TECHNICAL SUPPORT FOR
ROCKY MOUNTAIN ARSENAL

FINAL
INTEGRATED ENDANGERMENT ASSESSMENT/ RISK CHARACTERIZATION
VERSION 4.2

VOLUME IV of IV

Appendices D, E, and F

JULY 1994
CONTRACT NO. DAAA05-92-D-0002, Delivery Order 0004

Prepared by:

EBASCO SERVICES INCORPORATED
James M. Montgomery
International Dismantling & Machinery
Greystone Environmental
Hazen Research
Data Chem
BC Analytical
Terra Technologies

Prepared for:

U.S. Army Program Manager's Office for the
Rocky Mountain Arsenal

APPENDIX D

IMPLEMENTATION OF RISK MODELS

TABLE OF CONTENTS

TABLE OF CONTENTS (cont'd.)

LIST OF TABLES

LIST OF FIGURES

LIST OF ATTACHMENTS

LIST OF ACRONYMS AND ABBREVIATIONS

ASCII	American Standard Code for Information Interchange
Army	U.S. Army
BCRL	below certified reporting limit
BAF	bioaccumulation factor
BMF	biomagnification factor
CDF	cumulative distribution function
cm	centimeters
C_{max}	maximum concentration
CMP	Comprehensive Monitoring Program
COC	contaminant of concern
C_{ow}	chemical concentration in water
CR	cancer risk
CRL	certified reporting limit
C_{rep}	representative concentration
CrepMean	name of the HHRC code case (provided on diskettes) that uses C_{rep} = sample arithmetic mean
Crep95UCL	name of the HHRC code case (provided on diskettes) that uses C_{rep} = 95th upper confidence limit (bootstrap method) on the sample arithmetic mean
Crep95LCL	name of the HHRC code case (provided on diskettes) that uses C_{rep} = 95th lower confidence limit (bootstrap method) on the sample arithmetic mean
CSS	dust loading factor
DDE	dichlorodiphenyldichloroethene
DDT	dichlorodiphenyltrichloroethane
DT	critical toxicity values
EI	exposure index values
EPA	U.S. Environmental Protection Agency
ERC	Ecological Risk Characterization computer program
ESC	estimated soil concentration
F_{oc}	fraction of organic carbon content in soils or sediment
ft	foot, feet
g	gram
GIS	geographic information system
GCRL	greater than certified reporting limit
GW	groundwater
Hg	mercury
HHEA	Human Health Exposure Assessment
HHRC	Human Health Risk Characterization computer program
HI	hazard index
HQ	hazard quotient
IEA/RC	Integrated Endangerment Assessment/Risk Characterization
K_2	depuration rate coefficient

Kg	Kilogram
K_{oc}	soil-water partition coefficients normalized to organic carbon
LCL	lower confidence limit
LHS	Latin Hypercube sampling routines
ln	natural logarithm (logarithm to the base e, where e = 2.718...)
log	Base-10 logarithm
LSE	least squared error
LSP	least squares procedure
m	meter
MATC	maximum allowable tissue concentration
MB	megabyte
µg	micrograms
$\mu g/m^3$	micrograms per cubic meter
mg	milligrams
mg/m^3	milligrams per cubic meter
MLE	most likely estimate
NA	not applicable
NE	not estimated
PBC	probabilistic biota criteria
PDF	probability density function
PPLV	preliminary pollutant limit values
ppm	parts per million
Q	ratio of sediment to pore water chemical concentrations
QA/QC	quality assurance/quality control
RAF	relative absorption factor
RDM	Raima Data Manager, Version 3.2.1
RI	remedial investigation
RMA	Rocky Mountain Arsenal
RMADMS	Rocky Mountain Arsenal Data Management System
RME	reasonable maximum exposure
SAR	Study Area Report
SDK	Software Development Kit (Microsoft)
S-PLUS	S-PLUS Statistical Software
SPPPLV	single pathway preliminary pollutant limit value
TC	tissue concentration
TRV	toxicity reference value
UCL	upper confidence limit

D.1 RISK CHARACTERIZATION MODEL IMPLEMENTATION

D.1.1 MODEL IMPLEMENTATION OVERVIEW

Computer models for the Rocky Mountain Arsenal (RMA) human health risk characterization (HHRC) and ecological risk characterization (ERC) programs were developed to determine human health and ecological probabilistic risk-based criteria. The models are composed of sequences of equations that calculate risk-based criteria by simulating actual human or ecological exposure possibilities at RMA. Detailed descriptions of the computational methodologies behind the HHRC and ERC models are discussed in Appendices B and C, respectively.

Two Microsoft Windows-based software applications, the HHRC program and the ERC program, were created to implement the models and provide user interaction; the original ERC code has since been replaced by an improved method for calculating ecological health threats described in Section D.1.3. The HHRC program is a user-friendly interactive application that runs under Microsoft Windows, Version 3.1 (Microsoft Corporation 1992). It was written in Microsoft Visual C, Version 7.0, using the Microsoft Software Development Kit (SDK), Version 3.1. The database manager used by the HHRC code was developed by Raima Corporation and is called Raima Data Manager (RDM), Version 3.2.1 (Raima Corporation 1991).

The minimum hardware requirements for running the HHRC program is as follows:

Computer type:	IBM or 100 percent IBM compatible with 4 megabyte (MB) RAM (recommended 386 or higher with 6 MB of RAM)
Monitor type:	EGA/VGA
Floppy Drive type:	3.5-inch, high density
Hard Drive capacity:	100 MB minimum with 60 MB free (recommended 100 MB free)
Printer:	Hewlett Packard Laser Jet or compatible
Software:	Microsoft Windows Version 3.1
Miscellaneous:	Mouse device

D.1.2 HUMAN HEALTH RISK CHARACTERIZATION MODEL IMPLEMENTATION

The HHRC model discussed in Appendix B was developed to determine probabilistic preliminary pollutant limit values (PPLVs) and risks (throughout Appendix D the term "risk" represents both carcinogenic risks and noncarcinogenic hazard indices [HIs] unless stated otherwise) for five specific populations (biological worker, commercial worker, industrial worker, recreational visitor, and regulated/casual visitor) projected to frequent RMA after remediation. This section describes the computer programs that are used to implement this model.

The human health risk model implementation is broken into two parts. The first part consists of a series of preprocessor programs (Clipper programs [Nantucket Corporation 1990] and SampleCalc) that extract data from the primary sample database and calculate exposure point concentrations for every site and every depth horizon at RMA. These programs are discussed in detail in Section D.1.4.

The second part is an interactive Microsoft Windows-based program called the HHRC code. The HHRC code serves two purposes in addition to computation of PPLVs and risks. First, it allows the user to display and download the PPLVs and risks calculated for the IEA/RC. Second, it allows the user to re-run model calculations using input parameters differing from those used in the Integrated Endangerment Assessment/Risk Characterization (IEA/RC) and then to compare the resulting differences in PPLVs and risks. The HHRC program consists of four code files (RMA.EXE, VISTA.DLL, MOD.DLL, AND DBV.DLL) and several RDM database files (*.DBV) that contain input data and results. Use of the HHRC program is explained below in Section D.1.2.1.

D.1.2.1 HHRC Program

The HHRC program uses the Latin Hypercube sampling process to generate a set of input parameters from which it calculates probabilistic PPLVs and risks. Since the computational methodology and Latin Hypercube sampling process are explained elsewhere (Appendix Section B.1 and Appendix E, respectively), this section focuses on the logistics of using the HHRC program.

Each time the HHRC program is run with a new set of input parameters, it creates a new set of results (PPLVs and risks). The HHRC program saves each set of results in RDM database files, along with the corresponding set of input parameters. Each set of parameters and results is called a case. As shipped with the IEA/RC, the HHRC code contains the three cases evaluated in the report: CrepMean, Crep95UCL, and Crep95LCL. These cases contain results for exposure point concentrations $C_{rep,mean}$, $C_{rep,upper}$, and $C_{rep,lower}$, respectively. Each of these cases was generated using the parameter values defined in Appendix Section B.3 (these are the HHRC default parameters). These three cases differ only in the exposure point concentrations used in the calculations. For the CrepMean case, the sample arithmetic mean concentration ($C_{rep,mean}$) was used. For the Crep,upper case, the 95th percentile upper confidence limit (UCL) of the mean concentration ($C_{rep,upper}$) was used. For the Crep95LCL case, the 95th percentile lower confidence limit (LCL) of the mean concentration ($C_{rep,lower}$) was used. (For more information on exposure point concentrations, see Appendix Section B.1). Each of these cases can be displayed using the HHRC program; however, only one of these cases can be installed at a time. Since these cases contain already-generated results, they enable user review of PPLVs and risks without spending the time required to run the model. Running the HHRC code to generate new results for all sites and all chemicals takes several hours. (Instructions for running the model to generate a new set of results is described in Section D.1.2.1.1.)

The following paragraphs outline the structure of the HHRC code. It consists of five modules that allow the user to alter input parameters, to generate the Latin Hypercube samples, to generate a new set of results (PPLVs and risks), and to access model results (either by file or on screen). Each time the user activates a new module, a case must be opened. For example, if the user is in the PPLV Results Module in the CrepMean case and wishes to move to the Additivity Module, the CrepMean case would need to be opened again in the Additivity Module before any information could be displayed. A list of the five modules with a brief description of their functions is shown below:

- Uncertainty Module: Allows user to view probabilistic input parameters for existing cases or to edit probabilistic parameters, to generate new Latin Hypercube sampling results, and/or to calculate new PPLVs and risks for new cases.

- Fixed Parameter Module: Allows the user to view deterministic parameters for existing cases and to edit deterministic parameters for new cases.

- PPLV Results Module: Allows the user to view carcinogenic and noncarcinogenic direct and indirect single pathway preliminary pollutant limit values (SPPPLVs) and PPLVs on screen and/or to save them to file.

- Additivity Module: Allows the user to view risks, hazard indices (HIs), exposure indices (EIs), C_{rep} concentrations, and C_{max} concentrations on screen and/or to save them to file.

- Sensitivity Module: This module has been superseded by the S-Plus-based sensitivity analysis reported in Appendix Section B.5, and is no longer maintained.

Tables D.1-1a and D.1-1b contain a list of parameters and results the user might want to obtain (such as direct pathway PPLVs) and show where they can be accessed in the program. Some results reported in the IEA/RC need to be calculated outside the HHRC code using available data from the HHRC code. A list of these results and the calculations needed to obtain them are provided on Table D.1-1c. The sections that follow discuss the modules in more detail in the order in which they are executed in the program.

D.1.2.1.1 HHRC Uncertainty Module

The HHRC program uses several probabilistic parameters in calculating the PPLVs and risks. These include chemical-specific parameters such as soil-to-water partition coefficients (K_{oc}), population-specific exposure parameters (such as soil ingestion rates), and site-specific parameters (such as soil density). A complete list of the probabilistic parameters is provided in Appendix Section B.3. The model randomly selects values from these distributions using Latin Hypercube sampling. In turn, these values are used to calculate PPLVs and risks. The number of values selected from each distribution is called the Latin Hypercube sample size. For the IEA/RC, a sample size of 100 was used. Within the HHRC program, the Uncertainty Module controls the

probabilistic parameter distributions and generates the Latin Hypercube samples. This module can be used to accomplish the following:

- Change probabilistic input parameters from the default values described in Appendix Section B.3 when creating a new case
- Change the Latin Hypercube sampling parameters (such as the sample size) when creating a new case
- Begin model calculations for a new case
- View the probabilistic parameters or the LHS parameters from an existing case

The ability to change parameter distributions or Latin Hypercube sampling allows the user to compare new results (a new case) with those documented in the IEA/RC ($C_{rep,mean}$ case, $C_{rep,upper}$ case, and $C_{rep,lower}$ case) and see how these changes effect results. For more information on the Latin Hypercube sampling process and its use in PPLV and risk calculations, see Appendix E.

Creating a New Case

The steps necessary to create a new case and to generate a new set of results are listed below:

- Activate the Uncertainty Module.
- Under the File Menu, create a new case. Type the new case name in the designated box. At this point, the user has the option to copy the random seed (this determines the pseudorandom order in which parameter values will be selected from their respective distributions for the Latin Hypercube sampling result) from an existing case. For a valid comparison between HHRC cases, the random seed should be copied when defining a new case.
- Go to the Latin Hypercube Sample (LHS) Menu and change parameter distributions, if necessary.
- Go to the Fixed Parameters Module and edit fixed parameter values, if necessary (explained in Section D.1.2.1.2).
- Go back to the Uncertainty Module, go to the LHS Menu, and edit the sampling parameters. For a valid comparison with the IEA/RC cases, a sample size of 100 should be used. When editing Latin Hypercube sampling parameters, the user has several options from which to choose:

(a) The user can select the number of samples used to generate PPLV distributions (100 samples were used for the IEA/RC calculations).

(b) The user can choose between the following representative site concentrations: $C_{rep,mean}$, $C_{rep,upper}$, or $C_{rep,lower}$ case. This allows the user to choose which value of C_{rep} the model uses in the PPLV and risk calculations. (For more information on C_{rep}, see Appendix Section B.1.4.)

(c) The user can choose which cancer risk level to use in the PPLV and risk calculations (i.e., 10^{-4}, 10^{-5}, or 10^{-6}). A 10^{-6} cancer risk level was used in the IEA/RC cases. (For more information on cancer risk levels, see Appendix Section B.1.8.3.)

(d) The user can choose which percentile will be selected from the resulting PPLV distributions to be the final PPLV value. The 5th percentile was used in the IEA/RC cases. (For more information on the uncertainty in PPLV calculations, see Appendix E.)

