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#11697

# EVALUATION OF SITE-SPECIFIC RISK ASSESSMENT FOR CONTAMINATED LANDS

Submitted to:

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TABLE OF CONTENTS

SECTION PAGE

EXECUTIVE SUMMARY ..... iv

1.0 INTRODUCTION ..... 1

2.0 BACKGROUND ..... 2
2.1 Contaminated Sites Risk Assessment in the Canadian Private Sector ..... 2
2.2 Risk Assessment Principles, Variability and Uncertainty ..... 3
2.2.1 Risk Estimation and Toxicity Reference Values ..... 4
2.2.2 Dose Calculations ..... 5
2.2.3 Environmental Fate Model Calculations ..... 8
2.2.4 Sources of Variability ..... 9

3.0 METHODOLOGY ..... 9
3.1 Overview of Case Study ..... 9
3.2 Selection of Participants ..... 11
3.3 Data Analysis ..... 12
3.3.1 Background ..... 12
3.3.2 Tier I ..... 12
3.3.3 Tier II ..... 13
3.3.4 Tier III ..... 14

4.0 RESULTS ..... 14
4.1 Tier I ..... 14
4.1.1 Pathways ..... 15
4.1.2 Receptors and Exposure Scenarios ..... 16
4.1.3 Modes of Toxic Action ..... 16
4.1.4 Analytical Approach ..... 17
4.1.5 Risk Estimates ..... 17
4.1.6 Toxic Potency ..... 21
4.1.7 Dose Rates ..... 22
4.1.8 Relative Magnitude of Risk Estimates ..... 22
4.1.9 Relationship of Capability Score to Risk Estimates ..... 23
4.1.10 Regional Trends in Risk Estimates ..... 23
4.1.11 Sources of Variability in Risk Estimates ..... 23
4.2 Tier II ..... 24
4.3 Tier III Model Variability ..... 25
4.3.1 Environmental Fate And Transport For Dust Inhalation Pathway ..... 25
4.3.2 Environmental Fate And Transport For Soil Gas VOC Building Intrusion Pathway ..... 28

5.0 DISCUSSION ..... 32
5.1 Sources of Variability in Risk Estimates ..... 32
5.2 Environmental Fate Modelling ..... 35
5.2.1 Dust Inhalation Pathway ..... 35
5.2.2 Environmental Fate and Transport for Soil Gas VOC Outdoor Pathway ..... 36
5.2.3 Soil Gas Fate ..... 36
5.2.4 Comparison of Advective Soil Gas Flow Rates Using Three Models ..... 38
5.2.5 Summary of Soil Gas Modelling Results ..... 40
5.3 Relevance to Decision Making ..... 41

6.0 CONCLUSIONS ..... 43

REFERENCES ..... 45

TABLE 1 Exposure pathways amenable to statistical analyses and the number of participants employing a specific pathway/contaminant/receptor combination.
TABLE 2 Summary of non-cancer risks for future residents. Values representing the minimum, maximum and ratio are based on consideration of both adult and child receptors.
TABLE 3 Number of participants concluding acceptable versus non-acceptable risks by pathway and contaminant for non-carcinogenic endpoints.
TABLE 4 Summary of cancer risks for future residents. Values representing the minimum, maximum and ratio are based on consideration of both adult and child receptors.
TABLE 5 Number of participants concluding acceptable versus non-acceptable risks by pathway and contaminant for carcinogenic endpoints.
TABLE 6 Summary of Toxicity Reference Values
TABLE 7 Summary of Cancer Slope Factors
TABLE 8 Participant Ranking Based on Relative Magnitude of Hazard Quotients for Soil Ingestion (Child Receptors)
TABLE 9 Participant Ranking Based on Relative Magnitude of Hazard Quotients for Dust Inhalation Pathway (Child Receptors)
TABLE 10 Summary of Environmental Fate and Transport Modelling for Outdoor Dust Inhalation Pathway
TABLE 11 Summary of Selected Model Input Parameters for Outdoor Dust Pathway - Cadmium
TABLE 12 Summary of Environmental Fate and Transport Modeling for Indoor Soil Gas Intrusion Pathway
TABLE 13 Summary of Selected Model Input Parameters for Indoor Soil Gas Intrusion Pathway

TABLE 14 Comparison of Advective Soil Gas Flow Rates Through Building Envelope

FIGURE 1 Generalized Influence Diagram for the Human Health Risk Assessment Process
FIGURE 2 Non-Cancer Risks Associated with Zinc Exposure
FIGURE 3 Non-Cancer Risks Associated with Lead Exposure
FIGURE 4 Non-Cancer Risks Associated with Benzene Exposure
FIGURE 5 Acceptability of Total Risk (Hazard Index) for Non-Carcinogens
FIGURE 6 Cancer Risks Associated with Cadmium Exposure
FIGURE 7 Cancer Risks Associated with Benzene Exposure
FIGURE 8 Cancer Risks Associated with Vinyl Chloride Exposure
FIGURE 9 Acceptability of Total Risk for Carcinogens
FIGURE 10 Range in Reference Doses for Dust Inhalation Pathway
FIGURE 11 Dose Rates for Zinc Exposure (Non-Carcinogenic)
FIGURE 12 Dose Rates for Lead Exposure (Non-Carcinogenic)
FIGURE 13 Dose Rates for Benzene Exposure (Carcinogenic)
FIGURE 14 Dose Rates for Vinyl Chloride Exposure (Carcinogenic)
FIGURE 15 Component Variability for Risk and Dose Estimates
FIGURE 16 Particulate Emission Rate Versus Soil Mode Based on Cowherd's Unlimited Erosion Potential Model (Cowherd et al., 1985)

APPENDIX I Interim Results of Phase I Study
APPENDIX II Case Study
APPENDIX III Supplementary Results

## EXECUTIVE SUMMARY

This report provides the results of a two-phased study conducted for the Canada Mortgage and Housing Corporation (CMHC) to examine the practices and variability amongst practitioners of contaminated sites risk assessment in Canada. Phase I consisted of a survey of practitioners in the private and regulatory sectors. The intent of the private sector survey was to characterize the capabilities and experience of private firms engaged in human health risk assessment across Canada. Insight gained from the private sector survey was used to assist in the selection of participants for the round robin study which comprised Phase II. The intent of the regulatory survey was to gain insight on a regional basis with regards to regulator experience and acceptance of human health risk assessment of contaminated sites.

As part of Phase II, nine Canadian practitioners from various regions in Canada with varying levels of expertise performed a screening level risk assessment of a hypothetical case study. The purpose of the Phase II study was to assess the degree of variability in risk estimates among participants, and analyze the sources of variability and uncertainty. For Phase II, a hypothetical case study was designed and circulated to nine participants. The hypothetical case study consisted of a residential housing development proposed on former industrial lands and in this respect is reflective of a "brownfields development.

The results from Phase I of the study indicate that Canadian risk assessment practitioners have broad expertise in relevant disciplines such as toxicology, biology, environmental engineering, chemistry, and hydrology. Many of the firms supplement their in-house capabilities with external consultants. The results of the regulatory survey indicate that governmental agencies support the use of human health risk assessment in the management of contaminated sites. However, at present the approach is generally based on informal policy and not formally regulated.

The results of Phase II of the study indicate that Canadian firms vary considerably in their approach to performing human health risk assessments at contaminated sites. This results in risk estimates that ranged over several orders of magnitude for various chemical exposure pathways. The high variability in risk estimates was due to a combination of factors including differences in the assumed chemical toxicity, receptor characteristics, and differences in model type and assumptions used to predict vapour and dust concentrations

in air. Since the magnitude of risk estimates typically varied over several orders of magnitude for a particular chemical exposure pathway, it is likely that in some cases real world business and/or risk management decisions may be erroneously influenced by screening risk assessment practices. Depending on the results of a screening level risk assessment, risk assessors may incorrectly conclude that chemicals present at a site are not a human health concern when in fact the health risks are significant or conclude that chemicals present at a site are a human health concern when in fact the health risks are minimal. Direct application of these findings to a definitive risk assessment is discouraged because the latter situation is more likely to allow assessors greater time and effort to improve realism in risk estimates. However, it would seem prudent to provide guidance to practitioners and risk manager on how to apply risk assessment assumptions to encourage greater continuity in risk assessment and risk management.

## RÉSUMÉ

Ce rapport fait état des résultats d'une étude en deux phases effectuée pour le compte de la Société canadienne d'hypothèques et de logement (SCHL) dans le but d'examiner les méthodes des spécialistes qui évaluent les risques inhérents à des sites contaminés au Canada ainsi que la variabilité de leur travail. La phase I a consisté à faire un relevé des personnes qui effectuent ce genre d'évaluation dans le secteur privé et au sein d'organismes de réglementation. L'examen des activités du secteur privé devait permettre de caractériser les capacités et l'expérience des firmes privées actives dans le domaine de l'évaluation des risques pour la santé humaine au Canada. L'information recueillie lors de cette enquête a été utilisée pour choisir des participants à une étude comparative à effectuer en phase II. Pour ce qui est de l'examen des activités d'évaluation du risque des organismes de réglementation, il s'agissait d'obtenir des données régionales sur l'expérience de ces organismes et sur leur tolérance du risque pour la santé humaine que représentent les terrains contaminés.

Dans le cadre de la phase II, neuf spécialistes canadiens travaillant dans diverses régions du Canada et possédant divers niveaux de savoir-faire ont réalisé l'examen préalable d'une évaluation du risque pour un cas hypothétique. Le but de la phase II était d'évaluer la variabilité des évaluations effectuées par les participants et d'analyser les sources de variabilité et d'incertitude. Pour la phase II, donc, un cas hypothétique a été imaginé et transmis aux neuf participants. Le cas hypothétique consistait en un ensemble résidentiel devant être construit sur un ancien emplacement industriel, ce qui correspondait à un «aménagement sur friche contaminée».

Les résultats de la phase I de l'étude indiquent que les spécialistes canadiens de l'évaluation du risque possèdent une vaste expérience dans des disciplines pertinentes comme la toxicologie, la biologie, le génie de l'environnement, la chimie et l'hydrologie. Bien des firmes comblent leurs lacunes internes en ayant recours à des consultants. Le relevé des spécialistes travaillant pour les organismes de réglementation indique que les agences gouvernementales appuient le recours à l'évaluation du risque pour la santé humaine dans la gestion des terrains contaminés. Toutefois,

l'approche actuelle repose généralement sur des lignes de conduite officieuses et n'est pas officiellement réglementée.

Les résultats de la phase II de l'étude montrent que les firmes canadiennes ont des méthodes très variées pour évaluer les risques que représentent les terrains contaminés pour la santé humaine. Il en découle des évaluations qui varient de plusieurs ordres de grandeur pour divers modes d'exposition à des substances chimiques. La grande variabilité des évaluations du risque provient d'une association de facteurs comme les différences dans la toxicité chimique présumée, les caractéristiques des récepteurs ainsi que les différences touchant le type de modèle et les hypothèses utilisées pour prévoir les concentrations de vapeur et de poussière dans l'air. Comme l'importance de l'évaluation du risque a généralement varié de plusieurs ordres de grandeur pour un mode d'exposition chimique particulier, il est probable que, dans certains cas, les décisions d'affaires ou de gestion du risque qui sont prises dans la réalité soient mal orientées par un examen préalable à l'évaluation du risque. En se fiant aux résultats obtenus à partir d'un examen préalable, les évaluateurs du risque pourraient faussement conclure que les substances chimiques présentes sur un terrain ne constituent pas un risque pour la santé humaine alors qu'en fait les risques pour la santé sont importants ou, au contraire, croire que les substances chimiques qui se trouvent sur les lieux représentent un risque pour la santé humaine alors que ce risque est plutôt minime. L'utilisation directe de ces résultats aux fins d'une évaluation du risque décisive est déconseillée, car cette dernière situation est plus susceptible de permettre aux évaluateurs de consacrer plus de temps et d'effort à rendre plus réalistes leurs estimations du risque. Cela dit, il serait prudent de guider les spécialistes et les gestionnaires du risque quant à la façon d'utiliser les hypothèses d'évaluation du risque pour favoriser une meilleure continuité entre l'évaluation du risque et la gestion du risque.

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## 1.0 INTRODUCTION

This report provides the results of a two-phased study conducted for the Canada Mortgage and Housing Corporation (CMHC) to examine the practices and variability amongst practitioners of contaminated sites risk assessment in Canada. Golder Associates Ltd. (Golder) was retained by CMHC to design and conduct both phases of the study. Phase I consisted of a survey of practitioners in the private and regulatory sectors and Phase II consisted of the round robin study. Phase I was previously reported and is appended in the present document for reference.

At the federal level and at virtually all provincial levels in Canada, a risk assessment/risk management approach is permitted for management of contaminated sites. Risk assessment is recognized as being the tool or process whereby insight is gained respecting human health risks and is distinct from risk management (Health Canada, unpublished). This insight is communicated by the risk assessor to those involved in the risk management decision and, together with other considerations (e.g., local regulatory policies, stakeholder input, etc.), options are weighed and a decision rendered on the extent, if any, of remedial actions that are appropriate for the site.

A key issue associated with any health risk assessment is the level of uncertainty that exists in the assumptions and consequently the ultimate risk estimate. Uncertainty may arise as a consequence of incomplete information about the exposure scenario or through natural variability of the parameters used in the computation of the risk estimate. It follows that uncertainty can lead to differing risk estimates for a given site if risk assessors employ differing assumptions or data analysis techniques. The potential ramifications of this variability is that, theoretically, different risk management decisions could be rendered for the same site simply as a result of different risk assessments.

This latter issue has important ramifications on land value, business decisions and expenditures associated with remediation of the site. For example, a site may be considered to present acceptable health risks following assessment by one team, while a similar site/circumstances elsewhere in Canada is concluded to have unacceptable health risks by a different team. In reality the two sites may not differ substantially, yet there is potential for significantly different remedial actions and expenditures.

As the number of risk assessment/risk management projects is increasing together with professional practitioners, it is of interest to examine the variability amongst practitioners and gain insight as to what the major determinants are of the variability. This understanding could then assist in optimizing both the discipline and risk management process in Canada. The present study was designed to explore these issues by employing a round robin risk assessment of a hypothetical case study of a contaminated site.

## 2.0 BACKGROUND

### 2.1 Contaminated Sites Risk Assessment in the Canadian Private Sector

The current study examining human health risk assessment practices in Canada was structured in two phases. Phase I consisted of a survey of practitioners in the private and regulatory sectors. The intent of the private sector survey was to characterize the capabilities and experience of private firms engaged in human health risk assessment across Canada. Insight gained from the private sector survey was used to assist in the selection of participants for the round robin study which comprised Phase II. The intent of the regulatory survey was to gain insight on a regional basis with regards to regulator experience and acceptance of human health risk assessment of contaminated sites. Appendix I contains the technical memorandum which discusses the results of Phase I.

The results of the private sector survey suggest that practitioners have broad expertise in relevant disciplines such as toxicology, biology, environmental engineering, chemistry, and hydrology. Many of the firms supplement their in-house capabilities with external consultants. Many of the firms also have a high level of expertise modelling the fate of contaminants in soil gas, groundwater, fugitive dust and air.

The results of the regulatory survey indicate that governmental agencies support the use of human health risk assessment in the management of contaminated sites. However, at present the approach is generally based on informal policy and not formally regulated.

There appears to be a high level of variability in terms of private sector expertise and technical capabilities as well as regulatory experience and policy in the field of human health risk assessment. Based on this assessment, we would expect variability amongst

firms in both the type of risk assessments performed in Canada and their accompanying risk estimates.

In order to further understand and characterize the variability in risk estimates produced by different practitioners, Golder/CMHC undertook Phase II of the risk assessment study which is reported here. As part of Phase II, nine Canadian practitioners from various regions in Canada with varying levels of expertise performed a screening level risk assessment of a hypothetical case study. The purpose of the Phase II study was to assess the degree of variability in risk estimates among participants, and analyze the sources of variability and uncertainty. To this end, the case study employed in this round robin was not designed to have any "correct" answer.

This project does not purport to assess the acceptability of the participants' performance, and to this end all results are presented in a way to preserve participant anonymity.

## 2.2 Risk Assessment Principles, Variability and Uncertainty

All risk assessments have a component of uncertainty and variability associated with them. Uncertainty may arise from numerous sources and at various stages of the process. The magnitude of uncertainty will be governed to a large extent by the assumptions imposed by the assessor. This section provides a brief background to basic risk assessment principles and the sources of uncertainty in a risk estimate for the purposes of understanding the logic behind conducting a round robin risk assessment.

In order for chemicals to pose a risk, the following elements must be present:

- presence of a chemical at a potentially hazardous concentration;
- a mechanism of release to the environment;
- an exposure pathway through or in environmental media, such as air, soil, surface water, groundwater, or biota;
- a route of uptake, such as inhalation, trans-dermal absorption or ingestion; and
- a receptor (in this case humans) that can be exposed to the chemicals.

These may be conceptualized in an influence diagram, illustrated in Figure 1. The influence diagram depicts what factors are influenced by those below, not how they are influenced. Therefore, the health risk attributable to a chemical released from the site (Box 1) can be influenced by numerous factors as described below.

### 2.2.1 Risk Estimation and Toxicity Reference Values

Each of the contributors to a health risk is a function of dose (Figure 1, Box 2) and toxic potency (Box 3). *Toxicity reference values* (TRVs) reflect the toxic potency of chemicals and are typically presented as a *slope factor* (q*, $(mg/kg \cdot d)^{-1}$) for non-threshold (genotoxic) carcinogens or a *reference dose* (RfD, (mg/kg·d)) for threshold toxicants.

The *hazard quotient* (exposure ratio) is the conventional parameter employed for characterizing human health risks for contaminants which demonstrate threshold effects (non-carcinogenic chemicals and non-genotoxic carcinogens). The hazard quotient provides a basis by which to judge the acceptability or unacceptability of health risks by comparing the hazard quotient to a value of unity. The hazard quotient is calculated as follows:

$$\text{Hazard Quotient (unitless)} = \frac{\text{Dose Rate (mg/kg-day)}}{\text{Reference Dose (mg/kg-day)}}$$

A hazard quotient which exceeds unity is generally regarded as being indicative of an unacceptable exposure scenario which may potentially result in health effects (i.e., estimated exposure exceeds the accepted safe toxicity reference value). Conversely, a hazard quotient less than unity is generally regarded as being indicative of an acceptable exposure scenario (i.e., estimated exposure does not exceed the toxicity reference value). A *hazard index* is the sum of the hazard quotients calculated for exposure pathways of concern for each chemical and for chemicals with similar modes of action.

For contaminants which demonstrate non-threshold effects (i.e., genotoxic carcinogenic chemicals), incremental lifetime cancer risk (ILCR) is calculated by multiplying the dose rate (calculated over an averaging time) by the slope factor identified from carcinogenicity or epidemiological studies. The upper bound of acceptable lifetime cancer risks for a

residential scenario is generally one in a million ($1 \times 10^{-6}$) although this may vary among jurisdictions. The ILCR is calculated as follows:

ILCR (dimensionless probability) = dose rate x slope factor

In general, both the reference dose (RfD) and slope factor (SF or q*) are defined by regulatory agencies. Thus, there is less likelihood for these parameters to vary amongst assessors unless:

i. the assessor wishes to update the parameter based on new information; and

ii. different toxicity reference values exist for different receptors (e.g., lead for children vs. adults).

2.2.2 Dose Calculations

Dose is influenced by, or is a function of the concentration in the exposure medium (Figure 1, Box 4) and receptor characteristics (Box 5).

The concentration in the exposure medium represents the concentration of chemical in water, soil, or air which may be measured directly or estimated using models such as fugitive dust or soil vapour models. These models require a number of input parameters which may be generic or site-specific.

*Receptor characteristics* include physical characteristics (i.e., body weight, skin surface area, bioavailability, and ingestion or inhalation rates etc.) and characteristics which influence exposure (i.e., duration, frequency and rate of contact with chemicals), as illustrated in Figure 1 (Boxes 6-11).

The basic exposure equations used by the participants to calculate incidental soil ingestion, dermal contact with soils, and inhalation of fugitive dust and organic vapours were based on the US EPA (1991) approach which are summarized in the following.

Inhalation - Fugitive Dust

$$\text{Dose Rate}_{inh} = \frac{IR_{inh} \times ET \times C_{FD} \times C_{C\text{-}FD} \times UCF_1 \times EF \times ED \times BF}{BW \times AT \times UCF_2}$$

where:

$\text{Dose Rate}_{inh}$ = dose rate via inhalation of fugitive dust (mg/kg-day);
$IR_{inh}$ = inhalation rate ($m^3$/hr);
ET = exposure time outside (hr/day);
$C_{FD}$ = concentration of fugitive dust in air ($mg/m^3$);
$C_{C\text{-}FD}$ = concentration of contaminant in fugitive dust (mg/kg);
$UCF_1$ = unit conversion factor to convert mg of dust to kg of dust;
EF = exposure frequency (day/year);
ED = exposure duration (year);
BF = bioavailability factor (unitless);
BW = body weight (kg);
AT = averaging time (year);
$UCF_2$ = unit conversion factor to convert years to days.

Note that for each of these exposure the chemical concentration in the exposure media must either be measured directly or estimated by predictive models, both of which will introduce further variables and uncertainty to the overall equation.

Inhalation - Organic Vapours

$$\text{Dose Rate}_{inh} = \frac{IR_{inh} \times ET \times C_{AIR} \times EF \times ED \times BF}{BW \times AT \times UCF_1}$$

where:

$\text{Dose Rate}_{inh}$ = dose rate via inhalation of fugitive dust (mg/kg-day);
$IR_{inh}$ = inhalation rate ($m^3$/hr);
ET = exposure time outside (hr/day);
$C_{AIR}$ = concentration of organic vapours in air ($mg/m^3$);
EF = exposure frequency (day/year);
ED = exposure duration (year);
BF = bioavailability factor (unitless);
BW = body weight (kg);
AT = averaging time (year);
$UCF_1$ = unit conversion factor to convert years to days.

Ingestion of Soil (analogous to equations for water and food intake)

$$\text{Dose Rate}_{ing} = \frac{IR_{ing} \times C_s \times EF \times ED \times BF \times UCF_1 \times FI}{BW \times AT \times UCF_2}$$

where:

$\text{Dose Rate}_{ing}$ = dose rate via soil ingestion (mg/kg-day);
$C_s$ = contaminant concentration in soil(dust) (mg/kg);
$IR_{ing}$ = soil ingestion rate (mg/day);
EF = exposure frequency (day/year);
ED = exposure duration (year);
BF = bioavailability factor (unitless);
$UCF_1$ = unit conversion factor to convert mg soil to kg soil;
FI = fraction of daily soil ingestion derived from site (1.0, conservative);
BW = body weight of average adult worker (kg);
AT = averaging time (year).
$UCF_2$ = unit conversion factor to convert years to days.

Dermal Absorption of Soil

$$\text{Dose Rate}_d = \frac{C_s \times SDAF \times SA \times BF \times EF \times ED \times UCF_1 \times FI}{BW \times AT \times UCF_2}$$

where all variables as noted for soil ingestion apply, and:

Dose Rated = dose rate via dermal contact (mg/kg-day);

SDAF = soil/dust adherence factor ($mg/m^2$-day);

SA = body surface area exposed ($m^2$);

BF = bioavailability factor (unitless);

FI = fraction of daily dermal contact derived from site.

Note that for each of these exposure equations the chemical concentration in the exposure media must either be measured directly or estimated by predictive models, both of which will introduce further variables and uncertainty to the overall equation.

### 2.2.3 Environmental Fate Model Calculations

Environmental fate models (Figure 1, Box 12), which are often used to predict chemical concentrations in exposure media will also influence the variability in dose and risk computations. Examples of environmental fate models typically include prediction of fugitive dust in air, soil gas infiltration to indoor/outdoor air, and groundwater transport models. Fugitive dust models are used to predict chemical concentrations in airborne dust particles based on wind erosion on chemical concentrations in the soil. Soil gas infiltration models predict chemical concentrations in buildings based on chemical diffusive or advective flux rates and concentrations in soil or groundwater. Groundwater transport models predict the movement and concentrations of chemical in groundwater.

The models may vary considerably in complexity ranging from simple empirical relationships based on observed data to complex mechanistic models. Simple models typically require only a few input parameters while complex models may require numerous site-specific parameters. For instance, input parameters required by complex soil gas

infiltration models typically include building characteristics such as number of air exchanges per hour, building under-pressurization, floor crack spacing and width as well as soil characteristics (i.e., depth to contamination, moisture content, organic carbon content, etc.).

Since environmental fate models vary considerably in terms of their structure and complexity, there is no generic approach for predicting chemical concentrations in particular exposure media. It should be recognized that while models serve as predictive tools, they are seldom correct. Rather, they offer insight to the scenario of interest and must be carefully interpreted. Thus, the actual selection of a model, whether simple or complex will introduce a component of uncertainty based on the model's degree of deviation from the true system it is attempting to simulate. Factors which influence a fate/transport model include the source concentration, natural variability or stochasticity of input parameters, and the uncertainty in input parameters caused by incomplete information.

#### 2.2.4 Sources of Variability

Figure 1 infers that variability in risk estimates between participants of the Round Robin can be attributed to variability in dose rates and toxicity reference values employed by all the participants (Tier I). In addition, variability in dose rates will be influenced by variability in receptor characteristics and chemical concentrations in the exposure medium (either measured or predicted) (Tier II). Finally, variability in predicted chemical concentrations in the exposure medium will be influenced by differences in the types of environmental fate models and input parameters used to perform the calculations (Tier III).

## 3.0 METHODOLOGY

### 3.1 Overview of Case Study

A hypothetical case study was designed which was provided to nine participants. The hypothetical case study consisted of a residential housing development proposed on former industrial lands and in this respect is reflective of a "brownfields development". The developer and regulators have hired consultants (each of the nine participants) to

assess the potential human health risks to future residents. Participants were instructed to assume that the potential risk to workers had already been addressed in a separate risk assessment and was not part of the present scope of work. Details of the case study are provided in Appendix II which contains the documentation distributed to the participants.

The site was located on former industrial lands occupied by several different industries over the past 60-70 years. The site was located in a suburban area, was approximately 8 hectares in size and had been cleared of buildings and other structures. It was rectangular in shape, bounded on all four sides by paved roads, and adjacent properties were commercially developed. Several metals (cadmium, copper, lead, and zinc), benzene, and vinyl chloride were detected on the site. Elevated cadmium, copper, lead and zinc were measured in surface soils, elevated zinc and benzene in subsurface soils (3.0-3.5 m depth), and vinyl chloride in groundwater.

In order to provide sufficient information for the data analysis phase and to reduce bias in the results, the case study was designed and implemented in the following manner:

1. All participants were given the same case study and instructions.

2. The case study provided both descriptive and quantitative details of the site and proposed residential development. A core set of raw data relevant to the site was provided for participants to analyze as they considered appropriate. To the extent possible, the round robin was designed to introduce "real world" variability for participants to deal with accordingly.

3. The participants were instructed to focus their efforts on numerical risk calculations rather than other non-quantitative information. Nevertheless, the participants were given the opportunity to provide comments on methods to further refine risk calculations, mitigative measures, and other recommendations.

4. In order to minimize potential bias in the results, an attempt was made to help ensure that the level of effort was consistent amongst the various practitioners. Participants were instructed to perform a "preliminary risk assessment" with limited time and resources to allow developers to evaluate options at an early stage of the project. Participants were allocated a fixed sum of money and approximately 8 days (whichever was least constraining) to analyze the case study and provide numerical risk estimates for each exposure scenario identified by the

participants, and rationale and/or numerical assumptions supporting the calculation of the risk estimates.

5. Pre-formatted generic reporting forms were provided to ensure that the information required by Golder/CMHC for the data analysis phase was received. These forms were designed to facilitate the documentation of risk estimates, computational methods, and numerical assumptions.

6. To foster real world regional variability into the study, the participants were instructed to abide with the relevant polices of their home province, and apply appropriate criteria, guidelines, and methodologies.

7. CMHC and Golder were available for limited consultation to clarify ambiguities and/or provide sources for further information. However, technical guidance was not provided to any of the participants.

8. Although Golder was also a participant in the risk assessment, the case study was performed "blind" by personnel not involved in the overall project. No technical assistance or other information which could compromise the study were provided to individuals completing the risk assessment.

## 3.2 Selection of Participants

A total of ten participants were originally selected to participate in the round robin risk assessment. One participant withdrew and, therefore, only nine participants comprised the final group. The participants were selected based on geographic location and apparent risk assessment experience and capabilities determined by the results of Phase I of the study. Phase I of the study included a private sector survey with the intent to characterize the capabilities and experience of private firms across Canada (see Appendix I).

The experience and technical capabilities of the various firms which participated in Phase I of the study were ranked based on scores corresponding to questionnaire results. The questionnaire provided qualitative information on in-house capabilities, level of experience in various types of risk assessment, and technical capabilities in exposure assessment modelling, toxicity assessment, risk characterization, and risk management. A total score was derived for each firm based on the results of specific questions that were considered most relevant. In order to incorporate variability into the round robin, participants with *varying* apparent capabilities were selected. Four participants with high scores were

selected, three participants with medium scores were selected and two participants with slightly lower scores were selected. Firms with very low scores, reflecting minimal experience and/or capability, were not selected for participation. It is recognized that this selection process in itself may introduce some unknown bias to the study results, however it is believed to have been minimized by selecting a cross section of capabilities.

Broad regional representation was achieved, with representation from British Columbia, the prairie provinces, Quebec and the Maritimes. To ensure anonymity of the participants, only numerical identifiers are used in this report (i.e., Participant #1, 2, 3,....9).

## 3.3 Data Analysis

### 3.3.1 Background

One of the main purposes of the study is to gain insight on which parameters cause the most variability in risk estimates between participants. To accomplish this, a multi-stage or tiered approach was employed to systematically determine the sources of variability. The first level (Tier I) of analysis examines the sources of variability in risk estimates, the second level (Tier II) examines the sources of variability in dose rate estimates and the third level (Tier III) will identify the sources of variability in predicted concentrations in exposure media (Figure 1).

### 3.3.2 Tier I

The first tier included both a qualitative and quantitative component. The qualitative component describes exposure pathways identified and assessed by each participant, modes of toxic action (i.e., carcinogenic or non-carcinogenic), types of receptors (i.e., adult, child, composite, trespasser), and the type of quantitative analysis performed (i.e., stochastic or deterministic approach). The quantitative component includes (i) a description of the magnitude and variability in risk estimates provided for each chemical and exposure pathway, (ii) an analysis of the relationship between the apparent capability of the participants and their final risk estimates, and (iii) an analysis of the sources of variability in the risk estimates. As discussed in Section 2.2.3, any variability in risk estimates can be partitioned to variability in dose rates and toxicity reference values.

In order to determine if risk estimates reflected the experience and/or capabilities of the risk assessors, a linear correlation analysis was performed. Correlations were performed between risk estimates for various contaminant pathways (e.g., dust inhalation and soil ingestion for several contaminants) and the apparent capability of the participants. The apparent capabilities of the firms were assessed in Phase I of the study. Each of the firms were given a score based on their answers to a questionnaire (see Appendix I for details).

Analysis of the variability in the determinants of risk estimates was performed using correlation linear regression analysis. This technique was possible since risk estimates were calculated using standard equations which consider chemical intake rates and the toxicity of the chemical. By using linear regression analysis, it was possible to partition the variability in risk estimates according to variability in chemical intake rates or variability in toxicity reference values.

Statistical analyses were conducted on untransformed data. The variability in risk estimates were performed in steps. The initial step involved performing correlations between risk estimates, chemical intake rates, and toxicity reference values. The results of the correlations indicated whether chemical intake rates and toxicity reference values were co-dependent or colinear. If colinearity existed between the two parameters, additional analyses were not performed. However, if colinearity did not exist between the two parameters, a stepwise regression was conducted. The stepwise regression determined the proportion of variability in risk estimates which can explained by each of the two parameters.

### 3.3.3 Tier II

Tier II consists of a regression analysis of the sources of variability in the dose rates. Variability in the dose rate can be due to variability in receptor characteristics (i.e., breathing or ingestion rate, exposure duration, exposure frequency and duration, body weight, total exposure period, averaging time, chemical bioavailability), and variability in contaminant concentrations in the exposure medium (i.e., chemical concentration in soil, plant material, and air). The relative contributions of these sources to variability in the risk estimates were determined by using a stepwise regression analysis similar to that performed in Tier I.

For quality assurance purposes, data provided by the participants were transcribed into spreadsheets and the dose equations described in Section 2.2.2, employed to re-calculate and validate the dose rates for each of the chemicals and exposure pathways.

In the first step of the Tier II analyses, the variability in dose estimates was partitioned using the collective product of the receptor variables (i.e., defined in Section 2.2.) and either soil concentrations, calculated concentrations of fugitive dust in air, or volatile concentrations in air. Receptor data for each exposure pathway (i.e., ingestion, dermal, and inhalation) were provided by the participants.

The second step of Tier II analyses focused on partitioning dose variability according to individual receptor characteristics (i.e., assumed values for inhalation rate, body weight, exposure duration, etc.). Stepwise regressions were performed on data from several exposure pathways where there were sufficient degrees of freedom.

### 3.3.4 Tier III

Tier III focused on uncertainty introduced by models employed to predict chemical concentrations in exposure media. However, due to minimal replication of any one model, sensitivity analysis using stepwise regression was not possible. Consequently, the analysis was qualitative in nature and focused on describing the environmental fate models used for soil vapour and fugitive dust transport with discussion on inherent conservatism is the models (i.e., model uncertainty).

## 4.0 RESULTS

### 4.1 Tier I

Quantitative analyses were based on exposure pathways considered to be relevant by each participant. Constraints on regression analysis included the limited degrees of freedom due in part by the number of participants selected for study and due to the fact that not all exposure pathways were considered relevant by each participant. Statistical analyses were conducted on data for each exposure pathway where data were available for more than four companies. In some cases it was not possible to reproduce the intermediate calculations provided by the participants. In these cases, statistical analyses were not

performed on this data. For instance, statistical analyses were not performed on data received from participant #1, as a stochastic approach was used and it was not possible to reproduce the intermediate calculations in a deterministic manner. However, although participant #5 also employed a stochastic approach, in this case it was possible to reproduce the intermediate calculations. In addition, participant #6 provided a total dose for indoor and outdoor inhalation of VOCs, but the proportion of dose attributed to either indoor or outdoor exposure was not provided. Therefore, these data were not included in the statistical analysis. A list of the exposure pathways which were amenable to statistical analyses and the number of participants employing a specific pathway/contaminant/ receptor combination are provided in Table 1.

4.1.1 Pathways

The results indicate that the type and number of pathways included in the risk assessment varied between participants. For a particular contaminant source, some of the participants included a large number of exposure pathways while others included only a few (Table 1).

Of the exposure pathways considered for trace metals in surface soils, oral ingestion was the most commonly included pathway. Ingestion of surface soils by children was included as an exposure pathway by six or seven of the nine participants (the actual number depended on the type of chemical) and ingestion by adults was considered by four or five participants. Inhalation of fugitive dust by children was considered by five participants and dust inhalation by adults was considered by two participants. Dermal contact with contaminated soil by children was considered by five participants and dermal contact by adults was considered by three or four participants. Ingestion of home produce contaminated by trace metals was considered by only three participants for children and one participant for adults. Considering that the calculations are complex, time constraints imposed to conduct the preliminary assessment may have limited the number of participants assessing this pathway.

For benzene contamination of subsurface soils, considerable variation was noted in the type and number of exposure pathways assessed by the participants. Indoor and/or outdoor exposure to vapours emanating from the soil were the most common pathways considered. Other exposure pathways considered by certain participants included

ingestion of contaminated soil, dermal contact with chemical, and ingestion of home produce.

For vinyl chloride contamination of groundwater, a total of three pathways were considered (indoor inhalation of vapours, outdoor inhalation of vapours, and dermal contact with the contaminant) while two of the participants did not provide any risk estimates.

### 4.1.2 Receptors and Exposure Scenarios

All participants considered the future (proposed) residential landuse scenario, and additionally, one participant considered a baseline scenario. For the future residential scenario, potential on-site receptors considered by the participants included children, adults or composite receptors (Table 1). Composite receptors were used by two of the participants to estimate risks posed by carcinogenic chemicals present at the site. For the pre-development scenario (baseline), trespassers were considered as potential receptors of concern by one of the participants. None of the participants included off-site receptors.

### 4.1.3 Modes of Toxic Action

The chemicals were either assumed to behave as non-carcinogens (threshold), genotoxic carcinogens (non-threshold), or both (Table 1). All of the participants considered zinc, copper, and lead as threshold toxicants while one of the participants considered lead to also behave as a non-threshold carcinogen. The classification of lead as a non-threshold carcinogen reflects the position held by the U.S. EPA that lead is a probable carcinogen, although this is not a standard view held by Health Canada. One of the participants used a different method to estimate risks to children from lead exposure. They utilized the Integrated Exposure Uptake Biokinetic Model (U.S. EPA method) to estimate the probability that lead levels in blood would exceed 10 ug/dL. For cadmium, participants considered the route of exposure in determining whether it was assumed as a non-carcinogen or carcinogen. For ingestion and dermal contact pathways, cadmium was assumed to behave as a threshold toxicant, while for the dust inhalation pathway, it was considered a carcinogen or assessed for both carcinogenic and non-carcinogenic endpoints.

### 4.1.4 Analytical Approach

Two participants used a stochastic (probabilistic) approach while the remaining participants used a deterministic approach (point-estimate). Deterministic approaches provide a point estimate of risk with no definition of the underlying distribution and limited quantitative understanding of model uncertainty. Stochastic approaches are more complex and provide a distribution of risks and a robust quantification of model uncertainty.

### 4.1.5 Risk Estimates

#### 4.1.5.1 Non-Cancer Risks

*Variability*

Hazard quotients varied considerably between participants for similar exposure scenarios. Table 2 provides a summary of the range and magnitude difference (ratio) between the minimum and maximum risk estimates among participants, by contaminant and exposure pathway. For example, hazard quotients for ingestion of zinc in surface soils ranged from $7.0 \times 10^{-6}$ to $3.3 \times 10^{-2}$ which represents approximately four orders of magnitude difference between minimum and maximum values. It is important to note, however, that this range of difference encompasses consideration of both adult and child receptor; the difference would be smaller if the comparison was constrained to one receptor type. For inhalation of fugitive dust particles containing zinc, hazard quotients ranged from $2.0 \times 10^{-10}$ to $8.3 \times 10^{-1}$ which represents nine orders of magnitude difference between values. High levels of variability were also found for the other chemicals and exposure pathways. The greatest ranges in risk estimates were found for dust inhalation of copper, lead and zinc, with the ratio between maximum and minimum values exceeding one billion.

For surficial metal contamination, the pathway with the highest level of variability was generally the dust inhalation pathway. The only exception was risk estimates for cadmium. For this chemical, the variability in risk estimates was slightly higher for the soil ingestion and dermal contact pathways than the dust inhalation pathway. Figures 2 and 3 graphic examples of the variability in risk estimates for zinc and lead for each

exposure pathway. Additional scatter plots for other non-cancer health risks (i.e., other contaminants) are provided in Appendix III.

For benzene contamination of surface soils, only two participants derived risk estimates based on non-cancer endpoints. Nevertheless, for many exposure pathways, the variability in risk estimates was high (see Figure 4). For instance, hazard quotients for indoor exposure to vapours ranged from $2.9 \times 10^{-4}$ to $2.8 \times 10^{1}$ and for outdoor exposure ranged from $1.2 \times 10^{-5}$ to $5.2 \times 10^{-2}$. The variability in risk estimates for dermal contact with benzene was low because only one participant included this as an exposure pathway. Any variability in risk estimates for this pathway was due entirely to differences in receptor characteristics between children and adults (i.e., body weight, exposure frequency and duration, etc.). The soil ingestion, dust inhalation, and produce ingestion pathways are not shown in Figure 4 since either the pathway was not considered by any of the participants or risk estimates were zero.

For vinyl chloride contamination of groundwater, the variability in risk estimates was relatively low because only one participant considered this pathway, and the variability is due to reporting for two different receptors.

*Acceptability of Risks*

Table 3 shows the number of participants that would conclude acceptable versus unacceptable non-cancer health risks for each pathway and contaminant, based on hazard quotient estimates. In this assessment, hazard quotients exceeding unity were considered unacceptable. If a participant calculated hazard quotients for both children and adults but found that HQs for adults were less than unity while HQs for children were above unity, it was concluded that risks were unacceptable. Figure 5 shows the acceptability or un-acceptability of total risks based on hazard indices for each chemical. The figure shows the number of participants that concluded risks were acceptable or unacceptable for each chemical. It should be noted that some pathways were considered by some participants but not by others. Therefore, the number of pathways considered by a participant could influence whether or not total risks were acceptable or not.

For cadmium, the majority of participants would conclude that risks due to ingestion of soil, dermal contact, and dust inhalation were acceptable, while risks due to ingestion of produce were unacceptable. Based on these results, the majority of participants would conclude that the total risk due to cadmium exposure was unacceptable (5 of 8 participants). Hazard indices for cadmium ranged from $9.9 \times 10^{-2}$ to 13.

For copper, the majority of participants would conclude that risks due to ingestion of soil, dermal contact, and dust inhalation were acceptable, while only half the participants would conclude that risks due to ingestion of produce were acceptable. The majority of participants would conclude that risks due to copper exposure was unacceptable (4 of 7 participants). For copper, hazard indices for future residents ranged from $6.0 \times 10^{-2}$ to $3.2 \times 10^{2}$.

