# A Probabilistic Risk Assessment for Children Who Contact CCA-Treated Playsets and Decks

**Draft Final Report**

**November 10, 2003**

## Prepared by:

**W. Dang, J. Chen**

**U. S. Environmental Protection Agency**
**Office of Pesticide Programs, Antimicrobials Division**

**and**

**N. Mottl, L. Phillips, P. Wood, S. McCarthy,**
**R. Lee, M. Helmke, M. Nelson, and K. Coon**
**Versar, Inc.**

**DISCLAIMER**
This report has undergone internal EPA review through the Office of Research and Development (ORD) and the Office of Pesticide Programs (OPP). Some of the statutory provisions described in this report contain legally binding requirements. However, this report does not substitute for those provisions or regulations, nor is it regulation itself. Any decisions regarding a particular risk reduction process and remedy selection decision will be made based on the statute and regulation, and EPA decision makers retain the discretion to adopt approaches on a case-by-case basis.

**TABLE OF CONTENTS**

1.0 EXECUTIVE SUMMARY . . . . . 1-1

2.0 INTRODUCTION AND BACKGROUND . . . . . 2-1

2.1 Introduction . . . . . 2-2
2.2 Background . . . . . 2-2
2.2.1 Regulatory History of CCA . . . . . 2-2
2.2.2 Current Development of CCA Issue . . . . . 2-4
2.2.2.1 CPSC Activities . . . . . 2-5
2.2.2.2 Updated International Actions and Activities . . . . . 2-6
2.2.2.3 Updated State Actions and Activities . . . . . 2-8
2.2.3 Use Profile of CCA . . . . . 2-11
2.2.4 Overview of CCA Chemistry . . . . . 2-12
2.2.4.1 Speciation . . . . . 2-13
2.2.4.2 Fixation . . . . . 2-13
2.2.4.3 Leaching . . . . . 2-14
2.2.4.4 Environmental Fate . . . . . 2-18
2.2.5 CCA Use and Potential Exposures to Components of CCA . . . . . 2-19
2.2.6 Probabilistic Risk Assessment versus Deterministic Risk Assessment . . . . . 2-20
2.2.7 EPA and OPP Regulatory Approach to PRA . . . . . 2-22

3.0 EXPOSURE ASSESSMENT . . . . . 3-1

4.0 HAZARD ASSESSMENT . . . . . 4-1

4.1 Arsenic . . . . . 4-2
4.2 Chromium . . . . . 4-3
4.3 Summary Tables . . . . . 4-4
4.4 Early-Life Exposures . . . . . 4-5
4.5 Relative Bioavailability . . . . . 4-6
4.6 Dermal Absorption . . . . . 4-7

5.0 RISK CHARACTERIZATION . . . . . 5-1

5.1 Introduction . . . . . 5-1
5.2 Results . . . . . 5-3
5.2.1 Noncancer Effects . . . . . 5-4
5.2.2 Carcinogenic Effects . . . . . 5-22
5.3 Risk Reduction Assuming 0.01% Dermal Absorption Rate . . . . . 5-28
5.4 Summary . . . . . 5-37

**TABLE OF CONTENTS (CONTINUED)**

6.0 RISK REDUCTION IMPACTS . . . . . 6-1

6.1 Introduction . . . . . 6-1
6.1.1 Application of Sealant and Hand Washing Information to SHEDS-Wood . . . . 6-2
6.2 Risk Characterization for Mitigation Measures . . . . . 6-5
6.2.1 Summary of Results . . . . . 6-7
6.2.2 Risk Reduction Through the Use of Sealants . . . . . 6-8
6.2.3 Risk Reduction Through Hand Washing . . . . . 6-14
6.2.4 Risk Reduction Through Use of Sealants and Hand Washing . . . . . 6-17
6.3 Comparison of Residue and Soil Risks . . . . . 6-18
6.4 Summary . . . . . 6-24

7.0 UNCERTAINTY IN THE RISK ASSESSMENT . . . . . 7-1

7.1 Environmental Media Sampling and Analysis . . . . . 7-1
7.2 Chemical Fate . . . . . 7-3
7.3 Toxicity Data . . . . . 7-4
7.4 Exposure Assessment . . . . . 7-5
7.5 Risk Characterization . . . . . 7-6

8.0 REFERENCES . . . . . 8-1

## LIST OF TABLES


Table 1-1. Definitions of Key Terms Used in the SHEDS-Wood Risk Assessment . . . . . 1-2
Table 1-2. Summary of Risk Assessment Results . . . . . 1-6
Table 1-3. Arsenic Cancer Risks . . . . . 1-6
Table 1-4. Summary of Arsenic Risks Assuming Different Mitigation Measures for Warm Climate Conditions . . . . . 1-8
Table 2-1. International Regulatory Actions and Activities Related to CCA . . . . . 2-7
Table 2-2. State Regulatory Actions and Activities Related to CCA . . . . . 2-10
Table 2-3. Comparison of Deterministic and Probabilistic Risk Assessments . . . . . 2-20
Table 3-1. Arsenic ADDs (mg/kg/day) - Playsets and Decks . . . . . 3-3
Table 3-2. Chromium(VI) ADDs (mg/kg/day) - Playsets and Decks . . . . . 3-4
Table 3-3. Arsenic ADDs (mg/kg/day) - Playsets Only . . . . . 3-4
Table 3-4. Chromium(VI) ADDs (mg/kg/day) - Playsets Only . . . . . 3-5
Table 3-5. Arsenic ADDs (mg/kg/day) - Pica Ingestion . . . . . 3-5
Table 3-6. Arsenic LADDs (mg/kg/day) . . . . . 3-5
Table 3-7. Arsenic LADDs (mg/kg/day) - Mitigation with Sealant . . . . . 3-6
Table 3-8. Arsenic LADDs (mg/kg/day) - Mitigation with Hand Washing . . . . . 3-6
Table 3-9. Arsenic LADDs (mg/kg/day) - Mitigation with Hand Washing and Sealant . . . 3-7
Table 3-10. Arsenic LADDs (mg/kg/day) - Using 0.01% Dermal Absorption . . . . . 3-7
Table 4-1. Toxicological Endpoints for Assessing Exposures/Risks to Arsenic (V) . . . . . 4-5
Table 4-2. Toxicological Endpoints for Assessing Exposures/Risks to Chromium (VI) . . 4-5
Table 5-1. Summary of Risk Assessment Results . . . . . 5-4
Table 5-2. Arsenic Noncancer MOEs - Playset Only . . . . . 5-6
Table 5-3. Chromium (Cr(VI)) Noncancer MOEs - Playset Only . . . . . 5-6
Table 5-4. Arsenic Noncancer MOEs - Playset and Deck . . . . . 5-7
Table 5-5. Chromium (Cr(VI)) Noncancer MOEs - Playset and Deck . . . . . 5-7
Table 5-6. Probabilistic Short-Term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Based on Short-term ADDs in Table 18 From SHEDS-Wood Document) . . . . . 5-10
Table 5-7. Probabilistic Short-term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climates (Based on Short-Term ADDs in Table 19 From SHEDS-Wood Document) . . . . . 5-11
Table 5-8. Probabilistic Short-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Warm Climate (Soil Ingestion Only) . . . . . 5-12
Table 5-9. Probabilistic Short-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Cold Climate (Soil Ingestion Only) . . . . . 5-13
Table 5-10. Arsenic Noncancer Short-Term MOEs for Pica Children in Warm Climate (Based on Short-Term ADDs from Table 33 in SHEDS-Wood) . . . . 5-15

**LIST OF TABLES (CONTINUED)**

Table 5-11. Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Based on the ADDs in Table 16 From The SHEDS-Wood Document) . . . . . 5-18
Table 5-12. Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climates (Based on ADDs in Table 17 From the SHEDS-Wood Document) . . . . . 5-19
Table 5-13 Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Warm Climates (Soil Ingestion Only) . . . . . 5-20
Table 5-14 Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Cold Climates (Soil Ingestion Only) . . . . . 5-21
Table 5-15. Arsenic Cancer Risks . . . . . 5-25
Table 5-16. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Based on LADDs in Table 14 from the SHEDS-Wood document) . . . . . 5-26
Table 5-17. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climates (Based on LADDs in Table 15 from the SHEDS-Wood document) . . . . . 5-27
Table 5-18. Arsenic Cancer Risk Assuming 0.01% Dermal Absorption . . . . . 5-31
Table 5-19. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Dermal Residue Absorption Rate = 0.01%) (Based on LADDs in Table 35 from SHEDS-Wood document) . . . . . 5-32
Table 5-20. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climate (Dermal Residue Absorption Rate = 0.01%) (Based on LADDs in Table 36 From SHEDS-Wood Document) . . . . . 5-33
Table 5-21. Comparison of Arsenic Risks Between Baseline and 0.01% Dermal Absorption Warm Climate . . . . . 5-34
Table 6-1. Summary of Sealant Studies . . . . . 6-4
Table 6-2. Arsenic Mitigation Measures Evaluated . . . . . 6-6
Table 6-3. Summary of Risks Assuming Different Arsenic Mitigation Measures for Warm Climate Conditions . . . . . 6-7
Table 6-4. Cancer Risks Remaining Following Simulated Reduction in Residues from the Use of Sealants (Warm Climate only) . . . . . 6-9
Table 6-5. Probabilistic Arsenic Cancer Risk Distributions and Risk Ranges for Children in Warm Climates (reducing deck and playset residue concentration by 90%) (Based on the LADDs in Table 37 from the SHEDS-Wood document) . . . . . 6-12

## LIST OF TABLES (CONTINUED)

Table 6-6. Probabilistic Arsenic Cancer Risk Distributions and Risk Ranges for Children (Reducing Deck and Playset Residue Concentration by 99.5% in Warm Climates (Based on LADDs in Table 38 from the SHEDS-Wood document) . . . . . 6-13
Table 6-7. Cancer Risks Remaining Following Simulated Reductions from Hand Washing (Warm Climate Only) . . . . . 6-14
Table 6-8. Probabilistic Arsenic Cancer Risk Distributions and Risk Ranges for Children in Warm Climates (Reducing Exposure by Washing Hands After Playing on Deck or Playset) (Based on LADDs in Table 39 from the SHEDS-Wood document) . . . . . 6-16
Table 6-9. Comparison of Cancer Risks from Combined Mitigation Measures at $10^{-6}$ and $10^{-5}$ Risk Levels . . . . . 6-17
Table 6-10. Comparison of Total Risk to Residue Only Risk Under Different Mitigation Conditions (Playsets and Decks - Warm Climate) . . . . . 6-22
Table 6-11. Comparison of Mitigation Measures to Baseline at the $10^{-6}$ Risk Level (Warm Climate) . . . . . 6-25

## LIST OF FIGURES

Figure 1-1 Arsenic Cancer Risk at the 95% Percentile (Warm Climate) . . . . . 1-9
Figure 1-2 Arsenic Cancer Risk at the 50% Percentile (Warm Climate) . . . . . 1-10
Figure 1-3 Comparison of Residue Only Risks for Playsets and Decks for Warm Climate (Maximum Reduction, Moderate Reduction, Baseline) . . . . . 1-11
Figure 5-1. A Cumulative Distribution Function (CDF) for Cancer Risk . . . . . 5-2
Figure 5-2. MOE of Short-term ADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (separated by children with and playset only) . . . . . 5-8
Figure 5-3. MOE of Short-term ADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (separated by children with and playset only) . . . . . 5-9
Figure 5-4. MOE of Intermediate-term ADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (separated by children with and playset only) . . . . . 5-16
Figure 5-5. MOE of Intermediate-term ADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (separated by children with and playset only) . . . . . 5-17
Figure 5-6. Cancer Risk (Lifetime) for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (separated by children with and playset only) . . . . . 5-23
Figure 5-7. Cancer Risk (Lifetime) for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (separated by children with and playset only) . . . . . 5-24
Figure 5-8. Comparison of Total Risks from Decks and Playsets for Warm Climate - No Mitigation . . . . . 5-29
Figure 5-9. Comparison of Residue and Soil Total Exposures for Warm Climate - No Mitigation . . . . . 5-30
Figure 5-10. Cancer Risk from Lifetime LADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (Dermal Residue Absorption Rate = 0.01%) . . . . . 5-35

**LIST OF FIGURES (CONTINUED)**

Figure 5-11. Cancer Risk from Lifetime LADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (Dermal Residue Absorption Rate = 0.01%) . . . . . 5-36
Figure 6-1 Cancer Risk from Lifetime LADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (Reducing Deck and Playset Residue Concentration by 90%) . . . . . 6-10
Figure 6-2 Cancer Risk from Lifetime LADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (Reducing Deck and Playset Residue Concentration by 99.5%) . . . . . 6-11
Figure 6-3 Cancer Risk from Lifetime LADD for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (Reducing Deck and Playset Residue Concentration by Washing Hands after Playing on Deck or Playset) . . . . . 6-15
Figure 6-4 Comparison of Residue & Soil Total Arsenic Risks for Warm Climate 99.5% Reduction . . . . . 6-19
Figure 6-5 Comparison of Residue & Soil Total Arsenic Risks for Warm Climate 90% Reduction . . . . . 6-20
Figure 6-6 Comparison of Residue and Soil Arsenic Risks for Warm Climate 99.5% Reduction . . . . . 6-21
Figure 6-7 Comparison of Residue and Soil Arsenic Risks for Warm Climate 90% Reduction . . . . . 6-23

## APPENDICES


| | |
|---|---|
| Appendix A | Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium |
| Appendix B | Risk Spreadsheets |
| Appendix C | Comparison of Total Risks to Risk Reduction Impacts |
| Appendix D | Comparison of Residue and Soil Risk |
| Appendix E | Summary of Relative Bioavailability Studies |
| Appendix F | SAP Report No. 2001-12, FIFRA Scientific Advisory Panel Meeting |
| Appendix G | Effect of Hand Washing on Risks from Exposure to Residues |

## AUTHORS, CONTRIBUTORS, AND REVIEWERS

The National Exposure Research Laboratory of Exposure Assessment and Risk Assessment, Office of Research and Development, and the Antimicrobials Division, Office of Pesticide Programs, was responsible for the preparation of this document. A number of individuals have reviewed and/or have been contributing authors of this report including:

The **Probabilistic Exposure Assessment** portion of this report was developed by the following individuals:

V.G. Zartarian[1], J. Xue[1], H. Özkaynak[1], W. Dang[2]
U.S. Environmental Protection Agency
[1] Office of Research and Development, National Exposure Research Laboratory
[2] Office of Pesticide Programs, Antimicrobials Division

G. Glen, L. Smith, C. Stallings
ManTech Environmental Technology, Inc.

The **Probabilistic Risk Assessment** portion of this report was developed by the following individuals:

W. Dang, J. Chen
U.S. Environmental Protection Agency
Office of Pesticide Programs, Antimicrobials Division

N.Mottl, L.Phillips, P.Wood, S.McCarthy, R.Lee, M. Helmke,
M. Nelson, and K.Coon
Versar, Inc.

**For General Information Related to this Document Contact**:

W. Dang
U.S. Environmental Protection Agency
Office of Pesticide Programs, Antimicrobials Division
Mailcode 7510C
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

**Acknowledgments**

We would like to thank Jack Housenger, William Jordan, Najm Shamin, Timothy Leighton, Norm Cook, Nancy Chiu, Doreen Aviado, Siroos Mostaghimi, Tim McMahon, David Miller, Steven Nako, Greg Schweer, Brenda Foos, Andrew Schulman, Susan Griffin, Timothy Barry, Herman Gibb, Valerie Zartarian, Jianping Xue, Haluk Özkaynak, Francis Suhre, Elizabeth Margosches, Deborah Smegal and Bart Suhre of the U.S. Environmental Protection Agency, for assisting with selection of model inputs and reviewing the preliminary draft report. We would also like to thank the Consumer Product Safety Commission (CPSC), Michael Dong from CDPR of California EPA, and Cathy Campbell, John Worgan, Connie Moase, from PMRA of Health Canada, Graham Glen from ManTech Environmental Technology, Inc. for reviewing the preliminary drafts and providing valuable comments.

We would also like to thank Doreen Aviado, Norm Cook, Najm Shamin, Steve Malish, and Siroos Mostaghimi of Antimicrobials Division and Debbie Edwards, Director of Registration of OPP, US EPA for their support, drafting and reviewing the background documents in 2001.

**PREFACE**

The National Exposure Research Laboratory, EPA's Office of Research and Development and the Antimicrobials Division of EPA's Office of Pesticide Program has prepared this document to address exposures and risks to children from contact with Chromated Copper Arsenate (CCA)-treated wood in playsets and decks, and CCA-contaminated soil around these structures. In October 2001, OPP presented a proposed deterministic exposure assessment approach specific to CCA to the FIFRA (Federal Insecticide, Fungicide, and Rodenticide Act) Scientific Advisory Panel (SAP). One of the primary SAP recommendations was to use a probabilistic model to predict variability of absorbed doses for the population of interest. This document provides a probabilistic risk assessment based on exposure results from the model as recommended by the SAP.

In general, a risk assessment should include hazard identification, hazard assessment, exposure assessment, and risk characterization. In this document, OPP collaborated with ORD to develop comprehensive state-of-the-art techniques to complete a complex risk analysis of children exposed to CCA-treated wood at residential sites.

This report fulfills the following EPA basic guiding principles for assessing risk to CCA: (1) identifying the population (i.e., children) exposed to CCA-treated playsets and decks as part of the "problem formulation" phase, (2) gathering sufficient information to develop and model the exposures, (3) conducting sensitivity analysis, (4) discussing the correlation or dependencies between the input variables, (5) detailing information for each input and output distribution, including information on the stability of central tendency and higher end values, (6) comparing the results of deterministic and probabilistic assessments, and (7) using the best available toxicity information to combine with the exposure estimates to calculate risks. The current policy, Conditions for Acceptance and associated principles are not intended to apply to dose-response evaluations for human health risk assessments until this application has been studied further (Agency Policy Document, 5/15/1997). Currently, OPP does not have the Guidance to perform the probabilistic analysis of toxicity endpoints.

Three steps were used by OPP to complete this document. The first step used a deterministic risk assessment approach. The second used a custom-designed probabilistic model for wood preservative use exposure scenarios. The third step was the risk analysis based on the exposure model outputs and the toxicity endpoints recommended by the SAP and other EPA offices (OW, ORD, OCHP, Superfund and OPP).

This report provides the results of a study that used the output of (1) a probabilistic exposure assessment for children who come into contact with CCA-treated playsets and decks based on the Stochastic Human Exposure and Dose Simulation Model for the Wood Preservative Exposure Scenario (SHEDS-Wood) developed by the ORD; and (2) the toxicity data for arsenic

and chromium to develop probabilistic risk assessment for children who contact CCA-treated playsets and decks.

There are several other risk assessment reports related to children's exposure from CCA-treated playsets and decks. These reports have been developed by researchers outside of EPA/OPP. Additionally, research is ongoing that focuses on dislodgeable arsenic surface residues and risk reduction options based on the application of sealants. However, this assessment uses state-of-the-art analyses and is different from previous risk assessments in the available literature.

## LIST OF ACRONYMS

| | |
|---|---|
| ACC | American Chemistry Council |
| AD | Antimicrobials Division |
| ADD | Average Daily Dose |
| APVMA | Australian Pesticides & Veterinary Medicines Authority |
| Ar (V) | Arsenic (V) |
| As | Arsenic |
| ATSDR | Agency for Toxics Substances and Disease Registry |
| AWPA | American Wood-Preserver's Association |
| AWPI | American Wood Preservers Institute |
| BF | Bioavailability Factor |
| CAP | Consumer Awareness Program |
| CCA | Chromated Copper Arsenate |
| CCA-C | CCA Type C |
| CDF | Cumulative Density Function |
| CDHS | California Department of Health Services |
| CE | Cumulative Exposure |
| CFA | Consumer Federation of America |
| CHAD | Consolidated Human Activity Database |
| CPSC | Consumer Product Safety Commission |
| Cr | Chromium |
| Cr(VI) | Chromium (VI) |
| Cr(III) | Chromium (III) |
| CSIS | Consumer Safety Information Sheet |
| Cu | Copper |
| DA | Dislodgeable Arsenic |
| DEC | Department of Environmental Conservation |
| E | New Exposure |
| EC | European Commission |
| EFH | Exposure Factors Handbook |
| EMRA | Environmental Risk Management Authority |
| EPA | Environmental Protection Agency |
| ERDEM | Exposure Related Dose Estimation Model |
| EWG | Environmental Working Group |
| FIFRA | Federal Insecticide, Fungicide, Rodenticide Act |
| FR | *Federal Register* |
| FQPA | Food Quality Protection Act of 1996 |
| GI | Gastrointestinal |
| GM | Geometric Mean |
| GSD | Geometric Standard Deviation |
| HBN | Healthy Building Network |

| | |
|---|---|
| HED | Health Effects Division, OPP |
| HIARC | Hazard Identification Assessment Review Committee |
| IPEMA | International Play Equipment Manufacturers Association |
| IRIS | Integrated Risk Information System |
| LADD | Lifetime Average Daily Dose |
| $LD_{50}$ | Lethal Dose, 50% Kill |
| LOAEL | Lowest-Observed-Adverse-Effect Level |
| MCA | Monte Carlo Analysis |
| MDEP | Maine Department of Environmental Protection |
| MOE | Margin of Exposure |
| $MOE_{calc}$ | Calculated MOE |
| NAS | National Academy of Sciences |
| NCEA | National Center For Exposure Assessment |
| NCP | National Contingency Plan |
| NERL | National Exposure Research Laboratory |
| NHANES | National Health and Nutrition Examination Survey |
| NHAPS | National Human Activity Pattern Survey |
| NOAEL | No-Observed-Adverse-Effect Level |
| NOIC | Notice of Intent to Cancel |
| NRC | National Resource Council |
| OPP | Office of Pesticide Programs |
| OPPTS | Office of Prevention, Pesticides, and Toxic Substances |
| ORD | Office of Research and Development |
| PBPK | Physiologically-Based Pharmacokinetic |
| pcf | Pounds per cubic foot |
| PD | Position Document |
| PDF | Probability Density Function |
| PMRA | Pest Management Regulatory Agency |
| PND | Preliminary Notice of Determination |
| ppm | Parts per million |
| Pr | Probability |
| PRA | Probabilistic Risk Assessment |
| $Q_1$* | Slope Factor |
| RAG | Risk Assessment Guideline |
| RB | Relative Bioavailability |
| RED | Reregistration Eligibility Decision |
| RME | Reasonably Maximum Exposed Individual |
| RPAR | Notice of Rebuttable Presumption Against Registration and Continued Registration |
| RTI | Research Triangle Institute |
| SA | Surface Area |
| SAP | Science Advisory Panel |

| | |
|---|---|
| SCS | Soil Contact Survey |
| SCTEE | Scientific Committee on Toxicity, Ecotoxicity and the Environment |
| SF | Slope Factor |
| SHEDS | Stochastic Human Exposure and Dose Simulation Model |
| SHEDS-Wood | Stochastic Human Exposure and Dose Simulation Model for the Wood Preservative Scenario |
| SOPs | Standard Operating Procedures |
| TC | (Dermal) Transfer Coefficient |
| TE | (Dermal) Transfer Efficiency |
| USDA | U.S. Department of Agriculture |
| USPIRG | U.S. Public Interest Research Group |

# 1.0 EXECUTIVE SUMMARY

The U.S. Environmental Protection Agency's (EPA) Office of Pesticide Programs (OPP) is aware of increased concerns raised by the general public, municipal and state governments, and state/federal regulatory agencies regarding the safety of young children contacting arsenic and chromium residues while playing on Chromated Copper Arsenate (CCA) treated wood playground structures and decks. Because of this concern, OPP's Antimicrobials Division (AD), with the recommendation of the Federal Insecticide, Fungicide, Rodenticide Act (FIFRA)'s Scientific Advisory Panel (SAP) and the assistance of the Office of Research and Development (ORD), has conducted a probabilistic exposure assessment entitled the Stochastic Human Exposure and Dose Simulation Model for the Wood Preservative Exposure Scenario (SHEDS-Wood). SHEDS-Wood provides exposures reported as average daily doses (ADDs) and lifetime average daily doses (LADDs). Children's exposures may occur through touching CCA-treated wood and CCA-contaminated soil near treated wood structures, mouthing hands after touching CCA-treated wood, and eating CCA-contaminated soil. Since EPA has determined that the arsenic and chromium components of CCA pose the most significant toxicity concerns in comparison to copper, which is not a recognized or suspected carcinogen, the Agency focused on evaluating potential adverse short-term (1-day to 1-month), intermediate-term (1 to 6 months) noncancer exposure doses for total arsenic and chromium as Cr(VI), and lifetime average cancer exposure doses from total arsenic. Some of the key terms used in the SHEDS-Wood exposure report are summarized in Table 1-1.

OPP developed a preliminary deterministic risk assessment (Internal Draft Only) on May 30, 2001 (U.S. EPA, 2001a). In this internal draft, OPP reported on a preliminary exposure and risk assessment on the chromium and arsenic components of CCA to determine the potential health risks to children from contact with CCA-treated wood playground structures and CCA-contaminated soil resulting from use of CCA on lumber used in the fabrication of playground equipment and related structures commonly found in residential settings. The U.S. EPA (2001a) internal draft report was later revised and incorporated as a preliminary exposure assessment on September 27, 2001 (U.S. EPA, 2001b), which was later reviewed by the FIFRA SAP (U.S. EPA, 2001c). The exposure factors used in the U.S. EPA (2001a) assessment were primarily conservative upper bound estimates for short- and intermediate-term noncancer risk. The mean, or central tendency exposure factors were used for cancer risk.. The results of the U.S. EPA (2001a) arsenic cancer risk assessment were comparable to the upper bound estimates in this probabilistic risk assessment. Using an initial oral arsenic cancer slope factor (Q*) of 1.5 $(mg/kg/day)^{-1}$, U.S. EPA (2001a) reported a cancer risk of 2.0E-4 which would be equivalent to 5.0E-4 using the $Q_1$* of 3.67 $(mg/kg/day)^{-1}$ identified in this report. The arsenic probabilistic cancer risks presented in this report were 1.4E-4 for the 95$^{th}$ percentile, 2.3E-5 for the median, and 4.2E-5 for the mean. The results for the means in the probabilistic assessment are similar to the 75$^{th}$ percentile for several exposure scenarios.

## Table 1-1. Definitions of Key Terms Used in the SHEDS-Wood Risk Assessment

| Key Term | Definition |
|---|---|
| Population | OPP's primary population of interest for this assessment were children in the United States who frequently contact CCA-treated wood residues and/or CCA-containing soil from public playsets (e.g., at a playground, a school, a daycare center). Children playing on residential playsets were the secondary focus. SHEDS-Wood also examined a subset of these children who contact CCA-treated wood residues and/or CCA-containing soil from residential playsets and/or residential decks (i.e., at the child's own home or at another home). Results from both groups of children (those who contact public playsets only, and those who contact public and residential playsets) were presented in this report.<br><br>The focus of this assessment was on estimating the risk to children from contact with various sources of CCA-treated wood. The primary population considered in this assessment was children with public playsets. EPA believes that more young children are exposed to CCA-treated public playsets than residential playsets because children spend more time on public playsets at schools and daycare centers. EPA also believes that children playing on public playsets would affect a larger population of children. More data were available for public playsets than residential playsets. Further, CPSC and other groups have also focused their review on children exposed to public playsets. |
| Warm vs. Cold Scenarios | The SHEDS-Wood report referred to separate 'warm climate' and 'cold climate' scenarios. However, the Consolidated Human Activity Database (CHAD) diaries that were used in SHEDS-Wood were missing specific state locator information. Instead of using geographical locations, 'warm climate' and 'cold climate' were simulated by modifying inputs such as surface area of unclothed skin and time spent on playsets and decks. See the text and tables (e.g., Table 12) of Zartarian et al. (2003) for more details regarding the assumptions for warm vs. cold climates. |
| With and Without Decks | With or without decks was used to indicate whether or not the population of children examined in the assessment had a residential deck or not. The term "with deck" was used to indicate that a child was exposed to a residential deck (i.e., at the child's own home or at another home) and a playset. The term "without decks" was used to indicate that a child was exposed to a playset only (Zartarian et al., 2003). |
| Sealant (Moderate and Maximum Reduction) | Exposure reductions from sealants was assessed in SHEDS-Wood using two assumed reduction levels: moderate (90% residue reduction) and maximum (99.5% residue reduction). The use of the sealants reduced the arsenic residue concentrations which resulted in a corresponding reduction in arsenic exposure. EPA derived the assumed reduction estimates based upon the available literature on sealants and the comments provided by the FIFRA SAP in their review of sealant data (see Appendix F for more details). |
| Hand Washing | Hand washing was considered for all the modeled scenarios in SHEDS-Wood. Several different input distributions were used for hand washing events per day, hand washing removal, etc. In addition, a special analysis was simulated in SHEDS-Wood to estimate the exposure after a child washes his or her hands after playing on a playset or deck. In addition, exposures were modeled using hand washing in combination with a moderate reduction in residues because of the use of a sealant and hand washing in combination with a maximum reduction in residues because of the use of a sealant. |
| Time Periods | For the CCA assessment presented in this report, three exposure time periods were considered: short-term (represented in SHEDS-Wood by a 15 day averaging time; 1 day to 1 month), intermediate-term (represented in SHEDS-Wood by a 90 day averaging time; 1 to 6 months), and lifetime (6 years exposure over a 75-year lifetime). |
| Exposure Pathways | There were eight primary exposure pathways considered in SHEDS-Wood: dermal soil contact near decks; dermal residue contact from decks; soil ingestion near decks; residue ingestion from decks (via the wood-to-hand-to-mouth pathway); dermal soil contact near playsets; dermal residue contact from playsets; soil ingestion near playsets; and residue ingestion from playsets (via the wood-to-hand-to-mouth pathway). Dermal exposure was also computed separately for hands and body, and results were aggregated for decks and playsets, as well as over all pathways.<br><br>As pointed out by CPSC (2003a), it is possible in extreme cases that pre-schoolers may occasionally directly mouth portions of a wood play structure, although this behavior is not likely to be frequent for most playground users. Inhalation exposure to particulates for children that are present during sandblasting of CCA-treated surfaces would also be another potential pathway. These less common pathways were not included into the CCA risk assessment. Other potential sources of exposure not included in this assessment or other related CCA risk assessments include child exposures to picnic tables, porch railings and uprights, contact with pets and objects that have contacted treated wood, and CCA residues and soil that are brought indoors from outside. |
| Soil vs. Residue Exposure | SHEDS-Wood examined ingestion and dermal exposure routes for children from contact with CCA-contaminated soil and wood residues. Soil exposure refers to dermal contact with CCA-contaminated soil and soil ingestion. Residue exposure refers to dermal contact with CCA-treated wood and ingestion for residues from CCA-treated wood via hand-to-mouth contact. |

After review of the September 27, 2001 deterministic exposure assessment, SAP recommended that a probabilistic assessment be developed to examine the exposure scenarios U.S. EPA (2001c). In 2002, SHEDS-Wood probabilistic model was presented to the SAP for review and recommendations from the panel. After incorporation of comments from the SAP, a draft final report was prepared on September 25, 2003. The probabilistic exposure assessment present results for absorbed doses (both ADD and LADDs). The results of the draft final SHEDS-Wood probabilistic exposure assessment were used in this risk assessment. It should be noted, however, that the existing policy, Agency Policy Document (5/15/97), indicated that the "Conditions for acceptance and associated principles are not intended to apply to dose-response evaluations for human health risk assessments until this application has been studied further". Currently, OPP does not have the Guidance to perform the probabilistic analysis of toxicity endpoints. Some of the major findings from the probabilistic assessment include:

- Children who contact playsets only were found to have lower absorbed doses than children who contact both playsets and decks by a factor of 2.
- Warm climate bounding scenarios yielded higher results than cold climate scenarios.
- For children who contact both playsets and decks, the mean arsenic LADDs were reduced by a factor of 14 and median arsenic LADDs were reduced by a factor of 17 when residue concentrations were reduced by 99.5%.
- For children who contact both playsets and decks, the total mean and median arsenic LADDs were both reduced by a factor of 1.3 when hand washing was assumed to occur following exposure.
- Children with pica soil ingestion behavior had about 2-3 times higher absorbed mean doses (totaled over all pathways considered) of arsenic than non-pica children from CCA-treated playsets and decks. The risks estimated for children with pica soil ingestion behavior were higher than for non-pica children.
- Assuming a mean arsenic dermal absorption rate of 0.01% rather than 3% for children who contact playsets and decks in warm climates, the mean and median arsenic LADDs were 30% and 26% lower, respectively.
- The most significant exposure route for the population of interest for most scenarios was residue ingestion via hand-to-mouth contact, followed by dermal contact, soil ingestion, and dermal soil contact.

Risks that arise from the predicted exposures were quantified in this risk assessment. This report follows OPP guidance. This risk assessment includes a background chapter on issues related to children's exposure to CCA-treated wood and the reasons that EPA conducted a non-dietary probabilistic assessment (see Chapter 2.0); describes the arsenic and chromium exposures generated by the SHEDS-Wood model (see Chapter 3.0); summarizes the arsenic and chromium toxicity endpoints in a hazard assessment (see Chapter 4.0); characterizes the risks for the exposures generated by the SHEDS-Wood model (see Chapter 5.0); characterizes the reduction

impacts for the exposures generated by the SHEDS-Wood model (see Chapter 6.0); and discusses the uncertainty, strengths, and limitations associated with this risk assessment (see Chapter 7.0). In addition, the following appendices are provided:

| | |
|---|---|
| Appendix A | Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium |
| Appendix B | Risk Spreadsheets |
| Appendix C | Comparison of Total Risks to Risk Reduction Impacts |
| Appendix D | Comparison of Residue and Soil Risk |
| Appendix E | Summary of Relative Bioavailability Studies |
| Appendix F | SAP Report No. 2001-12, FIFRA Scientific Advisory Panel Meeting |
| Appendix G | Effects of Hand Washing on Risks from Exposure to Residues |

The goal of this risk assessment is to present the SAP with the calculated arsenic cancer risks to children (age 1-6) exposed to CCA-treated playsets and decks using a probabilistic risk analysis. It also identifies methods (e.g., sealants and hand washing) which can reduce the arsenic cancer risks to children. However, there is no concluding statement regarding the percentiles of the distribution or point estimates (e.g., mean, $50^{th}$ , $90^{th}$ , $95^{th}$, etc) at which risk management decisions will be made. OPP intends to provide recommendations on how risk managers should interpret the results of this risk assessment, after receiving technical comments from the FIFRA SAP on evaluating probabilistic risk distributions. OPP will carefully consider the FIFRA SAP's comments on this issue.

Noncancer Margins of Exposure (MOEs) and cancer risks to children exposed to CCA-treated playsets and decks and/or CCA-containing soil from these playsets and decks were calculated from doses generated using OPP/ORD's SHEDS-Wood model for chromium and arsenic. The exposure assessment considered children, ages 1 to 6 years old.[1] Risks due to possible exposure to Cr(VI) for the soil ingestion route were estimated, conservatively, by assuming 10% of total chromium was present as Cr(VI). For chromium, as Cr(VI), the toxicity value used was 0.5 mg/kg/day (a NOAEL) for noncancer effects. The toxicity value for total arsenic used in this assessment were 3.67 $(mg/kg/day)^{-1}$ (slope factor) for cancer effects and 0.05 mg/kg/day (a LOAEL) for noncancer effects. The Agency is currently considering recommendations by the National Research Council (NRC) and the arsenic slope factor may change in the final version of this risk assessment. The arsenic carcinogenic risk is a conservative estimate of the risk because the cancer slope factor is characterized as a upper-bound estimate. Therefore, the true risks to humans, while not identifiable, may not be likely to exceed the upper-bound estimates and in fact may be lower. Noncancer risks were evaluated against OPP's

[1] Exposure durations modeled were short-term (1 day to 1 month), intermediate-term (1 to 6 months), and lifetime (6 years averaged over 75 years).

guidance for MOE values for arsenic and chromium for short-term and intermediate-term exposure durations. Lifetime cancer risks from arsenic exposure were compared to EPA/OPP's risk range of $10^{-6}$ to $10^{-4}$. Risks were found to be greater under warm climate conditions than cold climate conditions (MOEs were lower for warm climates). Exposure to playsets and decks had higher risks than exposure to playsets alone. Noncancer MOEs for arsenic were found to be above EPA/OPP's guidance MOE of 30 for all exposures, except at the extreme upper end of the distribution. Cr(VI) risks were found to be above the guidance MOE of 100 for all doses. These noncancer MOEs are summarized in the upper portion of Table 1-2. Cancer risks exceeded the upper bound of the risk range, $10^{-4}$, at cumulative percentiles ranging from the 90th for warm climate conditions and exposure to CCA-treated playsets and decks, to the 99th for cold climate conditions and exposure to playsets only. Across all exposure scenarios, carcinogenic risks were found to be less than $10^{-6}$ at cumulative percentiles of the 9th and lower, meaning at least 91% of the simulated population had risks above $10^{-6}$. The lower portion of Table 1-2 presents the cumulative percentiles at the three levels of EPA's risk range.

Table 1-3 presents the arsenic cancer risks from four points on the cumulative probability curve: mean, median, 95$^{th}$ percentile, and 99$^{th}$ percentile. Risks at the mean and median were found to be in the range of $10^{-5}$ to $10^{-6}$. At the 95$^{th}$ percentile, the risk level for exposure to decks and playsets under warm climate conditions was at $10^{-4}$. Risk levels for other conditions of exposure at this percentile ranged from approximately $4 \times 10^{-5}$ to $8 \times 10^{-5}$.

The influence of the dermal absorption factor was evaluated. Baseline risks were determined using a dermal absorption factor of 2 to 3%. Risk levels were also calculated using the lower arsenic dermal absorption factor of 0.01%. Changing the dermal absorption factor by approximately two orders of magnitude reduced risk by 26% to 47%, depending on the exposure scenario and the cumulative percentile of interest.

An analysis comparing the arsenic risks from soil exposure versus residue exposure (i.e., contact with CCA treated wood surfaces only) was conducted for both sources of exposure: playsets alone and playsets with decks. The estimated risks should be viewed as approximations, however, because residue and soil risks were summed across routes at the quartile level and this incurs inaccuracies. Residue risks were found to be greater than soil risks. For contact with playsets only, this difference ranged from a factor of approximately 7 at the 50$^{th}$ percentile to 10 at the 99$^{th}$ percentile. Differences were larger for playsets and decks. At the 50$^{th}$ percentile, residue risk for playset and deck exposure was slightly greater than $10^{-5}$, and approximately $10^{-4}$ at the 95$^{th}$ percentile. Soil only risk for both playset only exposure, and playset and deck exposure exceeded $10^{-5}$ at the 95$^{th}$ percentile.

**Table 1-2. Summary of Risk Assessment Results**

*Noncancer MOEs for Arsenic and Chromium* [a]

| Source of Exposure | Climate | Duration of Exposure | Arsenic MOE <30 | Chromium MOE <100 |
|---|---|---|---|---|
| Playset Only | Warm | Short & Intermediate | >99.6th Percentile | None |
| | Cold | | | |
| Playset and Deck | Warm | Short & Intermediate | >99.6th Percentile | None |
| | Cold | | | |

a Chromium is represented as Cr(VI) for the soil ingestion route only.

*Cancer Risks for Arsenic* [b]

| Source of Exposure | Climate | Cumulative Percentiles at Specified Risk Levels | | |
|---|---|---|---|---|
| | | $10^{-6}$ | $10^{-5}$ | $10^{-4}$ |
| Playset Only | Warm | 3rd | 47th | 97th |
| | Cold | 9th | 69th | 99th |
| Playset and Deck | Warm | <1st | 23rd | 90th |
| | Cold | 2nd | 49th | 97th |

[b] Percentiles in this table represent the percent of the simulated population that have risks less than or equal to the stated risk level; e.g., at $10^{-6}$, 3% of the population have risks less than $10^{-6}$ and 97% have risks greater than $10^{-6}$.

**Table 1-3. Arsenic Cancer Risks**

| Scenario | Arsenic ($Q_1$*= 3.67 (mg/kg/day)$^{-1}$) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Playset and Deck | 4.2E-05 | 2.2E-05 | 2.3E-05 | 1.1E-05 | 1.4E-04 | 7.8E-05 | 3.1E-04 | 1.6E-04 |
| Playset Only | 2.3E-05 | 1.2E-05 | 1.1E-05 | 5.4E-06 | 8.3E-05 | 4.5E-05 | 2.4E-04 | 8.9E-05 |

In 2001, the FIFRA SAP recommended that additional research was needed to evaluate the performance and efficacy of different brands of coatings. EPA recently completed the protocol to begin additional research on the effectiveness of sealants on weathered CCA-treated wood. In the SHEDS-Wood exposure assessment, the concentration of wood surface residue was considered as the key variable based on the sensitivity analysis in the SHEDS-Wood exposure report. Therefore, using the existing data from Stilwell (1998) and CDHS (1987) (see Chapter 6), EPA assumed two reduction levels: moderate (90% residue reduction) and maximum (99.5% residue reduction) to assess reductions in exposure, and thus risk, based on the use of sealants.

Different mitigation measures to reduce exposure to arsenic-containing residues and, thus risk, were evaluated. Many of the recommendations to reduce arsenic concentrations are based on the activities of the homeowner and can only be considered guidance. The SHEDS-Wood model quantified exposures based on reduction in the residue concentration resulting from the use of sealants and/or hand washing. No reduction in soil exposure was considered as part of these mitigation simulations. Although soil concentrations may be reduced over time with the use of sealants, it was conservatively assumed that soil concentrations were the same as under baseline conditions. Results of five different mitigation conditions are summarized in this report. Two of the mitigation conditions simulated the effect of a hypothetical sealant on reducing exposure to dislodgeable residues. For moderately effective sealant conditions, the residue concentration was assumed to be reduced by 90%; for maximally effective sealant conditions, residue concentration was assumed to be reduced by 99.5%. The other type of mitigation measure simulated was increasing the frequency of hand washing. This was considered alone and in combination with the sealant conditions. These different mitigation measures were evaluated for the warm climate condition only, as that had the greater exposure and, thus, risk. The effect of reducing risk was considered at the $10^{-6}$ risk level. Increasing the frequency of hand washing alone or in combination with sealants had a minimal effect compared to no mitigation on the cumulative percentile at the $10^{-6}$ risk level. Note that although hand washing may not have had a significant impact on total risk from both (i.e., from both soil and residues), it did have a significant impact on the dermal and oral routes from the surface residue pathways. The largest change was for the maximum reduction assumption for contact with playsets only under warm climate conditions, where the $10^{-6}$ risk level was at the 57$^{th}$ percentile compared to the 3$^{rd}$ percentile at baseline. Table 1-4 compares the cumulative percentiles at the three risk levels for the various mitigation measures considered. Figure 1-1 shows a comparison of carcinogenic risks at the 95$^{th}$ percentile for baseline to the five different mitigation conditions described. Figure 1-2 shows the same comparison across baseline and mitigation conditions, but for the 50$^{th}$ percentile.

Figure 1-3 compares the effects of the residue mitigation measures to baseline conditions for playset and decks exposure. This plot shows the approximate residue only risk for the maximum and moderate reduction in residue concentration, and baseline conditions. Under the maximum reduction assumption, residue risks were decreased by over 2 orders of magnitude at the 95$^{th}$ percentile and by approximately 3 orders of magnitude at the 50$^{th}$ percentile.

**Table 1-4. Summary of Arsenic Risks Assuming Different Mitigation Measures for Warm Climate Conditions**

| Mitigation Measure | Cumulative Percentiles at Specified Risk Levels | | |
|---|---|---|---|
| | Risk Level of $10^{-6}$ | Risk Level of $10^{-5}$ | Risk Level of $10^{-4}$ |
| **Playset Only** | | | |
| 1. Sealant- moderate reduction | 27th | 94th | >99th |
| 2. Sealant – maximum reduction | 57th | 97th | >99th |
| 3. Hand washing | 5th | 59th | >99th |
| 4. Sealant-moderate + hand washing | 28th | 92nd | >99th |
| 5. Sealant-maximum + hand washing | 58th | 96th | >99th |
| 6. Baseline | 3rd | 47th | 97th |
| **Playset and Deck** | | | |
| 1. Sealant-moderate reduction | 10th | 80th | >99th |
| 2. Sealant-maximum reduction | 42th | 94th | >99th |
| 3. Hand washing | <1st | 28th | 95th |
| 4. Sealant-moderate + hand washing | 10th | 84th | >99th |
| 5. Sealant-maximum + hand washing | 44th | 93rd | >99th |
| 6. Baseline | <1st | 23rd | 90th |

Note: The baseline scenario includes a certain amount of hand washing. Hand washing, as a mitigation scenario, increases the frequency of this activity over baseline. See Appendix G for more information on hand washing.

**Figure 1-1 Arsenic Cancer Risk at the 95% Percentile (Warm Climate)**

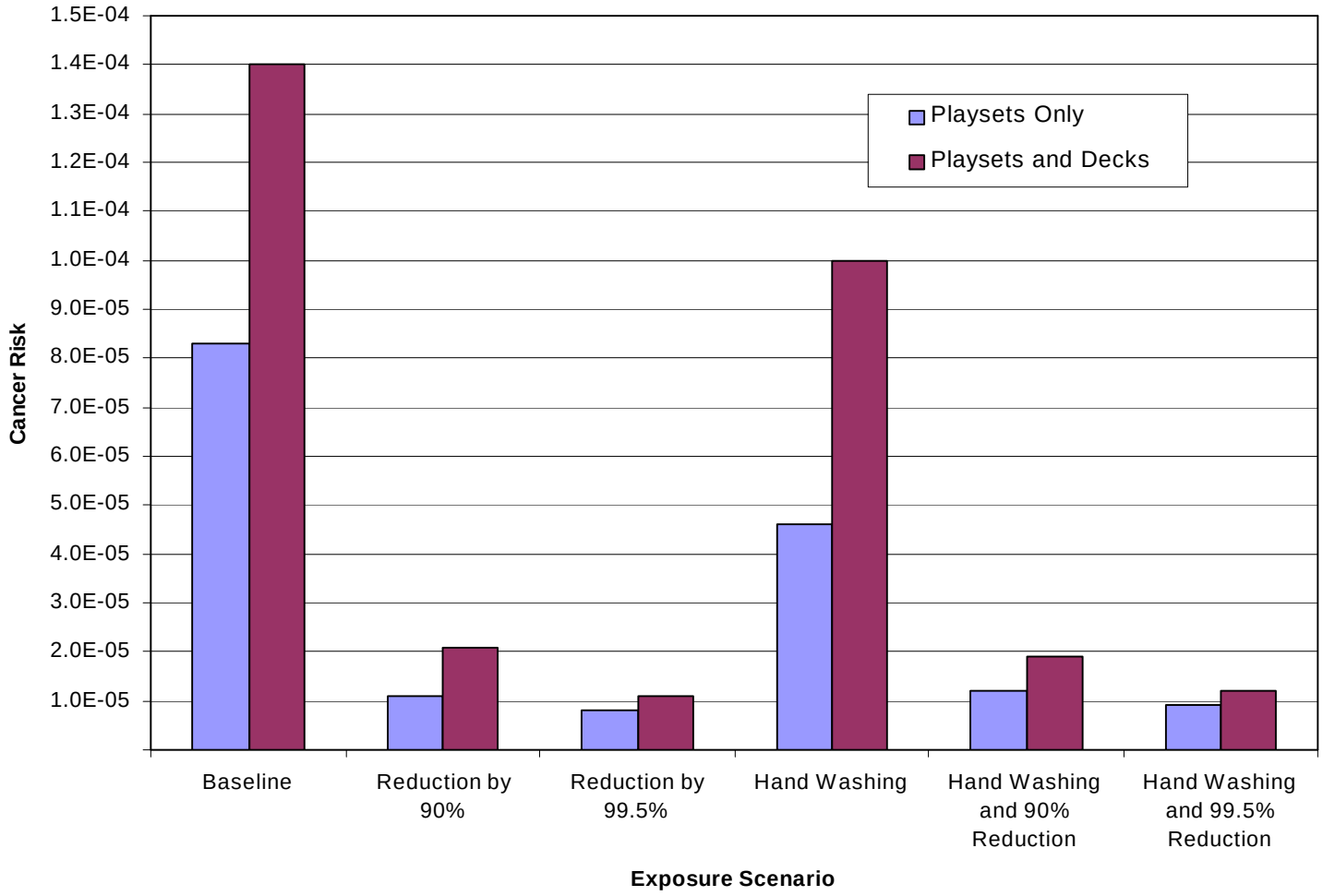


**Figure 1-2 Arsenic Cancer Risk at the 50% Percentile (Warm Climate)**

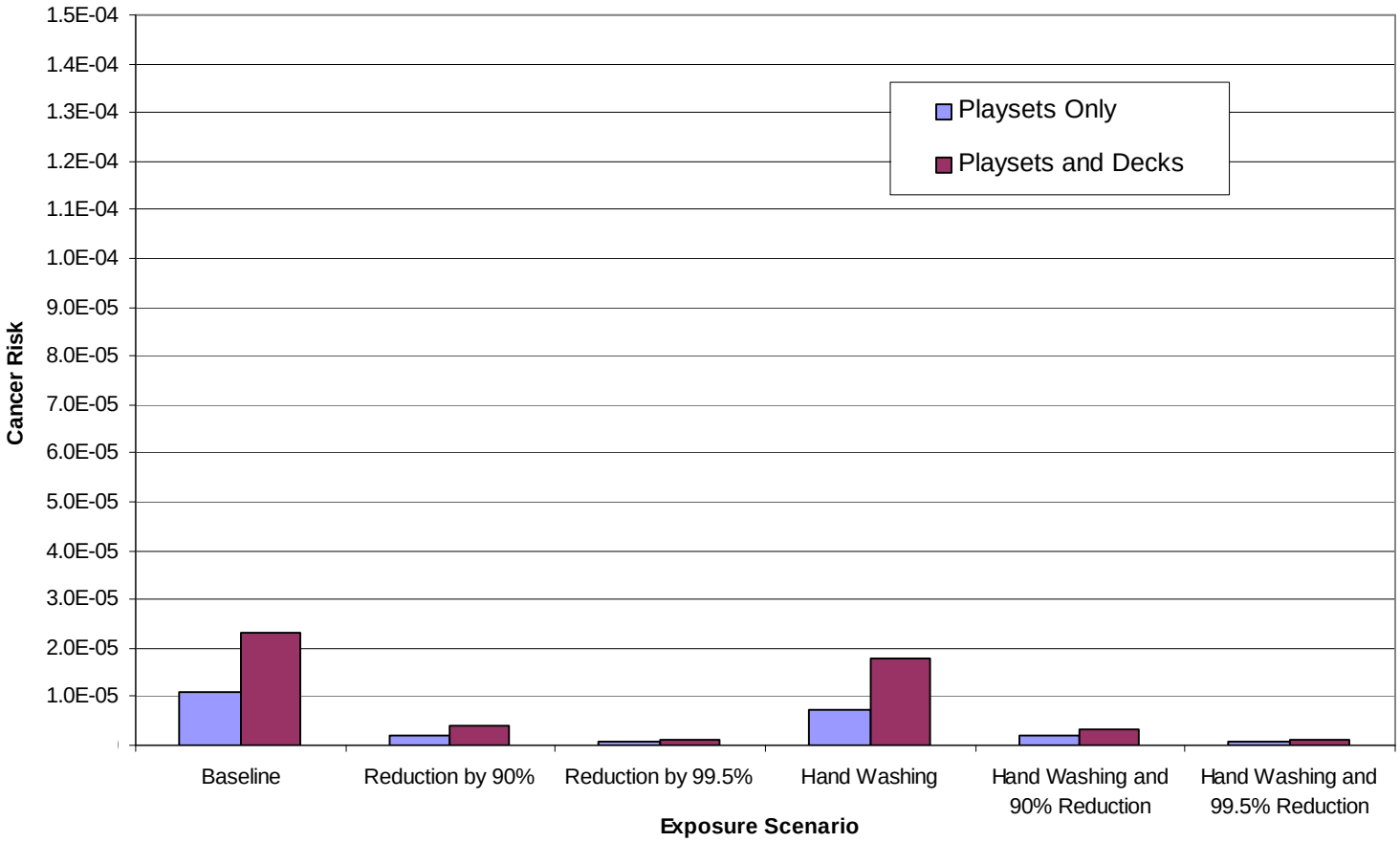
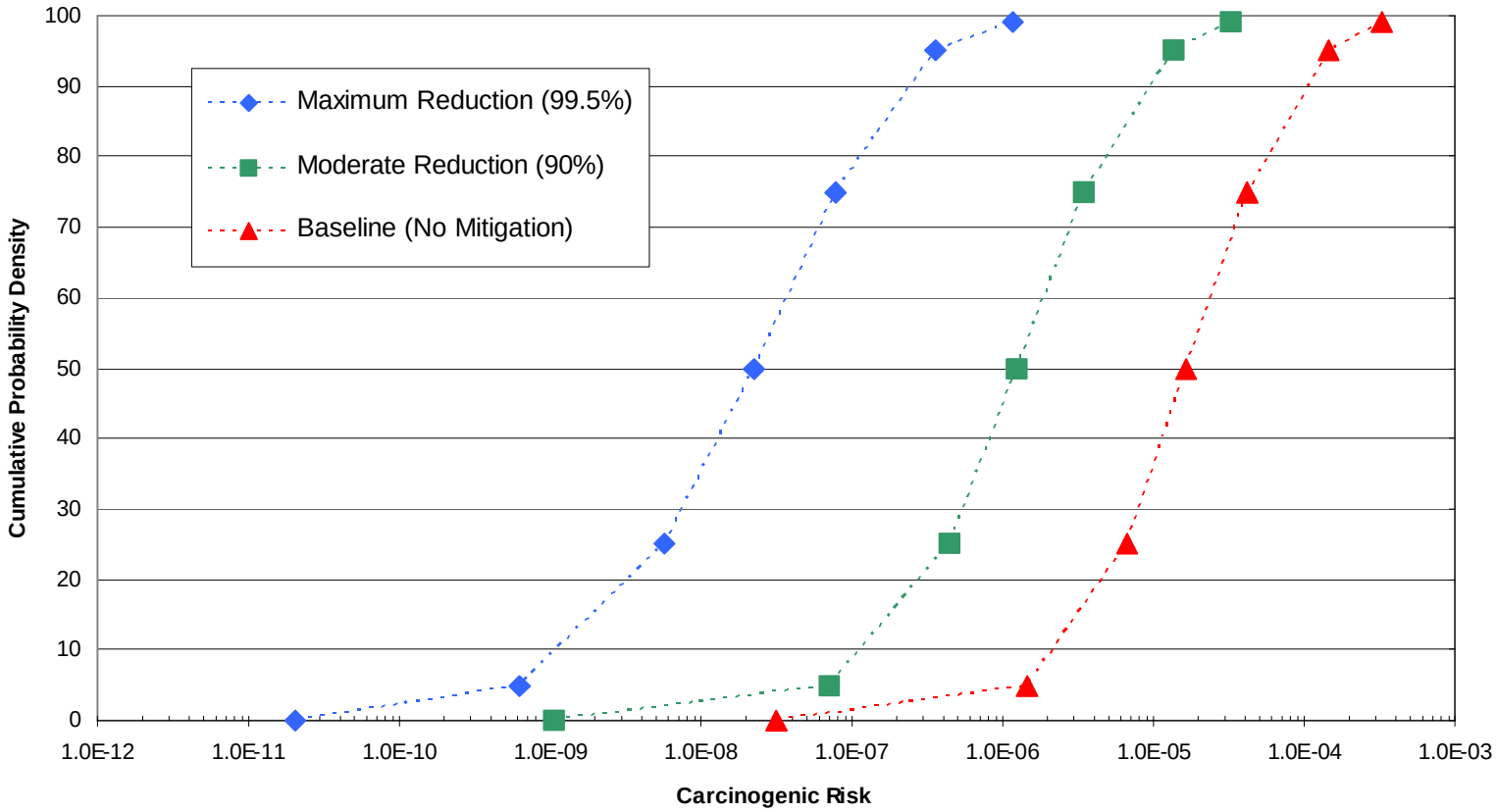


**Figure 1-3 Comparison of Residue Only Risks for Playsets and Decks for Warm Climate (Maximum Reduction, Moderate Reduction, Baseline)**



A qualitative assessment of uncertainty was conducted. Uncertainty in the risk characterization was a result of the combined uncertainty of the exposure assessment generated by SHEDS-Wood and the uncertainty in the toxicological factors. No quantitative evaluation of the uncertainty in the toxicity factors was conducted. A qualitative evaluation of the toxicity values showed that they were at the upper end of the range of a theoretical distribution because they incorporated several conservative assumptions. An in depth uncertainty and sensitivity analyses of the SHEDS-Wood exposure assessment was performed by Zartarian et al. (2003). For uncertainty, the two (out of six listed) most critical inputs were: transfer efficiency and residue concentration. Sensitivity analysis showed that the most influential variables were: transfer efficiency, residue concentration, fraction of hand mouthed, and amount of hand washing. Total uncertainty in the exposure assessment was estimated at a factor of 3-4.

Uncertainty was not modeled in this risk assessment. For carcinogenic risks, it is likely that the uncertainty is asymmetrical around the factor of 3-4 because slope factor accounts for several conservative assumptions. There is a low probability that the risks are higher, and a greater probability the risks are lower. For noncancer effects, the uncertainty is also asymmetrical. The MOE's are likely to be underestimated (i.e., they could be greater). Again, this is due to the LOAEL and NOAEL coming from the upper portion of the theoretical distribution. For chromium, there is the added conservative assumption that 10% of total chromium is present as Cr(VI). Taken together with the NOAEL, there is a much greater probability that the Cr(VI) MOEs are larger than those reported, and far lower probability that they are less than reported.

# 2.0 INTRODUCTION AND BACKGROUND

## 2.1 Introduction

The U.S. Environmental Protection Agency's (EPA) Office of Pesticide Programs (OPP) is aware of increased concerns raised by the general public, municipal and state governments, and state/federal regulatory agencies regarding the safety of children contacting arsenic and chromium residues while playing on Chromated Copper Arsenate- (CCA-) treated wood playground structures and decks. Because of this concern, OPP's Antimicrobials Division (AD), with the recommendation of the Science Advisory Panel (SAP) and the assistance of the Office of Research and Development (ORD), has conducted probabilistic assessments to evaluate potential childhood exposure to arsenic and chromium components of CCA-treated wood in decks, home playsets and public playground structures, and contaminated soils commonly found in these settings. This report focuses on the non-dietary assessment of CCA in treated wood.

OPP/AD's preliminary approach was reviewed by the SAP in 2001, which used a deterministic exposure assessment methodology for CCA-treated wood. SAP's primary recommendation to OPP was that a more comprehensive probabilistic assessment should be developed to examine the exposure scenarios presented in the deterministic assessment in 2001.

OPP requested the assistance of ORD in developing a model to conduct a probabilistic exposure assessment for CCA-treated wood. The Stochastic Human Exposure and Dose Simulation Model for the Wood Preservative Exposure Scenario (SHEDS-Wood), a probabilistic exposure model developed by the National Exposure Research Laboratory (ORD/NERL), was used to develop the exposure assessment for children exposed to CCA-treated playsets and decks. In 2002, SHEDS-Wood was presented to the SAP for model review and for recommendations from the panel. After incorporation of comments from the SAP, a draft document prepared by OPP and ORD in 2003, entitled *A Probabilistic Exposure Assessment for Children Who Contact CCA-treated Playsets and Decks Using the Stochastic Human Exposure and Dose Simulation Model for the Wood Preservative Exposure Scenario (SHEDS-Wood)* (Zartarian et al., 2003), together with this draft risk assessment report, *A Probabilistic Risk Assessment for Children Who Contact CCA-treated Playsets and Decks*, are scheduled to be reviewed by the SAP in December 2003. The SHEDS-Wood document provides exposures, reported as average daily doses (ADDs) and lifetime average daily doses (LADDs); it does not report risk estimates. The purpose of this report is to provide the results of a risk analysis, conducted by OPP, that uses the ADDs and LADDs generated by SHEDS-Wood in combination with toxicological endpoints for CCA (i.e., based on chromium and arsenic) selected by OPP. This document reports on children's risks to CCA using the multiple routes, multiple pathways, and dose estimates developed from the SHEDS-Wood draft document.

This OPP risk assessment provides background information on issues related to children's exposure to CCA-treated wood and the reasons that EPA conducted a non-dietary probabilistic assessment (see below); describes the exposures generated by SHEDS-Wood (see Chapter 3.0); summarizes the arsenic and chromium toxicity endpoints for children used in this risk assessment (see Chapter 4.0); characterizes the risks for the exposures presented in the SHEDS-Wood model (see Chapter 5.0); characterizes risk reduction impacts for the exposures presented in the SHEDS-Wood model (see Chapter 6.0); and discusses the uncertainties, strengths, and limitations of this risk assessment (see Chapter 7.0).

## 2.2 Background

Chromated Copper Arsenate (CCA) wood preservatives containing chromium (Cr), copper (Cu), and arsenic (As) as pesticidal compounds, protect wood from deterioration. They are predominantly used to pressure treat lumber intended for outdoor use in constructing a variety of residential landscape and building structures, as well as home, school, and community playground equipment. Children may potentially be exposed to the pesticide residues remaining on the surfaces of the treated wood structures as well as the residues leached into the surrounding soil. EPA is aware of increased concerns raised by the general public and state regulatory agencies regarding the safety of CCA-treated wood for residential applications. The children's risk assessment presented herein evaluates exposure routes and pathways anticipated as realistic, considering activity patterns and behavior of young children near residential playsets, public playsets, and residential decks. Children's exposure may occur through touching CCA-treated wood and CCA-contaminated soil near treated wood structures, mouthing hands after touching CCA-treated wood, and eating CCA-contaminated soil. Since EPA has determined that the arsenic and chromium components of CCA pose the most significant toxicity concerns in comparison to copper, which is not a recognized or suspected carcinogen, the Agency focused on evaluating potential adverse short-term, intermediate-term, and lifetime exposures and non-cancer/cancer risks to children from arsenic and chromium as Cr(VI). The SHEDS-Wood model developed by ORD was selected by OPP to conduct the probabilistic children's exposure and dose assessment for CCA (Zartarian et al., 2003). The exposure doses generated by SHEDS-Wood were used in conjunction with toxicity data for arsenic and chromium as Cr(VI) to estimate the risks presented in this report.

### 2.2.1 Regulatory History of CCA

Regulatory actions involving inorganic arsenical wood preservatives, including CCA, began nearly 25 years ago. An administrative review process was initiated in 1978 to consider whether the registration of certain wood preservative chemicals (pentachlorophenol; coal tar, creosote and coal tar neutral oil; and inorganic arsenicals) should be canceled or modified. A separate Notice of Rebuttable Presumption Against Registration and Continued Registration (RPAR) was issued for each heavy-duty wood preservative under consideration. A RPAR is

issued when the Agency determines that a pesticide meets or exceeds any of the risk criteria relating to acute and chronic toxic effects, as set forth under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Registrants then have the opportunity to submit evidence in rebuttal of the Agency's risk presumptions. The RPAR for inorganic arsenicals (43 FR 202) was published on October 18, 1978, along with a supporting Position Document (PD 1). According to that document, the risk criteria met or exceeded by inorganic arsenicals were: oncogenicity, mutagenicity, and fetotoxic/teratogenic effects. The RPAR generated substantial registrant comments, but these risks remained unrebutted after the RPAR process.

The Agency issued a Preliminary Notice of Determination (PND), concluding the RPAR process, which was published in the Federal Register of February 19, 1981 (46 FR 13020). This notice, along with the supporting Position Document (PD 2/3), stated the Agency's determination that the wood preservative chemicals continued to exceed the risk criteria which provided the basis of the RPARs. To reduce the risks, the Agency proposed certain modifications to the terms and conditions of registration, including certain protective clothing requirements, classifying all inorganic aresenical wood preservatives as Restricted Use (available to certified applicators only), and a mandatory program to provide users of treated wood with handling, use and disposal precautions.

The preliminary determinations described above were submitted to the FIFRA SAP and the U.S. Department of Agriculture (USDA) for review. Comments were also solicited from registrants and any other interested persons. The Agency considered the comments received and made modifications to the proposed decision announced in the PND. A public meeting was conducted on April 14, 1983 to allow interested persons to comment on the proposed changes. Their comments were considered in the development of the final determination, which was a Notice of Intent to Cancel (NOIC), published in the Federal Register of July 13, 1984 (49 FR 136), along with a supporting Position Document (PD 4).

Several trade associations and numerous registrants requested hearings to challenge the Agency's determinations in the July 13 NOIC. The Agency published a Federal Register Notice on October 31, 1984 (49 FR 43772), postponing the effective date of the labeling modifications for those registrants who filed applications for amended registration in response to the NOIC. On January 30, 1985, the Agency published an additional Federal Register Notice (50 FR 4269) announcing that persons other than registrants could continue to sell and distribute existing stocks of wood preservative products with existing labeling until further notice. Pre-hearing meetings were held between the Agency and some of the major parties who had requested hearings, during which alternative, mutually acceptable, mechanisms for achieving the regulatory goals set forth in the NOIC were discussed. After careful consideration of some of those alternatives, the Agency concluded that certain changes to the July 13, 1984 NOIC were appropriate and consistent with the Agency's goal of protecting the public from unreasonable adverse effects resulting from pesticide use. An amended NOIC, announcing these changes, was published in the Federal

Register of January 10, 1986 (51 FR 7). The modifications were mostly minor in scope, with the exception that the previous mandatory Consumer Awareness Program (CAP) was deleted from the labeling requirements. The wood preservative industry agreed to a voluntary CAP to educate consumers on the proper use and precautionary practices for treated wood.

Arsenic, chromium, and chromated arsenical compounds, used as wood preservatives, were evaluated under the Registration Standards Program in 1988. This program was established in order to provide a mechanism for pesticide products having the same active ingredient to be reviewed and brought into compliance with FIFRA. The outcome of the Registration Standard for arsenic, chromium, and chromated arsenical wood preservatives was as follows:

- Classification of inorganic arsenic and hexavalent chromium as Group A carcinogens;
- Acknowledgment that both arsenic and chromium have demonstrated the potential to cause teratogenic/fetotoxic effects through peritoneal exposure;
- Requirement of a reproduction study using a formulated chromated arsenical product to address the teratogenic/fetotoxic effects unless a metabolism study demonstrated that blood levels of chromium and arsenic are not increased above background levels;
- Requirement of metabolism data to assess the bioavailability of chromium and arsenic after exposure to a formulated product;
- Requirement of additional ecological effects and environmental fate data; and
- Reiteration of label restrictions set forth in the prior NOICs.

Currently, the only remaining use of arsenic acid is for wood preservation. The last remaining agricultural use of arsenic acid, as a desiccant on cotton, was voluntarily canceled in 1993 (58 FR 86, May 6, 1993). The voluntary cancellation was enacted following a NOIC issued for the cotton desiccant use of arsenic acid (56 FR 50576, October 7, 1991) due to the cancer risks to workers. The voluntary cancellation allowed the sale of existing stocks until December 31, 1993, after which they could be lawfully disposed of or sold to the wood preservative industry for reformulation or repackaging into registered wood preservative products.

### 2.2.2 Current Development of CCA Issue

On March 17, 2003 EPA granted the voluntary cancellation and use termination requests affecting virtually all residential uses of CCA-treated wood. Under this action, affected CCA products cannot be used after December 30, 2003 to treat lumber intended for use in most residential settings. This transition affects virtually all residential uses of wood treated with CCA, including play structures, decks, picnic tables, landscaping timbers, residential fencing, patios, and walkways/boardwalks. This action was proposed in February 2002 by the registrants of CCA pesticide products that are used to treat wood. Phase-out of the residential uses will reduce the

potential exposures and risks from arsenic, a known human carcinogen, thereby protecting human health, especially children's health, and the environment. The current action follows the February 2002 publication of a notice of receipt of voluntary cancellation/use termination requests, which also provided an opportunity for public comments to be submitted to EPA. A notice of the cancellation order was published in the Federal Register on April 9, 2003. Consumers may continue to buy and use the treated CCA wood for as long as it is available, but the transition to using the new generation treatment products is well underway. The Agency is deferring any action on two uses involved in the termination requests: (1) wood used in permanent wood foundations; and (2) wood used in fence posts for agricultural uses. Therefore, these two products may continue to be treated with CCA at this time. EPA is working with the registrant community and other stakeholders to ensure that safer, comparable alternatives will be available. EPA is continuing its work on an ongoing comprehensive reevaluation of CCA-treated wood that has been underway as part of the Agency's effort to re-evaluate older pesticides to ensure that they meet current health and safety standards. More information on CCA-treated wood is available at the following EPA website:
http://www.epa.gov/pesticides/factsheets/chemicals/1file.htm

The Agency is evaluating CCA under the reregistration process within OPP. Once OPP completes the reregistration review for CCA, the Reregistration Eligibility Decision (RED) document for Chromated Arsenicals will be released. The RED will include a comprehensive assessment of the potential human impacts (preliminary focus on occupational and environmental exposures/risks attributed to the use of CCA-treated wood and related inorganic chromated arsenical pesticides at the workplace) as well as potential impacts on the environment.

#### 2.2.2.1 CPSC Activities

On March 17, 2003, the U.S. Consumer Product Safety Commission (CPSC) staff held a Commission Briefing to respond to the petition from the Environmental Working Group (EWG) and the Healthy Building Network (HBN) to ban the CCA-treated wood being used in playground equipment and to review the safety of CCA-treated wood for general use (CPSC, 2003a). After briefing the Commissioners and the public on CPSC's deterministic risk assessment, CPSC staff recommended denial of the petition based on the actions of EPA (CPSC, 2003a). On November 4, 2003, CPSC voted unanimously that a ban was not necessary because the wood industry no longer uses CCA-treated wood for playsets. CPSC's decision was based on an agreement between CCA manufacturers and the Environmental Protection Agency (EPA) to phase out CCA treatment of wood for most consumer uses by the end of 2003. More information on CPSC's briefing on CCA-treated wood is available at the following website:
http://www.cpsc.gov/cpscpub/prerel/prhtml04/04026.html

#### 2.2.2.2 Updated International Actions and Activities

The European Commission (EC) has banned the sale of CCA-treated wood for most residential uses, effective June, 2004 (CPSC, 2003b; APVMA, 2003; EMRA, 2003a). However, none of the countries banned CCA-treated wood that is already in use (EMRA, 2003a). EC countries include Germany, Belgium, Luxembourg, France, Portugal, Spain, Italy, Greece, Austria, the United Kingdom, Ireland, Finland, Sweden, Denmark and The Netherlands (EMRA, 2003a).

The EC published a Marketing and Use Directive on January 6, 2003 stating "labeling requirements for CCA-treated wood, and banning the sale of CCA-treated wood unless structural integrity of the wood is needed for human or livestock safety and skin contact by the public is unlikely. The directive is to take effect by 30 June 2004 and applies only to CCA Type C preservatives. Situations in which CCA preservatives may not be used include residential or domestic constructions, where there is a risk of repeated skin contact, and where the wood may come into contact with intermediate or finished products intended for human consumption. The directive does not apply to CCA-treated wood already in use" (EMRA, 2003a).

"Restrictions on use of CCA already exist in a number of member states. Germany, Sweden, Austria, Finland, the Netherlands and Denmark had already initiated voluntary agreements or regulations restricting the use and marketing of CCA and CCA-treated wood." (EMRA, 2003a). However, for the United Kingdom, the Health and Safety Executive recommended to the government in 1999 to continue using CCA-treated wood with certain environmental data and occupational requirements (EMRA, 2003a).

Canada's Pest Management Regulatory Agency (PMRA) has reached an agreement with industry on the proposed transition away from the use of CCA-treated wood at residential sites. The PMRA agreement is identical to the voluntary label changes for CCA-treated wood that were proposed by EPA. Canada's Consumer Safety Information Sheet can be found at http://www.ccasafetyinfo.ca. and Fact Sheet information can be found at http://www.hc-sc.gc.ca/pmra-arla/english/pdf/fact/fs_cca-june2003-e.pdf

In Australia, the Australian Pesticides and Veterinary Medicines Authority (APVMA) has initiated reconsideration of the registration and associated label approval of products containing arsenic. It is anticipated that a draft report of APVMA's review of arsenic will be available for public comment in mid-2004 (APVMA, 2003).

## Table 2-1. International Regulatory Actions and Activities Related to CCA

| International Community | Summary of Action and Activities | | Website Source |
|---|---|---|---|
| USA | EPA<br>CCA is currently undergoing reregistration review by EPA. EPA granted the voluntary cancellation and use termination requests affecting virtually all residential uses of CCA-treated wood. Under this action, affected CCA products cannot be used after December 31, 2003, to treat lumber intended for use in most residential settings. EPA provides public health information on arsenic in pressure treated wood and provides safety recommendations for homeowners and additional information on their website. EPA is also awaiting results from ongoing work performing studies regarding the effectiveness of sealant on wood structures. | CPSC<br>On March 17, 2003, CPSC held a Commission Briefing to respond to the petition from the Environmental Working Group (EWG) and the Healthy Building Network (HBN). After briefing the Commissioners and the public on their deterministic risk assessment, CPSC deferred their decision on the petition pending final EPA action. CPSC provides public health information on arsenic in pressure treated wood and provides safety recommendations for homeowners and additional information on their website. | http://www.epa.gov<br>http://www.cpsc.gov |
| Canada | CCA is currently undergoing reregistration review by PMRA in collaboration with EPA. PMRA granted the voluntary cancellation and use termination requests affecting virtually all residential uses of chromated copper arsenate (CCA) treated wood. Under this action, affected CCA products cannot be used after December 31, 2003 to treat lumber intended for use in most residential settings (Personal Communication, 2003; PMRA, 2003). PMRA provides public health information on arsenic in pressure treated wood and provides safety recommendations for homeowners and additional information on their website. | | http://www.hc-sc.gc.ca/pmra-arla/english/index-e.html |
| Europe | EC will consider banning the sale of CCA-treated wood. Countries include Germany, Belgium, Luxembourg, France, Portugal, Spain, Italy, Greece, Austria, the United Kingdom, Ireland, Finland, Sweden, Denmark and The Netherlands (EMRA, 2003). The Scientific Committee on Toxicity, Ecotoxicity and the Environment (SCTEE) provided an assessment of risk to health and the environment of arsenic in wood preservatives in 1998. SCTEE recommended use of arsenic-based timber treatment to situations where it is 'absolutely necessary' (APVMA, 2003). EC member countries must publish the provisions necessary to comply with the directive in June 2003 and apply the provisions by June 2004. SCTEE provides public health information on arsenic in pressure treated wood and provides safety recommendations for homeowners and additional information on their website. | | http://www.europa.eu.int |
| Australia | The APVMA has initiated reconsideration of the registration and associated label approval of products containing arsenic. The reconsiderations will be made after APVMA has assessed all data and other information provided to it for this purpose. It is anticipated that a draft report of APVMA's review of arsenic will be available for public comment in mid-2004 (APVMA, 2003). APVMA provides public health information on arsenic in pressure treated wood and provided safety recommendations for homeowners on their website. | | http://apvma.gov.au |
| New Zealand | Additional research was commissioned on public health risks related to CCA, particularly around homes and playgrounds. EMRA has decided against a reassessment of registrations of CCA. However, EMRA is currently reviewing labeling procedures, disseminating public health information on CCA, assessing alternatives to CCA, etc. For public CCA-treated playsets, EMRA is not taking action on existing facilities. However, the government is working with schools on ways to reduce exposure to CCA (e.g., using coatings) on publically-maintained playsets (EMRA, 2003b). | | http://mfe.govt.nz |

New Zealand has also commissioned additional research on public health risks related to CCA, particularly around homes and playgrounds (EMRA, 2003a; APVMA, 2003). Based on an internal review of public health risks, Environmental Risk Management Authority (EMRA) has decided against a reassessment of registrations of CCA. However, EMRA is currently reviewing labeling procedures, disseminating public health information on CCA, assessing alternatives to CCA, etc. For public CCA-treated playsets, EMRA is not taking action on existing facilities. However, the government is working with schools on ways to reduce exposure to CCA (e.g., using coatings) on publically-maintained playsets (EMRA, 2003b). Table 2-1 summarizes international regulatory actions and activities related to CCA.

#### 2.2.2.3 Updated State Actions and Activities

In 1987, the California Department of Health Services (CDHS), Health and Welfare Agency, conducted a research study entitled, *Evaluation of Hazards Posed by the Use of Wood Preservatives on Playground Equipment,* and made recommendations to the Legislator in the State of California (CDHS, 1987). As a result of the findings and recommendations of that report, a new law was signed into effect in September 1987 (Div. 20 of the California Health and Safety Code, §25930.10.7) (Spease, 2002). The law stated that:

- State funds could not be used to purchase wooden playground or recreational equipment that may have been treated with arsenic (unless treated in accordance with AWPA standard C-17), pentachlorophenol or creosote;
- State funds may not be used for maintenance of the wooden playground or recreational equipment in question; and
- People installing any such structures must seal the structures with a non-toxic, non-slip sealer at the time of installation, and reseal the structure every two years.

Maine legislators approved the Nation's first ban on the sale of wood treated with arsenic on June 4, 2003. The bill states "that beginning April 1, 2004, Maine lumber dealers can no longer sell arsenic-treated lumber for use in residential construction" (Edgecomb, 2003). Additionally, retailers are prohibited from purchasing arsenic-treated wood for most residential uses (mid-September 2003). The Maine Department of Environmental Protection (MDEP) must complete a market evaluation of remaining uses. The Maine Bureau of Health must develop informational brochures to educate consumers by January 1, 2004, on what homeowners should know about hazards, and methods for reducing exposures with sealants. By January 1, 2005, the MDEP must develop plans to restrict the disposal of arsenic-treated wood (Our Stolen Future, 2003).

In New York, Section 37-0109 of the New York State Environmental Conservation Law makes it illegal for schools and public playgrounds to have playground equipment constructed from pressure treated lumber that contains CCA. The law requires that existing playground

equipment be sealed to stop CCA from leaching or escaping from the wood, and to cover the ground to protect children from arsenic that may have leached to the soil. The Department of Environmental Conservation (DEC) is to publish information on the dangers and hazards to public health and the environment from the use of CCA-treated lumber. The DEC is to compile and publish a list of less toxic materials that may be used on playgrounds as an alternative to CCA-treated lumber. The DEC will also compile and publish information on non-toxic methods and materials that are available to seal playground structures with CCA wood and to cover the ground (Healthy Schools Network, 2003).

Other state agencies such as the Connecticut Department of Public Health, the Massachusetts Department of Public Health, the Florida Department of Environmental Protection, and the Minnesota Department of Health have been actively investigating issues related to pressure-treated playground equipment. They have provided public health information on arsenic in pressure-treated wood, as well as safety recommendations for homeowners (see websites listed in Table 2-2). These recommendations include:

- Sealing CCA-treated structures (decks and playsets) every two years with oil-based stain;
- Preventing exposure to pressure-treated wood and dust;
- Washing hands after playing on wooden playground equipment;
- Inspecting structures for decay;
- Suggesting alternatives to CCA-treated pressure treated wood;
- Not placing food, drink or paper products on pressure treated wood;
- Never burning treated wood;
- Limiting use of under deck areas where arsenic may have accumulated in the soil;
- Not using treated wood on indoor surfaces; and
- Not using CCA-treated wood for wood chips or mulch.

Table 2-2 presents a summary of state regulatory activities and actions related to CCA.

**Table 2-2. State Regulatory Actions and Activities Related to CCA**

| State | Summary of Actions and Activities | Website Source |
|---|---|---|
| California | In 1987, the California Department of Health Services (CDHS), Health and Welfare Agency conducted a research study entitled, *Evaluation of Hazards Posed by the Use of Wood Preservatives on Playground Equipment* and made recommendations to the Legislator in the State of California (CDHS, 1987). As a result of the findings and recommendations of that report, a new law was signed into effect in September 1987 (Div. 20 of the California Health and Safety Code, §25930.10.7). Legislation required that publically-maintained wooden playground or recreation equipment be treated with a certain formulation of CCA. This legislation also required that existing publically-maintained wooden playground/recreation structures made with arsenic-treated wood be sealed with a non-toxic and non-slippery sealant every two years. CDHS provides public health information on arsenic in pressure-treated wood, safety recommendations for homeowners, as well as additional information on their website. | http://www.dhs.cawnet.gov |
| Connecticut | The Connecticut Department of Public Health provides public health information on arsenic in pressure treated wood, safety recommendations for homeowners, as well as additional information on their website. | http://www.state.ct.us/dph |
| Florida | Proposed legislation would prohibit the public use of CCA-treated wood in playground structures and associated ground covers that are constructed or contracted for by October 1, 2003. It would require that existing publically-maintained wooden playground/recreation structures made with arsenic treated wood be sealed with a non-toxic and non-slippery sealant every two years. Florida Department of Environmental Protection provides public health information on arsenic in pressure-treated wood, safety recommendations for homeowners, as well as additional information on their website. | http://www.dep.state.fl.us |
| Maine | Legislature approved a bill that states " beginning April 1, 2004, Maine lumber dealers can no longer sell arsenic-treated lumber for use in residential construction." Additionally retailers are prohibited from purchasing arsenic- treated wood for most residential uses (mid-September 2003). The Maine Department of Environmental Protection (MDEP) must complete a market evaluation of remaining uses. The Maine Bureau of Health must develop informational brochures by January 1, 2004, on what homeowners should do know about hazards, and methods for reducing exposures with sealant. By January 1, 2005, the MDEP must develop plans to restrict the disposal of arsenic treated wood. | http://www.state.me.us/dhs/boh<br>http://www.state.me.us/dep |

**Table 2-2. State Regulatory Actions and Activities Related to CCA (Continued)**

| State | Summary of Actions and Activities | Website Source |
|---|---|---|
| Massachusetts | The Massachusetts Department of Public Health provides public health information on arsenic in pressure-treated wood and safety recommendations for homeowners on their website. | http//:www.state.ma.us/dph |
| Minnesota | In Minnesota, a bill has been introduced that would ban the use and sale of CCA in the state. A second Minnesota bill would require that schools that use CCA-treated products seal the wood every two years (Environmental Health Perspectives, 2001). Minnesota Department of Health provides public health information on arsenic in pressure treated wood, safety recommendations for homeowners, as well as additional information on their website. | http//:www.health.state.mn.us |
| New York | In New York, Section 37-0109 of the New York State Environmental Conservation Law makes it illegal for schools and public playgrounds to construct playground equipment from pressure-treated lumber that contains CCA. The law requires that playgrounds be sealed to stop CCA from leaching or escaping from the wood, and to cover the ground to protect children from arsenic that may have leached to the soil. The New York Department of Environmental Conservation provides public health information on arsenic in pressure-treated wood, safety recommendations for homeowners, as well as additional information on their website. | http//:www.dec.ny.us |

### 2.2.3 Use Profile of CCA

CCA preservatives protect wood from deterioration from a variety of insects, fungi, and rot organisms. There are currently 26 CCA-containing wood preservative products registered with the EPA. CCA is used for pressure-treated lumber intended for outdoor use in constructing a variety of residential landscape and building structures, as well as home, school, and community playground equipment. However, it should be noted that EPA granted the voluntary cancellation and use termination requests of CCA-treated wood. The labels for the three CCA-containing preservatives that contained the non-pressure treatment uses were effectively canceled via a 6(f) notice on May 16, 2003. A final cancellation order was issued on May 28, 2003 for Osmose Special K-33 Preservative (EPA Registration 3008-21), Hollow Heart Concentrate (EPA Registration 75341-1) and Osmoplastic SD Wood Preserving Compound (EPA Registration 75341-7). The cancellation of these three products resulted in pressure treatment being the only allowable use for CCA-containing preservatives. CCA is used for pressure-treated lumber intended for outdoor use in constructing a variety of residential landscape and building structures, as well as home, school, and community playground equipment. However, it should be noted, EPA granted the voluntary cancellation and use termination requests affecting virtually all residential uses of CCA-treated wood. CCA-treated wood, predominantly of Southern yellow

pine, represents the majority of pressure-treated dimensional lumber marketed to the general consumer via lumberyards/hardware stores and other retailers. In some cases, CCA-treated lumber is recycled into wood chips which are stained, then sold to consumers as landscape mulch. Major commercial installations include utility poles, highway railings, roadway posts/barriers, bridges, bulkheads, and pilings. Industry cites advantages of CCA-treated wood over other pressure-treated wood, including superior durability, low-odor, and dry "non-oily" surfaces which can be painted or sealed.

There are three formulations of CCA, each containing varying ratios of arsenic pentoxide, chromic acid, and cupric oxide. CCA treatment solutions are typically classified by the American Wood-Preservers' Association (AWPA) as either type A, B, or C, with CCA type C (CCA-C) being the formulation most commonly used for pressure treating dimensional lumber for residential applications. AWPA's P5 Preservative Standard requires CCA-C composition to be 34.0% arsenic pentoxide ($As_2O_5$), 47.5% chromic acid ($CrO_3$), and 18.5% cupric oxide (CuO) (AWPA, 1998).

After pressure treatment and fixation, arsenic and chromium can be retained in the wood from 0.25 to 2.50 pounds per cubic foot (pcf), based on the retention of CCA-C in wood following AWPA treatment standards. Typical retention levels achieved depend on the intended applications of the treated lumber. Lower retention values are required for plywood, lumber, and timbers used for above-ground applications (0.25 pcf ), and for ground or freshwater contact uses (0.40 pcf). Higher retention levels are required for load bearing wood components, such as pilings, structural poles, and columns (0.60 - 0.80 pcf). The highest levels are required for wood foundations and saltwater applications (up to 2.50 pcf).

Nationwide, approximately 70% of single family homes have existing pressure-treated decks and porches, and approximately 14% of public playground equipment is made with treated wood. Based on current data from the American Chemistry Council (ACC), approximately 34% of CCA was used for decks and less than 1% was used in playground equipment (Zartarian et al., 2003; CPSC, 2003b). The potential for exposure to pesticide residues remaining on the surfaces of the existing aged treated wood structures as well as to the residues leached into the surrounding soil, may pose child health hazard concerns.

### 2.2.4 Overview of CCA Chemistry

CCA contains chromium (Cr), copper (Cu), and arsenic (As), each of which contributes to the wood-preservative properties of the compound. Copper acts as a fungicide in the CCA formulation and the arsenic protects against insect damage. Chromium, in the form of chromic acid, acts as a fixative (binding agent), whereby the Cr, Cu, and As metal ions present in the wood are fixed to the wood fibers. Most of the information presented in this overview is from U.S. EPA (2001b).

Metals go through various changes in environmental compartments such as soil, water, plants, and animals. The speciation of metals depends on sorption, desorption, redox reactions in soil and water, precipitation reactions, complexation reactions, etc. (Lebow, 1996). The different species of arsenic and chromium vary in their ability to be absorbed into the body and metabolized within the body, and differ in their toxicological profiles. Therefore, it is important to consider the species of arsenic or chromium present in soils surrounding CCA-treated wood and on the surface of the treated wood itself when assessing the exposure to these chemicals.

#### 2.2.4.1 Speciation

The FIFRA SAP (U.S. EPA, 2001c) noted that there is no reliable evidence on either the presence or absence of Cr(VI) in dislogeable residues on treated wood surfaces. However, since that meeting, more studies have indicated that Cr(III) is the primary component on treated wood surfaces. The FIFRA SAP also noted that some measurable Cr(VI) probably exists in certain soils, but it is unlikely to be 100 percent of the total chromium present. One approach recommended by FIFRA SAP in evaluating the hazards of chromium in the soil would be to use an estimate of 5 to 10 percent (or more conservatively 25 to 50 percent) Cr(VI).

More recent studies indicated that Cr(III) is the primary component in CCA pressure-treated wood surfaces of existing decks and playground structures (RTI International, 2003 (cited as ACC, 2003b in SHEDS-Wood Report); Cooper, 2003; Nico et al., 2003) and in the air of treatment plants (ACC, 2002). In fact, RTI International (2003) found that Cr(VI) was not detected in 142 of 145 wood surface dislogeable residue samples taken; Cr(VI) was not detected in any of the samples from existing aged decks, and only trace amounts were detected in the newly treated woods in the remaining samples. The registrants of CCA conducted a CCA treatment plant worker exposure study in 1999 (ACC, 2002). This study also indicated that the Cr(VI) in the air was undetectable (based on the sensitivity of the limit of detection of Cr(VI) used in that study). Nico et al. (2003) found that chromium and arsenic in CCA-treated wood were consistent between samples of fresh treated wood and aged wood, and between treated wood and dislogeable residue. The Nico et al. (2003) report indicated that a “chemical complex” type of matrix was formed between As-Cr-Wood. However, the Nico et al. (2003) report did not quantify the matrix type of CCA-treated wood and the free metal forms of arsenic and chromium.

#### 2.2.4.2 Fixation

After undergoing pressure treatment with CCA wood preservative, the chromium, copper and arsenic penetrate into the wood and become bound or fixated in the wood. The term, fixation, refers to the series of chemical reactions that take place after the wood has been pressure treated with CCA. These reactions render the CCA less likely to leach from the wood during service. The use of metal oxides in CCA formulations has been shown to aid in the fixation process. Fixation precedes the actual action of CCA to act as a wood preservative. The CCA penetration/fixation process preserves and protects the wood from pest attack. The absorption and fixation of CCA apparently occur in the cellulosic and lignin components of the wood (Kartal

and Lebow, 2000). Since lignin is thought to be a primary binding site for chromium to form chromium-lignin complexes, the use of woods with an increased lignin content may result in improved treatment. Softwood species, which have a high lignin content often perform better than hardwoods in terms of preservative treatment. Studies have shown that all of the three metals are fixed into the wood structure.

The initial reaction of fixation is the absorption of the CCA preservative into the cellulosic and lignin components of the wood. A second reaction occurs which converts Cr(VI) to Cr(III). This second reaction continues for a period of several hours to a few days. The reduction of Cr(VI) to Cr(III) is important in the formation of insoluble complexes in CCA-treated wood. Additionally, Cr(III) is less toxic than Cr(VI). The third reaction is the conversion of copper arsenate in the wood to basic copper arsenate with an arsenic valence state of +5. The complete fixation reaction may even take several months. Studies with treated pine have indicated that the copper and arsenic components of the CCA metals are "fixed" more rapidly than chromium. Some researchers have concluded that the fixation process is complete when the presence of Cr(VI) is no longer detected in the leachate or compensate of the treated wood. Cooper (2003) conducted research on CCA fixation using existing data and noted that virtually all of the chromium injected into the wood during the treating process is eventually reduced to low toxicity Cr(III) and there is no evidence that Cr(VI) is produced as a result of the oxidation of Cr(III) in the wood. The completion of the fixation process can be from a few days to a several months, depending on the ambient temperature of treatment plants.

#### 2.2.4.3 Leaching

The fixation process binds much of the chromium, copper, and arsenic into the wood fibers; however, some of the metals will not be "fixed" and will remain "free" on the surface of the treated wood. These will be susceptible to dislodging through washing off or by physical contact with other objects, including humans who have physical contact with the wood. The fixated metals can also slowly be leached from the treated wood by water.

Playground equipment constructed with treated wood can be in the form of many different types of items including swing sets, climbing bars, etc. The chromium, copper, and arsenic in/on the treated wood can be leached from the wood so that the metals fall vertically onto the soil under the equipment and leach laterally into the soil from the vertical pieces of treated wood that have contact with the playground soil. Metals also leach from ground-contact horizontal pieces of CCA-treated wood fabricated into playsets and related structures. Playground equipment may also have mulch placed under the equipment, and the mulch will receive leachate from the treated equipment pieces. Children playing on such equipment can be exposed to the CCA leachates either through contact with the CCA-treated wood or through contact with soil or mulch either under the equipment or immediately adjacent to the equipment.

A large amount of data are available regarding the leaching of chromium, copper, and arsenic from treated wood (Lebow, 1996). Much of the data are from studies that are not directly applicable to leaching from playground equipment. Some of the available data that are most applicable to playground equipment and decks constructed of CCA-treated wood are summarized below.

Leaching of chromium, copper, and arsenic from treated wood in an aqueous medium, which is most likely to simulate the playground use (where rainfall occurs), appears to be most rapid from freshly treated wood and is in the order of Cu > As > Cr. The release rate is also higher under acidic conditions; this would mean that leaching would be faster in the areas of the United States that have acid rain, such as the northeastern states. One study has shown that the leaching process from treated wood is aided by slow or drizzling rain rather than heavy showers. Leaching rates are generally lowest in wood that has been kiln-dried at high temperatures.

Most of the leaching from treated wood appears to take place in the first few days after treatment, but continues slowly over time (Lebow, 1996). Leaching rates depend on the size of the wood, type of wood, and on the fixation process. CCA leaches from hardwood more than soft wood. Pressure treated red pine leaches more than lodgepole pine and Douglas fir. A scheme has been proposed in the literature for the long-term leaching mechanism of CCA from wood: reversible disassociation of ion-exchanged metals and their redistribution to the wood surface and their loss; and physical or biological decay of the wood.

No leaching information was found to address the question of whether CCA metals leach from treated wood as copper or copper arsenate, or as complexes with inorganic or organic ligands, or as derivatives of wood-metal moieties or as water soluble extracts. Water mobility for the metal ions from CCA depend on many factors which give rise to a number of pathways. The metals can diffuse through the soils as complexes, simple salts or free ions, or can percolate through soils as insoluble substances.

Little data were found to estimate the level of CCA residues in soil or mulch under playground equipment constructed of treated wood. A Canadian study evaluated wooden play structures consisting mostly of CCA-treated lumber of various dimensions constructed in a range of designs (Riedel et al., 1991). The structural elements were comprised of beams and planks fastened together. Poles were cut and used to form rungs, ramps and ladders. Treated wood pieces were used to construct tower-like structures and to connect to swings, slides, ladders or horizontal monkey bars. Some structures incorporated hut-like shelters. Treated wood pieces were placed in vertical, horizontal and angled positions. Some structures were coated with an oil-based stain which had worn off in some areas. The structures were up to ten years old. The ground under the structures and surrounding the structures usually consisted of a layer of sand at least 25 centimeters deep which is replaced or replenished from time to time. The sand is carried onto the structures and contributes to the abrasion and wear on the treated wood pieces.

Sand and soil samples were taken from under each of the treated playground structures and a control soil sample was taken at a distance of ten meters (33 feet) from the treated playground structure. The sand samples were taken at similar locations under each structure; at the bottom of a slide, next to a support post, at the bottom of a support post holding the main structure, and underneath a wooden platform or underneath a structure approximately one meter from the wooden post. The samples were all taken in late fall and on a cloudy day. The soil samples were stored in plastic bags and taken to the laboratory for analyses. The samples were oven dried and analyzed using inductively coupled plasma mass spectrophotometry for total nitric acid soluble arsenic (not speciated). Neither chromium nor copper were analyzed in the sand and soil samples. The background levels of arsenic present in the control sand samples were generally less than 0.3 parts per million (ppm). The authors of the paper reported that the average arsenic residue level from samples taken from below the treated structures was 3.0 ppm with a range of 0.032 - 9.6 ppm. However, sand samples taken from other areas around the playground structures showed arsenic residues ranging from 0.13 ppm to 113.5 ppm under a structure next to a post. It should be noted that arsenic residues in sand sampled next to a treated post were less than 10 ppm except in the one playground with the high 113 ppm value. That study showed significantly higher sand residues than the other playground studies. There is no explanation for this difference, but could be due to reasons such as samples being taken near newly treated and replaced wood posts. Additionally, sand had been placed under the structures and leaching from wood posts into the sand may be more rapid and spread further from the post than would be the case for arsenic leaching into a clay soil. It could also be argued that if wood mulch rather than sand had been placed under the playground structures that, because of the surface area to weight relationship for this organic material, any arsenic residues leaching from treated wood could result in even higher arsenic residues in the mulch under the playground equipment.

The playground where arsenic residues were highest was ten years old and constructed of wood that had been stained, but on which the stain had been worn off. There did not appear to be a correlation between residue levels in the sand under and around the playground structures and whether the equipment had been stained or painted, or was left unsealed.

There are also data available showing soil residue levels that occur under wooden decks that have been constructed from CCA-treated wood. Children can play in the soil under and around a treated deck. While the deck data may exaggerate residue levels in soil compared to what would be expected under playground equipment, the data show that the level of CCA metals in soil under treated wood structures was greater than the background level of the metals in soil from the study location and show residue levels in soil where children could play.

