list of 20 to 30 compounds on Worksheet 3-5 is the initial list of indicator chemicals from which the final set of indicators is selected for the site. In most cases the initial list and final selection should be based on representative concentrations, although indicator scores based on maximum or peak concentrations may be used to modify the selection. There is no predetermined number of indicator chemicals appropriate for all sites; between 5 and 10 chemicals would be a manageable number and may be sufficient for most sites. However, if a very large number of chemicals has been detected at a site, it may be wise to select more indicators. The number and identity of indicator chemicals selected is a site-specific decision that must be made and documented for the site. Guidance for making the final selection is given in the following section.

## 3.2 Select Final Indicator Chemicals

Final selection of indicator chemicals is not based on a numerical ranking algorithm or set of precise decision rules. Instead, there are several chemical-specific factors to consider, plus a few general selection rules. The initial factor to consider is the relative indicator scores (IS) of the chemicals. The IS, based in part on concentrations at the site, has already been used to rank chemicals for the initial indicator chemical list (Worksheet 3-5). In general, higher ranking chemicals based on representative IS values should be selected toxicologic class (PC or NC). This rule can be modified, however, on the basis of the additional selection factors discussed below. Consideration should also be given to the quantity of chemicals found at the site. Some pollutants may not appear in very high concentration but may be distributed throughout the entire site, adding up to a substantial total quantity.

Because values of IS for PC and NC are not directly comparable, the IS value is not relevant to a determination of the relative number of PC and NC to select. In fact, this determination is subjective. Always include at least some of both classes, and consider the relative number of PC and NC present at the site (e.g., if 90 percent of the chemicals at a site are noncarcinogens, probably more noncarcinogens than carcinogens should be selected). In any case, include several top-ranked (by IS) PC and NC as indicator chemicals unless there are extremely strong site-specific reasons for doing otherwise.

Although IS is the initial selection factor, several additional factors are also important. These factors include five important chemical properties related to exposure potential: water solubility, vapor pressure, Henry's Law constant, organic carbon partition coefficient ($K_{OC}$), and persistence in various media. High or low values of any of these factors for a chemical found at a site may produce a high future exposure potential and may warrant inclusion of a particular chemical in the list of indicator chemicals despite a low IS score. Values for these factors are given in Appendix A for many chemicals. Record appropriate values for the preliminary indicator chemicals listed on Worksheet 3-5. For chemicals not listed in Appendix A, determine values using sources listed in Appendix A or other standard references. Also, estimation techniques are available for many physical/chemical parameters and have been summarized in Lyman et al. (1982) and Mabey et al. (1982). Use of estimation techniques in the absence of experimental data is encouraged, as long as the procedures are documented.

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 3-1

SCORING FOR INDICATOR CHEMICAL SELECTION: CONCENTRATIONS AND Koc VALUES IN VARIOUS ENVIRONMENTAL MEDIA

| Chemical (CAS No.) | Koc Value | Ground Water (mg/l) Range | Repres a/ | Ref b/ | Surface Water (mg/l) Range | Repres a/ | Ref b/ | Soil (mg/kg) Range c/ | Repres c/ | Ref b/ | Air (mg/m3) Range | Repres | Ref b/ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Arsenic (7440-38-2) | -- | <.010-.46 | .075 | B.112 | <.010 | - | B.112 | 1.7-36 | 7 | A.22 | - d/ | - | - |
| Tetrachloroethylene (127-18-4) | 364 | BDL e/-67 | 3.2 | A.18 | BDL-.012 | .003 | A.18 | BDL-13,000 | 2 | A.23 | - | - | - |
| Beryllium (7440-41-7) | -- | <.005-.05 | .006 | B.112 | <.005 | - | B.112 | <.25-2.6 | 0.6 | A.22 | - | - | - |
| 1,2,4-Trichlorobenzene (120-82-1) | 9200 | BDL-1.2 | .11 | A.18 | BDL-.026 | .002 | A.18 | BDL-210 | 15.8 | A.23 | - | - | - |

a/ Mean of reported values used as representative concentration for surface and ground water; zero used for all values reported as below detection limit.
b/ A = Feasibility Study document, B = Remedial Investigation document. Page numbers follow document designation.
c/ Soil concentration range is across surface, subsurface soils, and sediments; mean of the surface soil values used as representative concentration; zero used for all values reported as below detection limit.
d/ No data reported for this medium.
e/ BDL = below detection limits.

INSTRUCTIONS

1. Write down each chemical found at the site with its CAS Number and Koc value (see Appendix A).
2. If more than 20 chemicals are listed, identify those with the ten highest Koc values with an H and those with the ten lowest Koc values with an L.
3. Indicate the range of concentrations for each chemical in each medium and the source of the information (e.g., RI report).
4. Determine a "representative" concentration and enter it; indicate in footnotes the basis of the representative value.

ASSUMPTIONS

List all the major assumptions made in developing the data for this worksheet; also indicate any concerns about the monitoring data:

WORKSHEET 3-1
SCORING FOR INDICATOR CHEMICAL SELECTION:
CONCENTRATIONS AND $K_{oc}$ VALUES IN VARIOUS ENVIRONMENTAL MEDIA

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 3-2

SCORING FOR INDICATOR CHEMICAL SELECTION:
TOXICITY INFORMATION

| Chemical | Toxicologic Class | Rating Value/EPA Category a/ | $w_T$ b/ | $s_T$ b/ | $a_T$ b/ |
|---|---|---|---|---|---|
| Arsenic | PC | A | 4.1 | 2.0E-4 | 41 |
| | NC | 9 | 18 | 9.0E-4 | 180 |
| Tetrachloroethylene | PC | B2 | 8.9E-3 c/ | 4.4E-7 | 0.089 |
| | NC | 7 (oral)<br>10 (inhalation) | 9.6E-3 | 4.8E-7 | 0.028 |
| Beryllium | PC | B1 (inhalation) | -- | -- | 23 |
| | NC | 8 | -- | -- | 15,000 |
| 1,2,4-Trichlorobenzene | NC | 4 (oral)<br>1 (inhalation) | 0.21 | 1.1E-5 | 1.5 |

a/ Rating value is for severity of effect for noncarcinogens, range in 1(low) to 10(high); EPA category is a qualitative weight-of-evidence designation for potential carcinogens; explanation of the categories is presented in Exhibit B-2, Appendix B. Information taken from Appendix A.
b/ Data taken from Appendix A.
c/ 5E-3 is the same as 5.0 x 10-3.

## INSTRUCTIONS

1. Record compounds from Worksheet 3-1, then refer to Appendix A and note whether they are classed as PC or NC or both.

2. Record the rating value or EPA category for each compound in each class (see Appendix A). If there are route-specific differences, record both values.

3. Record the T values from Appendix A.

## ASSUMPTIONS

List all the major assumptions made in developing the data for this worksheet:

WORKSHEET 3-2
SCORING FOR INDICATOR CHEMICAL SELECTION:
TOXICITY INFORMATION

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 3-3

SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR CARCINOGENIC EFFECTS

| Chemical | Ground Water CT | | Surface Water CT | | Soil CT | | Air CT | | IS Value | | Tentative Rank | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Max | Repres | Max | Repres | Max | Repres | Max | Repres | Max | Repres | Max | Repres |
| Arsenic | 1.9 | 0.31 | - | - | 7.5E-3 | 1.4E-3 | - | - | 1.9 | 0.31 | 1 (A) a/ | 1 (A) |
| Tetrachloroethylene | 0.59 | 0.028 | 1E-4 | 2.6E-5 | 5.8E-3 | 8.8E-7 | - | - | 0.59 | 0.028 | 2 (B2) | 2 (B2) |
| Beryllium | - | - | - | - | - | - | - | - | - | - | - (B1) | - (B1) |

a/ EPA category in parentheses.

INSTRUCTIONS

1. List all of the chemicals to be considered as potential carcinogens.
2. Calculate concentration times toxicity (CT) values using the information from Worksheets 3-1 and 3-2. Calculate a CT based on both the maximum and representative concentration for all media in which the chemical was detected.
3. Sum the CT values across media, keeping the two types of concentration separate. Use only the highest CT value of ground water and surface water if both were contaminated. Record the sums in the IS column.
4. Rank the compounds based on both their maximum and representative IS values. Also, enter their EPA weight-of-evidence category in parentheses next to their rank.

ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 3-3
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR CARCINOGENIC EFFECTS

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 3-4

SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS

| Chemical | Ground Water CT | | Surface Water CT | | Soil CT | | Air CT | | IS Value | | Tentative Rank | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Max | Repres | Max | Repres | Max | Repres | Max | Repres | Max | Repres | Max | Repres |
| Arsenic | 8.3 | 0.14 | - | - | 3.2E-2 | 6.3E-3 | - | - | 8.3 | 0.14 | 1 | 1 |
| Tetrachloroethylene | 0.60 | 0.028 | 1.1E-4 | 2.7E-5 | 6.2E-3 | 9.6E-7 | - | - | 0.60 | 0.028 | 2 | 2 |
| Beryllium | - | - | - | - | - | - | - | - | - | - | - | - |
| Trichlorobenzene | 0.25 | 0.023 | 5.5E-3 | 4.2E-4 | 2.2E-3 | 1.7E-4 | - | - | 0.25 | 0.023 | 3 | 3 |

INSTRUCTIONS

1. List all of the chemicals to be considered for noncarcinogenic effects.
2. Calculate concentration times toxicity (CT) values using the information from Worksheets 3-1 and 3-2. Calculate CT values based on both maximum and representative concentrations for all media in which the chemical was detected.
3. Sum the CT values across media, keeping the two types of concentration separate. Use only the highest CT value of ground water and surface water if both were contaminated. Record the sums in the IS column.
4. Rank the compounds based on both their maximum and representative IS values.

ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 3-4
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 3-5

SCORING FOR INDICATOR CHEMICAL SELECTION:
EVALUATION OF EXPOSURE FACTORS AND FINAL CHEMICAL SELECTION

| Chemical | IS Values a/ PC | IS Values a/ NC | Ranking PC | Ranking NC | Water Solubility (mg/l) | Vapor Pressure (mm Hg) | Henry's Law Constant (atm-m3/mole) | Koc | Half-Life (Days) GW | Half-Life (Days) SW | Half-Life (Days) Soil | Half-Life (Days) Air | IC |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Arsenic | 0.31 | 0.14 | 1 | 1 | 1.5E+6 | 0 | 0 | - | >10,000 | >10,000 | >10,000 | 5 | + |
| Tetrachloroethylene | 0.028 | 0.028 | 2 | 2 | 150 | 18 | 0.026 | 364 | 264 | 1-30 | NA | 47 | + |
| Beryllium | - | - | - | - | 0.2 | 0 | 0 | - | >10,000 | >10,000 | >10,000 | NA | + |
| Trichlorobenzene | NA | 0.023 | NA | 3 | 30 | 0.29 | 0.0023 | 9,200 | >10,000 | 1.2 | NA | 50 | + |

a/ Based on representative concentrations.

INSTRUCTIONS

1. List the top 10 to 15 PC and NC based on IS scores, giving their IS values and their ranking.
2. Refer to Appendix C and record each chemical's solubility, vapor pressure, Henry's law constant, Koc, and half-lives in air, water, and soil.
3. Select the final indicator chemicals. Use your judgement -- if a compound has a high water solubility and a long half-life yet is ranked lower than a compound with minimal water solubility and a short half-life, you may wish to move it up in the ranking (refer to Section 3.2 for additional guidance on the final selection).
4. Document any changes in ranking made because of exposure factors.
5. In the last column indicate with a + those chemicals which have been selected as indicator chemicals (in this example all were selected because there are only four chemicals).

ASSUMPTIONS

List all major assumptions made in the development of data for this worksheet:



WORKSHEET 3-5
SCORING FOR INDICATOR CHEMICAL SELECTION:
EVALUATION OF EXPOSURE FACTORS AND FINAL CHEMICAL SELECTION

Clearly, other chemical properties could affect exposures and risks at a specific site. However, to limit the amount of data to be collected and considered, the indicator selection procedure focuses on the five properties listed above. These properties are important, but not exclusive, determinants of environmental transport and fate. Some of the properties have different implications for different exposure pathways. As a result, consideration of the potentially important exposure pathways at a site is important when applying physical/chemical factors in the selection process. A brief description of the relevance of each property to potential chemical release, transport, and fate is given below. Additional discussion of these parameters is available in numerous references, including Kenaga and Goring (1978), Lyman et al. (1982), Nelson et al. (1983), and Maki et al. (1980).

**Water solubility** is the maximum concentration of a chemical that dissolves in pure water at a specific temperature and pH. Solubility of an inorganic species can vary widely, depending on temperature, pH, Eh (redox potential), and the types and concentrations of complexing species present. Solubilities range from less than 1 ppb to greater than 100,000 ppm, with most common organics falling between 1 and 100,000 ppm (Lyman, 1982a). Water solubility is a critical property affecting environmental fate (Menzer and Nelson, 1980). Highly soluble chemicals can be rapidly leached from wastes and contaminated soil and are generally mobile in ground water. Solubility is one of the controlling factors affecting leachate strength and migration of chemicals from waste sites (along with sorption potential, soil type, and water infiltration). Soluble chemicals also tend to be more readily biodegradable than those with low solubility (Lyman, 1982a). Water solubility is especially important in the evaluation of aquatic exposure pathways. Solubility affects "leachability" into both ground water and surface water, and highly soluble compounds are usually less strongly adsorbed (thus more mobile) in both ground and surface water. Solubility, along with several other factors, also affects volatilization from water -- in general, high solubility is associated with lower volatilization rates (Menzer and Nelson, 1980).

Some chemicals may be measured at a site at concentrations higher than their water solubilities. This situation can arise in the case of non-aqueous phase liquids (i.e., liquids that are not dissolved in water and that form a second liquid layer, often floating on top of an aqueous phase or perched on top of an aquifer). In these cases almost pure contaminant may be found. Also, contaminants may be dissolved in the non-aqeous phase at concentrations higher than their water solubilities. Chemicals detected at concentrations higher than their water solubilities may warrant special consideration in selection of indicator chemicals.

**Vapor pressure** and **Henry's Law constant** are two measures of chemical volatility and thus are important in evaluating air exposure pathways. Vapor pressure is a relative measure of the volatility of a chemical in its pure state (Jaber et al., 1984). Vapor pressures of liquids range from 0.001 to 760 torr (mm Hg), with solids ranging down to $10^7$ (Grain, 1982). Vapor pressure is an important determinant of the rate of vaporization from waste sites, but other factors, including temperature and wind speed, degree of adsorption, water solubility, and soil conditions, are also important. Vapor pressure is most directly relevant to exposure pathways involving chemical releases to air from spills or contaminated surface soils. Henry's Law constant, which combines vapor pressure with solubility and molecular weight, is more appropriate for estimating releases to air from contaminated water (e.g., ponds, lagoons) and should be used to evaluate chemicals for which this type of pathway is expected. At sites where air exposure pathways are not important, these two factors should not be used in the selection of final indicator chemicals.

The **organic carbon partition coefficent ($K_{oc}$)** is a measure of relative sorption potential for organics and is a significant environmental fate determinant for all exposure pathways, especially aqueous pathways. The $K_{oc}$ indicates the tendency of an organic chemical to be adsorbed, and it is largely independent of soil properties (Lyman, 1982b). $K_{oc}$ is expressed as the ratio of amount of chemical adsorbed per unit weight of organic carbon to the chemical concentration in solution at equilibrium. Therefore:

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

The normal range of $K_{oc}$ values is from 1 to $10^7$, with higher values indicating greater sorption potential (Lyman, 1982b). Many other partition coefficients exist (e.g., $K_{om}$, $K_d$, $K_{ow}$), but $K_{oc}$ was selected for this purpose because it is chemical-specific (essentially independent of soil conditions) and for organics is directly related to soil and sediment sorption, both of which are significant chemical fate processes at many Superfund sites. For inorganics, some other parameter such as the distribution coefficient for a specific soil type ($K_d$) or the

maximum exchangeable mass may be a better measure of relative adsorption potential.

The significance and interpretation of $K_{oc}$ varies with different exposure pathways. For ground water, low $K_{oc}$ values indicate faster leaching from the waste source into an aquifer and relatively rapid transport through the aquifer (i.e., limited retardation of the chemical). $K_{oc}$ is directly proportional to the retardation factor, which is used in many ground-water transport models. Therefore, among chemicals with similar IS values due to ground-water pathways, high mobility (low $K_{oc}$) chemicals generally would be of more concern. If a chemical with a low $K_{oc}$ is present at a high concentration in soil but is not chosen because of a low IS value, consider adding it to the final indicator list.

For surface water pathways, $K_{oc}$ also has several significant implications. A high $K_{oc}$ indicates tight bonding of a chemical to soil, which means that less of the chemical will be dissolved in site runoff, but also implies that runoff of contaminated soil particles may occur over a longer time period. At some Superfund sites, direct recharge of surface water by ground water is important; in these situations, because of ground-water mobility considerations, chemicals with high $K_{oc}$ are of relatively lower concern. Once a chemical gets into surface water, however, a high $K_{oc}$ may be of great concern because it indicates a tendency to bioaccumulate ($K_{oc}$ is related to bioaccumulation potential). If aquatic food chain pathways are possibly significant, this implication of $K_{oc}$ should be considered. The $K_{oc}$ value also indicates the relative amount of sediment adsorption in surface waters.

An example of the consideration of $K_{oc}$ in indicator chemical selection follows. For a site with: (1) potential ground-water exposure pathways, (2) high soil concentrations of a chemical with low $K_{oc}$, and (3) low concentrations of the same chemical in available ground-water monitoring data, consideration should be given to selecting that chemical despite its probable low indicator score. The combination of low $K_{oc}$ and high soil concentration indicates that significant releases of the chemical to ground water are possible in the future.

The final chemical property to be considered in the indicator selection process is *persistence* in various environmental media. This property is a measure of how long a chemical will exist in a given medium, obviously a critical factor in assessing exposure potential. Important removal processes are phase transfer (e.g., water to air, soil to water), chemical transformation (hydrolysis, photolysis), and biological transformation. Available persistence data are given in Appendix A as ranges of overall half-lives (i.e., due to all removal processes) in air, soil, ground water, and surface water. If half-life values from other sources are used, be sure to determine whether they represent overall disappearance rates or whether they correspond to a specific removal mechanism.

Half-lives of chemicals vary from seconds to thousands of years. Small half-lives generally indicate a lower level of concern, although degradation products may have a higher toxicity or environmental mobility than the original chemical. In considering persistence as a secondary factor for selecting indicator chemicals, you must consider the exposure pathways contributing to the IS score (Worksheets 3-3 and 3-4). Do not use relative persistence in one medium to approximate it in another because the important removal processes may be very different.

One additional factor, to be considered for potential carcinogens only, is the qualitative weight-of-evidence rating. This rating is an indication of the quality and quantity of data underlying a chemical's designation as a potential human carcinogen. The categories of evidence for human carcinogenicity include sufficient, limited, and inadequate. Chemicals on the preliminary indicators list with sufficient evidence of human carcinogenicity (EPA Group A) and chemicals with limited human evidence and sufficient animal evidence (EPA Group B1) should generally be selected as final indicators unless there are convincing reasons to do otherwise. For chemicals with similar IS values, ones with stronger weight-of-evidence should usually be selected.

Using the preceding discussion as guidance, make the final selection of indicator chemicals. Starting with the initial chemical list given in Worksheet 3-5, consider IS scores and relevant additional factors in the final selection process. Indicate on Worksheet 3-5 the final selections and the rationale for each. If toxic organics and inorganics are both present at the site, be sure to include at least one of each on the final list of indicator chemicals.

* * * * *

By following the procedures described in this chapter, a subset of the chemicals present at the site has been selected to serve as indicator chemicals. The procedure has been structured to favor the selection of those chemicals that pose the greatest potential risks and therefore should serve as indicator chemicals. There are many components of the selection procedure that require individual judgment. Care must be taken to apply the general principles set forth in each step in a consistent manner so that the final scores are comparable. **The scores**

**developed here are used only for relative ranking and have no meaning outside the context of this procedure.** They should not be considered as a quantitative measure of a chemical's toxicity or exposure. As a next step in the quantitative analysis process, exposure pathways will be identified for these indicator chemicals and exposure point concentrations estimated.

# CHAPTER 4

# STEP 2: ESTIMATION OF EXPOSURE POINT CONCENTRATIONS OF INDICATOR CHEMICALS

This chapter describes methods for estimating baseline environmental concentrations of indicator chemicals so that the extent and duration of human exposure in the absence of any remedial action can be determined. During the remedial investigation, it is essential to collect sufficient environmental sampling data so that if contamination has reached a human exposure point, some actual data may be used in the evaluation of potential effects. However, at many Superfund sites, contamination has not yet reached the point of human exposure. As a result, it is necessary to estimate how and when such exposure will take place. Chemical fate and transport equations and models may be useful for predicting exposures. Many models, ranging widely in sophistication, data input requirements, cost, and reliability, are available. Ultimately, the remedial project manager must decide what model to use in exposure assessment. Consideration should be given to the complexity of the site and the environment, the precision needed, and the time available for analysis. The **Superfund Exposure Assessment Manual**, a companion to this manual, describes the various models available and provides guidance in selecting appropriate modeling techniques for each site. It should be recognized, however, that the uncertainty associated with modeling results can be significant.

At most sites, **a combination of site monitoring data and environmental modeling results** will be required to estimate chemical concentrations at exposure points. Alone, both types of information have considerable drawbacks. Taken together, site monitoring data and environmental modeling offer the best approach to estimating exposure levels.

Site monitoring data have the advantage of being actual measurements of chemical concentrations on and in the vicinity of the site. Within the accuracy and precision of the sampling and analysis procedures, these measurements are real chemical levels representative of the sampling time, location, and medium.[17] Consideration of site monitoring data alone, however, has several disadvantages for public health evaluation, particularly for assessment of long-term effects. Potential drawbacks include:

- Temporal representativeness-- Monitoring data may be representative of current and/or past conditions, but do not give a clear indication of future conditions. Often at Superfund sites the sampling history is too short to detect time trends, especially in ground water. Because it is necessary to predict future exposures to quantify long-term risks, especially if contaminants have yet to reach any exposure points, monitoring data must be supplemented by some kind of environmental fate modeling (or simple assumptions, such as that concentration will remain constant or continue to change at the observed trend for the next 70 years). Over-reliance on environmental monitoring data can lead to an underemphasis on chemicals not yet released from a source and on slow-moving chemicals that have not yet reached monitoring points. Source monitoring data can help identify these chemicals.
- Spatial representativeness -- Monitoring data are representative of their sampling locations, which may or may not be relevant to a risk assessment. In the past, monitoring at Superfund sites was often conducted on-site at or near a contaminant source. Because chemical concentrations are spatially variable, and available data may not cover off-site human exposure points, monitoring data usually must be supplemented by modeling to allow an adequate assessment of public health effects.

The extreme time and space variability of environmental concentration data at Superfund sites and the need for projections of future health risks, often at off-site exposure points, necessitate the use of chemical fate modeling along with site monitoring data. Monitoring usually represents a time "window" that is too small and a spatial distribution that is too limited to fully represent site conditions. However, at all sites the available monitoring data must be reviewed thoroughly and used to the extent possible. For example, monitoring data should always be used to assist in selection, calibration, and verification of chemical fate models and to help in the estimation of source terms (i.e., release rates) for these models.

Environmental fate modeling at Superfund sites also has significant disadvantages. However, models can

[17] Site monitoring data should be QA/QC validated before use in the risk assessment process.

project chemical concentrations over space and time and thus overcome the major drawback to site monitoring data. With all fate models, especially ones dealing with long-term subsurface transport, there is considerable uncertainty. Ground-water models have not been validated over the long time periods of concern, and many subsurface environments (e.g., anisotropic, heterogeneous) are not well suited to available models. More sophisticated computer models are expensive to use, often require extensive data inputs, and still may not be very accurate because of limitations in the characterization of the source term or other input data. Thus, simple environmental fate models using conservative (i.e., reasonable worst case) assumptions are usually most appropriate for Superfund sites.

In the event that data from human monitoring in the site vicinity (e.g., blood or tissue analyses, genetic testing data) are available or such monitoring is planned, the Agency for Toxic Substances and Disease Registry (ATSDR) should be consulted. ATSDR should take the lead in conducting any human monitoring and in assessing the current health status of people near the site based on human monitoring data.

At some Superfund sites, **background chemical contamination** is significant and should be accounted for in the public health evaluation. Background is defined here as chemical contamination due to a source other than the site under evaluation. Background can be either "natural," as in the case of certain inorganics such as arsenic, or from various anthropogenic sources (e.g., industrial point sources, other uncontrolled waste sites, agricultural pesticide applications). Try to define local background conditions for chemicals of concern based on recent monitoring data, such as RI site characterization results, at locations clearly unaffected by the site (e.g., upgradient, upwind). Three or four upgradient samples taken on one day are insufficient to establish background. However, if background conditions can be assessed with confidence based on available monitoring data, this information should be incorporated into the evaluation. Information resources such as the U.S. Geological Survey, the Soil Conservation Service, the Army Corps of Engineers, and state land use agencies may be helpful in determining background concentrations.

The recommended option for including background is to estimate all chemical concentrations, intakes, and risks for two scenarios: (1) actual conditions at the site, reflecting both background and site-specific contamination, and (2) background alone, as if the site did not exist. The first scenario allows an estimate of overall health risk at exposure points affected by the site, without attribution of the source of the risk. The second scenario indicates the probable risk due to sources other than the site, and comparison of the two scenarios gives information on the relative importance of the site to overall risk. For example, if background arsenic was 5 ppm in drinking water and projected exposure from all sources was 15 ppm, both values could be carried through the entire process, completing parallel worksheets for background and overall risk scenarios.

The methods for estimating environmental concentrations described in this chapter and the Superfund Exposure Assessment Manual should be applied to the selected indicator chemicals. Exhibit 4-1 diagrams the activities involved in estimating exposure point concentrations. The first task is a detailed exposure pathway analysis, which is described in Section 4.1. The second task, estimation of short-term and long-term concentrations for each indicator chemical at each human exposure point, is discussed in Section 4.2. These concentrations will generally be derived from a combination of site monitoring and modeling information. Short-term concentrations (STC) are averaged over a relatively short time period (10 to 90 days) and are used to evaluate potential effects of subchronic exposure; long-term concentrations (LTC) are averaged over longer time periods, up to a human lifetime (70 years), and are used in the assessment of effects of chronic exposure.

For assessment of potential carcinogenic risk, the LTC should usually be averaged over a lifetime. However, for assessment of other chronic health risks, the LTC should not necessarily be averaged over a 70-year period and for some chemicals it would clearly be incorrect to do so. The recommended approach is to average LTCs over the time period of highest exposure for assessment of noncarcinogenic effects and not to substantially reduce an LTC value by averaging over a full lifetime. However, if significant noncarcinogenic risk is projected using this approach, it may be necessary to refer to the specific toxicologic studies on which the toxicity values (i.e., reference dose) are based to determine the most appropriate averaging period.

For example, volatilization from a site may be rapid for a few months and then decrease substantially. The peak STC would be obtained by averaging concentrations over the 10- to 90-day period of greatest volatilization. The LTC for assessing cancer risk would be averaged over the entire 70-year period, beginning with the date of the site assessment. The LTC will always be less than or equal to the peak STC.

The concentrations derived in Step 2 of the public health evaluation process will be the inputs to Step 3 -- estimation of chemical intakes. The exposure

**Identify Potential Human Exposure Pathways**

**Estimate Exposure Point Concentrations of Indicator Chemicals Using Environmental Monitoring and Appropriate Models**

**Compare Projected Concentrations to Applicable or Relevant and Appropriate Requirements**

EXHIBIT 4-1
OVERVIEW OF STEP 2: ESTIMATING EXPOSURE POINT CONCENTRATIONS

point concentrations will also be compared to applicable or relevant and appropriate ambient concentration requirements, a task described in Section 4.3.

Worksheets are provided as a means for organizing and documenting the data collected for estimating exposure point concentrations. Filling in these worksheets will not be sufficient to complete the quantitative analyses required. Rather, they serve to direct and focus the analysis so that the results can be used directly in later steps of the public health evaluation. All procedures, assumptions, and calculations used to develop concentration estimates must be clearly documented in a format that will facilitate review.

## 4.1 Identify Exposure Pathways

This section describes an approach for identifying potential human exposure pathways at a Superfund site. An exposure pathway consists of four necessary elements: (1) a source and mechanism of chemical release to the environment, (2) an environmental transport medium (e.g., air, ground water) for the released chemical, (3) a point of potential human contact with the contaminated medium (referred to as the exposure point), and (4) a human exposure route (e.g., drinking water ingestion) at the contact point. Exhibit 4-2 illustrates the elements of an exposure pathway. Each pathway therefore describes a unique mechanism by which a population or an individual is exposed to contaminants originating from a site. The overall risks posed by a site are a composite of the set of individual pathway risks. Risks for individual pathways, however, may not be additive because they may represent risks to different populations.

The Superfund risk assessment process is based on concern for both individual risk and risk to exposed populations. One exposure point that should be evaluated for a pathway is the geographic point of highest individual exposure for a given release source/transport medium combination (i.e., the geographic location where human inhabitants are exposed to the highest predicted chemical concentrations). Exposure points with lower predicted chemical concentrations and large potentially exposed populations should also be evaluated. For example, a potentially vulnerable public water supply serving a large population should be included in the evaluation even if higher exposures are projected at a few private wells closer to the site.

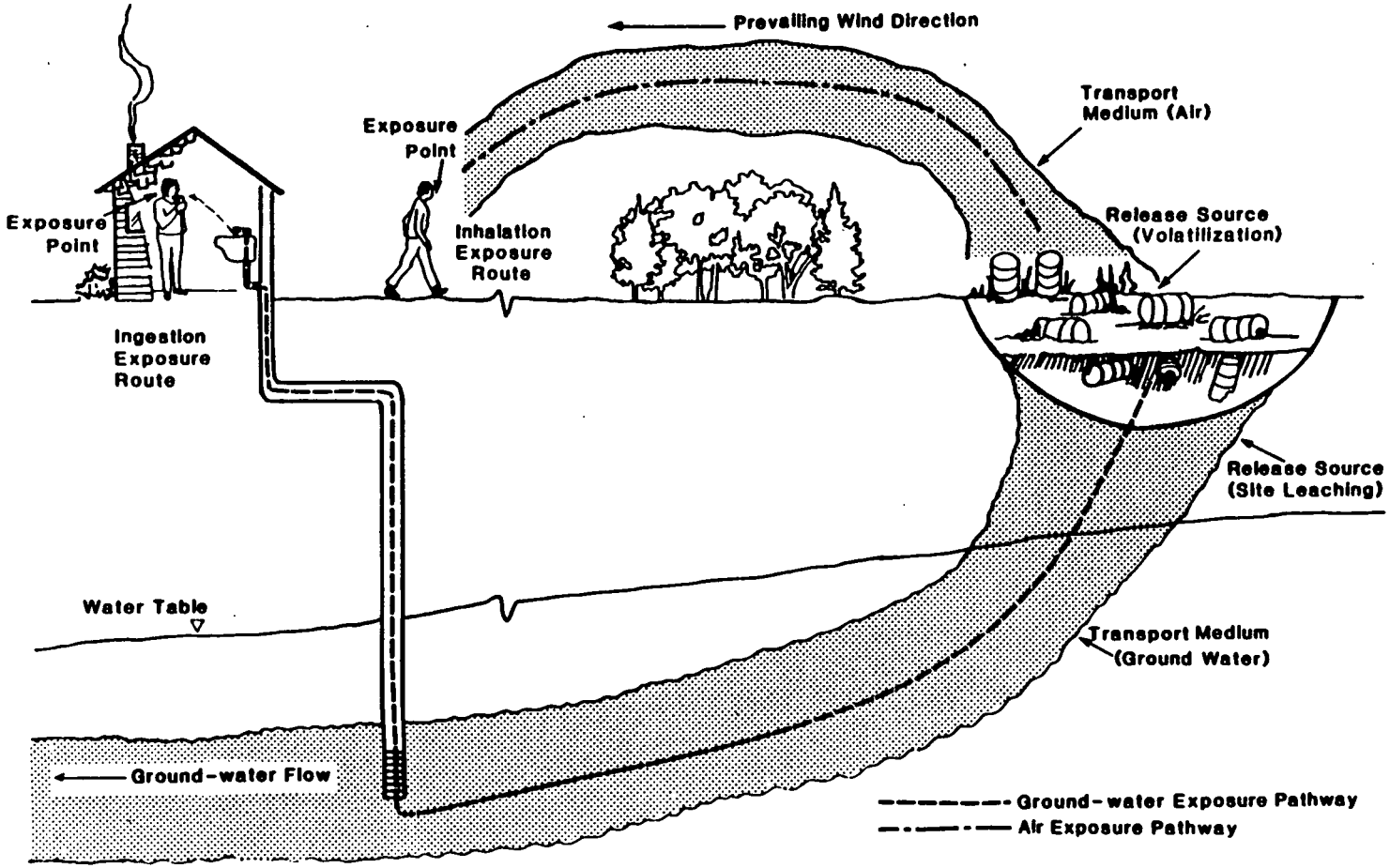


EXHIBIT 4-2 ILLUSTRATION OF EXPOSURE PATHWAYS

To identify possible exposure pathways, human activity patterns near the site should be defined and combined with chemical release source and transport media information. This task is accomplished using a qualitative, yet systematic procedure that relies on professional judgment and experience. Because chemical release and transport are more rigorously analyzed in the next phase of the exposure assessment (Section 4.2), the initial list of exposure pathways can be modified as the analysis proceeds. If there are questions or uncertainties about a possible exposure pathway, it should not be eliminated from the analysis until the next phase is completed.

The analysis described here is a first-cut organization of the relevant site information so that major exposure pathways can be defined. It is not intended as a time-consuming task in the overall public health evaluation process. Iterations of this procedure following the results of additional site sampling and/or modeling will confirm the important exposure pathways. A four-step framework for the exposure pathway analysis is described below.

### *4.1.1 Determine Possible Chemical Release Sources and Release Media*

To determine possible release sources for a site in the absence of remedial action, use all available site descriptions and data from preliminary assessment, site inspection, and remedial investigation. Also obtain and use any appropriate information being developed as part of the feasibility study. Monitoring data showing off-site contamination in excess of background levels are especially valuable because they demonstrate chemical release and transport from the site. Exhibit 4-3 lists some typical release sources at Superfund remedial sites, organized by release medium. In many cases the release, transport, and exposure media will be the same (i.e., release to air will result in transport and exposure via air). However, intermedia transfers can occur and may be critical at some sites (e.g., fish ingestion exposures, which result from releases to surface water).

Use Worksheet 4-1 to summarize the results of the initial release source analysis. Supplement Worksheet 4-1 with a site map that indicates locations of the release sources. At this point, combinations of release source/transport medium for a site (i.e., the first two components of exposure pathways) have been identified and the exposure points for each must now be determined.

### *4.1.2 Identify and Characterize Possible Human Exposure Points*

First, identify for each combination of release source and transport medium (Worksheet 4-1) the location of highest individual exposure to the general public (defined here as the "significant" exposure point). Next, determine the number of people potentially affected at each of the significant exposure points and record the basis for the estimate. Both short-term and long-term exposures must be considered. In addition, include any locations with the potential for exposure of large numbers of people (e.g., public drinking water supplies, shopping centers, industrial parks) or sensitive populations that may be at special risk (e.g., schools, hospitals). Some of these locations should be included as supplementary exposure points in the exposure and risk analysis to follow. In addition to identifying locations of exposure points, determine the probable routes of exposure at each. Guidance for identifying significant exposure points is given below for each transport medium.

Consider including the site itself as an exposure point, based on a reason- able future use scenario. Clearly, this consideration would be inappropriate at sites where future development is improbable, but some sites may have future human contact uses. Consult with local planning and zoning officials to determine a reasonable future use scenario. If the scenario includes human contact, include these on-site exposure pathways in the analysis.

<u>Air Exposure</u>. For air exposures, the individuals exposed to highest concentrations will generally be the people located downwind of and nearest to the source. This may not always be true; for example, the point of highest ambient ground-level concentration may be some distance from the source if the source is elevated. In these cases, the appropriate exposure point must be determined later, in conjunction with sampling or air modeling efforts (as described in Section 4.2). At the majority of Superfund sites, however, it can probably be assumed that the nearest population is the pertinent exposure point. Once the release sources into air are determined in the first task, it is relatively straightforward to locate the closest population. These populations can be located in residential, industrial, or commercial areas or at other points of human activity. Potential sources of this information include:

- site vicinity surveys;
- topographic maps;
- aerial photos of the site;
- county or city land-use maps; and
- census data.

On a map, indicate precisely for each air release source the direction and distance to the significant exposure point.

The point of highest short-term individual exposure by air may well be different from the point of highest long-term exposure. The highest short- term exposure point will generally be the closest

| Release Medium | Release Mechanism | Release Source |
|---|---|---|
| Air | Volatilization | Surface wastes -- lagoons, ponds, pits, spills<br>Contaminated surface soil<br>Contaminated wetlands<br>Leaking drums |
| | Fugitive dust generation | Contaminated surface soil<br>Waste piles |
| Surface water | Surface runoff | Contaminated surface soil |
| | Episodic overland flows | Lagoon overflow<br>Spills, leaking containers |
| | Ground-water seepage | Contaminated ground water |
| Ground water | Site leaching | Surface or buried wastes<br>Contaminated soil |
| Soil | Site leaching | Surface or buried wastes |
| | Surface runoff | Contaminated surface soil |
| | Episodic overland flows | Lagoon overflow<br>Spills |
| | Fugitive dust generation/ deposition | Contaminated surface soil<br>Waste piles |
| | Tracking | Contaminated surface soil |

EXHIBIT 4-3
COMMON CHEMICAL RELEASE SOURCES AT SITES IN THE ABSENCE OF REMEDIAL ACTION

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 4-1

## PRELIMINARY RELEASE SOURCE ANALYSIS FOR BASELINE SITE CONDITIONS

| Release Medium | Potential Release Source | Release Mechanism | Release Time Frame | Release Probability/ Amount |
|---|---|---|---|---|
| Air | Contaminated surface soil | Volatilization | C | 100% probability; amounts may be high |
| Surface water | On-site lagoon | Overflow | E | Low probability; relatively high amounts |
| Ground water | | | | |
| Soil | | | | |

## INSTRUCTIONS

1. For each medium, list potential release sources and mechanisms.
2. Estimate release time frame: chronic (C) or episodic (E).
3. Record any information, qualitative or quantitative, on release probabilities and amounts. If quantitative data from observations made during the remedial investigation on frequency, duration, probability, and quantity of releases are available, report those values here.
4. Attach a site map indicating locations of release sources.

## ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 4-1
PRELIMINARY RELEASE SOURCE ANALYSIS FOR BASELINE SITE CONDITIONS

population in any direction from the site, whereas the highest long-term exposure point will, in most cases, be downwind. Therefore, select the exposure point for determining long-term concentration within the downwind 90° arc from the emission source (45° on each side of the average downwind centerline as determined from historical wind data for locations near the site), unless it can be demonstrated that long-term concentrations will be higher elsewhere. Historical wind data are usually available for airports and some other locations through the National Oceanic and Atmospheric Administration (NOAA).

*Surface Water Exposure* The significant exposure points for surface water pathways depend on downstream uses of the water. Both withdrawal points and areas of in-stream use must be considered. Withdrawal uses to be considered include domestic water supply (drinking, cooking, bathing), agricultural use (livestock watering, irrigation), and industrial use. Relevant in-stream uses include swimming and other water contact sports and private and commercial fishing (resulting in ingestion of contaminated fish). Sources for identifying withdrawal points and uses include:

- site vicinity surveys;
- state water agency records;
- local water utility records;
- withdrawal permits; and
- EPA Office of Drinking Water data bases (Federal Reporting Data System, or FRDS).

Locate on a map the exact points of withdrawal in relation to the source from topographic maps. Indicate points of in-stream use from site vicinity surveys and possibly from local or state planning and recreation agencies.

At some sites, an important potential route of exposure via surface water is through the ingestion of contaminated fish or shellfish. Fish living in contaminated water concentrate contaminants from the water in their tissue. Due to the solubility of some contaminants in fats, many chemicals are bioconcentrated and appear in the tissue at concentrations higher than in the surrounding water. Consumption of fish from surface water near sites should be considered as a possible exposure route.

*Ground-Water Exposure*. Determining points of highest exposure to ground-water contaminants will often be difficult unless subsurface flow modeling is done. In general, nearby wells will have higher concentrations than distant wells, and wells in the direction of ground-water flow (often approximated by surface slope) will be higher. If comprehensive ground-water modeling is planned, do not determine the significant exposure point until it is completed. Determine instead the locations, depths, pumping rates, and uses of all wells in the immediate site vicinity and in the likely direction of flow. Specify the ground-water formations from which various wells are pumping, and determine the general extent of hydraulic connection among the multiple formations. Identify well information through state or local agency well logs or site vicinity surveys. This information can then be used in conjunction with monitoring and/or modeling results developed to determine the significant exposure points.

If subsurface modeling is not planned, determine the likely flow direction from geohydrologic data and assume that the closest domestic well in that direction is the highest individual exposure point. Locations and depths of public water supply wells should also be determined. In addition to domestic wells, locations of agricultural and industrial wells and any other relevant ground-water uses must be determined.

Hydraulic connections between ground water and the surface water exposure points identified above should also be determined.

*Soil Exposure*. Areas of highest direct exposure to contaminated surface soil will generally be on or directly adjacent to the waste site. If access to the site is not restricted or otherwise limited (e.g., by distance), the site itself usually can be assumed to be the point of highest individual exposure to surface soil. If site access is limited, the significant exposure point for soil often will be the nearest residence or other human use area (e.g., playground). If there is no evidence of surface soil contamination in the site vicinity, there may be no important direct exposure pathways resulting from soil contamination. A possible indirect route of exposure from soil contamination to be considered is chemical uptake by plants, with subsequent ingestion by humans.

Typical exposure points for the four environmental exposure media are summarized in Exhibit 4-4. This exhibit can be used as guidance for determining exposure points, but this determination is a site-by-site analysis and the possibility of other exposure points must be considered for each site.

### 4.1.3 Integrate Release Sources, Environmental Transport Media, Exposure Points, and Exposure Routes Into Exposure Pathways

Assemble the information developed in the previous two steps and determine the complete exposure pathways that exist for the site. Use Worksheet 4-2 to record the exposure pathway information. A complete exposure pathway is one that has all the necessary components: a source and mechanism of chemical release, an environmental transport

| Transport/Exposure Medium | Typical Exposure Point | Major Exposure Route |
|---|---|---|
| Air | Nearest residence to source | Inhalation |
| | Nearest population magnet (e.g., shopping center, school, industrial park) | Inhalation |
| | Other residence/population at point of highest concentration | Inhalation |
| Surface water | Withdrawal point for potable use | Ingestion, dermal, inhalation |
| | Withdrawal point for agricultural use | Inhalation, ingestion (food), dermal |
| | Withdrawal point for other uses (e.g., industrial) | Inhalation, dermal |
| | Nearest point for swimming/contact sports | Ingestion, dermal |
| | Nearest point for fishing | Ingestion (food) |
| Ground water | Nearest potable well (private or public) | Ingestion, dermal, inhalation |
| | Nearest agricultural well | Inhalation, ingestion (food), dermal |
| | Nearest well for other uses (e.g., industrial) | Inhalation, dermal |
| Soil | On-site | Dermal, ingestion |
| | Immediately adjacent to site (if site is restricted) | Dermal, ingestion |
| | Nearest cropland | Ingestion (food) |

EXHIBIT 4-4
TYPICAL EXPOSURE POINTS FOR CHEMICAL RELEASES FROM HAZARDOUS WASTE SITES

medium, a potential human exposure point, and a likely route of exposure. For example, if a release to ground water is projected but there is no ground-water use (or projected use) from the affected aquifer, then the exposure pathway is incomplete. The exposure points for the complete exposure pathways define the spatial locations at which chemical concentrations must be projected. The health risk estimates developed later in this process are based on exposures at these locations. The total number of people that may be exposed does not enter into the public health evaluation quantitatively; however, it may be important on a qualitative basis.

In some cases, exposures via identified pathways may be non-quantifiable. There are a number of possible reasons for this, including the absence of data on which to base estimates of chemical releases, environmental concentrations, or human intakes. If an exposure pathway is determined to be non-quantifiable during the exposure assessment procedure to follow, continue to include it as a potential pathway on all subsequent worksheets, designating it as non-quantified. This information can be taken into account in assessments of the uncertainty of the results.

### *4.1.4 Determine Presence of Sensitive Human Populations*

Review the information on the site area and determine if any population groups with high sensitivity to chemical exposure are present. Sensitive subpopulations that may be at higher risk include infants and children, elderly people, pregnant women, and people with chronic illnesses. Sites may be located in areas without readily identifiable sensitive subpopulations, but if such subpopulations are present, the number of people involved and their location should be determined.

To identify sensitive subpopulations in the site area, determine locations of schools, day care centers, hospitals, nursing homes, and retirement communities that are within three miles of the site or that use drinking water potentially affected by the site. Use local census data and information from local public health officials for this determination. Record this information on Worksheet 6-2 (see Chapter 6).

## 4.2 Estimate Exposure Point Concentrations

To the extent available, measured chemical concentration data should be reviewed for each chemical, exposure medium, and exposure point. Such monitoring data can be used to estimate peak short-term concentrations at exposure points. However, in addition to short-term indications of concentration, long-term concentrations (averaged over periods up to a human lifetime, 70 years) need to be estimated. Long-term concentrations are more difficult to estimate and usually require environmental fate modeling (see Sections 4.2.1 and 4.2.2). The short-term and long-term concentration estimates will be used in the next phase of the public health evaluation -- calculating human intake. By understanding the potential long-term exposures from a site, one will better understand the consequences of not taking any action. Short-term concentrations will be important in the evaluation of chemicals to which even short-term exposure is a concern and which can be contained by certain management practices. Note that the only chemicals being evaluated here are those that have been selected as indicator chemicals.

Relevant monitoring results from points of human exposure should be recorded on Worksheet 4-4 (near the end of Chapter 4) to provide short-term concentration values. Because several samples are generally taken, some measure of the variability of the estimate (confidence interval, range) should be recorded. Long-term concentrations on which to base lifetime exposures may be estimated on the basis of both monitoring data and the chemical release and fate models described in the Superfund Exposure Assessment Manual.

After potential exposure pathways are determined, environmental concentrations for each indicator chemical must be estimated at each of the significant and supplementary exposure point locations identified in Worksheet 4-2. Concentrations of substances need to be estimated as a function of time (i.e., short-term and long-term) in each environmental medium -- air, surface water, ground water, or soil -- through which potential exposures could occur. For example, if in completing Worksheet 4-2, it is determined that potential exposure routes for a nearby residential area are inhalation of contaminated air and ingestion of contaminated ground water, chemical concentrations over time must be predicted for both air and ground water at this location.

Estimating environmental concentrations at an exposure point is essentially a two-step process. First, quantify the amounts of chemicals that will be released to the environment by the various sources identified in the exposure pathway analysis. Given these release quantities, then predict the environmental transport and fate of each indicator substance in the identified medium of the exposure pathway. An example would be the movement of a contaminant released to ground water from contaminated soil and then transported to a drinking water well.

Numerous analytical techniques are available to perform the calculations required in these two steps. These techniques are described in detail in the

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 4-2

MATRIX OF POTENTIAL EXPOSURE PATHWAYS

| Release/ Transport Medium | Release/Source Mechanism | Exposure Point | Exposure Route | Number of People | Pathway Complete |
|---|---|---|---|---|---|
| Air | Contaminated soil/ volatilization | Nearest residences * (0.7 mile SW of site) | Inhalation | 50 | Yes |
| | Contaminated soil/ volatilization | Trailer park (2 miles south of site) | Inhalation | 600 | Yes |
| Ground water | Contaminated soil/ leaching | Wells at nearest residences* | Ingestion of drinking water | 50 | Yes |
| | Contaminated soil/ leaching | Wells at 2 miles serving neighborhood | Ingestion of drinking water | 900 | Yes |
| Surface water | | | | | |
| | | | | | |
| Soil | | | | | |
| | | | | | |

* Significant exposure point.

INSTRUCTIONS

1. List all release sources and mechanisms by release medium.
2. Describe the nature of the exposure point and its location with respect to release source (e.g., nearest residence to volatilization release site, 300 feet NW). Denote significant exposure points with an asterisk.
3. List exposure route (e.g., inhalation, ingestion).
4. Report the number of people potentially exposed at the exposure point.
5. Mark where exposure pathways are complete (i.e., where release source, transport medium, exposure point, and exposure route all exist).

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 4-2
MATRIX OF POTENTIAL EXPOSURE PATHWAYS

Superfund Exposure Assessment Manual. The techniques vary in sophistication from simple, desk-top methods that provide rapid, order-of-magnitude projections, to more rigorous approaches involving computer modeling that may give more accurate results, but require more time and resources to undertake. All techniques require certain chemical- and site-specific data, although the data requirements vary with the degree of sophistication of the method used. Regardless of the technique used, it is likely that numerous assumptions will be required because of gaps in available data. The appropriate level of sophistication will be influenced by data availability, and by the demands and bounds of the remedial investigation/feasibility study effort at a specific site. Relatively simple chemical release and transport models are usually appropriate for Superfund public health evaluation exposure assessments.

There are two recommended approaches for addressing the unavoidable estimation uncertainties likely to be encountered in the exposure assessment. One is to use a conservative (not necessarily "worst-case") approach in making the assumptions necessary for a particular estimation method. The consequence of making conservative assumptions is that risks may be substantially overstated but will not be understated in the final analysis. All assumptions and the basis for each should be recorded.

A second, and generally preferred, approach is to **calculate and present both best estimates and conservative upper bound estimates for all exposure point chemical concentrations.** If this approach is followed and both sets of concentration estimates are carried through the entire public health evaluation (ultimately resulting in two sets of risk estimates), the results will provide not only an estimate of the risk magnitude but also a good indication of the overall uncertainty of the analysis. Of course, this approach requires more calculation effort, but it is a straightforward way to account for analytical and data uncertainties. This approach, which yields an upper bound and best estimate of each risk projection, emphasizes the uncertainty involved by displaying it quantitatively. A large disparity between the upper bound and best estimates of risk would indicate relatively high uncertainty, and vice-versa. This approach requires that two sets of most subsequent worksheets be completed, one for the best estimate and one for the upper bound.

A third possible approach, generally beyond the scope of the Superfund public health evaluation process, is to model the important variables determining chemical concentration and risk stochastically. This allows estimation of a risk distribution, from which median and 90th percentile (or other upper bound) values can be determined. This approach is more complex and time-consuming than a deterministic approach, and it still only accounts for uncertainty due to the variables modeled stochastically. It does not address other sources of uncertainty, such as applicability of the release or transport models to the real site situation.

The following subsections explain how chemical release and transport models should be used and the types of outputs that are needed to continue the risk assessments process. Detailed guidance on chemical release, transport, and fate assessment at Superfund sites is contained in the Superfund Exposure Assessment Manual, which accompanies this manual. In addition, a set of background documents for EPA's proposed guidelines for exposure assessment (EPA, 1984b) is being prepared and will be a convenient source of this information when released.

### *4.2.1 Quantify Chemical Releases*

Chemical releases are quantified in terms of release rates. These rates are then used along with other factors to predict environmental fate and transport. Various methods are available for estimating release rates. They are fairly straightforward and can be verified with the use of site sampling data. Evidence of chemical release into an environmental medium such as ground water, air or surface water must have been observed to warrant a quantitative analysis. When release rates calculated from a model result in concentrations that do not make sense in light of the site sampling data, reexamine the selection of the model or the reliability of the sampling results.

To quantify releases, consider separately each release medium and the associated sources and mechanisms of release that have been identified in the exposure pathway analysis (Section 4.1) for a specific chemical. Calculate the mass loading of the chemical contaminant from each release source to the environmental medium. In some cases, it will be sufficient to calculate a constant, or steady-state loading rate, based on the assumption that insignificant reductions in contaminants occur at the source during the evaluation time period. In other instances, reductions in release rates over time may need to be accounted. Ultimately, professional judgment must be used to decide which course to take for each specific release source.

Brief descriptions of methods available to calculate releases are presented below for each of the four primary environmental media of interest -- air, surface water, ground water, and soil. References are also made to more detailed descriptions of the methods contained in other documents. A substantial amount of data is required to complete the analyses described. Recognizing that all of the

necessary data will rarely be available, the analyses can be conducted with proper application of professional judgment in making assumptions. Again, all assumptions and their basises should be recorded.