- Go to the LHS Menu and generate the Latin Hypercube sampling result.
- Go to the Analyze Menu and activate the Uncertainty Analysis command, which begins model calculations. The user can specify which sites and/or chemicals to analyze. Note that running the HHRC code for all sites and all chemicals will take several hours.

Once the Latin Hypercube sampling results have been generated, a new Latin Hypercube sample can be generated and parameter values changed only if a new case is created. This prevents the new data from accidentally overwriting the old. After the Latin Hypercube sampling result has been generated, the user can display histograms from the LHS Menu. These histograms compare the original parameter distributions with the data generated through Latin Hypercube sampling. This shows the user how closely the Latin Hypercube sampling result represents the overall sample population.

Parameter distributions can be compared to the reasonable maximum exposure (RME) values presented in the Human Health Exposure Assessment report (HHEA) (EBASCO 1990) by consulting the Help Menu. (The path Help\Index\Search\Parameter Tables\RME Values Table leads the user to a table of HHEA RME values; this table can then be printed and used for comparison.)

D.1.2.1.2 HHRC Fixed Parameter Module

Several fixed parameters (i.e., those not the subject of distribution development) are also used by the program to calculate risks. These fixed parameters are listed in Appendix Section B.3. The HHRC Fixed Parameter Module allows the user to edit all fixed parameters for new cases (as discussed in the previous section) and to view all fixed parameters for existing cases. In this module, toxicity parameter values (DT) for carcinogens are automatically adjusted to correspond to the cancer risk level designated in the HHRC Uncertainty Module.

Fixed parameters, like distributed parameters, may only be edited for a new case, and must be edited before PPLV and risk calculations are initiated. If changes are not made to the fixed parameters, default parameter values are used. These are detailed in Appendix Section B.3. As with distributed parameters, edited fixed parameters can be compared to RME values by consulting the Help Menu.

Once fixed parameters are edited, the user must go back to the HHRC Uncertainty Module and initiate the calculation of PPLVs by activating the Uncertainty Analysis command under the Analyze Menu (see "Creating a New Case" from the previous section). It should be noted that once the Uncertainty Analysis command has been activated, parameters can no longer be edited without creating a new case.

D.1.2.1.3 HHRC PPLV Results Module

Within this module, direct PPLVs, indirect PPLVs, and cumulative PPLVs (which contain both direct and indirect exposure pathways) are displayed by activating the Analyze Sites command under the Display Menu. PPLVs must be displayed separately for each region at RMA (including the Surficial Soil samples, which are organized as a "region" in the HHRC code). PPLVs can be displayed in four ways. First, the direct, indirect, and cumulative PPLVs for each chemical can be displayed in tables that may be printed for ease of reference. Second, the direct-pathway SPPPLVs can be displayed on screen. Third, the cumulative PPLV distributions for each chemical can be displayed on screen as cumulative distribution functions (CDFs). Fourth,

the cumulative PPLV distributions for each chemical can be displayed as histograms (also known as discrete probability density functions, or discrete PDFs).

Each value displayed on a PPLV table represents the percentile selected by the HHRC from the corresponding CDF curve. The user chooses the desired percentile for a new case in the Uncertainty Module; it cannot be altered for an existing case. For the CrepMean, Crep95UCL, and Crep95LCL cases, an uncertainty percentile of 5 percent was used, so the PPLV values displayed on the tables are the 6th lowest out of 100 calculated values (i.e., they are the values that exceed 5 percent of the values in the distribution). PPLV tables also contain the corresponding EI values, which are discussed in Appendix Section B.1. PPLV tables are classified by site, exposed population, and soil horizon. It should be noted that only cumulative PPLVs from Horizon 1 and Horizon 2 may be viewed graphically. Tabulated PPLV values may be carcinogenic or noncarcinogenic, depending upon the risk posed by the particular chemical. If the chemical poses both a carcinogenic risk and a noncarcinogenic health threat, only the carcinogenic PPLV is shown. Noncarcinogenic PPLVs for chemicals having both carcinogenic and noncarcinogenic effects must either be accessed from output files or calculated outside the HHRC code from Horizon 0 C_{rep} and HI data available in the Additivity Module as shown in Tables D.1-1b and D.1-1c.

RME direct PPLVs used in the HHEA (EBASCO 1990) can be compared with the risk characterization cumulative direct PPLV in this module by accessing the Help Menu (see Section D.1.2.1.1). Cumulative PPLVs can also be compared with the HHEA's cumulative PPLVs for site NCSA-3 (Basin F).

The linearity check (flux ratios) of the SPPPLV vapor equations (described in Appendix Section E.7.3) can also be accessed by the HHRC PPLV Results Module. The flux ratios are not available for on-screen display. However, they can be saved as ASCII text files (see Section D.1.2.1.6) for display outside the HHRC code, using commercially available spreadsheet, statistical, and graphics software packages.

D.1.2.1.4 HHRC Additivity Module

Cancer and noncancer risks from exposure to multiple chemicals (additive risks) are displayed in this module (under the Analyze Menu) for the chemicals present at each RMA site. Results are displayed in tables as cancer risks for carcinogens, and as chemical-specific hazard quotients (HQs) and additive HIs for noncarcinogens. The tables are classified by site, exposed population, soil horizon, type of health effect (carcinogen or noncarcinogen), and risk type (incremental or total), where total risk includes natural background risk (for metals), and incremental does not. Total and incremental risk are discussed in Section 3.2.2. A separate table exists for each combination (e.g., site C1A, biological worker, Horizon 1, carcinogen, incremental risk, etc.). The tables include the following information:

- A list of the chemical-specific risk (or HQ) for each chemical at a particular site, for both maximum and representative site concentrations (C_{max} and C_{rep}), and for both total and incremental risks (or HIs)
- Additive HIs for both maximum and representative site concentrations
- EIs corresponding to all HQs and HIs
- A list of the chemicals whose HQs contribute most to the HI at a particular site and their corresponding contributing percentages
- A residual additive site HI, which represents the HI that remains once contributing chemicals have been removed

The HHRC Additivity Module also ranks all RMA sites or specific site borings in order of risk, from highest to lowest estimated risk. The menu options provided enable selection among four ranking lists for sites or borings: total carcinogen, total noncarcinogen, incremental carcinogen, or incremental noncarcinogen. For more information on risk calculations, see Appendix Section B.1.8.

D.1.2.1.5 HHRC Sensitivity Module

The HHRC Sensitivity Module has been superseded by the S-Plus-based sensitivity analysis reported in Appendix Section B.5, and is no longer maintained. Within the HHRC Sensitivity Module, chemical-specific PPLV distributions from different user-specified cases were originally

displayed and compared graphically as PDFs or CDFs. The HHRC Sensitivity Module was used in the early stages of the IEA/RC to determine acceptable uncertainty and error factors about the PPLV distributions, and to determine how sensitive the results were to a revision of one or more of the parameters. This allowed the user to compare effects of user-specified changes to input parameters in a trial-and-error fashion. As noted in Appendix Section B.5, sensitivity of the results to variation or uncertainty in specific input parameters is better characterized using standard regression coefficients and partial correlation coefficients. Accordingly, their use has replaced this relatively simple (and potentially misleading) approach.

D.1.2.1.6 HHRC Output Data Summary Files

PPLVs and risks generated by the HHRC code as well as the input parameters used in their calculation can be saved to file and are referred to as HHRC output data summary files. These files enable the user to share custom cases with other users. These files also allow users to share HHRC model results electronically with others whose computer disk space is too limited to hold the large results files associated with the HHRC code (the largest such file occupies 17 MB of disk space). Finally, the data summary files provide electronic access to HHRC model results for use in quality assurance/quality control (QA/QC) procedures.

The HHRC Uncertainty, PPLV Results, and Additivity Modules also have menu tools that allow the user to save results or input parameters to file. It is important to note that not all of the data available on screen can to be saved to electronic files, and not all of the data saved to electronic files can be viewed on screen.

The following discussion describes the parameters and/or results that can be saved to electronic files from each module. All files are automatically saved to the c:\TMP directory unless directed otherwise. It is necessary to create the c:\TMP directory (or other desired computer directory) outside the HHRC code prior to using this feature, however.

Uncertainty

In the HHRC Uncertainty Module, variable and fixed parameters can be saved to electronic files under the LHS Menu. The user is able to select the report type (summary listing or complete listing) and the file format (fixed space or tab delimited). These files, saved as ASCII text files with a user-specified file name, can also be saved to any existing computer directory. (The default directory is c:\TMP.)

PPLV Results

Within the HHRC PPLV Results Module, indirect PPLVs and the corresponding linearity check flux ratios can also be saved to electronic files. The user can specify which indirect PPLVs (and ratios) are to be saved by designating an exposed population, a health effect type (carcinogen or noncarcinogen), and an exposure point concentration (C_{rep} or C_{max}). The files are saved as ASCII text files to any existing directory the user specifies. (Again, the default directory drive is c:\TMP.) The default file name, which can be modified by the user, refers to the particular set of PPLVs saved, using the file-naming system shown below.

File Name = "HPPLV" + (exposed population) + (health effect type) + (exposure point concentration) + ".TXT"

where:

Exposed Population	Health Effect	Exposure Point Concentration
B = Biological worker	C = Carcinogen	M = C_{max}
C = Commercial worker	H = Noncarcinogen	R = C_{rep}
I = Industrial worker		
F = Recreational visitor		
R = Regulated/casual visitor		

Two examples of default file names are the following:

- HPPLV**BCM**.TXT (the default name for biological worker/carcinogen/C_{max})
- HPPLV**BNR**.TXT (the default name for industrial worker/non-carcinogen/C_{rep})

Additivity

Additive cancer and noncancer risks and exposure point concentrations can be saved to electronic files under the Analyze Menu within the HHRC Additivity Module. The user can specify whether site risks or boring risks are to be saved, or whether risks or concentrations are to be saved. If the risk summary is selected, the user can further specify the risks by designating the exposed population, depth horizon, risk calculation type, and health effect type. If exposure point concentrations are selected (C_{rep} or C_{max}), concentrations are further specified by designating the depth horizon and the risk calculation type. These files are saved as ASCII text files to any existing directory the user specifies. (The default directory is c:\TMP.) The default file name refers to the specific set of risks or exposure point concentrations that was saved, using the file-naming system shown below.

Filename = "H" + (summary type) + (site type) + (risk type) + (variable percentile) + (chemical) + (health effect type) + (soil horizon) + "." + (exposed population) + (report type) + (data summary type)

where:

Summary Type	Site Type	Risk Type (not used for C_{rep} or C_{max} concentrations)	Variable Percentile (not used for C_{rep} or C_{max} concentrations)
S = site	SR = Study Area Report (SAR)	T = Total	5 = 5th
B = boring	SS = Surficial soils	I = Incremental	M = 50th

Health effect type			Depth Horizon			Exposed Population		
C	=	Carcinogen	0	=	Horizon 0	B	=	Biological worker
H	=	Noncarcinogen	2	=	Horizon 2	C	=	Commercial worker
@	=	Concentration	1	=	Horizon 1	I	=	Industrial worker
			S	=	Surficial soils	F	=	Recreational visitor
						R	=	Regulated/casual visitor
						@	=	Concentration

Chemical (not used for risk summaries)

_0 = Aldrin	14 = Endrin
_1 = Benzene	16 = Hexachlorocyclopentadiene*
_3 = Chlordane	17 = Isodrin
_4 = Chloroacetic Acid	18 = Methylene Chloride
_5 = Chlorobezene	19 = 1,1,2,2-Tetrachloroethane
_6 = Chloroform	20 = Tetrachloroethylene
_7 = Dichlorodiphenyldichloroethene (DDE)	21 = Toluene
_8 = Dichlorodiphenyltrichloroethane (DDT)	22 = Trichloroethylene
_9 = Dibromochloropropane	23 = Arsenic
10 = 1,2-Dichloroethane	24 = Cadmium
11 = 1,1-Dichloroethylene	25 = Chromium
12 = Dicyclopentadiene	26 = Lead
13 = Dieldrin	27 = Mercury

*Fluoroacetic Acid (number 15) is no longer an HHRC chemical of concern, as discussed in Appendix Section B.1.2.

Report Type			Data Summary Type		
R	=	Rank	M	=	C_{max}
M	=	Map	R	=	C_{rep}
D	=	Data summary	K	=	Risk

Two examples of default file name are the following:

- HSSRI5H1.RDK (the default name for HHRC, site, Study Area Report (SAR) site, incremental, 5th variable percentile, noncarcinogen, horizon 1, regulated/casual visitor, data summary, risk)

- HSSR_0@0.@DR (the default file name for HHRC, site, SAR site, aldrin, horizon 0, data summary, C_{rep}).

It is important to note that the program does not distinguish between different cases run with differences in C_{rep} (e.g., using $C_{rep,mean}$ or its 95 percent confidence limits or differences in exposure parameters) when it creates these default file names. To ensure that corresponding results from different cases are not overwritten, cases resulting from a change in parameters or C_{rep} must be saved in different directories.

D.1.3 ECOLOGICAL RISK CHARACTERIZATION MODEL IMPLEMENTATION

The ERC model was originally developed to determine multimedia probabilistic biota criteria (PBC) and health threats from bioaccumulated contaminants for four key upper trophic level predators (bald eagle, kestrel, great horned owl, and great blue heron) and one other key target species group (shorebird). The ERC computer code was created to implement this model in Microsoft Windows, Version 3.1 and provide easy user interaction. It consisted of two codes executable in Microsoft Windows: a preprocessor called ERC SampleCalc (BSC.EXE), which calculated maximum and representative site concentrations (C_{max} and C_{rep}), and an interactive risk-calculation code (BIO.EXE).