In one study conducted by Stilwell and Gorny (1997), soil from under seven decks constructed from CCA-treated wood were analyzed. Chromium levels ranged as high as 154 ppm under the treated decks and averaged 43 ppm, whereas, the control soils had an average of 20 ppm of chromium. Arsenic levels ranged as high as 350 ppm under the treated decks and averaged 76 ppm, whereas, the control soils had an average of 3.7 ppm of arsenic. No data are available for mulch under the treated deck, but residues in mulch may even be higher because of the surface

area weight relationship of mulch. The same study showed that those decks that had been coated tended to show a lesser degree of leaching of CCA metals. However, the degree of leaching from a deck that had been coated or sealed would most likely be dependent on the coating product used and on the age of the coating. The same study also showed that the age of the deck was a factor in the leachate residues found under the treated deck, with the older deck showing higher soil residues under the treated deck. This study does not reflect the soil CCA residue levels that could occur under treated playground equipment, but the generalization can be made that CCA residues in soil under treated playground equipment will be higher than soil background levels of the CCA metals in the surrounding area. The residue data from this study do not speciate the metals but determine total copper, chromium, and arsenic.

Lateral and vertical migration of CCA metal residues can also occur from vertical pieces of the playground equipment that have contact with the soil. In a study conducted by DeGroot et al. (1979), treated southern pine wooden stakes were placed in sandy soil, and the lateral and vertical migration of CCA metal residues were measured after 30 years. Both arsenic and chromium residues leached into the top six inches of a soil core, arsenic as high as 108 ppm and chromium as high as 25 ppm. Some increase in arsenic levels, but not chromium levels, was seen in the six- to twelve-inch core. In the twelve- to eighteen-inch core, there did not appear to be any increase in the arsenic and chromium level. In soils which have a high clay or organic content, metal leaching would be expected to be lower because of the metal binding to the soil particles. Lateral movement of residues in the soil surrounding the stakes appeared to be limited to the zero- to three-inch area surrounding the treated stakes. Based on the findings in this and other studies, CCA metal residues are not likely to leach from vertically-placed wood structures placed in contact with the soil to depths greater than twelve inches or to lateral distances from these treated wood pieces of greater than three inches.

In another study conducted in Florida with CCA-treated decks (Townsend et al., 2001), nine decks were studied (one deck could not be confirmed as treated with CCA). The decks were located in Gainesville, Miami, and Tallahassee and sampling was conducted in 1999. The decks varied in age from two to nineteen years old. A grid was set up under each deck before sampling where soil samples were collected. Surface samples, from the top inch of soil, and soil core samples, of approximately seven inches in depth, were taken. Soil control samples were also taken at locations away from the grid. The soil samples were digested and analyzed for total arsenic, copper, and chromium. Analyses were performed using an atomic absorption spectrophotometer. This method determines the total metal residue level and does not speciate the metals.

Arsenic residues were found in the soil beneath all of the CCA-treated decks. The average surface arsenic level was 39 ppm and the maximum level under one deck was 217 ppm. The maximum arsenic residue found under any of the other decks was 88 ppm. The maximum arsenic residues present in soil core samples were in the top two inches, but were present at levels of approximately 2-20 ppm over the depth range of two to eight inches. Control arsenic values average 1.5 ppm.

The average surface copper residues found in the soil beneath all of the CCA-treated decks was 40 ppm and the maximum level under one deck was 216 ppm (soil from the same deck reported high arsenic levels). The maximum copper residue found in soil under any of the other decks was 156 ppm. The maximum residues present in soil core samples were generally higher in the top few inches of soil, and were higher than those levels in control samples.

The average surface chromium residues found in the soil beneath all of the treated decks was 34 ppm and the maximum level under one deck was 198 ppm (soil from the same deck that reported high arsenic levels). The maximum chromium residue found in soil under any of the other decks was 114 ppm. The average control level was 9.8 ppm. Average chromium levels of up to 11.7 ppm were reported at depths of 4.5 inches.

The soils under the CCA-treated decks are described as ranging from beach sand to being dark in color with a sponge-like consistency and with a high percentage of volatiles given off during analysis. This latter observation seems to indicate a soil with high organic content. The site with the highest arsenic level was characterized as having relatively high volatile solids, and this correlation can be found in five of the nine deck sites. The lowest arsenic residues were found at sites with low volatile solids content (Townsend et al., 2001). This study indicates that CCA-treated decks increase arsenic, copper, and chromium levels in soil beneath treated decks.

Based on the available information from both CCA-treated playground equipment and decks, it appears that the primary source of soil exposure to children from playing on playground equipment constructed of CCA-treated wood or playing under treated decks would occur from the leaching of CCA metal residues from horizontal pieces of treated wood in the playground equipment and deck wood onto the soil. Maximum residue levels would likely be less than 200 ppm arsenic, copper, and chromium, and, on the average, would be less than 50 ppm for each of the metals. Maximum residues of arsenic would likely occur in sandy soil under treated wood. However, if an organic material such as wood mulch with a high surface to weight relationship were placed under CCA-treated playground equipment, residues of the metals could be absorbed and retained in the material with slow leaching from the mulch. All three of the leaching studies described above are suitable to show that residues of copper, chromium, and arsenic leach from treated wood onto the soil under playground equipment and decks constructed of treated wood. Additional studies would be desirable, which reflect the use of CCA-treated wood in playground equipment, specifically, studies designed to sample soils beneath/adjacent to CCA-treated playground structures from different (representative) geographic regions of the United States.

#### 2.2.4.4 Environmental Fate

Many studies in the recent literature (Lebow, 1996; Stilwell and Gorny, 1997; Stilwell, 1998; Townsend et al., 2001; Osmose, 2000) report on leaching into soils. These studies have shown that none of the three metals migrate large distances (twelve inches vertically and three inches laterally) from the treated wood structure. Some studies have shown that the contamination level is elevated in the soil compared to the natural background levels of these

metals. Such studies indicate that metals can be persistent in the soils, particularly on the soil surfaces, and can result in environmental exposure. The metals show various speciation characteristics in soils, depending on the types of soil.

The metals migrating into water bodies can result in aqueous contamination. Metals also show a tendency to speciate in water, and various species will be present in water depending on the pH of water as well as the salinity. If water is highly acidic, the leaching rates and amounts of leachates increase. Generally, in soil and water, the amounts of metals released are in the order of Cu > As > Cr. In some recent cases it has been shown that the order of release rates are: As > Cu > Cr. In all cases, the amounts of chromium released is least of the three metals.

Numerous studies on bioaccumulation in various aquatic organisms have also been carried out over a period of time. A number of these aquatic species have shown a degree of bioaccumulation, and toxic effects have been observed. The studies were conducted under varying conditions and very few studies reported depuration rates.

An overall robust fate assessment cannot be made at this time, as the studies were conducted under different laboratory or field conditions, which were not standardized. Hence, while one can determine the exposure and hazards of these metals on humans, plants, and aquatic organisms, a complete fate assessment is not possible.

### 2.2.5 CCA Use and Potential Exposures to Components of CCA

The Agency is aware of potential exposure concerns to arsenic and chromium components of CCA-treated wood in decks and playground structures, and contaminated soils commonly found in these settings. During the pressure treatment of wood, CCA undergoes a fixation process where it initially is absorbed into the cellulosic and lignin structures of the wood. Chromium in the form of Cr(VI) attaches itself to the 'carboxylic groups' of the cellulosic structure and converts into Cr(III). Copper arsenate converts into basic copper arsenate. In pressure-treated wood, arsenic leaches to the surface of the wood mostly as As(V), but there may be some As(III). Chromium leaches mostly as Cr(III); however, trace amounts of Cr(VI) may also be present. Copper is present as Cu(II) (U.S. EPA, 2001b).

Of the components in CCA, copper does not pose significant toxicity concerns compared to arsenic and chromium. Copper is an essential nutrient that functions as a component of several enzymes in humans, and the toxicity of copper in humans involves consumption of water contaminated with high levels of copper (U.S. EPA, 2001b). Because of the relatively low toxicity of copper, the Agency did not conduct an exposure/risk assessment for copper. For chromium, hazard data clearly show that Cr(VI) demonstrates more significant toxicity than Cr(III) (Zartarian et al., 2003). Thus, the Agency felt that it would not be credible to apply Cr(VI) toxicity endpoints to Cr(total) residue results to assess incidental ingestion and dermal exposures in children. Since the Agency has not identified any endpoints of concern for Cr(III), the short-term intermediate-term and lifetime risks to Cr(III) are not presented.

### 2.2.6 Probabilistic Risk Assessment versus Deterministic Risk Assessment

A probabilistic assessment (i.e., using SHEDS-Wood) was conducted to evaluate exposure to CCA (Zartarian et al., 2003). A probabilistic exposure assessment uses probability distributions for one or more variables in a exposure equation in order to quantitatively characterize variability and/or uncertainty. A Monte Carlo Analysis (MCA) is perhaps the most widely used probabilistic method. MCA uses computer simulations to combine multiple probability distributions in exposure or risk equations. In contrast, a deterministic assessment uses point estimates for each of the variables in the exposure algorithm. The result is a single estimate of exposure dose. The output of a probabilistic assessment is a probability distribution of exposures that reflects the combination of the input probability distributions. If the input distributions represent variability, then the output distribution can provide information on variability in the population of concern. The input/output uncertainties of this assessment are discussed in Zartarian et al. (2003) If the input distributions reflect uncertainty, then the output distribution can provide information about uncertainty in the estimate. Information from SHEDS-Wood can be used in combination with toxicity data to form a probabilistic risk assessment (PRA). The PRA can be used to make statements about the likelihood of exceeding a risk level of concern, given the estimated variability in elements of the risk equation. Since the results of point estimate methods generally do not lend themselves to this level of risk characterization (e.g., quantitative uncertainty assessment), the PRA can provide unique and important supplemental information that can be used in making risk management decisions.

Table 2-3 summarizes the key differences between deterministic and probabilistic risk assessment methods. From this table it is easy to understand why a probabilistic risk assessment was conducted in assessing the risks from CCA-treated deck and playground equipment.

**Table 2-3. Comparison of Deterministic and Probabilistic Risk Assessments**

| | Deterministic Risk Assessment | Probabilistic Risk Assessment |
|---|---|---|
| Data Input | Pesticide concentrations and potential exposure factors are expressed as single point estimates. | Takes into account all available information and considers the probability of an occurrence. |
| Risk Estimates | Expressed as a single point value. The variability and uncertainty of the value is not reflected. | Expressed as a distribution of values, with a probability assigned to each value. Distribution reflects variability and can provide risk manager with information helpful to determine what particular range of the risk estimate distribution most closely represents real life scenarios. |
| Resources | Less time and not resource intensive, calculation is relatively simple, but provides little information about the proportion of the population receiving the estimated exposure. | May require more time and resources for seeking credible software to use for specific site. |

**Table 2-3. Comparison of Deterministic and Probabilistic Risk Assessments (Continued)**

| | Deterministic Risk Assessment | Probabilistic Risk Assessment |
|---|---|---|
| Methods | Useful for screening method - easily described. | More complicated for risk manager who may need time to understand the methodology. |
| Risk Communication | Single point risk estimates are often viewed as "the answer"; public perception may be misled. | Communication of uncertainty in the risk assessment can help to build trust among stakeholders. |
| Uncertainties | Qualitative; importance of variability is sometimes lost. | Provide quantitative information and a more comprehensive characterization of variability associated with in input parameters. |
| Regulatory Concern | Does not quantify the probability that the risk estimate exceeds a regulatory level of concern. | Can identify the data gaps for further evaluation/data collection and can use wider variety of site-specific information. |
| Data Analysis | May not utilize all available data for characterizing variability and uncertainty in risk estimates; provides fewer incentives for collecting better and credible information | Complete use of available data when defining inputs to the risk equation; and can provide more comprehensive characterization of variability in risk estimates. |
| Sensitivity Analysis | Only limited to dominant exposure pathways and chemical of concern | Can identify the exposure variables, probability models, and model parameters that influence the estimates of risk. |

EPA recognizes that there are many parameters that affect the level of potential exposure and that each of these parameters may vary. Probabilistic (e.g., Monte Carlo) techniques are capable of using multiple data sets which reflect the variability of parameters to produce estimates of the distribution of potential exposures. OPP has identified a number of data sets that contain information on the variability of parameters affecting the levels of exposure to CCA residues experienced by children as a result of their playground activities.

Children playing on decks and playgrounds that are built out of CCA pressure treated wood can be exposed to arsenic and chromium residues on wood surfaces and soils via oral and dermal routes. OPP has considered four proposed exposure scenarios individually in their previous assessment; however, to more comprehensively assess risks to children from exposure to arsenic through deck and playground contact with wood and soil, all four scenarios must be considered concurrently. PRAs present the most flexible tool to examine combined activities concurrently. The advantages of conducting a probabilistic risk assessment are as follows:

- ? PRAs more comprehensively address the distributions and variabilities of multiple sets of data in both inputs and outputs;
- ? PRAs offer more in depth analysis of uncertainty for both inputs and outputs;
- ? PRAs present the most flexible tool to examine combined activities concurrently; For residential exposure, children may be exposed to residues from playsets, decks, and soil concurrently;
- ? PRAs allow for more subsets of data (e.g., warm or cold environments, hand washing, bathing, etc.) and allow the user to separate the data and consider different exposure considerations;
- ? PRAs characterize more of the statistical uncertainties and special sensitivities for certain population groups (e.g., pica children);
- ? PRAs may show the actual shape of the composite distribution. For example, the actual distributions of the data may be lognormal instead of normal distribution;
- ? PRAs account for covariance between variables. The variance of the product could be inflated if there is a positive correlation between the variables;
- ? PRAs show the influence of a particular data set on the exposure, and graphically depict the data;
- ? PRAs show the distributional quartiles;
- ? PRAs use sophisticated software that can reproduce the calculation quickly and accurately;
- ? PRAs allow for a comprehensive sensitivity analysis that can identify the exposure variables, probability models, and model parameters that influence risk; and
- ? PRAs more accurately quantify the upper bound high-end percentile of total risk to more accurately help the risk managers make decisions based on the data.

### 2.2.7 EPA and OPP Regulatory Approach to PRA

Agency policy is that risk assessments should be conducted in a tiered approach, proceeding from simple to more complex analyses as the risk management situation requires (Agency Policy Document, 5/15/97)(U.S. EPA, 1998a). More complex analyses require greater resources, and probabilistic assessments can represent high levels of complexity. In a deterministic assessment, exposure is expressed as a single value, which could represent an upper-bound scenario or a central tendency. If a deterministic analysis, based on conservative assumptions, leads to risk estimates that are below levels of concern, then there is no need to refine risk assessments with more complex techniques (U.S. EPA, 1998a). However, if a conservative deterministic assessment leads to estimates above the level of concern, more sophisticated risk assessments may be warranted.

Probabilistic techniques offer a higher level of sophistication. In contrast to deterministic techniques, probabilistic risk assessments more fully consider ranges of values regarding potential exposure, and then weights possible values by their probability of occurrence. Individual input values used to generate a point estimate are replaced by a distribution reflecting a range of potential values; a computer simulation then repeatedly selects individual values from each

distribution to generate a range and frequency of potential exposures. In accordance with Agency policy at the current time, such techniques will not be considered for dose-response evaluations of toxicological data (U.S. EPA,1998a), but are limited to exposure assessments.

# 3.0 EXPOSURE ASSESSMENT

**Note: This chapter only provides a summary of the SHEDS-Wood exposure doses used for the risk assessment. For the detailed probabilistic SHEDS-Wood exposure assessment please refer to Zartarian et al. (2003), *A Probabilistic Exposure Assessment for Children Who Contact CCA-Treated Playsets and Decks,* Draft Final Report, September 25, 2003.**

SHEDS-Wood, a probabilistic exposure model developed by ORD, was used to develop the exposure assessment for CCA. The exposure assumptions, pathways, exposure routes, algorithms, and methodologies for this model are explained in detail in a separate exposure report, Zartarian et al. (2003), prepared by ORD and OPP entitled, *A Probabilistic Exposure Assessment for Children Who Contact CCA-Treated Playsets and Decks*. The SHEDS-Wood document provides exposure doses as ADD and LADDs; it does not provide a risk analysis. A general description of the exposure assessment approach for this model and a brief description of how the probabilistic exposure assessment data were used in the risk analysis are described in the narrative below.

The SHEDS-Wood probabilistic exposure model evaluated exposure of children for two chemicals (arsenic and chromium) in two environmental media (soil and surface residues). Arsenic exposures were generated as both noncancer exposure doses (ADDs) and cancer exposure doses (LADDs). For chromium, only noncancer exposure doses (ADDs) were necessary (refer to Chapter 4 for a discussion of chromium carcinogenicity). Thus, the Zartarian et al. (2003) probabilistic exposure assessment provided only chromium total ADDs for both residues and soil (i.e., LADDs were not generated). However for chromium, OPP made a decision to assess noncancer risks using soil ingestion ADDs only, because of the lack of detectable surface residues for Cr(VI) (see Chapter 2) and because of no specific dermal toxicity end-point exists for Cr(VI) (see Chapter 4). In addition, OPP and the FIFRA SAP were concerned that combining total chromium doses with Cr(VI) toxicity endpoints would overestimate chromium risks. Because of this concern, ADDs for soil ingestion were developed specifically for Cr(VI) in this assessment. The Cr(VI) soil ingestion ADDs were derived based on the SAP Panel recommendations for chromium speciation (see Chapter 2). In summary, the total chromium soil ingestion ADDs originally provided in the SHEDS-Wood data files were adjusted by multiplying the data by 0.10 (10%) to account for the portion of total chromium that is assumed to be present as Cr(VI) because of speciation.

Because there are two routes of exposure (dermal and oral) and two environmental media (soil and surface residues), SHEDS-Wood evaluates exposure based on four scenarios. These scenarios are: dermal contact with CCA-treated wood, dermal contact from CCA-contaminated soil near treated wood structures, mouthing hands after touching CCA-treated wood, and ingesting CCA-contaminated soil. SHEDS-Wood was used to evaluate three exposure durations

short-term, intermediate-term, and average lifetime exposures (only for arsenic). The potentially exposed populations for this assessment are (1) children in the United States who contact CCA-treated wood (from decks and/or playsets) and/or (2) children in the United States who contact CCA-containing soil from public playsets (e.g., at a playground, a school, a daycare center). A subset of these children was also assumed to contact CCA-treated wood residues and/or CCA-containing soil from residential playsets (i.e., at the child's own home or at another home) and/or residential decks (i.e., at the child's own home or another home). This population was selected because of the particular focus by CPSC and other groups on playground playsets in conjunction with EPA's focus on estimating the risk to children from various primary sources of CCA-treated wood that children may contact (Zartarian et al., 2003).

Two bounding estimate climate scenarios (warm throughout the year and cold throughout the year) were considered, as well as three exposure time periods: short-term (one day to one month), intermediate-term (one month to six months), and average lifetime exposure (6 years over a 75 year lifetime). SHEDS-Wood calculated the predicted exposure (and dose) to arsenic and chromium using age and gender representative time-location activity data for children ages 1-6 years old (Zartarian et al., 2003).

It should be noted that this risk assessment report does not address certain high risk groups (e.g., children with autism, Down’s syndrome). This issue is, however, addressed qualitatively in the probabilistic exposure assessment (Zartarian et al., 2003). Exposures to arsenic for children with pica soil ingestion behavior were assessed. In addition, it should be noted that the probabilistic exposure assessment developed by Zartarian et al. (2003) contains additional exposure scenarios (e.g., Tables 32 and 34) that were not included in this risk assessment.

The ADDs and LADDs derived from the SHEDS-Wood report (Zartarian et al., 2003) are summarized in Tables 3-1 to 3-10. Tables 3-1 through 3-10 summarize the means, medians, 95 percentiles, and 99 percentiles for arsenic ADDs/LADDs and Cr(VI) ADDs. These exposure doses were used to generate MOEs and cancer risks for the risk characterization (see Chapter 5.0) and risk reduction impact (see Chapter 6.0) chapters. The ADDs and LADDs are also presented later in probabilistic risk tables in Chapters 5 and 6 along with the corresponding MOEs and cancer risks. With the exception of the Cr(VI) ADDs, Zartarian et al. (2003) presented all of the exposure doses used to generate these tables, and the footnotes in Tables 3-1 to 3-10 indicate which corresponding table from Zartarian et al. (2003) was referenced. For Cr(VI), the footnotes reference the ADDs that are presented later in Chapter 5.

Tables 3-1 to 3-2 present ADDs for arsenic and chromium for short- and intermediate-term exposures. These tables attempt to capture the population of children that play on both playsets and decks in warm and cold climate settings. Tables 3-3 to 3-4 present ADDs for children exposed to playsets only. Table 3-5 presents arsenic ADDs for children with a special

sensitivity of pica soil ingestion behavior in warm climates. Arsenic exposures for warm climates represent the highest risk concern for pica soil ingestion behavior. Table 3-6 presents the LADDs for arsenic. These tables present the risks for the population of children that play on playsets and decks, and playsets only based on warm and cold climate settings. Tables 3-7 to 3-9 are the LADDs for arsenic (used later in Chapter 5) and represent reductions in arsenic exposure from hand washing, and coating CCA-treated wood with sealants (assumes a hypothetical 90% and 99.5% reduction for typical sealants), or both hand washing and sealants combined. Only LADDs for the warm climate settings were assessed. Table 3-10 presents the exposure doses for arsenic using the Wester et al. (2003) dermal absorption factor for arsenic (i.e., 0.01 percent). This is considerably lower than the default dermal absorption factor recommended by the FIFRA SAP, a value in the range of 2-3 percent. Risks for these scenarios are provided later in Chapter 5.

**Table 3-1. Arsenic ADDs (mg/kg/day) - Playsets and Decks[a]**

| Time Frame[b] | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short[c] | 1.3E-4 | 6.7E-5 | 6.5E-5 | 2.5E-5 | 4.7E-4 | 2.2E-4 | 9.5E-4 | 7.0E-4 |
| Intermediate[d] | 1.3E-4 | 7.0E-5 | 6.8E-5 | 3.1E-5 | 4.5E-4 | 2.4E-4 | 9.6E-4 | 5.9E-4 |

[a] The ADDs represent the mean, median, 95%ile, and 99%ile total doses for both warm and cold climate residue and soil data for playsets and decks.

[b] Time frame considers short-term (1 day to 1 month) and intermediate-term (1-6 months) exposures.

[c] Data for short-term warm climate ADDs are based on the results of Table 18 in Zartarian et al. (2003). Data for short-term cold climate ADDs are based on the results of Table 19 in Zartarian et al. (2003). Also refer to Table 5-6 and Table 5-7 for short-term ADDs for warm and cold climates.

[d] Data for intermediate-term warm ADDs are based on the results of Table 16 in Zartarian et al. (2003). Data for intermediate-term cold ADDs are based on the results of Table 17 in Zartarian et al. (2003). Also refer to Table 5-11 and Table 5-12 for intermediate-term ADDs for warm and cold climates.

**Table 3-2. Chromium(VI) ADDs (mg/kg/day) - Playsets and Decks[a,b]**

| Time Frame[c] | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short[d] | 1.0E-5 | 2.5E-6 | 2.5E-7 | 2.9E-8 | 3.9E-6 | 1.0E-6 | 1.3E-5 | 3.7E-6 |
| Intermediate[e] | 9.1E-6 | 2.3E-6 | 2.3E-7 | 3.5E-8 | 3.5E-6 | 8.9E-7 | 1.0E-5 | 3.4E-6 |

[a] The exposure doses represent soil ingestion exposure only for Cr(VI).
[b] These ADDs represent the mean, median, 95%ile, and 99%ile total doses for both warm and cold climate soil ingestion data for playsets and decks.
[c] Time frame considers short-term (1 day to 1 month) and intermediate-term (1-6 month) exposures.
[d] Data for short-term ADDs were recalculated based on soil ingestion only exposures and a 10% adjustment to chromium(total) to account for Cr(VI). Refer to Table 5-8 and Table 5-9 for short-term ADDs for warm and cold climates.
[e] Data for intermediate-term ADDs were recalculated based on soil only exposures and a 10% adjustment to chromium(total) to account for Cr(VI). Refer to Table 5-13 and Table 5-14 for intermediate-term ADDs for warm and cold climates.

**Table 3-3. Arsenic ADDs (mg/kg/day) - Playsets Only[a]**

| Time Frame[b] | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short[c] | 8.4E-5 | 4.3E-5 | 3.0E-5 | 1.4E-5 | 2.9E-4 | 1.6E-4 | 8.2E-4 | 4.6E-4 |
| Intermediate[d] | 5.9E-5 | 3.7E-5 | 2.8E-5 | 1.1E-5 | 2.3E-4 | 1.2E-4 | 4.3E-4 | 3.9E-4 |

[a] The ADDs represent the mean, median, 95%ile, and 99%ile total doses for both warm and cold climate soil ingestion data for playsets only.
[b] Time frame considers short-term (1 day to 1 month) and intermediate-term (1-6 month) exposures.
[c] Data for short-term warm climate ADDs are based on Table 18 in Zartarian et al. (2003). Data for short-term cold climate ADDs were based on Table 19 in Zartarian et al. (2003). Also refer to Table 5-6 and Table 5-7 for short-term ADDs for warm and cold climates.
[d] Data for intermediate-term warm ADDs are based on Table 16 in Zartarian et al. (2003). Data for intermediate-term cold climate ADDs are based on Table 17 in Zartarian et al. (2003). Also refer to Table 5-11 and Table 5-12 for intermediate-term ADDs for warm and cold climates.

**Table 3-4. Chromium(VI) ADDs (mg/kg/day) - Playsets Only [a,b]**

| Time Frame[c] | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short[d] | 9.7E-6 | 1.8E-6 | 1.8E-7 | 1.5E-8 | 4.3E-6 | 6.9E-7 | 1.5E-5 | 3.0E-6 |
| Intermediate[e] | 8.9E-6 | 1.3E-6 | 1.6E-7 | 1.4E-8 | 3.5E-6 | 6.0E-7 | 1.6E-5 | 1.8E-6 |

[a] The exposure doses represent soil ingestion exposure only for Cr(VI) for playsets only.
[b] The ADDs represent the mean, median, 95%ile, and 99%ile total doses for both warm and cold climate soil ingestion data for playsets only.
[c] Time frame considers short-term (1-day to 1-month) and intermediate-term (1-6 month) exposures.
[d] Data for short-term ADDs are recalculated based on soil only exposures and a 10% adjustment to chromium(total) to account for Cr(VI). Refer to Table 5-8 and Table 5-9 for short-term ADDs for warm and cold climates.
[e] Data for intermediate-term ADDs were recalculated based on soil only exposures and a 10% adjustment to chromium(total) to account for Cr(VI). Refer to Table 5-13 and Table 5-14 for intermediate-term ADDs for warm and cold climates.

**Table 3-5. Arsenic ADDs (mg/kg/day) - Pica Ingestion[a]**

| Scenario | Mean | Median | 95%ile | 99%ile |
|---|---|---|---|---|
| Playsets and decks | 3.1E-4 | 1.5E-4 | 1.0E-3 | 1.6E-3 |
| Playsets only | 2.3E-4 | 9.9E-5 | 7.7E-4 | 2.2E-3 |

[a] The ADDs represent the mean, median, 95%ile, and 99%ile total doses for warm climate soil data. Data for short-term warm climate ADDs are based on Table 33 in Zartarian et al. (2003).

**Table 3-6. Arsenic LADDs (mg/kg/day)[a]**

| Scenario | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Playsets and decks | 1.1E-5 | 6.0E-6 | 6.1E-6 | 2.9E-6 | 3.9E-5 | 2.1E-5 | 8.4E-5 | 4.4E-5 |
| Playsets only | 6.4E-6 | 3.2E-6 | 3.0E-6 | 1.5E-6 | 2.3E-5 | 1.2E-5 | 6.5E-5 | 2.4E-5 |

[a] The LADDs represent the mean, median, 95%ile, and 99%ile total doses for playsets and decks and for playsets only in cold and warm climates. Data for warm climate LADDs are based on Table 14 in Zartarian et al. (2003). Data for cold climate LADDs are based on Table 15 in Zartarian et al. (2003). Also refer to Table 5-16 for LADDs for warm climates and Table 5-17 for LADDs for cold climates.

**Table 3-7. Arsenic LADDs (mg/kg/day) - Mitigation with Sealant[a]**

| Scenario | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Moderate (90%)[b] | Max. (99.5%)[c] | Moderate (90%)[b] | Max. (99.5%)[c] | Moderate (90%)[b] | Max. (99.5%)[c] | Moderate (90%)[b] | Max. (99.5%)[c] |
| Playsets and decks | 1.8E-6 | 8.0E-7 | 1.1E-6 | 3.6E-7 | 5.6E-6 | 2.9E-6 | 1.0E-5 | 7.8E-6 |
| Playsets only | 9.0E-7 | 5.5E-7 | 5.4E-7 | 2.2E-7 | 3.0E-6 | 2.2E-6 | 5.7E-6 | 4.6E-6 |

[a] The LADDs represent the mean, median, 95%ile, and 99%ile total dose for playsets and decks and for playsets only in warm climates, assuming reduction of sealants.

[b] Moderate reduction (assumes 90% reduction with sealant for warm climates only). Data for warm climate LADDs are based on Table 37 in Zartarian et al. (2003). Also refer to Table 6-5 for LADDs for warm climates.

[c] Maximum reduction (assumes 99.5% reduction with sealant for warm climates only). Data for warm climate LADDs are based on Table 38 in Zartarian et al. (2003). Also refer to Table 6-6 for LADDs for warm climates.

**Table 3-8. Arsenic LADDs (mg/kg/day) - Mitigation with Hand Washing[a]**

| Scenario | Mean | Median | 95%ile | 99%ile |
|---|---|---|---|---|
| Playsets and decks | 8.4E-6 | 4.8E-6 | 2.7E-5 | 7.0E-5 |
| Playsets only | 3.7E-6 | 2.1E-6 | 1.2E-5 | 2.0E-5 |

[a] The LADDs represent the mean, median, 95%ile, and 99%ile total dose for playsets and decks and playsets only in warm climates, assuming reduction by sealants. Data for warm climate LADDs were based on Table 39 in Zartarian et al. (2003). Also refer to Table 6-8 for LADDs for warm climates.

**Table 3-9. Arsenic LADDs (mg/kg/day) - Mitigation with Hand Washing and Sealant[a]**

| Scenario | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Moderate (90%)[b] | Max. (99.5%)[c] | Moderate (90%)[b] | Max. (99.5%)[c] | Moderate (90%)[b] | Max. (99.5%)[c] | Moderate (90%)[b] | Max. (99.5%)[c] |
| Playsets and decks | 1.6E-6 | 9.2E-7 | 8.9E-7 | 3.4E-7 | 5.2E-6 | 3.2E-6 | 8.4E-6 | 1.1E-5 |
| Playsets only | 9.8E-7 | 5.7E-7 | 5.2E-7 | 2.0E-7 | 3.2E-6 | 2.5E-6 | 6.2E-6 | 4.7E-6 |

[a] The LADDs represent the mean, median, 95%ile, and 99%ile total dose for playsets and decks and for playsets only in warm climates, assuming reduction of sealants.

[b] Moderate reduction assumes 90% reduction with sealant and hand washing. Data for warm climate LADDs are based on Table 40 in Zartarian et al. (2003). Also refer to Appendix C.

[c] High reduction assumes 99.5% reduction with sealant and hand washing. Data for warm climate LADDs are based on Table 41 in Zartarian et al. (2003). Also refer to Appendix C.

**Table 3-10. Arsenic LADDs (mg/kg/day) - Using 0.01% Dermal Absorption[a]**

| Scenario | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Playsets and decks | 7.9E-6 | 5.4E-6 | 4.5E-6 | 2.7E-6 | 2.7E-5 | 2.1E-5 | 6.4E-5 | 4.3E-5 |
| Playsets only | 4.0E-6 | 2.9E-6 | 2.0E-6 | 1.1E-6 | 1.2E-5 | 1.0E-5 | 3.3E-5 | 2.7E-5 |

[a] The LADDs represent the mean, median, 95%ile, and 99%ile total doses for playsets and decks and for playsets only for warm and cold climates, assuming reduction using a 0.01% dermal absorption factor. Data for warm climate LADDs are based on Table 35 in Zartarian et al. (2003). Data for cold climate LADDs are based on Table 36 in Zartarian et al. (2003). Also refer to Table 5-19 for LADDs for warm climates and Table 5-20 for LADDs for cold climates.

## 4.0 HAZARD ASSESSMENT

The purpose of the hazard assessment is to identify available evidence regarding the potential for the chemical of concern to cause adverse effects to the potential receptor (individual) and to provide, where possible, an estimate of the relationship between the extent of exposure to the chemical of concern and increased likelihood and/or severity of the adverse effects.

For noncancer toxic effects, available toxicology data are reviewed and no-observed adverse effect levels (NOAELs) and lowest observed adverse effect levels (LOAELs) are developed for each study. Subsequently, the reviewed data for the chemical of concern are presented to a committee of scientists within OPP who reach concurrence on toxicology endpoints that best represent the toxic effects expected from various routes of exposure and durations of exposure. Endpoints are selected for non-dietary exposures to represent short-term (1-30 days), intermediate-term (30-180 days), and long-term exposure scenarios, as needed. In addition, incidental oral exposure endpoints are selected for short-term and intermediate-term exposure durations to represent ingestion of the chemical of concern residues that may occur from hand-to-mouth behaviors. In general, toxicity endpoint selection should, to the extent possible, match the temporal and spatial characteristics of the exposure scenarios selected for use in the risk assessment. These endpoints are then used in conjunction with exposure values to calculate risks associated with various types of exposure, depending upon the uses of the chemical of concern (McMahon and Chen, 2003).

For carcinogenic effects of a chemical, a slope factor (SF), also know as potency factor, is derived. Slope factors are developed based on a dose-response curve for carcinogenicity of the specific chemicals. The slope factors are developed from human and animal studies and are designed to be health protective. The SF is used to estimate an upper-bound probability of an individual developing cancer as a result of exposure to a potential carcinogen. Carcinogens with EPA-derived slope factors are also given an EPA weight-of-evidence classification, whereby, potential carcinogens are grouped according to the likelihood that the chemical is a human carcinogen, depending on the quality and quantity of carcinogenic potency data for a given chemical.

For the current CCA risk assessment, arsenic and chromium were considered as the primary chemicals of concern. The current policy, Conditions for Acceptance and associated principles are not intended to apply to dose-response evaluations for human health risk assessments until this application has been studied further (Agency Policy Document, 5/15/1997) (U.S. EPA, 1997a). Currently, OPP does not have the Guidance to perform the probabilistic analysis of toxicity endpoints. For this risk assessment, OPP used the endpoints developed by U.S. EPA, (McMahon and Chen, 2003), which are provided in Appendix A. These endpoints were developed using guidance provided by the FIFRA SAP (U.S., EPA, 2001c). As stated in the Agency Policy Document, 5/15/97 (U.S. EPA, 1998), “For human health risk assessments, the

application of Monte Carlo and other probabilistic techniques has been limited to exposure assessments in the majority of cases. The current policy, Conditions for Acceptance and associated guiding principles are not intended to apply to dose-response evaluations for human health risks assessment until this application of probabilistic analysis has been studied further." Currently, OPP does not have guidance available to perform the probabilistic analysis of toxicity endpoints. According to Agency policy, endpoints used in assessments should be consistent with the exposure of concern (acute, subchronic, chronic), and should be those selected by the HED Hazard Identification Assessment Review Committee (HIARC), or selected in accordance with the *Draft Toxicology Endpoint Selection Process: A Guidance Document*, presented to the SAP in February 1997. Thus, point estimates have been used to characterize toxicity for the CCA risk assessment. Toxicology endpoints for both inorganic arsenic and chromium have been selected for the residential exposure assessment and are presented in Sections 4.1 and 4.2, respectively. Summary tables are provided in Section 4.3.

It also should be noted that the studies from Ginsberg (2003) and other researchers, and the recent work on early-life exposures by ORD in the *Draft Final Guidelines for Carcinogenic Risk Assessment* (U.S. EPA, 2003a) and *Supplemental Guidance for Assessing Cancer for Environment Assessment* (U.S. EPA, 2003b), discussed the criteria for assessing early-life exposure. A discussion of early-life exposures to arsenic is presented in Section 4.4. In addition, a brief discussion of the relative bioavailabilities and dermal absorption values of arsenic and chromium in surface residues and soil are presented in Sections 4.5 and 4.6, respectively.

## 4.1 Arsenic

Based on the registered use of CCA-treated lumber for fencing and decking materials in residential settings, both incidental oral and dermal exposures are expected. The studies selected for short- and intermediate-term incidental oral exposure were the human case reports of Franzblau and Lilis (1989) and Mizuta et al. (1956) (see Appendix A). The oral LOAEL of 0.05 mg/kg/day was selected, based on facial edema, gastrointestinal symptoms, neuropathy, and skin lesions observed at this dose level (see Appendix A). A Margin of Exposure (MOE) of 30 should be applied to the oral LOAEL. This value consists of a 10x factor for intraspecies variation and a 3x factor for extrapolating from a LOAEL to a NOAEL observed at the LOAEL of 0.05 mg/kg/day.

Since there were no appropriate dermal studies, the same studies selected for short- and intermediate-term incidental oral exposure were selected for short- (1-30 days ) and intermediate- (30-180 days) term dermal exposure scenarios (see Appendix A). OPP did not develop an exposure assessment for long-term exposures (see Zartarian et al., 2003). Thus, the oral LOAEL of 0.05 mg/kg/day was selected for dermal exposures, based on facial edema, gastrointestinal symptoms, neuropathy, and skin lesions observed at this dose level. The dermal absorption factor approach used in this assessment does not use a point estimate but uses a range of reported values

from the Wester et al. (1993) study which was recommended by the FIFRA Scientific Advisory Panel (U.S. EPA, 2001c). The same MOE of 30 was also selected for dermal exposure. No long-term incidental oral or dermal exposures are expected from residential exposure to arsenic in CCA-treated lumber. At the advice of the SAP, EPA decided not to quantify inhalation exposure to metals since such exposure would be minimal (U.S. EPA, 2001c).

For this risk assessment, an oral cancer slope factor of 3.67 $(mg/kg/day)^{-1}$ was used. This value is based on the Agency's risk assessment associated with inorganic arsenic in drinking water presented in 2000 (U.S. EPA, 2001e, personal communication with Andrew Schulman). It is consistent with the slope factor used by the Office of Water for the arsenic MCL. See Appendix A for more details regarding the carcinogenic slope factor used. Following the risk assessment associated with inorganic arsenic in drinking water, which was presented in 2000, EPA asked the National Research Council (NRC) to meet again to: (1) review EPA's characterization of potential human health risks from ingestion of inorganic arsenic in drinking water; (2) review the available data on the carcinogenic and noncancer effects of inorganic arsenic; (3) review the data on the metabolism, kinetics and mechanism(s)/mode(s) of action of inorganic arsenic; and (4) identify research needs to fill data gaps. In 2001, NRC published an update to the 1999 NRC report (NRC, 1999) and concluded that: (1) arsenic-induced bladder and lung cancers still should be the focus of arsenic-related cancer risk assessment; (2) southwestern Taiwan data are still the most appropriate for arsenic-related cancer risk assessment; and (3) present modes of action data are not sufficient to depart from the default assumption of linearity. However, the 2001 NRC update made specific recommendations with respect to the overall cancer risk estimates. The Agency is currently considering these recommendations and their potential impact on the cancer potency estimate. Based on the Agency's considerations of these recommendations, the current proposed cancer potency number may change in the final version of this risk assessment.

## 4.2 Chromium

For chromium, hazard data clearly show that Cr(VI) demonstrates more significant toxicity than Cr(III). During pressure treatment of wood, CCA undergoes a fixation process where it is initially absorbed into the cellulosic and lignin structures of the wood. Chromium, in the form of Cr(VI), attaches itself to the 'carboxylic groups' of the cellulosic structure and converts into Cr(III) (U.S. EPA, 2001c). More recent studies by the ACC and RTI International indicate that Cr(III) is the main component in CCA pressure-treated wood (RTI International, 2003; Nico et al., 2003; Cooper, 2003). Based on the non-detectable results of Cr(VI) in the CCA-treated wood in the Cooper (2003) and RTI International (2003) studies, the Agency felt that it was not credible to assign Cr(VI) toxicity endpoints to total Cr surface residue results, since evidence indicates most of the Cr wood surface residues in wood are primarily Cr(III). Therefore, OPP did not assess the risks from incidental ingestion exposures in wood surface residues for children in this assessment. However, the Agency felt that it would be appropriate to assess soil ingestion exposure to Cr(VI) (dermal toxicity endpoints were not identified for

Cr(VI)). Therefore, toxicity information to assess soil ingestion exposures to Cr(VI) was required. As discussed in the Exposure Chapter (3.0), the total chromium doses from SHEDS-Wood for soil ingestion were multiplied by 0.10 (10%) to estimate a Cr(VI) equivalent dose. This was done for both short- and intermediate-term chromium doses. Toxicity endpoints for Cr(VI) were then applied to the Cr(VI) equivalent doses to evaluate Cr(VI) risks.

Based on the registered use of CCA-treated lumber for fencing and decking materials in residential settings, incidental oral exposure to chromium is expected, based on potential ingestion of soil contaminated with chromium as a result of leaching from wood. The study selected for short- and intermediate-term incidental oral exposure was a developmental toxicity study in the rabbit conducted by Tyl et al. (1991) and submitted to the Agency under MRID #42171201 (see Appendix A for details regarding this study). Based on the Tyl et al. (1991) study, a maternal NOAEL of 0.5 mg/kg/day and a LOAEL of 2.0 mg/kg/day were selected as the short- and intermediate-term toxicity endpoints, based on the increased incidence of maternal mortality and decreased body weight gain (see Appendix A for more description of the studies used to develop the toxicity endpoints). An MOE of 100 was assigned by OPP for this endpoint (McMahon and Chen, 2003).

The U.S. EPA (1998b) IRIS document on Cr(VI) states that "chromium is one of the most common contact sensitizer in males in industrialized countries and is associated with occupational exposures to numerous materials and processes." In addition, it further states that "dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis." It was determined by the OPP HIARC that quantification of hazard from dermal exposure is not possible for chromium, due to the significant dermal irritation and sensitization observed. Therefore, no endpoints were determined by HIARC for Cr(VI) from dermal exposures. Dermal irritation and dermal sensitization are the primary concern for the dermal exposure route.

The members in the October 23-25, 2001, FIFRA SAP meeting agreed that the Agency should not consider the inhalation route of exposure for chromium in the risk assessment.

## 4.3 Summary Tables

All the selected non-cancer toxicological endpoints used for arsenic are summarized in Table 4-1. Table 4-2 presents the toxicological endpoints for Cr(VI). For child exposures in this assessment, only the incidental ingestion and dermal exposure pathways were considered.

**Table 4-1. Toxicological Endpoints for Assessing Exposures/Risks to Arsenic (V)**

| EXPOSURE SCENARIO | DOSE (mg/kg/day) | ENDPOINT | STUDY |
|---|---|---|---|
| Incidental Short- and Intermediate- Term Oral [a] | LOAEL= 0.05<br><br>MOE = 30 | Based on edema of the face, gastrointestinal, upper respiratory, skin, peripheral and neuropathy symptoms | Franzblau et al. (1989) and Mizuta et al. (1956) |
| Dermal Short- and Intermediate-Term [a,b] | LOAEL= 0.05<br><br>MOE = 30 | Based on edema of the face, gastrointestinal, upper respiratory, skin, peripheral and neuropathy symptoms | Franzblau et al. (1989) and Mizuta et al. (1956) |
| Carcinogenicity - Oral Ingestion<br>(Oral and Dermal Risks) | $Q_1$* = 3.67 $(mg/kg/day)^{-1}$ | Internal organ cancer (liver, lung and bladder) | Chronic epidemiological oral study on humans |

Note:

[a] MOE = Margin of Exposure; NOAEL = No observed adverse effect level; and LOAEL = Lowest observed adverse effect level.

[b] The dermal absorption factor approach used in this assessment does not use a point estimate but uses a range of reported values from the Wester et al. (1993) study which was recommended by the FIFRA Scientific Advisory Panel. The dermal absorptions are incorporated into the SHEDs-Wood model.

**Table 4-2. Toxicological Endpoints for Assessing Exposures/Risks to Chromium (VI)**

| EXPOSURE SCENARIO | DOSE (mg/kg/day) | ENDPOINT | STUDY |
|---|---|---|---|
| Incidental Short- and Intermediate- Term Oral [a] | NOAEL= 0.5<br><br>MOE = 100 | Increased mortality and decreased body weight gain in dams at 2.0 mg/kg/day. | Developmental/Rabbit<br>Tyl et al. (1991) |
| Dermal Short- and Intermediate-Term [b] | Because dermal irritation and dermal sensitization are the primary concern through the dermal exposure route, no toxicological end-point is selected for use in assessing dermal exposure risks to chromium. | | |

Note:

[a] MOE = Margin of Exposure; NOAEL = No observed adverse effect level; and LOAEL = Lowest observed adverse effect level.

[b] An oral NOAEL is used for the toxicity endpoint for soil ingestion. Dermal absorption factor of 1% is incorporated into SHEDS-Wood model.

## 4.4 Early-Life Exposures

Ginsberg (2003) mentions that for "a vast majority of chemicals that have cancer potency estimates on IRIS, the underlying database is deficient with respect to early-life exposures."

Ginsberg (2003) concluded that based on the results of his study "short-term exposures in early life are likely to yield a greater tumor response than short-term exposures in adults, but similar tumor response when compared to long-term exposures in adults." The risk attributable to early-life exposure often appears modest compared with the risk from lifetime exposure. It can be about 10-fold higher than the risk from an exposure of similar duration occurring later in life (Ginsberg, 2003).

ORD has recently published *Draft Final Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2003a) This document mentions the need to address early-life exposures from carcinogens. In addition, ORD has also published an external review draft document entitled the *Supplemental Guidance for Assessing Cancer Susceptibility from Early Life Exposure to Carcinogens* (U.S. EPA, 2003b). U.S. EPA (2003b) presents an approach for assessing cancer susceptibility from early-life exposure to carcinogens.

Much toxicity are available on arsenic; however, the data needed to account for an accurate representation of early-life exposure to arsenic appears to be insufficient. For example, the arsenic, the National Resource Council (NRC, 2001) reports that "few studies of the effects of arsenic on reproduction and development had been published" (NRC, 2001). NRC also concluded "that although a large amount of research is available on arsenic's mode of action, the exact nature of the carcinogenic action is not clear" (NRC, 2001). Finally, NRC concluded that inorganic arsenic and its metabolites have been shown to induce chromosomal alterations and large deletion of mutations, but not point mutations.

Although there is some new evidence indicating that exposure to arsenic from drinking water during pregnancy may be associated with decreased birth weights of newborns (Hopenhayn, 2003) and may increase the cancer incidence of the child in the later stage of life (Waalkes, 2003), the data needed to account for an accurate representation of early-life exposure of arsenic appears to be insufficient (NRC, 2001). However, because the cancer slope factor used in this cancer risk assessment is derived from the epidemiology study using the Southwestern Taiwan data, it is generally believed that the sensitive population exposed to inorganic arsenic through drinking water during the most sensitive period of time is already included in the exposed population. Therefore, an adjustment factor does not appear to be appropriate in the cancer risk assessment associated with arsenic exposure.

## 4.5 Relative Bioavailability

The absorption of a chemical of concern is dependent on the matrix to which it is exposed. It is generally assumed that the absorption of the chemical of concern from the gastrointestinal tract is nearly complete. The toxicological endpoints were selected based on the administered dose, not the absorbed dose. However, when the chemical is in a different matrix, it may have a different absorption rate because it may be present in water-insoluble forms or interact with other

constituents in the matrix. The relative bioavailability of the chemical of concern, after it is exposed (water vs. soil), was defined as the percentage of the chemical of concern absorbed into the body of a soil-dosed animal compared to that of an animal receiving a single dose of the chemical of concern in an aqueous solution.

The issue of arsenic and chromium relative bioavailability has already been discussed in the October 23-25, 2001, FIFRA SAP Meeting (see comments in Appendix F). The recommendations of the FIFRA SAP for both arsenic and chromium have been incorporated into the SHEDS-WOOD document to develop ADDs and LADDs (Zartanian et al., 2003). A summary of relative bioavailability studies for arsenic is presented in Appendix E.

*Arsenic*

Zartarian et al. (2003) used data from ACC (2003a; 2003b) to determine the relative bioavailability for arsenic in the matrix of concern (either CCA-treated wood surface residue or soil collected from areas around CCA-treated wood) vs. arsenic in water. According to Zartarian et al. (2003), the ACC data were fitted in SHEDS-Wood to a beta distribution, with a mean relative bioavailability of 0.273 (27.3%) for CCA-treated wood surface residue vs. arsenic in water. For arsenic in soil collected from an area close to CCA-treated wood, Zartarian et al. (2003) fitted the ACC (2003a) data to a beta distribution, with a mean relative bioavailability of 0.476 (47.6%).

*Chromium*

Zartarian et al. (2003), per FIFRA SAP (U.S. EPA, 2001c) recommendations, assumed a relative bioavailability of 100% for both chromium surface residues and soil vs. chromium in water.

## 4.6 Dermal Absorption

*Arsenic*

Although OPP reported a point estimate for dermal absorption from Wester et al. (1993) in the hazard assessment (see Appendix A), the dermal absorption factor approach used in the Zartarian et al. (2003) probabilistic exposure assessment, and in this risk assessment, used a range of reported values from Wester et al. (1993). The distribution of values selected from SHEDS-Wood is described in more detail in the SHEDS-Wood probabilistic assessment (Zartarian et al., 2003). It should be noted that the approach used in SHEDS-Wood was consistent with the recommendations of the FIFRA Scientific Advisory Panel (U.S. EPA, 2001c). Wester et al. (1993) *in vivo* results with monkeys ranged from 2.0% to 6.4%. In the OPP 2001 deterministic assessment, OPP used 6.4% and later also used this for the occupational risk assessment in the

reregistration document. The 2001 SAP recommended a value in the range of 3%." For the SHEDS-Wood assessment, ORD fit a triangular distribution to the Wester et al. (1993) data (Zartarian et al., 2003). Zartarain et al. (2003) also went on to say that "It was important to note that because of dermal removal processes (hand washing, bathing, and hand mouthing), the modeled daily absorption rate is lower than the user-specified value. For a 3% per day input, the actual amount absorbed is predicted at about 1% per day. This is consistent with the SAP 2001(U.S. EPA, 2001c) comment that the 2%-3% from the monkey studies may be too high because of real-world removal processes from skin noted above" (Zartarian et al., 2003).

*Chromium*

As noted in Section 4.2, dermal irritation and dermal sensitization are still the primary concern for the dermal exposure route. The FIFRA SAP noted that "it is unlikely that sufficient chromium could penetrate the skin and enter the circulation to cause systemic effects from dermal exposure. Skin penetration for chromium is estimated to be 1%. It is usually assumed that the contribution to systemic effects from dermal exposure is not likely to be significant relative to oral exposure."

# 5.0 RISK CHARACTERIZATION

## 5.1 Introduction

The objective of the risk characterization was to integrate toxicity data (see Chapter 4.0) with the results of the exposure assessment (Chapter 3.0) to evaluate potential human health impacts to children who are exposed to arsenic and chromium residues while playing on or near CCA-treated wood playgrounds and decks. Children can be exposed to arsenic and chromium residues via hand-to-mouth ingestion and dermal absorption of residues in wood and soil. This chapter presents the incremental risks from exposure to CCA-treated wood and does not address risks from exposure to all sources of arsenic and chromium in the environment. The probabilistic exposure assessment (Zartarian et al., 2003) used for this risk assessment was specific for exposure to surface residues from treated wood and surrounding soils.

This chapter presents a probabilistic risk characterization. Distributions were used for input variables of the exposure dose algorithm, and the output of the exposure assessment was a distribution of risks across all members of the population. This exposure distribution was combined with toxicity data to provide a risk distribution for members of the exposed population. A hypothetical example of a cumulative distribution function for cancer risk is shown in Figure 5-1. The x-axis of Figure 5-1 represents the excess lifetime cancer risk level and the y-axis represents the cumulative probability of the cancer risk level within the hypothetical population. The figure also shows various landmarks along the distribution curve, such as the 50th, 90th, 95th percentiles, etc. For example, in Figure 5-1, the 95th percentile corresponds to a cancer risk of 1.2E-06 and the 50th percentile corresponds to a cancer risk of 4.1E-07 (U.S. EPA, 2001d).

Risks due to exposure to CCA-treated wood were evaluated for noncancer and cancer effects. Cancer risk refers to the probability of increased cancer incidence resulting from exposure to proven or suspected carcinogenic chemicals. Cancer risk is generally expressed in scientific notation (e.g., an individual excess lifetime risk of 1 in 10,000 is represented as $1 \times 10^{4}$ or 1E-04). The impact of carcinogenic chemicals was assessed by combining chemical-specific estimates of doses and toxicity values (slope factors) and comparing the estimated risks to specified risk levels. Noncancer effects were evaluated by calculating the ratio of the NOAEL or the LOAEL to the projected or estimated intake (i.e., dose). The resulting value is termed the MOE. The larger the MOE, the more unlikely it is that a noncancer adverse effect would occur. EPA established a guidance MOE value of 30 for arsenic and 100 for chromium (Cr(VI)) to account for the uncertainties associated with the toxicity data and other factors. Some of the 2001 SAP Panel members cautioned that when the calculated MOE is below the acceptable MOE, it does not necessarily mean that health effects will occur. The presence or absence of health effects should not be drawn solely on whether there calculated MOEs exceed the acceptable MOEs (U.S. EPA,

2001c). Arsenic risks were evaluated for noncancer and cancer effects and Cr(VI) risks were evaluated for noncancer effects only.

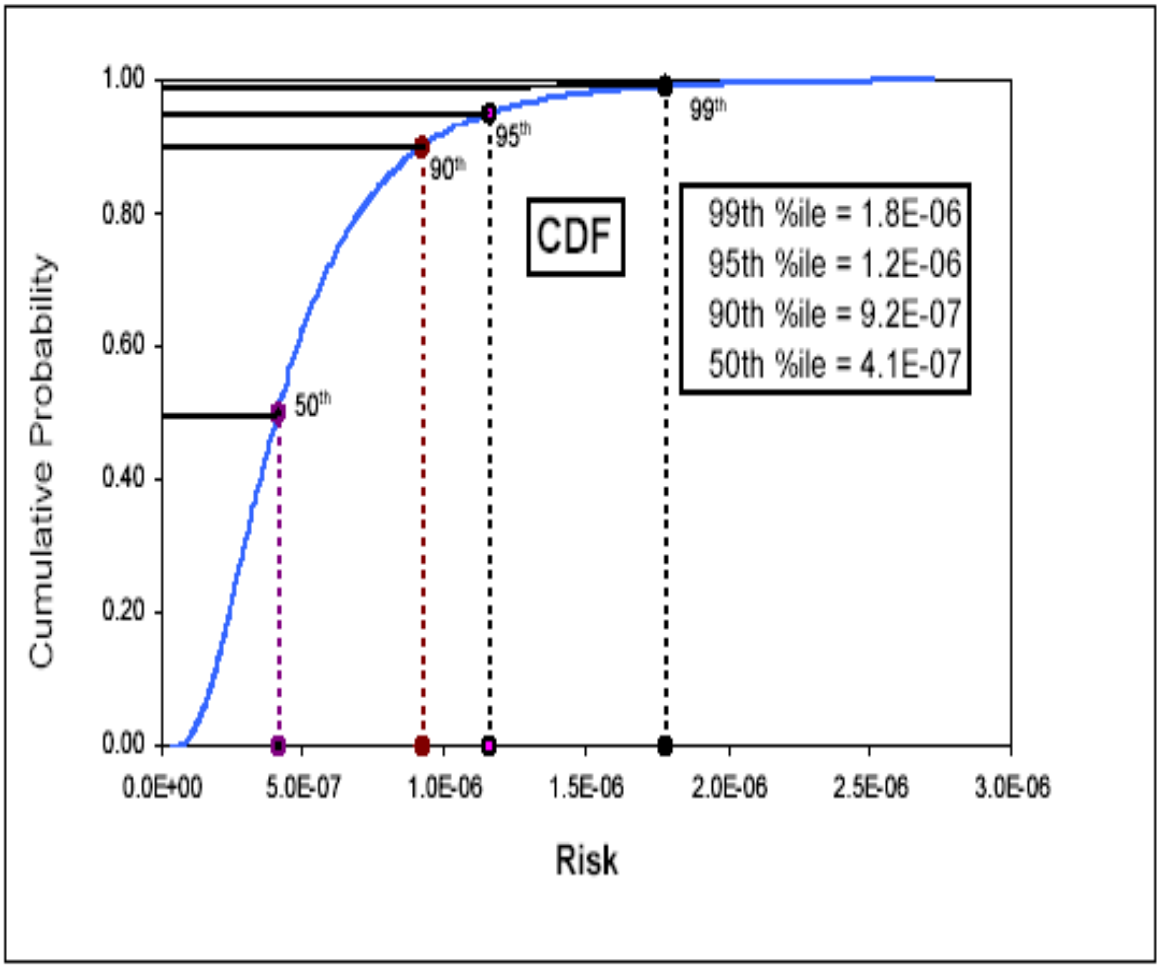



**Figure 5-1: A Cumulative Distribution Function (CDF) for Cancer Risk**

The interpretation of results in this risk assessment is somewhat unique. In traditional risk assessments, the intent is to inform risk managers whether or not a pre-established health effects threshold is exceeded. For example, in traditional cancer risk assessment, $1 \times 10^{6}$ is considered by OPP as the threshold of concern for residential scenarios. If this risk is exceeded, the risk manager then decides which remedial or mitigation measures are to be implemented to reduce the risks to an acceptable level. The intent of this present probabilistic risk assessment is slightly different. The goal of this risk assessment is to present the SAP with the calculated arsenic cancer risks to children (age 1-6) exposed to CCA-treated playsets and decks using a probabilistic risk analysis. It also identifies methods (e.g., sealants and hand washing) which can reduce the arsenic

cancer risks to children. However, there is no concluding statement regarding the percentiles of the distribution or point estimates (e.g., mean, $50^{th}$ , $90^{th}$ , $95^{th}$, etc) at which risk management decisions will be made. OPP intends to provide recommendations on how risk managers should interpret the results of this risk assessment, after receiving technical comments from the FIFRA SAP on evaluating probabilistic risk distributions. OPP will carefully consider the FIFRA SAP's comments on this issue.

## 5.2 Results

Noncancer MOEs and cancer risks were generated based on the exposure doses calculated by the SHEDS-Wood model (Zartarian et al., 2003), as summarized in Chapter 3 of this document, and the selected toxicological endpoint doses described in Chapter 4 of this document. Exposure doses were generated for the following:

- Two exposure routes - dermal and oral;
- Three durations - short, intermediate, and lifetime;
- Two sources of exposure - playset only, and playset and deck;
- Two climates - warm and cold; and
- Two chemicals - arsenic and chromium.

The exposed population of interest was children; ages 1 to 6 years. The durations of exposure were defined as short (1 day to 1 month); intermediate (1-6 months); and lifetime (6 years averaged over 75 years).

Table 5-1 presents a summary of the risk assessment results for noncancer and cancer risks. This table indicates which exposure conditions exceed specified risk levels. The summary is presented according to exposure scenario (i.e., source of exposure, climate, and duration of exposure for noncarcinogens). For the noncancer effects of arsenic, estimated MOEs were found to be greater than the guidance MOE of 30 for all exposures at the 99.6th percentile. For the noncancer effects of chromium, none of the exposure scenarios evaluated had estimated MOEs that fell below the target MOE of 100.

Cancer risks from arsenic were compared to the three levels of risk: $10^{-6}$, $10^{-5}$, and $10^{-4}$. Values reported in Table 5-1 are the cumulative probabilities above which the respective risk level has been exceeded. For example, for exposure to playsets only in a warm climate, the risk level of $10^{-6}$ was exceeded at the $3^{rd}$ percentile; 97% of the SHEDS-Wood simulated population had risks that exceeded $10^{-6}$. Cancer risks were found to be higher for the warm climate scenario than the cold climate scenario, reflecting the increased exposure in a warm climate. The $10^{-6}$ risk level was exceeded across all exposure scenarios at the very low end (i.e., less than $10^{th}$ percentile) of the cumulative probability distribution. The $10^{-4}$ risk level was

exceeded at the 90th percentile for exposure to playsets and decks in a warm climate and at the 97th percentile in a cold climate. For exposure to playsets only, the $10^{-4}$ level was exceeded at the 97th and 99th percentiles for warm and cold climates, respectively.

**Table 5-1. Summary of Risk Assessment Results**

*Noncancer MOEs for Arsenic and Chromium*

| Source of Exposure | Climate | Duration of Exposure | Arsenic MOE <30 | Chromium MOE <100 |
|---|---|---|---|---|
| Playset Only | Warm | Short & Intermediate | >99.6th Percentile | None |
| | Cold | | | |
| Playset and Deck | Warm | Short & Intermediate | >99.6th Percentile | None |
| | Cold | | | |

*Cancer Risks for Arsenic* [a]

| Source of Exposure | Climate | Cumulative Percentiles at Specified Risk Levels | | |
|---|---|---|---|---|
| | | $10^{-6}$ | $10^{-5}$ | $10^{-4}$ |
| Playset Only | Warm | 3rd | 47th | 97th |
| | Cold | 9th | 69th | 99th |
| Playset and Deck | Warm | <1st | 23rd | 90th |
| | Cold | 2nd | 49th | 97th |

[a] Percentiles in this table represent the percent of the simulated population that have risks less than or equal to the stated risk level; e.g., at $10^{-6}$, 3% of the population have risks less than $10^{-6}$ and 97% have risks greater than $10^{-6}$.

The remainder of this chapter presents the detailed results of the risk characterization. Noncancer MOE results are presented in Section 5.2.1 and cancer risk results are presented in Section 5.2.2.

### 5.2.1 Noncancer Effects

Noncancer effects were evaluated by calculating the ratio of the NOAEL or the LOAEL to the projected or estimated intake (i.e., dose). The resulting value is termed the MOE. The

larger the MOE, the more unlikely it is that a noncancer adverse effect would occur. EPA established an acceptable MOE value of 30 for arsenic and 100 for chromium (Cr(VI)) to account for the uncertainties associated with the toxicity data and other factors. When the calculated MOE is below the acceptable MOE, it does not necessary mean that health effects will occur. EPA uses the MOE approach in a screening level capacity only. That is, firm conclusions on the presence or absence of health effects should not be drawn solely on whether the calculated MOEs exceed the acceptable MOEs. For arsenic, the LOAEL used was 0.05 mg/kg/day and the target MOE was 30. For Cr(VI), the NOAEL used was 0.5 mg/kg/day and the guidance MOE was 100. The equation used for this calculation was:

MOE = NOAEL or LOAEL / ADD

Where:

NOAEL = No-Observed-Adverse-Effect Level (mg/kg/day);
LOAEL = Lowest-Observed-Adverse-Effect Level (mg/kg/day); and
ADD = Average Daily Dose (mg/kg/day).

Results are first presented for general population exposure and then for children with pica behavior.

Tables 5-2 and 5-3 present the MOEs for children who play on outdoor CCA-treated playsets only. The MOEs were calculated based on different exposure durations (short-term and intermediate) and climates (warm and cold). Data were generated, and are presented in this section, for the mean, median, 95$^{th}$ percentile, and 99$^{th}$ percentile of the distributions. Table 5-2 presents the arsenic MOEs for exposure to playsets only. The cold climate conditions were found to have a larger MOE than for the warm climate conditions. For all conditions, the MOEs were found to be substantially greater (minimum factor of 2) than the guidance MOE of 30. Table 5-3 presents the MOEs for Cr(VI) for the same scenarios.[1] All the chromium MOEs were found to be at least two orders of magnitude above the target MOE of 100.

[1] Cr(VI) exposures/risks from soil ingestion were calculated by assuming 10 percent of the chromium in soil was present as Cr(VI).

**Table 5-2. Arsenic Noncancer MOEs - Playset Only**

| Time Frame | Arsenic (guidance MOE = 30) LOAEL of 0.05 mg/kg/day | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short | 595 | 1,200 | 1,700 | 3,600 | 173 | 304 | 61 | 108 |
| Intermediate | 849 | 1,300 | 1,800 | 4,600 | 216 | 415 | 116 | 128 |

**Table 5-3. Chromium (Cr(VI)) Noncancer MOEs - Playset Only**

| Time Frame | Chromium (VI) (guidance MOE = 100) NOAEL of 0.5 mg/kg/day | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short | 7.2E+07 | 2.4E+08 | 1.7E+06 | 2.4E+07 | 1.0E+05 | 7.0E+05 | 3.1E+04 | 1.6E+05 |
| Intermediate | 2.2E+07 | 3.4E+08 | 1.8E+06 | 2.5E+07 | 1.3E+05 | 8.1E+05 | 3.0E+04 | 2.8E+05 |

The noncancer MOEs for exposure to both playsets and decks are presented in Table 5-4 for arsenic and Table 5-5 for chromium. The results for exposure to arsenic were similar to those for playsets alone. The MOEs for all exposures were found to be greater than the guidance value of 30. Cold climate conditions had higher MOEs (i.e., lower dose) than warm climate conditions. None of the MOEs for chromium (Table 5-5) were below the guidance value; even at the 99$^{th}$ percentile, these MOEs were orders of magnitude above the guidance MOE of 100.

**Table 5-4. Arsenic Noncancer MOEs - Playset and Deck**

| Time Frame | Arsenic (guidance MOE = 30) LOAEL of 0.05 mg/kg/day | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Short | 383 | 745 | 771 | 2,000 | 107 | 230 | 53 | 72 |
| Intermediate | 393 | 720 | 740 | 1,600 | 111 | 211 | 52 | 84 |

**Table 5-5. Chromium (Cr(VI)) Noncancer MOEs - Playset and Deck**

| **Time Frame** | **Chromium (VI) (guidance MOE = 100) NOAEL of 0.5 mg/kg/day** | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | **Mean** | | **Median** | | **95%ile** | | **99%ile** | |
| | **Warm** | **Cold** | **Warm** | **Cold** | **Warm** | **Cold** | **Warm** | **Cold** |
| Short | 3.9E+06 | 8.6E+07 | 1.2E+06 | 1.3E+07 | 1.2E+05 | 4.7E+05 | 3.7E+04 | 1.3E+05 |
| Intermediate | 3.6E+06 | 4.1E+07 | 1.3E+06 | 1.2E+07 | 1.2E+05 | 5.2E+05 | 4.7E+04 | 4.2E+05 |

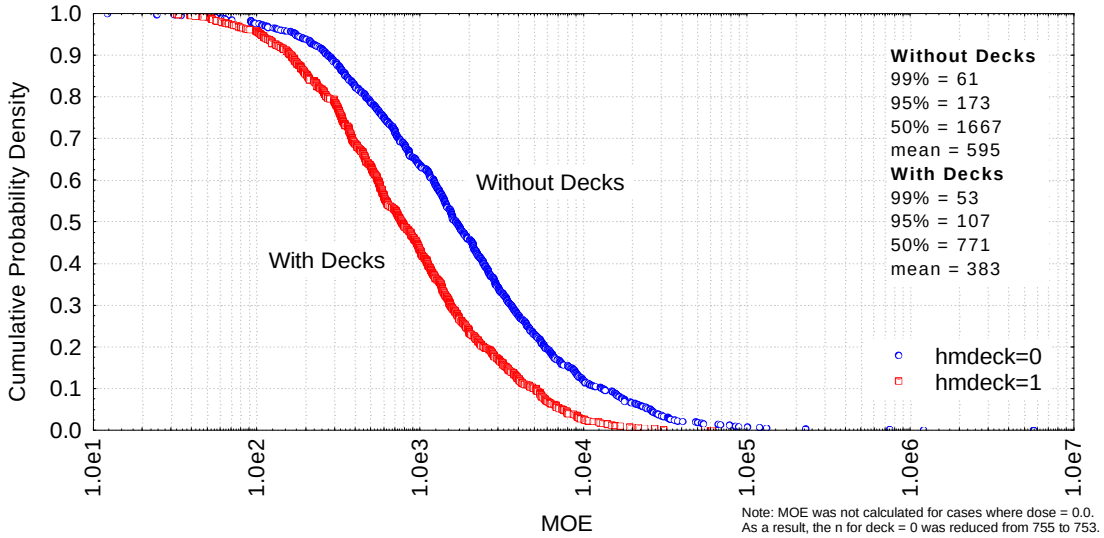
*Short-term MOEs*

Arsenic risk cumulative density functions and probability density functions (CDFs/PDFs) were plotted for all exposure scenarios. Short duration (i.e., 1 day to 1 month) risks are shown in Figure 5-2 for warm climate conditions and in Figure 5-3 for cold climate conditions. Each figure presents risks from exposure to playsets only (without decks) and playsets and decks (with decks). Probabilistic short-term MOE distributions and risk levels are presented in Table 5-6 for warm climates and Table 5-7 for cold climates for arsenic risks with playsets and decks, and playsets only. MOEs were found to be less than 30 only above the 99$^{th}$ percentile for warm climates for exposure to playsets only. However, MOEs were greater than 30 with playsets only under cold climate conditions.

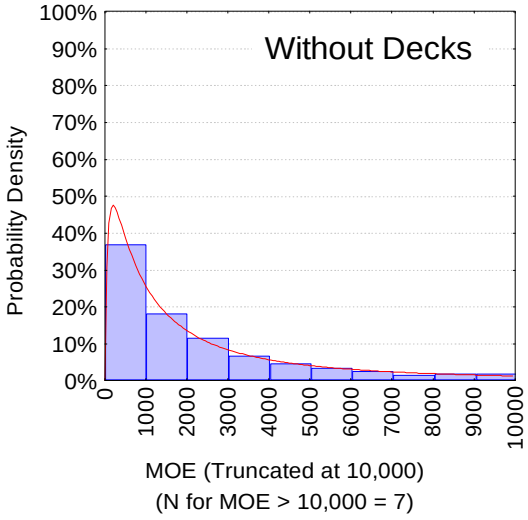
Cr(VI) probabilistic short-term MOE distributions and risk levels (soil ingestion only) for children with playsets and decks, and playsets only in warm and cold climates are presented in Tables 5-8 and 5-9, respectively. All MOEs are >100 for all climate scenarios.

Figure 5-2

MOE of Short-Term ADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(separated by children with and without decks)
Scatterplot (E5 (table 18).sta)




Histogram (E5 (table 18).sta)
y = 746 * 1004.83 * lognorm (x, 7.428197, 1.46652




Histogram (E5 (table 18).sta)
y = 710 * 1002.237 * lognorm (x, 6.71147, 1.30297

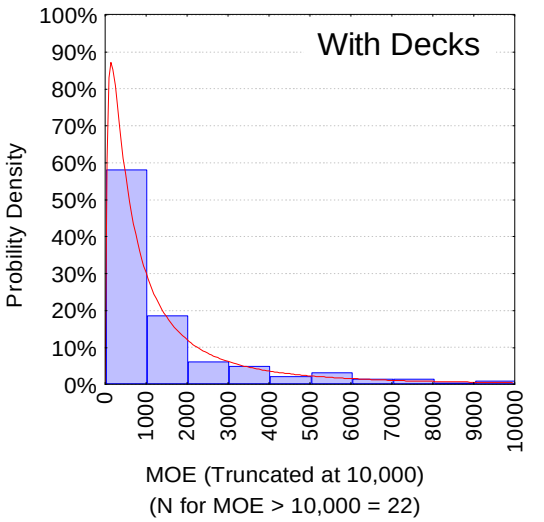


Figure 5-3

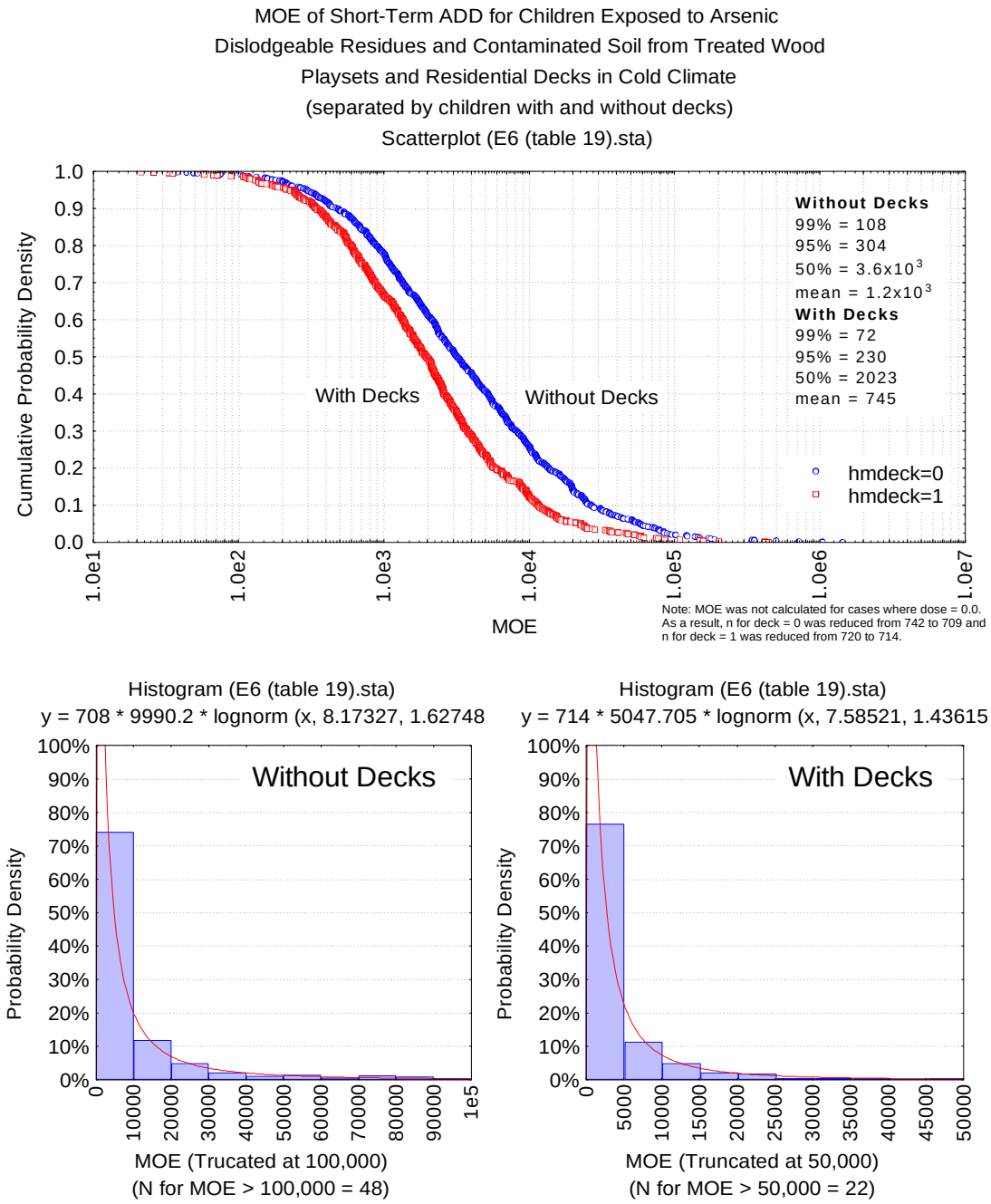

MOE of Short-Term ADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Cold Climate
(separated by children with and without decks)
Scatterplot (E6 (table 19).sta)
Cumulative Probability Density
MOE
With Decks
Without Decks
Without Decks
99% = 108
95% = 304
50% = 3.6x10^3
mean = 1.2x10^3
With Decks
99% = 72
95% = 230
50% = 2023
mean = 745
hmdeck=0
hmdeck=1
Note: MOE was not calculated for cases where dose = 0.0.
As a result, n for deck = 0 was reduced from 742 to 709 and
n for deck = 1 was reduced from 720 to 714.
Histogram (E6 (table 19).sta)
y = 708 * 9990.2 * lognorm (x, 8.17327, 1.62748)
Without Decks
Probability Density
MOE (Trucated at 100,000)
(N for MOE > 100,000 = 48)
Histogram (E6 (table 19).sta)
y = 714 * 5047.705 * lognorm (x, 7.58521, 1.43615)
With Decks
Probability Density
MOE (Truncated at 50,000)
(N for MOE > 50,000 = 22)

**Table 5-6. Probabilistic Short-Term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Based on Short-term ADDs in Table 18 from the SHEDS-Wood Document)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 4.1E-03 | 12 | |
| 99 | 8.2E-04 | 61 | |
| 95 | 2.9E-04 | 173 | |
| 90 | 1.8E-04 | 274 | |
| 50 | 3.0E-05 | 1.7E+03 | |
| 10 | 3.7E-06 | 1.3E+04 | |
| 5 | 1.9E-06 | 2.6E+04 | |
| 1 | 4.2E-07 | 1.2E+05 | |
| minimum dose | 0.0E+00 | N/A | |
| 99.6 | 1.6E-03 | 30 | |
| Note: Percentiles include cases where dose = 0 | | | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 1.5E-03 | 32 | |
| 99 | 9.5E-04 | 53 | |
| 95 | 4.7E-04 | 107 | |
| 90 | 3.1E-04 | 163 | |
| 50 | 6.5E-05 | 771 | |
| 10 | 9.7E-06 | 5.1E+03 | |
| 5 | 6.6E-06 | 7.5E+03 | |
| 1 | 3.0E-06 | 1.7E+04 | |
| minimum dose | 8.2E-07 | 6.1E+04 | |
| >99.9 | N/A | 30 | |
| Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency. | | | |

**Table 5-7. Probabilistic Short-term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climates (Based on Short-Term ADDs in Table 19 from the SHEDS-Wood Document)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 1.3E-03 | 38 | |
| 99 | 4.6E-04 | 108 | |
| 95 | 1.6E-04 | 304 | |
| 90 | 1.0E-04 | 497 | |
| 50 | 1.4E-05 | 3.6E+03 | |
| 10 | 9.5E-07 | 5.3E+04 | |
| 5 | 1.0E-07 | 5.0E+05 | |
| 1 | 0.0E+00 | N/A | |
| minimum dose | 0.0E+00 | N/A | |
| >99.9 | N/A | 30 | |
| Note: Percentiles include cases where dose = 0 | | | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 2.3E-03 | 21 | |
| 99 | 7.0E-04 | 72 | |
| 95 | 2.2E-04 | 230 | |
| 90 | 1.4E-04 | 351 | |
| 50 | 2.5E-05 | 2.0E+03 | |
| 10 | 4.0E-06 | 1.2E+04 | |
| 5 | 2.1E-06 | 2.4E+04 | |
| 1 | 1.2E-07 | 4.2E+05 | |
| minimum dose | 0.0E+00 | N/A | |
| 99.6 | 1.5E-03 | 30 | |
| Note: Percentiles include cases where dose = 0 | | | |
| Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency. | | | |

**Table 5-8. Probabilistic Short-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Warm Climate (Soil Ingestion Only)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 3.4E-05 | 1.5E+04 | |
| 99 | 1.5E-05 | 3.3E+04 | |
| 95 | 4.3E-06 | 1.2E+05 | |
| 90 | 1.9E-06 | 2.6E+05 | |
| 50 | 1.8E-07 | 2.8E+06 | |
| 10 | 1.5E-08 | 3.3E+07 | |
| 5 | 5.6E-09 | 8.9E+07 | |
| 1 | 7.8E-10 | 6.4E+08 | |
| minimum dose | 0.0E+00 | N/A | |
| >99.9 | N/A | 100 | |
| Note: Percentiles include cases where dose = 0 | | | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 4.2E-05 | 1.2E+04 | |
| 99 | 1.3E-05 | 3.8E+04 | |
| 95 | 3.9E-06 | 1.3E+05 | |
| 90 | 2.1E-06 | 2.4E+05 | |
| 50 | 2.5E-07 | 2.0E+06 | |
| 10 | 2.0E-08 | 2.4E+07 | |
| 5 | 1.2E-08 | 4.3E+07 | |
| 1 | 2.2E-09 | 2.3E+08 | |
| minimum dose | 8.1E-10 | 6.2E+08 | |
| >99.9 | N/A | 100 | |
| Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency. | | | |

**Table 5-9. Probabilistic Short-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Cold Climate (Soil Ingestion Only)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 1.9E-05 | 2.7E+04 | |
| 99 | 3.0E-06 | 1.6E+05 | |
| 95 | 6.9E-07 | 7.2E+05 | |
| 90 | 3.0E-07 | 1.7E+06 | |
| 50 | 1.5E-08 | 3.4E+07 | |
| 10 | 3.8E-10 | 1.3E+09 | |
| 5 | 6.4E-11 | 7.8E+09 | |
| 1 | 0.0E+00 | N/A | |
| minimum dose | 0.0E+00 | N/A | |
| >99.9 | N/A | 100 | |
| Note: Percentiles include cases where dose = 0 | | | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 1.5E-05 | 3.4E+04 | |
| 99 | 3.7E-06 | 1.3E+05 | |
| 95 | 1.0E-06 | 4.9E+05 | |
| 90 | 5.2E-07 | 9.6E+05 | |
| 50 | 2.9E-08 | 1.7E+07 | |
| 10 | 2.0E-09 | 2.5E+08 | |
| 5 | 8.0E-10 | 6.2E+08 | |
| 1 | 0.0E+00 | N/A | |
| minimum dose | 0.0E+00 | N/A | |
| >99.9 | N/A | 100 | |
| Note: Percentiles include cases where dose = 0 | | | |
| Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency. | | | |

Arsenic PDFs/CDFs and risk levels are not presented for pica children. However, short-term MOEs for arsenic for pica children and playsets only in warm climates are presented in Table 5-10. For all scenarios evaluated, the arsenic short-term MOEs were greater than 30 at the 95$^{th}$ percentile. For playset only exposure, the MOE was less than 30 near the 99$^{th}$ percentile. For deck and playset exposure, the MOE was slightly greater than 30 at the 99$^{th}$ percentile. This reversal of deck and playset and playset only risks was due to the exposure model's instability at this extreme dose levels. The SHEDS-Wood assessment did not calculate short-term MOEs for chromium for pica children. See Appendix B for additional short-term MOEs.

*Intermediate-term MOEs*

Arsenic risk PDFs/CDFs were plotted for all exposure scenarios. Intermediate duration (i.e., 1 to 6 months) risks are shown in Figure 5-4 for warm climate conditions and Figure 5-5 for cold climate conditions. Each figure presents risks from exposure to playsets only, and playsets and decks. Probabilistic intermediate-term MOE distributions and risk levels are presented in Table 5-11 for warm climates and Table 5-12 for cold climates for arsenic risks with playsets and decks, and playsets only. MOEs were shown to be less than 30 at the 99$^{th}$ percentile for both warm and cold climate conditions for exposure to playsets and decks and playsets only.

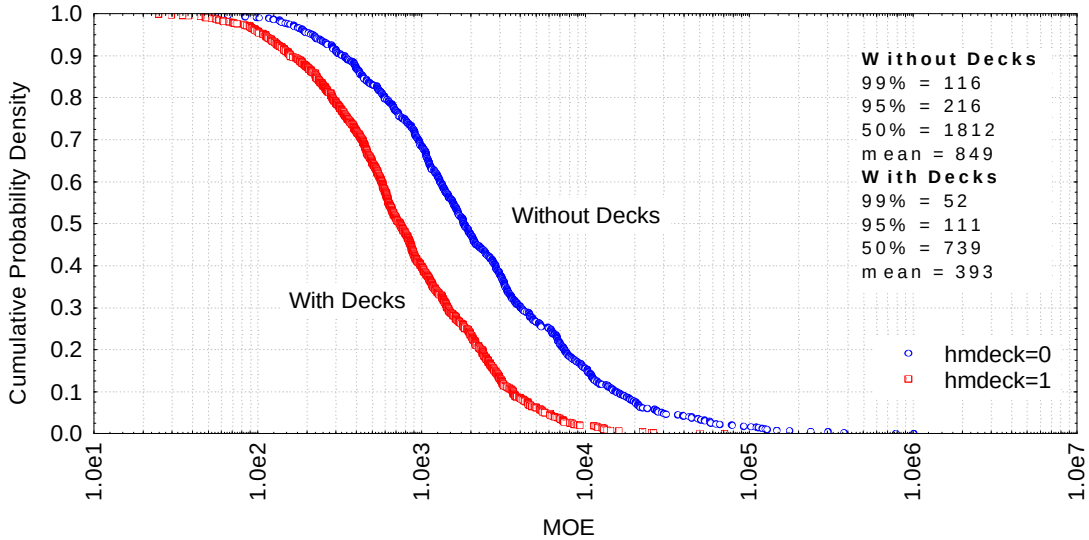
Chromium intermediate-term MOEs and risk levels (soil ingestion only) for children with playsets and decks, and playsets only at warm and cold climates are presented in Tables 5-13 and 5-14, respectively. All MOEs were found to be greater than 100 for exposure to playsets and decks, and playsets only for both climate scenarios.

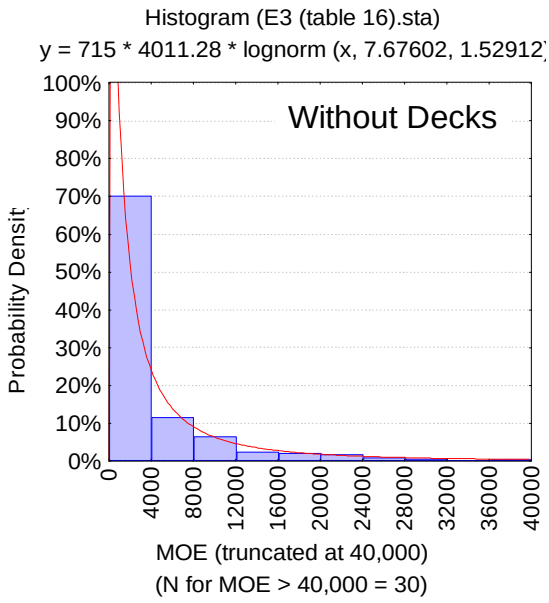
**Table 5-10. Arsenic Noncancer Short-Term MOEs for Pica Children in Warm Climate (Based on Short-Term ADDs from Table 33 in the SHEDS-Wood Document)**

| **Arsenic (guidance MOE = 30) LOAEL of 0.05 mg/kg/day** | | | | |
|---|---|---|---|---|
| Source of Exposure | Mean | Median | 95%ile | 99%ile |
| Playset Only | 219 | 503 | 65 | 23 |
| Playset and Deck | 163 | 339 | 49 | 31 |

Figure 5-4

MOE of Intermediate-Term ADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(separated by children with and without decks)
Scatterplot (E3 (table 16).sta)



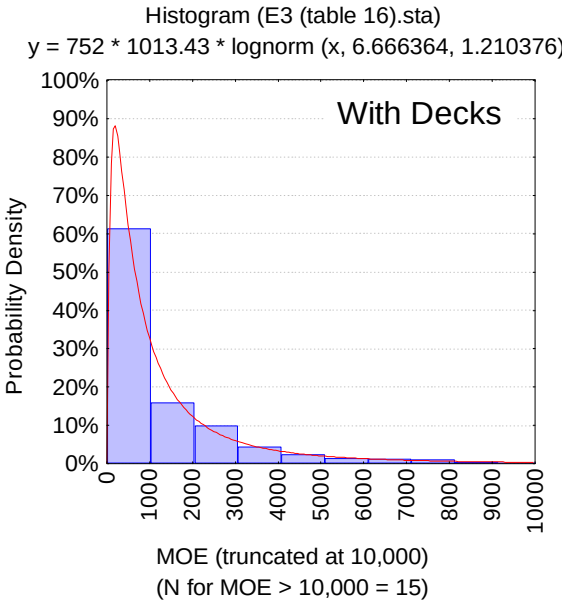
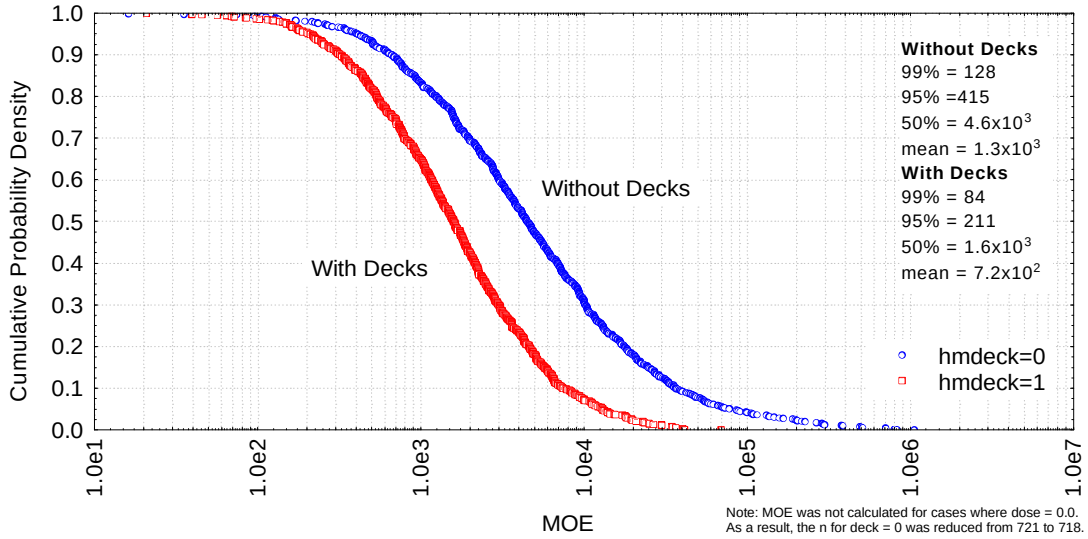
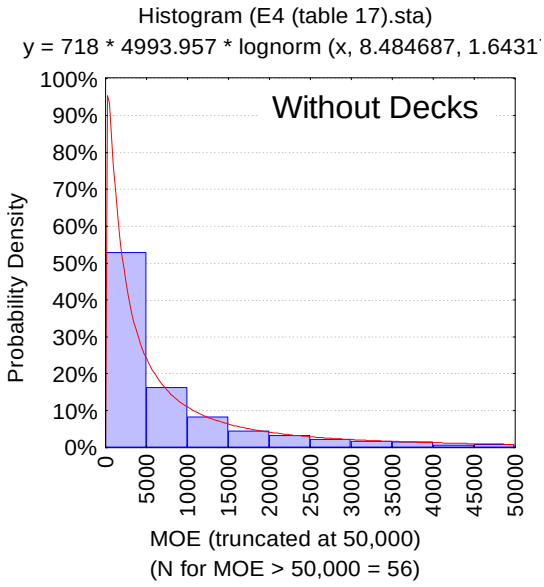



Figure 5-5

MOE of Intermediate-Term ADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Cold Climate
(separated by children with and without decks)
Scatterplot (E4 (table 17).sta)



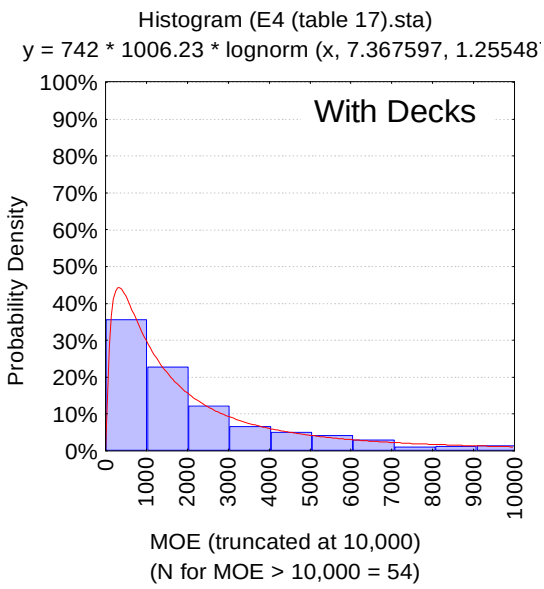



**Table 5-11. Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Based on the ADDs in Table 16 from the SHEDS-Wood Document)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 8.6E-04 | 58 | |
| 99 | 4.3E-04 | 116 | |
| 95 | 2.3E-04 | 216 | |
| 90 | 1.5E-04 | 339 | |
| 50 | 2.8E-05 | 1.8E+03 | |
| 10 | 3.2E-06 | 1.6E+04 | |
| 5 | 1.7E-06 | 3.0E+04 | |
| 1 | 3.9E-07 | 1.3E+05 | |
| minimum dose | 4.9E-08 | 1.0E+06 | |
| >99.9 | | 30 | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 2.0E-03 | 25 | |
| 99 | 9.6E-04 | 52 | |
| 95 | 4.5E-04 | 111 | |
| 90 | 3.1E-04 | 159 | |
| 50 | 6.8E-05 | 740 | |
| 10 | 1.4E-05 | 3.6E+03 | |
| 5 | 8.8E-06 | 5.7E+03 | |
| 1 | 3.7E-06 | 1.4E+04 | |
| minimum dose | 7.0E-07 | 7.1E+04 | |
| 99.6 | 1.4E-03 | 30 | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

**Table 5-12. Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climates (Based on ADDs in Table 17 from the SHEDS-Wood Document)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 3.1E-03 | 16 | |
| 99 | 3.9E-04 | 128 | |
| 95 | 1.2E-04 | 415 | |
| 90 | 7.5E-05 | 664 | |
| 50 | 1.1E-05 | 4.6E+03 | |
| 10 | 1.3E-06 | 3.8E+04 | |
| 5 | 5.7E-07 | 8.8E+04 | |
| 1 | 9.8E-08 | 5.1E+05 | |
| minimum dose | 0.0E+00 | N/A | |
| 99.7 | 1.4E-03 | 30 | |
| Note: Percentiles include cases where dose = 0 | | | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Average Daily Dose (ADD) mg/kg/day | MOE | Risk Level |
| | | | **MOE = 30** |
| maximum dose | 2.4E-03 | 21 | |
| 99 | 5.9E-04 | 84 | |
| 95 | 2.4E-04 | 211 | |
| 90 | 1.5E-04 | 325 | |
| 50 | 3.1E-05 | 1.6E+03 | |
| 10 | 6.7E-06 | 7.4E+03 | |
| 5 | 3.9E-06 | 1.3E+04 | |
| 1 | 1.5E-06 | 3.4E+04 | |
| minimum dose | 7.2E-07 | 6.9E+04 | |
| 99.7 | 1.3E-03 | 30 | |
| Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency. | | | |

**Table 5-13. Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Warm Climate (Soil Ingestion Only)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 3.6E-05 | 1.4E+04 | |
| 99 | 1.6E-05 | 3.0E+04 | |
| 95 | 3.5E-06 | 1.4E+05 | |
| 90 | 1.6E-06 | 3.0E+05 | |
| 50 | 1.6E-07 | 3.1E+06 | |
| 10 | 1.1E-08 | 4.6E+07 | |
| 5 | 5.0E-09 | 9.9E+07 | |
| 1 | 6.6E-10 | 7.6E+08 | |
| minimum dose | 1.0E-11 | 5.0E+10 | |
| >99.9 | N/A | 100 | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 6.6E-05 | 7.5E+03 | |
| 99 | 1.0E-05 | 4.9E+04 | |
| 95 | 3.5E-06 | 1.4E+05 | |
| 90 | 1.8E-06 | 2.8E+05 | |
| 50 | 2.3E-07 | 2.2E+06 | |
| 10 | 2.2E-08 | 2.2E+07 | |
| 5 | 9.4E-09 | 5.3E+07 | |
| 1 | 3.4E-09 | 1.5E+08 | |
| minimum dose | 6.1E-10 | 8.2E+08 | |
| >99.9 | N/A | 100 | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

**Table 5-14. Probabilistic Intermediate-Term MOE Distributions and Risk Levels for Children Exposed to Chromium (VI) in Cold Climate (Soil Ingestion Only)**

| | | | |
|---|---|---|---|
| **Playset Only** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 8.4E-06 | 5.9E+04 | |
| 99 | 1.8E-06 | 2.8E+05 | |
| 95 | 6.0E-07 | 8.3E+05 | |
| 90 | 2.7E-07 | 1.9E+06 | |
| 50 | 1.4E-08 | 3.5E+07 | |
| 10 | 6.5E-10 | 7.7E+08 | |
| 5 | 3.4E-10 | 1.5E+09 | |
| 1 | 3.5E-11 | 1.4E+10 | |
| minimum dose | 0.0E+00 | N/A | |
| >99.9 | N/A | 100 | |
| Note: Percentiles include cases where dose = 0 | | | |
| | | | |
| **Playset and Deck** | | | |
| Percentile of Exposure | Cr VI Average Daily Dose (ADD) mg/kg/day | Cr VI MOE | Risk Level |
| | | | **MOE = 100** |
| maximum dose | 1.2E-05 | 4.3E+04 | |
| 99 | 3.4E-06 | 1.5E+05 | |
| 95 | 8.9E-07 | 5.6E+05 | |
| 90 | 4.3E-07 | 1.2E+06 | |
| 50 | 3.5E-08 | 1.4E+07 | |
| 10 | 2.7E-09 | 1.9E+08 | |
| 5 | 1.3E-09 | 3.7E+08 | |
| 1 | 4.8E-10 | 1.0E+09 | |
| minimum dose | 1.4E-10 | 3.6E+09 | |
| >99.9 | N/A | 100 | |
| Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency. | | | |

### 5.2.2 Carcinogenic Effects

For carcinogens, risks were estimated as the probability of increased cancer incidence or excess lifetime cancer risk. The carcinogenic slope factor or $Q_1$* represents the 95 percent upper confidence limit (UCL) of the probability of response per unit intake of a chemical over a lifetime, and converts estimated intakes directly to incremental risk (U.S. EPA, 1990). Cancer risk was computed as follows:

$$\text{Risk} = \text{LADD} \times Q_1^*$$

Where:

LADD = Lifetime Average Daily Dose (mg/kg/day)
$Q_1$* = Carcinogenic slope factor [1/(mg/kg/-day)]

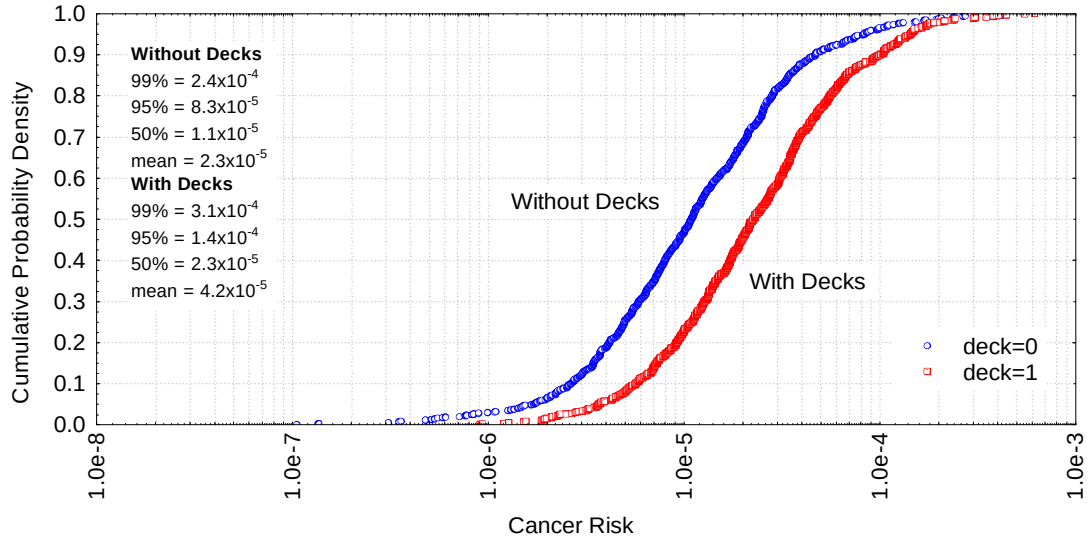
Doses (i.e., LADDs) corresponding to all risks calculated here are presented in Zartarian et al. (2003). The lifetime risk was based on a 6 year duration of exposure (ages 1-6 years) and averaged over a lifetime of 75 years.