Air Release Modeling. Releases of hazardous constituents to air from a remedial action site generally occur as a result of volatilization or fugitive dust generation. The calculation of the site volatilization rate depends on the situation in which the waste constituent exists in the environment. The rate differs according to whether the wastes are covered with soil, are concentrated on the surface, or are dissolved in water. Volatilization rate is determined primarily by the chemical properties of a given substance, the concentration of that substance, and environmental conditions such as wind speed and temperature.

There are a number of mathematical models available that describe volatilization rates for various types of physical situations. For a review and discussion of mathematical models describing volatile releases from hazardous waste sites and the selection of appropriate k-values, refer to the Superfund Exposure Assessment Manual.

Contaminated fugitive dusts from a waste site can result from many activities, including:

- wind erosion of wastes and soils
- vehicular traffic movement over contaminated roads
- heavy equipment activity at the site.

One or any combination of these activities can create emissions of toxic materials associated with the fugitive dust. In addition to the Superfund Exposure Assessment Manual, a manual recently prepared for EPA's Exposure Assessment Group, "Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites" (Cowherd et al., 1984) is a valuable reference for fugitive dust calculations.

Surface Water Release Modeling. Releases of hazardous constituents to surface water can occur due to the point discharge of treated runoff, leachate, or ground water (this mechanism is not usually relevant to assessment of the no-action alternative); contaminated surface runoff; recharge by contaminated ground water; or episodic overland flow from leaks, spills, or lagoon or pond overtopping. Refer to the Superfund Exposure Assessment Manual for additional guidance.

Ground-Water Release Modeling. Calculating releases to ground water involves the estimation of leachate migration from the site. For an uncontrolled site, one approach is to use site sampling data to determine the extent of soil contamination directly beneath the source of chemical release at the site, and convert these to release rates of constituents. For detailed guidance, refer to the Superfund Exposure Assessment Manual.

Soil Releases. Surface soils may become contaminated with toxic materials as a result of intentional placement of the wastes on the ground, or from spills, lagoons or pond failures, contaminated site runoff, or downwind deposition of contaminated airborne particulates. The substances of concern are generally those that adsorb to or are otherwise associated with the soil particles. Determine the extent of contamination of soils using the results of the sampling and analysis conducted during the remedial investigation phase. Monitoring is really the only practical method to provide direct quantification of soil contamination. The Superfund Exposure Assessment Manual gives more detailed guidance on estimating soil releases.

Worksheet 4-3 is provided as a convenient mechanism for compiling the results of the quantification of contaminant releases calculated for each exposure point. List the results of release calculations in the appropriate columns of the worksheet and attach all documentation for the release calculations.

#### *4.2.2 Predict Environmental Fate and Transport*

In the second step of the process for estimating environmental concentrations, use the estimates of mass loadings of chemicals released to predict the environmental fate and transport of chemicals from the release source to identified exposure points. For each chemical and each exposure pathway, the outcome of this exercise will be short-term and long-term environmental concentrations at the significant exposure point. To arrive at these concentrations, the entire concentration profile of a substance over time at the exposure point may have to be modeled; appropriate short-term and long-term values can then be determined from the profile.

To account for the behavior of all released chemicals, it is necessary to consider systematically the extent of chemical fate and transport in each environmental medium. In this way, the remedial project manager can consider the predominant mechanisms of chemical transport, transfer, and transformation, and disregard less significant processes. In the following sections, brief descriptions of the mechanisms for each of the major environmental release media are presented. More detailed descriptions of available techniques and computer models and their limitations are given in the Superfund Exposure Assessment Manual.

Air Transport Modeling The predominant mechanisms that affect the atmospheric fate and transport of substances released to the air are advection, dispersion and, in some cases, natural

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 4-3

RESULTS OF RELEASE QUANTIFICATION

Exposure Point: Nearest Residence

| | Chemical | Release Medium | Release Source/ Mechanism | Release Mass Load (mass/time) Best Estimate Short-Term | Best Estimate Long-Term | Upper-Bound Estimate Short-Term | Upper-Bound Estimate Long-Term |
|---|---|---|---|---|---|---|---|
| 1. | Benzene | Ground water | Leachate | 5 kg/day | 150 kg/yr | 50 kg/day | 2500 kg/yr |
| | | Air | Site volatilization | 0.1 kg/day | 2.5 kg/yr | 2 kg/day | 50 kg/yr |
| 2. | Lead | Ground water | Leachate | 1.5 kg/day | 35 kg/yr | 25 kg/day | 700 kg/yr |
| | | | | | | | |
| 3. | | | | | | | |
| | | | | | | | |

INSTRUCTIONS

1. List all indicator chemicals.
2. List the release media for each chemical: air, ground water, surface water, soil.
3. List all release sources.
4. Record best and upper-bound estimates for both short-term and long-term release mass load, as calculated. Attach the documentation for all calculations.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

decay. Ambient concentrations of a chemical at a specified downwind distance from the site can be determined as a direct function of chemical release rate when these key processes are considered. Refer to the Superfund Exposure Assessment Manual for guidance on appropriate modeling techniques.

At some sites, relatively precise estimates of chemical fate and transport in air may be required. Sophisticated computer models are available for predicting the behavior of chemicals released to the atmosphere. The models have varying capabilities, data requirements, computer resource requirements and sophistication of output. The Superfund Exposure Assessment Manual lists some computer models that are applicable to the analysis of remedial action sites. Exercise care in selecting the model most appropriate to the specific site and the hazardous substance characteristics. The reasons for selecting a particular model should be documented. Generally, for risk assessments in the feasibility study, the simplest model that reasonably represents the system should be used.

Surface Water Transport Modeling. The environmental fate of hazardous materials entering surface water bodies is highly dependent on the type of water body and the specific chemicals involved. Relatively simple, straightforward approaches are available for estimating environmental concentrations in rivers and streams. However, more complex methods are necessary for predicting concentrations resulting from releases to lakes, reservoirs, and estuaries. Applicable methods are described or referenced in the Superfund Exposure Assessment Manual. In addition, EPA's Water Quality Assessment documents (Mills et al., 1982) may be helpful in selecting water models.

Sophisticated computer models are also available for the analysis of environmental fate of hazardous substances in surface water bodies. As with the sophisticated air models, these vary in complexity, input data requirements, computer resource requirements, and model capabilities. Again, simple models are generally preferable. If a computer modeling approach is desired for a site, select the modeling procedure most appropriate to the circumstances under study. Again, document the rationale for selecting a particular model.

Ground-Water Transport Modeling. In describing the behavior of contaminants released to ground water from a hazardous waste site, two major subsurface zones must be considered: the unsaturated soil zone above the ground water (vadose zone), and the saturated zone, commonly called the aquifer. In general, after a substance is released, it first moves vertically down through the unsaturated soil zone to the ground water. Then, after initial mixing in the ground water, the substance travels horizontally because of the advective flow of the ground water underlying the site. The primary processes that affect the fate and transport of contaminants in these two zones are advection (including infiltration and leaching from the surface), dispersion, sorption (including reversible adsorption, ion exchange, complexation, and precipitation), and degradation. As a released substance flows away from the source area, these processes act to reduce its concentration.

Time plays a key role in the movement of contaminants in the subsurface environment. Unlike the air and surface water media where releases of chemicals generally result in downwind or downstream ambient concentrations within relatively short times after release (i.e., minutes, hours, or days), ground water moves slowly and takes much longer (years) to transport contaminants. Consequently, the estimation of ground-water concentrations at a given exposure point must be bounded by a specified time frame for which the public health evaluation will be conducted.

For purposes of evaluating individual risks for the no-action alternative at Superfund sites, ground-water concentrations should be estimated for at least 70 years. This period is selected because it approximates an average human life span, and it is the basis for establishment of the acceptable chronic chemical intakes contained in the health effects assessments (HEAs). Use the highest concentration value predicted at an exposure point during the 70-year period to represent the short-term concentration. For long-term concentrations, use a 70-year time-weighted average.

Numerous mathematical models are available that describe pollutant fate and transport in the subsurface environment. These models are described or referenced in the Superfund Exposure Assessment Manual. These models attempt to define waste migration over time and distance using the physical and chemical processes involved. The physical and chemical characteristics considered by these models include:

- Boundary conditions (hydraulic head distributions, recharge and discharge points, locations and types of boundaries);
- Material constants (hydraulic conductivity, porosity, transmissivity, extent of hydrogeologic units);
- Attenuation mechanisms (adsorption-desorption, ion exchange, complexing, nuclear decay, ion filtration, gas generation, precipitation-dissolution, biodegradation, chemical degradation);

- Molecular diffusion and hydrodynamic dispersion (transverse, longitudinal, and vertical); and
- Waste constituent concentrations (initial and background concentrations, boundary conditions).

These characteristics are incorporated into models by combining two sets of transport expressions: a ground-water flow equation and a chemical mass transport equation. The result is a prediction of solute transport in the ground-water system, with chemical reactions considered.

Separate models exist for predicting transport through both the unsaturated and saturated zones. Models are often linked into a comprehensive package to effectively simulate movement through both unsaturated and saturated soil zones. In addition, some ground-water models have the capability of predicting hazardous substance fate throughout both zones. Most of these models are designed to be used with a computer. The Superfund Exposure Assessment Manual lists some computer models applicable for site analysis.

Models for ground-water transport generally have not been fully verified, and their reliability is difficult to assess. Site-specific conditions and the analyst's ability to account for site-specific characteristics with quantitative input data influence the reliability of model results. Carefully applied professional judgment is therefore necessary both in using the models and in interpreting the results. Ground-water monitoring data collected in the vicinity of the site should be used whenever possible to test the reasonableness of model results. Models can sometimes be calibrated with the measurements taken during the RI. When no monitoring data are available, important sources of uncertainty should be noted and their impact on model results should be anticipated and recorded.

Worksheet 4-4 is provided as a format for recording the estimated chemical concentrations for each exposure point.

## 4.3 Compare to Requirements, Standards, and Criteria

At this point in the process, the projected baseline concentrations of indicator chemicals at exposure points should be compared to "applicable or relevant and appropriate requirements" (as defined by the NCP and originally identified in the CERCLA compliance with other environmental statutes policy memorandum that is an appendix to the NCP; additional requirements are identified in the CERCLA reauthorization statute). "Other criteria, advisories, and guidance" may also be compared to exposure point concentrations, if pertinent to site exposure conditions. The following subsections describe the procedure for comparing both to requirements and to other criteria. The user should be aware that EPA continues to update toxicological information and, based on these updated data, may issue revised standards and criteria.

This entire section of the manual focuses on numerical criteria that are in the form of ambient environmental concentration levels. In the case of applicable or relevant and appropriate requirements or other criteria expressed in intake or dose units (e.g., in mg/kg-day), the comparison should be deferred until the intake estimation step of this process is complete (see Chapter 5).

### *4.3.1 Compare to Applicable or Relevant and Appropriate Requirements*

If all indicator chemicals at a site have applicable or relevant and appropriate requirements (ARARs), then the remainder of the baseline process described in Chapters 5 through 7 is not necessary. In these cases, the comparison of predicted exposure point concentrations of indicator chemicals to ARARs will suffice as a baseline public health evaluation. At sites where some indicator chemicals do not have ARARs, make the comparison to requirements for those chemicals that have them and then proceed with the complete risk characterization process for all\ indicator chemicals. Therefore, in cases where ARARs are not available for all indicator chemicals, the baseline public health evaluation will include both a comparison to ARARs and a risk assessment as described in Chapters 5 through 7.

At the present time, EPA considers drinking water maximum contaminant levels (MCLs) and maximum contaminant level goals (MCLGs), federal ambient water quality criteria, national ambient air quality standards (NAAQS), and state environmental standards to be potentially applicable or relevant and appropriate requirements for ambient concentrations. Exhibits 4-5 and 4-6 list federal ARARs for ambient environmental concentrations of contaminants. RCRA design and operating requirements are also applicable or relevant and appropriate for design of remedial alternatives but, because they are not pertinent to the baseline public health analysis, they are not discussed further here (see Chapter 8).

The determination of exactly which requirements are applicable or relevant and appropriate to a particular Superfund site should be made on a site-specific basis. Potential ARARs will not necessarily be appropriate for every site. For potential ground-water and surface water exposure via drinking water, the most appropriate comparison values are Safe Drinking Water Act MCLs and MCLGs; for air exposure, national ambient air quality standards may be appropriate comparison values; for surface water

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 4-4

CONTAMINANT CONCENTRATIONS AT EXPOSURE POINTS

| Chemical | Release Medium | Exposure Point | Short-Term Concentration Best Estimate | Short-Term Concentration Upper Bound Estimate | Long-Term Concentration Best Estimate | Long-Term Concentration Upper Bound Estimate |
|---|---|---|---|---|---|---|
| 1. Benzene | Air | Nearest Residence* | 0.026 mg/m$^3$ | 0.50 | 0.0040 mg/m3 | 0.03 |
| | Ground water | Nearest Residence* | 0.20 mg/l | 8.0 | 0.0085 mg/l | 0.10 |
| 2. Lead | Ground water | Nearest Residence* | 0.045 mg/l | 2.0 | 0.0050 mg/l | 0.03 |
| | | | | | | |
| | | | | | | |
| 3. | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |

* Significant exposure point.

INSTRUCTIONS

1. List all indicator chemicals.
2. List all release media for each chemical: air, ground water, surface water, soil.
3. List all exposure points for each release medium. Indicate significant exposure point with an asterisk.
4. List projected short-term and long-term concentrations (best estimate and upper bound) for each exposure point, as calculated. Note that air concentrations are in mg/m3 units, water concentrations are in mg/l, and fish concentrations are in mg/kg. Attach all calculations documenting the concentration estimates to this worksheet.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

| CHEMICAL | SAFE DRINKING WATER ACT MCLs b/ (mg/l) | SAFE DRINKING WATER ACT MCLGs c/ (mg/l) | CLEAN AIR ACT NAAQS (ug/m3) |
|---|---|---|---|
| Arsenic | 0.05 | | |
| Barium | 1.0 | | |
| Benzene | | 0 | |
| Cadmium | 0.01 | | |
| Carbon monoxide | | | 40,000 (1-hour) d/<br>10,000 (8-hour) d/ |
| Carbon tetrachloride | | 0 | |
| Chlorophenoxys | | | |
| 2,4-Dichlorophenoxyacetic acid (2,4-D) | 0.1 | | |
| 2,4,5-Trichlorophenoxy-propionic acid (2,4,5-TP) | 0.01 | | |
| Chromium VI (hexavalent) | 0.05 | | |
| p-Dichlorobenzene | | 0.75 | |
| 1,2-Dichloroethane | | 0 | |
| 1,1-Dichloroethylene | | 0.007 | |
| Endrin | 0.0002 | | |
| Fluoride | 1.4-2.4 | | |
| Lindane (99% gamma-HCCH) | 0.004 | | |
| Hydrocarbons (non-methane) | | | 160 (3-hour) d/ |
| Lead | 0.05 | | 1.5 (90-day) e/ |
| Mercury | 0.002 | | |
| Methoxychlor | 0.1 | | |
| Nitrate (as N) | 10.0 | | |
| Nitrogen dioxide | | | 100 (1-year) f/ |
| Ozone | | | 235 (1-hour) d/ |
| Particulate Matter | | | 260 (24-hour) d/<br>75 (1-year) g/ |
| Radionuclides | | | |
| Radium-226 and 228 | 5 pCi/l | | |
| Gross alpha activity | 15 pCi/l | | |
| Tritium | 20,000 pCi/l | | |
| Strontium-90 | 8 pCi/l | | |
| Other man-made radionuclides | h/ | | |
| Selenium | 0.01 | | |
| Silver | 0.05 | | |
| Sulfur oxides | | | 365 (24-hour) d/<br>80 (1-year) f/ |

EXHIBIT 4-5
SELECTED APPLICABLE OR RELEVANT AND APPROPRIATE AMBIENT REQUIREMENTS

| CHEMICAL | SAFE DRINKING WATER ACT MCLs b/ (mg/l) | SAFE DRINKING WATER ACT MCLGs c/ (mg/l) | CLEAN AIR ACT NAAQS (ug/m3) |
|---|---|---|---|
| Toxaphene | 0.005 | | |
| 1,1,1-Trichloroethane | | 0.2 | |
| Trichloroethylene | | 0 | |
| Trihalomethanes (total) i/ | 0.1 | | |
| Vinyl chloride | | 0 | |

a/ Federal ambient water quality criteria (see Exhibit 4-6) and state environmental standards are also ARARs.

b/ EPA has also proposed MCLs for eight volatile organic chemicals: trichloroethylene, carbon tetrachloride, 1,1,1-trichloroethane, vinyl chloride, 1,2-dichloroethane, benzene, 1,1-dichloroethylene, and p-dichlorobenzene (50 Federal Register 46902-46933, November 13, 1985). Refer to Exhibit 4-7 for the proposed MCL values.

c/ EPA has also proposed MCLGs for 40 additional chemicals. Refer to Exhibit 4-7 for the proposed MCLG values.

d/ Maximum concentration not to be exceeded more than once per year.

e/ Three-month arithmetic mean concentration.

f/ Annual arithmetic mean concentration.

g/ Annual geometric mean concentration.

h/ Radionuclides in drinking water are limited to activity levels corresponding to a total body or any internal organ dose of 4 millirem/year, summed over all radionuclides present.

i/ Total trihalomethanes refers to the sum concentration of chloroform, bromodichloromethane, dibromochloromethane, and bromoform.

EXHIBIT 4-5 (CONTINUED)
SELECTED APPLICABLE OR RELEVANT AND APPROPRIATE AMBIENT REQUIREMENTS

| CHEMICAL | WQC (Concentrations in Parentheses Correspond to Midpoint of Risk Range for Potential Carcinogens Only) a/ | |
|---|---|---|
| | Aquatic Organisms and Drinking Water | Adjusted for Drinking Water Only b/ |
| Acenaphthene | 20 ug/l (Organoleptic) c/ | 20 ug/l (Organoleptic) |
| Acrolein | 320 ug/l | 540 ug/l |
| Acrylonitrile* | 0 (58 ng/l) | 0 (63 ng/l) |
| Aldrin* | 0 (0.074 ng/l) | 0 (1.2 ng/l) |
| Antimony* | 146 ug/l | 146 ug/l |
| Arsenic* | 0 (2.2 ng/l) | (25 ng/l) |
| Asbestos | 0 (30,000 fibers/l) | (30,000 fibers/l) |
| Benzene* | 0 (0.66 ug/l) | 0 (0.67 ug/l) |
| Benzidine* | 0 (0.12 ng/l) | 0 (0.15 ng/l) |
| Beryllium* | 0 (3.7 ng/l) | 0 (3.9 ng/l) |
| Cadmium* | 10 ug/l | 10 ug/l |
| Carbon tetrachloride* | 0 (0.4 ug/l) | 0 (0.42 ug/l) |
| Chlordane* | 0 (0.46 ng/l) | 0 (22 ng/l) |
| Chlorinated benzenes | | |
| Hexachlorobenzene* | 0 (0.72 ng/l) | 0 (21 ng/l) |
| 1,2,4,5-Tetrachlorobenzene* | 38 ug/l | 180 ug/l |
| Pentachlorobenzene* | 74 ug/l | 570 ug/l |
| Trichlorobenzene* | Insufficient data | Insufficient data |
| Monochlorobenzene* | 488 ug/l | 488 ug/l |
| Chlorinated ethanes | | |
| 1,2-Dichloroethane* | 0 (0.94 ug/l) | 0 (0.94 ug/l) |
| 1,1,1-Trichloroethane* | 18.4 mg/l | 19 mg/l |
| 1,1,2-Trichloroethane* | 0 (0.6 ug/l) | 0 (0.6 ug/l) |
| 1,1,2,2-Tetrachloroethane* | 0 (0.17 ug/l) | 0 (0.17 ug/l) |
| Hexachloroethane* | 0 (1.9 ug/l) | 0 (2.4 ug/l) |
| Monochloroethane | Insufficient data | Insufficient data |
| 1,1-Dichloroethane* | Insufficient data | Insufficient data |
| 1,1,1,2-Tetrachloroethane | Insufficient data | Insufficient data |
| Pentachloroethane | Insufficient data | Insufficient data |
| Chlorinated naphthalenes | Insufficient data | Insufficient data |
| Chlorinated phenols | | |
| 3-Monochlorophenol | 0.1 ug/l (Organoleptic) | 0.1 ug/l (Organoleptic) |
| 4-Monochlorophenol | 0.1 ug/l (Organoleptic) | 0.1 ug/l (Organoleptic) |
| 2,3-Dichlorophenol | 0.04 ug/l (Organoleptic) | 0.04 ug/l (Organoleptic) |
| 2,5-Dichlorophenol | 0.5 ug/l (Organoleptic) | 0.5 ug/l (Organoleptic) |
| 2,6-Dichlorophenol | 0.2 ug/l (Organoleptic) | 0.2 ug/l (Organoleptic) |
| 3,4-Dichlorophenol | 0.3 ug/l (Organoleptic) | 0.3 ug/l (Organoleptic) |
| 2,3,4,6-Tetrachlorophenol* | 1.0 ug/l (Organoleptic) | 1.0 ug/l (Organoleptic) |
| 2,4,5-Trichlorophenol* | 2600 ug/l | 2600 ug/l |

EXHIBIT 4-6
EPA AMBIENT WATER QUALITY CRITERIA (WQC) FOR PROTECTION OF HUMAN HEALTH

| CHEMICAL | WQC (Concentrations in Parentheses Correspond to Midpoint of Risk Range for Potential Carcinogens Only) a/ | |
|---|---|---|
| | Aquatic Organisms and Drinking Water | Adjusted for Drinking Water Only b/ |
| 2,4,6-Trichlorophenol* | 0 (1.2 ug/l) | 0 (1.8 ug/l) |
| 2-Methyl-4-chlorophenol | 1800 ug/l (Organoleptic) | 1800 ug/l (Organoleptic) |
| 3-Methyl-4-chlorophenol | 3000 ug/l (Organoleptic) | 3000 ug/l (Organoleptic) |
| 3-Methyl-6-chlorophenol | 20 ug/l (Organoleptic) | 20 ug/l (Organoleptic) |
| Chloroalkyl ethers | | |
| bis-(Chloromethyl) ether* | 0 (0.0038 ng/l) | 0 (0.0039 ng/l) |
| bis-(2-Chloroethyl) ether* | 0 (30 ng/l) | 0 (30 ng/l) |
| bis-(2-Chloroisopropyl) ether | 34.7 ug/l | 34.7 ug/l |
| Chloroform* | 0 (0.19 ug/l) | 0 (0.19 ug/l) |
| 2-Chlorophenol | 0.1 ug/l (Organoleptic) | 0.1 ug/l (Organoleptic) |
| Chromium Cr+6* | 50 ug/l | 50 ug/l |
| Cr+3* | 170 mg/l | 179 mg/l |
| Copper* | 1 mg/l (Organoleptic) | 1 mg/l (Organoleptic) |
| Cyanide* | 200 ug/l | 200 ug/l |
| DDT* | 0 (0.024 ng/l) | 0 (> 1.2 ng/l) |
| Dichlorobenzenes* (all isomers) | 400 ug/l | 470 ug/l |
| Dichlorobenzidines | 0 (10.3 ng/l) | 0 (20.7 ng/l) |
| Dichloroethylenes | | |
| 1,1-Dichloroethylene* | 0 (33 ng/l) | 0 (33 ng/l) |
| 1,2-Dichloroethylene | Insufficient data | Insufficient data |
| Dichloromethane* | See Halomethanes | See Halomethanes |
| 2,4-Dichlorophenol* | 3.09 mg/l | 3.09 mg/l |
| Dichloropropanes/Dichloropropenes | | |
| Dichloropropanes | Insufficient data | Insufficient data |
| Dichloropropenes | 87 ug/l | 87 ug/l |
| Dieldrin* | 0 (0.071 ng/l) | 0 (1.1 ng/l) |
| 2,4-Dimethylphenol | 400 ug/l (Organoleptic) | 400 ug/l (Organoleptic) |
| 2,4-Dinitrotoluene* | 0 (0.11 ug/l) | 0 (0.11 ug/l) |
| 1,2-Diphenylhydrazine* | 0 (42 ng/l) | 0 (46 ng/l) |
| Endosulfan* | 74 ug/l | 138 ug/l |
| Endrin | 1 ug/l | 1 ug/l |
| Ethylbenzene* | 1.4 mg/l | 2.4 mg/l |
| Fluoranthene | 42 ug/l | 188 ug/l |
| Haloethers | Insufficient data | Insufficient data |
| Halomethanes | 0 (0.19 ug/l) | 0 (0.19 ug/l) |
| Heptachlor* | 0 (0.28 ng/l) | 0 (11 ng/l) |
| Hexachlorobutadiene* | 0 (0.45 ug/l) | 0 (0.45 ug/l) |
| Hexachlorocyclohexanes (HCCH) | | |
| alpha-HCCH* | 0 (9.2 ng/l) | 0 (13 ng/l) |

EXHIBIT 4-6 (CONTINUED)
EPA AMBIENT WATER QUALITY CRITERIA (WQC) FOR PROTECTION OF HUMAN HEALTH

| CHEMICAL | WQC (Concentrations in Parentheses Correspond to Midpoint of Risk Range for Potential Carcinogens Only) a/ | |
|---|---|---|
| | Aquatic Organisms and Drinking Water | Adjusted for Drinking Water Only b/ |
| beta-HCCH* | 0 (16.3 ng/l) | 0 (23.2 ng/l) |
| gamma-HCCH* | 0 (12.3 ng/l) | 0 (17.4 ng/l) |
| delta-HCCH | Insufficient data | Insufficient data |
| epsilon-HCCH | Insufficient data | Insufficient data |
| Technical-HCCH | 0 (5.2 ng/l) | 0 (7.4 ng/l) |
| Hexachlorocyclopentadiene* | 206 ug/l | 206 ug/l |
| Isophorone* | 5.2 mg/l | 5.2 mg/l |
| Lead* | 50 ug/l | 50 ug/l |
| Mercury* | 144 ng/l | 10 ug/l |
| Naphthalene | Insufficent data | Insufficient data |
| Nickel* | 13.4 ug/l | 15.4 ug/l |
| Nitrobenzene* | 19.8 mg/l | 19.8 mg/l |
| Nitrophenols | | |
| 2,4-Dinitro-o-cresol | 13.4 ug/l | 13.6 ug/l |
| Dinitrophenol* | 70 ug/l | 70 ug/l |
| Mononitrophenol | Insufficient data | Insufficient data |
| Trinitrophenol | Insufficient data | Insufficient data |
| Nitrosamines | | |
| n-Nitrosodimethylamine* | 0 (1.4 ng/l) | 0 (1.4 ng/l) |
| n-Nitrosodiethylamine* | 0 (0.8 ng/l) | 0 (0.8 ng/l) |
| n-Nitrosodi-n-butylamine* | 0 (6.4 ng/l) | 0 (6.4 ng/l) |
| n-Nitrosodiphenylamine | 0 (4.9 ug/l) | 0 (7.0 ug/l) |
| n-Nitrosopyrrolidine* | 0 (16 ng/l) | 0 (16 ng/l) |
| Pentachlorophenol* | 1.01 mg/l | 1.01 mg/l |
| Phenol* | 3.5 mg/l | 3.5 mg/l |
| Phthalate esters | | |
| Dimethylphthalate | 313 mg/l | 350 mg/l |
| Diethylphthalate* | 350 mg/l | 434 mg/l |
| Dibutylphthalate* | 34 mg/l | 44 mg/l |
| Di-2-ethylhexylphthalate* | 15 mg/l | 21 mg/l |
| Polychlorinated biphenyls (PCBs)* | 0 (0.079 ng/l) | 0 (> 12.6 ng/l) |
| Polynuclear aromatic hydrocarbons (PAHs)* | 0 (2.8 ng/l) | 0 (3.1 ng/l) |
| Selenium* | 10 ug/l | 10 ug/l |
| Silver* | 50 ug/l | 50 ug/l |
| 2,3,7,8-TCDD* | 0 (0.000013 ng/l) | 0 (0.00018 ng/l) |
| Tetrachloroethylene* | 0 (0.8 ug/l) | 0 (0.88 ug/l) |
| Thallium* | 13 ug/l | 17.8 ug/l |

EXHIBIT 4-6 (CONTINUED)
EPA AMBIENT WATER QUALITY CRITERIA (WQC) FOR PROTECTION OF HUMAN HEALTH

| CHEMICAL | WQC (Concentrations in Parentheses Correspond to Midpoint of Risk Range for Potential Carcinogens Only) a/ | |
|---|---|---|
| | Aquatic Organisms and Drinking Water | Adjusted for Drinking Water Only b/ |
| Toluene* | 14.3 mg/l | 15 mg/l |
| Toxaphene* | 0 (0.71 ng/l) | 0 (26 ng/l) |
| Trichloroethylene* | 0 (2.7 ug/l) | 0 (2.8 ug/l) |
| Vinyl chloride* | 0 (2.0 ug/l) | 0 (2.0 ug/l) |
| Zinc* | 5 mg/l (Organoleptic) | 5 mg/l (Organoleptic) |

* Toxicity values necessary for risk characterization are given in Appendix A.

a/ The criterion value, which is zero for all potential carcinogens, is listed for all chemicals in the table. The concentration value given in parentheses for potential carcinogens corresponds to a risk of $10^{-6}$, which is the midpoint of the range of $10^{-5}$ to $10^{-7}$ given in water quality criteria documents. To obtain concentrations corresponding to risks of $10^{-5}$, the $10^{-6}$ concentrations should be multiplied by 10. To obtain concentrations corresponding to risks of $10^{-7}$, the $10^{-6}$ concentrations should be divided by 10.

b/ These adjusted criteria, for drinking water ingestion only, were derived from published EPA ambient water quality criteria (45 Federal Register 79318-79379, November 28, 1980) for combined fish and drinking water ingestion and for fish ingestion alone. The adjusted values are not official EPA ambient water quality criteria, but may be appropriate for Superfund sites with contaminated ground water. In the derivation of these values, intake was assumed to be 2 liters/day for drinking water and 6.5 grams/day for fish, and human body weight was assumed to be 70 kilograms. Values for bioconcentration factor, carcinogenic potency, and acceptable daily intake were those used for water quality criteria development.

c/ Criteria designated as organoleptic are based on taste and odor effects, not human health effects. Health-based water quality criteria are not available for these chemicals.

EXHIBIT 4-6 (CONTINUED)
EPA AMBIENT WATER QUALITY CRITERIA (WQC) FOR PROTECTION OF HUMAN HEALTH

contamination with possible exposure via ingestion of aquatic organisms, federal ambient water quality criteria may be appropriate. ARARs should correspond to the medium (e.g., air, water) for which they were developed and must be applicable or relevant and appropriate to site conditions. If requirements are available for all indicator chemicals, but are not appropriate to site exposure conditions, a full risk characterization should be completed.

Use Worksheet 4-5 to compare ARARs to environmental concentrations projected for exposure points. Calculate ratios between predicted concentrations and requirements, and designate whether concentrations exceed or fall below the requirements. Also, when risk levels associated with these requirements are known, they should be recorded. This information will be carried through to the end of the process and included in summary tables for the baseline public health evaluation. Factors in the development of the requirements listed in Exhibits 4-5 and 4-6 are discussed briefly in the following sections.

#### 4.3.1.1 Maximum Contaminant Levels (MCLs) and Maximum Contaminant Level Goals (MCLGs)

Drinking water standards under the Safe Drinking Water Act are promulgated as maximum contaminant levels (MCLs). MCLs are currently available for 16 specific chemicals (10 inorganics and 6 organic pesticides), total trihalomethanes (covers four chemicals), certain radionuclides, and microorganisms (40 CFR 141). Under the Safe Drinking Water Act amendments of 1986 (P.L. 99-339), EPA is required to promulgate MCLs for 83 contaminants within three years. Generally, an MCL for a toxic chemical represents the allowable lifetime exposure to the contaminant for a 70 kg adult who is assumed to ingest two liters of water per day. Total environmental exposure of a particular contaminant from various sources was considered in calculating specific MCLs. EPA estimated the amount of the substance to which the average person is likely to be exposed from all sources (e.g., air, food, water) and then determined the fraction of the total intake resulting from drinking water ingestion. Lifetime exposure limits were set at the lowest practical level to minimize the amount of contamination ingested from water, especially when exposure from other sources is large. The MCL calculation is adjusted by an exposure factor to reflect gastrointestinal absorption associated with water consumption.

In addition to health factors, an MCL is required by law to reflect the technological and economic feasibility of removing the contaminant from the water supply. The limit set must be feasible given the best available technology and treatment techniques. A safety factor is included in each of the standards to provide adequate protection for sensitive populations that may be at special risk such as infants and children. Safety factors vary from chemical to chemical because of the different risks associated with each.

As part of the process for developing final drinking water standards (i.e., MCLs), EPA develops maximum contaminant level goals (MCLGs).[18] MCLGs are entirely health-based; thus, they are always less than or equal to MCLs. EPA recently promulgated MCLGs for eight volatile organic chemicals (40 CFR 141.50; 50 *Federal Register* 46880-46901, November 13, 1985). Exhibit 4-5 lists the MCLs and MCLGs promulgated as of publication of this manual.

#### 4.3.1.2 National Ambient Air Quality Standards (NAAQS)

NAAQS are available for six chemicals or chemical groups and for airborne particulates; of these, the NAAQS for lead, hydrocarbons, and airborne particulates appear to be most useful for Superfund public health evaluations. In the development of primary NAAQS[19], sources of the contaminant that contribute to air pollution and all sources of exposure to the contaminant (e.g, food, water, air) are considered in determining the health risk. In addition, the statute states that primary NAAQS must be based exclusively on air quality criteria issued by EPA for each air pollutant. The Act does not require EPA to consider the costs (economics) of achieving the standards or the technological feasibility of implementing the standards. Standards can be promulgated as annual maximums, annual geometric means, annual arithmetic means, or for other time periods that vary from one hour to one year depending on the pollutant.

Primary standards must allow for an adequate margin of safety to account for unidentified hazards and effects. There is no rule used in setting the margin of safety for the standards. The law requires EPA to direct its efforts at groups of particularly sensitive citizens, such as bronchial asthmatics and emphysematics. In developing primary NAAQS, EPA must specify the nature and severity of the health effects of each contaminant, characterize the sensitive population involved, determine probable adverse health effect levels in sensitive persons,

[18] MCLGs were formerly known as recommended maximum contaminant levels (RMCLs).

[19] EPA also develops secondary NAAQS under the Clean Air Act to protect the public welfare from known or anticipated effects.

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 4-5

COMPARISON OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS TO ESTIMATED EXPOSURE POINT CONCENTRATIONS

Exposure Point: Private Drinking Water Wells at Nearest Residences

| | Chemical | Applicable/Relevant and Appropriate Requirement Being Compared | Value of Requirement/ Standard | Projected Exposure Point Concentration | Concentration: Standard Ratio |
|---|---|---|---|---|---|
| 1. | Lead | Drinking Water MCL | 0.05 mg/l | 0.045 mg/l (STC) | 0.9 |
| 2. | | | | | |
| 3. | | | | | |
| 4. | | | | | |

INSTRUCTIONS

1. List all indicator chemicals.
2. Indicate the identity of applicable or relevant and appropriate requirements (e.g., primary drinking water MCLs).
3. Obtain values for requirements from Exhibits 4-5 and 4-6 or, for state environmental standards, from the appropriate state agency.
4. Obtain the exposure point concentrations to be compared from Worksheet 4-4. Identify the values as short-term concentrations (STC) or long-term concentrations (LTC).
5. Record the ratios between exposure point concentrations and requirements; ratios greater than 1.0 indicate exceedance of the requirement.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 4-5
COMPARISON OF APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS TO ESTIMATED EXPOSURE POINT CONCENTRATIONS

and estimate the level below which an adequate margin of safety reduces or eliminates risks. Primary NAAQS are based for the most part on the direct health effects of chemicals to sensitive groups.

#### 4.3.1.3 Federal Ambient Water Quality Criteria

Federal ambient water quality criteria for the protection of human health have been developed for 62 out of 65 classes of toxic pollutants (a total of 95 individual chemicals have numerical health criteria). The health-based water quality criterion is an estimate of the ambient surface water concentration that will not result in adverse health effects in humans. In the case of suspect or proven carcinogens, concentrations associated with a

range of incremental cancer risks are provided to supplement a criterion of zero. The federal criteria are non-enforceable guidelines, which many states have used in the development of enforceable ambient water quality standards (see Section 4.3.1.4). Exhibit 4-6 lists federal ambient water quality criteria for specific chemicals.

For most chemicals, federal water quality criteria to protect human health are available for two different exposure pathways. One criterion is based on lifetime ingestion of both drinking water and aquatic organisms, and the other is based on lifetime ingestion of aquatic organisms alone. The calculations incorporate the assumption that a 70-kilogram adult consumes 2 liters of water and/or 6.5 grams of aquatic organisms daily for a 70-year lifetime. Of course, calculations can be made to derive an adjusted criterion for drinking water ingestion only, based on the two published criteria and the same intake assumptions (as was done for Exhibit 4-6). These adjusted criteria are more appropriate than non-adjusted criteria for Superfund sites with contamination of potential ground-water sources of drinking water because they are based on more realistic exposure assumptions (i.e., exclusion of aquatic organism ingestion as an exposure pathway).

Derivation of Criteria for Noncarcinogens. On the basis of a survey of the toxicology literature, EPA established a "no observed adverse effect level" (NOAEL) for each chemical. The NOAELs were usually based on animal studies, although human data were used whenever available. By applying a safety factor to account for the uncertainty in using available data to estimate human effects, an acceptable daily intake (ADI) was determined. Criteria (i.e., water concentrations) were then derived from the ADIs and the standard intake assumptions given above.

Derivation of Criteria for Carcinogens. The same exposure and intake assumptions were used for potential carcinogens. A literature search for human and animal carcinogenic effects formed the basis for EPA's estimate of the risk posed by potential human carcinogens. Because methods are not currently available to establish the presence of a threshold for carcinogenic effects, the criteria for all carcinogens state that the recommended concentration for maximum protection of human health is zero. EPA also estimated water concentrations corresponding to incremental risk levels, using a linear, non-threshold extrapolation model. Extrapolation models provide only an estimate of risk, but they represent the best available tool for describing the potential threat of a substance, given certain assumptions. In its published criteria, EPA provides water concentrations corresponding to incremental lifetime cancer risks of $10^{-7}$, $10^{-6}$, and $10^{-5}$.

#### 4.3.1.4 State Environmental Standards

State environmental standards are ARARs for Superfund remedial actions in that state. The availability of and numerical values for these standards vary widely from state to state. The remedial project manager is responsible for determining the availability of applicable or relevant and appropriate state standards for a site.

Water quality standards developed under the Clean Water Act are a commonly available type of state standard. These standards serve the dual purposes of establishing the water quality goals for a specific water body and as the regulatory basis for establishing water quality-based controls beyond the technology-based levels of treatment required by Sections 301(b) and 306 of the Clean Water Act. Water quality standards are adopted by states (or, where necessary, promulgated by EPA) to protect the public health or welfare, enhance the quality of the water, and serve the purposes of the Act. A water quality standard consists of basically two parts: (1) a "designated use" (or uses), which considers the water body's use and value for public water supplies, for propagation of fish, shellfish, and wildlife, and for recreational, navigation, agricultural, industrial, and other purposes; and (2) "criteria", which are numerical limits or narrative statements necessary to protect the designated use.

States must adopt appropriate water quality criteria sufficiently stringent to protect the designated uses. Numerical criteria may be based on ambient water quality criteria recommendations published by EPA (see Section 4.3.1.3) or developed by other scientifically defensible methods. States may also modify EPA's recommended criteria to reflect local environmental conditions and human exposure patterns before incorporation into water quality standards. When a criterion for the protection of human health must be developed for a chemical for which a national criterion has not been recommended, the state should consult EPA headquarters for assistance. Guidelines for deriving

human health-based water quality criteria were published on November 28, 1980 (EPA, 1980).

### *4.3.2 Compare to Other Criteria, Advisories, and Guidance*

In the absence of ARARs for all indicator chemicals, the remainder of the process as outlined in Chapters 5 through 7 should be completed. In addition, information on how exposure point concentrations compare to "other criteria, advisories, and guidance" (i.e., not ARARs) is useful as a supplement to the risk assessment and should be noted in the public health evaluation chapter in the feasibility study report. At sites where neither ARARs or appropriate toxicity values are available for some indicator chemicals, the comparison of ambient concentrations to other criteria may provide an important basis on which to judge the potential health effects of environmental concentrations of toxic substances.

For the purposes of Superfund public health evaluations, EPA considers drinking water health advisories and proposed drinking water standards to be pertinent for comparison with predicted concentrations, provided they are for the same exposure pathway. Exhibit 4-7 lists proposed MCLs and MCLGs and Exhibit 4-8 lists health advisories. Other standards may be used for comparison as well, provided they correspond to the environmental medium for which they were designed and are appropriate to site conditions. Criteria inappropriate for public health evaluation of long-term chemical exposures, such as $LD_{50}$ values and unadjusted occupational threshold limit values (TLVs), should not be used in this comparison.[20]

Some ambient concentration requirements or criteria will be pertinent to specific site conditions, while others can be adjusted to make them useful. For example, if a requirement applies to a different environmental medium or exposure route than the one threatened by a site, it would probably not be appropriate to use it without adjustment. As an illustration of this, ambient water quality criteria, which were developed for surface water, can be adjusted for ground water by recalculating without the assumption of fish ingestion (as in Exhibit 4-6). Concentration requirements and criteria may also be based on a different level, frequency, or duration of exposure than found at a specific site. Guidance on adjustment of standards for site-specific applications is currently under development by EPA.

For some chemicals several "other criteria, advisories, and guidance" may be available as comparison values. In this case choose the most suitable value for comparison. Suitability is determined in part by the pertinence of the criterion to exposure conditions at the site (e.g., exposed population characteristics, duration and timing of exposure, exposure pathways) and in part by how recently the value was developed. Some criteria have been developed recently and may reflect new information compared to older values. Some standards or criteria may have been scrutinized more closely than others and may consequently have more scientific credibility. Other standards may be current and scientifically accepted but not pertinent to exposure routes at the site and therefore unsuitable. Consequently, the most suitable comparison value is the most current, credible, and pertinent value available.

Use Worksheet 4-6 to compare "other criteria, advisories, and guidance" to environmental concentrations projected for exposure points. Calculate the ratios between predicted concentrations and requirements and be sure to designate whether concentrations exceed or fall below the requirements. This information will be carried through to the end of the process and included in summary tables for the baseline public health evaluation. The criteria and advisories in Exhibits 4-7 and 4-8 are discussed briefly in the following sections.

#### 4.3.2.1 Proposed MCLs and MCLGs

EPA has proposed MCLs for the same eight volatile organic chemicals for which final MCLGs were promulgated (50 *Federal Register* 46902-46933, November 13, 1985), and has proposed MCLGs for a larger group of inorganic chemicals, synthetic organic chemicals, and microorganisms (50 *Federal Register* 46936-47022, November 13, 1985). Exhibit 4-7 lists values for both proposed MCLs and proposed MCLGs. In general, proposed requirements, including proposed MCLs and MCLGs, should be used in the same manner as "other criteria, advisories, and guidance" (as defined in the CERCLA compliance with other environmental statutes policy memorandum; see Section 2.3). It should be recognized, however, that proposed requirements can be changed before they are promulgated; thus, final requirements may differ from proposed ones. After a proposed requirement that falls in the ARAR category becomes final, it should be added to the active list of ARARs.

[20] LD50 values and TLVs usually reflect short-term exposures. LD50 ("lethal dose-50") is the dose of a chemical that is fatal in 50 percent of the exposed population. TLVs are time-weighted average concentrations of chemicals in air that should not be exceeded for a given time period (usually 15 minutes or 8 hours).

| CHEMICAL | PROPOSED MCL (mg/l) a/ | PROPOSED MCLG (mg/l) b/ |
|---|---|---|
| Acrylamide | | 0 |
| Alachlor | | 0 |
| Aldicarb | | 0.009 |
| Aldicarb sulfoxide | | 0.009 |
| Aldicarb sulfone | | 0.009 |
| Arsenic | | 0.05 |
| Asbestos | | 7.1 c/ |
| Barium | | 1.5 |
| Benzene | 0.005 | |
| Cadmium | | 0.005 |
| Carbofuran | | 0.036 |
| Carbon tetrachloride | 0.005 | |
| Chlordane | | 0 |
| Chromium | | 0.12 |
| Copper | | 1.3 |
| Dibromochloropropane | | 0 |
| o-Dichlorobenzene | | 0.62 |
| p-Dichlorobenzene | 0.75 | |
| 1,2-Dichloroethane | 0.005 | |
| 1,1-Dichloroethylene | 0.007 | |
| 1,2-cis-Dichloroethylene | | 0.07 |
| 1,2-trans-Dichloroethylene | | 0.07 |
| 1,2-Dichloropropane | | 0.006 |
| 2,4-D | | 0.07 |
| Epichlorohydrin | | 0 |
| Ethylbenzene | | 0.68 |
| Ethylene dibromide (EDB) | | 0 |
| Heptachlor | | 0 |
| Heptachlor epoxide | | 0 |
| Lead | | 0.02 |
| Lindane | | 0.0002 |
| Mercury | | 0.003 |
| Methoxychlor | | 0.34 |
| Monochlorobenzene | | 0.06 |
| Nitrate | | 10 |
| Nitrite | | 1 |
| Polychlorinated biphenyls | | 0 |
| Pentachlorophenol | | 0.22 |
| Selenium | | 0.045 |
| Styrene | | 0.14 |
| Tetrachloroethylene | | 0 |
| 1,1,1-Trichloroethane | 0.2 | |

EXHIBIT 4-7
EPA PROPOSED MCLs AND MCLGs

| CHEMICAL | PROPOSED MCL (mg/l) a/ | PROPOSED MCLG (mg/l) b/ |
|---|---|---|
| Trichloroethylene | 0.005 | |
| Toluene | | 2 |
| Toxaphene | | 0 |
| 2,4,5-TP | | 0.052 |
| Vinyl chloride | 0.001 | |
| Xylene | | 0.44 |

a/ MCL = maximum contaminant level; proposed values taken from 50 Federal Register 46902 (November 13, 1985).

b/ MCLG = maximum contaminant level goal; proposed values taken from 50 Federal Register 46936 (November 13, 1985).

c/ Million fibers per liter.

EXHIBIT 4-7 (CONTINUED)
EPA PROPOSED MCLs AND MCLGs

EXHIBIT 4-8

EPA DRINKING WATER HEALTH ADVISORIES

| CHEMICAL | One-day (ug/l) 10 kg | Ten-day (ug/l) 10 kg | Longer-term a/ (ug/l) 10 kg | Longer-term a/ (ug/l) 70kg | Lifetime (ug/l) 70 kg | Reference Concentration for Potential Carcinogens b/ (ug/l) 70 kg |
|---|---|---|---|---|---|---|
| Acrylamide | 1500 | 300 | 20 | 70 | -- | 0.01 |
| Alachlor | 15000 | 15000 | NA | NA | NA | 0.15 |
| Aldicarb* | 12 | 12 | 12 | 42 | 42 | NA |
| Arsenic* | 50 | 50 | 50 | 50 | 50 | 0.0022 |
| Barium* | -- | -- | -- | -- | 1800 | NA |
| Benzene* | 233 | 233 | NA | NA | NA | 0.35 |
| Cadmium* | 43 | 8 | 5 | 18 | 18 | NA |
| Carbofuran | 50 | 50 | 50 | 180 | 180 | NA |
| Carbon Tetrachloride* | 4000 | 160 | 71 | 250 | -- | 0.3 |
| Chlordane* | 63 | 63 | -- | -- | -- | 0.0218 |
| Chlorobenzene* | 1800 | 1800 | 9000 | 30000 | 3150 | NA |
| Chromium* | 1400 | 1400 | 240 | 840 | 170 | NA |
| Cyanide* | 220 | 220 | 220 | 750 | 750 | NA |
| 2,4-D | 1100 | 300 | -- | -- | 350 | NA |
| Dibromochloropropane | 200 | 50 | NA | NA | NA | 0.025 |
| o-/m-Dichlorobenzene* | 8930 | 8930 | 8930 | 31250 | 3125 | NA |
| p-Dichlorobenzene | 10700 | 10700 | 10700 | 37500 | 3750 | NA |
| 1,2-Dichloroethane* | 740 | 740 | 740 | 2600 | NA | 0.95 |
| 1,1-Dichloroethylene* | 1000 | 1000 | 1000 | 3500 | -- | 0.24 |
| cis-1,2-Dichloroethylene | 4000 | 1000 | 1000 | 3500 | 350 | NA |
| trans-1,2-Dichloroethylene | 2720 | 1000 | 1000 | 3500 | 350 | NA |

EXHIBIT 4-8
EPA DRINKING WATER HEALTH ADVISORIES

EXHIBIT 4-8 (CONTINUED)
EPA DRINKING WATER HEALTH ADVISORIES

| CHEMICAL | One-day (ug/l) 10 kg | Ten-day (ug/l) 10 kg | Longer-term a/ (ug/l) 10 kg | Longer-term a/ (ug/l) 70kg | Lifetime (ug/l) 70 kg | Reference Concentration for Potential Carcinogens b/ (ug/l) 70 kg |
|---|---|---|---|---|---|---|
| Dichloromethane* | 13300 | 1500 | -- | -- | -- | 5 |
| 1,2-Dichloropropane | -- | 90 | -- | -- | -- | 0.56 |
| p-Dioxane | 5680 | 568 | -- | -- | -- | -- |
| Dioxin* | 0.001 | 0.0001 | 0.00001 | 0.000035 | -- | 2.2 x 10-7 |
| Endrin | 20 | 5 | 4.5 | 16 | 1.6 | NA |
| Epichlorohydrin* | 140 | 140 | 76 | 76 | -- | 3.54 |
| Ethylbenzene* | 21000 | 2100 | -- | -- | 3400 | NA |
| Ethylene Dibromide* | 8 | 8 | NA | NA | NA | 0.0005 |
| Ethylene Glycol* | 19000 | 5500 | 5500 | 19250 | -- | NA |
| Heptachlor* | 10 | 10 | -- | -- | -- | 0.0104 |
| Heptachlor Epoxide* | -- | -- | -- | -- | -- | 0.0006 |
| Hexachlorobenzene* | 50 | 50 | 50 | 175 | -- | 0.02 |
| n-Hexane | 13000 | 4000 | 4000 | 14000 | -- | NA |
| Lead* | -- | -- | 20 ug/day | 20 ug/day | 20 ug/day | 0.031 |
| Lindane* | 1200 | 1200 | 33 | 120 | -- | 0.02655 |
| Mercury* | -- | -- | -- | -- | 5.5 | NA |
| Methoxychlor | 6400 | 2000 | -- | -- | 1700 | NA |
| Methyl Ethyl Ketone* | 75000 | 7500 | 2500 | 8600 | 860 | NA |
| Nickel* | -- | 1000 | -- | -- | 350 | NA |
| Nitrate c/ | 10000 (4 kg)<br>111000 (10 kg) | 10000 (4 kg)<br>111000 (10 kg) | -- | -- | 10000 | NA |
| Nitrite c/ | 1000 (4 kg)<br>11000 (10 kg) | 1000 (4 kg)<br>11000 (10 kg) | -- | -- | 1000 | NA |

## EXHIBIT 4-8 (CONTINUED)
## EPA DRINKING WATER HEALTH ADVISORIES

| CHEMICAL | One-day (ug/l) 10 kg | Ten-day (ug/l) 10 kg | Longer-term a/ (ug/l) 10 kg | Longer-term a/ (ug/l) 70kg | Lifetime (ug/l) 70 kg | Reference Concentration for Potential Carcinogens b/ (ug/l) 70 kg |
|---|---|---|---|---|---|---|
| Oxamyl | 350 | 350 | -- | -- | 810 | NA |
| PCBs* | -- | -- | -- | -- | -- | -- |
| Pentachlorophenol* | 1000 | 300 | 300 | 1050 | 1050 | NA |
| Styrene* | 27000 | 20000 | 20000 | 70000 | -- | 0.014 |
| Tetrachloroethylene* | -- | 34000 | 1940 | 6800 | -- | 0.7 |
| Toluene* | 18000 | 6000 | -- | -- | 10800 | NA |
| Toxaphene* | 500 | 80 | -- | -- | -- | 0.031 |
| 2,4,5-TP* | 200 | 200 | -- | -- | 260 | NA |
| 1,1,1-Trichloroethane* | 140000 | 35000 | 35000 | 125000 | 1000 | 22000 |
| Trichloroethylene* | -- | -- | -- | -- | -- | 2.8 |
| Vinyl Chloride* | 2600 | 2600 | 13 | 46 | NA | 0.015 |
| Xylenes* | 12000 | 7800 | 7800 | 27300 | 2200 | NA |

* Toxicity values necessary for risk characterization are given in Appendix C.

a/ Longer term health advisories are for exposures ranging from several months to several years and should generally be compared only to estimated short-term concentrations (SIC).

b/ The concentration given corresponds to a potential carcinogenic risk of 10-6. To obtain concentrations corresponding to risks of 10-4 and 10-5, the 10-6 concentrations should be multiplied by 100 and 10, respectively. To obtain concentrations corresponding to risks of 10-7, the 10-6 concentrations should be divided by 10.

c/ The one- and ten-day health advisories for nitrate and nitrite are given for both a 4 kg newborn and a 10 kg infant.