An improved method for calculating ecological risks has since been developed, however, that involves analysis of the home ranges of biota in the bioaccumulation model. This home-range analysis can only be implemented using UNIX-based geographic information system (GIS) software. Accordingly, the Windows-based ERC computer code has been retired. The new ecological health threat calculation method, which uses the GIS-based home-range analysis and probabilistic bioaccumulation models implemented using @RISK simulation software and Microsoft Excel spreadsheets, is described in Sections D.1.3.1 through D.1.3.3.

D.1.3.1 Excel/@RISK - Based Biomagnification Spreadsheets

Food web models for the bioaccumulative COCs are coded in Microsoft Excel, Version 4.0 for the Macintosh and Microsoft Excel, Version 4.0 for Windows. Palisade Corporation's Excel add-in, @RISK Version 1.1, was used to code probabilistic model parameters and to perform Monte Carlo simulations. Monte Carlo simulations of up to 500 iterations were performed on several platforms, each of which met or exceeded the specifications of a Macintosh IIci with 4MB RAM and a math co-processor, or a 486P/50-based IBM compatible microcomputer. It should be noted that minor memory limitations were encountered on the Macintosh IIci. For example, at certain times that user was required to close unnecessary windows and applications and delay printing requests. Simulations were performed using @RISK's Latin Hypercube sampling algorithm.

The terrestrial food web spreadsheet was used to compute BMFs for the top predators for which RMA tissue concentration data were unavailable (kestrel, owl, heron, and eagle). It utilizes field BMFs generated by each of the three approaches, consensus BAFs and prey fractions. Linkages among the terrestrial spreadsheets are illustrated in Figure D.1-1. A printout of the terrestrial food web model spreadsheets (Figures D.1-2 through D.1-6) is provided below.

The aquatic model consists of 13 linked spreadsheets. This model is used to estimate population mean tissue concentrations, doses, and risks to the water bird, shorebird, great blue heron, and eagle trophic boxes from exposure to bioaccumulative COCs through the aquatic portion of the RMA food web. The spreadsheets are also used to estimate prey population mean tissue concentrations in cases for which field data were unavailable; to partition the total exposure of shorebird between the terrestrial and aquatic portions of its diet; and to compute RMA-wide averages of lake-specific exposure estimates. A summary of the spreadsheets used in aquatic spreadsheet model, showing how the individual spreadsheets ar linked together, is provided in Figure D.1-7. The individual spreadsheets are presented in Figures D.1-8 through D.1-20.

D.1.3.2 S-Plus-Based Computation of BMF_{obs}

S-Plus (StatSci Inc. 1993) is a programming language designed for statistical analysis. S-Plus programs were used in the estimation of BMF_{obs} using the Army approach. The advantages of

S-Plus for this step were the following: S-Plus has built-in functions for statistical analysis, including regression and graphical display, which were useful or required in analyzing BMF_{obs}. In many cases, EXCEL did not have these functions or was not flexible enough to incorporate the modifications required for a given specialized task (for example, the simulation-based LSE approach). In addition, S-Plus allowed a given set of formulas and actions to be implemented automatically for each of the large number of trophic box/chemical combinations, increasing the efficiency of performing repetitive tasks.

D.1.3.3 Arc/Info Computation of Spatially Averaged Ecological Risk

The areal extent of risk was estimated by assuming that all animals at RMA would be exposed to the average soil concentration within a defined species-specific exposure zone. These average exposure concentrations were calculated by first interpolating sample soil concentrations onto a regularly spaced grid using Techbase (described in Section D.1.4.4) and then averaging all grid points within a given circular exposure zone using the Grid package of Arc/Info, a GIS.

The Grid package performs a running average over the gridded concentrations within a given region and outputs a new grid that has each point assigned the average exposure concentration within the exposure zone centered at that point.

The problem of the "edge effect," where estimation at a point near the edge requires more data than is available, is common to all running average analyses (i.e., statistical smoothing or filtering). This problem introduces uncertainty into the estimation of average exposure concentrations for exposure zones overlapping the RMA borders. In addition, a value of NE (not estimated) was assigned to grid points within lakes, buildings, and areas where the soil data were not adequate to produce an interpolated estimate, because of a predominance of BCRLs, or in some cases, low sample density. The areas of NE are indicated on the soil concentration maps in Section 4 and Appendix C. The approach used to handle NE values is described below. For convenience, the areas outside RMA are hereafter referred to as NE areas.

In general, it is reasonable to estimate ESC (and risk) for a given exposure range if the fraction of its area assigned soil concentration estimates (as opposed to NEs) is sufficiently large. In cases where this fraction is small, the ESC and HQ estimates are highly uncertain and therefore should be replaced with NE. The criterion to decide if uncertainty due to NEs is acceptable was the following: ESC estimates were acceptable if they were based on soil concentration estimates in one half or more of the exposure range, while ESC estimates based on concentration estimates in less than half the exposure range area were considered highly uncertain. It was infeasible to precisely implement this rule because of the variability in the sizes of NE patches for each chemical and exposure ranges and therefore the procedure given below was applied.

STEP 1

ESC and HQ estimates were calculated for grid points with exposure ranges that contained at least one concentration estimate. The NE data within a given exposure range were ignored so that the average reflected only the average of the estimated concentrations.

STEP 2

Highly uncertain ESC estimates were replaced with NEs by superimposing onto the ESC grid, areas of NE which occurred on the corresponding soil concentration map (i.e. ESC estimates were replaced with NE in areas where NEs dominated the soil concentration map). This replacement varied with the size of the exposure range as follows.

(1) Predators with large exposure ranges (kestrel, owl, and eagle).

The soil concentration grids have areas of NE occurring at the outer edges of the Arsenal (and extending inward to the source area for some chemicals). These "outer" NE areas were expected to be large enough in general so that points within these areas would receive an estimate of ESC based on concentration estimates from less than half the exposure range. Therefore these outer areas of NE on the soil concentration grid were superimposed onto the ESC grid (i.e. no estimate of ESC was made for these areas). In contrast, the size of "inner" NE patches occurring infrequently within the source areas was expected to be very small relative to the size of the predator exposure ranges. Therefore the ESC estimates within

these small inner NE patches were considered acceptable. No ESC estimates were calculated for exposure ranges centered within lake boundaries.

(2) All other trophic boxes.

The remaining trophic boxes have exposure ranges which are small relative to the areas of both outer and inner NEs in the chemical concentration maps. Therefore the calculation of ESC for a grid point within even the smallest NE areas would tend to involve data from less than one half the exposure range and would therefore be uncertain. Therefore both inner and outer NEs from the soil concentration maps were superimposed onto the ESC map. Again no ESC estimates were calculated for exposure ranges centered within lake boundaries.

STEP 3

HQs were calculated from the final ESC grids. An NE value on the ESC grid directly translated to an NE value for the HQ grid.

STEP 4

HIs were calculated as the sum of HQs for each grid point. HIs were assigned NE if all HQs contributing to the sum were NE. Otherwise NEs were ignored in the sum (i.e. treated as zero). Uncertainties resulting from this approach are discussed below.

Uncertainty is introduced when an "incomplete" HI is calculated from the sum of HQs, where some HQs are NE and therefore treated as zero. The incomplete HIs may or may not provide significant underestimates of the true HI, depending on the true concentrations in BCRL and no data areas. (Both BCRL and no data areas are expected to have relatively low concentrations. No data areas arise when a given chemical was not analyzed because its presence was thought to be highly unlikely). It was infeasible to distinguish between complete and incomplete HI estimates in the maps. Ideally incomplete HI's would be assessed by comparing to the action level of 1.0: If the incomplete HI > 1 then the true HI is certainly in exceedance; if the incomplete HI < 1, then the true HI may or may not be in exceedance and could be distinguished from a complete HI < 1 as having additional

uncertainty. This comparison is complex to display and interpret for a HI map based on a single BMF, especially if spatial averaging is rigorously considered. The comparison is prohibitively complex to display and interpret for the report HI maps which present risks based on three BMFs. Therefore such a comparison was not attempted. As stated above, the chemical specific areas of NE are identified on the soil concentration maps given in the report.

D.1.4 CHEMICAL SAMPLING DATABASE AND SampleCalc PROGRAM

This section provides information on the primary RMA sample database used in the HHRC and ERC as well as on the formation of the subset databases used in the HHRC SampleCalc preprocessor program and the GIS home-range analysis. The ERC SampleCalc program was replaced by the GIS home-range analysis method (see Sections D.1.3.1 through D.1.3.3), and so is not described in this section. This section also describes some similarities and differences between the human health and ecological subset databases.

D.1.4.1 Description of Primary Sample Database

The primary RMA sample database (RMA Environmental Database) contains site-specific that were collected and compiled from numerous sampling programs managed by various contractors to the Army. This database contains data from soil, sediment, surface water, groundwater, and biota samples collected at RMA or at off-post control locations. (Since groundwater calculations are no longer used in the HHRC or ERC, groundwater sampling data are not included in this report.)

Soil samples were taken from soil borings and from surficial soil sampling locations. The soil boring data were collected under the remedial investigation (RI)/feasibility study sampling programs conducted from 1985 to 1993, during which approximately 7,000 borings from depths varying from 1 to 60 ft were collected. Because several samples were often collected from a single boring, and because each sample was analyzed for a number of contaminants, the number of soil boring database records is very large, approximately 410,000. The surficial soils data

were collected as part of the Surficial Soils Sampling Program, which was conducted from 1989 to 1993. This program resulted in approximately 11,300 additional soil database records.

Sediment and surface water data were taken from several sampling programs. Those soil boring samples that were collected in the RMA lakes and North Bog under the above-mentioned programs were designated as sediment samples. Under the Comprehensive Monitoring Program (CMP), surface water and sediment samples were collected from the years 1988 to 1990, resulting in approximately 391 surface water and sediment samples. These samples account for 30,000 database records. Further, under various other sampling programs, sediment samples (109 records) and surface water samples (2,733 records) were added to the primary sample database.

Biota tissues were collected and analyzed under the Biota Remedial Investigation (RI) program from 1986 to 1988 (ESE 1989), under the Biota CMP from 1988 to 1990 (RLSA 1990), and under the ERC field program in 1989 and 1990. Numbers of samples collected under these programs include 494 samples from the Biota RI, 61 samples from the Rosenlund study (Rosenlund et al. 1986), 1,080 samples from the CMP, and 48 samples from the ERC field program. An additional 406 data records from soil and sediment samples were provided to the risk characterization by the ERC field program.

The RMA Environment Database is maintained by D.P. Associates (DPA 1991) and is accessed through the Rocky Mountain Arsenal Environmental Database (RMAED). The database is currently stored and maintained on a VAX system at TENTIME in Denver, Colorado. RMAED became available for use in 1989; however, ongoing data validation tasks limited the usefulness of the database in supporting the IEA/RC effort until late 1990.

D.1.4.2 Modification of the Primary Sample Database for the HHRC Program

This section describes the general preprocessing approach that was taken to modify the RMA database for use in the HHRC. This approach consisted of filtering and modifying of the RMA Environmental Database for use in the SampleCalc program and further modifying the data by

SampleCalc for use in the HHRC program. The entire preprocessing system is shown in Figure D.1-21.

D.1.4.2.1 Primary Database Modification

The RMA database modification process involved a series of steps, each of which generated an intermediate data file as output. These steps and files are described below and shown in Figure D.1-21.

The first step consisted of pulling the appropriate soil and sediment records from RMADMS into a fixed-format file using ASCII-standard characters. This file was then loaded into dBASE (Version III or IV) using the dBASE LOAD or APPEND command, which resulted in a file named HHRC.DBF. The HHRC.DBF file is a subset of the primary RMA Environmental Database and contains more than 400,000 soil and sediment records.

Four programs (written using Clipper Version S87) were then run in series to modify, append, and filter the HHRC.DBF dBASE file for use in the HHRC risk calculations. The Clipper programs are compiled files that execute dBASE commands. These applications could have been accomplished manually using the dBASE user interface, but the Clipper programs were written to enhance the traceability and reproducibility of the process. The four programs are described below.

First, the Clipper program XY_REPL.PRG was applied to the HHRC.DBF file to add study area site names and x-y coordinates to the database records. This program also performed a general cleanup of the file (eliminating extra fields, correcting field names, etc.). The resulting dBASE file was called ALLCSO.DBF.

Second, the Clipper program RCNWBOR.PRG was applied to ALLCSO.DBF to append new fields that were used to further classify the data records and to identify potential problems or inconsistencies in the data. This program generated a dBASE output file called rcnwbor.dbf.

Third, the Clipper program COCNWBOR.PRG was applied to the RCNWBOR.DBF database to filter out data that was not usable for the HHRC program. It filtered out the following records:

- Any record that did not represent an intermediate chemical (ERC) or a COC (HHRC)
- Any record that represented a contaminated rinsate blank
- Any record that did not represent a study area site sample and that did not have a location (i.e., an x-y coordinate)
- Any record that represented a Basin F sample taken prior to the remediation of the site (i.e., prior to January 1, 1989) and that was taken from the 0- to 12-inch soil depth interval

This filtering program generated a dBASE output file called COCNWBOR.DBF.