For lead, most participants would conclude that risks due to ingestion of soil and produce were unacceptable, half the participants would conclude that risks due to dermal contact were acceptable, and the most participants would conclude that risks due to dust inhalation were acceptable. Hazard indices ranged from $1.4 \times 10^{-1}$ to $1.4 \times 10^{4}$.

For zinc, most participants would conclude that risks due to ingestion of soil and produce, dermal contact, and dust inhalation were acceptable. Seven out of eight participants would conclude that total risks from all exposure pathways were acceptable. Hazard indices for future residents were generally less than 1 ranging from $4.4 \times 10^{-3}$ to 10.

For benzene, most participants would conclude that risks due to ingestion of soil and produce, dermal contact, dust inhalation, and vapour inhalation were acceptable. Two out of three participants would conclude that total risks from all exposure pathways were acceptable. Hazard indices for future residents were generally less than 1 ranging from $3.0 \times 10^{-4}$ to 28.

For vinyl chloride, the single participant concluded that risks were acceptable from all pathways. Hazard indices ranged from $2.1 \times 10^{-3}$ to $3.5 \times 10^{-3}$.

The participant that considered risks to trespassers estimated hazard indices less than 1 for zinc, copper and cadmium, but greater than 1 for lead.

4.1.5.2 Cancer Risks

*Variability*

Incremental lifetime cancer risk (ILCR) estimates varied considerably between participants (Table 4). For instance, cancer risk estimates for inhalation of dust containing cadmium ranged from $3.0 \times 10^{-14}$ to $3.0 \times 10^{-4}$, risk estimates for indoor inhalation of vapours containing benzene ranged from $9.5 \times 10^{-9}$ to $3.5 \times 10^{-2}$, and risk estimates for indoor inhalation of vapours containing vinyl chloride ranged from $2.2 \times 10^{-9}$ to $2.4 \times 10^{-3}$. For lead contamination of surface soils, variability in cancer risk estimates was low because only one participant considered lead a carcinogenic agent, but different receptors were considered. Figures 6, 7 and 8 graphically display the variability in risk estimates for cadmium, benzene, and vinyl chloride for all exposure pathways. Additional scatter plots for other cancer health risks (i.e., other contaminants) are provided in Appendix III.

*Acceptability of Risks*

Table 5 shows the number of participants that would conclude acceptable versus unacceptable risks for each pathway and contaminant based on incremental lifetime cancer risks (ILCR). For purposes of this report, an ILCR greater than $1 \times 10^{-6}$ was considered unacceptable. Figure 9 shows the acceptability or un-acceptability of total cancer risks for each contaminant. The figure shows the number of participants that concluded risks were acceptable or unacceptable for each chemical.

For cadmium, two of five participants would conclude that the risks associated with dust inhalation were unacceptable. Cancer risks estimates were highly variable ranging from $3.0 \times 10^{-14}$ to $3.0 \times 10^{-4}$.

For lead, the single participant considering cancer risks would conclude that the risks associated with ingestion of soil and dust inhalation were unacceptable. Total cancer risks ranged from $7.0 \times 10^{-5}$ to $2.6 \times 10^{-4}$.

For benzene, virtually all assessors who addressed risks due to ingestion of soil and produce, dermal contact, and outdoor vapour inhalation would conclude these risks were

acceptable. Indoor vapour exposure would be considered unacceptable by 5 of 7 participants. On the basis of total ILCR, five out of eight participants would conclude that total risks from all benzene exposure pathways were unacceptable with total cancer risks ranging from 0 to $7.2 \times 10^{-4}$, three of eight assessors would conclude the risks were acceptable.

For vinyl chloride, the single assessor for this pathway would conclude that risks due to dermal contact were acceptable. The majority of assessors which addressed indoor and outdoor inhalation would conclude that risks were unacceptable. Total cancer risks ranged from $2.2 \times 10^{-9}$ to $2.4 \times 10^{-3}$, and virtually all assessors (five of six) would agree the health risks were unacceptable.

### 4.1.6 Toxic Potency

Most of the participants used similar toxicity reference values (i.e., reference doses and cancer slope factors). For a given chemical and pathway, toxicity reference values generally varied by three orders of magnitude or less (see Tables 6 and 7). Considerable variability in the magnitude of toxicity reference values was also observed, depending on the contaminant and pathway considered.

The greatest variability was seen for threshold toxicological endpoints. In the case of lead reference doses ranged over three orders of magnitude, although this was basically reflective of sensitivities between children and adult. Copper toxicity was relatively consistent for soil ingestion and dermal contact, but differed amongst assessors by five orders of magnitude in the case of dust inhalation. Zinc was also relatively consistent for ingestion of soil and produce, yet differed by three orders of magnitude for dust inhalation and dermal contact.

The range in reference doses was typically highest for the dust inhalation pathway and lowest for the soil ingestion, produce ingestion, and inhalation of organic vapours pathways. The variability in reference doses for the dust inhalation pathway is displayed in Figure 10.

For non-threshold carcinogens, the largest variability amongst assessors in slope factors was associated with dust inhalation of cadmium (a 90-fold difference), and vapour inhalation of vinyl chloride (a 2800 fold difference).

### 4.1.7 Dose Rates

There was considerable variability in dose rate estimates among participants. As examples, Figures 11 and 12 show dose rates provided by the participants for zinc and lead exposure for non-carcinogenic endpoints. Figures 13 and 14 show dose rates for benzene and vinyl chloride exposure for carcinogenic endpoints. For zinc and lead exposure, the lowest variability was found for the ingestion of home produce pathway (0.0038 to 0.67 mg/kg·d for residents) and the highest variability was found for the inhalation of surface soil pathway ($1.6 \times 10^{-12}$ to $1.2 \times 10^{-4}$ mg/kg·d for residents). For benzene and vinyl chloride exposure, the highest variability was found for the indoor inhalation of vapours pathway. Dose rates for benzene ranged from $3.3 \times 10^{-7}$ to 1.2 mg/kg·d for residents, and for vinyl chloride ranged from $7.2 \times 10^{-9}$ to $8.0 \times 10^{-3}$ mg/kg·d for residents. Additional scatter plots for other contaminants are provided in Appendix III.

### 4.1.8 Relative Magnitude of Risk Estimates

Based on the results for soil ingestion and dust inhalation pathways, the participants were ranked based on the relative magnitude of their risk estimates. The ranking was performed to assess whether or not specific companies consistently estimated relatively high or low risks for a specific pathway. Tables 8 and 9 show the results of the ranking procedure for the soil ingestion and dust inhalation pathways for child receptors. Lack of replication for other pathways precluded their analysis. For soil ingestion, participants 3 and 9 consistently calculated relatively high hazard quotients for the four metals, while participants 2 and 8 generally calculated moderate to low hazard quotients. For dust inhalation, participants 3 and 9 generally calculated high hazard quotients, while participants 2 and 5 calculated low hazard quotients.

### 4.1.9 Relationship of Capability Score to Risk Estimates

The results of correlations performed between risk estimates and the apparent capabilities scores of the participants were inconclusive. No significant trends were observed for the soil ingestion and dust inhalation pathways for cadmium, copper, lead and zinc. The highest correlation coefficient (r) was determined for ingestion of copper by adults ($r=0.81$, $n=4$, $0.05<P<0.1$) and the lowest correlation coefficient was determined for ingestion of copper by children ($r=0.0066$, $n=5$, $P>0.5$). The results of the correlation analyses are presented in Appendix III.

### 4.1.10 Regional Trends in Risk Estimates

Based on a visual inspection of the scatter plots for the various risk estimates (i.e., Figures 2, 3, 4, 6, 7, and 8), there were no apparent trends between a participant's home province or region and the magnitude of risk estimates. Although regional differences may explain some of the variability in risk estimates, there contribution appears to be minor. More of the variability is probably explained by differences in risk assessment assumptions (i.e., conservative versus more realistic) which may be driven more by conservatism in professional judgment, rather than region-specific policies/procedures.

### 4.1.11 Sources of Variability in Risk Estimates

Variability in risk estimates can be caused by variability in the dose rates and variability in the toxicity reference values. The Tier I regressions determined the proportion of the variability explained by each of the two components. A summary of the results of the Tier I analysis for zinc and benzene are shown in Figure 15.

For exposure to surficial metal contamination via ingestion and dermal contact, the majority of variability in risk estimates was generally due to variability in dose rates. For these pathways, risk estimates for individual substances and pathways were highly correlated with dose and less so with toxicity reference value. For instance, dose rates for ingestion of copper (adult), lead (adult & child) cadmium (child), and zinc (adult) accounted for 64 to 100% of the variability in risk estimates between participants, and dose rates for dermal contact of copper (child), cadmium (child and adult), and zinc

(adult) accounted for 69 to 97% of the variability in risk estimates. The only exceptions to this general trend were noted for (i) soil ingestion of copper (child), and dermal contact with lead (child) where variability in reference doses accounted for the most of the variability and (ii) soil ingestion of cadmium (adult) for which colinearity existed between the two variables and therefore a stepwise regression was not performed.

For the inhalation of fugitive dust pathways, most of the variability in risk estimates for cadmium (child) and zinc (child) were due to variability in either the RfD or cancer slope factor. For instance, 90% of the variability in risk estimates for cadmium was explained by variability in slope factor while 9% was explained by dose, and 35% of the variability in risk estimates for zinc was explained by variability in RfD while 24% of the variability was explained by dose. For zinc, the use of untransformed data resulted in a large unexplained component for this analysis. The correlations for copper and lead were not significant and stepwise regressions were not performed.

For indoor and outdoor inhalation of benzene, it was not possible to determine the percentage of the risk estimate attributable to the RfD or dose, since the two determinants were colinear.

## 4.2 Tier II

The Tier II regressions determined the proportion of the variability in dose estimates explained by either receptor characteristics or chemical concentrations in the exposure medium. A summary of the results of the Tier II analysis for zinc and benzene are also shown in Figure 15.

For exposure to surficial metal contamination via ingestion and dermal contact, the majority of variability in dose estimates was generally due to variability in receptor characteristics. For ingestion of zinc (child), cadmium (adult), copper (adult), and lead (adult & child), receptor characteristics accounted for 53 to 86% of the variability in dose rates. The only exception was ingestion of cadmium by children, where variability in the soil concentrations accounted for 50% of the variability in dose rates and receptor characteristics accounted for only 26% of the variability. For dermal contact with lead (child), cadmium (child and adult), and zinc (adult), receptor characteristics accounted for

80 to 99% of the variability in dose rates. The correlation performed on copper ingestion (child) and dermal contact with cadmium (child) were not significant.

For the inhalation of fugitive dust pathways, most of the variability in dose estimates for cadmium (child), copper (child), and zinc (child) were due to variability in predicted chemical concentrations in the air. Variability in air concentrations accounted for between 58 and 97% of the variability in dose estimates. The only exception to this trend was noted for lead exposure by children where receptor characteristics accounted for 71% of the variability and air concentrations accounted for only 7% of the variability.

For benzene exposure, both receptor characteristics and predicted chemical concentrations in air accounted for much of the variability in dose estimates. For indoor exposure, most of the variability was due to chemical concentrations in air (72%) while only 26% of the variability was explained by receptor characteristics. For outdoor exposure, most of the variability was due to variability in receptor characteristics (82%) while only 8.6% of the variability was explained by air concentrations.

A few of the correlations allowed for analysis of variability of dose estimates due to individual receptor characteristics. For the ingestion of zinc in soil pathway, the exposure frequency (82%) and ingestion rate (17%) were the major contributors to the dose rate.

## 4.3 Tier III Model Variability

### 4.3.1 Environmental Fate And Transport For Dust Inhalation Pathway

Environmental fate and transport modeling is required to predict potential dust generation and outdoor inhalation exposure based on measured metal concentrations in surface soil. Six of the nine participants estimated outdoor dust concentrations. The remaining three participants indicated that dust generation would not be a concern since ground would be either covered with vegetation or asphaltic paving therefore rendering this pathway insignificant. One of the three participants (#8) indicated that the dust inhalation pathway was not considered since dust is considered to be "a negligible exposure pathway for a residential project by provincial (i.e., Quebec) authorities". The models and input parameters used are further described below.

#### 4.3.1.1 Description of Models Used

The models used to predict outdoor dust concentrations are summarized in Table 10. Four participants (#2, 3, 4 and 7) used a two-component model consisting of (i) prediction of dust generation through wind erosion and (ii) prediction of exposure concentrations in air through atmospheric mixing of dust. One participant (#1) used an empirical approach based on typical dust measurements while one participant (#9) did not document the method used.

The model used by three participants (#2, 3 and 7) to predict dust generation consisted of the Cowherd rapid assessment model as referenced in Cowherd et al. (1985) and ASTM ES-1739-95. The particulate emission rate used by participants #2 and 7 was a generic default rate of 6.9 E-14 g/cm$^2$-sec provided in ASTM ES-1739-95 (the rate used by the third participant was not documented). The equation used to obtain this default emission rate value was not provided by any of the participants; however, one participant (#2) described the assumptions inherent in obtaining the particulate emission rate as follows:

- the mode of the surficial soil was 2 mm;
- the erosion potential is unlimited with no vegetative cover;
- the mean average wind speed was 4 m/sec;
- the site is uniformly contaminated with the concentration in respirable particulates matching the bulk contaminant concentration in surface soil; and
- emissions are assumed to be continuous and steady.

Based on the above information, it appears that the particulate emission rate model used is by Cowherd et al. (1985) for surfaces with unlimited erosion potential as represented by the following equation:

$$E_{10} = 0.036(1-V)\left(\frac{U_m}{U_t}\right)^3 F(x) \qquad \text{Eq. 1}$$

where $E_{10}$ = $PM_{10}$ emission factor i.e., annual average $PM_{10}$ emission rate per unit area of contaminated surface ($g/m^2$-hr)
0.036 = respirable fraction
V = fraction of contaminated surface vegetative cover (equals 0 for bare soil)
$U_m$ = mean annual wind speed (m/s)
x = 0.886 $U_t/U_m$ = dimensionless ratio
$U_t$ = erosion threshold wind speed at 7 m (m/s)
F(x) = function dependent on $U_m$ and $U_t$

It is noted the same particulate emission model is used in U.S. EPA (1991).

One participant (# 4) used a model for wind erosion from surfaces with limited erosion potential developed by Cowherd et al. (1985) as incorporated in the API-DSS software package. The following equation is used:

$$E_{10} = \frac{0.83\ f\ A\ P\ (u+)(1-V)}{\left(\frac{PE}{50}\right)^2} \qquad \text{Eq. 2}$$

where $E_{10}$ = annual average emission rate of particles less than 10 um in diameter (mg/hr)
f = frequency of disturbance per month ($mo^{-1}$)
A = area of contaminated soils ($m^2$)
P(u+) = 6.7 (u+ - $U_t$)
u+ = fastest mile speed (m/s)
$U_t$ = erosion threshold wind speed at 7 meters height (m/s)
PE = Thornthwaite's Precipitation Evaporation Index

Three participants used a box model to estimate a "volatization" factor which is used in the dose estimation equations. The volatization factor method is presented in ASTM ES-1739-95 and utilizes the following equation:

$$VF_p = \frac{P_e \times W}{UH} \times 10^3 \frac{cm^3 \cdot kg}{m^3 \cdot g} \qquad \text{Eq. 3}$$

where $VF_p$ = volatilization factor for dust
$P_e$ = particulate emission rate ($E_{10}$) ($g/cm^2$-sec)
W = width of the source parallel to the wind direction (cm)

U = wind speed (cm/s)
H = height of mixing zone (cm)

One participant (#3) used a Gaussian dispersion model (Screen 3) developed by the U.S. EPA (1985).

One participant (#1) used an empirical method to estimate dust concentrations based on a "typical" background outside dust level of 35 $\mu g/m^3$, and the assumption that 50 percent of the background dust (i.e., 17 $\mu g/m^3$) originates from the contaminated soil (Hawley, 1985).

#### 4.3.1.2 Description of Input Parameters Used

Selected model input parameters, as well as predicted exposure concentrations for one metal (cadmium) chosen as an example, are presented in Table 11. As shown, there is a significant range in predicted concentrations (about nine orders-of-magnitude). The exposure concentration calculations were not checked since for several participants, insufficient information was provided to enable checking of model equations.

The two parameters that showed the greatest variation were the particulate emission rate and width of the site for the box model. Strictly speaking the particulate emission rate is not an input parameter; nevertheless, it was included since participants did not indicate how the rate was calculated. The particulate emission rate varied over five orders-of-magnitude with the higher rate estimated using the Cowherd model which assumes limited erosion potential. The width of the site used varied from 1 m to 283 m. The site width is directly proportional to the volatilization factor and therefore a larger width will correspond to a higher exposure concentration. Several participants indicated the rationale for using a small width is that most of the site area will be covered with vegetation therefore reducing dust generation potential.

### 4.3.2 Environmental Fate And Transport For Soil Gas VOC Building Intrusion Pathway

Environmental fate and transport modeling is required to predict potential soil gas intrusion of VOC buildings and resulting inhalation exposure, based on measured benzene concentrations in soil, and vinyl chloride concentrations in groundwater. Seven of the nine

participants estimated indoor exposure concentrations. One participant (#3) included output data for a model (CalTOX) which appears to include the indoor pathway, but no indoor exposure concentration was reported. One participant (#4) did not address the indoor pathway since, in their consideration, the use of geomembrane vapour barriers (6 mil polyethylene) typically used for foundation construction would mitigate soil gas intrusion to non-significant levels.

The focus of the model and input parameter evaluation is benzene (soil-to-air pathway) since concepts are largely similar for vinyl chloride. The models and input parameters used are further described below.

#### 4.3.2.1 Description of Models Used

The models used to predict indoor exposure concentrations resulting from benzene soil contamination (i.e., soil-to-indoor-air pathway) are summarized in Table 12. The following observations are made with respect to the models:

1. *Source Depletion*: One participant assumed that benzene biodegradation occurs according to a first order decay function, (a biodegradation rate of 0.007 $day^{-1}$). The average soil benzene concentration over a 30 year exposure period was input into the exposure calculations. None of the participants incorporated source depletion using either a mass balance approach (i.e., mass depleted equals mass volatilized) or through groundwater infiltration and benzene leaching.

2. *Partitioning:* Six participants assumed that linear equilibrium chemical partitioning between the absorbed, aqueous and gaseous phase occurs. One participant (#8) utilized a semi-empirical method (Hamaker method as referenced in Lyman et al., 1990) to predict benzene mass flux in soil gas based on a measured soil concentration.

3. *Fate and Transport in Soil*: Six participants assumed one-dimensional steady-state upward diffusion of gas-phase and aqueous-phase benzene according to Fick's Law (i.e., chemical gradient). In all cases, a single homogeneous, isotropic soil layer was assumed. One participant (#8) used a semi-empirical method (Hamaker method) to predict benzene mass flux. None of the participants incorporated chemical retardation through biodegradation or adsorption. In all cases, the effective diffusivity was estimated using the Millington-Quirk relationship (Millington and Quirk, 1961).

4. *Fate and Transport Through Building Foundation*: Five participants assumed that one-dimensional upward diffusion occurs through dust-filled cracks; in addition, two participants (#5 and 9) assumed diffusion also occurs through intact concrete. One participant (#8) did not include diffusive mass flux.

   Four participants also assumed that mass flux through advection (i.e., pressure-driven flow) occurs. The pressure gradient is generated as a result of building underpressurization due to temperature differences between outside and indoor air, wind loading and/or mechanical ventilation. Two participants (#5 and 9) assumed that advective gas flow follows Darcy's Law. The concrete slab permeability was estimated based on relationships between fracture porosity and permeability developed for fractured rock (Snow, 1968; Freeze and Cherry, 1979). One participant (#1) used an idealized relationship for flow through a cylinder developed by Nazaroff (1988) while the remaining participant (#8) used an empirical method based on measured air leakage rates for building envelopes (Figley, 1996).

   One participant (#7) assumed that the building envelope provides no resistance to soil gas intrusion.

5. *Building Underpressurization*: Five participants utilized an assumed building underpressurization based on measured values for houses reported in the literature. One participant (#5) estimated the building underpressurization for the heating season (i.e., when there is a significant difference between the outdoor and indoor air temperatures) utilizing a relationship developed by Nazaroff (1992).

6. *Building Air Mixing*: Six of the seven participants assumed infiltrating VOCs are diluted according to a well-mixed single compartment box model. One participant (#7) utilized an outdoor box model, and assumed parameters for wind-speed and mixing height for the indoor case.

It is noted that the participant (#8) who estimated advective flux rates through the building envelope modified the method documented in Figley (1996). The following equation was used by participant #8 to estimate flux (F):

$$F = Q_t \times ELA \times K \qquad \text{Eq. 1}$$

where:
- $Q_t$ = total loss of chemical per unit area over some time t ($g/m^2$-sec)
- $ELA$ = Equivalent leakage area ($m^2$)
- $K$ = conversion factor (1E06 µg/g x 3600 sec/hr.)

$Q_t$ was obtained using the Hamaker method. The Figley (1996) method utilizes the following equations to estimate flux (F):

$$F = Qs \times C_{sg} \qquad \text{Eq. 2}$$

$$Q_s = 3.6\left(C \times \Delta P^n\right) \qquad \text{Eq. 3}$$

$$C = \frac{ELA}{\left\{0.0001157(\rho)^{0.5} \times 10^{n-0.5}\right\}} \qquad \text{Eq. 4}$$

where:
- $Q_s$ = soil gas flow rate ($m^3/hr$)
- $C_{sg}$ = average contaminant concentration in soil gas ($mg/m^3$)
- $C$ = gas flow coefficient ($L/s \cdot Pa^n$)
- $\Delta P$ = pressure difference ($Pa$)
- $n$ = flow coefficient (dimensionless)
- $\rho$ = soil gas density ($kg/m^3$)

4.3.2.2 Description of Input Parameters Used

Selected model input parameters, as well as predicted benzene exposure concentrations are presented in Table 13. As shown, there is a significant range in predicted concentrations (about five orders-of-magnitude). The exposure concentration calculations were not checked since for several participants, insufficient information was provided to enable checking of model equations. The following observations are made with respect to the input parameters.

1. *Soil Concentration*: Three participants used the maximum benzene concentration, one participant used the arithmetic mean while one participant used the 95th percentile concentration.

2. *Depth to Contamination*: The depth to contamination used ranged from 0.9 m (participant #7) to 3.0 m (participant #5). Based on the information provided for the case study, a depth of 1.0 m would be appropriate for the initial depth to contamination.

3. *Fraction Cracks to Total Foundation Area ($\eta$)*: Assumed values for this input parameter varied significantly (5.85E-05 for participant #8 to 0.01 for participant #2). The assumed value used by participant #8 is based on values

proposed by Figley (1996). It is noted that the equivalent leakage area is an *equivalent* leakage area and not equal to the physical leakage area. Information sources used to derive $\eta$ were poorly documented.

4. *Building Air Exchanges*: The air exchanges per hour ranged from 0.375 (#8) to 1.0 used by several participants. An air exchange rate of 0.375 is based on typical values proposed by Figley (1996) for ventilation rates for new Canadian houses in the Prairie Provinces.

5. *Building Underpressurization*: Three participants utilized an assumed underpressurization based on values published in the literature. One participant (#5) calculated the underpressurization for the heating season using a relationship developed by Nazaroff (1992). Using the average temperature difference provided by the participant (16.1 degrees Celsius), an underpressurization of approximately 2.4 Pa is obtained. The length of the heating season assumed by participant #5 is 7 months.

6. *Height of Building Mixing Zone*: Assumed values for this parameter ranged from 2.3 m (participants #9 and 2) to 6.9 m (#5). The low value conservatively assumes that mixing is limited to the basement and that the building ventilation system is not connected throughout the house.

For participant #8, it is noted that the predicted indoor concentration is equal to the outdoor concentration indicating that potential subsurface benzene intrusion had no effect on the indoor air quality.

## 5.0 DISCUSSION

### 5.1 Sources of Variability in Risk Estimates

The results of the round robin risk assessment indicate that the participants vary considerably in their approach to performing screening level human health risk assessments at contaminated sites. This results in risk estimates that differ over a considerable range for various chemical exposure pathways. The high variability in risk estimates was due to a combination of factors including differences in the assumed chemical toxicity, receptor characteristics, and differences in model type and assumptions used to predict vapour and dust concentrations in air. At a more fundamental level, difference existed amongst participants in terms of which pathways did or did not warrant consideration.

In general, most of the variability in risk estimates was due to variability in dose rates and not chemical toxicity. Nevertheless, in many cases toxicity reference values ranged over three orders of magnitude and for one of the pathways ranged over five orders of magnitude (i.e., inhalation of dust containing copper). The variation in toxicity reference values was likely due to differences in the source of the toxicity reference values and the type of adjustments made to toxicity values for the dust inhalation pathway. For instance, reference doses for copper (dermal pathway) were taken from various sources including Health Canada, provincial documents, the IRIS database developed by the U.S. Environmental Protection Agency, and journal articles. In some cases, TRVs for dust inhalation incorporated receptor specific data (i.e., inhalation rate and body weight which may vary between children and adults) to convert unit risks based on chemical concentrations in air ($mg/m^3$) to reference doses (mg/kg body weight/day). Considering that toxicity is a fixed and intrinsic characteristic of a chemical, the moderate to high variability in toxicity reference values utilized by the participants is notable because it could influence the overall outcome and conclusions of a risk assessment. This aspect of health risk assessment should be scrutinized carefully by the team's toxicologist. Uncertainty may arise when regulatory toxicity values (e.g., RfDs) and somewhat dated, or conversion of unit risk value to slope factors is conducted.

The source of variability in dose rate estimates varied depending on the complexity of the dose rate calculations. For instance, for the soil ingestion and dermal pathway which does not require environmental fate modelling to predict exposure concentrations, the source of variability in dose rates was generally differences in receptor characteristics such as body weight, exposure frequency and duration, inhalation or ingestion rates etc. For the fugitive dust and indoor vapour inhalation pathways which require complex modelling to estimate chemical concentrations in the air, the source of variability was generally differences in predicted chemical concentrations in air rather than receptor characteristics. Many types of models were used to predict dust and vapour concentrations, and the models were parameterized using both generic and site specific values. The variety of techniques and assumptions used to model fugitive dust and indoor vapour concentrations is discussed in more detail in section 5.1.3.

An interesting observation concerning analysis of the dose rates was the variation amongst the determinants of the dose. While exposure frequency (EF) was often seen as the

dominant factor, other determinants were found to be intercorrelated. This suggest that as assessors tended to use larger values for EF, they also tended concomitantly increase other exposure factors such as exposure duration, and intake rates (e.g., ingestion or breathing rates) and source concentration. There is no *a priori* why this should occur, and the observation suggests that at least some assessors are instilling conservatism across all (or most of) the dose parameters rather than applying conservatism to selected parameter. This "blanket conservatism" propagates considerable uncertainty and lack of realism in the final risk estimate.

As discussed in Section 4.1.9 and 4.1.10, there were no apparent trends between the magnitude of risk estimates and the apparent capabilities of the participants or between the magnitude of risk estimates and the home province or region of the participants. The lack of apparent trends could be due to the small sample size and due to the fact that the apparent capability of the participants or home region had less influence on the risk assessment results than the type of assumptions (conservative or non-conservative) and/or risk assessment techniques employed. This conclusion is supported by the results of the ranking of participants based on relative magnitude of risk estimates (Section 4.1.8). The results of the ranking procedure indicate that certain participants tended to estimate high risks while other estimated low risks for a variety of chemicals and exposure pathways.

An important consequence of the high variability is that the proportion of firms concluding acceptable versus non-acceptable risks would be highly dependent on the magnitude of soil concentrations provided in the case study. For example, based on the zinc concentrations provided in the case study, all of the participants would conclude that risks for the soil ingestion pathway were acceptable since hazard quotients were consistently below unity. However, if the zinc concentrations in soil were approximately three orders of magnitude higher, only half of the participants would conclude risks were acceptable, and if concentrations were approximately five orders of magnitude higher, all of the participants would conclude risks were unacceptable. The actual variability in risk estimates is expected to remain the same regardless of the absolute chemical concentration at the site.

Finally examination of the scatter on risk estimates for different pathways, indicates greater variability is present for the more complex pathways (e.g., fugitive dust inhalation

and indoor infiltration of soil vapour) versus the simpler direct pathways (e.g., soil ingestion, dermal contact). One may speculate that as greater paramertization of the exposure pathway occurs, the opportunity to assign conservative/non-conservative assumptions leads to greater variability amongst assessors. In order to prevent undue variability in risk estimates, a consistent approach should be considered.

## 5.2 Environmental Fate Modelling

### 5.2.1 Dust Inhalation Pathway

Most participants used a model which incorporates Cowherd's rapid assessment method (Cowherd, 1985) with a simple ambient air box model. Of concern is the significant difference (five orders-of-magnitude) in the particulate emission factor estimated using the limited erosion model and the assumed value based on the unlimited erosion model. The results were also unusual in that the results for the limited erosion model were greater than those for the unlimited erosion model which one would assume to be more conservative.

To further assess the particulate emission rates used, rates were estimated assuming Cowherd's unlimited erosion potential for a range of particle modes and the following assumptions:

$\mu$ = mean annual windspeed = 4.4 m/s
V = fraction vegetative cover = 0
$Z_o$ = roughness height = 5.0 cm and 50 cm (default values provided in Cowherd et al. (1985) for suburban residential dwellings, and suburban institutional buildings)

Using the above assumptions, the particulate emission factors versus particle mode is plotted in Figure 16. As shown, the results are highly sensitive to the particle mode. For most typical particle modes (i.e., clay to sand size), the default particulate emission rate of 6.9E-14 g/cm$^2$-sec referenced in ASTM ES-1739-95 would not be conservative based on the results in Figure 16.

A key implication arising from the study is that the models used to predict wind generated dust emissions are highly dependent on input parameters such as soil type, vegetative cover and size of the site. Therefore, it is important for screening-level risk assessments to

use appropriate site-specific data. In terms of the air mixing model, a simple box model is considered appropriate for a screening level risk assessment. The use of a dispersion model would be more appropriate for the case where receptors are removed some distance from the source.

### 5.2.2 Environmental Fate and Transport for Soil Gas VOC Outdoor Pathway

Environmental fate and transport modelling is required to predict volatilization, soil transport and ambient air exposure based on measured benzene concentrations in soil and vinyl chloride concentrations in groundwater. Five of the nine participants estimated outdoor air concentrations. Several participants indicated that the rationale for not including the outdoor pathway is that the indoor pathway is the more sensitive pathway (i.e., the outdoor pathway will result in lower predicted exposure concentrations).

The models used and input parameters chosen for soil gas fate and transport, and mixing in air, were identical to the models used for the indoor soil gas pathway. Therefore, a detailed discussion of the results is not repeated here. In most cases, a simple one-dimensional steady-state diffusion model in soil was used with one participant (#8) using an empirical approach (Hamaker method). In terms of mixing in air, most participants used a simple box model with one participant (#3) using a Gaussian dispersion model (Screen 3).

### 5.2.3 Soil Gas Fate

*General*

The modeling approach followed by the majority of the participants is based on the heuristic model developed by Johnson and Ettinger (1991) which has been, for the most, adopted by ASTM E1739-95. The models typically incorporate steady-state diffusion in soil, and diffusion and advection through a concrete building floor slab. The mass flux equations are solved analytically, or semi-analytically using iterative subroutines (e.g., "Solver" routine provided in Microsoft Excel™). The Johnson and Ettinger (1991) model is intended to be a relatively simple screening-level model; nevertheless, it is generally recognized that this model is likely highly conservative in most cases (Sanders and Stern (1994), Jeng et al. (1996), and Hers et al., (1997)).

*Source Depletion, Fate and Transport of Soil Gas*

Mechanisms that increase the realism of predictive models include contaminant source depletion, and biodegradation and adsorption of gas-phase VOCs during upward migration toward the building. Inclusion of source mass depletion provides a useful reality check in terms of the chemical mass that can be volatilized. For example, using the non-depleting steady-state mass flux rate predicted by participant #9, the available benzene would have depleted in about one month. When exposure is assumed to occur over 25 to 30 years, as often is the case for human health risk assessments, it is clear that not including source mass depletion can be extremely conservative.

It is noted that participant #7 assumed that benzene biodegradation occurs at the source (i.e., soil contamination zone). The assumed benzene biodegradation rate (0.007 $day^{-1}$) is a relatively low value based on reported range of degradation rates (five studies) for petroleum hydrocarbons documented in ASTM E1739-95. The reported degradation rates are for dissolved BTEX plumes and may not be applicable to degradation at source where the oxygen and other electron acceptors may be depleted. A more appropriate model would incorporate benzene gas-phase degradation as it migrates towards the ground surface and oxygenated zone.

A recent study by Jeng et al. (1996) demonstrated that model utilizing diffusion and gas-phase biodegradation (adapted from Jury et al., 1990) closely predicted vertical concentration profiles for BTEX. Incorporation of biodegradation was shown to potentially decrease the building air exposure concentrations by several orders-of-magnitude (depending on the soil type and resulting effective diffusivity). Another recent study (Fischer et. al, 1996) indicated that BTEX soil gas concentrations decreased sharply over a small vertical depth interval (0.1 m to 0.7 m below ground surface). The authors suggest that a partial physical barrier to vertical transport in combination with microbial degradation can account for the steep gradient.

*Chemical Partitioning*

Another potentially conservative aspect of the screening-level models typically used may be the partitioning model. In the case where non-aqueous phase liquid (NAPL) is present,

the equilibrium gas-phase concentration should be estimated using the chemical partial pressure (i.e., vapour pressure adjusted using Raoult's Law). Using this model, gas-phase chemical concentrations are constant and do not change with increasing NAPL concentration. Assuming only benzene was present, the benzene concentrations provided for the case study are below the saturation concentration for NAPL. It is noted that none of the participants estimated the benzene soil saturation concentration for NAPL to verify the appropriateness of the partitioning model used. A second partitioning issue is that the assumption of instantaneous equilibrium partitioning may not be appropriate and that there may be mass transfer rate limiting effects (i.e., kinetic effects) (e.g., Gong et al., 1996).

*Soil Gas Building Intrusion*

The case study results show a wide divergence in the models used to predict mass transport through the building foundation. Model assumptions ranged from diffusion only, advection only, and combined diffusion and advection. In terms of advection, methods ranged from an empirical approach based on measured soil gas entry rates, to a theoretical method based on Darcy's Law and an air permeability estimated using flow relationships for fractured rock. The results confirm the widely-held view by researchers that our understanding of soil gas migration through the building envelope is at a rudimentary stage.

Several field studies have suggested that if the soil permeability below the building floor slab is sufficiently high, a pressure differential can draw soil gas VOCs into the basement or ground floor at a significantly higher rate than would be predicted by diffusion alone (Hodgson et. al, 1992; Adomait, 1992). A sensitivity analysis conducted using a similar model to that utilized by participant #9, indicated that the advective mass flux through a cracked concrete floor slab dominated over diffusive flux for pressure gradients greater than 0.5 Pa, based on the input parameters assumed (Hers et. al, 1997).

### 5.2.4 Comparison of Advective Soil Gas Flow Rates Using Three Models

To further investigate advective soil gas flow rates through the building envelope, three different models used by participants are evaluated below using identical input parameters

for parameters common to all three models. The input parameters are defined in Table 14. The models used are described below:

1. *Model #1*: Soil gas advection ($Q_{sg}$) is estimated using Darcy's Law and relationships developed between fracture porosity and permeability for fractured rock. The pressure gradient across the concrete slab is estimated by taking into account soil gas advection in soil below the slab the resulting pressure drop in soil (i.e., resistance in soil).

$$Q_{sg} = \frac{9.8E-12 \times K_c \times A_b \times (\Delta P)}{(\mu \times T_s)} \qquad \text{Eq. 5}$$

$$K_c = \frac{10204 \times W_c^3}{6 \times S_c} \qquad \text{Eq. 6}$$

$$A_c = \frac{0.001 \times A_b \times W_c}{S_c} \qquad \text{Eq. 7}$$

$$\Delta P = P_b - P_{us} \qquad \text{Eq. 8}$$

$$P_{us} = \frac{Kc \times \frac{P_b}{T_s}}{\frac{Kc}{T_s} + \frac{k_{soil}}{D}} \qquad \text{Eq. 9}$$

2. *Model #2*: Advection estimated using an analytical solution for flow to a cylinder of length $x_{crack}$ and radius $r_{crack}$ located a depth $z_{crack}$ below ground surface using the following equation.

$$Q_{sg} = \frac{2\Pi \, \Delta P \, K_v \, x_{crack}}{\mu \ln\left(\frac{2 z_{crack}}{r_{crack}}\right)}; \quad \frac{r_{crack}}{z_{crack}} < 1 \qquad \text{Eq. 10}$$

$$r_{crack} = \frac{\eta A_b}{x_{crack}} \qquad \text{Eq. 11}$$

This is an idealized model for soil gas flow to cracks located at floor/wall seams.

3. *Model #3*: Advection estimated using the empirical methods presented by Figley (1996) and equations (2) to (4). It is noted that the equivalent leakage area chosen is an example value for a single family dwelling provided by Figley (1996).

To simplify the comparisons, it is assumed that soil gas transport occurs only through base of a concrete floor slab, or through cracks at the floor/wall interface, and not through the walls of a basement.

The results of the comparisons are shown in Table 12. It is emphasized that due to the significant differences in the models used, it is impossible to make direct comparisons and results can only be used to infer general predictive capabilities. In spite of the significant differences in model assumptions, all three models (excluding Model #1, Case #3) resulted in predicted soil gas advective flow rates which were within one order-of-magnitude. For Model #1, the difference between Cases 1 and 2 indicate that for the input parameters chosen the soil gas entry rates are not affected by the concrete permeability (i.e., concrete cracks are not significant) since the pressure gradient across the concrete slab is insignificant compared to that in soil. For Case #3, the soil parameters are changed (i.e., soil permeability increased and advective flow depth of influence decreased) resulting in a significant increase in advective soil gas flow rates.

While it is difficult to draw definitive conclusions based on the above results, it is clear that pressure coupling between the building envelope and soil is an important phenomenon that merits further study. Conceptually, factors that affect pressure coupling are the permeability of the concrete slab, permeability of the underlying of soil, presence of preferential conduits such as service penetrations and utility corridors, and the size of the building. Depending on site specific conditions, advective soil gas flow in some cases could potentially be controlled by resistance in soil while in other cases be controlled by resistance through the concrete slab. The concrete slab resistance could be the dominant mechanism when soil permeability is high and/or when there are preferential migration pathways below the slab.

### 5.2.5 Summary of Soil Gas Modelling Results

Soil gas fate and transport and building intrusion is a complex phenomena. The predicted exposure concentrations are highly dependent on the model assumptions, and site-specific parameters such as depth to contamination, soil properties (e.g., porosity, permeability and organic carbon fraction) and building characteristics (e.g., concrete cracks, drains and building underpressurization).

Conceptually, it is suggested that models for this pathway should couple fate and transport for soil gas with intrusion through the building envelope. It appears that incorporation of source depletion, biodegradation (when appropriate) and adsorption is important in increasing the realism of the subsurface component of the model. In terms of intrusion through the building envelope, advective soil gas intrusion rates will be highly dependent on the building floor slab and wall characteristics, and pressure coupling between the building and soil adjacent to the building.

It is clear that the relative importance of the different model characteristics will be highly dependent on the site specific conditions. In the case of relatively deeper contamination, overall VOC mass transport will be controlled primarily by diffusion through soil, and advection through the building envelope. In many cases, the "resistance" to mass transport provided by soil (i.e., diffusion biodegradation) will likely be greater than that provided by the building envelope. Therefore, the rate limiting process for this case would be mass transport through soil. In the case of shallow contamination, advective soil gas intrusion may be more significant particularly when there are preferential migration pathways (e.g., drains, utilities below the building) and when soil permeabilities are relatively high. Further analysis of case studies and detailed field measurements of soil gas transport and intrusion is required to refine predictive models and identify key controlling parameters.

## 5.3 Relevance to Decision Making

Contaminated sites risk assessment is intended to be a tool by which to obtain insight on health risks for purposes of assisting in making decisions. The basic areas of decision making in this context are either risk management decisions (i.e., steps required to mitigate health risks), and business decisions (i.e., land purchases, remediation for elimination of liability, etc.). In both cases the relevant point is that expenditures/investments are being made, in part, on insight gained from health risk assessment. Understandably there is both a need and desire for expenditures and decisions to be justified.

This present study provides some interesting perspectives on how risk estimates may affect business decisions, and to a lesser extent risk management decision. In the first

case, the wide variability in risk estimates, coupled with diversity of what is or is not an issue for consideration (e.g., inclusion versus exclusion of selected pathways), may give rise to very different perceptions about the liabilities intrinsic to a specific site. Thus, total risk estimated by one team may suggest health-related liabilities are virtually zero, while another team may conclude the warrant of a closer examination. If liabilities are perceived to be virtually zero, this may support the purchase of property, or perhaps a decision to sell without further remediation. A more conservative estimate of risk may support the opposite decision.

While agreement amongst participants on acceptability versus unacceptability of risk estimates was relatively good in this study, the wide spread in risk estimates suggest disagreement is highly likely if the contaminant source concentrations are of a magnitude to create borderline concerns.