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 4-6

COMPARISON OF OTHER FEDERAL AND STATE CRITERIA
TO ESTIMATED EXPOSURE POINT CONCENTRATIONS

Exposure Point: Private Drinking Water Wells at Nearest Residences

| | Chemical | App/Rel Requirement Available | Criterion Being Compared | Value of Criterion | Projected Exposure Point Concentration | Concentration: Standard Ratio |
|---|---|---|---|---|---|---|
| 1. | Benzene | No | Drinking Water Reference Concentration for Potential Carcinogenic Effects (Health Advisory Summary, Exhibit 4-8) | 0.00035 mg/l* | 0.0085 mg/l (LTC) | 24 |
| 2. | | | | | | |
| 3. | | | | | | |
| 4. | | | | | | |

* Reference concentration listed corresponds to 10-6 potential carcinogenic risk.

INSTRUCTIONS

1. List all indicator chemicals and designate for each whether it was compared to an applicable or relevant and appropriate requirement in Worksheet 4-5.
2. For each chemical identify the criterion/criteria being compared. In general each chemical should be compared to the criteria/criterion most appropriate to exposure conditions at the site.
3. Obtain values for criteria from Exhibit 4-7, 4-8, or other sources.
4. Obtain the exposure point concentrations to be compared from Worksheet 4-4 and identify each value as a short-term concentration (STC) or long-term concentration (LTC).
5. Record the ratios between exposure point concentrations and criteria; ratios greater than 1.0 indicate exceedance of the criterion.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 4-6
COMPARISON OF OTHER FEDERAL AND STATE CRITERIA TO ESTIMATE EXPOSURE POINT CONCENTRATIONS

#### 4.3.2.2 Drinking Water Health Advisories

In addition to MCLs and MCLGs, EPA provides drinking water suppliers with guidance on various chemicals that may be encountered in a water system. The Office of Drinking Water's nonregulatory health advisories are concentrations of contaminants in drinking water at which adverse effects would not be anticipated to occur. A margin of safety is included to protect sensitive members of the population. The health advisory numbers are developed from data describing noncarcinogenic end-points of toxicity. They do not incorporate quantitatively any potential carcinogenic risk from such exposure. The Office of Drinking Water has recently developed health advisories for 54 chemicals or chemicals groups, and these values are summarized in Exhibit 4-8.

Under certain circumstances and when the appropriate toxicological data are available, health advisories may be developed for one-day, ten-day, longer-term (several months to several years), and lifetime durations of exposure. One-day and ten-day health advisories are calculated for a 10 kg child (a one-year old infant) assumed to drink one liter of water per day. Lifetime health advisories are calculated for a 70 kg adult, assumed to drink two liters of water per day. Longer-term health advisories are calculated for both a 10 kg child and a 70 kg adult. For chemicals that are known or probable human carcinogens according to the proposed Agency classification scheme, non-zero one-day, ten-day, and longer-term health advisories may be derived, with attendant caveats. Health advisories for lifetime exposures are not recommended for this group of substances. For these potential carcinogens, drinking water concentrations associated with projected upper 95 percent con- fidence limit excess lifetime cancer risk of $10^{-6}$ are provided. Comparison of these values to measured or predicted drinking water concentrations can give an indication of the magnitude of potential carcinogenic risk.

* * * * *

This chapter, in conjunction with the Superfund Exposure Assessment Manual, has presented instructions for estimating exposure point concentrations of the indicator chemicals selected in Chapter 3. Important exposure pathways have been identified. Ambient concentrations of the indicator chemicals have been modeled from the point of release to the point of human exposure for important exposure pathways, and these estimated concentrations have been compared to applicable or relevant and appropriate requirements and other federal criteria, advisories, and guidance. If all indicator chemicals have applicable or relevant and appropriate requirements, the baseline public health evaluation is now complete. In this case, proceed to Chapter 8 to begin the analysis of remedial alternatives. Otherwise, the exposure point concentrations estimated here will be used in Chapter 5 to calculate chemical intakes, which subsequently will be used to estimate risk.

# CHAPTER 5
# STEP 3: ESTIMATION OF CHEMICAL INTAKES

To assess the potential adverse health effects associated with a site, the amount of human exposure to the selected contaminants must be determined. In this chapter, methods are presented for estimating human exposures using the environmental concentrations of substances that were estimated by the methods described in Chapter 4 and the Superfund Exposure Assessment Manual.

Human exposure is expressed in terms of intake, which is the amount of substance taken into the body per unit body weight per unit time.[21] Intakes are calculated separately for exposures to chemical contaminants in each environmental medium -- air, ground water, surface water, and soil. Then, for each exposed population-at-risk, intakes for the same route of exposure are summed, resulting in a total oral exposure and total inhalation exposure. Dermal exposure, if determined to be important, should be estimated separately. Exhibit 5-1 is an overview of the intake estimation step.

Because short-term (subchronic) exposures to relatively high concentrations of chemical contaminants can cause different toxic effects than those caused by long-term (chronic) exposures to lower concentrations, two intake levels are calculated for each chemical -- the subchronic daily intake (SDI) and the chronic daily intake (CDI). These calculated intakes are based on short-term and long-term concentrations derived for each chemical using the procedures in the preceding chapter. All intakes are expressed in mg/kg/day.

In circumstances where contamination already has reached a point of human exposure, intake calculations may be made based on personal air monitoring and body burden analysis data for exposed individuals. All human subject monitoring and assessment should be coordinated with the Agency for Toxic Substances and Disease Registry, Department of Health and Human Services. Results should be reported directly on Worksheets 5-1 through 5-4.

The sections that follow give standard methods to estimate human intakes through air, ground water, and surface water. If other exposure routes, such as dermal absorption and soil ingestion are important, contact the Exposure Assessment Group, Office of Research and Development, U.S. EPA, Washington, D.C. 20460, for additional guidance. Standard intake assumptions are given in Exhibit 5-2. **If more accurate site-specific information is available, it can be used to give a better representation of risk at the site.** See Exhibit 5-2 for an example of how to use the standard assumptions and how to make adjustments based on more accurate intake and body weight information for the exposed population. For example, higher than average fish consumption may be important for some sites where surface water contamination is a problem. In addition, the standard intake values do not account for reduced intakes resulting from human activity patterns that reduce human contact with the contamination (i.e., it is assumed that exposure occurs 24 hours per day for the entire period that contamination is present). This conservative approach can be modified based on site-specific information to the contrary. For example, if an industrial area is an inhalation exposure point, it may be appropriate to adjust the standard intake factor by the fraction of a year spent at the exposure point.

Worksheets are provided as a method of organizing information and keeping track of intake calculations. However, they will not generally be required as part of the final report. Only Worksheets 5-5, 5-6, and 5-7, the summary worksheets, will be required for submission with the final report.

## 5.1 Calculate Air Intakes

Human intake of contaminants present in the air is dependent on the contaminant concentration, the frequency and volume of inhalation, the duration of exposure, and in the case of particulates, particle size.

The measured or predicted atmospheric concentrations (short-term and long-term) of each contaminant at specific exposure points are given in Worksheet 4-4. Insert these values into the appropriate columns of Worksheet 5-1. Note that a separate worksheet must be prepared for each inhalation exposure point.

A standard human intake coefficient has been calculated for use in determining air exposures in the absence of more accurate site-specific intake

[21] The term intake is used instead of dose because the information required to estimate dose is often unavailable. To estimate dose, information indicating the amount of a chemical that may be absorbed (e.g., across lung or gastrointestinal tract lining or through the skin) and subsequently distributed to target organs or tissues would be needed. When absorption data are available they can be incorporated into the assessment. Because adequate absorption data for specific chemicals are relatively rare, they cannot be used consistently and are not included here.

**Adjust Standard Intake Assumptions for Site-Specific Factors, if Appropriate**

**Combine Adjusted Assumptions with Projected Chemical Concentrations to Estimate Intakes for Individual Exposure Routes**

**Sum Intakes Across Exposure Routes, as Appropriate**

EXHIBIT 5-1
OVERVIEW OF STEP 3: ESTIMATING HUMAN INTAKES

information. This value takes into account the frequency (breathing rate), volume, and duration of inhalation intake as well as an average human body weight. The intake coefficient is calculated by dividing the daily air intake by the average adult body weight to give a value in m3/kg/day. This coefficient has been inserted into Worksheet 5-1 and is based on the standard adult values given in Exhibit 5-2. For short-term exposures, include the duration of exposure on Worksheet 5-1.

Using Worksheet 5-1, estimate subchronic and chronic air intakes for each indicator chemical at all relevant exposure points. Note that absorption of chemicals into the body is not accounted for by the intake estimates (or by the critical toxicity values described in Chapter 6). However, if chemical-specific absorption data are available, they can be used to refine the assessment as long as the procedures and values are clearly documented.

## 5.2 Calculate Ground-Water Intakes

Human exposure to contaminated ground water can occur when contaminated wells are used as a drinking water source. The degree of exposure depends on the concentration of the contaminant in drinking water, the amount of water consumed per day, and the duration of exposure.

The measured or predicted concentrations (short-term and long-term) of each contaminant in ground water at each exposure point are given in Worksheet 4-4. Insert these values into appropriate columns of Worksheet 5-2. Note that separate worksheets must be prepared for each ground-water exposure point.

A standard human intake coefficient has been calculated for use in determining drinking water exposures. This value takes into account both average daily consumption of water and average body weight. The intake coefficient is calculated by dividing the standard drinking water intake by the average adult body weight to give a value in l/kg/day. This coefficient has been inserted into Worksheet 5-2 and is based on the standard adult values given in Exhibit 5-2. For short-term exposures, also include the duration of exposure on Worksheet 5-2.

Using Worksheet 5-2, estimate subchronic and chronic drinking water intakes for each indicator chemical at all relevant ground-water exposure points.

| Parameter | Standard Value | Reference |
|---|---|---|
| Average body weight, adult | 70 kg | EPA, 1980 |
| Average body weight, child | 10 kg | ICRP, 1975 |
| Amount of water ingested daily, adult | 2 liters | NAS, 1977 |
| Amount of water ingested daily, child | 1 liter | NAS, 1977 |
| Amount of air breathed daily, adult | 20 $m^3$ | EPA, 1980 |
| Amount of air breathed daily, child | 5 $m^3$ | FDA, 1970 |
| Amount of freshwater fish consumed daily, adult | 6.5 g | EPA, 1980 |

a/ Example 1: how to apply the standard assumptions.

If contaminant concentration is 3 mg/liter in drinking water:

(3 mg/liter x 2 liters/day water consumption) ÷ 70 kg body weight
= 0.086 mg/kg/day intake

Example 2: how to apply adjusted assumptions.

If site data indicate that the exposed population has a water consumption rate of 1.2 liters/day and an average weight of 60 kg, and the contaminant concentration is 3 mg/liter in drinking water:

(3 mg/liter x 1.2 liters/day water consumption) ÷ 60 kg body weight
= 0.06 mg/kg/day intake

EXHIBIT 5-2
STANDARD VALUES USED IN DAILY INTAKE CALCULATIONS

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 5-1

CALCULATE AIR INTAKES

Exposure Point: Nearest Residence

| | Chemical | Human Intake Factor (m3/kg/day) | Short-Term Concentration (mg/m3) | Subchronic Daily Intake (mg/kg/day) | Duration (fraction of year) | Long-term Concentration (mg/m3) | Chronic Daily Intake (mg/kg/day) |
|---|---|---|---|---|---|---|---|
| 1. | Benzene | 0.29 | 0.026 | 0.0075 | 0.5 | 0.0040 | 0.0012 |
| 2. | Lead | 0.29 | 0 | 0 | 0.5 | 0 | 0 |
| 3. | | 0.29 | | | | | |
| 4. | | 0.29 | | | | | |

INSTRUCTIONS

1. List all indicator chemicals.
2. List the short-term and long-term concentration of each chemical in air (from Worksheet 4-4) in the appropriate column.
3. Determine subchronic daily intake (SDI) using the following formula:

$$SDI = \text{Short-term Concentration} \times \text{Human Intake Factor}$$

Note: Human intake factor = standard air intake per day/standard body weight

4. Determine chronic daily intake (CDI) using the following formula:

$$CDI = \text{Long-term Concentration} \times \text{Human Intake Factor}$$

Note: Human intake factor = standard air intake per day/standard body weight

5. Include duration of subchronic exposure represented by the intake estimate, in fraction of year.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 5-2

CALCULATE GROUND-WATER INTAKES

Exposure Point: Private Drinking Water Wells

| | Chemical | Human Intake Factor (l/kg/day) | Short-Term Concentration (mg/l) | Subchronic Daily Intake (mg/kg/day) | Duration (fraction of year) | Long-term Concentration (mg/l) | Chronic Daily Intake (mg/kg/day) |
|---|---|---|---|---|---|---|---|
| 1. | Benzene | 0.029 | 0.20 | 0.0058 | 0.5 | 0.0085 | 0.00025 |
| 2. | Lead | 0.029 | 0.045 | 0.0013 | 0.5 | 0.0050 | 0.00015 |
| 3. | | 0.029 | | | | | |
| 4. | | 0.029 | | | | | |

INSTRUCTIONS

1. List all indicator chemicals.
2. List the short-term and long-term concentration of each chemical in ground water (from Worksheet 4-4) in the appropriate column.
3. Determine subchronic daily intake (SDI) using the following formula:

$$\text{SDI} = \text{Short-term Concentration} \times \text{Human Intake Factor}$$

Note: Human Intake Factor = standard drinking water intake per day/standard body weight

4. Determine chronic daily intake (CDI) using the following formula:

$$\text{CDI} = \text{Long-term Concentration} \times \text{Human Intake Factor}$$

Note: Human Intake Factor = standard drinking water intake per day/standard body weight

5. Include duration of subchronic exposure represented by the intake estimate, in fraction of year.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

## 5.3 Calculate Surface Water Intakes

For potential exposures to contaminated surface water, calculate intakes from ingestion of drinking water and ingestion of contaminated fish, as appropriate for the site being assessed.

Drinking Water. Human exposure to contaminated surface water can occur when the surface water is used as a drinking water source. The degree of exposure to contaminants present in drinking water derived from surface water depends on the same factors described for drinking water derived from ground water.

Obtain the concentrations (short-term and long-term) of each chemical present in surface water at each exposure point from Worksheet 4-4. Insert these values into the appropriate columns of Worksheet 5-3. The standard human intake coefficient for drinking water is the same as that used for calculating ground-water intakes and has been inserted into Worksheet 5-3. For short-term exposures, include the duration of exposure on Worksheet 5-3.

Using Worksheet 5-3, estimate subchronic and chronic drinking water intakes for each indicator chemical at all relevant surface water exposure points.

Fish Consumption. Another potential route of exposure from contaminated surface water is through the ingestion of contaminated fish. The factors that determine human exposure from contaminated fish are the contaminant concentration in the fish, the amount of fish consumed, and the duration of exposure.

The concentration of a contaminant in fish can be estimated by multiplying the concentration of the contaminant in surface water by the fish bioconcentration factor for that chemical. Obtain surface water concentrations for each chemical at each exposure point from Worksheet 4-4. Insert the appropriate values into the appropriate columns of Worksheet 5-4. Standard human intake coefficients are calculated by dividing standard freshwater fish intake per day by the average adult body weight. These coefficients have been inserted into the worksheet. Obtain the fish bioconcentration factor for each chemical from Appendix A or other sources. Again, for short-term exposures include the duration of exposure on Worksheet 5-4. If the concentration of contaminants in fish has been measured, this concentration can be used for short-term exposure. It should not necessarily be used for long-term exposure because surface water concentrations are likely to change over the 70-year period being considered, causing the concentration of contaminants in the fish to change over time.

Using Worksheet 5-4, estimate subchronic and chronic daily intakes from contaminated fish for each indicator chemical at all relevant surface water exposure points.

## 5.4 Calculate Intakes from Other Exposure Pathways

There are a number of other potentially important exposure pathways that are more difficult to quantify than those just described. Nevertheless, the human chemical intakes received though such pathways may be extremely important to certain populations-at-risk. For example, at some sites children playing outdoors may be exposed to contaminated soil through dermal absorption or through direct ingestion of soil. If young children have access to a site or adjacent area with contaminated surface soil, exposure for this subpopulation via soil ingestion can be estimated based on the following assumptions:

- Ingestion is primarily of concern for children between age two and six;
- Ingestion rate varies from 0.1 to 5 grams per day, with higher values representative of pica behavior; and
- Body weight of children in this age group averages 17 kg, and ranges from 10 to 25 kg.

These assumptions are based on EPA (1984), Kimbrough et al. (1984), and Anderson et al. (1984). In addition to exposures via soil ingestion, other soil-related pathways, particularly migration of contaminants to ground and surface waters, may be very important at a site and therefore should be considered.

Another potential exposure pathway could be agricultural land being irrigated with contaminated surface or ground water; human exposure would occur if produce is contaminated and ingested. Humans may also be exposed via consumption of game animals that reside in contaminated areas. Contaminated surface waters, in addition to providing drinking water, may be used for recreation and humans may be exposed by swimming in such waters. This may result in dermal, oral, and inhalation exposures. During bathing or showering, dermal or inhalation exposure may occur. Volatilization while cooking with contaminated water may result in inhalation exposure.

Formulas and worksheets for these less common exposure pathways have not been included in this manual because there has been little experience on which to base standard formulas. It should be noted, however, that at certain sites and for certain populations-at-risk, these less common routes of exposure may be significant. If one of these exposure pathways (e.g., exposure to soil, dermal exposure or surface water ingestion while swimming) has been

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 5-3

CALCULATE SURFACE WATER INTAKES

Exposure Point: Downstream Drinking Water

| | Chemical | Human Intake Factor (l/kg/day) | Short-Term Concentration (mg/l) | Subchronic Daily Intake (mg/kg/day) | Duration (fraction of year) | Long-term Concentration (mg/l) | Chronic Daily Intake (mg/kg/day) |
|---|---|---|---|---|---|---|---|
| 1. | Benzene | 0.029 | 0.0068 | 0.00020 | 0.5 | $1.5 \times 10^{-3}$ | $4.4 \times 10^{-5}$ |
| 2. | Lead | 0.029 | 0.00028 | $8.1 \times 10^{-6}$ | 0.5 | $1.0 \times 10^{-5}$ | $2.9 \times 10^{-7}$ |
| 3. | | 0.029 | | | | | |
| 4. | | 0.029 | | | | | |

INSTRUCTIONS

1. List all indicator chemicals.
2. List the short-term and long-term concentration of each chemical in surface water (from Worksheet 4-4) in the appropriate column.
3. Determine subchronic daily intake (SDI) using the following formula:

$$\text{SDI} = \text{Short-term Concentration} \times \text{Human Intake Factor}$$

Note: Human Intake Factor = standard drinking water intake per day/standard body weight

4. Determine chronic daily intake (CDI) using the following formula:

$$\text{CDI} = \text{Long-term Concentration} \times \text{Human Intake Factor}$$

Note: Human Intake Factor = standard drinking water intake per day/standard body weight

5. Include duration of subchronic exposure represented by the intake estimate, in fraction of year.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 5-4

CALCULATE INTAKES FROM
INGESTION OF CONTAMINATED FISH

Exposure Point: Nearby Stream

| | Chemical | Bio-concentration Factor | Human Intake Factor (kg fish/kg/day) | Short-term Concentration (mg/l) | Subchronic Daily Intake (mg/kg/day) | Duration (fraction of year) | Long-term Concentration (mg/l) | Chronic Daily Intake (mg/kg/day) |
|---|---|---|---|---|---|---|---|---|
| 1. | Benzene | 5.2 | .00009 | 0.010 | $4.7 \times 10^{-6}$ | 0.5 | 0.0025 | $1.2 \times 10^{-6}$ |
| 2. | Lead | 49 | .00009 | 0.00086 | $3.8 \times 10^{-6}$ | 0.5 | 0.00030 | $1.3 \times 10^{-6}$ |
| 3. | | | .00009 | | | | | |
| 4. | | | .00009 | | | | | |

INSTRUCTIONS

1. List all indicator chemicals.
2. List the short-term and long-term concentration of each chemical in surface water (from Worksheet 4-4) in the appropriate column.
3. Record the bioconcentration factor (BCF) for each chemical from Appendix C.
4. Determine subchronic daily intake (SDI) using the following formula:

$$\text{SDI} = \text{Short-term Concentration} \times \text{Human Intake Factor} \times \text{BCF}$$

Note: Human Intake Factor = standard fish ingestion per day/standard body weight

5. Determine chronic daily intake (CDI) using the following formula:

$$\text{CDI} = \text{Long-term Concentration} \times \text{Human Intake Factor} \times \text{BCF}$$

Note: Human Intake Factor = standard fish ingestion per day/standard body weight

6. Include duration of subchronic exposure represented by the intake estimate, in fraction of year.

ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

identified as significant, the Exposure Assessment Group at EPA headquarters should be contacted for guidance on a method for calculating chemical intakes.

## 5.5 Combine Pathway-Specific Intakes to Yield Total Oral and Total Inhalation Intakes

In this step, total exposure scenarios are developed for each exposure point, and the relevant route-specific intakes are combined for the affected population. This exposure summation gives the total daily oral intake and total daily inhalation intake of each chemical to which the population may be exposed.

In Chapter 4, chemical concentrations at the significant exposure point were estimated for each identified exposure pathway (see Worksheets 4-2 and 4-4). Recall that the significant exposure point for a pathway is the point of highest individual exposure, although locations with large exposed populations and lower exposure levels should also be included in the analysis as supplementary exposure points. Now the task is to determine, for each significant exposure point identified in Chapter 4, which of the other exposure pathways could contribute to total exposure at that point. Use Worksheet 5-5 to record this information. Be sure to list any potentially important non-quantified exposure pathways on Worksheet 5-5. If the populations-at-risk for different exposure pathways are mutually exclusive, do not sum intakes from both pathways for the same exposure point. For example, it is incorrect to sum the intakes associated with ingesting drinking water from different sources if each person's exposure is exclusively from one of the sources.

After a total exposure scenario has been developed for each significant exposure point (e.g., a population living near the site with private drinking water), combine the individual chemical intakes calculated for each of the oral exposure pathways identified for that exposure point. Do the same for inhalation. Referring to Worksheet 5-5, insert the appropriate intakes to be combined (from Worksheets 5-1 through 5-4) into Worksheet 5-6 (SDIs) and Worksheet 5-7 (CDIs). Note that some intake values from Worksheets 5-1 through 5-4 may need to be adjusted when applied to exposure points other than those specified. In situations where the significant exposure points of two pathways are relatively far apart, the project management team must judge whether the additional calculation effort is warranted or whether simply summing the intakes for the significant exposure points is sufficient. For example, if the significant exposure points for an air and a ground-water pathway differ, the project manager may choose to adjust the intakes from Worksheets 5-1 and 5-2 before using them for a total exposure estimate or may combine the unadjusted intakes for a conservative total exposure estimate.

The next step in the summation procedure is to add the intakes from fish and drinking water ingestion for each chemical to give the total oral SDI (Worksheet 5-6) and CDI (Worksheet 5-7) for the population-at-risk at each significant exposure point. The existence of any non-quantified exposure pathways should be noted on these summary intake worksheets. In addition, be sure to note the number of people exposed at each significant exposure point.

The intake summation procedure described here is most relevant to the estimation of total chronic exposure levels. When estimating total subchronic exposures, be sure not to sum peak intake values estimated for different time periods. Remember, the time period defined as short term is anywhere from a 10 to a 90 day period. If the SDI for one pathway is estimated to occur immediately and the SDI for another pathway affecting the same exposure point is predicted to occur in 5 years, it would be improper to sum these -- they would affect the same population, but at different times. In this situation, assessing short-term risks based on the higher of the two values usually will provide a reasonable assessment of short-term risks. However, exposures likely to occur immediately should also be assessed.

* * * * *

Human intakes for the indicator chemicals have been calculated from measured or predicted ambient exposure point concentrations. Intakes received from air, ground water, surface water, and fish consumption have been calculated separately for each exposure pathway and combined to give total oral and total inhalation intakes for each significant exposure point and each selected indicator chemical. These intake estimates will be combined with toxicity information gathered for Chapter 6 to perform the risk characterization for Chapter 7.

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 5-5

## PATHWAYS CONTRIBUTING TO TOTAL EXPOSURE

| Exposure Point | Exposure Pathways Contributing to Total Exposure | Comments |
|---|---|---|
| 1. Nearest downgradient residences on private wells* | Ground-water ingestion | |
| | Air inhalation | |
| | Soil contact | Non-quantified |
| 2. Residences 1 mile SW on vulnerable public wells | Ground-water ingestion | |
| | Air inhalation | Low exposure |
| 3. Hospital at 2 miles on public well (sensitive) | Ground-water ingestion | |
| | | |

* Significant exposure point.

## INSTRUCTIONS

1. List the exposure points for all exposure pathways being evaluated (from Worksheet 4-2).

2. Determine the exposure pathways contributing to total exposure for each listed exposure point.

3. Note in the comments column which exposure pathways are only short-term, which are non-quantified, and any other pertinent information.

## ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 5-5
PATHWAYS CONTRIBUTING TO TOTAL EXPOSURE

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 5-6

## TOTAL SUBCHRONIC DAILY INTAKE (SDI) CALCULATION

Total Exposure Point: Nearest Residences on Private Wells

Number of People: 40

| | Chemical | Ground-Water SDI | Surface Water SDI | Fish Ingestion SDI | Total Oral SDI | Total Air SDI |
|---|---|---|---|---|---|---|
| 1. | Benzene | 0.0058 | - | $4.7 \times 10^{-6}$ | 0.0058 | 0.0075 |
| 2. | Lead | 0.0013 | - | $3.8 \times 10^{-6}$ | 0.0013 | 0 |
| 3. | | | | | | |
| 4. | | | | | | |

### INSTRUCTIONS

1. List all indicator chemicals.
2. Refer to Worksheet 5-5 and determine which exposure pathways are relevant for the total exposure point.
3. Record SDIs (in mg/kg/day) for the total exposure point from Worksheets 5-1 through 5-4 in the appropriate columns. Be sure only to include SDIs estimated for the same time period.
4. For relevant exposure pathways that had intakes calculated for a different exposure point, adjust the intake estimates for the total exposure point. Record the rationale and calculations supporting any adjustments and attach to this worksheet.
5. Determine total oral SDI by adding the component SDIs (ground-water, surface water, fish) for each chemical.

### ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 5-6
TOTAL SUBCHRONIC DAILY INTAKE (SDI) CALCULATION

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 5-7

## TOTAL CHRONIC DAILY INTAKE (CDI) CALCULATION

Total Exposure Point: Nearest Residences on Private Wells

Number of People: 40

| | Chemical | Ground-Water CDI | Surface Water CDI | Fish Ingestion CDI | Total Oral CDI | Total Air CDI |
|---|---|---|---|---|---|---|
| 1. | Benzene | 0.00025 | - | $1.3 \times 10^{-6}$ | 0.00025 | 0.0012 |
| 2. | Lead | 0.00015 | - | $1.5 \times 10^{-6}$ | 0.00015 | 0 |
| 3. | | | | | | |
| 4. | | | | | | |

### INSTRUCTIONS

1. List all indicator chemicals.
2. Refer to Worksheet 5-5 and determine which exposure pathways are relevant for the total exposure point.
3. Record CDIs (in mg/kg/day) for the total exposure point from Worksheets 5-1 through 5-4 in the appropriate columns.
4. For relevant exposure pathways that had intakes calculated for a different exposure point, adjust the intake estimates for the total exposure point. Record the rationale and calculations supporting any adjustments and attach to this worksheet.
5. Determine total oral CDI by adding the component CDIs (ground-water, surface water, fish) for each chemical.

### ASSUMPTIONS

List all major assumptions in the development of data for this worksheet:

# CHAPTER 6
## STEP 4: TOXICITY ASSESSMENT

This chapter describes the critical toxicity values (i.e., numerical values describing a chemical's toxicity) needed for the risk characterization step of the Superfund public health evaluation process. An overview of the toxicity assessment step of the public health evaluation is shown in Exhibit 6-1. Toxicity information is used in conjunction with the results of the exposure assessment to characterize risk. EPA's verified reference doses (RfDs),[22] evaluations by EPA's Carcinogen Assessment Group, and Health Effects Assessment documents (HEAs) developed by EPA's Office of Research and Development serve as a consistent source of critical toxicity values for the Superfund public health evaluation process. **Critical toxicity values from these sources are summarized in Appendix A to this Manual and also are contained in PHRED (Public Health Risk Database).** EPA believes that these are currently the best available sources of toxicity values. However, this process is intended to accommodate new information and, as new toxicity data become available, Appendix A and PHRED will be updated to reflect these changes. Toxicity information for specific chemicals not covered in Appendix A may be available through the Environmental Criteria and Assessment Office (ECAO), U.S. EPA, 26 W. St. Clair Street, Cincinnati, Ohio 45268. In situations where Appendix A does not contain the necessary critical toxicity values for all indicator chemicals at a site, ECAO should be contacted for additional information. In some cases it may be necessary to derive appropriate values based on available toxicological or epidemiologic data.

Three values that describe the degree of toxicity posed by a chemical are required in the process:

- the acceptable intake for subchronic exposure (AIS);
- the acceptable intake for chronic exposure (AIC); and
- the carcinogenic potency factor (for potential carcinogenic effects only).

These values are based on empirical data and have not been adjusted for site-specific conditions. For some chemicals, separate critical toxicity values are available for ingestion and inhalation routes of exposure.

AIS and AIC values are required for all chemicals being evaluated. These values are derived from quantitative information available from studies in animals (or observations made in human epidemiologic studies) on the relationship between intake and noncarcinogenic toxic effects. They are designed to be protective of sensitive populations. The units for the AIS and AIC are the same as those developed for SDI and CDI in the human exposure phase of the public health evaluation -- mg/kg body weight/day. For teratogenic chemicals, AIS values are generally derived for the teratogenic effects.

AIS values are determined by a process similar to the procedure used to develop reference dose values, except that subchronic effects are the basis of the values instead of chronic effects. Most AIS values are based on subchronic (10-90 day) animal studies, although some are derived from human exposure data. For chemicals without appropriate human data, the highest subchronic exposure level not causing adverse effects, or no-observed-adverse- effect-level (NOAEL), is determined for all valid animal studies available in the literature. The NOAEL is then divided by appropriate uncertainty factors to give the AIS. Uncertainty factors usually include a factor of 10 to account for extrapolation from animal experiments to human effects and a factor of 10 for intraspecies variability (i.e., to account for the fact that two individuals of the same species may not react to the same quantity of a chemical with the same level of response).

In general, AIC values are based on long-term animal studies. For a few chemicals, however, adequate human data are available and are used. The highest chronic exposure level not causing an adverse effect (NOAEL) is determined by examining literature values from all appropriate animal studies. The NOAEL value is then divided by uncertainty factors as in AIS development. Again, a factor of 10 is used for extrapolation from animal effects to human effects, and a factor of 10 is used to account for intraspecies variability. If chronic studies are not available, subchronic NOAELs are used and divided by an additional factor of 10 to account for uncertainties in extrapolating from subchronic to chronic exposures.

The carcinogenic potency factor is expressed as the lifetime cancer risk per mg/kg body weight/day. This factor is equivalent to $q_1^*$ when it is based on animal study data evaluated by the multistage model. This

[22] Reference doses are for noncarcinogenic effects and are similar in concept to acceptable daily intakes (ADIs).

**Identify Critical Toxicity Values from EPA-Approved Sources (Summarized in Appendix C to Manual)**

**Develop Toxicity Values (in Conjunction with Headquarters EPA) for Additional Chemicals, if Necessary**

EXHIBIT 6-1
OVERVIEW OF STEP 4: ASSESSING TOXICITY

factor is an estimated upper 95 percent confidence limit of the carcinogenic potency of the chemical. From it, an upper bound estimate of cancer risk can be determined.

Although toxicity assessment is an integral part of the overall public health evaluation, in most cases limited new work will actually be required of the site analyst to complete this step. To prevent duplication of effort and ensure consistency among public health evaluations, the toxicity assessment step has already been done for many common toxic substances and is documented in a HEA or RfD summary. **If EPA has completed verification of a reference dose (RfD) for a specific chemical, then that value should be used as an AIC.** If critical toxicity values are not available in Appendix A, contact ECAO for further guidance. Worksheet 6-1 is provided as a format for summarizing the required toxicity data.

* * * * *

In this chapter, toxicity information was collected to combine with exposure information from the previous chapter to allow characterization of the health risks of the indicator chemicals. Three kinds of data were collected: chronic and subchronic acceptable intakes for noncarcinogenic effects and carcinogenic potency factors for potential carcinogenic effects. Using these data, long-term and short-term health risks can be characterized

Guidance for risk characterization is presented in Chapter 7.

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 6-1

## CRITICAL TOXICITY VALUES

| Chemical | AIS (mg/kg/day) | AIC (mg/kg/day) | Carcinogenic Potency Factor $(mg/kg/day)^{-1}$ |
|---|---|---|---|
| Inhalation Route | | | |
| 1. Benzene | -- | -- | 0.026(A)* |
| 2. Lead | -- | 0.00043 | N/A |
| 3. Methyl ethyl ketone | 2.2 | 0.22 | N/A |
| Ingestion Route | | | |
| 1. Benzene | -- | -- | 0.052(A)* |
| 2. Lead | -- | 0.0014 | N/A |
| 3. Methyl ethyl ketone | -- | 0.050 | N/A |

* EPA weight-of-evidence rating in parentheses for potential carcinogens.

### INSTRUCTIONS

1. List all indicator chemicals.
2. List AIS, AIC, and carcinogenic potency factor values and carcinogenicity weight-of-evidence ratings, obtained from Appendix A (or EPA/ECAO).
3. For teratogenic chemicals, list a separate AIS for that effect only.

### ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 6-1
CRITICAL TOXICITY VALUES

# CHAPTER 7
# STEP 5: RISK CHARACTERIZATION

In this final step of the baseline public health evaluation process, a comparison is made between projected intakes and reference levels for noncarcinogens and between calculated risks and target risks for potential carcinogens. In the following sections, the methodology for making these comparisons is described. There are separate discussions for noncarcinogenic and carcinogenic effects because the methodology differs for these two classes of chemical toxicity. Exhibit 7-1 is an overview of the risk characterization step.

Remember, comparisons to applicable or relevant and appropriate requirements and other standards and criteria should already have been made for those chemicals having them (see Section 4.3). This comparison to requirements, in addition to the risk characterization results, will be included in the final public health evaluation report in the feasibility study.

## 7.1 Noncarcinogenic Effects

Most sites being assessed will have more than one indicator chemical being evaluated for noncarcinogenic effects. To assess the overall potential for noncarcinogenic effects posed by multiple chemicals, a hazard index approach has been developed based on EPA's Guidelines for Health Risk Assessment of Chemical Mixtures (EPA, 1986d). This approach assumes that multiple sub-threshold exposures could result in an adverse effect and that the magnitude of the adverse effect will be proportional to the sum of the ratios of the sub-threshold exposures to acceptable exposures. This can be expressed as:

$$Hazard\,Index = \frac{E_1}{RL_1} + \frac{E_2}{RL_2} + \ldots + \frac{E_i}{RL_i}$$

where $E_i$ = Exposure level (or intake) for the $i^{th}$ toxicant

$RL_i$ = Reference level (or intake) for the $i^{th}$ toxicant

Any single chemical with an exposure level greater than the reference level will cause the hazard index to exceed unity, and when the index exceeds unity, there may be concern for a potential health risk. For multiple chemical exposures, the hazard index can exceed one even if no single chemical exceeds its acceptable level. However, the assumption of additivity reflected in the hazard index equation is most properly applied to compounds that induce the same effect by the same mechanism. Consequently, application of the equation to a mixture of compounds that are not expected to induce the same type of effects could overestimate the potential for effects. **If the hazard index results in a value greater than unity, segregate the compounds in the mixture by critical effect and derive separate hazard indices for each effect.** Critical effects are described in the Health Effects Assessment documents.

To make the comparison between estimated subchronic exposure to several chemicals and acceptable subchronic intakes, determine the subchronic hazard index by calculating and then summing the SDI:AIS ratios for all chemicals. Use Worksheet 7-1 to record this calculation and summation. A separate subchronic hazard index should be developed for each total exposure point. Be careful to sum ratios only for chemicals and exposure pathways for which the short-term concentration time period is the same.

If any chemicals with teratogenic effects are being assessed, calculate a separate subchronic hazard index for them. The subchronic daily intake (SDI) and the reference level for teratogenic effects should be used for assessment of teratogenic risk.

To make the comparison between estimated chronic exposure to indicator chemicals and acceptable chronic intake, follow a similar procedure, calculating and then summing the ratios of CDI:AIC for all chemicals to give a chronic hazard index. Calculate a separate index for each total exposure point, using Worksheet 7-2 to calculate and record the necessary information.

Throughout this entire public health evaluation process, intakes and risks from oral and inhalation exposure pathways have been estimated separately. This was done so that route-specific toxicity data could be used. However, the possible effects of multimedia exposure should be evaluated by summing the hazard indices for inhalation and oral exposures at each total exposure point. This will ensure that acceptable levels are not being exceeded by combined intakes when multiple exposure pathways exist.

It is emphasized that the hazard index is not a mathematical prediction of incidence or severity of effects. It is simply a numerical index to help identify potential exposure problems. Results for multiple chemicals should not be interpreted too strongly.

If some of the indicator chemicals do not have adequate toxicity information, thus preventing their inclusion in the hazard index, the hazard index may

**For Noncarcinogens, Compare Estimated Intakes to Reference Levels**

**For Carcinogens, Combine Estimated Intakes with Upper-Bound Carcinogenic Potency Factors to Calculate Risk**

EXHIBIT 7-1
OVERVIEW OF STEP 5: CHARACTERIZING RISKS

not be reflective of actual hazard at the site. Consideration of chemicals that do not have toxicity values could significantly increase the hazard index to levels of concern. Professional judgment is required to determine how to interpret the hazard index for a particular site.

## 7.2 Potential Carcinogenic Effects

For potential carcinogens, risks are estimated as probabilities. The carcinogenic potency factor, which is an upper 95 percent confidence limit on the probability of response per unit intake of a chemical over a lifetime (i.e., only 5 percent chance that the probability of response could be greater than the estimated value on the basis of the experimental data used), converts estimated intakes directly to incremental risk. If the exposure assessment is conservative, the resultant risk predicted is an upper-bound estimate. Consequently, predicted risk may overestimate the actual risk at a site. However, this method is used so that carcinogenic risk will not be underestimated.

Because relatively low intakes are most likely from environmental exposures, it can be assumed that the dose-response relationship will be in the linear portion of the dose-response curve. Under this assumption, the slope of the dose-response curve is equivalent to the carcinogenic potency factor, and risk will be directly related to intake at low levels of exposure. The carcinogenic risk equation is:

$$\text{Risk} = \text{CDI} \times \text{Carcinogenic Potency Factor}$$

The carcinogenic risk estimate will generally be an upper-bound estimate.

This equation is valid only at low risk levels. For sites where chemical intakes may be large (e.g., estimated carcinogenic risk above 0.01), an alternate model should be considered. For example, the one-hit equation, which is consistent with the linear low-dose model given above, may be useful:

$$\text{Risk} = 1 - \exp(-\text{CDI} \times \text{Carcinogenic Potency Factor})$$

In this situation, consult ECAO in Cincinnati for guidance on an appropriate model.

For multiple compounds, the risk equation may be generalized to:

$$\text{Risk} = \Sigma\,(\text{CDI}_i \times \text{Carcinogenic Potency Factor}_i)$$

This risk summation, also based on EPA's risk assessment guidelines, assumes that individual intakes are small. It also assumes independence of action by the compounds involved (i.e., that there are no synergistic or antagonistic chemical interactions and that all chemicals have the same endpoint, cancer). If these assumptions are incorrect, over- or under-estimation of the actual risk could result.

For Superfund public health evaluations, it also is assumed that cancer risks from various exposure routes are additive. Expressed mathematically this is:

$$\text{Carcinogenic Risk for chemical X} = [\text{CDI (inhalation)} \times \text{Potency Factor (inhalation)}] + [\text{CDI (oral)} \times \text{Potency Factor (oral)}]$$

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 7-1

## CALCULATION OF SUBCHRONIC HAZARD INDEX

Total Exposure Point: Nearest Residences on Private Wells

| | Chemical | Inhalation SDI | Inhalation AIS | Inhalation SDI:AIS | Oral SDI | Oral AIS | Oral SDI:AIS |
|---|---|---|---|---|---|---|---|
| 1. | Benzene | 0.0075 | 0.15* | 0.05 | 0.0058 | 0.15* | 0.04 |
| 2. | Lead | 0 | 0.5* | 0 | 0.0013 | 0.5* | 0.003 |
| 3. | | | | | | | |
| 4. | | | | | | | |

* Values for illustration only; not in Appendix C.

Sum of Inhalation SDI:AIS Ratios = 0.05
Sum of Oral SDI:AIS Ratios = 0.04
Sum Total of All Ratios = 0.09

### INSTRUCTIONS

1. List all indicator chemicals.

2. List the total inhalation SDI and total oral SDI (in mg/kg/day) from Worksheet 5-6 in the appropriate columns for each chemical.

3. List route-specific AIS values (from Worksheet 6-1) and calculate route-specific SDI:AIS ratios for each chemical.

4. Sum and record route-specific SDI:AIS ratios.

5. Sum and record total (inhalation plus oral) SDI:AIS ratios only if the SDIs for the two routes refer to the same time period. If total is less than 1, there is probably no subchronic health hazard. If the sum is greater than 1, separate the ratios according to health endpoint and do a separate worksheet for each endpoint.

### ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 7-1
CALCULATION OF SUBCHRONIC HAZARD INDEX

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 7-2

## CALCULATION OF CHRONIC HAZARD INDEX

Total Exposure Point: Nearest Residences on Private Wells

| | Chemical | Inhalation CDI | Inhalation AIC | Inhalation CDI:AIC | Oral CDI | Oral AIC | Oral CDI:AIC |
|---|---|---|---|---|---|---|---|
| 1. | Benzene | 0.0012 | 0.002* | 0.6 | 0.00025 | 0.002* | 0.1 |
| 2. | Lead | 0 | 0.00043 | 0 | 0.00015 | 0.0014 | 0.1 |
| 3. | | | | | | | |
| 4. | | | | | | | |

* Values for illustration only; not in Appendix C.

Sum of Inhalation CDI:AIC Ratios = 0.6
Sum of Oral CDI:AIC Ratios = 0.2
Sum Total of All Ratios = 0.8

### INSTRUCTIONS

1. List all indicator chemicals.
2. List the total inhalation CDI and total oral CDI (in mg/kg/day) from Worksheet 5-7 in the appropriate columns for each chemical.
3. List route-specific AIC values (from Worksheet 6-1) and calculate route-specific CDI:AIC ratios for each chemical.
4. Sum and record route-specific CDI:AIC ratios.
5. Sum and record total (inhalation plus oral) CDI:AIC ratios. If total is less than 1, there is probably no chronic health hazard. If the sum is greater than 1, separate the ratios according to health endpoint and do a separate worksheet for each endpoint.

### ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 7-2
CALCULATION OF CHRONIC HAZARD INDEX

Therefore, the total carcinogenic risk for a site is estimated by:

Total Risk = (Carcinogenic Risk for Chemical 1 + . . . + $Chemical_i$)

The result of the characterization will be upper-bound estimates of the potential carcinogenic risk for each total exposure point. Estimates for individual chemicals and pathways as well as estimates of aggregate risk should be developed and reported. Use Worksheet 7-3 to record the risk calculations for potential carcinogens.

## 7.3 Uncertainties

The public health evaluation process has been designed to rely on a subset of the chemicals present at a site. These indicator chemicals were identified on the basis of certain preliminary data. It is important at this time to review the original data used to select the indicator chemicals to make sure that it remains valid and that new indicator chemical candidates have not been uncovered during the evaluation process. It is wise to reevaluate the initial choice of indicator chemicals at this time to assure yourself that, having been through the entire process, they are still the appropriate chemicals on which to base the public health evaluation.

It is emphasized that all estimates of carcinogenic risk and hazard index are dependent on numerous assumptions, and many uncertainties are inherent in the risk assessment process. Probably without exception, information on site history and site characterization data will be lacking in some areas. Most toxicity information is derived from animal studies, and reputable scientists disagree about how to interpret these data. A single toxicity parameter based on an animal study does not convey the route of administration of test doses of the suspect chemicals, the organ(s) in which the response occurred, or the severity of endpoints in the animal experiment used to calculate the dose-response relationship. Consequently, extrapolation to humans is a source of uncertainty. Many toxicity studies are done at high doses relative to exposures associated with waste disposal sites; extrapolation from high to low doses also increases the uncertainty of risk numbers. Exposure modeling is based on many simplifying assumptions that add to the uncertainty. Often the quality or quantity of site-specific chemical monitoring data is inadequate. The additivity of toxicant risks and the additivity of doses of the same toxicant from different exposure routes are additional assumptions and additional sources of uncertainty. Consequently, the results of the baseline evaluation should not be taken as a characterization of absolute risk. An important use of these results is to highlight potential sources of risk at a site so that they may be dealt with effectively in the remedial process.

The procedures described in this chapter are not expected to supplant expert judgment nor can they be designed to include all of the information that may be available. If there are specific data germane to the assumption of additivity discussed above (e.g., if two compounds are present at the same site and it is known that the combination is five times more toxic than the sum of toxicities for the two compounds), then modify the risk estimate accordingly. If data on chemical interactions are available but are not good enough to support quantitative assessment, note the information on the "assumptions" portion of the appropriate worksheet.

A listing should be made of the most significant factors increasing the uncertainty of the risk assessment results, as illustrated in Worksheet 7-4.

* * * * *

As a result of the procedures described in Chapters 3 through 7, indicator chemicals at a site have been identified, releases calculated, exposure routes identified, and exposure point concentrations calculated. Applicable or relevant and appropriate requirements, when available, were compared to concentration estimates. Human intakes for each exposure pathway were calculated and summed, then combined with toxicity data to get risk estimates for both potential carcinogens and noncarcinogens. The results of the public health evaluation of baseline site conditions will now be used as a starting point for the formulation of numerical performance goals for management of migration remedial alternatives. These results also can be considered in the development of source control measures and as a check to make sure all potential sources of health risk at a site have been considered.

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 7-3

## CALCULATION OF RISK FROM POTENTIAL CARCINOGENS

Total Exposure Point: Nearest Residences on Private Wells

| | Chemical | Exposure Route | CDI (mg/kg/day) | Carcinogenic Potency Factor (mg/kg/day)-1 | Route-specific Risk | Total Chemical-specific Risk |
|---|---|---|---|---|---|---|
| 1. | Benzene | Oral | 0.00025 | 0.052 | $1 \times 10^{-5}$ | $4 \times 10^{-5}$ |
| | | Inhalation | 0.0012 | 0.026 | $3 \times 10^{-5}$ | |
| 2. | | | | | | |
| | | | | | | |

TOTAL UPPER BOUND RISK = $4 \times 10^{-5}$

### INSTRUCTIONS

1. List all potentially carcinogenic indicator chemicals.
2. List all exposure routes for each chemical.
3. Record CDIs (Worksheet 5-7) and carcinogenic potency factors (Worksheet 6-1) for each chemical and each exposure route.
4. Multiply the potency factor by the CDI to get the route-specific risk; then sum the route-specific risks for each chemical.
5. Sum all of the chemical-specific risks to give an upper bound estimate of total incremental risk due to potential carcinogens.

### ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

WORKSHEET 7-3
CALCULATION OF RISK FROM POTENTIAL CARCINOGENS

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 7-4

## SITE-SPECIFIC FACTORS INCREASING UNCERTAINTY

---

**(1) Sensitive Population(s):**

Yes, specifically: Hospital 1/2 mile southwest from site - 300 people potentially exposed via air and drinking water

**(2) Exposure Uncertainties:**

**A. Non-Quantifiable Exposure Routes**

Yes, minor pathways:
1. Ingestion of vegetables and livestock contaminated by spray irrigation
2. Ingestion/dermal absorption by swimmers

**B. Overall Data Adequacy**

The site characterization and sampling data is believed to be sufficiently detailed to allow a reasonable assessment; QA/QC is acceptable

**(3) Percentage of Chemicals Evaluated (number and volume):**

Approximately 10 percent of the total number of chemicals detected (represents over 70 percent of the total estimated volume)

**(4) Chemical or Biological Interactions:**

Yes, chemicals: 1. Benzene and PCBs
Extent of Interaction (if known):
Unknown, but PCBs increase metabolism of benzene

**(5) Other Factors:**

---

### INSTRUCTIONS

1. Complete worksheet, based on results of analysis of the listed factors at the site.

WORKSHEET 7-4
SITE-SPECIFIC FACTORS INCREASING UNCERTAINTY

# CHAPTER 8

# DEVELOPMENT OF PERFORMANCE GOALS AND ANALYSIS OF RISKS FOR REMEDIAL ALTERNATIVES

The baseline public health evaluation, using the procedures described in the preceding chapters, provides considerable information on the baseline health risks (i.e., in the absence of remedial action) from the site. This information about chemical releases, routes of exposure, human exposure points, and the level and timing of risk will be used as input to further development of the proposed remedial alternatives. This chapter describes the procedures for developing target chemical concentrations for remedial alternatives based on public health considerations and for comparison of health risks associated with each remedial alternative being considered. Conceptual alternatives will have already been developed as a concurrent part of the feasibility study process. By this time, site engineers should have defined the options available for remedial actions at a site based on feasibility and technical considerations. This chapter provides methods to compare public health risks among the remedial actions developed in other parts of the RI/FS process.

The NCP defines two different types of remedial alternatives that can be developed during the feasibility study process: ***source control*** alternatives and ***management of migration*** alternatives. This chapter provides guidance for developing performance goals and for estimating risks associated with a given level of control for management of migration alternatives.

Source control alternatives are those that control or remove the source of contamination before it has migrated much beyond the source. For example, site excavation and waste immobilization techniques are considered source control alternatives. Such remedial alternatives should be assessed and designed on the basis of applicable or relevant and appropriate requirements (as defined by the NCP; see Section 2.3) and best engineering judgment. However, best engineering judgment does not indicate how much to excavate or help to determine acceptable residual levels of chemicals in soil. The methods described in this chapter can be used to derive target soil concentrations associated with a target risk range. Otherwise, the procedures given in this chapter, with the exception of those described in Section 8.6 for assessing short-term effects, do not apply to source control alternatives.

Management of migration alternatives are those that address contaminants that have migrated away from the source. For example, pump and treat techniques for removing ground-water contamination are considered management of migration alternatives. These alternatives should be analyzed based on applicable or relevant and appropriate requirements and/or target health risk levels for population exposure points.

**The determination that proposed remedial alternatives attain, exceed, or fall below RCRA design and operating standards or any other applicable or relevant and appropriate requirement that is not an ambient concentration level is made independently of the procedures in this chapter.** Thus, although RCRA requirements are a key consideration in the development of remedial alternatives, they do not provide ambient concentration targets for specific chemicals and are not discussed further in this chapter. The procedures of this chapter allow development of ambient concentation goals to assist in refining remedial alternatives.

Some sites may have both source control and management of migration alternatives under evaluation. For these sites, follow the procedures described in this chapter for management of migration alternatives.

The remedial alternative ultimately chosen is a risk management decision that is made as part of the overall feasibility study. This chapter provides methods for a health-based comparison among alternatives. After a remedial action has been chosen, the target concentrations developed for the comparison can be used as performance goals for the remedial alternative and to calculate allowable release rates for contaminants at the site. When applicable or relevant and appropriate ambient requirements are available for all indicator chemicals at a site, the project manager will have specific environmental concentration levels for each chemical to use as performance goals.

When applicable or relevant and appropriate requirements are not available for all indicator chemicals, remedies considered should reduce ambient chemical concentrations to levels associated with a carcinogenic risk range of $10^{-4}$ to $10^{-7}$ (e.g., at least one remedial alternative being considered could be associated with a carcinogenic risk of $10^{-4}$, one with $10^{-6}$, and one with $10^{-7}$) where possible. For noncarcinogenic contaminants, exposure point concentrations should be reduced to correspond to acceptable intake levels. At sites where both potential carcinogens and noncarcinogens are involved, the potential carcinogens will generally drive the design

process because concentrations corresponding to the target risk range are usually lower than acceptable concentrations of noncarcinogens.