Fourth, the Clipper program HSOIL.PRG was applied to the COCNWBOR.DBF file to complete the filtering process and to convert the resulting database file to an ASCII file, hsoil.asc. This file filtered out the following records:

- Any record that did not represent one of the COCs (HHRC)
- Any record that represented a sediment sample
- Any record that did not represent a study area site boring

Once these four Clipper programs were run, the HSOIL.ASC file was imported into the risk characterization code along with numerous other import files, to create a risk characterization database in "RDM" format.

RDM (Version 3.2.1) is a database management system developed by The Raima Corporation. Unlike other commonly used databases (dBASE, RBASE, FoxPro), RDM does not significantly degrade user-access speed when the database becomes large. RDM was selected because speed was an important consideration for the Microsoft Windows programs, and the IEA/RC database is large relative to the capabilities of microcomputers.

The second page of Figure D.1-21 lists the RDM database files (*.DBD and *.DBV) that this process created. These RDM files provide the input data and structure for the SampleCalc program, that is described in the following section, and the HHRC program. Table D.1-2 lists the fields contained in the database structure that are used as input data for the SampleCalc program.

D.1.4.2.2 SampleCalc Program

The SampleCalc program (SC.EXE) preprocesses the RDM-formatted database for use in the HHRC program. This section describes why the processing steps are required, and then describes the SampleCalc program's data filtering and value calculation tasks.

The exposure models in the HHRC program require representative and maximum concentrations (C_{rep} and C_{max}, respectively) in order to calculate risk-based criteria (PPLVs), cancer risks (CRs), and HIs (see Appendix Section B.1 for PPLV and risk calculations). C_{rep} and C_{max} must be calculated for each soil horizon in each site at RMA. Moreover, indirect exposure models for open space and basement exposure in the HHRC require estimates of the upper and lower depth limits of contamination.

Extensive processing is required to generate this uncommonly large number of input values from the (even larger) RDM-formatted sample database. Since this processing takes a considerable amount of computer time, batch preprocessing of the RDM-formatted database is necessary to reduce the time the user spends running the HHRC program. The SampleCalc program was developed to make this possible. This program runs under Microsoft Windows, with the same hardware and software requirements as the HHRC and ERC programs.

The data analysis functions in the SampleCalc program include the calculation of C_{rep} and C_{max} as well as the determination of the vertical extent of contamination. Some of these functions are performed for both boring-specific and sitewide records; others are performed for sitewide records only. Figure D.1-22 shows the overall flow of the process used in the SampleCalc program.

Figure D.1-23 shows the process used to modify the contaminant concentrations and depths obtained from the database for composite borings before using them in SampleCalc computations. Surficial soil samples are exempt from this process, since they are grab samples from the top 2 inches of soil. For composite borings (data records with site type listed as "Comp") with depth listed as "0," the depth value was changed to "0.5" feet to reflect the average depth in the 0- to 1-foot depth interval. For those borings that are also hits, the concentration is doubled. This was necessary because many of the composite samples were created by mixing samples from the 0- to 1-foot depth interval and the 4- to 5-foot depth interval. Accordingly, there is the possibility that in some cases higher concentrations from the 0- to 1-foot depth interval could have been diluted by the addition of soil from the 4- to 5-foot depth interval. For example, if the soil from the 4- to 5-foot level were uncontaminated, the reported concentration for the composite sample would be half that of the original sample from the 0- to 1-foot depth interval. This is conservatively assumed to be the case for all composite samples. The reported concentrations are doubled for these samples to correct for this worst-case dilution factor. Concentration values were not changed for below certified reporting limit (BCRL) composite samples.

Multiple data records that refer to the same physical samples were filtered so that SampleCalc computations only used a single record per sample. This is because the presence of more than one data record for a sample would distort the C_{rep} calculation by artificially weighting multiple-record samples more heavily than single-record samples. The SampleCalc program recognizes data records as referring to the same sample when they have the same boring identification (ID) and the same depth. When some records referring to the same sample had different concentrations, the SampleCalc program selected the most representative record from among these multiple records. If at least one of the duplicate samples is a hit (i.e., limit = 0) or a greater than certified reporting limit (GCRL) sample (i.e., limit = 1), then the hit or GCRL sample with the largest concentration is used. The assumption that the largest hit is the best estimate of the true concentration for a multiple-record sample is a conservative one, but it was judged to be the most appropriate.

In cases where all of the records for the same sample are BCRL samples (i.e., limit = -1 or -2), then the sample with the smallest certified reporting limit (CRL) is used. The reason is that the BCRL record with the smallest certified reporting limit is a better estimate of the true sample concentration than the records with higher CRLs.

Once the composite and multiple-record samples have been modified and filtered, the SampleCalc programs sort and count the data records to obtain values needed in the calculation of C_{rep}, C_{max}, and vertical extent of contamination. These intermediate values include the number of samples (n_s) and number of hits (n_h) for each boring ID/contaminant/depth interval combination. Height and depth of the contaminated layer at each site are also determined. The methodology used to determine these values (the vertical extent of contamination) is shown in Figure D.1-24. The resulting values are then used in calculating C_{max} for surficial soil and for study area site borings for each depth horizon that has been defined in the analysis. These depth horizons are shown in the in-text table below (Section D.1.4.3). The SampleCalc program also calculates C_{rep}, C_{max}, and vertical extent of contamination values for SAR sites for each depth horizon that has been defined in the analysis. Three C_{rep} values are calculated for each site/contaminant/depth horizon combination: $C_{rep,mean}$, $C_{rep,upper}$, and $C_{rep,lower}$. C_{max} and C_{rep} calculations are described in Appendix Section B.1.

The resulting database generated by the SampleCalc program contains the calculated values of C_{rep}, C_{max} depth to the top and bottom of contamination for each site/contaminant/depth horizon combination, and values of C_{max} for each boring/contaminant/depth horizon combination.

D.1.4.3 Chemical Database for Human Health Risk

The chemical database used in the HHRC program was that created from the RMA primary sample database by the Clipper preprocessor and SampleCalc programs (as discussed in Sections D.1.4.2 and D.1.4.3). This database contains only chemical data relevant to the HHRC calculations. Selection criteria for relevant data are discussed in Section D.1.4.2. These data represent chemical concentrations and extents of contamination for all depth horizons of all SAR sites defined in the analysis. The soil horizons considered were as follows: Surficial Soil (0 to

2 inches), Horizon 0 (0 to 1 ft), Horizon 1 (0 to 10 ft), and Horizon 2 (10 ft to groundwater). The horizons and associated depth intervals are summarized in the table below. These concentration values and depth intervals were then used to calculate human health risks to the different populations from various exposure scenarios.

Depth By Exposure Pathway for HHRC

Horizon	Surficial Soil	Horizon 0	Horizon 1	Horizon 2
Depth Interval	0 to 2 inches	0 to 1 foot	0 to 10 feet	10 feet to groundwater
Exposure Pathway	Direct	Direct	Indirect	Indirect

D.1.4.4 Chemical Database for Ecological Risk

Media or chemical concentration data used for risk calculations are located in the RMA Environmental Database maintained by D.P. Associates (DPA 1991). Ecological risk was estimated for 14 COCs and 3 media: soil, sediment, and water.

The following table summarizes the media and depth interval information used to determine the soil concentrations for specific areas at RMA.

Depth Interval by Sampling Area and Soil Concentration			
Sampling Area	Designated Sites	Surficial Soils	Outside Designated Sites
Depth Interval Other Species	0 to 1 ft	0 to 2 inches	0 to 1 foot composite samples (0 to 1 ft, 4 to 5 ft)
Depth Interval Prairie Dogs	0 to 20 ft or 0 to GW[1]	0 to 2 inches	0 to 1 foot composite samples (0 to 1 ft, 4 to 5 ft)

1 The interval "0 to GW" was used for borings where groundwater was less than 20 feet beneath ground surface.

Depth Interval by Sampling Area and Sediment Concentration		
Sampling Area	Designated Sites	Outside Designated Sites
Depth Interval	0 to 1 ft	0 to 1 ft

Soil boring and surficial soil data concerning the biota COCs were downloaded in March 1993 from the existing RMA Environmental Database for use in estimating exposure concentrations. Several modifications were made to this data set before it was used. As part of the quality assurance evaluation of the soil data, extensive crosschecks were performed between locational coordinates and designated site assignments and boring identification numbers, and between surficial soil sample designations and sample depth values to ensure that locational data were complete. Soil sample data taken during the Basin F remediation were removed from the database. The data set was screened for replicate records so that any computations would include only a single record for each sample. Where replicate records were encountered, the sample with the largest concentration detected was retained in the database. If there were no hits among the

replicates, the sample with the lowest CRL was retained. Sample records in the database for composite borings were also modified prior to use in calculations. For records containing composite samples at a depth of 0, the depth value was changed to 0.5. The concentration was doubled for those samples that were hits, and the CRL was doubled for those samples that did not show detections. Surficial soil samples were not included in this process since they are grab samples taken from the top 2 inches of soil.

The resulting modified data set was the sample database used for soil concentration estimates of exposure soil concentrations. The sample database contained 174,121 records from 5,148 discrete sampling locations. Of these, 3,369 sampling locations (131,264 records) were within designated sites. The remaining 1,779 sampling locations (42,857 records), including 4,518 records from 314 surficial soil sample locations, were outside of designated sites. More than 85 percent (184,323 records) of this data set is BCRL.

The estimation of exposure area soil concentrations (ESC) from the modified soil database was a three-step process consisting of spatial interpolation of BCRL samples, modeled interpolation of soil concentrations into an RMA-wide grid, and spatial weighting of interpolated data using species home ranges.

First, spatial interpolation of BCRL values was used to estimate a value for each BCRL sample based on nearby hits using an inverse-distance squared algorithm. This algorithm computes an average inversely weighted by distance from each sample to the point being estimated. That is, a replacement value for a given BCRL was estimated as follows: $estimate_k = \Sigma xw$, where x is the value of the nearby hit and w is its weight. The first iteration estimation for each point was calculated on a chemical-specific basis using detected concentrations found within a specified search radius. Search radii used were 1,200 feet outside of designated sites (including Basin F Exterior and the wind dispersion area) and 400 feet for all other designated sites. A vertical search radius of 5 feet was used in all cases. Estimates for bores outside of designated sites utilized only data outside of designated sites in order to retain differences in soil concentration characteristics within and outside of designated sites. Other BCRL points were not factored into

the estimation and a maximum of six points was used. Calculated estimations were compared to CRLs to determine possible replacements after all estimations were completed. The estimations were used to replace BCRL points as follows: for calculated values less than CRL, BCRL was replaced with the calculated value; for calculated values greater than or equal to CRL, BCRL was replaced with CRL. No replacement was made for BCRL points where no concentration was estimated. Successive iterations continued this process, using all hits and replaced values to calculate a new estimation. Iterations after the second calculate new estimates only for BCRL points that have had previous estimations. Four iterations were completed using this method.

Second, the spatially interpolated data was modeled to produce an RMA-wide soil concentration profile. A 100- by 100-by 1-ft block grid was laid over the RMA boundary to represent the top 1 foot of soil. Concentrations were estimated at every block for each chemical, again using an inverse distance squared algorithm. Designated sites were grouped for modeling according to similarities in characteristics or location and assigned search radii as follows: outside designated sites, 1,200 feet; sites in South Plants, 800 feet; sites in the central and north-central areas, 750 feet; sites in the eastern, western, and southern areas and in North Plants, 400 feet; and Shell trenches and complex disposal trenches, 200 feet. Soil concentration estimations within each site group utilized data from the top 1 foot of borings and were constrained to borings located within that group.

Estimations for prairie dog analysis included a second soil model layer for the 1- to 20-foot interval. Concentrations were estimated for these blocks using the same algorithm and all data found below the 1-foot depth interval. After all blocks were modeled, each concentration was multiplied by the dietary fraction for that layer and these fractions were then summed to give a total soil concentration for the grid point. If there was no information to model a given block, that block was labeled as IN (insufficient information) in all cases.

Third, the modeled soil concentrations at grid points within species-specific exposure ranges for each trophic box were spatially weighted. (The development of the exposure ranges is explained in Appendix Section C.2.5.)

A single sediment exposure area concentration was calculated for each of the RMA lakes considered in the risk evaluations. For estimated sediment exposure area concentrations, sediment sample concentration data interpolated onto a grid by the process followed for soil data were used to compute a lakewide, area-averaged (i.e., arithmetic mean) concentration.

D.1.4.5 Comparison Between the Human Health Chemical Database and the Ecological Chemical Database

There are several differences in the HHRC and ERC databases, besides the actual depth intervals and the GIS-based implementation of home-range analysis for the ecological model. The ERC considers areas outside designated sites and defines different depth intervals for the C_{rep} and C_{max} concentration calculations. The HHRC considers designated sites only (with the exception of some surficial soil sample locations), and defines equivalent depth intervals regardless of C_{rep} and C_{max} calculations.

D.2 QA/QC PROCEDURES

Rigorous QA/QC procedures were performed for the HHRC code; however, because of the size of the HHRC code—more than 1,000 pages of source code—these QA/QC procedures were not intended to be exhaustive. The QA/QC process was broken into six parts, which are outlined below.

Logic Check

The logic check was conducted to verify that the individual functions of the HHRC code were being performed correctly, including, but not limited to, any logical branching in the HHRC code.

Parameter Check

All parameter data used in the HHRC code were checked against literature values to ensure correctness. QA/QC of input values and distributions was accomplished in a separate review process for each release version, each of which corresponded to the formal drafts of the IEA/RC. Parameter checks are discussed in more detail in Section D.2.1.