In the second case of decision making, risk management decisions, there is potential for a similar conundrum. However, it is imperative for risk managers to recognize the "weight-of-evidence" offered by screening risk estimates as developed in this Round Robin, versus the weight-of-evidence offered through *definitive* (i.e., detailed) risk assessment. As exemplified in the present study, screening risk estimates are "bounding estimates", designed to bound the reasonable upper limit of health risk. They are expected to be conservative (but not overly conservative) with the idea that even a borderline acceptance risk estimate is likely to be interpreted as acceptable owing to the inherent conservatism. Conservative estimates which are clearly *de minimus* (e.g., HQ < 0.01, or ILCR < 1E-7) are likely to be smaller in reality, and would not support the need for risk-reduction measures.

Where screening risk estimates suggest a substantial health hazard exists, the wide variability in results from this study would suggest risk management decisions not be made until more definitive computations are conducted.

This study provides a basis to benchmark the variability amongst risk assessors, under "screening risk assessment" conditions. The variability in this case is the product of differing views in applying conservatism in exposure assumptions, differences in analyzing

raw contaminant data, differences in perceived importance of specific exposure pathways and differences in the use of contaminant transport models and their inherent uncertainty.

The degree to which definitive risk estimates would vary amongst the same participants cannot be derived from this study. However, in theory one would expect a convergence amongst assessors, as more definitive (realistic and/or site specific) exposure assumptions are factored into the assessment, with a concomitant reduction in the variability of conservatism employed. In the final analysis, all risk assessments, whether screening or definitive in nature, should include some level of uncertainty analysis to allow the reviewer to appreciate the level of conservatism and range over which other possible value of health risk may apply. To this end, it is recommended that all contaminated sites health risk estimates be expressed at least as a possible range of values (e.g., reasonable minimum, reasonable maximum) and preferably with some aspect of probability associated with the assumption employed (e.g., mean, made or probability distribution). This would foster a better understanding of the health risks for both risk assessors and risk managers, and better support consequent risk management decisions.

## 6.0 CONCLUSIONS

Several conclusions were derived from this study. However, it is important to re-emphasize the present study was conducted as a screening risk assessment, not a definitive risk assessment. For some of the conclusions it may be reasonable to speculate that the same would hold true for a definitive assessment, but this may not apply in all cases.

1. Fundamentally, the type and number of pathways included in the risk assessments varied between participants. For trace metals in surface soils, oral ingestion was the most commonly included pathway. Fugitive dust inhalation and consumption of domestically grown produce were included/excluded by various participants.

2. Highly divergent risk estimates were demonstrated for all contaminants and exposure pathways. While general agreement existed amongst the acceptability of the risks, the divergence suggest lack of agreement could prevail if soil contaminant concentrations were appropriate.

3. The variability in risk estimates was primarily explained by variability in dose estimates. Thus, for improved conformity amongst assessors, both of these elements should be considered.

4. The variability in dose estimates via direct pathways (e.g., soil ingestion and dermal contact) were primarily explained by receptor characteristics. The variability in dose estimates for complex indirect pathways (e.g., dust inhalation and indoor gas inhalation) were primarily explained by model uncertainty, which affected the predicted exposure concentration.

5. Correlation amongst the various determinants of dose suggest assessors are applying conservatism to several variables. This suggests the need to re-visit the approach to applying conservatism, to avoid overly conservative risk assessments and uncertainty.

6. Models used to predict wind generated dust emissions are highly dependent on input parameters such as soil type, vegetative cover and size of the site. Therefore, it is important for screening-level risk assessments to use appropriate site-specific data.

7. Models used to predict soil gas fate and transport are highly dependent on the model assumptions, and site-specific parameters such as depth to contamination, soil properties (e.g., porosity, permeability and organic carbon fraction) and building characteristics (e.g., concrete cracks, drains and building underpressurization).

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**Table 1**
**Exposure pathways amenable to statistical analyses and the number of participants employing a specific pathway/contaminant/receptor combination.**

| Exposure Pathway | COPC | Mode of Toxicity | Adult Receptor | Child Receptor | Composite Receptor |
|---|---|---|---|---|---|
| Soil Ingestion | Zinc | non-carcinogenic | yes (n=5) | yes (n=6) | no (n=0) |
| | Copper | non-carcinogenic | yes (n=4) | yes (n=6) | no (n=0) |
| | Lead | non-carcinogenic | yes (n=4) | yes (n=7) | no (n=0) |
| | Lead | carcinogenic | no (n=1) | no (n=1) | no (n=0) |
| | Cadmium | non-carcinogenic | yes (n=5) | yes (n=6) | no (n=0) |
| | Benzene | non-carcinogenic | no (n=1) | no (n=1) | no (n=0) |
| | Benzene | carcinogenic | no (n=3) | no (n=2) | no (n=0) |
| Dermal Contact | Zinc | non-carcinogenic | yes (n=4) | yes (n=5) | no (n=0) |
| with Soil | Copper | non-carcinogenic | no (n=3) | yes (n=5) | no (n=0) |
| | Lead | non-carcinogenic | no (n=3) | yes (n=5) | no (n=0) |
| | Cadmium | non-carcinogenic | yes (n=4) | yes (n=5) | no (n=0) |
| | Benzene | carcinogenic | no (n=4) | no (n=2) | no (n=0) |
| | Benzene | non-carcinogenic | no (n=2) | no (n=2) | no (n=0) |
| | Vinyl Chloride | cancer | no (n=1) | no (n=1) | no (n=0) |
| | Vinyl Chloride | non-carcinogenic | no (n=1) | no (n=1) | no (n=0) |
| Inhalation | Zinc | non-carcinogenic | no (n=2) | yes (n=5) | no (n=0) |
| of Dust | Copper | non-carcinogenic | no (n=2) | yes (n=5) | no (n=0) |
| | Lead | non-carcinogenic | no (n=2) | yes (n=5) | no (n=0) |
| | Cadmium | carcinogenic | yes (n=4) | yes (n=3) | no (n=0) |
| | Cadmium | non-carcinogenic | no (n=1) | no (n=4) | no (n=0) |
| Ingestion of | Zinc | non-carcinogenic | no (n=1) | no (n=3) | no (n=0) |
| Produce | Copper | non-carcinogenic | no (n=1) | no (n=3) | no (n=0) |
| | Lead | non-carcinogenic | no (n=1) | no (n=3) | no (n=0) |
| | Cadmium | non-carcinogenic | no (n=1) | no (n=3) | no (n=0) |
| | Benzene | non-carcinogenic | no (n=1) | no (n=1) | no (n=0) |
| | Benzene | carcinogenic | no (n=2) | no (n=1) | no (n=0) |
| Inhalation of Volatiles | Benzene | carcinogenic | no (n=4) | yes (n=4) | no (n=2) |
| | Benzene | non-carcinogenic | no (n=1) | no (n=2) | no (n=0) |
| (Indoor) | Vinyl Chloride | cancer | no (n=4) | no (n=4) | no (n=1) |
| | Vinyl Chloride | non-carcinogenic | no (n=1) | no (n=1) | no (n=0) |
| Inhalation of Volatiles | Benzene | carcinogenic | yes (n=5) | no (n=3) | no (n=1) |
| | Benzene | non-carcinogenic | no (n=1) | no (n=2) | no (n=0) |
| (Outdoor) | Vinyl Chloride | carcinogenic | no (n=4) | no (n=3) | no (n=0) |
| | Vinyl Chloride | non-carcinogenic | no (n=1) | no (n=1) | no (n=0) |

Note:
"yes" indicates that exposure pathway was included in statistical analyses.
"no" indicates that exposure pathway was not included in the statistical analyses.
The number of participants that included the specific exposure pathway is provided in the parentheses (i.e., n=5).

**Table 2: Summary of non-cancer risks for future residents. Values representing the minimum, maximum and ratio are based on consideration of both adult and child receptors.**

| Exposure Pathway | COPC | Min | Max | Max:Min |
|---|---|---|---|---|
| Soil Ingestion | Cadmium | 1.0E-04 | 6.8E-01 | 6.8E+03 |
| | Copper | 3.0E-05 | 1.1E+00 | 3.7E+04 |
| | Lead | 2.0E-03 | 1.4E+04 | 6.9E+06 |
| | Zinc | 7.0E-06 | 3.3E-02 | 4.7E+03 |
| | Benzene[1] | 0.0E+00 | 0.0E+00 | - |
| Dermal Contact with Soil | Cadmium | 5.8E-03 | 3.8E+00 | 6.5E+02 |
| | Copper | 1.0E-09 | 3.2E+02 | 3.2E+11 |
| | Lead | 1.6E-02 | 4.0E+01 | 2.5E+03 |
| | Zinc | 3.2E-05 | 1.0E-01 | 3.2E+03 |
| | Benzene[1] | 0.0E+00 | 1.3E-07 | - |
| | Vinyl Choride | 2.1E-03 | 3.5E-03 | 1.7E+00 |
| Inhalation of Dust | Cadmium | 5.8E-05 | 3.7E-01 | 6.4E+03 |
| | Copper | 1.0E-09 | 3.2E+02 | 3.2E+11 |
| | Lead | 2.0E-08 | 3.8E+02 | 1.9E+10 |
| | Zinc | 2.0E-10 | 8.3E-01 | 4.2E+09 |
| Ingestion of Produce | Cadmium | 1.1E-01 | 1.3E+01 | 1.1E+02 |
| | Copper | 3.9E-03 | 8.3E+00 | 2.1E+03 |
| | Lead | 1.1E+00 | 5.8E+02 | 5.1E+02 |
| | Zinc | 1.3E-02 | 5.6E+00 | 4.5E+02 |
| | Benzene[1] | 0.0E+00 | 0.0E+00 | - |
| Inhalation of Volatiles, Indoor | Benzene | 2.9E-04 | 2.8E+01 | 9.8E+04 |
| | Vinyl Choride | 2.2E-02 | 9.7E-02 | 4.4E+00 |
| Inhalation of Volatiles, Outdoor | Benzene | 1.2E-05 | 5.2E-02 | 4.3E+03 |
| | Vinyl Choride | 9.3E-04 | 1.7E-02 | 1.8E+01 |

[1] One of the participants estimated exposure concentrations of 0 mg/kg benzene, which explains the risk estimates of 0.

**Table 3: Number of participants concluding acceptable versus non-acceptable risks by pathway and contaminant for non-carcinogenic endpoints.**

| Exposure Pathway | COPC | Acceptable Risks[1] | Non-acceptable Risks |
|---|---|---|---|
| Soil Ingestion | Cadmium | 8 | 0 |
| | Copper | 6 | 1 |
| | Lead | 1 | 6 |
| | Zinc | 8 | 0 |
| | Benzene | 1 | 0 |
| Dermal Contact with Soil | Cadmium | 5 | 2 |
| | Copper | 5 | 1 |
| | Lead | 3 | 3 |
| | Zinc | 7 | 0 |
| | Benzene | 2 | 0 |
| | Vinyl Choride | 1 | 0 |
| Inhalation of Dust | Cadmium | 4 | 0 |
| | Copper | 4 | 1 |
| | Lead | 4 | 1 |
| | Zinc | 5 | 0 |
| Ingestion of Produce | Cadmium | 1 | 3 |
| | Copper | 2 | 2 |
| | Lead | 0 | 4 |
| | Zinc | 3 | 1 |
| | Benzene | 1 | 0 |
| Inhalation of Volatiles, Indoor | Benzene | 1 | 1 |
| | Vinyl Choride | 1 | 0 |
| Inhalation of Volatiles, Outdoor | Benzene | 2 | 0 |
| | Vinyl Choride | 1 | 0 |
| All Pathways[2] | Cadmium | 3 | 5 |
| | Copper | 3 | 4 |
| | Lead | 0 | 7 |
| | Zinc | 7 | 1 |
| | Benzene | 2 | 1 |
| | Vinyl Choride | 1 | 0 |

Note:

[1] Acceptability of hazard quotient based on being less than unity.

[2] Based on hazard indices (sum of hazard quotients) for each contaminant.

**Table 4: Summary of cancer risks for future residents. Values representing the minimum, maximum and ratio are based on consideration of both adult and child receptors.**

| Exposure Pathway | COPC | Min | Max | Max:Min |
|---|---|---|---|---|
| Soil Ingestion | Lead | 1.3E-05 | 1.3E-04 | 9.4E+00 |
| | Benzene[1] | 0.0E+00 | 1.7E-10 | - |
| Dermal Contact with Soil | Benzene[1] | 0.0E+00 | 8.0E-10 | - |
| | Vinyl Choride | 1.7E-08 | 5.2E-08 | 3.0E+00 |
| Inhalation of Dust | Cadmium | 3.0E-14 | 3.0E-04 | 9.9E+09 |
| | Lead | 5.7E-05 | 1.3E-04 | 2.3E+00 |
| Ingestion of Produce | Benzene[1] | 0.0E+00 | 3.3E-07 | - |
| Inhalation of Volatiles, Indoor | Benzene | 9.5E-09 | 3.5E-02 | 3.7E+06 |
| | Vinyl Choride | 2.2E-09 | 2.4E-03 | 1.1E+06 |
| Inhalation of Volatiles, Outdoor | Benzene | 9.5E-09 | 2.6E-06 | 2.8E+02 |
| | Vinyl Choride | 5.5E-09 | 4.7E-05 | 8.4E+03 |

[1] One of the participants estimated exposure concentrations of 0 mg/kg benzene, which explains the risk estimates of 0.

**Table 5: Number of participants concluding acceptable versus non-acceptable risks by pathway and contaminant for carcinogenic endpoints.**

| Exposure Pathway | COPC | Acceptable Risks[1] | Non-Acceptable Risks |
|---|---|---|---|
| Soil Ingestion | Lead | 0 | 1 |
| | Benzene | 3 | 0 |
| Dermal Contact with Soil | Benzene | 4 | 0 |
| | Vinyl Choride | 1 | 0 |
| Inhalation of Dust | Cadmium | 2 | 3 |
| | Lead | 0 | 1 |
| Ingestion of Produce | Benzene | 2 | 0 |
| Inhalation of Volatiles, Indoor | Benzene | 2 | 5 |
| | Vinyl Choride | 1 | 5 |
| Inhalation of Volatiles, Outdoor | Benzene | 5 | 1 |
| | Vinyl Choride | 2 | 3 |
| All Pathways[2] | Cadmium | 2 | 3 |
| | Lead | 0 | 1 |
| | Benzene | 3 | 5 |
| | Vinyl Chloride | 1 | 5 |

Note:

[1] Acceptability of lifetime cancer risk based on being less than $10^{-6}$.

[2] Based on total ILCR (sum of individual ILCR) for each contaminant.

**Table 6: Summary of Toxicity Reference Values.**

| Exposure Pathway | COPC | Min | Max | Max:Min |
|---|---|---|---|---|
| Soil Ingestion | Cadmium | 5.0E-04 | 1.0E-03 | 2.0E+00 |
| | Copper | 4.0E-02 | 5.2E-01 | 1.3E+01 |
| | Lead | 1.0E-06 | 4.5E-03 | 4.5E+03 |
| | Zinc | 3.0E-01 | 3.3E-01 | 1.1E+00 |
| | Benzene | 3.7E-03 | 3.7E-03 | 1.0E+00 |
| Dermal Contact with Soil | Cadmium | 1.0E-05 | 2.5E-03 | 2.5E+02 |
| | Copper | 1.7E-02 | 2.0E+00 | 1.2E+02 |
| | Lead | 1.2E-06 | 3.6E-03 | 3.0E+03 |
| | Zinc | 1.5E-02 | 1.5E+01 | 1.0E+03 |
| | Benzene | 5.0E-03 | 5.0E-03 | 1.0E+00 |
| | Vinyl Chloride | 9.4E-06 | 9.4E-06 | 1.0E+00 |
| Inhalation of Dust | Cadmium | 9.3E-06 | 5.7E-05 | 6.1E+00 |
| | Copper | 2.4E-06 | 5.0E-01 | 2.1E+05 |
| | Lead | 1.0E-06 | 3.6E-03 | 3.6E+03 |
| | Zinc | 1.1E-04 | 3.0E-01 | 2.7E+03 |
| Ingestion of Produce | Cadmium | 8.1E-04 | 1.0E-03 | 1.2E+00 |
| | Copper | 4.0E-02 | 5.0E-01 | 1.3E+01 |
| | Lead | 1.0E-06 | 3.6E-03 | 3.6E+03 |
| | Zinc | 3.0E-01 | 3.0E-01 | 1.0E+00 |
| | Benzene | 3.7E-03 | 3.7E-03 | 1.0E+00 |
| Inhalation of Vapours | Benzene | 1.7E-03 | 2.6E-03 | 1.5E+00 |
| | Vinyl Chloride | 4.5E-03 | 4.5E-03 | 1.0E+00 |

**Table 7: Summary of Cancer Slope Factors.**

| Exposure Pathway | COPC | Min | Max | Max:Min |
|---|---|---|---|---|
| Soil Ingestion | Lead | 2.1E-02 | 2.1E-02 | 1.0E+00 |
| | Benzene | 2.7E-02 | 1.9E-01 | 7.2E+00 |
| Dermal Contact with Soil | Benzene | 3.4E-03 | 2.9E-02 | 8.5E+00 |
| | Vinyl Chloride | 6.1E+00 | 6.1E+00 | 1.0E+00 |
| Inhalation of Dust | Cadmium | 1.1E+00 | 1.0E+02 | 9.0E+01 |
| | Lead | 8.1E-01 | 8.1E-01 | 1.0E+00 |
| Ingestion of Produce | Benzene | 1.9E-01 | 1.9E-01 | 1.0E+00 |
| Inhalation of Vapours | Benzene | 5.0E-03 | 2.9E-02 | 5.8E+00 |
| | Vinyl Chloride | 2.5E-02 | 7.1E+00 | 2.8E+02 |

**Table 8**
**Participant Ranking Based on Relative Magnitude of Hazard Quotients for Soil Ingestion Pathway (Child Receptors).**

| Rank | Participant ID # | | | |
|---|---|---|---|---|
| | Copper | Zinc | Lead | Cadmium |
| 1 | 9 | 1 | 3 | 9 |
| 2 | 3 | 9 | 1 | 7 |
| 3 | 1 | 7 | 9 | 3 |
| 4 | 7 | 3 | 7 | 5 |
| 5 | 5 | 5 | 5 | 1 |
| 6 | 2 | 2 | 2 | 2 |

Note:
The lower the rank the higher the relative risk estimate

**Table 9**
**Participant Ranking Based on Relative Magnitude of Hazard Quotients for Dust Inhalation Pathway (Child Receptors).**

| Rank | Participant ID # | | | |
|---|---|---|---|---|
| | Copper | Zinc | Lead | Cadmium [1] |
| 1 | 3 | 3 | 3 | 3 |
| 2 | 1 | 1 | 5 | 5 |
| 3 | 5 | 5 | 1 | 1 |
| 4 | 7 | 7 | 7 | 7 |
| 5 | 2 | 2 | 2 | n/a |

Note:
The lower the rank the higher the relative risk estimate
n/a: not applicable

**Table 10**
**Summary of Environmental Fate and Transport Modelling for Outdoor Dust Inhalation Pathway**

| | #1 | #2 | #3 | #4 | #5 | #6 | #7 | #8 | #9 |
|---|---|---|---|---|---|---|---|---|---|
| Dust Generation | Empirical | Cowherd Rapid Assessment Model - Unlimited Erosion | Cowherd Rapid Assessment Model - Unlimited Erosion | Cowherd Rapid Assessment Model - Limited Erosion | * | NC | Cowherd Rapid Assessment Model - Unlimited Erosion | NC | NC |
| Air Model | Empirical | Single Compart-ment Box Model | Screen 3 Guassian Dispersion Model | Single Compart-ment Box Model | * | NC | Single Compart-ment Box Model | NC | NC |

Notes:
1) NC = Not conducted (i.e., pathway not considered); N/A = Not applicable (not included in model)
2) * = Not provided

**Table 11**
**Summary of Selected Model Input Parameters for Outdoor Dust Pathway - Cadmium**

| | #1 | #2 | #3 | #4 | #5 | #6 | #7 | #8 | #9 |
|---|---|---|---|---|---|---|---|---|---|
| Cadmium Soil Concentration (mg/kg) | 88.2 | 9.5 | | 88.2 | - | NC | 88.2 | NC | NC |
| Area ($m^2$) | N/A | N/A | N/A | 80,000 | * | NC | N/A | NC | NC |
| Fastest Mile Windspeed (u+) (m/s) | N/A | N/A | N/A | 29.2 | * | NC | N/A | NC | NC |
| Erosion Threshold Windspeed ($U_t$) (m/s) | N/A | N/A | N/A | 1 | * | NC | N/A | NC | NC |
| Freq. Disturbance per Month ($mo^{-1}$) | N/A | N/A | N/A | 15 | * | NC | N/A | NC | NC |
| Fraction Contaminated Surface Vegetative Cover (V) | N/A | N/A | N/A | 0.75 | * | NC | N/A | NC | NC |
| Thornthwaites Precipitation Evaporation Index (PE) | N/A | N/A | N/A | 80 | * | NC | N/A | NC | NC |
| Particulate Emission Rate ($g/cm^2$-sec) | N/A | 6.9E-14 | * | 6.4E-09[3] | * | NC | 6.9E-14 | NC | NC |
| Mean Annual Windspeed (U) (m/s) | N/A | 4.4 | 4.4 | 4.4 | * | NC | 4.4 | NC | NC |
| Mixing Height (H) | N/A | 2 | 2.0 | 2.0 | * | NC | 2.3 | NC | NC |
| Width of Site (W) (m) | N/A | 1[4] | 283 | 200[5] | * | NC | 10[4] | NC | NC |
| Exposure Concentration ($mg/m^3$) | 1.5E-06 | 7.38E-13 | 1.0E-05 | 2.56E-04 | 2E-04 | NC | 6.0E-11 | NC | NC |

Notes:
1) NC = Not conducted (i.e., pathway not considered); N/A = Not applicable (not included in model)
2) * = Not provided
3) Calculated based on input parameters provided
4) Parallel to predominant wind direction
5) Perpendicular to predominant wind direction

**Table 12**
**Comparison of Advective Soil Gas Flow Rates Through Building Envelope**

| Input Parameter | Definition (units) | Model 1 Case 1 | Model 1 Case 2 | Model 1 Case 3 | Model 2 | Model 3 |
|---|---|---|---|---|---|---|
| $A_b$ | Area of Square Slab ($m^2$) | 100 | 100 | 100 | 100 | 100 |
| $\Delta P$ | Pressure gradient between soil and building (Pa) | N/A | N/A | N/A | 5 | 5 |
| $P_b$ | Building Underpressurization (P) | 5 | 5 | 5 | N/A | N/A |
| $P_{us}$ | Underpressurization or vacuum directly below building slab (Pa) (calculated) | 4.997 | 4.997 | 4.86 | N/A | N/A |
| $\mu$ | Viscosity of Air (18 degrees Celsius) (g/sec-cm) | 0.000183 | 0.000183 | - | 0.000183 | N/A |
| $T_s$ | Thickness of Concrete (m) | 0.1 | 0.1 | 0.1 | N/A | N/A |
| $\rho$ | soil gas density ($kg/m^3$) | N/A | N/A | N/A | N/A | 1.2 |
| $\eta$ | ratio of cracks to total slab area | 0.001 | 0.001 | 0.001 | 0.001 | N/A |
| ELA | Equivalent Leakage Area ($m^2$) | N/A | N/A | N/A | N/A | 0.00072 [1] |
| $A_c$ | area concrete cracks ($m^2$) (calculated) | 0.1 | 0.1 | 0.1 | N/A | N/A |
| $x_{crack}$ | length of cylinder (m) | N/A | N/A | N/A | 40 | N/A |
| $r_{crack}$ | perimeter (i.e., cylinder) crack radius (cm) | N/A | N/A | N/A | 0.25 | N/A |
| $z_{crack}$ | depth of cylinder below ground surface (m) | N/A | N/A | N/A | 1 | N/A |
| D | Advective Flow Depth of Influence (m) | 1 | 1 | 0.1 | N/A | N/A |
| $W_c$ | Average crack width (mm) | 1 | 10 | 1 | N/A | N/A |
| $S_c$ | Average crack spacing (m) | 1 | 1 | 1 | N/A | N/A |
| n | flow coefficient (dimensionless) | N/A | N/A | N/A | N/A | 1 |
| $k_{soil}$ | permeability (darcy) | 10 | 10 | 50 | 10 | N/A |
| $K_c$ | permeability of concrete slab (darcy) (calculated) | 1700 | 1.7E+0.5 | 1700 | N/A | N/A |
| $Q_{sg}$ | Soil Gas Flow Rate Through Slab ($m^3/sec$) | 0.00027 | 0.00027 | 0.013 | 0.00010 | 0.0014 |

Notes:
1) Example equivalent leakage area for a new single family bungalow in Saskatoon provided by Figley (1996).

**Table 13**
**Summary of Selected Model Input Parameters for Indoor Soil Gas Intrusion Pathway**

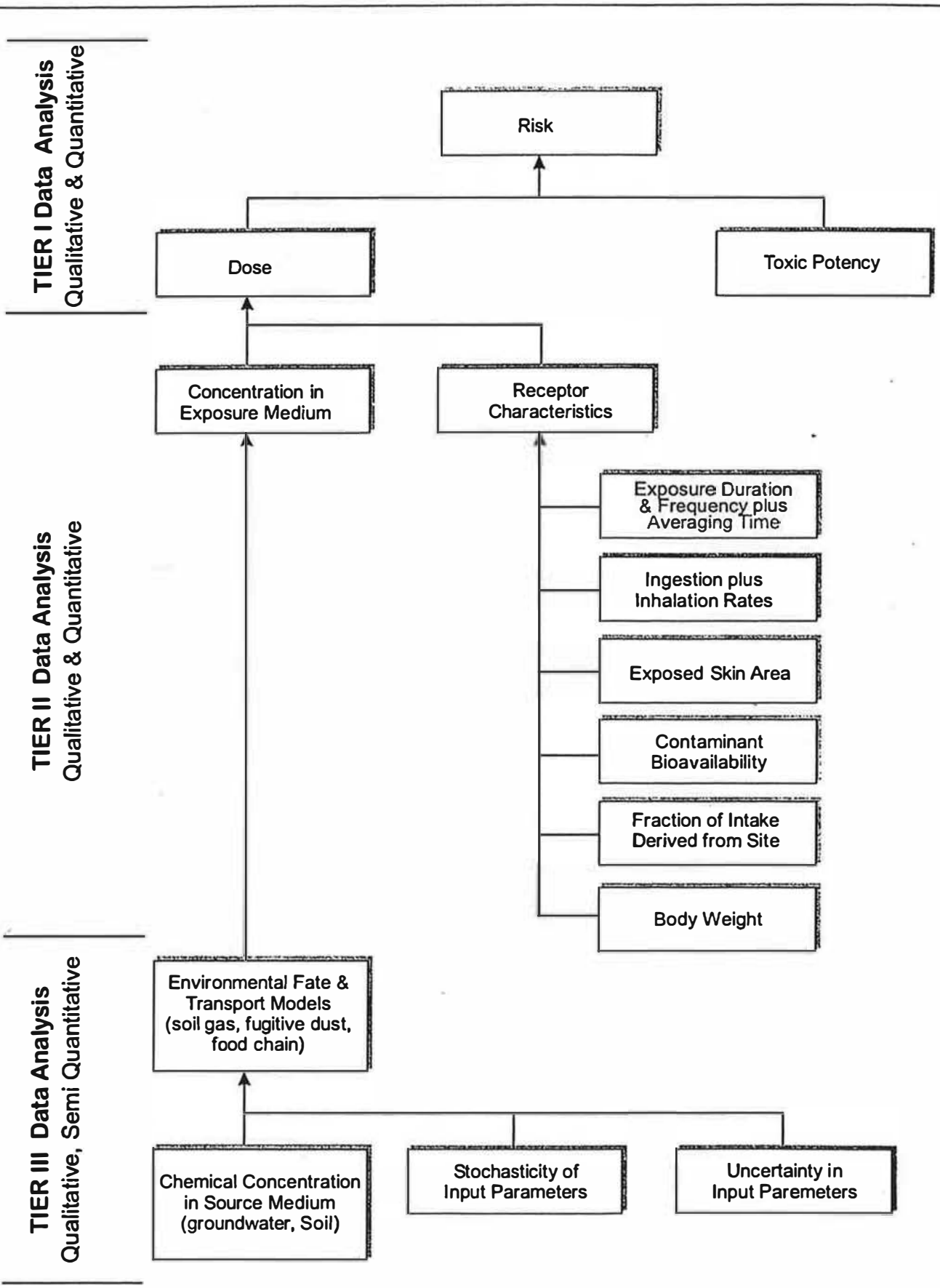
| Input/Output Parameter | #1 | #2 | #3 | #4 | #5 | #6 | #7 | #8 | #9 |
|---|---|---|---|---|---|---|---|---|---|
| Benzene Soil Concentration (mg/kg) | 12.6 | 1.06[5] | NC | NC | 12.6 | * | 0.29 | 1.49[6] | 12.6 |
| Depth to Contamination (m) | 1.5 | 1.0 | NC | NC | 3.0 | 1.0 | 0.9 | N/A | 1.0 |
| Crack Width (mm) | N/A | N/A | NC | NC | * | N/A | N/A | N/A | 1 |
| Crack Spacing (m) | N/A | N/A | NC | NC | 3 | N/A | N/A | N/A | 1 |
| Fraction Cracks to Total Foundation Area (dimensionless) | 0.001 | 0.01 | NC | NC | * | N/A | 0.01 | 5.85E-05 | 0.002 |
| Building Air Exchanges per hour | 1 | 0.5 | NC | NC | 1 | N/A | 0.4 | 0.375 | 1 |
| Building Under-pressurization (Pa) | 4.7 | N/A | NC | NC | Calculated | N/A | N/A | 10 | 5 |
| Height of Building Mixing Zone (m) | 6.3 | 2.3 | NC | Nc | 6.9 | N/A | 2.3 | 4.6[3] | 2.3 |
| Benzene Building Air Concentration ($mg/m^3$) | 0.0953 | 0.155 | NC | NC | 0.27[2] | 2.7E-06 | 0.025 | 0.0004 | 9.64 |

Notes:
1) NC = Not conducted (i.e., pathway not considered); N/A = Not applicable in terms of model used
2) 90th percentile value based on stochastic modeling
3) Based on a building area of 100 $m^2$
4) * = Value not provided
5) "95 percentile on average"
6) Arithmetic mean

**Table 14**
**Summary of Environmental Fate and Transport Modeling for Indoor Soil Gas Intrusion Pathway**

| Model Component | #1 | #2 | #3 | #4 | #5 | #6 | #7 | #8 | #9 |
|---|---|---|---|---|---|---|---|---|---|
| Source Depletion | N/A | N/A | N/A | NC | N/A | N/A | First Order Biodecay[6] | N/A | N/A |
| Partitioning | Linear Equilibrium | Linear Equilibrium | Fagacity[3] | NC | Linear Equilibrium | Linear Equilibrium | Linear Equilibrium | Hamaker Method | Linear Equilibrium |
| Fate & Transport in Soil | Gas & Aqueous Phase diffusion | Gas & Aqueous Phase diffusion | N/A | NC | Gas Phase diffusion | Gas & Aqueous Phase diffusion | Gas & Aqueous Phase diffusion[6] | Hamaker Method | Gas & Aqueous Phase diffusion |
| Fate & Transport through Building Foundation | Gas & Aqueous Phase diffusion dust-filled cracks in concrete, advection through cracks | Gas & Aqueous Phase diffusion through dust-filled cracks in concrete | Empirical[4] | NC | Gas & Aqueous Phase diffusion dust-filled cracks in concrete, advection through cracks | N/A | Gas & Aqueous Phase diffusion dust-filled cracks in concrete[6] | Empirical[2] | Gas & Aqueous Phase diffusion dust-filled cracks in concrete, advection through cracks |
| Building Underpressurization | Assumed | Assumed | N/A | NC | Empirical[5] (temperature difference) | N/A | Assumed | Assumed | Assumed |
| Building Air Mixing | Single compart-ment box model | Single compart-ment box model | N/A | NC | Single compart-ment box model | Outdoor box model | Single compart-ment box model [6] | Single compart-ment box model | Single compart-ment box model |

Notes:
1) NC = Not conducted; N/A = Not applicable
2) Empirical method based on measured air leakage rates (Figley, 1996)
3) CalToX model used - appears to be based on fugacity principles
4) CalToX model - appears to use an empirical attenuation factor of 10,000 between the soil gas VOC concentration at source, and building air VOC concentration
5) Based on empirical relationship between indoor and outdoor temperature difference (Nazaroff, 1992).
6) Based on ASTM E-1739-95.

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Golder Associates

**INFLUENCE DIAGRAM FOR CONTAMINATED SITES HEALTH RISKS**

Figure 1

**Figure 2. Non-Cancer Risks Associated With Zinc Exposure**

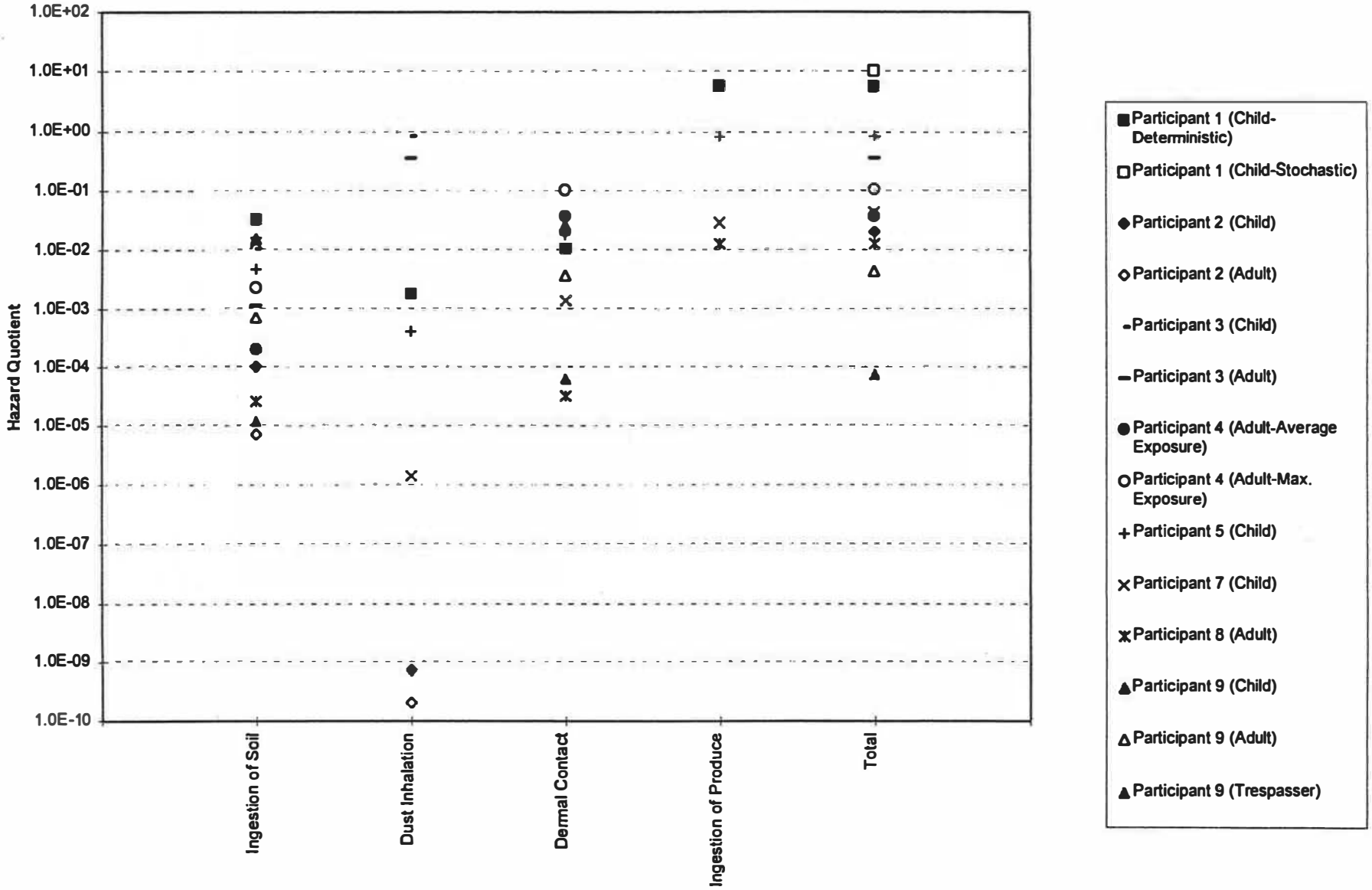


**Figure 3. Non-Cancer Risks Associated With Lead Exposure**

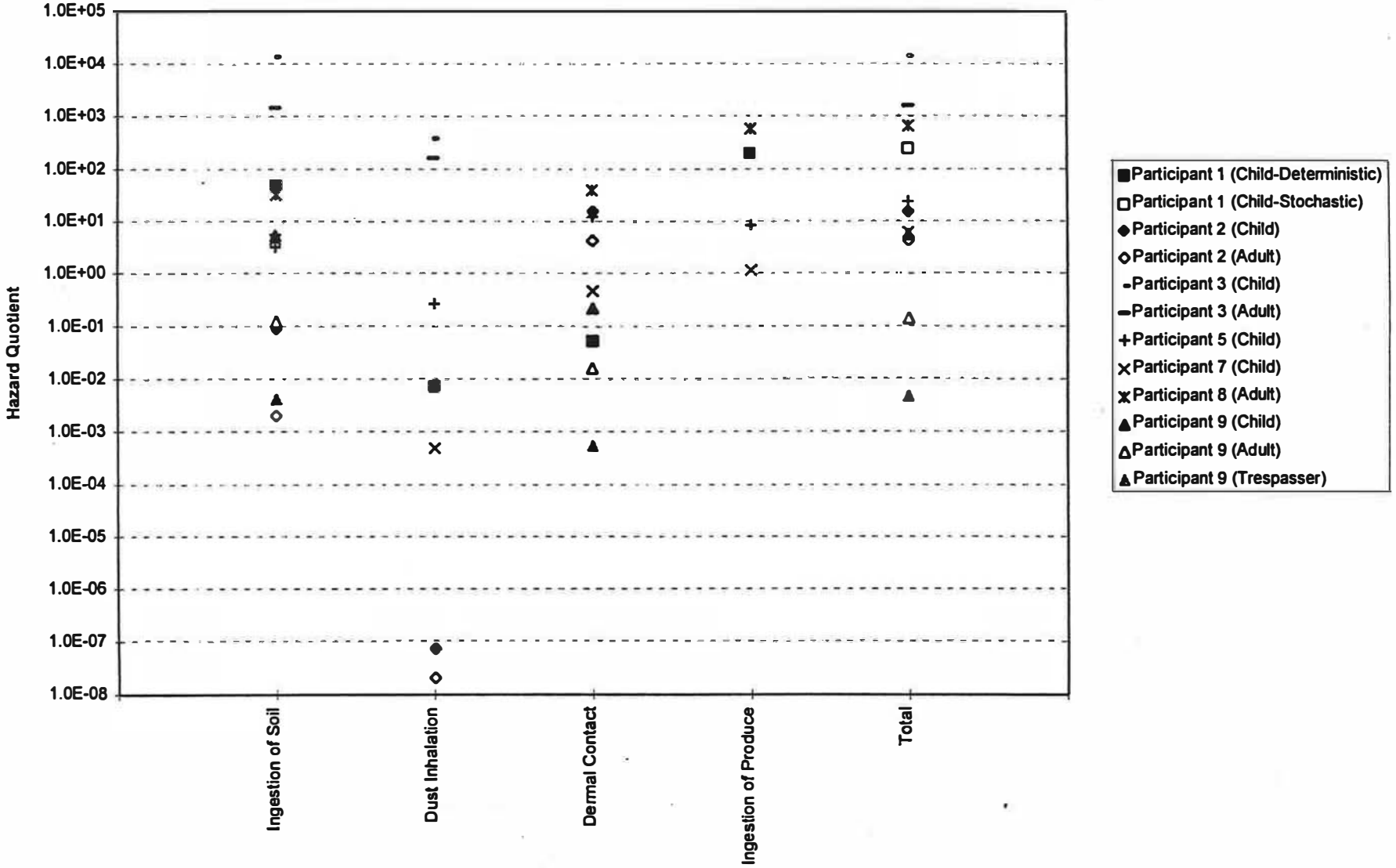


**Figure 4. Non-Cancer Risks Associated With Benzene Exposure**

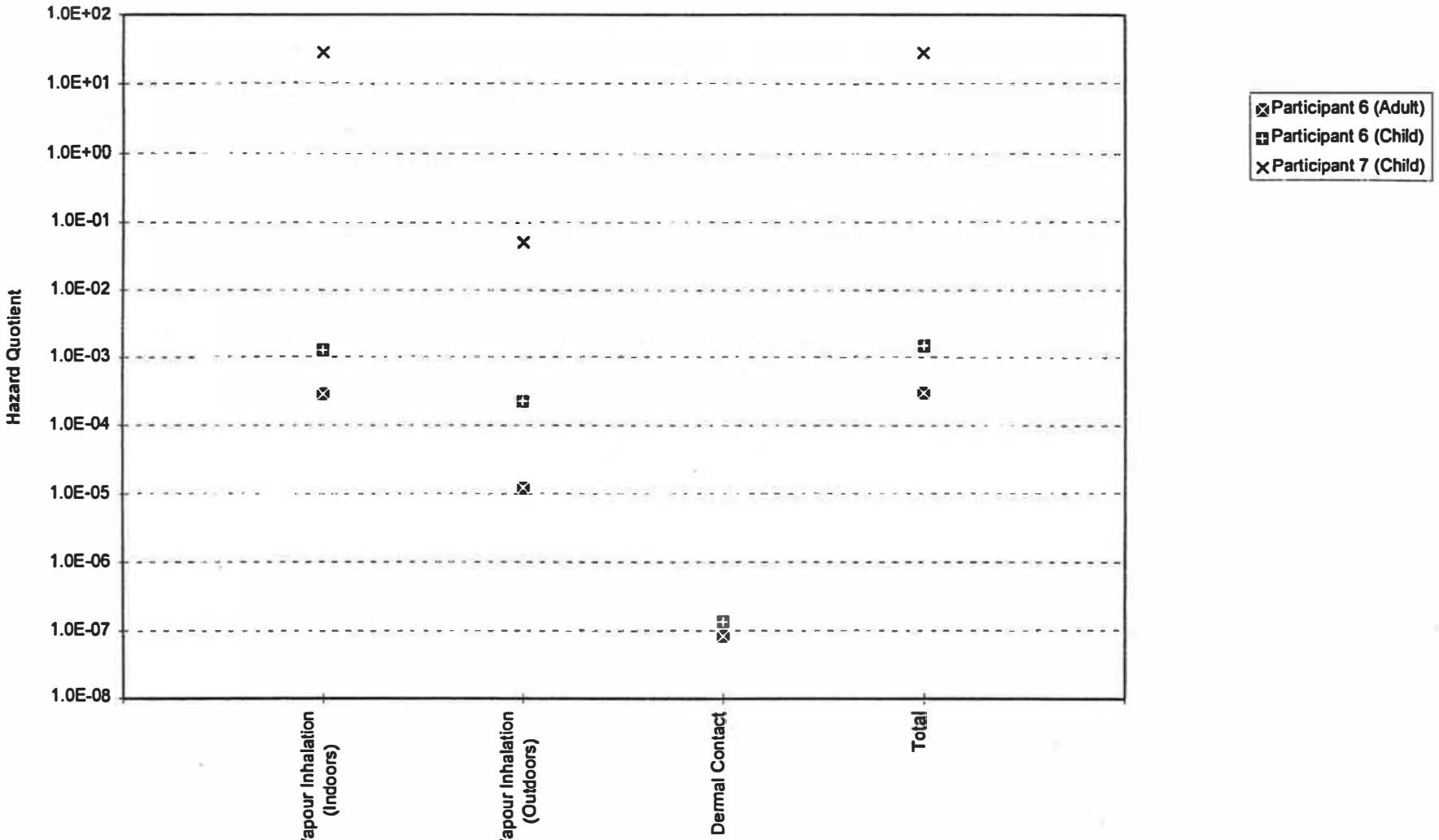


**Figure 5. Acceptability of Total Risk (Hazard Index) for Non-Carcinogens. Graph Displays Number of Participants Concluding Acceptable or Unacceptable Risk For Each Chemical.**