When some indicator chemicals have applicable or relevant and appropriate requirements and others do not, the preferred approach is first to evaluate remedial alternatives based on the total target carcinogenic risk range, as when there are no applicable or relevant and appropriate requirements. Then, for each chemical with a requirement, determine whether at least one alternative attains, one exceeds, and one falls below its requirement. Given the broad target range of carcinogenic risk, it is likely that these three conditions would be met. If not, additional remedial options may have to be developed to satisfy the proposed policy of considering options that exceed, attain, and fall below applicable or relevant and appropriate requirements.

A tiered approach for evaluating and comparing alternatives is described in this chapter. The first step is a reevaluation of the indicator chemicals to determine whether any additions are necessary due to treatability concerns. Second, human exposure pathways are determined for each remedial alternative. The next step is development of preliminary target concentrations, based either on applicable or relevant and appropriate requirements or the potential carcinogenic indicator chemicals at the site. The initial focus on potential carcinogens rather than noncarcinogens greatly simplifies the process, and it is a logical approach because potential carcinogens will usually drive the final design (i.e., environmental concentrations of potential carcinogens will generally have to be reduced to lower levels than concentrations of noncarcinogens). For sites without applicable or relevant and appropriate requirements, the next step, after developing preliminary target concentrations, is to estimate corresponding long-term concentrations of noncarcinogenic indicator chemicals to ensure that acceptable levels are attained. If necessary, the alternative should be modified to provide adequate control of noncarcinogens. The final steps of the tiered approach are to assess potential short-term health risks associated with the remedial alternative and to evaluate the potential health effects that could result from failure of the alternative.

Exhibit 8-1 presents a simple flowchart of the process for formulating performance goals. The remainder of this chapter describes specific procedures for comparing health risks and developing performance goals for management of migration remedial measures. The presentation of methods in this chapter assumes an understanding of the previous sections of the manual.

## 8.1 Reevaluate Indicator Chemicals

The first step in determining target concentrations for management of migration remedial alternatives is a review of indicator chemicals. Indicator chemicals have already been selected for assessing baseline site risks, but the list of indicators may need to be modified because of differences among chemicals in treatability, chemical class, and propensity to be released from specific remedial alternatives. Some chemicals may be more difficult to treat than those chosen as indicators for the baseline evaluation. These more recalcitrant chemicals should be considered in the design of remedial alternatives. It may be possible to use chemical class as a surrogate for treatability because chemicals within a class have similar physical and chemical properties. Consequently, chemical classes that were not important in the baseline evaluation may become important. In addition, some remedial alternatives will control or release different chemicals than others (e.g., volatiles will be of more concern for an air stripping alternative than a site capping alternative). Review the list of selected indicator chemicals (Worksheet 3-5) and the list of all chemicals present at the site (Worksheet 3-1) to determine whether additional chemicals should be included as indicators, taking into account treatability, chemical class, and new release sources associated with each specific alternative.

## 8.2 Identify Potential Exposure Pathways

The next step in determining target concentrations for management of migration remedial alternatives is identifying potential exposure pathways. Again, this task has been completed for the no-action alternative, but it should be reviewed in light of the particular remedial alternatives under consideration. Some exposure routes identified for the baseline analysis may not exist for certain remedial alternatives, while some new exposure routes may result. For example, long-term pumping and air-stripping treatment of ground water may result in air exposures not occurring under the no-action alternative. Therefore, for each management of migration remedial alternative remaining after initial screening (Chapter 2 of the Guidance for Feasibility Studies), determine the possible sources of chemical release, transport media, human exposure points, and exposure routes.

### *8.2.1 Determine Possible Sources of Chemical Release*

Based on available information from preliminary site assessments and the remedial investigation, identify and evaluate the sources of chemical release that could result from each remedial alternative being evaluated. Consider the possibilities of chemical releases to air, surface water, ground water, and soil

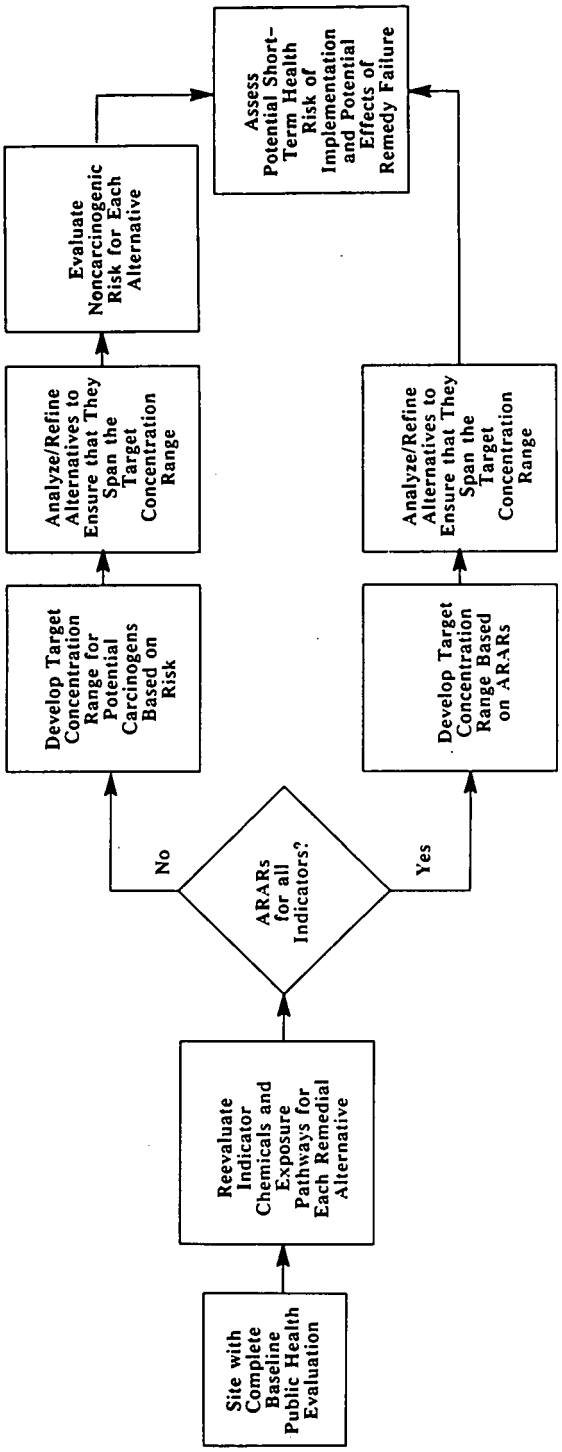


**EXHIBIT 8-1**
**FLOWCHART OF PERFORMANCE GOALS PROCESS**

from sources on the site itself and also from certain off-site sources resulting from the remedial action (e.g., a ground-water aeration tower away from the site). In all situations where contaminated materials are removed from the site and treated, stored, or disposed at a RCRA-permitted facility, it is not necessary to include the RCRA facility as a source for purposes of this assessment. Potential releases during transport of wastes from the site to the RCRA facility also need not be considered.[23]

Exhibit 8-2 gives some examples of sources of release to each medium resulting from typical remedial activities. Evaluate the sources given in Exhibit 8-2 and any others relevant to the site to determine whether each is important or unimportant, taking into consideration the potential quantity of waste that may be released, the frequency of releases, and any other important considerations. Be sure to consider the possibility of other release sources not listed in Exhibit 8-2.

Obtain descriptions and details of the remedial alternatives as a basis for identifying additional potential release sources. Use Worksheet 8-1 to list and qualitatively evaluate potential release sources for each remedial alternative. Worksheet 8-1 should be supplemented with a map that indicates the locations of the release sources for each alternative.

#### *8.2.2 Determine Human Exposure Points*

Review Worksheets 4-2 and 4-5 to determine whether the same populations included in the baseline evaluation will be affected by the specific remedial alternative under consideration. If so, note the same information previously collected. Any new significant or supplementary exposure points resulting from implementation of a remedial action should be noted (see Section 4.1.2, for definition and discussion of significant exposure points). Populations at these points will be characterized in a later step.

To assist in your evaluation of specific human exposure points, review Section 4.1. Exhibit 4-3 in that section lists common exposure points for chemical releases. Remember, the purpose of this task is to evaluate exposure pathways from a site after the implementation of remedial alternatives. In subsequent sections, methods are presented for modeling environmental transport processes from the point of exposure back to the source of contamination to define allowable releases.

As mentioned above, the affected populations may be identical to those defined in the baseline evaluation. If a new population might be exposed by the remedial alternative (e.g., a population that will be exposed to air emissions from an air stripping tower located at a distance from the site), this group must be identified and characterized.

#### *8.2.3 Integrate Release Sources, Transport Media, Exposure Points, and Exposure Routes into Exposure Pathways*

Assemble the information developed in the previous tasks and determine the complete exposure pathways that would exist for each remedial alternative. Use Worksheet 8-2 to integrate the exposure pathway information. A complete exposure pathway has four components -- a source of chemical release, an environmental transport medium, a point where human receptors could be exposed, and a likely exposure route. For example, if a release to ground water is projected but ground water from the affected aquifer is not now used or projected to be used, the exposure pathway is incomplete.

#### *8.2.4 Identify All Exposure Pathways for Each Exposure Point*

To determine the total exposure at each exposure point for a remedial alternative, review the pathways developed in Worksheet 8-2. Develop realistic total exposure scenarios (e.g., drinking contaminated ground water or contacting contaminated surface water) that combine the different pathways through which the population at an exposure point could conceivably be exposed. Record these on Worksheet 8-3.

## 8.3 Determine Target Concentrations at Human Exposure Points

This task involves analysis of each indicator chemical relevant to each significant exposure point (and supplementary exposure points, if necessary) to determine a target concentration range for each indicator chemical at the points of human exposure. Target concentrations will be calculated on the basis of applicable or relevant and appropriate requirements or the target cancer risk range of $10^{-4}$ to $10^{-7}$. If applicable or relevant and appropriate requirements are not available for all indicator chemicals, proceed to Section 8.3.2.

#### *8.3.1 Target Concentrations for Chemicals with Applicable or Relevant and Appropriate Requirements*

If all indicator chemicals have applicable or relevant and appropriate ambient concentration requirements, those requirements will be used as the basis for the

[23] Releases from waste transport and management in RCRA permitted facilities are regulated by applicable RCRA regulations (40 CFR 261 to 267) and are therefore not appropriate considerations for evaluating remedial alternatives under CERCLA.

| Release Medium | Release Mechanism | Release Source |
|---|---|---|
| Air | Volatilization | Aeration treatment processes<br>Residual contaminated soil or surface water |
| | Stack emission | Incineration |
| Surface water | Ground-water seepage | Residual contaminated ground water |
| | Effluent discharge | Treatment plant |
| | Site runoff | Residual contaminated surface soil |
| Ground water | Site leaching | Residual contaminated soil |
| | Effluent discharge | Treatment plant |
| Soil | Site leaching | Residual contaminated soil |
| | Surface runoff | Residual contaminated surface soil |

EXHIBIT 8-2
POSSIBLE CHEMICAL RELEASE SOURCES FOLLOWING REMEDIAL ACTIONS

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 8-1

## RELEASE SOURCE ANALYSIS

Remedial Alternative: Pumping and treatment of ground water using air stripping

| Medium | Potential Release Source/ Mechanism | Release Time Frame | Release Probability/ Amount |
|---|---|---|---|
| Air | Aeration treatment plant emissions | C | 100% probability for 10 years, then zero; amounts may be high for some volatile chemicals |
| | | | |
| Surface water | Aeration treatment plant discharge | C | 100% probability for 10 years, then zero; amounts may be high for non-volatile chemicals |
| | | | |
| Ground water | | | |
| | | | |
| Soil | | | |
| | | | |

### INSTRUCTIONS

1. For each medium, list potential release sources.
2. Estimate release time frame: chronic (C) or episodic (E).
3. Record any information, qualitative or quantitative, on release amounts and probabilities.
4. Attach a site map with sources located.

### ASSUMPTIONS

List all major assumptions made in developing data for this worksheet:

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 8-2

## MATRIX OF POTENTIAL EXPOSURE PATHWAYS FOR REMEDIAL ALTERNATIVES

Remedial Alternative: Limited excavation

| Release Medium | Release Source | Exposure Point | Exposure Route |
|---|---|---|---|
| Air | | | |
| | | | |
| Ground water | Remaining contaminated soil | Private well, 1/8 mile away (downgradient)* | Ingestion |
| Surface water | | | |
| | | | |
| Soil | | | |
| | | | |

*Significant exposure point.

### INSTRUCTIONS

1. List all potential release sources, by medium (see Worksheet 8-1).
2. Describe the nature of the exposure point (i.e., point of highest exposure) and its location with respect to release source (e.g., nearest residence to volatilization release area, 100 meters NW). Denote significant exposure points with an asterisk.
3. List Exposure Route: inhalation, oral, or dermal.

### ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 8-2
MATRIX OF POTENTIAL PATHWAYS FOR REMEDIAL ALTERNATIVES

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 8-3

## IDENTIFY ALL PATHWAYS FOR EXPOSURE POINTS

Remedial Alternative: Limited excavation

| Exposure Point | No. of People | Exposure Pathways: Source | Exposure Pathways: Exposure Route | Exposure Pathways: Exposure Medium |
|---|---|---|---|---|
| 1. Nearest residence on private wells | 100 | Site leachate | Ingestion | Drinking water |
| | | Site volatiles | Inhalation | Air |
| | | | | |
| 2. | | | | |
| | | | | |
| 3. | | | | |
| | | | | |

### INSTRUCTIONS

1. List each exposure point.
2. Note the number of people potentially exposed at each exposure point.
3. Record all exposure pathways relevant to each listed exposure point so that total exposure can be determined.

### ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 8-3
IDENTIFY ALL PATHWAYS FOR EXPOSURE POINTS

target concentration range. Otherwise, target concentrations will be based on the target carcinogenic risk range. Some chemicals may have more than one applicable or relevant and appropriate requirement. In these cases, the requirement most appropriate for site exposure conditions should be used. For drinking water exposures, for example, Safe Drinking Water Act MCLs should generally be used if available.

List on Worksheet 8-4 the numerical value and source of applicable or relevant and appropriate requirements for all of the indicator chemicals. The NCP requires consideration of remedies that attain, exceed, and fall below applicable or relevant and appropriate requirements.[24] Therefore, on Worksheet 8-4, list a target concentration that exceeds and one that falls below the applicable or relevant and appropriate requirements.

Once target concentrations have been determined for each medium affected, determine which of the concentrations can be achieved by each of the various remedial alternatives under consideration. Engineering judgment must be used to initially determine which remedies are likely to reduce chemicals to the various target concentrations. One approach is to review Worksheet 8-4 and consider which of the alternatives under consideration will reduce the most difficult chemical to treat to the most restrictive target concentration, the "exceeds requirements" category. Next, determine which alternative will reduce the most difficult chemical to treat to the level of the requirement. Then determine which remedy meets the "falls below requirement" category by reducing the concentration of the most difficult chemical to treat to the least restrictive level. Some of these options may actually be the same conceptual remedy modified to meet different operating levels, such as a pump and treat option with different levels of removal; conversely, they may be completely different remedies. Be sure to verify and document, using chemical release and transport modeling (see Section 4.2), that the target concentrations will be met.

Regardless of the "attain, exceed, and fall below requirements" policy, all remedies that eventually will be considered by the site decision-maker must be evaluated on public health grounds. This may be done for the remainder of the alternatives either by matching them with target concentrations or by using a public health evaluation as described in Chapters 3 through 7.

An example for a hypothetical site is provided in Worksheet 8-4. In this example, site contamination has polluted the ground water. Only two contaminants are present, cadmium and arsenic, both of which have applicable or relevant and appropriate requirements. Values for the standards and for concentrations exceeding and falling below requirements are included. Assume that four alternatives are being considered for the site: cap and slurry wall; pump, treat, and reinject; pump, treat, and discharge effluent to surface water; and provide an alternate water supply that meets the drinking water standards. The most restrictive concentration level is a concentration of 0.0001 mg/l for cadmium. Providing an alternate water supply would satisfy the "exceeds requirement" policy by reducing cadmium below that level. The pump/treat/reinject alternative can be designed to satisfy the arsenic and cadmium standards; by modifying the operating parameters, it can also satisfy the "falls below requirement" policy. Now the other two options under consideration must be assessed, either by determining what risks are likely as a result of their implementation (i.e., forward risk evaluation) or by back- calculating allowable release rates based on the target concentration range.

### *8.3.2 Target Concentrations for Chemicals Without Applicable or Relevant and Appropriate Requirements*

For situations where all indicator chemicals do not have applicable or relevant and appropriate requirements, target concentrations for potential carcinogens are calculated based on toxicity and chemical intake data. Potential carcinogens are evaluated first because target concentrations for potential carcinogens generally will be lower than acceptable concentrations for noncarcinogens; thus, potential carcinogens will usually drive the design process. Remedial alternatives under consideration must span the target carcinogenic risk range. Noncarcinogen exposures will subsequently be assessed to ensure that they are below acceptable levels.

The remedial alternatives under consideration should have been assessed to the extent that exposure points and routes have been determined for each alternative. This section describes how to quantify the target concentrations for each remedy at each exposure point. It is necessary to evaluate the risk of each alternative and to ensure that the proposed alternatives cover a wide range of risk. According to Agency policy, the target total individual carcinogenic risk resulting from exposures at a Superfund site may range anywhere between $10^{-4}$

[24] Reauthorization necessitates revision of the NCP; consequently, current policies regarding attainment of standards may be changed.

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 8-4

## TARGET CONCENTRATIONS FOR CHEMICALS WITH AMBIENT REQUIREMENTS

| | Chemical | Requirement Used/ Appropriate Medium | Target Concentration Exceeding Requirement | Applicable/ Relevant Ambient Requirement | Target Concentration Falling Below Standard |
|---|---|---|---|---|---|
| 1. | Cadmium | MCL/drinking water | .001 mg/l | 0.01 mg/l | 0.1 mg/l |
| 2. | Arsenic | MCL/drinking water | .005 mg/l | 0.05 mg/l | 0.5 mg/l |
| 3. | | | | | |
| 4. | | | | | |

### INSTRUCTIONS

1. List chemicals with applicable or relevant and appropriate ambient concentration requirements (see Exhibit 4-5).
2. List the numerical value of the requirement, the source of the requirement, and the appropriate exposure medium in the appropriate columns.
3. Determine a target concentration exceeding the standard.
4. Determine a concentration falling below the standard.

### ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 8-4
TARGET CONCENTRATIONS FOR CHEMICALS WITH AMBIENT REQUIREMENTS

to $10^{-7}$. Thus, remedial alternatives being considered should be able to reduce total potential carcinogenic risks to individuals to levels within this range. The Agency also encourages development of alternatives that eliminate carcinogenic risk where such a remedy is feasible. One remedy being considered could correspond to a carcinogenic risk of $10^{-7}$ one to $10^{-6}$, and one to $10^{-4}$. These may be the same conceptual alternative with different operating parameters or may be different alternatives altogether. In addition, the remaining remedial alternatives under consideration must also be evaluated either by calculating risks for those alternatives (i.e., forward risk evaluation as described in Chapters 3 through 7), or by back-calculating allowable release rates based on the target concentration range. For any remedial alternative which was developed by back-calculating release rates, a "forward" risk assessment of the proposed alternative should be performed to verify that it meets the risk level it was designed to achieve. This can be accomplished by following the steps described in Chapter 3 through 7.

#### 8.3.2.1 Apportion Total Potential Carcinogenic Risk Among Multiple Carcinogens

There are a number of ways of translating total risk levels into target concentrations for individual chemicals. Ultimately, the site assessor must judge how the carcinogenic exposure should be apportioned among multiple potential carcinogens and multiple routes of exposure. Two simple approaches to this problem are presented below as illustrative examples. The project manager is not restricted to these methods, though they will provide a reasonable starting point. These approaches assume low-dose additivity of carcinogenic risk, which is consistent with Agency risk assessment guidelines.

One method is to divide a target carcinogenic risk level by the number of indicator chemicals that are potential carcinogens. For example, at a target risk level of $10^{-6}$ where 5 potential carcinogens are of interest, the resulting target risk level for each individual potential carcinogen would be $2 \times 10^{-7}$ Once the target risk is determined, the target intake can be determined using the following formula:

Potential Carcinogenic Risk = (Chronic Daily Intake) x (Potency Factor)

Thus, if the potency factor for benzene is $5.2 \times 10^{-2}$ $(mg/kg/day)^{-1}$ the target benzene intake would be $3.8 \times 10^{-6}$ mg/kg/day:

$[2 \times 10^{-7}] \div [5.2 \times 10^{-2} (mg/kg/day)^{-1}] = 3.8 \times 10^{-6}$ mg/kg/day

The same calculations would then be repeated for each potential carcinogen and each level of the carcinogenic risk range. This approach is simple and conservative, ensuring that the target risk will not be exceeded if the target intakes are attained, but it may not result in the most efficient design.

Another approach is to let one or two chemicals drive the design process. One indicator chemical may be so difficult to treat or so potent (e.g., dioxin) that exposure levels must be extremely low so that the total risk falls within the target range. By designing remedies to reduce levels of such "bad actors" to within the range, concentrations of other indicator chemicals may become negligible by default, although it should still be demonstrated that these remaining concentrations of other indicator chemicals would not violate the risk range.

These approaches, however, may not be optimal with regard to engineering design or cost-effectiveness considerations. Thus, the specific means by which the target carcinogenic risk is apportioned must be determined on a site-by-site basis. Worksheet 8-5 illustrates a method for risk apportionment. This should be done for target risk levels between $10^{-4}$ and $10^{-7}$.

It is understood that this approach assumes additivity, while in fact there may be chemical interactions taking place. Until guidance is issued in this area, report any information available on chemical interaction among the substances of interest. In the unlikely event that quantitative data are available on the degree to which interactions affect risk, they should be used to adjust risk estimates.

Remember, the total individual risks from all routes of exposure must fall within the target range. If exposure to a chemical for a given population occurs by more than one route, the risk must be apportioned among routes in a similar manner to the apportionment among multiple chemicals. To determine where the most efficient reductions in risk can be made, one should first determine the target concentrations associated with both air and water routes of exposure independently. Then, the design engineers may refine the conceptual design iteratively so that the combined exposures from various routes fall within the stated range. These adjustments should be made based on the most risky routes of exposure and the most cost-effective way to reduce total carcinogenic risk from various exposure routes. The following sections present methods for calculating target concentrations in air and drinking water.

#### 8.3.2.2 Calculate Target Air Concentrations

Using the following formula, calculate the target long-term concentration in air for each potential carcinogen:

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 8-5

## APPORTIONING TOTAL TARGET RISK AMONG MULTIPLE POTENTIAL CARCINOGENS

Target Risk Level: $10^{-6*}$

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| Potential Carcinogen | Target Risk for Each Chemical | Potency Factor $^{-1}$ (mg/kg/day) | Target CDI (mg/kg/day) |
|---|---|---|---|
| 1. Benzene | $5x10^{-7}$ | 0.052 (oral) | $1x10^{-5}$ |
| 2. Chlordane | $5x10^{-7}$ | 1.61 (oral) | $3x10^{-7}$ |
| 3. | | | |
| 4. | | | |
| Total Target Risk = | $10^{-6}$ | | |

*Risk level used for illustrative purposes only.

### INSTRUCTIONS

1. Fill in target carcinogenic risk level under consideration.
2. List all potentially carcinogenic indicator chemicals.
3. Determine apportioned risk level for each chemical. Any method can be used as long as the total equals the target risk level. One method is equal apportionment, as follows:

$$\frac{\text{Total Target Risk}}{\text{Number of Potential Carcinogens}} = \text{Target Risk for Each Chemical}$$

4. List the potency factor for the appropriate exposure route for each chemical (obtained from Exhibit C-4 in Appendix C). Be sure to indicate the exposure route.
5. Calculate target intake (CDI) for each potential carcinogen:

Target Risk ÷ Potency Factor = Target Chronic Daily Intake

### ASSUMPTIONS

List all major assumptions in developing the data for this worksheet:

**WORKSHEET 8-5**
**APPORTIONING TOTAL TARGET RISK AMONG MULTIPLE POTENTIAL CARCINOGENS**

$$\text{Long-term Air Concentration} = \frac{\text{Target Chronic Daily Intake}}{\text{Human Intake Factor}}$$

Use Worksheet 8-6 to calculate target air concentrations for appropriate chemicals. This should be done for each remedial alternative. The human intake factor for air is given in the worksheet, and the target chronic daily intake is the intake corresponding to the target risk (see Worksheet 8-5).

#### 8.3.2.3 Calculate Target Drinking Water Concentrations

A population-at-risk can be exposed to contaminated surface or ground water (or both) by ingestion of drinking water. Calculate the target long-term concentration of potential carcinogens in drinking water using the following formula:

$$\text{Long-term DW Concentration} = \frac{\text{Target Chronic Daily Intake}}{\text{Human Intake Factor}}$$

Use Worksheet 8-7 to calculate the target concentrations for potential carcinogens in drinking water. The intake factor is given in the worksheet. If intakes from water exposure besides drinking water and fish ingestion, such as dermal exposure or intake of chemicals volatilizing from water, are important and can be quantified, those intakes should be included and standard intake assumptions should be adjusted.

The target chronic daily intake level represents total oral exposure. When drinking water is the only route of oral exposure, then the above calculation is appropriate. An added complication arises in cases where there is exposure to the same population through both drinking water and fish consumption. If the contaminated drinking water is from a different water source than the fish (i.e., ground water or a different surface water body), apportion the target oral intake between the two routes of ingestion. Use Worksheet 8-8 for this apportionment and Worksheet 8-9 to calculate target surface water concentrations based on intake via fish consumption. The illustrative apportionment on Worksheet 8-8 assigns equal chronic daily intake to drinking water and fish consumption. It is important to note that other apportionments are possible, permitting some tradeoffs between target concentrations for a drinking water source and surface water where fish are caught.

If exposure through drinking water and fish consumption originate from the same surface water body, consider both intake routes simultaneously in calculating target surface water concentrations. No apportionment is required because a single variable, the surface water concentration, controls the total intake. If there is simultaneous exposure to the population-at-risk via fish consumption and drinking water ingestion, calculate the target surface water concentration using the following equation:

Target Surface Water Concentration = Target Chronic Daily Intake ÷ (Bioconcentration Factor × Human Intake Factor for Fish) + Human Intake Factor for Drinking Water

Record the final target concentrations for each potential carcinogen on Worksheet 8-10. A separate worksheet should be completed for each target risk level being assessed between $10^{-4}$ and $10^{-7}$ Usually three risk levels should be assessed: the primary target ($10^{-6}$) and the extremes of the allowable range ($10^{-4}$ and $10^{-7}$). In Section 8.4, methods are described to convert the target environmental concentrations calculated here to allowable release rates of chemicals from the source.

### *8.3.3 Summarize Data*

Several data collection and calculation tasks have been completed thus far and now this information should be integrated to assist in the analysis and refinement of remedial alternatives. For each alternative, this involves combining the data from Worksheets 8-3 through 8-10. Worksheet 8-11 provides a format for this data collection.

## 8.4 Estimate Target Release Rates

Using environmental fate and transport models, target exposure point concentrations from the previous section can be applied to calculate target release rates at the identified sources of release for some remedial options. For options such as capping, slurry walls, and excavation, using models to calculate these releases is not a straightforward process. For other options such as pumping and treating, air stripping, and other point source treatment options with graded effectiveness, this step can be used to calculate allowable release rates. The estimated target chemical releases can eventually be incorporated into the remedial design. For example, the target effluent discharge levels from a contaminated ground-water treatment plant can be used to specify the treatment and removal efficiency of the facility.

Estimation of release rates requires the use of environmental fate and transport models. A great deal of uncertainty is inherent in the use of models, and it should be understood that the values generated by the models represent "ball park" estimates rather than precise values.

### *8.4.1 Predict Environmental Fate and Transport*

Because the concentration of contaminants changes as substances move from release sources to

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 8-6

## CALCULATION OF TARGET AIR CONCENTRATIONS

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| | Chemical | Target CDI (mg/kg/day) | Human Intake Factor ($m^3$/kg/day) | Target Long-Term Concentration (mg/$m^3$) |
|---|---|---|---|---|
| 1. | Benzene | N/A | 0.29 | N/A |
| 2. | | | 0.29 | |
| 3. | | | 0.29 | |
| 4. | | | 0.29 | |

## INSTRUCTIONS

1. List all indicator potential carcinogens with air as an exposure medium.
2. List the target chronic daily intake from Worksheet 8-5.
3. Determine the target long-term air concentration using the following formula:

Target Concentration = Target Chronic Daily Intake ÷ Human Intake Factor

## ASSUMPTIONS

List all major assumtions made in developing the data for this worksheet:

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 8-7

## CALCULATION OF TARGET DRINKING WATER CONCENTRATIONS

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| | Chemical | Target CDI (mg/kg/day) | Human Intake Factor (1/kg/day) | Target Long-Term Concentration (mg/l) |
|---|---|---|---|---|
| 1. | Benzene | $1x10^{-5}$ | 0.029 | $3.4x10^{-4}$ |
| 2. | | | 0.029 | |
| 3. | | | 0.029 | |
| 4. | | | 0.029 | |

### INSTRUCTIONS

1. List all indicator potential carcinogens with drinking water as an exposure route.

2. List the target chronic daily intake for each chemical from Worksheets 8-5 or 8-8.

3. Determine the target long-term drinking water concentration using the following formula:

Target Concentration = Target Chronic Daily Intake ÷ Human Intake Factor

### ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 8-8

## APPORTIONMENT OF TARGET ORAL INTAKE VIA DRINKING WATER AND FISH CONSUMPTION*

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| Chemical | Total Target Oral CDI (mg/kg/day) | Intake Via Drinking Water (mg/kg/day) | Intake Via Fish Consumption (mg/kg/day) |
|---|---|---|---|
| 1. Benzene | $1x10^{-5}$ | $5x10^{-6}$ | $5x10^{-6}$ |
| 2. | | | |
| 3. | | | |

*Not required when contaminated fish and drinking water originate from the same surface water source (see text for methods in this situation).

### INSTRUCTIONS

1. List potential carcinogens which have both drinking water and fish consumption as exposure routes and for which the fish originate from a different water source than the drinking water.

2. List total target oral intake for each of these (Worksheet 8-5).

3. List apportioned intakes for both drinking water and fish consumption, remembering that:

   Intake via drinking water + Intake via fish consumption = Total target oral intake

   As a first approximation, intake may be apportioned equally between the two (as in the example). Engineering and economic considerations may alter the apportionment on subsequent iterations.

### ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 8-8
APPORTIONMENT OF TARGET ORAL INTAKE VIA DRINKING WATER AND FISH CONSUMPTION

| | |
|---|---|
| Name of Site: | |
| Date: | |
| Analyst: | |
| QC: | |

## WORKSHEET 8-9

## CALCULATION OF TARGET SURFACE WATER CONCENTRATIONS BASED ON FISH CONSUMPTION

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| | Chemical | Target CDI (mg/kg/day) | Human Intake Factor (kg fish/ kg/day) | Bio-concentra-tion Factor | Target Surface Water Concentration (mg/l) |
|---|---|---|---|---|---|
| 1. | Benzene | $5x10^{-6}$ | .00009 | 5.2 | $1.1x10^{-2}$ |
| 2. | | | .00009 | | |
| 3. | | | .00009 | | |
| 4. | | | .00009 | | |

### INSTRUCTIONS

1. List all indicator potential carcinogens with fish consumption as an exposure pathway.
2. List the target chronic daily intake for each chemical (Worksheet 8-5 or 8-8).
3. Record the bioconcentration factors (Appendix C) for each chemical.
4. Determine target long-term surface water concentration using the following formula:

$$\text{Target Concentration} = \text{Target Chronic Daily Intake} \div \left[\text{Human Intake Factor} \times \text{Bioconcentration Factor}\right]$$

### ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 8-9
CALCULATION OF TARGET SURFACE WATER CONCENTRATIONS BASED ON FISH CONSUMPTION

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 8-10

## FINAL TARGET CONCENTRATIONS OF POTENTIAL CARCINOGENS

Target Risk Level: $10^{-6*}$

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| Exposure Route | Chemical | Target Concentration | Target Risk |
|---|---|---|---|
| Inhalation | N/A | N/A | N/A |
| | | | |
| Drinking water | Benzene | $1.7 \times 10^{-4}$ mg/l | $2 \times 10^{-7}$ |
| | | | |
| Surface water (fish consumption) | Benzene | $1.1 \times 10^{-2}$ mg/l | $2 \times 10^{-7}$ |
| | | | |

*Risk level used for illustrative purposes only.

### INSTRUCTIONS

1. Fill in target risk level.
2. List chemicals that account for exposures by each route.
3. List target concentrations from air route (Worksheet 8-6), drinking water route (Worksheet 8-7), and fish consumption route (Worksheet 8-9).
4. List target risk associated with each chemical concentration from Worksheet 8-5.

### ASSUMPTIONS

List all major assumptions made in developing data for this worksheet:

WORKSHEET 8-10
FINAL TARGET CONCENTRATIONS OF POTENTIAL CARCINOGENS

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 8-11

SUMMARY OF EXPOSURE PATHWAYS, EXPOSURE POINTS, AND TARGET CONCENTRATIONS

Remedial Alternative: Ground-water pumping/treatment

| Exposure Point | Number of People | Source | Exposure Route | Transport Medium | Target Concentrations at Point of Human Exposure: Chemicals | Target Concentration |
|---|---|---|---|---|---|---|
| Nearest group of residences on private wells | 100 | Effluent from water treatment (re-injected) | Ingestion of drinking water | Ground water | 1. Benzene | 1.7x10-4 mg/l |
| | | | | | 2. | |
| | | | | | 3. | |
| | | | | | 4. | |
| | | | | | 5. | |
| | | | | | 1. | |
| | | | | | 2. | |
| | | | | | 3. | |
| | | | | | 4. | |
| | | | | | 5. | |
| | | | | | 1. | |
| | | | | | 2. | |
| | | | | | 3. | |
| | | | | | 4. | |
| | | | | | 5. | |

INSTRUCTIONS

1. Record exposure pathway information from Worksheet 8-3.
2. Record all potential indicator carcinogens for each pathway and their target exposure point concentrations (see Worksheet 8-10).

ASSUMPTIONS

List all major assumptions made in developing data for this worksheet.

WORKSHEET 8-11
SUMMARY OF EXPOSURE PATHWAYS, EXPOSURE POINTS, AND TARGET CONCENTRATIONS

exposure points, environmental fate and transport must be assessed to project allowable releases. Each exposure pathway will have an identified medium of interest through which the contaminant travels, such as chemicals released to the subsurface that move through ground water to a well.

For each potential carcinogen moving through a specific transport medium, the output of this step will be a target release from the source, based on public health considerations at each exposure point. Using the pathways already identified for each chemical, systematically consider the extent of chemical fate and transport in each environmental medium. By doing so, the predominant mechanisms of chemical transport, transfer, and transformation can be considered and less significant processes disregarded.

Refer to the Superfund Exposure Assessment Manual for details on modeling environmental fate and transport for air, ground water, and surface water. Remember, in developing design criteria, you will be using "C", the concentration, to solve for "R", the release rate of a substance (mass/time). Some of the packaged computer models cannot be used for this because the software is designed only to determine concentration. Examine the chosen model carefully to ensure that it will work in this case. Otherwise, you may have to determine the release rate iteratively. That is, one could arbitrarily select a release rate and solve for concentration, repeating this step until the correct exposure point concentration is determined. The release rates calculated in this process can be used as design goals for the remedial alternatives of interest.

### *8.4.2 Summarize Data*

Use Worksheet 8-12 to present the average allowable release rates for each chemical and each source modeled for each remedial alternatives.

## 8.5 Assess Chronic Risk for Noncarcinogens

Now that remedial alternatives have been considered to reduce estimated carcinogenic risk to acceptable target levels, each alternative must be checked to ensure that it reduces noncarcinogenic risk to acceptable levels. This is done similarly to the quantitative analysis for noncarcinogens for the no-action alternative (Chapters 3 through 7).

Release sources and exposure routes for each remedial alternative have already been determined on Worksheets 8-1 and 8-2. Significant exposure points for each alternative have also been determined on Worksheet 8-3. Contaminant releases should be obtained or estimated from the remedial design specifications. These are then converted to environmental concentrations using chemical fate and transport models as described in Section 4.2. Human intakes for the environmental concentrations are calculated as described in Chapter 5. Worksheet 8-13 should be used to summarize the release and exposure data.

A Chronic Hazard Index should be calculated, as described in Section 7.1, to determine risk from noncarcinogens. Assessment of short-term risks is discussed in the next section. Remember, the equation for the Hazard Index is, for this situation:

$$HI = \frac{CDI_1}{AIC_1} + \frac{CDI_2}{AIC_2} + \ldots + \frac{CDI_i}{AIC_i}$$

where $CDI_i$ = Chronic daily intake for the $i^{th}$ toxicant

$AIC_i$ = Acceptable intake for chronic exposure for the $i^{th}$ toxicant (noncarcinogenic effects only).

Again, if the Hazard Index is less than one, no adverse effects are expected. If the value is near or greater than unity, the toxicants should be considered separately, according to the health endpoints they produce. If unity is exceeded for any health endpoint, consider revising the design to reduce the risk from noncarcinogens to a lower level. Worksheet 8-14 should be used to summarize the intake and toxicity information used to calculate the noncarcinogenic risk. Worksheets from Chapters 4 and 5 may be useful to organize this information.

## 8.6 Assess Potential Short-Term Health Effects of Remedial Alternatives

After remedial alternatives have been analyzed for chemical risks, the potential short-term public health effects of each alternative should be considered. Short-term health risks should not be used as a selection criterion for remedial alternatives, but should be used to determine appropriate management practices during implementation of the remedial action. In other words, if predicted short-term concentrations are likely to exceed short-term toxicity thresholds in the process of constructing or implementing a remedial alternative, certain management practices should be employed to reduce the potential risks. For example, a remedial option at a site may involve excavating and removing contaminated soil. In the absence of precautionary measures, fugitive dust generation by heavy equipment and remedial activities may create a short-term health hazard. These and other temporary sources of chemical release associated with construction and implementation of a remedy are not grounds for rejecting the remedial alternative. However, management practices, such as the temporary

Name of Site:
Date:
Analyst:
QC:

## WORKSHEET 8-12

## LONG-TERM TARGET RELEASES

Remedial Alternative: Limited excavation
Exposure Point: Nearest residence

| | Chemical | Exposure Pathway | Long-Term Target Release |
|---|---|---|---|
| 1. | Benzene | Site volatilization to air | 0.00027 kg/day |
| 2. | | | |
| 3. | | | |
| 4. | | | |

### INSTRUCTIONS

1. List indicator potential carcinogens.
2. Using Worksheet 8-2 indicate all the pathway/release sources identified for each chemical.
3. List the long-term target release rates calculated for each combination of chemical and pathway/release source, using the target concentrations listed in Worksheet 8-10. Release rates should be listed in units of mass per time (e.g. kg/day or lbs/hr).

### ASSUMPTIONS

List all assumptions made in developing the data for this worksheet:

WORKSHEET 8-12
LONG-TERM TARGET RELEASES

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 8-13

SUMMARY TABLE: EXPOSURE TO NONCARCINOGENS

Remedial Alternative: Ground-water pumping/treatment

| | Chemical | Exposure Pathway: Release Source | Exposure Pathway: Transport Medium | Exposure Pathway: Exposure Point | Release Rate | Long-Term Concentration at Exposure Point |
|---|---|---|---|---|---|---|
| 1. | Chlorobenzene | Effluent from water treatment | Surface water | Public drinking Water supply | 130g/day | 5.6 x 10-6 mg/l |
| 2. | Lead | Effluent from water treatment | Surface water | Public drinking Water supply | 2000g/day | 1.3 x 10-4 mg/l |
| 3. | | | | | | |
| 4. | | | | | | |

INSTRUCTIONS

1. List all noncarcinogenic indicator chemicals.
2. For each indicator list its release source, transport medium and exposure point.
3. List the release rate and appropriate units for each indicator.
4. Calculate and record the long-term concentration at the exposure point.

ASSUMPTIONS

List all major assumptions made in developing data for this worksheet:

WORKSHEET 8-13
SUMMARY TABLE: EXPOSURE TO NONCARCINOGENS

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 8-14

SUMMARY TABLE: CHRONIC INTAKES AND RISKS FROM NONCARCINOGENS

Remedial Alternative: Limited excavation  Exposure Point: Public drinking water supply and fish ingestion

| | Chemical | Chronic Daily Intake (mg/kg/day) | AIC (mg/kg/day) | CDI/AIC | Health Endpoint |
|---|---|---|---|---|---|
| 1. | Chlorobenzene | 1.6 x 10-6 | 0.027 | 5.4 x 10-5 | Nervous system |
| 2. | Lead | 4.0 x 10-5 | 1.4 x 10-3 | 2.9 x 10-2 | Blood/nervous system |
| 3. | | | | | |
| 4. | | | | | |
| 5. | | | | | |
| | | | TOTAL (Hazard Index) = | 0.029 | |

INSTRUCTIONS

1. List all noncarcinogenic indicator chemicals.
2. List chronic daily intake for each indicator noncarcinogen.
3. List the AIC value for each indicator noncarcinogen. These are listed in Exhibit C-6 in Appendix A.
4. Calculate the CDI/AIC ratio. Sum these to determine magnitude of the Hazard Index.
5. List the health endpoint for each noncarcinogenic indicator.

ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:

WORKSHEET 8-14
SUMMARY TABLE: CHRONIC INTAKES AND RISKS FROM NONCARCINOGENS

relocation of potentially exposed populations, should be considered to mitigate the health risks associated with temporary sources of release.

Data on acceptable short-term exposures are often difficult to obtain, and a qualitative analysis of short-term health effects from remedial actions may be all that is possible. Also remember that the remedial action itself, in addition to the initial implementation of an action, may increase short-term exposure at a site. For example, a pump and treat alternative for ground-water contamination may increase the concentration of volatiles in the air near a site until the clean-up at the site is completed, which could be several years.

Public health evaluation of short-term effects is similar to the preceding evaluation for chronic noncarcinogenic effects. However, because new exposures are possible, the exposure assessment must be reviewed. Review Section 4.2 to assist in identifying possible human exposure points and in characterizing sensitive human populations. Exhibit 8-3 lists some common types of release sources at sites during remedial action. Worksheet 8-15 should be completed to document potential short-term exposure pathways.

Environmental concentrations of the indicator chemicals at the site for the potential exposures must now be determined. Review Section 4.2 for the details of this process. Releases of chemicals will probably have to be estimated. Use any technical information available to generate a best approximation. Environmental fate and transport should be modeled from the release to obtain environmental concentrations. Intakes are calculated from the environmental concentration. Review Chapter 5 for the details of this process.

Short-term chemical concentrations are compared to the AIS, the acceptable intake of contaminants for subchronic exposures, to assess health risk. A Hazard Index should be calculated, as described in Section 7.1. Use Worksheet 8-16 to assess the short-term noncarcinogenic risk. If noncarcinogenic risk exceeds unity, management practices to mitigate or eliminate releases must be devised.

* * * * *

In this chapter, information from the baseline public health evaluation has been used as input to the analysis and refinement of remedial alternatives. For source control measures, best engineering judgment and applicable or relevant and appropriate requirements were used to refine remedial alternatives. For management of migration alternatives, applicable or relevant and appropriate requirements and health-based performance goals for potential carcinogens were used as inputs to the design process. Predicted exposure levels for noncarcinogens were checked to ensure that they would not be above their thresholds of safety. Short-term effects of remedial alternatives were also considered. All that remains to be done for the public health evaluation is organizing this information for use by the site decision-makers.

| Release Medium | Release Mechanism | Source of Released Materials |
|---|---|---|
| Air | Volatilization | Contaminated deep soil (during excavation)<br>Water/wastewater treatment facilities |
| | Fugitive dust generation | Contaminated surface soil<br>Contaminated deep soil (during excavation) |
| Surface water | Direct effluent discharge | Treatment of contaminated runoff<br>Treatment of contaminated ground water<br>Treatment of leachate |
| Ground water | Site runoff<br>Land application of effluents | Contaminated surface soil<br>Treatment of various waste streams |
| | Underground injection of effluents | Treatment of various waste streams |
| Soil | Land application | Treatment of various waste streams |

EXHIBIT 8-3
COMMON TEMPORARY CHEMICAL RELEASE SOURCES DURING IMPLEMENTATION OF A REMEDIAL ALTERNATIVE

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 8-15

MATRIX OF POTENTIAL SHORT-TERM EXPOSURE PATHWAYS

Remedial Alternative: Ground-water pumping/treatment

| Release Medium | Release Source | Exposure Point | Exposure Route | Number of People | Release Rate |
|---|---|---|---|---|---|
| Air | | | | | |
| | | | | | |
| Ground water | | | | | |
| | | | | | |
| Surface water | Increased volume of contaminated surface water resulting from groundwater treatment | Public drinking water intake, 2 km downstream from discharge point | Ingestion | 35000 | Low |
| Soil | | | | | |
| | | | | | |

INSTRUCTIONS

1. List all potential short-term release sources.
2. Describe the nature of the exposure point for the maximum exposed individual and its location with respect to release source (e.g., nearest residence to volatilization release source, 300 feet NW of site).
3. List exposure route: inhalation, oral, or dermal.
4. Record the number of people potentially exposed.
5. Record the expected potential release rate.

ASSUMPTIONS

List all major assumptions made in developing the data for this worksheet:



WORKSHEET 8-15
MATRIX OF POTENTIAL SHORT-TERM EXPOSURE PATHWAYS

Name of Site:

Date:

Analyst:

QC:

WORKSHEET 8-16

SUMMARY TABLE: SUBCHRONIC INTAKES AND RISKS

Remedial Alternative: Limited excavation  Exposure Point: Public drinking water supply and fish ingestion

| | Chemical | Subchronic Daily Intake (mg/kg/day) | AIS (mg/kg/day) | SDI/AIS | Health Endpoint |
|---|---|---|---|---|---|
| 1. | Chlorobenzene | 1.2 x 10-5 | 2.7 x 10-1 | 8.6 x 10-5 | Nervous system |
| 2. | Lead | 2.8 x 10-4 | 1.4 x 10-2* | 2.0 x 10-2 | Blood and nervous system |
| 3. | | | | | |
| 4. | | | | | |
| 5. | | | | | |
| | | | TOTAL (Hazard Index) = | 0.02 | |

* Value for illustration only; not in Appendix C.

INSTRUCTIONS

1. List all indicator chemicals.
2. List the subchronic daily intake (SDI) that has been calculated for each noncarcinogenic indicator.
3. List the AIS (acceptable intake for subchronic exposure). AIS values for some chemicals are listed in Exhibit A-6 in Appendix C.
4. Calculate the SDI:AIS ratio. Sum for all indicators to calculate the Hazard Index.
5. List the health endpoint for each indicator.

ASSUMPTIONS

List all major assumptions made in developing data for this worksheet:

WORKSHEET 8-16
SUMMARY TABLE: SUBCHRONIC INTAKES AND RISKS

# CHAPTER 9
# SUMMARIZING THE PUBLIC HEALTH EVALUATION

At this point in the public health evaluation process, the following analyses have been completed:

- Assessment of the baseline health risks posed by a site, and
- Assessment of the proposed remedial alternatives based on applicable or relevant and appropriate requirements and, for management of migration alternatives and soil excavation procedures, health-based performance goals.

The results of the public health evaluation should be reported to site decision-makers for consideration in the remedy selection process. For fund-financed remedial investigations/feasibility studies, **this reporting requirement will typically be fulfilled by a public health evaluation chapter in the feasibility study.** A separate handbook has been distributed for enforcement-lead sites; in general, the principles of public health evaluation for those sites will be similar.

This chapter provides guidance for summarizing and reporting the results of a Superfund public health evaluation. In general the report should provide a rationale for the level of detail of the analysis, a description of each of the steps discussed in Chapters 3 through 7, and a summary of the analysis of remedial alternatives. The worksheets listed in Exhibit 9-1 (or their equivalent) should be a part of the public health evaluation report.[25] Individual toxicity profiles are very useful and may be developed to describe potential effects of the indicator chemicals or other chemicals of concern. Relevant toxicity profiles also can be included as part of the public health evaluation report.

It is important to note that the narrative component of all public health evaluations plays a very important role. The narrative should be used to clearly explain the data used in the evaluation and the results of the evaluation. Recognizing that public health evaluation reports may be reviewed by the public and especially by members of the exposed or potentially exposed population, care must be taken to explain the major steps and the results of the evaluation in terms that are easily understood by the general public.

In addition to the narrative report and worksheets, the two summary exhibits described in this chapter (or their equivalent) should be included as a key part of the quantitative analysis report: Exhibit 9-2 for the baseline evaluation and Exhibit 9-3 for remedial alternatives. Both exhibits require qualitative and quantitative information. The qualitative entries are as important as the numbers and, in some cases, perhaps more important; consequently, be sure to complete the columns accurately and completely.

## 9.1 Summarize the Baseline Public Health Evaluation

Complete Exhibit 9-2 to provide a summary table for the baseline public health evaluation. First, list the indicator chemicals from Worksheet 3-5 which were used in the evaluation. Then describe the significant exposure points associated with the site. Describe where they are in relation to the site and how exposure might occur there. Next, discuss the exposure pathway qualitatively. List the release source, the transport media (e.g., ground water, surface water, air), and exposure routes (e.g., oral, inhalation, dermal) for each significant exposure point. The exposure pathway summary should be a combination of information from Worksheets 4-1 and 4-2. Also, from Worksheet 4-2, record the number of people at each significant exposure point and describe any other important populations that are nearby. For example, a town which draws water from a well down gradient from the point of maximum ground-water exposure or a school near the peak air exposure point might be included.

The next major topic of the exhibit is a summary of ambient concentration requirements that are relevant and appropriate or applicable to the site. You should list all requirements that were considered and compared to predicted ambient concentrations. In the next column, list any requirements that were violated. For this column you should include the type of requirement (e.g., Safe Drinking Water Act MCLs), the name of the chemicals which violated the requirements (e.g., arsenic) and the numerical value of the requirements (e.g., 0.05 mg/l). This information can be found on Worksheet 4-5.