Calculations Check

Hand calculations were completed to check results from each of the ERC and HHRC module and the SampleCalc program. Hand calculations were divided into two areas for QA/QC: model results (such as PPLVs) and report results (such as the additivity site-by-site summary tables).

Calculation checks for data sets were based on checking 20 percent of the results from a given data set. If an error was found with the check, then 40 percent were checked. If an error was still found, then the entire set was checked for errors. The calculations check was an iterative process in which individual errors were investigated and resolved before continuing the checks. Once the error or problem was identified and corrected, the results were rechecked and the problem documented. Several iterations were often required for completion of the process. For a more detailed description of HHRC hand-calculation procedures, see Section D.2.2.

Revisions to the HHRC model equations in response to discussions with the parties produced several phases of QA/QC. Each time changes were implemented in the HHRC code (including installation of developmental upgrades to the Microsoft Windows programming environment), hand calculation checks were re-initiated for a selected number of chemicals and sites. QA/QC data sets were selected to be typical of RMA chemicals and site information and to represent exposure from all pathways (soil ingestion, soil inhalation, dermal absorption, open space vapor inhalation, and enclosed space vapor inhalation.

Code Check

The HHRC code was tested to determine and eliminate all obvious unrecoverable application errors. Unrecoverable application errors are those that lock up the computer and force the user

to shut off and restart the computer. They may also cause serious corruption of the database if they occur during database reading and writing routines. This testing was not exhaustive but followed the general rules mentioned above. Additional testing was completed using Microsoft Windows debug tools from the Microsoft Windows Software Development Kit, Version 3.1.

Although testing was completed for all sections of the models, priority was given to the portion of the code that performed HHRC model calculations. This included a thorough check of all PPLV equations. For a more detailed description of the checks performed to verify the code describing the equations, see Section D.2.5.

Data Summary Check

Once all calculations performed in the HHRC and ERC programs were verified, the data summary files were checked to make sure that model results were correctly saved to file. For a more detailed description of this procedure, see Section D.2.4.

Checks Outside the HHRC Code

A QA/QC check for the GIS-based home-range analysis was conducted to ensure that resulting values were correct. In addition, values calculated from the @RISK-based spreadsheet models of biomagnification were also compared to those calculated from the original ERC program (benchmark test). A description of the home-range analysis is found in Section D.2.7 and a description of the benchmark test is found in Section D.2.6.

D.2.1 PARAMETER CHECKS

Fixed parameter values specified in the parameter packets were compared to those listed in the Excel input files, that were then imported into the HHRC code. These included chemical dose-response values, age-specific breathing rates, age-specific skin surfaces areas, pulmonary retention factor, annual exposure frequency, lifetime exposure duration, daily exposure period, soil porosity, soil density, soil moisture content, soil temperature, and chemical-specific molecular weights. Several values found to be in error were revised immediately. An additional check was then made of the revised values for accuracy.

Fixed parameter values were then compared from the master parameter list to those imported into the computer code. Several input values were in error and were corrected immediately in the input decks. The revised values were then verified in the code. Additionally, the dose-response values were verified as compared to their referenced sources (i.e., Integrated Risk Information System [EPA 1992] or Health Effects Assessment Summary Tables [EPA 1991]).

Probabilistic parameters were checked to make sure that the correct values were added to the Excel input files. This included checks of distribution types and arguments. Probabilistic parameters from the Excel files were then compared with the corresponding values imported into the code to make sure these values were also correct. Each time a fixed or probabilistic parameter was changed, the QA/QC checks described above were performed again to ensure that the changes were implemented correctly.

D.2.2 HHRC HAND-CALCULATION SUMMARY

The HHRC hand calculation process was conducted in four stages due to constant changes in the code during the QA/QC process. Stage I, performed in August and September 1991, checked computer code and reports that were not undergoing modifications. Stage II was accomplished at a later date (December 1991 and January 1992) to provide the QA/QC for those portions of the code that were still under development during Stage I. Stage III was performed in July and August 1993 to check modifications to the code. Stages I and II were used as a reference for Stage III. Since revisions were made to the code, most of the checks performed in Stage I were repeated in Stage III. Stage IV, performed in January 1994, verified correct implementation of input parameters that had be modified. Since no revisions to the structure of the HHRC model code calculations had been made, calculation checks were not necessary during Stage IV.

D.2.2.1 SampleCalc Program (SC.EXE)

D.2.2.1.1 Stage I Checks of SampleCalc

The QA/QC on the SampleCalc program was performed as follows. Chemical-specific representative and maximum soil concentrations were computed for ten chemicals on two sites (CSA-1a and NCSA-1g) and compared with model results. Several discrepancies were identified

in the QA process and were corrected. In addition to the verification of C_{rep} and C_{max}, the depths of the contaminated layer were checked for accuracy. These values were computed as described in Volume VI-A of the HHEA report (EBASCO 1990). The depth to the top of the contaminated layer (d) and the depth to the bottom of the contaminated layer (h) were computed and checked as follows for the following chemicals: site CSA-1a, aldrin, arsenic, chloroform, dieldrin, and toluene, and site NCSA-1g, arsenic and dieldrin. The QA/QC checks verified that all values had been correctly calculated correctly by the HHRC code.

D.2.2.1.2 Stage II Checks of SampleCalc

Significant expansion of the RMA Environmental Database occurred between Stages I and II. SampleCalc was run again using the new database, and QA/QC of the SampleCalc database was repeated as described in Stage I. Discrepancies uncovered in the new data records were identified, investigated, and resolved.

D.2.2.1.3 Stage III Checks of SampleCalc

Following the modification of the C_{rep} calculations in response and discussions with the parties (the change of C_{rep} to $C_{rep,mean}$ and the addition of the cases calculated using 95 percent confidence limits on C_{rep}), additional QA/QC procedures were conducted. The three new C_{rep} statistics (arithmetic mean, 95th upper and 95th lower confidence limits) were calculated for site CSA-1a (at Horizon 0 for aldrin) using an Excel spreadsheet. These calculation results were then compared with the program results. No discrepancies were found. For more information on the computation of $C_{rep,mean}$, $C_{rep,upper}$ and $C_{rep,lower}$,by the SampleCalc program, see Appendix Section B.1.

D.2.2.1.4 Stage IV Checks of SampleCalc

No additional checks of the SampleCalc program were necessary during Stage IV.

D.2.2.2 HHRC Uncertainty Module

D.2.2.2.1 Stage I Uncertainty Model Checks

Five random samples were obtained from the Latin Hypercube sampling distribution. Each sample contained a set of the distributed generic, chemical-specific, and population-specific input parameters. Each of these values was compared to the original parameter distribution to ensure that the Latin Hypercube sampling parameter value was within the range of the specified distribution, and all probabilistic parameter values were found to be within the assigned distribution range. In addition, the direct exposure pathway parameter values for aldrin under the industrial worker exposure setting were compared to the RME and most likely estimate (MLE) parameter values specified in Volume VII of the HHEA report (EBASCO 1990). These parameters were found to fall within the range of the MLE and the RME values.

D.2.2.2.2 Stage II Uncertainty Module Checks

For each of the distributed parameters checked during Stage II, the distribution type and the corresponding distribution input values were compared from the master parameter list to those imported into the HHRC code. Several values in the Excel input spreadsheets were in error and were corrected immediately. The revised values were then checked in the code to make sure they had been imported correctly. This was done by viewing parameters on screen or printing parameters to file, and verifying that the values matched those in the Excel input spreadsheets.

D.2.2.2.3 Stage III Uncertainty Module Checks

The checks described for Stage I were repeated, since changes in the computation of indirect SPPPLVs had been made.

D.2.2.2.4 Stage IV Uncertainty Module Checks

The distribution arguments for the aldrin, dieldrin, and chlordane (dermal Relative Absorption, factor [RAF_{dermal}]) dermal parameters and for the biological worker dust loading parameter (CSS) parameter (CSS) were checked (as described in Stage III) and verified to be correct both in the Excel input parameter spreadsheets and in the HHRC code.

D.2.2.3 HHRC PPLV Results Module

D.2.2.3.1 Stage I PPLV Results Module Checks

Cumulative PPLVs were obtained for the same five random samples as in the HHRC Uncertainty Module (see Section D.2.2.2). Cumulative PPLVs were then computed for each of the five random samples for Horizon 1 and Horizon 2, for each of the five exposed populations (regulated/casual visitor, recreational visitor, commercial worker, industrial worker, and biological worker), and for the same seven chemicals as were listed in Section D.2.2.1. (At this time, Horizon 0 was not yet defined in the code.) All SPPPLV computations were performed on Lotus 1-2-3 spreadsheets. One spreadsheet was developed for each chemical/exposed population/site combination, resulting in a total of 35 spreadsheets. Direct and indirect SPPPLV values were calculated as described in Appendix Section B.1.7. Spreadsheet calculations were verified by hand for one chemical, aldrin, over all pathways (soil ingestion, soil inhalation, dermal contact with soils, open space vapor inhalation, and enclosed space vapor inhalation) within the regulated/casual visitor and commercial worker settings. Hand calculations were not performed for the remaining three exposed populations, since the PPLVs evaluated for each of these populations are computed in the same manner as those for the regulated/casual visitor or commercial worker (with the exception of input parameter value differences). All intermediate value calculations were displayed in the spreadsheets so that those cumulative PPLV values that did not match the values produced by the HHRC code could be checked.

The following errors were discovered during QA/QC of the HHRC PPLV Results Module. Each of these errors was corrected, so the resolution is also listed.

- Error—For carcinogens under the regulated/casual visitor and recreational visitor exposure settings, intermediate intakes were not summed and then averaged over an 18-year period, but summed and then averaged over a 70-year period.

  Resolution—nMaxAge was redefined as 18 years for carcinogens.

- Error—For the soil inhalation pathway under the regulated/casual visitor and recreational visitor exposure settings, parentheses were missing in the denominators of the three equations that compute the intermediate soil inhalation rate, resulting in the exposure parameters being multiplied instead of divided.

Resolution—Parentheses were added to the denominator of the three equations.

- Error—The dust loading factor was entered into the input file in units of micrograms per cubic meter ($\mu g/m^3$), but was used in the soil inhalation single pathway PPLV equation in units of milligrams per cubic meter (mg/m^3).

  Resolution—A conversion factor of 1×10^{-3} mg/μg was added to the equations.

- Error—For the enclosed space vapor inhalation pathway, basement parameters (length and width of basement and length and width of zone of influence) were entered in units of meters, whereas basement height was in units of centimeters (cm). These values were included in the computation of volumes and areas without conversion to similar units.

  Resolution—Basement dimension parameters were converted to similar units and calculated volumes and areas were converted to the units needed to be used in the time-averaged flux (FAVN) equations.

- Error—For the enclosed space vapor inhalation pathway (where $d' < 100$ cm and the time that compares flux (T_d) is greater than 0 and less than 2.21×10^9 seconds), FAVN(1) should be calculated with t set equal to $(2.21 \times 10^9 - T_d)$. The second place in the FAVN(1) where t appears was hardcoded to set t equal to 2.21×10^9 seconds.

  Resolution—The second place where t appeared in the FAVN(1) equation was revised to set t equal to $(2.21 \times 10^9 - T_d)$.

- Error—For the enclosed space vapor inhalation pathway, the FAVN(2) equations were missing a conversion factor of 8.64×10^{11} $(mg\text{-}cm^2/day)/(g\text{-}m^2/sec)$.

  Resolution—The conversion factor was added in all cases where FAVN(2) was calculated.

To ensure that the changes noted above were made correctly, a second QA/QC was performed for aldrin on the recreational visitor and industrial worker population exposure setting. The comparison of results to histograms and CDFs was not performed during Stage I.

D.2.2.3.2 Stage II PPLV Results Module Checks

Cumulative PPLVs and input parameter values were obtained from the Latin Hypercube sampling distribution for five random samples. These values were provided in 56 files (*.CSV). Cumulative PPLVs were then computed for each of these samples for aldrin at site CSA-1a for all five exposed populations and exposure pathways using Excel spreadsheets. These

computations were performed to account for revisions that were made in the code since Stage I. The revisions included adding surficial soils (0 to 2"), adding Horizon 0 (0 to 1 ft), and changing the time-dependent variables (days worked, exposure frequency, exposure time) from fixed variables to distributed variables. One spreadsheet was developed for each of the five exposed populations. All SPPPLVs were computed using the same methodologies described in Stage I. The SPPPLVs were then compared to the values provided in the RESULT.CSV file for verification. All carcinogenic and noncarcinogenic SPPPLVs were verified for those chemicals that are only carcinogens or are both carcinogenic and noncarcinogenic. For noncarcinogens only was the cumulative direct pathway PPLV for Horizon 0 able to be verified since the RESULT.CSV file showed zero values for all other entries. This discrepancy was corrected, and all values were then verified. All cumulative PPLVs and EIs were compared with the results spreadsheets and no discrepancies were found.

Following the QA/QC of numeric PPLV results, the visual PPLV representations were also checked. The cumulative PPLVs for Horizons 1 and 2 presented in the RESULT.CSV file were compared to the histograms and CDFs for each exposed population for site CSA-1a. It was determined that all random sample values were reported correctly on the histograms and CDFs; however, there were two errors discovered with the visual representations: (1) the y axis was labeled "% pop." on both figures, although it should have been labeled "fractional pop" according to the values labeled on the y axis, and (2) it appeared that the line for the last sample was not being drawn on the CDFs, which resulted in CDFs that did not finish at a y-axis value of 1.0. Revisions were made to the code to resolve each of these errors.