Cancer risk results are presented in this section from four different perspectives. First, risks are presented in the same manner as the noncancer effects; namely, the four different exposure points (i.e., mean, median, 95$^{th}$ percentile, and 99$^{th}$ percentile) are presented. Second, risks are shown in cumulative probability density and probability density plots. Third, percentiles from the cumulative distribution that correspond to the three levels of EPA's risk range ($10^{-6}$, $10^{-5}$, and $10^{-4}$) are presented. And fourth, total risk is shown for two broad sources of exposure: soils and residues. The risk data for exposure to residues and soil in warm and cold climates are shown in line plots (Figures 5-6 and 5-7) for the different exposure scenarios (i.e., playset only and playset and deck) .

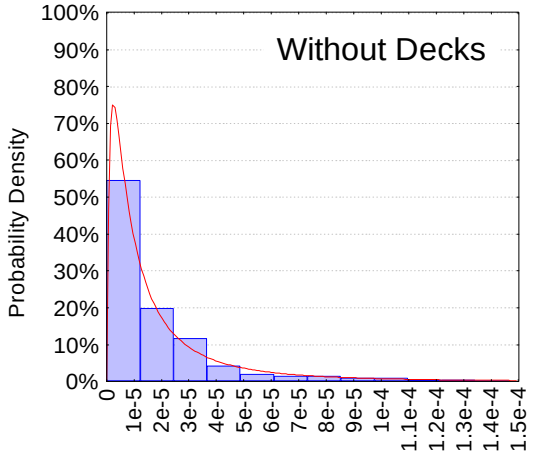
Table 5-15 summarizes the arsenic cancer risks for children who contact CCA-treated playsets and decks in warm and cold climates at the three exposure points of interest. Warm climate exposures were greater than cold climate exposures. Thus, cancer risks were correspondingly greater. Exposure at the mean and median were in the range $10^{-6}$ to$10^{-5}$. This range was exceeded with exposure to decks and playsets in warm climates at approximately the 95$^{th}$ percentile (1.4 x$10^{-4}$). At the 99$^{th}$ percentile, the $10^{-4}$ risk level was exceeded for playsets and decks exposure for cold climate conditions.

Figure 5-6

Cancer Risk (Lifetime Term) for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(separated by children with and without decks)
Scatterplot (E1 (table 14).sta)




Histogram (E1 (table 14).sta)
y = 728 * 0.000012 * lognorm (x, -11.42044, 1.220




Histogram (E1 (table 14).sta)
y = 738 * 0.00001 * lognorm (x, -10.689, 1.098477

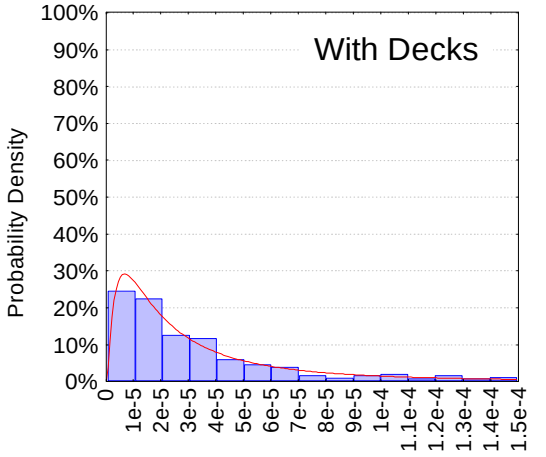


Figure 5-7

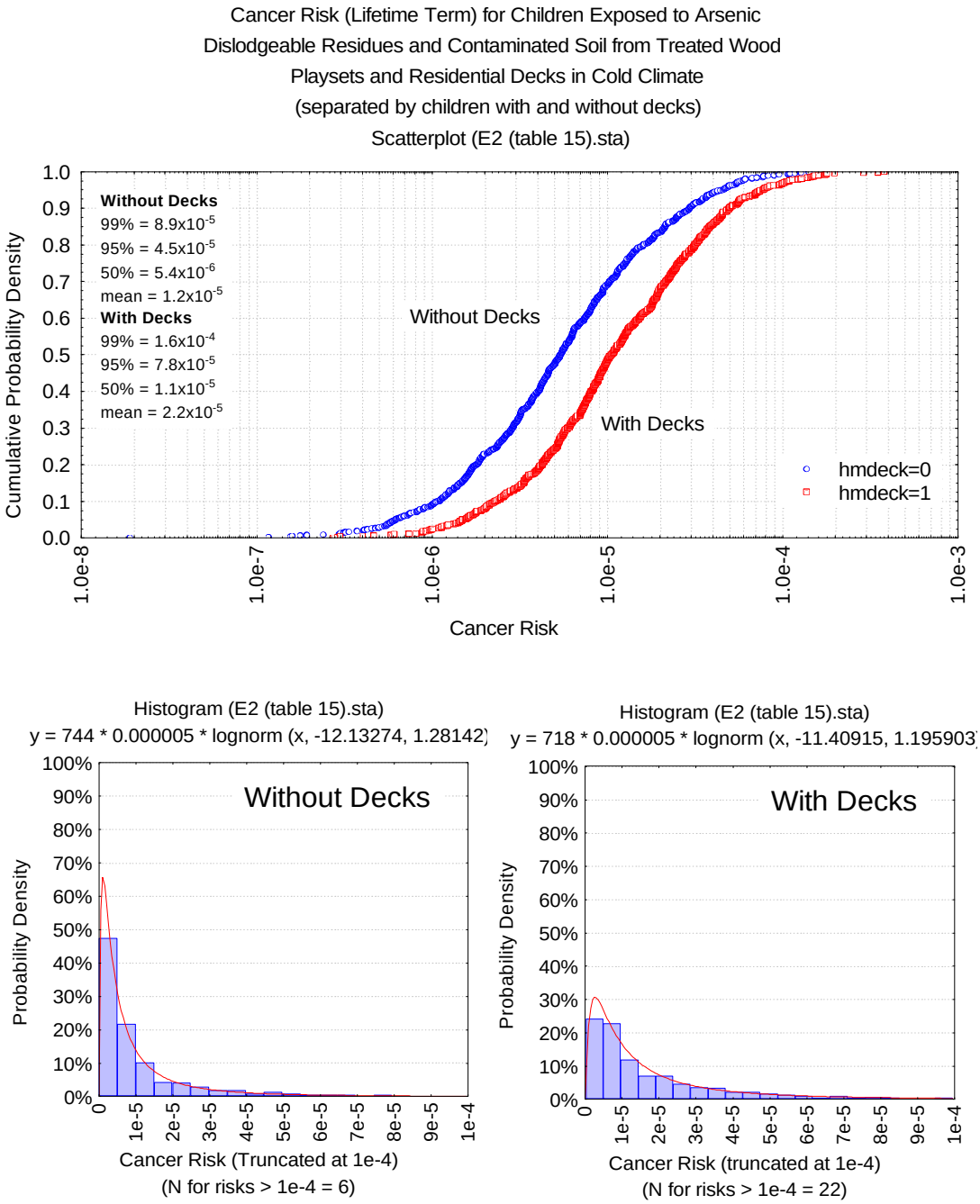

Cancer Risk (Lifetime Term) for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Cold Climate
(separated by children with and without decks)
Scatterplot (E2 (table 15).sta)
Without Decks
99% = 8.9x10-5
95% = 4.5x10-5
50% = 5.4x10-6
mean = 1.2x10-5
With Decks
99% = 1.6x10-4
95% = 7.8x10-5
50% = 1.1x10-5
mean = 2.2x10-5
Without Decks
With Decks
hmdeck=0
hmdeck=1
Cumulative Probability Density
Cancer Risk
Histogram (E2 (table 15).sta)
y = 744 * 0.000005 * lognorm (x, -12.13274, 1.28142)
Without Decks
Probability Density
Cancer Risk (Truncated at 1e-4)
(N for risks > 1e-4 = 6)
Histogram (E2 (table 15).sta)
y = 718 * 0.000005 * lognorm (x, -11.40915, 1.195903)
With Decks
Probability Density
Cancer Risk (truncated at 1e-4)
(N for risks > 1e-4 = 22)

**Table 5-15. Arsenic Cancer Risks**

| Scenario | Arsenic ($Q_1$*= 3.67 (mg/kg/day)$^{-1}$) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Playset and Deck | 4.2E-05 | 2.2E-05 | 2.3E-05 | 1.1E-05 | 1.4E-04 | 7.8E-05 | 3.1E-04 | 1.6E-04 |
| Playset Only | 2.3E-05 | 1.2E-05 | 1.1E-05 | 5.4E-06 | 8.3E-05 | 4.5E-05 | 2.4E-04 | 8.9E-05 |

The cumulative probability density and probability density plots for warm climates and cold climates are presented in Figures 5-6 and 5-7, respectively. Risks due to both types of exposure (i.e., playsets only, and playsets and decks) are shown on the cumulative probability density plot. Figure 5-6 shows that risks for warm climate conditions are less than the EPA's risk level of $10^{-6}$ only at extremely low cumulative probabilities (e.g., less than the 5$^{th}$ percentile for both exposure to playsets alone as well as with deck exposure). For cold climate conditions (Figure 5-7), the same pattern is evident. However, the cumulative probability curve shifted to the left slightly, as risks were lower due to lower levels of exposure.

Tables 5-16 and 5-17 present cumulative percentiles at the three specified risk levels for warm and cold climates playsets and decks, and playsets only. For the warm climate, risks were equal to $10^{-6}$ at the 3$^{rd}$ percentile for exposure to playsets only and less than the 1$^{st}$ percentile for exposure to playsets and decks. In cold climates, these percentiles are the 9$^{th}$ and 2$^{nd}$, respectively. Risks of $10^{-4}$ were exceeded at cumulative percentiles that range from the 90$^{th}$ percentile for exposure to playsets and decks in a warm climate to the 99$^{th}$ percentile for playsets only in a cold climate.

The last analysis of these data focused on evaluating the differing contributions to total risk from soil and disloadgeable residues. This was a screening level analysis using the summary statistics for each route of exposure. The estimated risks are considered approximations because inaccuracies occur when exposures are summed across routes at the quartile level. This is due to the way the Monte Carlo simulations were conducted and the outputs summarized. Errors are expected to be greatest at the lowest percentiles, minimal at the median, and then increase in the upper percentiles. Inaccuracy at the upper percentiles ranges from 10% to approximately greater than 20% depending on the exposure scenario. Appendix B shows how risks from ingestion and dermal absorption were summed across decks and playsets, and for residue and soil exposures. Plots presented are for warm climate, as this scenario had the highest risk.

**Table 5-16. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Based on LADDs in Table 14 from the SHEDS-Wood Document)**

| | | | | | |
|---|---|---|---|---|---|
| **Playset Only** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 1.3E-04 | 4.9E-04 | | | |
| 99 | 6.5E-05 | 2.4E-04 | | | |
| 95 | 2.3E-05 | 8.3E-05 | | | |
| 90 | 1.3E-05 | 4.7E-05 | | | |
| 50 | 3.0E-06 | 1.1E-05 | | | |
| 10 | 7.2E-07 | 2.6E-06 | | | |
| 5 | 4.6E-07 | 1.7E-06 | | | |
| 1 | 1.3E-07 | 4.8E-07 | | | |
| minimum | 2.9E-08 | 1.0E-07 | | | |
| 96.6 | 2.7E-05 | 1.0E-04 | | | |
| 46.7 | 2.7E-06 | 1.0E-05 | | | |
| 3.0 | 2.7E-07 | 1.0E-06 | | | |
| | | | | | |
| **Playset and Deck** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 1.7E-04 | 6.1E-04 | | | |
| 99 | 8.4E-05 | 3.1E-04 | | | |
| 95 | 3.9E-05 | 1.4E-04 | | | |
| 90 | 2.8E-05 | 1.0E-04 | | | |
| 50 | 6.1E-06 | 2.3E-05 | | | |
| 10 | 1.5E-06 | 5.6E-06 | | | |
| 5 | 1.0E-06 | 3.7E-06 | | | |
| 1 | 5.1E-07 | 1.9E-06 | | | |
| minimum | 2.5E-07 | 9.1E-07 | | | |
| 89.8 | 2.7E-05 | 1.0E-04 | | | |
| 22.9 | 2.7E-06 | 1.0E-05 | | | |
| 0.3 | 2.5E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

**Table 5-17. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climates (Based on LADDs in Table 15 from the SHEDS-Wood Document)**

| | | | | | |
|---|---|---|---|---|---|
| **Playset Only** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 5.4E-05 | 2.0E-04 | | | |
| 99 | 2.4E-05 | 8.9E-05 | | | |
| 95 | 1.2E-05 | 4.5E-05 | | | |
| 90 | 8.0E-06 | 2.9E-05 | | | |
| 50 | 1.5E-06 | 5.4E-06 | | | |
| 10 | 2.9E-07 | 1.1E-06 | | | |
| 5 | 1.7E-07 | 6.2E-07 | | | |
| 1 | 8.0E-08 | 2.9E-07 | | | |
| minimum | 5.1E-09 | 1.9E-08 | | | |
| 99.2 | 2.6E-05 | 1.0E-04 | | | |
| 69.1 | 2.7E-06 | 1.0E-05 | | | |
| 9.0 | 2.7E-07 | 1.0E-06 | | | |
| | | | | | |
| **Playset and Deck** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 1.0E-04 | 3.8E-04 | | | |
| 99 | 4.4E-05 | 1.6E-04 | | | |
| 95 | 2.1E-05 | 7.8E-05 | | | |
| 90 | 1.4E-05 | 5.0E-05 | | | |
| 50 | 2.9E-06 | 1.1E-05 | | | |
| 10 | 6.2E-07 | 2.3E-06 | | | |
| 5 | 4.1E-07 | 1.5E-06 | | | |
| 1 | 2.0E-07 | 7.4E-07 | | | |
| minimum | 7.5E-08 | 2.7E-07 | | | |
| 96.8 | 2.7E-05 | 1.0E-04 | | | |
| 48.5 | 2.7E-06 | 1.0E-05 | | | |
| 2.4 | 2.7E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

Figure 5-8 presents an approximate cumulative probability density plot for risks from soil and residue exposure. The lines in this plot are not true cumulative probabilities because risks were only calculated at the quartiles, as shown by the large dots; the lines merely connect the dots. Two observations are clearly shown in this plot. First, residue exposures have greater risk than soil exposure; and second, the difference between playset only versus playset and deck residue risk is less than the difference between residue and soil risk. At the 50th percentile, residue risk for playset and deck exposure is slightly greater than $10^{-5}$ and approximately $10^{-4}$ at the 95$^{th}$ percentile. The $10^{-4}$ risk level is exceeded for playset only exposure approaching the 99$^{th}$ percentile. Figure 5-9 is a bar chart of the risks from three different levels of exposure: 50th, 95th and 99th percentiles. At each level, there are four bars: soil and residue exposure for playsets alone and soil and residue exposure for both decks and playsets. Residue risk for playset only exposure exceeds the soil risk by a factor of approximately 6-7 at the 50$^{th}$ percentile and 95$^{th}$ percentiles, and by a factor of approximately 10 at the 99$^{th}$ percentile. For playset and deck exposure, residue risk is approximately 10 times greater that the soil risk at all three cumulative percentiles. Soil exposure risk exceeds $10^{-5}$ at the 95$^{th}$ percentile for both categories of exposure. Residue risk for playsets only is slightly less than $10^{4}$ and slightly greater than $10^{-4}$ for playsets and decks at the 95th percentile.

## 5.3 Risk Reduction Assuming 0.01% Dermal Absorption Rate

SHEDS-Wood used 2-3% as the arsenic dermal absorption rate (recommended by the FIFRA SAP) based on Wester et al. (1993). Zartarian et al.(2003) acknowledged that this value may be closer to 1% due to physical removal processes prior to absorption through the skin surface. However based on a dermal absorption study for monkeys reported by Wester et al. (2003), OPP also modeled exposure in SHEDS-Wood based on a 0.01% dermal absorption, which is considerably lower than previously reported by Wester et al. (1993). The 0.01% dermal absorption from the preliminary results of Wester et al. (2003) was based on urinary arsenic data following application of arsenic in CCA residue that had been weathered by the environment. Values reported for soluble arsenic were much higher, 3.6, 0.55, and 4.1 percent. Calculating risks for a lower dermal absorption value provided an indication of the sensitivity of the model to this parameter as well as risks under a fundamentally different assumption.

For this evaluation, risks were calculated for both warm and cold climate conditions. Table 5-18 presents the mean, median, 95$^{th}$, and 99$^{th}$ percentiles for the two exposure scenarios of playsets and decks, and playsets only. At the median, the lowest risk level was $4.2 \times 10^{6}$ for playset only exposure under cold climate conditions; the highest was $1.7 \times 10^{-5}$ for playset and deck exposure under warm climate conditions. At the 95$^{th}$ percentile, all exposure scenarios exceeded the $10^{-5}$ risk level. Tables 5-19 and 5-20 present the probabilistic cancer risk distributions and risk levels for exposure to arsenic in warm and cold climates, respectively, based on the assumed dermal absorption rate of 0.01%.

**Figure 5-8 Comparison of Total Arsenic Risks from Playsets and Decks for Warm Climate Baseline**

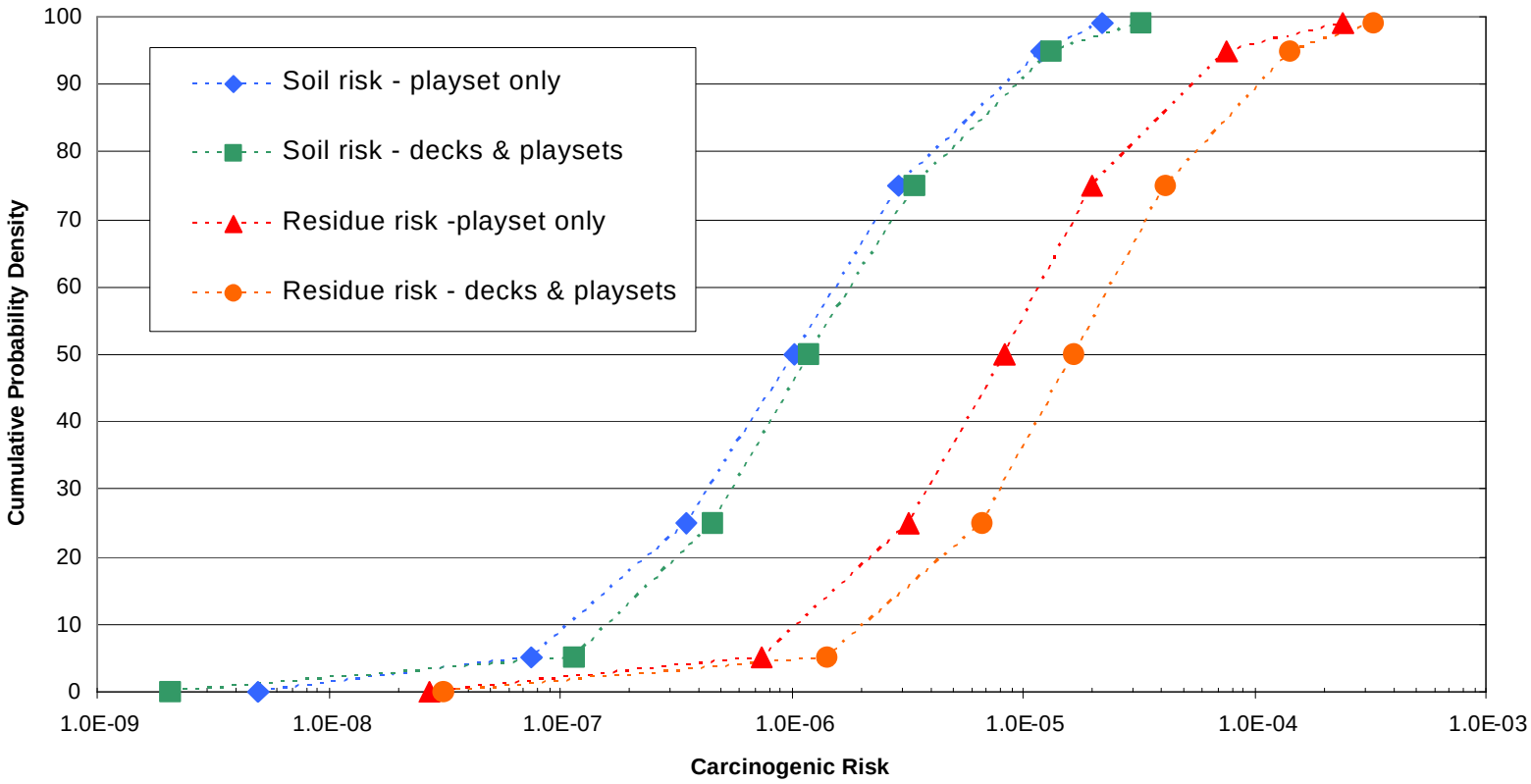


**Figure 5-9 Comparison of Residue and Soil Total Arsenic Risks for Warm Climate Baseline**

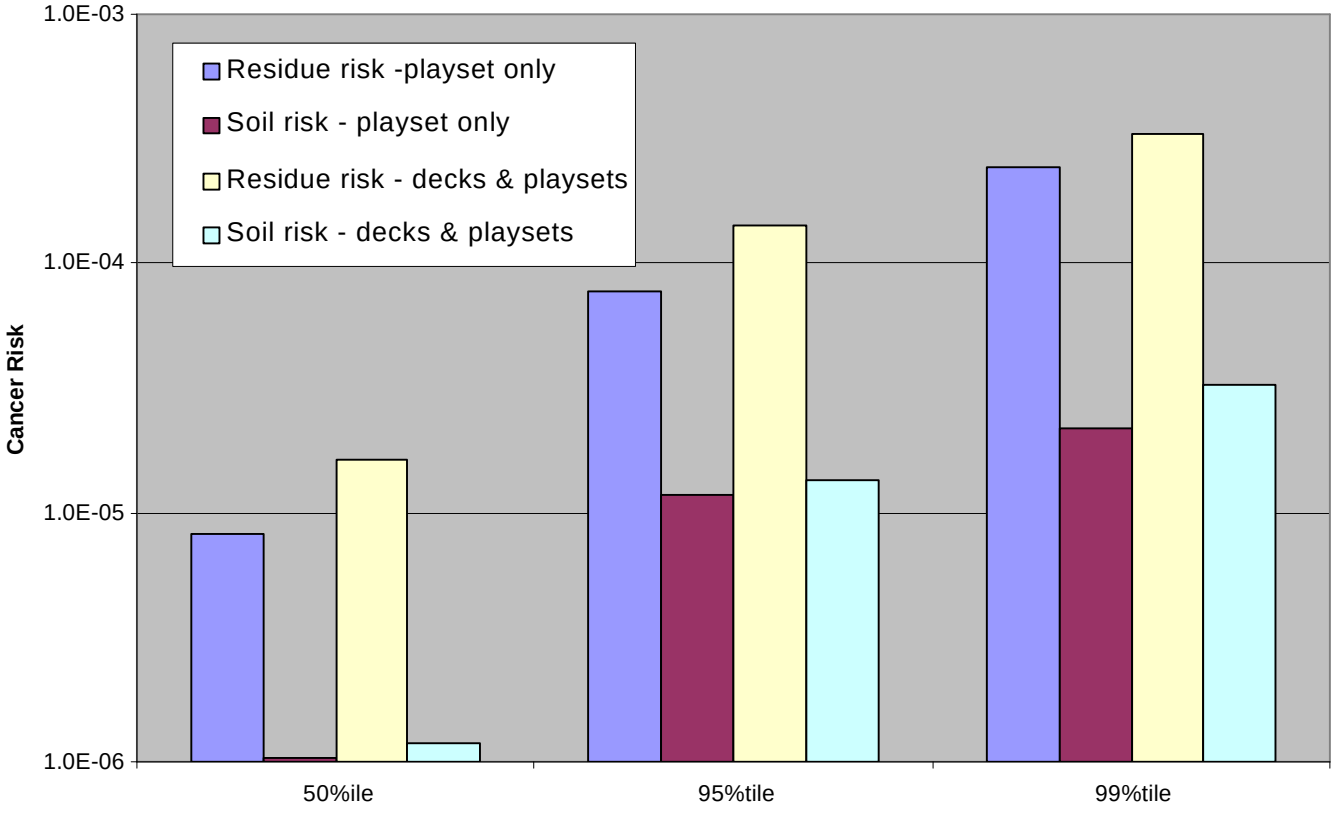


**Table 5-18. Arsenic Cancer Risk Assuming 0.01% Dermal Absorption**

| Scenario | Arsenic ($Q_1$*= 3.67 (mg/kg/day)$^{-1}$) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Warm | Cold | Warm | Cold | Warm | Cold | Warm | Cold |
| Playset and Deck | 2.9E-05 | 2.0E-05 | 1.7E-05 | 9.8E-06 | 9.8E-05 | 7.7E-05 | 2.3E-04 | 1.6E-04 |
| Playset Only | 1.5E-05 | 1.1E-05 | 7.3E-06 | 4.2E-06 | 4.4E-05 | 3.8E-05 | 1.2E-04 | 9.9E-05 |

**Table 5-19. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Warm Climates (Dermal Residue Absorption Rate = 0.01%) (Based on LADDs in Table 35 from the SHEDS-Wood Document)**

| | | | | | |
|---|---|---|---|---|---|
| **Playset Only** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 1.9E-04 | 7.1E-04 | | | |
| 99 | 3.3E-05 | 1.2E-04 | | | |
| 95 | 1.2E-05 | 4.4E-05 | | | |
| 90 | 7.8E-06 | 2.9E-05 | | | |
| 50 | 2.0E-06 | 7.3E-06 | | | |
| 10 | 4.3E-07 | 1.6E-06 | | | |
| 5 | 2.8E-07 | 1.0E-06 | | | |
| 1 | 6.1E-08 | 2.2E-07 | | | |
| minimum | 3.4E-09 | 1.2E-08 | | | |
| 98.2 | 2.4E-05 | 1.0E-04 | | | |
| 61.3 | 2.7E-06 | 1.0E-05 | | | |
| 4.7 | 2.6E-07 | 1.0E-06 | | | |
| | | | | | |
| **Playset and Deck** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 2.3E-04 | 8.6E-04 | | | |
| 99 | 6.4E-05 | 2.3E-04 | | | |
| 95 | 2.7E-05 | 9.8E-05 | | | |
| 90 | 1.6E-05 | 6.0E-05 | | | |
| 50 | 4.5E-06 | 1.7E-05 | | | |
| 10 | 9.1E-07 | 3.3E-06 | | | |
| 5 | 6.3E-07 | 2.3E-06 | | | |
| 1 | 2.8E-07 | 1.0E-06 | | | |
| minimum | 1.1E-07 | 4.1E-07 | | | |
| 95.1 | 2.7E-05 | 1.0E-04 | | | |
| 33.8 | 2.7E-06 | 1.0E-05 | | | |
| 0.9 | 2.5E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

**Table 5-20. Probabilistic Cancer Risk Distributions and Risk Levels for Children Exposed to Arsenic in Cold Climate (Dermal Residue Absorption Rate = 0.01%) (Based on LADDs in Table 36 From SHEDS-Wood Document)**

| | | | | | |
|---|---|---|---|---|---|
| **Playset Only** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 1.2E-04 | 4.3E-04 | | | |
| 99 | 2.7E-05 | 9.9E-05 | | | |
| 95 | 1.0E-05 | 3.8E-05 | | | |
| 90 | 6.2E-06 | 2.3E-05 | | | |
| 50 | 1.1E-06 | 4.2E-06 | | | |
| 10 | 2.3E-07 | 8.4E-07 | | | |
| 5 | 1.5E-07 | 5.6E-07 | | | |
| 1 | 5.4E-08 | 2.0E-07 | | | |
| minimum | 1.2E-08 | 4.4E-08 | | | |
| 99.1 | 2.7E-05 | 1.0E-04 | | | |
| 73.1 | 2.7E-06 | 1.0E-05 | | | |
| 12.6 | 2.7E-07 | 1.0E-06 | | | |
| | | | | | |
| **Playset and Deck** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Level | | |
| | | | **A = 1.0E-6** | **B = 1.0E-5** | **C = 1.0E-4** |
| maximum | 8.2E-05 | 3.0E-04 | | | |
| 99 | 4.3E-05 | 1.6E-04 | | | |
| 95 | 2.1E-05 | 7.7E-05 | | | |
| 90 | 1.2E-05 | 4.4E-05 | | | |
| 50 | 2.7E-06 | 9.8E-06 | | | |
| 10 | 6.0E-07 | 2.2E-06 | | | |
| 5 | 3.4E-07 | 1.3E-06 | | | |
| 1 | 1.6E-07 | 6.0E-07 | | | |
| minimum | 4.7E-08 | 1.7E-07 | | | |
| 96.6 | 2.7E-05 | 1.0E-04 | | | |
| 50.5 | 2.7E-06 | 1.0E-05 | | | |
| 3.5 | 2.7E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

For comparison of these risk levels to the baseline risks, Table 5-21 presents the median and 95th percentile risks for the baseline (2 to 4%) and Wester et al. (2003)(0.01%) dermal absorption values under warm climate conditions. Differences in risk levels between the two dermal assumptions for exposure to playsets and decks ranged from 26% at the median to 30% at the 95th percentile. For playsets only, the range was greater; 34% to 47%. Changing the dermal absorption factor by approximately two orders of magnitude had a much smaller effect on total risk because the dominant route of exposure was ingestion, not dermal uptake.

**Table 5-21. Comparison of Arsenic Risks Between Baseline and 0.01% Dermal Absorption Warm Climate**

| Dermal Absorption Assumption | Playset and Deck | | Playset Only | |
|---|---|---|---|---|
| | Median | 95th%ile | Median | 95th%ile |
| Baseline (2-4%) | 2.3E-05 | 1.4E-04 | 1.1E-05 | 8.3E-05 |
| Wester et al. (2003) (0.01%) | 1.7E-05 | 9.8E-05 | 7.3E-06 | 4.4E-05 |
| Difference | 26% | 30% | 34% | 47% |

Figure 5-10 presents the PDF and CDF distributions of the risks under warm climate conditions based on the assumed 0.01% dermal absorption. (The corresponding plots for cold climate conditions are in Figure 5-11.) Looking at the warm climate, the cumulative percentile at the lower end of EPA's risk level, $10^{-6}$, is approximately the 5th for playsets only exposure and less than the 1st for playsets and decks exposure (Table 5-19). For baseline warm conditions, the percentiles at this risk level are the 3rd and less than the 1st for playsets only, and playsets and decks exposure, respectively (Table 5-16). For dermal absorption at 0.01%, the upper end of the range, $10^{-4}$, is exceeded above the 98th percentile for playsets only and the 95th percentile for playsets and deck exposure (Table 5-19). Reducing the dermal absorption factor to 0.01% shifts the cumulative percentiles a small amount toward lower risks over baseline conditions.

Figure 5-10

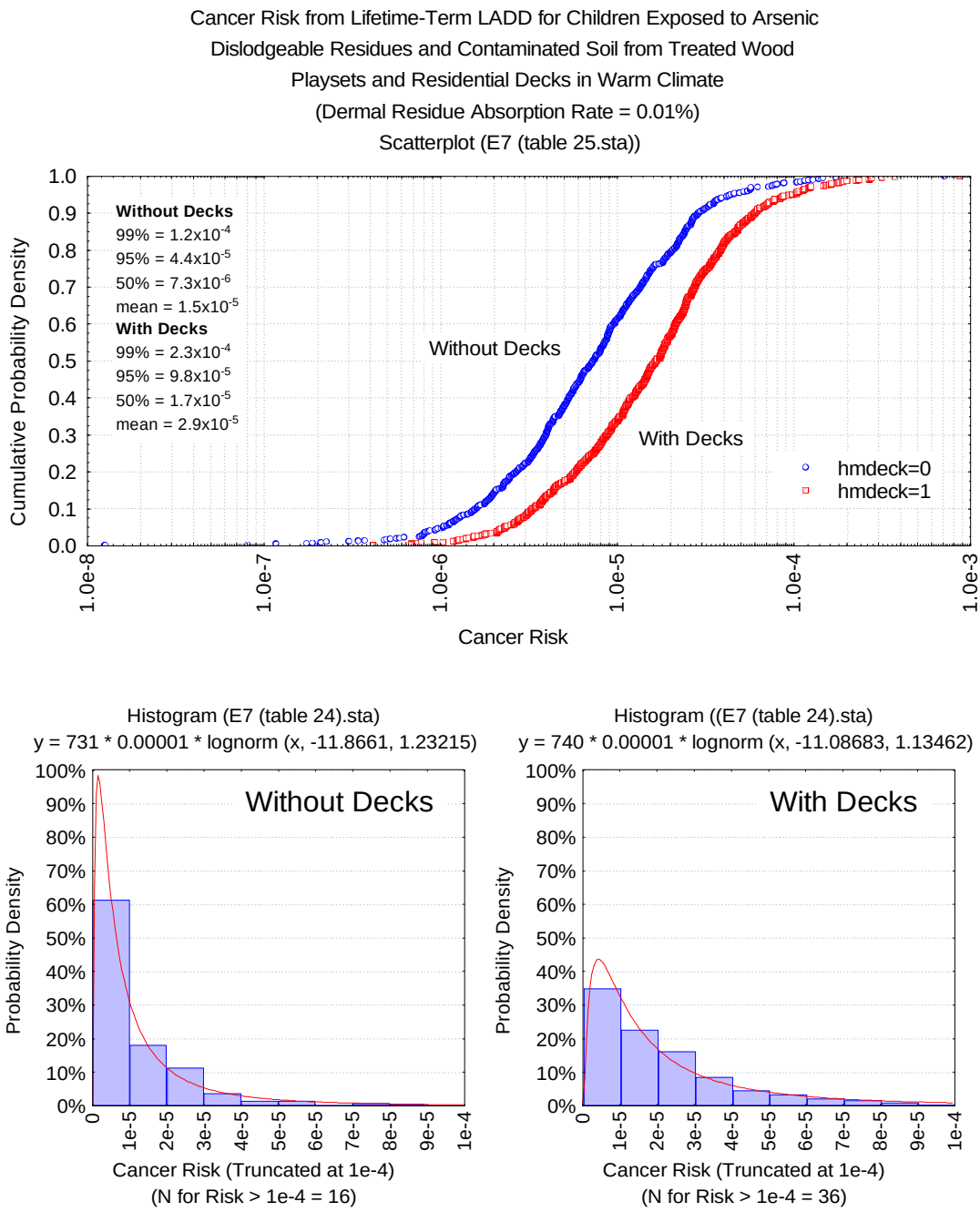
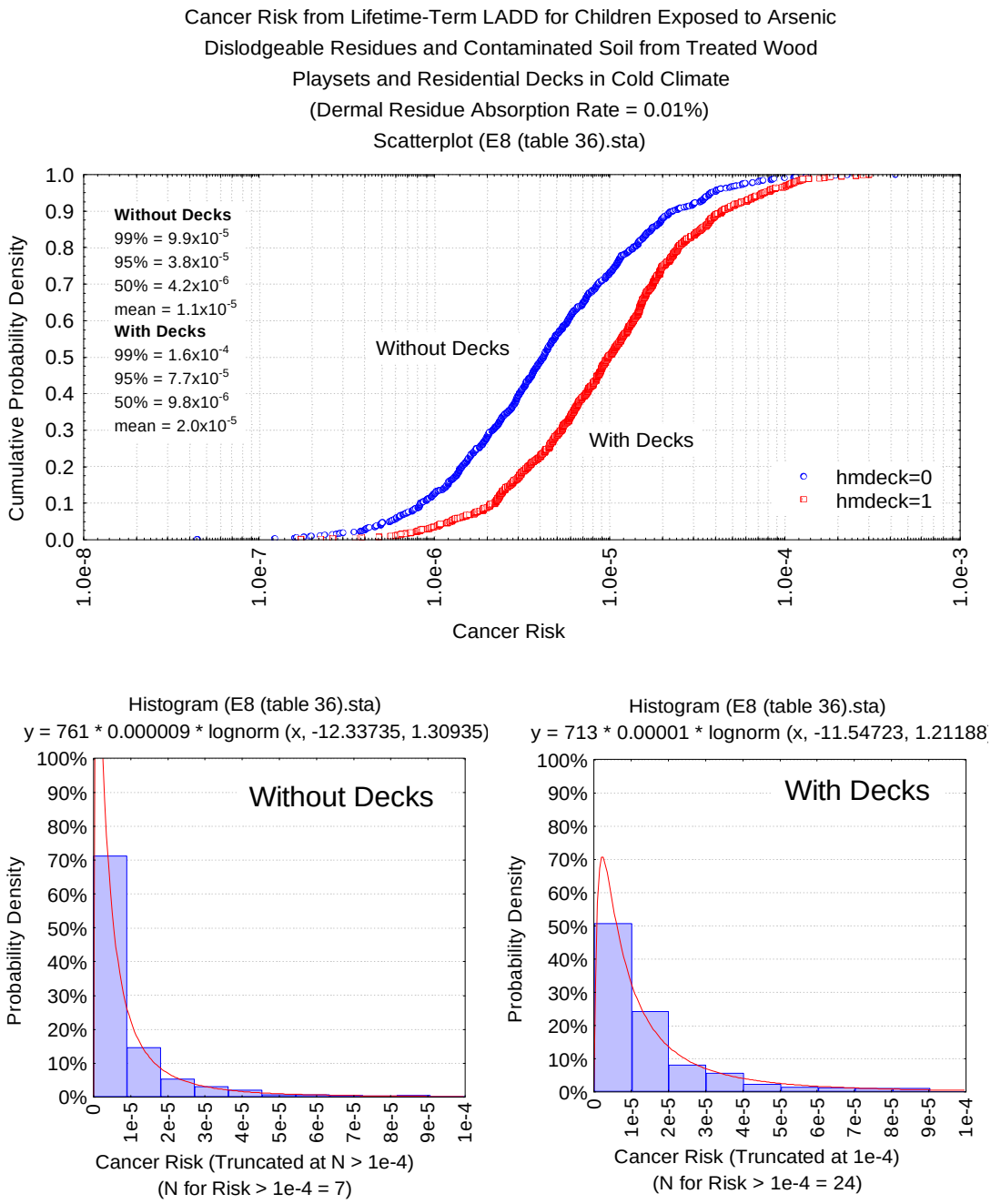

Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(Dermal Residue Absorption Rate = 0.01%)
Scatterplot (E7 (table 25.sta))
Without Decks
99% = 1.2x10-4
95% = 4.4x10-5
50% = 7.3x10-6
mean = 1.5x10-5
With Decks
99% = 2.3x10-4
95% = 9.8x10-5
50% = 1.7x10-5
mean = 2.9x10-5
Without Decks
With Decks
hmdeck=0
hmdeck=1
Cumulative Probability Density
Cancer Risk
Histogram (E7 (table 24).sta)
y = 731 * 0.00001 * lognorm (x, -11.8661, 1.23215)
Without Decks
Probability Density
Cancer Risk (Truncated at 1e-4)
(N for Risk > 1e-4 = 16)
Histogram ((E7 (table 24).sta)
y = 740 * 0.00001 * lognorm (x, -11.08683, 1.13462)
With Decks
Probability Density
Cancer Risk (Truncated at 1e-4)
(N for Risk > 1e-4 = 36)

Figure 5-11


Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Cold Climate
(Dermal Residue Absorption Rate = 0.01%)
Scatterplot (E8 (table 36).sta)
Cumulative Probability Density
Cancer Risk
Without Decks
99% = 9.9x10-5
95% = 3.8x10-5
50% = 4.2x10-6
mean = 1.1x10-5
With Decks
99% = 1.6x10-4
95% = 7.7x10-5
50% = 9.8x10-6
mean = 2.0x10-5
Without Decks
With Decks
hmdeck=0
hmdeck=1
Histogram (E8 (table 36).sta)
y = 761 * 0.000009 * lognorm (x, -12.33735, 1.30935)
Without Decks
Probability Density
Cancer Risk (Truncated at N > 1e-4)
(N for Risk > 1e-4 = 7)
Histogram (E8 (table 36).sta)
y = 713 * 0.00001 * lognorm (x, -11.54723, 1.21188
With Decks
Probability Density
Cancer Risk (Truncated at 1e-4)
(N for Risk > 1e-4 = 24)

## 5.4 Summary

The potentially exposed population for this assessment was assumed to be children (ages 1-6 years) in the United States who contact CCA-treated wood and/or CCA-containing soil from public playsets (e.g., at a playground, a school, a daycare center). A subset of these children was also assumed to contact CCA-treated wood residues and/or CCA-containing soil from residential playsets (i.e., at the child's own home or at another home) and/or residential decks (i.e., at the child's own home or another home). This population was selected because of the particular focus by CPSC and other groups on playground playsets in conjunction with EPA's focus on estimating the risk to children from various primary sources of CCA-treated wood (Zartarian et al., 2003).

Noncancer and cancer risks to children exposed to CCA-treated playsets and decks were calculated from doses generated using the SHEDS-Wood model. Noncancer risks were evaluated against OPP's guidance MOE values for arsenic and Cr(VI) for short- (1 day to 1 month) and intermediate-term (1 to 6 months) exposure duration. Lifetime (6 years of exposure averaged over 75 years) cancer risk from arsenic exposure was compared to risks ranging from $10^{-6}$ to $10^{-4}$. Noncancer risk for arsenic was above the guidance MOE of 30 for all exposure scenarios, up to the 99$^{th}$ percentile. Cr(VI) risks were above the guidance MOE of 100 for all doses. Cancer risk exceeded the upper bound of the risk range, $10^{-4}$, at cumulative percentiles ranging from the 90$^{th}$ for warm climate conditions and exposure to decks and playsets to the 99$^{th}$ for cold climate conditions and exposure to playsets only. Across all exposure scenarios, cancer risks were less than $10^{-6}$ at cumulative percentiles of the 9th and lower. Conversely, approximately 91% of the simulated exposures had risks exceeding $10^{-6}$.

A screening level analysis comparing the risks from soil exposure versus residue exposure was conducted. Residue risk was greater than soil risk for both categories of exposure. For playset and deck exposure, residue risks were approximately an order of magnitude greater; slightly lesser differences were seen for playset only exposure. At the 95$^{th}$ percentile, soil risks exceeded $10^{-5}$ for both categories of exposure and residue risks were slightly greater than $10^{-4}$ for playset and deck exposure.

Cancer risk was also evaluated using a lower dermal absorption factor for arsenic. When this factor was reduced by approximately two orders of magnitude, risks were reduced by up to 30% at the 95$^{th}$ percentile for exposure to playsets and decks. Lesser risk reduction was seen at the 50$^{th}$ percentile.

# 6.0 RISK REDUCTION IMPACTS

## 6.1 Introduction

The Agency describes risk reduction impacts in this chapter to aid the risk manager in identifying techniques to reduce risk, given the potential concerns based on cancer risk (see Chapter 5.0) from contact with CCA-treated wood. The effect of the mitigation strategies was only assessed for carcinogenic risks from arsenic, because the noncancer MOEs for arsenic and chromium were above OPP's guideline values. This Section presents background information regarding factors that may influence arsenic and chromium levels on or near CCA-treated wood. The impact of the various mitigation strategies considered are presented in Section 6.2.

There are many variables that can influence the amount of dislodgeable arsenic and chromium levels present in CCA-treated wood surfaces and in the soil residues from chromated arsenical wood products. These variables include:

- Level of retention of arsenic and chromium in the wood;
- Different types of CCA (e.g., CCA type A, B, or C);
- Different types of pressure treated wood (douglas fir, southern pine, western cedar, red oak, etc.);
- Variables in pressure treatment process (e.g., temperature and pH, air seasoning time, removal of water, oven drying, etc.) influence the retention of CCA in the wood;
- Use of a salt or an oxide formulation of CCA;
- Weather conditions (e.g, acid rain, wet wood can greatly increase the amount of dislodgeable residues);
- Different uses of wood (wood decks, construction or utility poles, marine timbers, fence posts, wood foundation lumber, plywood, and wood for playground structures, etc.);
- Age of the wood;
- Physical condition of the wood (e.g., sanding, surface dirt, or sawing); and
- Application of sealant (with oil-based or water-based).

Some of these variables, such as the pressure treatment, level of retention, the type of wood, age of wood, weather conditions, and formulation of CCA, are variables that are not controlled by the typical homeowner. However, there may be things that a homeowner can do to control or reduce the risks from CCA-treated wood in playsets and decks. The Introduction and Background Chapter (Chapter 2.0) summarized some of the state regulatory agency recommendations for homeowners to reduce risks to CCA-treated wood. These recommendations include:

- Sealing CCA-treated structures (decks and playsets) every two years with oil-based stain;
- Preventing exposure to pressure-treated wood and dust;
- Washing hands after playing on wooden playground equipment;
- Inspecting structures for decay;
- Using alternatives to CCA-treated pressure treated wood;
- Not placing food, drink or paper products on pressure treated wood;
- Never burning treated wood;
- Limiting use of under deck areas where arsenic may have accumulated in soil;
- Not using treated wood on indoor surfaces; and
- Not using CCA-treated wood for wood chips or mulch.

In addition, to providing recommendations to homeowners on how to reduce risks to CCA-treated wood, some states also have codified laws designed to reduce exposure and eliminate some types of CCA-treated wood. States such as California, Florida, Maine, Minnesota, and New York either have laws or are introducing legislation that eliminate the supply of certain types of CCA-treated wood or require the application of sealants to playground/recreation equipment made with CCA-treated wood (See Chapter 2.0).

Because of the potential concerns based on arsenic cancer risk (see Chapter 5.0) from contact with CCA-treated wood, the Agency is considering risk reduction impacts in this chapter to aid risk managers in identifying techniques to reduce risk. Obviously many of the recommendations to reduce arsenic are based on the activities of the homeowner that are not readily quantified. In 2001, the FIFRA SAP identified the need for information on the performance and efficacy of different types and brands of coatings (U.S. EPA, 1991c). EPA completed the protocol for ongoing research on the effectiveness of sealants on weathered CCA-treated wood. For the purposes of this risk assessment, assumptions were made with regard to the effectiveness of sealants. These assumptions were based on data such as Stilwell (1998) who reported over a 95% reduction for oil-based alkyl resins for samples tested one year after a sealant was applied. CDHS (1987) reported 96%, and 82% reduction from stained treated wood surfaces after one month and 2 years, respectively. Based on these two existing data sources, OPP assumed a 90% reduction in residues for a moderate reduction scenario, and a 99.5% reduction in residues for a maximum reduction scenario. These reductions in residue concentration were assumed to be constant over time. SHEDS-Wood estimated exposures based on residue reduction resulting from the use of sealants and/or hand washing. The risks associated with these reduced exposures are presented in Section 6.2.

### 6.1.1 Application of Sealant and Hand Washing Information to SHEDS-Wood

The FIFRA SAP recommended that “EPA inform the public of the ability of certain coatings to substantially reduce leachable and dislodgeable CCA chemicals and thus reduce

potential exposures to arsenic and chromium." While the Panel made recommendations regarding the need for additional studies in this area, it felt that the current evidence was sufficient to begin advising the public about the use of coatings. The Panel made the following observations.

- The weight-of-evidence from available studies indicated that certain coatings can substantially reduce dislodgeable and leachable CCA chemicals.
- Reductions of 70 to 95% or greater in dislodgeable arsenic were seen in all studies that subjected CCA wood to natural weathering. (U.S. EPA, 2001c).

Table 6-1 presents the results of the SAP's analysis of the available sealant studies. The Panel indicated that "there is no evidence that water repellents added directly to the treatment solution are effective in reducing leachable/dislodgeable CCA chemicals; current data are not adequate for identifying a particular coating as being clearly superior or inferior to reducing leachable/dislodgeable CCA chemicals. However, confidence is highest for polyurethane, as this coating was shown to result in substantial (70 to >95%) reduction in dislodgeable arsenic in a well controlled field study (a "real-world" application allowing for effects of use, and a short-term controlled laboratory study)" (U.S. EPA, 2001c).

Based on the results of the FIFRA SAP, EPA assumed a moderate reduction (90% reduction in residue concentrations with sealant for warm climates only) and a maximum feasible reduction (99.5% reduction in residue concentration with sealant for warm climates only) and incorporated this into the SHEDs-Wood model.

Hand washing was modeled in SHEDS-Wood. The SHEDS-Wood report (see Zartarian et al., 2003) specifically described how hand washing was estimated (Zartarian et al., 2003). Below is an excerpt from the text that describes the SHEDs-Wood modeling:

> "SHEDS–Wood is essentially a mass balance model that involves simulating the movement and fate of the pollutant of interest after it has come into contact with the exposed individual. The SHEDS-Wood model follows simulated individuals through time, keeping track of the additions and subtractions to the *cumulative exposure loading.* An 'exposure' is a new contact with the target chemical; hence 'exposure' can only occur at places where the chemical is present (in this case, decks or playsets). Once exposure occurs, the chemical remains present on or in an individual until it is removed. The cumulative exposure loading is the total amount of the chemical currently in contact with the person; this can be non-zero even when away from decks and playsets. It is analogous to a bank balance, with new exposures corresponding to deposits and removal processes to withdrawals. In the equations below, a distinction is made between the amount of new exposure E from a single macroactivity event, and the current loading or cumulative exposure CE . The size of CE cannot be determined solely from the record of exposures, but depends on the frequency and size of the removal terms (the withdrawals in the bank account analogy).

## Table 6-1. Summary of Sealant Studies

| Study | Design | Weathering | Sampling | Treatments | Results | Comments |
|---|---|---|---|---|---|---|
| Stilwell, 1998 (CT) | Purchased boards, placed outside, 4 coatings, 4 replicates, 5 time points out to 1 year | Outside, natural weathering, no human use | Standardized wipe method. Repeat rubbing of same surface under controlled pressure | Polyurethane, acrylic latex, Spar varnish, Oil-based stain. Brush applied, 2 coats. | > 95% reduction for polyurethane, acrylic resin, and varnish at all time points as compared to pretreatment. 80-97% reduction for oil stain. | Does not account for wear. Lacks temporal control. Aesthetic problems after 1 yr for spar varnish. |
| California DHS, 1987 (CA) | Fishing pier, 1 coating, 4 replicates, 2 time points out to 2 years | Outside, natural weathering, in use | Gauze wipe, 100 $cm^2$, with repeat rubbing to same surface | Polyurethane, no information on application methods | > 95% reduction at 2 years as compared to pretreatment levels. | Considers wear. Lacks temporal control. Limited sample sizes and coatings. |
| California DHS, 1987 (CA) | Single playground, 1 coating, ? replicates, 3 time points, out to 2 years | Outside, natural weathering, in use | Gauze wipe, 100 $cm^2$, with repeat rubbing of same surface | Oil-based stain, no information on application methods. | > 95% reduction at 6 months as compared to pretreatment levels. 70% reduction at 2 years. | Considers wear. Lacks temporal control. Limited sample sizes and coatings. |
| SCS, 1998 (lab) as cited in U.S. EPA 2001c | Purchased boards, used in laboratory, 3 coatings, 5 replicates, 1 time point apparently soon after coating applied. | Inside, no weathering, not subject to human use. | Kimwipes,100 $cm^2$, damp. Hand wipes, 500 $cm^2$, repeat rubbing of same surface | 3M sealant, Superdec stain, no information on application methods, Osmose water repellant | 60% - 80% reduction for 3M sealant as compared to pretreatment. No reduction for stain or water repellent. | Variable within type of coating. Does not account for wear. Not subject to natural aging and weathering. Short-term evaluation. |
| Cooper et al., 1997 (New Brunswick, CAN) | Laboratory prepared wood, fence & deck structures, placed outside, 1 coating, ? replicates, 2 time points, 4 mo, 2 yrs | Laboratory simulated aging, plus outside, natural weathering, no human use. | Collection of natural rain water contacting wood surface | Thompson's Water Seal (fence only) & Water Repellent in CCA treatment soln (fence & deck). | 70% reduction at 4 months and 80% reduction at 2 years for Thompson's. No reduction for water repellent added into treatment solution. | Does not account for wear. Includes temporal control. |
| Riedel et al., 1991 (Ontario, CAN) | 10 playgrounds, 2 to 10 years old. Some stained/painted, others not. 4 sampling points per structure. | Outside, natural weathering, in use | Gauze wipe, 250 or 500 $cm^2$ with repeat rubbing of same surface | Oil based stain on some though not all structures. | 4 structures treated with stain had on average 74% lower levels of dislodgeable As than average of 3 structures without any coating.[a] | Cross-sectional study with no site specific controls. Limited information on past application of coatings. Sampling locations vary across sites. |
| CPSC, 1990 (lab) | Purchased boards, used in laboratory, 2 treatments, 3 replicates, 2 wood types. | Inside, no weathering, not in use, no aging | Nylon cloth wipe, 400 $cm^2$ | Oil-based stain, water repellant, applied per manufacturer's label. | No clear evidence of reductions. | Considerable variability in the controls, short-term study with no weathering. |
| Lebow and Evans, 1990 as cited in U.S. EPA 2001c | Laboratory prepared wood 1 treatment, ? replicates, 1 time point at 17 weeks. | Laboratory simulated rainfall for 17 weeks. | Collection of natural rain water contacting wood surface | Water soluble acrylic polymer applied pre-CCA treatment. | 25-30% reduction in total As leached in artificial rainfall. | Coating applied pre-CCA treatment, so of limited relevance to post-treatment coatings. |

Source: U.S. EPA, 2001c (pp. 53-54); see Appendix F for text and references.

New dermal exposure, once contacted, remains on the skin until removed by one of a competing set of processes. These include washing and bathing, hand-to-mouth transfer, physical removal when load limits are exceeded, and dermal absorption. Because these processes compete, an increase in the frequency of hand washing will produce deceases in the amounts that can be removed by the other processes. Thus, the impact of washing on the absorbed dose can be estimated directly. For ingested (GI tract) exposures, the removal processes are gastrointestinal absorption and daily voiding of the GI tract.

The basis for the changes to the cumulative exposure loading is the macroactivity *time step*, or *event*. Within a single time step, the SHEDS-Wood model does not model processes as continuous changes in time, but instead treats them as a sequence of instantaneous adjustments or changes to the cumulative loading, one for each process under consideration. The order of the adjustments is: first, new exposure contact (if any) is added; second, the cumulative loadings are compared to the maximum dermal loading limits and reduced if necessary; third, the effects of absorption are determined; fourth, hand-to-mouth transfer occurs; fifth, hand washing (if present); and sixth, bathing (if present). The presence and the size of each adjustment is based on the activity events reported in the human activity diaries taken from the CHAD database and on the settings of several of the input variables" (Zartarian et al., 2003).

OPP was able to use the exposures of SHEDS-Wood to evaluate the effect of hand washing on cancer risks.

## 6.2 Risk Characterization for Mitigation Measures

Many of the methods to reduce arsenic exposure are based on the activities of the homeowner that are not readily quantified. SHEDS-Wood simulated reduced exposure doses resulting from two mitigation measures: the use of sealants and/or hand washing. These mitigation measures only apply to residue exposure and not to soil exposure. Although soil exposure may potentially be reduced through removal of contaminated soil, this was not modeled in SHEDS-Wood. The use of sealants may also reduce the rate of transfer of CCA to the adjacent soil over time, however, there are no available studies documenting this effect. The approach taken in SHEDS-Wood was conservative: exposures were most likely overestimated because soil concentrations were held constant while wood residue concentrations were reduced. Another issue is how SHEDS-Wood treated soil exposure. Residue exposures were assumed to be transferred to the gastrointestinal tract via hand-to-mouth behavior, but this was not the case for soils. Instead, soil ingestion was treated as a single term that included both direct eating of soil as well as soil-hand-mouth transfers.

Increased hand washing, as a mitigation strategy, was also evaluated. The way in which SHEDS-Wood handled hand washing was by only reducing residue exposure and not soil exposure. One supposes that hand washing could also provide some reduction in the soil-hand-mouth pathway, but this was not explicitly modeled in SHEDS-Wood.

Two types of mitigation measures that were evaluated: reducing exposure through the use of sealants and through hand washing. Table 6-2 summarizes the various mitigation measures considered.

**Table 6-2. Arsenic Mitigation Measures Evaluated**

| Title | Description |
|---|---|
| 1. Sealant- moderate reduction | Assumed sealant afforded a 90% reduction in residue concentration. |
| 2. Sealant – maximum reduction | Assumed sealant afforded a 99.5% reduction in residue concentration. |
| 3. Hand washing | Increased frequency of hand washing. |
| 4. Sealant-moderate + hand washing | 90% reduction in residue concentration with increased frequency of hand washing. |
| 5. Sealant-maximum + hand washing | 99.5% reduction in residue with increased frequency of hand washing. |

Risks based on mitigation strategies are presented in a similar format as in Chapter 5. First, for each mitigation measure considered, results are shown at four levels of exposure corresponding to the mean, median, 95$^{th}$ percentile and 99$^{th}$ percentile. Second, the CDF/PDFs are plotted. Lastly, a comparison of risks due to soil exposure versus residue exposure is presented.

### 6.2.1 Summary of Results

Results for the various mitigation scenarios are summarized in Table 6-3. Mitigation measures were only evaluated for the warm climate because this condition had the highest levels of risk. The table is shown in two parts: the top portion is for risk from exposure to playsets only and the lower portion is for risk from exposure to playsets and decks. Carcinogenic risks from arsenic are compared to the three levels of risk $10^{-6}$, $10^{-5}$, and $10^{-4}$ in Table 6-3. Values reported in the table are the cumulative probabilities above which the respective risk level is exceeded. Note that when exposure includes both playsets and decks (i.e., greater exposure), the cumulative percentile exceeding the risk level decreased at all levels except at $10^{4}$. The $10^{-4}$ risk level was at the extreme tail of the distributions under all mitigation conditions. It was slightly less extreme for hand washing only; for that mitigation scenario, the $10^{-4}$ risk level fell at the 95$^{th}$ percentile.

**Table 6-3. Summary of Risks Assuming Different Arsenic Mitigation Measures for Warm Climate Conditions**

| Mitigation Measure | Cumulative Percentiles at Specified Risk Levels | | |
|---|---|---|---|
| | Risk Level of $10^{-6}$ | Risk Level of $10^{-5}$ | Risk Level of $10^{-4}$ |
| **Playset Only** | | | |
| 1. Sealant- moderate reduction | 27$^{th}$ | 94$^{th}$ | >99$^{th}$ |
| 2. Sealant – maximum reduction | 57$^{th}$ | 97$^{th}$ | >99$^{th}$ |
| 3. Hand washing | 5$^{th}$ | 59$^{th}$ | >99$^{th}$ |
| 4. Sealant-moderate + hand washing | 28$^{th}$ | 92$^{nd}$ | >99$^{th}$ |
| 5. Sealant-maximum + hand washing | 58$^{th}$ | 96$^{th}$ | >99$^{th}$ |
| 6. Baseline | 3$^{rd}$ | 47$^{th}$ | 97$^{th}$ |
| **Playset and Deck** | | | |
| 1. Sealant-moderate reduction | 10$^{th}$ | 80$^{th}$ | >99$^{th}$ |
| 2. Sealant-maximum reduction | 42$^{nd}$ | 94$^{th}$ | >99$^{th}$ |
| 3. Hand washing | <1$^{st}$ | 28$^{th}$ | 95$^{th}$ |
| 4. Sealant-moderate + hand washing | 10$^{th}$ | 84$^{th}$ | >99$^{th}$ |
| 5. Sealant-maximum + hand washing | 44$^{th}$ | 93$^{rd}$ | >99$^{th}$ |
| 6. Baseline | <1$^{st}$ | 23$^{rd}$ | 90$^{th}$ |

Note: The baseline scenario includes a certain amount of hand washing. Hand washing, as a mitigation scenario, increases the frequency of this activity over baseline. See Appendix G for more information on hand washing.

The evaluation of risk remaining after the simulated mitigation measure was considered in reference to the baseline risk (i.e., no mitigation) discussed in Chapter 5. The following discussion focuses on the $10^{-6}$ risk level for comparison among the mitigation measures to baseline. For reference, the baseline percentiles at the $10^{-6}$ level (see Table 5-1) for warm climate conditions were the 3rd for playset only exposure and less than the 1st for exposure to playsets and decks. Several observations were made regarding the risks remaining following simulated mitigation measures. First, as would be expected, the greatest risk reduction occurred with the sealant under maximum residue reduction (99.5%). For example, for playset only exposure, the $10^{-6}$ risk level occurred at the 57th percentile with this mitigation as compared to the 3rd percentile for the baseline condition. For exposure to decks and playsets, the comparable percentile was the 42th. Second, hand washing provided the least reduction of risk; for playsets alone, the $10^{-6}$ risk level occurred at the 5th percentile and less than the 1st percentile for decks and playset exposure. Third, the simulation of combining sealant mitigation with hand washing had minimal impacts on the percentiles for the $10^{-6}$ risk level.[1]

### 6.2.2 Risk Reduction Through the Use of Sealants

Cancer risks for arsenic for children in warm climates were assessed for a moderate reduction in residue concentration (90% reduction in residue concentration) and a maximum reduction in residue concentration (99.5% reduction in residue concentration) to simulate the use of sealants. Table 6-4 presents the risks under the assumption of moderate and maximum reduction of exposure for the mean, median, and 95th percentile, and 99th percentile. For the maximum reduction simulation, risks were found to be within EPA's risk range for the four exposure levels shown here. Comparing risks from exposure to playsets and decks to exposure to playsets only, shows that the playset only risks were approximately 2 times less than the combined exposure. For the moderate reduction simulation, risks were still found to be within EPA's risk range for the four exposure levels shown here. Risk from exposure to playsets and decks were again approximately 2 times greater than risks from exposure to playsets alone. For comparison, baseline risks at the 95th percentile were $1.4 \times 10^{-4}$ for playset and deck exposure and $8.3 \times 10^{-5}$ for playsets only. At the 95th percentile, under the maximum reduction simulation, risks were reduced by approximately an order of magnitude over baseline.

[1] This may be due to the way SHEDS-Wood simulated the effect of hand washing, as noted in Section 6.2.

**Table 6-4. Cancer Risks Remaining Following Simulated Reduction in Residues from the Use of Sealants (Warm Climate only)**

| Scenario | Arsenic ($Q_1$*= 3.67 (mg/kg/day)$^{-1}$) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Mean | | Median | | 95%ile | | 99%ile | |
| | Moderate Reduction | Maximum Reduction | Moderate Reduction | Maximum Reduction | Moderate Reduction | Maximum Reduction | Moderate Reduction | Maximum Reduction |
| Playsets and Decks | 6.6E-06 | 2.9E-06 | 3.9E-06 | 1.3E-06 | 2.1E-05 | 1.1E-05 | 3.7E-05 | 2.9E-05 |
| Playsets only | 3.3E-06 | 2.0E-06 | 2.0E-06 | 8.1E-07 | 1.1E-05 | 8.2E-06 | 2.1E-05 | 1.7E-05 |

The cumulative probability density and probability density plots for moderate reduction and maximum reduction are shown in Figures 6-1 and 6-2, respectively. The probabilistic arsenic cancer risk distributions for moderate and maximum reduction are provided in Tables 6-5 and 6-6, respectively. Risks for both sources of exposure, playset only (without decks) and playset and deck (with decks) are plotted on the cumulative density plot. Under moderate reduction (Figure 6-1), the lower limit of EPA's risk range fell in the lower percentile range; at the 27$^{th}$ percentile for playset only exposure and at the 10$^{th}$ percentile for the combined exposure. For comparison, the corresponding percentiles under the baseline conditions were the 3$^{rd}$ for playset only exposure and less than the 1$^{st}$ for exposure to playsets and decks. At the maximum assumed reduction (Figure 6-2), the curve shifted to the left, so that the $10^{-6}$ risk occurred near the 50$^{th}$ percentile for both sources of exposure. For playsets only, it was slightly higher (57$^{th}$ percentile) and for playsets and decks it was slightly lower (42$^{nd}$ percentile). Risks exceeded the risk $10^{-4}$ range only for the moderate reduction assumption above the 99$^{th}$ percentile; under the maximum assumed reduction, risks did not exceed the risk range.

Figure 6-1

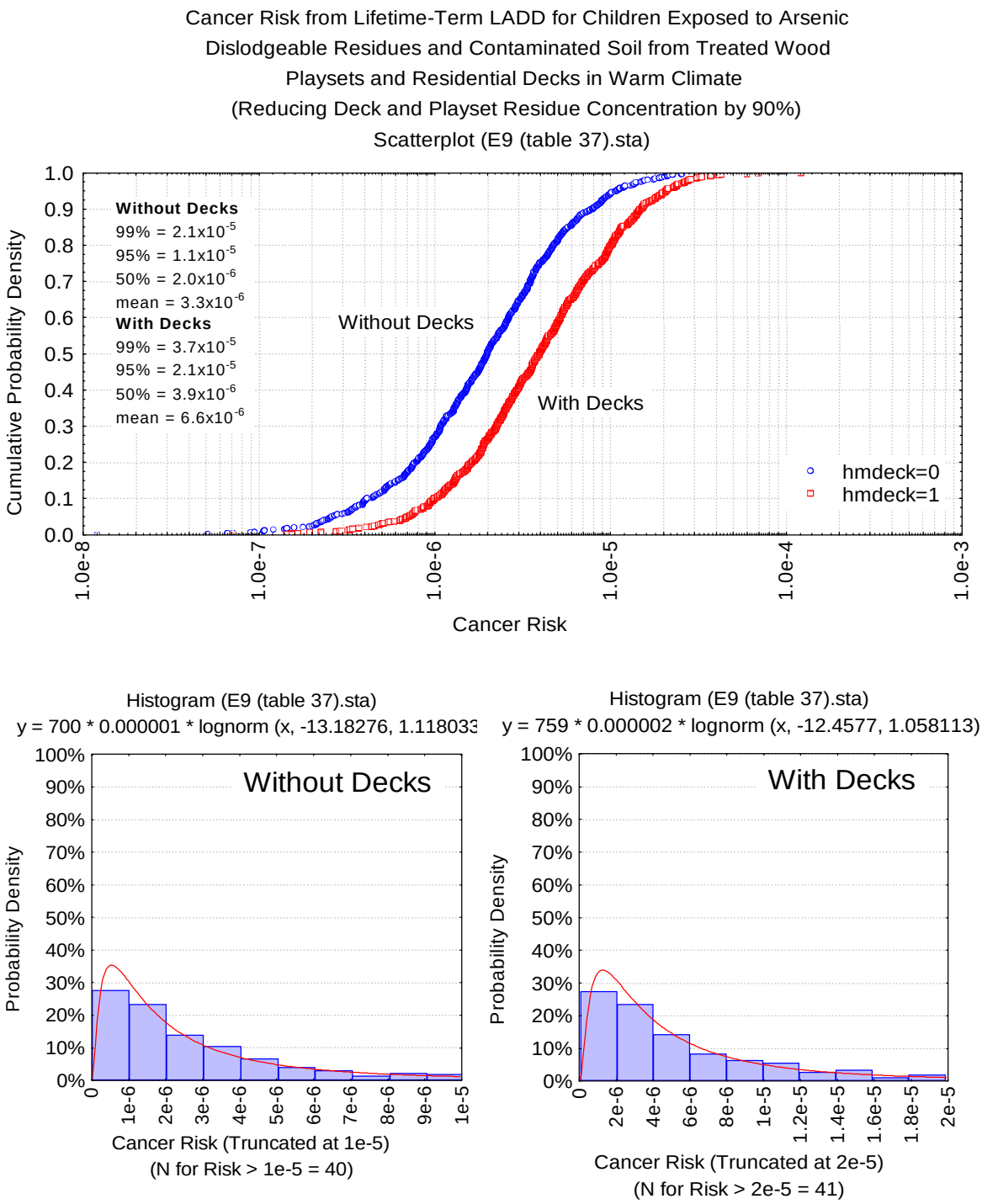

Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(Reducing Deck and Playset Residue Concentration by 90%)
Scatterplot (E9 (table 37).sta)
Without Decks
99% = 2.1x10-5
95% = 1.1x10-5
50% = 2.0x10-6
mean = 3.3x10-6
With Decks
99% = 3.7x10-5
95% = 2.1x10-5
50% = 3.9x10-6
mean = 6.6x10-6
Without Decks
With Decks
hmdeck=0
hmdeck=1
Cumulative Probability Density
Cancer Risk
Histogram (E9 (table 37).sta)
y = 700 * 0.000001 * lognorm (x, -13.18276, 1.118033
Without Decks
Probability Density
Cancer Risk (Truncated at 1e-5)
(N for Risk > 1e-5 = 40)
Histogram (E9 (table 37).sta)
y = 759 * 0.000002 * lognorm (x, -12.4577, 1.058113)
With Decks
Probability Density
Cancer Risk (Truncated at 2e-5)
(N for Risk > 2e-5 = 41)

Figure 6-2

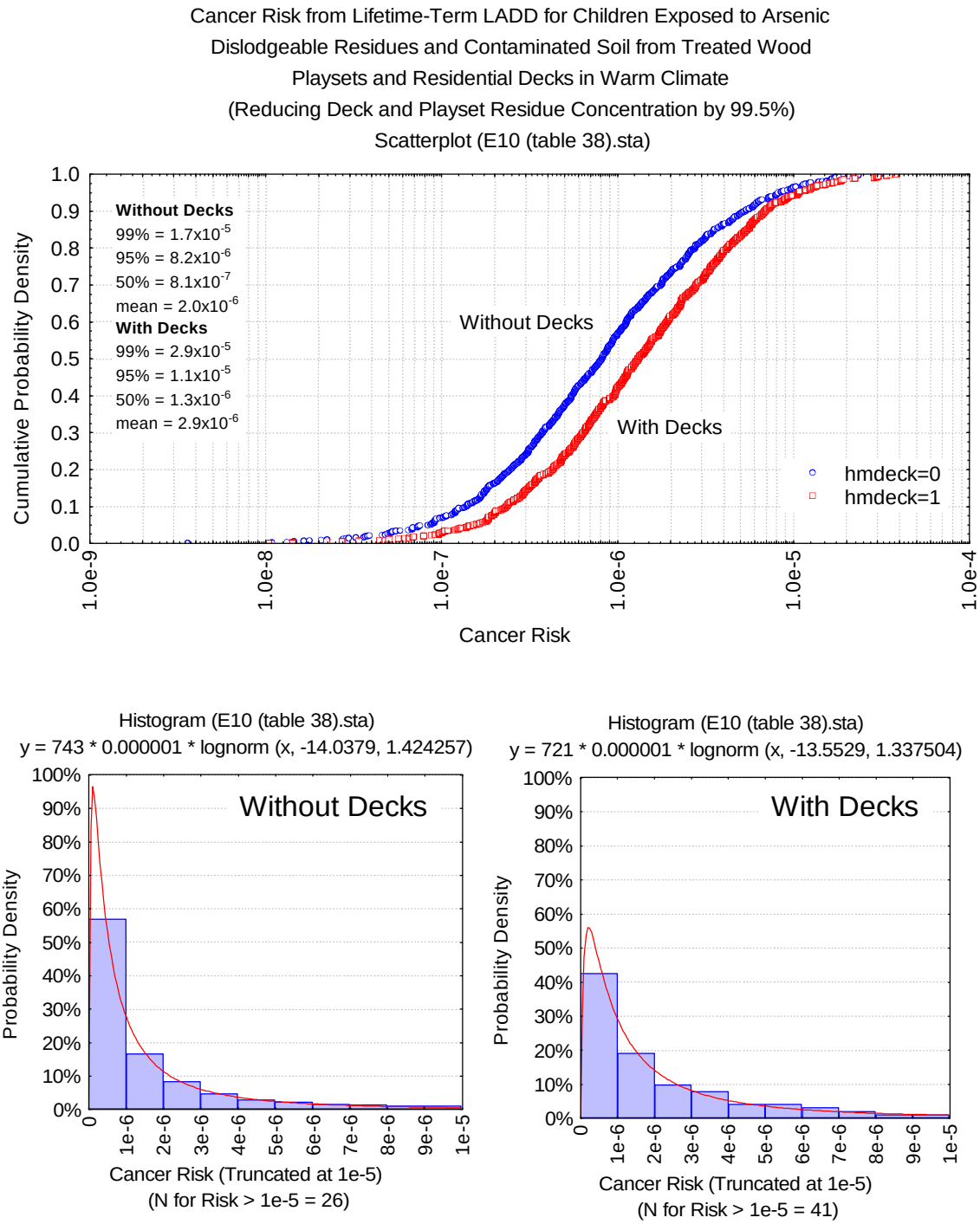

Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(Reducing Deck and Playset Residue Concentration by 99.5%)
Scatterplot (E10 (table 38).sta)
Without Decks
99% = 1.7x10-5
95% = 8.2x10-6
50% = 8.1x10-7
mean = 2.0x10-6
With Decks
99% = 2.9x10-5
95% = 1.1x10-5
50% = 1.3x10-6
mean = 2.9x10-6
Without Decks
With Decks
hmdeck=0
hmdeck=1
Cumulative Probability Density
Cancer Risk
Histogram (E10 (table 38).sta)
y = 743 * 0.000001 * lognorm (x, -14.0379, 1.424257)
Without Decks
Probability Density
Cancer Risk (Truncated at 1e-5)
(N for Risk > 1e-5 = 26)
Histogram (E10 (table 38).sta)
y = 721 * 0.000001 * lognorm (x, -13.5529, 1.337504)
With Decks
Probability Density
Cancer Risk (Truncated at 1e-5)
(N for Risk > 1e-5 = 41)

**Table 6-5. Probabilistic Arsenic Cancer Risk Distributions and Risk Ranges for Children in Warm Climates (reducing deck and playset residue concentration by 90%) (Based on the LADDs in Table 37 from the SHEDS-Wood document)**

**Playset Only**

| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Range | | |
|---|---|---|---|---|---|
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 1.2E-05 | 4.2E-05 | | | |
| 99 | 5.7E-06 | 2.1E-05 | | | |
| 95 | 3.0E-06 | 1.1E-05 | | | |
| 90 | 2.2E-06 | 7.9E-06 | | | |
| 50 | 5.4E-07 | 2.0E-06 | | | |
| 10 | 1.1E-07 | 4.1E-07 | | | |
| 5 | 7.1E-08 | 2.6E-07 | | | |
| 1 | 2.9E-08 | 1.1E-07 | | | |
| minimum | 3.3E-09 | 1.2E-08 | | | |
| >99.9 | 1.2E-05 | 1.0E-04 | | | |
| 94.3 | 2.7E-06 | 1.0E-05 | | | |
| 26.9 | 2.7E-07 | 1.0E-06 | | | |

**Playset and Deck**

| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Range | | |
|---|---|---|---|---|---|
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 3.3E-05 | 1.2E-04 | | | |
| 99 | 1.0E-05 | 3.7E-05 | | | |
| 95 | 5.6E-06 | 2.1E-05 | | | |
| 90 | 4.1E-06 | 1.5E-05 | | | |
| 50 | 1.1E-06 | 3.9E-06 | | | |
| 10 | 2.7E-07 | 9.9E-07 | | | |
| 5 | 1.9E-07 | 7.1E-07 | | | |
| 1 | 7.9E-08 | 2.9E-07 | | | |
| minimum | 1.9E-08 | 7.1E-08 | | | |
| 99.9 | 1.9E-05 | 1.0E-04 | | | |
| 80.0 | 2.7E-06 | 1.0E-05 | | | |
| 10.0 | 2.7E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

**Table 6-6. Probabilistic Arsenic Cancer Risk Distributions and Risk Ranges For Children in Warm Climates (Reducing Deck and Playset Residue Concentration by 99.5%)(Based on LADDs in Table 38 from the SHEDS-Wood document)**

| **Playset Only** | | | | | |
|---|---|---|---|---|---|
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Range | | |
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 9.0E-06 | 3.3E-05 | | | |
| 99 | 4.6E-06 | 1.7E-05 | | | |
| 95 | 2.2E-06 | 8.2E-06 | | | |
| 90 | 1.4E-06 | 5.3E-06 | | | |
| 50 | 2.2E-07 | 8.1E-07 | | | |
| 10 | 3.8E-08 | 1.4E-07 | | | |
| 5 | 2.4E-08 | 8.6E-08 | | | |
| 1 | 6.1E-09 | 2.2E-08 | | | |
| minimum | 9.8E-10 | 3.6E-09 | | | |
| >99.9 | 9.0E-06 | 1.0E-04 | | | |
| 96.5 | 2.7E-06 | 1.0E-05 | | | |
| 56.8 | 2.7E-07 | 1.0E-06 | | | |
| | | | | | |
| **Playset and Deck** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Range | | |
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 1.0E-05 | 3.8E-05 | | | |
| 99 | 7.8E-06 | 2.9E-05 | | | |
| 95 | 2.9E-06 | 1.1E-05 | | | |
| 90 | 1.9E-06 | 6.9E-06 | | | |
| 50 | 3.6E-07 | 1.3E-06 | | | |
| 10 | 6.4E-08 | 2.3E-07 | | | |
| 5 | 4.3E-08 | 1.6E-07 | | | |
| 1 | 1.4E-08 | 5.0E-08 | | | |
| minimum | 2.8E-09 | 1.0E-08 | | | |
| >99.9 | 1.0E-05 | 1.0E-04 | | | |
| 94.3 | 2.7E-06 | 1.0E-05 | | | |
| 42.3 | 2.7E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

### 6.2.3 Risk Reduction Through Hand Washing

Note: The baseline scenario includes a certain amount of hand washing. Hand washing, as a mitigation scenario, increases the frequency of this activity over baseline. See Appendix G for more information on hand washing.

Arsenic residues can be transferred from surface of wood to the surface of hands and subsequently be ingested by children through hand-to-mouth activity (in SHEDS-Wood, hand washing did not effect soil exposures, as noted in Section 6.2). The effectiveness of increased hand washing at reducing exposure, and thus, risk was evaluated. Table 6-7 presents the risks based on reduction by hand washing alone at the mean, median, 95$^{th}$ percentile, and 99$^{th}$ percentile. Risks for exposure to playsets and decks was approximately 2 times greater than risks to playsets alone. Risks were within EPA's risk range of $10^{-6}$ to $10^{-4}$ at all points except above the 95$^{th}$ percentile for exposure to playsets and decks. Baseline risks at the 95$^{th}$ percentile, for comparison, were 1.4 x $10^{-4}$ for exposure to playsets and decks and 8.3 x $10^{-5}$ for playsets alone. The pattern was similar at the other estimates of exposure. These baseline risks were only slightly greater than the risks when exposure was reduced by hand washing.

**Table 6-7. Cancer Risks Remaining Following Simulated Reductions from Hand Washing (Warm Climate Only)**

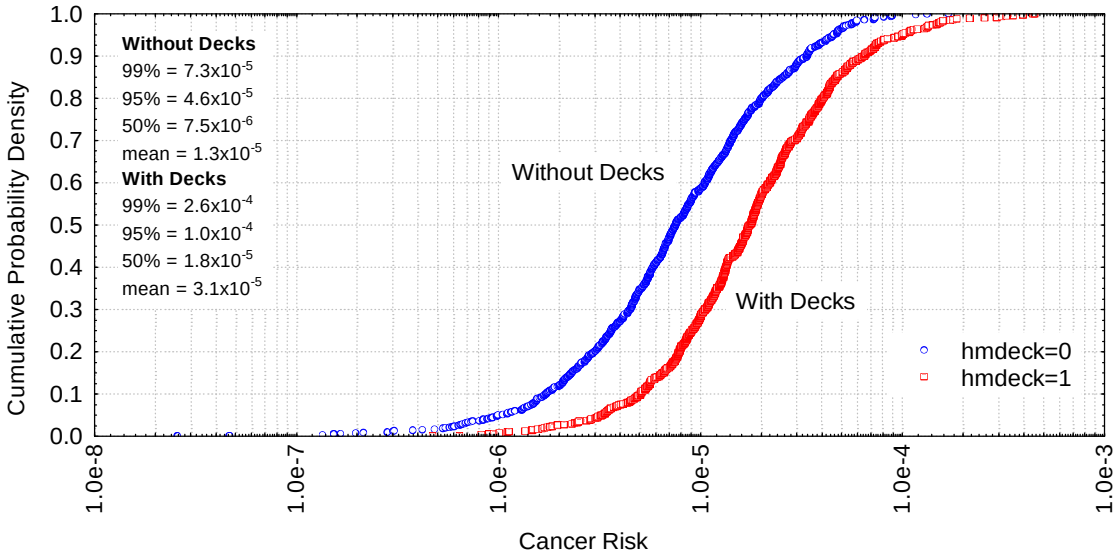
| Scenario | **Arsenic** ($Q_1$*= 3.67 (mg/kg/day)$^{-1}$) | | | |
|---|---|---|---|---|
| | **Mean** | **Median** | **95%ile** | **99%ile** |
| Playsets and decks | 3.1E-05 | 1.8E-05 | 1.0E-04 | 2.6E-04 |
| Playsets only | 1.3E-05 | 7.5E-06 | 4.6E-05 | 7.3E-05 |

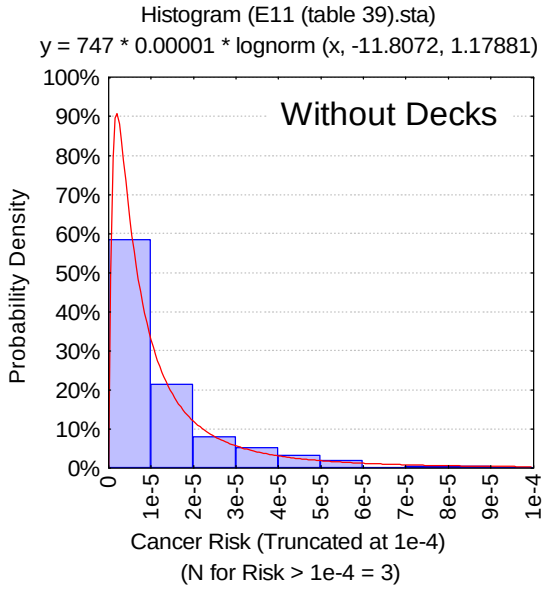
Note: The baseline scenario includes a certain amount of hand washing. Hand washing, as a mitigation scenario, increases the frequency of this activity over baseline. See Appendix G for more information on hand washing.

The cumulative probability function and probability density plots for the hand washing mitigation measure are shown in Figure 6-3. Cumulative percentiles and associated risks are compared to risk levels from $10^{-6}$ to $10^{-4}$ in Table 6-8. The cumulative probability at the $10^{-6}$ risk level was found to be at the very low end of the distribution: less than the 1$^{st}$ percentile for exposure to decks and playsets, and the 5$^{th}$ percentile for exposure to playsets alone. For the baseline scenario, the comparative percentiles were very similar: less than the 1$^{st}$ percentile for deck and playset exposure and 3$^{rd}$ percentile for playset only exposure.

Figure 6-3

Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(Reducing Exposure by Washing Hands after Playing on Deck or Playset)
Scatterplot (E11 (table 39).sta)



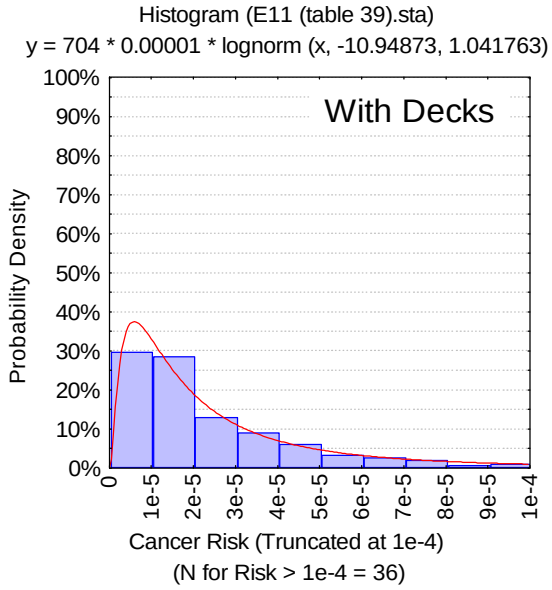



**Table 6-8. Probabilistic Arsenic Cancer Risk Distributions and Risk Ranges for Children in Warm Climates (Reducing Exposure by Washing Hands After Playing on Deck or Playset) (Based on LADDs in Table 39 from the SHEDS-Wood document)**

| **Playset Only** | | | | | |
|---|---|---|---|---|---|
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Range | | |
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 4.6E-05 | 1.7E-04 | | | |
| 99 | 2.0E-05 | 7.3E-05 | | | |
| 95 | 1.2E-05 | 4.6E-05 | | | |
| 90 | 9.2E-06 | 3.4E-05 | | | |
| 50 | 2.1E-06 | 7.5E-06 | | | |
| 10 | 4.7E-07 | 1.7E-06 | | | |
| 5 | 2.7E-07 | 1.0E-06 | | | |
| 1 | 7.7E-08 | 2.8E-07 | | | |
| minimum | 7.0E-09 | 2.6E-08 | | | |
| 99.6 | 2.4E-05 | 1.0E-04 | | | |
| 58.5 | 2.7E-06 | 1.0E-05 | | | |
| 5.1 | 2.7E-07 | 1.0E-06 | | | |
| | | | | | |
| **Playset and Deck** | | | | | |
| Percentile of Exposure | Lifetime Average Daily Dose (LADD) mg/kg/day | Cancer Risk | Risk Range | | |
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 1.2E-04 | 4.5E-04 | | | |
| 99 | 7.0E-05 | 2.6E-04 | | | |
| 95 | 2.7E-05 | 1.0E-04 | | | |
| 90 | 1.8E-05 | 6.5E-05 | | | |
| 50 | 4.8E-06 | 1.8E-05 | | | |
| 10 | 1.4E-06 | 5.1E-06 | | | |
| 5 | 9.0E-07 | 3.3E-06 | | | |
| 1 | 3.1E-07 | 1.1E-06 | | | |
| minimum | 1.3E-07 | 4.7E-07 | | | |
| 94.9 | 2.7E-05 | 1.0E-04 | | | |
| 28.1 | 2.7E-06 | 1.0E-05 | | | |
| 0.7 | 2.6E-07 | 1.0E-06 | | | |

Note: Shaded area indicates all the percentiles of the population that meet the risk level set by the Agency.

### 6.2.4 Risk Reduction Through Use of Sealants and Hand Washing

The reduction in risk through the use of sealants in combination with hand washing was simulated for both sealant effectiveness conditions, moderate (90% reduction in residue concentration) and maximum (99.5% reduction in residue concentration). Risks from the combined mitigation measures did not differ greatly from the sealant only mitigation. Therefore, the results of the combined mitigation measures are summarized below with the full results contained in Appendix C. Table 6-9 presents a comparison of the sealant alone percentiles to the sealant and hand washing percentiles at two risk levels, $10^{6}$ and $10^{-5}$. The highest risk level of the risk range, $10^{-4}$, was not included because all percentiles were greater than the 99$^{th}$ for these mitigation measures. Table 6-9 shows that, at the $10^{-6}$ risk level, adding hand washing increased the cumulative percentile (i.e., reducing risk) by less than 4% under the moderate sealant assumption and less than 2% at the maximum. At the $10^{-5}$ level, the cumulative percentiles decreased slightly. This is an artifact of a Monte Carlo simulation model. These differences are so small, and given the uncertainty in the model, it is concluded there are no substantive differences in mitigation effectiveness of sealants in combination with hand washing.

**Table 6-9. Comparison of Cancer Risks from Combined Mitigation Measures at $10^{-6}$ and $10^{-5}$ Risk Levels**

| Risk Level | Cumulative Percentile | | Difference | Cumulative Percentile | | Difference |
|---|---|---|---|---|---|---|
| | Moderate Reduction | Moderate + Hand wash | | Maximum Reduction | Maximum + Hand wash | |
| $10^{-6}$ | 27 | 28 | 3.7% | 57 | 58 | 1.8% |
| $10^{-5}$ | 94 | 92 | -2.1% | 97 | 96 | -1% |

Note: The baseline scenario includes a certain amount of hand washing. Hand washing, as a mitigation scenario, increases the frequency of this activity over baseline. See Appendix G for more information on hand washing.

Appendix C contains further detail on these combined mitigation measures: the tables of mean, median, 95$^{th}$ percentile, and 99$^{th}$ percentile and the cumulative probability density and probability density plots.

## 6.3 Comparison of Residue and Soil Risks

The most significant exposure route for the population of interest for most scenarios (As and Cr, warm and cold, all time periods) was residue ingestion via hand-to-mouth contact, followed by dermal residue contact. Zartarian et al. (2003) found that under baseline conditions, doses from residues were shown to be from a factor of 6 to a factor of 10 greater than soil doses, depending on the source of exposure. Thus, it was of interest to examine how the distribution of risks changed under the assumed mitigation conditions.

The various mitigation measures were evaluated for the differing contribution to total risk from dislodgeable residues. This is the same type of analysis as was conducted for the baseline condition discussed in Chapter 5. In this chapter, only the maximum and moderate reduction sealant simulation results are presented; figures for all other mitigation measures are shown in Appendix D. Figures 6-4 and 6-5 are line graphs of the estimated cumulative probability density for the maximum and moderate sealant mitigation conditions, respectively. Figure 6-4 clearly shows that exposure to soil accounts for the majority of risk when exposure to residues is reduced by 99.5%. The difference in residue risk between playset only and playset and deck exposures were greater for residues than for soil. Under the moderate residue reduction assumption (Figure 6-5), the difference between residue and soil risks was less. Compared to baseline conditions, the residue risk lines for maximum and moderate reduction shifted to the left (i.e., reduced risk) significantly (see Figure 5-8, Chapter 5). For example, at the 99$^{th}$ percentile, the playset and deck residue risk under the maximum reduction was at $10^{-6}$; under baseline conditions this same risk was greater than $10^{-4}$. Figures 6-6 for maximum reduction and 6-7 for moderate reduction are bar charts for three points from the cumulative distribution curves: median, and 95$^{th}$ percentile and 99$^{th}$ percentile.

In Figure 6-6, soil risks exceed residue risks at the median by a factor of 200 for playset only exposure and by a factor of 150 for playset and deck exposure. These differences decrease to 65 and 50 for playset only and playset and deck exposure, respectively, at the 95$^{th}$ percentile. This same bar chart shows that at the 95$^{th}$ percentile, soil risk from exposure to decks and playsets is at $10^{-5}$; playset only soil risk is slightly less. In Table 6-10, total risk is compared to residue risk at four points on the cumulative distribution, mean, median, 95$^{th}$ percentile, and 99$^{th}$ percentile under the two reduced residue simulated conditions. These risk levels are for playset and deck exposure under warm climate conditions. The first line is total risk and the second line is residue risk. For total risk, the difference between the two mitigation assumptions at the median is a factor of 3; at the 99$^{th}$ percentile this decreases to less than a factor of 1.3. These differences are relatively small because soils now account for the majority of risk (see Figures 6-4 and 6-5). The residue risks show a larger difference between the moderate and maximum reduction assumption. At the median, the difference is a factor of 54 and at the 99$^{th}$ percentile the difference decreases to a factor of 27.