Information about carcinogenic risk will be summarized next. First, enter the total carcinogenic risk due to all potential carcinogens. This risk value can be found on Worksheet 7-3. If possible include some measure of the reliability of this information (e.g., 95% confidence level, standard deviation). At many sites one, two, or three chemicals will be responsible for most of the risk at the site because of high toxicity, large projected releases, or high

[25] Other worksheets from Chapters 3 through 8 may be included as an appendix to the feasibility study.

| Title | Number |
|---|---|
| Scoring for Indicator Chemical Selection: Koc Values and Concentrations in Various Environmental Media | 3-1 |
| Scoring for Indicator Chemical Selection: Evaluation of Exposure Factors | 3-5 |
| Matrix of Potential Exposure Pathways | 4-2 |
| Contaminant Concentrations at Exposure Points | 4-4 |
| Comparison of Applicable or Relevant and Appropriate Requirements to Estimated Exposure Point Concentrations | 4-5 |
| Comparison of Other Federal and State Criteria to Estimated Exposure Point Concentrations | 4-6 |
| Pathways Contributing to Total Exposure | 5-5 |
| Total Subchronic Daily Intake (SDI) Calculation | 5-6 |
| Total Chronic Daily Intake (CDI) Calculation | 5-7 |
| Calculation of Subchronic Hazard Index | 7-1 |
| Calculation of Chronic Hazard Index | 7-2 |
| Calculation of Risk from Potential Carcinogens | 7-3 |
| Matrix of Potential Exposure Pathways for Remedial Alternatives | 8-2 |
| Summary of Exposure Pathways, Exposure Points, and Target Concentrations | 8-11 |
| Summary Table: Chronic Intakes and Risks from Noncarcinogens | 8-14 |
| Summary Tables: Subchronic Intakes and Risks | 8-16 |

EXHIBIT 9-1
WORKSHEETS THAT SHOULD BE INCLUDED IN A PUBLIC HEALTH EVALUATION SUMMARY

EXHIBIT 9-2

SUMMARY OF THE BASELINE PUBLIC HEALTH EVALUATION

Site: _____ Indicator Chemicals: _____

| Human Exposure Point a/ | Exposure Pathway b/ | Number of People Potentially Exposed c/ | Requirements/Criteria | | Potential Carcinogenic Risk | | | Noncarcinogenic Risk | | | | Significant Sources of Uncertainty m/ | Comments n/ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Compared d/ | Standard concentration ratio e/ | Risk Estimate f/ | Dominant Chemicals g/ | Weight-of-Evidence for Dom. Chem. h/ | Chronic Hazard Index i/ | Dominant Chemicals j/ | Severity Rating for Dom. Chem. k/ | Subchronic Hazard Index l/ | | |
| 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. |
| | | | 2. | 2. | | 2. | 2. | | 2. | 2. | | | |
| | | | 3. | 3. | | 3. | 3. | | 3. | 3. | | | |
| | | | 4. | 4. | | | | | | | | | |
| | | | 5. | 5. | | | | | | | | | |
| 2. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. |
| | | | 2. | 2. | | 2. | 2. | | 2. | 2. | | | |
| | | | 3. | 3. | | 3. | 3. | | 3. | 3. | | | |
| | | | 4. | 4. | | | | | | | | | |
| | | | 5. | 5. | | | | | | | | | |

a/ List each human exposure point evaluated.
b/ Include information on release source, transport medium, and exposure route.
c/ List the population potentially exposed for each exposure point. Nearby populations also warrant listing separately if they are large or especially sensitive.
d/ List all requirements/criteria that were compared to ambient concentration values for indicator chemicals. Record the chemical and its numerical value, and indicate whether it is an applicable or relevant and appropriate requirement or other criterion.
e/ Record the ratio between the projected exposure point concentration and the requirement criterion.
f/ List the total potential carcinogenic risk for each exposure point. Include best estimate and upper-bound estimate, if available.
g/ List the dominant chemicals that contribute most to the carcinogenic risk at the site. These chemicals may dominate because of high toxicity, high concentration, or large quantity.
h/ Weight-of-evidence is a qualitative, graded scale based on the EPA classification scheme, to capture differences in amount and quality of toxicity data (see Exhibit B-2).
i/ Chronic Hazard Index is calculated for all noncarcinogenic indicator chemicals. If unity is exceeded, segregate chemicals by their health endpoints and list separately for each endpoint. Include the health endpoint and the hazard index. Report best estimate and upper-bound estimate, if available.
j/ The chemicals that contribute most significantly to noncarcinogenic risk, whether due to high toxicity, high concentration, or large quantity.
k/ A qualitative, graded scale to capture differences in health endpoint severities (see Exhibit B-1).
l/ Subchronic Hazard Index is calculated for short-term exposures for all indicator chemicals.
m/ Sources of uncertainty for the assessment process may include data gaps, incomplete toxicity information, sample variation, uncertainty due to modeling.
n/ Comments may be necessary to explain assumptions, difficulties, results, or conclusions relating to the assessment process. Where available, background concentrations should be noted. Organoleptic (taste and odor) thresholds may be relevant to compare to environmental concentrations and toxicity values.

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EXHIBIT 9-2
SUMMARY OF THE BASELINE PUBLIC HEALTH EVALUATION

EXHIBIT 9-3

SUMMARY OF THE PUBLIC HEALTH EVALUATION OF REMEDIAL ALTERNATIVES

Site: _____ Target Risk Level for Potential Carcinogens: _____ a/

| Remedy b/ | Potential Exposure Pathways c/ | Indicator Chemicals d/ | App/Rel Requirements e/ | Target Concentrations f/ | Target Risk For Each Pot'l. Carcinogen g/* | Noncarcinogenic Risk Summary h/* | Short-term Risk Summary i/ | Significant Sources of Uncertainty j/ | Comments k/ |
|---|---|---|---|---|---|---|---|---|---|
| 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. |
| | | 2. | 2. | 2. | 2. | | | | |
| | | 3. | 3. | 3. | 3. | | | | |
| | | 4. | | 4. | 4. | | | | |
| | | 5. | | 5. | 5. | | | | |
| | | 6. | | 6. | 6. | | | | |
| | 2. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. |
| | | 2. | 2. | 2. | 2. | | | | |
| | | 3. | 3. | 3. | 3. | | | | |
| | | 4. | | 4. | 4. | | | | |
| | | 5. | | 5. | 5. | | | | |
| | | 6. | | 6. | 6. | | | | |
| 2. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. | 1. |

* Column does not apply if applicable/relevant requirements available for all indicator chemicals.

a/ Separate sheets must be done for carcinogenic risk levels spanning the range from $10^{-4}$ to $10^{-7}$ unless all indicator chemicals have applicable or relevant and appropriate requirements.
b/ List the remedies under consideration.
c/ Include information on sources of contaminants, transport media and routes, exposure points, and populations involved.
d/ List chemicals considered for remedial alternatives.
e/ Enter the identity of and value for all applicable or relevant and appropriate requirements for indicator chemicals.
f/ List the concentrations calculated for potential carcinogens to achieve target risk level; also enter the EPA weight-of-evidence category. However, if all indicators have applicable or relevant and appropriate requirements, enter the value of the requirement.
g/ Record the target risk apportioned to each potential carcinogen.
h/ Include Chronic Hazard Index. If subdivided by health endpoint, list both hazard index and health endpoint. Include the chemicals that contribute most significantly to noncarcinogenic risk.
i/ Describe possible short-term risks, and include synopsis of how these risks can be managed or eliminated.
j/ Sources of uncertainty may include incomplete toxicity information, apportionment of risk between chemicals, modeling difficulties, and other data gaps.
k/ Comments may be necessary to explain assumptions, difficulties, results or conclusions relating to the design goals process. Organoleptic (taste and odor) thresholds may be included to compare to environmental concentrations and target risks.

EXHIBIT 9-3
SUMMARY OF THE PUBLIC HEALTH EVALUATION OF REMEDIAL ALTERNATIVES

concentrations. List these especially important chemicals here. The weight-of-evidence rating, a qualitative scale based on the amount, relevance, and quality of the toxicity data, should be included. This value can be found in Appendix A or on Worksheet 3-2.

Health risk due to noncarcinogens should be summarized in the next section. From Worksheet 7-2, list the chronic hazard index calculated for all noncarcinogens. If the index exceeds unity and was recalculated for each health endpoint, that information should be included. For noncarcinogens, as for carcinogens, one or two chemicals may dominate the risks. This (or these) chemical(s) should be listed along with their severity rating, a qualitative scale indicating the severity of their health endpoint (the severity rating scale is given in Exhibit B-1). Also, list the subchronic hazard index calculated for short-term exposures for all indicator noncarcinogens. This index can be found on Worksheet 7-1. Subchronic hazards may require qualitative description.

Sources of uncertainty, such as data gaps, incomplete toxicity information, sample variation, and uncertainty contributed by modeling, that were encountered in a particular assessment should be discussed briefly. If ranges of uncertainty or confidence levels for particular circumstances are known, they should be included. Finally, any comments that are necessary to explain assumptions, difficulties, results, or conclusions relating to the assessment should be written in the final column.

Organoleptic (taste and odor) thresholds should be included if they are known because they may affect consumption. Background concentration may be important for some sites. Timing of exposures should also be noted if it can be determined.

## 9.2 Summarize Analysis of Remedial Alternatives

Exhibit 9-3 provides a format for a table to summarize remedial alternatives. For each site, relevant information should be provided for all remedial alternatives being considered and should include alternatives spanning a carcinogenic risk range of $10^{-4}$ to $10^{-7}$. Several remedies under consideration for a site can be included on a single summary table as long as they correspond to the same risk level.

Describe the remedial action under consideration in the first column of Exhibit 9-3. This action might be excavation, removal, a pump-and-treat remedy, or air stripping. Next, qualitatively summarize the significant potential exposures pathways. The exposure pathways might be an air release from air stripping towers or migration of contaminated ground water. Sources of contaminants, the transport media and routes, possible exposure points, timing and amount of releases should be included. The exposure pathway column should be a synthesis of information appearing in Worksheets 8-1, 8-2, 8-3, and 8-11.

The indicator chemicals used in the assessment of a particular remedy should be listed in the next column. Any applicable or relevant and appropriate requirements should also be listed. Include both the identity of the requirement and its numerical value in this column. Next to this, list target concentrations for potentially carcinogenic indicator chemicals. Values for each chemical and each transport medium of concern have been summarized on Worksheet 8-10 and should be recorded here also. In the next column, list the individual target risks due to each potential carcinogen. These target risks were the bases for the calculated target concentrations in the previous column. The target risk column should display how carcinogenic risk has been apportioned among the chemicals at the site, as determined on Worksheet 8-10.

Noncarcinogenic risk should be summarized in the next column. Results of the chronic hazard index calculation should be included and risks from each remedial alternative should be described. If no risks are expected, that should be noted also. Information on noncarcinogenic risks can be found on Worksheets 8-13 and 8-14. Short-term risks should also be qualitatively described. Identify each and briefly discuss how they can be managed at the site. These risks were identified on Worksheet 8-16.

The possible effects and public health consequences of remedy failure, discussed in Section 8.9, should be summarized in the next column. Any information concerning the significant sources of uncertainty involved in the calculations, assumptions, or data inputs for the performance goals portion of the risk assessment should be discussed next. Comments about assumptions, difficulties, results, and conclusions should be written in the final column.

The process of public health evaluation is complete when all remedies under consideration, including the no-action alternative, have been summarized. Site decision-makers can use this information along with other elements of the feasibility study (e.g. engineering reliability of alternatives, life-cycle costs, and cost-effectiveness) in the selection of a remedial alternative.

# CHAPTER 10
# REFERENCES


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## *APPENDIX A*

## *SUMMARY TABLES FOR CHEMICAL-SPECIFIC DATA*

Appendix A contains the following six summary data tables:

- Exhibit A-1: Physical, Chemical, and Fate Data
- Exhibit A-2: Half-Lives in Various Media
- Exhibit A-3: Toxicity Data for Potential Carcinogenic Effects -- Selection of Indicator Chemicals Only
- Exhibit A-4: Toxicity Data for Potential Carcinogenic Effects -- Risk Characterization
- Exhibit A-5: Toxicity Data for Noncarcinogenic Effects -- Selection of Indicator Chemicals Only
- Exhibit A-6: Toxicity Data for Noncarcinogenic Effects -- Risk Characterization

These tables summarize key quantitative parameters for more than 300 chemicals or chemical groups that were evaluated as part of the Superfund reportable quantity (RQ) adjustment process or the intra-agency reference dose (RfD) review process. These specific chemicals are included because of the amounts of readily available toxicity information. This list should not be interpreted as a complete list of chemicals of concern at Superfund sites. Other substances may be important at certain sites. However, this appendix covers many toxic chemicals commonly detected at Superfund sites.

Chemical-specific parameters listed in the tables are primarily those referred to in this manual, although a limited amount of other useful information (e.g., CAS number, molecular weight) is also provided. Values for physical, chemical, and fate parameters given in Exhibits A-1 and A-2 are provided for the convenience of the user and have not been fully peer reviewed within EPA. Conversely, values given in Exhibits A-4 and A-6 for acceptable intake level and/or carcinogenic potency have been reviewed within EPA and should generally be used in the public health evaluation process at Superfund sites. The sources of values and data transformation procedures, if any, are described in the following sections.

In addition to the six data summary tables described above, a list of chemicals for which EPA Health Effects Assessment documents are available is provided in Exhibit A-7.

### A-1 Exhibit A-1: Physical, Chemical, and Fate Data

The physical, chemical, and fate data shown in Exhibit A-1 were either recorded directly from standard secondary references or were derived based on information contained in such references. A general hierarchy of sources was established, and values were taken from sources in order of the hierarchy. The hierarchy was ordered with documents developed specifically for the Superfund program at the top, followed by other relevant EPA data compilations, and then general reference texts at the bottom. In general, succeeding references were used only when a value could not be obtained from a reference higher in the hierarchy. Priority was given to more recent sources, and measured values were chosen over estimated values even if obtained from a source lower on the hierarchy. The hierarchy of sources used to select values for Exhibit A-1 is shown below and is lettered to correspond with the sources referenced in the exhibit. More complete reference information for each of these sources is in the reference list for Appendix A. A brief description of the derivation of values for each parameter in Exhibit A-1 follows the hierarchy listed below.

A) ECAO, EPA, Health Effects Assessments, 1985
B) Jaber *et al.*, 1984
C) Mabey *et al.*, 1982
D) Callahan *et al.*, 1979
E) ORD, EPA, 1981
F) Dawson *et al.*, 1980
G) Lyman *et al.*, 1982
H) OWRS, EPA, 1980
I) Weast *et al.*, 1979
J) Verschueren, 1983
K) Windholz *et al.*, 1976
L) Perry and Chilton, 1973
M) OSW, EPA, 1984b
N) OSW, EPA, 1984a

Water Solubility is the maximum concentration of a chemical that dissolves in pure water at a specific temperature and pH. It is a critical property affecting environmental fate and transport. Values for water solubility, in mg/l, were recorded in Exhibit A-1 directly using the hierarchy of sources and general decision rules outlined above. Values are given for a neutral pH and a temperature range of 20 to 30°C. Chemicals listed in the literature as being "infinitely soluble" were assigned a solubility value of 1,000,000 mg/l.

Vapor Pressure is a relative measure of the volatility of a chemical in its pure state and is an important determinant of the rate of vaporization from waste sites. Values for this parameter, in units mm Hg, were recorded directly from the hierarchy of sources described above. Values are given for a temperature range of 20 to 30° C.

Henry's Law Constant is another parameter important in evaluating air exposure pathways. Values for Henry's Law Constant (H) were calculated using the following equation and the values previously recorded for solubility, vapor pressure, and molecular weight:

$$H(atm-m^3/mole)=\frac{vapor\ pressure(atm)\times MW(g/mole)}{water\ solubility\ (g/m^3)}$$

Organic Carbon Partition Coefficient (Koc) is a measure of the tendency for organics to be adsorbed by soil and sediment and is expressed as:

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

The Koc is chemical specific and is largely independent of soil properties. Most Koc values in Exhibit A-1 were recorded directly from the above hierarchy of sources. However, some Koc values were estimated using methods specified in Lyman (1982). Estimated values are clearly designated as such.

Octanol-Water Partition Coefficient (Kow) is a measure of how a chemical is distributed at equilibrium between octanol and water. Although Kow is not directly referenced in the text of this manual, it is an important parameter and is used often in the assessment of environmental fate and transport for organic chemicals. Additionally, Kow is a key variable used in the estimation of other properties. For the convenience of the user, values for log Kow have been included in Exhibit A-1. These values were recorded directly from the hierarchy of sources referenced above.

Bioconcentrateion Factor as used in this manual is a measure of the tendency for a chemical contaminant in water to accumulate in fish tissue. The equilibrium concentration of a contaminant in fish can be estimated by multiplying the concentration of the chemical in surface water by the fish bioconcentration factor for that chemical. This parameter is therefore an important determinant for human intakes via the aquatic food ingestion route. Values for bioconcentration factors shown in Exhibit A-1 were recorded directly from the above hierarchy of sources.

## A.2 Exhibit A-2 : Half-Lives in Various Media

Chemical Half-Lives are used in this manual as measures of persistence, or how long a chemical will remain, in various environmental media. Exhibit A-2 presents values for overall half-lives, which are the result of all removal processes (e.g., phase transfer, chemical transformation, and biological transformation) acting together rather than a single removal mechanism. All of the half-life values in Exhibit A-2 were recorded directly from two sources, ECAO Health Effects Assessments (ECAO, 1985) and exposure profiles for the RCRA Risk-Cost Analysis Model (OSW, 1984b). The same source lettering convention was followed for Exhibit A-2 as for Exhibit A-1.

## A.3 Exhibit A-3: Toxicity Data for Potential Carcinogenic Effects--Selection of Indicator Chemicals Only

For the risk assessment process outlined in this manual, data presented in Exhibit A-3 are used only in the selection of indicator chemicals and not in actual risk characterization. These data were obtained from information contained in the Reportable Quantity (RQ) data base (OHEA, 1986). The procedures used to convert source data to the values given in Exhibit A-3 are described briefly below.

The 10% Effective Dose ($ED_{10}$) represents the dose at which a 10 percent incremental carcinogenic response is observed. This parameter was calculated for both oral and inhalation routes by taking the reciprocal of the Potency Factor Estimate (PFE) given in the RQ data base (this source defines PFE = $1/ED_{10}$; therefore, $ED_{10}$ = 1/PFE). The $ED_{10}$ is in units of mg/kg/day.

Toxicity Constants vary for different exposure media. As such, Exhibit A-3 contains toxicity constant values specific to water (wTc) and soil (sTc) for the oral route, and a value for air (aTc) for the inhalation route. Each of these constants for potential carcinogens is based on the $ED_{10}$, standard intake assumptions for the respective media, and a standard body weight. The specific equations and assumptions used to calculate the various toxicity constants are presented and discussed in further detail in Appendix B.

## A.4 Exhibit A-4: Toxicity Data for Potential Carcinogenic Effects--Risk Characterization

Data presented in Exhibit A-4 are for use in risk characterization, as opposed to the selection of indicator chemicals. Values in this exhibit were derived in the following manner.

Carcinogenic Potency Factors are upper 95 percent confidence limits on the slope of the dose-response curve. These values were recorded directly from HEAs or CAG summary tables, with the actual source cited in the exhibit for each value and then fully referenced at the end of the exhibit. Potency factors are used to estimate potential carcinogenic risk. These factors, specific to different exposure routes, are given in Exhibit A-4 in units of $(mg/kg/day)^{-1}$.

Weight of Evidence ratings qualify the level of evidence that supports designating a chemical as a human carcinogen. Exhibit A-4 lists ratings based on EPA categories for potential carcinogens, which are fully itemized in Exhibit B-2. The ratings were recorded directly from the RQ data base. (Note: Weight-of-evidence ratings are also used in the procedure for selecting indicator chemicals.)

## A.5 Exhibit A-5: Toxicity Data for Noncarcinogenic Effects--Selection of Indicator Chemicals Only

The data in Exhibit A-5 were generated based on information contained in the RQ data base for chronic effects (ECAO, 1984). Values for the parameters in Exhibit A-5, which are used in the selection of indicator chemicals but not in risk characterization, were derived in the following manner. In addition, chemicals marked in Exhibit A-5 with "@" also exhibit potential carcinogenic effects. The reader is referred to Exhibits A-3 and A-4 for information concerning these effects.

To determine the human Minimum Effective Dose (MED), the RQ data base was reviewed to identify the studies with the highest composite score (a score that combines MED and severity of effect) for oral and for inhalation exposure routes. These MEDs were recorded under the appropriate exposure route in Exhibit A-5. If composite score values were reported to be equal, the study that yielded the lowest MED was used. For metals, one MED value was derived from all studies for the various compounds of a given metal. Human MED values are expressed in Exhibit A-5 in terms of mg/day. If an MED was available for only one exposure route, it was recorded in Exhibit A-5 for the other exposure routes without modification unless the toxic effect was at the site of entry.

Severity of effect Ratings, or RVe's, were recorded from the RQ data base for the same study used to determine MED values. These rating constants are unitless integers ranging from 1 to 10, corresponding to various levels of severity of effects. The severity scale is presented in Exhibit B-1.

Toxicity Constants for noncarcinogenic effects, like those for carcinogens, are specific to water, soil, and air and are designated in Exhibit A-5 as wTn, sTn, and aTn, respectively. Again, these toxicity constants are used only in the indicator chemical selection step of the process. Values in Exhibit A-5 are based on standard intake assumptions as well as a chemical's RVe and MED values. Refer to Appendix B for the specific toxicity constant equations and for a discussion on their application.

## A.6 Exhibit A-6: Toxicity Data for Noncarcinogenic Effects-- Risk Characterization

Exhibit A-6 gives values for parameters that are used in actual risk characterization. The methods used to derive these values are described below. Although the data in Exhibit A-6 are for noncarcinogenic effects, several of the chemicals listed in the exhibit (those marked with an "@") also exhibit potential carcinogenic effects. Exhibits A-3 and A-4 should be referred to for information concerning carcinogenic effects.

Subchronic acceptable intake (AIS) values are short-term acceptable intake levels and are recorded directly from the appropriate HEA. Likewise, values for chronic acceptable intake (AIC), which is the long-term acceptable intake level for noncarcinogenic effects, were recorded directly from the appropriate HEA or from compilations of Agency-verified reference dose (RfD) values. These verified reference doses were developed by an EPA work group chaired by the Office of Research and Development in 1985 and 1986. The actual source used for each value is cited in Exhibit A-6 and is referenced fully at the end of the exhibit. AIS and AIC are used to characterize risks of noncarcinogenic effects. Both AIS and AIC values are in units of mg/kg/day.

## *REFERENCES FOR APPENDIX A*


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Callahan *et al.*, 1979. Water-Related Environmental Fate of 129 Priority Pollutants, Volumes I and II, Office of Water Planning and Standards, Office of Water and Waste Management, U.S. EPA, EPA Contract Nos. 68-01-3852 and 68-01-3867. [Source D*]

Dawson, *et al.*, 1980. Physical/Chemical Properties of Hazardous Waste Constituents. Prepared By Southeast Environmnetal Research Laboratory for U.S. EPA. [Source F*]

ECAO, U.S. EPA, 1985. Health Effects Assessment for [Specific Chemical]. [Note: 58 individual documents available for specific chemicals or chemical groups] [Source A*]

ECAO, U.S. EPA, 1984. Summary Data Tables for Chronic Noncarcinogenic Effects. [Note: Prepared during RQ adjustment process]

Jaber, *et al.*, 1984. Data Acquisition for Environmental Transport and Fate Screening. Office of Health and Environmental Assessment, U.S. EPA, Washington, DC, EPA 600/6-84-009 [Source B*]

Lyman, 1982. Adsorption Coefficient for Soils and Sediments. Chapter 4 in Lyman *et al.*, *Handbook of Chemical Property Estimation Methods*. McGraw-Hill, New York.

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Mabey, *et al.*, 1982. Aquatic Fate Process Data for Organic Priority Pollutants. Prepared by SRI International, EPA Contract Nos. 68-01-3867 and 68-03-2981, prepared for Monitoring and Data Support Division, Office of Water Regulations and Standards, Washington, DC. [Source C*]

OHEA, U.S. EPA, 1986. Methodology for Evaluating Reportable Quantity Adjustments Pursuant to CERCLA Section 102, External Review Draft. OHEA-C-073.

ORD, U.S. EPA, 1981. Treatability Manual, Volume I, EPA 600/2-82-001a. [Source E*]

OSW, U.S. EPA, 1984a. Characterization of Constituents from Selected Waste Streams Listed in 40 CFR Section 261. Prepared by Environ Corporation. [Source N*]

OSW, U.S. EPA, 1984b. Exposure Profiles for RCRA Risk-Cost Analysis Model. Prepared by Environ Corporation. [Source M*]

OWRS, U.S. EPA, 1980. Ambient Water Quality Criteria Documents for [Specific Chemical]. [Source H*]

Perry and Chilton, 1973. *Chemical Engineers' Handbook*, McGraw-Hill, 5th Ed. [Source L*]

Verschueren, 1983. *Handbook of Environmental Data for Organic Chemicals*. Van Nostrand Reinhold Co., New York, 2nd ed. [Source J*]

Weast *et al.*, 1979. CRC *Handbook of Chemistry and Physics*. [Source I*]

Windholz, et al., 1976. *The Merck Index*. [Source K*] .


*Source letters correspond to Exhibits A-1 and A-2.

Date Prepared: October 1, 1986

EXHIBIT A-1

PHYSICAL, CHEMICAL, AND FATE DATA

| Chemical Name | CAS # | Mole Weight (g/mole) | Water Solubility (mg/l) | S* | Vapor Pressure (mm Hg) | S* | Henry's Law Constant (atm-m3/mol) | Koc (ml/g) | S* | Log Kow | S* | Fish BCF (l/kg) | S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Acenaphthene | 83-32-9 | 154 | 3.42E+00 | C | 1.55E-03 | C | 9.20E-05 | 4600 | C | 4.00 | C | 242 | H |
| Acenaphthylene | 208-96-8 | 152 | 3.93E+00 | C | 2.90E-02 | C | 1.48E-03 | 2500 | C | 3.70 | C | | |
| Acetone | 67-64-1 | 58 | 1.00E+06 | # | 2.70E+02 | J | 2.06E-05 | 2.2 | & | -0.24 | J | | |
| Acetonitrile | 75-05-8 | 41 | 1.00E+06 | # | 7.40E+01 | F | 4.00E-06 | 2.2 | & | -0.34 | F | | |
| 2-Acetylaminofluorene | 53-96-3 | 223 | 6.50E+00 | B | | | NA | 1600 | & | 3.28 | B | | |
| Acrylic Acid | 79-10-7 | 72 | 1.00E+06 | # | 4.00E+00 | F | | | | 0.13 | F | 0 | F |
| Acrylonitrile | 107-13-1 | 53 | 7.90E+04 | C | 1.00E+02 | C | 8.84E-05 | 0.85 | C | 0.25 | C | 48 | G |
| Aflatoxin B1 | 1162-65-8 | 312 | | | | | NA | | | | | | |
| Aldicarb | 116-06-3 | 190 | | | | | | | | | | | |
| Aldrin | 309-00-2 | 365 | 1.80E-01 | C | 6.00E-06 | C | 1.60E-05 | 96000 | C | 5.30 | C | 28 | H |
| Allyl Alcohol | 107-18-6 | 58 | 5.10E+05 | B | 2.46E+01 | B | 3.69E-06 | 3.2 | & | -0.22 | B | | |
| Aluminum Phosphide | 20859-73-8 | 58 | | | | | | | | | | | |
| 4-Aminobiphenyl | 92-67-1 | 169 | 8.42E+02 | B | 6.00E-05 | B | 1.59E-08 | 107 | & | 2.78 | B | | |
| Amitrole | 61-82-5 | 84 | 2.80E+05 | B | | | NA | 4.4 | & | -2.08 | B | | |
| Ammonia | 7664-41-7 | 17 | 5.30E+05 | F | 7.60E+03 | F | 3.21E-04 | 3.1 | & | 0.00 | F | 0 | F |
| Anthracene | 120-12-7 | 178 | 4.50E-02 | A | 1.95E-04 | A | 1.02E-03 | 14000 | C | 4.45 | A | | |
| Antimony and Compounds | 7440-36-0 | 122 | | | 1.00E+00 | N | NA | | | | | 1 | H |
| Arsenic and Compounds | 7440-38-2 | 75 | | | 0.00E+00 | E | NA | | | | | 44 | H |
| Asbestos | 1332-21-4 | NA | NA | | NA | | NA | NA | | NA | | 0 | D |
| Auramine | 2465-27-2 | 267 | 2.10E+00 | B | | | NA | 2900 | & | 4.16 | B | | |
| Azaserine | 115-02-6 | 173 | 1.36E+05 | B | | | NA | 6.6 | & | -1.08 | B | | |
| Aziridine | 151-56-4 | 43 | 2.66E+06 | B | 2.55E+02 | B | 5.43E-06 | 1.3 | & | -1.01 | | | |
| Barium and Compounds | 7440-39-3 | 137 | | | | | NA | | | | | | |
| Benefin | 1861-40-1 | 335 | | | | | | | | | | | |
| Benzene | 71-43-2 | 78 | 1.75E+03 | A | 9.52E+01 | A | 5.59E-03 | 83 | G | 2.12 | A | 5.2 | H |
| Benzidine | 92-87-5 | 184 | 4.00E+02 | C | 5.00E-04 | C | 3.03E-07 | 10.5 | C | 1.30 | C | 87.5 | H |
| Benz(a)anthracene | 56-55-3 | 228 | 5.70E-03 | C | 2.20E-08 | C | 1.16E-06 | 1380000 | G | 5.60 | C | | |
| Benz(c)acridine | 225-51-4 | 229 | 1.40E+01 | B | | | NA | 1000 | & | 4.56 | B | | |
| Benzo(a)pyrene | 50-32-8 | 252 | 1.20E-03 | A | 5.60E-09 | A | 1.55E-06 | 5500000 | C | 6.06 | C | | |
| Benzo(b)fluoranthene | 205-99-2 | 252 | 1.40E-02 | C | 5.00E-07 | C | 1.19E-05 | 550000 | C | 6.06 | A | | |
| Benzo(ghi)perylene | 191-24-2 | 276 | 7.00E-04 | A | 1.03E-10 | A | 5.34E-08 | 1600000 | C | 6.51 | A | | |
| Benzo(k)fluoranthene | 207-08-9 | 252 | 4.30E-03 | C | 5.10E-07 | C | 3.94E-05 | 550000 | C | 6.06 | C | | |
| Benzotrichloride | 98-07-7 | 195 | | | | | | | | | | | |
| Benzyl Chloride | 100-44-7 | 127 | 3.30E+03 | F | 1.00E+00 | E | 5.06E-05 | 50 | & | 2.63 | F | | |
| Beryllium and Compounds | 7440-41-7 | 9 | | | 0.00E+00 | E | NA | | | | | 19 | H |
| 1,1-Biphenyl | 92-52-4 | 154 | | | | | | | | | | | |
| Bis(2-chloroethyl)ether | 111-44-4 | 143 | 1.02E+04 | C | 7.10E-01 | C | 1.31E-05 | 13.9 | C | 1.50 | C | 6.9 | H |
| Bis(2-chloroisopropyl)ether | 108-60-1 | 171 | 1.70E+03 | C | 8.50E-01 | C | 1.13E-04 | 61 | C | 2.10 | C | 0 | D |
| Bis(chloromethyl)ether | 542-88-1 | 115 | 2.20E+04 | C | 3.00E+01 | C | 2.06E-04 | 1.2 | C | 0.38 | C | 0.63 | H |
| Bis(2-ethylhexyl)phthalate (DEHP) | 117-81-7 | 391 | | | | | | | | | | | |
| Bromomethane | 74-83-9 | 95 | | | | | | | | | | | |
| Bromoxynil Octanoate | 1689-99-2 | 403 | | | | | | | | | | | |
| 1,3-Butadiene | 106-99-0 | 54 | 7.35E+02 | F | 1.84E+03 | F | 1.78E-01 | 120 | & | 1.99 | F | | |
| n-Butanol | 71-36-3 | 74 | | | | | | | | | | | |
| Butylphthalyl Butylglycolate | 85-70-1 | 336 | | | | | | | | | | | |
| Cacodylic Acid | 75-60-5 | 138 | 8.30E+05 | F | | | NA | 2.4 | & | 0.00 | F | | |
| Cadmium and Compounds | 7740-43-9 | 112 | | | 0.00E+00 | E | NA | | | | | 81 | H |
| Captan | 133-06-2 | 301 | 5.00E-01 | E | 6.00E-05 | E | 4.75E-05 | 6400 | & | 2.35 | F | | |
| Carbaryl | 63-25-2 | 201 | 4.00E+01 | E | 5.00E-03 | E | | | | 2.36 | F | | |
| Carbon Disulfide | 75-15-0 | 76 | 2.94E+03 | E | 3.60E+02 | E | 1.23E-02 | 54 | & | 2.00 | F | 0 | F |
| Carbon Tetrachloride | 56-23-5 | 154 | 7.57E+02 | A | 9.00E+01 | A | 2.41E-02 | 110 | & | 2.64 | A | 19 | H |
| Chlordane | 57-74-9 | 410 | 5.60E-01 | A | 1.00E-05 | A | 9.63E-06 | 140000 | C | 3.32 | A | 14000 | H |
| Chlorobenzene | 108-90-7 | 113 | 4.66E+02 | A | 1.17E+01 | A | 3.72E-03 | 330 | C | 2.84 | A | 10 | H |
| Chlorobenzilate | 510-15-6 | 325 | 2.19E+01 | B | 1.20E-06 | B | 2.34E-08 | 800 | & | 4.51 | B | | |
| Chlorodibromomethane | 124-48-1 | 208 | | | 1.50E+01 | D | NA | | | 2.09 | D | | |

Date Prepared: October 1, 1986

EXHIBIT A-1
(Continued)

PHYSICAL, CHEMICAL, AND FATE DATA

| Chemical Name | CAS # | Mole Weight (g/mole) | Water Solubility (mg/l) | S* | Vapor Pressure (mm Hg) | S* | Henry's Law Constant (atm-m3/mol) | Koc (ml/g) | S* | Log Kow | S* | Fish BCF (l/kg) | S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Chloroform | 67-66-3 | 119 | 8.20E+03 | A | 1.51E+02 | A | 2.87E-03 | 31 | & | 1.97 | A | 3.75 | H |
| Chloromethyl Methyl Ether | 107-30-2 | 81 | | | | | NA | | | 0.00 | F | | |
| 4-Chloro-o-toluidine Hydrochloride | 3165-93-3 | 142 | | | | | NA | | | | | | |
| Chromium III and Compounds | 7440-47-3 | 52 | | | 0.00E+00 | E | NA | | | | | 16 | H |
| Chromium VI and Compounds | 7440-47-3 | 52 | | | 0.00E+00 | E | NA | | | | | 16 | H |
| Chrysene | 218-01-9 | 228 | 1.80E-03 | A | 6.30E-09 | A | 1.05E-06 | 200000 | C | 5.61 | A | | |
| Copper and Compounds | 7440-50-8 | 64 | | | 0.00E+00 | G | NA | | | | | 200 | D |
| Creosote | 8001-58-9 | NA | | | | | NA | | | | | | |
| Cresol | 1319-77-3 | 108 | 3.10E+04 | E | 2.40E-01 | J | 1.10E-06 | 500 | G | 1.97 | F | 0 | F |
| Crotonaldehyde | 123-73-9 | 70 | | | | | | | | | | | |
| Cyanides | 57-12-5 | NA | | | | | NA | | | | | | |
| -- Barium Cyanide | 542-62-1 | 189 | | | | | | | | | | | |
| -- Calcium Cyanide | 502-01-8 | 92 | | | | | | | | | | | |
| -- Copper Cyanide | 544-92-3 | 90 | | | | | | | | | | | |
| -- Cyanogen | 460-19-5 | 52 | 2.50E+05 | K | | | | | | | | | |
| -- Cyanogen Chloride | 506-77-4 | 61 | 2.50E+03 | F | 1.00E+03 | J | | | | 0.00 | F | | |
| -- Hydrogen Cyanide | 74-90-8 | 27 | 1.00E+06 | # | 6.20E+02 | E | | | | -0.25 | F | 0 | F |
| -- Nickel Cyanide | 557-19-7 | 182 | | | | | | | | | | | |
| -- Potassium Cyanide | 151-50-8 | 65 | 5.00E+05 | K | | | | | | | | | |
| -- Potassium Silver Cyanide | 506-61-6 | 199 | | | | | | | | | | | |
| -- Silver Cyanide | 506-64-9 | 134 | | | | | | | | | | | |
| -- Sodium Cyanide | 143-33-9 | 49 | 8.20E+05 | H | | | | | | | | | |
| -- Zinc Cyanide | 557-21-1 | 117 | | | | | | | | | | | |
| Cyclophosphamide | 50-18-0 | 261 | 1.31E+09 | B | | | NA | 0.042 | & | -3.22 | B | | |
| Dalapon | 75-99-0 | 143 | | | | | | | | | | | |
| DDD | 72-54-8 | 320 | 1.00E-01 | C | 1.89E-06 | C | 7.96E-06 | 770000 | C | 6.20 | C | | |
| DDE | 72-55-9 | 318 | 4.00E-02 | C | 6.50E-06 | C | 6.80E-05 | 4400000 | C | 7.00 | C | 51000 | G |
| DDT | 50-29-3 | 355 | 5.00E-03 | A | 5.50E-06 | A | 5.13E-04 | 243000 | G | 6.19 | J | 54000 | H |
| Decabromodiphenyl Ether | 1163-19-5 | 959 | | | | | | | | | | | |
| Diallate | 2303-16-4 | 274 | 1.40E+01 | B | 6.40E-03 | B | 1.65E-04 | 1000 | & | 0.73 | B | | |
| 2,4-Diaminotoluene | 95-80-7 | 122 | 4.77E+04 | B | 3.80E-05 | B | 1.28E-10 | 12 | & | 0.35 | B | | |
| 1,2,7,8-Dibenzopyrene | 189-55-9 | 305 | 1.10E-01 | B | | | NA | 1200 | & | 6.62 | B | | |
| Dibenz(a,h)anthracene | 53-70-3 | 278 | 5.00E-04 | C | 1.00E-10 | C | 7.33E-08 | 3300000 | C | 6.80 | C | | |
| 1,2-Dibromo-3-chloropropane | 96-12-8 | 236 | 1.00E+03 | B | 1.00E+00 | B | 3.11E-04 | 98 | & | 2.29 | B | | |
| Dibutylnitrosamine | 924-16-3 | 152 | | | | | NA | | | | | | |
| Dibutyl Phthalate | 84-74-2 | 278 | 1.30E+01 | C | 1.00E-05 | C | 2.82E-07 | 170000 | C | 5.60 | C | | |
| 1,2-Dichlorobenzene | 95-50-1 | 147 | 1.00E+02 | C | 1.00E+00 | C | 1.93E-03 | 1700 | C | 3.60 | C | 56 | H |
| 1,3-Dichlorobenzene | 541-73-1 | 147 | 1.23E+02 | C | 2.28E+00 | C | 3.59E-03 | 1700 | C | 3.60 | C | 56 | H |
| 1,4-Dichlorobenzene | 106-46-7 | 147 | 7.90E+01 | C | 1.18E+00 | C | 2.89E-03 | 1700 | C | 3.60 | C | 56 | H |
| 3,3'-Dichlorobenzidine | 91-94-1 | 253 | 4.00E+00 | C | 1.00E-05 | C | 8.33E-07 | 1553 | C | 3.50 | C | 312 | H |
| Dichlorodifluoromethane | 75-71-8 | 121 | 2.80E+02 | C | 4.87E+03 | | | 58 | C | 2.16 | D | | |
| 1,1-Dichloroethane | 75-34-3 | 99 | 5.50E+03 | A | 1.82E+02 | A | 4.31E-03 | 30 | C | 1.79 | A | | |
| 1,2-Dichloroethane (EDC) | 107-06-2 | 99 | 8.52E+03 | A | 6.40E+01 | A | 9.78E-04 | 14 | C | 1.48 | A | 1.2 | H |
| 1,1-Dichloroethylene | 75-35-4 | 97 | 2.25E+03 | A | 6.00E+02 | A | 3.40E-02 | 65 | C | 1.84 | A | 5.6 | H |
| 1,2-Dichloroethylene (trans) | 540-59-0 | 97 | 6.30E+03 | A | 3.24E+02 | A | 6.56E-03 | 59 | C | 0.48 | A | 1.6 | H |
| 1,2-Dichloroethylene (cis) | 540-59-0 | 97 | 3.50E+03 | A | 2.08E+02 | A | 7.58E-03 | 49 | & | 0.70 | A | 1.6 | H |
| Dichloromethane | 75-09-2 | 85 | 2.00E+04 | C | 3.62E+02 | C | 2.03E-03 | 8.8 | C | 1.30 | C | 5 | H |
| 2,4-Dichlorophenol | 120-83-2 | 163 | 4.60E+03 | C | 5.90E-02 | C | 2.75E-06 | 380 | C | 2.90 | C | 41 | H |
| 2,4-Dichlorophenoxyacetic Acid (2,4-D) | 94-75-7 | 221 | 6.20E+02 | F | 4.00E-01 | F | 1.88E-04 | 20 | G | 2.81 | F | | |
| 4-(2,4-Dichlorophenoxy)butyric Acid (2,4-DB) | 94-82-6 | | | | | | | | | | | | |
| Dichlorophenylarsine | 696-28-6 | 223 | | | | | NA | | | | | | |
| 1,2-Dichloropropane | 78-87-5 | 113 | 2.70E+03 | C | 4.20E+01 | C | 2.31E-03 | 51 | C | 2.00 | C | | |

Date Prepared: October 1, 1986

EXHIBIT A-1
(Continued)

PHYSICAL, CHEMICAL, AND FATE DATA

| Chemical Name | CAS # | Mole Weight (g/mole) | Water Solubility (mg/l) | S* | Vapor Pressure (mm Hg) | S* | Henry's Law Constant (atm-m3/mol) | Koc (ml/g) | S* | Log Kow | S* | Fish BCF (l/kg) | S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1,3-Dichloropropene | 542-75-6 | 111 | 2.80E+03 | C | 2.50E+01 | C | 1.30E-03 | 48 | C | 2.00 | C | 1.9 | H |
| Dieldrin | 60-57-1 | 381 | 1.95E-01 | C | 1.78E-07 | C | 4.58E-07 | 1700 | C | 3.50 | C | 4760 | H |
| Diepoxybutane | 1464-53-5 | 86 | | | | | NA | | | | | | |
| Diethanolnitrosamine | 1116-54-7 | 134 | | | | | NA | | | | | | |
| Diethyl Arsine | 692-42-2 | 134 | 4.17E+02 | B | 3.50E+01 | B | 1.48E-02 | 160 | & | 2.97 | B | | |
| 1,2-Diethylhydrazine | 1615-80-1 | 88 | 2.88E+07 | B | | | NA | 0.3 | & | -1.68 | B | | |
| Diethylnitrosamine | 55-18-5 | 102 | | | 5.00E+00 | F | NA | | | 0.48 | F | | |
| Diethyl Phthalate | 84-66-2 | 222 | 8.96E+02 | C | 3.50E-03 | C | 1.14E-06 | 142 | C | 2.50 | C | 117 | G |
| Diethylstilbestrol (DES) | 56-53-1 | 268 | 9.60E-03 | B | | | NA | 28 | & | 5.46 | B | | |
| Dihydrosafrole | 94-58-6 | 164 | 1.50E+03 | B | | | NA | 78 | & | 2.56 | B | | |
| Dimethoate | 60-51-5 | 229 | 2.50E+04 | J | 2.50E-02 | J | | | | 2.71 | J | | |
| 3,3'-Dimethoxybenzidine | 119-90-4 | 244 | | | | | NA | | | | | | |
| Dimethylamine | 124-40-3 | 45 | 1.00E+06 | F | 1.52E+03 | F | 9.02E-05 | 2.2 | & | -0.38 | F | 0 | F |
| Dimethyl Sulfate | 77-78-1 | 126 | 3.24E+05 | B | 6.80E-01 | B | 3.48E-07 | 4.1 | & | -1.24 | B | | |
| Dimethyl Terephthalate | 120-61-6 | 194 | | | | | | | | | | | |
| Dimethylaminoazobenzene | 60-11-7 | 225 | 1.36E+01 | B | 3.30E-07 | B | 7.19E-09 | 1000 | & | 3.72 | B | | |
| 7,12-Dimethylbenz(a)anthracene | 57-97-6 | 256 | 4.40E-03 | B | | | NA | 476000 | G | 6.94 | B | | |
| 3,3'-Dimethylbenzidine | 119-93-7 | 212 | | | | | | | | | | | |
| Dimethylcarbamoyl Chloride | 79-44-7 | 108 | 1.44E+07 | B | 1.95E+00 | B | 1.92E-08 | 0.5 | & | -1.32 | B | | |
| 1,1-Dimethylhydrazine | 57-14-4 | 60 | 1.24E+08 | B | 1.57E+02 | B | 1.00E-07 | 0.2 | & | -2.42 | B | | |
| 1,2-Dimethylhydrazine | 540-73-8 | 60 | | | | | NA | | | | | | |
| Dimethylnitrosamine | 62-75-9 | 74 | 1.00E+06 | # | 8.10E+00 | C | 7.90E-07 | 0.1 | C | -0.68 | C | 0 | D |
| 1,3-Dinitrobenzene | 99-65-0 | 168 | 4.70E+02 | J | | | NA | 150 | & | 1.62 | F | | |
| 4,6-Dinitro-o-cresol | 534-52-1 | 198 | 2.90E+02 | C | 5.00E-02 | C | 4.49E-05 | 240 | C | 2.70 | C | 0 | E |
| 2,4-Dinitrophenol | 51-28-5 | 184 | 5.60E+03 | C | 1.49E-05 | C | 6.45E-10 | 16.6 | C | 1.50 | C | 0 | D |
| 2,3-Dinitrotoluene | 602-01-7 | 182 | 3.10E+03 | B | | | NA | 53 | & | 2.29 | B | 3.8 | H |
| 2,4-Dinitrotoluene | 121-14-2 | 182 | 2.40E+02 | C | 5.10E-03 | C | 5.09E-06 | 45 | C | 2.00 | C | 3.8 | H |
| 2,5-Dinitrotoluene | 619-15-8 | 182 | 1.32E+03 | B | | | NA | 84 | & | 2.28 | B | 3.8 | H |
| 2,6-Dinitrotoluene | 606-20-2 | 182 | 1.32E+03 | B | 1.80E-02 | C | 3.27E-06 | 92 | C | 2.00 | C | 3.8 | H |
| 3,4-Dinitrotoluene | 610-39-9 | 182 | 1.08E+03 | B | | | NA | 94 | & | 2.29 | B | 3.8 | H |
| Dinoseb | 88-85-7 | 240 | 5.00E+01 | J | | | | | | | | | |
| 1,4-Dioxane | 123-91-1 | 88 | 4.31E+05 | B | 3.99E+01 | B | 1.07E-05 | 3.5 | & | 0.01 | B | | |
| N,N-Diphenylamine | 122-39-4 | 169 | 5.76E+01 | B | 3.80E-05 | B | 1.47E-07 | 470 | & | 3.60 | B | 30 | G |
| 1,2-Diphenylhydrazine | 122-66-7 | 184 | 1.84E+03 | C | 2.60E-05 | C | 3.42E-09 | 418 | C | 2.90 | C | 25 | H |
| Dipropylnitrosamine | 621-64-7 | 130 | 9.90E+03 | C | 4.00E-01 | C | 6.92E-06 | 15 | C | 1.50 | C | | |
| Disulfoton | 298-04-4 | 274 | | | | | | | | | | | |
| Endosulfan | 115-29-7 | 407 | | | | | | | | | | | |
| Epichlorohydrin | 106-89-8 | 93 | 6.00E+04 | J | 1.57E+01 | B | 3.19E-05 | 10 | & | 0.15 | B | | |
| Ethanol | 64-17-5 | 46 | 1.00E+06 | # | 7.40E+02 | G | 4.48E-05 | 2.2 | & | -0.32 | J | | |
| Ethyl Acetate | 141-78-6 | 88 | | | | | | | | | | | |
| Ethyl Methanesulfonate | 62-50-0 | 124 | 3.69E+05 | B | 2.06E-01 | B | 9.12E-08 | 3.8 | & | 0.21 | B | | |
| Ethylbenzene | 100-41-4 | 106 | 1.52E+02 | A | 7.00E+00 | A | 6.43E-03 | 1100 | C | 3.15 | A | 37.5 | H |
| Ethyl-4,4'-dichlorobenzilate | 510-15-6 | 352 | | | | | | | | | | | |
| Ethylene Dibromide (EDB) | 106-93-4 | 188 | 4.30E+03 | J | 1.17E+01 | B | 6.73E-04 | 44 | G | 1.76 | B | | |
| Ethylene Oxide | 75-21-8 | 44 | 1.00E+06 | B | 1.31E+03 | B | 7.56E-05 | 2.2 | & | -0.22 | B | | |
| Ethylenethiourea | 96-45-7 | 102 | 2.00E+03 | F | | | NA | 67 | & | -0.66 | J | 0 | F |
| 1-Ethyl-nitrosourea | 759-73-9 | 117 | 3.31E+08 | B | | | NA | 0.1 | & | | | | |
| Ethylphthalyl Ethyl Glycolate | 84-72-0 | 280 | | | | | | | | | | | |
| Ferric Dextran | 9004-66-4 | 7500 | | | | | NA | | | | | | |
| Fluoranthene | 206-44-0 | 202 | 2.06E-01 | A | 5.00E-06 | A | 6.46E-06 | 38000 | C | 4.90 | A | 1150 | H |
| Fluorene | 86-73-7 | 116 | 1.69E+00 | C | 7.10E-04 | C | 6.42E-05 | 7300 | C | 4.20 | C | 1300 | G |
| Fluorides | 7782-41-4 | NA | | | | | NA | | | | | | |
| Fluridone | 59756-60-4 | 329 | | | | | | | | | | | |
| Formaldehyde | 50-00-0 | 30 | 4.00E+05 | K | 1.00E+01 | E | 9.87E-07 | 3.6 | & | 0.00 | F | 0 | F |

Date Prepared: October 1, 1986

EXHIBIT A-1
(Continued)

PHYSICAL, CHEMICAL, AND FATE DATA

| Chemical Name | CAS # | Mole Weight (g/mole) | Water Solubility (mg/l) | S* | Vapor Pressure (mm Hg) | S* | Henry's Law Constant (atm-m3/mol) | Koc (ml/g) | S* | Log Kow | S* | Fish BCF (l/kg) | S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Formic Acid | 64-18-6 | 46 | 1.00E+06 | | 4.00E+01 | E | | | | -0.54 | F | 0 | F |
| Furan | 110-00-9 | 68 | | | | | | | | | | | |
| Glycidaldehyde | 765-34-4 | 72 | 1.70E+08 | B | 1.97E+01 | B | 1.10E-08 | 0.1 | & | -1.55 | B | | |
| Glycol Ethers | NA | NA | | | | | NA | | | | | | |
| -- Diethylene Glycol, Monoethyl Ether | 111-90-0 | 134 | | | | | | | | | | | |
| -- 2-Ethoxyethanol | 110-80-5 | 90 | 1.00E+06 | F | | | | | | 0.00 | F | | |
| -- Ethylene Glycol, Monobutyl Ether | 111-76-2 | 118 | 1.00E+06 | F | | | | | | 0.00 | F | | |
| -- 2-Methoxyethanol | 109-86-4 | 76 | 1.00E+06 | K | | | | | | | | | |
| -- Propylene Glycol, Monoethyl Ether | 52125-53-8 | 104 | | | | | | | | | | | |
| -- Propylene Glycol, Monomethyl Ether | 107-98-2 | 90 | | | | | | | | | | | |
| Heptachlor | 76-44-8 | 374 | 1.80E-01 | C | 3.00E-04 | C | 8.19E-04 | 12000 | C | 4.40 | C | 15700 | H |
| Heptachlor Epoxide | 1024-57-3 | 389 | 3.50E-01 | C | 3.00E-04 | C | 4.39E-04 | 220 | C | 2.70 | C | 14400 | G |
| Hexachlorobenzene | 118-74-1 | 285 | 6.00E-03 | A | 1.09E-05 | A | 6.81E-04 | 3900 | G | 5.23 | A | 8690 | H |
| Hexachlorobutadiene | 87-68-3 | 261 | 1.50E-01 | A | 2.00E+00 | A | 4.57E+00 | 29000 | C | 4.78 | A | 2.8 | H |
| Hexachlorocyclopentadiene | 77-47-4 | 273 | 2.10E+00 | A | 8.00E-02 | A | 1.37E-02 | 4800 | C | 5.04 | A | 4.3 | H |
| alpha-Hexachlorocyclohexane (HCCH) | 319-84-6 | 291 | 1.63E+00 | C | 2.50E-05 | C | 5.87E-06 | 3800 | C | 3.90 | C | 130 | H |
| beta-HCCH | 319-85-7 | 291 | 2.40E-01 | C | 2.80E-07 | C | 4.47E-07 | 3800 | C | 3.90 | C | 130 | H |
| gamma-HCCH (Lindane) | 58-89-9 | 291 | 7.80E+00 | C | 1.60E-04 | C | 7.85E-06 | 1080 | G | 3.90 | C | 130 | H |
| delta-HCCH | 319-86-8 | 291 | 3.14E+01 | C | 1.70E-05 | C | 2.07E-07 | 6600 | C | 4.10 | C | 130 | H |
| Hexachloroethane | 67-72-1 | 237 | 5.00E+01 | C | 4.00E-01 | C | 2.49E-03 | 20000 | C | 4.60 | C | 87 | H |
| Hexachlorophene | 70-30-4 | 407 | 4.00E-03 | F | | | NA | 91000 | & | 7.54 | F | | |
| Hydrazine | 302-01-1 | 32 | 3.41E+08 | B | 1.40E+01 | B | 1.73E-09 | 0.1 | & | -3.08 | B | | |
| Hydrogen Sulfide | 7783-06-4 | 34 | 4.13E+03 | K | | | | | | | | | |
| Indeno(1,2,3-cd)pyrene | 193-39-5 | 276 | 5.30E-04 | C | 1.00E-10 | C | 6.86E-08 | 1600000 | C | 6.50 | C | | |
| Iodomethane | 77-88-4 | 142 | 1.40E+04 | J | 4.00E+02 | J | 5.34E-03 | 23 | & | 1.69 | J | | |
| Iron and Compounds | 15438-31-0 | 56 | | | | | NA | | | | | | |
| Isobutanol | 78-83-1 | 74 | | | | | | | | | | | |
| Isoprene | 78-79-5 | 68 | | | 4.00E+02 | E | NA | | | | | | |
| Isosafrole | 120-58-1 | 168 | 1.09E+03 | B | 1.60E-08 | B | 3.25E-12 | 93 | & | 2.66 | B | | |
| Isophorone | 78-59-1 | 138 | | | | | | | | | | | |
| Isopropalin | 33820-53-0 | 309 | | | | | | | | | | | |
| Kepone | 143-50-0 | 491 | 9.90E-03 | B | | | NA | 55000 | & | 2.00 | B | 8400 | G |
| Lasiocarpine | 303-34-4 | 412 | 1.60E+03 | B | | | NA | 76 | & | 0.99 | B | | |
| Lead and Compounds (Inorganic) | 7439-92-1 | 207 | | | 0.00E+00 | E | NA | | | | | 49 | H |
| Linuron | 330-55-2 | 249 | | | | | | | | | | | |
| Malathion | 121-75-7 | 330 | 1.45E+02 | E | 4.00E-05 | E | | | | 2.89 | J | 0 | F |
| Manganese and Compounds | 7439-96-5 | 55 | | | | | NA | | | | | | |
| Melphalan | 148-82-3 | 305 | | | | | | | | | | | |
| Mercury and Compounds (Alkyl) | 7439-97-6 | | | | | | NA | | | | | 3750 | H |
| Mercury and Compounds (Inorganic) | 7439-97-6 | 201 | | | 2.00E-03 | E | NA | | | | | 5500 | H |
| Mercury Fulminate | 628-86-4 | 285 | | | | | | | | | | | |
| Methanol | 67-56-1 | 32 | | | | | | | | | | | |
| Methyl Chloride | 74-87-3 | 50 | 6.50E+03 | C | 4.31E+03 | B | 4.40E-02 | 35 | & | 0.95 | B | | |
| Methyl Ethyl Ketone | 78-93-3 | 72 | 2.68E+05 | A | 7.75E+01 | A | 2.74E-05 | 4.5 | & | 0.26 | A | 0 | F |
| Methyl Ethyl Ketone Peroxide | 1338-23-4 | 176 | | | | | | | | | | | |
| Methyl Isobutyl Ketone | 108-10-1 | 100 | | | | | | | | | | | |
| Methyl Methacrylate | 80-62-6 | 100 | 2.00E+01 | F | 3.70E+01 | E | 2.43E-01 | 840 | & | 0.79 | F | | |
| Methyl Parathion | 298-00-0 | 263 | 6.00E+01 | E | 9.70E-06 | E | 5.59E-08 | 460 | & | 1.91 | F | 45 | F |
| 2-Methyl-4-chlorophenoxyacetic Acid | 94-74-6 | 201 | | | | | | | | | | | |
| 2(2-Methyl)-4-Chlorophenoxy-propionic Acid | 93-65-2 | 215 | | | | | | | | | | | |