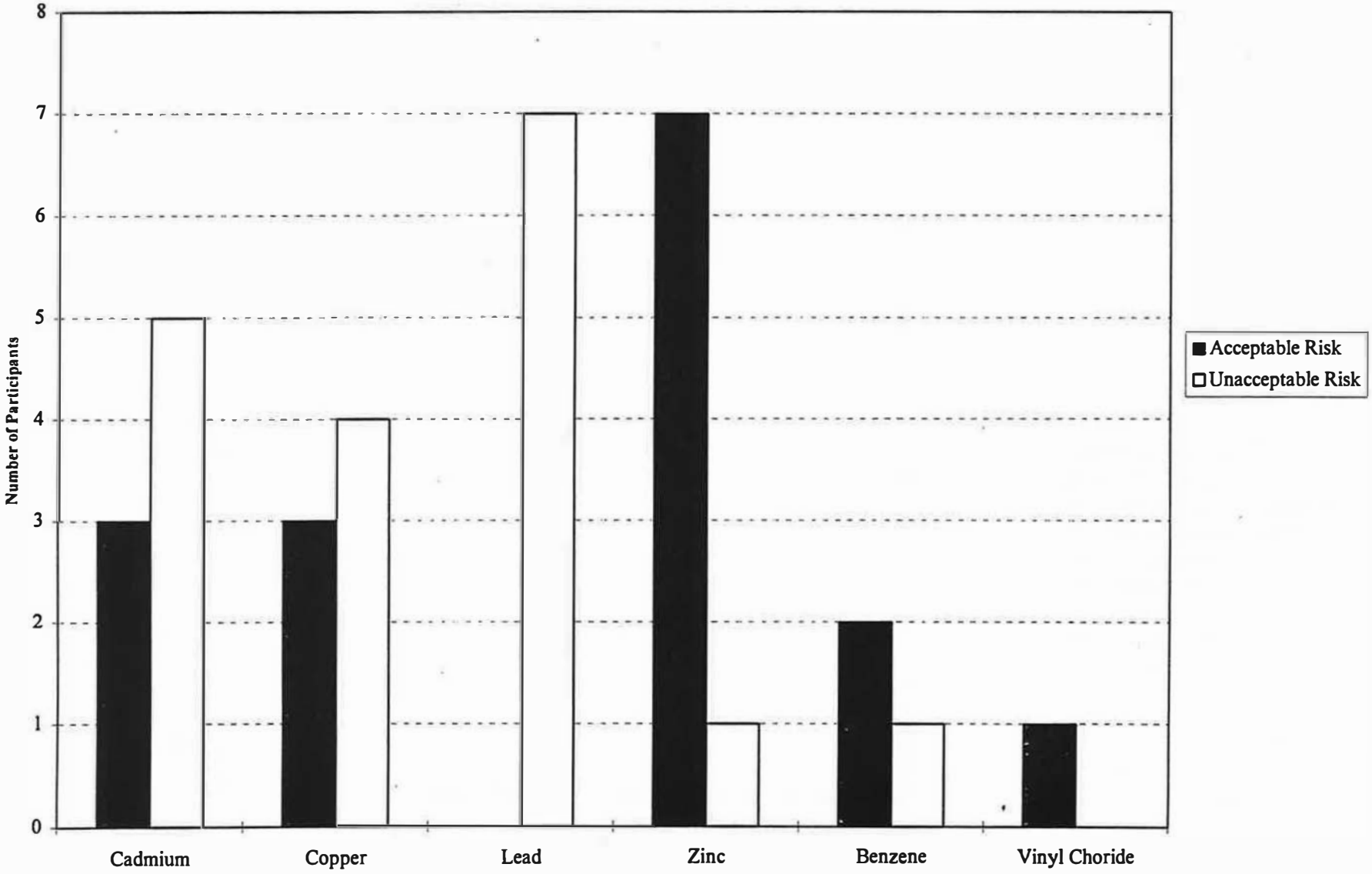


**Figure 6. Cancer Risks Associated With Cadmium Exposure**

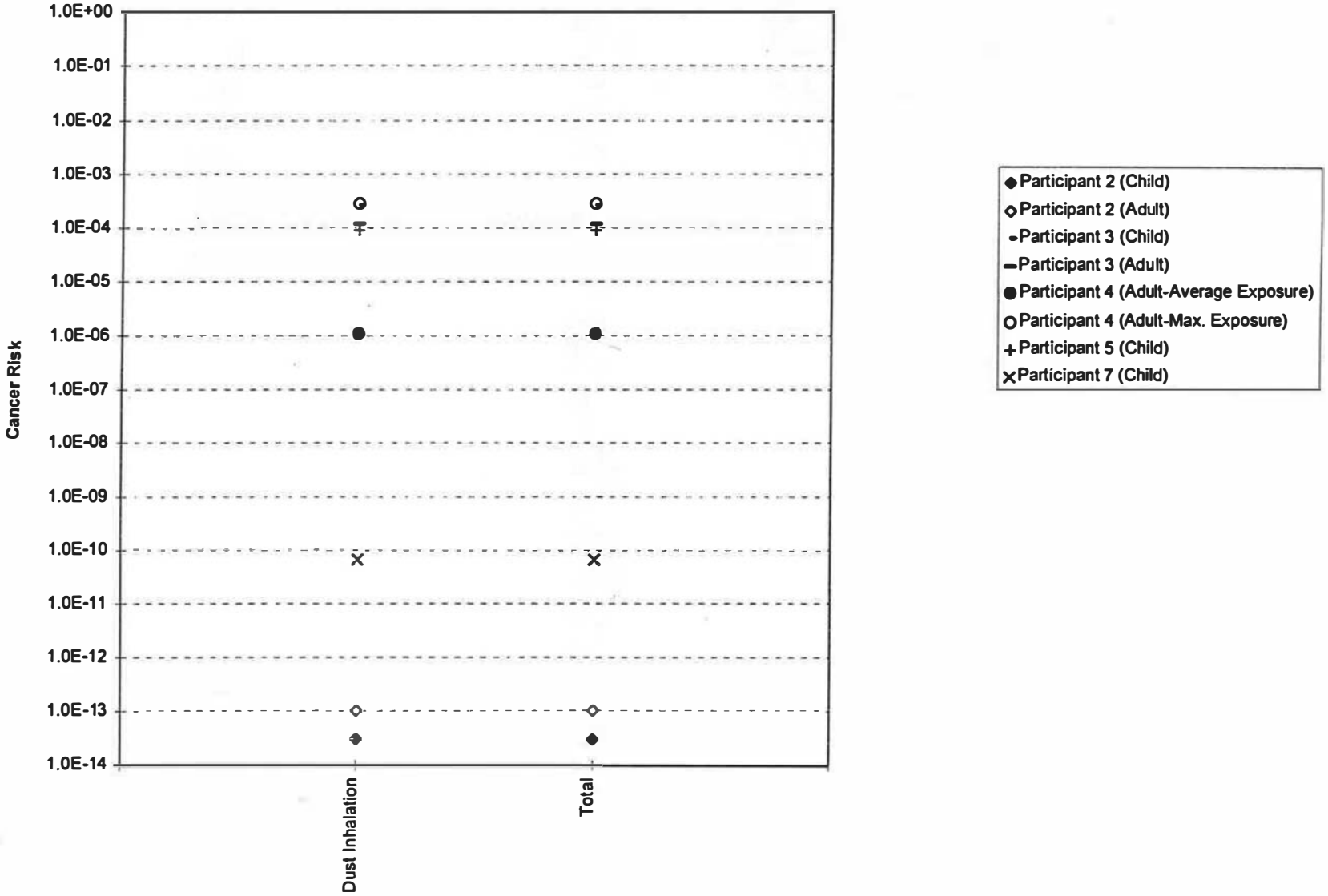


**Figure 7. Cancer Risks Associated With Benzene Exposure**

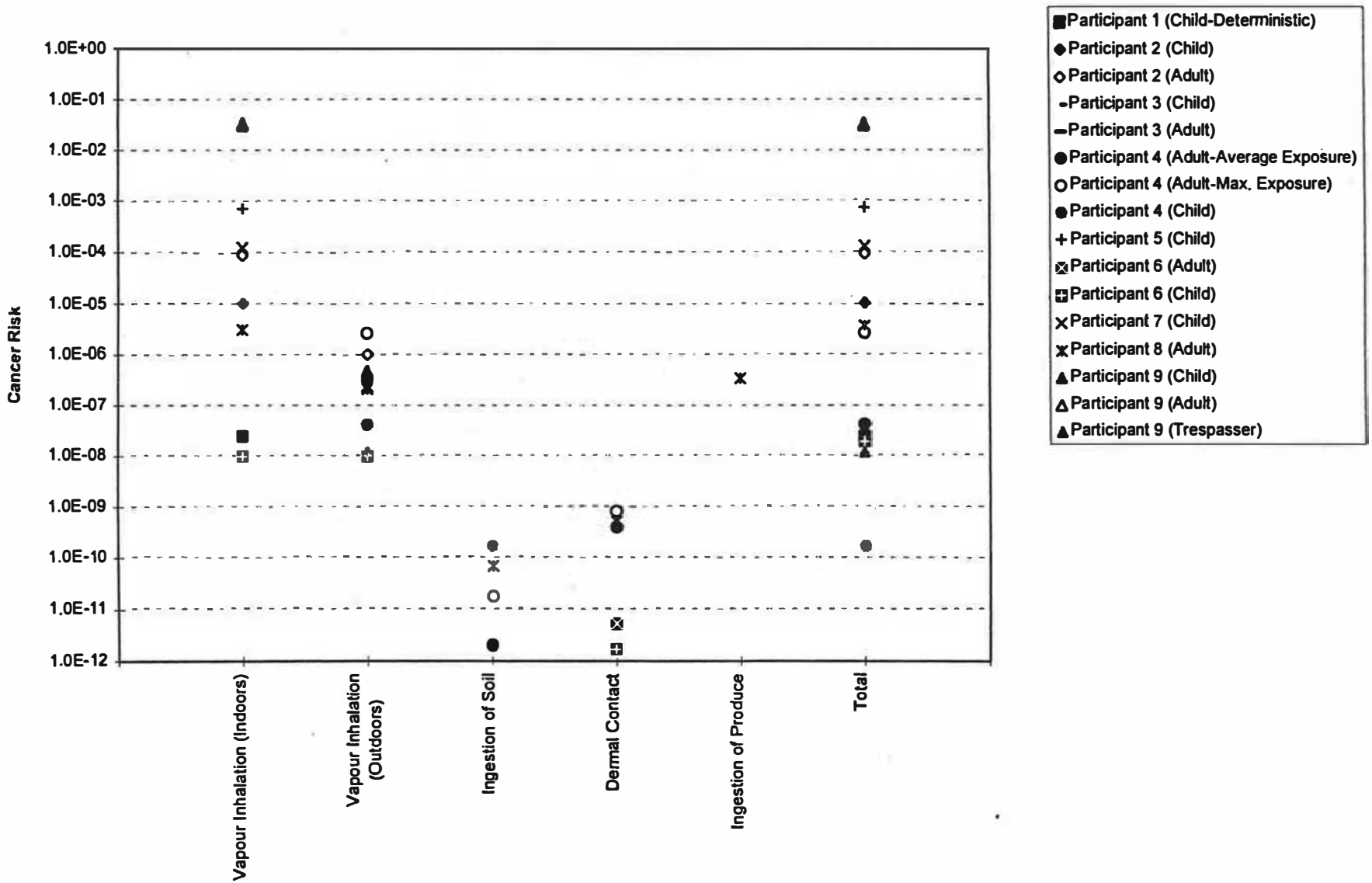


**Figure 8. Cancer Risks Associated With Vinyl Chloride Exposure**

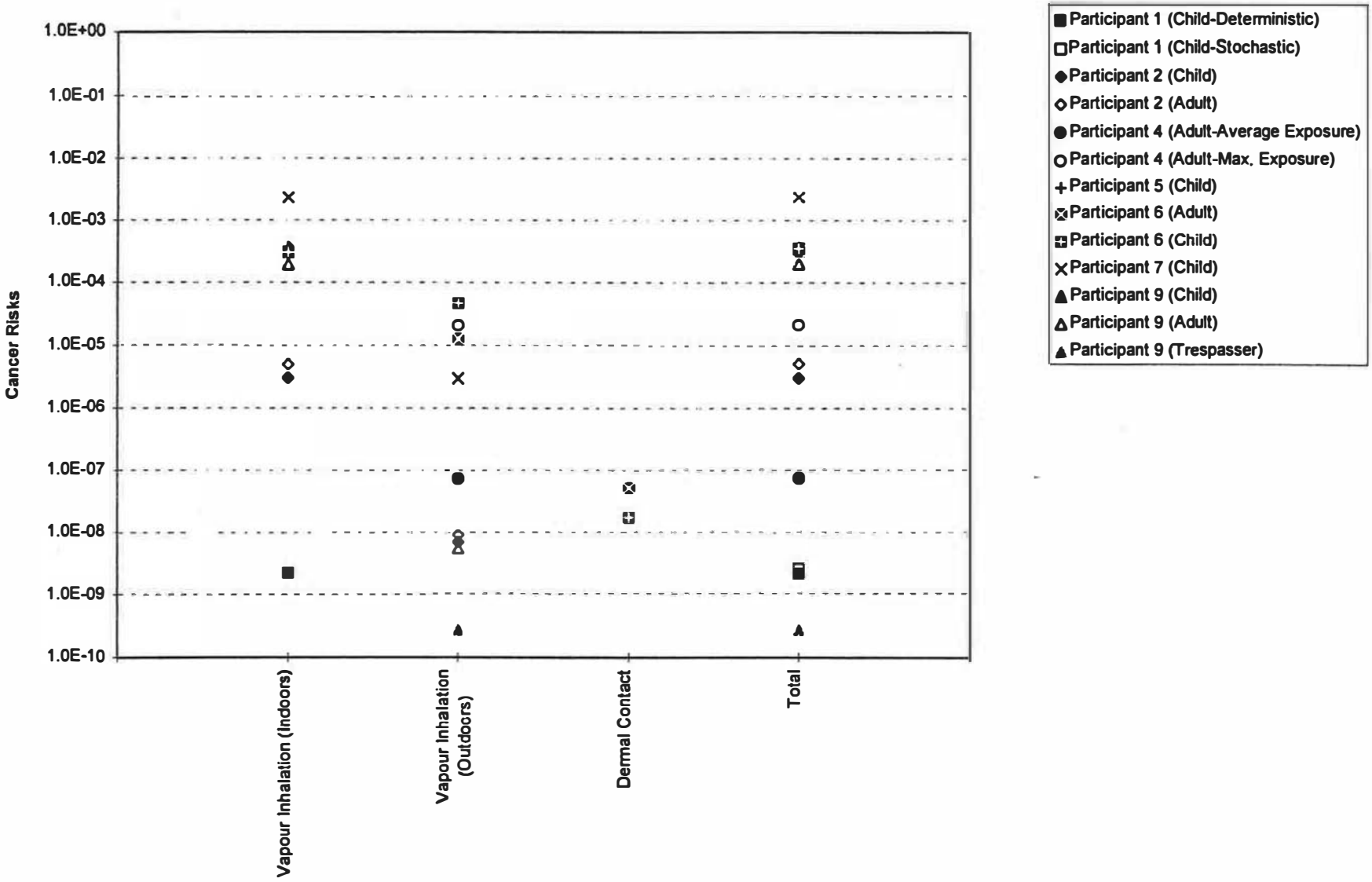


**Figure 9. Acceptability of Total Risk for Carcinogens. Graph Displays Number of Participants Concluding Acceptable or Unacceptable Risk For Each Chemical.**

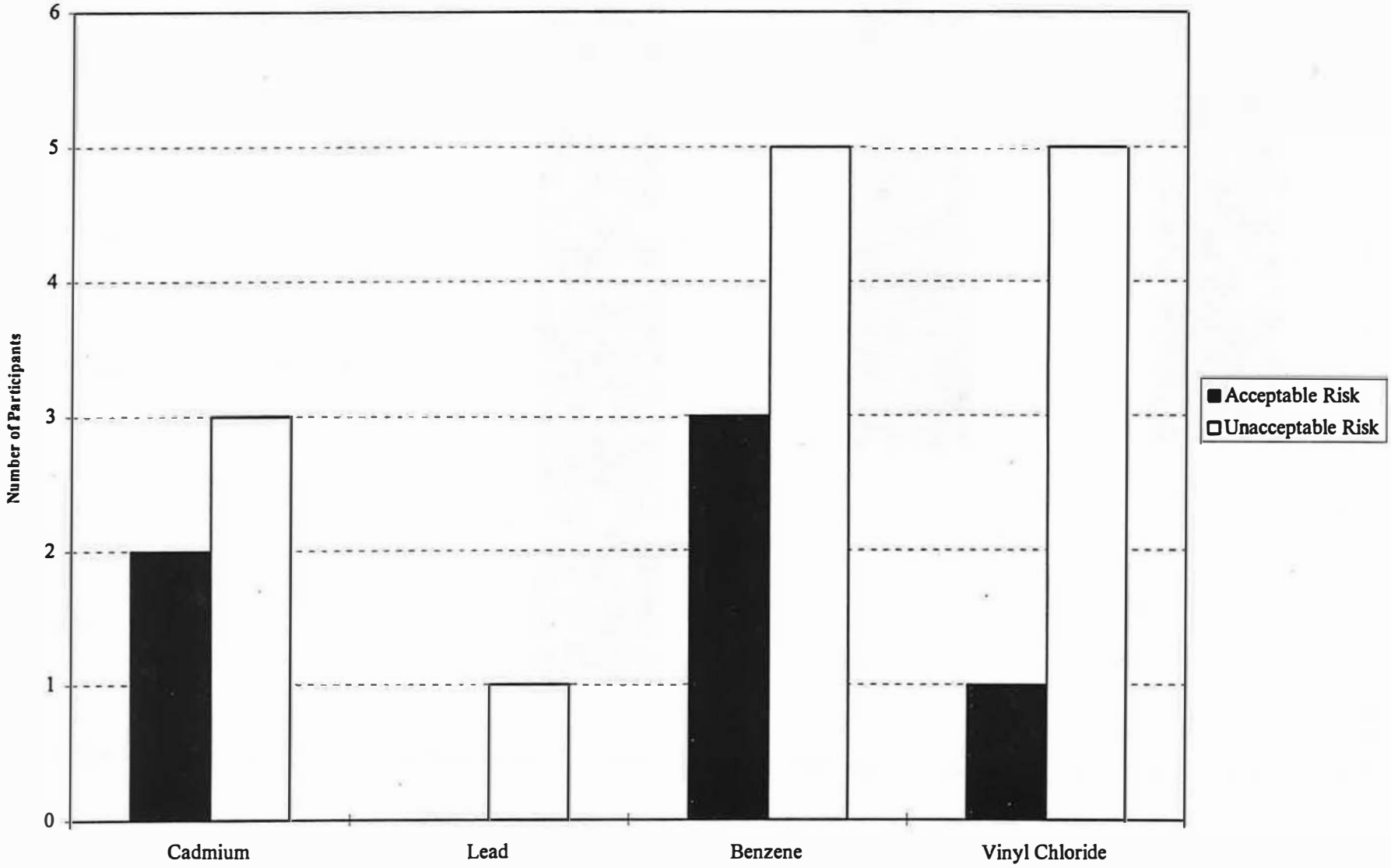


**Figure 10. Range in Reference Doses for Dust Inhalation Pathway**

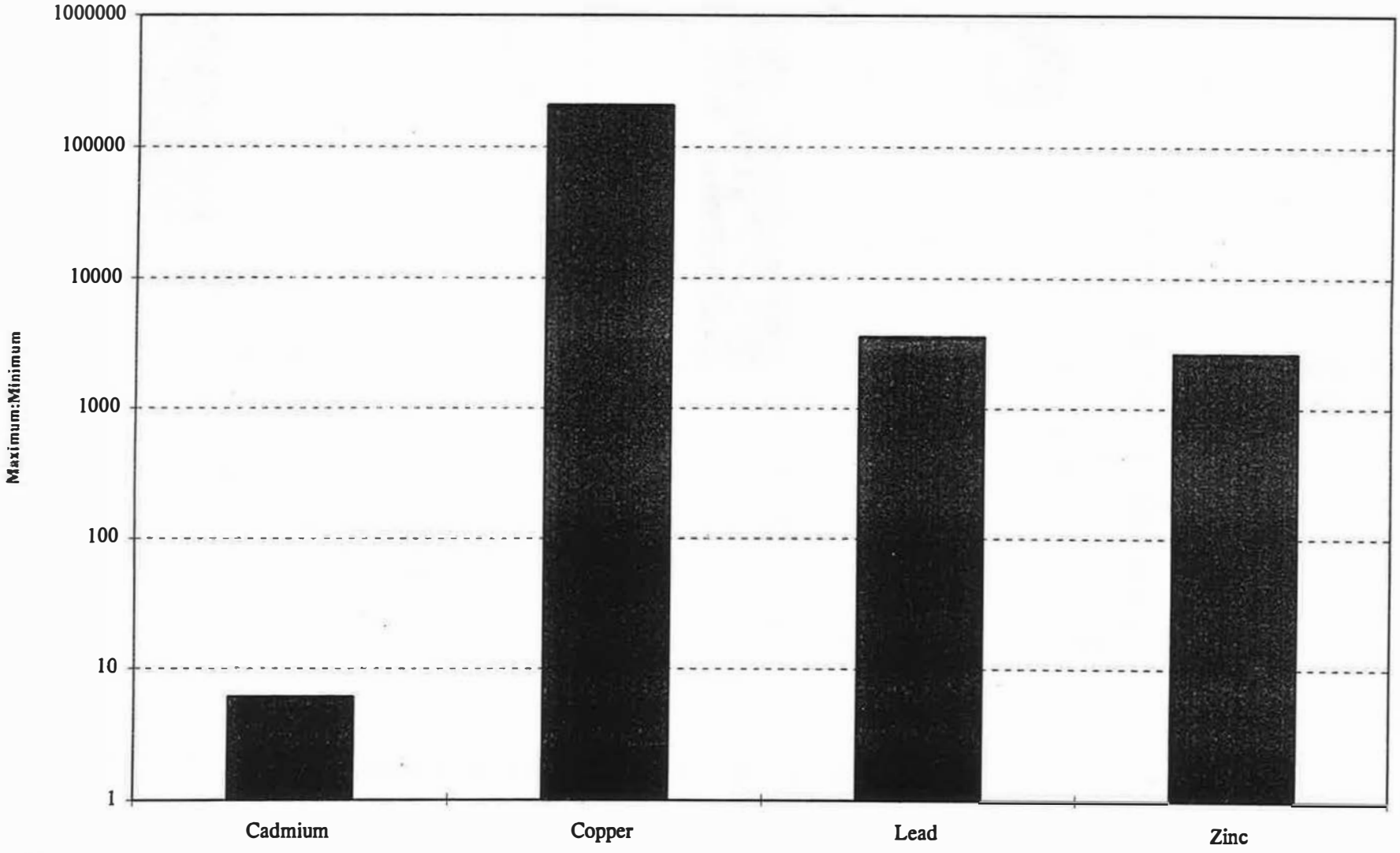


**Figure 11. Dose Rates for Zinc Exposure (Non-Carcinogenic)**

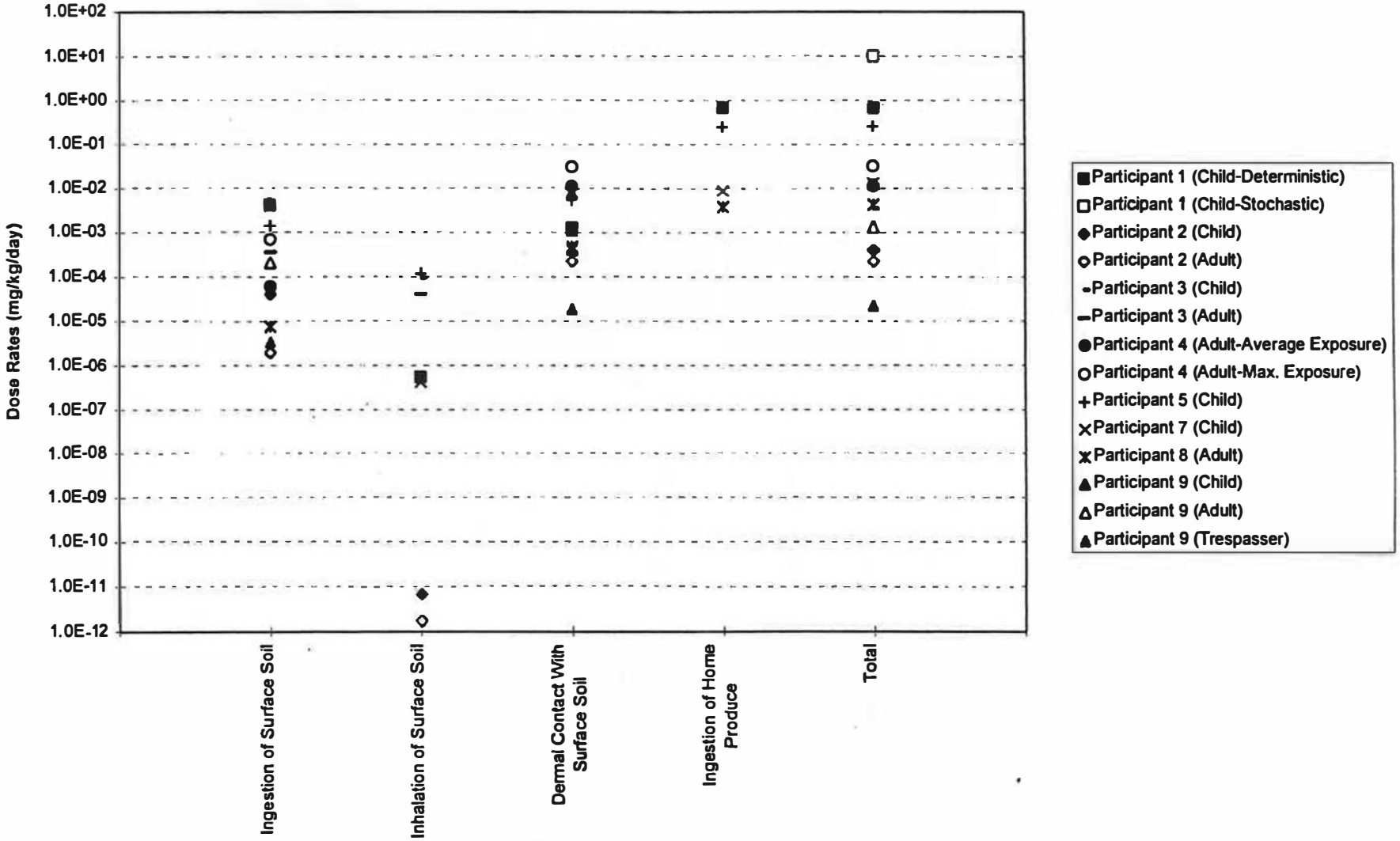


**Figure 12. Dose Rates for Lead Exposure (Non-Carcinogenic)**

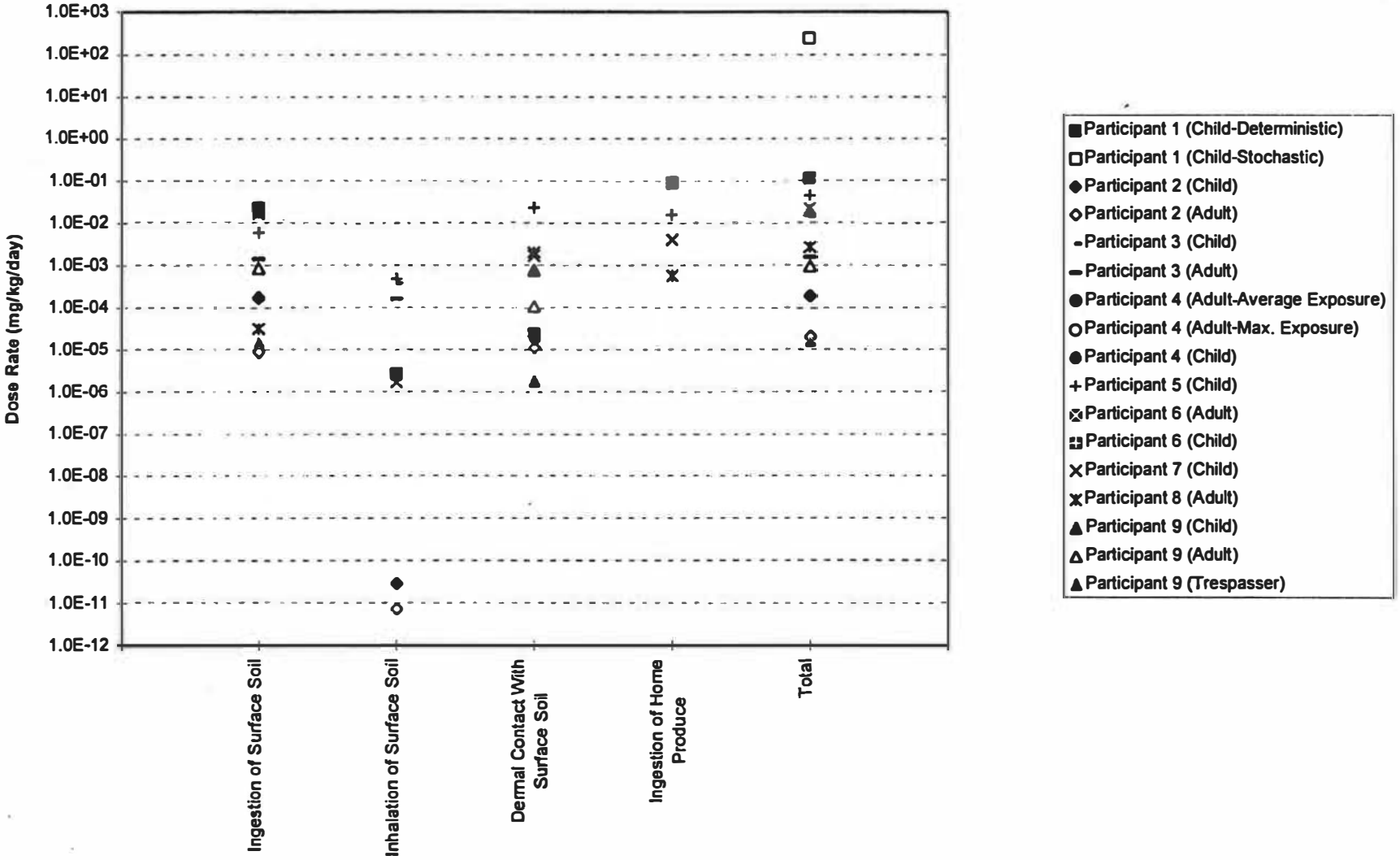


**Figure 13. Dose Rates for Benzene Exposure (Carcinogenic)**

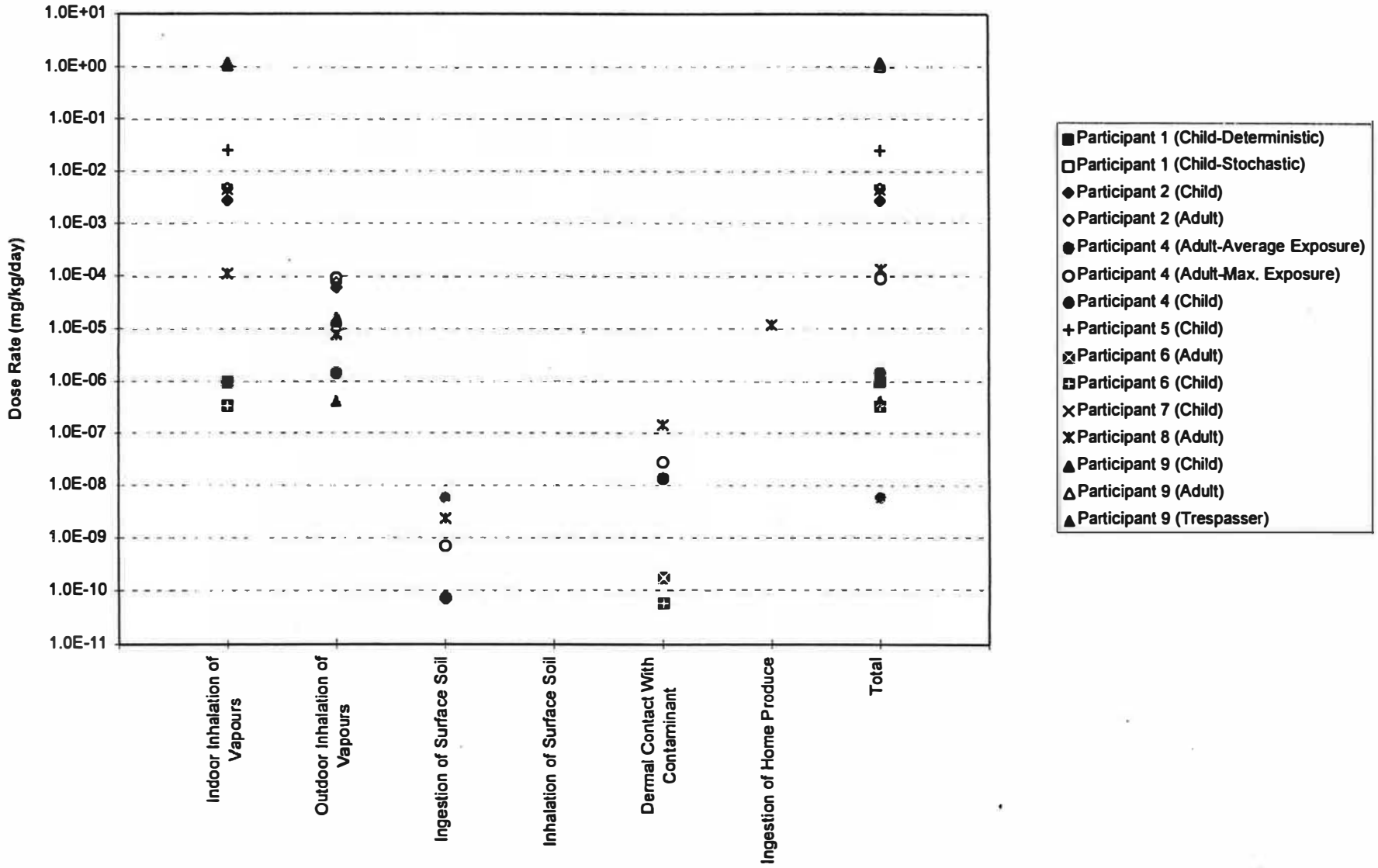
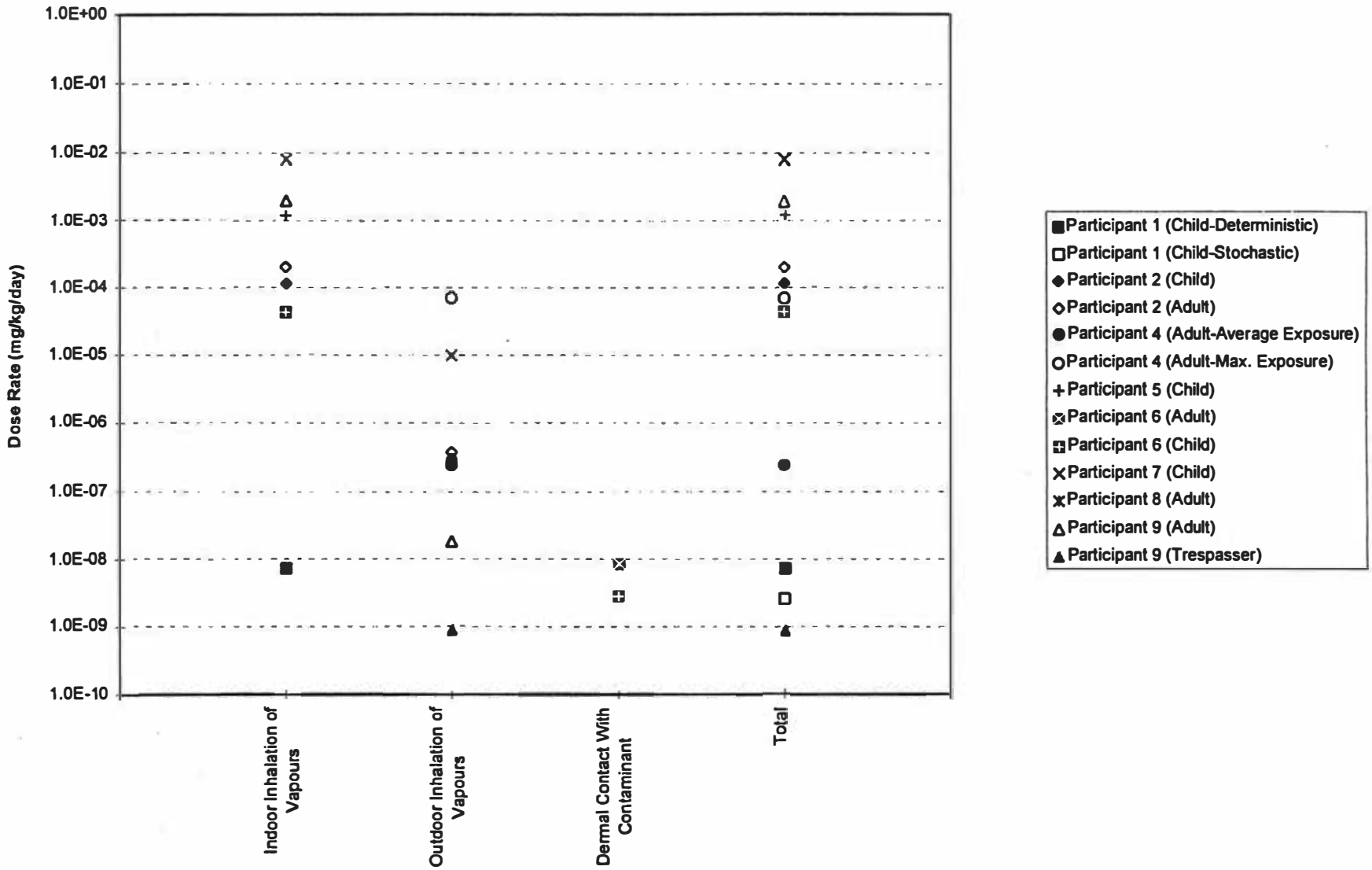
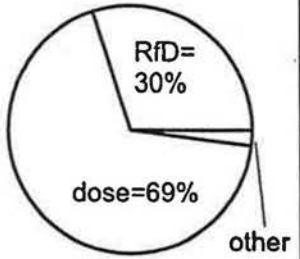
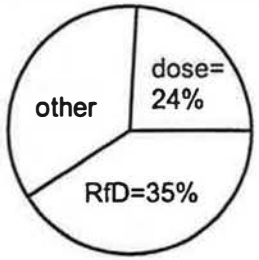
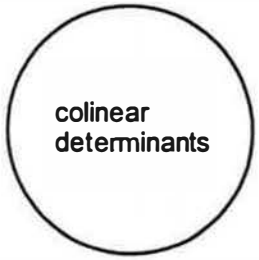
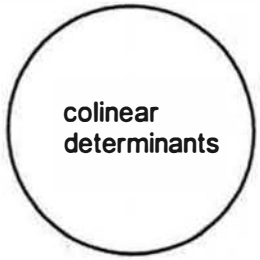
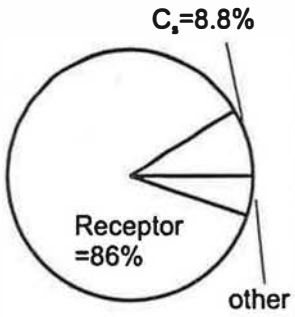
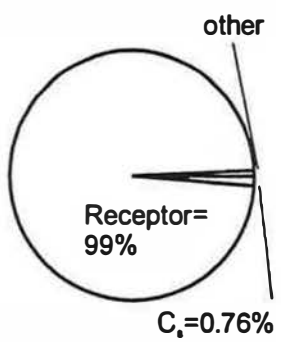
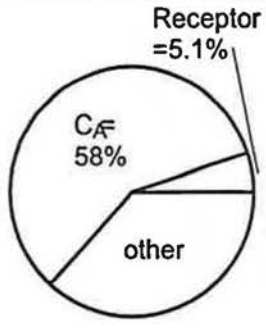
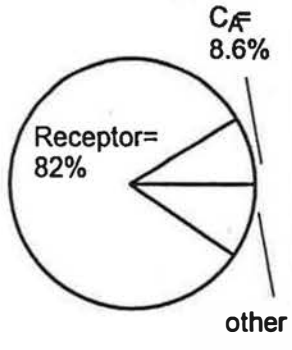
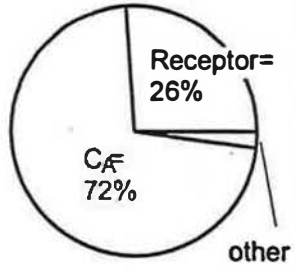