**Figure 6-4 Comparison of Residue & Soil Total Arsenic Risks for Warm Climate 99.5% Reduction**

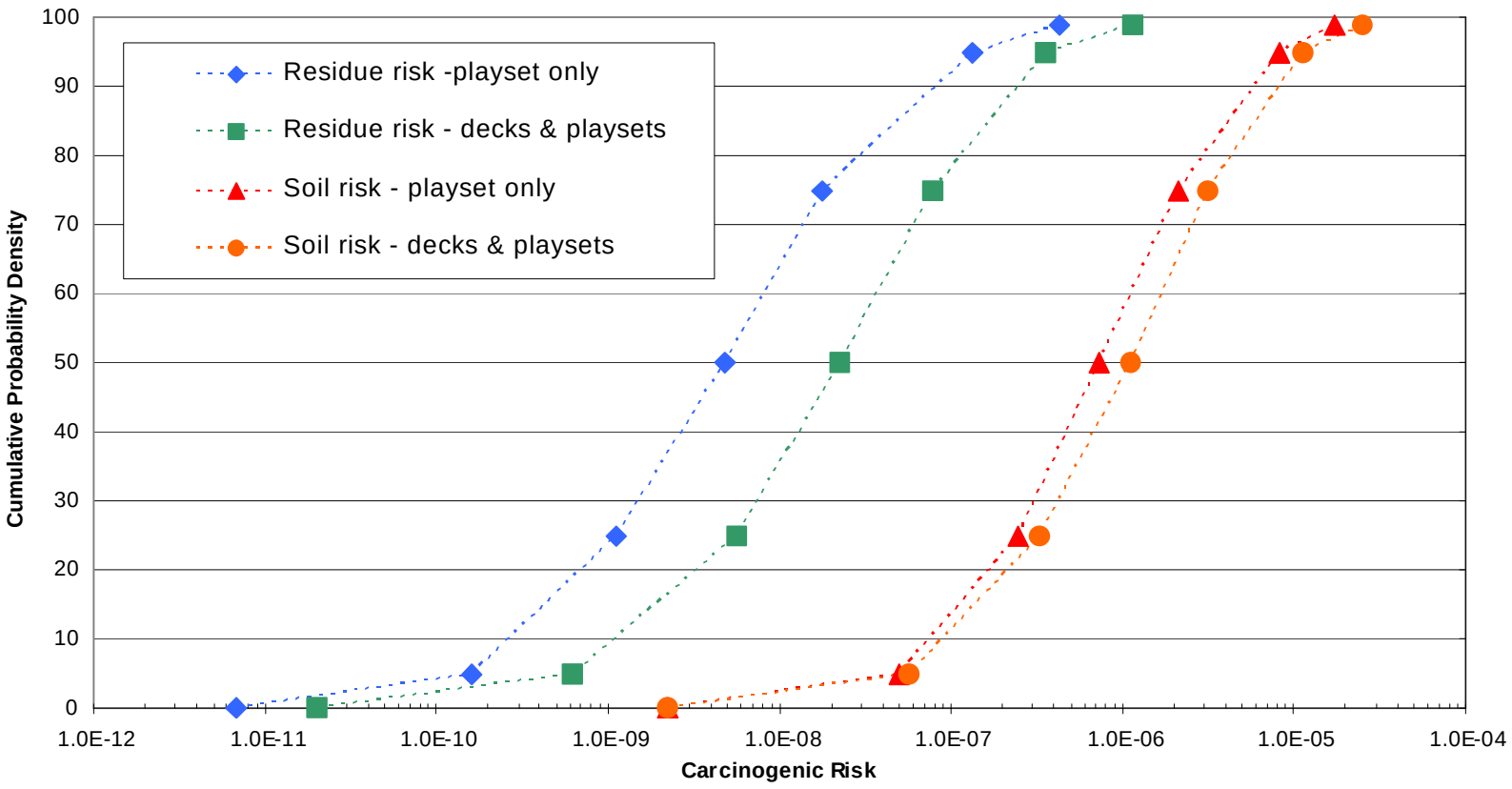


**Figure 6-5 Comparison of Residue & Soil Total Arsenic Risks for Warm Climate 90% Reduction**

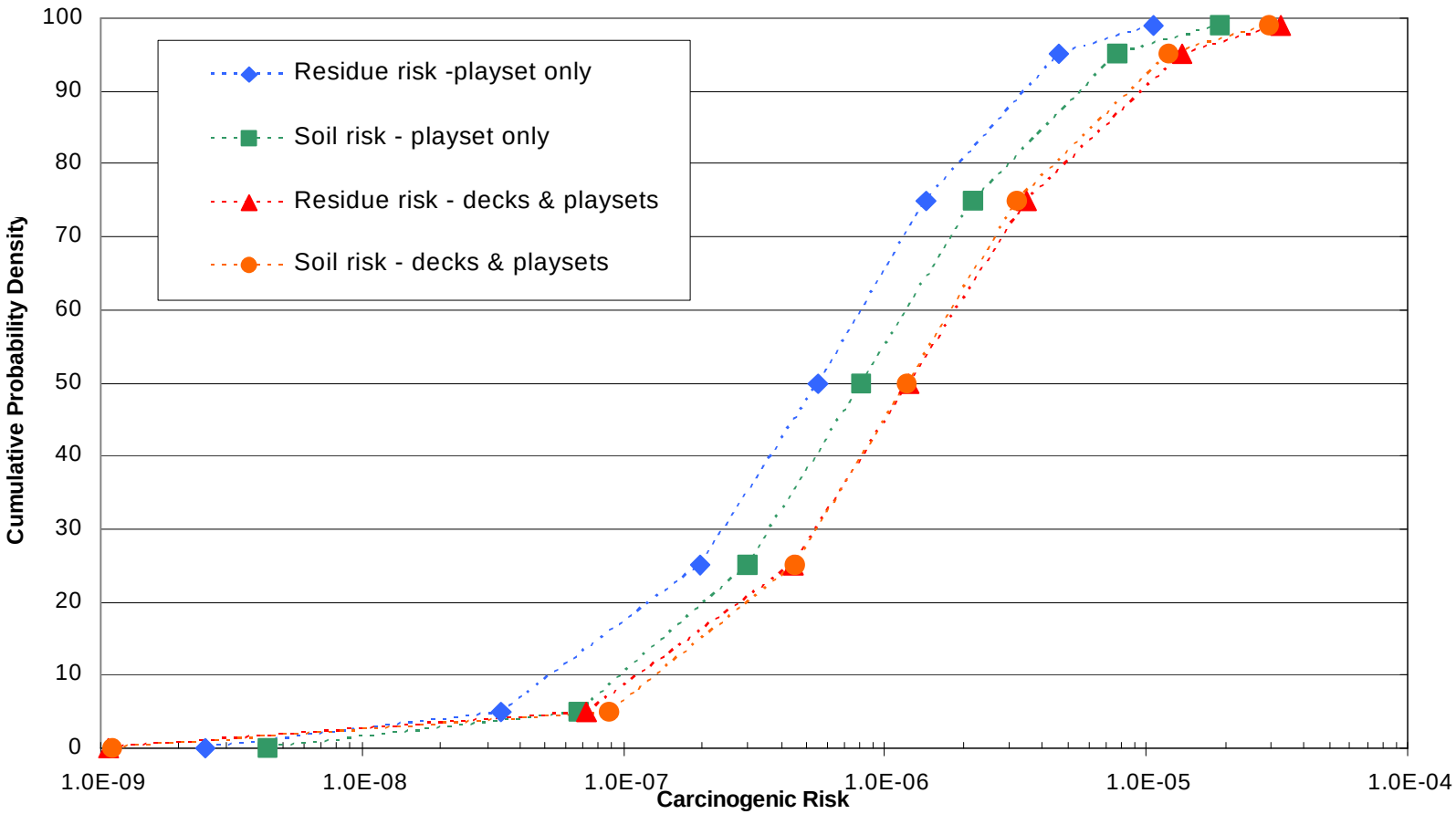


**Figure 6-6 Comparison of Residue and Soil Arsenic Risks for Warm Climate 99.5% Reduction**

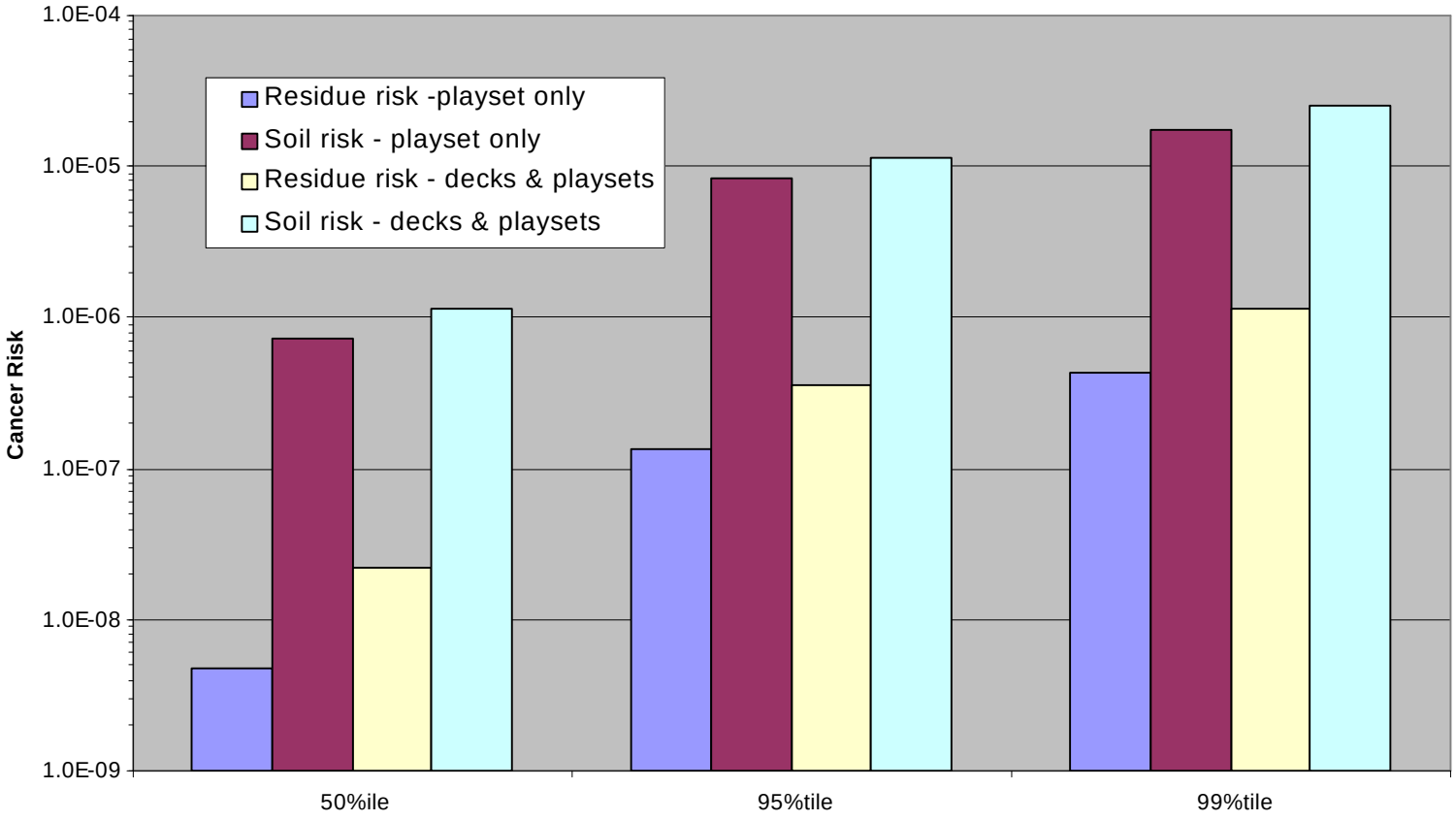
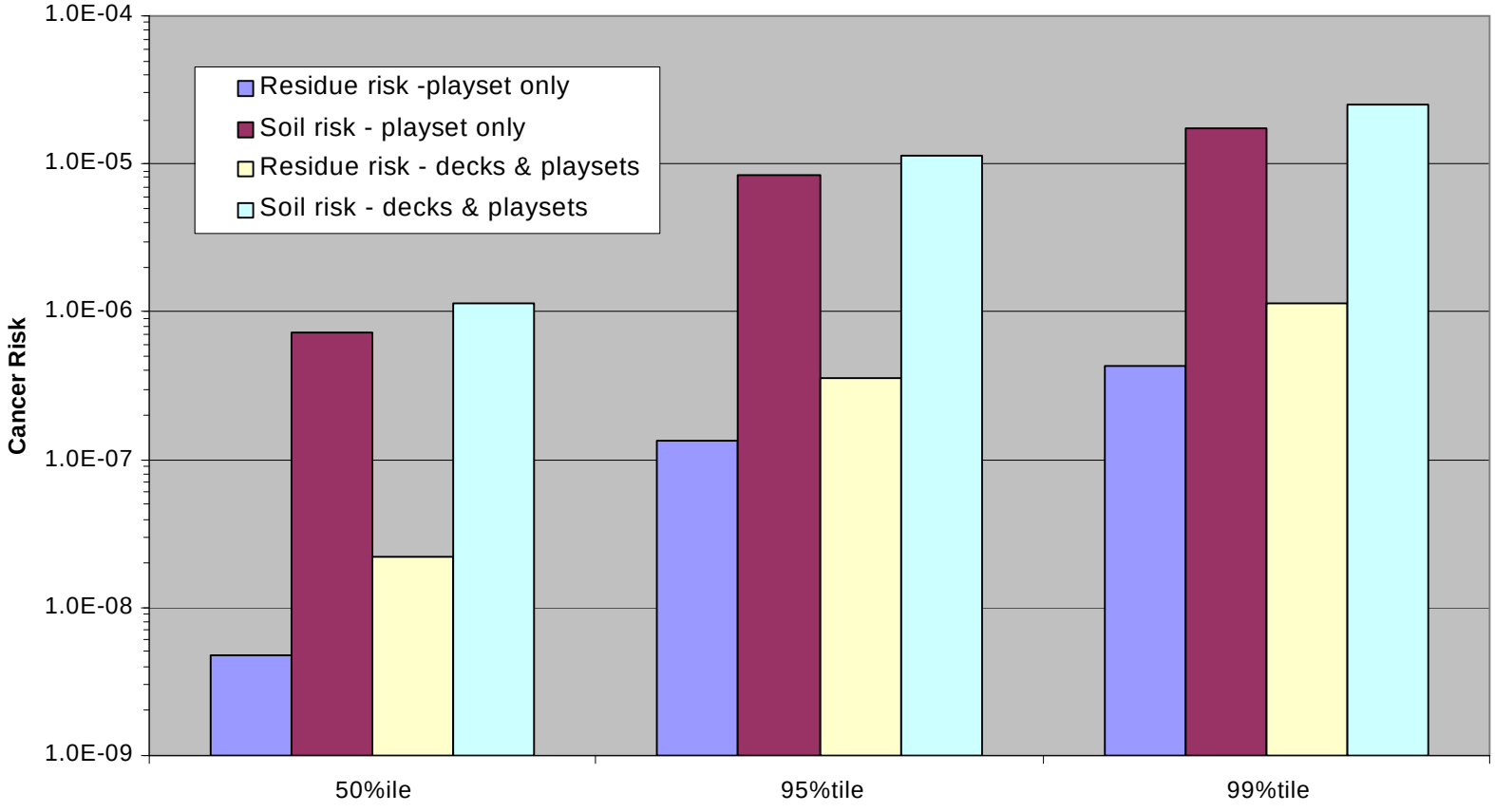


**Table 6-10. Comparison of Total Risk to Residue Only Risk Under Different Mitigation Conditions (Playsets and Decks - Warm Climate)**

| Scenario | Mean | | Median | | 95%ile | | 99%ile | |
|---|---|---|---|---|---|---|---|---|
| | Moderate Reduction | Maximum Reduction | Moderate Reduction | Maximum Reduction | Moderate Reduction | Maximum Reduction | Moderate Reduction | Maximum Reduction |
| Total Risk (Playsets and Decks) | 6.6E-06 | 2.9E-06 | 3.9E-06 | 1.3E-06 | 2.1E-05 | 1.1E-05 | 3.7E-05 | 2.9E-05 |
| Residue Risk (Playsets and Decks) | 3.5E-06 | 9.5E-08 | 1.2E-06 | 2.2E-08 | 1.4E-05 | 3.6E-07 | 3.3E-05 | 1.2E-06 |

**Figure 6-7 Comparison of Residue and Soil Arsenic Risks for Warm Climate 90% Reduction**



## 6.4 Summary

In this chapter, the results from five different mitigation conditions are summarized. Two of the mitigation conditions simulated the effect of a sealant on reducing exposure to dislodgeable residues. For moderately effective sealant conditions, residues were assumed to be reduced by 90%; for maximally effective sealant conditions, residues assumed to be reduced by 99.5%. The other type of mitigation measure simulated was hand washing. In SHEDS-Wood, simulated hand washing only was applicable to residue exposure and not to soil exposure. Hand washing was considered alone and in combination with the sealant conditions. These different mitigation measures were evaluated for the warm climate condition only, as that has the greater exposure, and associated risk. Soil exposures were assumed to be the same as under baseline (i.e., no reduction in exposure due to the use of sealants).

In probabilistic risk assessments, risk reduction is seen as a shifting of the cumulative probability curve towards lower risks (i.e. to the left). Because it is difficult to discuss an entire curve, the cumulative percentiles were compared at the $10^{-6}$ risk level. Table 6-11 presents the cumulative percentiles at the $10^{-6}$ risk level for baseline and each mitigation measure. Increase the frequency of hand washing alone was the least effective mitigation measure. For the sealant only mitigation condition, maximum reduction in exposure shifted the cumulative percentile to the 57$^{th}$ percentile for playsets alone and the 42$^{nd}$ percentile for playset and deck exposure. There was a much similar shift for the moderate reduction measure. Hand washing combined with either sealant mitigation simulation had a minimal effect on the cumulative percentile at the $10^{-6}$ risk level. The differences between baseline conditions and any of the mitigation measures was consistently less for exposure to both playsets and decks than it was for playsets only.

**Table 6-11. Comparison of Mitigation Measures to Baseline at the $10^{-6}$ Risk Level (Warm Climate)**

| | Baseline | Moderate Sealant Reduction | Maximum Sealant Reduction | Hand Washing | Moderate + Hand Washing | Maximum + Hand Washing |
|---|---|---|---|---|---|---|
| Playsets Only | | | | | | |
| Cumulative Percentile | 3 | 27 | 57 | 5 | 28 | 58 |
| Mitigation – Baseline difference | | 24 | 54 | 2 | 25 | 55 |
| Playsets & Decks | | | | | | |
| Cumulative Percentile | 1 | 10 | 42 | <1 | 10 | 44 |
| Mitigation – Baseline difference | | 9 | 41 | 0 | 9 | 43 |

# 7.0 UNCERTAINTY IN THE RISK ASSESSMENT

In risk assessment, uncertainty refers to a lack of knowledge in the underlying science, while variability considers that some individuals in a population have more or less risk than others because of differences in exposure, dose-response relationship or both. Uncertainties are inherent in the risk assessment process. In order to appreciate the limitation and significance of the risk estimates, it is important to have an understanding of the sources and magnitudes of these uncertainties. Sources of uncertainty in this risk assessment, include:

- Environmental media sampling and analysis;
- Chemical fate;
- Toxicity data;
- Exposure assessment modeling; and
- Risk characterization.

Over the course of EPA's evaluation of risks from exposure to CCA, the FIFRA SAP has made recommendations regarding input data and default assumptions used in risk assessment. These recommendations included criteria to evaluate the quality of data included in the modeling effort and appropriate decisions to be made in the absence of adequate data. The SAP provided the Agency with clear criteria to judge data quality in 1999, and these were recognized in support documents provided to the panel. Under conditions of moderate or high uncertainty (absence of sufficient data to fully capture the variability in exposure from these sources), the SAP suggested that the Agency should develop clear default assumptions to be employed until sufficient data are secured. They also recommended that these assumptions should err on the side of overestimation of exposure, or factors that contribute to exposure (U.S. EPA, 2001c).

The uncertainty in a risk assessment reflects the combined uncertainty of all the input variables that are used to estimate an exposure dose combined with the uncertainty of the toxicological parameters. Zartarian et al. (2003) conducted a thorough uncertainty analysis, evaluating model sensitivity and uncertainty through hundreds of iterations of the SHEDS-Wood model. Toxicological parameters have only been evaluated in a qualitative manner due to constraints of time and resources. Therefore, this uncertainty analysis is considered semi-quantitative.

## 7.1 Environmental Media Sampling and Analysis

Analytical data for chromium and arsenic residues on CCA-treated surfaces, as used in the SHEDS-Wood model, were taken from several literature sources, as described in Zartarian et al. (2003). The variability distributions for arsenic and chromium residues and soil concentrations are shown in Table 7 of Zartarian et al. (2003). Summaries of the sources of data used to develop

residue and soil concentrations are presented in Table 8 of Zartarian et al. (2003). Likewise, data for arsenic and chromium residues in CCA-treated decks, as used by SHEDS-Wood to estimate exposure doses, are described in Zartarian et al. (2003). The data sets used to generate the exposure point concentrations are described in the SHEDS-Wood exposure report and summarized in Table 12 of that report. Uncertainty in the exposure point concentration arises from how accurately these various data sets characterize the soil and dislodgeable residue concentrations in the underlying population of treated playsets and decks.

There are many significant variables that can affect the measurement of dislodgeable arsenic and chromium in CCA-treated wood surfaces and in the soil surrounding the treated wood products. Some of these variables include the following:

- The fraction of arsenic and chromium retained in the wood (retentions of CCA type C in wood can range anywhere from 0.25-2.50 pounds per cubic foot (pcf) depending on the different AWPA standards;
- The type of CCA formulation used to treat the wood (CCA treatment solutions are typically classified as either type A, B, or C since they vary in the proportion of arsenic to chromium compounds; however, CCA type C is most commonly used to treat dimensional lumber for above-ground residential applications). Data from type C was most often used in the study data used in this assessment;
- The type of pressure-treated wood (e.g., Douglas fir, southern pine, western cedar, red oak, etc.) can affect leaching and/or transfer of residues;
- The end use of wood (e.g., wood decks, construction or utility poles, marine timbers, fence posts, wood foundation lumber, plywood, and wood for playground structures or decks) determines the amount of CCA used for treatment;
- The degree that the wood has been sanded can affect residue levels;
- Variables in the pressure treatment process can influence the retention of CCA in wood (e.g., temperature and pH, too short of air seasoning time, rapid removal of water, rapid oven drying, etc.);
- The moisture content of the wood can affect CCA content and leaching; and
- The age of the CCA-treated wood can affect residue levels and leaching of CCA to surrounding soil.

To the extent that the data sets used in SHEDS-Wood represent these variables, then these sources of variability are accounted for. However, it is not known how these factors are distributed across the underlying population of decks and playsets and if they are represented in the input data sets.

In addition to the variables mentioned above, wood finishes such as oil stain, varnish, paint or sealant (e.g., polyurethane, acrylic or spar varnish) applied to pressure-treated wood may decrease the amounts of dislodgeable residues in CCA pressure-treated wood surfaces. For

mitigation, this study assumed that sealants can reduce overall residue concentration by 90% and 99.5%. This assumption is based on the results of various studies listed in Chapter 6.0. The following uncertainties regarding coatings should be considered.

- The weight-of-evidence from available studies indicates that certain coatings can substantially reduce dislodgeable and leachable CCA chemicals;
- Reductions of 70 to 95% in dislodgeable arsenic were seen in all studies that subjected CCA wood to natural weathering;
- There is no evidence that water repellents added directly to the CCA treatment solution are effective in reducing leachable/dislodgeable CCA chemicals;
- Current data are not adequate for identifying a particular coating as being clearly superior or inferior to reducing leachable/dislodgeable CCA chemicals; and
- Confidence is highest for polyurethane as this coating has been shown to result in substantial 70 to >95% reduction in dislodgeable arsenic in a well controlled field study, a "real-world" application allowing for effects of use, and a short-term controlled laboratory study.

There is considerable uncertainty regarding the representativeness of the assumed exposure reduction based on the use of sealants. The assumed reductions were selected to evaluate different mitigation control measures. They are reasonable scientific judgements; however, they are not based on a study that would allow extrapolation to the underlying population of decks and playsets implied in this study. Therefore, there is higher uncertainty associated with the dislodgeable residue exposure point concentrations for the mitigation scenarios than with the residue exposure point concentrations used in the baseline risk assessment.

## 7.2 Chemical Fate

Conservative assumptions were made regarding the fate of arsenic and chromium in the environment. For arsenic, it was assumed that concentrations are relatively persistent and immobile. Thus, individuals were assumed to be exposed to the same concentration for the entire duration of exposure (i.e., 6 years). For chromium, all studies used to develop the probability density functions for exposure point concentrations reported total chromium, Cr(III) and Cr(VI). There was concern that assessing chromium(total) doses would overestimate the exposure. Therefore, an attempt was made to determine the speciation of chromium in soil. One study (RTI International, 2003) analyzed Cr(VI) concentrations for a limited subset of samples. All of these samples were below the detection limit of the method. Due to the lack of data on Cr(VI), the Agency decided to make a conservative assumption about speciation in soil. OPP adjusted the ADDs by multiplying by 0.10 (10%) to approximate Cr(VI) speciation. This conservative assumption most likely overestimates exposure to Cr(VI). This overestimation means that uncertainties around the Cr(VI) values are asymmetrical; the probability that concentrations are lower is much greater than the probability that concentrations are higher.

Migration, dispersion, dilution, retardation, degradation, and other attenuation or transformation processes may occur over time that could change the chemical concentrations in residues or soil. It has been conservatively assumed that the concentrations of arsenic are relatively persistent and immobile in both media. With reference to soil, this is an important factor to consider when evaluating the mitigation measures presented in Chapter 6.0. In calculating the exposure doses for these mitigation conditions, SHEDS-Wood used the same soil input distributions as were used for the baseline condition; only exposure to residue concentrations were reduced. Conceptually, a sealant would limit the migration of CCA to surrounding soils, however, there are no data available describing the change in soil concentrations due to the use of sealants. Thus, the approach to estimating overall risk to surrounding soils may be conservative (i.e., risks are overestimated).

## 7.3 Toxicity Data

Varying degrees of uncertainty surround the assessment of adverse health effects in potentially exposed populations to arsenic. Some sources of uncertainty for toxic effects in humans may include:

- Extrapolating from a LOAEL to a NOAEL;
- Extrapolation of data due to intraspecies variation; and
- Extrapolation of epidemiological data from adult populations to children.

In general, cancer risk is a conservative estimate of the risk because the cancer slope factor is characterized as a upper-bound estimate. Therefore, the true risks to humans, while not identifiable, may likelynot exceed the upper-bound estimates and in fact may be lower.

For inorganic arsenic, in this assessment, the slope factor used was 3.67 $(mg/kg/day)^{-1}$. This is the mean slope factor derived from the higher risk approach for both lung and bladder cancers. This slope factor was used by the EPA s Office of Water when it established the MCL for arsenic in drinking water (US EPA, 2001e). In 2001, NRC published an update to the 1999 NRC report and made some specific recommendations with respect to the EPA's Office of Water cancer risk estimate.

The Agency is currently considering NRC's recommendations and their potential impact on the cancer potency estimate. Based on the Agency's considerations of these recommendations, the current proposed cancer potency number may change in the final version of this risk assessment. The slope factor published by EPA's Integrated Risk Information System (IRIS), 1.5 $(mg/kg/day)^{-1}$ is also being revisited in FY2003 due to the recommendation by the NRC in 2001. The uncertainties in the hazard assessment are discussed in detail in Appendix A.

For noncancer effects, OPP assessed exposure using the MOE approach. This approach shows how many times the NOAEL or LOAEL exceed the predicted exposure. The MOE is a ratio of the LOAEL or NOAEL to the predicted exposure, thus, the uncertainties derive from the toxicity value and the manner in which exposure is estimated. Several conservative assumptions are considered in setting the acceptable amount by which the predicted exposure should exceed the LOAEL or NOAEL. Given the conservative assumptions used to generate the LOAEL and NOAEL, it is likely that the total uncertainty of the MOE is assymetrical. There is a greater probability that the true MOE is higher, and a lesser probability that the true MOE is lower.

## 7.4 Exposure Assessment

Exposure assessment is perhaps the most critical step in achieving a reliable estimate of health risks to humans. Little direct data exist to measure arsenic and chromium exposure in children. The exposure estimates used in this assessment were based on absorbed doses calculated by the SHEDS-Wood model (see Zartarian et al., 2003). This model predicted exposure and dose to arsenic and chromium using age and gender time-location-activity diaries for children 1-6 years old. All the data from SHEDS-Wood have been either recommended from the SAP or from the survey of studies in the EPA's Exposure Factor's Handbook (US EPA, 1997b). This information is used by several Agencies to estimate children's exposure durations at outdoor playsets and decks.

The SHEDS-Wood results showed that the significant exposure routes, in order of descending importance, were: residue ingestion via hand-to-mouth contact, dermal residue contact, soil ingestion, and dermal soil contact. The variability and uncertainty in the ADDs and LADDs from SHEDS-Wood were evaluated. Sensitivity of the model to the various input parameters was also evaluated. The following discussion summarizes these results; the full text can be found in Zartarian et al. (2003) (see Section 5).

Variability: Generally, there were several orders of magnitude difference in absorbed dose between the low end and high end percentiles for the various population estimates. This was due to differences in activity patterns, soil and residue concentrations, and exposure factors.
Uncertainty: Uncertainty was modeled using a non-parametric bootstrap approach for ADDs. The estimated uncertainty was indicated by a factor of 3 at the median and 4 at the 95$^{th}$ percentile.
Sensitivity: The most critical input variables to the model results were: wood surface residue-to-skin transfer efficiency, deck wood surface residue concentration, fraction of hand surface area mouthed, and hand washing events per day.

The estimated uncertainty of a factor of 3 to 4 appeared to be approximately symmetrical around the predicted absorbed dose.

## 7.5 Risk Characterization

There are various sources of uncertainty in each step of the risk assessment process. In the final estimate of risk, the uncertainty in the toxicity value is combined with the uncertainty in the absorbed dose estimate. This combined uncertainty is greater than the uncertainty in the exposure estimate, however, the question is - how much greater? This is not a simple question to answer. It is beyond the scope of this risk assessment to address that question in a quantitative manner. Thus, it is addressed in conceptual manner. Further limitations of this discussion are that it applies to the carcinogenic risk from exposure to arsenic, as that is the most critical and it is more appropriate to the baseline risk results (Chapter 5.0) and less so the mitigation risks (Chapter 6.0). (This is due to the lack of any information on the uncertainty of the assumed effectiveness of the mitigation measures.)

Uncertainty can be considered a cloud surrounding a value. If risks are presented as a CDF, then there is a cloud surrounding the entire curve. The shape of this cloud is determined by the uncertainty of the input variables and how these uncertainties are combined mathematically. There are standard approaches for estimating the combined uncertainty for the simple case of two input variables where the uncertainty of each has been quantified and it is symmetrical around the a central value of the variable. More sophisticated approaches are required when either of the uncertainties are asymmetrical.

In this risk assessment, only the uncertainty in the absorbed dose was characterized; the uncertainties in the toxicity values were not characterized. To generate an uncertainty estimate for the risk characterization, similar to the absorbed dose uncertainty estimate, would require: (1) knowledge of the uncertainty in the slope factor and NOAEL/LOAEL, and (2) running a Monte Carlo simulation to calculate risk that included a distribution function for the slope factor. Thus, the uncertainty of the risk characterization step can not be quantified.

What is known about the uncertainty around the input variables is very different. The slope factor in this assessment is not a mean value. It is at the high end of the distribution, based on the various conservative factors that are included in the formulation of the final value (e.g., low dose extrapolation and extrapolation from adults to children). Therefore, the “uncertainty cloud” around the slope factor is asymmetrical. How asymmetrical is not known; however, it is likely that the vast majority of the cloud is below the slope factor value and very little is above it. By contrast, uncertainty in the absorbed dose estimate appears to be symmetrical based on the analysis presented in the SHEDS-Wood report (Zartarian et al., 2003).

The uncertainty in the risk estimate is a combination of the symmetrical uncertainty in the absorbed dose and the asymmetrical uncertainty in the slope factor. It is beyond the scope of this assessment to mathematically combine those uncertainties. However, a relative estimate of the shape and location of the uncertainty cloud is possible. The first order estimate is based on the

absorbed dose uncertainty. Combining the slope factor uncertainty would increase the size (dispersion) of the cloud and shift it so that it was asymmetrical around the risk estimate (i.e., more of the cloud is below the risk estimate than above it).

Therefore, it is concluded that the arsenic carcinogenic risk, (especially under the assumed mitigation measures) are conservative estimates of risk. The Cr(VI) noncancer MOEs are also considered to be conservative (i.e., MOEs are most likely higher) due to the assumption that 10% of total chromium was assumed to be present as Cr(VI). The noncancer arsenic MOEs are also considered to be conservative estimates, given the assumptions for the LOAEL.

## 8.0 REFERENCES*

ACC, 2002. Assessment of Potential Inhalation and Dermal Exposure Associated with Pressure Treatment of Wood with Arsenical Products. MRID 455021-01.

ACC, 2003a. CCA Workgroup, Relative Bioavailability of Dislodgeable Arsenic from CCA-Treated Wood, Prepared by Veterinary Medical Diagnostic Laboratory Medicine. University of Missouri, Syracuse Research Corporation, Denver, Colorado.

ACC, 2003b. CCA Workgroup, Relative Bioavailability of Arsenic in Soil Affected by CCA-Treated Wood. Prepared by Veterinary Medical Diagnostic Laboratory College of Veterinary Medicine. University of Missouri, Syracuse Research Corporation, Denver, Colorado.

APVMA, 2003. Arsenic Timber Treatments (CCA and Arsenic Trioxide) Review Scope Document. March, 2003. http://www.apvma.gov.au/chemrev/arsenic_scope.pdf Accessed September 17, 2003.

AWPA, 1998. Book of Standards. P5-P98 Standard for Waterbourne Preservatives.

CDHS, 1987. Condensed Report to the Legislature: Evaluation of Hazards Posed By the Use of Wood Preservatives on Playground Equipment. State of California. Office of Environmental Health Hazard Assessment, Department of Health Services, Health and Welfare Agency.

Cooper, P. and Y.T. Ung 1997. Effect of water repellents on leaching of CCA from treated fence and deck units. An update presented at the 28th Annual Meeting of the International Research Group on Wood.

CPSC, 1990. Estimation of Hand-to-Mouth Activity by Children Based on Soil Ingestion for Dislodgeable Arsenic Exposure Assessment. Memorandum from B. Lee to E.A. Tyrell.

CPSC, 2003a. Briefing Package. Petition to Ban Chromated Copper Arsenate (CCA)-Treated Wood in Playground Equipment (Petition HP 01-3). February 2003.

CPSC, 2003b. Briefing Package. Staff Recommendation to Ban Use of Chromated Copper Arsenate (CCA)-Treated Wood in Playground Equipment (Petition HP 01-3) October 2003.

Cooper, P.A., 2003. CCA Fixation and its Implications on Availability of Hexavalent Chromium (Cr VI) for Dislodgeability and Leaching. Prepared for the American Chemistry Council Arsenicals Wood Preservative Task Force. August 20, 2003.

DeGroot, R.C., T.W. Popham, L.R. Gjovik, and T. Forehand, 1979. Distribution Gradients of Arsenic, Copper, and Chromium Around Preservative-treated Wooden Stakes. J. Environ. Qual., 8(1):39-41.

Edgecomb, M, 2003. Bangor News. Legislators Ban Lumber Treated With Arsenic. June 4,2003. http://www.bangornews.com/editorialnews/article.cfm/ID/402406/cfid/9206765/cftoken/3105964 Accessed September 17, 2003.

EMRA, 2003a. Report on Copper, Chromium and Arsenic (CCA) Treated Timber. Report prepared by Deborah Read, Public Health Physician, EMRA. April. ISBN 0-478-21521-5.

EMRA, 2003b. Copper Chromium Arsenic (CCA) Treatment of Timber. Report from the Environmental Risk Management Authority. Niel Walter, Chair. May.

Environmental Health Perspectives, 2001. Volume 109, Number 6, June 2001. http:\\Ehpnet1.niehs.nih.gov/docs/2001/109-6/focus.pdf. Accessed September 18, 2003.

Franzblau, A. and Lilis, R., 1989. Acute Arsenic Intoxication from Environmental Arsenic Exposure. Archives of Envir. Health 44(6). 385-390.

Ginsberg, G., 2003. Assessing Cancer Risks from Short-term Exposures in Children. Risk Analysis, Vol 23, No. 1. Society for Risk Analysis.

Healthy Schools Network, 2003. The Guide to Playgrounds and Arsenic Wood. Undated http://www.healthyschools.or/documents/CCA_Guide.pdf
Accessed September 15, 2003.

Hopenhayn,C.; Ferreccio, C.; Browning, S.R.; Huang, B. Peralta, C.; Gibb, H.; and Hertz-Picciotto, I. 2003. Arsenic Exposure from Drinking Water and Birth Weight. Epidemiology. 14:593-602.

Kartal, S. and Lebow, S., 2000. Effect of Compression Wood on Leaching of Chromium, Copper, and Arsenic From CCA-C Treated Red Pine (*Pinus resinosa* Ait.) USDA Forest Service, Forest Products Laboratory, Madison, WI, USA.

Lebow, S., 1996. Leaching of Wood Preservative Components and their Mobility in the Environment- Summary of Pertinent Literature. Gen. Tech. Rep. FPL-GTR-93. Madison, WI: U.S. Department of Agriculture, Forest Service, Forest Products Laboratory, 36 p.

McMahon and Chen, 2003. Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium. Prepared by the Office of Pesticide Programs Antimicrobial Division. Draft Version, November 4, 2003.

Mizuta, N, Mizuta, et al., 1956. An Outbreak of Acute Arsenic Poisoning Caused by Arsenic-Containing Soy-Sauce (Shoyu). A Clinical Report of 220 Cases. Bull Yamaguchi Med Sch 4(2-3):131-149.

Nico, P., Fendorf, S., Lownery, Y., Holm, S. and Ruby, M., 2003. Chemical Structure of Arsenic and Chromium in CCA-Treated Wood: Implications of Environmental Weathering. Draft Version August 2003, Unpublished.

NRC (National Research Council), 2001. Arsenic in Drinking Water. 2001 update. National Academy Press, Washington, D.C.

NRC (National Research Council), 1999. Arsenic in Drinking Water. National Academy Press, Washington, D.C.

Osmose, 2000. Metal Removal from CCA-treated Lumber Under Simulated Normal Use Conditions. Osmose Research Division. Osmose Wood Preserving Company. Buffalo, New York.

Our Stolen Future, 2003. Arsenic Treated Wood Banned in Maine. June 4, 2003. http://www.ourstolenfuture.org/Commentary/News/2003/2003-0604-BDN-arsenicban.htm. Accessed September 15, 2003.

Personal Communication, 2003. Email from Cathy Campbell, PMRA to Winston Dang, U.S.EPA. 9/16/2003. Re: Canada's Action on CCA.

PMRA, 2003. Re-evaluation Note- Chormated Copper Arsenate (CCA). April 3, 2002. http://www.hc-sc.gc.ca/pmra-arla/english/pdf/rev/rev2002-030e.pdf Accessed on September 17, 2003.

Riedel, D., D. Galarneau, J. Harrison, D.C. Gregoire and N. Bertrand. February, 1991. Residues of Arsenic, Chromium and Copper On and Near Playground Structures Built of Wood Pressure-treated with CCA Type Preservatives. Health and Welfare Canada. (Unpublished).

RTI International, 2003. Assessment of Exposure to Metals in CCA-Preserved Wood: Full Study. Prepared for American Chemistry Council CCA Task Force. Prepared by RTI International. Research Triangle Park, North Carolina. June 20, 2003. (This reference is same as ACC 2003a in SHEDS-Wood document)

Spease, 2002. CA Playground Safety Regulation. July 13, 2002. http://www.spease.com/Wood%20Preservatives.html. Accessed September 15, 2003.

Stilwell, DE and Gorny, KD, 1997. Contamination of Soil with Copper, Chromium, and Arsenic Under Decks Built from Pressure Treated Wood. Bulletin of Environmental Contamination and Toxicology 58:22-29. Springer Verlag New York, Inc.

Stilwell, D., 1998. Arsenic From CCA-treated Wood Can be Reduced by Coating. Frontiers of Plant Science 51(1):6-8.

Townsend, T.G., K. Stook, T.M. Tolaymat, J.K. Song, H. Solo-Gabriele, N. Hosein, and B. Khan, 2001. New lines of CCA-treated wood research: In-service and disposal issues - Final Technical Report #00-12. Submitted to the Florida Center for Solid and Hazardous Waste, Gainesville, Florida.

Tyl, R.W., Marr, M., and Meyers, C.B., 1991. Developmental Toxicity Evaluation of Chromic Acid Administered by Gavage to New Zealand White Rabbits. Research Triangle Institute, Reserch Triangle Park, NC Study No. 60C-4808-30/40. Unpublished.

U.S. EPA, 2003a. Draft Final Guidelines for Carcinogenic Risk Assessment. Risk Assessment Forum. National Center for Environmental Assessment. Washington D.C. EPA/630/P-03/001A.

U.S. EPA, 2003b. Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens. Risk Assessment Forum. National Center for Environmental Assessment. Washington D.C. EPA/630/R-03/003.

U.S. EPA, 2001a. An Evaluation of the Non-Dietary Exposures and Risks to Children from Contact with CCA-Treated Wood Playground Structures and CCA-Contaminated Soil (Internal Draft Only). Prepared by the Office of Pesticide Programs Antimicrobial Division. Internal Draft Version, May 30, 2001.

U.S. EPA, 2001b. Children's Exposure to CCA-Treated Wood Playground Equipment and CCA-Contaminated Soil (Final Report to SAP 9/27/01). Prepared by the Office of Pesticide Programs Antimicrobial Division. Draft Version, September 27, 2001.

U.S. EPA, 2001c. Memorandum: Transmittal of the Final Report for the FIFRA Scientific Advisory Panel (SAP) Meeting Held October 23-25, 2001. From Olga Odiott and Larry Dorsey, Office of Science Coordination and Policy to Marcia Mulkey, Director Office of Pesticide Programs. October 25, 2001.

U.S. EPA, 2001d. Process for Conducting Probabilistic Risk Assessment. Risk Assessment Guidance for Superfund. Volume 3. Part A. December 31, 2001.

U.S. EPA, 2001e. National Primary Drinking Water Regulations: Arsenic and Clarifications to Compliance and New Source Contaminants Monitoring; Final Rule. 40CFR Parts 9, 141, and 142. EPA-815-Z-01-001.

U.S. EPA, 1990. National Oil and Hazardous Substances Pollution Contingency Plan. Final Rule. 40 CFR 300: 55 *Federal Register*, 8666-8865, March 8.

U.S. EPA, 1998a. Guidance for Submission of Probabilistic Exposure Assessments to the Office of Pesticide Programs' Health Effects Division. Prepared by the Office of Pesticide Programs Health Effects Division. Draft Version, February 6, 1998.

U.S. EPA 1998b. IRIS, Chromium (VI), 1998; CASRN 18540-29-9, September 3, 1998.

U.S. EPA, 1997a. Policy for Use of Probabilistic Analysis in Risk Assessment. Prepared by the Science Policy Council. http://epa.gov/esp/osp/spc/probpd.htm.

U.S. EPA, 1997b. Exposure Factors Handbook. Volume I. Prepared by the Office of Research and Development. Washington D.C., 20460. EPA/600/P-95/002Fa.

Waalkes, M.P.; Ward, J.M.; Liu, J. and Diwan, B.A. 2003. Transplacental carcinogenicity of inorganic arsenic in the drinking water: induction of hepatic, ovarian, pulmonary, and adrenal tumors in mice. Toxicology and Applied Pharmacology: 186:7-17

Wester, R.C., Hui, X., Barbadillo, S., Maibach, MI., Lowney, Y.W., Schoof, R.A., Holm, S.E., and Ruby, M.V., 2003. In Vivo Percutaneous Absorption of Arsenic from Water and CCA-treated Wood Residue Draft. August 2003.

Wester, R.C., Maibach, H.I., Sedik, L. Melendres, J., and Wader, M., 1993. In Vivo and in Vitro Percutaneous Absorption and Skin Decontamination of Arsenic From Water and Soil. Fundamental and Applied Toxicology 20:336-340

Waalkes, M.P.; Ward, J.M.; Liu, J. and Diwan, B.A. 2003. Transplacental carcinogenicity of inorganic arsenic in the drinking water: induction of hepatic, ovarian, pulmonary, and adrenal tumors in mice. Toxicology and Applied Pharmacology: 186:7-17

Zartarian, V.G., Xue, J., Özkaynak, H., Dang, W., Glen, G. and Stallings C., 2003. A Probabilistic Exposure Assessment for Children Who Contact CCA-Treated Playsets and Decks Using the Stochastic Human Exposure and Dose Simulation Model for the Wood Preservative Exposure Scenario (SHEDS-Wood). Prepared by Office of Research and Development, National Exposure Research Laboratory and Office of Pesticide Programs, Antimicrobial Division. Draft Report September 25, 2003.

*Additional references used to develop exposures and toxicity can be found in Zartarian et al. (2003) and Appendix A, respectively.

# APPENDICES

# A Probabilistic Exposure and Risk Assessment for Children Who Contact CCA-Treated Playsets and Decks

**Draft Final Report**

**November 10, 2003**

Prepared by

U. S. Environmental Protection Agency
Office of Pesticide Programs, Antimicrobials Division
and
Office of Research and Development
National Exposure Research Laboratory

**DISCLAIMER**
This report has undergone internal EPA review through the Office of Research and Development (ORD) and the Office of Pesticide Programs (OPP). It is currently undergoing additional external review through OPP. Thus, this report should not be considered final until it has been submitted to OPP's Scientific Advisory Panel. Some of the statutory provisions described in this report contain legally binding requirements. However, this report does not substitute for those provisions or regulations, nor is it regulation itself. Any decisions regarding a particular risk reduction process and remedy selection decision will be made based on the statute and regulation, and EPA decision makers retain the discretion to adopt approaches on a case-by-case basis.

# Appendix A

## Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium

**November 10, 2003**

# Hazard Identification and Toxicology Endpoint Selection for Inorganic Arsenic and Inorganic Chromium

November 10, 2003

Timothy F. McMahon, Ph.D. and Jonathan Chen, Ph.D.
U.S. Environmental Protection Agency
Office of Pesticide Programs
Antimicrobials Division

TABLE OF CONTENTS

0.0 INTRODUCTION ..... 3

1.0 HAZARD CHARACTERIZATION ..... 4
1.1 Hazard Characterization - Arsenic ..... 4
1.1.1 Acute Toxicity ..... 4
1.1.2 Non-Acute Toxicity ..... 5
1.1.3 Metabolism ..... 9
1.2 Hazard Characterization - Chromium ..... 10
1.2.1 Acute Toxicity ..... 11
1.2.2 Non-Acute Toxicity ..... 13
1.2.3 Metabolism ..... 19

2.0 DOSE-RESPONSE ASSESSMENT ..... 20
2.1 Inorganic Arsenic-Endpoint Selection ..... 22
2.1.1 Acute Reference Dose (RfD) ..... 22
2.1.2 Chronic Reference Dose (RfD) ..... 22
2.1.3 Short (1-30 days ) and Intermediate (30-180 days) Incidental Oral Exposure ..... 24
2.1.4 Dermal Absorption ..... 24
2.1.5 Short (1-30 days ) and Intermediate (30-180 days) Dermal Exposure ..... 25
2.1.6 Long-Term Dermal Exposure ..... 25
2.1.7 Short-, Intermediate-, and Long-term Inhalation Exposure ..... 25
2.1.8 Carcinogenicity ..... 26
2.2 Inorganic Chromium Endpoint Selection ..... 31
2.2.1 Acute Reference Dose (RfD) ..... 31
2.2.2 Chronic Reference Dose (RfD) ..... 31
2.2.3 Short-Term (1-30 days) and Intemediate-Term (30-180 days) Incidental Oral Exposure ..... 31
2.2.4 Dermal Absorption ..... 32
2.2.5 Short-, Intermediate-, and Long- term Dermal Exposure ..... 33
2.2.6 Inhalation Exposure (all durations) ..... 33
2.2.7 Carcinogenicity ..... 34

3.0 REFERENCES ..... 36

## 0.0 INTRODUCTION

The Agency has become aware in recent years of concerns raised by the public regarding the potential hazards associated with the continued use of CCA-treated lumber, especially the use of this material in playground equipment to which infants and children may be exposed through direct dermal contact with the treated wood and/or soil around the treated wood structure, or through oral ingestion of chemical residue from touching of wood and/or soil and subsequent hand-to-mouth behaviors. As a result of these concerns, the Agency has embarked upon a process to assess the exposures and risks associated with the current uses of CCA-treated lumber, including exposures and risks associated with use of this wood in playground structures. In any such assessment, the toxicity of the pesticide chemical must first be adequately described, either through submission of guideline toxicology studies that are reviewed by the Agency, or through citation of scientific studies in the peer-reviewed literature. For the present assessment, the Agency recognizes that inorganic arsenic and inorganic chromium are the compounds of toxicological concern with respect to exposure to CCA-treated wood. The following sections characterize the hazards of inorganic arsenic and inorganic chromium. Information was summarized from submitted toxicology studies, the open scientific literature, and from published documents by the USEPA and the Agency for Toxic Substances and Disease Registry (ATSDR). It is noted for inorganic arsenic that in most cases, human data (in the form of epidemiology studies and case reports) provide the basis for the hazard identification, as most laboratory animal models appear to be substantially less susceptible to arsenic toxicity than humans.

For chromium, hazard data show clearly that Cr(VI) demonstrates more significant toxicity than Cr (III). The Agency has not identified any endpoints of concern for Cr(III). For exposure to Cr(VI), the Agency has identified toxicological endpoints of concern and has used these endpoints in conjunction with exposure to Cr(VI) for evaluating risks associated with Cr (VI).

Copper as a component of CCA-treated wood is not considered in this document. Copper is an essential nutrient which functions as a component of several enzymes in humans, and toxicity of copper in humans involves consumption of water contaminated with high levels of copper, suicide attempts using copper sulfate, or genetic disorders such as Wilson's disease.

# 1.0 HAZARD CHARACTERIZATION

## 1.1 Hazard Characterization - Arsenic

Arsenic is a naturally occurring element present in soil, water, and food. In the environment, arsenic exists in many different forms. In water, for example, arsenic exists primarily as the inorganic forms As +3 (arsenite) and As +5 (arsenate), while in food, arsenic exists primarily in organic forms (seafood, for example, contains arsenic as arsenobetaine, a form which is absorbed but rapidly excreted unchanged). Human activities also result in the release of arsenic into the environment, such as residual arsenic from former pesticidal use, smelter emissions, and the use of chromated copper arsenicals (CCA) in the pressure-treatment of wood for construction of decks, fences, playgrounds, and other structural uses.

Inorganic arsenic, prior to 1991, was used as an agricultural pesticide. In 1991, the Agency proposed cancellation of the sole remaining agricultural use of arsenic acid (As+5) on cotton. Subsequently, this registration was voluntarily canceled by the sponsor and made immediately effective by the Agency (Federal Register, 1993). However, inorganic arsenic contained within CCA-treated wood continues to be widely used for decking and fencing lumber as well as playground equipment.

### 1.1.1 <u>Acute Toxicity</u>

The acute toxicity summary of inorganic arsenic (arsenic acid 7.5%) is summarized in **Table 1**. Humans are very sensitive to arsenic toxicity when compared with other experimental animals. Inorganic arsenic is acutely toxic, and ingestion of large doses leads to gastrointestinal symptoms, disturbances of cardiovascular and nervous system functions, and eventually death. The effects seen after short-term arsenic exposure (appearance of edema, gastrointestinal or upper respiratory symptoms) differ from those after longer exposure (symptoms of skin and neuropathy). Some of the effects after short-term exposure tended to subside gradually from the 5th day of the illness, despite continuous intakes of the poison. In contrast, symptoms of peripheral neuropathy appeared in some individuals even after the cessation of arsenical intakes

The acute oral toxicity of inorganic arsenic in humans shows lethal effects in the range of 22-121 mg/kg, which is consistent with results of animal studies showing lethality in the range of 15-175 mg/kg. There are no studies reporting death in humans after dermal exposure to inorganic arsenic, which is consistent with results of animals studies showing no mortality at dermal doses up to 1000 mg/kg. Mortality in humans from short-term inhalation exposure to inorganic arsenic has not been observed in occupational settings at air levels up to 100 $mg/m^3$. One study in pregnant rats reported lethality of inorganic arsenic at a concentration of 20 $mg/m^3$. Arsenic has been shown to result in contact dermatitis in humans exposed occupationally, and animal studies are also suggestive of mild to severe dermal irritation after application of arsenic to skin. Severe ocular irritation was observed in an acute eye irritation study (MRID # 00026356). Arsenic does not produce skin sensitization in a guinea pig model (MRID # 40646201).

### 1.1.2 Non-Acute Toxicity

Subchronic studies with arsenic in experimental animal models have produced only generalized toxicity, i.e., weight loss, and decreased survival, while data from human exposures have shown more specific toxic effects, such as neurotoxicity and hyperkeratoses of the skin of the hands and feet (ATSDR, 2000a).

Chronic toxicity studies with inorganic arsenic in experimental animals also show a lack of specific toxic effects, whereas the scientific literature that describes chronic human exposure shows a clear relationship between chronic exposure to inorganic arsenic and the development of skin cancer as well as cancers of the lung, liver, and bladder (ATSDR, 2000; NRC, 1999).
The most notable example of this is the data of Tseng, (1968, 1977) who conducted epidemiological studies of chronic oral exposure of humans to arsenic contained in food and water. From these studies it was noted that hyperpigmentation, keratosis and possible vascular complications [Blackfoot disease] occurred at a dose of 0.17 mg arsenic per liter of water, equivalent of 0.014 mg/kg/ day. Several follow-up studies of the Taiwanese population exposed to inorganic arsenic in drinking water showed an increase in fatal internal organ cancers as well as an increase in skin cancer. Other investigators found that the standard mortality ratios (SMR) and cumulative mortality rates for cancers of the bladder, kidney, skin, lung, and liver were significantly greater in the Blackfoot disease endemic area of Taiwan when compared with the age adjusted rates for the general population of Taiwan.

Data on the developmental and reproductive toxicity of inorganic arsenic in humans is not extensive. One study conducted in Sweden among copper smelter workers showed significantly reduced live birth weights in offspring of women employed at the copper smelter and increased incidence of spontaneous abortion among those who worked at the smelter or lived i nproximity to it. However, effects from exposure to lead or copper in this study could not be ruled out. Hopenhayn-Rich (2000) conducted a retrospective study of late fetal, neonatal and postnatal mortality in Antofagasta, Chile for the years 1950 to 1996. The data from this study indicated an elevation in late fetal, neonatal and postnatal mortality compared to a comparison group in Valparaiso, Chile during the period when drinking water in Antofagasta was contaminated [860 ug/L] with arsenic (1958 to 1970). There was a decline in late fetal, neonatal and postnatal mortality when the concentration of arsenic in the drinking water declined due to installation of a water treatment plant. After installation of the plant, the mortality rates in Antofagasta were indistinguishable from those in Valparaiso. It was noted that the mothers involved in this incident had characteristic arsenic-induced skin lesions. A prospective cohort study was conducted in these two cities during the period in the period when drinking water arsenic levels in Antofagasta is 40µg/L and in Valparaiso is less than 1µg/L. By comparing the preganancy and birth

## Table 1. Acute Toxicity Summary of Arsenic Acid (75%)

| Guideline Reference No. | Study Type | MRID/ Data Accession No. | Results | Toxicity Category |
|---|---|---|---|---|
| 81-1<br><br>(OPPTS 870.1100) | Acute Oral | 404090-01 | Mouse<br>$LD_{50}$ = ? 141 mg/kg<br>= ? 160 mg/kg<br>M+F = 150 mg/kg | II |
| | | 26356 | Rat<br>$LD_{50}$ = ? 76 mg/kg<br>= ? 37 mg/kg<br>M+F = 52 mg/kg | I |
| 81-2<br><br>(OPPTS 870.1200) | Acute Dermal | 26356 | Rabbit<br>$LD_{50}$ = ? 1750 mg/kg<br>= ? 2300 mg/kg | II |
| 81-3<br><br>(OPPTS 870.1300) | Acute Inhalation | 404639-02 | Mouse<br>$LC_{50}$ = ? 1.153 mg/L<br>= ? 0.79 mg/L<br>M+F = 1.040 mg/L | II |
| 81-4<br><br>(OPPTS 870.2400) | Primary Eye Irritation | 26356 | Rabbit<br>3/6 animals died by day 7. The 3 surviving animals were sacrificed on day 9 because of severe ocular irritation and corrosion. | I |
| 81-5<br><br>(OPPTS 870.2500) | Primary Skin Irritation | 26356 | Rabbit<br>At 30 minutes, all animals showed moderate to severe erythema and slight to severe edema. All animals died prior to the 24 hour observation. | I |
| 81-6<br><br>(OPPTS 870.2600) | Dermal Sensitization | 406462-01 | Guinea Pig<br>Not a Sensitizer | |

information form these two cities, the results suggests that moderate arsenic exposure (<50µg/L) durign preganancy may associated with reduction in birth weight (Hopenhayn et al., 2003).

In laboratory animals, the major teratogenic effect induced by inorganic arsenic is neural tube defect, characterized by exencephaly and encephalocele. However, this effect has not been observed in humans (IPCS, 2001). In addition, data on the developmental and reproductive toxicity of inorganic arsenic submitted to the Agency show effects on offspring only at doses that are maternally toxic.

In a developmental toxicity study (Nemac, 1968b), pregnant Crl:CD-1(ICR)BR mice (25 per dose group) received a single daily gavage of aqueous Arsenic Acid (75%) from day 6 through 15 of gestation. Doses were 0, 10, 32 and 64 mg/kg/day. Controls received deionized water. Body weights were recorded at six hour periods. Cesarean section was on day 18. Fetuses were weighed, sexed and examined for external skeletal and soft tissue malformations and variations. At the high dose, two dams died. Signs included lethargy, decreased urination and defecation, soft stool or mucoid feces. Brown urogenital matting, and red material around the eyes. Necropsy showed bilateral reddening of cortico-medullary junction (kidneys) and a red areas in the stomach. At mid and (especially) top dose, the dams showed weight loss and an elevated incidence of total litter resorption. An increase in exencephaly occurred in the both the low (1/231 fetuses per 1 litter) and the high (2/146 fetuses per 1 litter) doses, but statistical significance was not seen. The Maternal Toxicity NOAEL was determined to be 32 mg/kg/day, and the Maternal toxicity LOAEL was determined to be 64 mg/kg/day, based on increased total litter resorption, reduced body weight, and increased maternal mortality. The Developmental Toxicity NOAEL was determined to be 32 mg/kg/day and the Developmental Toxicity LOAEL was determined to be 64 mg/kg/day, based on reduced mean viable fetuses, reduced fetal weights, increased post implantation loss and increased incidence of exencephaly (not statistically significant).

In a prenatal developmental toxicity study (Nemec, 1988a), artificially inseminated New Zealand White rabbits (20/dose) received aqueous arsenic acid (75%) by gavage from days 6 through 18 of gestation inclusive at doses of 0, 0.25, 1, and 4 mg/kg/ day. At the 4 mg/kg/day dose level, seven dams died or were sacrificed in extremis. Reduced body weight gain, clinical signs of toxicity (prostration, ataxia, decreased defacation and urination, mucoid feces), and histo-logical alterations in dams sacrificed or dead at the high dose (pale, soft, or mottled kidneys; pale and soft liver; dark red areas of the stomach; dark red lungs) were observed. Fetal data showed increased post-implantation loss at the 4 mg/kg/day dose (1.8 vs. 0.5 in control) and reduced mean viable fetuses (4.9 vs. 6.7 in control). There was no evidence from the data of increased incidence of fetal alterations (variations, malformations) related to treatment with test article. The Maternal NOAEL was determined to be 1 mg/kg/day, and the Maternal LOAEL was determined to be 4 mg/kg/day, based on increased mortality, decreased body weight gain, clinical signs, and histological alterations of the kidney and liver. The Developmental NOAEL was determined to be 1 mg/kg/day, and the Developmental LOAEL was determined to be 4 mg/kg/day, based on increased post-implantation loss and decreased viable fetuses.

With regard to the susceptibility of offspring to the toxicity of inorganic arsenic, DeSesso, (1998) in a review paper exploring the reproductive and developmental toxicity of arsenic acid (As+5) noted that in three repeated oral dose studies carried out under EPA guidelines for assaying developmental toxicity, arsenic acid was not teratogenic in: mice by oral gavage (10 to 64 mg/kg/day), rabbits by oral gavage (1 to 4 mg/kg/day) and in a mouse two-generation feeding study (20 to 500 ppm). Other animal developmental and reproductive toxicity data based on the published literature also showed no increased sensitivity to arsenic (+5) when given orally by repeated doses.

In a tranplacental carcinoginicity study (Waalkes et al., 2003), pregnant C3H mice were given drinking water containing sodium arsenic at 0, 42,5 and 85 ppm ad libitum from day 8 to 18 of gestation. These dosages were well tolerated and did not decrease the body weight of the dams during gestation and the birth weight of the offspring after birth. However, after weaning at 4 weeks, the offsprings were put into separate gender-base groups according to maternal exposure level. The offspring received no additional arsenic treatment. The study lasted 74 weeks in males and 90 weeks in females. A complete necropsy was performed on all mice and tissues were examined. In male, there was a dose-related increases in the incidences of heptatocellular carcinoma, liver tumor, adrenal tumor. In females offsprin, dose-related increases occurred in ovarian tumors incidence and lung carcinoma incidence were observed.(Waalkes et al., 2003)

The same authors note that "there is a paucity of human data regarding inorganic arsenic exposure during pregnancy and potential adverse effects on progeny. The available epidemiological studies were neither rigorously designed nor well controlled. These studies failed to find a definitive or consistent association between arsenic exposure and adverse pregnancy outcome. Consequently, claims of potential adverse effects of inorganic arsenic on human development remain unsubstantiated." This conclusion is consistent with ATSDR (2000a), which noted that " Although several studies have reported marginal associations between prolonged low-dose human arsenic exposure and adverse reproductive outcomes, including spontaneous abortion, stillbirth, developmental impairment, and congenital malformation, none of these studies have provided convincing evidence for such effects. "

The January 22, 2001 Federal Register Notice (Vol. 66, No. 14, pages 7027-7028), in which the arsenic drinking water standard was discussed in relation to susceptibility of certain human subpopulations including infants and children also supports the view that inorganic arsenic does not pose a special sensitivity to children. In that notice, the Agency agreed with a report by the National Research Council noting "that there is a marked variation in susceptibility to arsenic-induced toxic effects which may be influenced by factors such as genetic polymorphisms, life stage at which exposures occur, sex, nutritional status, and concurrent exposures to other agents or environmental factors." However, the view was also shared between the EPA and NRC that "there is insufficient scientific information to permit separate cancer risk estimates for potential subpopulations...and that factors that influence sensitivity to or expression of arsenic-associated cancer and non-cancer effects need to be better characterized. The EPA agrees with the NRC that there is not enough information to make risk conclusions regarding any specific

subpopulations." In the latest update to this issue (NRC, 2001), it is noted that while "evidence from human studies suggests the potential for adverse effects on several reproductive endpoints... "there are no reliable data that indicate heightened susceptibility of children to arsenic."

Neurotoxicity of inorganic arsenic is not evident in studies with experimental animals. However, there is a large body of epidemiology studies and case reports which describe neurotoxicity in humans after both acute and chronic exposures, characterized by headache, lethargy, seizures, coma, encephalopathy (after acute exposures of 2 mg/kg/day and above), and peripheral neuropathy (after repeated exposures to 0.03-0.1 mg/kg/day) (ATSDR, 2000a).

Mutagenicity studies using inorganic arsenic have shown mixed results. Sodium arsenite is not genotoxic to Chinese hamster ovary (CHO) cells (Rossman et al., 1980) or Syrian hamster embryo cells (Lee et al., 1985b) when selecting for ouabain- (ATPase) or thioguanine-resistant (hypoxanthine phosphoribosyl transferase, HPRT) mutants. In the L5178Y mouse lymphoma assay, sodium arsenite is weakly genotoxic at the thymidine kinase locus without metabolic activation (Oberly et al., 1982; Moore et al., 1997a). Sodium arsenate is even a weaker mutagen with (Oberly et al., 1982) and without metabolic activation (Moore et al., 1997a). The type of effects reported by Moore et al. (1997a) were chromosomal aberrations, micronuclei (arsenite only) polyploidy and endoreduplication.

Sodium arsenate and sodium arsenite induce sister chromatid exchanges and chromosomal aberrations in hamster embryo cells ($10^{-7}$mol/litre-$10^{-4}$mol/litre) (Larramendy et al., 1981; Lee et al., 1985b; Kochhar et al., 1996). The aberrations are characterized by chromatid gaps, breaks, and fragmentation, endoreduplication and chromosomal breaks. These clastogenic effects are observed at lower doses of arsenite than arsenate. The difference may be due to greater *in vitro* cellular uptake of arsenite than arsenate (Lerman et al., 1983; Bertolero et al., 1987). GaAs (2.5-10 µg/ml) did not induce micronuclei in Syrian hamster embryo cells (Gibson et al., 1997).

Recently, methylated trivalent forms of arsenic have been shown to nick and/or completely degrade f X174 DNA in vitro (Mass et al., 2001), while sodium arsenite, arsenate, and the pentavalent methylated forms of arsenic were without effect. In the single-cell gel assay (COMET assay)using human lymphocytes, inorganic arsenite and arsenate produced concentration-dependent linear increases in DNA damage, but the methylated trivalent forms of arsenic were observed to be 54-77 times more potent in this assay than the non-methylated forms. DNA damage occurred in the absence of metabolic activation in both assays.

### 1.1.3 Metabolism

Metabolism of inorganic arsenic first proceeds through non-enzymatic reduction of arsenate to arsenite, which can then undergo enzymatic methylation to the products monomethylarsinic acid and dimethylarsinic acid. These products are then reduced to the monomethylarsinous acid and dimethylarsinous acid produts. The major site of methylation appears to be liver, where the methylation reaction is mediated by methyltransferase enzymes using S-adenylmethionine as a

cosubstrate. The products of inorganic arsenic metabolism in urine have been identified as As(+3), As(+5), monomethylarsinous acid, and dimethylarsinous acid. Urinary products appear similar among species studied (ATSDR, 2000a), but the relative proportions of these products vary greatly.

## 1.2 Hazard Characterization - Chromium

Chromium is a naturally occurring element found in animals, plants, rocks, in soil, and in volcanic dust and gases. In the trivalent (+3) state, chromium compounds are stable and occur in nature in this state in ores such as ferrochromite. Chromium (VI) is second-most stable relative to the (+3) form, but rarely occurs naturally and is usually produced from anthropogenic sources (ATSDR, 2000b). The general population is exposed to chromium by inhalation of ambient air, ingestion of food, and drinking of water. Dermal contact with chromium can also occur from skin contact with products containing chromium or from soils containing chromium.

In humans and animals, chromium (III) is an essential nutrient that plays a role in glucose, fat, and protein metabolism. The biologically active form of chromium exists as a complex of chromium (III), nicotninc acid, and possibly the amino acids glycine, cysteine, and glutamic acid to form glucose tolerance factor. GTF is believed to function by facilitating the interaction of insulin with its cellular receptor sites although the exact mechanism is not known. The National Research Council recommends a dietary intake of 50-200 micrograms per day for chromium III.

Chromium in the ambient air occurs from natural sources, industrial and product uses, and burning of fossil fuels and wood. The most important industrial sources of chromium in the atmosphere originate from ferrochrome production. Ore refining, chemical and refractory processing, cement-producing plants, automobile brake lining and catalytic converters for automobiles, leather tanneries, and chrome pigments also contribute to the atmospheric burden of chromium (Fishbein, 1981). Chromate chemicals used as mist inhibitors in cooling towers and the mist formed during chrome plating are probably the primary sources of Cr(VI) emitted as mists in the atmosphere (Towill et al., 1978).

Surface runoff, deposition from air, and release of municipal and industrial waste waters are the sources of chromium in surface waters.

Ingested hexavalent chromium is efficiently reduced to the trivalent form in the gastrointestinal tract (DeFlora et al., 1987). In the lungs, hexavalent chromium can be reduced to the trivalent form by ascorbate and glutathione. Given the rapid reduction of Cr(VI) to Cr(III) in vivo, it is relevant to consider whether environmental exposures to Cr(VI) or administration of Cr(VI) in controlled animal experiments is essentially identical to environmental exposures to Cr(III) or administration of Cr(III) in controlled experiments. For chromium, hazard data show clearly that Cr (VI) demonstrates more significant toxicity than Cr (III). The Agency has not identified any endpoints of concern for Cr(III). For exposure to Cr(VI), the Agency has identified toxicological endpoints of concern and has used these endpoints in conjunction with exposure to Cr(VI) for

evaluating risks associated with Cr (VI).

### 1.2.1 Acute Toxicity

The acute toxicity summary of the Chromium (VI) is summarized in **Table 2**. In acute toxicity animal studies, administration of chromium (VI) (as chromic acid) by the oral, dermal, and inhalation routes resulted in significant acute toxicity as measured by lethality. The measured oral LD50 in rats was reported as 52 mg/kg, the dermal LD50 as 57 mg/kg, and the inhalation LC50 as 0.217 mg/L, placing chromium (VI) in Toxicity Category I for acute lethality. Human reports of death after ingestion of chromium show lethality at similar dose levels (ATSDR, 1998). Chromium (VI) is a significant eye and skin irritant, and severe allergic reactions consisting of redness and swelling of the skin have also been noted in exposed animals and humans. Case reports of humans who have intentionally or accidentally ingested chromium have also shown severe respiratory effects (pulmonary edema, bronchitis, bronchopneumonia), cardiovascular effects (cardiac arrest), and gastrointestinal effects (hemorrhage, ulceration).

In contrast to the acute toxicity of chromium (VI), acute toxicity data for chromium (III) show less severe acute toxicity, with oral LD50 values in rats reported as 183-200 mg/kg or 2365 mg/kg. There are no reports of lethality in experimental animals after acute inhalation or acute dermal exposure to chromium (III). However, skin irritation and sensitization have also been observed from exposure to chromium (III).

The dermal irritancy and sensitization potential of chromium compounds are worthy of note. The potent skin allergenicity of chromium has been well documented in the literature, and chromium compounds have been reported to be the most frequent sensitizing agents in man (IRIS, 2000). The prevalence of Cr(VI) sensitivity among the general U.S. population is estimated to be 0.08%, based on studies conducted by Proctor et al (1998). Most of the occurrences of contact dermatitis and sensitization cited are from the result of occupational exposures, but include the wood preserving industry (Burrows, 1983). For previously sensitized individuals, very low dosage of Cr(VI) can elicit allergic contact dermatitis. Several studies document the sensitization reactions

**Table 2**: Acute Toxicity Summary of the Chromium (VI)

| **Guideline** | **Study Type** [Substance] | **MRID/Literature** | **Results** | **Toxicity Category** |
|---|---|---|---|---|
| 81-1<br><br>(OPPTS 870.1100) | Acute Oral/Rat<br><br>[Chromic Acid, 100% a.i.] | 434294-01 | $LD_{50}$ = **?** 56 mg/kg<br>= **?** 48 mg/kg<br>M+F = 52 mg/kg | I |
| 81-2<br><br>(OPPTS 870.1200) | Acute Dermal/Rabbit<br><br>[Chromic Acid, 100% a.i.] | 434294-02 | $LD_{50}$ = **?** >48 mg/kg<br>= **?** 48 mg/kg<br><br>M+F = 57 mg/kg | I |
| 81-3<br><br>(OPPTS 870.1300) | Acute Inhalation/Rat<br><br>[Chromic Acid, 100% a.i.] | 434294-03 | $LC_{50}$ = **?** 0.263 mg/L<br>= **?** 0.167 mg/L<br>M+F = 0.217 mg/L | I |
| 81-4<br><br>(OPPTS 870.2400) | Primary Eye Irritation<br><br>[Various Cr(VI) compounds] | Literature | Waiver<br><br>Corrosive | I |
| 81-5<br><br>(OPPTS 870.2500) | Primary Dermal Irritation<br><br>[Various Cr(VI) compounds] | Literature | Waiver<br><br>Corrosive | I |
| 81-6<br><br>(OPPTS 870.2600) | Dermal Sensitization /Guinea Pig<br><br>[Various Cr(VI) compounds] | Literature | Strong sensitizer | |

observed in humans previously exposed dermally to chromium (VI) compounds. Sensitization can also be observed in humans with chromium (III) if exposure concentration is high enough (ATSDR, 2000b). Bagdon (1991) collected skin hypersensitivity data for trivalent chromium compounds in human subjects and concluded that the threshold level for evoking hypersensitivity reactions from trivalent chromium compounds is approximately 50-fold higher than for hexavalent chromium compounds.

Experimental animal models also show that sensitization to chromium compounds can occur, and in some cases, the sensitization response observed is similar using an equivalent dose of either chromium (VI) or chromium (III) (ATSDR, 2000b).

### 1.2.2 Non-Acute Toxicity

Subchronic toxicity studies in experimental animals have demonstrated hematologic and hepatic effects from repeated oral exposure to chromium (VI). In a 9 week study in which male and female Sprague-Dawley rats were fed diets containing potassium dichromate at dose levels of 0, 15, 50, 100, or 400 ppm potassium dichromate [NTP, 1996], there were no treatment related findings noted in mean body weights, water and feed consumption, organ weights or microscopic pathology of the liver, kidneys and ovaries. Hematology findings effects consisted of decreases in mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) at the high dose (8.4 and 9.8 mg/kg/day in male and female rats respectively). There were no reported hepatic effects in this study. However, Kumar and Rana (1992) reported increased accumulation of hepatic lipids after gavage treatment of rats with 13.5 mg/kg chromium (VI) (as potassium chromate) after 20 days of treatment.

In a 9-week feeding study in mice conducted by the National Toxicology Program (1996) in which mice were fed diets containing 1.1, 3.5, 7.4, and 32 mg/kg/day chromium (males) or 1.8, 5.6, 12, and 48 mg/kg/day chromium (females), hepatic cytoplasmic vacuolization was observed to be slightly increased at the high dose in males and females, and the appearance of the vacuoles was suggestive of lipid accumulation. Additional endpoints examined in this study included body weights, feed and water consumption, organ weights, microscopic evaluation of the liver, kidney and ovaries, hematology, histology of the testis and epididymis for Sertoli nuclei, and preleptotene spermatocyte counts in Stage X or XI tubules and chromatin analysis. Slight decreases in body weight were observed during this study, but there was no significant effect of treatment on clinical signs, necropsy findings, or microscopic histology. Hematologic effects were observed and consisted of a 2-4% decrease in MCV at weeks 3, 6, and 9 in high dose males and females and at week 6 in the 100 ppm females. The MCV returned to normal in the female mice after the recovery period (week 17); however the MCV increased 2.8% in the 400 ppm males.

The MCV changes at weeks 3, 6 and 9 were, in general associated with small decreases in the RBC, and small decreases in the MCH, although only the MCH values from the 400 ppm males (week 9), the 400 ppm females (Weeks 3 and 6), the 15 and 100 ppm females (week 3) were

decreased.

Occupational exposure to chromium by inhalation has been studied in the chromate manufacturing and ferrochromium industries; however, exposures all include mixed exposures to both Cr(III) and Cr(VI). The Cr(VI) species is widely considered to be the causative agent in reports of excess cancer risk in chromium workers. However, studies are inadequate to rule out a contribution by Cr(III), and Cr(VI) cannot be unequivocally demonstrated to be the causative agent for noncarcinogenic effects following inhalation.

A number of epidemiologic studies have considered the association between inhalation of chromium and noncarcinogenic endpoints, including upper respiratory irritation and atrophy, lower respiratory effects, and systemic effects. Symptoms reported from inhalation exposure to mists and dusts containing chromium have included nasal tissue damage, perforated septum, ulcerated septum, chrome holes, nosebleed, inflamed mucosa, nasal septal perforation, and nasal septal ulceration (USEPA IRIS, 1998). Exposure to vapors of chromium salts has also been suspected as a cause of asthma, coughing, wheezing, and other respiratory distress in ferrochromium workers.

Despite the consistency of the reported effects from inhalation of chromium contained in dusts and mists, the actual Cr(III) and Cr(VI) exposure levels in many of the studies attributing respiratory effects to chromium were unknown. In addition, data on other confounding factors such as smoking were frequently unavailable. These caveats significantly complicate determination of the potential health effects associated with inhalation exposure to chromium (ATSDR, 2000b).

Although human data examining developmental endpoints are scarce, animal studies have consistently shown that chromium, particularly chromium(VI), is a developmental toxicant. Oral ingestion of chromium (VI) compounds in experimental animals results in significant developmental toxicity. Studies describing the effects observed have been published in the IRIS Toxicological Reviews for both chromium (VI) and chromium (III) as well as from submitted studies to the Agency and are summarized here.

Trivedi et al. (1989) exposed mice to 250, 500, and 1,000 ppm potassium dichromate daily through drinking water during the entire gestational period. The authors reported decreased fetal weight, increased resorptions, and increased abnormalities (tail kinking, delayed ossification of the cranium) in exposed mice. The medium- and high-dose groups registered significant reductions in body weight gain when compared to controls. The most significant finding of the study was the complete absence of uterine implantation in the high-dose group. The 250 and 500 ppm dose groups also showed significant incidences of resorption as compared to controls. The authors observed significant increases in preimplantation and postimplantation losses and dose-dependent reductions in total weight and crown-rump length in the lower dose groups. Additional effects included treatment-related increases in abnormalities in the tail, wrist forelimbs and subdermal hemorrhagic patches in the offspring.

Junaid et al. (1996) exposed female Swiss albino mice to 250, 500, or 750 ppm potassium dichromate in drinking water to determine the potential embryotoxicity of hexavalent chromium during days 6-14 of gestation. No notable changes in behavior or clinical signs were observed in the control or treated dams. Chromium levels in blood, placenta, and fetus increased in a dose-dependent fashion over the course of the study. The authors reported retarded fetal development and embryo- and fetotoxic effects including reduced fetal weight, reduced number of fetuses (live and dead) per dam, and higher incidences of stillbirths and postimplantation loss in the 500 and 750 ppm dosed mothers. Significantly reduced ossification in nasal, frontal, parietal, interparietal, caudal, and tarsal bones was observed in the high-dose group, while reduced ossification in only the caudal bones was observed in the 500 ppm dose group. Based on the body weight of the animals (30 +/- 5 g) and the drinking water ingested by the animals in the 250 ppm dose group (8.0 ml/mouse/day), the dose level in the 250 ppm group can be identified as 67 mg/kg-day. The maternal NOAEL was 63 [22.3] mg/kg/day while the LOAEL was 42.1 mg/kg/day and was based on a decreased gestational body weight. At the lowest dose tested, the incidence of resorptions was increased and a developmental NOAEL was, therefore, not determined.

Kanojia et al. (1996) exposed female Swiss albino rats to 250, 500, or 750 ppm potassium dichromate in drinking water for 20 days 3 months prior to gestation to determine the potential teratogenicity of hexavalent chromium. No notable changes in behavior or clinical signs were observed in the control or treated dams. Chromium levels in blood, placenta, and fetus were significantly increased in the dams of the 500 and 750 ppm dose groups. The authors reported a reduced number of corpora lutea and implantations, retarded fetal development, and embryo- and fetotoxic effects including reduced number of fetuses (live and dead) per dam and higher incidences of stillbirths and postimplantation loss in the 500 and 750 ppm dosed mothers. Significantly reduced parietal and interparietal ossification was observed in the high-dose group. Based on the body weight of the animals (175 +/- 25 g) and the drinking water ingested by the animals in the 250 ppm dose group (26 ml/mouse/day) the dose level in the 250 ppm group can be identified as 37 mg/kg-day.

Tyl (1991) examined the developmental and maternal effects of daily administration of chromic acid (55.0% a.i.) at dosages of 0, 0.1, 0.5, 2.0 or 5.0 mg/kg/day by gavage in rabbits. Clinical signs of toxicity , including diarrhea, and slow, audible or labored breathing were observed in predominately in the 2.0 and 5.0 mg/kg/day groups. However, these signs did not show a dose-response and were observed in lesser incidence at 5.0 mg/kg/day vs. 2.0 mg/kg/day. However, the incidence of mortality (at 2.0 mg/kg/day, one doe died on gestation day (GD) 28; at 5.0 mg/kg/day, 5 does died (one each on GD 10, 14, and two on GD 15) and the magnitude of decreased body weight gain during the dosing period (average weight loss of 48 grams at 2.0 mg/kg/day, and average weight loss of 140 grams at 5.0 mg/kg/day during gestation days 7-19) were observed to occur in a dose-related fashion at 2.0 and 5.0 mg/kg/day. Food efficiency was also observed to be significantly lower during the dosing period in the 5.0 mg/kg/day dose group. Cesarean section observations were unremarkable in this study at any dose level. No treatment related effects on either fetal malformations or variations were observed.

The Maternal NOAEL = 0.5 [0.12] mg/kg/day and LOAEL = 2.0 [0.48] mg/kg/day (based on the increased incidence of maternal mortality and decreased body weight gain ). The Developmental NOAEL = 2.0 [0.48] mg/kg/day and LOAEL > 2.0 [>0.48] mg/kg/day based on the lack of developmental effects at any dose level tested.

By contrast to effects of chromium (VI), effects on development and reproduction from exposure to Cr (III) show either negative results or effects only at high doses. For example, male and female rats treated with 1,806 mg Cr(III) kg/day as Cr(III) oxide 5 days/week for 60 days before gestation and throughout the gestation period had normal fertility, gestational length, and litter size (Ivankovic and Preussman, 1975). Elbetieha and Al-Hamood (1997) examined fertility following chromium chloride exposures in mice. Sexually mature male and female mice were exposed to 1,000, 2,000, or 5,000 mg/L chromium chloride in drinking water for 12 weeks. Exposure of male mice to 5,000 ppm trivalent chromium compounds for 12 weeks had adverse impacts on male fertility. Testes weights were increased in the males exposed in the 2,000 and 5,000 mg/L dose groups, while seminal vesicle and preputial gland weights were reduced in the 5,000 mg/L exposed males. The number of implantation sites and viable fetuses were significantly reduced in females exposed to 2,000 and 5,000 mg/L chromium chloride. Water consumption was not reported precluding calculation of the doses received. However it is evident that adverse effects were observed only at a high dose of Cr (III).

The National Toxicology Program recently conducted a three-part study to investigate oral ingestion of hexavalent chromium in experimental animals (NTP, 1996a,b, 1997). The study included a determination of the potential reproductive toxicity of potassium dichromate in Sprague-Dawley rats, a repeat of the study of Zahid et al. (1990) using BALB/C mice, and a Reproductive Assessment by Continuous Breeding study in BALB/C mice. The study in the Sprague-Dawley rat (NTP, 1996a) was conducted in order to generate data in a species commonly used for regulatory studies. Groups of 24 males and 48 females were exposed to 0, 15, 50, 100, or 400 ppm potassium dichromate daily in the diet for 9 weeks followed by a recovery period of 8 weeks. Six male and 12 female rats were sacrificed after 3, 6, or 9 full weeks of treatment or after the full recovery period. Animals were examined for body weights; feed and water consumption; organ weights; microscopic evaluation of the liver, kidney, and ovaries; hematology; histology of the testis and epididymus for Sertoli nuclei and preleptotene spermatocyte counts in Stage X or XI tubules; and chromatin analysis. No treatment-related hematology findings were reported except for slight decreases in MCV and MCH values in the male and female treatment groups receiving 400 ppm potassium dichromate (24 mg/kg-day). While the trends in MCV and MCH were not large and were within the reference ranges, they are consistent with the findings of the companion studies in BALB/C mice and were characterized by the authors as suggestive of a potential bone marrow/erythroid response. The authors considered the 100 ppm (6 mg/kg-day) dose group to be representative of the NOAEL for the study.

The reproductive study in BALB/C mice (NTP, 1996b) was conducted to reproduce the conditions utilized by Zahid et al. (1990) in their examination of comparative effects of trivalent

and hexavalent chromium on spermatogenesis of the mouse. Groups of 24 male and 48 female BALB/C mice were exposed to 0, 15, 50, 100, or 400 ppm potassium dichromate in the diet for 9 weeks followed by a recovery period of 8 weeks. Six male and 12 female mice were sacrificed after 3, 6, or 9 full weeks of treatment or after the full recovery period. Animals were examined for body weights; feed and water consumption; organ weights; microscopic evaluation of the liver, kidney, and ovaries; hematology; histology of the testis and epididymus for Sertoli nuclei and preleptotene spermatocyte counts in Stage X or XI tubules; and chromatin analysis. Treatment-related effects included a slight reduction in the mean body weights in the 400 ppm males and the 100 ppm females, a slight increase in food consumption at all dose levels, a slight decrease in MCV and MCH at 400 ppm, and cytoplasmic vacuolization of the hepatocyte at 50, 100 and 400 ppm. None of the effects on spermatogenesis reported by Zahid et al. (1990) were observed in this study. On the basis of the cytoplasmic vacuolization of the hepatocyte in the 50, 100, and 400 ppm dose groups, the authors selected 15 ppm (4 mg/kg-day) as the NOAEL.

Increased resorptions and increased post-implantation loss as well as gross fetal abnormalities were observed in offspring of pregnant mice exposed to potassium dichromate at 57 mg/kg/day in drinking water during gestation (ATSDR, 2000b). At a higher dose of 234 mg/kg/day, no implantations were observed in maternal mice. In a second study in mice, potassium dichromate was administered in the diet for 7 weeks at dose levels of 15.1 and 28 mg/kg/day. Reduced sperm counts and degeneration of the outer layer of the seminiferous tubules was observed at the 15.1 mg/kg/day dose, and morphologically altered sperm was observed at the 28 mg/kg/day dose.

In male rats administered 20 mg/kg/day chromium trioxide for 90 days by gavage, reduced testicular weight, decreased testicular testosterone, and reduced Leydig cell number was observed (Chowdhury and Mitra, 1995).

Despite the wealth of animal studies on the developmental and reproductive toxicity of chromium VI, there are too few human data with which to make any reliable conclusion regarding the susceptibility of the developing fetus, infants, or children to the toxic effects of chromium VI. The evidence available suggests similar toxic effects in adults and children from ingestion of chromium VI (ATSDR, 2000b).

Hexavalent chromium (Cr VI) is known to be carcinogenic in humans by the inhalation route of exposure. Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both Cr(III) and Cr(VI) compounds. Because only Cr(VI) has been found to be carcinogenic in animal studies, however, it was concluded that only Cr(VI) should be classified as a human carcinogen.

Animal data are consistent with the human carcinogenicity data on hexavalent chromiumby the inhalation route. Hexavalent chromium compounds are also carcinogenic in animal bioassays by other routes of exposure, such as: intramuscular injection site tumors in rats and mice,

intrapleural implant site tumors for various Cr(VI) compounds in rats, intrabronchial implantation site tumors for various Cr(VI) compounds in rats, and subcutaneous injection site sarcomas in rats (IRIS, 2001). However, these routes of administration are not relevant to exposures of chromium in CCA-treated wood.

Data addressing human carcinogenicity from exposures to Cr(III) alone are not available, and data are inadequate for an evaluation of human carcinogenic potential. Two oral studies located in the available literature (Schroeder et al., 1965; Ivankovic and Preussman, 1975) reported negative results for rats and mice. Several animal studies have been performed to assess the carcinogenic potential of Cr(III) by inhalation. These studies have not found an increased incidence of lung tumors following exposure either by natural routes, intrapleural injection, or intrabronchial implantation (Baetjer et al., 1959; Hueper and Payne, 1962; Levy and Venitt, 1975; Levy and Martin, 1983).

The data from oral and inhalation exposures of animals to trivalent chromium do not support determination of the carcinogenicity of trivalent chromium. IARC (1990) concluded that animal data are inadequate for the evaluation of the carcinogenicity of Cr(III) compounds. Furthermore, although there is sufficient evidence of respiratory carcinogenicity associated with exposure to chromium, the relative contributions of Cr(III), Cr(VI), metallic chromium, or soluble versus insoluble chromium to carcinogenicity cannot be elucidated.

In vitro data are suggestive of a potential mode of action for hexavalent chromium carcinogenesis. Hexavalent chromium carcinogenesis may result from the formation of mutagenic oxidatitive DNA lesions following intracellular reduction to the trivalent form. Cr(VI) readily passes through cell membranes and is rapidly reduced intracellularly to generate reactive Cr(V) and Cr(IV) intermediates a reactive oxygen species. A number of potentially mutagenic DNA lesions are formed during the reduction of Cr(VI). Hexavalent chromium is mutagenic in bacterial assays, yeasts, and V79 cells, and Cr(VI) compounds decrease the fidelity of DNA synthesis in vitro and produce unscheduled DNA synthesis as a consequence of DNA damage. Chromate has been shown to transform both primary cells and cell lines (ATSDR, 2000b).

Intracellular reduction of Cr(VI) generates reactive chromium V and chromium IV intermediates as well as hydroxyl free radicals (OH) and singlet oxygen. A variety of DNA lesions are generated during the reduction of Cr(VI) to Cr(III), including DNA strand breaks, alkali-labile sites, DNA-protein and DNA-DNA crosslinks, and oxidative DNA damage, such as 8-oxo-deoxyguanosine. The relative importance of the different chromium complexes and oxidative DNA damage in the toxicity of Cr(VI) is unknown.

Hexavalent chromium has been shown to be genotoxic only in the presence of appropriate reducing agents in vitro or in viable cell systems in vitro or in vivo. Hexavalent chromium has been shown to be mutagenic in bacterial systems in the absence of a mammalian activating system, and not mutagenic when a mammalian activating system is present. Hexavalent chromium is also mutagenic in eukaryotic test systems and clastogenic in cultured mammalian cells.

Hexavalent chromium in the presence of glutathione has been demonstrated to produce genotoxic DNA adducts that inhibit DNA replication and are mutagenic (IRIS, 2000). Chromium (III) has also produced positive mutagenic responses in vitro (IRIS, 2000).

### 1.2.3 Metabolism

Absorption of chromium by the oral route ranges from essentially zero for the insoluble chromium III compound chromic oxide to 10% for potassium chromate. Absorption through exposure in the diet, in water, or from contaminated soil is consistently low, with values reported in the range of 1-5% (ATSDR, 2000b; USEPA, 1998). Hexavalent chromium can be reduced to the trivalent form in the epithelial lining fluid of the lungs by ascorbate and glutathione as well as by gastric juice in the stomach, which contributes to the low oral absorption. Absorption by the dermal route is also low (1.3% after 24 hours as reported by Bagdon et al., 1991)

Once absorbed, chromium compounds are distributed to all organs of the body without any preferential distribution to any one organ. However, exposures to higher levels of chromium, such as can occur in the chrome plating industry and chrome refining plants, may result in accumulation of chromium in tissues. Witmer et al. (1989, 1991) studied chromium distribution in tissues of rats administered chromium via gavage. In one experiment, the highest dose of sodium chromate [5.8 mg Cr(VI)/kg/day for 7 days] resulted in concentrations of chromium in the tissues in the following order: liver (22 ?g chromium/whole organ) > kidney (7.5 ?g) > lung (4.5 ?g) > blood (2 ?g) > spleen (1 ?g). These tissues combined retained about 1.7% of the administered dose; however, some tissues were not analyzed. At the two lower doses administered (1.2 or 2.3 mg/kg/day), very little chromium was detected (<0.5 µg/organ) in the organs analyzed.

Maruyama (1982) studied the chromium content in major organs of mice exposed to potassium dichromate [Cr(VI)] or chromium trichloride ([Cr(III)] for 1 year in drinking water. Groups of mice received 4.4, 5.0 or 14.2 mg Cr(VI)/kg/day or 4.8, 6.1 or 12.3 mg Cr(III)/kg/day. Examination of organs and blood in mice that received Cr(VI) revealed that the liver and spleen had the highest levels of chromium, although some chromium accumulation was observed in all tissues. In mice that received Cr(III), the liver was the only organ with detectable amounts of chromium, and at levels that were about 40-90 times less than in mice that received the Cr(VI) compound. MacKenzie et al. (1958) reported that in rats following the administration of similar concentrations of Cr(VI) as potassium chromate or Cr(III) as chromium trichloride in drinking water for 1 year, tissue levels were approximately 9 times greater in rats that received the Cr(VI) compound, compared to rats that received the Cr(III) compound.

If hexavalent chromium is absorbed, it can readily enter red blood cells through facilitated diffusion, where it will be reduced to the trivalent form by glutathione. During reduction to the trivalent form, chromium may interact with cellular macromolecules, including DNA (Wiegand et al., 1985), or may be slowly released from the cell (Bishop and Surgenor, 1964). Chromium III can be cleared rapidly from the blood but more slowly from tissues, which may be related to the formation of trivalent chromium complexes with proteins or amino acids (Bryson and Goodall,

1983).

The liver is a primary site of chromium metabolism and has been studied in animals. Incubation of Cr(VI) with rat liver microsomes in the presence of the enzyme cofactor nicotinamide adenine dinucleotide phosphate (NADPH) resulted in the reduction of Cr(VI) to Cr(III) (ATSDR, 2000b). Exclusion of the co-factors necessary for the production of NADPH resulted in a large decrease in the reduction of Cr(VI) to Cr (III).

Chromium metabolism can result in the formation of species that interact with deoxyribonucleic acid (DNA). The reduction of Cr(VI) to a Cr(V) intermediate involves a single electron transfer from the microsomal electron-transport cytochrome P-450 system (Jennette 1982). These reactive Cr(V) complexes/ intermediates are relatively unstable and persist for approxim-ately 1 hour *in vitro*. During this time the Cr(V) complexes/ intermediates can interact with deoxyribonucleic acid (DNA), which may eventually lead to cancer. When Cr(VI) interacts with glutathione, Cr(V) complexes and glutathione thonyl radicals were produced, and when Cr(VI) interacts with DNA and glutathione, DNA adducts were formed (Aiyar et al. 1989). The formation of Cr(V) was found to correlate with DNA adduct formation. Following reactions of Cr(VI) with hydrogen peroxide, hydroxyl radicals were produced; the addition of DNA resulted in the formation of an 8-hydroxy guanine adduct and DNA strand breakage.

The elimination of chromium after oral exposure has been studied in both humans and animals. In one study, human volunteers received an acute oral dose of radiolabeled Cr(III) or Cr(VI) (Donaldson and Barreras 1966). Fecal samples were collected for 24 hours, and urine samples were collected for 6 days and analyzed for chromium. Approximately 99.6% of the Cr(III) compound was recovered in the 6-day fecal sample, while 89.4% of the Cr(VI) compound was recovered. The results of the analysis of the 24-hour urine samples indicated that 0.5% and 2.1% of the administered dose of the Cr(III) and the Cr(VI) compounds, respectively, were recovered in the urine. Other potential routes of excretion include hair, fingernails and breast milk (ATSDR 2000b).

In several studies in which rats and hamsters were fed Cr(VI) compounds, fecal excretion of chromium varied slightly from 97% to 99% of the administered dose, and urinary excretion of chromium, administered as Cr(III) or Cr(VI) compounds, varied from 0.6% to 1.4% of the dose (Donaldson and Barreras 1966, Henderson et al. 1979, Sayato et al. 1980). Following the gavage administration of 13.92 mg chromium/kg/day as calcium chromate for 8 days, the total urinary and fecal excretion of chromium on days 1 and 2 of dosing were <0.5% and 1.8%, respectively (Witmer et al. 1991). The total urinary and fecal excretion of chromium on days 7 and 8 of dosing were 0.21% and 12.35%, respectively. Donaldson et al. (1984), reported that excretion of Cr(III) and creatinine clearance were almost equal suggesting that tubular absorption or reabsorption of chromium in the kidneys was minimal.

## 2.0 DOSE-RESPONSE ASSESSMENT

The process of dose-response assessment as part of a total risk assessment involves describing the quantitative relationship between the exposure to a chemical and the extent of toxic injury or disease. Following the process of hazard identification, in which the available toxicology data is reviewed and selection of NOAELs and LOAELs is made for each study, the reviewed data for a pesticide chemical is presented to a committee of scientists within the Office of Pesticide Programs who reach concurrence on toxicology endpoints that best represent the toxic effects expected from various routes of exposure and durations of exposure. For most pesticide chemicals, the process results in selection of acute and chronic Reference Dose values (which can be used as benchmark values for acute and chronic dietary risk calculations), as well as endpoint values for non-dietary risk assessments involving occupational and/or residential exposures by the oral, dermal, and inhalation routes. Endpoints are selected for non-dietary exposures to represent short-term (1-30 days), intermediate-term (30-180 days), and long-term exposure scenarios, as needed. In addition, incidental oral exposure endpoints are selected for short-term and intermediate term exposure durations to represent ingestion of pesticide chemical residues that may occur from hand-to-mouth behaviors. In general, toxicity endpoint selection should, to the extent possible, match the temporal and spatial characteristics of the exposure scenarios selected for use in the risk assessment. These endpoints are then used in conjunction with exposure values to calculate risks associated with various types of exposure, depending upon the uses of the pesticide chemical.

Toxicology endpoints for both inorganic arsenic and chromium have been selected for the residential exposure assessment and are presented below:

## 2.1 Inorganic Arsenic-Endpoint Selection

On August 21, 2001, the OPP's Hazard Identification Assessment Review Committee (HIARC) evaluated the toxicology data base of **Inorganic Arsenic** and established the toxicological endpoints for occupational exposure risk assessments. On October, 23-25 2001, the FIFRA Scientific Advisory Panel (SAP) met and discussed some issues about the end points proposed by the HIARC. The inorganic arsenic toxicological end-points selected for CCA occupational risk assessment are summarized in **Table 4**.