Date Prepared: October 1, 1986

EXHIBIT A-1
(Continued)

PHYSICAL, CHEMICAL, AND FATE DATA

| Chemical Name | CAS # | Mole Weight (g/mole) | Water Solubility (mg/l) | S* | Vapor Pressure (mm Hg) | S* | Henry's Law Constant (atm-m3/mol) | Koc (ml/g) | S* | Log Kow | S* | Fish BCF (l/kg) | S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 3-Methylcholanthrene | 56-49-3 | 268 | | | | | NA | | | | | | |
| 4,4'-Methylene-bis-2-chloroaniline | 101-14-4 | 267 | | | | | NA | | | | | | |
| Methylnitrosourea | 684-93-5 | 103 | 6.89E+08 | B | | | NA | 0.1 | & | -3.81 | B | | |
| Methylthiouracil | 56-04-2 | 142 | | | | | NA | | | | | | |
| Methylvinylnitrosamine | 4549-40-0 | 86 | 7.60E+05 | B | 1.23E+01 | B | 1.83E-06 | 2.5 | & | -0.23 | B | | |
| N-Methyl-N'-nitro-N-nitrosoguanadin | 70-25-7 | 147 | | | | | NA | | | | | | |
| Mitomycin C | 50-07-7 | 334 | | | | | NA | | | | | | |
| Mustard Gas | 505-60-2 | 159 | 8.00E+02 | B | 1.70E-01 | B | 4.45E-05 | 110 | & | 1.37 | B | | |
| 1-Napthylamine | 134-32-7 | 143 | 2.35E+03 | B | 6.50E-05 | B | 5.21E-09 | 61 | & | 2.07 | B | | |
| 2-Napthylamine | 91-59-8 | 143 | 5.86E+02 | B | 2.56E-04 | B | 8.23E-08 | 130 | & | 2.07 | B | | |
| Nickel and Compounds | 7440-02-0 | 59 | | | 0.00E+00 | D | NA | | | | | 47 | H |
| Nitric Oxide | 10102-43-9 | 30 | | | | | | | | | | | |
| Nitrobenzene | 98-95-3 | 123 | 1.90E+03 | C | 1.50E-01 | B | | 36 | C | 1.85 | D | | |
| Nitrogen Dioxide | 10102-44-0 | 46 | | | | | | | | | | | |
| Nitrosomethylurethane | 615-53-2 | 132 | | | | | NA | | | | | | |
| N-Nitrosopiperidine | 100-75-4 | 114 | 1.90E+06 | B | 1.40E-01 | B | 1.11E-08 | 1.5 | & | -0.49 | B | | |
| N-Nitrosopyrrolidine | 930-55-2 | 100 | 7.00E+06 | B | 1.10E-01 | B | 2.07E-09 | 0.8 | & | -1.06 | B | | |
| 5-Nitro-o-toluidine | 99-55-8 | 152 | | | | | NA | | | | | | |
| Osmium Tetroxide | 20816-12-0 | 254 | | | | | | | | | | | |
| Pentachlorobenzene | 608-93-5 | 250 | 1.35E-01 | F | | | NA | 13000 | & | 5.19 | F | 2125 | H |
| Pentachloronitrobenzene | 82-68-8 | 295 | 7.11E-02 | B | 1.13E-04 | B | 6.18E-04 | 19000 | & | 5.45 | B | | |
| Pentachlorophenol | 87-86-5 | 266 | 1.40E+01 | C | 1.10E-04 | C | 2.75E-06 | 53000 | C | 5 | C | 770 | G |
| Phenacetin | 62-44-2 | 179 | | | | | NA | | | | | | |
| Phenanthrene | 85-01-8 | 178 | 1.00E+00 | A | 6.80E-04 | A | 1.59E-04 | 14000 | C | 4.46 | A | 2630 | G |
| Phenobarbital | 50-06-6 | 232 | 1.00E+03 | B | | | NA | 98 | & | -0.19 | B | | |
| Phenol | 108-95-2 | 94 | 9.30E+04 | A | 3.41E-01 | A | 4.54E-07 | 14.2 | C | 1.46 | A | 1.4 | H |
| Phenylalanine Mustard | 148-82-3 | 305 | | | | | NA | | | | | | |
| m-Phenylenediamine | 108-45-2 | 108 | | | | | | | | | | | |
| Phenyl Mercuric Acetate | 62-38-4 | 337 | 1.67E+03 | K | | | | | | | | | |
| Phosphine | 7803-51-2 | 34 | | | | | | | | | | | |
| Polychlorinated Biphenyls (PCBs) | 1336-36-3 | 328 | 3.10E-02 | C | 7.70E-05 | C | 1.07E-03 | 530000 | C | 6.04 | C | 100000 | G |
| Propane Sultone | 1120-71-4 | 122 | | | | | NA | | | | | | |
| Propylenimine | 75-55-8 | 57 | 9.44E+05 | B | 1.41E+02 | B | 1.12E-05 | 2.3 | & | -0.48 | B | | |
| Pyrene | 129-00-0 | 202 | 1.32E-01 | A | 2.50E-06 | A | 5.04E-06 | 38000 | C | 4.88 | A | | |
| Pyridine | 110-86-1 | 79 | 1.00E+06 | F | 2.00E+01 | F | | | | 0.66 | F | | |
| Saccharin | 81-07-2 | 183 | | | | | NA | | | | | | |
| Safrole | 94-59-7 | 162 | 1.50E+03 | B | 9.10E-04 | B | 1.29E-07 | 78 | & | 2.53 | B | | |
| Selenium and Compounds | 7782-49-2 | 79 | | | 0.00E+00 | E | NA | | | | | 16 | H |
| -- Selenious Acid | 7783-00-8 | 129 | | | | | | | | | | | |
| -- Selenourea | 630-10-4 | 123 | | | | | | | | | | | |
| -- Thallium Selenite | 12039-52-0 | 488 | | | | | | | | | | | |
| Silver and Compounds | 7440-22-4 | 108 | | | 0.00E+00 | D | NA | | | | | 3080 | D |
| Sodium Diethyldithiocarbamate | 148-18-5 | 171 | | | | | | | | | | | |
| Streptozocin | 18883-66-4 | 457 | | | | | NA | | | | | | |
| Strychnine | 57-24-9 | 334 | 1.56E+02 | E | | | | | | | | | |
| Styrene | 100-42-5 | 104 | | | | | | | | | | | |
| 1,2,4,5-Tetrachlorobenzene | 95-94-3 | 216 | 6.00E+00 | F | | | NA | 1600 | & | 4.67 | F | 1125 | H |
| 2,3,7,8-TCDD (Dioxin) | 1746-01-6 | 322 | 2.00E-04 | A | 1.70E-06 | A | 3.60E-03 | 3300000 | C | 6.72 | A | 5000 | H |
| 1,1,1,2-Tetrachloroethane | 630-20-6 | 168 | 2.90E+03 | J | 5.00E+00 | J | 3.81E-04 | 54 | & | | | | |
| 1,1,2,2-Tetrachloroethane | 79-34-5 | 168 | 2.90E+03 | A | 5.00E+00 | A | 3.81E-04 | 118 | C | 2.39 | A | 42 | H |
| Tetrachloroethylene | 127-18-4 | 166 | 1.50E+02 | A | 1.78E+01 | A | 2.59E-02 | 364 | C | 2.6 | A | 31 | H |
| 2,3,4,6-Tetrachlorophenol | 58-90-2 | 232 | 1.00E+03 | F | | | NA | 98 | & | 4.1 | F | 240 | H |
| 2,3,5,6-Tetrachloroterephthalate Acid (DCPA) | 1861-32-1 | 332 | | | | | | | | | | | |

Date Prepared: October 1, 1986

EXHIBIT A-1
(Continued)

PHYSICAL, CHEMICAL, AND FATE DATA

| Chemical Name | CAS # | Mole Weight (g/mole) | Water Solubility (mg/l) | S* | Vapor Pressure (mm Hg) | S* | Henry's Law Constant (atm-m3/mol) | Koc (ml/g) | S* | Log Kow | S* | Fish BCF (l/kg) | S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Tetraethyl Lead | 78-00-2 | 323 | 8.00E-01 | J | 1.50E-01 | J | 7.97E-02 | 4900 | & | | | | |
| Thallium and Compounds | 7440-28-0 | 204 | | | 0.00E+00 | E | | | | | | | |
| -- Thallium Acetate | 563-68-8 | 263 | | | | | | | | | | | |
| -- Thallium Carbonate | 6533-73-9 | 469 | | | | | | | | | | | |
| -- Thallium Chloride | 7791-12-0 | 240 | 2.90E+03 | E | 0.00E+00 | E | | | | | | | |
| -- Thallium Nitrate | 10102-45-1 | 266 | | | | | | | | | | | |
| -- Thallic Oxide | 1314-32-5 | 457 | | | | | | | | | | | |
| -- Thallium Sulfate | 7446-18-6 | 505 | 2.00E+02 | E | 0.00E+00 | E | | | | | | | |
| Thioacetamide | 62-55-5 | 75 | | | | | NA | | | -0.46 | J | | |
| Thiourea | 62-56-6 | 76 | 1.72E+06 | B | | | NA | 1.6 | & | -2.05 | B | | |
| o-Tolidine | 119-93-7 | 212 | 7.35E+01 | B | | | NA | 410 | & | 2.88 | B | | |
| Toluene | 108-88-3 | 92 | 5.35E+02 | A | 2.81E+01 | A | 6.37E-03 | 300 | C | 2.73 | A | 10.7 | H |
| o-Toluidine Hydrochloride | 636-21-5 | 144 | 1.50E+04 | J | 1.00E-01 | J | 9.39E-07 | 22 | & | 1.29 | J | | |
| Toxaphene | 8001-35-2 | 414 | 5.00E-01 | C | 4.00E-01 | C | 4.36E-01 | 964 | C | 3.3 | C | 13100 | H |
| Tribromomethane (Bromoform) | 75-25-2 | 253 | 3.01E+03 | C | 5.00E+00 | C | 5.52E-04 | 116 | C | 2.4 | C | | |
| 1,2,4-Trichlorobenzene | 120-82-1 | 181 | 3.00E+01 | C | 2.90E-01 | C | 2.31E-03 | 9200 | C | 4.3 | C | 2800 | G |
| 1,1,1-Trichloroethane | 71-55-6 | 133 | 1.50E+03 | A | 1.23E+02 | A | 1.44E-02 | 152 | C | 2.5 | C | 5.6 | H |
| 1,1,2-Trichloroethane | 79-00-5 | 133 | 4.50E+03 | A | 3.00E+01 | A | 1.17E-03 | 56 | C | 2.47 | A | 5 | H |
| Trichloroethylene | 79-01-6 | 131 | 1.10E+03 | A | 5.79E+01 | A | 9.10E-03 | 126 | C | 2.38 | A | 10.6 | H |
| Trichlorfon | 52-68-6 | 257 | 1.54E+05 | E | 7.80E-06 | E | 1.71E-11 | 6.1 | & | 2.29 | A | | |
| Trichloromonofluoromethane | 75-69-4 | 137 | 1.10E+03 | C | 6.67E+02 | C | | 159 | C | 2.53 | D | | |
| 2,4,5-Trichlorophenol | 95-95-4 | 197 | 1.19E+03 | A | 1.00E+00 | A | 2.18E-04 | 89 | & | 3.72 | A | 110 | H |
| 2,4,6-Trichlorophenol | 88-06-2 | 197 | 8.00E+02 | A | 1.20E-02 | A | 3.90E-06 | 2000 | C | 3.87 | A | 150 | H |
| 2,4,5-Trichlorophenoxyacetic Acid | 93-76-5 | 255 | | | | | | | | | | | |
| 1,2,3-Trichloropropane | 96-18-4 | 147 | | | | | | | | | | | |
| 1,1,2-Trichloro-1,2,2,-trifluoroethane | 76-13-1 | 187 | 1.00E+01 | F | 2.70E+02 | F | | | | 2.00 | F | | |
| Tris(2,3-dibromopropyl)phosphate | 126-72-7 | 698 | 1.20E+02 | B | | | NA | 310 | & | 4.12 | B | 2.7 | G |
| Trinitrotoluene (TNT) | 118-96-7 | 227 | | | | | | | | | | | |
| Trypan Blue | 72-57-1 | 961 | | | | | NA | | | | | | |
| Uracil Mustard | 66-75-1 | 252 | 6.41E+02 | B | | | NA | 120 | & | -1.09 | B | | |
| Uranium and Compounds | 7440-61-1 | 238 | | | | | NA | | | | | | |
| Urethane | 51-79-6 | 89 | | | | | NA | | | | | | |
| Vanadium and Compounds | 7440-62-2 | 51 | | | | | NA | | | | | | |
| Vinyl Chloride | 75-01-4 | 63 | 2.67E+03 | A | 2.66E+03 | A | 8.19E-02 | 57 | & | 1.38 | A | 1.17 | H |
| Warfarin | 81-81-2 | 308 | | | | | | | | | | | |
| o-Xylene | 95-47-6 | 106 | 1.75E+02 | F | 1.00E+01 | E | | | | 2.95 | F | | |
| m-Xylene | 108-38-3 | 106 | 1.30E+02 | F | 1.00E+01 | F | | | | 3.26 | F | | |
| p-Xylene | 106-42-3 | 106 | 1.98E+02 | F | 1.00E+01 | F | | | | 3.15 | F | | |
| Xylene (mixed) | 1330-20-7 | 106 | 1.98E+02 | F | 1.00E+01 | F | 7.04E-03 | 240 | & | 3.26 | F | | |
| Zinc and Compounds | 7440-66-6 | 65 | | | 0.00E+00 | D | NA | | | | | 47 | H |
| -- Zinc Phosphide | 1314-84-7 | 258 | | | | | | | | | | | |
| Zineb | 12122-67-7 | 276 | | | | | | | | | | | |

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* Letters denote the source of the data, as listed in Section 3.1.
\# Solubility of 1,000,000 mg/l assigned because of reported "infinite solubility" in the literature.
& Koc estimated by the following equation: log Koc = (-0.55*logS) + 3.64 (Note: S in mg/l).

Date Prepared: October 1, 1986

EXHIBIT A-2

HALF-LIVES IN VARIOUS MEDIA

| Chemical Name | CAS # | Half-Life Range (Days) Soil Low | Soil High | Soil S* | Air Low | Air High | Air S* | Surface Water Low | Surface Water High | Surface Water S* | Ground Water Low | Ground Water High | Ground Water S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Acenaphthene | 83-32-9 | | | | | | | | | | | | |
| Acenaphthylene | 208-96-8 | | | | 5.50 | - | M | 0.125 | - | M | | | |
| Acetone | 67-64-1 | | | | | | | | | | | | |
| Acetonitrile | 75-05-8 | | | | 390.00 | - | M | 7.00 | - | M | | | |
| 2-Acetylaminofluorene | 53-96-3 | | | | | | | | | | | | |
| Acrylic Acid | 79-10-7 | | | | | | | | | | | | |
| Acrylonitrile | 107-13-1 | | | | 3.90 | - | M | 2.10 | 7.00 | M | | | |
| Aflatoxin B1 | 1162-65-8 | | | | | | | | | | | | |
| Aldicarb | 116-06-3 | | | | | | | | | | | | |
| Aldrin | 309-00-2 | | | | | | | | | | | | |
| Allyl Alcohol | 107-18-6 | | | | | | | 5.00 | - | M | | | |
| Aluminum Phosphide | 20859-73-8 | | | | | | | | | | | | |
| 4-Aminobiphenyl | 92-67-1 | | | | | | | | | | | | |
| Amitrole | 61-82-5 | | | | | | | | | | | | |
| Ammonia | 7664-41-7 | | | | | | | | | | | | |
| Anthracene | 120-12-7 | | | | | | | | | | | | |
| Antimony and Compounds | 7440-36-0 | | | | 4.80 | - | M | PERS** | - | M | | | |
| Arsenic and Compounds | 7440-38-2 | | | | 5.00 | - | M | PERS | - | M | | | |
| Asbestos | 1332-21-4 | | | | 4.80 | - | M | PERS | - | M | | | |
| Auramine | 2465-27-2 | | | | | | | | | | | | |
| Azaserine | 115-02-6 | | | | | | | | | | | | |
| Aziridine | 151-56-4 | | | | | | | | | | | | |
| Barium and Compounds | 7440-39-3 | | | | 4.80 | - | M | PERS | - | M | | | |
| Benefin | 1861-40-1 | | | | | | | | | | | | |
| Benzene | 71-43-2 | | | | 6.00 | - | A | 1.00 | 6.00 | A | | | |
| Benzidine | 92-87-5 | | | | | | | | | | | | |
| Benz(a)anthracene | 56-55-3 | | | | 5.50 | - | M | 0.10 | 5.00 | M | | | |
| Benz(c)acridine | 225-51-4 | | | | | | | | | | | | |
| Benzo(a)pyrene | 50-32-8 | 420.00 | 480.00 | A | 1.00 | 6.00 | A | 0.40 | - | A | | | |
| Benzo(b)fluoranthene | 205-99-2 | | | | 5.50 | - | M | 1.00 | 2.00 | M | | | |
| Benzo(ghi)perylene | 191-24-2 | | | | | | | | | | | | |
| Benzo(k)fluoranthene | 207-08-9 | | | | | | | | | | | | |
| Benzotrichloride | 98-07-7 | | | | | | | | | | | | |
| Benzyl Chloride | 100-44-7 | | | | | | | 1.50 | - | M | | | |
| Beryllium and Compounds | 7440-41-7 | | | | | | | | | | | | |
| 1,1-Biphenyl | 92-52-4 | | | | | | | | | | | | |
| Bis(2-chloroethyl)ether | 111-44-4 | | | | | | | | | | | | |
| Bis(2-chloroisopropyl)ether | 108-60-1 | | | | | | | | | | | | |
| Bis(chloromethyl)ether | 542-88-1 | | | | 0.14 | 2.00 | M | 0.0007 | - | M | | | |
| Bis(2-ethylhexyl)phthalate (DEHP) | 117-81-7 | | | | | | | | | | | | |
| Bromomethane | 74-83-9 | | | | | | | | | | | | |
| Bromoxynil Octanoate | 1689-99-2 | | | | | | | | | | | | |
| 1,3-Butadiene | 106-99-0 | | | | | | | | | | | | |
| n-Butanol | 71-36-3 | | | | | | | | | | | | |
| Butylphthalyl Butylglycolate | 85-70-1 | | | | | | | | | | | | |
| Cacodylic Acid | 75-60-5 | | | | | | | | | | | | |
| Cadmium and Compounds | 7740-43-9 | | | | 4.80 | - | M | PERS | - | M | | | |
| Captan | 133-06-2 | | | | | | | | | | | | |
| Carbaryl | 63-25-2 | | | | | | | | | | | | |
| Carbon Disulfide | 75-15-0 | | | | | | | | | | | | |
| Carbon Tetrachloride | 56-23-5 | | | | 8030.00 | - | A | 0.30 | 300.00 | A | | | |
| Chlordane | 57-74-9 | | | | 40.00 | - | M | 420.00 | 500.00 | A | | | |
| Chlorobenzene | 108-90-7 | | | | 3.50 | - | A | 0.30 | - | A | | | |
| Chlorobenzilate | 510-15-6 | | | | | | | | | | | | |

Date Prepared: October 1, 1986

EXHIBIT A-2
(Continued)

HALF-LIVES IN VARIOUS MEDIA

| Chemical Name | CAS # | Half-Life Range (Days) Soil Low | Soil High | Soil S* | Air Low | Air High | Air S* | Surface Water Low | Surface Water High | Surface Water S* | Ground Water Low | Ground Water High | Ground Water S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Chlorodibromomethane | 124-48-1 | | | | | | | | | | | | |
| Chloroform | 67-66-3 | | | | 80.00 | - | A | 0.30 | 30.00 | A | | | |
| Chloromethyl Methyl Ether | 107-30-2 | | | | | | | | | | | | |
| 4-Chloro-o-toluidine Hydrochloride | 3165-93-3 | | | | | | | | | | | | |
| Chromium III and Compounds | 7440-47-3 | | | | | | | | | | | | |
| Chromium VI and Compounds | 7440-47-3 | | | | 4.80 | - | M | 3.00 | - | M | | | |
| Chrysene | 218-01-9 | | | | 5.50 | - | M | 0.20 | - | M | | | |
| Copper and Compounds | 7440-50-8 | | | | | | | | | | | | |
| Creosote | 8001-58-9 | | | | | | | | | | | | |
| Cresol | 1319-77-3 | | | | | | | | | | | | |
| Crotonaldehyde | 123-73-9 | | | | | | | | | | | | |
| Cyanides | 57-12-5 | | | | 73000.00 | - | M | 0.33 | 0.80 | M | | | |
| -- Barium Cyanide | 542-62-1 | | | | | | | | | | | | |
| -- Calcium Cyanide | 502-01-8 | | | | | | | | | | | | |
| -- Copper Cyanide | 544-92-3 | | | | | | | | | | | | |
| -- Cyanogen | 460-19-5 | | | | | | | | | | | | |
| -- Cyanogen Chloride | 506-77-4 | | | | | | | | | | | | |
| -- Hydrogen Cyanide | 74-90-8 | | | | | | | | | | | | |
| -- Nickel Cyanide | 557-19-7 | | | | | | | | | | | | |
| -- Potassium Cyanide | 151-50-8 | | | | | | | | | | | | |
| -- Potassium Silver Cyanide | 506-61-6 | | | | | | | | | | | | |
| -- Silver Cyanide | 506-64-9 | | | | | | | | | | | | |
| -- Sodium Cyanide | 143-33-9 | | | | | | | | | | | | |
| -- Zinc Cyanide | 557-21-1 | | | | | | | | | | | | |
| Cyclophosphamide | 50-18-0 | | | | | | | | | | | | |
| Dalapon | 75-99-0 | | | | | | | | | | | | |
| DDD | 72-54-8 | | | | | | | | | | | | |
| DDE | 72-55-9 | | | | | | | | | | | | |
| DDT | 50-29-3 | 1000.00 | 5500.00 | A | | | | 56.00 | 110.00 | A | | | |
| Decabromodiphenyl Ether | 1163-19-5 | | | | | | | | | | | | |
| Diallate | 2303-16-4 | | | | | | | | | | | | |
| 2,4-Diaminotoluene | 95-80-7 | | | | | | | | | | | | |
| 1,2,7,8-Dibenzopyrene | 189-55-9 | | | | | | | | | | | | |
| Dibenz(a,h)anthracene | 53-70-3 | | | | 5.50 | - | M | 0.0208 | 2.08 | M | | | |
| 1,2-Dibromo-3-chloropropane | 96-12-8 | | | | | | | | | | | | |
| Dibutylnitrosamine | 924-16-3 | | | | | | | | | | | | |
| Dibutyl Phthalate | 84-74-2 | | | | | | | | | | | | |
| 1,2-Dichlorobenzene | 95-50-1 | | | | 26.00 | - | M | 1.50 | 8.50 | M | | | |
| 1,3-Dichlorobenzene | 541-73-1 | | | | | | | | | | | | |
| 1,4-Dichlorobenzene | 106-46-7 | | | | 23.00 | - | M | 1.50 | 8.50 | M | | | |
| 3,3'-Dichlorobenzidine | 91-94-1 | | | | | | | | | | | | |
| Dichlorodifluoromethane | 75-71-8 | | | | | | | | | | | | |
| 1,1-Dichloroethane | 75-34-3 | | | | 45.00 | - | A | 1.00 | 5.00 | A | | | |
| 1,2-Dichloroethane (EDC) | 107-06-2 | | | | 36.00 | 127.00 | A | 0.17 | - | A | | | |
| 1,1-Dichloroethylene | 75-35-4 | | | | 2.00 | - | A | 1.00 | 6.00 | A | | | |
| 1,2-Dichloroethylene (trans) | 540-59-0 | | | | 2.10 | - | A | 1.00 | 6.00 | A | | | |
| 1,2-Dichloroethylene (cis) | 540-59-0 | | | | 1.30 | - | A | 1.00 | 6.00 | A | | | |
| Dichloromethane | 75-09-2 | | | | 53.20 | - | M | 1.20 | 5.80 | M | | | |
| 2,4-Dichlorophenol | 120-83-2 | | | | 2.30 | - | M | 6.00 | - | M | | | |
| 2,4-Dichlorophenoxyacetic Acid (2,4-D) | 94-75-7 | | | | | | | | | | | | |
| 4-(2,4-Dichlorophenoxy)butyric Acid (2,4-DB) | 94-82-6 | | | | | | | | | | | | |

Date Prepared: October 1, 1986

EXHIBIT A-2
(Continued)

HALF-LIVES IN VARIOUS MEDIA

| Chemical Name | CAS # | Half-Life Range (Days) Soil Low | Soil High | Soil S* | Air Low | Air High | Air S* | Surface Water Low | Surface Water High | Surface Water S* | Ground Water Low | Ground Water High | Ground Water S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Dichlorophenylarsine | 696-28-6 | | | | | | | | | | | | |
| 1,2-Dichloropropane | 78-87-5 | | | | 80.00 | - | M | 1.40 | 7.70 | M | | | |
| 1,3-Dichloropropene | 542-75-6 | | | | | | | 1.00 | - | M | | | |
| Dieldrin | 60-57-1 | | | | | | | | | | | | |
| Diepoxybutane | 1464-53-5 | | | | | | | | | | | | |
| Diethanolnitrosamine | 1116-54-7 | | | | | | | | | | | | |
| Diethyl Arsine | 692-42-2 | | | | | | | | | | | | |
| 1,2-Diethylhydrazine | 1615-80-1 | | | | | | | | | | | | |
| Diethylnitrosamine | 55-18-5 | | | | | | | | | | | | |
| Diethyl Phthalate | 84-66-2 | | | | | | | | | | | | |
| Diethylstilbestrol (DES) | 56-53-1 | | | | | | | | | | | | |
| Dihydrosafrole | 94-58-6 | | | | | | | | | | | | |
| Dimethoate | 60-51-5 | | | | | | | | | | | | |
| 3,3'-Dimethoxybenzidine | 119-90-4 | | | | | | | | | | | | |
| Dimethylamine | 124-40-3 | | | | | | | | | | | | |
| Dimethyl Sulfate | 77-78-1 | | | | | | | | | | | | |
| Dimethyl Terephthalate | 120-61-6 | | | | | | | | | | | | |
| Dimethylaminoazobenzene | 60-11-7 | | | | | | | | | | | | |
| 7,12-Dimethylbenz(a)anthracene | 57-97-6 | | | | | | | | | | | | |
| 3,3'-Dimethylbenzidine | 119-93-7 | | | | | | | | | | | | |
| Dimethylcarbamoyl Chloride | 79-44-7 | | | | | | | | | | | | |
| 1,1-Dimethylhydrazine | 57-14-4 | | | | | | | | | | | | |
| 1,2-Dimethylhydrazine | 540-73-8 | | | | | | | | | | | | |
| Dimethylnitrosamine | 62-75-9 | | | | | | | | | | | | |
| 1,3-Dinitrobenzene | 99-65-0 | | | | | | | 96.00 | - | M | | | |
| 4,6-Dinitro-o-cresol | 534-52-1 | | | | | | | | | | | | |
| 2,4-Dinitrophenol | 51-28-5 | | | | | | | | | | | | |
| 2,3-Dinitrotoluene | 602-01-7 | | | | | | | | | | | | |
| 2,4-Dinitrotoluene | 121-14-2 | | | | 133.00 | - | M | 0.40 | 10.00 | M | | | |
| 2,5-Dinitrotoluene | 619-15-8 | | | | | | | | | | | | |
| 2,6-Dinitrotoluene | 606-20-2 | | | | | | | | | | | | |
| 3,4-Dinitrotoluene | 610-39-9 | | | | | | | | | | | | |
| Dinoseb | 88-85-7 | | | | | | | | | | | | |
| 1,4-Dioxane | 123-91-1 | | | | | | | | | | | | |
| N,N-Diphenylamine | 122-39-4 | | | | | | | | | | | | |
| 1,2-Diphenylhydrazine | 122-66-7 | | | | | | | | | | | | |
| Dipropylnitrosamine | 621-64-7 | | | | | | | | | | | | |
| Disulfoton | 298-04-4 | | | | | | | | | | | | |
| Endosulfan | 115-29-7 | | | | | | | | | | | | |
| Epichlorohydrin | 106-89-8 | | | | 2.70 | - | M | 3.50 | 10.80 | M | | | |
| Ethanol | 64-17-5 | | | | | | | | | | | | |
| Ethyl Acetate | 141-78-6 | | | | | | | | | | | | |
| Ethyl Methanesulfonate | 62-50-0 | | | | | | | | | | | | |
| Ethylbenzene | 100-41-4 | | | | 1.46 | - | A | 1.50 | 7.50 | A | | | |
| Ethyl-4,4'-dichlorobenzilate | 510-15-6 | | | | | | | | | | | | |
| Ethylene Dibromide (EDB) | 106-93-4 | | | | | | | | | | | | |
| Ethylene Oxide | 75-21-8 | | | | | | | | | | | | |
| Ethylenethiourea | 96-45-7 | | | | | | | | | | | | |
| 1-Ethyl-nitrosourea | 759-73-9 | | | | | | | | | | | | |
| Ethylphthalyl Ethyl Glycolate | 84-72-0 | | | | | | | | | | | | |
| Ferric Dextran | 9004-66-4 | | | | | | | | | | | | |
| Fluoranthene | 206-44-0 | | | | 5.50 | - | M | 1.00 | 2.00 | M | | | |
| Fluorene | 86-73-7 | | | | | | | | | | | | |

Date Prepared: October 1, 1986

EXHIBIT A-2
(Continued)

HALF-LIVES IN VARIOUS MEDIA

| Chemical Name | CAS # | Half-Life Range (Days) Soil Low | Soil High | Soil S* | Air Low | Air High | Air S* | Surface Water Low | Surface Water High | Surface Water S* | Ground Water Low | Ground Water High | Ground Water S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Fluorides | 7782-41-4 | | | | | | | | | | | | |
| Fluridone | 59756-60-4 | | | | | | | | | | | | |
| Formaldehyde | 50-00-0 | | | | 0.80 | - | M | 0.90 | 3.50 | M | | | |
| Formic Acid | 64-18-6 | | | | | | | | | | | | |
| Furan | 110-00-9 | | | | | | | | | | | | |
| Glycidaldehyde | 765-34-4 | | | | | | | | | | | | |
| Glycol Ethers | NA | | | | | | | | | | | | |
| -- Diethylene Glycol, Monoethyl Ether | 111-90-0 | | | | | | | | | | | | |
| -- 2-Ethoxyethanol | 110-80-5 | | | | | | | | | | | | |
| -- Ethylene Glycol, Monobutyl Ether | 111-76-2 | | | | | | | | | | | | |
| -- 2-Methoxyethanol | 109-86-4 | | | | | | | | | | | | |
| -- Propylene Glycol, Monoethyl Ether | 52125-53-8 | | | | | | | | | | | | |
| -- Propylene Glycol, Monomethyl Ether | 107-98-2 | | | | | | | | | | | | |
| Heptachlor | 76-44-8 | | | | 40.00 | - | M | 0.96 | - | M | | | |
| Heptachlor Epoxide | 1024-57-3 | | | | | | | | | | | | |
| Hexachlorobenzene | 118-74-1 | 1100.00 | 2200.00 | A | 80.00 | - | M | 0.30 | 300.00 | A | | | |
| Hexachlorobutadiene | 87-68-3 | | | | 0.20 | - | M | 29.00 | 2300.00 | A | | | |
| Hexachlorocyclopentadiene | 77-47-4 | | | | 0.14 | - | M | 0.007 | - | M | | | |
| alpha-Hexachlorocyclohexane (HCCH) | 319-84-6 | | | | | | | | | | | | |
| beta-HCCH | 319-85-7 | | | | | | | | | | | | |
| gamma-HCCH (Lindane) | 58-89-9 | | | | | | | | | | | | |
| delta-HCCH | 319-86-8 | | | | | | | | | | | | |
| Hexachloroethane | 67-72-1 | | | | 7900 | - | M | 1.10 | 9.50 | M | | | |
| Hexachlorophene | 70-30-4 | | | | | | | | | | | | |
| Hydrazine | 302-01-1 | | | | | | | | | | | | |
| Hydrogen Sulfide | 7783-06-4 | | | | | | | | | | | | |
| Indeno(1,2,3-cd)pyrene | 193-39-5 | | | | 5.50 | - | M | 0.0208 | 2.08 | M | | | |
| Iodomethane | 77-88-4 | | | | | | | | | | | | |
| Iron and Compounds | 15438-31-0 | | | | | | | | | | | | |
| Isobutanol | 78-83-1 | | | | | | | | | | | | |
| Isoprene | 78-79-5 | | | | | | | | | | | | |
| Isosafrole | 120-58-1 | | | | | | | | | | | | |
| Isophorone | 78-59-1 | | | | | | | | | | | | |
| Isopropalin | 33820-53-0 | | | | | | | | | | | | |
| Kepone | 143-50-0 | | | | | | | | | | | | |
| Lasiocarpine | 303-34-4 | | | | | | | | | | | | |
| Lead and Compounds (Inorganic) | 7439-92-1 | | | | 4.80 | - | M | PERS | - | M | | | |
| Linuron | 330-55-2 | | | | | | | | | | | | |
| Malathion | 121-75-7 | | | | | | | | | | | | |
| Manganese and Compounds | 7439-96-5 | | | | | | | | | | | | |
| Melphalan | 148-82-3 | | | | | | | | | | | | |
| Mercury and Compounds (Alkyl) | 7439-97-6 | | | | | | | | | | | | |
| Mercury and Compounds (Inorganic) | 7439-97-6 | | | | 4.80 | - | M | PERS | - | M | | | |
| Mercury Fulminate | 628-86-4 | | | | | | | | | | | | |
| Methanol | 67-56-1 | | | | | | | | | | | | |
| Methyl Chloride | 74-87-3 | | | | | | | 1.00 | - | M | | | |
| Methyl Ethyl Ketone | 78-93-3 | | | | 0.58 | - | A | 10.00 | - | A | | | |
| Methyl Ethyl Ketone Peroxide | 1338-23-4 | | | | | | | | | | | | |
| Methyl Isobutyl Ketone | 108-10-1 | | | | | | | | | | | | |

Date Prepared: October 1, 1986

EXHIBIT A-2
(Continued)

HALF-LIVES IN VARIOUS MEDIA

| Chemical Name | CAS # | Half-Life Range (Days) Soil Low | Soil High | Soil S* | Air Low | Air High | Air S* | Surface Water Low | Surface Water High | Surface Water S* | Ground Water Low | Ground Water High | Ground Water S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Methyl Methacrylate | 80-62-6 | | | | | | | 15.00 | - | M | | | |
| Methyl Parathion | 298-00-0 | | | | | | | | | | | | |
| 2-Methyl-4-chlorophenoxyacetic Acid | 94-74-6 | | | | | | | | | | | | |
| 2(2-Methyl)-4-Chlorophenoxy-propionic Acid | 93-65-2 | | | | | | | | | | | | |
| 3-Methylcholanthrene | 56-49-3 | | | | | | | | | | | | |
| 4,4'-Methylene-bis-2-chloroaniline | 101-14-4 | | | | | | | | | | | | |
| Methylnitrosourea | 684-93-5 | | | | | | | | | | | | |
| Methylthiouracil | 56-04-2 | | | | | | | | | | | | |
| Methylvinylnitrosamine | 4549-40-0 | | | | | | | | | | | | |
| N-Methyl-N'-nitro-N-nitrosoguanadin | 70-25-7 | | | | | | | | | | | | |
| Mitomycin C | 50-07-7 | | | | | | | | | | | | |
| Mustard Gas | 505-60-2 | | | | | | | | | | | | |
| 1-Napthylamine | 134-32-7 | | | | | | | | | | | | |
| 2-Napthylamine | 91-59-8 | | | | | | | | | | | | |
| Nickel and Compounds | 7440-02-0 | | | | | | | | | | | | |
| Nitric Oxide | 10102-43-9 | | | | | | | | | | | | |
| Nitrobenzene | 98-95-3 | | | | | | | 12.50 | - | M | | | |
| Nitrogen Dioxide | 10102-44-0 | | | | | | | | | | | | |
| Nitrosomethylurethane | 615-53-2 | | | | | | | | | | | | |
| N-Nitrosopiperidine | 100-75-4 | | | | | | | | | | | | |
| N-Nitrosopyrrolidine | 930-55-2 | | | | | | | | | | | | |
| 5-Nitro-o-toluidine | 99-55-8 | | | | | | | | | | | | |
| Osmium Tetroxide | 20816-12-0 | | | | | | | | | | | | |
| Pentachlorobenzene | 608-93-5 | | | | | | | | | | | | |
| Pentachloronitrobenzene | 82-68-8 | | | | | | | | | | | | |
| Pentachlorophenol | 87-86-5 | | | | 21.00 | - | M | 5.00 | - | M | | | |
| Phenacetin | 62-44-2 | | | | | | | | | | | | |
| Phenanthrene | 85-01-8 | | | | | | | 0.38 | 2.00 | A | | | |
| Phenobarbital | 50-06-6 | | | | | | | | | | | | |
| Phenol | 108-95-2 | | | | 0.62 | 9.00 | A | 0.62 | 9.00 | A | | | |
| Phenylalanine Mustard | 148-82-3 | | | | | | | | | | | | |
| m-Phenylenediamine | 108-45-2 | | | | | | | | | | | | |
| Phenyl Mercuric Acetate | 62-38-4 | | | | | | | | | | | | |
| Phosphine | 7803-51-2 | | | | | | | | | | | | |
| Polychlorinated Biphenyls (PCBs) | 1336-36-3 | | | | 58.00 | - | M | 2.00 | 12.90 | M | | | |
| Propane Sultone | 1120-71-4 | | | | | | | | | | | | |
| Propylenimine | 75-55-8 | | | | | | | | | | | | |
| Pyrene | 129-00-0 | | | | 0.08 | 2.00 | A | | | | | | |
| Pyridine | 110-86-1 | | | | | | | 2.00 | - | M | | | |
| Saccharin | 81-07-2 | | | | | | | | | | | | |
| Safrole | 94-59-7 | | | | | | | | | | | | |
| Selenium and Compounds | 7782-49-2 | | | | | | | | | | | | |
| -- Selenious Acid | 7783-00-8 | | | | | | | | | | | | |
| -- Selenourea | 630-10-4 | | | | | | | | | | | | |
| -- Thallium Selenite | 12039-52-0 | | | | | | | | | | | | |
| Silver and Compounds | 7440-22-4 | | | | | | | | | | | | |
| Sodium Diethyldithiocarbamate | 148-18-5 | | | | | | | | | | | | |
| Streptozocin | 18883-66-4 | | | | | | | | | | | | |
| Strychnine | 57-24-9 | | | | | | | | | | | | |
| Styrene | 100-42-5 | | | | | | | | | | | | |
| 1,2,4,5-Tetrachlorobenzene | 95-94-3 | | | | | | | | | | | | |
| 2,3,7,8-TCDD (Dioxin) | 1746-01-6 | 3650.00 | 4380.00 | A | | | | 365.00 | 730.00 | A | | | |

Date Prepared: October 1, 1986

EXHIBIT A-2
(Continued)

HALF-LIVES IN VARIOUS MEDIA

| Chemical Name | CAS # | Half-Life Range (Days) Soil Low | Soil High | Soil S* | Air Low | Air High | Air S* | Surface Water Low | Surface Water High | Surface Water S* | Ground Water Low | Ground Water High | Ground Water S* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1,1,1,2-Tetrachloroethane | 630-20-6 | | | | | | | 1.40 | - | M | | | |
| 1,1,2,2-Tetrachloroethane | 79-34-5 | | | | 584.00 | - | A | 0.04 | - | A | | | |
| Tetrachloroethylene | 127-18-4 | | | | 47.00 | - | A | 1.00 | 30.00 | A | | | |
| 2,3,4,6-Tetrachlorophenol | 58-90-2 | | | | | | | | | | | | |
| 2,3,5,6-Tetrachloroterephthalate Acid (DCPA) | 1861-32-1 | | | | | | | | | | | | |
| Tetraethyl Lead | 78-00-2 | | | | | | | | | | | | |
| Thallium and Compounds | 7440-28-0 | | | | | | | | | | | | |
| -- Thallium Acetate | 563-68-8 | | | | | | | | | | | | |
| -- Thallium Carbonate | 6533-73-9 | | | | | | | | | | | | |
| -- Thallium Chloride | 7791-12-0 | | | | | | | | | | | | |
| -- Thallium Nitrate | 10102-45-1 | | | | | | | | | | | | |
| -- Thallic Oxide | 1314-32-5 | | | | | | | | | | | | |
| -- Thallium Sulfate | 7446-18-6 | | | | | | | | | | | | |
| Thioacetamide | 62-55-5 | | | | | | | | | | | | |
| Thiourea | 62-56-6 | | | | | | | | | | | | |
| o-Tolidine | 119-93-7 | | | | | | | | | | | | |
| Toluene | 108-88-3 | | | | 1.30 | - | A | 0.17 | - | A | | | |
| o-Toluidine Hydrochloride | 636-21-5 | | | | | | | | | | | | |
| Toxaphene | 8001-35-2 | | | | 40.00 | - | M | 2.00 | 14.20 | M | | | |
| Tribromomethane (Bromoform) | 75-25-2 | | | | | | | | | | | | |
| 1,2,4-Trichlorobenzene | 120-82-1 | | | | | | | 1.20 | - | M | | | |
| 1,1,1-Trichloroethane | 71-55-6 | | | | 803.00 | 1752.00 | A | 0.14 | 7.00 | A | | | |
| 1,1,2-Trichloroethane | 79-00-5 | | | | 24.00 | - | A | 1.90 | - | A | | | |
| Trichloroethylene | 79-01-6 | | | | 3.70 | - | A | 1.00 | 90.00 | A | | | |
| Trichlorfon | 52-68-6 | | | | | | | | | | | | |
| Trichloromonofluoromethane | 75-69-4 | | | | | | | | | | | | |
| 2,4,5-Trichlorophenol | 95-95-4 | 72.00 | - | A | | | | | | | | | |
| 2,4,6-Trichlorophenol | 88-06-2 | 5.00 | - | A | 1.00 | - | A | 1.00 | 19.00 | A | | | |
| 2,4,5-Trichlorophenoxyacetic Acid | 93-76-5 | | | | | | | | | | | | |
| 1,2,3-Trichloropropane | 96-18-4 | | | | | | | | | | | | |
| 1,1,2-Trichloro-1,2,2,-trifluoroethane | 76-13-1 | | | | | | | | | | | | |
| Tris(2,3-dibromopropyl)phosphate | 126-72-7 | | | | | | | | | | | | |
| Trinitrotoluene (TNT) | 118-96-7 | | | | | | | | | | | | |
| Trypan Blue | 72-57-1 | | | | | | | | | | | | |
| Uracil Mustard | 66-75-1 | | | | | | | | | | | | |
| Uranium and Compounds | 7440-61-1 | | | | | | | | | | | | |
| Urethane | 51-79-6 | | | | | | | | | | | | |
| Vanadium and Compounds | 7440-62-2 | | | | | | | | | | | | |
| Vinyl Chloride | 75-01-4 | | | | 1.20 | - | A | 1.00 | 5.00 | A | | | |
| Warfarin | 81-81-2 | | | | | | | | | | | | |
| o-Xylene | 95-47-6 | | | | | | | | | | | | |
| m-Xylene | 108-38-3 | | | | | | | | | | | | |
| p-Xylene | 106-42-3 | | | | | | | | | | | | |
| Xylene (mixed) | 1330-20-7 | | | | 0.50 | - | M | 1.50 | 9.00 | M | | | |
| Zinc and Compounds | 7440-66-6 | | | | 4.80 | 20.00 | M | PERS | - | M | | | |
| -- Zinc Phosphide | 1314-84-7 | | | | | | | | | | | | |
| Zineb | 12122-67-7 | | | | | | | | | | | | |

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* Letters denote the source of the data, as listed in Section C.1.
** PERS indicates the chemical is persistent for that medium.