D.2.2.3.3 Stage III PPLV Results Module Checks

Cumulative PPLV calculations were checked (using the same procedures described in Stages I and II) for Horizon 0, Horizon 1, and Horizon 2 for the following sites:

- SPSA-10—Biological worker; chloroform
- NCSA-1a—Biological worker; methylene chloride
- CSA-1a—Industrial worker; aldrin

No errors were found during the test.

Changes to the code included the addition of a flux ratio calculation. The flux ratios were added to measure deviations from linearity in the relationship between soil concentration and flux in the vapor models. A description of the flux ratios and the reasoning behind their use is included in Appendix Section E.7.3. A description of the vapor model equations is included in Appendix Section B.1.7.2. Flux ratios were obtained from the PPLV output data files for the same sites listed above. Corresponding flux ratios were the calculated as described in Appendix Section E.7.3 and compared to the model values. All values were verified to be correct.

D.2.2.3.4 Stage IV PPLV Results Module Checks

No checks of the PPLV Results Module were needed in Stage IV because changes had not been made to the PPLV Results Module.

D.2.2.4 HHRC Additivity Module

D.2.2.4.1 Stage I Additivity Module Checks

The HHRC Additivity Module was checked for the following: (1) to verify that all chemicals that have both carcinogenic and noncarcinogenic effects have a noncarcinogenic HI; (2) to verify cancer risk and HI values; (3) to verify cumulative site cancer risk and HI calculations; and (4) to verify RMA-wide ranking of the sites.

It was found that all chemicals that are both carcinogenic and noncarcinogenic had a noncarcinogenic HI. Cancer risks and HIs were verified for most chemicals. At site CSA-1a under the recreational visitor exposure setting, however, it appeared that the reported HI for toluene was not the sum of Horizons 1 and 2 as it should be, but the value for Horizon 1 only. Although this anomaly was not found for any other chemical, it was, nevertheless, determined that the Horizon 1 value was driving the cumulative HI for most other chemicals. This discrepancy was investigated and correct values were verified in Stage II.

The cumulative site cancer risk and HI were verified as functioning correctly. It should be noted, however, that these values are difficult to duplicate exactly since the printout only displays results to one decimal place, and the code, which uses double precision, displays results about 17 decimal places. In addition, the portion of this module that subtracts the contribution of the driver contaminants to the cumulative cancer risk or HI and computes the residual risk or HI that would exist if those driver contaminants were removed was also verified as functioning correctly.

The site priority designation and RMA-wide ranking of sites were not checked for accuracy during Stage I.

D.2.2.4.2 Stage II Additivity Module Checks

The HHRC Additivity Module was checked in Stage II using a method similar to that used in Stage I of the QA/QC process. Only the industrial worker exposure setting was checked for accuracy, however, since QA/QC of this population would cover all possible combinations (soil ingestion, soil inhalation, dermal absorption, open space vapor inhalation, and enclosed space vapor inhalation) of additive risks. Changes to the code since Stage I included the addition of Horizon 0 as well as surficial soils. The HHRC Additivity Module was therefore revised to provide a report for Horizon 0, Horizons 1 and 2 combined, and surficial soils, which is done on a boring-by-boring basis.

For carcinogens, risk values were compared directly from the results tables to the additivity tables, where the risk is equal to the EI multiplied by the 1×10^{-6} cancer risk level. All values were verified. For the noncarcinogens, the HIs were calculated as the chemical concentration divided by the PPLV. These values, which were obtained from those provided in the RESULT.CSV and SAMPCALC.CSV files, were verified. The cumulative site HIs and cancer risks were also verified, as were the contributing contaminants. Finally, the computation of residual risk was verified, although it should be noted that the residual HI was found to be in error. On the noncarcinogen additivity sheets, it appeared that the carcinogenic residual risk was being duplicated, instead of the residual HI. This necessitated a minor revision to the code, after which the residual HI was verified. Additional QA/QC steps included verifying the transfer of

all additivity table footnotes from the SC_BITS.XLS file and verifying all site priority designations and RMA-wide site rankings.

D.2.2.4.3 Stage III Additivity Module Checks

Changes to the code since the Stage II included the separation of Horizon 1 and Horizon 2 additive risks and HIs. The calculation of total risk for metals was also added to the code. (For more information on total vs. incremental risk, see Section 3.2.2). QA/QC checks were performed in Stage III to verify that these changes were correct.

Additivity tables, site ranking reports, boring ranking reports, and the database results file RESULT.CSV were generated for the biological worker, site CSA-1a and boring 3173@. Boring 3173@ was originally reported as several separate single-depth-interval "borings 3173@ and several others" with boring ID ending in A, B, etc. These were consolidated into a single boring ID, replacing the letter with the @sign. Risks, EIs, and HQs were checked to make sure that Horizon 1 and Horizon 2 results were reported separately and that these values were correct. This QA/QC process followed the same procedures described in Stage II. All values were verified to be correct.

Total and incremental risks and HIs (for metals) were also checked for the same site and boring listed above. Values reported on the additivity tables, ranking reports, and RESULT.CSV were checked against Excel spreadsheet calculations. Risks and HIs were calculated using the additivity equations described in Appendix Section B.1.7, and the incremental risk equation described in Section 3.2.2. All values were verified to be correct.

D.2.2.4.4 Stage IV Additivity Module Checks

No checks of the additivity module were necessary during Stage IV because the Additivity Module had not been changed since Stage III.

D.2.2.5 HHRC Sensitivity Module

D.2.2.5.1 Stage I Sensitivity Module Checks

The Sensitivity Module was still under development during Stage I, so QA/QC checks were not required during this stage.

D.2.2.5.2 Stage II Sensitivity Module Checks

The HHRC Sensitivity Module was reviewed first to determine whether the plotting of histograms and CDFs was functioning properly, and then to verify the computation of PPLVs for risk levels of 1×10^{-4} and 1×10^{-5}. Histograms and CDFs were checked by comparing the cumulative Horizons 1 and 2 representative PPLVs for each random sample of a 5-sample run to the plots. A second 10-sample run was also included on the figures in order to determine whether the scaling was working properly. While reviewing the histograms and CDFs associated with the sensitivity module, the following errors were detected:

- The last sample was not being drawn on the plot for the CDFs.
- The histogram graph routines were not scaling the "% pop." properly. The highest value reported on the y axis was typically one less than the value that should be labelled (e.g., a value of 59 was reported when it should have been 60).
- The histogram graph legends were found to be inconsistent. Sometimes the legend reported the fifth percentile cumulative PPLV and other times it reported the wrong percentile.

These problems were satisfactorily corrected in the code. Once the plots were functioning properly, three 5-sample cases were run for aldrin on site CSA-1a and dieldrin on site CSA-2a. The three cases consisted of runs that demonstrated the code's ability to compute PPLVs for the 1×10^{-4}, 1×10^{-5}, and 1×10^{-6} risk levels. These were achieved by revising the dose-response values for each chemical to reflect the value at the risk level of interest. PPLVs were found to be computed correctly, and the three cases for each chemical were plotted on single histograms and CDFs to accurately depict the results.

D.2.2.5.3 Stages III and IV Checks of the Sensitivity Module

The HHRC Sensitivity Module was replaced with the sensitivity analysis discussed in Appendix Section B.5 and is no longer maintained. For this reason checks of the Sensitivity Module were not necessary during Stages III and IV.

D.2.3 ERC HAND-CALCULATIONS SUMMARY

ERC hand calculations for QA/QC of the original ERC computer code were performed in October through December 1991. The original QA/QC procedures were used as a reference for additional checks that were performed during development of the ERC model in response to discussions with the parties. As noted in Section D.1.1, the original ERC code has since replaced by a more effective ERC model implemented using @RISK spreadsheets and home-rage analysis using UNIX-based GIS software.

D.2.4 OUTPUT DATA SUMMARY FILES

Once HHRC code calculations were verified to be correct, QA/QC procedures were performed to verify that these values were saved correctly to file. This was done by comparing the values in the output files to those shown on screen, and making sure that they matched. Values were also checked by accessing model results stored in the RDM-formatted (RESULT.CSV) and comparing these values to corresponding values found in the output files. This comparison was performed using Excel spreadsheets containing a cell-matching formula. HHRC output data summary files for the PPLV Results and Additivity Modules were checked. All files were verified to contain correct data.

D.2.5 CODE EQUATIONS CHECK

All HHRC PPLV equations and all ERC food-web equations were checked for accuracy in the HHRC and ERC codes, respectively. This check was initiated after all revisions had been made to the codes. Any errors found were corrected immediately. Attachment D-1 contains the equations that were used in this verification.

D.2.6 ERC CODE/ @RISK SPREADSHEET BENCHMARK TEST

BMFs computed using the @RISK-based spreadsheet model of the food web at RMA were compared with BMFs computed using the same input parameters in the ERC code. The purpose of this "benchmark" comparison test was to demonstrate equivalence between the @RISK and ERC code implementations of the food-web model before retiring the ERC code and replacing it with the @RISK ERC spreadsheets. Arithmetic means of terrestrial BMFs computed from 100-sample runs using literature-based BAFs were compared for each of the five top predators. Similarly, aquatic BMFs from 100-sample runs were compared for each of the three top predators (bald eagle, great blue heron, shorebird) that have aquatic prey.

D.2.7 QA/QC OF GIS-BASED HOME-RANGE ANALYSIS

The ArcInfor Grid package was used to estimate the ESC associated with each organism, and to produce the HQ, HI, and predicted TC maps that were used in the risk and uncertainty analyses. The main task involved in these analyses (exposure range averaging) was performed using a built-in ArcInfo Grid function. ArcInfo programs were constructed to calculate exposure area average soil concentrations (ESC), match associated input files (e.g., BMFs, PBCs, exposure range radii) with the soil concentration grids, and to perform other tasks such as special handling of NE grid points and summing averaged HQs to produce HIs. The input files were verified independently by two individuals. The algorithms for specific tasks were verified by performing hand calculations on numerous randomly selected points.

D.3 REFERENCES

Ambrose, R.B., R.A. Wool, J.P. Connolly and R.W. Scharz. 1988. WASP4. A Hydrodynamic and Water Quality Model. Model Theory User's Manual and Programmer's Guide. EPA/600/3-87/039. Environmental Research Laboratory.

Becker, et al. 1988. The New S Language: A Programming Environment for Data Analysis and Graphics. R.A. Becker, J.M. Chambers and A.R. Wilks, AT&T Bell Laboratories. Wadsworth & Brooks/Cole Computer Science Series, Pacific Grove, CA.

DPA (D.P. Associates). 1991. RMAEDB Database.

EBASCO. 1990. Human Health Exposure Assessment for Rocky Mountain Arsenal. Final,Version 4.1.

EPA (U.S. Environmental Protection Agency). 1991. Health Effects Assessment Summary Tables. (HEAST) Office of Health and Environmental Assessment.

EPA. 1992. Integrated Risk Information System (IRIS). Online Database. Office of Health and Environmental Assessment.

RTIC 89173R02
ESE (Environmental Science and Engineering). 1989. Biota Remedial Investigation Report. Final, Version 3.2.

Microsoft Corporation. 1992. Guide to Programming Microsoft® Windows™ Version 3.1 Software Development Kit.

Nantucket Corporation. 1990. Clipper® 5.0 Programming and Utilities, and Clipper® 5.0 Reference.

Raima Corporation. 1991. Raima Data Manager™ User's Guide. First Edition, Raima Data Manager Version 3.2.1.

RTIC 86041R02
Rosenlund, B., D. Jannings, W. Kuray, T. Jackson, 2nd, E. Bergason. 1986. Contaminants in the Aquatic Systems at the Rocky Mountain Arsenal. USFWS, Denver, CO.

RTIC 90280R02
RLSA (R.L. Stollar & Associates). 1990. Comprehensive Monitoring Program. Final Biota Annual Report for 1989, Version 2.0.

Statistical Sciences, Inc. 1993. S-PLUS for Windows User's Manual, Version 3.1. Statistical Sciences, Inc., Seattle, WA, March 1993.

Table D.1-1a HHRC Data Available for Screen Display**

Result	Module	Menu	Tool Pathway
Probabilistic Input Parameters	Uncertainty	LHS	View Distribution List
Deterministic (Fixed) Input Parameters	Fixed Parameters	View	Fixed Values
LHS Sampling Parameters	Uncertainty	LHS	View Sampling Parameters
LHS Sample Histograms	Uncertainty	Display	LHS Histograms
Cumulative PPLVs* (includes all pathways)	PPLV Results	Display	Analyze Sites
Cumulative PPLV* Histograms	PPLV Results	Display	Analyze Sites/Pick Chemical/PPLV Histogram
Cumulative PPLV* CDFs	PPLV Results	Display	Analyze Sites/Pick Chemical/PPLV CDF
Direct Pathway PPLVs*	PPLV Results	Display	Analyze Sites (PPLVs for Horizon 0 are direct pathway only)
Indirect Pathway PPLVs*	PPLV Results	Display	Analyze Sites*** (shown for Horizon 1 and Horizon 2)
Direct Pathway SPPPLVs*	PPLV Results	Display	Analyze Sites/Pick Chemical/Direct Pathway SPPPLVs
Site C_{rep} Concentrations	PPLV Results	Display	Analyze Sites/Pick Chemical/ Concentration Values
Site C_{max} Concentrations	PPLV Results	Display	Analyze Sites/Pick Chemical/ Concentration Values
Site Cancer Risks	Additivity	Analyze Display	Site Additivity Report Carcinogenic Additivity
Site Noncancer Health Threats (Hazard Indices)	Additivity	Analyze Display	Site Additivity Report Noncarcinogenic Additivity
Sites ranked in order of decreasing Cancer Risks	Additivity	Analyze Display	Site Ranking Report Cancer Risk Ranking
Sites ranked in order of decreasing noncancer health threats (Hazard Indices)	Additivity	Analyze Display	Site Ranking Report Hazard Index Ranking
Boring Cancer Risks	Additivity	Analyze Display	Boring Additivity Report Carcinogenic Additivity
Boring Hazard Indices	Additivity	Analyze Display	Boring Additivity Report Noncarcinogenic Additivity
Borings ranked in order of decreasing Cancer Risks	Additivity	Analyze Display	Boring Ranking Report Cancer Risk Ranking
Borings ranked in order of decreasing Hazard Indices	Additivity	Analyze Display	Boring Ranking Report Hazard Index Ranking

* PPLVs displayed are carcinogenic for chemicals having a carcinogenic effect and noncarcinogenic for chemicals having a noncarcinogenic effect. For chemicals having both carcinogenic and noncarcinogenic effects, only the carcinogenic PPLVs are displayed. Noncarcinogenic PPLVs for these chemicals must either be accessed from output files or calculated from available data as shown on Tables D.1-1b and D.1-1c.