### 2.1.1 Acute Reference Dose (aRfD)

In the Office of Pesticide Program (OPP) in EPA, the acute reference dose (aRfD) was used in the risk assessment associated with oral exposure to food related chemicals. Inorganic arsenic is not registered for any food uses and there are no existing tolerances. For inorganic arsenic as contained within CCA-treated wood, therefore, an acute RfD is not relevant to the exposures from registered use.

### 2.1.2 Chronic Reference Dose (cRfD)

The U.S. EPA has published a chronic RfD value for inorganic arsenic (USEPA IRIS, 1998). However, as with the acute RfD, in OPP, the chronic RfD in OPP was considered for evaluating risks associated with food and/or drinking water related chemical uses. Because there are no exposure scenarios relevant to the currently registered uses of inorganic arsenic, and specifically the registered uses in CCA-treated lumber. No chronic RfD value is need for the current inorganic arsenic use in CCA-treated wood use. However, if the Agency determines in the future that an aggregate assessment is needed for calculation of risk from exposure to arsenic in treated lumber and exposure in drinking water and/or food, the chronic RfD value can be utilized.

### 2.1.3 Short (1-30 days ) and Intermediate (30-180 days) Incidental Oral Exposure

Based on the registered use of CCA-treated lumber for fencing and decking materials in residential settings, incidental oral exposure is expected, based on potential ingestion of soil contaminated with arsenic as a result of leaching from wood, and from ingestion of arsenic residues from the palm as a result of direct dermal contact with treated wood. The studies selected for short- and intermediate-term incidental oral exposure are the human case reports of Franzblau and Lilis (Arch. of Envir. Health 44(6): 385-390, 1989) and Mizuta et al. (Bull. Yamaguchi Med. Sch. 4(2-3): 131-149, 1956). The LOAEL of 0.05 mg/kg/day was selected, based on facial edema, gastrointestinal symptoms, neuropathy, and skin lesions observed at this dose level

Franzblau et al., (1989) reported 2 cases of subchronic (2 months) arsenic intoxication resulting from ingestion of contaminated well water (9-10.9 mg/L) sporadically (once or twice a week) for about 2 months. Acute gastrointestinal symptoms, central and peripheral neuropathy, bone

marrow suppression, hepatic toxicity and mild mucous membrane and cutaneous changes were presented. The calculated dose was 0.03 - 0.08 mg/kg/day based on a body weight of 65 Kg and ingestion of from 238 to 475 ml water/day.

Mizuta et al. (1956) reported a poisoning incident involving the presence of arsenic [probably calcium arsenate] contained in soy-sauce. The duration of exposure was 2-3 weeks. The arsenic content was estimated at 0.1 mg/ml. Out of 417 patients, the authors reported on 220 (age not specified for all patients. The age of the 46 paints with age information are ranging from 15 - 69). An early feature of the poisoning was appearance of facial edema that was most marked on the eyelids. Other symptoms presented included multifaceted gastrointestinal symptoms, liver enlargement, upper respiratory symptoms, peripheral neuropathy and skin disorders. In the majority of the patients, the symptoms appeared within two days of ingestion and then declined even with continued exposure. There was evidence of minor gastrointestinal bleeding (occult blood in gastric and duodenal juice). There were abnormalities in electrocardiograms (altered Q-T intervals and P and T waves). These changes were not evident on reexamination after recovery from the clinical symptoms. An abnormal patellar reflex was evident in >50% of the cases. This effect did not return to normal during the course of the investigation.

Based on the consumption of the arsenic in the contaminated soy-sauce, the pattern of soy-sauce consumption and on measured urinary arsenic levels, the authors estimated consumption of arsenic at 3 mg/day. Although the body weight was not reported, the EPA assumes an average body weight of 55 kg in the Asian population. The estimated exposure was, therefore, 0.05 mg/kg/day and was considered the LOAEL. **The LOAEL= 0.05 mg/kg/day (edema of the face; gastrointestinal, upper respiratory, skin, peripheral and neuropathy symptoms).**

These two case reports are appropriate for both short- and intermediate-term incidental oral endpoints for the following reasons:

1) Symptoms reported in the Mizuta study (gastrointestinal disorders, neuropathy, liver toxicity) occurred after 2-3 weeks of exposure, making this endpoint appropriate for the short-term (1-30 days) exposure period. This study also examined toxicity by the relevant route of exposure (oral).

2) Similar symptoms were observed in the Franzblau study, and are appropriate for the intermediate-term endpoint as they were observed to occur after longer-term (2 months) exposure.

A Margin of Exposure (MOE) of 30 applied to the LOAEL was suggested the majority of the SAP panel members in the 2001 meeting.

USEPA Region 8 has also published a report on selection of acute and chronic Reference Doses for Inorganic Arsenic, intended to apply to exposures of 1-14 days and 15 days-7 years (USEPA Region 8, 2001). The use of the term “reference dose” in the Region 8 report “apply to readily

soluble forms of arsenic and are intended to include total oral exposure to inorganic arsenic, that is drinking water, food, and soil. " The report concludes that a NOAEL value of 0.015 mg/kg/day from a study by Mazumder et al (Int. J. Epidem. 27: 871-877) can be used for acute and subchronic reference dose values, with an uncertainty factor of 1. Alternately, the LOAEL of 0.05 mg/kg/day and an uncertainty factor of 3 (for extrapolation from the LOAEL to the NOAEL) could be selected from this same study. A full factor of 10 was not employed by Region 8 based on the reasoning that a No Adverse Effect Level "is likely at an exposure only slightly below the effect level" (USEPA Region 8, 2001). However, this report did not discuss severity or irreversibility of effects observed in the Mizuta et al. report as a factor in selecting the uncertainty factor, which was taken into consideration by the OPP HIARC. Further, the effect observed in the Mazumder et al. study of hyperkeratosis is a result of chronic exposure and not short- or intermediate-term exposure and was thus felt to be inappropriate for determination of short- and intermediate-term incidental oral risk. The Region 8 report was part of the background documents presented to the 2001 SAP.

For the risk assessment, based on the recommendations of the SAP, the Agency decided to use a Margin of Exposure (MOE) of 30. This value of 30 was recommended on the basis that the severity of symptoms near or moderately above the LOAEL (0.05mg/kg/day) warranted a full uncertainty factor of 10 and an uncertainty factor of 3 for protection of children.

### 2.1.4 Dermal Absorption

Dermal absorption of inorganic arsenic is represented by the study of Wester et al. (Fund. Appl. Toxicol. 20: 336-340, 1993). In this study, the percutaneous absorption of arsenic acid ($H_3AsO_4$) from water and soil both *in vivo* using rhesus monkeys and *in vitro* with human skin was examined. *In vivo*, absorption of arsenic acid from water (loading 5 µl/cm$^2$ skin area) was 6.4 ± 3.9% at the low dose (0.024 ng/cm$^2$) and 2.0 ± 1.2% at the high dose (2.1 µg/cm$^2$). Absorption from soil (loading 0.04 g soil/cm$^2$ skin area) *in vivo* was 4.5 ± 3.2% at the low dose (0. 04 ng/cm$^2$) and 3.2 ± 1.9% at the high dose (0.6 µg/cm$^2$). Thus, *in vivo* in the rhesus monkey, percutaneous absorption of arsenic acid is low from either soil or water vehicles and does not differ appreciably at doses more than 10,000-fold apart. Wester et al. (1993) also reported that for human skin, at the low dose, 1.9% was absorbed from water and 0.8% from soil over a 24-h period.

The value of 6.4% dermal absorption was chosen based on the use of non-human primates for derivation of this value and the fact that this was a well-conducted study. It is observed in this study that a higher dose on the skin resulted in lower dermal absorption as noted above, but the data in this and other studies suggests sufficient variability in the absorption such that use of the 6.4% dermal absorption value is sufficiently but not overly conservative. For children playing around playground equipment, however, it is assumed the dermal exposure would be arsenic in wood surface residue and/or arsenic in soil, a dermal absorption value of 3% will be used (SAP, 2001).

### 2.1.5 Short (1-30 days ) and Intermediate (30-180 days) Dermal Exposure

Since there is no appropriate dermal studies, same as studies selected for short- and intermediate-term incidental oral exposure, the case reports of Franzblau and Lilis (Arch. of Envir. Health 44(6): 385-390, 1989) and Mizuta et al. (Bull. Yamaguchi Med. Sch. 4(2-3): 131-149, 1956) were selceted for short (1-30 days ) and intermediate (30-180 days) term dermal exposure scenarios. The LOAEL of 0.05 mg/kg/day was selected, based on facial edema, gastrointestinal symptoms, neuropathy, and skin lesions observed at this dose level. An Margin of Exposure (MOE) of 30 should be applied to the LOAEL. This value consists of a 10x factor for intraspecies variation and a 3x factor for extrapolating from a LOAEL to a NOAEL.

### 2.1.6 Long-Term Dermal Exposure

While no long-term dermal exposures are expected from residential exposure to arsenic in CCA-treated lumber, long-term dermal exposure is expected in the occupational setting. Thus, for this exposure scenario, the dose and endpoint selected are the NOAEL of 0.0008 mg/kg/day from the Tseng et al. (1968) study, which examined chronic non -cancer and cancer effects from arsenic exposure through well water in a large cohort in Taiwan.

In Taiwan, Tseng, (1977), Tseng, (1968) [U.S. EPA, 1998] noted that hyperpigmentation, keratosis and possible vascular complications were seen at the LOAEL of 0.17 mg/L, converted to 0.014 mg/kg/day.

The NOAEL was based on the arithmetic mean of 0.009 mg/L in a range of arsenic concentration of 0.001 to 0.017 mg/L. The NOAEL also included estimation of arsenic from food. Since oral arsenic exposure data were missing, arsenic concentrations in sweet potatoes and rice were estimated as 0.002 mg/day. Other assumptions included consumption of 4.5 L water/day and 55 kg body weight (Abernathy, (1989). Thus, the converted NOAEL = [(0.009 mg/L x 4.5 L/day) + 0.002 mg/day]/55 kg = **0.0008 mg/kg/day**. The LOAEL dose was estimated using the same assumptions as the NOAEL starting with an arithmetic mean water concentration from Tseng, (1977) of 0.17 mg/L. LOAEL = [(0.17 mg/L x 4.5 L/day)+ 0.002 mg/day]/55 kg = **0.014 mg/kg/day**. Therefore the NOAEL = 0.0008 mg/kg and the LOAEL= 0.014 mg/kg/day (based on hyperpigmentation, keratosis and possible vascular complications )

An MOE of 3 is applied to this risk assessment. A factor of 3 and not 10 is used based on the large sample size of the Tseng study (> 40,000) and is in agreement with the published value and rationale in the 1998 IRIS document on inorganic arsenic.

### 2.1.7 Short-, Intermediate-, and Long-term Inhalation Exposure

Short-, intermediate-, and long-term endpoints were not identified in the HIARC report for inhalation exposures to arsenic. Since no inhalation studies are available, committee selected the same studies as for the dermal risk assessments. Since the dose identified for inhalation risk assessments are from oral studies, route-to-route extrapolation should be as follows:

Step I: The inhalation exposure component (i.e., g a.i./day) using a 100% (default) absorption rate and application rate should be converted to an equivalent oral dose (mg/kg/day);

Step II: The dermal exposure component (i.e., mg/kg/day) using 6.4 % absorption factor and application rate should be converted to an equivalent oral dose. The dose should be combined with the converted oral dose in Step I.

Step III: To calculate the MOE's, the combined dose from Step I and II should then be compared to the oral LOAEL of 0.05 mg/kg/day for short and intermediate term exposure and the oral NOAEL of 0.0008 mg/kg/day for long-term exposure.

As discussed in endpoints selected for dermal exposure scenarios (Sections 2.1.5 and 2.1.6), acceptable Margin of Exposure (MOE) of 30 should be applied to the short- and intermediate inhalation scenarios. For long-term inhalation exposure scenarios, an acceptable margin of exposure of 3 should be applied.

### 2.1.8 Carcinogenicity

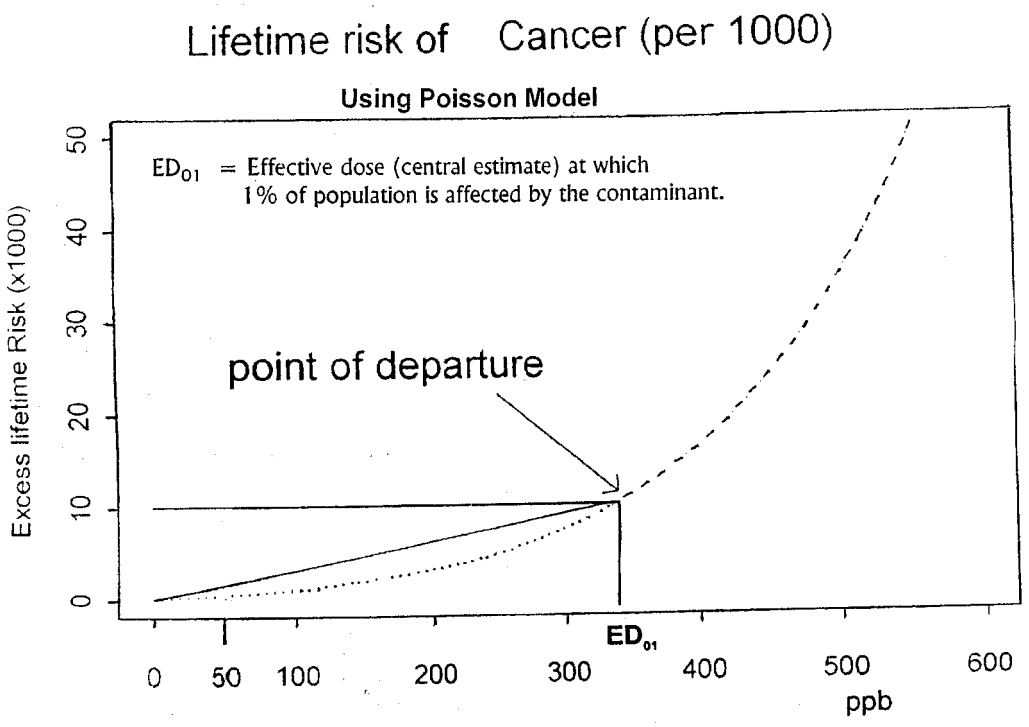
There is sufficient evidence from human data indicating arsenic exposure can cause cancer. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and increased incidences of skin cancer were observed in populations consuming drinking water high in inorganic arsenic. In order to evaluate the cancer risk associated with arsenic exposure in drinking water, in 1997, at EPA's request, the National Academy of Sciences' (NAS) Subcommittee on Arsenic of the Committee on Toxicology of the National Research Council (NRC) met. Their charge was to review EPA's assessments of arsenic. The NAS/NRC Subcommittee finished their work in March 1999. In general, the NRC report confirms and extends concerns about human carcinogenicity of drinking water containing arsenic and offers perspective on dose-response issues and needed research. The NRC recommended that EPA analyze risks of internal cancers both separately and combined.

EPA applied many of the recommendations from the 1999 NRC report in the risk characterization used to support the January 2001 revised arsenic drinking water regulation. In the risk assessment, EPA used risk estimates taken from Morales et al. (2000). Morales et al. fit a variety of dose-response models to lung and bladder cancer data from an arseniasis-endemic region of southwestern Taiwan. Risk was assumed to increase linearly with dose, from zero to the effective dose (central estimate) at which 1% of population is affected by the chemical (ED01). The slope of the line extrapolated from ED01 to the origin was calculated and used as the cancer slope factor for cancer risk assessment (see Plot 1 as an example). In the risk assessment associated with inorganic arsenic in drinking water in 2000 (EPA, 2001), EPA presented two sets of risk estimates, higher and lower:

- **For the higher set of risks:** For the higher set of risks: EPA used the theoretical risk estimates taken directly from Morales et al. (2000). Assumed drinking water consumption in Taiwanese population is 3.5 L/day for male and 2.0 L/day for female.

- **For the lower set of risks:** For the lower set of risks: EPA adjusted the theoretical risks to take into account possible higher arsenic consumption in Taiwan. For these estimates, EPA assumed that people in Taiwan consumed an additional 1 L/d of water in cooking, due to dehydration of rice and sweet potatoes, and a further 50 µg/d of arsenic directly from their food.

Following the risk assessment associated with inorganic in drinking water are presented in 2000, EPA asked the National Research Council (NRC) to meet again to: (1) review EPA's characterization of potential human health risks from ingestion of inorganic arsenic in drinking water;(2) review the available data on the carcinogenic and non-carcinogenic effects of inorganic arsenic; (3) review the data on the metabolism, kinetics and mechanism(s)/mode(s) of action of inorganic arsenic; and (4) identify research needs to fill data gaps.




PLOT 1: Example of how the cancer risk estimations are derived

In 2001, NRC published an update to the 1999 NRC report and concluded that (1) arsenic- -induced bladder and lung cancers still should be the focus of an arsenic-related cancer risk assessment; (2) the southwestern Taiwan data are still the most appropriate for arsenic-related cancer risk assessments; and (3) present modes of action data are not sufficient to depart from the default assumption of linearity. The 2001 NRC update also made specific recommendations with respect to the overall cancer risk estimate.

The Agency is currently considering the best way to address all the NRC's recommendations. Based on the Agency's considerations of these recommendations, the current proposed cancer potency number may change in the final version of this risk assessment. For this risk assessment, an oral cancer slope factor of 3.67 $(mg/kg/day)^{-1}$ was used. This is the mean slope factor derived from the higher risk approach for both lung and bladder cancers. This slope factor was used by the EPA's Office of Water when it established the MCL for arsenic in drinking water (U.S. EPA, 2001) and also by the Consumer Product Safety Commission when it performed its deterministic assessment for children's risks from CCA-treated playsets in March 2003 (CPSC, 2003).

The slope factor published by EPA's Integrated Risk Information System (IRIS), 1.5 (mg/kg/day)-1 , is also being revisited in FY2003 due to the recommendation by the NRC in 2001. If the Agency had used the current IRIS cancer slope factor (1.5 (mg/kg/day)-1 ) instead of the slope factor used in the Office of Water's arsenic MCL document (3.67 (mg/kg/day)-1)(U.S. EPA, 2001), the cancer risk would be approximately 41% of the current cancer risk estimates in this document . For example, a reported cancer risk of 5.0E-4 using the cancer slope factor of 3.67$(mg/kg/day)^{-1}$ would be equivalent to 2.0E-4 using the IRIS cancer slope factor of 1.5 $(mg/kg/day)^{-1}$.

For inhalation exposure route, in this risk assessment, the cancer endpoint is $4.3 \times 10^{-3}$ ( g/m3)$^{-1}$ which is equivalent to a cancer slope factor of 15.1 $(mg/kg/day)^{-1}$ (EPA, 1989). All the EPA proposed $Q_1$* are summarized in Table 3.

**Table 3. Cancer Slope Factor used for Assessing Cancer Risks Associated with Occupational Exposures/Risks to Inorganic Arsenic**

**Oral Slope Factors (Q1*) Based on the EPA's Integrated Risk Information System (IRIS) (Last Revised -- 04/10/1998)**

**Based on Internal organ cancer (liver, kidney, lung and bladder) and skin cancer**

Slope Factor (Q1*) [(a)] 1.5E+0 per (mg/kg)/day

**Oral Slope Factors (Q1*) Based on the EPA's Risk Assessment Associated with Drinking Water (EPA, 2001)**

| | Higher Set [(a)] (per (mg/kg)/day) | Lower Set [(a)] (per (mg/kg)/day) |
|---|---|---|
| **Based on Bladder Cancer** | | |
| Male: | 1.49 | 0.18 |
| Female: | 2.15 | 0.25 |
| **Based on Lung Cancer:** | | |
| Male: | 1.60 | 0.23 |
| Female: | 2.10 | 0.27 |
| **Based on Bladder+Lung Cancer:** | | |
| Male: | 3.09 | 0.30 |
| Female: | 4.25 | 0.37 |
| Combine Male and Female | 3.67 [(b)] | 0.33 |

**Inhalation Slope Factors (Q1*) Based on the EPA's Integrated Risk Information System (IRIS) (Last Revised -- 04/10/1998)**

**Based on Skin Cancer**

Slope Factor (Q1*) [(c)] 15.1 per (mg/kg)/day

**Note:**

(a). EPA's assumptions for its high and low risk estimates is as follows:

| | | Low risk | High risk |
|---|---|---|---|
| Drinking water consumption, U.S. | L/d | 1 | 1.2 |
| Cooking water consumption, Taiwan | L/d | 1 | 0 |
| Additional As consumed in food, Taiwan | µg/d | 50 | 0 |

(b). Q1* of 3.67 per (mg/kg)/day is the number be used in this risk assessment. Attachment 1 presents how the slope factor was derived.

(c). Q1* derived from a inhalation unit risk of 4.3 x 10-3 ( g/m3)$^{-1}$. Unit risk= Q1* x 1/70 kg x 20 m$^3$/day x 1 g/1,000 mg (EPA, 1989).

**Table 4. Toxicological Endpoints for Assessing Occupational Exposures/Risks to Arsenic (V)**

| EXPOSURE SCENARIO | DOSE (mg/kg/day) | ENDPOINT | STUDY |
|---|---|---|---|
| Acute Dietary | This risk assessment is not required. | | |
| Chronic Dietary | This risk assessment is not required. | | |
| Incidental Short- and Intermediate- Term Oral | LOAEL= 0.05<br><br>MOE = 30 | Based on edema of the face, gastrointestinal, upper respiratory, skin, peripheral and neuropathy symptoms | Franzblau et al.(1989) and Mizuta et al. (1956) |
| Dermal Short- and Intermediate-Term [(a) (b)] | LOAEL= 0.05<br><br>MOE = 30 | Based on edema of the face, gastrointestinal, upper respiratory, skin, peripheral and neuropathy symptoms | Franzblau et al.(1989) and Mizuta et al. (1956) |
| Dermal Long-Term [(a) (b)] | NOAEL= 0.0008<br><br>MOE = 3 | Based on hyperpigmentation, keratosis and possible vascular complications. | Tseng et al. (1968) and Tseng (1977) |
| Inhalation Short- and Intermediate-Term[(c)] | LOAEL= 0.05<br><br>MOE = 30 | Based on edema of the face, gastrointestinal, upper respiratory, skin, peripheral and neuropathy symptoms | Franzblau et al.(1989) and Mizuta et al. (1956) |
| Inhalation, Long-Term | NOAEL= 0.0008<br><br>MOE = 3 | Based on hyperpigmentation, keratosis and possible vascular complications. | Tseng et al. (1968) and Tseng (1977) |
| Carcinogenicity - Inhalation (Inhalation Unit Risk) | $Q_1$*= 15.1 [(d)] $(mg/kg/day)^{-1}$ | Lung cancer | Chronic epidemiological inhalation study on humans |
| Carcinogenicity - Oral Ingestion (Oral and Dermal Risks) | $Q_1$* = 3.67 $(mg/kg/day)^{-1}$ | Internal organ cancer (liver, kidney, lung and bladder) and skin cancer | Chronic epidemiological oral study on humans |

Note:

(a). MOE = Margin of Exposure; NOAEL = No observed adverse effect level; and LOAEL = Lowest observed adverse effect level.

(b). The dermal absorption factor = 6.4%. (Note: The FIFRA Scientific Advisory Panel recommended use of a lower value of 2-3%. The occupational assessment in the PRA uses 6.4 percent dermal absorption because the handlers and workers are exposed to the arsenic residue from the aqueous solution during mixing, loading, and handling or are exposed to newly treated, or "wet' wood which has arsenic residues on the surface of the wood).

(c). For inhalation exposure, a default absorption factor of 100% is used. Route-to-route extrapolation is used to estimate the exposed dose.

(d). $Q_1$* derived from a inhalation unit risk of $4.3 \times 10^{-3}$ $(\mu g/m^3)^{-1}$. Unit risk= $Q_1$* x 1/70 kg x 20 $m^3$/day x 1 mg/1,000 µg (EPA, 1989).

## 2.2 Inorganic Chromium Endpoint Selection

On August 28, 2001, the OPP's Hazard Identification Assessment Review Committee (HIARC) evaluated the toxicology data base of **Cr(VI)** and established the toxicological endpoints for occupational exposure risk assessments. On October, 23-25 2001, the FIFRA Scientific Advisory Panel (SAP) met and discussed some issues about the end points proposed by the HIARC. The conclusions related to inorganic arsenic are summarized in **Table 5.**

### 2.2.1 Acute Reference Dose (aRfD)

An acute RfD value was not selected for inorganic chromium. Inorganic chromium is not registered for any food uses and there are no existing tolerances. For inorganic chromium as contained within CCA-treated wood, therefore, an acute RfD is not relevant to the exposures from registered uses.

### 2.2.2 Chronic Reference Dose (cRfD)

The U.S. EPA has published a chronic RfD value for inorganic chromium (USEPA IRIS, 1998). However, as with the acute RfD, there are no exposure scenarios relevant to the currently registered uses of inorganic chromium, and specifically the registered uses in CCA-treated lumber. If the Agency determines in the future that an aggregate assessment is needed for calculation of risk from exposure to chromium in treated lumber and exposure in drinking water and/or food, the chronic RfD value can be utilized.

### 2.2.3 Short-Term (1-30 days) and Intemediate-Term (30-180 days) Incidental Oral Exposure

Based on the registered use of CCA-treated lumber for fencing and decking materials in residential settings, incidental oral exposure to chromium is expected, based on potential ingestion of soil contaminated with chromium as a result of leaching from wood, and from ingestion of chromium residues from the palm as a result of direct dermal contact with treated wood. The study selected for short- and intermediate-term incidental oral exposure is a developmental toxicity study in the rabbit conducted by Tyl and submitted to the Agency under MRID # 42171201. The executive summary is shown below.

In a developmental toxicity study [ MRID 421712-01], artificially inseminated New Zealand White rabbits (16 females/dose group) received aqueous chromic acid (55.0%) by gavage once daily on gestation days 7 through 19 at dose levels of 0.0, 0.1, 0.5, 2.0, or 5.0 mg/kg/day in deionized/distilled water.

Clinical signs of toxicity , including diarrhea, and slow, audible or labored breathing were observed predominately in the 2.0 and 5.0 mg/kg/day groups. These signs were observed in slightly higher incidence at the 2.0 mg/kg/day dose level than at the 5.0 mg/kg/day dose level.

However, the incidence and temporal occurrence of mortality (at 2.0 mg/kg/day, one doe died on gestation day (GD) 28; at 5.0 mg/kg/day, 5 does died (one each on GD 10, 14, and two on GD 15) and the magnitude of decreased body weight gain during the dosing period (average weight loss of 48 grams at 2.0 mg/kg/day and average weight loss of 140 grams at 5.0 mg/kg/day during gestation days 7-19) were observed to occur in a dose-related fashion at 2.0 and 5.0 mg/kg/day. Overall weight gain was decreased 24% at 2.0 mg/kg/day and 20% at 5.0 mg/kg/day. Food efficiency was also observed to be significantly lower during the dosing period in the 5.0 mg/kg/day dose group. Cesarean section observations were unremarkable in this study at any dose level tested. There were no significant treatment-related effects on the incidence of external, visceral, or skeletal malformations in the offspring in this study.

**The Maternal NOAEL = 0.5 [0.12] mg/kg/day and LOAEL = 2.0 [0.48] mg/kg/day (based on the increased incidence of maternal mortality and decreased body weight gain ). The Developmental NOAEL = 2.0 [0.48] mg/kg/day and LOAEL > 2.0 [>0.48] mg/kg/day based on the lack of developmental effects at any dose level tested.**

The developmental toxicity study in the rabbit was chosen for selection of the short-term and intermediate-term incidental oral exposure endpoint. This study and endpoint is felt to be appropriate for both short- and intermediate-term incidental oral exposures, based on the occurrence of toxic effects after short-term dosing (mortality, clinical signs, weight loss), and supporting data from the open literature showing similar effects after longer-term exposures at similar dose levels. A study by Zhang and Li (1987) detailed toxic effects observed in 155 human subjects exposed long-term to chromium in drinking water at a concentration of approximately 20 mg/L (USEPA IRIS, 1998), or 0.66 mg/kg/day. These effects included mouth sores, diarrhea, stomach ache, indigestion, vomiting, and elevated white cell count. Although precise concentrations of chromium in the water, exposure durations, and confounding factors were not discussed in this paper, the data suggest gastrointestinal effects at a level of approximately 0.66 mg/kg/day. Thus, the choice of the NOAEL value of 0.5 mg/kg/day from the developmental toxicity study in rabbits (a well-conducted multi-dose animal study) for the incidental oral endpoint is felt to be protective of the gastrointestinal effects observed in humans at a similar dose. The choice of this endpoint is also felt to be protective of the non-lethal effect observed in humans based on a more severe effect observed in animals (i.e. mortality).

### 2.2.4 Dermal Absorption

For inorganic chromium, a dermal absorption value of 1.3% was selected, based upon the data of Bagdon (1991). The executive summary of this study is presented below.

Sodium chromate (Cr(VI)) was applied to the skin of guinea pigs and the skin permeation was determined by assay of $^{51}Cr$ content present in the excreta (1.11%) and organs (0.19%) after 24 hours. In this study in guinea pigs, skin penetration of chromium amounted to 1.30% of the applied dose after 24 hours. Using another *in vivo* method, a weighed amount of the agent was patched to the skin of guinea pigs and the concentration followed by determination of the remaining agent at the application site after different intervals. Skin penetration was concentration

dependent. The range used was 0.0048 to 1.689 M. Dermal penetration for hexavalent chromium amounted to 2.6% of the applied dose of 0.0175 M/5 hours and 4.0% at 0.261 M/5 hours. At 0.261 M, the skin permeation rate was 700 mμM/cm$^2$/hr. This procedure may overestimate skin penetration because chromium present in the skin depot would be calculated as part of the residual test material at the skin's surface.

### 2.2.5 Short-, Intermediate-, and Long- term Dermal Exposure

The 1998 EPA IRIS document on chromium (VI) states that "chromium is one of the most common contact sensitizers in males in industrialized countries and is associated with occupational exposures to numerous materials and processes.." In addition, it is stated further that "dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis." The relative potency of this effect appears to differ between the (VI) and (III) species of chromium. Bagdon (1991) collected skin hypersensitivity data for trivalent chromium compounds in human subjects and concluded that the threshold level for evoking hypersensitivity reactions from trivalent chromium compounds is approximately 50-fold higher than for hexavalent chromium compounds.Nontheless, it is apparent that both forms of chromium cause hypersensitivity reactions in humans.

It was determined by the Hazard Identification Assessment Review Committee (HIARC) of the Office of Pesticide Programs that quantification of hazard from dermal exposure is not possible for chromium, due to the significant dermal irritation and sensitization observed. Therefore, no endpoints were determined by HIARC for hexavalent chromium from dermal exposures.

### 2.2.6 Inhalation Exposure (all durations)

Although chromium is not considered a volatile agent when present in soil, inhalation of soil dust contaminated with chromium may present a potential inhalation risk given the significant irritant properties of chromium and the potential for nasal deposition of the chemical after inhalation of contaminated soil dust. Linberg, 1983 studied respiratory symptoms, lung function and changes in nasal septum in 104 workers (85 males, 19 females exposed in chrome plating plants. Workers were interviewed using a standard questionnaire for the assessment of nose, throat and chest symptoms. Nasal inspections and pulmonary function testing were performed as part of the study.

The median exposure time for the entire group of exposed subjects (104) in the study was 4.5 years (0.1-36 years). A total of 43 subjects exposed almost exclusively to chromic acid experienced a mean exposure of 2.5 years (0.2-23.6 years). The subjects exposed almost exclusively to chromic acid were divided into a low exposure group (8-hr TWA below 0.002 mg/m$^3$, N=19) and a high-exposure group (8-hr TWA above 0.002 mg/m$^3$, N=24). Exposure measurements using personal air samplers were performed for 84 subjects in the study on 13 different days. Exposure for the remaining workers 20 workers was assumed to be similar to that measured for workers in the same area. Nineteen office employees were used as controls for nose and throat symptoms. A group of 119 auto mechanics whose lung function had been evaluated by

similar techniques was selected as controls for lung function measurements. Smoking habits of workers were evaluated as part of the study.

At mean exposures below 0.002 mg/m$^3$, 4/19 workers from the low-exposure group experienced subjective nasal symptoms. Atrophied nasal mucosa were reported in 4/19 subjects from this group and 11/19 had smeary and crusty and septal mucosa, which was statistically higher than the controls. No one exposed to levels below 0.001 mg/m$^3$ complained of subjective symptoms. At mean concentrations of 0.002 mg/m$^3$ or above, approximately 1/3 of the subjects had reddened, smeary or crusty nasal mucosa. Atrophy was seen in 8/24 workers, which was significantly different from controls. Eight subjects had ulcerations in the nasal mucosa and 5 had perforations of the nasal septum. Atrophied nasal mucosa was not observed in any of the 19 controls, but smeary and crusty septal mucosa occurred in 5/19 controls.

Short-term effects on pulmonary function were evaluated by comparing results of tests taken on Monday and Thursday among exposed groups and controls. No significant changes were seen in the low-exposure group or the control group. Non-smokers in the high-exposure group experienced significant differences in pulmonary function measurements from the controls, but the results were within normal limits.

The authors concluded that 8 hour exposure to chromic acid above 0.002 mg/m$^3$ may cause a transient decrease in lung function, and that short-term exposure to greater than 0.002 mg/m3 may cause ulceration and perforation. Based on the result of this study, a LOAEL of 0.002 mg/m$^3$ can be identified for incidence of nasal septum atrophy following exposure to chromic acid mists in chrome plating facilities. Therefore, the LOAEL of continuous exposure of 0.002 mg/m$^3$ was based on ulcerations, perforations of the nasal septum and pulmonary function changes. A MOE of 100 selected (3x to extrapolate from LOAEL to NOAEL, 3X to account for the uncertainty associated with using an epidemiological study and 10X for intraspecies extrapolation). The 100 mg/m$^3$ can not be considered as NOAEL because it is just a level reported with no subjective symptoms.

### 2.2.7 <u>Carcinogenicity</u>

The cancer endpoint for inhalation exposure is classified as group A (known human carcinogen) with an inhalation unit risk of $1.2 \times 10^{-2}$ (?g/m$^3$)$^{-1}$ (Table 5). Human carcinogenicity by the oral route of exposure cannot be determined and chromium is classified as group D.

**Table 5. Toxicological Endpoints for Assessing Occupational Exposures/Risks to Chromium (VI)**

| EXPOSURE SCENARIO | DOSE (mg/kg/day) | ENDPOINT | STUDY |
|---|---|---|---|
| Acute Dietary | This risk assessment is not required. | | |
| Chronic Dietary | This risk assessment is not required. | | |
| Incidental Short- and Intermediate-Term Oral | [a] NOAEL= 0.5of chromic acid [0.12 of Cr(VI)]<br><br>MOE = 100 | based on the increased incidence of maternal mortality and decreased body weight gain at LOAEL of 2.0 [0.48] | Developmental/Rabbit Tyl, 1991 |
| Dermal Exposure [b] (All Durations) | Because dermal irritation and dermal sensitization are the primary concern through the dermal exposure route, no toxicological end-point is selected for use in assessing dermal exposure risks to chromium. | | |
| Inhalation Exposure (All Durations) | [a] LOAEL= 0.002 $mg/m^3$; (or $2.3 \times 10^{-4}$ mg/kg/day)<br>MOE = 100 | nose and throat symptoms observed at the 0.002 $mg/m^3$ level | Linberg and Hedenstierna, 1983. |
| Carcinogenicity - Inhalation (Inhalation Unit Risk) | $Q_1$* = 40.6 $(mg/kg/day)^{-1}$ [c] | Lung tumors | IRIS |

Note:

(a). MOE = Margin of Exposure; NOAEL = No observed adverse effect level; and LOAEL = Lowest observed adverse effect level.

(b). The dermal absorption factor for Cr(VI) = 1.3% for handler dermal contact with chromated arsenical pesticides.

(c) The inhalation $Q_1$* is $1.16 \times 10^{-2}$ $(\mu g/m^3)^{-1}$ which can also be expressed as 0.0116 $m^3$/?g.. To convert the air concentration to a dose to yield units of kg-day/mg or $(mg/kg/day)^{-1}$ the unit risk is expressed mathematically as 0.0116 $m^3$/?g x day/20 $m^3$ x 1000 ?g/mg x 70 kg = 40.6 $(mg/kg/day)^{-1}$ (EPA, 1989).

## 3.0 REFERENCES


Aiyar J, Borges K, Floyd RA, et al. 1989. Role of chromium(V), glutathione thiyl radical and hydroxyl radical intermediates in chromium(VI)-induced DNA damage. Toxicol Environ Chem 22:135-148.

Amdur, MO; Doull, J; Klaassen, CD. (1993) Casarett and Doull's Toxicology. New York: McGraw Hill.

ATSDR (2000a). Toxicological Profile for Arsenic.: U.S. Department of Health and Human Services, Public Health Service.

ATSDR (2000b): Toxicological Profile for Chromium. U.S. Department of Health and Human Services, Public Health Service.

*Author not stated. 1985 Acute Oral Toxicity Study, Bio/dynamics, Inc. Project 5465-84. May 30, 1985. Data Accession No. 26356. Unpublished.

*Author not stated. 1984. Acute Dermal Toxicity Study, Bio/dynamics Inc. Project 5466-84. Nov, 1984. Data Accession No. 26356. Unpublished.

*Author not stated. 1984. Primary Eye Irritation Study, Bio/dynamics, Inc. Project 5468-84. April 24, 1984. Data Accession No. 26356. Unpublished.

*Author not stated. 1984. Primary Dermal Irritation Study, Bio/dynamics, Inc. Project 5467-84. April 18, 1985. Data Accession No. 26356. Unpublished.

Baetjer, AM; Lowney, JF; Steffee, H; et al. (1959) Effect of chromium on incidence of lung tumors in mice and rats. Arch Ind Health 20:124-135.

Bagdon, R.E. and Hazen, R.E. (1991): Skin Permeation and Cutaneous Hypersensitivity as a Basis for Making Risk Assessments of Chromium As a Soil Contaminant. Env. Hlth. Perspec. 92: 111-119.

Bertolero F, Pozzi G, Sabbioni E, et al. 1987. Cellular uptake and metabolic reduction of pentavalent to trivalent arsenic as determinants of cytotoxicity and morphological transformation. Carcinogenesis 8:803-808.

Bishop, C; Surgenor, M, eds. (1964) The red blood cell: a comprehensive treatise. New York: Academic Press.

Bryson WG, Goodall CM. 1983. Differential toxicity and clearance kinetics of chromium(III) or (VI) in mice. Carcinogenesis 4(12):1535-1539.

Chowdhury AR, Mitra C. 1995. Spermatogenic and steroidogenic impairment after chromium treatment in rats. Indian J Exp Biol 33:480-484.

Cohen, Y., Winer, A.M., Creelman, L., and Mabuni, C. 1999.A Critical Assessment of Chromium in the Environment. Critical Rev. in Environmental Science and Technology 29(1): 1-46.

CPSC, 2003. Briefing Package. Petition to Ban Chromated Copper Arsenate (CCA)-Treated Wood in Playground Equipment (Petition HP 01-3). February 2003.

De Flora S, Badolati GS, Serra D, et al. 1987a. Circadian reduction of chromium in the gastric environment. Mutat Res 192:169-174.

Donaldson DL, Smith CC, and Yunice AA. 1984. Renal excretion of chromium-51 chloride in the dog. Am J Physiol 246:F870-F878.

Donaldson RM and Barreras RF. 1966. Intestinal absorption of trace quantities of chromium. J Lab Clin Med 68:484-493.

Federal Register, May 6, 1993, Vol 58, p. 26975, [as cited in Federal Register, Vol 58, No 234/Wednesday, Dec. 8, 1993/Notices, p. 64580-64582]

Fishbein L. 1981. Sources, transport and alterations of metal compounds: An overview. I. Arsenic,beryllium, cadmium, chromium and nickel. Environ Health Perspect 40:43-64.

Franzblau, A. and Lilis, R. 1989. Acute Arsenic Intoxication from Environmental Arsenic Exposure. Archives of Envir. Health 44(6). 385-390.

Freeman, GB., Johnson, J.D., Killinger, J.M., Liao, S.C., Davis, A.O., Ruby, M.V., Chaney, R.L., Lovre, S.C., and Bergstrom, P.D. 1993. Bioavailability of Arsenic in Soil Impacted by Smelter Activities Following Oral Administration in Rabbits. Fundamental and Applied Toxicology 21:83-88

Freeman, G.B., Schoof, R.A., Ruby, M.V., Davis, A.O., Dill, J.A., Liao, S.C., Lapin, C.A., and Bergstrom, P.D. 1995. Bioavailability of Arsenic in Soil and House Dust Impacted by Smelter Activities Following Oral Administration in Cynomologus Monkeys. Fundamental and Applied Toxicology 28:215-222

Gibson DP, Brauninger R, Shaffi HS, et al. 1997. Induction of micronuclei in Syrian hamster embryocells: comparison of results in the SHE cell transformation assay for national toxicology program test chemicals. Mutat Res 392(1-2):61-70.

Groen, K., Vaesen, H.A.G., Klest, J.I.G. deBar, J.L.M., von Ooik, T. Timmerman, A. and Vlug, R.G. 1993. Bioavailability of Inorganic Arsenic from Bog Ore-Containing Soil in the Dog. Environmental Health Perspective 102: 182-184.

Henderson RF, Rebar AH, Pickrell JA, et al. 1979. Early damage indicators in the lung. III. Biochemical and cytological response of the lung to inhaled metal salts. Toxicol Appl Pharmacol 50:123-136.

Hopenhayn-Rich et al., 1998: Lung and Kidney Cancer Mortality Associated with Arsenic in Drinking Water in Cordoba, Argentina. Epidemiology 27: 561-569.

Hopenhayn-Rich et al., 2000: Chronic Arsenic Exposure and Risk of Infant Mortality in Two Areas of Chile. Env. Hlth. Perspec. 108: 667-673, July 2000.

Hopenhayn, C, Ferreccio, C, Browning, SR, Huang, B. et al. (2003) Arsenic Exposure from Drinking Water and Birth Weight. Epidemiology 14:593-602.

Hueper, WC; Payne, WW. (1962) Experimental studies in metal carcinogenesis--Chromium,nickel, iron, arsenic. Arch Environ Health 5:445-462.

International Agency for Research on Cancer (IARC). 1990. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Vol. 49. Some metals and metallic compounds. Lyon, France: World Health Organization.

IRIS. 2000. Chromium VI. Integrated Risk Information System. U.S. Environmental ProtectionAgency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Ivankovic, S; Preussman, R. 1975. Absence of toxic and carcinogenic effects after administrations of high doses of chronic oxide pigment in subacute and long term feeding experiments in rats. Food Cosmet Toxicol 13:347-351.

Jennette KW. 1982. Microsomal reduction of the carcinogen chromate produced chromium(V). J Am Chem Soc 104:874-875.

Junaid M, Murthy RC, Saxena DK. 1996a. Embryo- and fetotoxicity of chromium in pregestationally exposed mice. Bull Environ Contam Toxicol 57:327-334.

Kanojia RK, Junaid M, Murthy RC. 1996. Chromium induced teratogenicity in female rat. ToxicolLett 89:207-213.

Kenyon, E.M. and Hughes, M.F. 2001.: A concise review of the toxicity and carcinogenicity of dimethylarsinic acid. Toxicology 160: 227-236.

Kochhar TS, Howard W, Hoffman S, et al. 1996. Effect of trivalent and pentavalent arsenic in causing chromosome alterations in cultured Chinese hamster ovary (CHO) cells. Toxicol Lett 84(1):37-42.

Lerman S, Clarkson TW, Gerson RJ. 1983. Arsenic uptake and metabolism by liver cells is dependent on arsenic oxidation state. Chem Biol Interact 45:401-406.

Larramendy ML, Popescu NC, DiPaolo J. 1981. Induction by inorganic metal salts of sister chromatid exchanges and chromosome aberrations in human and Syrian hamster strains. Environ Mutagen 3:597-606.

Lebow, S. 1996. Leaching of Wood Preservative Components and their Mobility in the Environment- Summary of Pertinent Literature. Gen. Tech. Rep. FPL-GTR-93. Madison, WI: U.S. Department of Agriculture, Forest Service, Forest Products Laboratory, 36 p.

Lee, T-C, et al. 1985. Comparison of arsenic-induced cell transformation, cytotoxicity, mutation, and cytogenetic effects in Syrian hamster embryo cells in culture. Carcinogenesis 6(10): 1421-1426.

Levy, LS; Venitt, S. 1975. Carcinogenic and mutagenic activity of chromium-containing materials. Br J Cancer 32:254-255.

Levy, LS; Martin, PA. 1983. The effects of a range of chromium-containing materials on rat lung. Dye Color Manufacturers Association.

Maruyama, Y.(1982): The health effect of mice given oral administration of trivalent and hexavalent chromium over a long term. Acta Scholae Medicinalis Universitatis in Gifu 31:24-36.

Mass, M.J. et al. 2001.: Methylated Trivalent Arsenic Species are Genotoxic. Chem. Res. Toxicol. 14: 355-361.

Mizuta, N, Mizuta, et al. 1956. An Outbreak of Acute Arsenic Poisoning Caused by Arsenic-Containing Soy-Sauce (Shoyu). A Clinical Report of 220 Cases. Bull Yamaguchi Med Sch 4(2-3):131-149.

Moore MM, Harrington-Brock K, Doerr CL. 1997. Relative genotoxic potency of arsenic and its methylated metabolites. Mutat Res 386(3):279-290.

Morales, K. H.; Ryan, L.; Kuo, T.; Wu, M.; and Chen, C.. 2000. Risk of Internal Cancers from Arsenic in Drinking Water. Environ. Health Perspect 108:655-661.

National Research Council: Arsenic in Drinking Water: 2001 Update. September, 2001, National Academy Press, Washington, D.C.

National Toxicology Program (NTP). 1996. Final report on the reproductive toxicity of potassium dichromate (hexavalent)(CAS No. 7778-50-9) administered in diet to SD rats. Dec. 16, 1996. U.S. Department of Commerce, National Technical Information Service, PB97125355.

National Toxicology Program (NTP). 1997a. Final report on the reproductive toxicity of potassium dichromate (hexavalent) (CAS No. 7778-50-9) administered in diet to BALB/C mice. Jan 10, 1997. U.S. Department of Commerce, National Technical Information Service, PB97125363.

NTP, Public Health Service, U.S. Department of Health and Human Services. 1997. Final report. Potassium dichromate (hexavalent): reproductive assessment by continuous breeding when administered to BALB/c mice in the diet. February 18, 1997. Available from: National Institute of Environmental Health Sciences, Research Triangle Park, NC.

NRC (National Research Council). 1999. Arsenic in Drinking Water. National Academy Press, Washington, D.C.

Oberly TJ, Piper CE, McDonald DS. 1982. Mutagenicity of metal salts in the L5178Y mouse lymphoma assay. J Toxicol Environ Health 9:367-376.

Rossman, T.G. et al. 1980.: Absence of arsenite mutagenicity in E. coli and Chinese hamster cells. Environ. Mut. 2: 371-379.

Sayato Y, Nakamuro K, Matsui S, et al. 1980. Metabolic fate of chromium compounds. I. Comparative behavior of chromium in rat administered with $Na_2{}^{51}CrO_4$ and $^{51}CrCl_3$. J Pharm Dyn 3:17-23.

Schroeder, HA; Balassa, JJ; Vinton, WH, Jr. 1965. Chromium, cadmium and lead in rats: effects on lifespan, tumors, and tissue levels. J Nutr 86:51-66.

Suzuki Y, Fukuda K. 1990. Reduction of hexavalent chromium by ascorbic acid and glutathione with special reference to the rat lung. Arch Toxicol 64:169-176.

Towill, LE; Shriner, CR; Drury, JS; et al. 1978. Reviews of the environmental effects of pollutants. III. Chromium. Prepared by the Health Effects Research Laboratory, Office ofResearch and Development, U.S. Environmental Protection Agency, Cincinnati, OH. Report No. ORNL/EIS-80. EPA 600/1-78-023. NTIS PB 282796.

Trivedi B, Saxena DK, Murthy RC, et al. 1989. Embryotoxicity and fetotoxicity of orally administered hexavalent chromium in mice. Reprod Toxicol 3:275-278.

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

Tseng W-P. 1977. Effects and dose-response relationships of skin cancer and Blackfoot disease with arsenic. Environ Health Perspect 19:109-119.

USEPA. Bioavailability of Arsenic and Lead in Environmental Substrates. 1. Results of an Oral Dosing Study of Immature Swine. Superfund/Office of Environmental Assessment, Region 10, EPA 910/R-96-002, 1996.

USEPA. 2001. National Primary Drinking Water Regulation; Arsenic and Clarifications to Compliance and New Source Contaminants Monitoring; Final Rule. *Federal Register*. Vol. 66, No. 14. p. 6975, January 22, 2001.

USEPA Region 8, 2001: Derivation of Acute and Subchronic Oral Reference Doses for Inorganic Arsenic.

USEPA, IRIS, Chromium (VI), 1998; CASRN 18540-29-9, 9/3/1998.

Waalkes, MP; Ward, JM; Liu, J. and Diawan, BA. 2003. Transplacental carcinogenicity of Inorganic Arsenic in the Drinking Water: Induction of Hepatic, Ovarian, Pulmary, and Adrenal Tumors in Mice. Toxicology and Applied Pharmacology: 186:7-17.

Wiegand, HJ; Ottenwalder, H; Bolt, HM. 1985. Fast uptake kinetics *in vitro* of 51 Cr(VI) by red blood cells of man and rat. Arch Toxicol 57:31-34.

Wester, R.C., Maibach, H.I., Sedik, L. Melendres, J., and Wader, M. 1993. In Vivo and in Vitro Percutaneous Absorption and Skin Decontamination of Arsenic From Water and Soil. Fundamental and Applied Toxicology 20:336-340

Williams, T.W. : Rawlins, B.G.; Smith, B.; and Breward, N. 1998. In-Vitro Determination of Arsenic Bioavailability in Contaminated Soil and Mineral Benefication Waste from Ron Phibun, Southern Thailand: A Basis for Improved Human Risk Assessment. Environmental Geochemistry and Health: 20

Witmer, C.M, Harris R and Shupack SI. 1991. Oral bioavailability of chromium from a specific site. Environ Health Perspect 92:105-110.

Zahid,Z.R., Al-Hakkak ZS, Kadhim AHH, et al. 1990. Comparative effects of trivalent and hexavalent chromium on spermatogenesis of the mouse. Toxicol Environ Chem 25:131-136.

# Attachment 1

## US EPA's Risk Assessment for Arsenic in Drinking Water

**US EPA's Risk Estimates for Arsenic in Drinking Water**
8 March 2002

This spreadsheet contains the U.S. Environmental Protection Agency's estimates of the average lifetime excess risk of death from lung and bladder cancers due to arsenic in drinking water. EPA developed these risk estimates to support its January 2001 National Primary Drinking Water Regulation for arsenic (US EPA, 2001).

The calculations in this spreadsheet describe the theoretical, population-averaged risks of death for a single person drinking a given concentration of arsenic for their entire lifetime. Changes in exposure over time, aggravating or mitigating factors, and ordinary variation between people mean that any one person's risk will be different from the risks computed here. However, the risks in this spreadsheet represent EPA's best estimate of the average risks to people exposed to given levels of arsenic in drinking water in the U.S.

The risk estimates computed here provide only one piece of EPA's arsenic risk assessment. For its risk assessment, EPA combined estimates of risk for a single "average" person with information about the distributions of arsenic occurrence, sex, body weight, and drinking water consumption in the U.S, in order to estimate the expected numbers of lives saved under different regulatory options. EPA also considered information about other, nonquantifiable risks. Only one of these factors, the theoretical average risk of lung and bladder cancers, is described in this spreadsheet. For more information about EPA's arsenic risk assessment, see US EPA (2000, 2001).

To estimate the risks due to arsenic in drinking water, EPA used risk estimates taken from Morales et al. (2000). Morales et al. fit a variety of dose-response models to lung and bladder cancer data from an arseniasis-endemic region of southwestern Taiwan. Of the models in Morales et al., EPA used estimates from Model 1, fit with no comparison population. Model 1 is a Poisson regression model in which the logarithm of the hazard (rate of death among those surviving to a given age) increases linearly with arsenic dose and quadratically with age. The risk estimates from Model 1 are contained in the yellow cells of the "Calculations" worksheet. The estimates are characterized there (as in Morales et al.) by ED01, the level at which a person's lifetime risk of death increases by 1% (for example, from 2% to 3%), and LED01, a 95% lower confidence bound for ED01.

EPA presented two sets of risk estimates, higher and lower, in its final arsenic rule. For the higher set of risks, EPA used the theoretical risk estimates taken directly from Morales et al. (2000). Risk was assumed to increase linearly with dose, from zero to the ED01. For the lower set of risks, EPA adjusted the theoretical risks to take into account possible higher arsenic consumption in Taiwan. For these estimates, EPA assumed that people in Taiwan consumed an additional 1 L/d of water in cooking, due to rehydration of rice and sweet potatoes, and a further 50 ug/d of arsenic directly from their food. These assumptions resulted in lower risk estimates for the U.S., since the same number of cancer deaths in Taiwan were assumed to have resulted from higher arsenic exposure there. EPA also assumed a lower mean drinking water consumption in the U.S. A summary of EPA's assumptions for its high and low risk estimates is as follows:

| | | Low risk | High risk |
|---|---|---|---|
| Drinking water consumption, U.S. | L/d | 1 | 1.2 |
| Cooking water consumption, Taiwan | L/d | 1 | 0 |
| Additional arsenic consumed in food, Taiwan | ug/d | 50 | 0 |

The attached "Calculations" worksheet contains all of the necessary assumptions, intermediate calculations, and results to derive EPA's theoretical mean risk estimates.

For more information about this spreadsheet or EPA's arsenic risk assessment, contact:

Andrew Schulman
202-564-5244
schulman.andrew@epa.gov

or

John Bennett
202-564-4690
bennett.johnb@epa.gov
at EPA's Office of Ground Water and Drinking Water. Or contact:

Targeting and Analysis Branch
Standards and Risk Management Division
Office of Ground Water and Drinking Water
1200 Pennsylvania Ave. NW, MS 4607M
Washington, DC 20460
202-564-4660

**Converting from ppb and (ppb)$^{-1}$**
**to µg/kg/d and (µg/kg/d)$^{-1}$**

Suppose a person consumes *A* ppb (µg/L) of arsenic in drinking water. They weigh *K* kg and drink *C* L/d of water. Then in µg/kg/d, their exposure is

$$A\frac{\mathbf{ug}}{\mathbf{L}}\cdot\frac{C\,\mathbf{L/d}}{K\,\mathbf{kg}}=\frac{AC}{K}\frac{\mathbf{ug}}{\mathbf{kg\cdot d}}$$

where the liters have cancelled from the numerator and denominator.

On the risk side, suppose that the risk slope is *S* per ppb (ppb$^{-1}$). For the same person as above drinking *C* L/d of water and weighing *K* kg, the risk per µg/kg/d is

$$\frac{S}{\mathbf{ug/L}}\cdot\frac{K\,\mathbf{kg}}{C\,\mathbf{L/d}}=\frac{SK}{C}\frac{}{\mathbf{ug/kg/d}}$$

Again the liters cancel. This is just simple algebra, there's nothing more to it.

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| **Exposure assumptions** | | | | | | | |
| | | | | | | | |
| Arsenic concentration in drinking water | US ppb | | | 50 | | | |
| | | | | | | | |
| **Risk assumptions** | | | | | | | |
| | | Taiwan | | U.S. | | | |
| | | M | F | M | F | | |
| Body weight | kg | 55 | 50 | 70 | 65 | | |
| Drinking water consumption | L/d | 3.5 | 2.0 | 1.2 | 1.2 | | |
| Cooking water consumption | L/d | 0.0 | 0.0 | | | | |
| Additional As consumed in food | ug/d | 0 | 0 | | | | |
| | | | | | | | |
| **Results: Lifetime excess risk of death, bladder and lung cancer** | | | | | | | |
| | | bladder cancer | | lung cancer | | bladder + lung | |
| | | M | F | M | F | M | F |
| Morales ED01 | US ppb | 395 | 252 | 364 | 258 | 189 | 127 |
| Morales LED01 | US ppb | 326 | 211 | 294 | 213 | 163 | 111 |
| | | | | | | | |
| Slope of risk function, for the given occurrence level | /(US ppb) | 0.0000253 | 0.0000397 | 0.0000275 | 0.0000388 | 0.0000528 | 0.0000784 |
| Standard error | /(US ppb) | 0.0000033 | 0.0000047 | 0.0000040 | 0.0000050 | 0.0000051 | 0.0000068 |
| | | | | | | | |
| Lifetime excess risk of death | 1 | 0.0012658 | 0.0019841 | 0.0013736 | 0.0019380 | 0.0026394 | 0.0039221 |
| Standard error | 1 | 0.0001629 | 0.0002344 | 0.0001988 | 0.0002489 | 0.0002570 | 0.0003419 |
| | | | | | | | |
| Note: the risk slopes and ED01s don't depend on the occurrence level unless the "Additional As consumed in food" in Taiwan is >0. | | | | | | | |

References:

Morales, K.H., L. Ryan, T.-L. Kuo, M.-M. Wu and C.-J. Chen. 2000. Risk of internal cancers from arsenic in drinking water. Environmental Health Perspectives 108:655-661.

US EPA. 2000. Arsenic Economic Analysis. EPA 815-R-00-026. Office of Ground Water and Drinking Water, Washington, DC.

US EPA. 2001. National Primary Drinking Water Regulations; Arsenic and Clarifications to Compliance and New Source Contaminants Monitoring. 66 FR 14:6976-7066. Office of Ground Water and Drinking Water, Washington, DC.

## 3.0 REFERENCES


Aiyar J, Borges K, Floyd RA, et al. 1989. Role of chromium(V), glutathione thiyl radical and hydroxyl radical intermediates in chromium(VI)-induced DNA damage. Toxicol Environ Chem 22:135-148.

Amdur, MO; Doull, J; Klaassen, CD. (1993) Casarett and Doull's Toxicology. New York: McGraw Hill.

ATSDR (2000a). Toxicological Profile for Arsenic.: U.S. Department of Health and Human Services, Public Health Service.

ATSDR (2000b): Toxicological Profile for Chromium. U.S. Department of Health and Human Services, Public Health Service.

*Author not stated. 1985 Acute Oral Toxicity Study, Bio/dynamics, Inc. Project 5465-84. May 30, 1985. Data Accession No. 26356. Unpublished.

*Author not stated. 1984. Acute Dermal Toxicity Study, Bio/dynamics Inc. Project 5466-84. Nov, 1984. Data Accession No. 26356. Unpublished.

*Author not stated. 1984. Primary Eye Irritation Study, Bio/dynamics, Inc. Project 5468-84. April 24, 1984. Data Accession No. 26356. Unpublished.

*Author not stated. 1984. Primary Dermal Irritation Study, Bio/dynamics, Inc. Project 5467-84. April 18, 1985. Data Accession No. 26356. Unpublished.

Baetjer, AM; Lowney, JF; Steffee, H; et al. (1959) Effect of chromium on incidence of lung tumors in mice and rats. Arch Ind Health 20:124-135.

Bagdon, R.E. and Hazen, R.E. (1991): Skin Permeation and Cutaneous Hypersensitivity as a Basis for Making Risk Assessments of Chromium As a Soil Contaminant. Env. Hlth. Perspec. 92: 111-119.

Bertolero F, Pozzi G, Sabbioni E, et al. 1987. Cellular uptake and metabolic reduction of pentavalent to trivalent arsenic as determinants of cytotoxicity and morphological transformation. Carcinogenesis 8:803-808.

Bishop, C; Surgenor, M, eds. (1964) The red blood cell: a comprehensive treatise. New York: Academic Press.

Bryson WG, Goodall CM. 1983. Differential toxicity and clearance kinetics of chromium(III) or (VI) in mice. Carcinogenesis 4(12):1535-1539.

Chowdhury AR, Mitra C. 1995. Spermatogenic and steroidogenic impairment after chromium treatment in rats. Indian J Exp Biol 33:480-484.

Cohen, Y., Winer, A.M., Creelman, L., and Mabuni, C. (1999): A Critical Assessment of Chromium in the Environment. Critical Rev. in Environmental Science and Technology 29(1): 1-46.

De Flora S, Badolati GS, Serra D, et al. 1987a. Circadian reduction of chromium in the gastric environment. Mutat Res 192:169-174.

Donaldson DL, Smith CC, and Yunice AA. 1984. Renal excretion of chromium-51 chloride in the dog. Am J Physiol 246:F870-F878.

Donaldson RM and Barreras RF. 1966. Intestinal absorption of trace quantities of chromium. J Lab Clin Med 68:484-493.

Federal Register, May 6, 1993, Vol 58, p. 26975, [as cited in Federal Register, Vol 58, No 234/Wednesday, Dec. 8, 1993/Notices, p. 64580-64582]

Fishbein L. 1981. Sources, transport and alterations of metal compounds: An overview. I. Arsenic,beryllium, cadmium, chromium and nickel. Environ Health Perspect 40:43-64.

Franzblau, A. and Lilis, R. 1989. Acute Arsenic Intoxication from Environmental Arsenic Exposure. Archives of Envir. Health 44(6). 385-390.

Freeman, GB., Johnson, J.D., Killinger, J.M., Liao, S.C., Davis, A.O., Ruby, M.V., Chaney, R.L., Lovre, S.C., and Bergstrom, P.D. 1993. Bioavailability of Arsenic in Soil Impacted by Smelter Activities Following Oral Administration in Rabbits. Fundamental and Applied Toxicology 21:83-88

Freeman, G.B., Schoof, R.A., Ruby, M.V., Davis, A.O., Dill, J.A., Liao, S.C., Lapin, C.A., and Bergstrom, P.D. 1995. Bioavailability of Arsenic in Soil and House Dust Impacted by Smelter Activities Following Oral Administration in Cynomologus Monkeys. Fundamental and Applied Toxicology 28:215-222

Gibson DP, Brauninger R, Shaffi HS, et al. 1997. Induction of micronuclei in Syrian hamster embryocells: comparison of results in the SHE cell transformation assay for national toxicology program test chemicals. Mutat Res 392(1-2):61-70.

Groen, K., Vaesen, H.A.G., Klest, J.I.G. deBar, J.L.M., von Ooik, T. Timmerman, A. and Vlug, R.G. 1993. Bioavailability of Inorganic Arsenic from Bog Ore-Containing Soil in the Dog. Environmental Health Perspective 102: 182-184.

Henderson RF, Rebar AH, Pickrell JA, et al. 1979. Early damage indicators in the lung. III. Biochemical and cytological response of the lung to inhaled metal salts. Toxicol Appl Pharmacol 50:123-136.

Hopenhayn-Rich et al., 1998: Lung and Kidney Cancer Mortality Associated with Arsenic in Drinking Water in Cordoba, Argentina. Epidemiology 27: 561-569.

Hopenhayn-Rich et al., 2000: Chronic Arsenic Exposure and Risk of Infant Mortality in Two Areas of Chile. Env. Hlth. Perspec. 108: 667-673, July 2000.

Hueper, WC; Payne, WW. (1962) Experimental studies in metal carcinogenesis--Chromium,nickel, iron, arsenic. Arch Environ Health 5:445-462.

International Agency for Research on Cancer (IARC). (1990) IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Vol. 49. Some metals and metallic compounds. Lyon, France: World Health Organization.

IRIS. 2000. Chromium VI. Integrated Risk Information System. U.S. Environmental ProtectionAgency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

Ivankovic, S; Preussman, R. (1975) Absence of toxic and carcinogenic effects after administrations of high doses of chronic oxide pigment in subacute and long term feeding experiments in rats. Food Cosmet Toxicol 13:347-351.

Jennette KW. 1982. Microsomal reduction of the carcinogen chromate produced chromium(V). J Am Chem Soc 104:874-875.

Junaid M, Murthy RC, Saxena DK. 1996a. Embryo- and fetotoxicity of chromium in pregestationally exposed mice. Bull Environ Contam Toxicol 57:327-334.

Kanojia RK, Junaid M, Murthy RC. 1996. Chromium induced teratogenicity in female rat. ToxicolLett 89:207-213.

Kenyon, E.M. and Hughes, M.F. (2001): A concise review of the toxicity and carcinogenicity of dimethylarsinic acid. Toxicology 160: 227-236.

Kochhar TS, Howard W, Hoffman S, et al. 1996. Effect of trivalent and pentavalent arsenic in causing chromosome alterations in cultured Chinese hamster ovary (CHO) cells. Toxicol Lett 84(1):37-42.

Lerman S, Clarkson TW, Gerson RJ. 1983. Arsenic uptake and metabolism by liver cells is dependent on arsenic oxidation state. Chem Biol Interact 45:401-406.

Larramendy ML, Popescu NC, DiPaolo J. 1981. Induction by inorganic metal salts of sister chromatid exchanges and chromosome aberrations in human and Syrian hamster strains. Environ Mutagen 3:597-606.

Lebow, S. (1996): Leaching of Wood Preservative Components and their Mobility in the Environment- Summary of Pertinent Literature. Gen. Tech. Rep. FPL-GTR-93. Madison, WI: U.S. Department of Agriculture, Forest Service, Forest Products Laboratory, 36 p.

Lee, T-C, et al. (1985): Comparison of arsenic-induced cell transformation, cytotoxicity, mutation, and cytogenetic effects in Syrian hamster embryo cells in culture. Carcinogenesis 6(10): 1421-1426.

Levy, LS; Venitt, S. (1975) Carcinogenic and mutagenic activity of chromium-containing materials. Br J Cancer 32:254-255.

Levy, LS; Martin, PA. (1983) The effects of a range of chromium-containing materials on rat lung. Dye Color Manufacturers Association.

Maruyama, Y.(1982): The health effect of mice given oral administration of trivalent and hexavalent chromium over a long term. Acta Scholae Medicinalis Universitatis in Gifu 31:24-36.

Mass, M.J. et al. (2001): Methylated Trivalent Arsenic Species are Genotoxic. Chem. Res. Toxicol. 14: 355-361.

Mizuta, N, Mizuta, et al. 1956. An Outbreak of Acute Arsenic Poisoning Caused by Arsenic-Containing Soy-Sauce (Shoyu). A Clinical Report of 220 Cases. Bull Yamaguchi Med Sch 4(2-3):131-149.

Moore MM, Harrington-Brock K, Doerr CL. 1997. Relative genotoxic potency of arsenic and its methylated metabolites. Mutat Res 386(3):279-290.

National Research Council: Arsenic in Drinking Water: 2001 Update. September, 2001, National Academy Press, Washington, D.C.

National Toxicology Program (NTP). 1996. Final report on the reproductive toxicity of potassium dichromate (hexavalent)(CAS No. 7778-50-9) administered in diet to SD rats. Dec. 16, 1996. U.S. Department of Commerce, National Technical Information Service, PB97125355.

National Toxicology Program (NTP). 1997a. Final report on the reproductive toxicity of potassium dichromate (hexavalent) (CAS No. 7778-50-9) administered in diet to BALB/C

mice. Jan 10, 1997. U.S. Department of Commerce, National Technical Information Service, PB97125363.

NTP, Public Health Service, U.S. Department of Health and Human Services. (1997) Final report. Potassium dichromate (hexavalent): reproductive assessment by continuous breeding when administered to BALB/c mice in the diet. February 18, 1997. Available from: National Institute of Environmental Health Sciences, Research Triangle Park, NC.

NRC (National Research Council). 1999. Arsenic in Drinking Water. National Academy Press, Washington, D.C.

Oberly TJ, Piper CE, McDonald DS. 1982. Mutagenicity of metal salts in the L5178Y mouse lymphoma assay. J Toxicol Environ Health 9:367-376.

Roberts, S.M.; Welmar, W.R.; Venson, J.R.; Munson, J.W.; and Bergeron. Measurement of Arsenic Bioavailability from Soils Using a Primate Model. Abstract.

Rossman, T.G. et al. (1980): Absence of arsenite mutagenicity in E. coli and Chinese hamster cells. Environ. Mut. 2: 371-379.

Sayato Y, Nakamuro K, Matsui S, et al. 1980. Metabolic fate of chromium compounds. I. Comparative behavior of chromium in rat administered with $Na_2{}^{51}CrO_4$ and $^{51}CrCl_3$. J Pharm Dyn 3:17-23.

Schroeder, HA; Balassa, JJ; Vinton, WH, Jr. (1965) Chromium, cadmium and lead in rats: effects on lifespan, tumors, and tissue levels. J Nutr 86:51-66.

Suzuki Y, Fukuda K. 1990. Reduction of hexavalent chromium by ascorbic acid and glutathione with special reference to the rat lung. Arch Toxicol 64:169-176.

Towill, LE; Shriner, CR; Drury, JS; et al. (1978) Reviews of the environmental effects of pollutants. III. Chromium. Prepared by the Health Effects Research Laboratory, Office ofResearch and Development, U.S. Environmental Protection Agency, Cincinnati, OH. Report No. ORNL/EIS-80. EPA 600/1-78-023. NTIS PB 282796.

Trivedi B, Saxena DK, Murthy RC, et al. 1989. Embryotoxicity and fetotoxicity of orally administered hexavalent chromium in mice. Reprod Toxicol 3:275-278.

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

Tseng W-P. 1977. Effects and dose-response relationships of skin cancer and Blackfoot disease with arsenic. Environ Health Perspect 19:109-119.

USEPA. Relative Bioavailability of Arsenic in Mining Wastes, Region 8, Document Control No. 4500-88-AORH, 1997.

USEPA. Bioavailability of Arsenic and Lead in Environmental Substrates. 1. Results of an Oral Dosing Study of Immature Swine. Superfund/Office of Environmental Assessment, Region 10, EPA 910/R-96-002, 1996.

USEPA Region 8, 2001: Derivation of Acute and Subchronic Oral Reference Doses for Inorganic Arsenic.

U.S. EPA, IRIS, Chromium (VI), 1998; CASRN 18540-29-9, 9/3/1998.

Wiegand, HJ; Ottenwalder, H; Bolt, HM. (1985) Fast uptake kinetics *in vitro* of 51 Cr(VI) by red blood cells of man and rat. Arch Toxicol 57:31-34.

Wester, R.C., Maibach, H.I., Sedik, L. Melendres, J., and Wader, M. 1993. In Vivo and in Vitro Percutaneous Absorption and Skin Decontamination of Arsenic From Water and Soil. Fundamental and Applied Toxicology 20:336-340

Williams, T.W. : Rawlins, B.G.; Smith, B.; and Breward, N. 1998. In-Vitro Determination of Arsenic Bioavailability in Contaminated Soil and Mineral Benefication Waste from Ron Phibun, Southern Thailand: A Basis for Improved Human Risk Assessment. Environmental Geochemistry and Health: 20

Witmer, C.M, Harris R and Shupack SI. 1991. Oral bioavailability of chromium from a specific site. Environ Health Perspect 92:105-110.

Zahid,Z.R., Al-Hakkak ZS, Kadhim AHH, et al. 1990. Comparative effects of trivalent and hexavalent chromium on spermatogenesis of the mouse. Toxicol Environ Chem 25:131-136.

# Appendix B

# Risk Spreadsheets

Table 1. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (separated by children with and without decks) [Based on LADDs in Table 14 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 728 | 2.3E-05 | 4.4E-05 | 1.1E-05 | 1.0E-07 | 1.7E-06 | 4.9E-06 | 2.5E-05 | 8.3E-05 | 2.4E-04 | 4.9E-04 |
| Playset Total Dose | 0 | 728 | 2.3E-05 | 4.4E-05 | 1.1E-05 | 1.0E-07 | 1.7E-06 | 4.9E-06 | 2.5E-05 | 8.3E-05 | 2.4E-04 | 4.9E-04 |
| Playset Surf Inges-HandToMouth Dose | 0 | 728 | 1.4E-05 | 3.2E-05 | 5.3E-06 | 1.0E-08 | 4.2E-07 | 1.9E-06 | 1.3E-05 | 5.4E-05 | 1.7E-04 | 3.8E-04 |
| Playset Soil Inges-Direct Dose | 0 | 728 | 2.3E-06 | 4.2E-06 | 7.7E-07 | 9.6E-10 | 4.4E-08 | 2.2E-07 | 2.4E-06 | 1.0E-05 | 2.0E-05 | 4.0E-05 |
| Playset Surf Derm Dose | 0 | 728 | 6.5E-06 | 1.2E-05 | 3.0E-06 | 1.7E-08 | 3.3E-07 | 1.3E-06 | 6.7E-06 | 2.3E-05 | 7.1E-05 | 1.1E-04 |
| Playset Soil Derm Dose | 0 | 728 | 4.3E-07 | 5.3E-07 | 2.6E-07 | 4.0E-09 | 3.2E-08 | 1.3E-07 | 5.3E-07 | 1.4E-06 | 2.3E-06 | 5.8E-06 |
| Total Dose | 1 | 738 | 4.2E-05 | 5.9E-05 | 2.3E-05 | 9.1E-07 | 3.7E-06 | 1.1E-05 | 4.7E-05 | 1.4E-04 | 3.1E-04 | 6.1E-04 |
| Playset Total Dose | 1 | 738 | 2.0E-05 | 3.0E-05 | 1.1E-05 | 4.3E-08 | 1.5E-06 | 4.7E-06 | 2.2E-05 | 6.7E-05 | 1.4E-04 | 3.7E-04 |
| Playset Surf Inges-Hand To Mouth Dose | 1 | 738 | 1.1E-05 | 2.2E-05 | 4.5E-06 | 1.4E-08 | 3.1E-07 | 1.8E-06 | 1.2E-05 | 4.5E-05 | 9.6E-05 | 2.7E-04 |
| Playset Soil Inges-Direct Dose | 1 | 738 | 2.4E-06 | 4.4E-06 | 7.6E-07 | 5.6E-10 | 6.8E-08 | 2.9E-07 | 2.5E-06 | 1.0E-05 | 2.4E-05 | 4.0E-05 |
| Playset Surf Derm Dose | 1 | 738 | 5.7E-06 | 8.9E-06 | 2.6E-06 | 7.2E-09 | 2.5E-07 | 1.0E-06 | 6.5E-06 | 2.2E-05 | 4.8E-05 | 1.0E-04 |
| Playset Soil Derm Dose | 1 | 738 | 4.8E-07 | 7.7E-07 | 3.0E-07 | 1.5E-09 | 4.4E-08 | 1.4E-07 | 5.7E-07 | 1.4E-06 | 3.0E-06 | 1.6E-05 |
| Deck Total Dose | 1 | 738 | 2.2E-05 | 3.5E-05 | 1.0E-05 | 1.0E-08 | 1.2E-06 | 4.6E-06 | 2.4E-05 | 7.7E-05 | 1.8E-04 | 5.1E-04 |
| Deck Surf Inges-HandToMouth Dose | 1 | 738 | 1.3E-05 | 2.2E-05 | 5.7E-06 | 2.7E-09 | 5.0E-07 | 2.4E-06 | 1.4E-05 | 4.7E-05 | 1.2E-04 | 2.0E-04 |
| Deck Soil Inges-Direct Dose | 1 | 738 | 3.4E-07 | 8.4E-07 | 8.6E-08 | 3.2E-12 | 3.0E-09 | 2.2E-08 | 2.7E-07 | 1.3E-06 | 4.7E-06 | 1.1E-05 |
| Deck Surf Derm Dose | 1 | 738 | 8.1E-06 | 1.6E-05 | 3.7E-06 | 7.5E-09 | 3.8E-07 | 1.5E-06 | 8.8E-06 | 2.9E-05 | 6.4E-05 | 3.1E-04 |
| Deck Soil Derm Dose | 1 | 738 | 7.7E-08 | 1.2E-07 | 3.5E-08 | 5.0E-13 | 2.0E-09 | 1.3E-08 | 9.6E-08 | 2.7E-07 | 6.0E-07 | 1.2E-06 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in Table 14 of the SHEDS-Wood document.
The slope factor ($Q_1$*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 2. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (separated by children with and without decks) [Based on LADDs in Table 15 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 744 | 1.2E-05 | 1.8E-05 | 5.4E-06 | 1.9E-08 | 6.2E-07 | 2.4E-06 | 1.3E-05 | 4.5E-05 | 8.9E-05 | 2.0E-04 |
| Playset Total Dose | 0 | 744 | 1.2E-05 | 1.8E-05 | 5.4E-06 | 1.9E-08 | 6.2E-07 | 2.4E-06 | 1.3E-05 | 4.5E-05 | 8.9E-05 | 2.0E-04 |
| Playset Surf Inges-HandToMouth Dose | 0 | 744 | 1.0E-05 | 1.6E-05 | 4.4E-06 | 1.8E-08 | 4.6E-07 | 1.8E-06 | 1.0E-05 | 3.9E-05 | 7.8E-05 | 1.9E-04 |
| Playset Soil Inges-Direct Dose | 0 | 744 | 1.0E-07 | 3.4E-07 | 1.8E-08 | 1.0E-12 | 6.5E-10 | 4.6E-09 | 6.7E-08 | 4.2E-07 | 1.4E-06 | 4.9E-06 |
| Playset Surf Derm Dose | 0 | 744 | 1.5E-06 | 2.0E-06 | 7.7E-07 | 1.3E-09 | 9.2E-08 | 3.5E-07 | 1.8E-06 | 5.4E-06 | 9.5E-06 | 2.0E-05 |
| Playset Soil Derm Dose | 0 | 744 | 7.7E-09 | 1.8E-08 | 2.4E-09 | 5.5E-13 | 1.2E-10 | 8.9E-10 | 6.6E-09 | 3.1E-08 | 8.7E-08 | 2.6E-07 |
| Total Dose | 1 | 718 | 2.2E-05 | 3.4E-05 | 1.1E-05 | 2.7E-07 | 1.5E-06 | 5.2E-06 | 2.6E-05 | 7.8E-05 | 1.6E-04 | 3.8E-04 |
| Playset Total Dose | 1 | 718 | 1.0E-05 | 1.6E-05 | 4.7E-06 | 1.4E-07 | 5.5E-07 | 2.0E-06 | 1.2E-05 | 3.8E-05 | 7.0E-05 | 2.0E-04 |
| Playset Surf Inges-HandToMouth Dose | 1 | 718 | 8.8E-06 | 1.5E-05 | 3.8E-06 | 8.9E-08 | 3.5E-07 | 1.6E-06 | 9.9E-06 | 3.4E-05 | 6.3E-05 | 1.9E-04 |
| Playset Soil Inges-Direct Dose | 1 | 718 | 1.2E-07 | 6.2E-07 | 2.2E-08 | 1.2E-11 | 7.5E-10 | 6.0E-09 | 7.9E-08 | 4.0E-07 | 1.5E-06 | 1.5E-05 |
| Playset Surf Derm Dose | 1 | 718 | 1.4E-06 | 1.8E-06 | 7.2E-07 | 1.0E-08 | 7.8E-08 | 3.0E-07 | 1.7E-06 | 5.1E-06 | 8.9E-06 | 1.9E-05 |
| Playset Soil Derm Dose | 1 | 718 | 1.0E-08 | 3.1E-08 | 3.1E-09 | 3.5E-12 | 1.8E-10 | 1.0E-09 | 7.7E-09 | 4.2E-08 | 1.2E-07 | 6.0E-07 |
| Deck Total Dose | 1 | 718 | 1.2E-05 | 2.1E-05 | 5.4E-06 | 1.9E-08 | 6.7E-07 | 2.6E-06 | 1.3E-05 | 4.3E-05 | 8.5E-05 | 3.3E-04 |
| Deck Surf Inges-HandToMouth Dose | 1 | 718 | 9.7E-06 | 1.8E-05 | 4.2E-06 | 1.5E-08 | 3.8E-07 | 1.7E-06 | 1.1E-05 | 3.8E-05 | 7.4E-05 | 2.7E-04 |
| Deck Soil Inges-Direct Dose | 1 | 718 | 3.2E-07 | 8.8E-07 | 9.2E-08 | 5.1E-11 | 4.4E-09 | 2.4E-08 | 2.6E-07 | 1.4E-06 | 3.7E-06 | 1.7E-05 |
| Deck Surf Derm Dose | 1 | 718 | 1.8E-06 | 3.2E-06 | 8.7E-07 | 3.1E-09 | 8.6E-08 | 3.8E-07 | 2.0E-06 | 6.5E-06 | 1.2E-05 | 5.9E-05 |
| Deck Soil Derm Dose | 1 | 718 | 2.3E-08 | 3.0E-08 | 1.3E-08 | 1.9E-11 | 9.7E-10 | 5.1E-09 | 2.8E-08 | 8.1E-08 | 1.6E-07 | 2.9E-07 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in Table 15 of the SHEDS-Wood document.
The slope factor ($Q_1$*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 3. Probabilistic Estimates of Intermediate-Term MOEs for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (separated by children with and without decks) [Based on intermediate-term ADDs in Table 16 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 715 | 849 | 532 | 1812 | 1.0E+06 | 3.0E+04 | 6.1E+03 | 787 | 216 | 116 | 58 |
| Playset Total Dose | 0 | 715 | 849 | 532 | 1812 | 1.0E+06 | 3.0E+04 | 6.1E+03 | 787 | 216 | 116 | 58 |
| Playset Surf Inges-HandToMouth Dose | 0 | 715 | 1419 | 719 | 4112 | 5.9E+06 | 9.0E+04 | 1.2E+04 | 1.4E+03 | 309 | 146 | 71 |
| Playset Soil Inges-Direct Dose | 0 | 715 | 7318 | 2920 | 39739 | 1.8E+07 | 1.1E+06 | 1.5E+05 | 9.4E+03 | 1.5E+03 | 592 | 2.9E+02 |
| Playset Surf Derm Dose | 0 | 715 | 3258 | 2047 | 7693 | 8.3E+06 | 1.9E+05 | 2.3E+04 | 2.9E+03 | 806 | 387 | 2.6E+02 |
| Playset Soil Derm Dose | 0 | 715 | 3.3E+04 | 2.0E+04 | 8.1E+04 | 1.4E+07 | 1.4E+06 | 2.3E+05 | 3.1E+04 | 8.1E+03 | 4.2E+03 | 2.4E+03 |
| Total Dose | 1 | 752 | 393 | 263 | 739 | 70965 | 5681 | 1902 | 354 | 111 | 52 | 25 |
| Playset Total Dose | 1 | 752 | 779 | 417 | 1978 | 2.1E+06 | 2.6E+04 | 5.3E+03 | 800 | 190 | 78 | 45 |
| Playset Surf Inges-HandToMouth Dose | 1 | 752 | 1.4E+03 | 651 | 4.6E+03 | 3.7E+06 | 7.5E+04 | 1.7E+04 | 1.5E+03 | 301 | 121 | 60 |
| Playset Soil Inges-Direct Dose | 1 | 752 | 5.8E+03 | 1.8E+03 | 3.2E+04 | 1.6E+08 | 1.1E+06 | 1.3E+05 | 7.9E+03 | 1.5E+03 | 408 | 93 |
| Playset Surf Derm Dose | 1 | 752 | 2.9E+03 | 1.3E+03 | 8.1E+03 | 5.0E+06 | 1.3E+05 | 2.4E+04 | 2.9E+03 | 712 | 270 | 81 |
| Playset Soil Derm Dose | 1 | 752 | 3.0E+04 | 1.6E+04 | 7.2E+04 | 3.2E+08 | 1.3E+06 | 2.2E+05 | 2.8E+04 | 7.7E+03 | 3.3E+03 | 1625 |
| Deck Total Dose | 1 | 752 | 795 | 463 | 1905 | N/A | 2.8E+04 | 5.6E+03 | 706 | 225 | 85 | 46 |
| Deck Surf Inges-HandToMouth Dose | 1 | 752 | 1.3E+03 | 7.4E+02 | 3.4E+03 | N/A | 6.7E+04 | 1.1E+04 | 1.2E+03 | 343 | 1.3E+02 | 75 |
| Deck Soil Inges-Direct Dose | 1 | 752 | 4.9E+04 | 2.0E+04 | 2.3E+05 | N/A | 1.4E+07 | 9.6E+05 | 6.4E+04 | 1.1E+04 | 4.1E+03 | 1.2E+03 |
| Deck Surf Derm Dose | 1 | 752 | 2.2E+03 | 1.1E+03 | 5.2E+03 | N/A | 9.5E+04 | 1.7E+04 | 2.0E+03 | 622 | 2.5E+02 | 71 |
| Deck Soil Derm Dose | 1 | 752 | 2.0E+05 | 1.1E+05 | 6.0E+05 | N/A | 2.2E+07 | 1.8E+06 | 2.0E+05 | 5.3E+04 | 2.2E+04 | 9.8E+03 |

* MOEs calculated by dividing the LOAEL by the ADD reported in Table 16 of the SHEDS-Wood document.
The LOAEL of 0.05 mg/kg/day is used in this assessment.

Table 4. Probabilistic Estimates of Intermediate-Term MOEs for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (separated by children with and without decks) [Based on intermediate-term ADDs in Table 17 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 721 | 1.3E+03 | 355 | 4.6E+03 | | 8.8E+04 | 1.3E+04 | 1.6E+03 | 415 | 128 | 16 |
| Playset Total Dose | 0 | 721 | 1.3E+03 | 355 | 4.6E+03 | | 8.8E+04 | 1.3E+04 | 1.6E+03 | 415 | 128 | 16 |
| Playset Surf Inges-HandToMouth Dose | 0 | 721 | 1.5E+03 | 420 | 5.9E+03 | | 1.2E+05 | 1.8E+04 | 1.9E+03 | 477 | 135 | 20 |
| Playset Soil Inges-Direct Dose | 0 | 721 | 1.7E+05 | 3.6E+04 | 1.4E+06 | | 1.2E+08 | 8.4E+06 | 3.4E+05 | 4.5E+04 | 1.3E+04 | 1.9E+03 |
| Playset Surf Derm Dose | 0 | 721 | 1.1E+04 | 2.2E+03 | 3.3E+04 | | 7.7E+05 | 1.1E+05 | 1.3E+04 | 3.4E+03 | 1.3E+03 | 92 |
| Playset Soil Derm Dose | 0 | 721 | 1.9E+06 | 5.4E+05 | 1.2E+07 | | 3.7E+08 | 4.6E+07 | 3.3E+06 | 5.2E+05 | 1.2E+05 | 3.6E+04 |
| Total Dose | 1 | 742 | 7.2E+02 | 357 | 1.6E+03 | 6.9E+04 | 1.3E+04 | 3.6E+03 | 676 | 211 | 84 | 21 |
| Playset Total Dose | 1 | 742 | 1.5E+03 | 667 | 4.4E+03 | | 8.0E+04 | 1.4E+04 | 1.5E+03 | 404 | 162 | 39 |
| Playset Surf Inges-HandToMouth Dose | 1 | 742 | 1.8E+03 | 740 | 5.5E+03 | | 1.0E+05 | 1.7E+04 | 1.8E+03 | 479 | 192 | 44 |
| Playset Soil Inges-Direct Dose | 1 | 742 | 1.3E+05 | 3.1E+04 | 1.3E+06 | | 6.6E+07 | 6.8E+06 | 2.8E+05 | 2.7E+04 | 9.8E+03 | 2.0E+03 |
| Playset Surf Derm Dose | 1 | 742 | 1.2E+04 | 5.8E+03 | 3.4E+04 | | 6.9E+05 | 1.2E+05 | 1.1E+04 | 3.1E+03 | 1.1E+03 | 400 |
| Playset Soil Derm Dose | 1 | 742 | 1.5E+06 | 4.4E+05 | 8.4E+06 | | 2.8E+08 | 3.2E+07 | 2.4E+06 | 4.0E+05 | 9.9E+04 | 3.4E+04 |
| Deck Total Dose | 1 | 742 | 1.4E+03 | 467 | 3.5E+03 | | 4.1E+04 | 9.7E+03 | 1.5E+03 | 358 | 161 | 22 |
| Deck Surf Inges-HandToMouth Dose | 1 | 742 | 1.6E+03 | 503 | 4.5E+03 | | 7.4E+04 | 1.4E+04 | 1.8E+03 | 418 | 177 | 23 |
| Deck Soil Inges-Direct Dose | 1 | 742 | 6.0E+04 | 2.5E+04 | 2.3E+05 | | 8.9E+06 | 9.9E+05 | 6.8E+04 | 1.5E+04 | 5.3E+03 | 2.2E+03 |
| Deck Surf Derm Dose | 1 | 742 | 1.0E+04 | 5.3E+03 | 2.6E+04 | | 4.8E+05 | 7.2E+04 | 9.7E+03 | 2.7E+03 | 1.5E+03 | 3.7E+02 |
| Deck Soil Derm Dose | 1 | 742 | 7.7E+05 | 4.3E+05 | 1.7E+06 | | 3.1E+07 | 4.7E+06 | 7.2E+05 | 2.1E+05 | 8.0E+04 | 3.7E+04 |

* MOEs calculated by dividing the LOAEL by the ADD reported in Table 17 of the SHEDS-Wood document.
The LOAEL of 0.05 mg/kg/day is used in this assessment.

Table 5. Probabilistic Estimates of Short-Term MOEs for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (separated by children with and without decks)[Based on short-term ADDs in Table 18 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 755 | 595 | 230 | 1667 | N/A | 26449 | 4623 | 605 | 173 | 61 | 12 |
| Playset Total Dose | 0 | 755 | 595 | 230 | 1667 | N/A | 26449 | 4623 | 605 | 173 | 61 | 12 |
| Playset Surf Inges-HandToMouth Dose | 0 | 755 | 951 | 304 | 3361 | N/A | 76029 | 10925 | 1126 | 263 | 90 | 16 |
| Playset Soil Inges-Direct Dose | 0 | 755 | 6.0E+03 | 2.0E+03 | 2.9E+04 | N/A | 8.3E+05 | 1.2E+05 | 7.8E+03 | 1.2E+03 | 470 | 109 |
| Playset Surf Derm Dose | 0 | 755 | 2.3E+03 | 911 | 7.1E+03 | N/A | 156896 | 2.4E+04 | 2455 | 6.7E+02 | 192 | 60 |
| Playset Soil Derm Dose | 0 | 755 | 3.2E+04 | 1.6E+04 | 7.7E+04 | N/A | 1.1E+06 | 2.5E+05 | 3.1E+04 | 8.6E+03 | 3.1E+03 | 1.4E+03 |
| Total Dose | 1 | 710 | 383 | 261 | 771 | 61169 | 7520 | 1908 | 333 | 107 | 53 | 32 |
| Playset Total Dose | 1 | 710 | 686 | 404 | 1747 | N/A | 24985 | 5164 | 612 | 175 | 93 | 45 |
| Playset Surf Inges-Hand To Mouth Dose | 1 | 710 | 1.1E+03 | 565 | 3.4E+03 | N/A | 74934 | 1.3E+04 | 1102 | 274 | 118 | 64 |
| Playset Soil Inges-Direct Dose | 1 | 710 | 6.4E+03 | 2.0E+03 | 3.1E+04 | N/A | 8.2E+05 | 1.3E+05 | 8.1E+03 | 1609 | 427 | 134 |
| Playset Surf Derm Dose | 1 | 710 | 2.7E+03 | 1.5E+03 | 8.0E+03 | N/A | 1.5E+05 | 2.6E+04 | 2.5E+03 | 663 | 342 | 146 |
| Playset Soil Derm Dose | 1 | 710 | 2.7E+04 | 1.2E+04 | 7.6E+04 | N/A | 1.2E+06 | 2.2E+05 | 2.6E+04 | 6.8E+03 | 2.9E+03 | 694 |
| Deck Total Dose | 1 | 710 | 868 | 438 | 2380 | N/A | N/A | 8816 | 853 | 198 | 75 | 50 |
| Deck Surf Inges-Hand To Mouth Dose | 1 | 710 | 1.3E+03 | 643 | 4.4E+03 | N/A | N/A | 1.7E+04 | 1327 | 304 | 138 | 60 |
| Deck Soil Inges-Direct Dose | 1 | 710 | 6.5E+04 | 2.3E+04 | 3.7E+05 | N/A | N/A | 2.4E+06 | 1.0E+05 | 1.3E+04 | 5.0E+03 | 2.1E+03 |
| Deck Surf Derm Dose | 1 | 710 | 2.6E+03 | 1.1E+03 | 8.4E+03 | N/A | N/A | 3.3E+04 | 3.0E+03 | 643 | 254 | 100 |
| Deck Soil Derm Dose | 1 | 710 | 2.8E+05 | 1.5E+05 | 8.6E+05 | N/A | N/A | 3.7E+06 | 2.7E+05 | 6.9E+04 | 3.3E+04 | 1.4E+04 |

* MOEs calculated by dividing the LOAEL by the ADD reported in Table 18 of the SHEDS-Wood document.
The LOAEL of 0.05 mg/kg/day is used in this assessment.

Table 6. Probabilistic Estimates of Short-Term MOEs for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks
Cold Climate (separated by children with and without decks)[Based on short-term ADDs in Table 19 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 742 | 1.2E+03 | 482 | 3.6E+03 | N/A | 5.0E+05 | 1.3E+04 | 1.15E+03 | 304 | 108 | 38 |
| Playset Total Dose | 0 | 742 | 1.2E+03 | 482 | 3.6E+03 | N/A | 5.0E+05 | 1.3E+04 | 1.15E+03 | 304 | 108 | 38 |
| Playset Surf Inges-Hand To Mouth Dose | 0 | 742 | 1.3E+03 | 533 | 4.4E+03 | N/A | 5.6E+05 | 1.6E+04 | 1.34E+03 | 360 | 111 | 40 |
| Playset Soil Inges-Direct Dose | 0 | 742 | 1.2E+05 | 2.7E+04 | 1.4E+06 | N/A | 8.8E+08 | 6.8E+06 | 2.9E+05 | 3.0E+04 | 5.1E+03 | 1.7E+03 |
| Playset Surf Derm Dose | 0 | 742 | 1.0E+04 | 3.7E+03 | 3.4E+04 | N/A | 5.3E+06 | 1.0E+05 | 1.1E+04 | 2.7E+03 | 976 | 1.8E+02 |
| Playset Soil Derm Dose | 0 | 742 | 1.9E+06 | 4.8E+05 | 1.0E+07 | N/A | 1.2E+09 | 4.9E+07 | 2.8E+06 | 4.4E+05 | 1.5E+05 | 2.3E+04 |
| Total Dose | 1 | 720 | 745 | 309 | 2023 | N/A | 2.43E+04 | 4.78E+03 | 739 | 230 | 72 | 21 |
| Playset Total Dose | 1 | 720 | 1.3E+03 | 454 | 4455 | N/A | N/A | 1.48E+04 | 1.44E+03 | 328 | 118 | 26 |
| Playset Surf Inges-Hand To Mouth Dose | 1 | 720 | 1.4E+03 | 490 | 5454 | N/A | N/A | 1.86E+04 | 1.69E+03 | 369 | 126 | 28 |
| Playset Soil Inges-Direct Dose | 1 | 720 | 1.0E+05 | 1.9E+04 | 1.6E+06 | N/A | N/A | 6.6E+06 | 3.2E+05 | 2.7E+04 | 6.8E+03 | 1.0E+03 |
| Playset Surf Derm Dose | 1 | 720 | 1.2E+04 | 5.3E+03 | 3.8E+04 | N/A | N/A | 1.2E+05 | 1.2E+04 | 2.9E+03 | 1.1E+03 | 431 |
| Playset Soil Derm Dose | 1 | 720 | 1.8E+06 | 6.7E+05 | 1.0E+07 | N/A | N/A | 4.4E+07 | 2.5E+06 | 3.7E+05 | 1.5E+05 | 6.2E+04 |
| Deck Total Dose | 1 | 720 | 1.8E+03 | 563 | 6845 | N/A | N/A | 1.69E+05 | 2.19E+03 | 488 | 175 | 30 |
| Deck Surf Inges-Hand To Mouth Dose | 1 | 720 | 2.1E+03 | 621 | 9294 | N/A | N/A | 1.02E+06 | 2.70E+03 | 556 | 218 | 32 |
| Deck Soil Inges-Direct Dose | 1 | 720 | 6.5E+04 | 2.1E+04 | 6.0E+05 | N/A | N/A | 1.3E+08 | 1.0E+05 | 1.3E+04 | 4.8E+03 | 1.4E+03 |
| Deck Surf Derm Dose | 1 | 720 | 1.5E+04 | 5.6E+03 | 5.8E+04 | N/A | N/A | 4.1E+06 | 1.7E+04 | 3.6E+03 | 1.5E+03 | 350 |
| Deck Soil Derm Dose | 1 | 720 | 9.2E+05 | 3.7E+05 | 3.7E+06 | N/A | N/A | 9.1E+08 | 1.0E+06 | 1.9E+05 | 9.0E+04 | 2.1E+04 |

* MOEs calculated by dividing the LOAEL by the ADD reported in Table 19 of the SHEDS-Wood document.
The LOAEL of 0.05 mg/kg/day is used in this assessment.