Date Prepared October 1, 1986

## EXHIBIT A-3

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY [1]

| Chemical Name | Oral Route: 10% Effective Dose (ED10) mg/kg/day | Oral Route: Toxicity Constant Water (wTc) 1/mg | Oral Route: Toxicity Constant Soil (sTc) kg/mg | Inhalation Route: 10% Effective Dose (ED10) mg/kg/day | Inhalation Route: Air Toxicity Constant (aTc) (m3/mg) |
|---|---|---|---|---|---|
| 2-Acetylaminofluorene | 2.60E-02 | 1.10E+00 | 5.50E-05 | 2.60E-02 | 1.10E+01 |
| Acrylonitrile | 4.39E-01 | 6.51E-02 | 3.26E-06 | 4.39E-01 | 6.51E-01 |
| Aflatoxin B1 | NA | NA | NA | NA | NA |
| Aldrin | 1.52E-02 | 1.88E+00 | 9.40E-05 | 1.52E-02 | 1.88E+01 |
| Amitrole | 1.89E-01 | 1.51E-01 | 7.56E-06 | 1.89E-01 | 1.51E+00 |
| Arsenic and Compounds | 7.03E-03 | 4.07E+00 | 2.03E-04 | 7.03E-03 | 4.07E+01 |
| Asbestos | NA | NA | NA | NA | NA |
| Auramine | 1.08E+00 | 2.66E-02 | 1.33E-06 | 1.08E+00 | 2.66E-01 |
| Azaserine | NA | NA | NA | NA | NA |
| Aziridine | 3.60E-03 | 7.93E+00 | 3.97E-04 | 3.60E-03 | 7.93E+01 |
| Benzene | 3.70E+00 | 7.71E-03 | 3.86E-07 | 3.70E+00 | 7.71E-02 |
| Benzidine | 4.50E-04 | 6.34E+01 | 3.17E-03 | 4.50E-04 | 6.34E+02 |
| Benz(a)anthracene | 4.92E-02 | 5.81E-01 | 2.91E-05 | 4.92E-02 | 5.81E+00 |
| Benz(c)acridine | 6.67E-05 | 4.29E+02 | 2.14E-02 | 6.67E-05 | 4.29E+03 |
| Benzo(a)pyrene | 6.28E-03 | 4.55E+00 | 2.28E-04 | 6.28E-03 | 4.55E+01 |
| Benzo(b)fluoranthene | NA | NA | NA | NA | NA |
| Benzo(k)fluoranthene | NA | NA | NA | NA | NA |
| Benzotrichloride | 8.91E-03 | 3.21E+00 | 1.60E-04 | 8.91E-03 | 3.21E+01 |
| Benzyl Chloride | NA | NA | NA | NA | NA |
| Beryllium and Compounds | NA | NA | NA | 1.25E-02 | 2.28E+01 |
| Bis(2-chloroethyl)ether | 8.23E-02 | 3.47E-01 | 1.74E-05 | 8.23E-02 | 3.47E+00 |
| Bis(chloromethyl)ether | 7.22E-04 | 3.96E+01 | 1.98E-03 | 7.22E-04 | 3.96E+02 |
| Bis(2-ethylhexyl)phthalate (DEHP) | 5.00E+01 | 5.71E-04 | 2.86E-08 | 5.00E+01 | 5.71E-03 |
| Cacodylic Acid | NA | NA | NA | NA | NA |
| Cadmium and Compounds | NA | NA | NA | 1.73E-02 | 1.65E+01 |
| Carbon Tetrachloride | 1.52E-02 | 1.88E+00 | 9.41E-05 | 1.52E-02 | 1.88E+01 |
| Chlordane | 6.61E-02 | 4.32E-01 | 2.16E-05 | 6.61E-02 | 4.32E+00 |
| Chloroform | 5.08E-01 | 5.63E-02 | 2.81E-06 | 5.08E-01 | 5.63E-01 |
| 4-Chloro-o-toluidine Hydrochloride | 8.13E-01 | 3.51E-02 | 1.76E-06 | 8.13E-01 | 3.51E-01 |
| Chromium VI and Compounds | NA | NA | NA | 2.57E-03 | 1.11E+02 |
| Chrysene | NA | NA | NA | NA | NA |
| Cyclophosphamide | 5.70E-02 | 5.01E-01 | 2.50E-05 | 5.70E-02 | 5.01E+00 |
| DDD | 7.69E-01 | 3.71E-02 | 1.86E-06 | 7.69E-01 | 3.71E-01 |
| DDE | 2.53E-01 | 1.13E-01 | 5.64E-06 | 2.53E-01 | 1.13E+00 |
| DDT | 1.79E-01 | 1.59E-01 | 7.97E-06 | 1.79E-01 | 1.59E+00 |
| Diallate | 4.24E-01 | 6.74E-02 | 3.37E-06 | 4.24E-01 | 6.74E-01 |

Date Prepared: October 1, 1986

## EXHIBIT A-3
## (Continued)

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| | Oral Route | | | Inhalation Route | |
|---|---|---|---|---|---|
| | 10% Effective Dose (ED10) | Toxicity Constant Water (wTc) | Toxicity Constant Soil (sTc) | 10% Effective Dose (ED10) | Air Toxicity Constant (aTc) |
| Chemical Name | mg/kg/day | 1/mg | kg/mg | mg/kg/day | m3/mg |
| Diaminotoluene (mixed) | 3.40E-01 | 8.40E-02 | 4.20E-06 | 3.40E-01 | 8.40E-01 |
| 1,2,7,8-Dibenzopyrene | NA | NA | NA | NA | NA |
| Dibenz(a,h)anthracene | 2.83E-03 | 1.01E+01 | 5.04E-04 | 2.83E-03 | 1.01E+02 |
| 1,2-Dibromo-3-chloropropane | 6.00E-03 | 4.76E+00 | 2.38E-04 | 6.00E-03 | 4.76E+01 |
| Dibutylnitrosamine | 2.29E-02 | 1.25E+00 | 6.24E-05 | 2.29E-02 | 1.25E+01 |
| 3,3'-Dichlorobenzidine | 1.20E-01 | 2.39E-01 | 1.19E-05 | 1.20E-01 | 2.39E+00 |
| 1,2-Dichloroethane (EDC) | 4.88E-01 | 5.86E-02 | 2.93E-06 | 4.88E-01 | 5.86E-01 |
| 1,1-Dichloroethylene | 2.33E-01 | 1.23E-01 | 6.14E-06 | 2.33E-01 | 1.23E+00 |
| Dichloromethane | NA | NA | NA | NA | NA |
| Dieldrin | 7.81E-03 | 3.66E+00 | 1.83E-04 | 7.81E-03 | 3.66E+01 |
| Diepoxybutane | 3.58E-02 | 7.98E-01 | 3.99E-05 | 3.58E-02 | 7.98E+00 |
| Diethanolnitrosamine | NA | NA | NA | NA | NA |
| Diethyl Arsine | NA | NA | NA | NA | NA |
| 1,2-Diethylhydrazine | NA | NA | NA | NA | NA |
| Diethylnitrosamine | 1.03E-03 | 2.77E+01 | 1.38E-03 | 1.03E-03 | 2.77E+02 |
| Diethylstilbestrol (DES) | 2.11E-04 | 1.35E+02 | 6.77E-03 | 2.11E-04 | 1.35E+03 |
| Dihydrosafrole | 9.26E-01 | 3.09E-02 | 1.54E-06 | 9.26E-01 | 3.09E-01 |
| 3,3'-Dimethoxybenzidine | 2.00E+01 | 1.43E-03 | 7.14E-08 | 2.00E+01 | 1.43E-02 |
| Dimethyl Sulfate | NA | NA | NA | NA | NA |
| Dimethylaminoazobenzene | 9.52E-03 | 3.00E+00 | 1.50E-04 | 9.52E-03 | 3.00E+01 |
| 7,12-Dimethylbenz(a)anthracene | 5.23E-06 | 5.46E+03 | 2.73E-01 | 5.23E-06 | 5.46E+04 |
| 3,3'-Dimethylbenzidene | 3.70E-02 | 7.71E-01 | 3.86E-05 | 3.70E-02 | 7.72E+00 |
| Dimethylcarbamoyl Chloride | 1.98E-03 | 1.44E+01 | 7.22E-04 | 1.98E-03 | 1.44E+02 |
| 1,1-Dimethylhydrazine | 7.44E-02 | 3.84E-01 | 1.92E-05 | 7.44E-02 | 3.84E+00 |
| 1,2-Dimethylhydrazine | 1.87E-04 | 1.53E+02 | 7.65E-03 | 1.87E-04 | 1.53E+03 |
| Dimethylnitrosamine | 3.91E-02 | 7.30E-01 | 3.65E-05 | 3.91E-02 | 7.30E+00 |
| Dinitrotoluene (mixed) | 2.62E-01 | 1.09E-01 | 5.46E-06 | 2.62E-01 | 1.09E+00 |
| 2,4-Dinitrotoluene | 2.62E-01 | 1.09E-01 | 5.46E-06 | 2.62E-01 | 1.09E+00 |
| 2,6-Dinitrotoluene | NA | NA | NA | NA | NA |
| 1,4-Dioxane | 2.94E+01 | 9.71E-04 | 4.86E-08 | 2.94E+01 | 9.71E-03 |
| 1,2-Diphenylhydrazine | 2.19E-01 | 1.31E-01 | 6.53E-06 | 2.19E-01 | 1.31E+00 |
| Dipropylnitrosamine | NA | NA | NA | NA | NA |
| Epichlorohydrin | 2.70E+00 | 1.06E-02 | 5.29E-07 | 2.70E+00 | 1.06E-01 |
| Ethyl-4,4'-dichlorobenzilate | 5.59E-01 | 5.11E-02 | 2.56E-06 | 5.59E-01 | 5.11E-01 |
| Ethylene Dibromide (EDB) | 2.56E-03 | 1.11E+01 | 5.57E-04 | 2.56E-03 | 1.11E+02 |
| Ethylene Oxide | 4.13E-01 | 6.91E-02 | 3.46E-06 | 4.13E-01 | 6.91E-01 |

Date Prepared: October 1, 1986

# EXHIBIT A-3
## (Continued)

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| | Oral Route | | | Inhalation Route | |
|---|---|---|---|---|---|
| | 10% Effective Dose (ED10) | Toxicity Constant | | 10% Effective Dose (ED10) | Air Toxicity Constant (aTc) |
| Chemical Name | mg/kg/day | Water (wTc) 1/mg | Soil (sTc) kg/mg | mg/kg/day | m3/mg |
| Ethylenethiourea | 7.69E-01 | 3.71E-02 | 1.86E-06 | 7.69E-01 | 3.71E-01 |
| Ethyl Methanesulfonate | 5.58E-03 | 5.12E+00 | 2.56E-04 | 5.58E-03 | 5.12E+01 |
| 1-Ethyl-nitrosourea | 1.14E-01 | 2.50E-01 | 1.25E-05 | 1.14E-01 | 2.50E+00 |
| Formaldehyde | 4.90E-02 | 5.83E-01 | 2.92E-05 | 4.90E-02 | 5.83E+00 |
| Glycidaldehyde | 3.45E-01 | 8.29E-02 | 4.14E-06 | 3.45E-01 | 8.29E-01 |
| Heptachlor | 8.93E-03 | 3.20E+00 | 1.60E-04 | 8.93E-03 | 3.20E+01 |
| Heptachlor Epoxide | 3.45E-03 | 8.28E+00 | 4.14E-04 | 3.45E-03 | 8.28E+01 |
| Hexachlorobenzene | 8.51E-02 | 3.36E-01 | 1.68E-05 | 8.51E-02 | 3.36E+00 |
| Hexachlorobutadiene | 1.69E+00 | 1.69E-02 | 8.43E-07 | 1.69E+00 | 1.69E-01 |
| alpha-Hexachlorocyclohexane (HCCH) | 1.83E-02 | 1.56E+00 | 7.79E-05 | 1.83E-02 | 1.56E+01 |
| beta-HCCH | 5.75E-01 | 4.97E-02 | 2.49E-06 | 5.75E-01 | 4.97E-01 |
| gamma-HCCH (Lindane) | 5.46E-01 | 5.23E-02 | 2.61E-06 | 5.46E-01 | 5.23E-01 |
| Hexachloroethane | 1.25E+01 | 2.29E-03 | 1.14E-07 | 1.25E+01 | 2.29E-02 |
| Hydrazine | 1.27E-02 | 2.25E+00 | 1.13E-04 | 1.27E-02 | 2.25E+01 |
| Indeno(1,2,3-cd)pyrene | NA | NA | NA | NA | NA |
| Iodomethane | NA | NA | NA | NA | NA |
| Isosafrole | 1.67E+00 | 1.71E-02 | 8.57E-07 | 1.67E+00 | 1.71E-01 |
| Kepone | 2.09E-02 | 1.37E+00 | 6.85E-05 | 2.09E-02 | 1.37E+01 |
| Lasiocarpine | 2.66E-02 | 1.08E+00 | 5.38E-05 | 2.66E-02 | 1.08E+01 |
| Melphalan | 9.09E-04 | 3.14E+01 | 1.57E-03 | 9.09E-04 | 3.14E+02 |
| Methyl Chloride | 1.05E+01 | 2.71E-03 | 1.36E-07 | 1.05E+01 | 2.71E-02 |
| 3-Methylcholanthrene | 4.64E-02 | 6.16E-01 | 3.08E-05 | 4.64E-02 | 6.16E+00 |
| 4,4'-Methylene-bis-2-chloroaniline | 8.20E-01 | 3.49E-02 | 1.74E-06 | 8.20E-01 | 3.49E-01 |
| Methylnitrosourea | 9.48E-05 | 3.01E+02 | 1.51E-02 | 9.48E-05 | 3.01E+03 |
| Methylnitrosourethane | NA | NA | NA | NA | NA |
| Methylthiouracil | 3.50E-02 | 8.16E-01 | 4.08E-05 | 3.50E-02 | 8.16E+00 |
| Methylvinylnitrosamine | NA | NA | NA | NA | NA |
| N-Methyl-N'-nitro-N-nitrosoguanadine | 1.79E-02 | 1.59E+00 | 7.97E-05 | 1.79E-02 | 1.59E+01 |
| Mitomycin C | NA | NA | NA | NA | NA |
| 1-Napthylamine | NA | NA | NA | NA | NA |
| 2-Napthylamine | 1.98E-01 | 1.44E-01 | 7.21E-06 | 1.98E-01 | 1.44E+00 |
| Nickel and Compounds | NA | NA | NA | 1.00E-01 | 2.85E+00 |
| N-Nitrosopiperidine | 3.88E-02 | 7.37E-01 | 3.68E-05 | 3.88E-02 | 7.37E+00 |
| N-Nitrosopyrrolidine | 5.36E-03 | 5.33E+00 | 2.66E-04 | 5.36E-03 | 5.33E+01 |
| 5-Nitro-o-toluidine | 7.14E+00 | 4.00E-03 | 2.00E-07 | 7.14E+00 | 4.00E-02 |
| Pentachloronitrobenzene | 7.04E-01 | 4.06E-02 | 2.03E-06 | 7.04E-01 | 4.06E-01 |

Date Prepared: October 1, 1986

## EXHIBIT A-3
## (Continued)

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| | Oral Route | | | Inhalation Route | |
|---|---|---|---|---|---|
| | 10% Effective Dose (ED10) | Toxicity Constant | | 10% Effective Dose (ED10) | Air Toxicity Constant (aTc) |
| Chemical Name | mg/kg/day | Water (wTc) l/mg | Soil (sTc) kg/mg | mg/kg/day | m3/mg |
| Pentachlorophenol | NA | NA | NA | NA | NA |
| Phenacetin | 1.25E+01 | 2.29E-03 | 1.14E-07 | 1.25E+01 | 2.29E-02 |
| Polychlorinated Biphenyls (PCBs) | 5.00E-02 | 5.71E-01 | 2.86E-05 | 5.00E-02 | 5.71E+00 |
| Polynuclear Aromatic Hydrocarbons | NA | NA | NA | NA | NA |
| Propane Sultone | 2.85E-02 | 1.00E+00 | 5.01E-05 | 2.85E-02 | 1.00E+01 |
| 1,2-Propylenimine | 3.35E-02 | 8.53E-01 | 4.27E-05 | 3.35E-02 | 8.53E+00 |
| Saccharin | 2.44E+02 | 1.17E-04 | 5.86E-09 | 2.44E+02 | 1.17E-03 |
| Safrole | 5.00E+00 | 5.71E-03 | 2.86E-07 | 5.00E+00 | 5.71E-02 |
| Streptozocin | 9.17E-03 | 3.12E+00 | 1.56E-04 | 9.17E-03 | 3.12E+01 |
| 2,3,7,8-TCDD (Dioxin) | 8.33E-06 | 3.43E+03 | 1.71E-01 | 8.33E-06 | 3.43E+04 |
| 1,1,1,2-Tetrachloroethane | 1.20E+00 | 2.37E-02 | 1.19E-06 | 1.20E+00 | 2.37E-01 |
| 1,1,2,2-Tetrachloroethane | 6.02E-01 | 4.74E-02 | 2.37E-06 | 6.02E-01 | 4.74E-01 |
| Tetrachloroethylene | 3.23E+00 | 8.86E-03 | 4.43E-07 | 3.23E+00 | 8.86E-02 |
| Thioacetamide | 4.04E-02 | 7.07E-01 | 3.54E-05 | 4.04E-02 | 7.07E+00 |
| Thiourea | 9.52E-01 | 3.00E-02 | 1.50E-06 | 9.52E-01 | 3.00E-01 |
| o-Toluidine hydrochloride | 6.37E-01 | 4.49E-02 | 2.24E-06 | 6.37E-01 | 4.49E-01 |
| Toxaphene | 1.02E-01 | 2.80E-01 | 1.40E-05 | 1.02E-01 | 2.80E+00 |
| 1,1,2-Trichloroethane | 2.78E+00 | 1.03E-02 | 5.14E-07 | 2.78E+00 | 1.03E-01 |
| Trichloroethylene | 6.67E+00 | 4.29E-03 | 2.14E-07 | 6.67E+00 | 4.29E-02 |
| 2,4,6-Trichlorophenol | 1.25E+01 | 2.29E-03 | 1.14E-07 | 1.25E+01 | 2.29E-02 |
| Tris(2,3-dibromopropyl)phosphate | 1.02E-01 | 2.79E-01 | 1.39E-05 | 1.02E-01 | 2.79E+00 |
| Trypan Blue | 2.78E+00 | 1.03E-02 | 5.14E-07 | 2.78E+00 | 1.03E-01 |
| Uracil Mustard | NA | NA | NA | NA | NA |
| Urethane | 1.56E+00 | 1.83E-02 | 9.14E-07 | 1.56E+00 | 1.83E-01 |
| Vinyl Chloride | 6.67E+00 | 4.29E-03 | 2.14E-07 | 6.67E+00 | 4.29E-02 |

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[1] The list of chemicals presented in this exhibit is based on EPA's Reportable Quantities Analysis and should not be considered an all-inclusive list of suspected carcinogens. Refer to Exhibit A-4 for toxicity data for risk characterization for the chemicals listed here.

Date Prepared: October 1, 1986

# EXHIBIT A-4

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- RISK CHARACTERIZATION [1]

| Chemical Name | Oral Route: Potency Factor (PF) (mg/kg/d)-1 | Oral Route: Source[2] | Oral Route: EPA Weight of Evidence | Inhalation Route: Potency Factor (PF) (mg/kg/d)-1 | Inhalation Route: Source[2] | Inhalation Route: EPA Weight of Evidence |
|---|---|---|---|---|---|---|
| 2-Acetylaminofluorene | | | B2 | | | B2 |
| Acrylonitrile | | | B1 | 2.40E-01 | CAG | B1 |
| Aflatoxin B1 | 2.90E+03 | CAG | B2 | | | B2 |
| Aldrin | 1.14E+01 | CAG | B2 | | | B2 |
| Amitrole | | | B2 | | | B2 |
| Arsenic and Compounds | 1.50E+01 | HEA | A | 5.00E+01 | HEA | A |
| Asbestos | | | A | | | A |
| Auramine | | | B2 | | | B2 |
| Azaserine | | | B2 | | | B2 |
| Aziridine | | | B2 | | | B2 |
| Benzene | 5.20E-02 | HEA | A | 2.60E-02 | HEA | A |
| Benzidine | | | A | 2.30E+02 | CAG | A |
| Benz(a)anthracene | | | B2 | | | B2 |
| Benz(c)acridine | | | C | | | C |
| Benzo(a)pyrene | 1.15E+01 | HEA | B2 | 6.10E+00 | HEA | B2 |
| Benzo(b)fluoranthene | | | B2 | | | B2 |
| Benzo(k)fluoranthene | | | D | | | D |
| Benzotrichloride | | | B2 | | | B2 |
| Benzyl Chloride | | | C | | | C |
| Beryllium and Compounds | NA | | B1 | 4.86E+00 | CAG | B1 |
| Bis(2-chloroethyl)ether | 1.10E+00 | CAG | B2 | | | B2 |
| Bis(chloromethyl)ether | | | A | 9.30E+03 | CAG | A |
| Bis(2-ethylhexyl)phthalate (DEHP) | 6.84E-04 | CAG | B2 | | | B2 |
| Cacodylic Acid | | | D | | | D |
| Cadmium and Compounds | NA | | | 6.10E+00 | HEA | B1 |
| Carbon Tetrachloride | 1.30E-01 | HEA | B2 | | | B2 |
| Chlordane | 1.61E+00 | HEA | B2 | | | B2 |
| Chloroform | 8.10E-02 | HEA | B2 | | | B2 |
| 4-Chloro-o-toluidine Hydrochloride | | | B2 | | | B2 |
| Chromium VI and Compounds | NA | | | 4.10E+01 | HEA | A |
| Chrysene | | | B2 | | | B2 |
| Cyclophosphamide | | | B1 | | | B1 |
| DDD | | | B2 | | | B2 |
| DDE | | | B2 | | | B2 |
| DDT | 3.40E-01 | HEA | B2 | | | B2 |

Date Prepared: October 1, 1986

# EXHIBIT A-4
## (Continued)

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Potency Factor (PF) (mg/kg/d)-1 | Oral Route: Source[2] | Oral Route: EPA Weight of Evidence | Inhalation Route: Potency Factor (PF) (mg/kg/d)-1 | Inhalation Route: Source[2] | Inhalation Route: EPA Weight of Evidence |
|---|---|---|---|---|---|---|
| Diallate | | | C | | | B2 |
| Diaminotoluene (mixed) | | | B2 | | | B2 |
| 1,2,7,8-Dibenzopyrene | | | B2 | | | B2 |
| Dibenz(a,h)anthracene | | | B2 | | | B2 |
| 1,2-Dibromo-3-chloropropane | | | B2 | | | B2 |
| Dibutylnitrosamine | 5.40E+00 | CAG | B2 | | | B2 |
| 3,3'-Dichlorobenzidine | 1.70E+00 | CAG | B2 | | | B2 |
| 1,2-Dichloroethane (EDC) | 9.10E-02 | HEA | B2 | 3.50E-02 | HEA | B2 |
| 1,1-Dichloroethylene | 5.80E-01 | HEA | C | 1.16E+00 | HEA | C |
| Dichloromethane | 7.50E-03 | HEA | B2 | 1.43E-02 | HEA | B2 |
| Dieldrin | 3.00E+01 | CAG | B2 | | | B2 |
| Diepoxybutane | | | B2 | | | B2 |
| Diethanolnitrosamine | | | B2 | | | B2 |
| Diethyl Arsine | | | D | | | D |
| 1,2-Diethylhydrazine | | | B2 | | | B2 |
| Diethylnitrosamine | 4.40E+01 | CAG | B2 | | | B2 |
| Diethylstilbestrol (DES) | | | A | | | A |
| Dihydrosafrole | | | B2 | | | B2 |
| 3,3'-Dimethoxybenzidine | | | B2 | | | B2 |
| Dimethyl Sulfate | | | B2 | | | B2 |
| Dimethylaminoazobenzene | | | B2 | | | B2 |
| 7,12-Dimethylbenz(a)anthracene | | | B2 | | | B2 |
| 3,3'-Dimethylbenzidene | | | B2 | | | B2 |
| Dimethylcarbamoyl Chloride | | | B2 | | | B2 |
| 1,1-Dimethylhydrazine | | | B2 | | | B2 |
| 1,2-Dimethylhydrazine | | | B2 | | | B2 |
| Dimethylnitrosamine | 2.60E+01 | CAG | B2 | | | B2 |
| Dinitrotoluene (mixed) | | | B2 | | | B2 |
| 2,4-Dinitrotoluene | 3.10E-01 | CAG | B2 | | | B2 |
| 2,6-Dinitrotoluene | | | C | | | C |
| 1,4-Dioxane | | | B2 | | | B2 |
| 1,2-Diphenylhydrazine | 7.70E-01 | CAG | B1 | | | B2 |
| Dipropylnitrosamine | | | B2 | | | B2 |
| Epichlorohydrin | 9.90E-04 | CAG | B2 | | | B2 |
| Ethyl-4,4'-dichlorobenzilate | | | B2 | | | B2 |
| Ethylene Dibromide (EDB) | 4.10E+01 | CAG | B2 | | | B2 |

Date Prepared: October 1, 1986

# EXHIBIT A-4
## (Continued)

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Potency Factor (PF) (mg/kg/d)-1 | Oral Route: Source[2] | Oral Route: EPA Weight of Evidence | Inhalation Route: Potency Factor (PF) (mg/kg/d)-1 | Inhalation Route: Source[2] | Inhalation Route: EPA Weight of Evidence |
|---|---|---|---|---|---|---|
| Ethylene Oxide | | | B1/B2 | 3.50E-01 | CAG | B1/B2 |
| Ethylenethiourea | | | B2 | | | B2 |
| Ethyl Methanesulfonate | | | B2 | | | B2 |
| 1-Ethyl-nitrosourea | 3.30E+01 | CAG | B2 | | | B2 |
| Formaldehyde | | | B2 | | | B2 |
| Glycidaldehyde | | | B2 | | | B2 |
| Heptachlor | 3.40E+00 | CAG | B2 | | | B2 |
| Heptachlor Epoxide | 2.60E+00 | CAG | B2 | | | B2 |
| Hexachlorobenzene | 1.69E+00 | HEA | B2 | | | B2 |
| Hexachlorobutadiene | 7.75E-03 | HEA | C | | | C |
| alpha-Hexachlorocyclohexane (HCCH) | 1.10E+01 | CAG | B2 | | | B2 |
| beta-HCCH | 1.80E+00 | CAG | C | | | C |
| gamma-HCCH (Lindane) | 1.33E+00 | HEA | B2/C | | | B2/C |
| Hexachloroethane | 1.40E-02 | CAG | C | | | C |
| Hydrazine | | | B2 | | | B2 |
| Indeno(1,2,3-cd)pyrene | | | C | | | C |
| Iodomethane | | | C | | | C |
| Isosafrole | | | B2 | | | C |
| Kepone | | | B2 | | | B2 |
| Lasiocarpine | | | B2 | | | B2 |
| Melphalan | | | B1 | | | B1 |
| Methyl Chloride | | | C | | | C |
| 3-Methylcholanthrene | | | B2 | | | B2 |
| 4,4'-Methylene-bis-2-chloroaniline | | | B2 | | | B2 |
| Methylnitrosourea | 3.00E+02 | CAG | B2 | | | B2 |
| Methylnitrosourethane | | | B2 | | | B2 |
| Methylthiouracil | | | B2 | | | B2 |
| Methylvinylnitrosamine | | | B2 | | | B2 |
| N-Methyl-N'-nitro-N-nitrosoguanadine | | | B2 | | | B2 |
| Mitomycin C | | | B2 | | | B2 |
| 1-Napthylamine | | | C | | | C |
| 2-Napthylamine | | | A | | | A |
| Nickel and Compounds | NA | | A | 1.19E+00 | HEA | A |
| N-Nitrosopiperidine | | | B2 | | | B2 |
| N-Nitrosopyrrolidine | 2.10E+00 | CAG | B2 | | | B2 |
| 5-Nitro-o-toluidine | | | C | | | C |

Date Prepared: October 1, 1986

# EXHIBIT A-4
## (Continued)

## TOXICITY DATA FOR POTENTIAL CARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Potency Factor (PF) (mg/kg/d)-1 | Oral Route: Source[2] | Oral Route: EPA Weight of Evidence | Inhalation Route: Potency Factor (PF) (mg/kg/d)-1 | Inhalation Route: Source[2] | Inhalation Route: EPA Weight of Evidence |
|---|---|---|---|---|---|---|
| Pentachloronitrobenzene | | | C | | | C |
| Pentachlorophenol | | | D | | | D |
| Phenacetin | | | B2 | | | B2 |
| Polychlorinated Biphenyls (PCBs) | 4.34E+00 | HEA | B2 | | | B2 |
| Polynuclear Aromatic Hydrocarbons | 1.15E+01 | HEA | | 6.11E+00 | HEA | |
| Propane Sultone | | | B2 | | | B2 |
| 1,2-Propylenimine | | | B2 | | | B2 |
| Saccharin | | | C | | | C |
| Safrole | | | B2 | | | B2 |
| Streptozocin | | | B2 | | | B2 |
| 2,3,7,8-TCDD (Dioxin) | 1.56E+05 | HEA | B2 | | | B2 |
| 1,1,1,2-Tetrachloroethane | | | B2 | | | C |
| 1,1,2,2-Tetrachloroethane | 2.00E-01 | HEA | C | | | C |
| Tetrachloroethylene | 5.10E-02 | HEA | B2 | 1.70E-03 | HEA | B2 |
| Thioacetamide | | | B2 | | | B2 |
| Thiourea | | | B2 | | | B2 |
| o-Toluidine hydrochloride | | | B2 | | | B2 |
| Toxaphene | 1.10E+00 | CAG | B2 | | | B2 |
| 1,1,2-Trichloroethane | 5.73E-02 | HEA | C | | | C |
| Trichloroethylene | 1.10E-02 | HEA | B2 | 4.60E-03 | HEA | B2 |
| 2,4,6-Trichlorophenol | 1.98E-02 | HEA | B2 | | | B2 |
| Tris(2,3-dibromopropyl)phosphate | | | B2 | | | B2 |
| Trypan Blue | | | B2 | | | B2 |
| Uracil Mustard | | | B2 | | | B2 |
| Urethane | | | B2 | | | B2 |
| Vinyl Chloride | 2.30E+00 | HEA | A | 2.50E-02 | HEA | A |

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[1] The list of chemicals presented in this exhibit is based on EPA's Reportable Quantities Analysis and should not be considered an all-inclusive list of suspected carcinogens. Refer to Exhibit A-3 for toxicity constants for indicator selection for the chemicals listed here.

[2] Sources for Exhibit A-4:

HEA = Health Effects Assessment, prepared by the Environmental Criteria and Assessment Office, U.S. EPA, Cincinnati, Ohio, 1985 (updated in May 1986).

CAG = Evaluation by Carcinogen Assessment Group, U.S. EPA, Washington, D.C., 1985.

Date Prepared: October 1, 1986

# EXHIBIT A-5

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY [1]

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) 1/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| Acenaphthene @ | | | | | | | |
| Acenaphthylene @ | | | | | | | |
| Acetone | | | | | | | |
| Acetonitrile | | | | | 1.23E+02 | 8 | 1.31E+00 |
| 2-Acetylaminofluorene @ | | | | | | | |
| Acrylic Acid | | | | | | | |
| Acrylonitrile @ | 2.99E+01 | 9 | 6.02E-01 | 3.01E-05 | 4.34E+01 | 10 | 4.61E+00 |
| Aflatoxin B1 @ | | | | | | | |
| Aldicarb | | | | | | | |
| Aldrin @ | | | | | | | |
| Allyl Alcohol | 3.54E+00 * | 6 | 3.39E+00 | 1.69E-04 | 3.54E+00 | 6 | 3.39E+01 |
| Aluminum Phosphide | | | | | | | |
| 4-Aminobiphenyl @ | | | | | | | |
| Amitrole @ | | | | | | | |
| Ammonia | 8.80E-01 | 3 | 6.82E+00 | 3.41E-04 | 4.25E+01 | 5 | 2.35E+00 |
| Anthracene @ | | | | | | | |
| Antimony and Compounds | 4.60E+00 | 10 | 4.35E+00 | 2.17E-04 | 7.00E-01 | 8 | 2.29E+02 |
| Arsenic and Compounds @ | 1.00E+00 | 9 | 1.80E+01 | 9.00E-04 | 1.00E+00 * | 9 | 1.80E+02 |
| Asbestos @ | | | | | 2.70E-02 | 10 | 7.41E+03 |
| Auramine @ | | | | | | | |
| Azaserine @ | | | | | | | |
| Aziridine @ | | | | | | | |
| Barium and Compounds | 4.90E+00 | 10 | 4.08E+00 | 2.04E-04 | 4.90E+00 * | 10 | 4.08E+01 |
| Benefin | | | | | | | |
| Benzene @ | 8.55E+01 | 5 | 1.17E-01 | 5.85E-06 | 1.70E+00 | 10 | 1.18E+02 |
| Benzidine @ | 2.24E+01 | 8 | 7.14E-01 | 3.57E-05 | 1.19E+01 | 7 | 1.18E+01 |
| Benz(a)anthracene @ | | | | | | | |
| Benz(c)acridine @ | | | | | | | |
| Benzo(a)pyrene @ | 6.00E-01 | 8 | 2.67E+01 | 1.33E-03 | 6.28E+00 | 6 | 1.91E+01 |
| Benzo(b)fluoranthene @ | | | | | | | |
| Benzo(ghi)perylene @ | | | | | | | |
| Benzo(k)fluoranthene @ | | | | | | | |
| Benzotrichloride @ | | | | | | | |
| Benzyl Chloride @ | | | | | | | |
| Beryllium and Compounds @ | | | | | 1.10E-02 | 8 | 1.45E+04 |
| 1,1-Biphenyl | | | | | | | |
| Bis(2-chloroethyl)ether @ | | | | | | | |
| Bis(2-chloroisopropyl)ether | 7.43E+02 | 10 | 2.69E-02 | 1.35E-06 | 7.43E+02 * | 10 | 2.69E-01 |
| Bis(chloromethyl)ether @ | | | | | | | |
| Bis(2-ethylhexyl)phthalate (DEHP) @ | | | | | | | |
| Bromomethane | | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) 1/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| Bromoxynil Octanoate | | | | | | | |
| 1,3-Butadiene | 2.39E+00 | 4 | 3.35E+00 | 1.67E-04 | 2.39E+00 * | 4 | 3.35E+01 |
| n-Butanol | | | | | | | |
| Butylphthalyl Butylglycolate | | | | | | | |
| Cacodylic Acid @ | | | | | | | |
| Cadmium and Compounds @ | 4.49E+00 | 10 | 4.45E+00 | 2.23E-04 | 4.46E-01 | 8 | 3.59E+02 |
| Captan | 9.85E+02 | 10 | 2.03E-02 | 1.02E-06 | 9.85E+02 * | 10 | 2.03E-01 |
| Carbaryl | | | | | | | |
| Carbon Disulfide | 3.30E+01 * | 7 | 4.24E-01 | 2.12E-05 | 3.30E+01 | 7 | 4.24E+00 |
| Carbon Tetrachloride @ | 6.30E+01 * | 10 | 3.17E-01 | 1.59E-05 | 6.30E+01 | 10 | 3.17E+00 |
| Chlordane @ | | | | | | | |
| Chlorobenzene | 5.60E+01 | 4 | 1.43E-01 | 7.14E-06 | 7.18E+01 | 1 | 2.79E-01 |
| Chlorobenzilate @ | | | | | | | |
| Chlorodibromomethane | 6.60E+00 | 6 | 1.82E+00 | 9.09E-05 | 6.60E+00 * | 6 | 1.82E+01 |
| Chloroform @ | | | | | | | |
| Chloromethyl Methyl Ether @ | | | | | 5.90E+00 | 7 | 2.37E+01 |
| 4-Chloro-o-toluidine Hydrochloride@ | | | | | | | |
| Chromium III and Compounds | | | | | | | |
| Chromium VI and Compounds @ | | | | | 6.40E+00 | 8 | 2.50E+01 |
| Chrysene @ | | | | | | | |
| Copper and Compounds | 1.40E+01 | 5 | 7.14E-01 | 3.57E-05 | 1.40E+01 * | 5 | 7.14E+00 |
| Creosote @ | | | | | | | |
| Cresol | 1.34E+00 * | 4 | 5.97E+00 | 2.99E-04 | 1.34E+00 | 4 | 5.97E+01 |
| Crotonaldehyde | | | | | | | |
| Cyanides (n.o.s.) [2] | | | | | | | |
| -- Barium Cyanide | | | | | | | |
| -- Calcium Cyanide | | | | | | | |
| -- Cyanogen | | | | | | | |
| -- Cyanogen Chloride | | | | | | | |
| -- Copper Cyanide | | | | | | | |
| -- Hydrogen Cyanide | | | | | | | |
| -- Nickel Cyanide | | | | | | | |
| -- Potassium Cyanide | | | | | | | |
| -- Potassium Silver Cyanide | | | | | | | |
| -- Silver Cyanide | | | | | | | |
| -- Sodium Cyanide | | | | | | | |
| -- Zinc Cyanide | | | | | | | |
| Cyclophosphamide @ | | | | | | | |
| Dalapon | | | | | | | |
| DDD @ | | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) l/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| DDE @ | | | | | | | |
| DDT @ | | | | | | | |
| Decabromodiphenyl Ether | | | | | | | |
| Diallate @ | | | | | | | |
| 2,4-Diaminotoluene @ | | | | | | | |
| 1,2,7,8-Dibenzopyrene @ | | | | | | | |
| Dibenz(a,h)anthracene @ | | | | | | | |
| 1,2-Dibromo-3-chloropropane @ | | | | | | | |
| Dibutylnitrosamine @ | | | | | | | |
| Dibutyl Phthalate | 4.20E+02 | 8 | 3.81E-02 | 1.90E-06 | 4.20E+02 * | 8 | 3.81E-01 |
| 1,2-Dichlorobenzene | 1.54E+02 | 4 | 5.19E-02 | 2.60E-06 | 2.77E+02 * | 5 | 3.61E-01 |
| 1,3-Dichlorobenzene | 1.54E+02 | 4 | 5.19E-02 | 2.60E-06 | 2.77E+02 | 5 | 3.61E-01 |
| 1,4-Dichlorobenzene | 1.54E+02 | 4 | 5.19E-02 | 2.60E-06 | 2.77E+02 | 5 | 3.61E-01 |
| 3,3'-Dichlorobenzidine @ | | | | | | | |
| Dichlorodifluoromethane | | | | | | | |
| 1,1-Dichloroethane | 5.42E+02 * | 7 | 2.58E-02 | 1.29E-06 | 5.42E+02 | 7 | 2.58E-01 |
| 1,2-Dichloroethane (EDC) @ | 1.14E+03 | 10 | 1.76E-02 | 8.80E-07 | 1.45E+02 | 8 | 1.10E+00 |
| 1,1-Dichloroethylene @ | 3.77E+01 | 7 | 3.71E-01 | 1.86E-05 | 1.77E+01 | 5 | 5.65E+00 |
| 1,2-Dichloroethylene (cis) | 1.89E+02 * | 5 | 5.29E-02 | 2.65E-06 | 1.89E+02 | 5 | 5.29E-01 |
| 1,2-Dichloroethylene (trans) | 1.89E+02 * | 5 | 5.29E-02 | 2.65E-06 | 1.89E+02 | 5 | 5.29E-01 |
| Dichloromethane @ | 2.18E+04 | 10 | 9.20E-04 | 4.60E-08 | 2.18E+04 * | 10 | 9.20E-03 |
| 2,4-Dichlorophenol | 1.21E+02 | 5 | 8.26E-02 | 4.13E-06 | 1.21E+02 * | 5 | 8.26E-01 |
| 2,4-Dichlorophenoxyacetic Acid (2,4-D) | 1.29E+02 | 8 | 1.24E-01 | 6.20E-06 | 1.29E+02 * | 8 | 1.24E+00 |
| 4-(2,4-Dichlorophenoxy)butyric Acid (2,4-DB) | | | | | | | |
| Dichlorophenylarsine @ | | | | | | | |
| 1,2-Dichloropropane | 2.00E+02 * | 10 | 1.00E-01 | 5.00E-06 | 2.00E+02 | 10 | 1.00E+00 |
| 1,3-Dichloropropene | 6.00E-01 | 1 | 3.33E+00 | 1.67E-04 | 3.24E+00 | 5 | 3.09E+01 |
| Dieldrin @ | | | | | | | |
| Diepoxybutane @ | | | | | | | |
| Diethanolnitrosamine @ | | | | | | | |
| Diethyl Arsine @ | | | | | | | |
| 1,2-Diethylhydrazine @ | | | | | | | |
| Diethylnitrosamine @ | | | | | | | |
| Diethyl Phthalate | 2.99E+04 | 4 | 2.67E-04 | 1.34E-08 | 2.99E+04 * | 4 | 2.67E-03 |
| Diethylstilbestrol (DES) @ | | | | | | | |
| Dihydrosafrole @ | | | | | | | |
| Dimethoate | | | | | | | |
| 3,3'-Dimethoxybenzidine @ | | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) l/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| Dimethylamine | 3.70E+01 * | 6 | 3.24E-01 | 1.62E-05 | 3.70E+01 | 6 | 3.24E+00 |
| Dimethyl Sulfate @ | | | | | | | |
| Dimethyl Terephthalate | | | | | | | |
| Dimethylaminoazobenzene @ | | | | | | | |
| 7,12-Dimethylbenz(a)anthracene @ | | | | | | | |
| 3,3'-Dimethylbenzidine @ | | | | | | | |
| Dimethylcarbamoyl Chloride @ | | | | | | | |
| 1,1-Dimethylhydrazine @ | | | | | | | |
| 1,2-Dimethylhydrazine @ | | | | | | | |
| Dimethylnitrosamine @ | | | | | | | |
| 1,3-Dinitrobenzene | 1.35E+00 | 6 | 8.89E+00 | 4.44E-04 | 1.35E+00 * | 6 | 8.89E+01 |
| 4,6-Dinitro-o-cresol | 2.45E+00 | 8 | 6.53E+00 | 3.27E-04 | 2.45E+00 * | 8 | 6.53E+01 |
| 2,4-Dinitrophenol | 1.40E+01 | 8 | 1.14E+00 | 5.71E-05 | 1.40E+01 * | 8 | 1.14E+01 |
| 2,3-Dinitrotoluene @ | | | | | | | |
| 2,4-Dinitrotoluene @ | 2.05E+01 | 9 | 8.78E-01 | 4.39E-05 | 2.05E+01 * | 9 | 8.78E+00 |
| 2,5-Dinitrotoluene @ | | | | | | | |
| 2,6-Dinitrotoluene @ | 2.99E+01 | 9 | 6.02E-01 | 3.01E-05 | 2.99E+01 * | 9 | 6.02E+00 |
| 3,4-Dinitrotoluene @ | | | | | | | |
| Dinoseb | | | | | | | |
| 1,4-Dioxane @ | | | | | | | |
| N,N-Diphenylamine @ | | | | | | | |
| 1,2-Diphenylhydrazine @ | 5.98E+01 | 10 | 3.34E-01 | 1.67E-05 | 5.98E+01 * | 10 | 3.34E+00 |
| Dipropylnitrosamine @ | | | | | | | |
| Disulfoton | | | | | | | |
| Endosulfan | | | | | | | |
| Epichlorohydrin @ | | | | | | | |
| Ethanol | 2.40E+04 | 10 | 8.33E-04 | 4.17E-08 | 2.40E+04 * | 10 | 8.33E-03 |
| Ethyl Acetate | | | | | | | |
| Ethyl Methanesulfonate @ | | | | | | | |
| Ethylbenzene | 7.24E+02 * | 4 | 1.10E-02 | 5.52E-07 | 7.24E+02 | 4 | 1.10E-01 |
| Ethyl-4,4'-dichlorobenzilate @ | | | | | | | |
| Ethylene Dibromide (EDB) @ | | | | | | | |
| Ethylene Oxide @ | | | | | | | |
| Ethylenethiourea @ | | | | | | | |
| 1-Ethyl-nitrosourea @ | | | | | | | |
| Ethylphthalyl Ethyl Glycolate | | | | | | | |
| Ferric Dextran @ | | | | | | | |
| Fluoranthene @ | | | | | | | |
| Fluorene @ | | | | | | | |
| Fluorides | 8.01E+00 | 5 | 1.25E+00 | 6.24E-05 | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) l/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| Fluridone | | | | | | | |
| Formaldehyde | | | | | 1.00E+00 | 7 | 1.40E+02 |
| Formic Acid | | | | | | | |
| Furan | | | | | | | |
| Glycidaldehyde @ | | | | | | | |
| Glycol Ethers (n.o.s.) | | | | | | | |
| -- Diethylene Glycol, Monoethyl Ether | | | | | | | |
| -- 2-Ethoxyethanol | | | | | | | |
| -- Ethylene Glycol, Monobutyl Ether | | | | | | | |
| -- 2-Methoxyethanol | | | | | | | |
| -- Propylene Glycol, Monoethyl Ether | | | | | | | |
| -- Propylene Glycol, Monomethyl Ether | | | | | | | |
| Heptachlor @ | | | | | | | |
| Heptachlor Epoxide @ | | | | | | | |
| Hexachlorobenzene @ | 5.00E+01 | 10 | 4.00E-01 | 2.00E-05 | 5.00E+01 * | 10 | 4.00E+00 |
| Hexachlorobutadiene @ | | | | | | | |
| Hexachlorocyclopentadiene | | | | | | | |
| alpha-Hexachlorocyclohexane (HCCH)@ | | | | | | | |
| beta-HCCH @ | | | | | | | |
| gamma-HCCH (Lindane) @ | | | | | | | |
| delta-HCCH @ | | | | | | | |
| Hexachloroethane @ | 1.81E+03 | 6 | 6.62E-03 | 3.31E-07 | 4.49E+02 | 10 | 4.45E-01 |
| Hexachlorophene | 2.99E+01 | 9 | 6.02E-01 | 3.01E-05 | 2.99E+01 * | 9 | 6.02E+00 |
| Hydrazine @ | | | | | | | |
| Hydrogen Sulfide | | | | | | | |
| Indeno(1,2,3-cd)pyrene @ | | | | | | | |
| Iodomethane @ | | | | | | | |
| Iron and Compounds | | | | | | | |
| Isobutanol | | | | | | | |
| Isoprene | 5.50E+02 * | 4 | 1.45E-02 | 7.27E-07 | 5.50E+02 | 4 | 1.45E-01 |
| Isosafrole @ | | | | | | | |
| Isophorone | | | | | | | |
| Isopropalin | | | | | | | |
| Kepone @ | | | | | | | |
| Lasiocarpine @ | | | | | | | |
| Lead and Compounds (Inorganic) | 2.24E+01 | 10 | 8.93E-01 | 4.46E-05 | 2.24E+01 * | 10 | 8.93E+00 |
| Linuron | | | | | | | |
| Malathion | | | | | | | |
| Manganese and Compounds | | | | | | | |
| Melphalan @ | | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) 1/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| Mercury and Compounds (Alkyl) | | | | | | | |
| Mercury and Compounds (Inorganic) | 7.60E-01 | 7 | 1.84E+01 | 9.21E-04 | 8.60E-01 | 8 | 1.86E+02 |
| Mercury Fulminate | | | | | | | |
| Methanol | | | | | | | |
| Methyl Chloride | 2.21E+02 * | 10 | 9.05E-02 | 4.52E-06 | 2.21E+02 | 10 | 9.05E-01 |
| Methyl Ethyl Ketone | 2.58E+03 * | 10 | 7.75E-03 | 3.87E-07 | 2.58E+03 | 10 | 7.75E-02 |
| Methyl Ethyl Ketone Peroxide | | | | | | | |
| Methyl Isobutyl Ketone | | | | | | | |
| Methyl Methacrylate | 1.76E+03 | 4 | 4.55E-03 | 2.28E-07 | 1.22E+02 | 7 | 1.15E+00 |
| Methyl Parathion | 1.07E+01 | 10 | 1.87E+00 | 9.35E-05 | 2.40E-02 | 5 | 4.17E+03 |
| 2-Methyl-4-Chlorophenoxyacetic Acid | | | | | | | |
| 2(2-Methyl-4-Chlorophenoxy) propionic Acid | | | | | | | |
| 3-Methylcholanthrene @ | | | | | | | |
| 4,4'-Methylene-bis-2-chloroaniline@ | | | | | | | |
| Methylnitrosourea @ | | | | | | | |
| Methylthiouracil @ | | | | | | | |
| Methylvinylnitrosamine @ | | | | | | | |
| N-Methyl-N'-nitro-N-nitrosoguanadine@ | | | | | | | |
| Mitomycin C @ | | | | | | | |
| Mustard Gas @ | | | | | | | |
| 1-Napthylamine @ | | | | | | | |
| 2-Napthylamine @ | | | | | | | |
| Nickel and Compounds @ | 4.70E+00 | 10 | 4.26E+00 | 2.13E-04 | 1.27E+00 | 10 | 1.57E+02 |
| Nitric Oxide | | | | | | | |
| Nitrobenzene | | | | | | | |
| Nitrogen Dioxide | | | | | | | |
| Nitrosomethylurethane @ | | | | | | | |
| N-Nitrosopiperidine @ | | | | | | | |
| N-Nitrosopyrrolidine @ | | | | | | | |
| 5-Nitro-o-toluidine @ | | | | | | | |
| Osmium Tetroxide | | | | | | | |
| Pentachlorobenzene | 8.62E+02 | 10 | 2.32E-02 | 1.16E-06 | 8.62E+02 * | 10 | 2.32E-01 |
| Pentachloronitrobenzene @ | 2.20E-01 | 6 | 5.45E+01 | 2.73E-03 | 2.20E-01 * | 6 | 5.45E+02 |
| Pentachlorophenol | | | | | | | |
| Phenacetin @ | | | | | | | |
| Phenanthrene @ | | | | | | | |
| Phenobarbital @ | | | | | | | |
| Phenol | 5.98E+01 | 3 | 1.00E-01 | 5.02E-06 | 8.02E+01 | 10 | 2.49E+00 |
| Phenylalanine Mustard @ | | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
(Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS
## -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) l/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| m-Phenylenediamine | | | | | | | |
| Phenyl Mercuric Acetate | | | | | | | |
| Phosphine | | | | | | | |
| Polychlorinated Biphenyls (PCBs) @ | | | | | | | |
| Propane Sultone @ | | | | | | | |
| Propylenimine @ | | | | | | | |
| Pyrene @ | | | | | | | |
| Pyridine | | | | | | | |
| Saccharin @ | | | | | | | |
| Safrole @ | | | | | | | |
| Selenium and Compounds (n.o.s.) | 1.90E-01 | 10 | 1.05E+02 | 5.26E-03 | 1.90E-01 * | 10 | 1.05E+03 |
| -- Selenious Acid | | | | | | | |
| -- Selenourea | | | | | | | |
| -- Thallium Selenite | | | | | | | |
| Silver and Compounds | 1.00E-01 | 1 | 2.00E+01 | 1.00E-03 | 1.00E-01 * | 1 | 2.00E+02 |
| Sodium Diethyldithiocarbamate | | | | | | | |
| Streptozocin @ | | | | | | | |
| Strychnine | | | | | | | |
| Styrene | | | | | | | |
| 1,2,4,5-Tetrachlorobenzene | 2.05E+01 | 1 | 9.76E-02 | 4.88E-06 | 2.05E+01 * | 1 | 9.76E-01 |
| 2,3,7,8-TCDD (Dioxin) @ | | | | | | | |
| 1,1,1,2-Tetrachloroethane @ | | | | | | | |
| 1,1,2,2-Tetrachloroethane @ | 2.20E+01 * | 5 | 4.55E-01 | 2.27E-05 | 2.20E+01 | 5 | 4.55E+00 |
| Tetrachloroethylene @ | 1.46E+03 | 7 | 9.62E-03 | 4.81E-07 | 7.27E+03 | 10 | 2.75E-02 |
| 2,3,4,6-Tetrachlorophenol | 1.07E+01 | 8 | 1.50E+00 | 7.48E-05 | 1.07E+01 * | 8 | 1.50E+01 |
| 2,3,5,6-Tetrachloroterephthalate Acid (DCPA) | | | | | | | |
| Tetraethyl Lead @ | 1.40E-03 | 5 | 7.14E+03 | 3.57E-01 | 2.50E+00 | 5 | 4.00E+01 |
| Thallium and Compounds (n.o.s.) | | | | | | | |
| -- Thallium Acetate | | | | | | | |
| -- Thallium Carbonate | | | | | | | |
| -- Thallium Chloride | | | | | | | |
| -- Thallium Nitrate | | | | | | | |
| -- Thallic Oxide | | | | | | | |
| -- Thallium Sulfate | | | | | | | |
| Thioacetamide @ | | | | | | | |
| Thiourea @ | | | | | | | |
| o-Tolidine @ | | | | | | | |
| Toluene | 2.69E+03 * | 7 | 5.20E-03 | 2.60E-07 | 2.69E+03 | 7 | 5.20E-02 |
| o-Toluidine Hydrochloride @ | | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-5
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- SELECTION OF INDICATOR CHEMICALS ONLY

| Chemical Name | Oral Route: Minimum Effective Dose (MED) mg/day | RVe | Toxicity Constant: Water (wTn) 1/mg | Toxicity Constant: Soil (sTn) kg/mg | Inhalation Route: Minimum Effective Dose (MED) mg/day | RVe | Air Toxicity Constant (aTn) m3/kg |
|---|---|---|---|---|---|---|---|
| Toxaphene @ | | | | | | | |
| Tribromomethane (Bromoform) | 6.60E+00 | 6 | 1.82E+00 | 9.09E-05 | 6.60E+00 * | 6 | 1.82E+01 |
| 1,2,4-Trichlorobenzene | 3.73E+01 | 4 | 2.14E-01 | 1.07E-05 | 1.32E+01 | 1 | 1.52E+00 |
| 1,1,1-Trichloroethane | 5.45E+03 * | 2 | 7.33E-04 | 3.67E-08 | 5.45E+03 | 2 | 7.33E-03 |
| 1,1,2-Trichloroethane @ | | | | | | | |
| Trichloroethylene @ | 9.50E+00 | 5 | 1.05E+00 | 5.26E-05 | 2.70E+00 | 4 | 2.96E+01 |
| Trichlorofon | 4.52E+01 | 10 | 4.42E-01 | 2.21E-05 | 4.52E+01 * | 10 | 4.42E+00 |
| Trichloromonofluoromethane | | | | | | | |
| 2,4,5-Trichlorophenol | 1.18E+02 | 6 | 1.02E-01 | 5.10E-06 | 1.18E+02 * | 6 | 1.02E+00 |
| 2,4,6-Trichlorophenol @ | | | | | | | |
| 2,4,5-Trichlorophenoxyacetic Acid | | | | | | | |
| 1,2,3-Trichloropropane | | | | | | | |
| 1,1,2-Trichloro-1,2,2-trifluoroethane | | | | | | | |
| Tris(2,3-dibromopropyl)phosphate @ | | | | | | | |
| Trinitrotoluene (TNT) | | | | | | | |
| Trypan Blue @ | | | | | | | |
| Uracil Mustard @ | | | | | | | |
| Uranium and Compounds | 1.70E+00 | 6 | 7.06E+00 | 3.53E-04 | 1.70E+00 * | 6 | 7.06E+01 |
| Urethane @ | | | | | | | |
| Vanadium and Compounds | 1.40E+01 | 1 | 1.43E-01 | 7.14E-06 | 1.40E+01 * | 1 | 1.43E+00 |
| Vinyl Chloride @ | 2.28E+02 * | 10 | 8.77E-02 | 4.39E-06 | 2.28E+02 | 10 | 8.77E-01 |
| Warfarin | | | | | | | |
| o-Xylene | | | | | | | |
| m-Xylene | | | | | | | |
| p-Xylene | | | | | | | |
| Xylenes (mixed) | | | | | | | |
| Zinc and Compounds | 1.50E+02 | 8 | 1.07E-01 | 5.33E-06 | 1.50E+02 | 8 | 1.07E+00 |
| -- Zinc Phosphide | | | | | | | |
| Zineb | | | | | | | |

@ Potential carcinogenic effects also. See Exhibits A-3 and A-4.

* MED and RVe values marked with an asterisk are based on values for the other exposure route.

[1] Refer to Exhibit A-6 for toxicity data for risk characterization for the chemicals listed here.

[2] N.O.S. = not otherwise specified.