**Figure 14. Dose Rates for Vinyl Chloride Exposure (Carcinogenic)**



| | Soil Ingestion Adult Zinc | Dermal Contact Adult Zinc | Dust Inhalation Child Zinc | Outdoor Soil Gas Inhalation Adult Benzene | Indoor Soil Gas Inhalation Child Benzene |
|---|---|---|---|---|---|
| Risk Estimate |  |  |  |  |  |
| Dose Estimate |  |  |  |  |  |

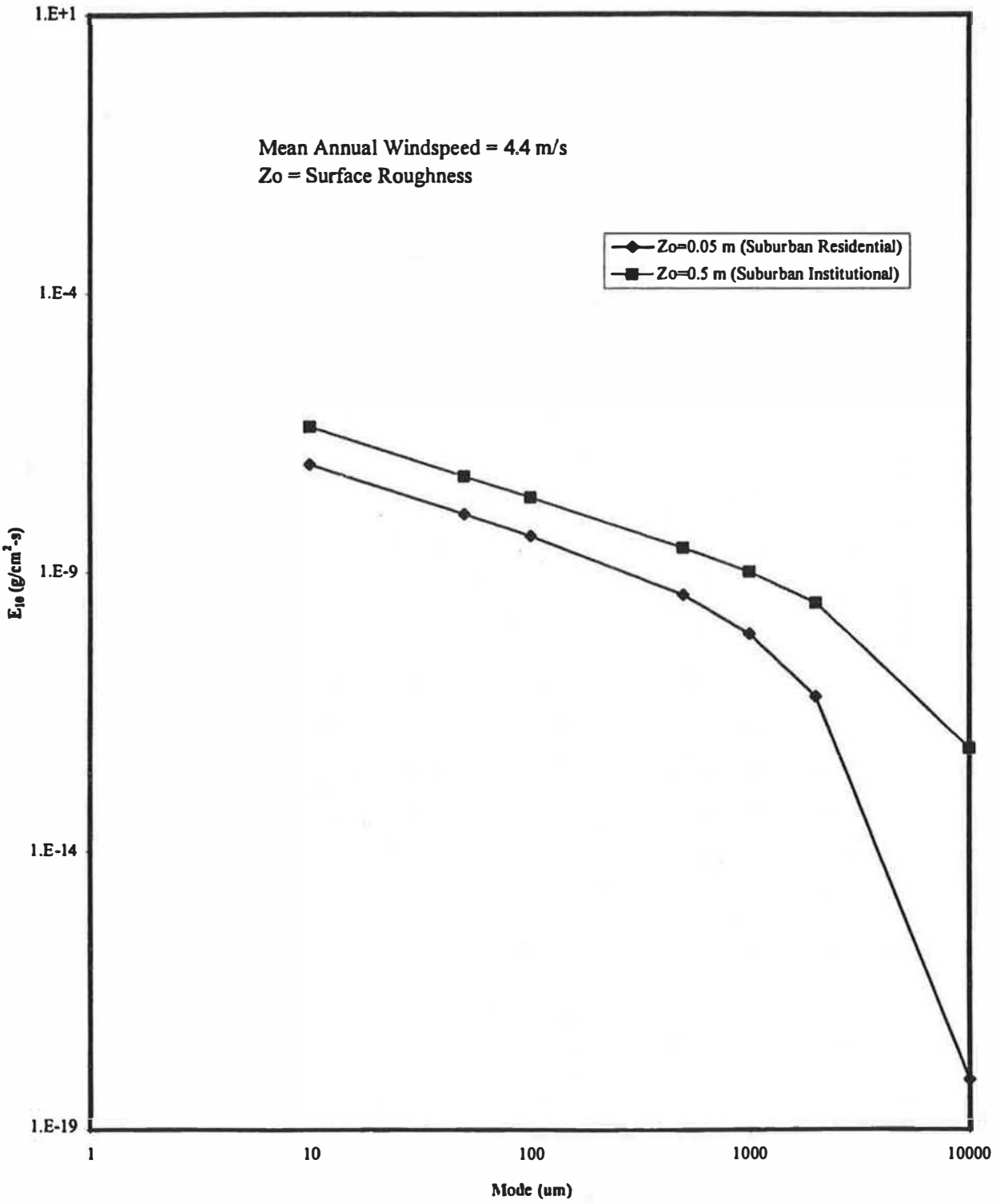
**$C_A$** - Concentration in air
**$C_s$** - Concentration in soil
**RfD** - Toxicity reference value
**SF** - Slope factor
**Receptor** - Receptor characteristics

Golder Associates

## COMPONENT VARIABILITY FOR RISK AND DOSE ESTIMATES

Figure 15

**Figure 16: Particulate Emission Rate Versus Soil Mode Based on Cowherd's Unlimited Erosion Potential Model (Cowherd et al., 1985)**



# APPENDIX I

# INTERIM RESULTS OF PHASE I STUDY

**TECHNICAL MEMORANDUM ON**

# INTERIM REPORT: RESULTS OF RISK ASSESSMENT SURVEY (PHASE I)

Submitted to:

Canada Mortgage and Housing Corporation (CMHC)
National Office
700 Montreal Road
Ottawa, ON
K1A 0P7

Attention: Mr. Don Fugler

DISTRIBUTION:

| | |
|---|---|
| 2 copies - | CMHC<br>Ottawa, Ontario |
| 2 copies - | Golder Associates Ltd.<br>Burnaby, B.C. |

March 3, 1997 962-1828

## 1.0 INTRODUCTION

The Canada Mortgage and Housing Corporation (CMHC) retained Golder Associates Ltd. (Golder) to conduct a study of human health risk assessment practices of contaminated sites in Canada. The study was structured in two phases. Phase I consisted of a survey of practitioners in the private sector and regulatory sectors. The intent of the private sector survey was to characterize the capabilities and experience of private firms. Insight gained from this survey was used to assist in the selection of ten participants for a Round Robin Study, which constituted Phase II of the project. The intent of the regulatory survey was to gain insight on a regional basis in regards to regulator experience and acceptance of human health risk assessment of contaminated sites. This interim report provides the results of the Phase I surveys.

## 2.0 METHODOLOGY

Two surveys were developed by Golder with review and input from CMHC. The surveys were similar but differed slightly to reflect private sector versus regulatory audiences (Attachment 1a, b).

### 2.1 Private Sector Survey

The private sector survey was designed to first characterize the training of persons conducting risk assessments (RAs) by determining the academic qualifications of each firm's risk assessment team. The survey then focused on the risk assessment experience the firm has accumulated, by assessing the number and type of relevant projects conducted over the past five years. Finally, the survey addressed the technical capabilities and experience of each firm through a series of questions focusing on technical issues in human health risk assessment. Questionnaires were sent to practitioners based on a compilation of industry contacts identified by both CMHC and Golder. Analysis of the survey was based on simple descriptive statistics only.

A subset of the questions (not revealed to the participants) was used for scoring purposes to provide an objective method of ranking the overall experience and capabilities of the practitioners. This score assisted in selecting participants for the Round Robin Study

(Phase II) but was not the sole basis for selection. The scoring criteria are listed in Attachment 1c.

### 2.2 Regulatory Survey

The survey of regulatory officials was simpler in nature and addressed issues such as whether human health risk assessments were allowed (regulated, policy or otherwise) in the management of contaminated sites, and if so, whether certain technical methods have were either allowed or encountered in the past. As with the private sector survey, only simple descriptive statistics were used to analyze the regulatory survey results.

## 3.0 SURVEY RESULTS

### 3.1 Private Sector Survey

Of the approximately 100 questionnaires sent, 25 were completed and returned. While this response rate was somewhat low, it appears to be a consequence of several factors. The initial mailing list was overly presumptive in that the identified audience did not consist entirely of bona-fide human health risk assessment practitioners. For instance, a number of firms sub-contract risk assessment services and consequently did not respond to the survey, in spite of the questionnaire providing for such arrangements.

The nature of the technical questions in the survey likely were a deterrent to individuals not intimately involved in human health risk assessments. Other reasons for not responding included lack of interest, lack of staff availability, and concerns over confidentiality.

When interpreting the survey results, the following points must be considered:

- The various HHRA (Human Health Risk Assessment) practitioners were self-evaluated and not evaluated by an independent party;
- The respondents represented a sub-set of HHRA practitioners, and the sub-set may not entirely represent the technical capabilities of the larger group; and

- The apparent capabilities of the respondents reported in the survey was not necessarily indicative of the quality of work performed by the respondents.

With regard to in-house capabilities (Question 2), most of the firms indicated that they have broad expertise. Out of nine major disciplines considered relevant to human health risk assessment, most of the respondents have staff with academic qualifications in 5 or 6 of these disciplines (Figure 1). Eight percent of respondents have staff members with academic qualifications in all nine disciplines. The predominant disciplines were biology, environmental engineering, chemical fate and transport, and hydrogeology (Figure 2). The least predominant disciplines were statistics and analytical chemistry. Some of the firms also indicated that they have in-house risk communication specialists, meteorologists, chemical engineers, process engineers, and land use specialists. Many of the firms also indicated that they supplement their in-house HHRA capabilities with external consultants with varying qualifications.

The questionnaire asked participants to indicate the number of risk assessments that they have performed at contaminated sites over the past five years (Question 3, Figure 3). The results suggest that a wide range of experience exists between firms:

- 16% of respondents have not performed qualitative RAs at contaminated sites over the past five years;
- 12% have not performed quantitative-deterministic RAs; and
- 32% have not completed quantitative-probabilistic RAs.

However, several firms appear to be quite experienced:

- 36% of respondents have completed more than 15 quantitative-deterministic RAs; and
- 16% of respondents have completed more than 15 quantitative-probabilistic RAs.

The above results may be affected by inconsistent interpretation amongst participants with respect to "qualitative" versus "quantitative" risk assessment projects. For example, most quantitative risk assessment studies invariably involve an initial qualitative phase.

Question 4 addressed the project experience specific to residential sites and is summarized in Figure 4. Close to 50% of the firms indicated they have some level of quantitative-probabilistic experience with residential sites. Approximately 76% of respondents indicated some level of quantitative-deterministic RA experience with residential sites, and about 60% had qualitative RA experience with residential sites over the past five years.

Based on the survey results, most of the respondents appear to be highly experienced in performing risk assessments in Canada with a smaller component being less technically experienced (Figure 5). The majority of firms are able to perform quite sophisticated quantitative assessments. For instance, many of the firms utilize simulation models to predict human exposure to soil gas, groundwater, and dust. In addition, most respondents perform risk assessments of carcinogenic and non-carcinogenic chemicals, evaluate risks from varying exposure duration, conduct sensitivity analyses, and evaluate, design, and implement risk management or remedial strategies.

The respondents with modeling capabilities indicated that they utilize a diverse array of environment fate models to perform exposure assessments. Many of the respondents utilize in-house models to predict the transport of *soil gas* volatile organic carbons. Externally developed soil gas models identified include MEPAS, CALTOX, Farmer's model (U.S. EPA), ASTM RBCA and others. Contaminant fate and transport in *groundwater* is modeled using a combination of in-house models, SOLUTE, MODFLOW, PHREEQE, FLOWPATH, and many others. Respondents model wind and vehicle generated erosion using in-house models, Cowherd's model, U.S. EPA's model, HEC, SEDIMOT, AERIS, AP-42, CAPCOA and others. A very large array of models are used for *air dispersion* modeling. The most common models include ISCST2/3, ISCLT2/3, in-house models, DEGADIS, SCREEN2, and MOEE 308/346.

## 3.2 Score Ranking and Selection Process for Round-Robin Participants

Ten participants have been selected for the second stage (i.e., Round Robin Study) which consisted of undertaking a screening level risk assessment of a hypothetical case study. The participants were selected based on geographic location and apparent risk assessment experience and capabilities as derived from the scoring of select responses.

In order to ensure broad regional representation, firms from each of the following geographic regions were chosen:

1) British Columbia;
2) The prairie provinces (Alberta, Saskatchewan and Manitoba);
3) Ontario and Quebec; and
4) The Maritimes.

The experience and technical capabilities of each firm were ranked based on the questionnaire results (Figure 5 and Table 1). The questionnaire provided qualitative information on each of the firms in-house capabilities, level of experience in various types of risk assessment, and technical capabilities in exposure assessment, modeling, toxicity assessment, risk characterization, and risk management. A total score was derived for each firm based on the results of specific questions that were considered the most relevant (Questions 3, 4, 6c, 7a-j, 8a, 9e, see Attachment 1c). The highest total score possible was 39. In order to incorporate additional variability into the Round Robin, participants with *varying* apparent capabilities were selected. Four participants with a score above 30 were selected, three participants with a score from 20 to 30 were selected, and two participants with a score between 10 and 20 were selected.

## 3.3 Regulatory Survey

Of 19 surveys sent to regulators from both provincial and federal agencies, 11 were completed and returned. Respondents included representatives from the following government agencies:

1) BC Environment
2) Alberta Health
3) Saskatchewan Health
4) Manitoba Environment
5) Ontario Ministry of Environment and Energy
6) Ministère de Environment et de la Faune de Quebec
7) New Brunswick Department of Environment
8) Newfoundland and Labrador Department of Environment and Labour
9) Department of Environment of Nova Scotia
10) Environment Canada.

There was consensus among regulators regarding the use of human health risk assessment in the management of contaminated sites. All of the respondents indicated that their region supports the use of human health risk assessments (Question 1a). In addition, all of the respondents indicated that their risk assessment approach is based on informal policy, and is not included in legislated regulation (Question 1c). However, BC Environment has included human health risk assessment protocols in their Draft Contaminated Site Regulation to the Waste Management Amendment Act (Bill 26). Most of the respondents also indicated that they cooperate with other agencies to integrate policies from other regions (Question 1e).

The level of experience of regulatory agencies in human health risk assessments at contaminated sites was highly variable and the sample size was insufficient to determine any trends (Questions 3 and 4).

All of the respondents who completed Question 5 (two of the respondents did not answer this question) indicated that their agency allowed the use of a tiered risk assessment approach where risk-based criteria are developed for specific exposure pathways.

With regards to problem formulation, most of the agencies required that both human and ecological receptors were considered, and most of the agencies allowed screening of chemical concentrations (Question 7).

With regards to exposure assessment and modeling (Question 8), all of the respondents who answered this question, specified that their agency allowed the use of exposure-specific bioavailabilities for chemicals and incremental risk. However, not all the agencies prescribed to the use of composite receptors, microenvironments, and probabilistic methods for exposure assessment. There was considerable variability in the answers to Question 8f-c. Some of the agencies provided guidance for the use of exposure models to predict contaminant fate for all pathways specified in these questions, some of the agencies provided limited guidance, while others did not provide any guidance.

There was also considerable variation in the source of toxicity reference values suggested by the agencies (Question 9). Some prescribed to the use of externally derived toxicity

reference values (TRVs), Health Canada TRVs, and non-Canadian TRVs, while others did not. Some of this variability could be attributed to the availability or lack of provincially derived TRVs. However, the results suggest that most of the agencies do use Health Canada's system for classifying carcinogens in conjunction with other systems such as IARC or U.S. EPA.

## 4.0 CONCLUSIONS

As part of the first phase of the CMHC study, questionnaires were sent to several Canadian human health risk assessment practitioners and regulators. The intent of the surveys was to characterize private firms' capabilities and expertise, and regulators' experience and acceptance of human health risk assessments of contaminated sites. In addition, private sector respondents were scored based on their responses to specific questions regarded as most relevant. Based on the scores and geographic location of the respondents, nine practitioners were selected to participate in a round-robin risk assessment (Phase II).

The results of the private sector survey suggest that practitioners have broad expertise in relevant disciplines such as toxicology, biology, environmental engineering, chemistry, and hydrology. Many of the firms supplement their in-house capabilities with external consultants. Many of the firms also have a high level of expertise modeling the fate of contaminants in soil gas, groundwater, fugitive dust and air.

The results of the regulatory survey indicate that governmental agencies support the use of human health risk assessment in the management of contaminated sites. However, the approach is generally based on informal policy and is not formally regulated.

There appears to be a high level of variability in terms of private sector expertise and technical capabilities as well as regulator experience and policy in the field of human health risk assessment. Based on this assessment, we would expect both regional and between firm variability in the type of risk assessments performed in Canada and their accompanying risk estimates. This variability has significant implications for business expenditures associated with re-development and the overall state of the Canadian environment.

In order to further understand and characterize the variability in risk estimates produced by different practitioners, Golder/CMHC will undertake Phase II of the risk assessment study. As part of Phase II, nine Canadian practitioners from various regions in Canada with varying levels of expertise performed a screening level risk assessment of a hypothetical case study. The case study consisted of a residential exposure scenario designed to re-create a situation that might be encountered in the real world. The purpose of the Phase II study was be to assess the degree of variability in risk estimates among participants, and analyze the sources of variability and uncertainty.

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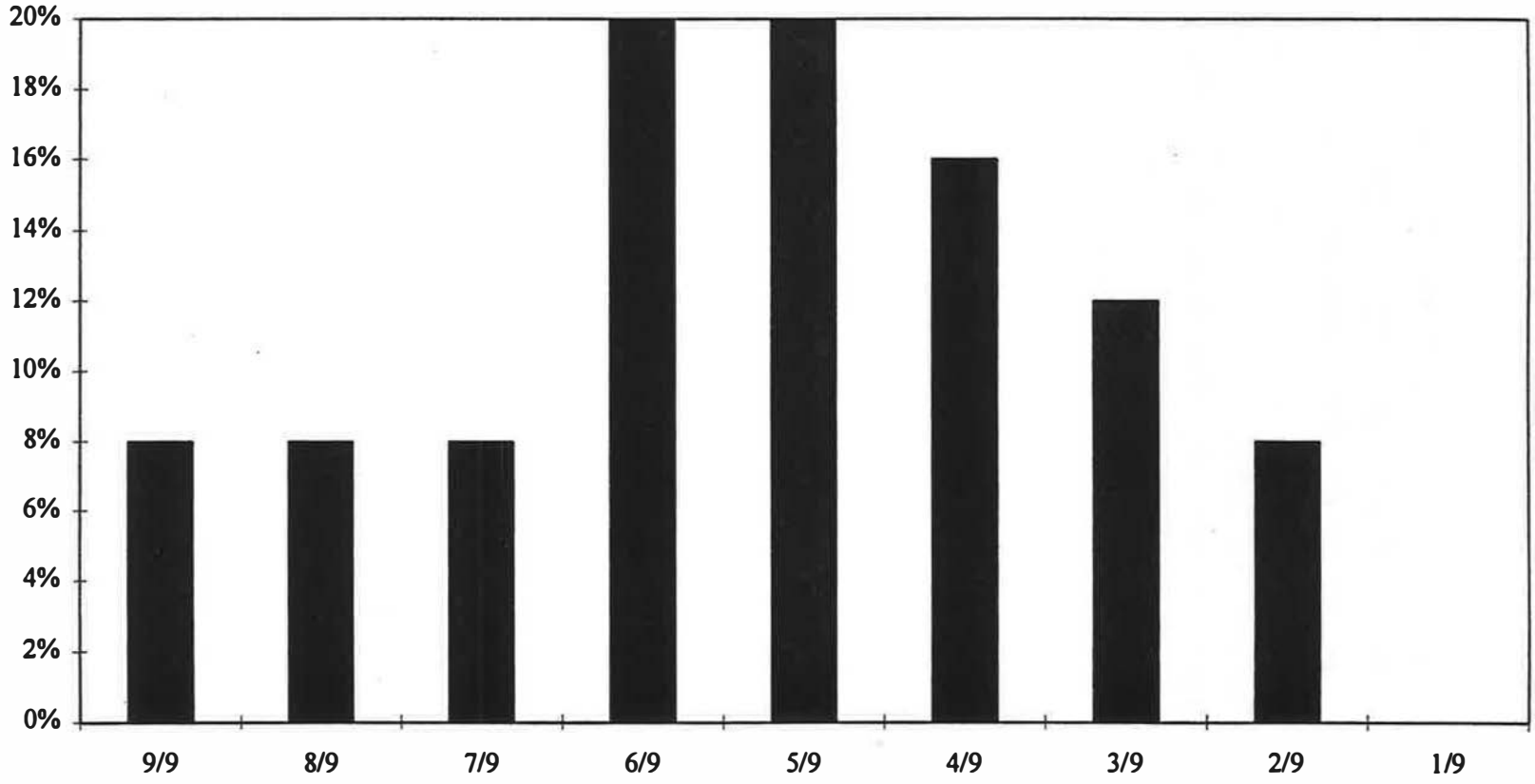
Figure 1. Percentage of respondents with in-house capabilities for 9 risk assessment related disciplines[1].




[1]Toxicologist (human health), toxicologist (ecological), biologist, chemist (chemical fate and transport), chemist (analytical), statistician, hydrogeologist, environmental engineer, soil scientist

Figure 2. Percent of respondents with in-house capabilities

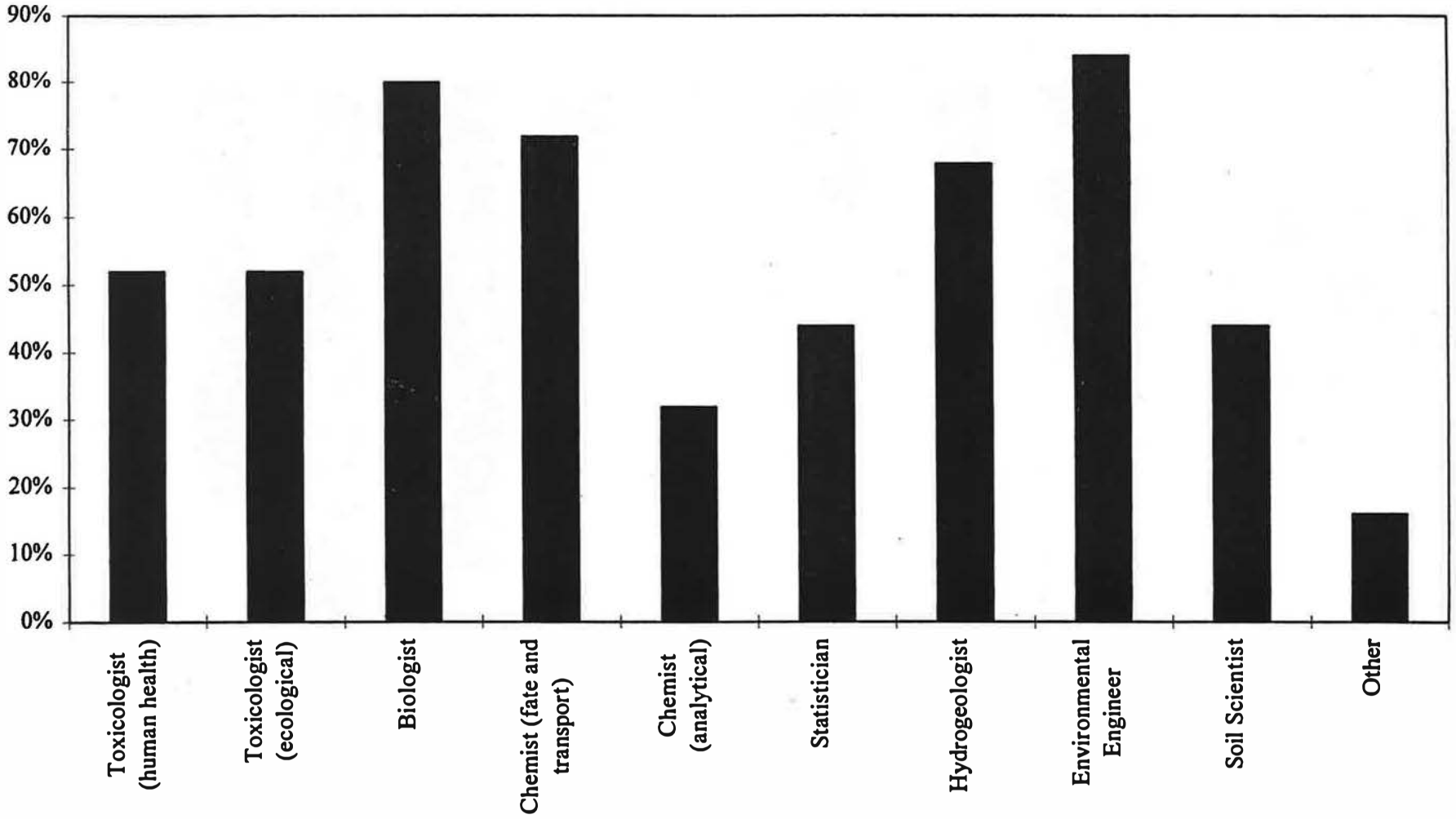


Figure 3. Percent of respondents that conducted human health risk assessments at contaminated sites within last 5 years

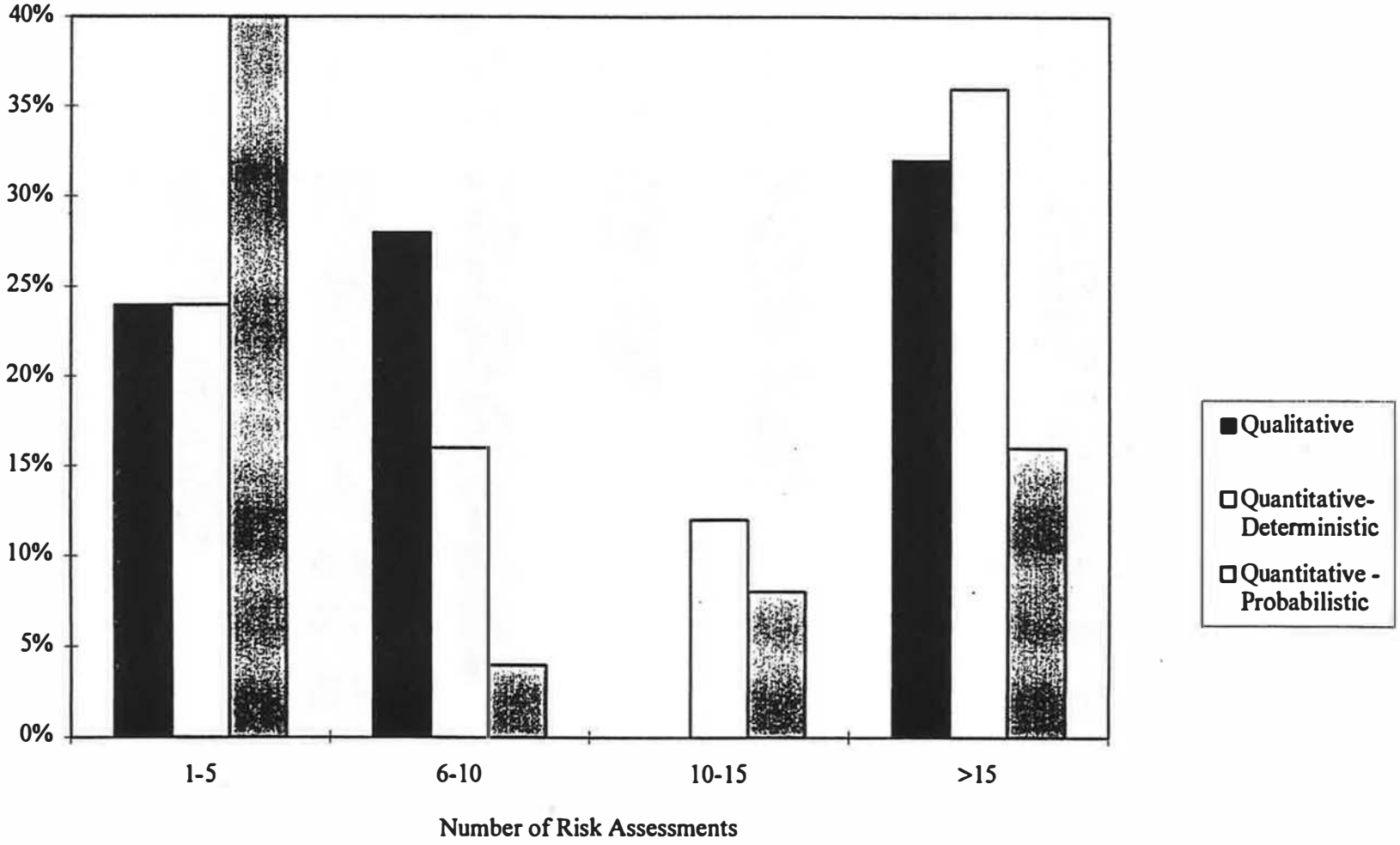


Figure 4. Percent of respondents that conducted human risk assessments (in the last 5 years) at contaminated sites which included a residential land-use scenario

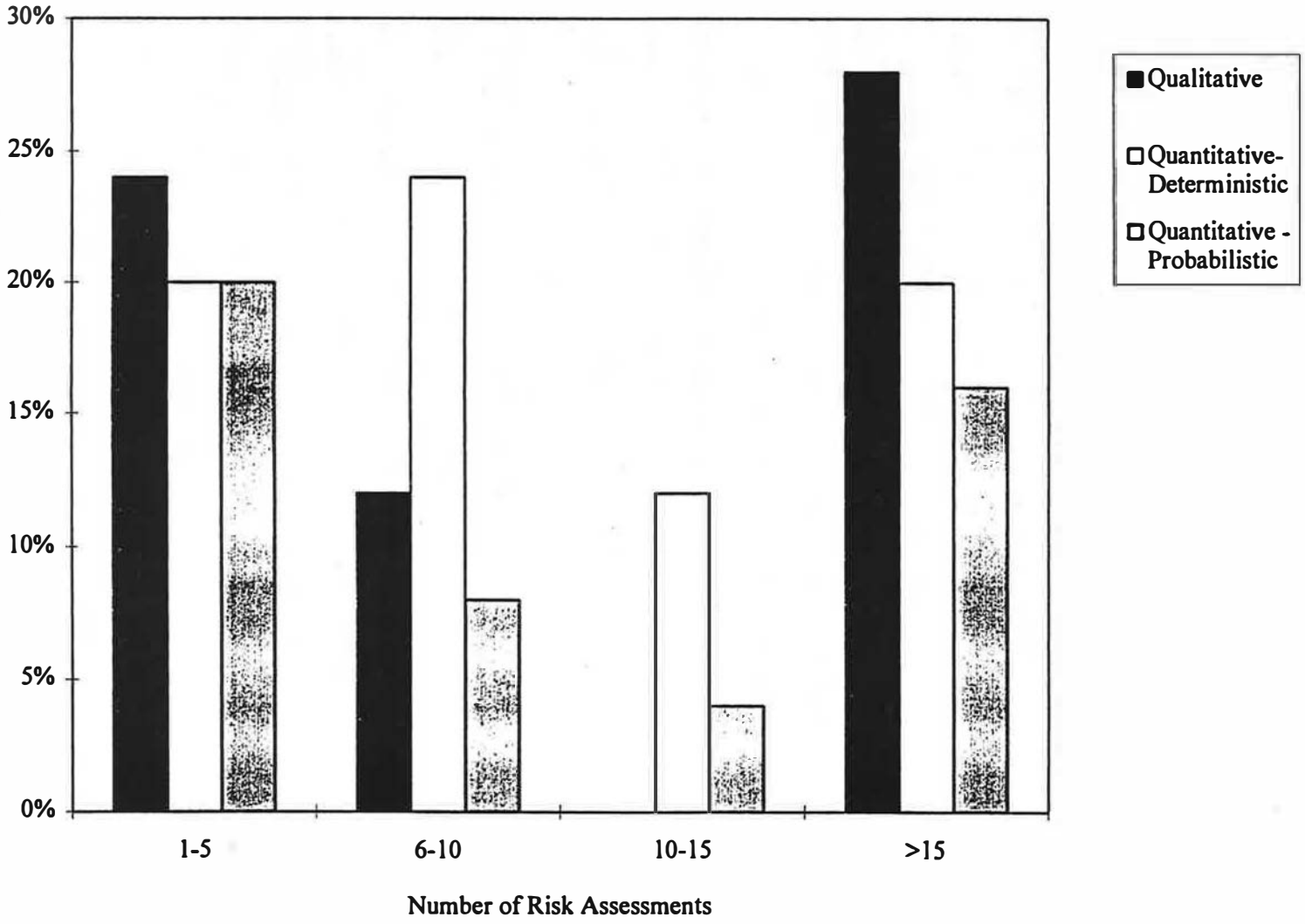


Figure 5. Summary of questionnaire responses

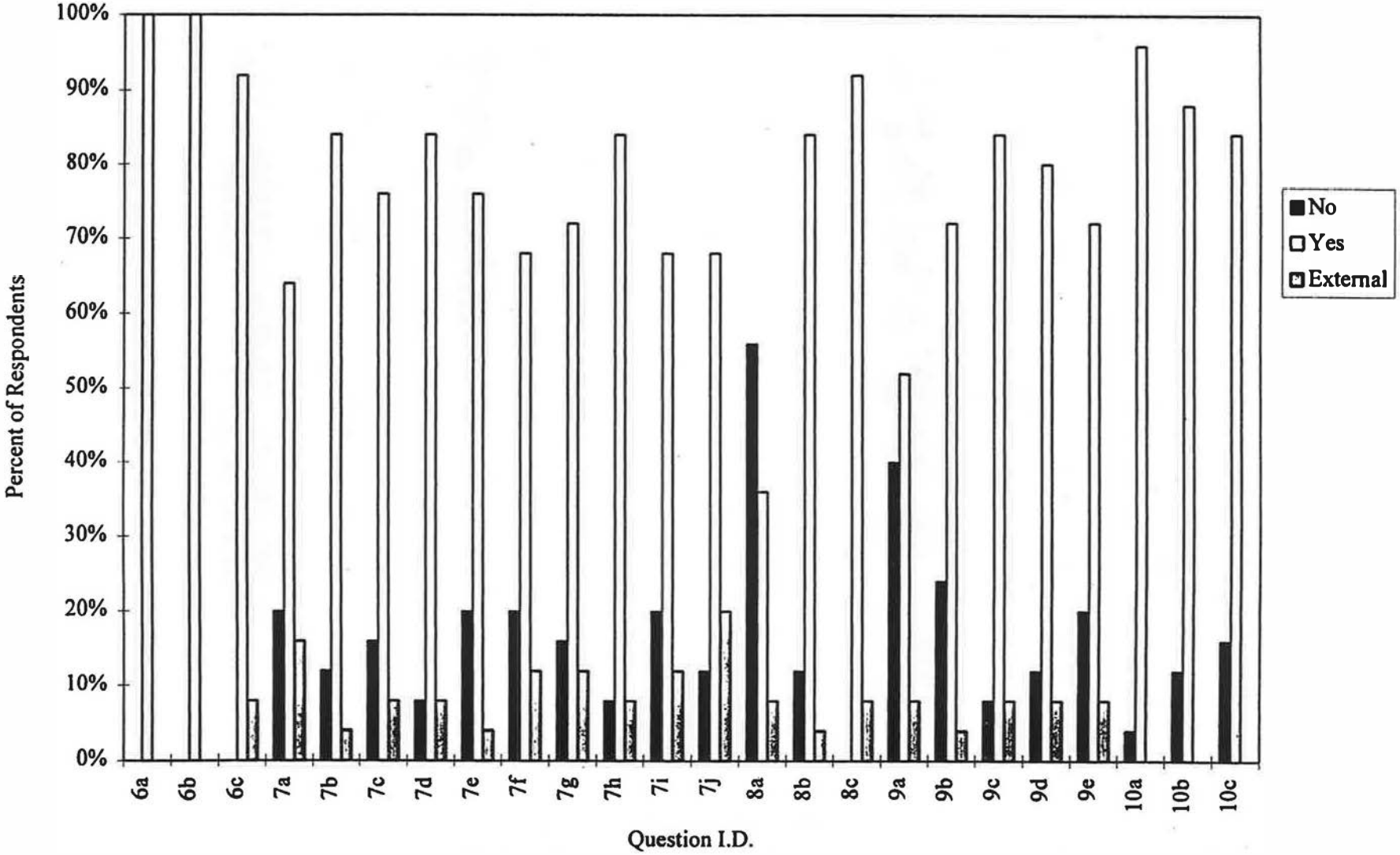


Figure 6. Total score of each respondent

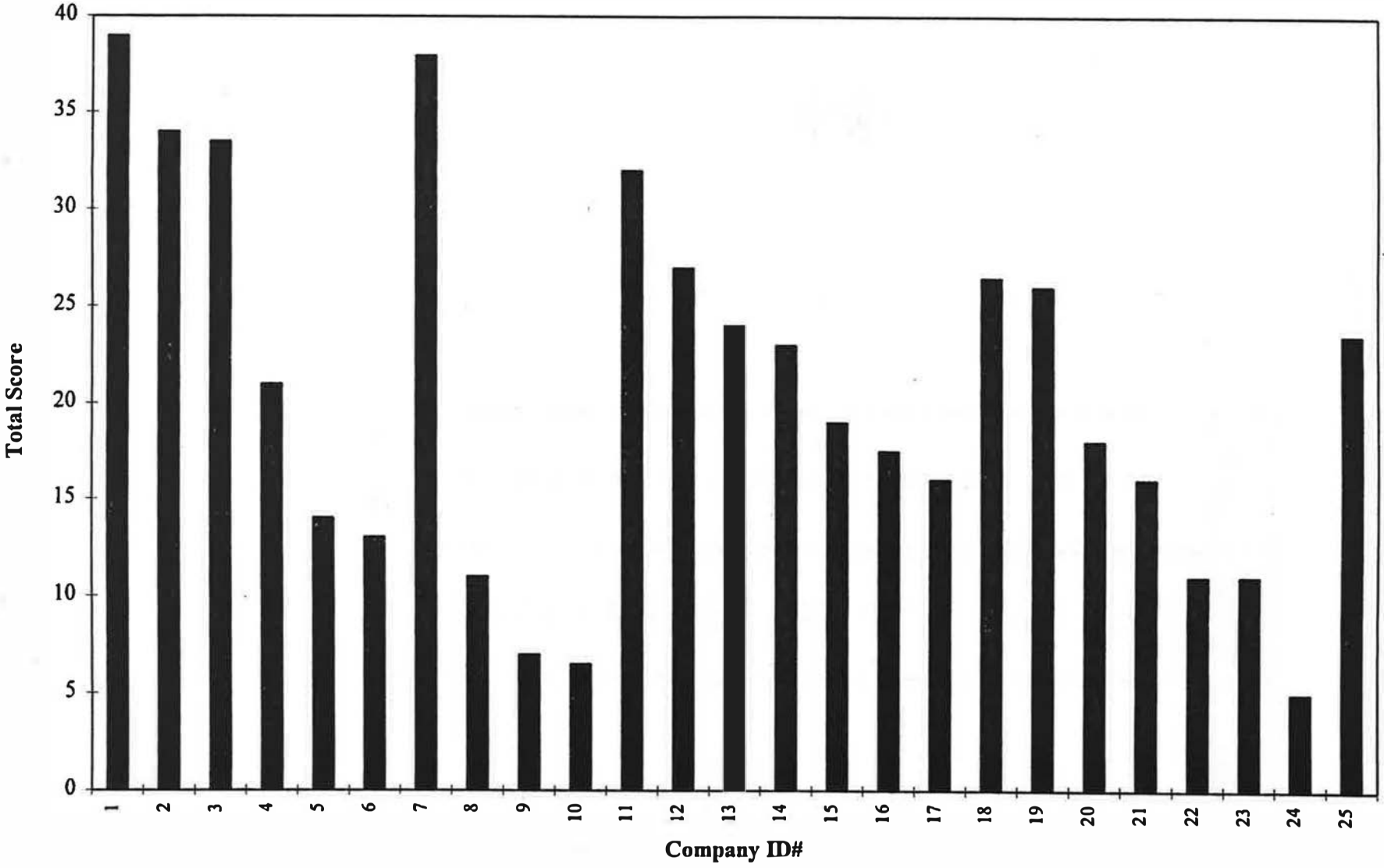


Table 1. Summary of total scores and ranks based on questionnaire results.

| Company ID# | TOTAL | RANK |
|---|---|---|
| 1 | 39 | 1 |
| 2 | 34 | 3 |
| 3 | 33.5 | 4 |
| 4 | 21 | 12 |
| 5 | 14 | 18 |
| 6 | 13 | 19 |
| 7 | 38 | 2 |
| 8 | 11 | 20 |
| 9 | 7 | 23 |
| 10 | 6.5 | 24 |
| 11 | 32 | 5 |
| 12 | 27 | 6 |
| 13 | 24 | 9 |
| 14 | 23 | 11 |
| 15 | 19 | 13 |
| 16 | 17.5 | 15 |
| 17 | 16 | 16 |
| 18 | 26.5 | 7 |
| 19 | 26 | 8 |
| 20 | 18 | 14 |
| 21 | 16 | 16 |
| 22 | 11 | 20 |
| 23 | 11 | 20 |
| 24 | 5 | 25 |
| 25 | 23.5 | 10 |

## Attachment 1a. CMHC Human Health Risk Assessment Questionnaire for Practitioners

**COMPANY INFORMATION**

**INTRODUCTION:**

The following questionnaire is presented to develop a general profile on the technical approaches of risk assessment practitioners. Additionally, it will provide an objective basis for selection of participants in a Round-Robin Risk Assessment study. Confidentiality is ensured on all information and subsequent presentation of results. There is no intent to solicit confidential business information; should you not wish to respond to some questions on this basis, please indicate "CBI".