** All screen tables (such as those with cumulative PPLVs, cancer risks, hazard indices, and ranking reports) can be printed from screen.

*** Indirect pathway PPLVs are unique to each horizon at each site. This is because indirect PPLVs are dependent upon the soil concentration. Indirect PPLVs are shown separately for Horizon 1. (Horizon 2 PPLVs are indirect only.)

Table D.1-1b HHRC Data Available for File Output*

Result	Module	Menu	Tool Pathway
Probabilistic Input Parameters	Uncertainty	LHS	Save Variable Parameters to File
Deterministic (Fixed) Input Parameters	Uncertainty	LHS	Save Fixed Parameters to File
Indirect Pathway PPLVs (carcinogenic)	PPLV Results	Display	Save Indirect PPLVs to File
Indirect Pathway PPLVs (noncarcinognic)	PPLV Results	Display	Save Indirect PPLVs to File
Flux Ratios (Vapor Models Linearity Check)	PPLV Results	Display	Save Indirect PPLVs to File (included in same file as indirect PPLVs)
Site C_{rep} Concentrations	Additivity	Analyze	Save Site Summary to File
Site C_{max} Concentrations	Additivity	Analyze	Save Site Summary to File
Boring C_{max} Concentrations	Additivity	Analyze	Save Boring Summary to File
Site Cancer Risks	Additivity	Analyze	Save Site Summary to File
Site Noncancer Health Threats (Hazard Indices)	Additivity	Analyze	Save Site Summary to File
Boring Cancer Risks	Additivity	Analyze	Save Boring Summary to File
Boring Hazard Indices	Additivity	Analyze	Save Boring Summary to File

* For a more complete description of the output data summary files and default naming conventions, see Section D.1.2.6.

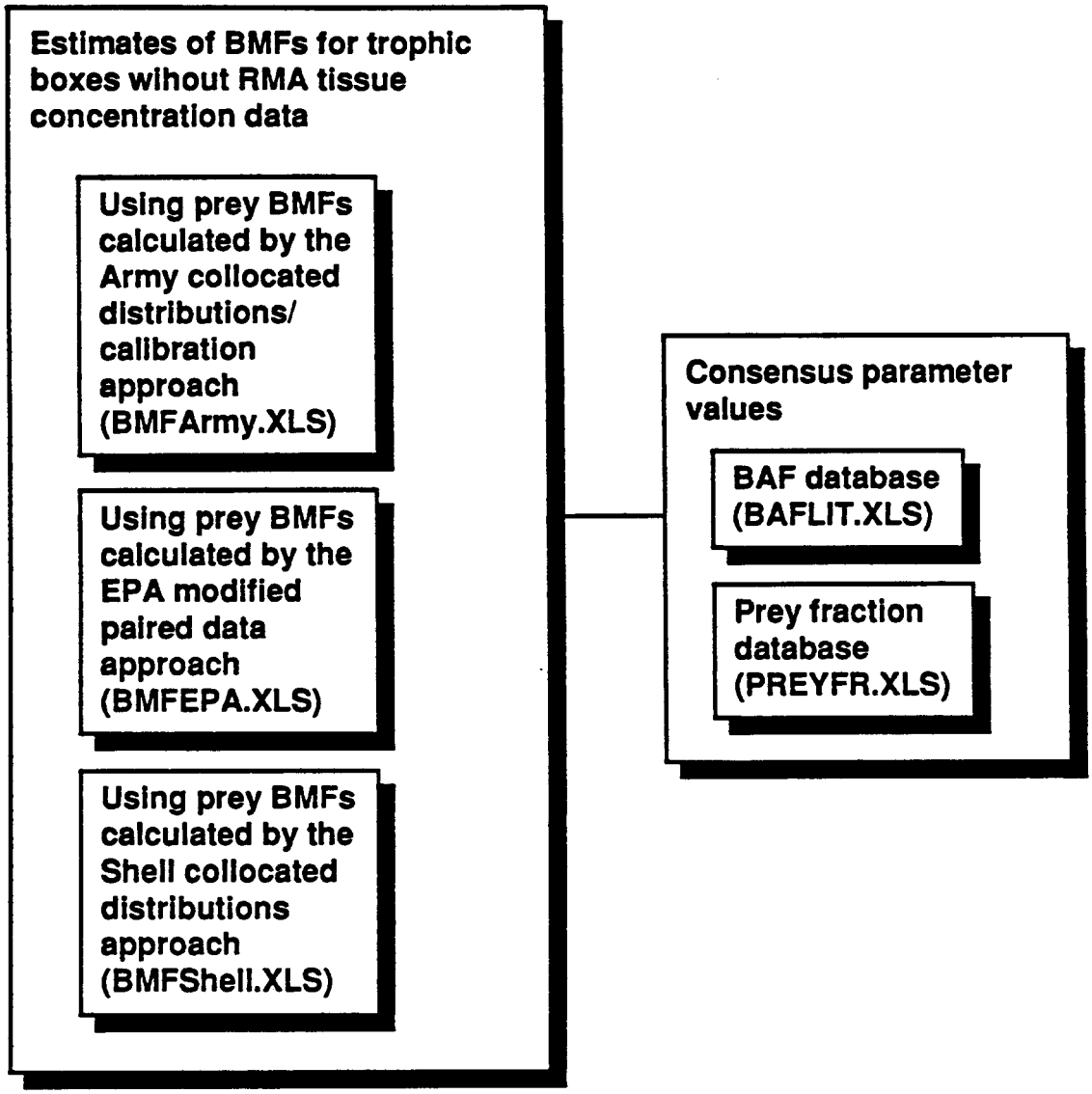
Table D.1-1c HHRC Results Calculated From Available Data*

Result	Data Needed for Calculation	Calculation
Direct Pathway PPLVs (noncarcinogenic for chemicals with both carcinogenic and noncarcinogenic effects)	Horizon 0 C_{rep}; Horizon 0 Hazard Index for C_{rep}	PPLV = C_{rep}/HI
Indirect Pathway PPLVs (noncarcinogenic for chemicals with both carcinogenic and noncarcinogenic effects)	Horizon 2 C_{rep}; Horizon 2 Hazard Index for C_{rep}	PPLV = C_{rep}/HI

* For a more complete description of the calculations performed to generate noncarcinogenic PPLV data used in the IEA/RC, see corresponding tables in Appendix B, Section B.4

Table D.1-2 RDM Database Structure Relevant to the SampleCalc Program

Structure Name	Field Name	Field Contents
Region	Region	Region name
	RegionAbbrev	Abbreviation or acronym for region
Site	Number	Index number for the site
	LakeSite	Flag that identifies lake sites
	Site	Site name
	CapRise	Capillary rise
	XoF	Dispersion coefficient
	Depth2GWavg	Average depth to groundwater
Boring	Boring	Boring identification number
	Boring Date	Date the boring was taken
	BoringX	X coordinate of the boring
	BoringY	Y coordinate of the boring
Boring Depth	Soil Depth	Sample depth within a boring
ChemConc	SampleType	Sample type: single bore or composite
	Conc	Chemical concentration (mg per g of medium)
	Limit	Flag to show if concentration is below, above, or within CRL limits
Chemical	Number	Chemical number of the data record
	ChemName	Chemical name
	ChemAbbrev	USATHEMA abbreviation for chemical name
	Chemical	Bit fields
	CRL	Certified Reporting Limit



RMA IEA/RC 2.94.dr

Figure D.1-1

Terrestrial Model Spreadsheet Linkages

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E
1					
2					
3	**Expected Values of BAFlit Distributions for Terrestrial Trophic Boxes**				
4		Ald/Did	DDE/DDT	Endrin	Mercury
5	**Trophic Box**				
6	Soil	1	1	1	1
7	TerPlt	6.49E-01	1.17E+00	6.49E-01	4.84E-01
8	Worm	4.95E+00	3.55E+00	1.32E+01	1.45E+00
9	Insect	1.91E+01	3.92E+00	1.03E+01	9.04E-01
10	SmBird	6.60E+00	1.84E+01	1.12E+00	7.77E-01
11	SmMammal	1.12E+00	7.10E-01	8.00E-02	2.42E+01
12	MdMammal	1.92E+00	7.10E-01	1.61E-01	2.42E+01
13	Hrp	3.58E+00	2.70E+00	3.58E+00	1.55E+00
14	Kestrel	1.05E+01	1.84E+01	1.12E+00	7.77E-01
15	Owl	2.11E+01	6.41E+01	1.12E+00	7.77E-01
16	Shorebird	1.33E+01	1.84E+01	1.12E+00	7.77E-01
17	Heron	1.60E+01	9.35E+01	1.12E+00	8.67E+00
18	Eagle	1.59E+01	3.98E+01	1.12E+00	7.77E-01

RMA IEA/RC 2.94.dr

Figure D.1-2 (page 1 of 2)
BAFLIT.XLS
Consensus Database of BAF Values for Terrestrial Trophic Boxes

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

BAFlit Distributions for Terrestrial Trophic Boxes

Trophic Box	Ald/Did	DDE/DDT	Endrin	Mercury
Soil	1	1	1	1
TerPlt	=RiskLognorm2(-1.109,1.163)	=RiskLognorm2(-0.151,0.788)	=RiskLognorm2(-1.109,1.163)	=RiskTriang(0.001,0.45,1)
Worm	=RiskLognorm2(1.253,0.833)	=RiskLognorm2(0.956,0.788)	=RiskLognorm2(0.916,1.825)	=RiskLognorm2(0.095,0.742)
Insect	=RiskTriang(6.5,8.8,42)	=RiskLognorm2(0.405,1.386)	=RiskLognorm2(1.988,0.833)	=RiskLognorm2(-0.117,0.182)
SmBird	=RiskNormal(6.6,1.8)	=RiskUniform(7.7,29)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.33,2)
SmMammal	=RiskUniform(0.64,1.6)	=RiskUniform(0.44,0.98)	=RiskLognorm2(-2.526,0.001)	=RiskTriang(0.001,22.5,50)
MdMammal	=RiskUniform(0.64,3.2)	=RiskUniform(0.44,0.98)	=RiskLognorm2(-1.833,0.095)	=RiskTriang(0.001,22.5,50)
Hrp	=RiskTriang(0.73,3.6,6.4)	=RiskNormal(2.7,0.5)	=RiskTriang(0.73,3.6,6.4)	=RiskLognorm2(0.405,0.262)
Kestrel	=RiskNormal(10.5,1.2)	=RiskUniform(7.7,29)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.33,2)
Owl	=RiskNormal(21.1,3.4)	=RiskLognorm2(3.777,0.875)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.33,2)
Shorebird	=RiskNormal(13.3,4.2)	=RiskUniform(7.7,29)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.33,2)
Heron	=RiskNormal(16,5.1)	=RiskNormal(93.5,20)	=RiskLognorm2(0,0.47)	=RiskLognorm2(1.411,1.224)
Eagle	=RiskNormal(15.9,3.9)	=RiskLognorm2(3.3,0.875)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.33,2)

RMA IEA/RC 2.94.dr

Figure D.1-2 (page 2 of 2)
BAFLIT.XLS
Consensus Database of BAF Values for Terrestrial Trophic Boxes

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E	F	G	H	I	J	K	L	M	N
1	Aquatic Prey Fraction Database												k = predator	
2													j = prey	
3		FR(k, j)												
4	Trophic Box	k=	1	2	3	4	5	6	7	8	9	10	11	12
5	Water	j=1	1	1	1	1	1	0	0	0	0.02	0	0.07	0
6	Sediment	2	0	0	0	0	0	0	0.130	0	0.04	0.160	0	0
7	Plankton	3	0	0	0	0	0	0	0	0	0	0	0	0
8	quatic Plant	4	0	0	0	0	0	0	0.01	0	0.94	0	0	0
9	quatic Invert.	5	0	0	0	0	0	1	0.86	0.05	0	0.11	0.02	0
10	Amphibian	6	0	0	0	0	0	0	0	0	0	0	0.01	0
11	Small Fish	7	0	0	0	0	0	0	0	0.95	0	0	0.19	0
12	Large Fish	8	0	0	0	0	0	0	0	0	0	0	0.6	0
13	Waterbird	9	0	0	0	0	0	0	0	0	0	0	0	0.030
14	Shorebird	10	0	0	0	0	0	0	0	0	0	0	0	0
15	Heron	11	0	0	0	0	0	0	0	0	0	0	0	0
16	Eagle	12	0	0	0	0	0	0	0	0	0	0	0	0
17	SUM(j)(FR(k, j)		1	1	1	1	1	1	1	1	1	0.27	0.891	0.03
18														
19	This worksheet contains estimates of relative importance of aquatic prey trophic boxes in a predator'													
20	The numbers are weight fractions. For example it is estimated that the diet for trophic box 7 (small													
21	comprised of 13% sediment, 0.1% plankton, 0.7% aquatic plants, and 86.2% aquatic invertebrates													

RMA IEA/RC 6.94.dr

Figure D.1-3

PREYFR.XLS

Consensus Database of Prey Fractions for Trophic Boxes

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

BMFArmy.XLS- Page 1 of 2

Pre-Calibration (Army Collocated Distributions) BMFs

This table contains the expected values of BMF distributions derived by the Army collocated distributions for kestrel, owl, heron, and eagle were subsequently calculated using the RMA terrestrial food web model concentration data were unavailable for these top predators. The kestrel, owl, heron, and eagle BMFs are from post-calibration BMFs.