Date Prepared: October 1, 1986

# EXHIBIT A-6

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION [1]

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source[2] | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source[2] |
|---|---|---|---|---|---|---|
| Acenaphthene @ | | | | | | |
| Acenaphthylene @ | | | | | | |
| Acetone | | 1.00E-01 | RfD | 3.00E+01 | 3.00E+00 | HEA |
| Acetonitrile | | | | | | |
| 2-Acetylaminofluorene @ | | | | | | |
| Acrylic Acid | | 8.00E-02 | RfD[3] | | | |
| Acrylonitrile @ | | | | | | |
| Aflatoxin B1 @ | | | | | | |
| Aldicarb | | 1.00E-02 | RfD | | | |
| Aldrin @ | | 3.00E-05 | RfD | | | |
| Allyl Alcohol | | 5.00E-03 | RfD | | | |
| Aluminum Phosphide | | 4.00E-04 | RfD | | | |
| 4-Aminobiphenyl @ | | | | | | |
| Amitrole @ | | | | | | |
| Ammonia | | | | | | |
| Anthracene @ | | | | | | |
| Antimony and Compounds | | 4.00E-04 | RfD | | | |
| Arsenic and Compounds @ | | | | | | |
| Asbestos @ | | | | | | |
| Auramine @ | | | | | | |
| Azaserine @ | | | | | | |
| Aziridine @ | | | | | | |
| Barium and Compounds | | 5.10E-02 | HEA | 1.4E-3(T)[4] | 1.40E-04 | HEA |
| Benefin | | 3.00E-01 | RfD | | | |
| Benzene @ | | | | | | |
| Benzidine @ | | | | | | |
| Benz(a)anthracene @ | | | | | | |
| Benz(c)acridine @ | | | | | | |
| Benzo(a)pyrene @ | | | | | | |
| Benzo(b)fluoranthene @ | | | | | | |
| Benzo(ghi)perylene @ | | | | | | |
| Benzo(k)fluoranthene @ | | | | | | |
| Benzotrichloride @ | | | | | | |
| Benzyl Chloride @ | | | | | | |
| Beryllium and Compounds @ | | 5.00E-04 | RfD | | | |
| 1,1-Biphenyl | | 5.00E-02 | RfD | | | |
| Bis(2-chloroethyl)ether @ | | | | | | |
| Bis(2-chloroisopropyl)ether | | | | | | |
| Bis(chloromethyl)ether @ | | | | | | |
| Bis(2-ethylhexyl)phthalate (DEHP) @ | | 2.00E-02 | RfD | | | |
| Bromomethane | | 4.00E-04 | RfD | | | |
| Bromoxynil Octanoate | | 3.00E-02 | RfD | | | |
| 1,3-Butadiene | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-6
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| n-Butanol | | 1.00E-01 | RfD | | | |
| Butylpthalyl Butylglycolate | | 1.00E+00 | RfD | | | |
| Cacodylic Acid @ | | 1.00E-02 | RfD | | | |
| Cadmium and Compounds @ | | 2.90E-04 | HEA | | | |
| Captan | | | | | | |
| Carbaryl | | 1.00E-01 | RfD | | | |
| Carbon Disulfide | | 1.00E-01 | RfD | | | |
| Carbon Tetrachloride @ | | | | | | |
| Chlordane @ | | 5.00E-05 | RfD | | | |
| Chlorobenzene | 2.70E-01 | 2.70E-02 | HEA | 5.30E-02 | 5.70E-03 | HEA |
| Chlorobenzilate @ | | | | | | |
| Chlorodibromomethane | | | | | | |
| Chloroform @ | | 1.00E-02 | RfD | | | |
| Chloromethyl Methyl Ether @ | | | | | | |
| 4-Chloro-o-toluidine Hydrochloride@ | | | | | | |
| Chromium III and Compounds | 1.40E+01 | 1.00E+00 | RfD | | 5.10E-03 | HEA |
| Chromium VI and Compounds @ | 2.50E-02 | 5.00E-03 | HEA | | | |
| Chrysene @ | | | | | | |
| Copper and Compounds | 3.70E-02 | 3.70E-02 | HEA | | 1.00E-02 | HEA |
| Creosote @ | | | | | | |
| Cresol | | 5.00E-02 | RfD | | 1.00E-01 | HEA |
| Crotonaldehyde | | 1.00E-02 | RfD | | | |
| Cyanides (n.o.s.) [5] | | 2.00E-02 | RfD | | | |
| -- Barium Cyanide | | 7.00E-02 | RfD | | | |
| -- Calcium Cyanide | | 4.00E-02 | RfD | | | |
| -- Cyanogen | | 4.00E-02 | RfD | | | |
| -- Cyanogen Chloride | | 5.00E-02 | RfD | | | |
| -- Copper Cyanide | | 7.00E-02 | RfD | | | |
| -- Hydrogen Cyanide | | 2.00E-02 | RfD | | | |
| -- Nickel Cyanide | | 2.00E-02 | RfD | | | |
| -- Potassium Cyanide | | 5.00E-02 | RfD | | | |
| -- Potassium Silver Cyanide | | 2.00E-01 | RfD | | | |
| -- Silver Cyanide | | 1.00E-01 | RfD | | | |
| -- Sodium Cyanide | | 4.00E-02 | RfD | | | |
| -- Zinc Cyanide | | 5.00E-02 | RfD | | | |
| Cyclophosphamide @ | | | | | | |
| Dalapon | | 8.00E-02 | RfD | | | |
| DDD @ | | | | | | |
| DDE @ | | | | | | |
| DDT @ | | 5.00E-04 | RfD | | | |
| Decabromodiphenyl Ether | | 1.00E-02 | RfD | | | |
| Diallate @ | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-6
(Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| 2,4-Diaminotoluene @ | | | | | | |
| 1,2,7,8-Dibenzopyrene @ | | | | | | |
| Dibenz(a,h)anthracene @ | | | | | | |
| 1,2-Dibromo-3-chloropropane @ | | | | | | |
| Dibutylnitrosamine @ | | | | | | |
| Dibutyl Phthalate | | 1.00E-01 | RfD | | | |
| 1,2-Dichlorobenzene | | | | | | |
| 1,3-Dichlorobenzene | | | | | | |
| 1,4-Dichlorobenzene | | | | | | |
| 3,3'-Dichlorobenzidine @ | | | | | | |
| Dichlorodifluoromethane | | 2.00E-01 | RfD | | | |
| 1,1-Dichloroethane | 1.20E+00 | 1.20E-01 | HEA | 1.38E+00 | 1.38E-01 | HEA |
| 1,2-Dichloroethane (EDC) @ | | | | | | |
| 1,1-Dichloroethylene @ | | 9.00E-03 | RfD | | | |
| 1,2-Dichloroethylene (cis) | | | | | | |
| 1,2-Dichloroethylene (trans) | | | | | | |
| Dichloromethane @ | | 6.00E-02 | RfD | | | |
| 2,4-Dichlorophenol | | 3.00E-03 | RfD | | | |
| 2,4-Dichlorophenoxyacetic Acid (2,4-D) | | | | | | |
| 4-(2,4-Dichlorophenoxy)butyric Acid (2,4-DB) | | 8.00E-03 | RfD | | | |
| Dichlorophenylarsine @ | | | | | | |
| 1,2-Dichloropropane | | | | | | |
| 1,3-Dichloropropene | | | | | | |
| Dieldrin @ | | | | | | |
| Diepoxybutane @ | | | | | | |
| Diethanolnitrosamine @ | | | | | | |
| Diethyl Arsine @ | | | | | | |
| 1,2-Diethylhydrazine @ | | | | | | |
| Diethylnitrosamine @ | | | | | | |
| Diethyl Phthalate | | 1.30E+01 | RfD | | | |
| Diethylstilbestrol (DES) @ | | | | | | |
| Dihydrosafrole @ | | | | | | |
| Dimethoate | | 2.00E-02 | RfD | | | |
| 3,3'-Dimethoxybenzidine @ | | | | | | |
| Dimethylamine | | | | | | |
| Dimethyl Sulfate @ | | | | | | |
| Dimethyl Terephthalate | | 1.00E-01 | RfD | | | |
| Dimethylaminoazobenzene @ | | | | | | |
| 7,12-Dimethylbenz(a)anthracene @ | | | | | | |
| 3,3'-Dimethylbenzidine @ | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-6
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| Dimethylcarbamoyl Chloride @ | | | | | | |
| 1,1-Dimethylhydrazine @ | | | | | | |
| 1,2-Dimethylhydrazine @ | | | | | | |
| Dimethylnitrosamine @ | | | | | | |
| 1,3-Dinitrobenzene | | | | | | |
| 4,6-Dinitro-o-cresol | | | | | | |
| 2,4-Dinitrophenol | | 2.00E-03 | RfD | | | |
| 2,3-Dinitrotoluene @ | | | | | | |
| 2,4-Dinitrotoluene @ | | | | | | |
| 2,5-Dinitrotoluene @ | | | | | | |
| 2,6-Dinitrotoluene @ | | | | | | |
| 3,4-Dinitrotoluene @ | | | | | | |
| Dinoseb | | 1.00E-03 | RfD | | | |
| 1,4-Dioxane @ | | | | | | |
| N,N-Diphenylamine @ | | | | | | |
| 1,2-Diphenylhydrazine @ | | | | | | |
| Dipropylnitrosamine @ | | | | | | |
| Disulfoton | | 4.00E-03 | RfD | | | |
| Endosulfan | | 1.50E-05 | RfD | | | |
| Epichlorohydrin @ | | 2.00E-03 | RfD | | | |
| Ethanol | | | | | | |
| Ethyl Acetate | | 9.00E-01 | RfD | | | |
| Ethyl Methanesulfonate @ | | | | | | |
| Ethylbenzene | 9.70E-01 | 1.00E-01 | RfD | | | |
| Ethyl-4,4'-dichlorobenzilate @ | | | | | | |
| Ethylene Dibromide (EDB) @ | | | | | | |
| Ethylene Oxide @ | | | | | | |
| Ethylenethiourea @ | | | | | | |
| 1-Ethyl-nitrosourea @ | | | | | | |
| Ethylphthalyl Ethyl Glycolate | | 3.00E+00 | RfD | | | |
| Ferric Dextran @ | | | | | | |
| Fluoranthene @ | | | | | | |
| Fluorene @ | | | | | | |
| Fluorides | | 6.00E-02 | RfD | | | |
| Fluridone | | 8.00E-02 | RfD | | | |
| Formaldehyde | | | | | | |
| Formic Acid | | 2.00E+00 | RfD | | | |
| Furan | | 1.00E-03 | RfD | | | |
| Glycidaldehyde @ | | | | | | |
| Glycol Ethers (n.o.s.) | | | | | | |
| -- Diethylene Glycol, Monoethyl Ether | 5.00E+00 | 2.00E+00 | HEA | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-6
(Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| -- 2-Ethoxyethanol | 4.7E-1(T) | 3.60E-01 | HEA | 6.9E-2(T) | 5.00E-02 | HEA |
| -- Ethylene Glycol, Monobutyl Ether | | | | 1.60E-01 | 1.60E-02 | HEA |
| -- 2-Methoxyethanol | | | | 5.9E-2(T) | 2.40E-02 | HEA |
| -- Propylene Glycol, Monoethyl Ether | 6.80E+00 | 6.80E-01 | HEA | | | |
| -- Propylene Glycol, Monomethyl Ether | 6.80E+00 | 6.80E-01 | HEA | 4.90E+00 | 4.90E-01 | HEA |
| Heptachlor @ | | | | | | |
| Heptachlor Epoxide @ | | 3.00E-05 | RfD | | | |
| Hexachlorobenzene @ | | | | | | |
| Hexachlorobutadiene @ | | 2.00E-03 | RfD | | | |
| Hexachlorocyclopentadiene | 7.00E-02 | 7.00E-03 | RfD | 2.90E-03 | 6.60E-05 | HEA |
| alpha-Hexachlorocyclohexane (HCCH)@ | | | | | | |
| beta-HCCH @ | | | | | | |
| gamma-HCCH (Lindane) @ | | 3.00E-04 | RfD | | | |
| delta-HCCH @ | | | | | | |
| Hexachloroethane @ | | | | | | |
| Hexachlorophene | | | | | | |
| Hydrazine @ | | | | | | |
| Hydrogen Sulfide | | 3.00E-03 | RfD | | | |
| Indeno(1,2,3-cd)pyrene @ | | | | | | |
| Iodomethane @ | | | | | | |
| Iron and Compounds | | | | | 8.60E-03 | HEA |
| Isobutanol | | 3.00E-01 | RfD | | | |
| Isoprene | | | | | | |
| Isosafrole @ | | | | | | |
| Isophorone | | 2.00E-01 | RfD | | | |
| Isopropalin | | 3.00E-02 | RfD | | | |
| Kepone @ | | | | | | |
| Lasiocarpine @ | | | | | | |
| Lead and Compounds (Inorganic) | | 1.40E-03 | HEA | | 4.30E-04 | HEA |
| Linuron | | | | | | |
| Malathion | | 2.00E-02 | RfD | | | |
| Manganese and Compounds | 5.30E-01 | 2.20E-01 | HEA | 3.00E-04 | 3.00E-04 | HEA |
| Melphalan @ | | | | | | |
| Mercury and Compounds (Alkyl) | 2.80E-04 | 3.00E-04 | RfD | 1.00E-04 | 1.00E-04 | HEA |
| Mercury and Compounds (Inorganic) | 2.00E-03 | 2.00E-03 | RfD | 5.10E-04 | 5.10E-05 | HEA |
| Mercury Fulminate | | 3.00E-03 | RfD | | | |
| Methanol | | 5.00E-01 | RfD | | | |
| Methyl Chloride | | | | | | |
| Methyl Ethyl Ketone | | 5.00E-02 | RfD | 2.20E+00 | 2.20E-01 | HEA |

Date Prepared: October 1, 1986

# EXHIBIT A-6
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| Methyl Ethyl Ketone Perioxide | | 8.00E-03 | RfD | | | |
| Methyl Isobutyl Ketone | | 5.00E-02 | RfD | | | |
| Methyl Methacrylate | | | | | | |
| Methyl Parathion | | | | | | |
| 2-Methyl-4-Chlorophenoxyacetic Acid | | 1.00E-03 | RfD | | | |
| 2(2-Methyl-4-Chlorophenoxy) propionic Acid | | 3.00E-03 | RfD | | | |
| 3-Methylcholanthrene @ | | | | | | |
| 4,4'-Methylene-bis-2-chloroaniline@ | | | | | | |
| Methylnitrosourea @ | | | | | | |
| Methylthiouracil @ | | | | | | |
| Methylvinylnitrosamine @ | | | | | | |
| N-Methyl-N'-nitro-N-nitrosoguanadine@ | | | | | | |
| Mitomycin C @ | | | | | | |
| Mustard Gas @ | | | | | | |
| 1-Napthylamine @ | | | | | | |
| 2-Napthylamine @ | | | | | | |
| Nickel and Compounds @ | 2.00E-02 | 1.00E-02 | HEA | | | |
| Nitric Oxide | | 1.00E-01 | RfD | | | |
| Nitrobenzene | | 5.00E-04 | RfD | | | |
| Nitrogen Dioxide | | 1.00E+00 | RfD | | | |
| Nitrosomethylurethane @ | | | | | | |
| N-Nitrosopiperidine @ | | | | | | |
| N-Nitrosopyrrolidine @ | | | | | | |
| 5-Nitro-o-toluidine @ | | | | | | |
| Osmium Tetroxide | | 1.00E-05 | RfD | | | |
| Pentachlorobenzene | | 8.00E-04 | RfD | | | |
| Pentachloronitrobenzene @ | | 8.00E-03 | RfD | | | |
| Pentachlorophenol | 3.0E-2(T) | 3.00E-02 | RfD | | | |
| Phenacetin @ | | | | | | |
| Phenanthrene @ | | | | | | |
| Phenobarbital @ | | | | | | |
| Phenol | 1.00E-01 | 1.00E-01 | RfD | 1.90E-01 | 2.00E-02 | HEA |
| Phenylalanine Mustard @ | | | | | | |
| m-Phenylenediamine | | 6.00E-03 | RfD | | | |
| Phenyl Mercuric Acetate | | 8.00E-05 | RfD | | | |
| Phosphine | | 3.00E-04 | RfD | | | |
| Polychlorinated Biphenyls (PCBs) @ | | | | | | |
| Propane Sultone @ | | | | | | |
| Propylenimine @ | | | | | | |
| Pyrene @ | | | | | | |
| Pyridine | | 2.00E-03 | RfD | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-6
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| Saccharin @ | | | | | | |
| Safrole @ | | | | | | |
| Selenium and Compounds (n.o.s.) | 3.20E-03 | 3.00E-03 | HEA | | 1.00E-03 | HEA |
| -- Selenious Acid | | 3.00E-03 | RfD | | | |
| -- Selenourea | | 5.00E-03 | RfD | | | |
| -- Thallium Selenite | | 5.00E-04 | RfD | | | |
| Silver and Compounds | | 3.00E-03 | RfD | | | |
| Sodium Diethyldithiocarbamate | | 3.00E-02 | RfD | | | |
| Streptozocin @ | | | | | | |
| Strychnine | | 3.00E-04 | RfD | | | |
| Styrene | | 2.00E-01 | RfD | | | |
| 1,2,4,5-Tetrachlorobenzene | | 3.00E-04 | RfD | | | |
| 2,3,7,8-TCDD (Dioxin) @ | | | | | | |
| 1,1,1,2-Tetrachloroethane @ | | | | | | |
| 1,1,2,2-Tetrachloroethane @ | | | | | | |
| Tetrachloroethylene @ | | 2.00E-02 | RfD | | | |
| 2,3,4,6-Tetrachlorophenol | | 1.00E-02 | RfD | | | |
| 2,3,5,6-Tetrachloroterephthalate Acid (DCPA) | | 5.00E-02 | RfD | | | |
| Tetraethyl Lead @ | | 1.00E-07 | RfD | | | |
| Thallium and Compounds (n.o.s.) | | 4.00E-04 | RfD | | | |
| -- Thallium Acetate | | 5.00E-04 | RfD | | | |
| -- Thallium Carbonate | | 4.00E-04 | RfD | | | |
| -- Thallium Chloride | | 5.00E-04 | RfD | | | |
| -- Thallium Nitrate | | 5.00E-04 | RfD | | | |
| -- Thallic Oxide | | 4.00E-04 | RfD | | | |
| -- Thallium Sulfate | | 5.00E-04 | RfD | | | |
| Thioacetamide @ | | | | | | |
| Thiourea @ | | | | | | |
| o-Tolidine @ | | | | | | |
| Toluene | 4.30E-01 | 3.00E-01 | RfD | 1.50E+00 | 1.50E+00 | HEA |
| o-Toluidine Hydrochloride @ | | | | | | |
| Toxaphene @ | | | | | | |
| Tribromomethane (Bromoform) | | | | | | |
| 1,2,4-Trichlorobenzene | | 2.00E-02 | RfD | | | |
| 1,1,1-Trichloroethane | | 5.40E-01 | HEA | 1.10E+01 | 6.30E+00 | HEA |
| 1,1,2-Trichloroethane @ | | | | | | |
| Trichloroethylene @ | | | | | | |
| Trichlorofon | | | | | | |
| Trichloromonofluoromethane | | 3.00E-01 | RfD | | | |
| 2,4,5-Trichlorophenol | 1.00E+00 | 1.00E-01 | RfD | | | |
| 2,4,6-Trichlorophenol @ | | | | | | |

Date Prepared: October 1, 1986

# EXHIBIT A-6
## (Continued)

## TOXICITY DATA FOR NONCARCINOGENIC EFFECTS -- RISK CHARACTERIZATION

| Chemical Name | Oral Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Oral Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Oral Route: Source | Inhalation Route: Acceptable Intake Subchron (AIS) --mg/kg/day-- | Inhalation Route: Acceptable Intake Chronic (AIC) --mg/kg/day-- | Inhalation Route: Source |
|---|---|---|---|---|---|---|
| 2,4,5-Trichlorophenoxyacetic Acid | | 3.00E-02 | RfD | | | |
| 1,2,3-Trichloropropane | | 1.00E-01 | RfD | | | |
| 1,1,2-Trichloro-1,2,2-Trifluoroethane | | 3.00E+01 | RfD | | | |
| Tris(2,3-dibromopropyl)phosphate @ | | | | | | |
| Trinitrotoluene (TNT) | | 2.00E-04 | RfD | | | |
| Trypan Blue @ | | | | | | |
| Uracil Mustard @ | | | | | | |
| Uranium and Compounds | | | | | | |
| Urethane @ | | | | | | |
| Vanadium and Compounds | | 2.00E-02 | RfD | | | |
| Vinyl Chloride @ | | | | | | |
| Warfarin | | 3.00E-04 | RfD | | | |
| o-Xylene | 1.00E-01 | 1.00E-02 | HEA | 9.6E-1(T) | 2.00E-01 | HEA |
| m-Xylene | 1.00E-01 | 1.00E-02 | HEA | 1.00E+00 | 2.00E-01 | HEA |
| p-Xylene | | | | | | |
| Xylenes (mixed) | 1.00E-01 | 1.00E-02 | HEA | 6.9E-1(T) | 4.00E-01 | HEA |
| Zinc and Compounds | 2.10E-01 | 2.10E-01 | HEA | 1.00E-01 | 1.00E-02 | HEA |
| -- Zinc Phosphide | | 3.00E-04 | RfD | | | |
| Zineb | | 5.00E-02 | RfD | | | |

@ Potential carcinogenic effects also. See Exhibits A-3 and A-4.

[1] Refer to Exhibit A-5 for toxicity data for indicator selection for the chemicals listed here.

[2] Sources for Exhibit A-6:

RfD = Agency-wide reference dose value, developed by an inter-office work group chaired by the Office of Research and Development, U.S. EPA, Washington, D.C., 1986.

HEA = Health Effects Assessment document, prepared by the Environmental Criteria and Assessment Office, U.S. EPA, Cincinnati, Ohio, 1985 (updated in May 1986).

[3] The RfD values listed here are EPA-verified numbers. All RfD values were derived based on oral exposure; however, in the absence of other more specific data, these values may also be useful in assessing risks of inhalation exposure.

[4] T indicates that teratogenic or fetotoxic effects are the basis for the AIS value listed.

[5] N.O.S. = not otherwise specified.

## EXHIBIT A-7

## CHEMICALS AND CHEMICAL GROUPS HAVING EPA HEALTH EFFECTS ASSESSMENT (HEA) DOCUMENTS [1]

| CHEMICAL | NTIS[2] PB NUMBER |
|---|---|
| Acetone | 86 134277/AS |
| Arsenic and Compounds | 86 134319/AS |
| Asbestos | 86 134608/AS |
| Barium and Compounds | 86 134327/AS |
| Benzene | 86 134483/AS |
| Benzo(a)pyrene | 86 134335/AS |
| Cadmium and Compounds | 86 134491/AS |
| Carbon Tetrachloride | 86 134509/AS |
| Chlordane | 86 134343/AS |
| Chlorobenzene | 86 134517/AS |
| Chloroform | 86 134210/AS |
| Chromium III and Compounds | 86 134467/AS |
| Chromium VI and Compounds | 86 134301/AS |
| Coal Tars | 86 134350/AS |
| Copper and Compounds | 86 134368/AS |
| Cresol | 86 134616/AS |
| Cyanides | 86 134228/AS |
| DDT | 86 134376/AS |
| 1,1-Dichloroethane | 86 134384/AS |
| 1,2-Dichloroethane (EDC) | 86 134137/AS |
| 1,1-Dichloroethylene | 86 134624/AS |
| 1,2-cis-Dichloroethylene | 86 134269/AS |
| 1,2-trans-Dichloroethylene | 86 134525/AS |
| Dichloromethane | 86 134392/AS |
| Ethylbenzene | 86 134194/AS |
| Glycol Ethers | 86 134632/AS |
| Hexachlorobenzene | 86 134285/AS |
| Hexachlorobutadiene | 86 134640/AS |
| Hexachlorocyclopentadiene | 86 134129/AS |
| gamma-Hexachlorocyclohexane (Lindane) | 86 134673/AS |
| Iron and Compounds | 86 134657/AS |
| Lead and Compounds (Inorganic) | 86 134665/AS |
| Manganese and Compounds | 86 134681/AS |
| Mercury | 86 134533/AS |
| Methyl Ethyl Ketone | 86 134145/AS |
| Naphthalene | 86 134251/AS |
| Nickel and Compounds | 86 134293/AS |
| Pentachlorophenol | 86 134541/AS |
| Phenanthrene | 86 134400/AS |
| Phenol | 86 134186/AS |
| Polychlorinated Biphenyls (PCBs) | 86 134152/AS |

EXHIBIT A-7
(Continued)

## CHEMICALS AND CHEMICAL GROUPS HAVING EPA HEALTH EFFECTS ASSESSMENT (HEA) DOCUMENTS [1]

| CHEMICAL | NTIS[2] PB NUMBER |
|---|---|
| Polynuclear Aromatic Hydrocarbons | 86 134244/AS |
| Pyrene | 86 134418/AS |
| Selenium and Compounds | 86 134699/AS |
| Sodium Cyanide | 86 134236/AS |
| Sulfuric Acid | 86 134426/AS |
| 2,3,7,8-TCDD (Dioxin) | 86 134558/AS |
| 1,1,2,2-Tetrachloroethane | 86 134434/AS |
| Tetrachloroethylene | 86 134202/AS |
| Toluene | 86 134442/AS |
| 1,1,1-Trichloroethane | 86 134160/AS |
| 1,1,2-Trichloroethane | 86 134566/AS |
| Trichloroethylene | 86 134574/AS |
| 2,4,5-Trichlorophenol | 86 134459/AS |
| 2,4,6-Trichlorophenol | 86 134582/AS |
| Vinyl Chloride | 86 134475/AS |
| Xylene | 86 134178/AS |
| Zinc and Compounds | 86 134590/AS |
| Complete Set of 58 HEAs | 86 134111/AS |

[1] As of the date of publication for this manual.

[2] National Technical Information Service.

# APPENDIX B

## DETAILED PROCEDURES FOR DETERMINING TOXICITY CONSTANTS FOR INDICATOR CHEMICAL SELECTION

The method for selecting indicator chemicals for a site, described in Chapter 3 of this manual, requires the determination of toxicity constants (T). For many chemicals, these values are given in Appendix A. This appendix (Appendix B) presents methods for calculating toxicity constants for chemicals not listed in Appendix A. If, in the process of preparing a public health evaluation for a site, such chemicals are found, you should request help from EPA headquarters before doing these calculations. As new information becomes available or new chemicals are identified as problems, the list in Appendix A will be updated and expanded.

Toxicity constants, T, are medium-specific. A toxicity constant for use with drinking water concentrations is referred to as ${}^{w}T$, whereas one for concentrations in air is ${}^{a}T$, and and one for concentrations in soil is ${}^{s}T$. Toxicity constants for potential carcinogens are based on the $ED_{10}$[1]; for noncarcinogens they are based on the minimum effective dose (MED) and a severity of effects rating. All toxicity constants also have standard intake assumptions built in. Units of toxicity constants are the inverse of concentration units.

Values of ${}^{a}T$, ${}^{s}T$, and ${}^{w}T$ for a variety of compounds are given in Appendix A. In the event that values are not present in Appendix A, they can be calculated as follows:

<u>Potential Carcinogens</u>

$$ {}^{w}Tc = \frac{2 \text{ liters drinking water/day}}{70 \text{ kg} \cdot ED_{10}} \qquad [1] $$

$$ {}^{s}Tc = \frac{0.0001 \text{ kg soil/day}}{70 \text{ kg} \cdot ED_{10}} \qquad [2] $$

$$ {}^{a}Tc = \frac{20 \text{ m}^3 \text{ air/day}}{70 \text{ kg} \cdot ED_{10}} \qquad [3] $$

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[1] $ED_{10}$ = dose in mg/kg/day at which 10% incidence above control is observed for a tumor type showing a statistically significant incidence.

where the $ED_{10}$ is derived from carcinogenicity dose-response data and is expressed in mg/kg/day.

Noncarcinogens

$$^{w}Tn = \frac{2 \text{ liters drinking water/day} \bullet RVe}{MED \text{ (oral)}} \quad [4]$$

$$^{s}Tn = \frac{0.0001 \text{ kg soil/day} \bullet RVe}{MED \text{ (oral)}} \quad [5]$$

$$^{a}Tn = \frac{20 \text{ m}^3 \text{ air/day} \bullet RVe}{MED \text{ (inhalation)}} \quad [6]$$

where RVe is a rating value based on the severity of effect and scored as indicated in Exhibit B-1, and MED is the human minimum effective dose in mg/day for a given effect. If the MED is given in mg/kg/day, multiply it by 70 and then substitute it into the above equation.

The soil toxicity constant ($^{s}T$) is incorporated as a way to estimate the overall exposure that might be contributed by contaminated soil. Inclusion of $^{s}T$ in the indicator selection process is a way to use the soil concentration data gathered in most site characterizations, in part so that compounds found in soil and not in air and water could be considered in indicator compound scoring. The $^{s}T$ equation is based on a child's consumption of contaminated soil as detailed in a recent ORD risk assessment of contaminated soil (EPA, 1984).

The ORD document estimates that children between the ages of two and six consume at least 100 mg of soil per day, and that in situations of direct ingestion of soil (i.e., pica) the rate could go as high as 5 g per day. The lower value was selected for this procedure because it was more comparable to the standard consumption values used in calculating the other T values. The 5 g per day value is representative of a pathologic state (pica), and using it to calculate $^{s}T$ would correspond to assuming 8 liters or more as the daily consumption of water (to reflect the diabetic who consumes 8 liters of water per day).

Although Equations 2 and 5 are based on ingestion by a child, the intake is not normalized to an equivalent lifetime intake. The equations use an intake rate during childhood rather than an lifetime average daily intake to ensure that compounds are identified on the basis of their potential to harm a child. Thus, the equations compare a child's daily intake rate to a lifetime average daily intake (expressed as an MED or an $ED_{10}$), which, strictly speaking, may be inappropriate. Unfortunately, the most appropriate data to use, dose-response information for children, do not exist, and even data for dose-response relationships in immature animals are rare. What little

## EXHIBIT B-1

## RATING CONSTANTS (RVe) FOR NONCARCINOGENS[1]

| Effect | Severity Rating (RVe) |
|---|---|
| Enzyme induction or other biochemical change with no pathologic changes and no change in organ weights. | 1 |
| Enzyme induction and subcellular proliferation or other changes in organelles but no other apparent effects. | 2 |
| Hyperplasia, hypertrophy or atrophy, but no change in organ weights. | 3 |
| Hyperplasia, hypertrophy or atrophy with changes in organ weights. | 4 |
| Reversible cellular changes: cloudy swelling, hydropic change, or fatty changes. | 5 |
| Necrosis, or metaplasia with no apparent decrement of organ function. Any neuropathy without apparent behavioral, sensory, or physiologic changes. | 6 |
| Necrosis, atrophy, hypertrophy, or metaplasia with a detectable decrement of organ functions. Any neuropathy with a measurable change in behavioral, sensory, or physiologic activity. | 7 |
| Necrosis, atrophy, hypertrophy, or metaplasia with definitive organ dysfunction. Any neuropathy with gross changes in behavior, sensory, or motor performance. Any decrease in reproductive capacity, any evidence of fetotoxicity. | 8 |
| Pronounced pathologic changes with severe organ dysfunction. Any neuropathy with loss of behavioral or motor control or loss of sensory ability. Reproductive dysfunction. Any teratogenic effect with maternal toxicity. | 9 |
| Death or pronounced life-shortening. Any teratogenic effect without signs of maternal toxicity. | 10 |

[1] Rating scale identical to that used by EPA in the RQ adjustment process, as described in EPA (1983).

information is available seems to indicate that the young are generally more sensitive to the toxic effects of chemicals than adults. Although this approach is not strictly accurate it errs on the more protective side, while at the same time achieving the goal of being a simple way to incorporate soil concentration information into the indicator selection process.

Although not used directly in the calculation of indicator scores for potential carcinogens, a qualitative weight-of-evidence rating is considered in the final selection of indicators. The EPA weight-of-evidence criteria (EPA, 1986) are given in Exhibit B-2 and should be used to categorize potential carcinogens not listed in Appendix A. The EPA approach for determining weight of evidence is similar to the International Agency for Research on Cancer (IARC) approach, differing primarily by having an additional category for "no evidence of carcinogenicity in humans" and revised criteria for defining evidence as "sufficient", "limited", or "inadequate."

## REFERENCES FOR APPENDIX B

U.S. EPA, 1983. Methodology and Guidelines for Reportable Quantity Determinations Based on Chronic Toxicity Data, External Review Draft. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment. ECAO-CIN-R245.

U.S. EPA, 1986. Guidelines for Carcinogen Risk Assessment. Federal Register 51:33992.

U.S. EPA, 1984. Risk Analysis of TCDD Contaminated Soil. Prepared by the Exposure Assessment Group, Office of Health and Environmental Assessment. EPA 600/8-84-031.

## EXHIBIT B-2

## EPA WEIGHT-OF-EVIDENCE CATEGORIES FOR POTENTIAL CARCINOGENS

| EPA Category | Description of Group | Description of Evidence |
|---|---|---|
| Group A | Human Carcinogen | Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer |
| Group B1 | Probable Human Carcinogen | Limited evidence of carcinogenicity in humans from epidemiologic studies |
| Group B2 | Probable Human Carcinogen | Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans |
| Group C | Possible Human Carcinogen | Limited evidence of carcinogenicity in animals |
| Group D | Not Classified | Inadequate evidence of carcinogenicity in animals |
| Group E | No Evidence of Carcinogenicity in Humans | No evidence for carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies |

# APPENDIX C

## MEMORANDUM OF UNDERSTANDING
## BETWEEN
## THE AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY
## AND
## THE UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

April 2, 1985

### 1. PURPOSE

The Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA) agree that guidance is required to define and coordinate joint and respective responsibilities under the Comprehensive Environmental Response, Compensation, and Liability Act (Public Law 96-510, 94 Stat. 2796, 42 USC 9601 et seq; CERCLA), Executive Order 12316 (Responses to Environmental Damage), and the National Oil and Hazardous Substances Contingency Plan (NCP; 40 CFR Part 300). This Memorandum of Understanding (MOU) establishes policies and procedures for conducting response and non-response health activities related to releases of hazardous substances.

### 2. AUTHORITY

CERCLA section 104 authorizes the President to respond to releases or substantial threats of releases into the environment of hazardous substances and certain releases of pollutants or contaminants. CERCLA also establishes the Hazardous Substance Response Trust Fund. CERCLA section 104(i) authorizes ATSDR (part of the Department of Health and Human Services (HHS)) to effectuate and implement specific health-related activities with the cooperation of EPA and other agencies. Executive Order 12316 further delegates to the Secretary of HHS certain investigatory authorities vested in the President under CERCLA section 104 for conducting activities with the cooperation of other agencies, relating to illness, disease or complaints thereof. Executive Order 12316 delegates to EPA the primary response authority under CERCLA section 104 relating to release or extent of release of hazardous substances, pollutants, or contaminants, and determination of the presence of an imminent and substantial danger to the public health or welfare or the environment. Exceptions to this authority include responses to releases from Department of Defense (DOD) facilities or vessels (delegated to DOD) and releases involving the coastal zone, Great Lakes waters, ports, and harbors (delegated to the U.S. Coast Guard).

## 3. SCOPE OF RESPONSIBILITIES

This MOU covers the coordination of health-related activities by ATSDR and EPA as authorized by CERCLA and delegated by Executive Order 12316. ATSDR has statutory responsibilities under CERCLA and Executive Order 12316 for activities related to illness, disease, or complaints thereof, for disease registries and other responsibilities related to response actions. EPA has statutory authority under CERCLA and Executive Order 12316 for activities related to release or threat of release of hazardous substances, pollutants or contaminants, and for determination of the extent of danger to public health, welfare or the environment, as well as, other responsibilities related to response actions.

ATSDR and EPA will carry out their responsibilities according to CERCLA, Executive Order 12316, the NCP, and this MOU. ATSDR's major responsibility will be the evaluation of populations with current or potential exposure to waste sites, development of health advisories, and the follow up on populations for the evaluation of future health effects. EPA's major responsibility in the health area will be risk assessment and risk management as defined herein. Health advisories will be based on ATSDR's evaluations of current health effects and will adapt EPA's risk assessments at a site or sites. ATSDR will not perform risk assessments as defined herein, using the funds made available from the Hazardous Substances Response Trust Fund. If risk assessments are not available ATSDR will consult EPA on a case-by-case basis. ATSDR will conduct some of its activities through interagency agreements with other participating agencies of the Public Health Service through cooperative agreements with State health departments, and through contractual arrangements whenever appropriate. Such interagency agreements include those with the Centers for Disease Control to conduct health studies and conduct research and provide assistance on worker health and safety issues; with the Library of Medicine to establish and maintain the needed data bases on health effects of toxic substances; and with the National Toxicology Program to conduct standard toxicological assays.

Definitions for the key terms used in this section follow:

- <u>Health Consultation</u>: Immediate or short-term consultation by ATSDR to provide health advice and/or health effects information regarding a specific site.

- <u>Health Assessment</u>: Initial multi-disciplinary reviews by ATSDR of all readily available data to evaluate

the nature and magnitude of any threat to human health at a site. These evaluations will adapt EPA's risk assessment for the characterization of potential health threats at a site or sites, and may include literature searches, information summarization and evaluation of existing environmental data, pilot samples, testing for food chain contamination, and similar activities.

- Public Health Advisory: An advisory issued by ATSDR based on the results of its health assessment.

- Epidemiologic Studies: Long-term epidemiologic study by ATSDR involving a comprehensive protocol designed to add knowledge of the health effects of a specific substance or substances at a site or sites.

- Health Registry: A site-specific or adverse health effects-specific registry established and maintained to track specific diseases and illnesses and long-term health effects to persons exposed to toxic substances.

- Pilot Study: A preliminary or short term medical, laboratory, or epidemiologic study on a limited human population to decide if additional, large scale studies are warranted. The study populations can include those living at, or near, a site and those not residing at, or near, a site (control or reference population).

- Risk Assessment: A qualitative/quantitative process conducted by EPA to characterize the nature and magnitude of potential risks to public health from exposure to hazardous substances, pollutants or contaminants released from specific sites. This process consists of hazard identification, dose-response assessment, exposure assessment, and risk characterization and supports EPA's risk management process.

- Risk Management: The process conducted by EPA to determine the nature and extent of remedy for a site, including alternative selection.

A. Removal Actions

Removal actions are Superfund response activities involving the short-term cleanup or removal of released hazardous substances that pose an immediate hazard. These actions generally are limited by CERCLA to $1 million in cost and six months in duration.

ATSDR activities in support of specific removal actions involve health consultations and health advisories. In addition, ATSDR may monitor the health of residents who have been exposed to the hazardous substances or who live near the release site. ATSDR may also provide technical assistance to EPA on matters of worker health and safety during the removal and may provide community relations assistance to EPA. ATSDR may become involved in removal actions through a variety of mechanisms and at various stages of a removal action. The On-Scene Coordinator (OSC) shall recommend that ATSDR be called in at any time during the removal action, at the time that the criteria under Section B.3 are met, unless in the OSC's opinion there is no need for further public health input into the removal action. Alternatively, the recommendation for ATSDR involvement may be initiated by ATSDR itself, the State, or the EPA Regional Administrator.

## B. Remedial Response

Remedial actions are those response actions consistent with a permanent remedy at a site. Remedial action is preceded by detailed planning. This section discusses coordination of ATSDR and EPA efforts during the remedial response process, which involves five major stages:

- Site discovery, preliminary assessment, and site inspection;
- Site ranking and NPL listing;
- Remedial investigation (RI);
- Feasibility study (FS); and
- Remedial design and construction.

The roles of ATSDR and EPA during these stages are discussed in the subsections below.

### B.1 Site Discovery, Preliminary Assessment, and Site Inspection

There are different methods for identifying sites for potential remedial response under the Superfund program. CERCLA section 103 requires certain parties to notify the National Response Center when they have knowledge of a release of a hazardous substance equal to or in excess of the reportable quantity for that substance. Notification is forwarded to EPA and the affected State. In addition to this formal notification process, EPA may receive notification of a potential or actual release from a local, State, or Federal agency that discovers the release in the performance of its responsibilities. Following notification of a potential or actual release, EPA conducts a preliminary assessment of the site to determine whether further investigation and Hazard Ranking System (HRS) scoring is warranted.

Site discovery, preliminary assessment, and site inspection are primarily the responsibility of EPA. If ATSDR discovers a potential or actual release during the performance of its responsibilities, ATSDR will notify EPA of this release. EPA may perform preliminary assessments and site inspections of such releases, as warranted, and will determine whether further investigation is necessary.

### B.2 Site Ranking and NPL Listing

CERCLA section 105(8) requires the President to develop criteria for determining priorities among releases or threatened releases of hazardous substances and, based upon those criteria, publish and amend the NPL. Executive Order 12316, section 1(c) delegates to EPA "[t]he responsibility for. . .all of the. . .functions vested in section 105" of CERCLA.

Decisions regarding specific site scoring and listing of sites on the NPL are the responsibility of EPA. If ATSDR discovers any information about potential candidates for the NPL during the performance of its responsibilities, ATSDR will submit that information to EPA. To facilitate this, EPA Headquarters will notify ATSDR prior to each amendment of the NPL to allow ATSDR to recommend sites to be considered for the NPL, and EPA will consider such recommendations, based upon the data used by ATSDR to make the recommendation, before publishing the amended NPL. EPA may decide to rank sites identified by ATSDR, retain the site information on EPA files for future reference, or seek further information about such sites, and will notify ATSDR of its decision.

### B.3 Remedial Investigation

CERCLA section 104(b) authorizes the President to undertake "such investigations, monitoring, surveys, testing, and other information gathering" necessary to "identify the existence and extent of the release or threat thereof, the source and nature of hazardous substances, pollutants or contaminants involved, and the extent of danger to public health or welfare or the environment." Section 2(a) of Executive Order 12316 delegates to the Secretary of HHS in cooperation with other agencies, those functions of Section 104(b) "relating to illness, disease, or complaints thereof." HHS's responsibilities are performed by ATSDR. Section 2(e) delegates to EPA most of the remaining authorities under section 104, including those functions under section 104(b) listed above as they relate to the occurrence or potential occurrence of a release.

The EPA Regional Administrator, or his designee, will determine as early as possible in the RI/FS process for a site whether concurrent ATSDR involvement in the RI/FS is

necessary. In deciding whether to request concurrent ATSDR involvement, the Regional Administrator, or his designee, will consider the following criteria:

- Whether the presence of toxic substances has been confirmed at the site;

- Whether pathways of human exposure to toxic substances have been demonstrated to exist at the site, especially if such pathways involve direct contact with toxic substances; and

- Whether a human population has been exposed to toxic substances via the identified pathways, and whether there exists a threat of current or future health effects to the population being so exposed, after considering EPA's risk assessments or health effects information from other sources.

If these criteria are met, the EPA Regional Administrator, or his designee, shall request concurrent ATSDR involvement, unless in his opinion there is no need for further public health input into the RI/FS. Alternatively, the recommendation for ATSDR involvement may be initated by ATSDR itself, or the State.

Elements of the remedial investigation in which ATSDR participates may include review of site sampling plans and analysis protocols, site sampling, data analysis and interpretation, worker health and safety, community relations, and the remedial investigation report. The division of responsibilities and coordination between EPA and ATSDR in conducting these activities is described in the following paragraphs. EPA and ATSDR will agree to strict time schedules on a site-specific basis for all activities to be performed by ATSDR, to ensure that the response process is not delayed. Any changes in the time schedule will be mutually agreed upon by EPA and ATSDR.

Site Sampling. Where EPA has requested concurrent ATSDR involvement, ATSDR will advise EPA during the preparation of sampling and analysis protocols to ensure collection of data useful to ATSDR for health assessments and epidemiological studies. EPA will be responsible for the development and conduct of any environmental and biological (other than human) sampling, and developing the tests therefor. ATSDR will consult with appropriate health agencies and will summarize recommendations regarding the necessity for testing of human subjects. If human subject testing is determined to be necessary, ATSDR will be responsible for any such testing. EPA shall review the protocols or sampling plans for such testing to ensure collection of data useful to EPA in performing subsequent risk assessment and risk management.

Sampling Protocol. Where EPA has requested concurrent ATSDR involvement, EPA and ATSDR will submit a draft of all protocols to each other for review prior to institution of any site sampling or monitoring. Any changes in the sampling protocols will also be provided to ATSDR for review. With regard to the review of non-site specific protocols, (e.g., protocols for standard Contract Laboratory Program analysis) EPA will provide these to ATSDR for review as early as possible to avoid the necessity of ATSDR review of these protocols on a site specific basis.

Data Analysis and Interpretation. At sites where EPA has requested concurrent ATSDR involvement, EPA will provide its data from environmental, toxicological and other biological sampling and testing to ATSDR. ATSDR will review all available data for a site, including EPA's hazard identification, dose-response assessment, exposure assessment, and risk characterization information, drawing conclusions about any threats to public health associated with the site. Based on its interpretation of the site data, ATSDR will characterize the health threats based on its evaluation of current health effects and in consultation with EPA concerning the magnitude and timing of potential future health effects. ATSDR will communicate all health concerns to regional EPA staff and will provide copies of health assessments and advisories to EPA.

Worker Health and Safety. EPA may request assistance from ATSDR on worker health and safety issues during a remedial investigation, including consultation on the design of worker health and safety plans and monitoring of plan implementation. ATSDR will make arrangements for laboratory and field testing related to worker health and safety and worker surveillance.

Community Relations. ATSDR may provide, at EPA's request, assistance in conducting community relations activities during the remedial investigation. Such assistance may include:

- Preparation of technical and non-technical information material for the public describing human health threats posed by substances at a site;
- Reviewing and commenting on human health-related documents prepared and submitted by citizens (e.g., citizen-generated health survey protocols);
- Participation in public meetings, small group meetings, and workshops; and
- Preparing responses to specific public inquiries regarding human health impacts of site problems.

Remedial Investigation Report. At the conclusion of the remedial investigation at sites where ATSDR is involved, EPA will send a copy of the remedial investigation report to ATSDR. ATSDR will review health-related data and interpretations of such data in the report and provide comments to EPA within a mutually agreed upon time frame.

If EPA and ATSDR agree that ATSDR involvement is not required at a site, ATSDR will not participate in the remedial planning process at that site. ATSDR may undertake other statutory activities, such as epidemiological studies or disease registries, at a site or sites. ATSDR will coordinate all such activities with EPA and will advise EPA of imminent threats to human health at any site and at any time during EPA's remedial process. In addition, EPA may request ATSDR assistance in disseminating health information to the public and in responding to health concerns of local citizens.

B.4 Feasibility Study

EPA has the final authority for determining the extent of remedy at a site and selecting a specific remedy during the feasibility study. In conducting feasibility studies, EPA will develop, evaluate, and select remedial options using the approach described in its feasibility study guidance. For those sites where there has been concurrent ATSDR involvement, EPA staff will consult ATSDR for its assessment of any human health data (e.g., clinical, epidemiologic) and EPA's risk assessment resulting from the remedial investigation. EPA will be responsible for performing qualitative/quantitative risk assessments evaluating long-term risks to the public that may result from exposure to hazardous substances from Superfund sites.

It is the responsibility of EPA (Office of Solid Waste and Emergency Response) to incorporate the results of the risk assessment process and of health assessments by ATSDR into risk management determinations of the extent of remedy for a site. The goal of this process is to ensure that the remedial action is adequate with regard to eliminating or mitigating the existing and future public health threats. EPA may consider and incorporate applicable information provided by ATSDR on the current status of public health at the site into the selection of the preferred remedy. At the discretion of the appropriate Regional Administrator, EPA staff may also consult with ATSDR staff for any interpretation of human health data at sites where ATSDR is not concurrently involved. In addition, EPA may request ATSDR assistance at any site in disseminating health information to the public and in responding to health concerns of local citizens. In the course of performing its health activities, should ATSDR discover any site which, in its opinion, poses

an imminent threat to public health, ATSDR will immediately notify the relevant EPA Regional Office and EPA Headquarters of this finding.

For each remedial response site where ATSDR involvement is requested , EPA will provide ATSDR with a copy of the draft feasibility study, and where appropriate with rough draft sections of the feasibility study relating to human health and interpretation, prior to the public comment period if possible. ATSDR will review the interpretation of the human health data in the draft feasibility study and provide comments to EPA during the public comment period. ATSDR will also provide to EPA any health information it possesses on the site during the public comment period

B.5 Remedial Design and Construction

The design and construction of the selected remedy at Superfund sites is EPA's responsibility. The Regional Administrator may, at his discretion, request a health assessment from ATSDR with regard to certain elements of the remedial design. At the conclusion of the design stage, EPA should provide advance copies of the Remedial Design and Construction Plans to ATSDR whenever possible if they wish review and comment by ATSDR. ATSDR will notify EPA if the remedial design does not, in its opinion, eliminate or mitigate the public health threat.

C. Cost Recovery

Under CERCLA, EPA is authorized to recover from responsible parties all government costs incurred during a response action. ATSDR agrees to conform with all procedures and requirements for documenting costs that are to be recovered.

D. Funding

All costs incurred by ATSDR in performing its CERCLA responsibilities are funded by ATSDR through funds provided for this purpose. Funding for ATSDR activities performed under CERCLA is from the Hazardous Substances Response Trust Fund and is provided by EPA through the budget task force required by Section 7 of Executive Order 12316 or through separate interagency agreements for specific health studies. ATSDR will comply with the financial and reporting requirements outlined in the Interagency Agreements that transfer Fund monies to ATSDR.

4. PERIOD OF AGREEMENT

This Memorandum of Understanding will continue in effect until modified or amended by the assent of both parties or terminated by either party upon a thirty (30) day advance

written notice of the other party. Nothing in the Memorandum is intended to diminish or otherwise alter statutory authority of the agencies involved.

5. AMENDMENTS

This Memorandum may be amended at any time by the agreement of both parties. Each amendment must be in writing and signed by the appropriate ATSDR and EPA officials.

6. EFFECTIVE DATE

This Memorandum will become effective at noon on the date of the last signature below.

Date: MAY 29 1985

For the Agency for Toxic Substances and Disease Registry

Date: 4-25-85

For the United States Environmental Protection Agency

## *GLOSSARY*

## LIST OF ACRONYMS

| Acronym | Meaning |
|---|---|
| ACL | Alternate Concentration Limit |
| ADI | Acceptable Daily Intake |
| AIC | Acceptable Intake for Chronic Exposures |
| AIS | Acceptable Intake for Subchronic Exposures |
| ARAR | Applicable or Relevant and Appropriate Requirement |
| ATSDR | Agency for Toxic Substances and Disease Registry |
| CAG | Carcinogen Assessment Group, U.S. EPA |
| CERCLA | Comprehensive Environmental Response, Compensation, and Liability Act |
| CDI | Chronic Daily Intake |
| ECAO | Environmental Criteria and Assessment Office, U.S. EPA |
| $ED_{10}$ | Ten Percent Effective Dose |
| FRDS | Federal Reporting Data System |
| FS | Feasibility Study |
| HEA | Health Effects Assessment |
| HRS | Hazard Ranking System |
| IARC | International Agency for Research on Cancer |
| IS | Indicator Score |
| $LD_{50}$ | Median Lethal Dose |
| LTC | Long-term Concentration |
| MCL | Maximum Contaminant Level |
| MCLG | Maximum Contaminant Level Goal |
| MED | Minimum Effective Dose |
| MOU | Memorandum of Understanding |
| NAAQS | National Ambient Air Quality Standards |
| NC | Noncarcinogen |
| NCP | National Oil and Hazardous Substances Pollution Contingency Plan |
| NOAA | National Oceanic and Atmospheric Administration |
| NOAEL | No Observed Adverse Effect Level |
| NPL | National Priorities List |
| OERR | Office of Emergency and Remedial Response, U.S. EPA |
| OHEA | Office of Health Effects Assessment, U.S. EPA |
| ORD | Office of Research and Development, U.S. EPA |
| OSWER | Office of Solid Waste and Emergency Response, U.S. EPA |
| PC | Potential Carcinogen |
| PHE | Public Health Evaluation |
| PHRED | Public Health Risk Evaluation Database |
| QA/QC | Quality Assurance/Quality Control |
| RCRA | Resource Conservation and Recovery Act |
| RfD | Reference Dose |
| RI | Remedial Investigation |
| RMCL | Recommended Maximum Contaminant Level |
| SDI | Subchronic Daily Intake |
| SDWA | Safe Drinking Water Act |
| SEAM | Superfund Exposure Assessment Manual |
| SPHEM | Superfund Public Health Evaluation Manual |
| STC | Short-term Concentration |
| WQC | Water Quality Criteria |

## DEFINITIONS OF TERMS DEVELOPED SPECIFICALLY FOR THE SUPERFUND PUBLIC HEALTH EVALUATION PROCESS

| Acronym | Definition |
|---|---|
| STC | **Short-Term Concentration.** The projected chemical concentration in an exposure medium averaged over a short time period (10 to 90 days). The peak STC (i.e., highest one projected over the entire evaluation period, usually 70 years) is used for subchronic risk characterization. Unless otherwise stated, the STC refers to a best estimate concentration value, not an upper bound estimate. |
| LTC | **Long-Term Concentration.** The projected chemical concentration at an exposure point averaged over a long time period, up to 70 years (assumed to be a human lifetime). The LTC for the 70-year period beginning with the date of the RI/FS is used for carcinogenic risk characterization. Unless otherwise stated, the LTC refers to a best estimate concentration value, not an upper bound estimate |
| SDI | **Subchronic Daily Intake.** The projected human intake of a chemical averaged over a short time period, expressed as mg/kg/day. The SDI is calculated by multiplying peak STC by human intake and body weight factors and is used for subchronic risk characterization. |
| CDI | **Chronic Daily Intake** The projected human intake of a chemical averaged over a long time period, up to 70 years, and expressed as mg/kg/day. The CDI is calculated by multiplying LTC by human intake and body weight factors and is used for chronic risk characterization. |
| AIS | **Acceptable Intake for Subchronic Exposure.** The highest human intake of a chemical, expressed as mg/kg/day, that does not cause adverse effects when exposure is short-term (but not acute). The AIS is usually based on subchronic animal studies. |
| AIC | **Acceptable Intake for Chronic Exposure.** The highest human intake of a chemical, expressed as mg/kg/day, that does not cause adverse effects when exposure is long-term (lifetime). The AIC is usually based on chronic animal studies. |
| IS | **Indicator Score.** A unitless score that is the product of a media-specific concentration of a chemical and the media-specific toxicity constant for that chemical. The indicator score is one of the factors considered in the selection of indicator chemicals. |



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