**PART I**

**The following section is designed to provide general information on firms which conduct human health risk assessments in order to relate this to subsequent responses or technical questions in Part II.**

1. Please indicate the number of years your company has actively been conducting human health risk assessments of contaminated sites.

☐

***For the following sections, please respond with a checkmark in applicable boxes.***

2. Please indicate capabilities in terms of the primary academic qualifications of staff conducting risk assessments.

| Staff | In-House Capabilities | | | External* |
|---|---|---|---|---|
| | Ph.D. | Masters | Bachelors | Capabilities |
| Toxicologist, Human Health | | | | |
| Toxicologist, Ecological | | | | |
| Biologist | | | | |
| Chemist, Fate and Transport | | | | |
| Chemist, Analytical | | | | |
| Statistician | | | | |
| Hydrogeologist | | | | |
| Environmental Engineer | | | | |
| Soil Scientist | | | | |
| Other (please list) | | | | |

* Subconsultants which your company uses for risk assessment projects.

3. Please indicate the total number of ***human health*** risk assessments your risk assessment team has conducted (in the last 5 years) at contaminated sites specified by type of risk assessment.

| Type of Risk Assessment (size/scope) | Number of Risk Assessments | | | |
|---|---|---|---|---|
| | 1-5 | 6-10 | 10-15 | >15 |
| Qualitative | | | | |
| Quantitative - Deterministic | | | | |
| Quantitative - Probabilistic | | | | |

4. Please indicate the number of ***human health*** risk assessments your risk assessment team has conducted (in the last 5 years) at contaminated sties which have included a *residential* (i.e., housing) land-use scenario.

| Type of Risk Assessment (size/scope) | Number of *Residential* Risk Assessments | | | |
|---|---|---|---|---|
| | 1-5 | 6-10 | 10-15 | >15 |
| Qualitative | | | | |
| Quantitative - Deterministic | | | | |
| Quantitative - Probabilistic | | | | |

5. Please indicate **(with a checkmark)** the different regions of Canada and client types for which human health risk assessments have been conducted by your risk assessment team.
For each region, please indicate **(with an "X")** who prompted HHRA instead of clean-up to criteria levels.

| Client Type | B.C. | | Prairies | | Ontario | | Quebec | | Maritimes | | Territories | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | ✓ | × | ✓ | × | ✓ | × | ✓ | × | ✓ | × | ✓ | × |
| Real Estate Developers | | | | | | | | | | | | |
| Industry | | | | | | | | | | | | |
| Government | | | | | | | | | | | | |

**PART II**

**The following questions are intended to survey the technical procedures incorporated in human health risk assessments**

**conducted by your risk assessment team. Please indicate whether your team has used these procedures in HHRA.**

| | Your Firm Yes | Your Firm No | External Consultant |
|---|---|---|---|
| ***6. Problem Formulation*** | | | |
| a) In conducting risk assessments at contaminated sites, do you consider both human and ecological receptors? | ☐ | ☐ | ☐ |
| b) Do you screen site chemical concentrations against applicable criteria? | ☐ | ☐ | ☐ |
| c) Do you develop a conceptual exposure model of the problem formulation? | ☐ | ☐ | ☐ |
| ***7. Exposure Assessment and Modelling*** | | | |
| Has your risk assessment team conducted risk assessments where: | | | |
| a) "composite receptors" have been selected for exposure assessments? (a composite receptor differs from age-specific receptors in that it reflects physiological traits over the corresponding age classes during maturation from childhood to adult) | ☐ | ☐ | ☐ |
| b) the bioavailability of chemicals have been estimated? | ☐ | ☐ | ☐ |
| c) microenvironments have been considered? | ☐ | ☐ | ☐ |
| Does your risk assessment team typically: | | | |
| d) estimate incremental risk (i.e., health risk from exposure beyond background exposure) for a chemical of concern? | ☐ | ☐ | ☐ |
| e) consider temporal variations of contaminants? | ☐ | ☐ | ☐ |
| Has your risk assessment team conducted risk assessments where exposure models were used to: | | | |
| f) predict soil gas volatile organic compounds fate and transport to *indoor* air? | ☐ | ☐ | ☐ |
| If yes, please specify which models (i.e., name of product or in-house) _____ | | | |
| g) predict soil gas volatile organic compounds fate and transport to *outdoor* air? | ☐ | ☐ | ☐ |
| If yes, please specify which models (i.e., name of product or in-house) _____ | | | |
| h) predict contaminant fate and transport in groundwater? | ☐ | ☐ | ☐ |
| If yes, please specify which models (i.e., name of product or in-house) _____ | | | |
| i) predict dust generation through wind erosion or vehicle generated erosion? | ☐ | ☐ | ☐ |
| If yes, please specify which models (i.e., name of product or in-house) _____ | | | |
| j) predict contaminant fate and transport in air dispersion modelling? | ☐ | ☐ | ☐ |
| If yes, please specify which models (i.e., name of product or in-house) _____ | | | |

k) For the various model input parameters noted in items (f) through (j), please indicate what values are *typically* used:
arithmetic/geometric mean___ 95th percentile___ 99th percentile___ 99.9th percentile___ range probability distribution function___

l) Briefly list the main input variables for indoor air modelling (e.g., air exchange rate, building height, etc.)

_____

m) Briefly list the main input variables for outdoor air modelling (e.g., meteorological parameters, soil parameters, etc.)

_____

| | | | |
|---|---|---|---|
| ***8. Toxicity Assessment*** | | | |
| Please indicate which toxicity reference values are used by your risk assessment team: | | | |
| a) Toxicity reference values derived in-house? | ☐ | ☐ | ☐ |
| b) RfD's or RsD's from Health Canada? | ☐ | ☐ | ☐ |
| c) RfD's or slope factors from non-Canadian agencies (e.g., IRIS, HEAST, WHO)? | ☐ | ☐ | ☐ |
| ***9. Risk Characterization*** | | | |
| Please indicate how your risk assessment team characterizes risk: | | | |
| a) an exposure ratio value approach? | ☐ | ☐ | ☐ |
| b) a hazard quotient approach? | ☐ | ☐ | ☐ |
| c) a numerical cancer risk estimate approach? | ☐ | ☐ | ☐ |
| d) Have you conducted a risk assessment where it was necessary to evaluate risk from short exposure duration's as well as lifetime exposure? | ☐ | ☐ | ☐ |
| e) Have you conducted a risk assessment where it was necessary to conduct sensitivity analyses? | ☐ | ☐ | ☐ |
| ***10. Risk Management*** | | | |
| a) Have you evaluated risk management or remedial options? | ☐ | ☐ | ☐ |
| b) Have you designed and implemented risk management or remedial designs? | ☐ | ☐ | ☐ |
| c) In the projects which your team has conducted, is monitoring typically required? | ☐ | ☐ | ☐ |

11. Please use this space and any additional pages necessary to provide other comments which you feel may be of assistance.

_____

_____

_____

## Attachment 1b. CMHC Human Health Risk Assessment Questionnaire for Regulatory Agencies

### REGULATORY INFORMATION

**PART 1: The following section is designed to characterize agency policy and regulatory experience with human health risk assessment.**

***For the following sections, please respond with a checkmark in applicable boxes.***

1. a) Please indicate whether your region supports the use of human health risk assessments in the management of contaminated sites. Yes _____ No _____

   b) If no, please go to Question 10 and describe how your regulatory agency approaches management and remediation of contaminated sites.

   c) Is this an informal policy or a legislated regulation?

   Informal Policy _____ Regulation _____

   d) If this is a legislated policy, please cite the appropriate act, regulation and/or guideline.

   _____

   e) Briefly, how does your agency's policy integrate with other regionally applicable policies/regulations (e.g., cooperative, supersedes others, etc.)

   _____

   _____

2. Please indicate the number of years your agency has actively been reviewing human health risk assessments of contaminated sites. ☐

3. Please indicate the total number of human health risk assessments your agency has received (in the last 5 years) for contaminated sites specified by type of risk assessment.

| Type of Risk Assessment (size/scope) | Number of Risk Assessments 1-5 | 6-10 | 10-15 | >15 |
|---|---|---|---|---|
| Qualitative | | | | |
| Quantitative - Deterministic | | | | |
| Quantitative - Probabilistic | | | | |

4. Please indicate the number of human health risk assessments your agency has received (in the last 5 years) for contaminated sites which have included a residential (i.e., housing) land-use scenario.

| Type of Risk Assessment (size/scope) | Number of *Residential* Risk Assessments 1-5 | 6-10 | 10-15 | >15 |
|---|---|---|---|---|
| Qualitative | | | | |
| Quantitative - Deterministic | | | | |
| Quantitative - Probabilistic | | | | |

5. Over the past decade, a generic numerical concentration approach and site-specific risk assessment approach emerged as two options for contaminated sites management. Does your agency also allow the use of a tiered risk assessment approach where risk-based criteria are developed for specific exposure pathways? Yes _____ No _____

6. With regard to the acceptable risk levels used in your region, does your agency support the use of
   $1 \times 10^{-6}$ for an acceptable lifetime cancer risk (LCR)? Yes _____ No _____
   1 for a hazard quotient (HQ)/exposure ratio (ER)? Yes _____ No _____

If your region does not support the use of the above risk levels, please specify which risk levels your region considers acceptable: _____

**PART II:**

**The following questions are intended to determine the technical procedures incorporated in human health risk assessments accepted by your region.**
**Please indicate whether your agency approves of these procedures in human health risk assessments.**

| | Yes | No | N/A |
|---|---|---|---|
| ***7. Problem Formulation*** | | | |
| a) Does your agency require consideration of both human and ecological receptors? | | | |
| b) Does your agency allow screening of chemical concentrations. | | | |
| ***8. Exposure Assessment and Modelling*** | | | |
| Does your agency allow use of: | | | |
| a) a composite receptor in conducting a human health risk assessment? | | | |
| (a composite receptor differs from age-specific receptors in that it reflects physiological traits over the corresponding age classes during maturation from childhood to adult) | | | |
| b) exposure-specific bioavailabilities for chemicals? | | | |
| c) microenvironments? | | | |
| d) incremental risk (i.e., health risk from exposure beyond background exposure)? | | | |
| e) probabilistic methods to be employed in the exposure assessment? | | | |
| Does your agency provide specific guidance for the use of exposure models to: | | | |
| f) predict soil gas volatile organic compounds fate and transport to *indoor* air? | | | |
| g) predict soil gas volatile organic compounds fate and transport to *outdoor* air? | | | |
| h) predict contaminant fate and transport in groundwater? | | | |
| i) predict dust generation through wind erosion or vehicle generated erosion? | | | |
| j) predict contaminant fate and transport in air dispersion modelling? | | | |
| k) Does your agency specify models to be used for the above? If so, please specify: _____ | | | |
| l) Does your agency specify exposure factors for receptors? If so, are they from CEPA? If not, please specify the source below: _____ | | | |
| ***9. Toxicity Assessment*** | | | |
| Please indicate which toxicity reference values are suggested for use: | | | |
| a) Does your agency allow derivation of toxicity reference values? | | | |
| b) Does your agency specify use of RfD's from Health Canada? | | | |
| c) Does your agency allow use of RfD's from non-Canadian agencies (e.g., IRIS, HEAST, WHO?) | | | |
| Please specify the sources and hierarchy of preference for toxicity reference values employed in health risk assessments: _____ | | | |
| d) In regards of possible carcinogenic potential, please specify the classification system to which your agency subscribes: Health Canada _____ IARC _____ U.S.EPA _____ Other _____ | | | |
| e) Does your agency allow *probabilistic* methods to be employed in the *toxicity assessment*? | | | |

10. Please use this space and any additional pages necessary to provide other comments which you feel may be of assistance.

_____
_____
_____
_____
_____

Attachment 1c. Scoring of private sector survey results

| Question | Response | Score |
|---|---|---|
| 3 | Qualitative (0 RAs) | 0 |
| | Qualitative (1-5 RAs) | 1 |
| | Qualitative (6-10 RAs) | 2 |
| | Qualitative (10-15 RAs) | 3 |
| | Qualitative (>15 RAs) | 4 |
| | | |
| | Quantitative-Deterministic (0 RAs) | 0 |
| | Quantitative-Deterministic (1-5 RAs) | 1 |
| | Quantitative-Deterministic (6-10 RAs) | 2 |
| | Quantitative-Deterministic (10-15 RAs) | 3 |
| | Quantitative-Deterministic (>15 RAs) | 4 |
| | | |
| | Quantitative-Probabilistic (0 RAs) | 0 |
| | Quantitative-Probabilistic (1-5 RAs) | 2 |
| | Quantitative-Probabilistic (6-10 RAs) | 3 |
| | Quantitative-Probabilistic (10-15 RAs) | 4 |
| | Quantitative-Probabilistic (>15 RAs) | 5 |
| 4 | Same scoring procedure as Question 3 | |
| 6c | Yes | 1 |
| | No | 0 |
| | External | 0 |
| | Yes/External | 0.5 |
| 7a-j | Yes | 1 |
| | No | 0 |
| | External | 0 |
| | Yes/External | 0.5 |
| 8a | Yes | 1 |
| | No | 0 |
| | External | 0 |
| | Yes/External | 0.5 |
| 9e | Yes | 1 |
| | No | 0 |
| | External | 0 |
| | Yes/External | 0.5 |

Note:

1) Questions with no response were given a score of 0.

2) The highest possible score was 39.

# APPENDIX II

# CASE STUDY

E/96/1043
July 25, 1996 962-1828

«Company»
«Address1»
«Address2»
«City»

Attention: «attention»

**RE: ROUND ROBIN RISK ASSESSMENT STUDY**

Dear «dear»:

Thank you for agreeing to participate in the Canada Mortgage and Housing Corporation (CMHC) Round Robin Risk Assessment Study. We are pleased to provide you with a description of the hypothetical case study.

Background

The growing application of human health risk assessment (HHRA) as a tool for improving risk management decisions in the redevelopment of contaminated sites has lead to an increase in the number of risk assessment techniques and assumptions employed. In addition, regulatory policy has developed to various degrees within and among countries. For these reasons, considerable variability may exist in HHRA methods and assumptions among Canadian practitioners. It is important to characterize and understand this variability because risk estimates may have significant implications on business expenditures associated with re-development.

A Round Robin Assessment has been chosen by CMHC to assess the variability among Canadian HHRA practitioners. It is a well established method for assessing interlaboratory variability, or feasibility and practicality of proposed protocols. Round Robins have enjoyed wide application in toxicology and other disciplines.

The Round Robin Risk Assessment Study is the second phase of the overall project. In the first phase, a questionnaire was circulated to a large number of risk assessment practitioners and regulators throughout Canada. The survey responses provided a profile of both the technical capabilities of practitioners in risk assessment and the regulatory/policy environment in which risk assessments are being conducted in Canada. CMHC greatly appreciates the input received from you.

As part of this final phase, we have asked your firm and nine other Canadian practitioners to undertake a screening level risk assessment of a hypothetical case study. The case study consists of a residential exposure scenario designed to re-create a situation which approximates a condition that may be encountered in the real world. Details of the case study are included in this package. The purpose of this phase of the study is to assess the degree of variability in risk estimates among participants, and analyze the sources of the variability and uncertainty. It is important to note that there is no preconceived "correct answer" to the case study and participants should not be concerned about being right or wrong. Additionally, all results and communication of the results will be structured to ensure anonymity of the participants. Once the results of the Round Robin have been compiled, they will be circulated to each participant. This will allow individual participants to compare their results with those of the group as a whole.

As part of a related study, Figley Consultants Associates Ltd. prepared a report for CMHC discussing a methodology for estimating indoor concentrations of soil gas which considers building design characteristics.

Overview of Round-Robin

1. All participants have been provided with an identical case study which focuses on potential residential development of a former industrial parcel of land contaminated with various substances.

2. The hypothetical case study provides both a descriptive and quantitative assessment of the site and its proposed development. A core set of raw data relevant to the site is provided for participants to analyze as they consider appropriate. CMHC do not wish to influence or bias participants in their approach or computations. To the extent possible, the Round Robin attempts to introduce "real world" variability for participants to deal with accordingly.

3. While the focus of the Round Robin is in risk assessment, we are also interested in your preliminary ideas regarding steps to further refine the risk calculations, mitigative measures, and other recommendations. Therefore, please provide brief comments on these issues, but understand that the largest effort should be dedicated to numerical derivation of health risks.

4. CMHC/Golder are available for limited consultation to clarify ambiguities and/or provide sources for further information. However, discussions concerning technical guidance will not be provided.

5. To further minimize potential bias in results, it is important that the level of effort be consistent amongst the various practitioners. Therefore, this case study simulates a request from a client who needs a preliminary risk assessment with limited time and resources to allow developers to evaluate options at an early

stage of the project. All participants will therefore be allocated $4500 and 8 days (whichever is least constraining) in which to analyze the case study, and provide:

- numerical risk estimates for each relevant potential exposure scenario identified by the participants; and
- rationale and/or numerical assumptions supporting the calculation of the risk estimates.

6. Pre-formatted generic reporting forms (hardcopy and electronic copy) are provided to ensure the information required for analysis by CMHC/Golder is received. These forms are designed to facilitate the documentation of risk estimates, computational methods, and numerical assumptions.

7. In order to include regional variability in the study, please assume that the site is located in your home province. Apply appropriate criteria, guidelines, and methodologies.

We wish you the best of luck in conducting the Round Robin Study. If you require clarification or if you have any concerns regarding any aspect of the study, please do not hesitate to contact the undersigned. We look forward to receiving your results via fax or mail by **23 August 1996**.

Yours truly,

**GOLDER ASSOCIATES LTD.**

M. Rankin, M.Sc.
Senior Toxicologist

I. Hers, P.Eng.
Senior Environmental Engineer

MR/IH/vw
962-1828

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*Hypothetical Case Study*

A residential housing development has been proposed on former industrial lands. The developer and regulators have hired a consultant (your firm) to assess the potential human health risks to future residents. The potential risk to workers at the site has already been addressed in a separate risk assessment and is not part of the present scope of work.

## 1. SITE DESCRIPTION

- The site is located on former industrial lands occupied by several different industries over the past 60-70 years.

- Several metals, benzene and vinyl chloride have been detected on site. The benzene and zinc contamination is confined to the top 1/2 metre of the native sand layer and is attributed to early industrial activity on the site. Following demolition of the factory and placement of fill, the more recent industrial activity has led to surface soil metal contamination. Previous site activities upgradient have resulted in vinyl chloride contamination of the groundwater.

- The site has been vacant for the past 8 years.

- The site is cleared of buildings/structures and is approximately 20 acres (8 hectares) in size. The land is covered with low lying vegetation (e.g., berry bushes, grasses etc.) and some refuse/construction materials.

- The site is located in suburban area of an urban centre with a population of approximately 500,000.

- The meteorological conditions measured at the main city airport located 25 km northwest of the site are shown in Table 1.

- The site is rectangular in shape and is bounded on all four sides by paved roads. The adjacent properties are commercially developed.

## 2. SITE INVESTIGATION

A site investigation was recently completed to assess the soil characteristics, hydrogeology, and the existing level of site contamination.

*Geology*

- The site investigation revealed a vertical soil structure comprised of the following units in sequence from ground surface (Figure 1):
  - fill consisting of sand (0 to 3 m depth)
  - native sand (3 to 7 m depth)
  - native silt (below 7 m depth)

- The average properties of the fill, native sand, and silt layers are shown in Table 2.

*Hydrogeology*

- The depth to the water table is 5 m and should be assumed constant.

- The Darcy velocity of the groundwater is 10 m/yr.

*Contaminant Levels*

- Thirty soil core samples were collected on an approximate grid basis from across the site. Subsamples were collected from varying depths and analyzed for a suite of metals and organic constituents.

- Elevated concentrations of four metals (cadmium, copper, lead and zinc) were found at 0 - 0.5 m depth (Table 3).

- Elevated concentrations of benzene and zinc were found at a depth of 3.0 - 3.5 m (Table 3).

- Groundwater samples were taken from 15 wells across the site and measured for volatile hydrocarbons. Only vinyl chloride was detected and the results are presented in Table 4. Recognizing that the upgradient source of vinyl chloride has been removed, assume that the vinyl chloride concentration in the groundwater will not increase in the future.

## 3. DEVELOPMENT PROPOSAL

- The proposed development is a suburban residential community consisting of approximately 60 single family dwellings. Included in the development will be small park/recreational area.

- Each lot will be 35x110 ft. (10.7x33.5 m).

- Each dwelling will have two stories and a full height basement (below ground). The dwelling will be 1800 $ft.^2$ (167 $m^2$) in area and have 3 bedrooms, 2 bathrooms, 1 powder room, and a 2 car garage. It will be heated using forced air and built under current building codes. Additional building specifications are provided in Table 5.

- The dwelling will be built on a non-structural slab with a thickness of 0.1 m and all service penetrations/drains will be trapped.

- The front yard will have a paved walkway with the remaining area covered by a grass lawn. The backyard will be mostly covered with grass and will include some planted shrubs and flowers.

- The drinking water will be municipally supplied.

## 4. PRESENTATION OF RESULTS

A standardized format (template) is provided for presentation of the risk assessment results. Both a hardcopy and electronic copy (MS Excel 5.0 spreadsheet file) of the forms has been provided for your convenience. Either a hardcopy or electronic version of the forms is to be completed and returned by 23 August 1996. The standardized form were developed to simplify reporting, maintain consistency and ensure that information required for analysis purposes is documented.

1) Use the enclosed reporting form to document the results of the risk assessment. DO NOT include a full written report as the time and resources should be applied to refining computations rather than report text. Space is provided on the form for written comments.

2) For each exposure scenario, fill out a separate exposure scenario form (make as many copies of the generic reporting form as required). A separate form must be filled out for each exposure pathway, receptor, and chemical. In order to avoid unnecessary repetition, if specific information is identical for more than one exposure scenarios, please reference the form where information is first documented.

3) Fill out a single summary form to integrate the findings of the various exposure scenarios and discuss overall recommendations.

**Figure 1: Building & Site Characteristics**

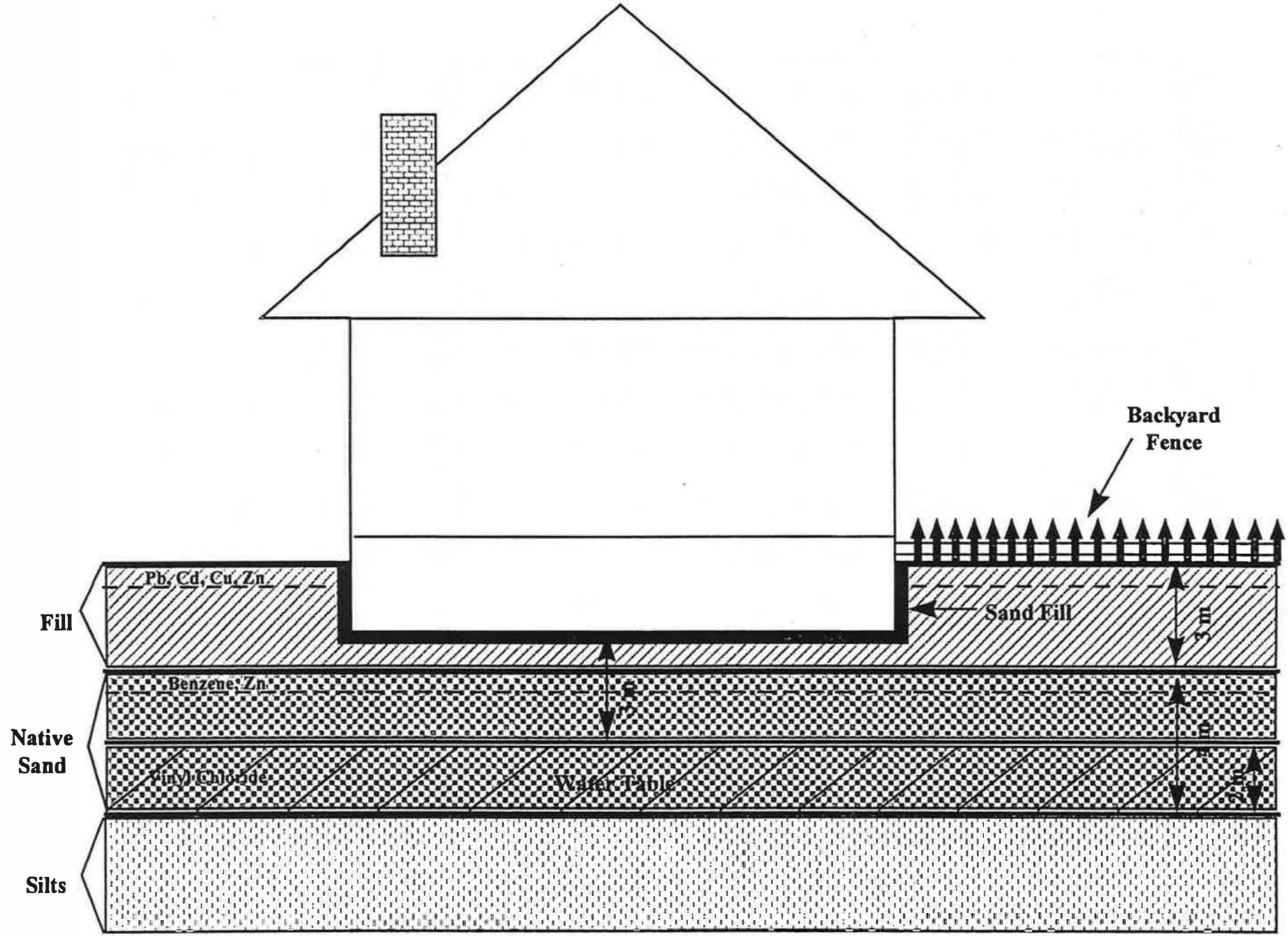


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Table 1. Meteorological Data

| | Jan | Feb | Mar | Apr | May | Jun | Jul | Aug | Sep | Oct | Nov | Dec | Year |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Temperature* | | | | | | | | | | | | | |
| Daily Maximum (°C) | -3.6 | -0.5 | 3.3 | 10.6 | 16.4 | 20.6 | 23.2 | 22.7 | 17.4 | 12.6 | 2.9 | -2.3 | 10.3 |
| Daily Minimum (°C) | -15.7 | -12.3 | -8.4 | -2.4 | 3.0 | 7.4 | 9.5 | 8.6 | 3.8 | -1.2 | -9.0 | -14.4 | -2.6 |
| Daily Mean (°C) | -9.6 | -6.3 | -2.5 | 4.1 | 9.7 | 14.0 | 16.4 | 15.7 | 10.6 | 5.7 | -3.0 | -8.3 | 3.9 |
| Extreme Maximum (°C) | 16.5 | 18.9 | 22.8 | 29.4 | 32.4 | 35.0 | 36.1 | 35.6 | 33.3 | 29.4 | 22.8 | 19.4 | |
| Extreme Minimum (°C) | -44.4 | -45.0 | -37.2 | -30.0 | -16.7 | -3.3 | -0.6 | -2.2 | -13.3 | -25.7 | -35.0 | -42.8 | |
| *Degree-Days* | | | | | | | | | | | | | |
| Above 18°C | 0.0 | 0.0 | 0.0 | 0.0 | 1.0 | 7.0 | 17.9 | 15.6 | 2.2 | 0.2 | 0.0 | 0.0 | 44.0 |
| Below 18°C | 857.6 | 687.6 | 638.1 | 417.1 | 258.1 | 126.6 | 68.7 | 88.3 | 224.0 | 380.5 | 631.3 | 816.9 | 5195.0 |
| Above 5°C | 2.1 | 3.3 | 6.4 | 47.2 | 153.8 | 27.6 | 352.2 | 330.3 | 179.0 | 79.3 | 8.9 | 1.9 | 1435.0 |
| Below 0°C | 318.3 | 204.5 | 124.7 | 20.1 | 0.3 | 0.0 | 0.0 | 0.0 | 1.3 | 15.7 | 134.5 | 276.4 | 1096.0 |
| *Precipitation* | | | | | | | | | | | | | |
| Rainfall (mm) | 0.2 | 0.2 | 1.5 | 9.2 | 43.9 | 76.7 | 69.9 | 48.7 | 42.7 | 6.4 | 0.6 | 0.1 | 300.3 |
| Snowfall (cm) | 18.0 | 14.9 | 18.7 | 20.4 | 10.2 | 0.3 | 0.0 | 0.0 | 6.4 | 11.5 | 16.0 | 199.0 | 135.4 |
| Precipitation (mm) | 12.2 | 9.9 | 14.7 | 25.1 | 52.9 | 76.9 | 69.9 | 48.7 | 48.1 | 15.5 | 11.6 | 13.2 | 398.8 |
| Extreme Daily Rainfall (mm) | 7.6 | 6.4 | 23.4 | 37.1 | 65.0 | 79.2 | 95.3 | 80.8 | 92.6 | 45.7 | 5.6 | 6.4 | |
| Extreme Daily Snowfall (mm) | 25.4 | 27.7 | 24.1 | 45.7 | 48.4 | 24.9 | 0.3 | 6.1 | 22.9 | 29.7 | 35.6 | 21.8 | |
| Extreme Daily Precip. (mm) | 25.4 | 27.7 | 24.1 | 45.7 | 65.0 | 79.2 | 95.3 | 80.8 | 92.6 | 45.7 | 35.6 | 21.8 | |
| Month-end Snow Cover (cm) | 7 | 5 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 4 | 6 | |
| *Days With* | | | | | | | | | | | | | |
| Maximum Temperature >0°C | 15 | 16 | 22 | 28 | 31 | 30 | 31 | 31 | 30 | 30 | 20 | 15 | 298 |
| Measurable Rainfall | - | - | - | 3 | 10 | 13 | 12 | 10 | 9 | 3 | - | - | 62 |
| Measurable Snowfall | 10 | 8 | 10 | 7 | 2 | - | 0 | 0 | 2 | 4 | 7 | 9 | 58 |
| Measurable Precipitation | 9 | 8 | 9 | 8 | 11 | 13 | 12 | 10 | 9 | 6 | 7 | 8 | 111 |
| Freezing Precipitation | - | 1 | 1 | - | - | 0 | - | 0 | - | - | 1 | - | 6 |
| Fog | 2 | 3 | 3 | 2 | - | - | - | 1 | 1 | 2 | 3 | 2 | 22 |
| Thunderstorms | - | 0 | - | - | 3 | 7 | 8 | 5 | 2 | - | 0 | 0 | 25 |
| Sunshine (hrs) | 113.8 | 136.8 | 174.0 | 214.8 | 256.0 | 285.5 | 320.1 | 284.8 | 201.8 | 179.0 | 125.4 | 102.5 | 2394.6 |
| Station Pressure (kPa) | 88.8 | 88.8 | 88.7 | 88.9 | 88.9 | 89.0 | 89.2 | 89.2 | 89.1 | 89.0 | 88.8 | 88.8 | 88.9 |
| *Moisture* | | | | | | | | | | | | | |
| Vapour Pressure (kPa) | 0.24 | 0.28 | 0.35 | 0.45 | 0.64 | 0.90 | 1.08 | 1.05 | 0.76 | 0.52 | 0.34 | 0.26 | 0.57 |
| Rel. Humidity - 0600L (%) | 67 | 68 | 73 | 72 | 71 | 72 | 76 | 78 | 77 | 69 | 71 | 68 | |
| Rel. Humidity - 1500L (%) | 59 | 57 | 55 | 43 | 42 | 44 | 44 | 43 | 45 | 43 | 56 | 60 | |
| *Wind* | | | | | | | | | | | | | |
| Speed (km/h) | 16 | 15 | 16 | 17 | 18 | 17 | 15 | 14 | 15 | 15 | 15 | 16 | 16 |
| Most Frequent Direction | W | S | S | N | NW | NW | NW | NW | S | S | S | W | N |
| Maximum Hourly Speed (km/h) | 84 | 89 | 85 | 105 | 90 | 82 | 82 | 97 | 84 | 90 | 84 | 100 | |
| Direction | N | NW | NW | N | NW | NW | NW | N | NW | W | W | N | |
| Maximum Gust Speed (km/h) | 127 | 126 | 114 | 116 | 121 | 127 | 122 | 109 | 111 | 117 | 113 | 111 | |
| Direction | W | N | N | N | N | S | N | N | NW | NW | W | N | |

Table 2. Soil Properties

| Parameter | Fill | Native Sand | Native Silts |
|---|---|---|---|
| Thickness (m) | 3 | 4 | - |
| Total Porosity Above the Water Table (ratio) | 0.4 | 0.4 | 0.35 |
| Soil Dry Bulk Density (kg-soil/L-soil) | 1.7 | 1.7 | 1.7 |
| Organic Carbon Fraction (kg-OC/kg-soil) | 0.004 | 0.008 | 0.01 |
| Soil Temperature (°C) | 15 | 15 | 15 |
| Particle Density (kg-soil/L-soil) | 2.65 | 2.65 | 2.65 |
| Moisture Content Above Water Table (L-water/L-soil) | 0.11 | 0.21 | * |
| Effective Porosity | 0.35 | 0.35 | 0.25 |

* Saturated

Table 3: Contaminant Concentrations (ug/g dry weight) in soil at 2 depth intervals.

| | 0-0.5 m depth | | | | 3.0-3.5 m depth | |
|---|---|---|---|---|---|---|
| Sample | Lead | Cadmium | Copper | Zinc | Zinc | Benzene |
| 1 | 823.8 | 8.2 | 3315.4 | 128.2 | 1955.4 | 0.81 |
| 2 | 1076.2 | 10.7 | 250.4 | 50.1 | 1607.8 | 0.90 |
| 3 | 345.1 | 3.4 | 1012.7 | 368.9 | 2976.6 | 0.71 |
| 4 | 2068.3 | 20.6 | 1637.4 | 92.8 | 206.1 | 0.17 |
| 5 | 597.2 | 5.9 | 140.2 | 159.8 | 430.1 | 12.65 |
| 6 | 535.9 | 5.3 | 1463.1 | 226.6 | 245.5 | 1.39 |
| 7 | 3023.0 | 30.2 | 163.0 | 103.0 | 1764.7 | 6.57 |
| 8 | 293.7 | 2.9 | 4462.5 | 388.3 | 189.6 | 1.08 |
| 9 | 280.4 | 2.8 | 2012.9 | 606.6 | 395.1 | 0.22 |
| 10 | 735.6 | 7.3 | 709.4 | 39.2 | 33.9 | 0.52 |
| 11 | 699.5 | 0.7 | 1173.3 | 32.8 | 418.9 | 1.25 |
| 12 | 323.1 | 3.2 | 8967.6 | 139.0 | 668.7 | 0.39 |
| 13 | 469.2 | 4.6 | 1817.0 | 161.2 | 1118.5 | 0.89 |
| 14 | 639.0 | 6.3 | 271.1 | 63.1 | 300.3 | 0.67 |
| 15 | 733.1 | 7.3 | 208.8 | 727.3 | 343 | 0.57 |
| 16 | 639.2 | 6.3 | 829.9 | 32.0 | 2095.2 | 1.62 |
| 17 | 176.4 | 1.7 | 457.5 | 230.0 | 656.7 | 0.22 |
| 18 | 84.6 | 8.0 | 2031.7 | 421.4 | 896.8 | 2.89 |
| 19 | 989.0 | 36.9 | 503.3 | 59.3 | 152.8 | 0.82 |
| 20 | 1413.1 | 14.1 | 53.0 | 60.2 | 4884.5 | 0.37 |
| 21 | 699.8 | 6.9 | 3154.2 | 224.9 | 2978.6 | 0.62 |
| 22 | 1212.1 | 12.1 | 152.1 | 144.2 | 696.9 | 0.22 |
| 23 | 497.4 | 4.9 | 1211.7 | 364.6 | 717.3 | 2.05 |
| 24 | 1913.2 | 19.1 | 3918.2 | 111.5 | 3308.3 | 0.27 |
| 25 | 1092.5 | 10.9 | 848.5 | 461.1 | 320.3 | 0.66 |
| 26 | 1388.0 | 13.8 | 166.6 | 137.2 | 1633.8 | 0.71 |
| 27 | 1014.0 | 10.1 | 493.0 | 79.9 | 632.8 | 2.25 |
| 28 | 650.8 | 6.5 | 75.6 | 171.6 | 2063.5 | 0.26 |
| 29 | 586.8 | 88.2 | 1219.0 | 96.8 | 509.4 | 0.83 |
| 30 | 433.2 | 4.3 | 1234.2 | 94.4 | 1081.8 | 2.25 |

Table 4: Dissolved Contaminant Concentrations in Groundwater (mg/L)

| Well | Vinyl Chloride |
|---|---|
| 1 | 0.0054 |
| 2 | 0.0408 |
| 3 | <0.0005 |
| 4 | 0.0014 |
| 5 | 0.0975 |
| 6 | 0.0176 |
| 7 | 0.0395 |
| 8 | 0.0537 |
| 9 | 0.0023 |
| 10 | <0.0005 |
| 11 | 0.0897 |
| 12 | 0.0014 |
| 13 | 0.0236 |
| 14 | 0.0056 |
| 15 | 0.0085 |

Table 5. Building Details

| *Parameter* | Value |
|---|---|
| Thickness of Concrete in Floor Slab and Walls (m) | 0.1 |
| Footprint Area of Building ($m^2$) | 100 |
| Height of Each Floor of Building (m) | 2.3 |
| Thickness of Sand Fill Below Concrete Slab (m) * | 0.2 |

* Assume sand is from regional source and material typically used for such purposes

# APPENDIX III

## SUPPLEMENTARY RESULTS

**Part A:** Tier I and II Regressions and Correlations
**Part B:** Scatter plots of Risk Estimates and Dose Rates
**Part C:** Correlations between Risk Estimate and Capability Scores

# PART A

## TIER I AND II REGRESSIONS AND CORRELATIONS

Risk Estimate Correlations

Soil Ingestion Pathway
Zinc
Adult

| Company | HQ | RfD | Dose |
|---|---|---|---|
| 2 | 7.00E-06 | 0.3 | 2.03E-06 |
| 3 | 0.0011 | 0.3 | 0.000363 |
| 4 (average) | 0.0002 | 0.3 | 5.98E-05 |
| 8 | 2.60E-05 | 0.3 | 7.66E-06 |
| 9 | 0.00069 | 0.3 | 0.000208 |

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | #DIV/0! | 1 | |
| Dose | 0.998606 | #DIV/0! | 1 |