Table 7. Probabilistic Estimates of Short-Term MOEs for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (children with PICA behavior) [Based on short-term ADD from Table 33 in SHEDS-Wood document]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Playset Total Dose | 0 | 288 | 219 | 125 | 503 | 2.3E+05 | 7.9E+03 | 1.3E+03 | 207 | 65 | 23 | 14 |
| Playset Surf Inges-HandToMouth Dose | 0 | 288 | 1.3E+03 | 781 | 3.3E+03 | 8.0E+05 | 5.4E+04 | 9.7E+03 | 1.4E+03 | 250 | 170 | 121 |
| Playset Soil Inges-Direct Dose | 0 | 288 | 299 | 135 | 955 | 5.7E+05 | 1.8E+04 | 2.3E+03 | 336 | 69 | 24 | 15 |
| Playset Surf Derm Dose | 0 | 288 | 2.6E+03 | 1.4E+03 | 6.6E+03 | 1.4E+06 | 1.5E+05 | 2.4E+04 | 2.4E+03 | 624 | 266 | 144 |
| Playset Soil Derm Dose | 0 | 288 | 2.5E+04 | 1.3E+04 | 5.7E+04 | 4.8E+07 | 2.2E+06 | 2.3E+05 | 2.6E+04 | 6.2E+03 | 2.2E+03 | 1.6E+03 |
| | | | | | | | | | | | | |
| Total Dose | 1 | 318 | 163 | 94 | 339 | 1.2E+04 | 1.4E+03 | 6.5E+02 | 155 | 49 | 31 | 7 |
| Playset Total Dose | 1 | 318 | 207 | 102 | 549 | 3.0E+04 | 3.6E+03 | 1.2E+03 | 193 | 59 | 32 | 8 |
| Playset Surf Inges-HandToMouth Dose | 1 | 318 | 1.1E+03 | 416 | 3.7E+03 | 1.4E+06 | 4.9E+04 | 8.9E+03 | 1.3E+03 | 233 | 93 | 32 |
| Playset Soil Inges-Direct Dose | 1 | 318 | 287 | 115 | 955 | 3.3E+04 | 6.8E+03 | 2.3E+03 | 3.3E+02 | 66 | 33 | 8 |
| Playset Surf Derm Dose | 1 | 318 | 2.6E+03 | 1.4E+03 | 6.5E+03 | 9.6E+05 | 1.3E+05 | 1.9E+04 | 2.7E+03 | 698 | 288 | 157 |
| Playset Soil Derm Dose | 1 | 318 | 2.6E+04 | 1.7E+04 | 5.9E+04 | 4.4E+06 | 8.0E+05 | 1.6E+05 | 2.3E+04 | 6.0E+03 | 3.6E+03 | 2.3E+03 |
| Deck Total Dose | 1 | 318 | 7.8E+02 | 404 | 2.3E+03 | | | 7.3E+03 | 834 | 183 | 72 | 52 |
| Deck Surf Inges-HandToMouth Dose | 1 | 318 | 1.4E+03 | 609 | 6.1E+03 | | | 2.5E+04 | 2.0E+03 | 302 | 116 | 68 |
| Deck Soil Inges-Direct Dose | 1 | 318 | 3.7E+03 | 1.5E+03 | 1.4E+04 | | | 1.1E+05 | 3.8E+03 | 787 | 461 | 104 |
| Deck Surf Derm Dose | 1 | 318 | 3.1E+03 | 1.2E+03 | 1.2E+04 | | | 4.5E+04 | 3.8E+03 | 822 | 304 | 90 |
| Deck Soil Derm Dose | 1 | 318 | 2.8E+05 | 1.2E+05 | 1.0E+06 | | | 6.9E+06 | 3.3E+05 | 6.2E+04 | 2.2E+04 | 1.2E+04 |

* MOEs calculated by dividing the LOAEL by the ADD reported in Table 33 of the SHEDS-Wood document.
The LOAEL of 0.05 mg/kg/day is used in this assessment.

Table 8. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (Dermal Residue Absorption Rate =0.01%)[Based on LADDs in Table 35 from the SHEDS-Wood Document]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 731 | 1.5E-05 | 3.3E-05 | 7.3E-06 | 1.2E-08 | 1.0E-06 | 3.4E-06 | 1.6E-05 | 4.4E-05 | 1.2E-04 | 7.1E-04 |
| Playset Total Dose | 0 | 731 | 1.5E-05 | 3.3E-05 | 7.3E-06 | 1.2E-08 | 1.0E-06 | 3.4E-06 | 1.6E-05 | 4.4E-05 | 1.2E-04 | 7.1E-04 |
| Playset Surf Inges-HandToMouth Dose | 0 | 731 | 1.1E-05 | 3.2E-05 | 4.0E-06 | 7.7E-09 | 3.7E-07 | 1.6E-06 | 1.1E-05 | 3.6E-05 | 1.1E-04 | 7.1E-04 |
| Playset Soil Inges-Direct Dose | 0 | 731 | 2.7E-06 | 5.7E-06 | 9.5E-07 | 9.5E-10 | 5.3E-08 | 3.1E-07 | 2.9E-06 | 1.1E-05 | 2.8E-05 | 8.9E-05 |
| Playset Surf Derm Dose | 0 | 731 | 1.8E-08 | 3.8E-08 | 8.4E-09 | 2.6E-11 | 9.6E-10 | 3.4E-09 | 2.1E-08 | 6.0E-08 | 1.4E-07 | 7.5E-07 |
| Playset Soil Derm Dose | 0 | 731 | 6.1E-07 | 7.7E-07 | 4.0E-07 | 6.8E-10 | 4.7E-08 | 1.8E-07 | 7.5E-07 | 1.8E-06 | 3.7E-06 | 9.4E-06 |
| Total Dose | 1 | 740 | 2.9E-05 | 5.0E-05 | 1.7E-05 | 4.1E-07 | 2.3E-06 | 7.3E-06 | 3.2E-05 | 9.8E-05 | 2.3E-04 | 8.6E-04 |
| Playset Total Dose | 1 | 740 | 1.4E-05 | 2.1E-05 | 7.6E-06 | 6.3E-08 | 1.0E-06 | 3.3E-06 | 1.6E-05 | 4.1E-05 | 1.2E-04 | 2.9E-04 |
| Playset Surf Inges-HandToMouth Dose | 1 | 740 | 1.1E-05 | 2.1E-05 | 5.5E-06 | 3.8E-08 | 4.0E-07 | 1.9E-06 | 1.3E-05 | 3.9E-05 | 1.1E-04 | 2.9E-04 |
| Playset Soil Inges-Direct Dose | 1 | 740 | 1.7E-06 | 3.3E-06 | 5.5E-07 | 4.1E-09 | 3.6E-08 | 2.1E-07 | 1.7E-06 | 7.8E-06 | 1.5E-05 | 3.2E-05 |
| Playset Surf Derm Dose | 1 | 740 | 1.9E-08 | 3.1E-08 | 9.9E-09 | 5.1E-11 | 1.2E-09 | 4.3E-09 | 2.1E-08 | 6.1E-08 | 1.6E-07 | 3.7E-07 |
| Playset Soil Derm Dose | 1 | 740 | 3.7E-07 | 4.7E-07 | 2.2E-07 | 1.7E-09 | 2.8E-08 | 1.1E-07 | 4.5E-07 | 1.1E-06 | 2.7E-06 | 5.2E-06 |
| Deck Total Dose | 1 | 740 | 1.6E-05 | 3.3E-05 | 6.9E-06 | 1.3E-07 | 6.9E-07 | 2.7E-06 | 1.7E-05 | 5.6E-05 | 1.3E-04 | 5.7E-04 |
| Deck Surf Inges-HandToMouth Dose | 1 | 740 | 1.5E-05 | 3.3E-05 | 6.4E-06 | 5.9E-08 | 4.9E-07 | 2.3E-06 | 1.7E-05 | 5.6E-05 | 1.2E-04 | 5.7E-04 |
| Deck Soil Inges-Direct Dose | 1 | 740 | 3.3E-07 | 1.0E-06 | 8.8E-08 | 5.3E-11 | 2.8E-09 | 2.2E-08 | 2.6E-07 | 1.3E-06 | 3.8E-06 | 1.6E-05 |
| Deck Surf Derm Dose | 1 | 740 | 2.9E-08 | 5.9E-08 | 1.4E-08 | 8.8E-11 | 1.2E-09 | 5.5E-09 | 3.2E-08 | 8.7E-08 | 1.9E-07 | 9.0E-07 |
| Deck Soil Derm Dose | 1 | 740 | 7.9E-08 | 1.1E-07 | 4.2E-08 | 1.6E-12 | 1.3E-09 | 1.5E-08 | 9.3E-08 | 3.0E-07 | 5.6E-07 | 9.3E-07 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 35 of the SHEDS-Wood document.
The slope factor ($Q_1$*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 9. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Cold Climate (Dermal Residue Absorption Rate =0.01%)[Based on LADDs in the Table 36 from the SHEDS-Wood Document]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 761 | 1.1E-05 | 2.4E-05 | 4.2E-06 | 4.4E-08 | 5.6E-07 | 1.8E-06 | 1.1E-05 | 3.8E-05 | 9.9E-05 | 4.3E-04 |
| Playset Total Dose | 0 | 761 | 1.1E-05 | 2.4E-05 | 4.2E-06 | 4.4E-08 | 5.6E-07 | 1.8E-06 | 1.1E-05 | 3.8E-05 | 9.9E-05 | 4.3E-04 |
| Playset Surf Inges-HandToMouth Dose | 0 | 761 | 1.0E-05 | 2.4E-05 | 3.9E-06 | 4.4E-08 | 4.1E-07 | 1.6E-06 | 1.0E-05 | 3.8E-05 | 9.9E-05 | 4.3E-04 |
| Playset Soil Inges-Direct Dose | 0 | 761 | 1.9E-07 | 4.9E-07 | 4.5E-08 | 1.3E-10 | 1.6E-09 | 1.1E-08 | 1.6E-07 | 7.2E-07 | 2.8E-06 | 6.6E-06 |
| Playset Surf Derm Dose | 0 | 761 | 5.3E-09 | 1.1E-08 | 2.3E-09 | 6.0E-11 | 3.4E-10 | 1.0E-09 | 5.6E-09 | 1.8E-08 | 4.3E-08 | 1.9E-07 |
| Playset Soil Derm Dose | 0 | 761 | 1.5E-08 | 2.8E-08 | 5.5E-09 | 3.4E-11 | 4.3E-10 | 2.0E-09 | 1.5E-08 | 5.4E-08 | 1.4E-07 | 3.0E-07 |
| Total Dose | 1 | 713 | 2.0E-05 | 3.1E-05 | 9.8E-06 | 1.7E-07 | 1.3E-06 | 4.5E-06 | 2.0E-05 | 7.7E-05 | 1.6E-04 | 3.0E-04 |
| Playset Total Dose | 1 | 713 | 9.4E-06 | 1.6E-05 | 4.3E-06 | 2.9E-08 | 4.6E-07 | 1.9E-06 | 9.8E-06 | 3.5E-05 | 9.1E-05 | 1.6E-04 |
| Playset Surf Inges-HandToMouth Dose | 1 | 713 | 9.3E-06 | 1.6E-05 | 4.2E-06 | 2.9E-08 | 4.2E-07 | 1.9E-06 | 9.7E-06 | 3.5E-05 | 9.1E-05 | 1.6E-04 |
| Playset Soil Inges-Direct Dose | 1 | 713 | 4.6E-08 | 1.4E-07 | 9.7E-09 | 1.5E-12 | 3.6E-10 | 2.6E-09 | 3.6E-08 | 2.0E-07 | 5.8E-07 | 2.7E-06 |
| Playset Surf Derm Dose | 1 | 713 | 4.9E-09 | 7.6E-09 | 2.4E-09 | 1.8E-11 | 3.2E-10 | 1.2E-09 | 5.8E-09 | 1.6E-08 | 3.4E-08 | 8.1E-08 |
| Playset Soil Derm Dose | 1 | 713 | 3.6E-09 | 7.8E-09 | 1.2E-09 | 9.0E-12 | 1.0E-10 | 4.3E-10 | 3.5E-09 | 1.5E-08 | 3.3E-08 | 1.1E-07 |
| Deck Total Dose | 1 | 713 | 1.0E-05 | 1.7E-05 | 5.1E-06 | 3.6E-08 | 5.9E-07 | 2.1E-06 | 1.1E-05 | 3.7E-05 | 9.0E-05 | 1.6E-04 |
| Deck Surf Inges-HandToMouth Dose | 1 | 713 | 1.0E-05 | 1.7E-05 | 4.8E-06 | 2.0E-08 | 3.7E-07 | 1.8E-06 | 1.0E-05 | 3.7E-05 | 8.8E-05 | 1.6E-04 |
| Deck Soil Inges-Direct Dose | 1 | 713 | 2.9E-07 | 6.9E-07 | 8.1E-08 | 2.3E-10 | 3.3E-09 | 2.4E-08 | 2.4E-07 | 1.2E-06 | 3.4E-06 | 9.9E-06 |
| Deck Surf Derm Dose | 1 | 713 | 5.9E-09 | 9.7E-09 | 3.0E-09 | 2.8E-11 | 3.5E-10 | 1.4E-09 | 6.9E-09 | 1.9E-08 | 4.7E-08 | 1.3E-07 |
| Deck Soil Derm Dose | 1 | 713 | 2.2E-08 | 3.1E-08 | 1.2E-08 | 1.7E-11 | 8.6E-10 | 4.5E-09 | 2.6E-08 | 7.9E-08 | 1.6E-07 | 3.8E-07 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 39 of the SHEDS-Wood document.
The slope factor ($Q_1$*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 10. Probabilistic Cancer Risk Estimates of Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (reducing deck and playset residue concentration by 90%) [Based on LADDs in Table 37 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 700 | 3.3E-06 | 4.0E-06 | 2.0E-06 | 1.2E-08 | 2.6E-07 | 9.4E-07 | 3.9E-06 | 1.1E-05 | 2.1E-05 | 4.2E-05 |
| Playset Total Dose | 0 | 700 | 3.3E-06 | 4.0E-06 | 2.0E-06 | 1.2E-08 | 2.6E-07 | 9.4E-07 | 3.9E-06 | 1.1E-05 | 2.1E-05 | 4.2E-05 |
| Playset Surf Inges-Hand To Mouth Dose | 0 | 700 | 8.2E-07 | 1.4E-06 | 3.5E-07 | 7.2E-10 | 1.8E-08 | 1.2E-07 | 9.1E-07 | 3.0E-06 | 7.1E-06 | 1.7E-05 |
| Playset Soil Inges-Direct Dose | 0 | 700 | 1.8E-06 | 3.3E-06 | 6.3E-07 | 1.2E-09 | 3.5E-08 | 2.0E-07 | 1.9E-06 | 7.0E-06 | 1.7E-05 | 4.2E-05 |
| Playset Surf Derm Dose | 0 | 700 | 4.3E-07 | 6.5E-07 | 2.0E-07 | 1.8E-09 | 1.6E-08 | 7.6E-08 | 5.1E-07 | 1.6E-06 | 3.5E-06 | 5.8E-06 |
| Playset Surf Derm Dose | 0 | 700 | 2.7E-07 | 3.1E-07 | 1.8E-07 | 3.1E-09 | 3.2E-08 | 9.4E-08 | 3.2E-07 | 7.8E-07 | 1.9E-06 | 2.6E-06 |
| Total Dose | 1 | 759 | 6.6E-06 | 8.3E-06 | 3.9E-06 | 7.1E-08 | 7.1E-07 | 1.9E-06 | 8.6E-06 | 2.1E-05 | 3.7E-05 | 1.2E-04 |
| Playset Total Dose | 1 | 759 | 4.0E-06 | 5.1E-06 | 2.3E-06 | 2.5E-09 | 3.1E-07 | 1.0E-06 | 4.9E-06 | 1.4E-05 | 2.7E-05 | 3.9E-05 |
| Playset Surf Inges-Hand To Mouth Dose | 1 | 759 | 8.8E-07 | 1.9E-06 | 2.6E-07 | 2.3E-10 | 1.3E-08 | 7.8E-08 | 8.2E-07 | 3.7E-06 | 9.1E-06 | 2.1E-05 |
| Playset Soil Inges-Direct Dose | 1 | 759 | 2.4E-06 | 4.3E-06 | 8.8E-07 | 1.2E-10 | 5.3E-08 | 3.1E-07 | 2.5E-06 | 9.6E-06 | 2.5E-05 | 3.9E-05 |
| Playset Surf Derm Dose | 1 | 759 | 4.3E-07 | 7.2E-07 | 1.7E-07 | 1.1E-10 | 1.2E-08 | 6.1E-08 | 4.6E-07 | 1.6E-06 | 3.7E-06 | 6.2E-06 |
| Playset Soil Derm Dose | 1 | 759 | 3.1E-07 | 3.7E-07 | 2.0E-07 | 9.5E-10 | 2.9E-08 | 1.1E-07 | 3.8E-07 | 9.8E-07 | 1.8E-06 | 3.9E-06 |
| Deck Total Dose | 1 | 759 | 2.5E-06 | 4.9E-06 | 1.2E-06 | 7.0E-09 | 1.6E-07 | 5.6E-07 | 2.8E-06 | 8.4E-06 | 2.0E-05 | 9.4E-05 |
| Deck Surf Inges-Hand To Mouth Dose | 1 | 759 | 1.4E-06 | 3.7E-06 | 4.7E-07 | 4.0E-10 | 2.7E-08 | 1.8E-07 | 1.4E-06 | 5.6E-06 | 1.4E-05 | 7.6E-05 |
| Deck Soil Inges-Direct Dose | 1 | 759 | 2.9E-07 | 5.4E-07 | 9.0E-08 | 7.9E-12 | 3.2E-09 | 2.5E-08 | 2.8E-07 | 1.4E-06 | 2.9E-06 | 4.4E-06 |
| Deck Surf Derm Dose | 1 | 759 | 7.5E-07 | 1.3E-06 | 3.2E-07 | 3.2E-10 | 2.0E-08 | 1.3E-07 | 8.0E-07 | 2.8E-06 | 5.8E-06 | 1.8E-05 |
| Deck Soil Derm Dose | 1 | 759 | 6.0E-08 | 6.9E-08 | 3.5E-08 | 2.9E-11 | 2.1E-09 | 1.3E-08 | 8.0E-08 | 2.1E-07 | 3.4E-07 | 4.8E-07 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 37 of the SHEDS-Wood document.
The slope factor ($Q_1$*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 11. Probabilistic Estimates of Cancer Risk for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks
Warm Climate (reducing deck and playset residue concentration by 99.5%)[Based on the Table 38 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 743 | 2.0E-06 | 3.5E-06 | 8.1E-07 | 3.6E-09 | 8.6E-08 | 3.1E-07 | 2.3E-06 | 8.2E-06 | 1.7E-05 | 3.3E-05 |
| Playset Total Dose | 0 | 743 | 2.0E-06 | 3.5E-06 | 8.1E-07 | 3.6E-09 | 8.6E-08 | 3.1E-07 | 2.3E-06 | 8.2E-06 | 1.7E-05 | 3.3E-05 |
| Playset Surf Inges-HandToMouth Dose | 0 | 743 | 2.1E-08 | 8.2E-08 | 2.5E-09 | 2.1E-12 | 7.7E-11 | 5.5E-10 | 9.8E-09 | 8.0E-08 | 2.8E-07 | 1.6E-06 |
| Playset Soil Inges-Direct Dose | 0 | 743 | 1.9E-06 | 3.4E-06 | 6.9E-07 | 1.0E-09 | 4.5E-08 | 2.3E-07 | 2.0E-06 | 8.1E-06 | 1.7E-05 | 3.3E-05 |
| Playset Surf Derm Dose | 0 | 743 | 1.2E-08 | 3.3E-08 | 2.3E-09 | 4.7E-12 | 8.2E-11 | 5.8E-10 | 8.0E-09 | 5.4E-08 | 1.5E-07 | 5.2E-07 |
| Playset Soil Derm Dose | 0 | 743 | 6.0E-08 | 1.0E-07 | 3.3E-08 | 1.2E-09 | 5.2E-09 | 1.5E-08 | 6.8E-08 | 1.9E-07 | 4.5E-07 | 2.0E-06 |
| Total Dose | 1 | 721 | 2.9E-06 | 4.6E-06 | 1.3E-06 | 1.0E-08 | 1.6E-07 | 5.3E-07 | 3.4E-06 | 1.1E-05 | 2.9E-05 | 3.8E-05 |
| Playset Total Dose | 1 | 721 | 2.5E-06 | 4.3E-06 | 1.1E-06 | 2.7E-09 | 8.9E-08 | 3.7E-07 | 2.9E-06 | 1.0E-05 | 2.2E-05 | 3.7E-05 |
| Playset Surf Inges-HandToMouth Dose | 1 | 721 | 1.2E-08 | 4.1E-08 | 1.9E-09 | 2.8E-12 | 3.8E-11 | 5.3E-10 | 8.1E-09 | 4.4E-08 | 2.1E-07 | 6.1E-07 |
| Playset Soil Inges-Direct Dose | 1 | 721 | 2.5E-06 | 4.3E-06 | 1.0E-06 | 1.5E-09 | 5.0E-08 | 2.9E-07 | 2.8E-06 | 9.9E-06 | 2.2E-05 | 3.7E-05 |
| Playset Surf Derm Dose | 1 | 721 | 7.2E-09 | 2.0E-08 | 1.6E-09 | 3.7E-12 | 4.9E-11 | 4.7E-10 | 5.6E-09 | 2.6E-08 | 1.0E-07 | 2.5E-07 |
| Playset Soil Derm Dose | 1 | 721 | 5.5E-08 | 7.3E-08 | 3.1E-08 | 6.2E-10 | 4.3E-09 | 1.4E-08 | 6.5E-08 | 1.9E-07 | 3.6E-07 | 7.3E-07 |
| Deck Total Dose | 1 | 721 | 4.0E-07 | 7.7E-07 | 1.8E-07 | 1.8E-09 | 1.8E-08 | 7.3E-08 | 4.3E-07 | 1.4E-06 | 3.3E-06 | 1.2E-05 |
| Deck Surf Inges-HandToMouth Dose | 1 | 721 | 4.7E-08 | 1.6E-07 | 1.1E-08 | 4.7E-12 | 2.3E-10 | 2.5E-09 | 3.8E-08 | 1.8E-07 | 5.6E-07 | 2.5E-06 |
| Deck Soil Inges-Direct Dose | 1 | 721 | 3.0E-07 | 7.2E-07 | 8.8E-08 | 3.9E-11 | 2.4E-09 | 2.3E-08 | 3.0E-07 | 1.2E-06 | 2.8E-06 | 1.1E-05 |
| Deck Surf Derm Dose | 1 | 721 | 2.8E-08 | 7.8E-08 | 8.1E-09 | 8.9E-12 | 3.0E-10 | 2.1E-09 | 2.7E-08 | 1.0E-07 | 2.9E-07 | 1.5E-06 |
| Deck Soil Derm Dose | 1 | 721 | 2.0E-08 | 2.4E-08 | 1.3E-08 | 8.8E-11 | 1.4E-09 | 6.0E-09 | 2.6E-08 | 6.7E-08 | 1.3E-07 | 1.9E-07 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 38 of the SHEDS-Wood document.
The slope factor (Q*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 12. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (reducing exposure washing hands after playing on deck or playset) [Based on LADDs in the Table 39 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 747 | 1.3E-05 | 1.6E-05 | 7.5E-06 | 2.6E-08 | 1.0E-06 | 3.6E-06 | 1.7E-05 | 4.6E-05 | 7.3E-05 | 1.7E-04 |
| Playset Total Dose | 0 | 747 | 1.3E-05 | 1.6E-05 | 7.5E-06 | 2.6E-08 | 1.0E-06 | 3.6E-06 | 1.7E-05 | 4.6E-05 | 7.3E-05 | 1.7E-04 |
| Playset Surf Inges-HandToMouth Dose | 0 | 747 | 6.0E-06 | 9.5E-06 | 2.7E-06 | 9.6E-09 | 1.9E-07 | 9.4E-07 | 7.1E-06 | 2.1E-05 | 3.9E-05 | 1.1E-04 |
| Playset Soil Inges-Direct Dose | 0 | 747 | 2.1E-06 | 4.5E-06 | 6.7E-07 | 1.3E-09 | 3.7E-08 | 2.0E-07 | 2.2E-06 | 8.1E-06 | 2.4E-05 | 5.2E-05 |
| Playset Surf Derm Dose | 0 | 747 | 5.0E-06 | 6.7E-06 | 2.7E-06 | 4.3E-09 | 2.5E-07 | 1.2E-06 | 6.3E-06 | 1.9E-05 | 3.3E-05 | 5.5E-05 |
| Playset Soil Derm Dose | 0 | 747 | 3.9E-07 | 5.4E-07 | 2.4E-07 | 4.0E-09 | 2.4E-08 | 1.1E-07 | 4.6E-07 | 1.3E-06 | 2.9E-06 | 6.4E-06 |
| Total Dose | 1 | 704 | 3.1E-05 | 4.6E-05 | 1.8E-05 | 4.7E-07 | 3.3E-06 | 9.1E-06 | 3.5E-05 | 1.0E-04 | 2.6E-04 | 4.5E-04 |
| Playset Total Dose | 1 | 704 | 1.4E-05 | 2.0E-05 | 8.2E-06 | 2.3E-07 | 1.2E-06 | 4.1E-06 | 1.6E-05 | 4.5E-05 | 1.0E-04 | 2.0E-04 |
| Playset Surf Inges-Hand To Mouth Dose | 1 | 704 | 5.9E-06 | 1.2E-05 | 2.7E-06 | 1.1E-08 | 2.4E-07 | 1.1E-06 | 5.8E-06 | 2.0E-05 | 6.1E-05 | 1.5E-04 |
| Playset Soil Inges-Direct Dose | 1 | 704 | 2.3E-06 | 4.0E-06 | 8.4E-07 | 1.3E-08 | 6.1E-08 | 3.0E-07 | 2.6E-06 | 8.7E-06 | 2.0E-05 | 4.8E-05 |
| Playset Surf Derm Dose | 1 | 704 | 5.0E-06 | 8.5E-06 | 2.6E-06 | 4.0E-08 | 2.6E-07 | 1.1E-06 | 5.8E-06 | 1.5E-05 | 3.8E-05 | 1.0E-04 |
| Playset Soil Derm Dose | 1 | 704 | 5.0E-07 | 6.0E-07 | 3.1E-07 | 3.8E-09 | 4.7E-08 | 1.4E-07 | 6.3E-07 | 1.7E-06 | 2.8E-06 | 6.1E-06 |
| Deck Total Dose | 1 | 704 | 1.7E-05 | 3.2E-05 | 7.5E-06 | 5.7E-09 | 7.4E-07 | 3.3E-06 | 1.8E-05 | 5.6E-05 | 1.7E-04 | 3.8E-04 |
| Deck Surf Inges-Hand To Mouth Dose | 1 | 704 | 8.5E-06 | 1.6E-05 | 3.3E-06 | 3.1E-09 | 2.5E-07 | 1.3E-06 | 8.8E-06 | 2.9E-05 | 9.1E-05 | 1.7E-04 |
| Deck Soil Inges-Direct Dose | 1 | 704 | 3.1E-07 | 6.9E-07 | 7.9E-08 | 1.9E-11 | 2.3E-09 | 2.1E-08 | 2.8E-07 | 1.4E-06 | 3.6E-06 | 7.3E-06 |
| Deck Surf Derm Dose | 1 | 704 | 8.2E-06 | 1.8E-05 | 3.4E-06 | 2.4E-09 | 2.8E-07 | 1.4E-06 | 8.4E-06 | 2.9E-05 | 6.5E-05 | 2.2E-04 |
| Deck Soil Derm Dose | 1 | 704 | 7.5E-08 | 1.2E-07 | 3.6E-08 | 3.3E-11 | 1.7E-09 | 1.2E-08 | 8.9E-08 | 2.9E-07 | 4.9E-07 | 1.3E-06 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 39 of the SHEDS-Wood document.
The slope factor ($Q_1$*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 13. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (simulating 90% residue reduction and hand washing to reduce exposure) [Based on LADDs in the revised Table 40 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 713 | 3.6E-06 | 5.3E-06 | 1.9E-06 | 1.6E-08 | 2.7E-07 | 9.1E-07 | 4.5E-06 | 1.2E-05 | 2.3E-05 | 6.8E-05 |
| Playset Total Dose | 0 | 713 | 3.6E-06 | 5.3E-06 | 1.9E-06 | 1.6E-08 | 2.7E-07 | 9.1E-07 | 4.5E-06 | 1.2E-05 | 2.3E-05 | 6.8E-05 |
| Playset Surf Inges-HandToMouth Dose | 0 | 713 | 5.9E-07 | 1.2E-06 | 2.1E-07 | 6.9E-10 | 1.1E-08 | 6.9E-08 | 5.9E-07 | 2.5E-06 | 6.5E-06 | 1.0E-05 |
| Playset Soil Inges-Direct Dose | 0 | 713 | 2.1E-06 | 4.4E-06 | 7.8E-07 | 1.2E-09 | 4.5E-08 | 2.3E-07 | 2.1E-06 | 9.1E-06 | 1.7E-05 | 6.5E-05 |
| Playset Surf Derm Dose | 0 | 713 | 5.6E-07 | 1.6E-06 | 2.2E-07 | 2.2E-09 | 1.2E-08 | 8.5E-08 | 5.7E-07 | 1.7E-06 | 4.8E-06 | 3.7E-05 |
| Playset Soil Derm Dose | 0 | 713 | 3.0E-07 | 4.1E-07 | 1.7E-07 | 2.4E-09 | 2.8E-08 | 8.4E-08 | 3.5E-07 | 1.0E-06 | 2.0E-06 | 4.6E-06 |
| Total Dose | 1 | 756 | 5.8E-06 | 9.2E-06 | 3.3E-06 | 1.4E-07 | 6.5E-07 | 1.8E-06 | 6.8E-06 | 1.9E-05 | 3.1E-05 | 1.9E-04 |
| Playset Total Dose | 1 | 756 | 3.6E-06 | 4.3E-06 | 2.0E-06 | 3.8E-09 | 3.0E-07 | 9.7E-07 | 4.5E-06 | 1.4E-05 | 2.1E-05 | 3.2E-05 |
| Playset Surf Inges-HandToMouth Dose | 1 | 756 | 6.1E-07 | 1.5E-06 | 1.7E-07 | 2.6E-10 | 8.9E-09 | 5.2E-08 | 5.2E-07 | 2.4E-06 | 7.7E-06 | 2.0E-05 |
| Playset Soil Inges-Direct Dose | 1 | 756 | 2.2E-06 | 3.5E-06 | 8.2E-07 | 4.9E-10 | 5.4E-08 | 3.1E-07 | 2.7E-06 | 9.2E-06 | 1.7E-05 | 2.8E-05 |
| Playset Surf Derm Dose | 1 | 756 | 4.5E-07 | 8.0E-07 | 1.9E-07 | 1.0E-09 | 1.4E-08 | 6.6E-08 | 4.5E-07 | 1.8E-06 | 4.7E-06 | 7.6E-06 |
| Playset Soil Derm Dose | 1 | 756 | 3.1E-07 | 3.7E-07 | 2.0E-07 | 1.3E-09 | 3.5E-08 | 9.6E-08 | 3.8E-07 | 9.6E-07 | 2.0E-06 | 3.0E-06 |
| Deck Total Dose | 1 | 756 | 2.2E-06 | 6.5E-06 | 9.9E-07 | 1.8E-09 | 1.1E-07 | 4.6E-07 | 2.2E-06 | 7.7E-06 | 1.7E-05 | 1.6E-04 |
| Deck Surf Inges-HandToMouth Dose | 1 | 756 | 1.1E-06 | 4.3E-06 | 2.9E-07 | 4.5E-10 | 1.7E-08 | 1.0E-07 | 8.8E-07 | 4.1E-06 | 1.1E-05 | 1.1E-04 |
| Deck Soil Inges-Direct Dose | 1 | 756 | 2.8E-07 | 5.9E-07 | 7.5E-08 | 7.2E-11 | 2.0E-09 | 2.2E-08 | 2.7E-07 | 1.4E-06 | 2.5E-06 | 8.4E-06 |
| Deck Surf Derm Dose | 1 | 756 | 8.3E-07 | 2.3E-06 | 3.2E-07 | 9.8E-10 | 2.4E-08 | 1.2E-07 | 8.1E-07 | 3.4E-06 | 7.8E-06 | 4.9E-05 |
| Deck Soil Derm Dose | 1 | 756 | 6.0E-08 | 8.9E-08 | 3.3E-08 | 1.3E-11 | 1.9E-09 | 1.1E-08 | 7.5E-08 | 2.0E-07 | 3.9E-07 | 1.2E-06 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 40 of the SHEDS-Wood document.
The slope factor (Q*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

Table 14. Probabilistic Estimates of Cancer Risks for Children Exposed to Arsenic Dislodgeable Residues and Contaminated Soil from Treated Wood Playsets and Residential Decks in Warm Climate (simulating 99.5% residue reduction and hand washing to reduce exposure[Based on LADDs in the revised Table 41 from the SHEDS-Wood Document.]*

| Pathway | Deck | n | mean | std | p50 | min | p05 | p25 | p75 | p95 | p99 | max |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total Dose | 0 | 729 | 2.1E-06 | 3.5E-06 | 7.5E-07 | 1.3E-08 | 7.2E-08 | 2.6E-07 | 2.1E-06 | 9.3E-06 | 1.7E-05 | 3.0E-05 |
| Playset Total Dose | 0 | 729 | 2.1E-06 | 3.5E-06 | 7.5E-07 | 1.3E-08 | 7.2E-08 | 2.6E-07 | 2.1E-06 | 9.3E-06 | 1.7E-05 | 3.0E-05 |
| Playset Surf Inges-HandToMouth Dose | 0 | 729 | 9.7E-09 | 2.8E-08 | 1.9E-09 | 3.0E-12 | 6.3E-11 | 4.9E-10 | 7.7E-09 | 4.3E-08 | 1.1E-07 | 3.7E-07 |
| Playset Soil Inges-Direct Dose | 0 | 729 | 2.0E-06 | 3.5E-06 | 6.7E-07 | 3.2E-09 | 3.6E-08 | 2.0E-07 | 2.0E-06 | 9.2E-06 | 1.7E-05 | 3.0E-05 |
| Playset Surf Derm Dose | 0 | 729 | 9.6E-09 | 2.2E-08 | 2.7E-09 | 4.9E-12 | 1.1E-10 | 7.3E-10 | 8.2E-09 | 4.0E-08 | 1.2E-07 | 2.5E-07 |
| Playset Soil Derm Dose | 0 | 729 | 5.9E-08 | 7.7E-08 | 3.4E-08 | 6.6E-10 | 5.1E-09 | 1.5E-08 | 7.2E-08 | 1.9E-07 | 3.7E-07 | 7.7E-07 |
| Total Dose | 1 | 738 | 3.4E-06 | 6.7E-06 | 1.3E-06 | 4.0E-08 | 1.6E-07 | 5.3E-07 | 3.4E-06 | 1.2E-05 | 4.2E-05 | 6.8E-05 |
| Playset Total Dose | 1 | 738 | 3.0E-06 | 6.4E-06 | 1.0E-06 | 5.5E-09 | 1.1E-07 | 3.8E-07 | 2.8E-06 | 1.1E-05 | 4.1E-05 | 6.4E-05 |
| Playset Surf Inges-HandToMouth Dose | 1 | 738 | 9.0E-09 | 3.4E-08 | 1.0E-09 | 3.2E-12 | 2.8E-11 | 2.5E-10 | 4.2E-09 | 3.4E-08 | 1.7E-07 | 4.8E-07 |
| Playset Soil Inges-Direct Dose | 1 | 738 | 2.9E-06 | 6.4E-06 | 8.8E-07 | 4.2E-09 | 7.0E-08 | 3.2E-07 | 2.6E-06 | 1.0E-05 | 4.1E-05 | 6.4E-05 |
| Playset Surf Derm Dose | 1 | 738 | 7.8E-09 | 2.7E-08 | 1.5E-09 | 4.7E-12 | 5.0E-11 | 3.6E-10 | 5.7E-09 | 2.7E-08 | 1.1E-07 | 3.9E-07 |
| Playset Soil Derm Dose | 1 | 738 | 5.4E-08 | 7.4E-08 | 3.1E-08 | 7.2E-10 | 4.3E-09 | 1.4E-08 | 6.4E-08 | 1.7E-07 | 3.5E-07 | 8.2E-07 |
| Deck Total Dose | 1 | 738 | 4.1E-07 | 6.7E-07 | 1.7E-07 | 1.1E-09 | 1.9E-08 | 6.6E-08 | 4.8E-07 | 1.5E-06 | 3.6E-06 | 5.5E-06 |
| Deck Surf Inges-Hand To Mouth Dose | 1 | 738 | 3.2E-08 | 9.9E-08 | 6.1E-09 | 7.6E-12 | 1.4E-10 | 1.6E-09 | 2.3E-08 | 1.4E-07 | 3.7E-07 | 1.4E-06 |
| Deck Soil Inges-Direct Dose | 1 | 738 | 3.3E-07 | 6.5E-07 | 8.8E-08 | 9.4E-11 | 2.2E-09 | 2.2E-08 | 3.6E-07 | 1.3E-06 | 3.5E-06 | 5.5E-06 |
| Deck Surf Derm Dose | 1 | 738 | 2.8E-08 | 6.9E-08 | 7.9E-09 | 2.3E-11 | 2.4E-10 | 2.0E-09 | 2.5E-08 | 1.1E-07 | 2.8E-07 | 7.7E-07 |
| Deck Soil Derm Dose | 1 | 738 | 2.0E-08 | 3.1E-08 | 1.2E-08 | 4.5E-12 | 1.3E-09 | 5.4E-09 | 2.4E-08 | 6.5E-08 | 1.1E-07 | 6.0E-07 |

* Cancer Risks calculated by multiplying the slope factor (Q1*) times the LADD reported in the revised Table 41 of the SHEDS-Wood-Wood document.
The slope factor (Q1*) of 3.67 $(mg/kg/day)^{-1}$ is used in this assessment.

# Appendix C

## Comparison of Total Risks to Risk Reduction Impacts

**Figure C-1 Arsenic Cancer Risk at the 95% Percentile (Warm Climate)**

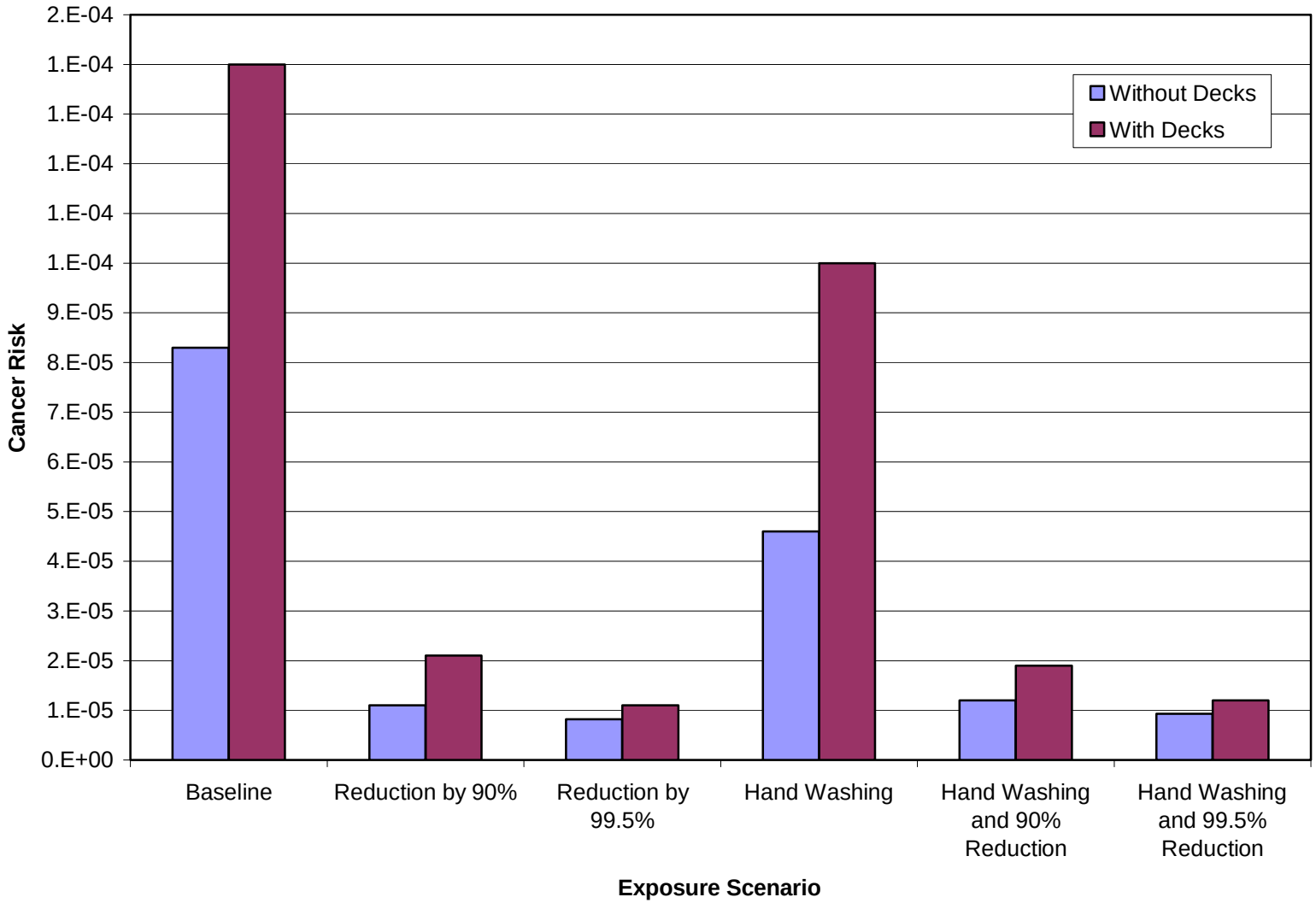


**Figure C-2 Arsenic Cancer Risk at the 50% Percentile (Warm Climate)**

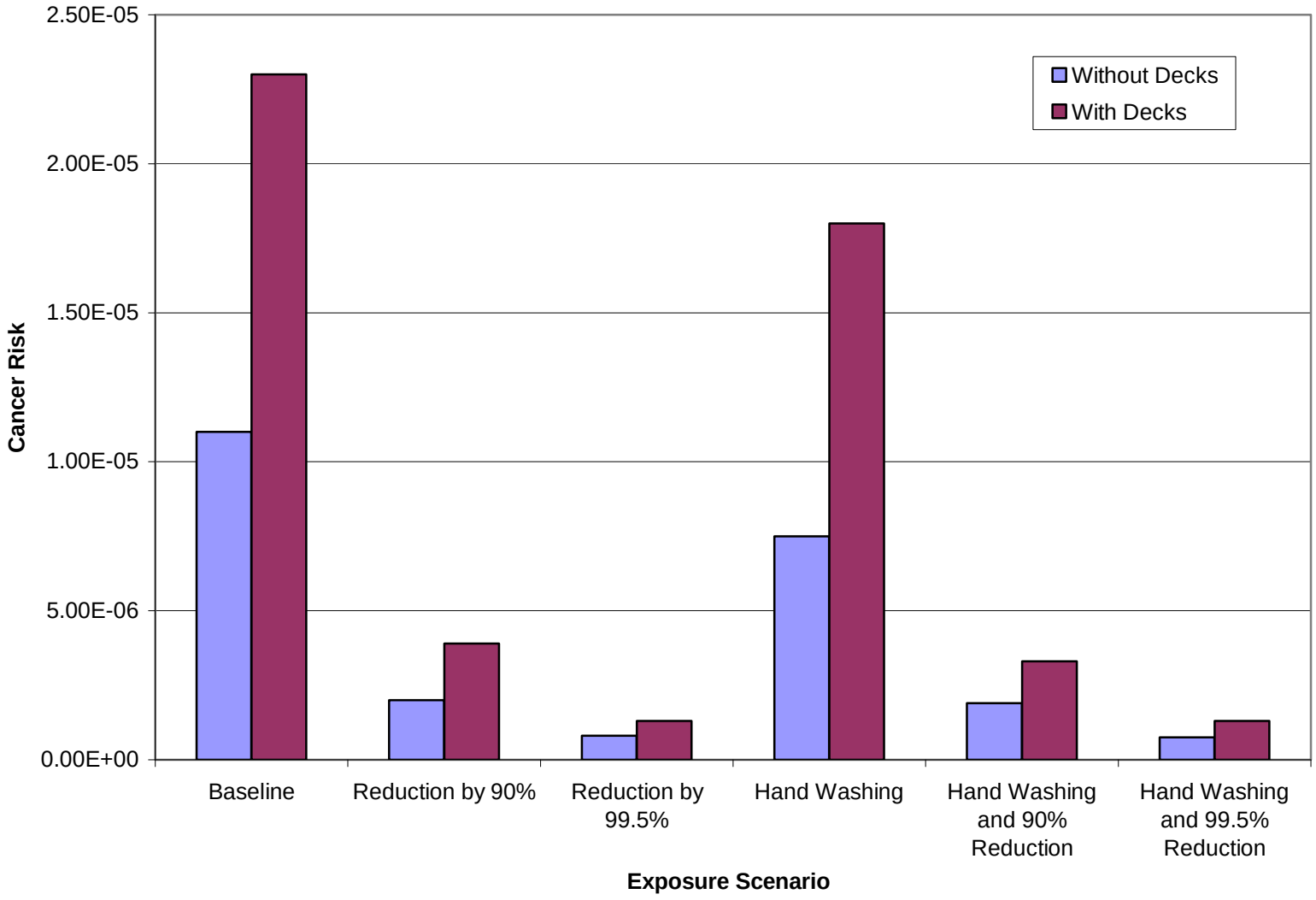


**Figure C-3 Arsenic Cancer Risk for the Mean Population (Warm Climate)**

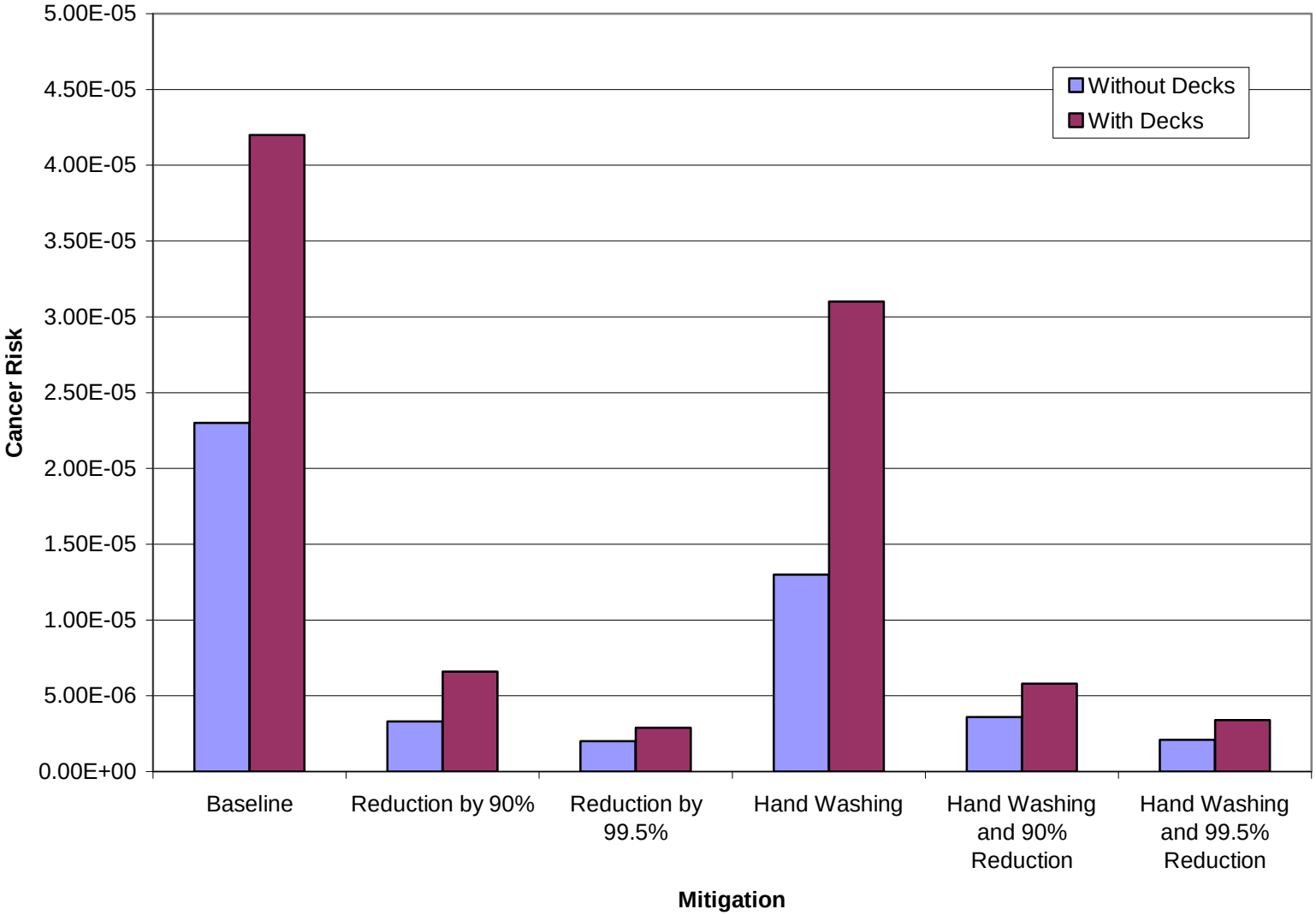


**Figure C-4 Arsenic Cancer Risk for the 95% Population (Cold Climate)**

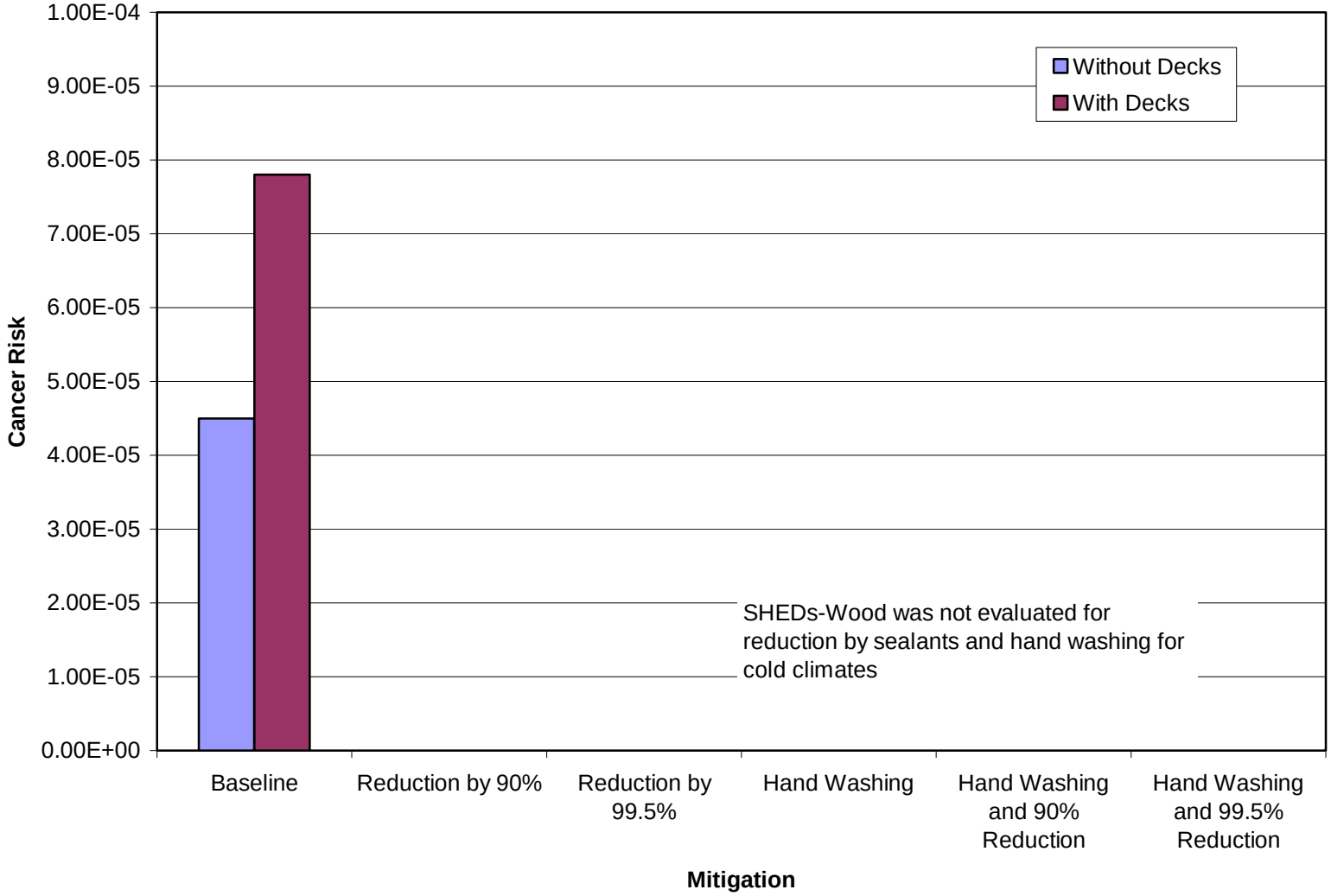


**Figure C-5 Arsenic Cancer Risk for the 50% Population (Cold Climate)**

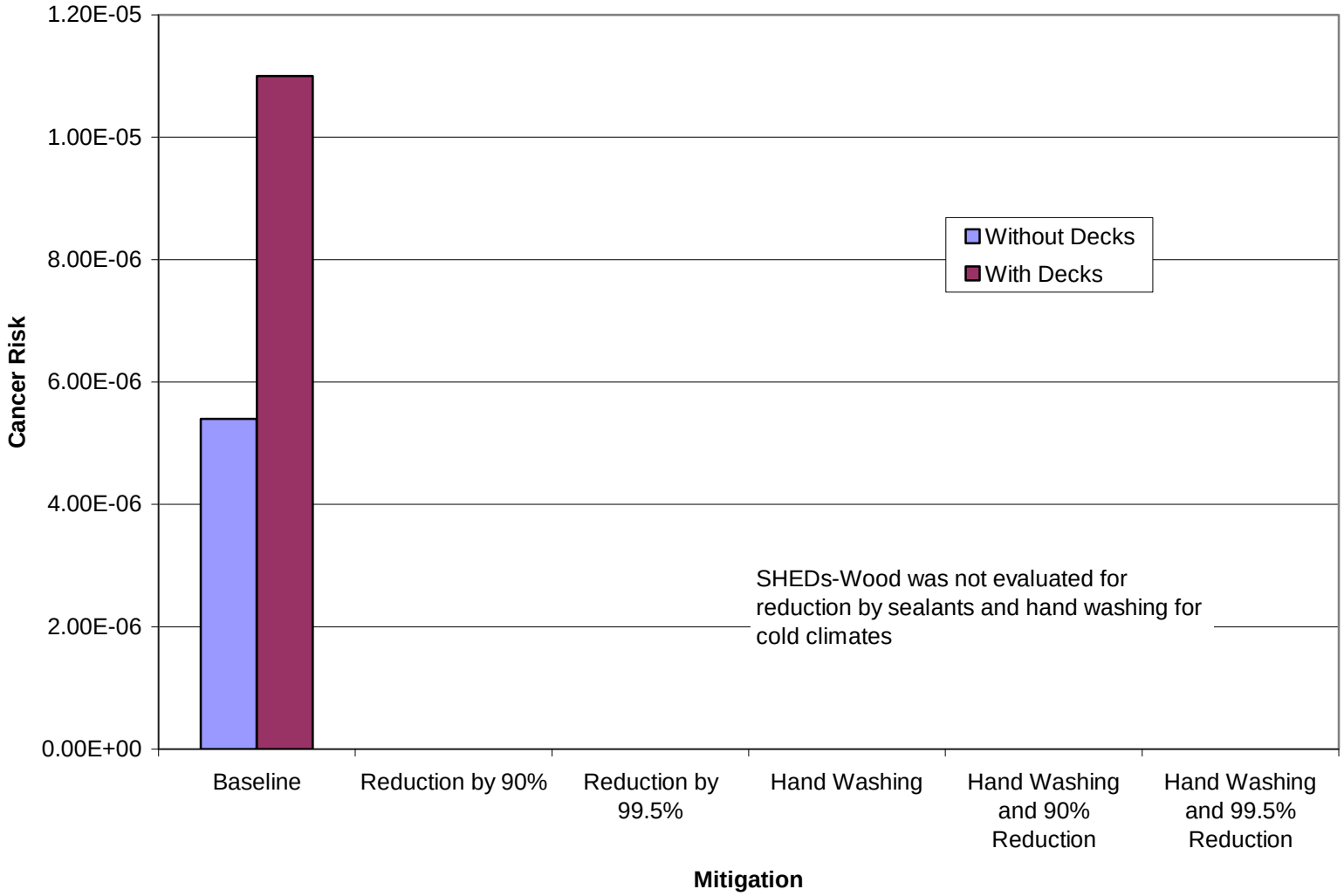


**Figure C-6 Arsenic Cancer Risk for the Mean Population (Cold Climate)**

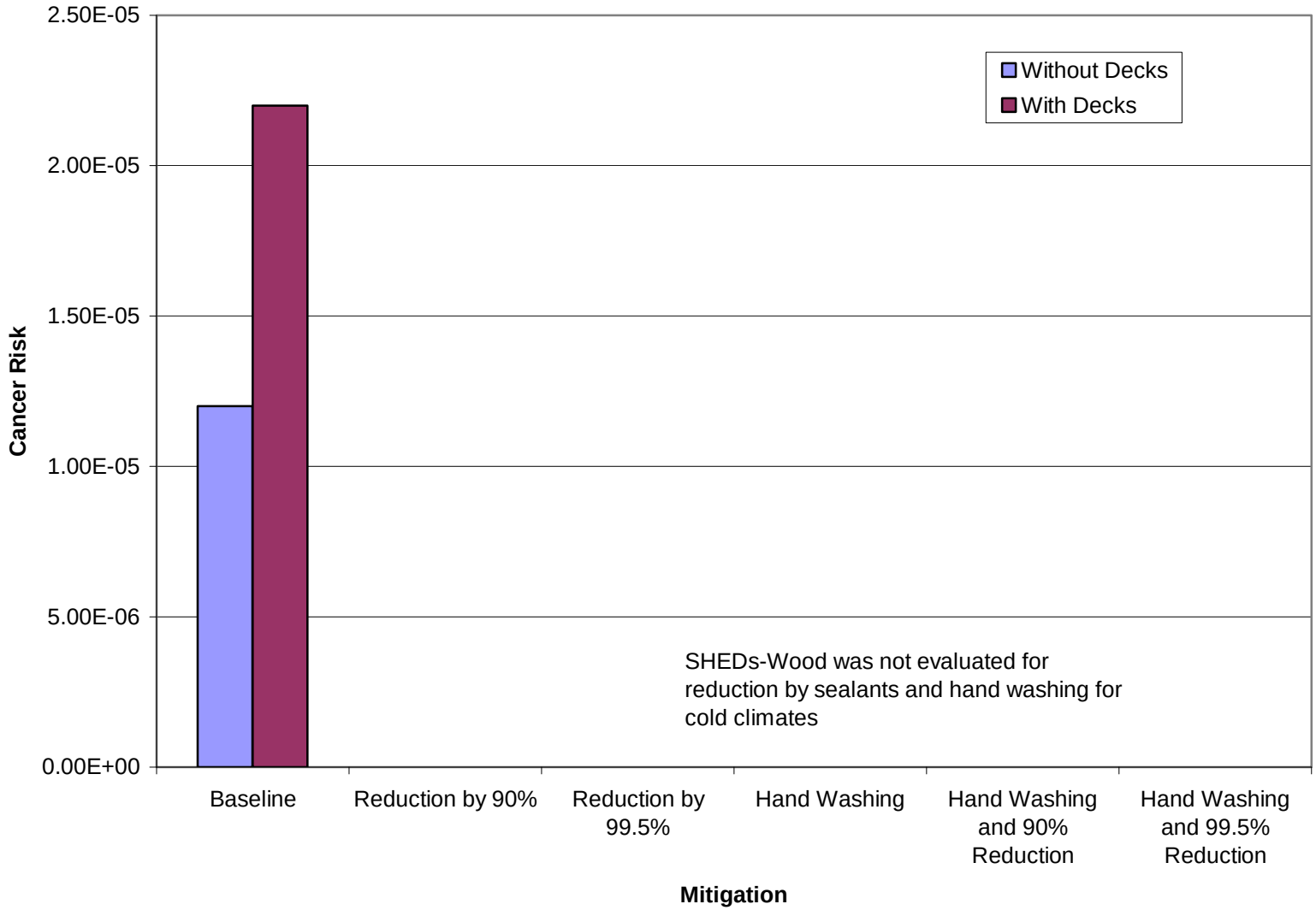
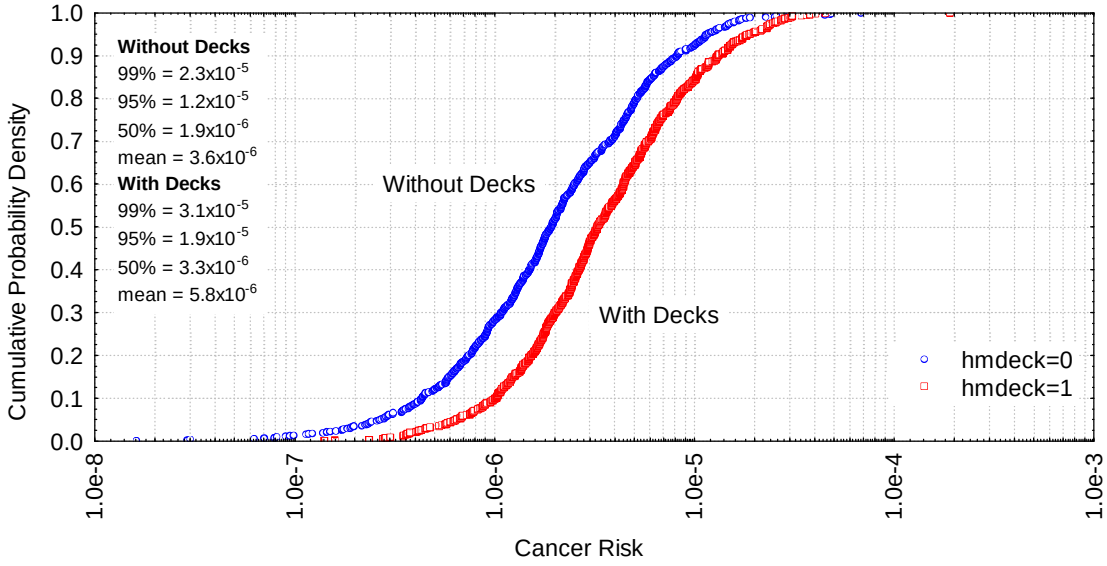
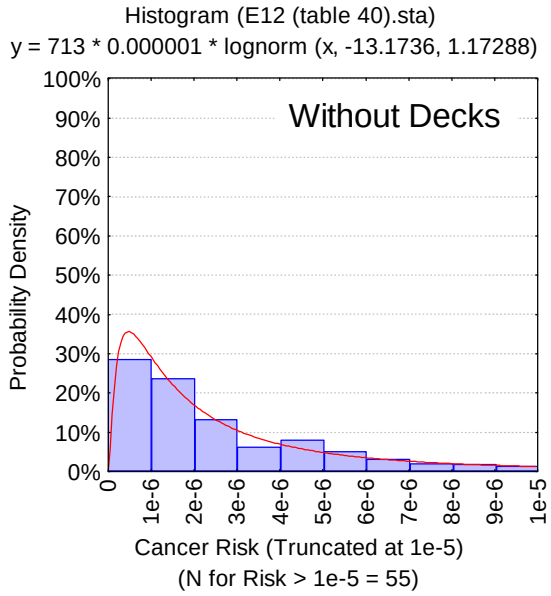


Figure C-7.

Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(Simulating 90% Residue Reduction and Hand Washing to Reduce Exposure)
Scatterplot (E12 (table 40).sta)



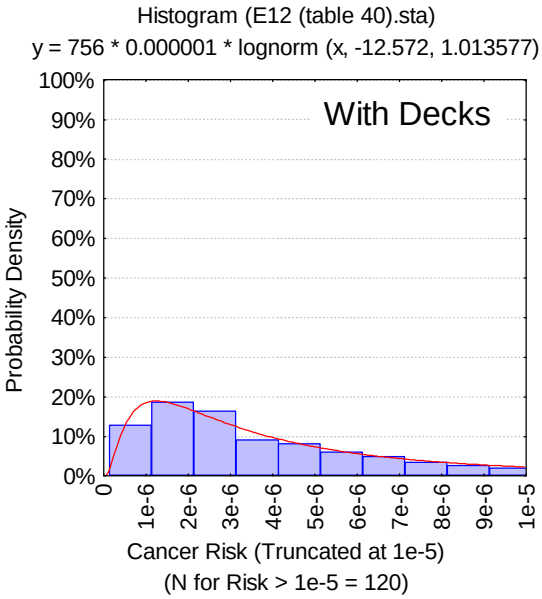
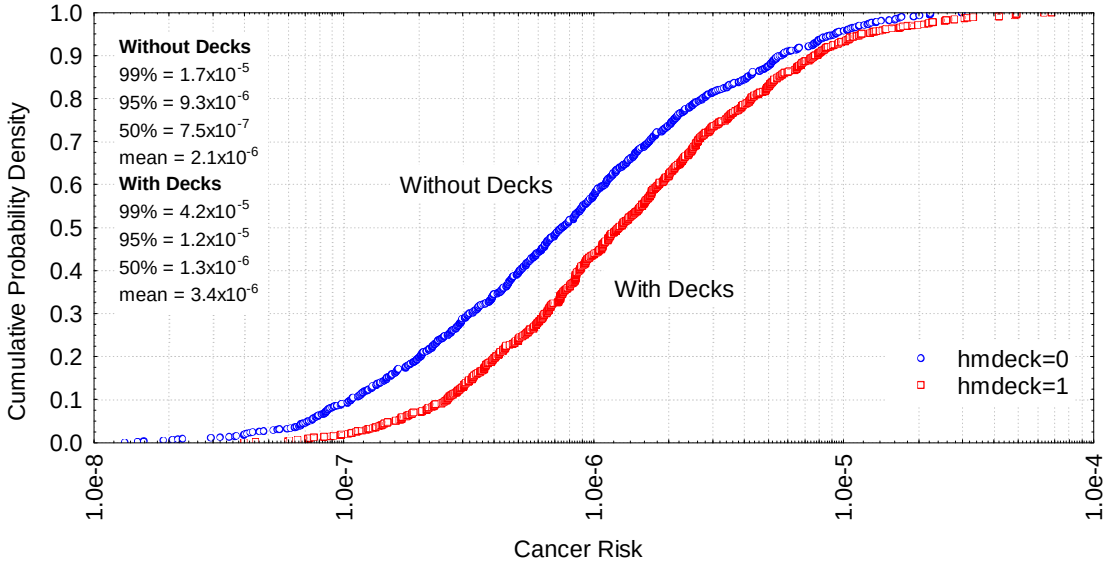
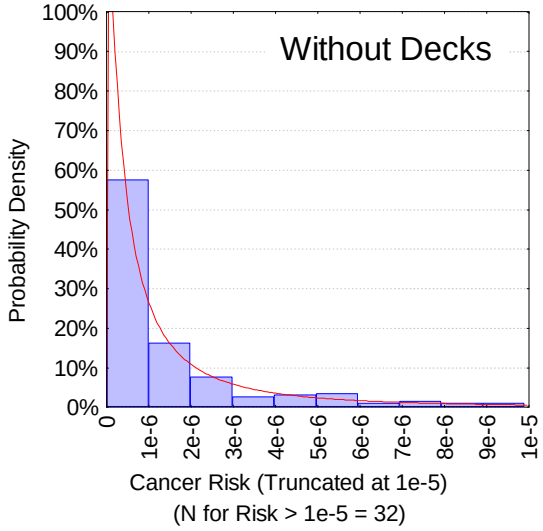



Figure C-8.

Cancer Risk from Lifetime-Term LADD for Children Exposed to Arsenic
Dislodgeable Residues and Contaminated Soil from Treated Wood
Playsets and Residential Decks in Warm Climate
(Simulating 99.5% Residue Reduction and Hand Washing to Reduce Exposure)
Scatterplot (E13 (table 41).sta)




Histogram (E13 (table 41).sta)
y = 729 * 0.000001 * lognorm (x, -14.09607, 1.485913)




Histogram (E13 (table 41).sta)
y = 738 * 0.000001 * lognorm (x, -13.52167, 1.33602)

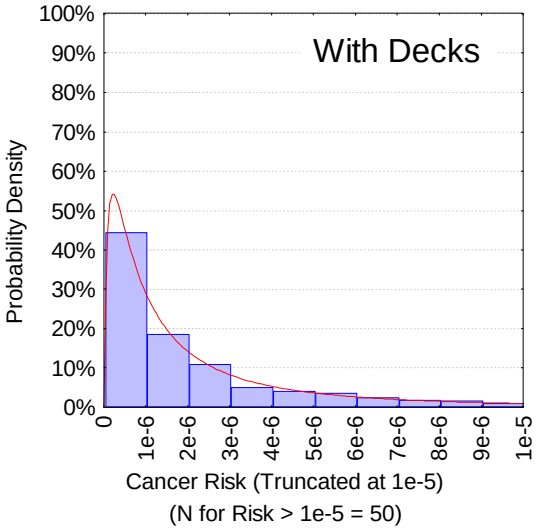


Table C-1. Probabilistic cancer risk distributions and management goals for children exposed to arsenic dislodgeable residues and contaminated soil from treated wood playsets and residential decks in warm climate (reducing exposure by washing hands and reducing deck and playset residue concentration by 90%) (Based on LADDs in Table 40 from the SHEDS-Wood document)

**Without Decks**

| Percentile of Exposure | Lifetime Average Daily Dose (LADD) ug/kg/day | Cancer Risk | Proposed Management Goals | | |
|---|---|---|---|---|---|
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 1.9E-05 | 6.8E-05 | | | **X** |
| 99 | 6.2E-06 | 2.3E-05 | | | |
| 95 | 3.2E-06 | 1.2E-05 | | | |
| 90 | 2.2E-06 | 8.2E-06 | | **X** | |
| 50 | 5.2E-07 | 1.9E-06 | | | |
| 10 | 1.2E-07 | 4.4E-07 | **X** | | |
| 5 | 7.3E-08 | 2.7E-07 | | | |
| 1 | 2.4E-08 | 9.0E-08 | | | |
| minimum | 4.4E-09 | 1.6E-08 | | | |
| >99.9 | 1.9E-05 | 1.0E-04 | | | **X** |
| 92.3 | 2.7E-06 | 1.0E-05 | | **X** | |
| 28.1 | 2.7E-07 | 1.0E-06 | **X** | | |

**With Decks**

| Percentile of Exposure | Lifetime Average Daily Dose (LADD) ug/kg/day | Cancer Risk | Proposed Management Goals | | |
|---|---|---|---|---|---|
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 5.2E-05 | 1.9E-04 | | | |
| 99 | 8.4E-06 | 3.1E-05 | | | **X** |
| 95 | 5.2E-06 | 1.9E-05 | | | |
| 90 | 3.6E-06 | 1.3E-05 | | | |
| 50 | 8.9E-07 | 3.3E-06 | | **X** | |
| 10 | 2.8E-07 | 1.0E-06 | | | |
| 5 | 1.8E-07 | 6.5E-07 | **X** | | |
| 1 | 9.5E-08 | 3.5E-07 | | | |
| minimum | 3.8E-08 | 1.4E-07 | | | |
| 99.9 | 1.2E-05 | 1.0E-04 | | | **X** |
| 84.1 | 2.7E-06 | 1.0E-05 | | **X** | |
| 9.7 | 2.7E-07 | 1.0E-06 | **X** | | |

**X** = Meets Management Goal

Table C-2. Probabilistic cancer risk distributions and management goals for children exposed to arsenic dislodgeable residues and contaminated soil from treated wood playsets and residential decks in warm climate (reducing exposure by washing hands and reducing deck and playset residue concentration by 99.5%) (Based on LADDs in Table 41 from the SHEDS-Wood document)

**Without Decks**

| Percentile of Exposure | Lifetime Average Daily Dose (LADD) ug/kg/day | Cancer Risk | Proposed Management Goals | | |
|---|---|---|---|---|---|
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 8.1E-06 | 3.0E-05 | | | **X** |
| 99 | 4.7E-06 | 1.7E-05 | | | |
| 95 | 2.5E-06 | 9.3E-06 | | **X** | |
| 90 | 1.5E-06 | 5.6E-06 | | | |
| 50 | 2.0E-07 | 7.5E-07 | **X** | | |
| 10 | 2.9E-08 | 1.1E-07 | | | |
| 5 | 2.0E-08 | 7.2E-08 | | | |
| 1 | 8.0E-09 | 2.9E-08 | | | |
| minimum | 3.6E-09 | 1.3E-08 | | | |
| >99.9 | 8.1E-06 | 1.0E-04 | | | **X** |
| 95.6 | 2.6E-06 | 1.0E-05 | | **X** | |
| 57.5 | 2.7E-07 | 1.0E-06 | **X** | | |

**With Decks**

| Percentile of Exposure | Lifetime Average Daily Dose (LADD) ug/kg/day | Cancer Risk | Proposed Management Goals | | |
|---|---|---|---|---|---|
| | | | **A = 1.0e-6** | **B = 1.0e-5** | **C = 1.0e-4** |
| maximum | 1.9E-05 | 6.8E-05 | | | **X** |
| 99 | 1.1E-05 | 4.2E-05 | | | |
| 95 | 3.2E-06 | 1.2E-05 | | | |
| 90 | 2.1E-06 | 7.7E-06 | | **X** | |
| 50 | 3.4E-07 | 1.3E-06 | | | |
| 10 | 7.1E-08 | 2.6E-07 | **X** | | |
| 5 | 4.4E-08 | 1.6E-07 | | | |
| 1 | 2.0E-08 | 7.3E-08 | | | |
| minimum | 1.1E-08 | 4.0E-08 | | | |
| >99.9 | 1.9E-05 | 1.0E-04 | | | **X** |
| 93.2 | 2.7E-06 | 1.0E-05 | | **X** | |
| 43.6 | 2.7E-07 | 1.0E-06 | **X** | | |

**X** = Meets Management Goal

# Appendix D

## Comparison of Residue and Soil Risks

**Figure D-1 Comparison of Total Arsenic Risks from Playsets and Decks for Warm Climate Baseline**

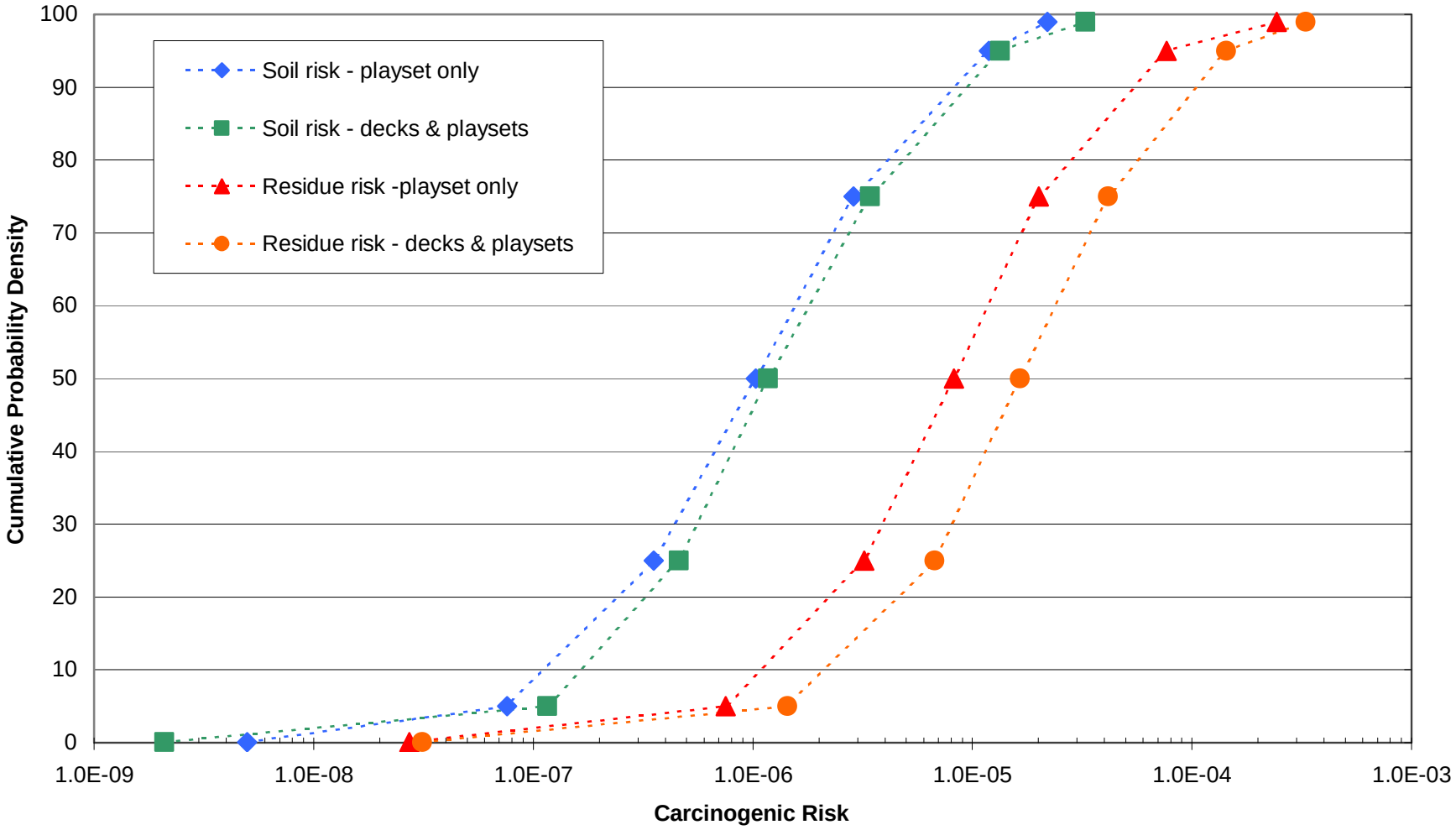


**Figure D-2 Comparison of Total Arsenic Risks from Decks & Playsets for Warm Climate**
**90% Reduction and Hand Washing**

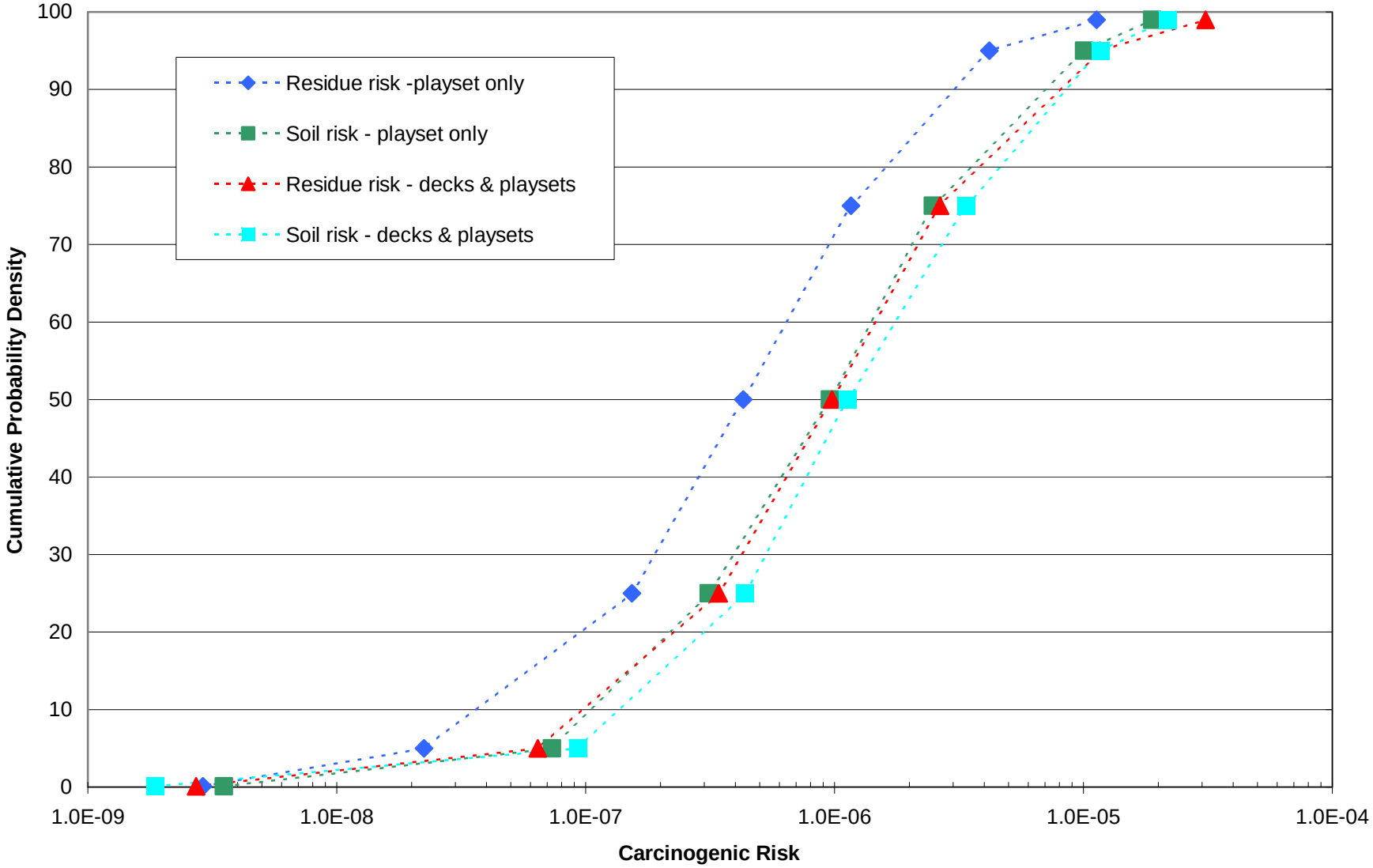


**Figure D-3 Comparison of Residue & Soil Total Arsenic Risks for Warm Climate**
**Dermal Residue Absorption Rate =0.01%**

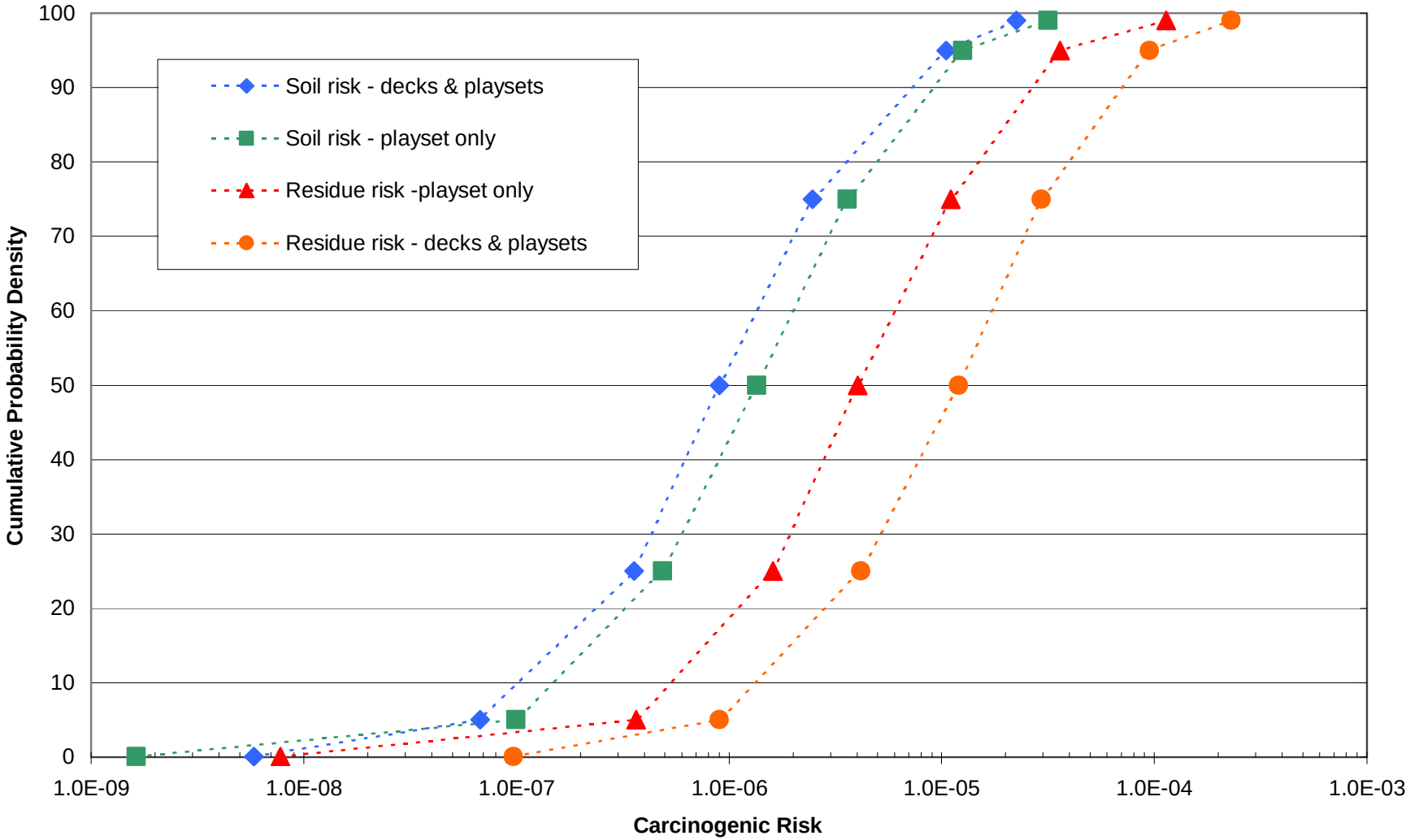


**Figure D-4 Comparison of Total Arsenic Risks from Decks and Playsets for Warm Climate**
**90% Reduction**

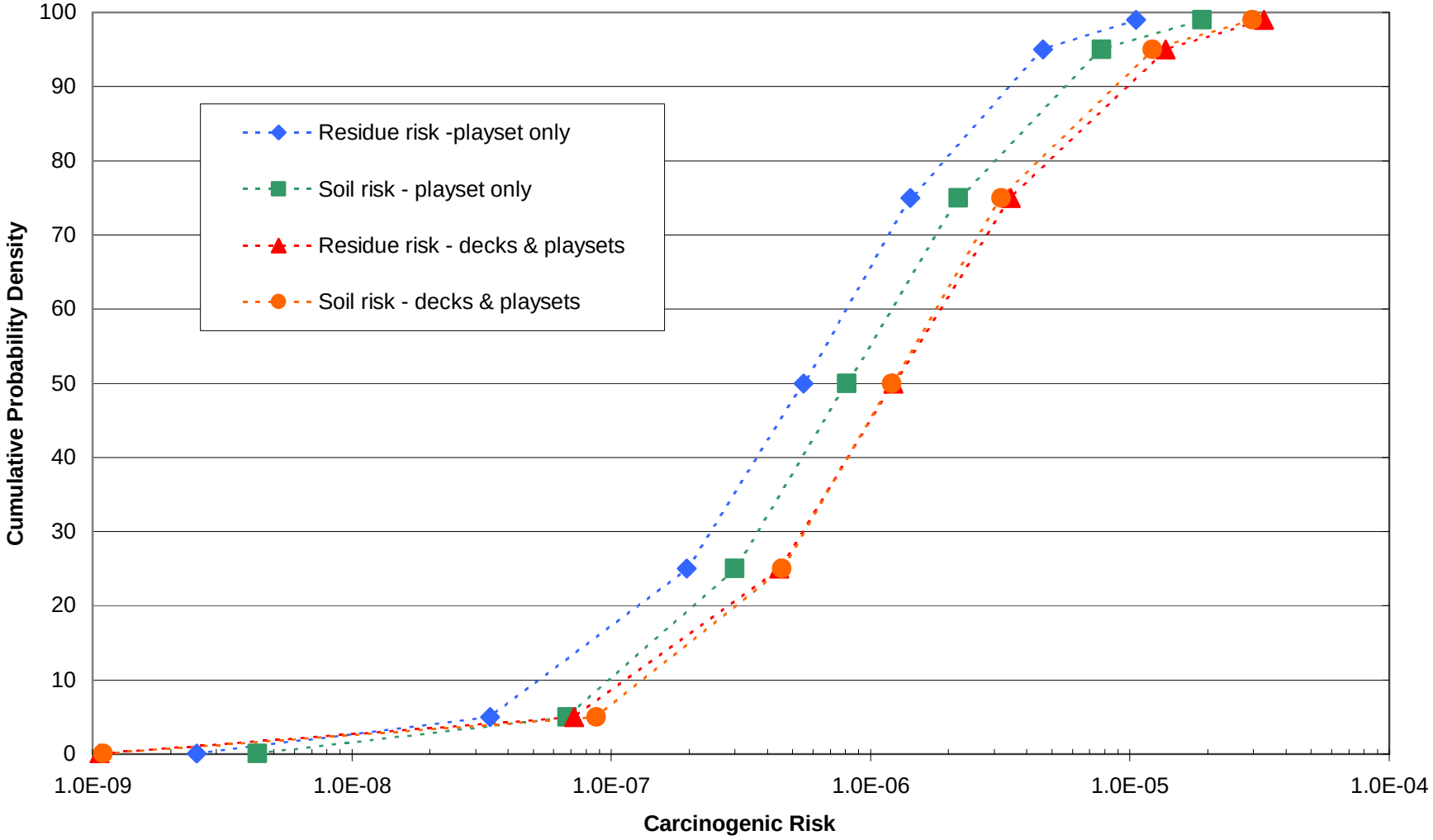


**Figure D-5 Comparison of Residue & Soil Total Arsenic Risks for Warm Climate**
**99.5% Reduction + Hand Wash**

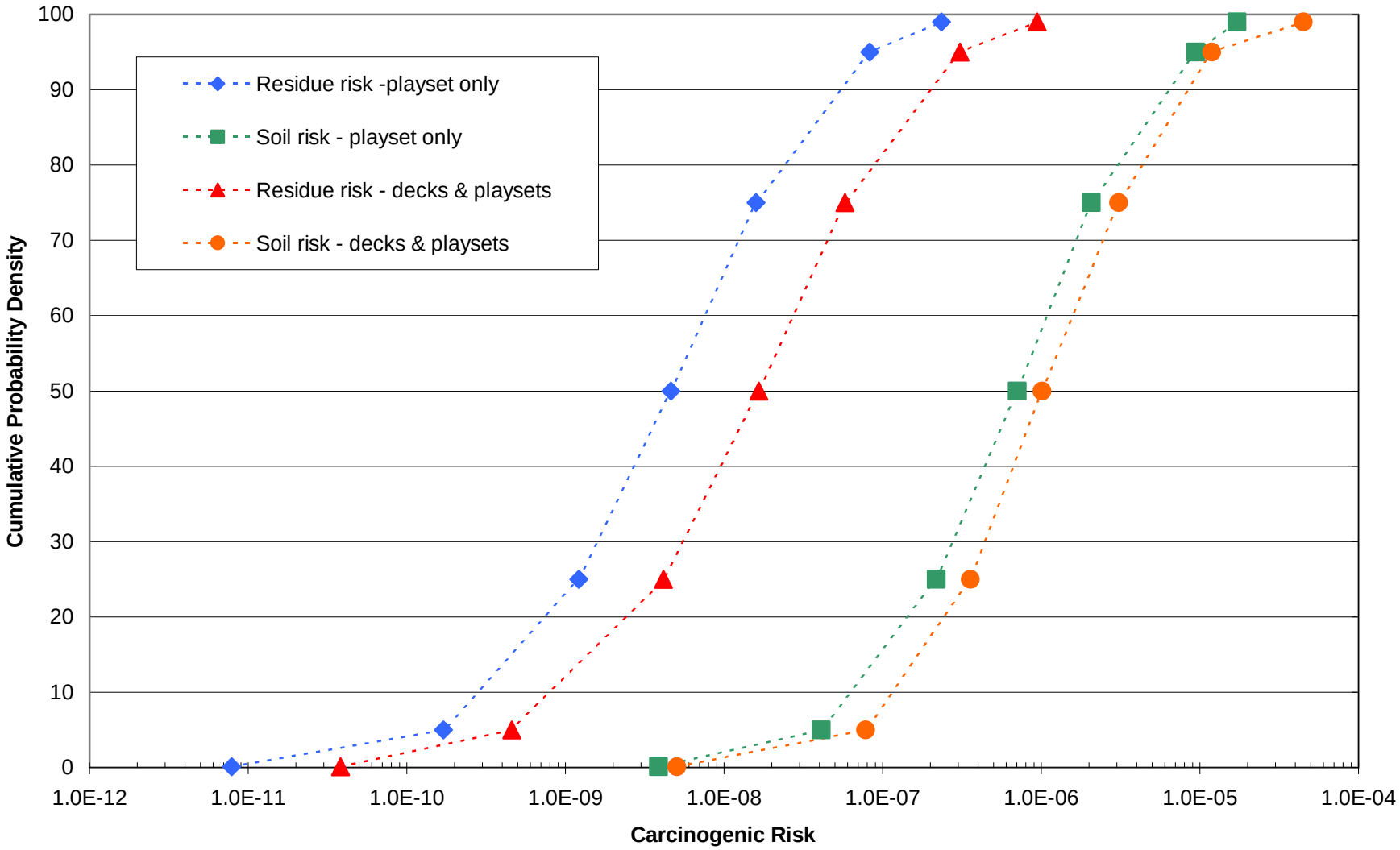


**Figure D-6 Comparison of Residue and Soil Total Arsenic Risks for Warm Climate**
**99.5% Reduction**

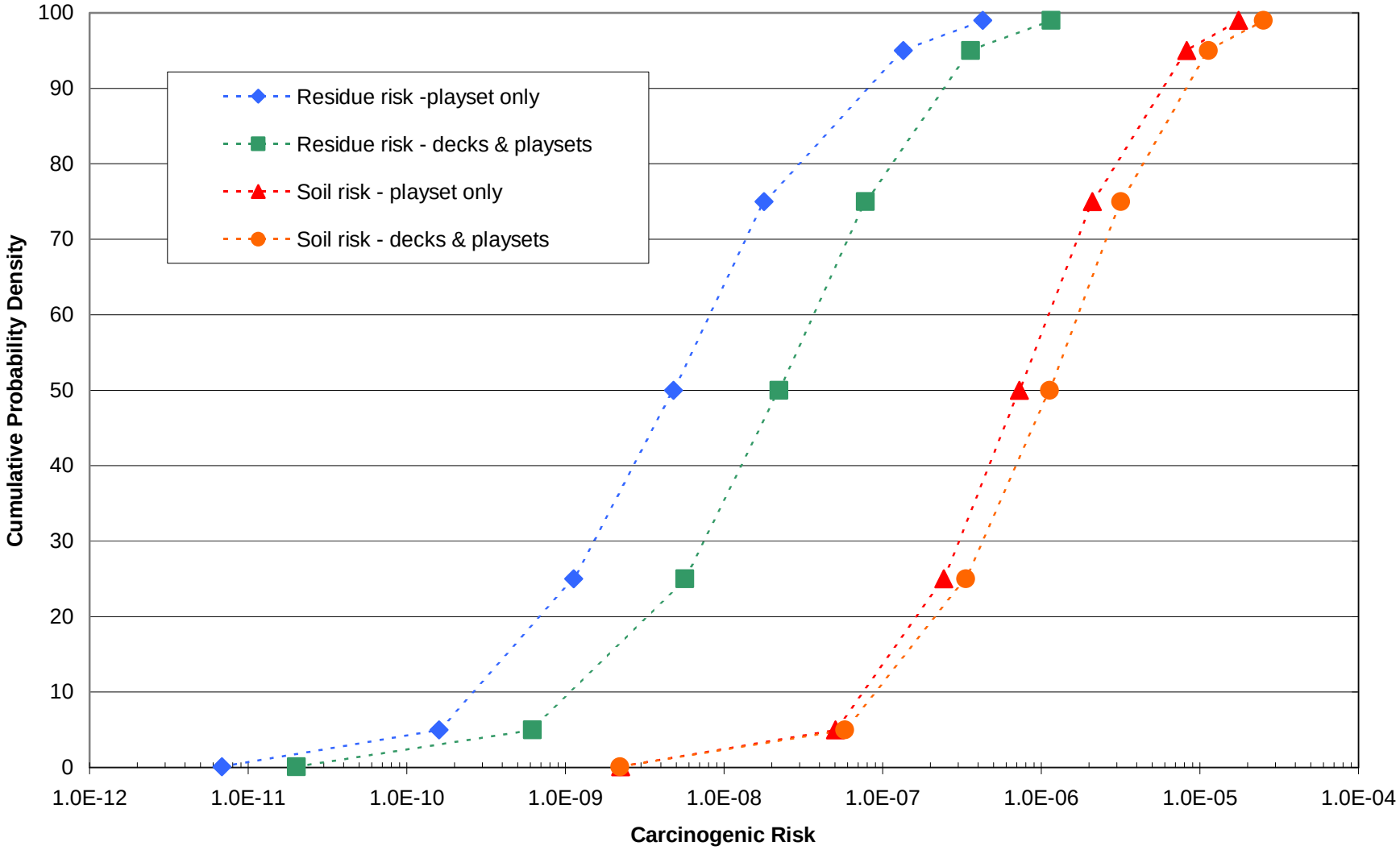
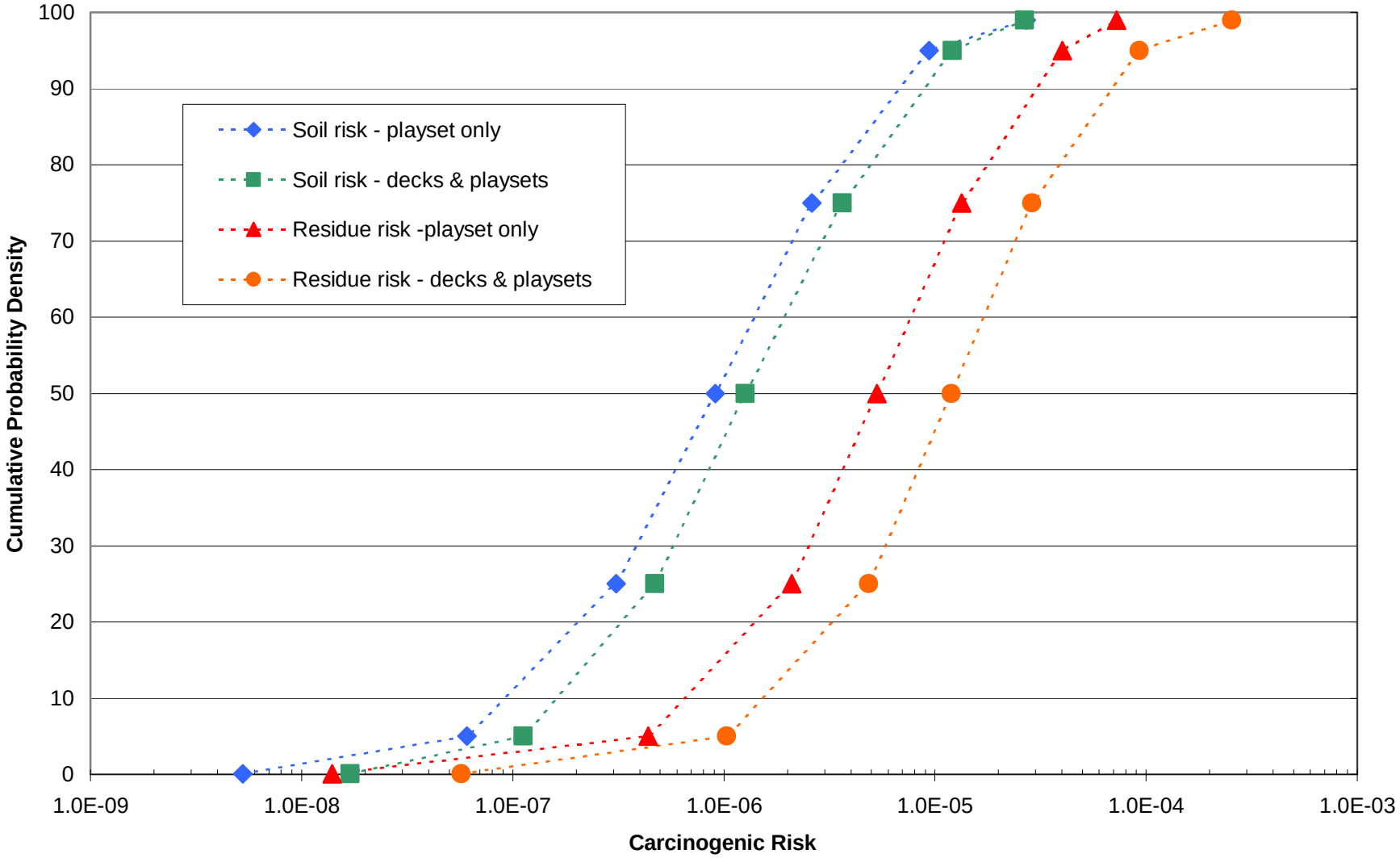


**Figure D-7 Comparison of Residual & Soil Total Arsenic Risks for Warm Climate Hand Wash**



# Appendix E

## Summary of Relative Bioavailability Studies
**Prepared by Jonathan Chen, Ph.D 9-15-2003**

**SUMMARY OF RELATIVE BIOAVAILABILITY STUDIES**
**Prepared by Jonathan Chen, Ph.D 9-15-2003**

The bioavailability of absorbed inorganic arsenic is dependent on the matrix in which it is exposed to. Arsenic in drinking water is in a water-soluble form, and it is generally assumed that its absorption from the gastrointestinal tract is nearly complete. Arsenic in soils, however, may be incompletely absorbed because they may be present in water-insoluble forms or interact with other constituents in the soil. The relative bioavailability of arsenic after it is been exposed (water versus soil) was defined as the percentage of arsenic absorbed into the body of a soil-dosed animal compared to that of animal receiving an single dose of arsenic in aqueous solution. This is a route specific issue. The relative bioavailability through oral route for both arsenic in soil vs. arsenic in water and arsenic in dislodgeable wood residue vs arsenic in water are discussed.