No regression done, because RfD data are identical

Correlation using untransformed doses, and one factor for receptor characteristics
Soil Ingestion Pathway, Zinc, Adult

receptor = (IR x EF x ED x FI)/(BW x AT)

| Dose | Cs | Receptor |
|---|---|---|
| 2.03E-06 | 181.4 | 4.1 |
| 0.000363 | 265 | 500.0 |
| 5.98E-05 | 727.3 | 30.0 |
| 7.66E-06 | 199 | 14.0 |
| 0.000208 | 727.3 | 104.3 |

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.135605 | 1 | |
| Receptor | 0.929834 | -0.1698 | 1 |

therefore, receptor = 0.929^2
0.864591

Do regression on both:

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.976364 |
| R Square | 0.953287 |
| Adjusted | 0.906574 |
| Standard E | 4.75E-05 |
| Observatio | 5 |

therefore, Cs is 0.953-0.864 = 0.088696

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regression | 2 | 9.21E-08 | 4.6E-08 | 20.40733 | 0.046713 |
| Residual | 2 | 4.51E-09 | 2.26E-09 | | |
| Total | 4 | 9.66E-08 | | | |

| | *Coefficient* | *ndard Err* | *t Stat* | *P-value* | *ower 95%* | *pper 95%* | *ower 95.0* | *pper 95.0%* |
|---|---|---|---|---|---|---|---|---|
| Intercept | -3.6E-05 | 4.63E-05 | -0.78708 | 0.513692 | -0.00024 | 0.000163 | -0.00024 | 0.000163 |
| Cs | 1.66E-07 | 8.54E-08 | 1.948713 | 0.190665 | -2E-07 | 5.34E-07 | -2E-07 | 5.34E-07 |
| Receptor | 7.25E-07 | 1.15E-07 | 6.326718 | 0.024084 | 2.32E-07 | 1.22E-06 | 2.32E-07 | 1.22E-06 |

Risk Estimate Correlations

Soil Ingestion Pathway
Copper
Child

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.0005 | 0.517 | 0.00027 | 1209 | 81 |
| 3 | 0.482 | 0.0571 | 0.02753 | 2153 | 4667 |
| 5 | 0.035 | 0.5 | 0.0174 | 8967.6 | 672 |
| 7 | 0.095 | 0.5 | 0.04747 | 8967.6 | 1931 |
| 9 | 1.1037 | 0.05 | 0.05518 | 8967.6 | 2246 |

receptor = (IR x EF x ED x FI)/(BW x AT)

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | [redacted] | 1 | |
| Dose | 0.685852589 | -0.503366341 | 1 |

$R^2$: 0.7835317

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.927827142 |
| R Square | 0.860863205 |
| Adjusted R Square | 0.72172641 |
| Standard Error | 0.24624269 |
| Observations | 5 |

Dose: 0.0773315

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.750323999 | 0.375162 | 6.1871714 | 0.139136795 |
| Residual | 2 | 0.121270924 | 0.060635 | | |
| Total | 4 | 0.871594923 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.58655399 | 0.343975853 | 1.705219 | 0.2302709 | -0.893455682 |
| RfD | -1.362593935 | 0.575147096 | -2.36912 | 0.1413484 | -3.837253882 |
| Dose | 6.740069015 | 6.392813806 | 1.05432 | 0.4023029 | -20.76600791 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.675620051 | 1 | |
| Receptor | 0.455260031 | -0.156912065 | 1 |

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.882860128 |
| R Square | 0.779442005 |
| Adjusted R Square | 0.55888401 |
| Standard Error | 0.014803595 |
| Observations | 5 |

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.001548907 | 0.000774 | 3.5339549 | 0.220557995 |
| Residual | 2 | 0.000438293 | 0.000219 | | |
| Total | 4 | 0.0019872 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -0.010108326 | 0.016331601 | -0.61894 | 0.5990592 | -0.080377585 |
| Cs | 4.26257E-06 | 1.87134E-06 | 2.277823 | 0.1504254 | -3.78914E-06 |
| Receptor | 7.23004E-06 | 4.22474E-06 | 1.711359 | 0.2291449 | -1.09475E-05 |

Risk Estimate Correlations

Soil Ingestion Pathway
Copper
Adult

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.00003 | 0.517 | 1.35E-05 | 1209 | 4 |
| 3 | 0.05 | 0.0571 | 0.00295 | 2153 | 500 |
| 8 | 0.00141 | 0.04 | 5.64E-05 | 1465 | 14 |
| 9 | 0.051243 | 0.05 | 0.002562 | 8967.6 | 104 |

receptor = (IR x EF x ED x FI)/(BW x AT)

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.57109898 | 1 | |
| Dose | [redacted] | -0.559966634 | 1 |

$R^2$: 0.986063

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.993172906 |
| R Square | 0.986392422 |
| Adjusted R Square | 0.959177265 |
| Standard Error | 0.005823102 |
| Observations | 4 |

RfD 0.00033

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.002457976 | 0.001229 | 36.24423 | 0.116651525 |
| Residual | 1 | 3.39085E-05 | 3.39E-05 | | |
| Total | 3 | 0.002491884 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.001139672 | 0.006417018 | 0.177602 | 0.888102 | -0.080395922 |
| RfD | -0.00269904 | 0.017334579 | -0.155703 | 0.901666 | -0.222954808 |
| Dose | 17.89949027 | 2.569689812 | 6.965623 | 0.090774 | -14.75137471 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.580443703 | 1 | |
| Receptor | [redacted] | -0.042065098 | 1 |

$R^2$: 0.622374

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999791303 |
| R Square | 0.99958265 |
| Adjusted R Square | 0.998747949 |
| Standard Error | 5.58758E-05 |
| Observations | 4 |

Cs: 0.377208

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 7.47765E-06 | 3.74E-06 | 1197.534 | 0.020429153 |
| Residual | 1 | 3.1221E-09 | 3.12E-09 | | |
| Total | 3 | 7.48077E-06 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -0.00036466 | 4.69176E-05 | -7.772301 | 0.081461 | -0.000960799 |
| Cs | 2.62294E-07 | 8.72466E-09 | 30.06355 | 0.021168 | 1.51437E-07 |
| Receptor | 5.49877E-06 | 1.37996E-07 | 39.84718 | 0.015973 | 3.74536E-06 |

Risk Estimate Correlations

Soil Ingestion Pathway
Lead
Child

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.09 | 0.002 | 0.000175 | 785.3 | 81 |
| 3 | 13821 | 0.000001 | 0.01382 | 1081 | 4667 |
| 5 | 0 | 0.00185 | 0 | 3023 | 672 |
| 7 | 0.00 | 0.00357 | 0.000 | 3023 | 1931 |
| 9 | 0 | 0.0035 | 0 | 3023 | 2246 |

receptor = (IR x EF x ED x FI)/(BW x AT)

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.834098641 | 1 | |
| Dose | [redacted] | -0.837576323 | 1 |

$R^2$: 0.999849

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999943977 |
| R Square | 0.999887957 |
| Adjusted R Square | 0.999775913 |
| Standard Error | 92.52554081 |
| Observations | 5 |

RfD 3.91E-05

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 152798413.3 | 76399207 | 8924.12 | 0.000112043 |
| Residual | 2 | 17121.9514 | 8560.976 | | |
| Total | 4 | 152815535.3 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -175.9006425 | 165.278234 | -1.06427 | 0.3987 | -887.0359823 |
| RfD | 48324.93514 | 57873.82792 | 0.835005 | 0.49157 | -200686.2219 |
| Dose | 1012707.152 | 13743.75527 | 73.68489 | 0.00018 | 953572.505 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | -0.548254914 | 1 | |
| Receptor | [redacted] | -0.149792729 | 1 |

$R^2$: 0.741339

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.959776772 |
| R Square | 0.921171453 |
| Adjusted R Square | 0.842342906 |
| Standard Error | 0.002446446 |
| Observations | 5 |

Cs: 0.179832

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.000139881 | 6.99E-05 | 11.6858 | 0.078828547 |
| Residual | 2 | 1.19702E-05 | 5.99E-06 | | |
| Total | 4 | 0.000151851 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.002515473 | 0.003078086 | 0.81722 | 0.49967 | -0.010728472 |
| Cs | -2.29912E-06 | 1.07635E-06 | -2.136029 | 0.16619 | -6.93028E-06 |
| Receptor | 2.76731E-06 | 6.97401E-07 | 3.96803 | 0.05804 | -2.33368E-07 |

Risk Estimate Correlations

Soil Ingestion Pathway
Lead
Adult

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.002 | 0.002 | 8.79E-06 | 785.3 | 4 |
| 3 | 1481 | 0.000001 | 0.0014 | 1081 | 500 |
| 8 | 32.6 | 0.000001 | 3.26E-05 | 848 | 14 |
| 9 | 0.1233878 | 0.007 | 0.000864 | 3023 | 104 |

receptor = (IR x EF x ED x FI)/(BW x AT)

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.46720438 | 1 | |
| Dose | [redacted] | 0.130575564 | 1 |

$R^2$: 0.646492

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.989735543 |
| R Square | 0.979576445 |
| Adjusted R Square | 0.938729335 |
| Standard Error | 181.9848409 |
| Observations | 4 |

RfD 0.333084

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1588464.155 | 794232.1 | 23.9815 | 0.142911004 |
| Residual | 1 | 33118.48234 | 33118.48 | | |
| Total | 3 | 1621582.638 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 119.9794462 | 141.0976244 | 0.850329 | 0.55138 | -1672.828177 |
| RfD | -129549.2526 | 32079.19921 | -4.038419 | 0.15453 | -537152.3794 |
| Dose | 954413.9168 | 156323.9477 | 6.10536 | 0.10335 | -1031861.658 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.393024062 | 1 | |
| Receptor | [redacted] | -0.029206389 | 1 |

$R^2$: 0.823706

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999931364 |
| R Square | 0.999862732 |
| Adjusted R Square | 0.999588197 |
| Standard Error | 1.37571E-05 |
| Observations | 4 |

Cs: 0.176157

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.37857E-06 | 6.89E-07 | 3642.02 | 0.011716125 |
| Residual | 1 | 1.89259E-10 | 1.89E-10 | | |
| Total | 3 | 1.37876E-06 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower95%* |
|---|---|---|---|---|---|
| Intercept | -0.000221175 | 1.38813E-05 | -15.93331 | 0.0399 | -0.000397553 |
| Cs | 2.66848E-07 | 7.449E-09 | 35.8233 | 0.01777 | 1.722E-07 |
| Receptor | 2.66513E-06 | 3.39604E-08 | 78.47761 | 0.00811 | 2.23363E-06 |

Risk Estimate Correlations

Soil Ingestion Pathway
Cadmium
Child

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.002 | 0.001 | 2.12E-06 | 9.5 | 81 |
| 3 | 0.47 | 0.0005 | 0.000234 | 18.27 | 4667 |
| 5 | 0.34 | 0.0005 | 0.000171 | 88.2 | 672 |
| 7 | 0.5742 | 0.00081 | 0.000465 | 88.2 | 1931 |
| 9 | 0.678 | 0.0008 | 0.00054 | 88.2 | 2246 |

receptor = (IR x EF x ED x FI)/(BW x AT)

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.332891203 | 1 | |
| Dose | [redacted] | -0.032677639 | 1 |

$R^2$: 0.901686

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.996445694 |
| R Square | 0.99290402 |
| Adjusted R Square | 0.985808041 |
| Standard Error | 0.031162764 |
| Observations | 5 |

RfD 0.091218

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.271767076 | 0.135884 | 139.925 | 0.00709598 |
| Residual | 2 | 0.001942236 | 0.000971 | | |
| Total | 4 | 0.273709312 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.358906294 | 0.057752332 | 6.214577 | 0.02493 | 0.110417894 |
| RfD | -362.9991939 | 71.59079768 | -5.070473 | 0.03676 | -671.0297494 |
| Dose | 1118.715128 | 70.95040364 | 15.76757 | 0.004 | 813.4399671 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | [redacted] | 1 | |
| Receptor | 0.389587384 | -0.16379355 | 1 |

$R^2$: 0.503359

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.875351426 |
| R Square | 0.76624012 |
| Adjusted R Square | 0.532480239 |
| Standard Error | 0.000150239 |
| Observations | 5 |

Receptor 0.262882

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.47976E-07 | 7.4E-08 | 3.27789 | 0.23375988 |
| Residual | 2 | 4.51436E-08 | 2.26E-08 | | |
| Total | 4 | 1.9312E-07 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -9.11786E-05 | 0.000161693 | -0.563899 | 0.62962 | -0.000786888 |
| Cs | 4.27703E-06 | 1.86537E-06 | 2.29286 | 0.14888 | -3.74901E-06 |
| Receptor | 6.4375E-08 | 4.29247E-08 | 1.499719 | 0.27246 | -1.20315E-07 |

Risk Estimate Correlations

Soil Ingestion Pathway
Cadmium
Adult

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.00010 | 0.001 | 1.06E-07 | 9.5 | 4 |
| 3 | 0.05 | 0.0005 | 0.000025 | 18.27 | 500 |
| 4 | 0.0073 | 0.001 | 7.25E-06 | 88.2 | 30 |
| 8 | 0.000465 | 0.001 | 4.65E-07 | 12.1 | 14 |
| 9 | 0.0315 | 0.0008 | 2.52E-05 | 88.2 | 104 |

receptor = (IR x EF x ED x FI)/(BW x AT)

TIER I

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.971394088 | 1 | |
| Dose | 0.949494065 | -0.849825893 | 1 |

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999468363 |
| R Square | 0.998937008 |
| Adjusted | 0.997874016 |
| Standard E | 0.001017666 |
| Observatio | 5 |

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.001946474 | 0.000973 | 939.741 | 0.001062992 |
| Residual | 2 | 2.07129E-06 | 1.04E-06 | | |
| Total | 4 | 0.001948546 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.060133025 | 0.004589061 | 13.10356 | 0.00577 | 0.040387876 |
| RfD | -59.65087198 | 4.406479048 | -13.53708 | 0.00541 | -78.61043428 |
| Dose | 779.0045492 | 76.34973958 | 10.20311 | 0.00947 | 450.4979051 |

TIER II

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.391950737 | 1 | |
| Receptor | [redacted] | -0.20314489 | 1 |

$R^2$: 0.535369

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.916616348 |
| R Square | 0.84018553 |
| Adjusted | 0.68037106 |
| Standard E | 7.14871E-06 |
| Observatio | 5 |

Cs: 0.304816

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 5.37334E-10 | 2.69E-10 | 5.25726 | 0.15981447 |
| Residual | 2 | 1.02208E-10 | 5.11E-11 | | |
| Total | 4 | 6.39543E-10 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -2.52937E-06 | 5.79693E-06 | -0.436329 | 0.70518 | -2.74716E-05 |
| Cs | 1.73249E-07 | 8.87042E-08 | 1.953107 | 0.19004 | -2.08415E-07 |
| Receptor | 5.08872E-08 | 1.73605E-08 | 2.931205 | 0.09935 | -2.38091E-08 |

Risk Estimate Correlations

Dermal Contact Pathway
Zinc
Adult

| Company | HQ | RfD | Dose |
|---|---|---|---|
| 2 | 2.00E-02 | 0.015 | 0.000219 |
| 4 (average) | 0.037 | 0.3 | 0.0112 |
| 8 | 3.20E-05 | 0.3 | 0.000475 |
| 9 | 0.00372 | 0.3 | 0.001117 |

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.18946 | 1 | |
| Dose | 0.835153 | 0.380769 | 1 |

Therefore Dose is .835^2
0.69748

Regressions on both parameters

| HQ | RfD | Dose |
|---|---|---|
| 0.02 | 0.015 | 0.000219 |
| 0.037 | 0.3 | 0.0112 |
| 0.000032 | 0.3 | 0.000475 |
| 0.00372 | 0.3 | 0.001117 |

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.99933 |
| R Square | 0.99866 |
| Adjusted | 0.995979 |
| Standard | 0.001074 |
| Observati | 4 |

Therefore RfD is .998 - .697 = 0.30118

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 0.000859 | 0.000429 | 372.5573 | 0.03661 |
| Residual | 1 | 1.15E-06 | 1.15E-06 | | |
| Total | 3 | 0.00086 | | | |

| | *Coefficient* | *andard Err* | *t Stat* | *P-value* | *ower 95* | *pper 95* | *ower 95.0* | *pper 95.0%* |
|---|---|---|---|---|---|---|---|---|
| Intercept | 0.020317 | 0.001131 | 17.9689 | 0.035392 | 0.00595 | 0.034684 | 0.00595 | 0.034684 |
| RfD | -0.07052 | 0.004705 | -14.9905 | 0.042405 | -0.1303 | -0.01075 | -0.1303 | -0.01075 |
| Dose | 3.382754 | 0.126214 | 26.80174 | 0.023742 | 1.779061 | 4.986448 | 1.779061 | 4.986448 |

Correlation using untransformed doses, and one factor for receptor characteristics
Dermal Contact Pathway, Zinc, Adult

| Company | Dose | Cs | Receptor |
|---|---|---|---|
| 2 | 0.000219 | 181.4 | 440 |
| 4 (ave) | 0.0112 | 727.3 | 5616 |
| 8 | 0.000475 | 199 | 870 |
| 9 | 0.001117 | 727.3 | 561 |

where:
Receptor = (EF*SA*SDAF*BA)/BW

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.631965 | 1 | |
| Receptor | 0.995012 | 0.562734 | 1 |

Therefore, receptor is 0.995^2
0.990025

Regression on both:

| Dose | Cs | Receptor |
|---|---|---|
| 0.000219 | 181.4 | 440.2357 |
| 0.0112 | 727.3 | 5616 |
| 0.000475 | 199 | 870.3846 |
| 0.001117 | 727.3 | 560.5357 |

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.998821 |
| R Square | 0.997643 |
| Adjusted | 0.992929 |
| Standard E | 0.000447 |
| Observatio | 4 |

Therefore Cs is 0.997-.990 = 0.007618

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regression | 2 | 8.44E-05 | 4.22E-05 | 211.6285 | 0.04855 |
| Residual | 1 | 2E-07 | 2E-07 | | |
| Total | 3 | 8.46E-05 | | | |

| | *Coefficient* | *ndard Err* | *t Stat* | *P-value* | *ower 95%* | *pper 95%* | *ower 95.0* | *pper 95.0* |
|---|---|---|---|---|---|---|---|---|
| Intercept | -0.001293 | 0.000443 | -2.919348 | 0.210094 | -0.006918 | 0.004333 | -0.006918 | 0.004333 |
| Cs | 1.81E-06 | 1.01E-06 | 1.794982 | 0.323584 | -1.1E-05 | 1.46E-05 | -1.1E-05 | 1.46E-05 |
| Receptor | 1.99E-06 | 1.25E-07 | 15.93165 | 0.039907 | 4.02E-07 | 3.57E-06 | 4.02E-07 | 3.57E-06 |

Risk Estimate Correlations

Dermal Contact Pathway
Copper
Child

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.09 | 0.017 | 0.00154 | 1209 | 466 |
| 5 | 0.13 | 0.5 | 0.067 | 8967.6 | 11553 |
| 7 | 0.0099 | 0.5 | 0.004926 | 8967.6 | 200 |
| 9 | 2.5365497 | 0.05 | 0.1268 | 8967.6 | 5162 |

receptor = (EF*SA*SDA*BA)/BW

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.541885274 | 1 | |
| Dose | [redacted] | -0.231008796 | 1 |

$R^2$: 0.7676087

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.943058862 |
| R Square | 0.889360018 |
| Adjusted R Squar | 0.668080053 |
| Standard Error | 0.709196487 |
| Observations | 4 |

RfD: 0.1217513

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 4.042952651 | 2.021476 | 4.01916 | 0.332625889 |
| Residual | 1 | 0.502959658 | 0.50296 | | |
| Total | 3 | 4.545912308 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.304519895 | 0.702380922 | 0.433554 | 0.73956 | -8.620037662 |
| RfD | -1.63704962 | 1.560562011 | -1.04901 | 0.48477 | -21.46578509 |
| Dose | 16.45351006 | 7.09076979 | 2.320412 | 0.25904 | -73.64287664 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.545086613 | 1 | |
| Receptor | 0.591511675 | 0.486311228 | 1 |

Risk Estimate Correlations

Dermal Contact Pathway
Lead
Child

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 15.2 | 0.0000012 | 1.82E-05 | 785.3 | 8 |
| 5 | 12.2 | 0.00185 | 0.02 | 3023 | 11597 |
| 7 | 0.4653 | 0.00357 | 0.001664 | 3023 | 200 |
| 9 | 0.222098 | 0.0035 | 0.000778 | 3023 | 94 |

receptor = (EF*SA*SDA*BA)/BW

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | [redacted] | 1 | |
| Dose | 0.395074048 | -0.098233806 | 1 |

$R^2$: 0.907894

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999832783 |
| R Square | 0.999665594 |
| Adjusted | 0.998996783 |
| Standard E | 0.247324868 |
| Observatio | 4 |

Dose: 0.091772

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 182.8591804 | 91.42959 | 1494.69 | 0.01828676 |
| Residual | 1 | 0.06116959 | 0.06117 | | |
| Total | 3 | 182.92035 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 15.19443205 | 0.247412589 | 61.41333 | 0.01037 | 12.05077051 |
| RfD | -4276.308867 | 85.14166981 | -50.2258 | 0.01267 | -5358.13172 |
| Dose | 217.8585502 | 13.15093546 | 16.56601 | 0.03838 | 50.76078758 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.381685373 | 1 | |
| Receptor | [redacted] | 0.344020117 | 1 |

$R^2$: 0.997696

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999669355 |
| R Square | 0.999338819 |
| Adjusted | 0.998016456 |
| Standard E | 0.000485934 |
| Observatio | 4 |

Cs: 0.001643

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 0.0003569 | 0.000178 | 755.722 | 0.025713445 |
| Residual | 1 | 2.36132E-07 | 2.36E-07 | | |
| Total | 3 | 0.000357136 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -0.000328232 | 0.000667672 | -0.49161 | 0.70912 | -0.008811769 |
| Cs | 4.20996E-07 | 2.67052E-07 | 1.576455 | 0.35987 | -2.97221E-06 |
| Receptor | 1.86762E-06 | 5.19765E-08 | 35.93196 | 0.01771 | 1.2072E-06 |

Risk Estimate Correlations

Dermal Contact Pathway
Cadmium
Child

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.4 | 0.00001 | 3.68E-06 | 9.5 | 141 |
| 5 | 1.3 | 0.0005 | 0.0007 | 88.2 | 11252 |
| 7 | 0.0058 | 0.00081 | 4.7E-06 | 88.2 | 20 |
| 9 | 0.4725 | 0.0008 | 0.000378 | 88.2 | 1564 |

receptor = (EF*SA*SDA*BA)/BW

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.19240311 | 1 | |
| Dose | [redacted] | 0.227920359 | 1 |

$R^2$: 0.81055398

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.98858648 |
| R Square | 0.97730323 |
| Adjusted R Square | 0.93190969 |
| Standard Error | 0.14189078 |
| Observations | 4 |

RfD: 0.16674925

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 0.866909174 | 0.433455 | 21.5296 | 0.150654476 |
| Residual | 1 | 0.020132993 | 0.020133 | | |
| Total | 3 | 0.887042168 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.42201058 | 0.142053427 | 2.970788 | 0.20671 | -1.382941611 |
| RfD | -607.607573 | 224.1678241 | -2.7105 | 0.22501 | -3455.917639 |
| Dose | 1701.18592 | 264.304303 | 6.436467 | 0.09812 | -1657.104282 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.53962369 | 1 | |
| Receptor | [redacted] | 0.38422534 | 1 |

$R^2$: 0.80569283

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.92205504 |
| R Square | 0.8501855 |
| Adjusted R Square | 0.5505565 |
| Standard Error | 0.00021341 |
| Observations | 4 |

Cs: 0.04449267

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 2.58453E-07 | 1.29E-07 | 2.83746 | 0.387058783 |
| Residual | 1 | 4.5543E-08 | 4.55E-08 | | |
| Total | 3 | 3.03996E-07 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -2.0639E-05 | 0.000239795 | -0.08607 | 0.94534 | -0.003067514 |
| Cs | 1.8482E-06 | 3.39149E-06 | 0.544963 | 0.68235 | -4.12446E-05 |
| Receptor | 4.7876E-08 | 2.47851E-08 | 1.931638 | 0.30412 | -2.67047E-07 |

Risk Estimate Correlations

Dermal Contact Pathway
Cadmium
Adult

| Company | HQ | RfD | Dose | Cs | Receptor |
|---|---|---|---|---|---|
| 2 | 0.2 | 0.00001 | 2.26E-06 | 9.5 | 88 |
| 4(ave.) | 1.36 | 0.001 | 0.00137 | 88.2 | 5616 |
| 8 | 0.0115 | 0.0025 | 2.89E-05 | 12 | 870 |
| 9 | 0.067725 | 0.0008 | 5.42E-05 | 88.2 | 224 |

receptor = (EF*SA*SDA*BA)/BW

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.1613038 | 1 | |
| Dose | [illegible] | -0.039398874 | 1 |

$R^2$: 0.97831912

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.99664843 |
| R Square | 0.99330808 |
| Adjusted R Square | 0.97992425 |
| Standard Error | 0.09045016 |
| Observations | 4 |

RfD: 0.01498896

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.214373044 | 0.607187 | 74.217 | 0.081804137 |
| Residual | 1 | 0.008181232 | 0.008181 | | |
| Total | 3 | 1.222554275 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.15018441 | 0.076809039 | 1.955296 | 0.30096 | -0.825762783 |
| RfD | -75.1930924 | 50.24205555 | -1.49662 | 0.375 | -713.5762017 |
| Dose | 936.255163 | 77.87379329 | 12.02272 | 0.05283 | -53.22095942 |

**TIER II**

| | *Dose* | *Cs* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Cs | 0.59941406 | 1 | |
| Receptor | [illegible] | 0.537833769 | 1 |

$R^2$: 0.98366505

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.99488304 |
| R Square | 0.98979227 |
| Adjusted R Square | 0.9693768 |
| Standard Error | 0.00011744 |
| Observations | 4 |

Cs: 0.00612722

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.33738E-06 | 6.69E-07 | 48.4825 | 0.101033322 |
| Residual | 1 | 1.37924E-08 | 1.38E-08 | | |
| Total | 3 | 1.35117E-06 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -0.00011311 | 9.5341E-05 | -1.18634 | 0.44587 | -0.001324524 |
| Cs | 1.3932E-06 | 1.79817E-06 | 0.77476 | 0.58037 | -2.14547E-05 |
| Receptor | 2.4006E-07 | 3.05449E-08 | 7.859161 | 0.08057 | -1.48051E-07 |

Risk Estimate Correlations

Inhalation of Fugitive Dust Pathway
Zinc
Child

| Company | HQ | RfD | Dose |
|---|---|---|---|
| 2 | 7.00E-10 | 0.01 | 6.63E-12 |
| 3 | 0.83 | 0.000112 | 9.35E-05 |
| 5 | 4.00E-04 | 0.3 | 0.000119 |
| 7 | 1.40E-06 | 0.3 | 9.50E-10 |

Note: calculated value used for Company No. 7 (they reported data as <1%)

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | -0.59636 | 1 | |
| Dose | 0.433009 | 0.103741 | 1 |

Therefore RfD is 0.596^2
0.35564

Regressions on both parameters

| HQ | RfD | Dose |
|---|---|---|
| 7E-10 | 0.01 | 6.63E-12 |
| 0.83 | 0.000112 | 9.35E-05 |
| 0.0004 | 0.3 | 0.000119 |
| 1.4E-06 | 0.3 | 9.5E-10 |

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.776663 |
| R Square | 0.603206 |
| Adjusted | -0.19038 |
| Standard | 0.452711 |
| Observati | 4 |

Therefore Dose is 0.603 - 0.355 0.247566

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 0.311561 | 0.155781 | 0.7601 | 0.629916 |
| Residual | 1 | 0.204947 | 0.204947 | | |
| Total | 3 | 0.516509 | | | |

| | *Coefficient* | *andard Err* | *t Stat* | *P-value* | *ower 95* | *pper 95* | *ower 95.0* | *pper 95.0%* |
|---|---|---|---|---|---|---|---|---|
| Intercept | 0.271232 | 0.382103 | 0.70984 | 0.607015 | -4.58382 | 5.126286 | -4.58382 | 5.126286 |
| RfD | -1.57914 | 1.542799 | -1.02356 | 0.492589 | -21.1822 | 18.02389 | -21.1822 | 18.02389 |
| Dose | 3336.121 | 4223.558 | 0.789884 | 0.574393 | -50329 | 57001.28 | -50329 | 57001.28 |

Correlation using untransformed doses, and one factor for receptor characteristics
Inhalation Pathway, Zinc, Child

receptor factor = (InhR * EF * ET)/BW

| Dose | Cair | Receptor factor |
|---|---|---|
| 6.63E-12 | 1.41E-11 | 4118.4 |
| 9.35E-05 | 0.000146 | 5600 |
| 0.000119 | 0.001684 | 614.5988 |
| 9.51E-10 | 4.96E-10 | 16800 |

| | *Dose* | *Cair* | *ceptor factor* |
|---|---|---|---|
| Dose | 1 | | |
| Cair | 0.76284 | 1 | |
| Receptor f | -0.64671 | -0.61314 | 1 |

Therefore Cair is 0.762^2
0.581925

Do regression on both

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.795777 |
| R Square | 0.633261 |
| Adjusted | -0.10022 |
| Standard | 6.53E-05 |
| Observati | 4 |

Therefore, receptor is 0.633-0.581 = 0.051336

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 7.35E-09 | 3.68E-09 | 0.863367 | 0.60559 |
| Residual | 1 | 4.26E-09 | 4.26E-09 | | |
| Total | 3 | 1.16E-08 | | | |

| | *Coefficient* | *ndard Err* | *t Stat* | *P-value* | *ower 95* | *pper 95* | *ower 95.0* | *pper 95.0* |
|---|---|---|---|---|---|---|---|---|
| Intercept | 5.01E-05 | 7.36E-05 | 0.680055 | 0.619802 | -0.00089 | 0.000985 | -0.00089 | 0.000985 |
| Cair | 0.04452 | 0.058142 | 0.765708 | 0.583983 | -0.69424 | 0.783277 | -0.69424 | 0.783277 |
| Receptor f | -2.6E-09 | 6.82E-09 | -0.37414 | 0.77208 | -8.9E-08 | 8.41E-08 | -8.9E-08 | 8.41E-08 |

Risk Estimate Correlations

Inhalation Pathway
Copper
Child

| Company | HQ | RfD | Dose | Conc | Receptor |
|---|---|---|---|---|---|
| 2 | 0.000000004 | 0.01 | 4.42E-11 | 9.4E-11 | 4118 |
| 3 | 316 | 0.0000024 | 0.00076 | 0.00119 | 5600 |
| 5 | 0.003 | 0.5 | 0.00147 | 2.1E-02 | 6741 |
| 7 | 0.0000105 | 0.5 | 5.24E-06 | 6.1E-09 | 16800 |

receptor = (InhR*EF*ET)/BW

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | [redacted] | 1 | |
| Dose | 0.190362918 | 0.286709548 | 1 |

$R^2$: 0.346859

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.698179982 |
| R Square | 0.487455287 |
| Adjusted R Square | -0.53763414 |
| Standard Error | 195.9215529 |
| Observations | 4 |

Dose: 0.140596

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regression | 2 | 36506.26946 | 18253.13 | 0.47552 | 0.715922 |
| Residual | 1 | 38385.25488 | 38385.25 | | |
| Total | 3 | 74891.52435 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *ower 95%* |
|---|---|---|---|---|---|
| Intercept | 127.8273936 | 153.7668578 | 0.831307 | 0.55848 | -1825.96 |
| RfD | -387.6031354 | 413.1048706 | -0.93827 | 0.52027 | -5636.58 |
| Dose | 87763.96688 | 167569.6466 | 0.523746 | 0.69285 | -2041401 |

**TIER II**

| | *Dose* | *Conc* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Conc | [redacted] | 1 | |
| Receptor | -0.355949353 | -0.203964935 | 1 |

$R^2$: 0.790047

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.906573601 |
| R Square | 0.821875695 |
| Adjusted R Square | 0.465627084 |
| Standard Error | 0.00051508 |
| Observations | 4 |

Receptor: 0.031829

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.22415E-06 | 6.12E-07 | 2.30703 | 0.422048 |
| Residual | 1 | 2.65308E-07 | 2.65E-07 | | |
| Total | 3 | 1.48945E-06 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *ower 95%* |
|---|---|---|---|---|---|
| Intercept | 0.00042116 | 0.00056105 | 0.750664 | 0.59006 | -0.00671 |
| Conc | 0.059477311 | 0.03010687 | 1.975539 | 0.29831 | -0.32307 |
| Receptor | -2.23017E-08 | 5.27581E-08 | -0.42272 | 0.74539 | -6.9E-07 |

Risk Estimate Correlations

Inhalation Pathway
Lead
Child

| Company | HQ | RfD | Dose | Conc | Receptor |
|---|---|---|---|---|---|
| 2 | 0.00000007 | 0.00043 | 2.87E-11 | 6.1E-11 | 4118 |
| 3 | 382 | 0.000001 | 0.000382 | 0.0006 | 5600 |
| 5 | 0.27 | 0.00185 | 0.000494 | 6.9E-03 | 6741 |
| 7 | 0.000495 | 0.00357 | 1.77E-06 | 2.1E-09 | 700 |

receptor = (InhR*EF*ET)/BW

**TIER I**

| | *HQ* | *RfD* | *Dose* |
|---|---|---|---|
| HQ | 1 | | |
| RfD | [redacted] | 1 | |
| Dose | 0.423141119 | -0.292987818 | 1 |

$R^2$: 0.365577

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.657093147 |
| R Square | 0.431771404 |
| Adjusted R Square | -0.704685789 |
| Standard Error | 249.3177009 |
| Observations | 4 |

Dose: 0.066194

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 47232.07404 | 23616.04 | 0.37993 | 0.753809391 |
| Residual | 1 | 62159.31601 | 62159.32 | | |
| Total | 3 | 109391.39 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 142.7203927 | 247.2082006 | 0.577329 | 0.66668 | -2998.344159 |
| RfD | -62291.67816 | 93404.7994 | -0.6669 | 0.62556 | -1249107.098 |
| Dose | 200345.7938 | 586991.0784 | 0.34131 | 0.79061 | -7258051.074 |

**TIER II**

| | *Dose* | *Conc* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Conc | 0.770045529 | 1 | |
| Receptor | [redacted] | 0.669014636 | 1 |

$R^2$: 0.713667

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.888619651 |
| R Square | 0.789644883 |
| Adjusted R Square | 0.36893465 |
| Standard Error | 0.000203745 |
| Observations | 4 |

Cair: 0.075978

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.55831E-07 | 7.79E-08 | 1.87693 | 0.45864487 |
| Residual | 1 | 4.15121E-08 | 4.15E-08 | | |
| Total | 3 | 1.97343E-07 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -8.38828E-05 | 0.000233658 | -0.359 | 0.78058 | -0.003052778 |
| Conc | 0.028415911 | 0.047281844 | 0.60099 | 0.6555 | -0.5723543 |
| Receptor | 5.83416E-08 | 6.03365E-08 | 0.966936 | 0.5107 | -7.08303E-07 |

Risk Estimate Correlations

Inhalation Pathway
Cadmium (Carcinogen)
Child

| Company | LCR | SF | Dose | Conc | Receptor |
|---|---|---|---|---|---|
| 2 | 3E-14 | 1.125 | 2.48E-14 | 7.38E-13 | 4118 |
| 3 | 0.000278 | 101 | 3E-06 | 1.01E-05 | 5600 |
| 5 | 9.1E-05 | 6.3 | 1.4E-05 | 2.0E-04 | 6741 |
| 7 | 6.65E-11 | 6.3 | 4.19E-10 | 6.02E-11 | 16800 |

receptor = (InhR*EF*ET)/BW

**TIER I**

| | *LCR* | *SF* | *Dose* |
|---|---|---|---|
| LCR | 1 | | |
| SF | [illegible] | 1 | |
| Dose | 0.196703841 | -0.109101842 | 1 |

$R^2$: 0.906693

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.999067554 |
| R Square | 0.998135977 |
| Adjusted R Square | 0.994407932 |
| Standard Error | 9.80022E-06 |
| Observations | 4 |

Dose: 0.091443

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 5.14292E-08 | 2.57E-08 | 267.7371 | 0.043174328 |
| Residual | 1 | 9.60443E-11 | 9.6E-11 | | |
| Total | 3 | 5.15253E-08 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | -9.78541E-06 | 7.14951E-06 | -1.36868 | 0.4017 | -0.000100628 |
| SF | 2.67513E-06 | 1.17913E-07 | 22.68737 | 0.028042 | 1.17691E-06 |
| Dose | 5.820000964 | 0.830945104 | 7.004074 | 0.090283 | -4.738112419 |

**TIER II**

| | *Dose* | *Conc* | *Receptor* |
|---|---|---|---|
| Dose | 1 | | |
| Conc | [illegible] | 1 | |
| Receptor | -0.259408302 | -0.201053067 | 1 |

$R^2$: 0.975482

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.98961534 |
| R Square | 0.979338521 |
| Adjusted R Square | 0.938015564 |
| Standard Error | 1.70547E-06 |
| Observations | 4 |

Receptor: 0.003857

ANOVA

| | *df* | *SS* | *MS* | *F* | *Significance F* |
|---|---|---|---|---|---|
| Regression | 2 | 1.37867E-10 | 6.89E-11 | 23.69962 | 0.143741012 |
| Residual | 1 | 2.90863E-12 | 2.91E-12 | | |
| Total | 3 | 1.40776E-10 | | | |

| | *Coefficients* | *Standard Error* | *t Stat* | *P-value* | *Lower 95%* |
|---|---|---|---|---|---|
| Intercept | 1.40585E-06 | 1.85325E-06 | 0.75859 | 0.586849 | -2.21418E-05 |
| Conc | 0.067143707 | 0.010105962 | 6.64397 | 0.095105 | -0.06126417 |
| Receptor | -7.54268E-11 | 1.74579E-10 | -0.43205 | 0.740369 | -2.29365E-09 |

Risk Estimate Correlations

Inhalation of Volatiles Pathway, Outdoor
Benzene
Adult

| Company | LCR | SF | Dose |
|---|---|---|---|
| 2 | 1.00E-06 | 0.019 | 7.00E-05 |
| 4(ave) | 4.00E-08 | 0.0291 | 1.00E-06 |
| 8 | 2.05E-07 | 0.027 | 8.00E-06 |
| 9 | 4.10E-07 | 0.029 | 1.42E-05 |

Note: Company #6 not used since they report totaol indoor/outdoor dose

| | *LCR* | *SF* | *Dose* |
|---|---|---|---|
| LCR | 1 | | |
| SF | -0.91178 | 1 | |
| Dose | 0.980178 | -0.96763 | 1 |

No regression possible because of colinearity

Correlation using untransformed doses, and one factor for receptor characteristics
Inhalation Pathway, Benzene, Outdoors, Adult

receptor factor = (InhR*EF*ET*ED)/(BW*AT)

| Dose | Cair | Receptor factor |
|---|---|---|
| 7.5E-05 | 1.8E-03 | 364.2122 |
| 1.4E-06 | 2.2E-03 | 5.70769 |
| 1.2E-08 | 2.7E-06 | 38.57143 |
| 7.6E-06 | 4.0E-04 | 168.4615 |
| 1.4E-05 | 6.5E-04 | 191.8857 |

| | *Dose* | *Cair* | *ceptor factor* | |
|---|---|---|---|---|
| Dose | 1 | | | |
| Cair | 0.431018 | 1 | | |
| Receptor f | 0.905723 | 0.155266 | 1 | Therefore, receptor = 0.905^2 |
| | | | | 0.820334 |

Do regression on both

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.952231 |
| R Square | 0.906744 |
| Adjusted | 0.813488 |
| Standard | 1.35E-05 |
| Observati | 5 |

Therefore, Cair is 0.906-0.820 = 0.086409

ANOVA

| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 3.57E-09 | 1.78E-09 | 9.723162 | 0.093256 |
| Residual | 2 | 3.67E-10 | 1.83E-10 | | |
| Total | 4 | 3.93E-09 | | | |

| | *Coefficient* | *ndard Err* | *t Stat* | *P-value* | *ower 95* | *pper 95* | *ower 95.0* | *pper 95.0%* |
|---|---|---|---|---|---|---|---|---|
| Intercept | -1.9E-05 | 1.13E-05 | -1.71826 | 0.227888 | -6.8E-05 | 2.93E-05 | -6.8E-05 | 2.93E-05 |
| Cair | 0.009896 | 0.007269 | 1.36131 | 0.306497 | -0.02138 | 0.041172 | -0.02138 | 0.041172 |
| Receptor f | 1.89E-07 | 4.81E-08 | 3.932188 | 0.059008 | -1.8E-08 | 3.96E-07 | -1.8E-08 | 3.96E-07 |

Risk Estimate Correlations

Inhalation of Volatiles Pathway, Indoor
Benzene
Child

| Company | LCR | SF | Dose |
|---|---|---|---|
| 2 | 1.00E-05 | 0.005 | 3.00E-03 |
| 5 | 7.20E-04 | 0.3 | 2.47E-02 |
| 6 | 9.50E-09 | 0.029 | 3.27E-07 |
| 7 | 1.30E-04 | 0.027 | 4.40E-03 |

| | *LCR* | *SF* | *Dose* |
|---|---|---|---|
| LCR | 1 | | |
| SF | 0.986953 | 1 | |
| Dose | 0.994014 | 0.98016 | 1 |

No regression possible because of colinearity

Correlation using untransformed doses, and one factor for receptor characteristics
Inhalation Pathway, Benzene, Indoors, Child

receptor factor = (InhR*EF*ET*ED)/(BW*AT)

| Dose | Cair | Receptor factor |
|---|---|---|
| 0.0027 | 0.16 | 6.128571 |
| 0.025 | 0.27 | 41.18972 |
| 3.27E-07 | 2.69E-06 | 38.016 |
| 0.0044 | 0.03 | 56 |

| | *Dose* | *Cair* | *ceptor factor* |
|---|---|---|---|
| Dose | 1 | | |
| Cair | 0.846265 | 1 | |
| Receptor f | 0.219621 | -0.3153 | 1 |

Therefore, Cair is 0.846^2
0.716164

Do regression on both

SUMMARY OUTPUT

| *Regression Statistics* | |
|---|---|
| Multiple R | 0.989403 |
| R Square | 0.978918 |
| Adjusted | 0.936755 |
| Standard | 0.002882 |
| Observati | 4 |

Therefore, receptor is 0.978-0.716 = 0.262754

ANOVA

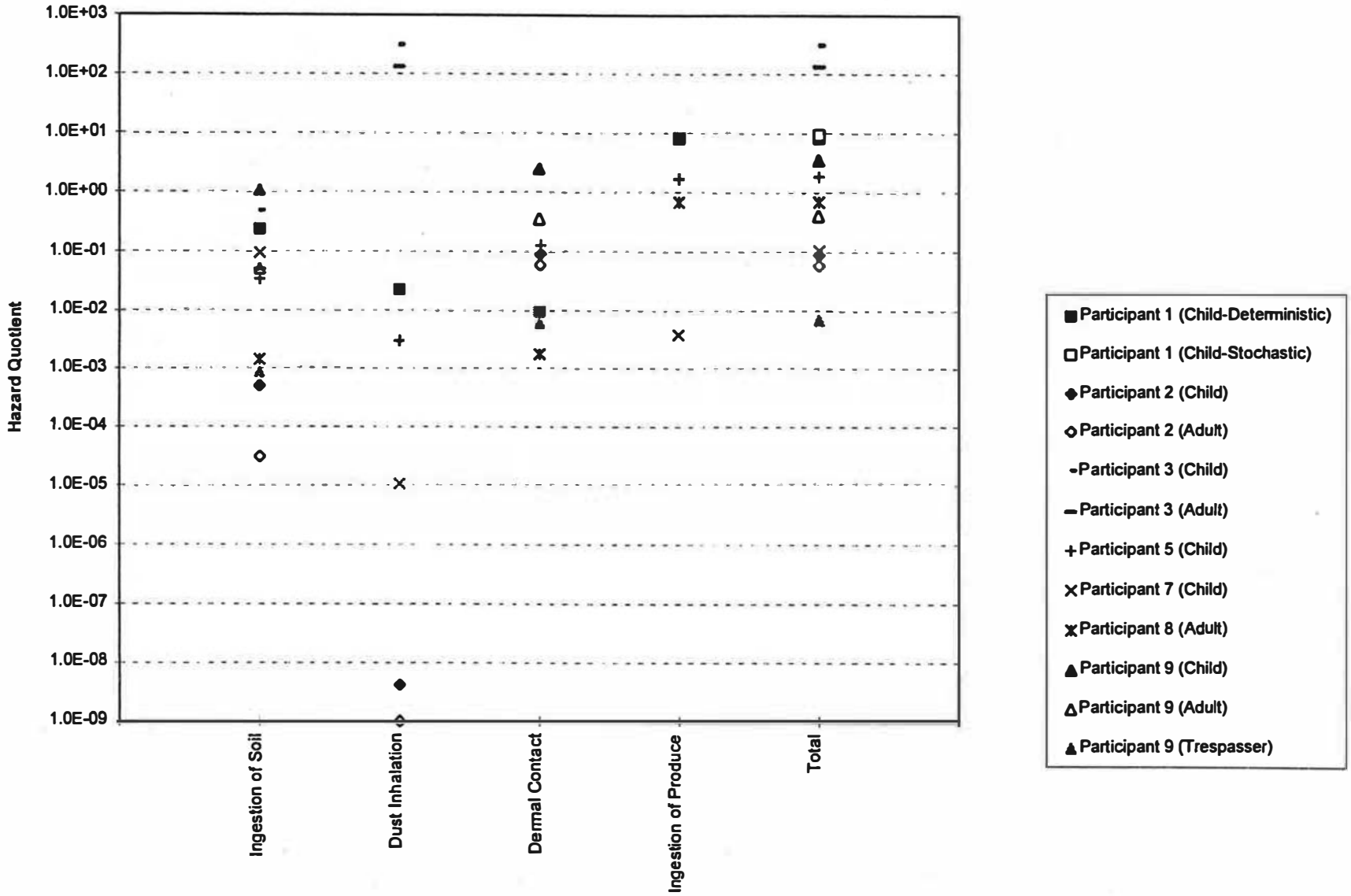
| | *df* | *SS* | *MS* | *F* | *gnificance F* |
|---|---|---|---|---|---|
| Regressio | 2 | 0.000386 | 0.000193 | 23.21732 | 0.145195 |
| Residual | 1 | 8.31E-06 | 8.31E-06 | | |
| Total | 3 | 0.000394 | | | |

| | *Coefficient* | *ndard Err* | *t Stat* | *P-value* | *ower 95* | *pper 95* | *ower 95.0* | *pper 95.0* |
|---|---|---|---|---|---|---|---|---|
| Intercept | -0.01316 | 0.004053 | -3.24631 | 0.190234 | -0.06466 | 0.038344 | -0.06466 | 0.038344 |
| Cair | 0.093581 | 0.014084 | 6.644299 | 0.095101 | -0.08538 | 0.272539 | -0.08538 | 0.272539 |
| Receptor f | 0.000295 | 8.35E-05 | 3.530389 | 0.175723 | -0.00077 | 0.001356 | -0.00077 | 0.001356 |

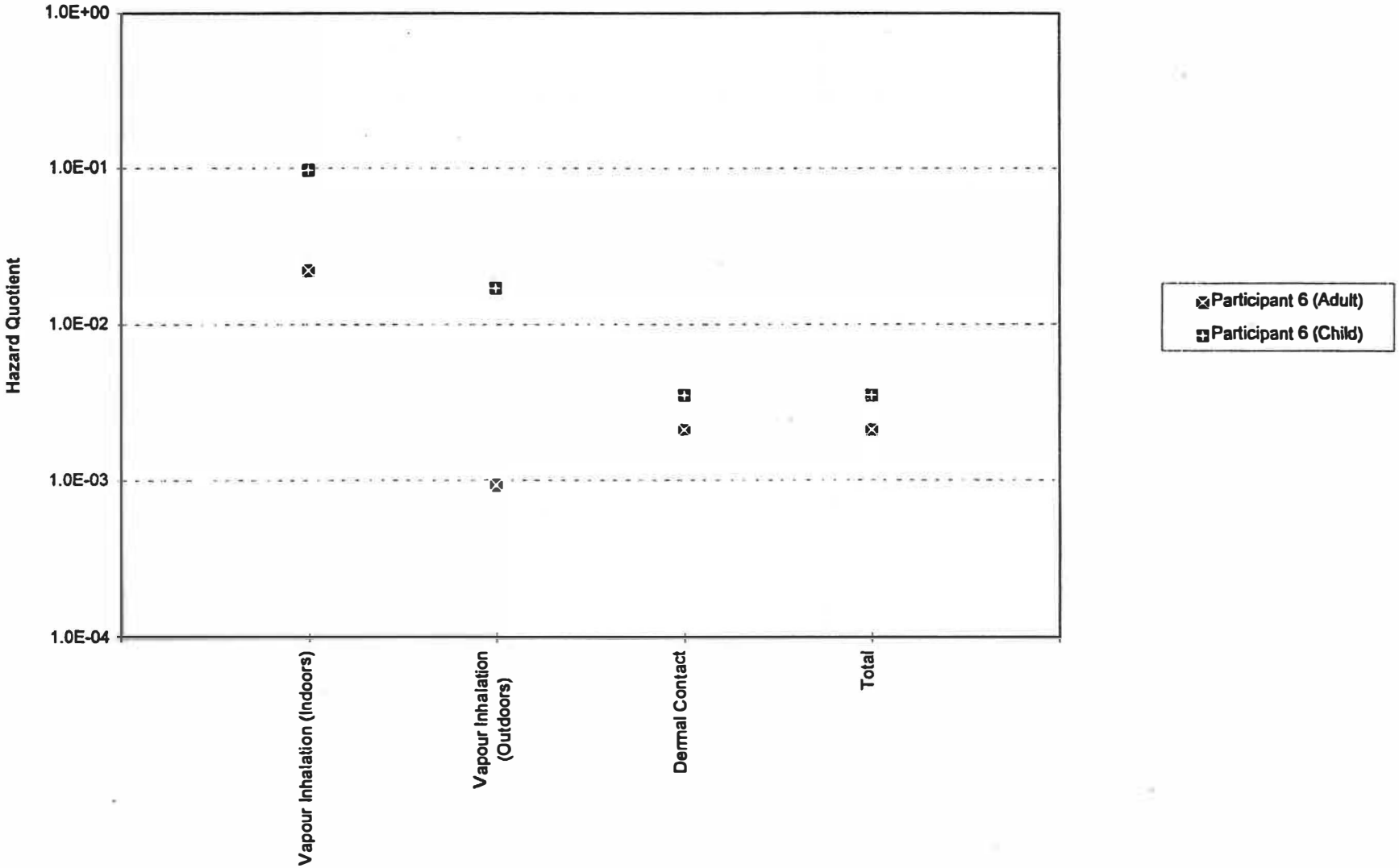
# PART B

# SCATTER PLOTS OF RISK ESTIMATES AND DOSE RATES

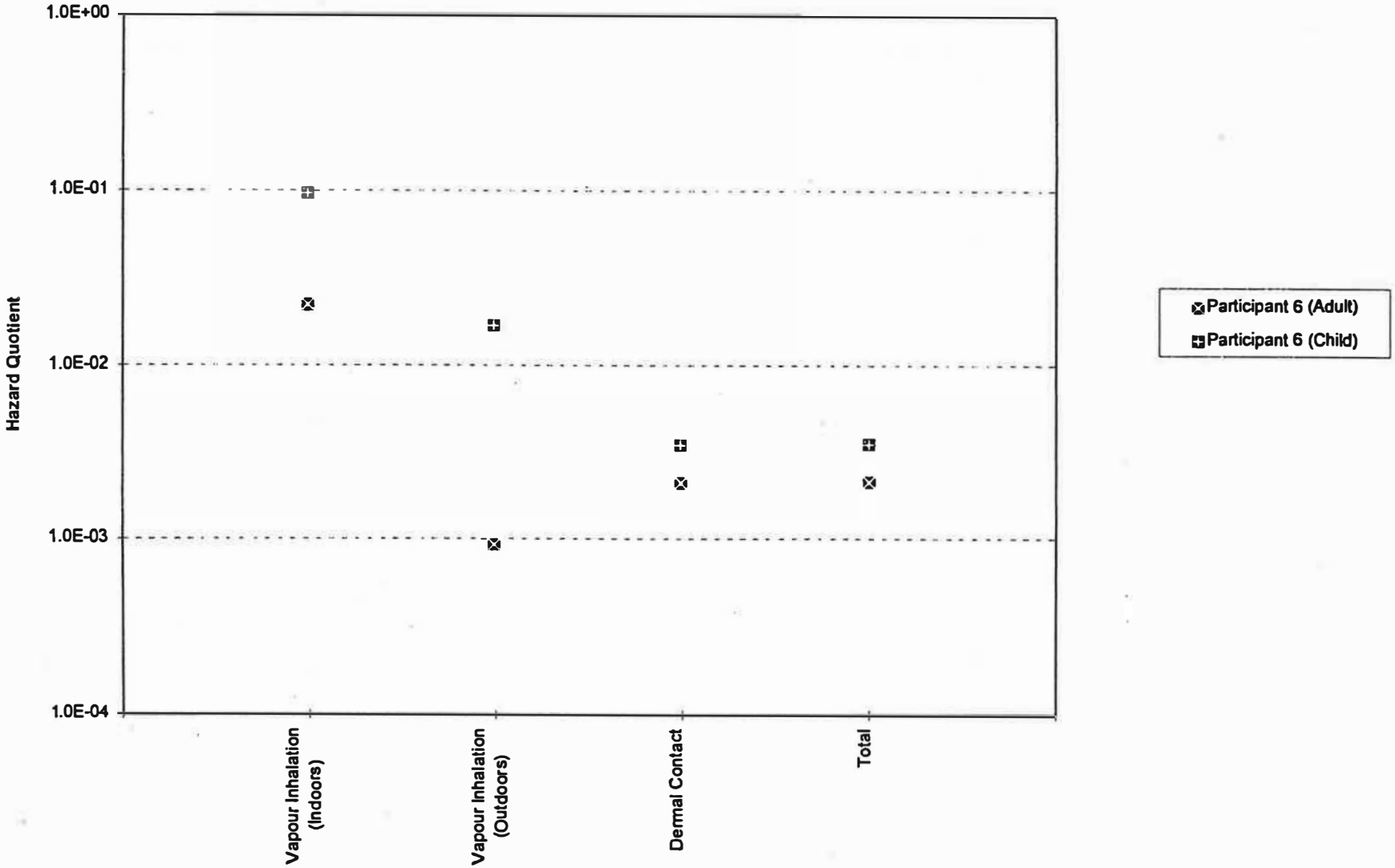
## Copper - Non-Cancer Risks



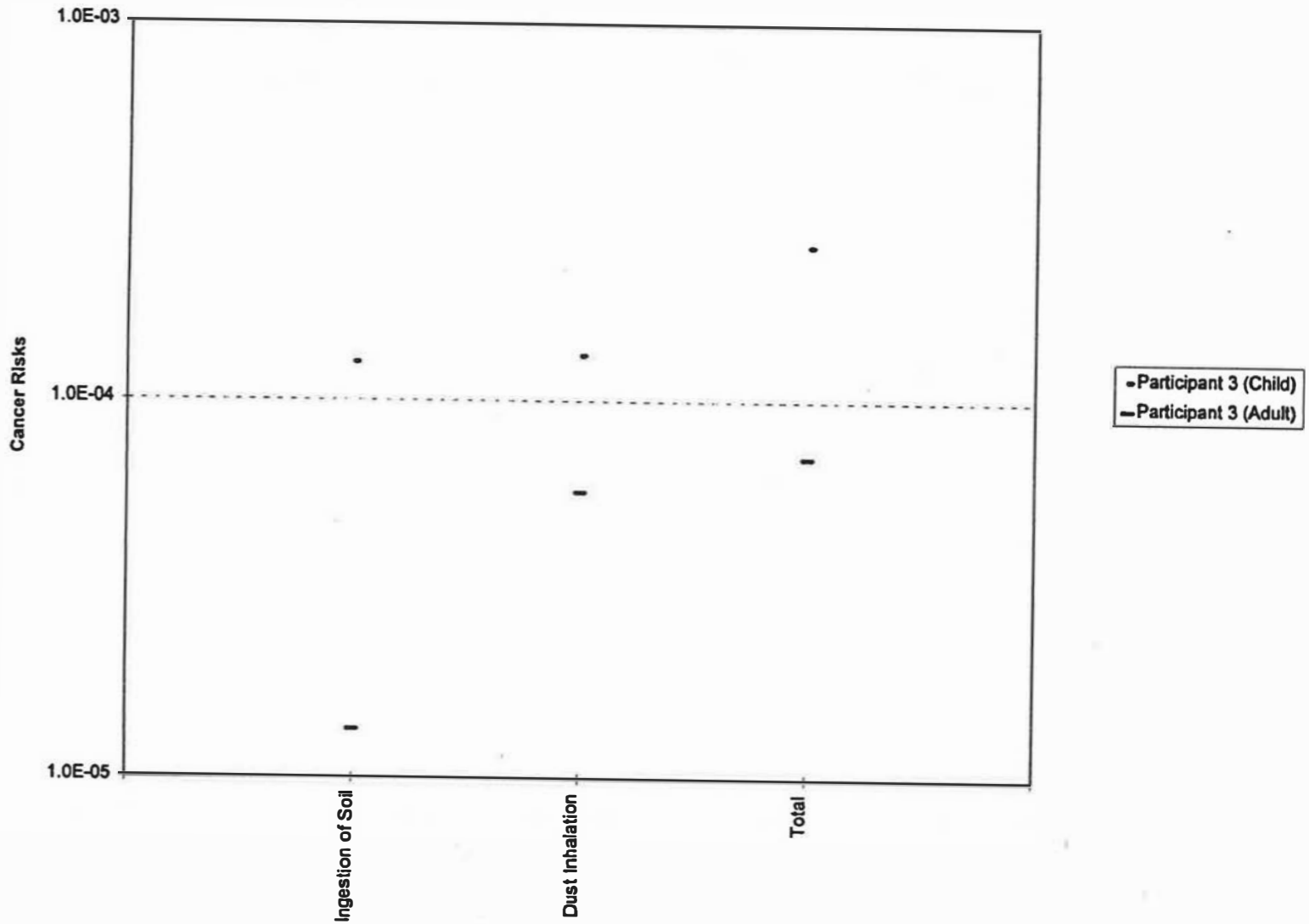
## Vinyl Chloride - Non-Cancer Risks



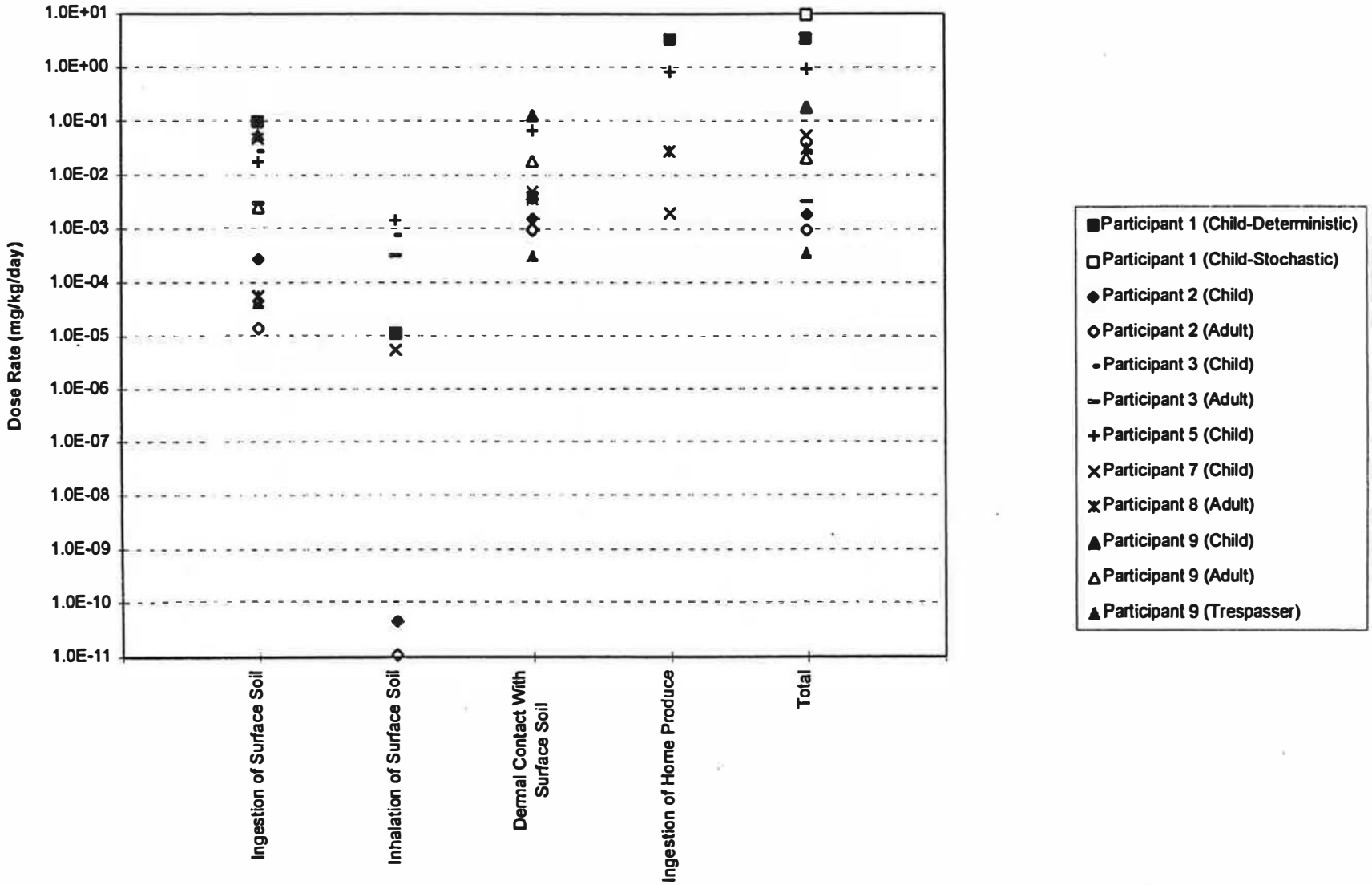
## Vinyl Chloride - Non-Cancer Risks



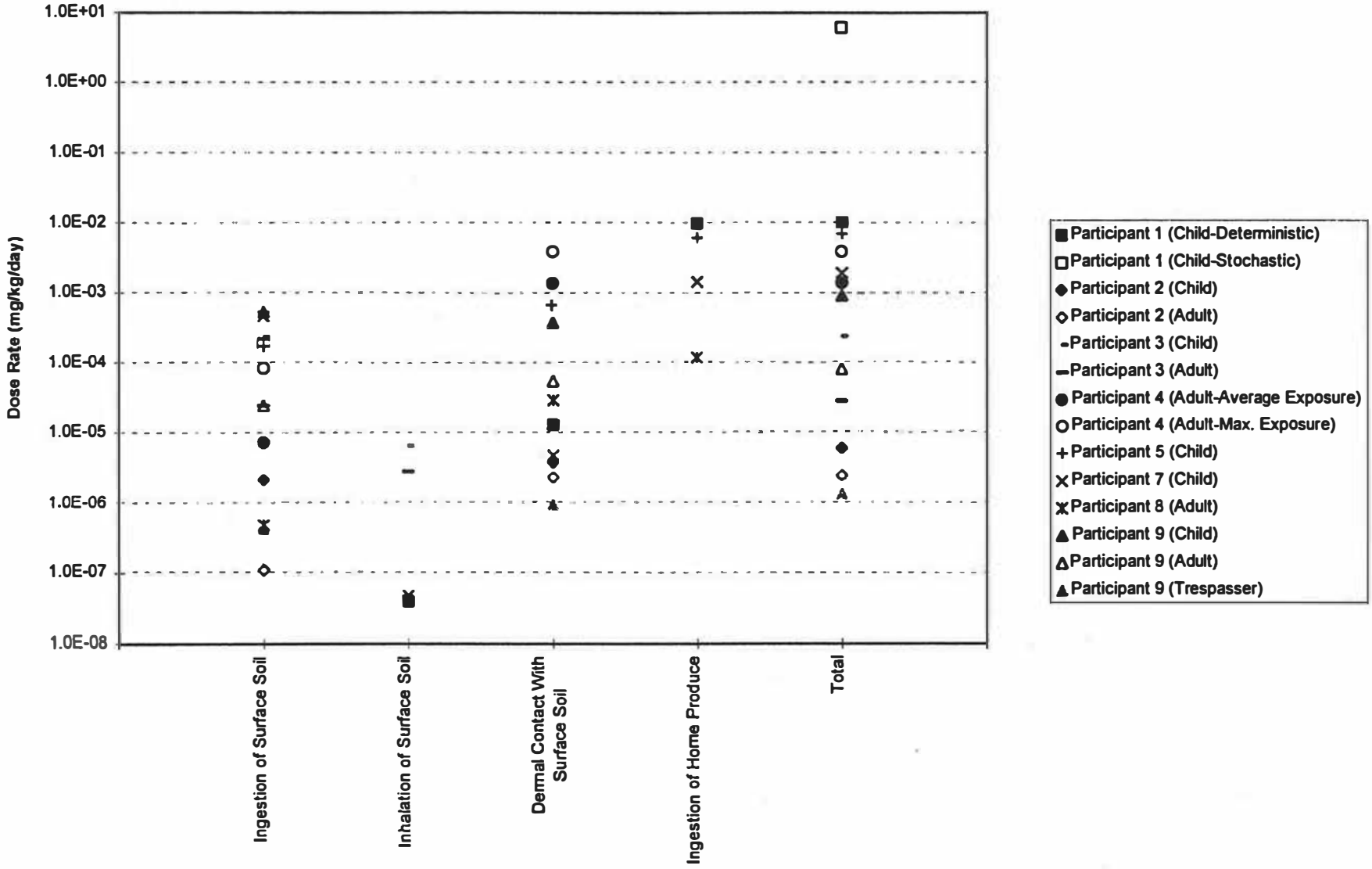
## Lead - Cancer Risks



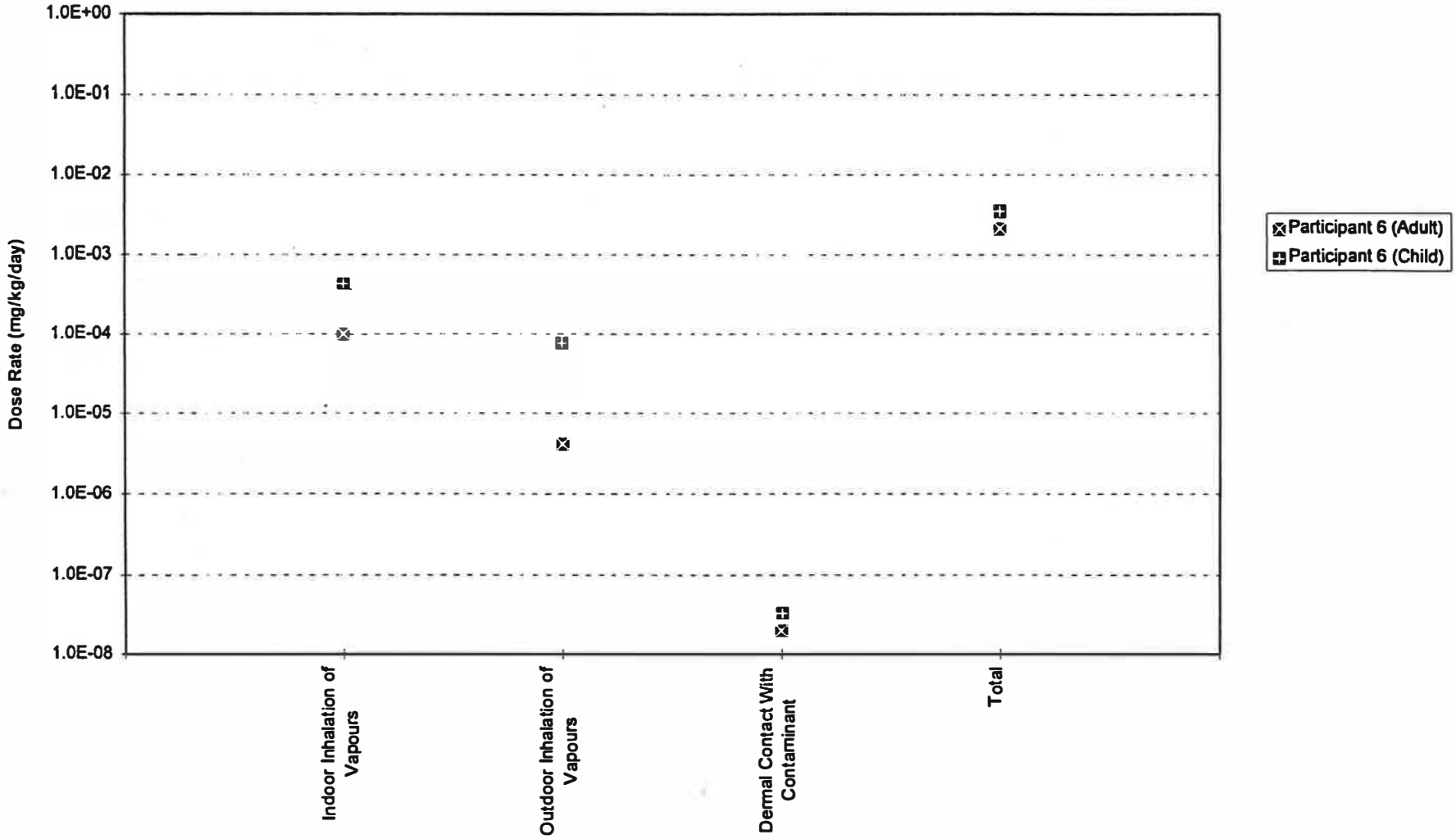
## Dose Rates - Copper (Non-Carcinogen)



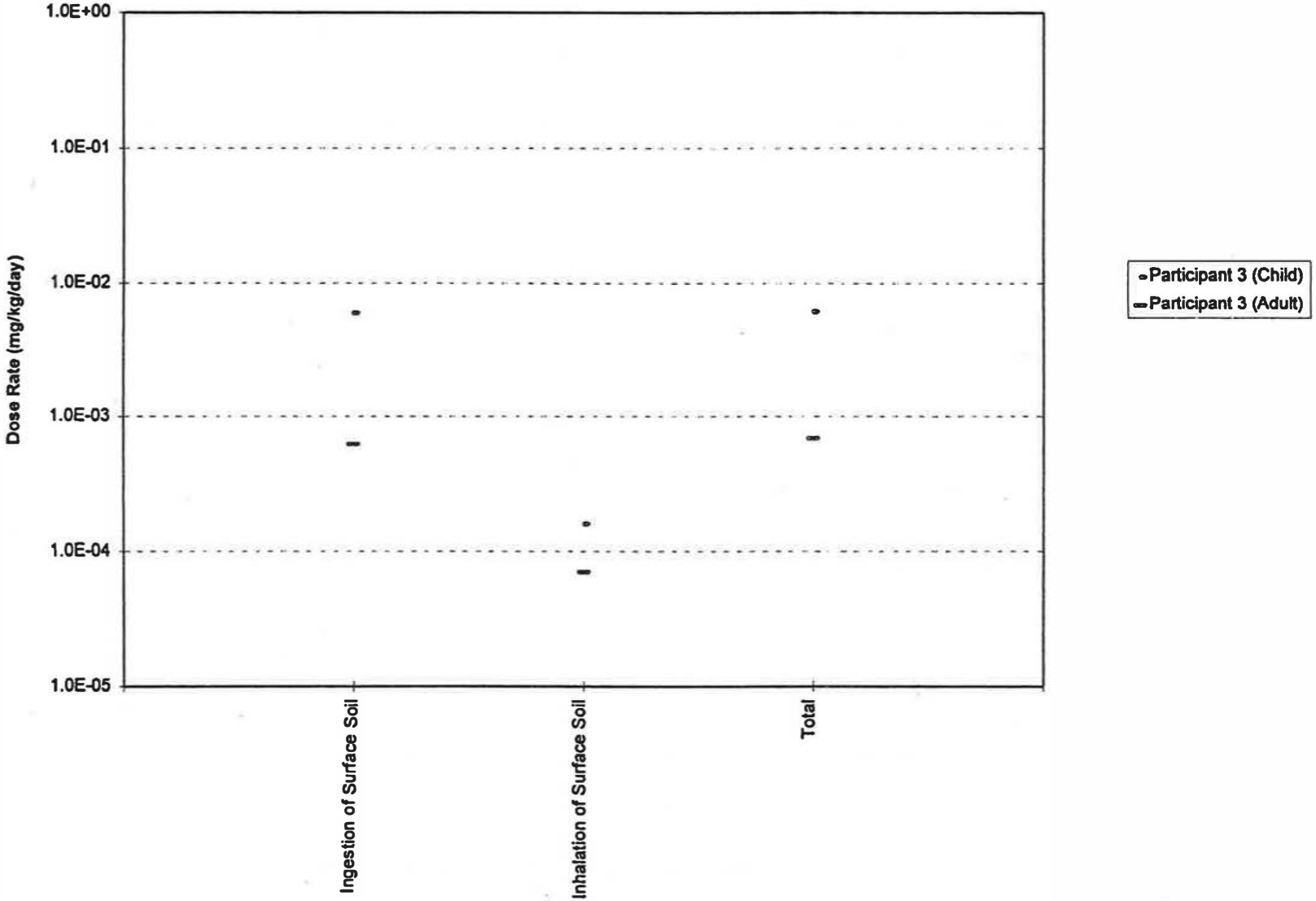
## Cadmium - Dose Rates (Non-Carcinogen)

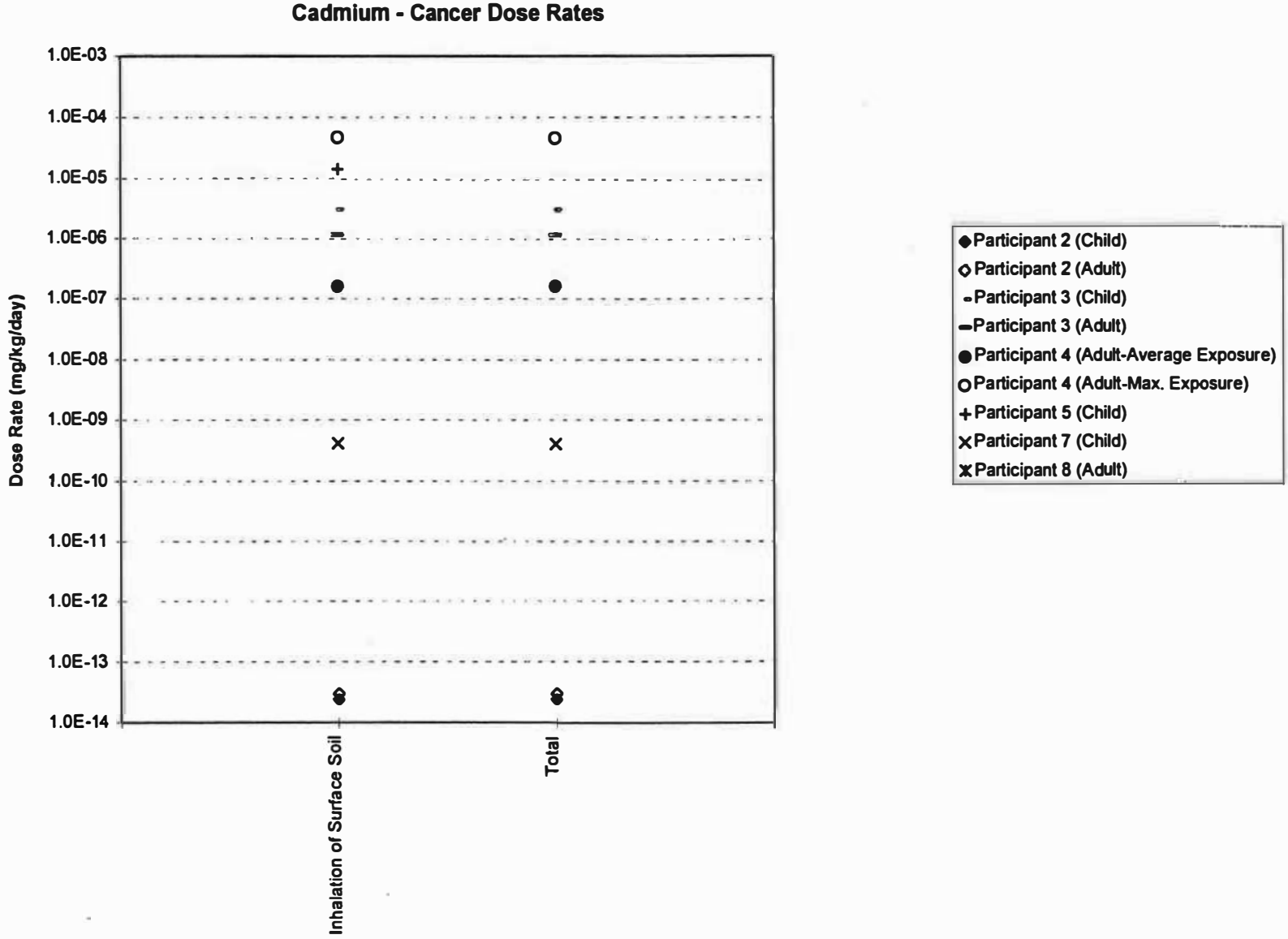


## Dose Rates - Vinyl Chloride (Non-Carcinogen)



## Lead - Cancer Dose Rates




Cadmium - Cancer Dose Rates
Dose Rate (mg/kg/day)
1.0E-03
1.0E-04
1.0E-05
1.0E-06
1.0E-07
1.0E-08
1.0E-09
1.0E-10
1.0E-11
1.0E-12
1.0E-13
1.0E-14
Inhalation of Surface Soil
Total
Participant 2 (Child)
Participant 2 (Adult)
Participant 3 (Child)
Participant 3 (Adult)
Participant 4 (Adult-Average Exposure)
Participant 4 (Adult-Max. Exposure)
Participant 5 (Child)
Participant 7 (Child)
Participant 8 (Adult)

# PART C

# CORRELATIONS BETWEEN RISK ESTIMATES AND CAPABILITY SCORES

## Risk Estimate Correlations with Capability

Soil Ingestion Pathway
Copper
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.0005 | 23.5 |
| 3 | 0.482 | 26.5 |
| 5 | 0.035 | 38 |
| 7 | 0.095 | 39 |
| 9 | 1.1037 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | 0.006566747 | 1 |

Risk Estimate Correlations with Capability

Soil Ingestion Pathway
Copper
Adult

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.00003 | 23.5 |
| 3 | 0.05 | 26.5 |
| 8 | 0.00141 | 16 |
| 9 | 0.051243 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | 0.81265422 | 1 |

Risk Estimate Correlations with Capability

Soil Ingestion Pathway
Lead
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.09 | 23.5 |
| 3 | 13821 | 26.5 |
| 5 | 3.2 | 38 |
| 7 | 4.48 | 39 |
| 9 | 5 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | -0.46076279 | 1 |

Risk Estimate Correlations with Capability

Soil Ingestion Pathway
Lead
Adult

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.002 | 23.5 |
| 3 | 1481 | 26.5 |
| 8 | 32.6 | 16 |
| 9 | 0.1233878 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | 0.11740924 | 1 |

Risk Estimate Correlations with Capability

Soil Ingestion Pathway
Cadmium
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.002 | 23.5 |
| 3 | 0.47 | 26.5 |
| 5 | 0.34 | 38 |
| 7 | 0.5742 | 39 |
| 9 | 0.678 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | 0.608142948 | 1 |

## Risk Estimate Correlations with Capability

### Soil Ingestion Pathway
### Cadmium
### Adult

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.00010 | 23.5 |
| 3 | 0.05 | 26.5 |
| 4 | 0.0073 | 11 |
| 8 | 0.000465 | 16 |
| 9 | 0.0315 | 34 |

**Correlation with Risk and Compatibility**

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibi | 0.632713832 | 1 |

Risk Estimate Correlations with Capability

Dermal Contact Pathway
Copper
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.09 | 23.5 |
| 5 | 0.13 | 38 |
| 7 | 0.0099 | 39 |
| 9 | 2.5365497 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | 0.025284226 | 1 |

Risk Estimate Correlations with Capability

Dermal Contact Pathway
Lead
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 15.2 | 23.5 |
| 5 | 12.2 | 38 |
| 7 | 0.4653 | 39 |
| 9 | 0.222098 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibi | -0.589920777 | 1 |

Risk Estimate Correlations with Capability

Dermal Contact Pathway
Cadmium
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.4 | 23.5 |
| 5 | 1.3 | 38 |
| 7 | 0.0058 | 39 |
| 9 | 0.4725 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | 0.15965723 | 1 |

Risk Estimate Correlations with Capability

Dermal Contact Pathway
Cadmium
Adult

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.2 | 23.5 |
| 4(ave.) | 1.36 | 11 |
| 8 | 0.0115 | 16 |
| 9 | 0.067725 | 34 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | -0.65155794 | 1 |

Risk Estimate Correlations with Capability

Inhalation Pathway
Copper
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.000000004 | 23.5 |
| 3 | 316 | 26.5 |
| 5 | 0.003 | 38 |
| 7 | 0.0000105 | 39 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | -0.443010693 | 1 |

Risk Estimate Correlations with Capability

Inhalation Pathway
Lead
Child

| Company | HQ | Capability |
|---|---|---|
| 2 | 0.00000007 | 23.5 |
| 3 | 382 | 26.5 |
| 5 | 0.27 | 38 |
| 7 | 0.000495 | 39 |

Correlation with Risk and Compatibility

| | *HQ* | *Capability* |
|---|---|---|
| HQ | 1 | |
| Compatibility | -0.442745158 | 1 |

Risk Estimate Correlations with Capability

Inhalation Pathway
Cadmium (Carcinogen)
Child

| Company | LCR | Capability |
|---|---|---|
| 2 | 3E-14 | 23.5 |
| 3 | 0.000278 | 26.5 |
| 5 | 9.1E-05 | 38 |
| 7 | 6.65E-11 | 39 |

Correlation with Risk and Compatibility

| | *LCR* | *Capability* |
|---|---|---|
| LCR | 1 | |
| Compatibility | -0.287172982 | 1 |