# I. ARSENIC IN SOIL

## I -A STUDY SUMMARY

The arsenic relative bioavailability from soils were studied in different animal models . These studies are summarized below.

### I- A-1 Freeman et al. 1993

The relative bioavailability of arsenic from soil samples from Anaconda, Montana was measured. After a fasting period of approximately 16 hours, prepubescent male and female SPF New Zealand White rabbits (5/sex/group) were given a single oral (capsule) administration of soil (3900ppm As) at three dose levels (0.2, 0.5, and 1.0 g of soil/kg, corresponding to 0.78, 1.95 and 3.9 mg As/kg, respectively). Control groups included untreated controls, and an intravenous sodium arsenate group (1.95 mg As/kg). The relative bioavailability of arsenic in the soil was approximately 37 - 56 % (based on the As concentration in the excreted urine).

### I- A-2 Groen et al. 1993

Arsenic was administered as an intravenoussolution ($As_2O_5$) or orally as As in soil to groups of six beagle dogs, and urine was collected in 24-hour fractions for 120 hours. After 120 hours, 88% ± 16% of the dose administered intravenously was excreted in the urine, compared to only 7.0 ± 1.5% excreted in the urine after oral soil administration. The calculated bioavailability of inorganic As from urininary excretion was 8.3 ± 2.0%.

### I- A-3 Freeman et al. 1995

Oral absorption of arsenic in a group of three female Cynomolgus monkeys from a soluble salt, soil, and household dust was compared with absorption of an intravenous dose of sodium arsenate (Freeman et al. 1995). Mean absolute percentage bioavailability based on urine arsenic excretion was reported at 67.6±2.6% (gavage), 19.2±1.5% (oral dust), and 13.8±3.3% (oral soil). Mean absolute percentage bioavailability based on blood arsenic levels was reported at 91.3±12.4% (gavage), 9.8±4.3% (oral dust), and 10.9±5.2% (oral soil). The relative bioavailabilities of arsenic in the dust and soil were approximately

28.4% and 20.4% respectively (based on urine).

**I- A-4 USEPA Region 10, 1996**

The relative bioavailability of arsenic and lead in soil or slag from the Ruston/North Tacoma Superfund Site has been studied in immature swine that received one single oral dose of soil or sodium arsenate (EPA, 1996). Following a 12 hour overnight fast, each animal was given a single administration of the appropriate test material. Solutions of sodium arsenate and lead acetate were administered separately and not mixed together prior to administration. The group receiving environmental media received a single oral administration od one of four quantities of soils at 25, 60, 100 or 150 mg soil/kg of body weight (BW) (0.04, 0.10, 0.16, or 0.24 mg As/kg BW and 0.03, 0.08, 0.14, or 0.20 mg pb / kg BW). Control groups include intravenous or gavage doses of solution arsenic, untreated controls (received aqueous vehicle only), and an intravenous sodium arsenate group (1.95 mg As/kg). Because several urine samples were lost during sampling procedure, urinary arsenic excretion was not used as an biomarker in estimating bioavailability. Based on the blood level of arsenic, the relative bioavailability of arsenic (soil versus water) in the soil was 78% (56 - 111%).

**I- A-5 USEPA Region 8, 1997**

The bioavailability of arsenic in soil has been studied in juvenile swine that received daily oral doses of soil or sodium arsenate (in food or by gavage) for 15 days (EPA 1997). The soils were obtained from various mining and smelting sites and contained, in addition to arsenic at concentrations of 100-300 µg/g, lead at concentrations of 3,000-14,000 µg/g. The arsenic doses ranged from 1 to 65.4 µg/kg/day. The fraction of the arsenic dose excreted in urine was measured on days 7 and 14 and the relative bioavailability of the soil-borne arsenic was estimated as the ratio of urinary excretion fractions, soil arsenic:sodium arsenate. The mean relative bioavailability of soil-borne arsenic ranged from 0 to 98% in soils from seven different sites (mea±SD, 45% ±32). Estimates for relative bioavailability of arsenic in samples of smelter slag and mine tailings ranged from7 to 5l% (mean±SD, 35%±27).

**I- A-6 Roberts et al. 2001**

The relative bioavailability of arsenic from selected soil samples was measured in a primate model. Sodium arsenate was administered to five male *Cebus apella* monkeys by the intravenous and oral routes, and urine and feces were collected over a four-day period. Pharmacokinetic behavior of arsenic and the fractions of dose excreted in urine and feces were consistent with previous observations in humans. Soil samples from four waste sites in Florida (one from an electrical substation, one from a wood preservative treatment (CCA) site, one from a pesticide application site, and one from a cattle dip vat site) were dried and sieved. Soil doses were prepared from these samples and administered orally to the monkeys. Relative bioavailability was assessed based on urinary excretion of arsenic following the soil dose compared with excretion following an oral dose of arsenic in solution. Relatively consistent bioavailability measurements were obtained among monkeys given the same soil sample. Differences in bioavailability were observed for different sites, with relative bioavailability ranging from 10.7±14.9% (mean±SD) to

24.7±3.2% for the four soil samples.

**I- A-7 American Chemistry Council (ACC), 2003a.**

The bioavailability of arsenic in soil affected by CCA-treated wood has been studied in juvenile swine (ACC, 2003a). The soil was collected near the base of utility poles treated with CCA Type C wood. The poles were installed on the site for around 5 years. The arsenic concentration in the utility pole soil was 320 µg/g. Groups of five swine were given oral doses of sodium arsenate or utility pole soil twice a day for 15 days. The amount of arsenic absorbed by each animal was evaluated by measuring the amount of arsenic excreted in the urine (as measured on days 8 to 9 and 10 to 11). The urinary excretion fraction (UEF) (the ratio of the amount excreted per 48 hours divided by the dose given per 48 hours) was calculated for sodium arsenate and the utility pole soil using linear regression analysis. By using sodium arsenate as a relative frame of reference, the mean RBA estimate for the soil affected by the CCA-treated wood is 49% (90th % CI = 41% - 58%).

The study design, the soil types and the results of these studies are summarized in **Table I-1.**

### I-B DISCUSSION AND CONCLUSION

The issue has been discussed in the October 23- 25, 2001 FIFRA Scientific Advisory Panel Meeting. In the meeting, the Agency as the panel members to comment on the choice of the data set and value chosen for representation of the relative bioavailability of inorganic arsenic from ingestion of arsenic-contaminated soil. The panel considered that a research is needed to obtain data on the relative bioavailability of arsenic from soil contamination specifically resulting from CCA-treated wood applications. Based on this consideration, ACC (2003) conducted the study with soil contaminated directly from CCA-treated wood with the juvenile swine model. This is the only study using soil that is contaminated with CCA-treated soil. Although, only one soil type is involved in the study, after evaluating all available information, the Agency decide to use 49% as the relative bioavailabilty value in the risk assessment.

## II. ARSENIC IN WOOD RESIDUE

In the October 23- 25, 2001 FIFRA Scientific Advisory Panel Meeting, the panel member also suggested the Agency to look into the relative bioavailability issue associated with the absorption of arsenic from non-soil substances (such as wood chips or other buffer material) that might be subject to incidental ingestion. For playground equipment, the dislodgeable arsenic from CCA-treated wood become the primary concern.

To address this issue, ACC sponsored a study (2003b). A study using juvenile

swine as test animals was performed to measure the gastrointestinal absorption of arsenic in dislodgeable material obtained from the surface of chromated copper arsenate (CCA)-treated wood. The CCA residue was collected from the surface of 1,456 CCA-treated boards of wood (Southern Yellow Pie or Ponderosa Pine) that had been weathered in the environment for 1 to 4 years. The arsenic concentration in the dislodgeable arsenic material was 3500 µg/g. Groups of five swine were given oral doses of sodium arsenate or dislodgeable arsenic twice a day for 12 days. The amount of arsenic absorbed by each animal was evaluated by measuring the amount of arsenic excreted in the urine (as measured on days 6 to 7, 8 to 9, and 10 to 11). The urinary excretion fraction (UEF) (the ratio of the amount excreted per 48 hours divided by the dose given per 48 hours) was calculated for sodium arsenate and the dislodgeable arsenic using linear regression analysis. Using sodium arsenate as a relative frame of reference, the RBA estimate for the test material is 29% (90th % CI = 26% - 32%).

The Agency consider this is a valid study and the result (29%) will be used as the relative bioavailability of dislodgeable srsenic from CCA-treated wood.

## III. REFERENCES


ACC, 2003a. CCA Workgroup, Relative Bioavailability of Arsenic in Soil Affected by CCATreated Wood. Prepared by Veterinary Medical Diagnostic Laboratory College of Veterinary Medicine. University of Missouri, Syracuse Research Corporation, Denver, Colorado

ACC, 2003b. CCA Workgroup, Relative Bioavailability of Dislodgeable Arsenic from CCATreated Wood, Prepared by Veterinary Medical Diagnostic Laboratory Medicine. University of Missouri, Syracuse Research Corporation, Denver, Colorado.

Freeman, G.B., Schoof, R.A., Ruby, M.V., Davis, A.O., Dill, J.A., Liao, S.C., Lapin, C.A., and Bergstrom, P.D. 1995. Bioavailability of Arsenic in Soil and House Dust Impacted by Smelter Activities Following Oral Administration in Cynomologus Monkeys. Fundamental and Applied Toxicology 28:215-222

Freeman, GB., Johnson, J.D., Killinger, J.M., Liao, S.C., Davis, A.O., Ruby, M.V., Chaney, R.L., Lovre, S.C., and Bergstrom, P.D. 1993. Bioavailability of Arsenic in Soil Impacted by Smelter Activities Following Oral Administration in Rabbits. Fundamental and Applied Toxicology 21:83-88

Groen, K., Vaesen, H.A.G., Klest, J.I.G. deBar, J.L.M., von Ooik, T. Timmerman, A. and Vlug, R.G. 1993. Bioavailability of Inorganic Arsenic from Bog Ore-Containing Soil in the Dog. Environmental Health Perspective 102: 182-184.

Roberts, S.M.; Welmar, W.R.; Venson, J.R.; Munson, J.W.; and Bergeron 2001. Meausrement of Arsenic Bioavailability from Soils Using a Primate Model. Unpublished.

US. EPA, 1996.. Bioavailability of Arsenic and Lead in Environmental Substrates. 1. Results of an Oral Dosing Study of Immature Swine. Superfund/Office of Environmental Assessment,Region 10, EPA 910/R-96-002,

US. EPA. 1997. Relative Bioavailability of Arsenic in Mining Wastes, Region 8, Document Control No. 4500-88-AORH, 1997.

U.S. EPA, 2001. Children's Exposure to CCA-Treated Wood Playground Equipment and CCAContaminated Soil (Final Report to SAP 9/27/01). Prepared by the Office of Pesticide Programs Antimicrobial Division. Draft Version, September 27, 2001.

**Appendix F**

**SAP Report No. 2001-12**
**FIFRA Scientific Advisory Panel Meeting,**
**October 23- 25, 2001, held at the Sheraton Crystal City Hotel, Arlington, Virginia**

**A Set of Scientific Issues Being Considered by the Environmental Protection Agency Regarding:**

**Preliminary Evaluation of the Non-dietary Hazard and Exposure to Children from Contact with Chromated Copper Arsenate (CCA)-treated Wood Playground Structures and CCA-contaminated Soil.**

**UNITED STATES ENVIRONMENTAL PROTECTION AGENCY**
WASHINGTON, D.C. 20460

OFFICE OF
PREVENTION, PESTICIDES, AND
TOXIC SUBSTANCES

December 12, 2001

**MEMORANDUM**

**SUBJECT:** Transmittal of the Final Report for the FIFRA Scientific Advisory Panel (SAP) Meeting Held October 23 - 25, 2001

**TO:** Marcia E. Mulkey, Director
Office of Pesticide Programs

**FROM:** Olga Odiott, Designated Federal Official
FIFRA Scientific Advisory Panel
Office of Science Coordination and Policy

Larry Dorsey, Executive Secretary
FIFRA Scientific Advisory Panel
Office of Science Coordination and Policy

**THRU:** Vanessa T. Vu, Ph.D., Director
Office of Science Coordination and Policy

Please find attached the Report for the FIFRA SAP open meeting held October 23-25,2001: **Preliminary Evaluation of the Non-dietary hazard and Exposure to Children from Contact with Chromated Copper Arsenate Treated Wood Playground Structures and Contaminated Soil.**

cc:

Stephen Johnson
Susan Hazen
Janet Andersen
Don Barnes (SAB)
James Jones
Denise Keehner
Elizabeth Leovey
Anne Lindsay
Douglas Parsons
Lois Rossi
Frank Sanders
Richard Schmitt
Margaret Stasikowski
OPP Docket

# SAP Report No. 2001-12

## FIFRA Scientific Advisory Panel Meeting, October 23- 25, 2001, held at the Sheraton Crystal City Hotel, Arlington, Virginia

## A Set of Scientific Issues Being Considered by the Environmental Protection Agency Regarding:

# Preliminary Evaluation of the Non-dietary Hazard and Exposure to Children from Contact with Chromated Copper Arsenate (CCA)-treated Wood Playground Structures and CCA-contaminated Soil.

# SAP Report No. 2001-12

## FIFRA Scientific Advisory Panel Meeting, October 23 - 25, 2001, held at the Sheraton Crystal City Hotel, Arlington, Virginia

## A Set of Scientific Issues Being Considered by the Environmental Protection Agency Regarding:

# Preliminary Evaluation of the Non-dietary Hazard and Exposure to Children from Contact with Chromated Copper Arsenate (CCA)-treated Wood Playground Structures and CCA-contaminated Soil.


Olga Odiott, M.S.
Designated Federal Official
FIFRA Scientific Advisory Panel
Date: December 12, 2001

Stephen M. Roberts, Ph.D.
FIFRA SAP Session Chair
FIFRA Scientific Advisory Panel
Date: December 12, 2001

## NOTICE

This report has been written as part of the activities of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), Scientific Advisory Panel (SAP). This report has not been reviewed for approval by the United States Environmental Protection Agency (Agency) and, hence, the contents of this report do not necessarily represent the views and policies of the Agency, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute a recommendation for use.

The FIFRA SAP was established under the provisions of FIFRA, as amended by the Food Quality Protection Act (FQPA) of 1996, to provide advice, information, and recommendations to the Agency Administrator on pesticides and pesticide-related issues regarding the impact of regulatory actions on health and the environment. The Panel serves as the primary scientific peer review mechanism of the EPA, Office of Pesticide Programs (OPP) and is structured to provide balanced expert assessment of pesticide and pesticide-related matters facing the Agency. Food Quality Protection Act Science Review Board members serve the FIFRA SAP on an ad-hoc basis to assist in reviews conducted by the FIFRA SAP. Further information about FIFRA SAP reports and activities can be obtained from its website at http://www.epa.gov/scipoly/sap/ or the OPP Docket at (703) 305-5805. Interested persons are invited to contact Larry Dorsey, SAP Executive Secretary, via e-mail at dorsey.larry@.epa.gov.

**CONTENTS**

PARTICIPANTS ---------- 1

PUBLIC COMMENTERS ---------- 3

INTRODUCTION ---------- 5

CHARGE ---------- 6

DETAILED RESPONSE TO THE CHARGE ---------- 15

ADDITIONAL PANEL RECOMMENDATIONS ---------- 55

REFERENCES ---------- 59

**Federal Insecticide, Fungicide, and Rodenticide Act**
**Scientific Advisory Panel Meeting**
**October 23 –25, 2001**

# Preliminary Evaluation of the Non-dietary Hazard and Exposure to Children from Contact with Chromated Copper Arsenate (CCA)-treated Wood Playground Structures and CCA-contaminated Soil.

## PARTICIPANTS

### FIFRA SAP Chair

Stephen M. Roberts, Ph.D., Director, Center for Environmental and Human Toxicology, University of Florida

### Scientific Advisory Panel Members

Fumio Matsumura, Ph.D., Institute of Toxicology and Environmental Health, University of California at Davis

Mary Anna Thrall, D.V.M., Department of Pathology, College of Veterinary Medicine & Biomedical Sciences, Colorado State University

### FQPA Science Review Board Members

John L. Adgate, Ph.D., University of Minnesota School of Public Health, Division of Environmental and Occupational Health

Michael Bates, Ph.D., Visiting Researcher, School of Public Health, University of California, Berkeley

James V. Bruckner, Ph.D., Department of Pharmaceutical and Biomedical Sciences, College of Pharmacy, University of Georgia

Karen Chou, Ph.D., Institute for Environmental Toxicology, Institute of International Health, Dept. of Animal Science, Michigan State University

Harvey Clewell, M.S., ENVIRON International Corporation, Ruston, Louisiana

M. Rony Francois, M.D., Ph.D.c, University of South Florida, College of Public Health

Natalie Freeman, Ph.D., Department of Environmental and Community Medicine, Robert Wood Johnson Medical School, University of Medicine and Dentistry of New Jersey

Gary L. Ginsberg, Ph.D., Connecticut Department of Public Health, Environmental Epidemiology, and Occupational Health, CT Dept. of Public Health

Terry Gordon, Ph.D., NYU School of Medicine

Steven Heeringa, Ph.D., Director, Statistical Design and Analysis, Institute for Social Research University of Michigan

Claudia Hopenhayn-Rich, M.P.H., Ph.D., Department of Preventive Medicine and Environmental Health, University of Kentucky

John Kissel, Ph.D., Department of Environmental Health, School of Public Health and Community Medicine, University of Washington

Michael J. Kosnett, M.D., M.P.H., Division of Clinical Pharmacology and Toxicology, University of Colorado Health Sciences Center

Peter S.J. Lees, Ph.D., C.I.H., Johns Hopkins University, Bloomberg School of Public Health, Department of Environmental Health Sciences, Division of Environmental Health Engineering,

Ross B. Leidy, Ph.D., Director, Pesticide Residue Research Laboratory, Department of Toxicology, North Carolina State University

Peter D.M. Macdonald, D.Phil., Department of Mathematics and Statistics, McMaster University, Hamilton, Ontario, Canada

David W. Morry, Ph.D., Office of Environmental Health Hazard Assessment, California Environmental Protection Agency

Paul Mushak, Ph.D., PB Associates, Durham, NC

Xianglin Shi, Ph.D., Pathology and Physiology Research Branch, National Institute for Occupational Safety and Health, Morgantown, WV

Andrew Smith, SM, ScD, Director, Environmental Toxicology Program, Maine Department of Human Services

Helena Solo-Gabriele, Ph.D., P.E., Department of Civil,Arch., and Environmental Engineering, University of Miami, FL

Jacob J. Steinberg, M.D., Albert Einstein College of Medicine, Director, Autopsy Division, Director, Residency Training Program, Environmental Medicine and Pathology Laboratory, Montefiore Medical Center, N.Y.

Miroslav Styblo, Ph.D., Department of Pediatrics, School of Medicine, and Department of

Nutrition, School of Public Health, Department of Pediatrics, University of North Carolina

John Wargo, Ph.D., Environmental Policy and Risk Analysis, Yale University, CT

## PUBLIC COMMENTERS

**Oral statements were made by:**
Jane Houlihan, Environmental Working Group, Washington, D.C.

Cristopher Williams, Ph.D., Ecology and Environment Inc.,Tallahassee, FL

Ligia Mora-Applegate, M.S.P., M.P.A., M.P.H., Florida Department of Environmental Protection

Pascal Kamdem, Ph.D., Michigan State University, on behalf of American Forest and Paper Association

H. Vasken Aposhian, Ph.D., University of Arizona, on behalf of Arch Chemicals, Inc.

Jay Feldman, Beyond Pesticides / National Coalition Against the Misuse of Pesticides

Yvette Lowney, M.P.H., E$^{x}$ponent, on behalf of the American Chemistry Council

Barbara Beck, Ph.D., DABT, Gradient Corporation, on behalf of Osmose and Arch Chemicals, Inc.

Bill Walsh, Healthy Building Network, Washington, DC

John Butala, M.S., Toxicology Consultants, Inc., on behalf of American Chemistry Council Arsenical Wood Preservatives Task Force

Joyce Tsuji, Ph.D., E$^{x}$ponent, on behalf of American Forest and Paper Association

Scott Conklin, Universal Forest Products, Inc.

Robert Turkewitz, Ness, Motley, Loadholt, Mount Pleasant, SC

Steven Lamm, M.D., Consultants in Epidemiology & Occupational Health, Inc., Washington, DC

**Written statements were made by:**
The Accord Group, on behalf of Osmose and Arch Chemicals, Inc.

Mr. Marc Leathers, Leathers & Associates, Inc.

Ligia Mora-Applegate, M.S.P., M.P.A., M.P.H., Florida Department of Environmental Protection

# INTRODUCTION

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), Scientific Advisory Panel (SAP) has completed its review of the set of scientific issues being considered by the Agency pertaining to its review of the Office of Pesticide Programs' (OPP) Preliminary Evaluation of the Non-dietary Hazard and Exposure to Children from Contact with Chromated Copper Arsenate (CCA)-treated Wood Playground Structures and CCA-contaminated Soil. Advance notice of the meeting was published in the *Federal Register* on September 19, 2001. The review was conducted in an open Panel meeting held in Arlington, Virginia, October 23- 25, 2001. The meeting was chaired by Stephen M. Roberts, Ph.D. Ms. Olga Odiott served as the Designated Federal Official.

The scientific issues addressed by the FIFRA SAP were complex and varied. Panel members were selected to serve because of their expertise in one or more of the subject areas being discussed. The Panel was asked to evaluate the scientific soundness and OPP' s evaluation of the exposure and hazard data available to the Agency for CCA. Specifically, the Panel was asked to 1) review the exposure scenarios and hazard endpoints that the Agency intends to use in its CCA-risk characterization for children; and 2) provide recommendations concerning additional data needed to reduce the uncertainties of this risk characterization.

## CHARGE

**Issue: Short- and Intermediate-term Endpoint Selection for Inorganic Arsenic**

For inorganic arsenic, the data of Franzblau et al (1989) and Mizuta et al (1956) using a LOAEL value of 0.05 mg/kg/day is proposed for selection of short-term and intermediate-term incidental oral endpoints as well as short-term and intermediate-term dermal endpoints. An acceptable Margin of Exposure value of 100 is also proposed. The acceptable Margin of Exposure value includes a 10x factor for intraspecies variation as well as a 10x factor for use of a LOAEL value and the severity of the effects observed in the Mizuta study.

**Question 1: Please comment on the Agency's selection of the 0.05 mg/kg/day LOAEL value for use in assessing risks to the general population as well as children from short-term and intermediate-term incidental oral and dermal exposures, and the appropriateness of the use of a 10x factor for severity of the toxic effects observed in the Mizuta study. Please provide an explanation and scientific justification for your conclusions as to whether the presented data are adequate or whether other data should be considered for selection of this endpoint.**

**Issue: Relative Bioavailability of Inorganic Arsenic**

The bioavailability of inorganic arsenic is dependent on the matrix in which it exists. For purposes of this discussion, the relative bioavailability of inorganic arsenic after ingestion of arsenic-contaminated soil is defined as the percentage of arsenic absorbed into the body from soil compared to that of arsenic administered in drinking water. Arsenic in drinking water is in a water-soluble form, and bioavailability by this route is high (i.e. 95-100%). Arsenic in soil, however, has reduced bioavailability due to existence in a water-insoluble form or its interaction with other soil constituents that impair absorption.

The available data on urinary and fecal recovery of arsenic after an intravenous dose of sodium arsenate in experimental animals compared to recovery after administration of sodium arsenate to experimental animals in soil was examined. Based on these data, a value of 25% bioavailability was selected for arsenic from soil ingestion. This value is based upon the data of Roberts et al. (2001) and Freeman et al. (1995) using non-human primates. These data were felt to best represent relative bioavailability of inorganic arsenic in soil based on the use of non-human primates and the physiological similarity in the pattern of metabolism with humans, and the use of CCA-contaminated soil in the study for estimation of bioavailability.

**Question 2: Please comment on the choice of this data set and value chosen for representation of the relative bioavailability of inorganic arsenic from ingestion of arsenic-contaminated soil. Please discuss the strengths and weaknesses of the selected data and also provide an explanation as to whether this 25% value is appropriate for estimation of bioavailability in children.**

**Issue: Dermal Absorption Value for Inorganic Arsenic**

A value of 6.4% for the dermal absorption of arsenic was selected to represent absorption from dermal contact with inorganic arsenic. This value is based upon the data of Wester et al. (1993) and represents percent absorbed dose of arsenic applied to the skin in a water solution. Although this value is slightly higher than the value of 4.5% obtained for arsenic applied in soil, the mean values for absorption from water and soil both showed significant variability (i.e. 6.4% ± 3.9% in water, 4.5% ±3.2% in soil) such that use of the 6.4% dermal absorption value was selected. It is observed in this study that a higher dose on the skin resulted in lower dermal absorption as noted above, but the data in this study suggests sufficient variability in the absorption such that use of the 6.4% dermal absorption value is sufficiently but not overly conservative.

**Question 3: Please comment on the selection of the value of 6.4% for dermal absorption of inorganic arsenic and whether or not this value will be appropriate for use in all scenarios involving dermal exposure to arsenic from CCA-treated wood, including children's dermal contact with wood surface residues and contaminated soils.**

**Issue: Selection of Hazard Database for Hazard Characterization of Inorganic Chromium in CCA-Treated Wood**

Hazard data show clearly that Cr (VI) demonstrates more significant toxicity than Cr (III). However, there is little data delineating the valence state of chromium in compounds that leach from in-service CCA-treated wood (Lebow, 1996). Interconversion of Cr (VI) and Cr (III) in the environment is observed (Cohen et al., 1999), and at least one study has reported measurable levels of hexavalent chromium in soils (Lebow, 1996). In-service CCA-treated wood contains mainly chromium (III), due to reduction of chromium (VI) during fixation. However, when fixation conditions are not ideal or when storage temperatures are low, Cr (VI) is observed to be present in leachate from the treated wood and in addition, conditions in some soil types can result in conversion of leached Cr (III) to Cr (VI).

**Question 4: As available monitoring data do not differentiate among chromium species found in CCA dislodgeable residues on wood surfaces and in soils, and as Cr (VI) is the more toxic species of chromium, please comment on whether use of the**

**hazard data for chromium (VI) is the best choice for characterizing hazard and risk from exposure to chromium as a component of CCA-treated wood. Please provide a scientific explanation and justification for your recommendation on the choice of either the chromium (III) or chromium (VI) hazard database.**

**Issue: Short- and Intermediate-term Endpoint Selection for Inorganic Chromium**

For inorganic chromium (VI), OPP proposes using the developmental toxicity study of Tyl (MRID 42171201) with a NOAEL value of 0.5 mg/kg/day [0.12 mg/kg/day chromium equivalents] and an MOE of 100 (10x for interspecies variation, 10x for intraspecies variation) for selection of short-term and intermediate-term incidental oral endpoints.

**Question 5: Please comment on the Agency's selection of the 0.5 mg/kg/day NOAEL value for use in assessing risks to the general population as well as children from short-term and intermediate-term incidental oral exposures to inorganic chromium as contained in CCA-treated wood. Please provide an explanation and scientific justification for your conclusions as to whether the presented data are adequate or whether other data should be considered for selection of these endpoints.**

**Issue: Selection of Endpoints for Dermal Risk Assessments for Inorganic Chromium**

Dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis, and chromium is also one of the most common contact sensitizers in males in industrialized countries (IRIS, 2000). The relative potency of Cr (VI) and Cr (III) in causing dermal effects has been estimated to differ by approximately 50-fold (Bagdon,1991) but both produce irritation and dermal sensitization. In the OPP HIARC review of selection of dermal toxicity endpoints, it was concluded that skin irritation and skin allergenicity are the primary effects of concern from dermal exposure to Cr(VI), as these effects are the predominant response from dermal exposure to inorganic chromium. Thus, endpoints based on systemic effects from dermal exposure were not selected.

**Question 6: Please comment on whether the significant non-systemic dermal effects from dermal exposure to inorganic chromium should form the basis of dermal residential risk assessments, and, if so, how the Agency should establish a dermal endpoint for such an assessment.**

**Issue: Selection of Parameters and Methodology for Characterizing Child Exposures**

OPP intends to develop realistic exposure scenarios and dose estimates for characterizing potential dermal/oral ingestion exposures to children in playground settings from contact with dislodgeable *As* and *Cr* residues on CCA-treated wood playground structures and in CCA-contaminated soils. In keeping with EPA policy, OPP would like its estimates to

characterize both the middle and upper end of the range of potential exposure values. (The "high end" of exposure is defined as a level of exposure which is likely to be higher than experienced by at least 90% of the population, but not higher than the level experienced by the maximally exposed individual.) Following EPA guidance on conducting exposure assessments, OPP intends to rely on "mean value" (central tendency) data for calculating the lifetime average daily doses (LADDs) used for the cancer assessment, and "maximum value" (high end) data for calculating the average daily doses (ADDs) used for the non-cancer assessment.
OPP expects to use a combination of central tendency and high end values for the different parameters of the exposure equations, as identified below.

**Exposure Parameters Proposed for Use in Conducting the Child Exposure Assessment**

| | | | |
|---|---|---|---|
| *General Variables:* | Age | 3 yr old | central tendency |
| | Body Weight | 15 kg | central tendency |
| | Surface Area: hands, arms, legs | 1640 $cm^2$ | high end |
| | 3 fingers | 20 $cm^2$ | central tendency |
| | Playground activity: hours / day | 1 hr | central tendency |
| | days / year | 130 days | central tendency |
| | years / lifetime | 6 yrs out of a 75 yr lifetime | central tendency |
| *Scenario Specific Variables:* • Dermal Contact with Soil | Soil Adherence Factor | 1.45 $mg/cm^2$ | central tendency |
| • Oral Ingestion of Residues from Hand-to-Mouth Contact with Wood | Exposure time (hrs/day spent for hand-to-mouth activity) | 1 hr/day and 3 hrs/day | central tendency and high end |
| | Hand-to-Mouth Frequency (events/hour) | 9.5 events/hr and 20 events/hr | central tendency and high end |
| | Fraction Ingestion | 50% removal efficiency | central tendency |
| • Oral Ingestion of Soil Residues | Soil Ingestion Rate | 100 mg/day and 400 mg/day | central tendency and high end |

**Question 7. Please comment on whether OPP's choices of central tendency and high end values for different parameters should, collectively, produce estimates of the middle and high end of the range of potential exposures. If the Panel thinks that OPP's approach may not estimate the high ends of the exposure range (because it produces values that are either higher or lower than the upper end of the exposure**

**range), please comment on what specific values should be modified to produce estimates of the high end of potential exposure.**

EPA recognizes that there are many parameters that affect the level of potential exposure and that each of these parameters may vary. Probabilistic (e.g., Monte Carlo) techniques are capable of using multiple data sets which reflect the variability of parameters to produce estimates of the distribution of potential exposures. OPP has identified a number of data sets that contain information on the variability of parameters affecting the levels of exposure to CCA residues experienced by children as a result of their playground activities. Nonetheless, OPP intends to develop deterministic estimates of potential exposure using selected values (either central tendency or high end) for different parameters, in such a manner that the estimates describe both the middle and high end of the range of exposures.

**Question 8: Please comment on whether the existing data bases on the variability of the different parameters affecting potential exposure are adequate to support the development of probabilistic estimates of potential exposure. If the Panel regards the data bases as adequate for that purpose, please identify which parameters should be addressed using a distribution of values and which data bases should be used to supply the distribution for particular parameters.**

**Issue: Transfer of Residues from Wood Surface to Skin**

In lieu of appropriate data on residue transfer from wood to skin surfaces, OPP proposes to rely on assumptions for residue transfer from turf as a surrogate. A one-to-one relationship is assumed between the transferable residues on turf and the surface area of the skin after contact (i.e., if the transferable residue on the turf is 1 $mg/cm^2$, then the residue on the human skin is also 1 $mg/cm^2$ after contact with the turf). This is based on OPP' s Residential SOP' s (April, 2000). OPP plans to apply this one-to-one relationship to the current assessment, assuming a one-to-one relationship between the dislodgeable residues on the wood surface and the surface of the skin after contact.

**Question 9: OPP is assuming that a one-to-one relationship applies to the transfer of residues from wood to skin. The Panel is asked to address whether this is a reasonable assumption, and if not, to provide guidance on other approaches.**

**Issue: Selection of a Soil Adherence Factor**

The Soil Adherence Factor (AF) is defined as the amount of soil which adheres to the skin. The AF is highly dependent on the soil type, moisture content of soil and skin, amount of time the soil contacts the skin, and human activities. OPP adopted a dermal exposure scenario for children touching CCA-contaminated soil which relies on an AF of 1.45 $mg/cm^2$ (U.S.EPA' s Superfund RAG, 1989) for hand contacting commercial potting

soil in lieu of playground soil. A recently drafted report (U.S.EPA' s Superfund RAG, Part E., Supplemental Guidance for Dermal Risk Assessment, draft, 2000), recommended an activity-specific surface area weighted AF value for a child resident at a day care center (1 to 6 years old) of 0.2 $mg/cm^2$.

**Question 10: The Panel is asked to comment on whether the proposed AF of 1.45 $mg/cm^2$ for hand contact with commercial potting soil is a realistic value for use in estimating the transfer of residues from playground soil to skin in this assessment.**

**Issue: Variability of Residue Data Available for Soil and Wood.**

The soil and wood residue data being considered for this assessment has been generated over the last 25 years. There are several variables influencing the consistency of the data:

- Data were gathered and analyzed by several different research laboratories
- Data were collected at different geographic sites
- Differences in wood types and treatments between data sets

Additionally, the leaching rates of arsenic and chromium (to both the wood surface and the soil) are highly dependent on factors such as wood type, degree of weathering, age of wood, moisture content, pressure treatment process and retention time, use of coatings/sealants, and variations in the analytical and sampling techniques between laboratories.

OPP summarized the residue data by selecting and recommending some of the mean and maximum values from each study in order to compare the degree of leaching from the wood and the level of contamination in the soils. The "mean" data will be used to develop the lifetime average daily doses (LADDs) for the cancer assessment, and "maximum" data used in developing the average daily doses (ADDs) for the non-cancer assessment.

**Question 11: OPP will need to calculate intermediate-term, and possibly long-term exposures in this assessment using available wood/soil residue data. The Panel is asked to recommend a credible approach for selecting residue data values for use in OPP's risk assessment, taking into consideration the inherent variability of the data sets. Please advise us on which values are best for representing central tendency and high-end exposures. Also, the Panel is asked to discuss the feasibility of combining data from individual data sets.**

**Issue: Combining Multiple Exposure Scenarios into a Comprehensive Estimate of Risk**

Children playing on playgrounds containing CCA-treated wood structures will be exposed to arsenic and chromium residues on wood surfaces and in soils via oral and dermal routes. OPP has discussed four proposed exposure scenarios individually in the exposure assessment; however, to adequately assess the risks to children from exposure

to arsenic and chromium residues through playground contact with wood and soil media, all four scenarios must be considered concurrently.

**Question 12: Does the Panel have any recommendations for combining the four scenarios (oral/wood, dermal/wood, oral/soil, dermal/soil) such that a realistic aggregate of these exposure routes may be estimated?**

**Issue: Inhalation Exposure Potential from Wood/Soil Media**

The Agency has selected a NOAEL value of 2.4 x 10-4 mg/m $^3$ taken from the 1998 IRIS update for Cr(VI) using the study of Lindberg and Hedenstierna (Arch Environ Health 38(6):367-374) who observed ulcerations, perforations of the nasal septum and pulmonary function changes in 104 workers (85 males, 19 females) exposed in chrome plating plants at a concentration of 7.14 x 10-4 mg/m $^3$. The NOAEL value selected is intended to represent an endpoint for use in inhalation risk assessments representative of any duration of exposure.

OPP does not propose to evaluate potential exposures via the inhalation route for the child playground exposure assessment. The Agency anticipates that the inhalation potential from contact with either CCA-treated wood or CCA-contaminated soil is negligible. Neither arsenic *As(V)* nor chromium *Cr(VI)* residues are volatile on the surfaces of treated wood, or readily available as respirable airborne particulate concentrations. During play activities in CCA-contaminated soil, any airborne soil-bound residues that a child might inhale through the nose or mouth are not anticipated to contribute significantly to the overall exposure (i.e., exposure will be insignificant compared to the oral dose attributed to soil ingestion or hand-to-mouth activities).

**Question 13: Can the Panel comment on whether OPP should conduct a child playground inhalation exposure assessment, taking into consideration the hazard profile for chromium (VI) as an irritant to mucous membranes? If so, can the Panel comment on whether the endpoint described above is appropriate for assessing the risk to children from such an exposure?**

**Issue: Consideration of Buffering Materials as a Source of Exposure**

The CPSC specifies suitable loose-fill surfacing materials (e.g., wood chips/mulch, sand, gravel, and shredded rubber tires) for use under and around public playground equipment as shock-absorbing buffers (i.e., "buffering materials") to protect children from injury during a fall. (Handbook for Public Playground Safety, U.S. CPSC, Pub. No. 325) **.** Concerns surrounding use of these buffers include the potential for CCA compounds to leach from the CCA-treated playground equipment and absorb into the buffering materials. In addition, these buffers may include wood mulch products originating from recycled construction and demolition (C&D) debris that may contain varying quantities of CCA-treated wood. Coupling CCA-treated playground equipment with playground

barriers made from recycled wood mulch containing CCA-treated wood may increase background levels of arsenic and chromium, posing greater human exposure and health concerns.

Leaching studies conducted in Florida by Townsend et al. (2001) on new CCA-treated wood samples (wood blocks, chipped wood mulch, and sawdust) indicated that the concentrations of metals in leachate solutions were higher for wood processed into chips/mulch or sawdust over wood blocks. The degree to which wood leaches appears to be dependent on particle size since wood chips/mulch have increased surface areas available for leaching, and consequently exposure, over dimensional lumber.

Currently there are limited data available which adequately address the effects of leaching of CCA-treated wood compounds from playground structures to buffering materials used under and around these structures. A recent report released by Florida' s Alachua County Board of County Commissioners (2001) presents soil and mulch data from limited arsenic sampling conducted by Environmental Protection Department staff at five county owned parks. Tire chip and wood mulch buffering materials sampled at half-depth (2"-6") from areas immediately adjacent to CCA wood playground borders, playground posts, and within playground areas (between borders/posts) yielded arsenic concentrations for wood mulch of 43.1 - 61.2 mg/kg ( border) and 0.5 mg/kg (play areas), and for tire chips 3.5 - 70.3 mg/kg ( border), 10.3 - 80.3 mg/kg (posts) and 0.4 - 0.9 mg/kg (play areas). Each park had a liner in place between the mulch material and the bare soil.

**Question 14: Data on the effectiveness of reducing exposure by using buffering materials are limited. Does the Panel have recommendations as to whether additional studies to obtain this information are warranted? Does the Panel have suggestions on how OPP can best evaluate child exposures attributed to contact with CCA-contaminated buffering materials ?**

**Issue: Effectiveness of Stains/Sealants/Coatings at Reducing Leaching of CCA Compounds from Treated Wood**

Several researchers have reported that stains/sealants/coatings can reduce the rate of leaching of CCA compounds from treated wood and that the effectiveness of these coating materials over time varies greatly, depending on the type of surface coating used and environmental conditions effecting the wood. Stilwell (1998) reported over a 95% reduction in dislodgeable arsenic residues from CCA-treated wood surfaces coated with polyurethane, acrylic or spar varnish, and an average of 90% reduction for oil-based alkyl resins for samples tested one year after a sealant was applied. CDHS (1987) reported 96%, and 82% reductions in dislodgeable arsenic from stained CCA-treated wood surfaces after one month and 2 years, respectively. Lebow and Evans (1999) reported that staining CCA-treated wood surfaces reduced leaching of arsenic by 25%.

**Question 15: The Panel is asked to comment as to whether stains, sealants and other coating materials should be recommended as a mitigation measure to reduce exposure to arsenic and chromium compounds from CCA treated wood. If so, can the Panel comment on the most appropriate way for the Agency to recommend effective coating materials when the current data on long-term performance are limited and sometimes inconsistent, and should the Agency specify a time interval for the re-application of these selected coating materials? Can the Panel make recommendations for additional studies?**

## DETAILED RESPONSES TO THE CHARGE

**Question 1 Please comment on the Agency's selection of the 0.05 mg/kg/day LOAEL value for use in assessing risks to the general population as well as children from short-term and intermediate-term incidental oral and dermal exposures, and the appropriateness of the use of a 10x factor for severity of the toxic effects observed in the Mizuta study. Please provide an explanation and scientific justification for your conclusions as to whether the presented data are adequate or whether other data should be considered for selection of this endpoint.**

Recommendation

There was consensus by the Panel that 0.05 mg As/kg per day is an appropriate LOAEL for short- (1 to 30 day) and intermediate- (31 to 180 day) human ingestion of the chemical. The majority of Panel members expressing an opinion recommended a margin of exposure (MOE) of 30 from this LOAEL to afford protection from non-cancer health effects. Some Panel members thought an MOE of 10 would be adequate.

Discussion

Both Mizuta et al. (1956) and Fanzblau and Lilis (1989) described symptoms and clinical signs of arsenic poisoning in persons believed to have consumed 0.03 – 0.08 mg/kg per day for up to several weeks. Confidence in these dose estimates is low. Mizuta et al. (1956) did not provide information on their analytical method or on the basis for estimating the extent of arsenic consumption [from soy sauce] in patients experiencing arsenic toxicity. The information in Franzblau and Lilis (1989) pertaining to dose is derived in part from a retrospective estimate of water ingestion rates by two individuals who sporadically utilized an arsenic contaminated well.

Despite reservations about the dose estimates from these two studies, confidence in 0.05 mg/kg per day as an appropriate LOAEL is quite high in that several other clinical studies have reported the emergence of adverse signs and symptoms associated with the ingestion of inorganic arsenic at similar doses. These include accounts of gastrointestinal disturbances and less commonly mild peripheral neuropathy in individuals consuming medicinal preparations such as Fowler' s solution or liquor arsenicalis at doses of 5 to 10 mg of arsenite per day over a period of days to months (Stockman, 1902; Pope, 1902; Harter and Novitch, 1967). Daily doses of arsenic that were probably in the range of 1 to 5 mg arsenite per day for weeks to months resulted in gastrointestinal and peripheral neurological findings during the Manchester beer epidemic of 1900 (Reynolds, 1901; Kelynack and Kirby, 1901). Arsenic exposure in drinking water for 1 to 4 months was observed to result in gastrointestinal, neurological, and skin symptoms at doses estimated to be > 0.05 mg/kg per day (Wagner, 1979 as summarized in Benson, 2001). While each of these studies individually has limitations in terms of establishing a LOAEL, there is reassurance in the relative consistency of the LOAEL value they collectively provide. Confidence is further enhanced by the large overall number of subjects, the ethnic diversity of the subjects, and the inclusion of potentially sensitive subpopulations (including

children) across studies.

Several members of the Panel expressed the opinion that the severity of symptoms noted in some patients near or moderately above a LOAEL of 0.05 mg/kg per day warranted a full uncertainty factor of 10. Reports of peripheral neuropathy, gastrointestinal bleeding, liver damage, low blood counts, CNS dysfunction, and abnormal electrocardiograms were mentioned as examples of signs and symptoms of concern in these patients. Humans appear to be more sensitive than most laboratory animals to arsenic toxic effects, and there is little information on the shape of the dose-response curve for these effects in humans. Without knowledge of the dose-response relationships, it is difficult to forecast acceptable margins of safety. This uncertainty contributed to the recommendation that a 10X uncertainty factor be applied to the LOAEL of 0.05 mg/kg/day.

The Panel was divided on whether the MOE should include an additional uncertainty factor. The majority of Panel members expressing an opinion recommended that a MOE include an additional intraspecies uncertainty factor of 3 to provide for protection of children. They pointed out that there may be subpopulations of children at special risk of arsenic toxicity, such as individuals with concomitant toxic exposures and/or vitamin and nutritional deficiencies that might impact arsenic kinetics. They noted that there is a paucity of information on the toxicokinetics of arsenic and its metabolites in children, and that it is unclear whether there are age-dependent differences in GI absorption or biotransformation of arsenic that might influence toxicity. It was acknowledged that data available at present generally indicate that responses of children and adults to arsenic do not differ significantly qualitatively or quantitatively. However, the opinion was expressed that these data pertain largely to effects on the skin, and that the immature central and peripheral nervous systems may be quite another matter. Short and intermediate term exposure to sufficiently high doses of arsenic are neurotoxic in adults, and data offering insight as to the arsenic doses associated with neurological effects in children were viewed by these Panel members as lacking. Specifically, an absence of adequate studies monitoring neurological indices in children exposed at or near the proposed arsenic LOAEL was cited. Some Panel members expressed concern for childhood exposure to arsenic in combination with other neurotoxic metals, and for synergy with other toxicants. Toxicity data on combinations of arsenic and other toxicants are extremely limited, and some Panel members questioned whether a NOAEL developed for inorganic arsenic alone is applicable under circumstances of exposure to other components of CCA.

Some Panel members argued that an additional intraspecies uncertainty factor of 3X was not required, and that an overall MOE of 10 would be adequate to protect human health. It was noted that an MOE of 30 would identify doses above 0.0017 mg/kg per day as having the potential to produce adverse non-cancer effects with exposures of 180 days or less. For a 15 kg child, 0.0017 mg/kg per day is equivalent to 25 micrograms of arsenic per day. Since it is estimated that the background diet of a 3 year old includes approximately 5 micrograms of inorganic arsenic (NRC, 1999), the 30-fold margin applied to a 15 kg child would be akin to expressing concern regarding short- or intermediate-term doses greater than an additional 20 micrograms of arsenic per day. There are no data demonstrating acute or subchronic noncancer effects at this approximate level of exposure. The United States experience with respect to

existing levels of arsenic in drinking water was cited as evidence for this. The US EPA has estimated that there are in excess of 1200 public drinking water systems in the United States that deliver drinking water with arsenic concentrations in excess of 20 ug/L (US EPA, 2001). Since the level of water consumption by some 3 year olds is 60 ml/kg (90 $^{th}$ percentile estimate) (NRC, 2001), there appear to be many communities in the United States where young children have already been consuming >25 micrograms day. There are no reliable reports in the medical literature documenting or suggesting that adverse health effects from arsenic have occurred in these children. Several health surveys conducted in U.S. communities where the arsenic concentration in drinking water was several hundred micrograms per liter have also not detected adverse non-cancer effects (Harrington et al., 1978; Kreiss et al., 1983; Southwick et al., 1983). It was pointed out that both the Agency for Toxic Substances and Disease Registry (ATSDR) and U.S. EPA Region 8 have established health criteria for short- and intermediate-term exposure to arsenic of 0.005 mg/kg-day or higher, which is equivalent to an MOE of 10 or less [from a LOAEL of 0.05 mg/kg-day]. Finally, it was noted by one Panel member that clinical studies on children exposed to arsenic in drinking water associated the increased severity of observed multisystemic adverse effects in children compared to adults to a higher dose rate in children, and not to intrinsically increased susceptibility (Zaldivar, 1977; Zaldivar and Gullier, 1977; Zaldivar and Ghai, 1980).

Some Panel members cautioned that exposures above the MOE do not necessarily mean that health effects will occur and that the Agency should use the MOE in a screening level capacity only. That is, firm conclusions on the presence or absence of health effects should not be drawn solely on the basis of doses calculated to exceed the MOE.

**Question 2: Please comment on the choice of this data set and value chosen for representation of the relative bioavailability of inorganic arsenic from ingestion of arsenic-contaminated soil. Please discuss the strengths and weaknesses of the selected data and also provide an explanation as to whether this 25% value is appropriate for estimation of bioavailability in children.**

Recommendation

Panel members expressed a diversity of opinions regarding the designation of 25% as a value for the estimated relative bioavailability of inorganic arsenic from ingestion of arsenic-contaminated soil. Several members of the Panel felt that EPA should consider alternatives to a fixed value of 25% for the relative bioavailability of arsenic in soil in the vicinity of CCA contamination, while others felt that 25% was a reasonable interim value. Many members suggested an interim value of 50%. Several Panel members recommended that a range of values be considered: for some the suggested range was 25 to 50%, while another member suggested consideration of the full range of bioavailability for arsenic in soil reported in the literature (near zero to 98%).

In addition to oral absorption of arsenic from soil, consideration should be given to absorption of arsenic from nonsoil substances (such as wood chips or other buffer material) that might be subject to incidental ingestion.

Research is needed to obtain data on the relative bioavailability of arsenic from numerous sites that encompass the broad range of soil types and arsenic contamination specifically resulting from CCA-treated wood applications. These studies should be conducted in appropriate animal models preferably at doses that simulate the anticipated level of exposure of children playing on or around structures or sites subject to CCA contamination.

Discussion

There is general scientific consensus that a number of physical, chemical, and biological factors may impact the extent of gastrointestinal absorption of a substance present in ingested soil relative to absorption of the same substance ingested in solution. For arsenic, as with several other metals, solubility of the form of arsenic present in soil is a key factor, such that increased solubility or extractability of the metal from soil to an aqueous solution is positively correlated with increased absorption. Chemical and physical factors influencing the solubility or extractability of arsenic from the soil include 1) the molecular form of the arsenic species; 2) the nature of its chemical and/or physical interaction with the constituents of the soil matrix (e.g., chemical bonding, sorption, complexation, rinding, or encapsulation); and 3) the size, porosity, compaction, and surface area of the arsenic-containing soil particulates or agglomerations. Biological factors may also influence the absorption of an ingested metal present in soil, including 1) the species-specific metabolism of the metal, including metabolism by microflora within the gastrointestinal tract (Hall et al., 1997), 2) the physical condition of the animal at the time of ingestion (e.g., the effect of drugs, physical stress, toxins, nutritional perturbations, or disease states on the animal' s physiology), 3) the presence of other ingested material (food, drugs, or other substances) in the intestinal tract, and 4) in some cases the age and/or developmental stage of the animal. The dose regimen that characterizes the ingestion of the metal and the soil matrix may also exert influence on the absorption, in terms of either absolute amounts or the percent of the dose administered. For example, data on absorption of lead from soils (Kierski, 1992; Mushak, 1998) suggest that bolus administration of a large mass of metal and/or metal-containing soil matrix may be associated with a lesser degree of gastrointestinal absorption, in terms of percent of total ingested amount, than might result from administration of the same mass in smaller, divided doses.

Members of the Panel expressed concern that the findings of Roberts et al. (2001) and Freeman et al. (1995) have not provided a sufficient basis to establish a relative bioavailability of 25% for arsenic present in soil as a consequence of CCA related release or contamination. The single, high dose, bolus administration of arsenate and arsenic-containing soils used in the studies by Roberts et al. (2001) and Freeman et al. (1995) does not reasonably simulate the relatively low dose, repeated ingestion of arsenic-containing soil that would be anticipated with hand-to-mouth behavior of a child playing in the vicinity of a CCA application. The arsenic concentration of the test soils (ranging from 101 to 743 mg/kg) appears high relative to those measured in the vicinity of CCA-treated structures in children' s playgrounds in several recent investigations. The experimental design used by the investigators resulted in these soils being introduced into the monkey test subjects in single high mass boluses. For example, in the case of soil obtained from a "wood treatment site", it may be calculated that the soil-associated arsenic dose of 0.3 mg As/kg body weight was achieved by administering a 3 kg monkey a single oral dose of 9000 mg of soil. In like manner, in Freeman et al. (1995), the monkeys (which weighed between 2 to 3 kg)

were given single, oral doses of 3000 to 4500 mg of soil containing 410 ppm arsenic. Enhanced confidence in the generalizability of the relative bioavailability values from such studies might be obtained from experimental designs that utilize multiple, smaller soil doses spanning a range of relevant arsenic concentrations.

There is uncertainty regarding the extent to which the test soils used in the studies by Roberts et al. (2001) and Freeman et al. (1995) reflect arsenic speciation and chemical and physical characteristics of the soil matrix in the vicinity of CCA contamination at a playground. Although a soil sample from the investigation by Roberts et al (2001) was identified as coming from a "wood treatment site," this sample was not characterized further. The arsenic in that soil may have resulted in part from direct spillage of raw CCA product onto the soil, rather than leaching of arsenic from a weathered piece of CCA-treated wood.

The animal model used in the studies by Roberts et al. (2001) and Freeman et al. (1995) were the Cebus apella monkey and the cynomolgus monkey, respectively. Intravenous dosing with sodium arsenate suggested that these nonhuman primates were similar to humans with respect to excreting absorbed arsenic almost entirely through the urine (<5% of the recovered dose occurred in the feces). Also, the extent of excretion of an oral dose of sodium arsenate in urine and feces was quite similar between these monkeys and humans. At this point in time, the Panel is not aware of information regarding the biomethylation patterns of arsenic species in these nonhuman primates. This is an issue of some concern for some Panel members because other nonhuman primates, such as the marmoset monkey, do not biomethylate arsenic and exhibit prolonged retention of some arsenic species in vivo. These Panel members thought that this could potentially result in an underestimation of relative bioavailability if a significant proportion of the arsenic specie(s) present in the test soils was retained in the body for a longer period of time *relative* to the reference material, sodium arsenate in solution. Underestimation could also result if arsenic present in the test soil underwent greater relative biliary excretion compared to sodium arsenate. Other Panel members acknowledged these possibilities but expressed the opinion that these factors were not likely to significantly affect the findings.

At the present time, little is known regarding differential absorption and metabolism of arsenic in juvenile versus adult animals. Some Panel members expressed concern that the developmental age of the animal model might be a potentially significant variable, since it is known that infants and even older children as well as very young animals, sometimes have the potential for increased uptake of contaminants. Although the swine models have utilized juvenile pigs, the current monkey bioavailability data were obtained with adult animals. To the extent that the nutritional or dietary status of children and experimental test animals may affect the uptake of other substances, the absorption of arsenic (particularly arsenate) in the face of phosphorous deficiency is of potential concern for these Panel members. They noted, for example, that arsenate uptake by cells has been shown to be increased in low phosphate media (Huang and Lee, 1996) and suggested the need for further research on the impact of nutritional and developmental factors on bioavailability determinations. Other Panel members pointed out that the absorption of arsenite and arsenate, in absolute terms, is already extensive in adult animals and humans. As a result, the potential for greater absorption in children is limited, and consequently they did not think that use of arsenic bioavailabilty values from adult animals was

a significant concern (the Panel members thought that arsenic bioavailability values from adult animals were applicable to immature subjects).

As discussed in more detail elsewhere in this report, the interactive effect of metal combinations may influence arsenic absorption, biotransformation, and excretion. For example, when administered together with selenite, some inorganic arsenic compounds undergo increased biliary excretion (Levander, 1977; Gailer et al., 2000), a factor that may potentially serve to underestimate relative bioavailability in models that examine relative urinary excretion as a marker of relative bioavailability.

Panel members noted several other studies that have investigated the oral bioavailability of arsenic in soils. Widely divergent results for relative bioavailability have been reported, a finding that is not unexpected given the variability in soil-associated arsenic compounds, soil matrices, animal models, and experimental design. For example, Casteel et al. (2001), under the auspices of U.S. EPA Region VIII, recently examined the relative bioavailability of arsenic in soils from the VBI70 superfund site in Denver, CO. Using a swine model that investigated six soil specimens spanning a range of arsenic concentrations, the mean relative bioavailability was 31%, with a 95% upper confidence limit of 42%. This latter value (42%) has been utilized in risk calculations contained in the site' s baseline risk assessment (US EPA, 2001). Other relative bioavailability studies have been noted or reviewed in the Inorganic Arsenic Report of the Hazard Identification Assessment Review Committee (HIARC, 8/21/2001), and a recent publication by Ruby et al. (1999). Results for relative bioavailability have ranged from near zero to 50%, with the exception of two soils from Aspen, CO, that yielded much higher results, albeit with extremely wide confidence intervals (62% ± 55, 98% ± 86; Casteel et al., 1997; Ruby et al., 1999).

**Question 3: Please comment on the selection of the value of 6.4% for dermal absorption of inorganic arsenic and whether or not this value will be appropriate for use in all scenarios involving dermal exposure to arsenic from CCA-treated wood, including children's dermal contact with wood surface residues and contaminated soils.**

Recommendations

The Panel recommends that EPA use a value less than 6.4%, probably in the range 2-3%, for dermal absorption of inorganic arsenic. The Agency should consider using a figure for absorption rate (e.g., percent exposure absorbed per hour) rather than a value for percent absorption.

Research, using arsenic in more appropriate chemical form (that it is present in dislodgeable CCA residues and in soil beneath CCA-treated sites) and in a relevant matrix, should be carried out to improve estimates of dermal absorption.

Discussion

The Panel accepted the EPA view that the publication by Wester et al. (1993) provided the most appropriate available data for addressing this question. This research included a study that used seven rhesus monkeys. The absorptions of the pentavalent arsenic species $H_3AsO_4$, radiolabeled with $As^{73}$, in water solution and added to soil, were compared. For both water and soil, a high-exposure and a low-exposure group were used. The low exposure groups represented "the minimum arsenic that could be used given the specific activity of the compound." These exposures were 0.00004 $\mu g/cm^2$ for the soil group and 0.000024 $\mu g/cm^2$ for the water formulation group. The high exposure group was described as "representative of what would be encountered in more contaminated areas." These exposures were 0.6 $\mu g/cm^2$ for the soil exposure group and 2.1 $\mu g/cm^2$ for the water group. The skin exposures were for 24 hours and confined within a non-occlusive cover. Urine was collected for seven days following the beginning of the exposure period. Results were adjusted for excretion by other routes than urine and for retention in the body using results from monkeys that had been treated with an intravenous dose of arsenic. The data showed that 80.1% of the intravenous dose administered to the monkeys was excreted in the urine over a period of seven days.

The Panel noted a number of limitations in the reporting of the design and conduct of the study by Wester et al. These included:

- Uncertainty whether separate monkeys had been used for the dose groups or the same monkeys had been reused. If the latter, then it raised the possibility of cross-contamination, such that there could have been continued "slow leaking" of arsenic from body reservoirs that would have affected latter parts of the experiment.

- The large particle size of the soil used. Particle size is likely to affect bioavailability because of differences in surface-to-volume ratio.

- The procedure by which arsenic was added to the soil was not described. The contact period prior to skin application appears likely to have been very short relative to the time that would be necessary for binding to the soil particles.

- The water in which the arsenic was dissolved was not adequately described in terms of its chemical characterization.

- There was no information on whether the cage was washed to collect radioactivity and, if so, how this was taken into account for the purposes of calculating the absorbed dose.

- To keep the soil on the skin, a device made of two aluminum eyeguards sandwiched around a Goretex membrane was taped over the soil after application to the skin. The soil used had a particle size distribution larger than would typically be expected to adhere to skin. It is likely that soil fell to the bottom of the dosing device (which had a larger volume than the volume of the soil applied) and was not uniformly distributed on skin.

- The applicability of soil and water data to arsenic residues derived from wood surfaces is unknown.

Results of the study by Wester et al. (1993) were as shown in the following table:

***In vivo* percutaneous absorption of arsenic from water and soil on Rhesus Monkeys**

| **Percent applied dose** | | | |
|---|---|---|---|
| **Water** | | **Soil** | |
| Low exposure | High exposure | Low exposure | High exposure |
| 6.4 ± 3.9 | 2.0 ± 1.2 | 4.5 ± 3.2 | 3.2 ± 1.9 |

An inverse relationship between exposure concentrations and percent absorbed was noted. The lowest exposure (water formulation) was associated with the highest percent absorbed – 6.4%. This value was proposed by EPA as the default value for skin absorption.

The Panel considered that, on general toxicological principles, for the purpose of extrapolating the results to humans, it would be more appropriate to have results obtained using a more realistic level of exposure, namely the higher exposure level used in the experiment. In that regard, it was also noted that the soil group had a higher degree of arsenic absorption (3.2%) than the water formulation group (2.0%). There were two possible reasons why this might have happened: 1) a factor in the soil that promoted exposure or 2) random biological variation because of the small numbers of monkeys in the groups (three monkeys in the water formulation group and four in the soil group). The Panel considered the second reason the more likely.

On this basis, the Panel considered that a value for skin absorption in the range 2-3% would be more appropriate than 6.4%. However, it was felt that this was likely to overestimate actual absorption, because the monkeys had been exposed for 24 hours and the form of arsenic used in the experiment was probably more water soluble than arsenic from CCA treatment. Also, the treated soil had had no opportunity to "age," a process that could bind the arsenical molecules more tightly to the soil matrix and reduce absorption. The possibility of considering a measure of absorption that took into account time of exposure (i.e., absorption rate) was felt to be worthy of further consideration.

A separate *in vitro* experiment reported by Wester et al. involved measuring absorption of arsenic from water and soil on human skin. This gave a result of 0.76 % absorption from soil and 1.9 % from water.

It was also noted that there was a lack of information on the chemical form of arsenic in CCA residues. The assumption was that residues were likely to be in the pentavalent form. If there were trivalent arsenic present, then the kinetics of arsenic absorption could be different. However, there is no available information on skin absorption of trivalent arsenic compounds.

In view of the limitations of the research on which this evaluation was based, the Panel considered that there was an urgent need for further research on skin absorption of CCA residues, employing the form of arsenic found in dislodgeable residues and soil from CCA-treated installations.

The Panel also considered a proposal by two of the public presenters – Gradient and E$^{X}$ponent – to adjust the percent absorbed (as determined by Wester et al.) by a factor representing the relative bioavailability of gastrointestinal absorption of CCA arsenic residues. The rationale for this adjustment was that the Wester experiment had used a more water soluble form of arsenic than was present in CCA residues, and it had not had the opportunity to "age" after being added to soil. The Panel accepted that the form of arsenic used in the experiments by Wester et al. (1993) was not ideal, but considered it inappropriate to adopt this proposal for an adjustment factor, since it would involve a form of "double-counting" of soil-related factors that reduce absorption. It was felt that the recommended research would better address the issue identified by Gradient and E$^{X}$ponent.

**Question 4: As available monitoring data do not differentiate among chromium species found in CCA dislodgeable residues on wood surfaces and in soils, and as Cr (VI) is the more toxic species of chromium, please comment on whether use of the hazard data for chromium (VI) is the best choice for characterizing hazard and risk from exposure to chromium as a component of CCA-treated wood. Please provide a scientific explanation and justification for your recommendation on the choice of either the chromium (III) or chromium (VI) hazard database.**

Recommendation

It is the Panel's conclusion that, at present, there is no reliable evidence on either the presence or absence of Cr(VI) in dislodgeable residues on treated wood surfaces. Some measurable Cr(VI) probably exists in certain soils, but it is unlikely to be 100% of total chromium. One approach would be to use an estimate of 25 to 50% hexavalent chromium. Some Panel members suggested 5 to 10% would be conservative. In order to be health protective, it would be scientifically reasonable to use the Cr(VI) hazard database with respect to a range of chromium fractions. The Panel strongly recommends that EPA conduct studies of chromium speciation (in both dislodgeable residues and soil samples) in their proposed studies.

Discussion

There was little disagreement that available information on the valence state of chromium did not establish either the presence or absence of Cr(VI) in dislodgeable residues. The speciation data presented at this meeting are limited. However, the limited Florida data suggest that Cr(VI) may not be a major environmental hazard, even as we acknowledge that the hexavalent form of chromium is the more significant health hazard. Further research on valence speciation of chromium at sites where treated wood is being used is warranted.

Several members of the Panel noted that detection of Cr(VI) would be confounded by the fact that Cr(VI) in dislodgeable residues would be much more soluble and therefore more mobile to transport off treated surfaces with rain events. This complication could be accounted for in the future pilot studies being planned by EPA and CPSC. The Panel did not elaborate further on the issue of Cr(VI) in dislodgeable residues, and the rest of the Panel' s focus was on valency issues for soil chromium.

Several members of the Panel noted that there is some evidence from two types of studies that Cr(VI) can exist in soils in measurable amounts. The fraction of Cr(VI) in soils is highly dependent on such soil characteristics as moisture content, pH, binding sites for adsorption on mineral and organic components, etc. One line of study entailed experimental evaluation of Cr(VI) formation and stability in soils of differing chemical, moisture, and complexing types (Bartlett, 1991; Bartlett et al., 1983; Bartlett et al., 1979).

A critical factor in formation of Cr(VI) in undisturbed, non-acid soils of typical moisture content is the oxidation of Cr(III) to Cr(VI) by manganese oxide. Bartlett also reported that Cr(VI) can be adsorbed and stabilized to some extent.

A second body of information consists of determination of the fraction of Cr(VI) in soils in Hudson County, NJ, which received alkaline chromite ore processing residues. The fraction of Cr(VI) ranged from 1-50% (Burke et al., 1991). At this site, the amount of Cr(VI) was seldom more than 10%. In situations where the chromium was in solution in surface water, chromium blooms (crystallization) occurred on the soil surface as the soil dried out and contained up to 50% Cr(VI).

The overall discussion by the Panel of what fraction of Cr(VI) in soils should be adopted by EPA in any preliminary risk assessment efforts elicited a range of values and a variety of conclusions as to their significance. This variety of opinions included a desire to wait for the pilot studies being planned nationally before going any further. Some members indicated a range from 5-10% would be conservative. Other Panel members indicated a range of 25 to 50%. One Panel member noted that it was inappropriate to consider the chromite ore residue data in New Jersey.

It was generally the view that it would be unlikely that 100% of total chromium would be present as Cr(VI) in playground and deck areas. Conversely, no Panel member tendered the view that Cr(VI) would never exist in any soils associated with playgrounds and/or decks constructed of CCA-treated wood.

The Panel generally was interested in having the planned studies by EPA and CPSC include as much chemical speciation data as possible -- much more than the agencies had indicated in their draft protocols.

One Panel member, with assent from others, noted that the issue of chromium valency in soils is not subordinated to the subsequent redox transformations that might be assumed to occur in the stomachs of children ingesting soils containing Cr(VI). That is, any transformation of Cr(VI) to Cr(III) in receiving body compartments (lung, GI tract) occurs via uptake of Cr(VI) and

formation of intermediate, bioreactive valencies that may be linked to the mechanism of the toxicity of Cr(VI).

**Question 5: Please comment on the Agency's selection of the 0.5 mg/kg/day NOAEL value for use in assessing risks to the general population as well as children from short-term and intermediate-term incidental oral exposures to inorganic chromium as contained in CCA-treated wood. Please provide an explanation and scientific justification for your conclusions as to whether the presented data are adequate or whether other data should be considered for selection of these endpoints.**

Recommendation

The Panel expressed concerns regarding the selection of the 0.5 mg/kg/day NOAEL for short-term and intermediate-term incidental oral exposures to inorganic chromium. In general, these concerns involved the appropriateness of the study selected by EPA (Tyl, 1991) to derive this value. It is the Panel' s recommendation that the Agency re-review the literature and consider other potentially more relevant studies.

Discussion

The SAP agreed that the most appropriate toxicology data for the development of the NOAEL should involve the same species of chromium as present in CCA-dislodgeable materials and contaminated soils which are the subject of the risk assessment. Given the absence of appropriate data, this decision will ultimately depend on the results of field studies such as the playground studies proposed by EPA/CPSC.

The Panel questioned whether the study proposed for the derivation of the NOAEL (Tyl, 1991) actually demonstrated the purported effect. The Panel was divided on this issue; some thought the study adequate and appropriate to support the proposed NOAEL while others thought the study to be flawed and inappropriate.

- The selected study had the primary purpose of evaluating the reproductive and developmental effects of exposure to Cr(VI). While no developmental effects were observed, maternal effects were noted and were used to derive the NOAEL. Rabbits were given a bolus of chromic acid ($CrO_3$) diluted in distilled water by gavage for twelve consecutive days. Dosing was at 0, 0.1, 0.5, 2.0, 5.0 mg/kg/day levels; the 5.0 mg/kg/day dose was noted to have a pH of 1.52. Maternal effects observed included mortality in the 2.0 and 5.0 mg/kg/day groups and reduced weight gain, decreased food absorption efficiency, labored breathing, and diarrhea in the 5.0 mg/kg/day group. No pathologic abnormalities were noted in any group.

Several members of the Panel questioned the attribution of the observed effects to the Cr( VI) dosing; they believed that an acid effect could not be ruled out. Others discounted this possibility stating that dietary residues could readily neutralize the acid. Specifically, it was noted that rabbits retain half their ingested diet 24 hours after conventional fasting began

(Carmichael et al., 1945) and this residue would serve to buffer against acid injury from acidic media given after conventional fasting. There was no resolution of this difference in interpretation.

Studies cited by the Agency (MacKenzie, 1958) and Zhang and Li (1987) are generally supportive of the NOAEL value derived from Tyl, but, as noted by OPP, these studies suffer from a lack of definitive exposure information and are of inappropriate duration for use in deriving a short or intermediate measure.

**Question 6: Please comment on whether the significant non-systemic dermal effects from dermal exposure to inorganic chromium should form the basis of dermal residential risk assessments, and, if so, how the Agency should establish a dermal endpoint for such an assessment.**

Recommendation

The Panel advises that EPA should base risk assessments for noncancer health effects of dermal exposure to hexavalent chromium on direct dermal effects – irritant and allergic contact dermatitis. The Panel was unable to provide EPA with methods for establishing endpoints and determining dose response relationships for these effects.

Discussion

It is unlikely that sufficient chromium could penetrate the skin and enter the circulation to cause systemic effects from dermal exposure. Skin penetration for chromium is estimated to be 1%. It is usually assumed that the contribution to systemic effects from dermal exposure is not likely to be significant relative to oral exposure. Direct dermal effects (irritation and allergenicity) are therefore likely to be the controlling endpoints as far as dermal exposures are concerned. The Panel therefore advises that EPA base its residential risk assessments for the dermal route on these direct dermal effects. In order to make sure this route is inconsequential for systemic effects, one could run a PBPK model and compare the target tissue doses from the oral and dermal routes.

The Panel believes that EPA should consult with the New Jersey Department of Environmental Protection (see Bagdon and Hazen, 1991) concerning establishing dermal endpoints and performing dose response assessments for dermal exposure to chromium using direct skin effect endpoints. The main problem will be determining the appropriate endpoint and obtaining a usable dose estimate from the published literature using data from exposure of workers and other exposed individuals (Bagdon and Hazen, 1991; Burke et al., 1991) or possibly from animal experiments mentioned in the ATSDR document (Mor et al., 1988; Gross et al., 1968; Jansen and Berrens, 1968).

**Question 7. Please comment on whether OPP's choices of central tendency and high end values for different parameters should, collectively, produce estimates of the middle and high end of the range of potential exposures. If the Panel thinks that OPP's approach may not estimate the high ends of the exposure range**

**(because it produces values that are either higher or lower than the upper end of the exposure range), please comment on what specific values should be modified to produce estimates of the high end of potential exposure.**

Recommendation

The Panel offers the following recommendations:

- Particularly when using point estimates it is important to do subset analyses for specific regions of the country (for example, the South compared to the North or Midwest) and for age groups (for example, one year olds compared to 5-6 year olds).
- The averaging of exposure over a 75-year lifetime may underestimate risk. More research is needed to understand the uncertainty associated with this form of temporal averaging.
- More research is needed on the amount of soil ingested, as this is still a source of uncertainty.
- For fully evaluating high end exposures it would be necessary to include exposure of children with Pica.
- A probabilistic assessment as discussed in question 8 is recommended.

Discussion

Comments of OPP' s choices of central tendency and high-end values for different parameters have been approached in two ways: assessing the quality of the specific values OPP has presented and evaluating whether the point estimates used in the Agency' s calculations will provide reasonable estimates of the high-end exposure range.

Specific values

The prototypical three year old behaviorally does not represent either a one year old or a six year old. In addition, the surface area used for fingers, 20 $cm^2$, while appropriate for a three year old, would be an overestimate for young children, since both the surface area of the hand and the proportion of the surface area which is fingers are different for younger children. This is an argument for either doing subset analyses for smaller age groups, or doing probabilistic evaluations.

Time spent at play outdoors may be an overestimate for the measure of central tendency. Both NHAPS data and that reported by Silvers et al. (1994) suggest that most of the time children are at play outdoors is on grass or paved areas, neither of which represent the types of substrates typically found around CCA wood play structures.

The assumption that the average child spends 130 days playing on these structures is also not a realistic central tendency measure. The National Human Activity Pattern Survey (NHAPS) data for children 1-4 years old suggests that on any day only 50% of children may play outdoors and that, of those, approximately 40% would play on the types of substrates on which play equipment is found. Data presented from Florida suggest that there may be major regional differences in these estimates which would not be treated well with point estimates unless regional subset analyses were done.

The hand to mouth frequencies proposed are based on both indoor and outdoor mouthing periods. Freeman et al. (2001) found that, among children in Minnesota, mouthing rates were significantly higher indoors than outdoors (approximately 3 times higher indoors). At the same time, if residues or soil adheres to hands, the ingestion of that material may not take place outdoors, but occur indoors after the child has played on the equipment. Freeman in her own evaluations has shifted to using the median value of Reed' s data (8.5/hr) rather than using the mean of 9.5/hr as a more conservative value for a measure of central tendency. It should be noted that most mouthing behaviors occur indoors during quiet times such as when watching TV. It has been infrequently observed during active outdoor play other than by infants and very young toddlers.

The issue of whether replenishment occurs after mouthing needs to be addressed. Contacts with surfaces and objects are "fast and furious," with the average contact duration of 4 seconds and hundreds occurring per hour. If total replenishment occurs after 4-5 contacts (Rodes et al., 2001), then it is likely that the fingers are fully loaded between mouthing events.

Some Panel members noted that the central value for soil ingestion rates of 100 mg/day is probably an overestimate. Median values reported by both Stanek and Calabrese (1995), and Davis (1990) range from 0-96 and 25-81 mg/day, depending of the tracer used. Other estimates range between 35-70 mg/day and may be more realistic (Sedman, 1989 and 1994; Calabrese, 1995). These values also represent total soil consumption for the day, and not just from the 1-3 hours of play by CCA treated equipment, which would be something less than 100% of daily soil consumption. In addition, the use of 400 mg/day as the high-end value is also an overestimate based on Calabrese and Stanek' s work.

In considering high-end exposures, the Agency might consider including an evaluation of children with Pica behaviors. This is an area for which there are little data but may be important for understanding the high end exposures.

<u>Reasonable estimates</u>

The dermal and ingestion models proposed are very simplistic, but there is no harm with trying them and trying a variety of inputs as a first step in understanding exposure and risk. There may be no point trying to agree on a correct set of inputs at this time. Additional data on dermal and ingestion exposure will improve the models and reduce uncertainties. All of the coefficients and parameters seem to be conservatively biased toward overestimating exposure. When inflated "central tendency" values are put into the deterministic exposure calculation, they can be expected to overestimate the expected or "central tendency" exposure. If the distribution of exposure is highly positively skewed, this bias may be considerable. In some cases the arithmetic mean values presented are substantially skewed and should be replaced by median values as a better indicator of central tendency.

Working with the high-end values will be even worse, as the result will correspond to the very rare event of an exposure that is extreme in every aspect and hence will be higher than is ever observed in reality.

Looking at the general variables that the Agency proposes to use for characterizing child exposures through dermal and oral routes, the general conclusion is that using a range of values in a probabilistic evaluation should be the way to approach evaluating child exposures. Issues related to a probabilistic model are discussed in Question 8.

If the deterministic model is used, any parameters that are unnecessarily inflated should be reduced. One value to look at first is the calculation of skin surface area, which could be replaced by the "effective skin surface area." The hours per day of playground activity could also be looked at, the days per year will probably vary regionally.

The models proposed by the Agency for the study of children's acute and chronic CCA metals exposures (ADD-average daily dose and LADD-lifetime average daily dose) from play structures involve the composition (through multiplication and division) of stochastic variables from multiple sources and transitions in the dermal and oral exposure routes. The true composite exposure distribution is expected to be right skewed (e.g., log normal or similar distribution). It is also expected that the distribution is left censored—rarely at zero exposure—but at other exposures not related to playground or play structure use.

Estimates of ADD and LADD distributions, their means, medians, and quantiles, should reflect the distributional parameters (means and variability) of each of the exposure components, the covariance of the exposure components, and, through sensitivity analysis, the uncertainty (variance and bias) of the sample-based or postulated value of these parameters. In addition, the influence of covariates (e.g., region, climate) not explicitly included in the estimation model must be taken into account.

The proposed estimators of ADD and LADD are simple product and/or ratio statistics. In the simple case of a product of two variables, the product of central tendency values is

$$E(X)\,E(Y) = E(XY) - Cov(X, Y)$$

This implies that for positively correlated X, Y the product of means will underestimate the mean of the product.

Furthermore, if these central tendency measures are estimated from sample data, the approximate variance of the product is

$$Var(X\,Y) = Var(X)\,E^2(Y) + Var(Y)\,E^2(X) + 2\,Cov(X, Y)\,E(X)\,E(Y)$$

That is, the variance of the product will be inflated if there is a positive correlation between the variables. For statistics such as the proposed estimators of the ADD or LADD, the properties illustrated here for means of two distributions will be propagated through calculations involving means of more than two variables.

The median of the product X Y may not be the product of the median even if the two distributions are uncorrelated. To illustrate, take two simple discrete uniform distributions, X=(1,

2, 3), Y = (2, 8, 14). Generate all possible (X, Y) pairs and create the distribution of their product (e.g., 1 x 2=2, 1 x 8 = 8, etc.) assuming no correlation. The composite product distribution includes 9 equally likely values X Y=(2, 4, 6, 8, 14, 16, 24, 28, 42) with median = 14, but the product of the medians of the X and Y distributions is 2 x 8 =16.

Likewise, the product of the medians and other distributional quantiles (e.g., $Q_{90}$, $Q_{95}$) for X and Y that are positively or negatively correlated will be biased for the quantile of their product distribution. The direction and magnitude of the bias will depend on the size of the correlation and the shape (symmetry and variability about mean) of the distributions of X and Y. The theory here is based on Dirichlet distributions for the products of order statistics.

The alternative to the deterministic approach that is proposed is a probabilistic modeling of the exposure routes. Bayesian methods, possibly with flat priors over the range of measured parameter values, might be considered as the probabilistic approach is developed.

**Question 8: Please comment on whether the existing data bases on the variability of the different parameters affecting potential exposure are adequate to support the development of probabilistic estimates of potential exposure. If the Panel regards the data bases as adequate for that purpose, please identify which parameters should be addressed using a distribution of values and which data bases should be used to supply the distribution for particular parameters.**

Recommendation

In view of its concerns that the deterministic model reviewed in Question 7 will not correctly estimate the central tendency or percentiles of the exposure distribution, the SAP recommends that the EPA immediately begin to take steps toward the development and progressive refinement of probabilistic models of exposure. The probabilistic models will give high-end values that are interpretable as a percentile of the modeled exposure distribution rather than a biased approximation of the upper limit of exposure. The existing databases on the variability of the different parameters affecting potential exposures of children using CCA-treated playground structures are adequate to begin the development of probabilistic estimates of potential exposure provided the uncertainty associated with these data is reflected in the exposure modeling. As noted above, the Panel views the development of a probabilistic assessment as a process of progressive learning and refinement. New or more detailed data on states and transition factors are needed and will contribute to improvements in the exposure models as they become available.

Discussion

The Monte Carlo risk assessment of CCA metals exposures presented by the Environmental Working Group (EWG), while it contains several deterministic and simplifying assumptions, is a good start and illustrates what can be done with existing data. The use of a probabilistic approach avoids the arbitrariness and artificiality of selecting single values to represent factors that are known to vary considerably across individual exposures. This advantage is of particular

importance for the case of the RME estimates, where it has been demonstrated that the selection of reasonable upper bounds for several distributed parameters used as independent variables in a calculation can result in an estimate of the dependent variable (e.g., exposure) that is unreasonably far out in the tail of the probability distribution for that variable.

EPA guidelines for probabilistic exposure assessment and software programs like SHED, ReX, LifeLine, and Calendex can be used to perform a multi-dimensional analysis (separating, to the extent possible, variability and uncertainty). The LifeLine and Calendex models that have been reviewed by prior SAPs and proposed for studies of cumulative (including residential) exposure to organophosphates provide basic algorithms for beginning probabilistic approaches. One advantage of these programs is that they permit inspection of individual exposure values and the cumulative contribution of individual components to aggregate exposures. In these software systems, varying degrees of deterministic analysis can be forced by simply limiting the variability of the stochastic distributions.

The Panel recognizes that probabilistic exposure assessment is a relatively recent advance in risk assessment methodology. Therefore, a parallel approach is suggested, in which deterministic exposure estimates may be determined quickly and in advance of probabilistic estimates primarily to develop an initial understanding of which parameters have the greatest leverage on the final distribution of exposure outcomes. In addition, it is recommended that a limited variability analysis, similar to that presented by EWG, be performed as well as a full variability/uncertainty analysis. If a probabilistic approach is used, the 50 $^{th}$ and appropriate upper percentiles can be used as the central tendency and high-end estimates, respectively. Even if the Agency determines that a probabilistic approach cannot be used for the exposure estimates, the probabilistic results will play an important role in the risk characterization, for characterizing the variability of individual risk, the impact of uncertainty on the risk estimates, and the suitability of the selected central tendency and "high-end" values used in the deterministic calculations.

As mentioned above, a limited variability analysis could be performed in a similar fashion to the analysis presented by EWG. In this case, the Monte Carlo analysis would only vary parameters for which substantial data on variability are available. In the EWG analysis these included daily soil ingestion, dislodgeable arsenic, soil arsenic, and body weight. The approach used by EWG for body weight and surface area, following a child from 12 to 84 months of age with the opportunity for moving, is recommended, since it permits the incorporation of other age-dependent parameters such as mouthing behavior.

The details of the Monte Carlo analysis will need to be determined by considering the presumed nature of the anticipated exposures and the available data. For example, if it is assumed that a child uses a single playset for the entire 6 years, then a single randomly selected data set for dislodgeable arsenic can be associated with each child. If it is assumed that a child would use more than one playset, a more complicated sampling approach would be necessary. Alternatively, a very simple approach would be to repeatedly select at random from the totality of the data for dislodgeable and soil arsenic and use these selected values in the deterministic formulas together with the assumed values for the other parameters. The Panel encourages the

Agency to plan for modeling the exposure risks not only of special scenarios of individual exposures but also exposures in special high-risk populations. Such high-risk populations might include children in day care settings and living in warm climates, where the exposures to CCA treated wood may be more frequent and of longer duration than in the general population at large.

The more complete two-dimensional analysis should include distributions for all of the parameters in the exposure calculations. To the extent possible, separate distributions would be developed to describe variability (inherent variation) and uncertainty (lack of certainty regarding the correct value). The resulting exposure estimates would take the form of a "distribution of distributions" in which the variability around the central estimate would be displayed in one dimension while the uncertainty in the central estimate would be displayed in the second dimension. The multiple curves presented by EWG for different parameter assumptions is a (simpler) example of such a concept. However, instead of just showing the variability results for discrete alternatives of the uncertain parameters, distributions would be presented. The distributions used to describe uncertainty are necessarily much simpler than those informed by variability data. For example, a parameter may be characterized as having a uniform distribution (with equal likelihood of being anywhere in a given range), or by a triangular distribution (with a peak at the best estimate and vertices at the extreme values). The biggest problem with this approach is that there is often considerable uncertainty whether the observed differences in the value of a parameter represent variability or uncertainty or variability compounded by experimental bias (which introduces uncertainty). It is usually valuable to perform an analysis on the probabilistic approach that evaluates the sensitivity of the conclusions to alternative decisions that could be made regarding the variability/uncertainty distributions. As mentioned earlier, even if it is decided that the use of such an analysis for the risk assessment would not be appropriate, the results of the analysis would be very useful for characterizing the potential impact of uncertainty on a risk assessment using a partial probabilistic/deterministic approach.

Publications and presentations given to the Panel indicate that more data are needed to characterize other sources of variation and that there are more factors that need to be included in the model.

One area of improvement that could be addressed immediately is better representation of age-specific differences in children' s body size and behaviors. For modeling population exposures, a first improvement over the current assumption of a fixed age and body weight for exposed children is to draw on data for children that are available from major survey data sets such as the National Health and Nutrition Examination Survey (NHANES). The EWG simulation study presented to the SAP by Dr. Houlihan used this approach. These samples of children provide representative age- and gender-specific data on body weights and heights for U.S. children. Modeling of exposure should adopt the methodology employed in LifeLine and other software that "ages" the child through the exposure window. As the child ages in the probabilistic simulation, the appropriate age-specific activity and exposure factor data are applied to estimate time-dependent contributions to short- and long-term exposures.

Another major source of uncertainty in the current model of exposure is the data on the

distribution of frequency and duration of children' s exposures to CCA-treated wood play structures. The deterministic model proposed in the EPA presentation to the SAP makes a very simplifying assumption of constant daily and annual exposure frequency for six years of life. Obtaining precise, nationally representative time and activity data for children in the relevant age ranges would be tremendously costly. However, small local studies and existing small-sample data from the National Human Activity Pattern Survey (NHAPS), the Child Supplement to the Panel Study of Income Dynamics (PSID; http://www.isr.umich.edu/src/child-development/publications.html ), and other studies could be used to better approximate the variability in frequency and duration of outdoor play. These data could then be interpolated to approximate time spent on CCA-treated play structures. We need more detailed information on the relative time spent on the structure and in the substrate. These data might be obtained from existing or new observational studies. Activity patterns will very likely depend on the weather, as children may, for example, avoid sand that is too hot or too wet. Data on the correlation between As/Cr in the structure and its substrate will be needed to use this information.

The EPA is planning a new survey of existing playground structures and substrates. These should be executed as one combined survey to look for correlations between existing structures and their substrate. All possible covariates should be recorded in the hope that the "unexplained variation" in As and Cr levels could be reduced from what we have seen in studies to date. Covariates might include the following: evidence of construction debris (sawdust) in the substrate, nature of substrate (clay, sand, etc.), source of wood, age of structure, condition of surface (new, aged, worn to a shine), climate. Initially, the probabilistic modeling could rely on the empirical distributions provided by Townsend, et al. and Stillwell data on soil and surface residue concentrations. The Panel expects that the new survey data will be substituted when they become available. In its response to Question 11, the Panel recommends that data obtained in studies of CCA treated decks not be viewed as representative of dislodgeable residues on CCA treated play structures or in the soils or substrates beneath these structures.

Panel members also identified the transfers of CCA residues from surfaces and soils as a major uncertainty factor in the modeling of exposure. For example, it is possible that wet-weather play and play on damp structures bring increased risk of uptake, but there seems to be no information other than wet-hand/dry-hand wipe studies. The Panel strongly recommends that the EPA explore and evaluate alternatives (by comparison) to the hand loading transfer efficiency in modeling the transfer of CCA metals from surfaces and soils to the child' s hands and other skin surfaces. Specifically, the Panel recommends that the EPA conduct direct hand loading measurements in samples of children (preferably) or adults (if human subjects concerns intervene). The best empirical data may actually be collected through sampling of children who are actively involved in playing on CCA treated structures. The Panel also cautions that empirical distributions of arsenic and chromium concentrations measured in these hand loading studies not be used as the concentration values for dermal exposure through non-hand skin surfaces. One Panel member noted that probabilistic exposure models should allow occasional events like splinters and abraded skin to be included in the exposure pathway.

The Panel also noted that the better distributional data on children' s outdoor hand-to-mouth frequency and the fraction of residue transfer are needed to improve the probabilistic modeling

of children' s exposure. Data from Dr. Natalie Freeman and her colleagues is expected in the Spring of 2002. Preliminary results will be presented at a conference in early November, 2001.

In its response to Question 8, some Panel members noted that there ultimately should be a biomonitoring study that does a reality check on the predictions of the model, perhaps arranging a sample of children to play in a CCA-free environment for several months and comparing some measure of arsenic uptake with the same measure in a matched sample using existing CCA-treated playgrounds. The suggestion was made to collect urine samples from children during the time period after they had actively played on treated and untreated structures. A further comment was made that any analysis of arsenic in urine should examine the speciation of the arsenic. The topic of biomonitoring studies was discussed at length at the conclusion of the question responses. The reader is referred to the general summary of this discussion for a summary of the Panel' s recommendations on the need and design issues for biomonitoring studies.

**Question 9: OPP is assuming that a one-to-one relationship applies to the transfer of residues from wood to skin. The Panel is asked to address whether this is a reasonable assumption, and if not, to provide guidance on other approaches.**

Recommendation

The Panel does not recommend assuming that a one-to-one relationship applies to the transfer of CCA chemical residues from wood to skin as proposed by the Agency. It is the Panel' s opinion that the underlying conceptual model is questionable. Sufficient justification for a one-to-one relationship was not provided and the limited available empirical data contradict the validity of the assumed one-to-one relationship.

The Panel strongly recommends that the Agency expand its planned joint study with CPSC to measure dislodgeable CCA chemicals from an appropriate sample of play structures, so as to obtain information of more direct value for exposure assessment. Ideally CCA chemical loadings on the hands (and possibly other skin surfaces) of children using play structures would be measured in addition to corresponding dislodgeable residues. At a minimum, some Panelists would accept gathering of data sufficient to more adequately support implementation of OPP' s current conceptual model (e.g., matched adult volunteer hand and cloth wipe samples to better establish the relationship between these two measures as well as the constancy of any relationship as a function of surface area sampled).

The Panel was divided on an interim recommendation for the Agency while it awaits collection of these additional data for the EPA/CPSC study. Some Panel members were willing to endorse interim use of existing hand or fabric wipe data if described probabilistically. One Panel member voiced strong opposition to any use of cloth wipe data until the Agency obtained additional information establishing the validity of the assumption of a constant loading as a function of wiped surface area. At least one Panel member opposed use of a "transfer

efficiency" approach, preferring a "transfer factor" approach which cannot be implemented without further data collection.

Discussion

The Panel was not given any explanation or justification of how OPP' s Residential SOP – with its assumption of a one-to-one transfer of pesticide residues from surface to skin – was derived and whether there is general agreement on its principle and use. [Note: The SAP meeting at which the proposed residential SOPs were discussed was held in September of 1999. The reference to April, 2000 is unclear. No source bearing that date was provided as background material or is cited in the background document.]

The Panel noted a number of factors that make an assessment of the appropriateness of a one-to-one transfer relationship difficult. Variables that might influence surface-to-skin transfer include the nature of the initial CCA treatment, type of wood (softwood, hardwood, etc.), condition of wood (age, moisture content, etc.), orientation of wood member (vertical or horizontal), nature of the surface residue (particle-bound, dissolved, crystalline, etc.), condition of skin (moist/dry, intact/broken, clean/dirty), and nature of contact (pressure, duration, static/dynamic, etc.). The Panel discussed the extent to which both published peer reviewed literature and new information presented at the meeting provided empirical data for evaluating the assumed one-to-one transfer of CCA chemicals from wood surfaces. Dr. Stillwell reported transfers of 30 to > 90% when CCA chemicals were applied to a glass surface using his cloth swipe technology. The higher levels of transfer were observed when using damp cloth. Similarly high transfer factors were reported for a study (Rodes et al., 2001) with hand presses to remove household dust. This study showed that the magnitude of transfer was sensitive to surface material (stainless steel > vinyl > carpet) and hand moisture content (wet > damp > dry), although the applicability of this study to dislodgeable CCA wood chemicals was unclear. The Panel was presented with an unpublished study by Scientific Certification Systems (SCS, 2001) that compared loadings of hand swipes versus "KimWipe" tissue swipes of CCA wood. This study reported that transfer efficiencies for damp adult hands were lower than those observed using dry "KimWipe" tissue swipes of CCA wood. Damp hand swipes were reported to be 7.5% of results obtained using "KimWipe" tissue swipes of new CCA wood surfaces that had not been treated with a sealant. The damp hand swipes were 44% of "KimWipe" tissue swipes of aged CCA wood. The Panel noted that these comparisons reflect different surface areas swiped by hand (500 $cm^2$) versus tissue (100 $cm^2$), and expressed concern over potential nonlinearity in loading as a function of surface area. Exponent presented an analysis of existing data indicating that hand swipes were on average about 25% of cloth/tissue wipes, but the Panel noted variability and uncertainties related to the size of the surface area sampled, the type of contact and consistency across testers, and humidity of contact surface confounded the interpretation of results.

One Panel member stated that the Agency' s proposed model for computing hand loading of CCA chemicals appeared capable of substantially underestimating and overestimating the amount of transfer, based on comparing predicted hand loadings from cloth and tissue swipe data with observed hand loading data. The Panel member strongly urged the Agency to make use of current data with both hand and cloth swipe data (e.g., Lu and Fenske, 1999; California DHS, 1987; SCS, 1998) to validate their conceptual model. It was emphasized that the assumption of

a constant transfer efficiency as a function of surface area wipe had not been established and indeed there were data to argue to the contrary.

It was noted by the Panel that all the transfer studies discussed use the hand only, and the transfer may be different with different body surfaces that may contact the wood. The issue was also raised that there are no studies showing that transfer efficiency is constant across different surface area sizes or types. It should also be noted that Rodes (2000) found that skin loading of dust particles reaches a maximum after typically 4-5 contacts. After that, there may be dislodging of particles from skin.

Addition of collection of hand wipe samples from children engaged in unstaged activity on play structures has been recommended for the proposed CPSC-EPA field project. Wipe samples should include body parts other than hands, or non-hand surfaces should be removed from the dermal/dislodgeable residue scenario, as loading on body parts other than hands will probably be much lower than loading on hands. If the Agency intends to do these transfer evaluations, it needs to adopt an appropriate standardized sampling protocol for surface collection since that will affect the outcome. Specifically, the Agency needs to include validation of the assumption of constant transfer efficiency as a function of the sampled surface area.

In conclusion, the Panel agrees that a one-to-one relationship for transfer of residues from wood to skin is not justified at this time. The Panel also agrees on the need to collect empirical data that realistically reflect the activities of children on CCA-treated wood play structures and other possible points of contact such as decks and walkways. If a probabilistic risk assessment is to be conducted before new, relevant empirical data are generated, a wide range of possible transfer efficiencies (TEs) should be used in a manner that reflect the uncertainty and variability in the available data.

**Question 10: The Panel is asked to comment on whether the proposed Soil Adherence Factor (AF) of 1.45 mg/cm$^2$ for hand contact with commercial potting soil is a realistic value for use in estimating the transfer of residues from playground soil to skin in this assessment.**

<u>Recommendation</u>
Use of an AF of 1.45 mg/cm$^2$ is not recommended. The proposed AF was derived from an unpublished study of very limited scope. EPA has funded subsequent research to derive more representative values.

Discussion

The proposed AF represents a fairly high hand level and is too high for whole-exposed-body-surface average. Soil loadings on non-hand body parts are typically lower than loadings on hands. The soon-to-be-released RAGS Part E provides an estimate of a surface-area-weighted average soil adherence factor for children. However that number reflects multiple activities on multiple surface types. For purposes of evaluating use of CCA-treated wood in play structures, consideration of media found under play structures is required. Adherence factors relevant to loose media appear most appropriate. A possible alternative to the value recommended in RAGS Part E would be the children-playing data from Kissel et al., 1998. Those data were collected from 8-12 year olds playing in a bed of sandy loam installed in a greenhouse. (The data can be downloaded from http://depts.washington.edu/jkspage/greenpost.html .) Most of the data were collected under wet soil conditions. The wet soil data should be conservative for soil and may be adequate for buffer materials. No data describing adherence of buffering materials (bark, pea gravel, ground tires) to skin are known to exist.

The proposed dermal/soil scenario utilizes an absorption factor derived from 24-hour experiments, implying that the exposure period is also 24 hours. Soil exposures occur intermittently and are interrupted by bathing events. (For instance it is unlikely that soil loadings equivalent to those observed in the greenhouse experiments noted above would be maintained for 24 hours.) A probabilistic approach incorporating temporal description of both exposure and absorption is preferable to the deterministic approach proposed by OPP. To the extent possible, variation with age, season, and geographical region should also be incorporated.

**Question 11: OPP will need to calculate intermediate-term, and possibly long-term exposures in this assessment using available wood/soil residue data. The Panel is asked to recommend a credible approach for selecting residue data values for use in OPP's risk assessment, taking into consideration the inherent variability of the data sets. Please advise us on which values are best for representing central tendency and high end exposures. Also, the Panel is asked to discuss the feasibility of combining data from individual data sets.**

Recommendation

The proposed USEPA/CPSC study of wood and soil residues associated with CCA-treated playground equipment provides a unique opportunity to generate a substantial data set on the variability of residue levels for the playground scenario using a standardized sampling and analytical methodology. This study should help to resolve uncertainty regarding the relative contribution of true, inherent variability in residues versus variability due to differences in methodology. It is critical, therefore, that the protocol be highly detailed regarding sampling methods, locations, and frequencies and that the protocol be rigidly followed. Basic scientific criteria for acceptance of the final data set should be laid out first and include: standardized collection methods, precision, accuracy, reproducibility, and other measures of QA/QC.

The Agency should not combine data with quite differing levels of precision and conservativeness, and use one set of data to drive other model considerations. The model cannot be fully evaluated without real world (i.e., biomonitoring or soil consumption) data for comparison, and that comparison cannot be made without a representation of both variability and uncertainty in model outputs.

Discussion

There are few studies related to children' s playgrounds, and no study contains all of the data that the Panel considers critical to getting an accurate determination of what children are being exposed to on playgrounds and on CCA-treated decks. The Panel believes that separate studies should be conducted (i.e., those looking at residue levels on playgrounds, and those examining home decks and home playgrounds). The residue data should not be combined from decks and playgrounds. Data from piers, walkways in wetlands, and similar structures do not fit the playground scenario and should be ignored. The Panel recommends that the Agency expand the upcoming study to 25 playgrounds and 25 home decks/home playground combinations in each of the three U.S. study areas (e.g., Northeast) in order to determine what children are being exposed to. An extensive sampling regimen must be undertaken.

The critical data required for risk analyses should include the following information and samples:

- Soils selected should mirror those most commonly found in each region;
- History of the playground equipment (e.g., wood type, age, coatings/sealants etc.) must be collected;
- Representative soils should be collected in order to determine speciation and profiles of As, including organic arsenic species and chromium in the soil profile at each site; soils must be collected from throughout the area below and adjacent to the play equipment/deck and analyzed separately to determine the primary sites of residue levels that are unique to each playground/deck studied; adequate control soils must be collected from adjacent areas;
- Soils, including controls should be characterized thoroughly (e.g., clay, sand and silt content, pH, organic matter, moisture, etc.);
- Wood borings from sections of the playground equipment known to have frequent contact by children at play (this can be accomplished by video) for residue analyses should be collected to determine residue levels in wood and relate these to residue levels that have leached to the surface (the treatment process is not uniform due to knots, growth rings, etc., and there probably are "hot spots" of As and Cr) (this is related to Question 9);
- Wipes have been used as a means of determining dislodgeability, but there is no standard technique that provides reliability and uniformity to data collected from various surfaces;

- Consider collecting hand, arm and leg rinses from a representative sampling of children playing on the equipment and tie these to biomonitoring analyses;
- Analyses of buffering materials from play areas including borders should be included in the study (related to Question 14).

Each of the available data sets should be critically evaluated to determine whether they have been obtained 1) from a relevant structure and 2) using acceptable sampling and analysis methodologies.

The following studies present some representative soil data and can be used until additional data are collected:

- Playground equipment: Riedel, D. et al. 1991. Residues of arsenic, chromium and copper on and near playground structures built of wood pressure-treated with "CCA" type preservatives. Draft report to Health and Welfare Canada, 49 pp. (10 playgrounds examined); Malcom Pirnie. 2001. Report results of soil sampling analysis. Chromated copper arsenate treated wood at playground structures. Draft appendices. Prepared for Am. Chem. Council. (4 playgrounds in U.S.)
- Decks: Stillwell, D. E. and K. D. Gorny. 1997. Contamination of soil with copper, chromium and arsenic under decks built from pressure treated wood. Bull. Environ. Contam. Toxicol. 58:22-29 (7 decks); Scientific Certification Systems. 2000. Study of arsenic leaching into soils underneath CCA treated wood decks. Prepared for Osmose, Inc., 47 pp. (10 decks 5-5 to 10 yr and 5-10 to 15 yr old); Townsend, T., H. Solo-Gabriele. 2001. Metal concentrations in soils below decks made of CCA-treated wood. FL Center for Solid and Hazardous Waste Management, Gainesville, FL, 88 pp. (9 structures in FL).

The Agency asked for advice on values for best representing central tendency and high-end exposures. The best measure of the central tendency depends on the shape of the distribution. One Panel Member noted that it has been suggested (Crump, 1998) that the arithmetic mean, as opposed to the geometric mean, is the preferred measure of central tendency for exposure when the concern is for health effects. The best approach for estimating central tendency and high-end exposures and for dealing substantively with the process would be a two-stage probabilistic analysis that evaluates both variability and uncertainty. The use of distributional analyses for CCA exposures should rely on firmly established and transparent criteria that are common to all probabilistic analyses. Many of the same principles that have been incorporated into the assessments of food and residential exposure guidance, for example, should be incorporated into the CCA assessments. In order to do this it is important to develop clear and consistent criteria for both the modeling process and methods for dealing with model uncertainty, model variability, and input uncertainty. All three of these must be addressed systematically throughout the process.

This three-point framework for describing model variability and uncertainty is based on the points outlined in Cullen and Frey (1999), which is a useful guidebook and starting point for addressing the issues raised by this question. Once these principles are clearly articulated and inculcated, decisions based on application of this framework should be easier to justify.

Given the multiple models, data sets, and analyses involved in developing an assessment of CCA, probabilistic methods are the preferred approach for estimating exposures and risks. That said, the use of uniform distributions, which are generally used in cases where data are sparse or inconsistent, are better than point estimates. Fitted distributions should be used when there is

some underlying rationale, such as processes driven by physical parameters where some data exist or in cases where there are fairly good data, such as soil consumption rates. There are several options for combining these data. When appropriate, multiple data sets could simply be combined into a single, "global" data set that could be used as input for a probabilistic exposure assessment. However, this simple approach ignores the "inadvertent" weighting associated with combining data from experiments with different numbers of samples. Appropriate weighting factors could be applied to each data set to correct for this effect.

Alternatively, weightings could be applied on the basis of a judgment concerning the "representative nature" of a particular data set. The specifics of the approach for combining these data should be determined by a qualified statistician in conjunction with scientists familiar with the data. A similar analysis has previously been performed to obtain a "global" distribution for the hair:blood partition coefficient for methylmercury (Clewell et al. 1999).

It is inevitable that dissimilar data will be combined once exposures are aggregated, but uncertainty and variability should be distinguished. The Agency should not combine data with quite differing levels of precision and conservativeness, and use one set of data to drive other model considerations. The model cannot be fully evaluated without real world (i.e., biomonitoring or soil consumption) data for comparison, and that comparison cannot be made without a representation of both variability and uncertainty in model outputs.

In performing a probabilistic analysis the Panel suggests using intervals (i.e., uniform distributions) rather than point estimates when data are sparse/uncertain. This approach reduces the burden of data collection and parameterization and, although it is simpler than second-order Monte Carlo simulations that formally separate variability from uncertainty, it still distinguishes the two. Using intervals in a Monte Carlo simulation avoids creating a mix of partially probabilistic and partially deterministic estimates.

**Question 12: Does the Panel have any recommendations for combining the four scenarios (oral/wood, dermal/wood, oral/soil, dermal/soil) such that a realistic aggregate of these exposure routes may be estimated?**

The Panel offers the following recommendations:

- The Panel encourages the Agency to aggregate exposure estimates across all potential sources. This should occur in a way that makes the contribution of various sources of exposure transparent and tracks separate species of arsenic and chromium. Although data at present are limited, it is possible that the different species of arsenic encountered from distinct exposure scenarios may differ with respect to their hazard. For example, arsenic in the form of a complex of copper chromated arsenate ingested from direct contact with freshly treated wood might be metabolized and excreted differently than arsenate leached from weathered wood and ingested incidentally in soil.

- The suggested scenarios *(oral/wood, dermal/wood, oral/soil, dermal/soil)* capture the exposures that may occur on playscapes and decks. Inhalation exposure may be a route

that should be included, however at this point in time, data are insufficient to estimate the distribution of possible inhalation exposures. Refer to the response to question 13 for further analysis.

- However, in terms of the aggregate exposure assessment, the proposed scenarios do not capture sources of exposure that appear to be significant. The Panel suggests that the Agency broaden its inquiry to consider the diversity of possible exposures to arsenic, chromium and copper. Some Panel members felt that the Agency should expand its formal analyses of exposure to include other media under the jurisdiction of other EPA offices—drinking water, air, and waste—to avoid a fragmented and incremental approach to risk assessment and management of arsenic, chromium and copper species.

- Probabilistic methods should be used to estimate exposure and risk. This demands selection of best available data sets to construct distributions. This must be done with considerable care. The EWG approach seems conceptually reasonable; however, their method combines point estimates with distributions, and this may introduce bias into the estimates. The Lifeline method is especially well adapted to aggregate exposures across diverse routes, while preserving estimates at the level of the individual. The Agency should be encouraged to develop this model in the immediate future while closing data gaps.

- Uncertainty should be carefully characterized, distributions characterized, and clear criteria applied to judge the quality of available data for each parameter included in the assessment. The Agency should further develop Table 4 in the EPA support document *Children's Exposure to CCA Treated Wood Playground Equipment and CCA Contaminated Soil*. Table 8 in the Gradient Corporation submission provides a similar model that attempts to identify ranges of factors potentially affecting exposure, tracks the sources of data, and provides a preliminary characterization of uncertainty.

- Regarding uncertainty and default assumptions the Agency should confront two questions directly: When are data of sufficient quality to include in a modeling effort? What should be done until data are adequate? The SAP provided the Agency with clear criteria to judge data quality in 1999, and these were recognized in support documents provided to this panel. Under conditions of moderate or high uncertainty (absence of sufficient data to fully capture the variability in exposure from these sources), the Agency should develop clear default assumptions to be employed until sufficient data are secured. These assumptions should err on the side of overestimation of exposure, or factors that contribute to exposure, and reduced if and when credible data are presented.

- The Agency should develop methods that aggregate exposure and risk estimates for individuals. These may then be aggregated by various demographic characteristics—age, income, ethnicity, and location (north/south; urban/rural), or specific behavioral characteristics.

- The literature on childhood behavior and activity patterns that may be associated with CCA exposures is quite young. It provides only a limited basis for understanding the associations between behavior and exposure. The Panel recommends that the Agency undertake studies of childhood behavior and activity patterns to clarify these possible associations, as children move through their daily life. These studies would be useful in EPA assessment of exposures to many different hazardous substances. The Agency' s efforts in developing the Exposure Factors Handbook and the more recent Children' s Exposure Factors Handbook are very positive and important contributions that support data based, behavioral scenario-building.

- The Panel anticipates considerable year-to-year variability in exposure among children ages 1-6. Toddlers between 1 and 2 years of age play, behave, eat and dress very differently than 6 year olds, and these are likely to affect contact with contaminated media.

- Residential, educational, day-care, recreational, and occupational environments all offer the possibility of childhood exposures to CCA. Specific locations where CCA is in common use include decks, playscapes, railings, docks, piers, pilings, fencing, and exposed untreated interiors of structures, especially those close to the ground such as sills. Picnic tables, mulch, and contact with wood scraps, smoke from intentional and unintentional fires, and ashes from burned construction debris could all be sources of exposure.

- CCA-treated wood is increasingly being used for interior construction, and if unfinished and left exposed, may be an additional source of childhood exposure. It would be helpful for the Agency to estimate the extent of these uses.

- The Panel encourages the Agency to consider possible high exposure scenarios defined by overlapping risk factors. For example, a toddler living in the Southwest, may experience high drinking water concentrations of arsenic. At the same time the warm climate encourages extended periods of outdoor recreation. If this child is enrolled in a day care facility with decks and play structures made from CCA-treated wood, the aggregate exposure may be high. The identification of populations that are both physiologically susceptible and highly exposed would provide a logical basis for strategic risk management.

**Question 13: Can the Panel comment on whether OPP should conduct a child playground inhalation exposure assessment, taking into consideration the hazard profile for chromium (VI) as an irritant to mucous membranes? If so, can the Panel comment on whether the endpoint described above is appropriate for assessing the risk to children from such an exposure?**

Recommendation

The Panel notes that both the trivalent and hexavalent forms of chromium are of concern in the inhalation route of exposure and that arsenic should also be considered in the inhalation route of exposure.

However, the Panel agrees that calculations of probable exposure concentrations suggest that the Agency should not consider the inhalation route of exposure to inhaled metals in their risk assessment. The SAP strongly suggests, however, that exposure concentrations be monitored via personal and area sampling to validate such a conclusion.

Discussion

The contribution of inhaled metals to the risk for children using playground equipment constructed of CCA wood is dependent on the airborne level of metals. Unfortunately, there are no data on the ambient concentrations of metals in the vicinity of CCA-wood play structures. There is a need for determination of the range of background ambient exposure levels to chromium and arsenic and to compare these values to potential exposure levels for a 15-kg child during a 1-3 hours of play.

Soil in the immediate vicinity of play structures is frequently disturbed during child play and inhalable particles can be resuspended and re-entrained in air. Although the question posed to the SAP referred to the volatility of the inhaled metals, the primary concern is a resuspended dirt scenario, and not the volatility of chromium and arsenic, which should be considered. The questions posed to the Panel also referred to respirable particles. Most mechanically generated particles are very large. Thus, inhalable (particles which can be inhaled into the nasal or oral passages; generally less than 100 μm in aerodynamic size) and not respirable (particles which reach the gas exchange region of the lung; generally less than 3 or 4 μm) particles are of concern in terms of the nasal effects of chromium. These particles deposit in the nasal cavity, are cleared towards the back of the throat, and swallowed, thus ultimately resulting in an oral delivery.

It is likely that the assumption of 100% hexavalent chromium is an overestimate of its proportion in the soil and dislodgeable residue. In addition, there are very sparse published data on hexavalent vs. trivalent chromium in CCA-treated wood and none (except for what was presented by Drs. Stillwell and Townsend) for soil. Such a data set regarding the valence state of chromium in soil needs to be developed.

Because no data have been developed for airborne metals in the vicinity of playgrounds built with CCA-treated wood, one must use surrogate values to calculate a potential risk. The Panel members introduced three arguments against the need for an inhalation examination of the potential effects of playground exposure to chromium and arsenic. First, workers are exposed to

much higher concentrations of CCA-treated wood dust in occupational settings (Decker et al., 2001) and the occupational exposure limit for chromium is not exceeded. It must be considered, of course, that an occupational exposure limit (OEL) is set for a healthy adult worker over an 8-hour period.

Second, using the NOAEL given in the EPA document for adverse nasal effects in workers, one can calculate relative inhalable concentrations of chromium for a child. Using a rough assumption of the volume of air inhaled by a child, the level of exposure to inhalable chromium is insignificant:

- Using the following rough assumptions, one can calculate that a 15 kg child inhales approximately 5.4 $m^3$ in a 24 hour period:

  0.25 l/breath x 15 breaths/min x 60 min/hr x 24 hour/day = 5.4 $m^{3.}$

- Assumption of a NOAEL of $2.4 \times 10^{-4}$ $mg/m^3$ (noted in the EPA document) yields:

  0.086 μg/kg-day for 24 hr (0.000086 mg/kg-day for 24 hr exposure) for a 15 kg child
  or
  0.0036 μg/kg-day for 1hr (i.e., 0.000036 mg/kg-day for 1 hour exposure).

Therefore, in comparison to the LOAEL of 0.05 mg/kg-day (or 50 μg/kg-day) considered in Question 1, this exposure level is below that of concern.

Of course, an assumption of $2.4 \times 10^{-4}$ $mg/m^3$ as a NOAEL for hexavalent chromium could be high, but as noted previously, it is unknown what the exposure levels are for airborne particles in playground areas.

Third, one can calculate a worst-case scenario for inhaled resuspended soil and compare it to the central tendency value for the oral ingestion of soil. If one uses a central tendency value of 100 mg soil ingested/day for a child (as suggested by EPA), this can be compared with the potential concentration of airborne soil particles which would need to be inhaled to equal this amount of soil delivered via the oral route. Using a rough assumption of 5 $m^3$ for the volume of air inhaled in a day suggests that a child would have to be exposed to an airborne concentration of 20 $mg/m^3$.

$$100 \text{ mg/day} / 5 \text{ m}^3\text{/day} = 20 \text{ mg/m}^3$$

This value of 20 $mg/m^3$ for inhalable particles is exceedingly high and is very unlikely in a playground setting. It was noted by Panel members that the soil, sand, or buffering material below the playground structure may influence the degree of resuspension of dislodgeable CCA or soil.

Thus, it appears to be unlikely that an inhalation pathway needs to be considered in the EPA risk assessment of the use of CCA-treated wood in playground settings. However, the Panel feels it

would be prudent to develop a data set for airborne/reentrained soil particles to validate this recommendation. It is suggested that personal or area monitoring be added to the proposed EPA-CPSC playground study. In addition, this data set should include airborne arsenic as there is no justification to exclude it in an inhalable CCA-treated wood risk assessment.

**Question 14: Data on the effectiveness of reducing exposure by using buffering materials are limited. Does the Panel have recommendations as to whether additional studies to obtain this information are warranted? Does the Panel have suggestions on how OPP can best evaluate child exposures attributed to contact with CCA-contaminated buffering materials?**

Recommendation

The consensus of the Panel is that additional studies are warranted to obtain information needed to assess the exposures associated with using buffering materials.

Buffering materials do not appear to provide a means to reduce exposures to CCA leached from play structures. Rather, buffering materials can present important risk scenarios that differ from the four scenarios currently proposed for analysis by the Agency. These additional scenarios include: 1) exposure to buffer materials that become contaminated with metals from CCA leached from play structures and 2) exposure to buffer materials that contain CCA because they consist of recycled construction/demolition debris which contains CCA-treated wood.

Exposure to buffer materials that become contaminated with metals from CCA needs to be examined. This effort should be aided by the generation of data describing the amount and nature of exposure in young children who play on or with these materials. It may also be possible to generate bounding estimates of exposure from these materials for the purpose of screening the relative importance of this scenario compared to other play structure-related scenarios.

Exposure to buffer materials that contain CCA-bearing mulch will likely result in a sufficient hazard potential to warrant a modification of the recycling practices that lead to the introduction of this mulch into children's play environments.

Discussion

Buffering materials refer to those materials that are placed below a play set to minimize injury in the event of a fall. Examples of buffering materials include sand, pea gravel, wood mulch, and tire chips.

There are two sets of issues imbedded in the concern over buffering materials. The first is the potential that the materials can become contaminated due to their close proximity and contact with CCA-treated wood play structures as CCA leached from the play structures coats the buffers. The second issue is the potential presence of CCA-containing wood in construction debris that is recycled into wood mulch used as buffering material. Both of these issues are of

particular concern because children may be attracted to the buffering materials due to their unusual textures, colors, and ready availability. Therefore, where there is CCA contamination of buffering materials, exposure potential to young children may be particularly high.

Regarding buffer materials that become contaminated by CCA leached from the play structure, the following assumptions can be used to help conceptualize the issue:

- Leached CCA forms a surface coating that is dislodgeable and thus available to children who handle these materials. This implies that the proper way to analyze these materials for the purposes of risk assessment is to find out how much dislodgeable residue is present on the buffer material surfaces in ug/cm2 surface area as has been done with play structure surfaces, rather than analyzing the entire material to get a ppm readout of arsenic or chromium on a mass unit basis. Expressing the contamination as metal concentration per surface area has the advantage of direct applicability to risk assessment equations since children can be modeled to take up some or all of the dislodgeable residue through mouthing behavior (putting entire chip or stone in mouth for brief period) or via chip-to-hand transfer followed by hand-to-mouth behavior. However, it is unlikely that children will actually ingest an entire chip and so the ppm type measurement would be less relevant. The Panel noted that the Alachua County, Florida data presented to the Panel, while limited in number of samples, suggested similar ppm contamination of buffer material (shredded rubber in this case) as neighboring soil. However, since buffers will not be ingested on a mg/day basis, the exposure implications of ppm in soil and ppm in buffer are not the same.

The recommendation that comes from these considerations is that buffering materials that are passive recipients of CCA leached from play structures should be chemically analyzed by finding out how much dislodgeable metal is available per square centimeter surface area. A suggestion for how to conduct this analysis is to put a representative number of chips/stones into dilute acid solution to extract the metals, analyze the metal content in the extract (e.g., ICP) and then calculate the dislodgeable residue by dividing the total metal extracted by the surface area of the chips/stones placed into the dilute acid solution. This would result in an estimate of the maximum metal loading concentration that children may dislodge onto hands or extract in their mouth.

The Panel is not aware of any survey or videotaping data of children' s behaviors with respect to buffer materials. Therefore, exposure assessment in this area will be uncertain and data collection is important. The most direct and empirical form of data collection may be the sampling of children' s hands and other exposed dermal surfaces at the end of a play event to find out how much of the dislodgeable material (measured by analyzing the buffer materials as described above, from various portions of the playground) is transferred onto children' s hands. This may allow for an estimation of buffer-to-hand (or skin) transfer efficiency. Given that this transfer will be highly age and behavior dependent (types of interactions with the buffer materials and location on playground where child interacts with buffer), there will be much variability in the data. Therefore, a sizable dataset would be needed to obtain a representative distribution of dermal loading per play event. The value of such distributions obtained in this

way is that they could be suitable inputs to probabilistic (Monte Carlo style) exposure calculations.

Another option for data collection pertinent to this exposure scenario is obtaining observational (e.g., videotape) data on children' s play activities to compile information on the frequency of contact with the buffer materials and classification of contact into categories such as superficial (brief touches) vs. intimate (handling/playing with the chips) vs. mouthing of chips. This information could then be combined with study data on how much CCA is dislodged from the chips from superficial vs. intimate vs. mouthing of chips in studies involving children or adult volunteers performing these types of activities. Of course, the mouthing of chips would not be performed but would have to be simulated in some way or assumptions used regarding percent extraction of residues from chip/stone residence for brief periods in the mouth.

Actual empirical data are most desirable for input into this or any other human health risk assessment. However, to evaluate the need to collect field data and perform a refined analysis for this particular scenario, the Agency may want to consider performing bounding, screening level calculations. Given that without the types of empirical data described above there will be greater uncertainty in exposure estimates, the defaults used should necessarily be high-end bounding assumptions. Such assumptions may look like the following:

- The dislodgeable residue loading on wood/rubber/gravel buffers is equal to the dislodgeable residue on the wood play structure. This may tend to be a reasonable upper bound on the relationship between wood structure to recipient buffer material loading since the buffer material will not only receive leached CCA but will also have such residues washed off in rain events, and so some equilibrium (probably no higher than what is on the donor wood) would be established on the buffer materials.

- The chip-to-hand transfer efficiency is 1:1, that is, whatever surface loading that is assumed to be on the chips will also exist on the hands. This assumption implies intimate contact with the chips such that the hands would be in equilibrium with the chips. Lower levels of loading are also possible from less intimate contact but would not represent an upper bound default. Higher levels of loading in which the hand actually accumulates dislodgeable residue might also theoretically occur, but empirical data would be needed to find out the circumstances under which this might be possible.

- Mouthing of chips/gravel would extract all the dislodgeable residue from that buffer material; the amount of residue available is based upon the surface area of the chip (empirical measurements for different buffering materials should be used) and the loading on the chip (as discussed above).

- The Panel did not have an opportunity to explore what might be reasonable bounding assumptions on frequency of mouthing behavior, time spent playing in chips relative to time spent on play structure itself, hand-to-mouth frequency, etc. (This might be based upon the same videotaping studies used for the hand-to-mouth assumptions for children on play structures.)

In summary, the Panel recommends that empirical data be gathered with respect to children' s exposures to dislodgeable residues of CCA from buffer materials via actual children' s play studies or from observational data on children' s play behavior combined with data describing the range of chip-to-hand transfer for different degrees of contact. An additional study design mentioned during Panel deliberations was to have a single playscape underlain with differing buffering materials including no buffer (native soil) around different portions of the play structure. After a period of environmental loading and equilibration of buffer material, then children could be instructed to play on one of the buffer types to allow a comparison of exposure potential across the range of different materials.

Whatever study data are generated, they should be of sufficient diversity and robustness to characterize the distribution of behaviors and residue transfer efficiencies. However, if appropriate empirical datasets are not available for probabilistic assessments, EPA may consider the option of screening level deterministic assessments using reasonable upper bound defaults based upon available empirical data.

The possibility that these buffers might lead to a decrease in exposure to CCA relative to native soils does not look promising, based upon the evidence that leached CCA can lead to elevated arsenic concentrations near CCA-treated wood structures (Alachua County data). However, additional studies along these lines or as described above may better answer this question.

Unfortunately, when construction debris is recycled into mulch, a percentage of the wood may contain CCA. Data are available (Tolaymat et al., 2000, Townsend et al. 2001, and Solo-Gabriele 1998, 1999) that indicate that CCA-treated wood is found in mulches produced from construction and demolition facilities within Florida. During 1996, the mulch from 12 different construction and demolition facilities was found to contain 6% CCA-treated wood by weight, on average. A study at 3 facilities during 1999 found that concentrations of CCA-treated wood in mulch varied between 9 and 30%. Mulch samples collected from retail establishments showed evidence of CCA as observed by arsenic leaching tests.

If CCA-wood bearing mulches are used as buffering material under play structures, then there could a higher exposure potential from children' s hand contact and mouthing of CCA wood mulch. As the wood degrades into smaller pieces and dust, there might be a higher opportunity for bulk ingestion of material containing high concentrations of metals both from direct mouthing of small objects and from hand to mouth transfer. Additionally, the dust generated from playing with the mulch might become an inhalation risk issue, particularly with respect to the Cr(VI) that may be present in the dust particles.

Rather than conduct a risk assessment on this portion of the scenario, the Panel recommends survey, intervention, and education activities on the part of the Agency, perhaps coordinated with other agencies, to prevent this exposure pathway to the extent possible. The survey would be of the wood mulch marketplace to determine to what degree CCA-treated wood enters the playscape environment, including practices at the municipal and homeowner/residential levels. This survey information should be followed with focused intervention and education/warnings to

prevent or modify the practices that lead to this type of wood entering children' s play environments.

**Question 15: The Panel is asked to comment as to whether stains, sealants and other coating materials should be recommended as a mitigation measure to reduce exposure to arsenic and chromium compounds from CCA treated wood. If so, can the Panel comment on the most appropriate way for the Agency to recommend effective coating materials when the current data on long-term performance are limited and sometimes inconsistent, and should the Agency specify a time interval for the re-application of these selected coating materials? Can the Panel make recommendations for additional studies?**

Recommendation

The Panel offers the following conclusions and recommendations:

- The Panel recommends that the EPA inform the public of the ability of certain coatings to substantially reduce leachable and dislodgeable CCA chemicals and thus reduce potential exposure to arsenic and chromium. While the Panel makes recommendations below regarding the need for additional studies in this area, it feels that the current evidence is sufficient to begin advising the public about the use of coatings now.
- The weight-of-evidence from available studies indicates that certain coatings can substantially reduce dislodgeable and leachable CCA chemicals.
- Reductions of 70 to 95% in dislodgeable arsenic were seen in all studies that subjected CCA wood to natural weathering.
- There is no evidence that water repellents added directly to the CCA treatment solution are effective in reducing leachable/dislodgeable CCA chemicals;
- Current data are not adequate for identifying a particular coating as being clearly superior or inferior to reducing leachable/dislodgeable CCA chemicals.
- Confidence is highest for polyurethane as this coating has been shown to result in substantial 70 to >95% reduction in dislodgeable arsenic in a well controlled field study, a "real-world" application allowing for effects of use, and a short-term controlled laboratory study.
- Current data support a treatment frequency of once per year, although for some products this may be too frequent (e.g., possibly polyurethane where one study noted up to 95% reduction in dislodgeable arsenic out to 2 years). This is an area in need of additional study.
- More studies are needed to evaluate the performance / efficacy of different types and brands of coatings.

Discussion

Important definitions when evaluating coating data include the differences between treated and untreated wood. Treated wood typically implies that the wood is CCA treated. Untreated wood refers to virgin wood without the addition of CCA wood treatment chemicals. Both treated and untreated wood can be either coated or uncoated. Coatings or sealants refer to paints, stains, varnishes, and polyurethane resins applied to wood surfaces. For years the manufacturers of CCA wood have recommended the use of surface coatings to reduce the checking and cracking of wood resulting from effects of weather, such as rain, temperature, humidity, solar radiation (http://www.preservedwood.com/faqs/faqs.html ). Intuitively, reductions in leachable and dislodgeable CCA chemicals should be expected to the extent that coatings establish a barrier to moisture contacting and entering wood and as a barrier to direct hand contact with the wood surface. Likewise, the surface area available for leaching, including access to deeper wood layers which are less depleted of CCA chemicals, should be reduced given that such coatings reduce checking and cracking of CCA wood.

Evaluation of Coating Data

Sealant studies evaluated were separated into three groups: Studies that evaluated the impacts of coatings on dislodgeable arsenic, impacts on leaching of arsenic, and related studies.

Studies available to the Panel that evaluated the impact of coatings on dislodgeable arsenic included: Stilwell 1998, Scientific Certification Systems 1998, California Department of Health Services 1987, and the Consumer Products Safety Commission 1990. Stilwell, 1998, evaluated boards with four different coatings (polyurethane, latex/acrylic, oil stain, and spar varnish). His results indicate that these coatings remained very effective in reducing dislodgeable CCA chemicals for at least one year after they are applied. This study did not evaluate the performance of sealants beyond one year. The boards were subjected to natural weathering processes but the study did not include the effects of wear from human use. Also, the experimental design would have benefited with the inclusion of a temporal control.

The SCS 1998 study evaluated the impacts of two post-treatment coatings (Superset stain, 3M clear sealer/polyurethane -) and one coating incorporated into the CCA-treatment process (Osmose water repellent). The other two coatings were applied after the wood was CCA-treated. The SCS study was a laboratory-based study using a series of boards. It is assumed that the measurements of the dislodgeable arsenic were taken shortly after the coatings were applied. Wear and tear from human use was not simulated, nor was rainfall or other weather related effects taken into consideration. Results from this study were variable indicating that the 3M polyurethane sealant was effective at reducing dislodgeable arsenic whereas the Superdec stain and the Osmoses water repellent were not effective. Results from this work suggest that there is variability in the reduction of dislodgeable arsenic by different types and brands of coatings.

The California Department of Health Services 1987 study was the only study that evaluated structures (a fishing pier treated with polyurethane and a playset treated with an oil-based stain) that were in current use and therefore included the effects of wear and tear upon the efficacy of the coatings. Results of this study suggest that coatings provide a considerable reduction in the amount of dislodgeable arsenic and, in the case of polyurethane, out to two years post-treatment.

One drawback of the study is the lack of an uncoated temporal control. However, the decrease in dislodgeable arsenic was very large that even in the absence of such a control, the data are considered to be meaningful.

The last study, sponsored by the Consumer Products Safety Commission in 1990, evaluated an oil-based stain and a water repellent in a laboratory setting. The results of this study were variable which is reflected in the high standard deviation observed in the uncoated CCA-treated control. This variability confounds the ability to interpret the data and therefore the results are considered inconclusive.

Only one study, Cooper et al. 1997, evaluated the ability of coatings to reduce the leachability of arsenic from CCA-treated wood. This study evaluated the ability of Thompson' s water seal (applied after CCA treatment) to reduce leaching of CCA from fences. The efficacy of water repellents applied as part of the CCA treatment chemical were evaluated for fences and decks. Results show that Thompson' s water seal (applied after CCA treatment) significantly reduced the quantity of arsenic for a period of two years after the application of the water seal. The water repellents applied as part of the CCA treatment chemical were not effective at reducing leachable arsenic concentrations.

Related studies cited as part of the EPA review include Riedel et al. 1991 and Lebow and Evans, 1999. Riedel et al., 1991, focused on collecting dislodgeable arsenic data from 10 CCA-treated playsets. Some playsets were coated with sealants and some were uncoated. This study is useful in providing a range of dislodgeable arsenic variables from playsets. However, there are many confounding factors when comparing the coated set of playsets with the uncoated sets. These confounding factors include differences in retention levels between one playset and another, wear and tear, locations sampled, and absence of any information on time elapsed from when a coating was last applied. Given these confounding factors it is difficult to conclude whether or not coatings reduce the quantities of dislodgeable arsenic.

Lebow and Evans et al., 1999, evaluated the use of an innovative pre-stain (water soluble acrylic polymer with an iron oxide) which was applied prior to treatment with CCA. Results from this study found that the pre-stain was able to reduce the release of arsenic by 25 to 30%. This was a laboratory study that simulated natural rainfall over a 17-week period.

Conclusions from studies

Table 1 summarizes design features of the various studies and results in terms of percent reduction in dislodgeable or leachable arsenic. Results of these studies as a whole support that surface coatings (applied after CCA treatment) are effective at reducing the quantities of dislodgeable and leachable arsenic. Reductions of 70 to 90% in dislodgeable arsenic were observed across the studies as CCA-treated wood was subjected to natural weathering. Conflicting results were obtained from the laboratory studies. It is noted that no studies looked at both dislodgeable and leachable arsenic fractions. The current data are not sufficient to identify a superior coating. The evidence is strongest for polyurethane, based on results from Stilwell 1998, California DHS 1987, and SCS 1998. Future experiments should evaluate the efficacy of different types of brands of coatings on both the quantities of dislodgeable and leachable arsenic. Such studies should include a validated and consistent measure of dislodgeable (e.g., Stilwell, 1998) and leachable CCA chemicals and should evaluate performance over at least a 2-year and preferably a 3-year period. Furthermore, studies should also focus on the durability of the coatings when subjected to wear and tear and include natural weathering conditions.

The Panel recommends that the Agency inform the public of the potential benefits associated with coatings in reducing leachable and dislodgeable CCA chemicals. Polyurethane should be recommended for the time being. It should be also mentioned that other coatings show some promise including acrylic/latex, oil-based stains, and some consumer applied water sealants, although data are more limited. Furthermore, recommendations should mention that some coatings will change the surface properties of the wood making it necessary for additional traction on floors and deck portions of playsets. It is recommended that the decks be sealed at least once per year. More definitive information concerning the use of coatings should be provided to the public once additional data are available.

One Panel member recommended that the coating applied be clearly visible so that the effects of wear can be easily observed. The Panel member indicated this is especially important in light of the fact that there are limited data on the durability of the coatings against wear. In areas of heavy wear, the coating should be applied more frequently than once per year if the coating is visibly removed from these high-wear areas.

Another Panel member, who supported the use of coatings, voiced the concern that the CCA chemical may accumulate below the coatings which if pealed and ingested could result in an elevated risk to children. This comment emphasizes the need to periodically inspect the coatings to minimize this potential exposure route.

It is important to keep in mind that none of the studies cited in this review have been published in peer-reviewed journals. The strength of the overall conclusions made through this review relies on the relative consistency between the results observed between some studies.

**Table 1**

| Study | Design | Weathering | Sampling | Treatments | Results | Comments |
|---|---|---|---|---|---|---|
| Stilwell, 1998 (CT) | Purchased boards, placed outside, 4 coatings, 4 replicates, 5 time points out to 1 year | Outside, natural weathering, no human use | Standardized wipe method. Repeat rubbing of same surface under controlled pressure | Polyurethane, acrylic latex, Spar varnish, Oil-based stain. Brush applied, 2 coats. | > 95% reduction for polyurethane, acrylic resin, and varnish at all time points as compared to pretreatment. 80-97% reduction for oil stain. | Does not account for wear. Lacks temporal control. Aesthetic problems after 1 yr for spar varnish. |
| California DHS, 1987 (CA) | Fishing pier, 1 coating, 4 replicates, 2 time points out to 2 year | Outside, natural weathering, in use | Gauze wipe, 100 $cm^2$ with repeat rubbing to same surface | Polyurethane, no information on application methods | > 95% reduction at 2 years as compared to pretreatment levels. | Considers wear. Lacks temporal control. Limited sample sizes and coatings. |
| California DHS, 1987 (CA) | Single playground, 1 coating, ? replicates, 3 time points, out to 2 years | Outside, natural weathering, in use | Gauze wipe, 100 $cm^2$, with repeat rubbing of same surface | Oil-based stain, no information on application methods. | > 95% reduction at 6 months as compared to pretreatment levels. 70% reduction at 2 years. | Considers wear. Lacks temporal control. Limited sample sizes and coatings. |
| SCS, 1998 (lab) | Purchased boards, used in laboratory, 3 coatings, 5 replicates, 1 time point apparently soon after coating applied. | Inside, no weathering, not subject to human use. | Kimwipes,100 $cm^2$, damp. Hand wipes, 500 $cm^2$, repeat rubbing of same surface | 3M sealant, Superdec stain, no information on application methods, Osmose water repellant | 60% - 80% reduction for 3M sealant as compared to pretreatment. No reduction for stain or water repellent. | Variable within type of coating. Does not account for wear. Not subject to natural aging and weathering. Short-term evaluation. |
| Cooper et al., 1997 (New Brunswick, CAN) | Laboratory prepared wood, fence & deck structures, placed outside, 1 coating, ? replicates, 2 time points, 4 mo, 2 yrs | Laboratory simulated aging, plus outside, natural weathering, no human use. | Collection of natural rain water contacting wood surface | Thompson's Water Seal (fence only) & Water Repellent in CCA treatment soln (fence & deck). | 70% reduction at 4 months and 80% reduction at 2 years for Thompson' s. No reduction for water repellent added into treatment solution. | Does not account for wear. Includes temporal control. |
| Riedel et al., 1991 (Ontario, CAN) | 10 playgrounds, 2 to 10 years old. Some stained / painted, others not. 4 sampling points per structure. | Outside, natural weathering, in use | Gauze wipe, 250 or 500 $cm^2$ with repeat rubbing of same surface. | Oil based stain on some though not all structures. | 4 structures treated with stain had on average 74% lower levels of dislodgeable As than average of 3 structures without any coating.[a] | Cross-sectional study with no site specific controls. Limited information on past application of coatings. Sampling locations vary across sites. |
| CPSC, 1990 (lab) | Purchased boards, used in laboratory, 2 treatments, 3 replicates, 2 wood types. | Inside, no weathering, not in use, no aging | Nylon cloth wipe, 400 $cm^2$ | Oil-based stain, water repellant, applied per manufacturer' s label. | No clear evidence of reductions. | Considerable variability in the controls, short-term study with no weathering. |
| Lebow and | Laboratory prepared wood | Laboratory | Collection of | Water soluble acrylic | 25-30% reduction in total As | Coating applied pre-CCA |

| | | | | | | |
|---|---|---|---|---|---|---|
| Evans, 1990 | 1 treatment,<br>? replicates,<br>1 time point at 17 weeks | simulated rainfall for 17 weeks. | natural rain water contacting wood surface | polymer applied pre-CCA treatment. | leached in artificial rainfall. | treatment, so of limited relevance to post-treatment coatings. |

Estimate of 77% reduction based on comparing mean or the means for playgrounds designated A, B, and H (reported as no prior treatment with stain or paint) to mean of means for playgrounds designated as D, E, G, I and J (identified as having a stain applied). Playgrounds C and F not included because of ambiguity about application of stains. 50-60% reduction in leachable arsenic suggested from comparison of data on soil samples.

## ADDITIONAL PANEL RECOMMENDATIONS

### A. Biomonitoring study

Recommendation

The Panel recommends that a biomonitoring study of children normally exposed to CCA-treated play equipment and decks be conducted. This study should be designed according to well-accepted epidemiological principles, with adequate sample size, to resolve the issue of whether there is substantive exposure of children to arsenic (and possibly chromium) residues. The study should include urinary arsenic measurements as a biomarker of exposure. If practicable, skin wipe samples should be collected in the same study. It would be used to provide exposure information that could be used directly in the risk assessment and also to validate the proposed exposure models.

Planning and study design should begin immediately, as there is a need for such information, irrespective of the final form of the proposed exposure models.

Discussion

In the course of its deliberations the Panel noted two particular things: Firstly, the high degree of uncertainty inherent in the assumptions and default measures proposed for use in the exposure assessment pathway. The cumulative uncertainty in the resulting exposure assessment (and, therefore, the risk assessment) was likely to be substantial. Secondly, the Panel noted the absence of data on exposure of children to arsenic and chromium residues from playing on CCA-treated playground equipment and decks.

There was general consensus that there was an urgent need to obtain biomonitoring data for two main purposes: to obtain data that could be directly used in risk assessments and to validate the exposure assessment models.

The ultimate risk assessments that would employ the exposure data would be of two kinds: risk assessments involving acute or short-term toxicity endpoints and assessments of chronic toxicity endpoints, particularly carcinogenicity. The former would involve relatively large differences in exposure between children exposed and unexposed to CCA-treated timber structures. These differences could be detected in studies using smaller numbers of children than would be necessary for assessments of carcinogenic risk, when smaller incremental exposures might be important.

Concerns about the potential difficulties of carrying out epidemiological studies of this nature were raised. These included the possibility of confounding by other sources of arsenic exposure and whether it would be possible to obtain a representative sample of children.

In response, it was pointed out that other exposures to arsenic would only cause confounding if they were correlated with exposures to arsenic from CCA. *A priori*, this seemed unlikely.

Provided a sufficient sample size was used and data on any potential confounding factors were collected, confounding would not necessarily be a problem and, if necessary, could be adjusted for in the statistical analysis.

In regard to whether a representative sample was necessary, it was agreed that such a sample should not be necessary in the first instance, as the primary issue was one of causal inference – whether there was evidence that children were substantially exposed to arsenic and chromium from CCA-treated timber. A first study could be done in a potentially "worst case" situation. If such a study provided evidence of minimal exposure then it would be likely that children actually did not receive substantial exposure to residues from CCA-treated timber. On the other hand, if such a study did show evidence of exposure then further studies in other settings would be appropriate for refinement of the exposure assessments.

Ideally, the proposed study would include collection of skin (particularly hand) wipe data from the children. This could lead to an improved understanding of the relationship between skin exposure and actual absorption. However, it is important that collection of such wipe samples does not lead to underestimates of the amount of arsenic absorbed and that the collection process does not alter the normal play activities of the children.

It was generally agreed that such studies, involving children, are inherently difficult and need to be designed and carried out very carefully. Because of the potentially long lead time for such studies, it is advisable to begin the planning process for such a study as soon as possible. At the same time, the risk assessment process should not be delayed pending final results of the biomonitoring study.

## B. Effects of Metal-Metal Interactions on Toxicokinetics of Arsenic from CCA-Contaminated Materials and Environmental Media (Soil, Dislodgeable Material)

Recommendation

Detailed information must be provided about total composition of metals and metalloids that are introduced into CCA-treated wood and that are present in contaminated soil and dislodgeable materials. Information about known interactions between arsenic, chromium, and copper should be included into the risk assessment related to CCA-treated wood. Additional studies are needed to obtain more data about chemical and biological interactions of arsenic, chromium, copper, and other metal (metalloid) contaminants found in CCA-treated (contaminated) materials.

Discussion

Exposures to CCA-treated wood components or to CCA-contaminated environmental media represent in fact combined exposures to three metals (metalloids), arsenic, chromium, and copper. It is generally recognized that biological effects associated with a co-exposure to a mixture of metals may significantly differ from effects caused by an exposure to each metal separately. The presence of chromium and copper may affect toxicokinetics (e.g., absorption, tissue distribution/retention, biotransformation, biliary and urinary excretion) of arsenic and *vice*

*versa*. Because bulk chemical agents are used for the CCA treatment, other minor chemical contaminants are also of concern in their effect on the subsequent disposition of arsenic with co-ingestion by exposed children.

Metabolic and toxicological interactions between the three major metallic components of the CCA mixture have previously been reported:

- Co-exposures to arsenic are known to cause a profound accumulation of copper by the kidney cortex (Ademuyiwa, et al., 1996). Although, copper is a relatively nontoxic metal, possible adverse consequences associated with its accumulation in the kidney should be considered under these exposure conditions.
- Co-exposures to arsenic affect tissue levels of chromium in laboratory animals with no significant effects on hypoglycemic properties of inorganic chromium (Aguilar et al., 1997).
- Combined acute exposure to Cr(VI), arsenate, and copper causes a marked decreased in fetal weight and increased incidence of fetal resorption and abnormality formation in rats, while none of the metals is teratogenic when administered (i.p.) separately (Mason et al., 1989).

One concern is the extent to which impurities would affect the process of biliary excretion of arsenic. Any effect on biliary excretion, for example, would complicate easy comparisons and computational adjustments for biliary excretion when doing relative bioavailability studies. That is, these substances would affect arsenic in dislodgeable residues or receiving soils but obviously would not affect any toxicokinetics of the reference, soluble As(V) or As(III) dosing solution. This creates a miscomparison. For example, inorganic selenium is known to modify excretion of arsenic in bile. It has previously been shown that the biliary excretion of arsenic is strongly dependent on glutathione levels in hepatic tissue (Gyurasics et al., 1991). Co-exposures to selenite dramatically increase levels of arsenic in bile in rats (Gregus et al., 1998). Metabolic interactions between arsenic, selenium, and glutathione are responsible for this effect. A complex, seleno-bis(*S*-glutathionyl) arsinium ion, has recently been identified in the bile of rabbits injected with selenite and arsenite ( Gailert et al., 2000). Similar biliary interactions have also been reported for other metalloids (Gregus et al., 1998). Although effects of copper and/or chromium on biliary excretion of arsenic are unknown, a report of Peoples et al., (1979) provided to the Panel indicate that they exist. Here, dogs were fed with food containing sawdust from CCA and ACA treated wood. The first dog received 6 mg As in CCA sawdust; the second dog received 13.2 mg As in ACA sawdust (ACA does not contain chromium). Based on urinary excretion, about 40% of the arsenic dose was absorbed in the first dog over 8 days. In contrast, about 60-70% was absorbed in the second dog, indicating that the presence of chromium in CCA sawdust may decrease absorption and/or urinary excretion of arsenic. The fact that these are one-animal and one-dose data prevents more extensive evaluation of these results.

Other interactions with arsenic that are of concern are those that may potentially affect uptake of arsenic in various media as a function of nutritional status. As noted in responses to Question 2, the arsenic pathway interacts with the phosphorus pathway in biological systems so that a child's

nutritional status with respect to phosphorus deficiency or adequacy could possibly affect the parameter of arsenic uptake.

## C. Bioavailability of Dislodgeable CCA Residues

The Panel agreed with the Agency' s decision to assume on an interim basis 100% relative bioavailability of ingested dislodgeable CCA residue. During the public comment period, results of an unpublished study were presented in which the absolute oral bioavailability of dislodged material from CCA-treated wood was measured in hamsters. The Panel recommended not using this information in the risk assessment at this time because the material dosed has not been characterized and concerns about the animal model. However, the Panel recognizes that oral absorption is a critical variable in the assessment of dose from oral exposure to CCA residues and encourages further research to characterize it.

## REFERENCES


Ademuyiwa O., Elsenhans B., Nguyen P.T., Forth W. (1996) Arsenic-copper interaction in the kidney of the rat: influence of arsenic metabolites. Pharmacol. Toxicol. 78:154-160.

Aguilar M.V., Martinez-para M.C. and Gonzales M.J. (1997) Effects of Arsenic(V)-Chromium(III) interaction on plasma glucose and cholesterol levels in growing rats. Annals Nutr. Metab. 41:189-195.

Bartlett R.J. (1991) Chromium cycling in soils and water: links, gaps, and methods. Environ. Health Perspect. 92: 17-24.

Bartlett R.D. and James, B.R. (1983) Behavior of chromium in soils. V. Fate of organically-complexed Cr added to soil. J. Environ. Qual. 12: 169-172.

Bartlett R.J. and James B.R. (1979) Oxidation of chromium in soils. J. Environ. Qual. 8: 31-35.

Bagdon, R.E. and Hazen, R.E. (1991) Skin permeation and cutaneous hypersensitivity as a basis of making risk assessments of chromium as a soil contaminant. Environ. Health Perspect. 92, 111-119.

Burke, T., Fagliano, J., Goldoft, M., Hazen, R.E., Iglewicz, R. and McKee, T. (1991) Chromite ore processing residue in Hudson County, New Jersey. Environ. Health Perspect. 92, 131-137.

Carmichael E.B., Strickland, J.T., and Driver, R.L. (1945) The contents of the stomach, small intestine, cecum and colon of normal and fasting rabbits. Amer. J. Physiol. 143: 562-566, 1945.

Calabrese, E.J. and Stanek, E.J. (1995) Resolving inter-tracer inconsistencies in soil ingestion estimation. Environmental Health Perspectives 103:454-457.

California Department of Health Services. (1987) Condensed report to the Legislature: Evaluation of hazards posed by the use of wood preservatives on playground equipment. State of California. Office of Environmental Health Hazard Assessment, Department of Health Services, Health and Welfare Agency.

Casteel, S. W.; Brown, L. D. and Dunsmore, M. E.. (1997) Relative bioavailability of arsenic in mining wastes; Document Control No. 4500-88-AORH; U.S. Environmental Protection Agency: Region VIII, Denver, CO.

Casteel S.W., Evans, T. and Dunsmore, M.E. Relative bioavailability of arsenic in soils from the vbi70 site. (prepared for US EPA, Region VIII). Final Report, January, 2001

Clewell, H.J., Gearhart, J.M., Gentry, P.R., Covington, T.R., VanLandingham, C.B., Crump, K.S., and Shipp, A.M. 1999. Evaluation of the uncertainty in an oral Reference Dose for methylmercury due to interindividual variability in pharmacokinetics. Risk Anal 19:541-552.

Cooper, P. and Y.T. Ung. (1977b) Effect of water repellents on leaching of CCA from treated fence and deck units – An update. Presented at the 28th Annual Meeting of the International Research Group on Wood Preservation, May 26-30, 1977, Whistler, Canada

Crump, K. (1998) On summarizing group exposures: Is an arithmetic mean or a geometric mean more appropriate? Risk Anal 18:293-297.

Cullen, A., and H.C. Frey. (1999) Probabilistic techniques in exposures assessment: A handbook for dealing with variability and uncertainty in models and inputs. Plenum Press.

Davis, S., Waller, P., Buschbon, R., Ballou, J., and White, P. (1990) Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: Population based estimates using aluminum, silicon, and titanium as soil tracer elements. Arch. Environ. Health. 45: 112-122.

Decker, P., Cohen, B., Butala, J.H., and Gordon, T. Exposure to wood dust and heavy metals in workers using CCA pressure-treated wood. Amer Ind Hyg Assoc J (in press).

EPA. Baseline Human Health Risk Assessment . Vasquez Boulevard and I-70 Superfund Site. US EPA, Region VIII. August, 2001

Freeman, G.B., Schoof, R.A., Ruby, M.V., Davis, A.O., Dill, J.A., Liao, S.C., Lapin, C.A., and Bergstrom, P.D. (1995) )Bioavailability of arsenic soil and house dust impacted by smelter activities following oral administration in cynomologus monkeys. Fundamental and Applied Toxicology 28: 215-222

Freeman, N.C.G., Jimenez, M. and Reed, K.J. (2001) Quantitative analysis of children' s micro activity patterns: the Minnesota children' s pesticide exposure study. J.Exposure Analysis and Environmental Epidemiology 11:

Gailer, J., George, G.N., Pickering, I.J., Prince, R.C., Ringwald, S.C., Pemberton, J.E., Glass, R.S., Younis, H.S., DeYoung, D.W., and Aposhian, H.V. (2000) A metabolic link between arsenite and selenite: The seleno-bis(S-glutathionyl) arsinium ion. J. Am. Chem. Soc., 122, 4637-4639.

Gregus, Z., Gyurasics, Á., and Koszorús, L. (1998) Interactions between selenium and group Va-metalloids (arsenic, antimony and bismuth) in the biliary excretion. Environ. Toxicol. Pharmacol., 5:89-99.

Gross, P.R., Katz, S.A., and Samitz, M.H. (1968) Sensitization of guinea pigs to chromium salts. J. Invest. Dermatol. 50, 424-427.

Gyurasics, Á., Varga, F., and Gregus, Z. (1991) Effects of arsenicals on biliary excretion of endogenous glutathione and xenobiotics with glutathione-dependent hepatobiliary transport. Biochem. Pharmacol., 41:937-944.

Gyurasics, Á, Varga, F., and Gregus, Z. (1991) Glutathione-dependent biliary excretion of arsenic. Biochem. Pharmacol. 42:465-468.

Hall, L.L., George S.E., Kohan M.G. Styblo, M., and Thomas, D.J. (1997) In vitro methylation of inorganic arsenic in mouse caecum. Toxicol. Appl. Pharmacol. 147:101-109.

Huang, R.N., Lee, T.C. (1996) Cellular uptake of trivalent arsenite and pentavalent arsenate in KB cells cultured in phosphate-free medium. Tox Appl Pharmacol 136:243-249.

Jansen, L.H., and Berrens, L. (1968) Sensitization and partial desensitization of guinea pigs to hexavalent chromium. Dermatologica 137, 65-73.

Kierski, M.W. (1992). The Oral bioavailability of soil-lead in the Weanling rabbit. Ph.D. Thesis, Minneapolis, MN: University of Minnesota Diss. Abs. Int. B: 53: 2819-2820.

Kissel J.C., Shirai, J.H., Richter, K.Y., and Fenske, R.A. (1998) Investigation of dermal contact with soil in controlled trials, J.Soil Contam. 7(6):737-752

Lebow, S.T. and Evans, J.W. (1999) Effect of prestain on the release rate of cooper, chromium, and arsenic from Western hemlock. USDA Forest Service, Forest Products Laboratory, Madison, Wisconsin. Research Note FPL-RN-0271

Levander, O.A. (1977) Metabolic interrelationships between arsenic and selenium. Environ Health Perspect 19:159-164. (A review).

Lu, C. and Fenske, R.A. (1999) Dermal transfer of chlorpyrifos residues from residential surfaces: Comparison of hand press, hand drag, wipe, and polyurethane foam roller measurements after broadcast and aerosol pesticide applications. Environ Health Perspect 107:463-467.

MacKenzie, R.D., Byerrum, R.U., and Decker, C.F. (1958) Chronic toxicity studies II. Hexavalent and trivalent chromium administered in drinking water to rats. A.M.A. Arch Industrial Health 18: 232-234.

Mason R.W., Edwards I.R., and Fisher L.C. (1989) Teratogenicity of combinations of sodium dichromate, sodium arsenate, and copper sulphate in the rat. Comp. Biochem. Physiol. 93C:407-411.

Mor, S., Ben Efraim, S., and Leibovici, J.L. (1988) Successful contact sensitization to chromate in mice. Int. Arch. Allergy Appl. Immunol. 85, 452-457.

Mushak, P. (1998) Uses and limits of empirical data in measuring and modeling human lead exposure. Environ. Health Perspect. 106 (Suppl. 6) 1467-1484.

Peoples, S.A. and Parker, H.R. (1979) The absorption and excretion of arsenic from the ingestion of sawdust of arsenical treated wood by dogs. University of California, School of Veterinary Medicine, Davis, CA. July.

Roberts, S.M., Weimar W.R., Vinson, J.R., Munson, J.W., and Bergeron R.J. (2001) Measurement of arsenic bioavailability from soils using a primate model. Toxicological Sciences 60: 436 Presented at the 2001 Annual Meeting of the Society of Toxicology.

Rodes, C.E., Newsome, J.R., and Vanderpool, R.W. (2001) Experimental methodologies and preliminary transfer factor data for estimation of dermal exposure to particles. J. Exposure Analysis and Environmental Epidemiology 11: 123-139.

Ruby, M.V., Schoof, R., and Brattin, W. (1999) Advances in evaluating the oral bioavailability of inorganics in soil for use in human health risk assessment. Environ Sci Tech 33:3697-3705

Sedman, R.M. (1989) The development of applied action levels for soil contact: A scenario for the exposure of humans to soil in a residential setting. Environmental Health Perspectives 79: 291-313.

Sedman, R.M. and mahmood, R.J. (1994) Soil ingestion by children and adults reconsidered using the results of recent tracer studies. Journal of the Air and Waste Management Association 44:141-144.

Silvers, A. Florence, B.T., Rourke, D.L. and Lorimer, R.J. (1994) How children spend their time: A sample survey for use in exposure and risk assessments. Risk Analysis 14: 931-944.

Solo-Gabriele, H.M. and Townsend, T.G. (1999) Disposal practices and management alternatives for CCA-treated wood waste. Waste Management Research 17: 378-389.

Stanek, E.J. and Calabrese, E.J. (1995) Daily estimates of soil ingestion in children. Environmental Health Perspectives 103: 276-285.

Stilwell, D. (1998) Arsenic from CCA-treated wood can be reduced by coating. Frontiers of Plant Science 51(1): 6-8.

Tolaymat, T.M., Townsend, T.G., and H. Solo-Gabriele (2000) Chromated copper arsenate treated wood in recovered wood. Environmental Engineering and Science . 17(1) : 19-28.

Townsend, T.G., K. Stook, Tolaymat, T.M., Song, J.K., H. Solo-Gabriele, Hosein, N., and Khan, B. (2001) New lines of CCA-treated wood research: In-service and disposal issues – Final Technical Report #00-12. Submitted to the Florida Center for Solid and Hazardous Waste, Gainesville, Florida.

Tyl, R.W., Marr, M., and Meyers, C.B. (1991) Developmental toxicity evaluation of chromic acid administered by gavage to New Zealand white rabbits. Research Triangle Institute, Research Triangle Park, NC Study No. 60C-4808-30/40. Unpublished.

U.S. Consumer Product Safety Commission (CPSC). (1990) Estimate of risk of skin cancer from dislodgeable arsenic on pressure treated wood playground equipment.

Wester, R.C., Maibach, H.I., Sedik, L., Melendres, J., and Wader, M. (1993) In vivo and in vitro percutaneous absorption and skin decontamination of arsenic from water and soil. Fundamental and Applied Toxicology 20: 336-340.

Zaldivar, R. (1977) Ecological investigations on arsenic dietary intake and endemic chronic poisoning in man: dose-response curve. Zbl Bakt Hyg, I Art Orig B 164:481-484.

Zaldivar, R., and Ghai, G.L. (1980) Cllinical epidemiological studies on endemic chronic arsenic poisoning in children and adults, including observations on children with high and low intake of dietary arsenic. Zbl Bakt Hyg, I Abt Orig B 170:409-421.

Zaldivar, R., and Guillier, A. (1977) Environmental and clinical investigations on endemic chronic arsenic poisoning in infants in children. Zbl Bakt Hyg, I Abt Orig B 165:226-234.

Zhang, J.D. and Li, S.K. (1997) Cancer mortality in a Chinese population exposed to hexavalent chromium in water. J.Occ. Env. Med. 39 (4): 315-319.

# Attachment 1 for Appendix F

## The Office of Pesticides Programs (OPP) Responses to Scientific Advisory Panel (SAP) Recommendations for CCA Children Exposure and Risk Assessment

**The Office of Pesticides Programs (OPP) Responses to Scientific Advisory Panel (SAP) Recommendations for CCA Children Exposure and Risk Assessment**

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 1. Please comment on the choice of this data set and value chosen for representation of the relative bioavailability of inorganic arsenic from ingestion of arsenic-contaminated soil. Please discuss the strengths and weaknesses of the selected data and also provide an explanation as to whether this 25% value is appropriate for estimation of bioavailability in children. | • Four different suggestions 25%, 50%., 25 to 50%, 0-98%<br>• Oral absorption of arsenic from soil, consideration should be given to absorption of arsenic from nonsoil substances (such as wood chips or other buffer material) that might be subject to incidental ingestion.<br>• Research is needed to obtain data on the relative bioavailability of arsenic from numerous sites that encompass the broad range of soil types and arsenic contamination specifically resulting from CCA-treated wood applications. These studies should be conducted in appropriate animal models preferably at doses that simulate the anticipated level of exposure of children playing on or around structures or sites subject to CCA contamination. | ? As suggested , a study of relative bioavailabile study of arsenic in soil affected by CCA- treated wood and wood residues collected from surface of CCA-treated wood were conducted in juvenile swine. (ACC, 2003)<br>? As suggested , a study of relative bioavailabile study of arsenic in soil affected by wood residues collected from surface of CCA-treated wood were conducted in juvenile swine (ACC 2003) |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 2. Please comment on the Agency's selection of the 0.05 mg/kg/day LOAEL value for use in assessing risks to the general population as well as children from short-term and intermediate-term incidental oral and dermal exposures, and the appropriateness of the use of a 10x factor for severity of the toxic effects observed in the Mizuta study. Please provide an explanation and scientific justification for your conclusions as to whether the presented data are adequate or whether other data should be considered for selection of this endpoint. | • 0.05 mg As/kg per day is an appropriate LOAEL for short- (1 to 30 day) and intermediate- (31 to 180 day) human ingestion of the chemical.<br>• MOE of 30 from this LOAEL is recommended for non-cancer health effects<br>• MOE of 10 is also suggested by some panel members | ? As suggested. A LOAEL of 0.05 mg As/kg per day with a MOE value of 30 will be used for short- (1 to 30 day) and intermediate- (31 to 180 day) human ingestion of As. |
| 3. Please comment on the selection of the value of 6.4% for dermal absorption of inorganic arsenic and whether or not this value will be appropriate for use in all scenarios involving dermal exposure to arsenic from CCA-treated wood, including children's dermal contact with wood surface residues and contaminated soils. | • In the range 2-3%, for dermal absorption of inorganic arsenic should be used.<br>• Using arsenic in more appropriate chemical form (that it is present in dislodgeable CCA residues and in soil beneath CCA-treated sites) and in a relevant matrix, should be carried out to improve estimates of dermal absorption. | ? As suggest, 3% will be used in the risk assessment for children playing around playground equipment.<br>? As suggested , a study of dermal absorption of arsenic in the wood residue collected from the surface of CCA-treated wood was conducted. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 4. As available monitoring data do not differentiate among chromium species found in CCA dislodgeable residues on wood surfaces and in soils, and as Cr (VI) is the more toxic species of chromium, please comment on whether use of the hazard data for chromium (VI) is the best choice for characterizing hazard and risk from exposure to chromium as a component of CCA-treated wood. Please provide a scientific explanation and justification for your recommendation on the choice of either the chromium (III) or chromium (VI) hazard database.. | • One approach would be to use an estimate of 25 to 50% hexavalent chromium. Some Panel members suggested 5 to 10% would be conservative.<br>• The Panel strongly recommends that EPA conduct studies of chromium speciation (in both dislodgeable residues and soil samples) in their proposed studies.. | ? As suggested , 10% will be used in the risk assessment. |
| 5. Please comment on the Agency's selection of the 0.5 mg/kg/day NOAEL value for use in assessing risks to the general population as well as children from short-term and intermediate-term incidental oral exposures to inorganic chromium as contained in CCA-treated wood. Please provide an explanation and scientific justification for your conclusions as to whether the presented data are adequate or whether other data should be considered for selection of these endpoints. | • The Panel expressed concerns regarding the selection of the 0.5 mg/kg/day NOAEL for short-term and intermediate-term incidental oral exposures to inorganic chromium. In general, these concerns involved the appropriateness of the study selected by EPA (Tyl, 1991) to derive this value. It is the Panel's recommendation that the Agency re-review the literature and consider other potentially more relevant studies.<br>• The Panel questioned whether the study proposed for the derivation of the NOAEL (Tyl, 1991).actually demonstrated the purported effect. The Panel was divided on this issue; some thought the study adequate and appropriate to support the proposed NOAEL while others thought the study to be flawed and inappropriate. | ? The issue of using Tyl (1991) has been discussed in the OPP's Hazard Identification Assessment Review Committee (HIARC). HIARC considered this is a good study with appropriate exposure duration for the study scenarios. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 6. Please comment on whether the significant non-systemic dermal effects from dermal exposure to inorganic chromium should form the basis of dermal residential risk assessments, and, if so, how the Agency should establish a dermal endpoint for such an assessment. | • EPA should base risk assessments for noncancer health effects of dermal exposure to hexavalent chromium on direct dermal effects irritant and allergic contact dermatitis | ? Because the thresholds for the dermal effects usually are very low and individual variations usually are big, therefore, the dermal effects will be address in a qualitative risk assessment. |
| 7. Please comment on whether OPP's choices of central tendency and high end values for different parameters should, collectively, produce estimates of the middle and high end of the range of potential exposures. If the Panel thinks that OPP's approach may not estimate the high ends of the exposure range (because it produces values that are either higher or lower than the upper end of the exposure range), please comment on what specific values should be modified to produce estimates of the high end of potential exposure. | • Particularly when using point estimates it is important to do subset analyses for specific regions of the country (for example, the South compared to the North or Midwest) and for age groups (for example, one year olds compared to 5-6 year olds).<br>• The averaging of exposure over a 75-year lifetime may underestimate risk. More research is needed to understand the uncertainty associated with this form of temporal averaging.<br>• More research is needed on the amount of soil ingested, as this is still a source of uncertainty.<br>• For fully evaluating high end exposures it would be necessary to include exposure of children with Pica.<br>• A probabilistic assessment as discussed in question 8 is recommended. | ? OPP will conduct a probabilistic risk assessment which will include regional and seasonal subset analyses.<br>? 75- years old lifetime is recommended by the EPA Exposure handbook which has been presented to the SAP in 1999.<br>? OPP/ORD used more updated data from Stnek and Calabrease (2000) and Stanek et al. (2001) and a workshop report by ATSDR on soil pica workshop (ATSDR, 2001)<br>? The probabilistic risk assessment will address the issue of children with Pica. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 8. Please comment on whether the existing data bases on the variability of the different parameters affecting potential exposure are adequate to support the development of probabilistic estimates of potential exposure. If the Panel regards the data bases as adequate for that purpose, please identify which parameters should be addressed using a distribution of values and which data bases should be used to supply the distribution for particular parameters. | • In view of its concerns that the deterministic model reviewed in Question 7 will not correctly estimate the central tendency or percentiles of the exposure distribution, the SAP recommends that the EPA immediately begin to take steps toward the development and progressive refinement of probabilistic models of exposure. The probabilistic models will give high-end values that are interpretable as a percentile of the modeled exposure distribution rather than a biased approximation of the upper limit of exposure. The existing databases on the variability of the different parameters affecting potential exposures of children using CCA-treated playground structures are adequate to begin the development of probabilistic estimates of potential exposure provided the uncertainty associated with these data is reflected in the exposure modeling. As noted above, the Panel views the development of a probabilistic assessment as a process of progressive learning and refinement. New or more detailed data on states and transition factors are needed and will contribute to improvements in the exposure models as they become available. | ? Based on the panel recommendation, EPA has already started to develop a probabilistic risk assessment using the SHEDS-Wood model.<br>? OPP agrees with the SAP panel recommendations in regards to the development of the probabilistic risk assessment. The panel recommendations will be incorporated into the development of the risk assessment for CCA. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 9. OPP is assuming that a one-to-one relationship applies to the transfer of residues from wood to skin. The Panel is asked to address whether this is a reasonable assumption, and if not, to provide guidance on other approaches. | • The Panel does not recommend assuming that a one-to-one relationship applies to the transfer of CCA chemical residues from wood to skin as proposed by the Agency. It is the Panel's opinion that the underlying conceptual model is questionable. Sufficient justification for a one-to-one relationship was not provided and the limited available empirical data contradict the validity of the assumed one-to-one relationship.<br>• The Panel strongly recommends that the Agency expand its planned joint study with CPSC to measure dislodgeable CCA chemicals from an appropriate sample of play structures, so as to obtain information of more direct value for exposure assessment. Ideally CCA chemical loadings on the hands (and possibly other skin surfaces) of children using play structures would be measured in addition to corresponding dislodgeable residues. At a minimum, some Panelists would accept gathering of data sufficient to more adequately support implementation of OPP's current conceptual model (e.g., matched adult volunteer hand and cloth wipe samples to better establish the relationship between these two measures as well as the constancy of any relationship as a function of surface area sampled).<br>• The Panel was divided on an interim recommendation for the Agency while it awaits collection of these additional data for the EPA/CPSC study. Some Panel members were willing to endorse interim use of existing hand or fabric wipe data if described probabilistically. One Panel member voiced strong opposition to any use of cloth wipe data until the Agency obtained additional information establishing | ? OPP agrees with the panel approach related to this question and will follow their recommendations for the development of the risk assessment.<br>? A new transfer efficiency based on ACC (2003) and CPSC (2003) studies had been developed for probablistic assessment. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 10. The Panel is asked to comment on whether the proposed Soil Adherence Factor (AF) of 1.45 mg/cm$^2$ for hand contact with commercial potting soil is a realistic value for use in estimating the transfer of residues from playground soil to skin in this assessment. | • Use of an AF of 1.45 mg/cm$^2$ is not recommended. The proposed AF was derived from an unpublished study of very limited scope. EPA has funded subsequent research to derive more representative values. | ? OPP agrees with the panel and will use the study conducted by Holmes et. Al. (1999) based on lognormal distribution, a geometric mean of 0.11 mg/cm$^2$ and a geometric standard derivation of 2.0 was selected.<br>? The probabilistic risk assessment will address the seasonal and geographic variations. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 11. OPP will need to calculate intermediate-term, and possibly long-term exposures in this assessment using available wood/soil residue data. The Panel is asked to recommend a credible approach for selecting residue data values for use in OPP's risk assessment, taking into consideration the inherent variability of the data sets. Please advise us on which values are best for representing central tendency and high end exposures. Also, the Panel is asked to discuss the feasibility of combining data from individual data sets. | • The proposed USEPA/CPSC study of wood and soil residues associated with CCA-treated playground equipment provides a unique opportunity to generate a substantial data set on the variability of residue levels for the playground scenario using a standardized sampling and analytical methodology. This study should help to resolve uncertainty regarding the relative contribution of true, inherent variability in residues versus variability due to differences in methodology. It is critical, therefore, that the protocol be highly detailed regarding sampling methods, locations, and frequencies and that the protocol be rigidly followed. Basic scientific criteria for acceptance of the final data set should be laid out first and include: standardized collection methods, precision, accuracy, reproducibility, and other measures of QA/QC.<br>• The Agency should not combine data with quite differing levels of precision and conservativeness, and use one set of data to drive other model considerations. The model cannot be fully evaluated without real world (i.e., biomonitoring or soil consumption) data for comparison, and that comparison cannot be made without a representation of both variability and uncertainty in model outputs. | ? OPP followed the panel recommendations and obtained pertinent data from the sampling study conducted in cooperation with the CPSC.<br>? The CPSC's wood residue study (2003) was combined with ACC's (2003) study for the surface residue concentration distribution |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 12. Does the Panel have any recommendations for combining the four scenarios (oral/wood, dermal/wood, oral/soil, dermal/soil) such that a realistic aggregate of these exposure routes may be estimated? | • The Panel encourages the Agency to aggregate exposure estimates across all potential sources. This should occur in a way that makes the contribution of various sources of exposure transparent and tracks separate species of arsenic and chromium. Although data at present are limited, it is possible that the different species of arsenic encountered from distinct exposure scenarios may differ with respect to their hazard. For example, arsenic in the form of a complex of copper chromated arsenate ingested from direct contact with freshly treated wood might be metabolized and excreted differently than arsenate leached from weathered wood and ingested incidentally in soil.<br>• The suggested scenarios (oral/wood, dermal/wood, oral/soil, dermal/soil) capture the exposures that may occur on playscapes and decks. Inhalation exposure may be a route that should be included, however at this point in time, data are insufficient to estimate the distribution of possible inhalation exposures. Refer to the response to question 13 for further analysis.<br>• However, in terms of the aggregate exposure assessment, the proposed scenarios do not capture sources of exposure that appear to be significant. The Panel suggests that the Agency broaden its inquiry to consider the diversity of possible exposures to arsenic, chromium and copper. Some Panel members felt that the Agency should expand its formal analyses of exposure to include other media under the jurisdiction of other EPA offices—drinking water, air, and waste—to avoid a fragmented and incremental approach to risk assessment and management of arsenic, chromium and copper species. | ? After the completion of the probabilistic modeling, OPP had combined the results from the four scenarios to obtain an aggregate risk assessment.<br>? OPP believes that the probabilistic modeling will answer most of the panel's questions in regards to this issue.<br>? OPP agree that inhalation route should not be included.<br>? OPP agree with panel's recommendation and look at into EWG's raw data as consideration. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| Question 12 (Continued) | • Probabilistic methods should be used to estimate exposure and risk. This demands selection of best available data sets to construct distributions. This must be done with considerable care. The EWG approach seems conceptually reasonable; however, their method combines point estimates with distributions, and this may introduce bias into the estimates. The Lifeline method is especially well adapted to aggregate exposures across diverse routes, while preserving estimates at the level of the individual. The Agency should be encouraged to develop this model in the immediate future while closing data gaps.<br><br>• Uncertainty should be carefully characterized, distributions characterized, and clear criteria applied to judge the quality of available data for each parameter included in the assessment. The Agency should further develop Table 4 in the EPA support document Children's Exposure to CCA Treated Wood Playground Equipment and CCA Contaminated Soil. Table 8 in the Gradient Corporation submission provides a similar model that attempts to identify ranges of factors potentially affecting exposure, tracks the sources of data, and provides a preliminary characterization of uncertainty.<br><br>• Regarding uncertainty and default assumptions the Agency should confront two questions directly: When are data of sufficient quality to include in a modeling effort? What should be done until data are adequate? The SAP provided the Agency with clear criteria to judge data quality in 1999, and these were recognized | ? Uncertainty analysis was conducted based on 2-stage Monte Carlo simulation.<br>? In SAP 2002, OPP and ORD had develop the method including warm and cold climate (see SHED-Wood report, 2003).<br>? OPP agree with panel's comments, the childhood behavior and activity data are needed.<br>? In SHEDS-Wood, the age is considered in the simulation |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| Question 12 (Continued) | ? The Agency should develop methods that aggregate exposure and risk estimates for individuals. These may then be aggregated by various demographic characteristics—age, income, ethnicity, and location (north/south; urban/rural), or specific behavioral characteristics.<br><br>? The literature on childhood behavior and activity patterns that may be associated with CCA exposures is quite young. It provides only a limited basis for understanding the associations between behavior and exposure. The Panel recommends that the Agency undertake studies of childhood behavior and activity patterns to clarify these possible associations, as children move through their daily life. These studies would be useful in EPA assessment of exposures to many different hazardous substances. The Agency's efforts in developing the Exposure Factors Handbook and the more recent Children's Exposure Factors Handbook are very positive and important contributions that support data based, behavioral scenario-building.<br><br>? The Panel anticipates considerable year-to-year variability in exposure among children ages 1-6. Toddlers between 1 and 2 years of age play, behave, eat and dress very differently than 6 year olds, and these are likely to affect contact with contaminated media.<br><br>? CCA-treated wood is increasingly being used for interior construction, and if unfinished and left | |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| Question 12 (Continued) | ? Residential, educational, day-care, recreational, and occupational environments all offer the possibility of childhood exposures to CCA. Specific locations where CCA is in common use include decks, playscapes, railings, docks, piers, pilings, fencing, and exposed untreated interiors of structures, especially those close to the ground such as sills. Picnic tables, mulch, and contact with wood scraps, smoke from intentional and unintentional fires, and ashes from burned construction debris could all be sources of exposure.<br><br>? The Panel encourages the Agency to consider possible high exposure scenarios defined by overlapping risk factors. For example, a toddler living in the Southwest, may experience high drinking water concentrations of arsenic. At the same time the warm climate encourages extended periods of outdoor recreation. If this child is enrolled in a day care facility with decks and play structures made from CCA-treated wood, the aggregate exposure may be high. The identification of populations that are both physiologically susceptible and highly exposed would provide a logical basis for strategic risk management. | ? SHED-Wood focus on children 1-6 year old to public playset in daycare center, and should be the primary focus. The exposure to the home playset is considered as secondary scenarios.<br><br>? OPP agree panel's comments it will be helpful if a biomonitoring study can be conducted, such as in South west area. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 13. Can the Panel comment on whether OPP should conduct a child playground inhalation exposure assessment, taking into consideration the hazard profile for chromium (VI) as an irritant to mucous membranes? If so, can the Panel comment on whether the endpoint described above is appropriate for assessing the risk to children from such an exposure? | • The Panel notes that both the trivalent and hexavalent forms of chromium are of concern in the inhalation route of exposure and that arsenic should also be considered in the inhalation route of exposure.<br><br>• However, the Panel agrees that calculations of probable exposure concentrations suggest that the Agency should not consider the inhalation route of exposure to inhaled metals in their risk assessment. The SAP strongly suggests, however, that exposure concentrations be monitored via personal and area sampling to validate such a conclusion. | ? Opp does not have a plan to perform inhalation exposure assessment.<br><br>? In General, OPP assumes that because of the low volatility of the CCA, conducting an inhalation study is not possible. However, OPP will try to obtain any data available related to the inhalation of CCA |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 14. Data on the effectiveness of reducing exposure by using buffering materials are limited. Does the Panel have recommendations as to whether additional studies to obtain this information are warranted? Does the Panel have suggestions on how OPP can best evaluate child exposures attributed to contact with CCA-contaminated buffering materials? | • The consensus of the Panel is that additional studies are warranted to obtain information needed to assess the exposures associated with using buffering materials.<br><br>• Buffering materials do not appear to provide a means to reduce exposures to CCA leached from play structures. Rather, buffering materials can present important risk scenarios that differ from the four scenarios currently proposed for analysis by the Agency. These additional scenarios include: 1) exposure to buffer materials that become contaminated with metals from CCA leached from play structures and 2) exposure to buffer materials that contain CCA because they consist of recycled construction/demolition debris which contains CCA-treated wood.<br><br>• Exposure to buffer materials that become contaminated with metals from CCA needs to be examined. This effort should be aided by the generation of data describing the amount and nature of exposure in young children who play on or with these materials. It may also be possible to generate bounding estimates of exposure from these materials for the purpose of screening the relative importance of this scenario compared to other play structure-related scenarios.<br><br>• Exposure to buffer materials that contain CCA-bearing mulch will likely result in a sufficient hazard potential to warrant a modification of the recycling practices that lead to the introduction of this mulch into children's play environments. | ? OPP agrees that there is a need for further studies.<br><br>? OPP will try to obtain all available data related to buffers and the extent of the contamination of buffers used in CCA-treated playgrounds. |

| OPP Questions to SAP | SAP RECOMMENDATIONS to OPP | OPP RESPONSE |
|---|---|---|
| 15. The Panel is asked to comment as to whether stains, sealants and other coating materials should be recommended as a mitigation measure to reduce exposure to arsenic and chromium compounds from CCA treated wood. If so, can the Panel | ● The Panel recommends that the EPA inform the public of the ability of certain coatings to substantially reduce leachable and dislodgeable CCA chemicals and thus reduce potential exposure to arsenic and chromium. While the Panel makes recommendations below regarding the need for additional studies in this area, it feels that the current evidence is sufficient to begin advising the public about the use of coatings now.<br>● The weight-of-evidence from available studies indicates that certain<br>● Reductions of 70 to 95% in dislodgeable arsenic were seen in all studies that subjected CCA wood to natural weathering.<br>● There is no evidence that water repellants added directly to the CCA treatment solution are effective in reducing leachable/dislodgeable CCA chemicals.<br>● Confidence is highest for polyurethane as this coating has been shown to result in substantial 70 to >95% reduction in dislodgeable arsenic in a well controlled field study, a "real-world" application allowing for effects of use, and a short-term controlled laboratory study.<br>● Current data support a treatment frequency of once per year, although for some products this may be too frequent (e.g., possibly polyurethane where one study noted up to 95% reduction in dislodgeable arsenic out to 2 years). This is an area in need of additional study.<br>● More studies are needed to evaluate the performance / efficacy of different types and brands of coatings.<br>. | ? The Agency is looking into this issue and is considering the panel's as well as the industry's recommendations for the most appropriate way to advise the public for suitable sealants to be used on the CCA-treated wood.<br><br>? SHEDS-Wood used average 90% as reduction factor (from existing articles) and assuned 99.5 as maximum effect sealant in the simulation |

# Appendix G

## Effect of Hand Washing on Risks from Exposure to Residues

**Effect of Hand Washing on Risks from Exposure to Residues**

**November 7, 2003**

The effect of hand washing on risk from residue only exposure was examined. Two pathways, dermal absorption and ingestion, were summed to determine the risk due to residue only exposure. Comparisons were then made for this residue risk between baseline and the hand washing mitigation scenario for playsets alone and deck and playset exposure. Results are presented in bar charts for the 50$^{th}$ and 95$^{th}$ percentiles. The results at the 99$^{th}$ percentile are somewhat unstable so they are not presented. Figure 1 is for playset only exposure and Figure 2 is for playset and deck exposure. For playset only exposure, residue risks with hand washing were reduced by approximately 36% at the median and slightly more (48%) at the 95$^{th}$ percentile. For playset and deck exposure these differences are less. At the median, playset and deck residue risk under baseline conditions were reduced by approximately 27%; at the 95$^{th}$ percentile they were reduced by 35%.

When total risk is calculated by combining residue and soil risk the effect of hand washing is less. Table 1 shows a comparison for total risk between baseline and the hand washing mitigation scenario. For playset only exposure the difference between baseline and hand washing at the 50$^{th}$ and 95$^{th}$ percentiles is 32% and 45%, respectively. For playset and deck exposure, risks are reduced by 22% and 29% at these same percentiles. See Zartarian et al. (2003) for a more complete description of the hand washing scenario.

**Figure 1. Comparison of Playset Only Residue Risk for Baseline and Hand Washing**

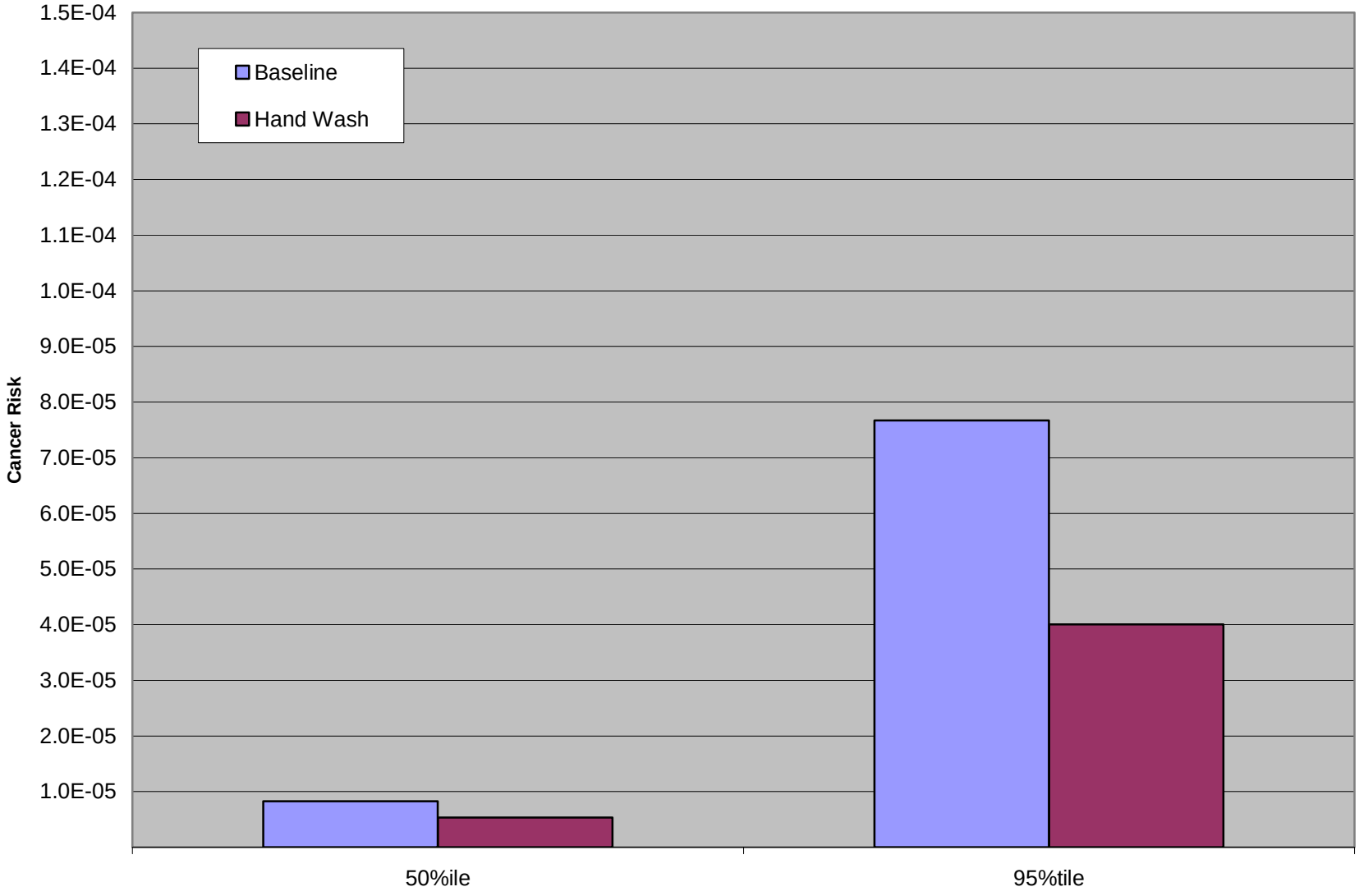


**Figure 2. Comparison of Playset and Deck Residue Risk for Baseline and Hand Washing**

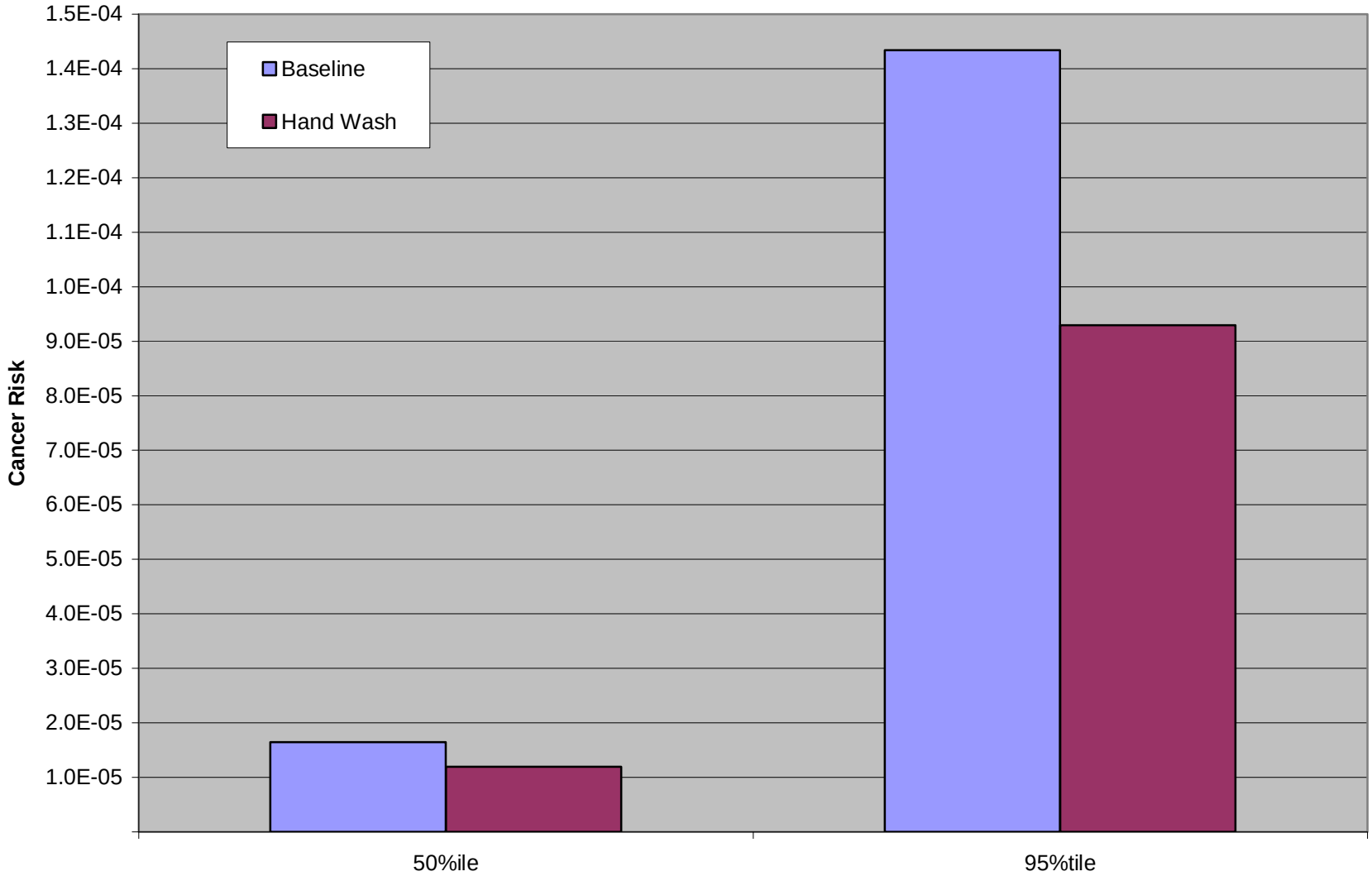


**Table 1 Comparison for Total Risk (Residue and Soil) Between Baseline and the Hand Washing Mitigation Scenario**

| Total Risk:Playset Only | 50th Percentile | 95th Percentile |
|---|---|---|
| Baseline | 1.10E-05 | 8.30E-05 |
| Hand Wash | 7.50 E-06 | 4.60E-05 |
| Percent Reduction | 32% | 45% |

| Total Risk: Playset and Deck | 50th Percentile | 95th Percentile |
|---|---|---|
| Baseline | 2.30E-05 | 1.40E-04 |
| Hand Wash | 1.80E-05 | 1.00E-04 |
| Percent Reduction | 22% | 29% |