Trophic Box	Ald/Dld	DDE/DDT	Endrin	Mercury
Soil	1	1	1	1
TerPlt	1.6E-02	6.6E-01	1.4E-01	3.5E-02
Worm	2.3E-01	1.4E+00	4.0E-01	6.2E-01
Insect	7.4E-02	7.5E-01	1.0E-01	1.1E-02
SmBird	2.1E-01	5.4E-01	1.1E-01	1.7E-01
SmMammal	6.1E-01	4.6E-01	1.7E-01	2.5E-03
MdMammal	3.8E-01	3.2E+00	1.6E-01	2.8E-01
Hrp	2.4E+00	2.5E+00	1.0E+00	6.0E-01
Kestrel	na	na	na	na
Owl	na	na	na	na
Shorebird	3.6E+00	4.8E+01	9.9E-01	1.2E+00
Heron	na	na	na	na
Eagle	na	na	na	na

Post-Calibration Army BMFs

The table below contains the Army's BMFs (post-calibration). The calibration process involved overlaying tissue concentration field data on tissue concentration prediction maps, where TCpred = ESC * BMFArm and adjusting the pre-calibration BMF as necessary to obtain reasonable corroboration of the RMA tissue data, as determined by professional judgement. The six enlarged bold entires are the BMFs that were cha calibration process from the pre-calibration (Army collocated distributions) values.

For kestrel, owl, heron, and eagle:

BMFArmy(k) = BAFlit(k) * SUM(j)(FR(k, j)*BMFArmy(j)) (equation 1-Army)

Nomenclature

BMFArmy = mean biomagnification factor (post-calibration) predicted by the Army approach or equati (tissue conc./estimated exposure soil conc.)

BAFlit = mean bioaccumulation factor distribution derived from literature values (predator tissue conc. / prey tissue conc.)

FR = mass fraction of total prey (dimensionless)

k = trophic box index variable for predator (dimensionless)

j = trophic box index variable for prey (dimensionless)

SUM(j)(f(j)) = summation over j of f(j)

Trophic Box	Ald/Dld	DDE/DDT	Endrin	Mercury
Soil	1	1	1	1
TerPlt	1.6E-02	6.6E-01	1.4E-01	3.5E-02
Worm	2.3E-01	1.4E+00	4.0E-01	6.2E-01
Insect	7.4E-02	7.5E-01	1.0E-01	1.1E-02
SmBird	2.1E-01	5.4E-01	1.1E-01	**1.1E-01**
SmMammal	**2.7E-01**	4.6E-01	1.7E-01	**5.5E-01**
MdMammal	3.8E-01	**4.9E-01**	**3.3E-02**	2.8E-01
Hrp	2.4E+00	**1.3E+00**	1.0E+00	6.0E-01
Kestrel	2.6E+00	9.9E+00	1.9E-01	3.2E-01
Owl	8.0E+00	3.2E+01	8.8E-02	2.6E-01
Shorebird	3.6E+00	4.8E+01	9.9E-01	1.2E+00
Heron	2.9E+00	1.1E+01	1.1E-01	6.8E-01
Eagle	6.1E+00	1.9E+01	6.7E-02	2.3E-01

Figure D.1-4 (page 1 of 2)

BMFARMY.XLS

Calculation of Top Predator BMFs by the Army Approach

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

Pre-Calibration (Army Collocated Distributions) BMFs

This table contains the BMF distributions derived by the Army collocated distributions approach.

Trophic Box	Ald/Did	DDE/DDT
Soil	1	1
TerPlt	=RiskNormal(0.016,0.002)	=RiskNormal(0.664,0.046)
Worm	=RiskNormal(0.232,0.031)	=RiskNormal(1.373,0.173)
Insect	=RiskNormal(0.074,0.011)	=RiskNormal(0.754,0.025)
SmBird	=RiskNormal(0.214,0.009)	=RiskNormal(0.536,0.045)
SmMammal	=RiskNormal(0.607,0.051)	=RiskNormal(0.457,0.062)
MdMammal	=RiskNormal(0.379,0.049)	=RiskNormal(3.18,1.35)
Hrp	=RiskNormal(2.396,0.179)	=RiskNormal(2.527,0.096)
Kestrel	na	na
Owl	na	na
Shorebird	=RiskNormal(3.611,0.201)	=RiskNormal(47.571,2.053)
Heron	na	na
Eagle	na	na

Trophic Box	Endrin	Mercury
Soil	1	1
TerPlt	=RiskNormal(0.143,0.017)	=RiskNormal(0.035,0.0003)
Worm	=RiskNormal(0.399,0.055)	=RiskNormal(0.621,0.088)
Insect	=RiskNormal(0.102,0.019)	=RiskNormal(0.011,0.001)
SmBird	=RiskNormal(0.111,0.013)	=RiskNormal(0.167,0.0004)
SmMammal	=RiskNormal(0.165,0.022)	=RiskNormal(0.0025,0.001)
MdMammal	=RiskNormal(0.161,0.015)	=RiskNormal(0.283,0.055)
Hrp	=RiskNormal(1.026,0.07)	=RiskNormal(0.596,0.011)
Kestrel	na	na
Owl	na	na
Shorebird	=RiskNormal(0.994,0.059)	=RiskNormal(1.154,0.016)
Heron	na	na
Eagle	na	na

Post-Calibration Army BMFs

This spreadsheet shows the formulas used to calculate the Army's kestrel, owl, heron and eagle BMFs. The cell references shown in the formulas can be located on the spreadsheets BAFLIT.XLS, PREYFRAC.XLS, and page 1 of BMFARMY.XLS.

Trophic Box	Ald/Did	DDE/DDT
Soil	1	1
TerPlt	1.6E-02	6.6E-01
Worm	2.3E-01	1.4E+00
Insect	7.4E-02	7.5E-01
SmBird	2.1E-01	5.4E-01
SmMammal	2.7E-01	4.6E-01
MdMammal	3.8E-01	4.9E-01
Hrp	2.4E+00	1.3E+00
Kestrel	=BAFLIT.XLS!$B$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,$B49:$B56)	=BAFLIT.XLS!$C$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,$C49:$C56)
Owl	=BAFLIT.XLS!$B$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,$B49:$B57)	=BAFLIT.XLS!$C$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,$C49:$C57)
Shorebird	3.6E+00	4.8E+01
Heron	=BAFLIT.XLS!$B$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,$B49:$B59)	=BAFLIT.XLS!$C$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,$C49:$C59)
Eagle	=BAFLIT.XLS!$B$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,$B49:$B60)	=BAFLIT.XLS!$C$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,$C49:$C60)

Trophic Box	Endrin	Mercury
Soil	1	1
TerPlt	1.4E-01	3.5E-02
Worm	4.0E-01	6.2E-01
Insect	1.0E-01	1.1E-02
SmBird	1.1E-01	1.1E-01
SmMammal	1.7E-01	5.5E-01
MdMammal	3.3E-02	2.8E-01
Hrp	1.0E+00	6.0E-01
Kestrel	=BAFLIT.XLS!$D$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,$D49:$D56)	=BAFLIT.XLS!$E$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,$E49:$E56)
Owl	=BAFLIT.XLS!$D$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,$D49:$D57)	=BAFLIT.XLS!$E$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,$E49:$E57)
Shorebird	9.9E-01	1.2E+00
Heron	=BAFLIT.XLS!$D$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,$D49:$D59)	=BAFLIT.XLS!$E$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,$E49:$E59)
Eagle	=BAFLIT.XLS!$D$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,$D49:$D60)	=BAFLIT.XLS!$E$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,$E49:$E60)

RMA IEA/RC 2.94.dr

Figure D.1-4 (page 2 of 2)

Calculation of Top Predator BMFs by the Army Approach

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

BMFEPA.XLS- Page 1 of 2

BMF Distributions Calculated by the EPA Approach

This table displays the expected values of BMF distributions derived by the EPA approach. BMFs for kestrel, owl, heron, and eagle were calculated using the RMA terrestrial food web model because tissue concentration data were unavailable for these top predators. The remaining BMFs were calculated by the EPA modified paired data approach.

For kestrel, owl, heron, and eagle:

BMFEPA(k) = BAFlit(k) * SUM(j)(FR(k, j)*BMFEPA(j)) (equation 1-EPA)

Nomenclature

BMFEPA = mean biomagnification factor predicted by the modified paired data approach or equation 1-EPA (tissue conc./estimated exposure soil conc.)

BAFlit = mean bioaccumulation factor distribution derived from literature values (predator tissue conc. / prey tissue conc.)

FR = mass fraction of total prey (dimensionless)

k = trophic box index variable for predator (dimensionless)

j = trophic box index variable for prey (dimensionless)

SUM(j)(f(j)) = summation over j of f(j)

Trophic Box	Ald/Dld	DDE/DDT	Endrin	Mercury
Soil	1	1	1	1
TerPlt	1.8E-01	5.2E+00	1.3E+00	3.1E-01
Worm	2.5E+00	7.8E+00	1.1E+00	8.1E-01
Insect	4.2E-01	3.9E+00	3.6E-01	2.7E-01
SmBird	6.8E-01	3.3E+00	9.1E-01	3.4E-01
SmMammal	3.0E+00	2.8E+00	1.5E+00	1.7E-01
MdMammal	1.9E+00	6.0E+00	1.2E+00	7.3E+00
Hrp	7.7E+00	6.3E+00	1.5E+00	8.2E-01
Kestrel	2.3E+01	5.5E+01	1.3E+00	1.8E-01
Owl	4.1E+01	3.4E+02	1.4E+00	4.8E+00
Shorebird	6.2E+00	1.5E+02	1.1E+00	1.8E-02
Heron	8.6E+00	4.2E+01	1.6E-01	7.6E-01
Eagle	2.8E+01	2.2E+02	1.3E+00	5.4E+00

RMA IEA/RC 6.94.dr

Figure D.1-5 (page 1 of 2)

BMFEPA.XLS

Calculation of Top Predator BMFs by the EPA Approach

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

This table contains the BMF distributions derived by the EPA modified paired approach, as well as the formulas used to calculate kestrel, owl, heron and eagle BMFs. The cell references shown in the formulas can be located on the spreadsheets BAFLIT.XLS, PREYFRAC.XLS, and page 1 of BMFEPA.XLS.

Trophic Box	Ald/Dld	DDE/DDT
Soil	1	1
TerPlt	=RiskNormal(0.1822,0.3474/SQRT(112))	=RiskNormal(5.1603,7.7157/SQRT(96))
Worm	=RiskNormal(2.5136,2.8447/SQRT(28))	=RiskNormal(7.796,21.5409/SQRT(28))
Insect	=RiskNormal(0.4178,0.6061/SQRT(55))	=RiskNormal(3.9162,4.3206/SQRT(55))
SmBird	=RiskNormal(0.6808,0.9625/SQRT(69))	=RiskNormal(3.2581,4.4091/SQRT(69))
SmMammal	=RiskNormal(2.9504,9.6134/SQRT(44))	=RiskNormal(2.7703,3.2284/SQRT(44))
MdMammal	=RiskNormal(1.8816,6.5122/SQRT(124))	=RiskNormal(5.9566,4.8128/SQRT(105))
Hrp	=RiskNormal(7.7388,14.9489/SQRT(7))	=RiskNormal(6.3151,10.565/SQRT(7))
Kestrel	=BAFLIT.XLS!$B$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,B21:B28)	=BAFLIT.XLS!$C$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,C21:C28)
Owl	=BAFLIT.XLS!$B$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,B21:B29)	=BAFLIT.XLS!$C$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,C21:C29)
Shorebird	=RiskNormal(6.2377,6.7652/SQRT(10))	=RiskNormal(153.7983,247.2037/SQRT(10))
Heron	=BAFLIT.XLS!$B$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,B21:B31)	=BAFLIT.XLS!$C$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,C21:C31)
Eagle	=BAFLIT.XLS!$B$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,B21:B32)	=BAFLIT.XLS!$C$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,C21:C32)

Trophic Box	Endrin	Mercury
Soil	1	1
TerPlt	=RiskNormal(1.3008,2.2946/SQRT(112))	=RiskNormal(0.3106,0.2283/SQRT(80))
Worm	=RiskNormal(1.0898,1.0001/SQRT(24))	=RiskNormal(0.8098,0.4959/SQRT(27))
Insect	=RiskNormal(0.3595,0.8454/SQRT(55))	=RiskNormal(0.2702,0.1852/SQRT(41))
SmBird	=RiskNormal(0.9062,1.7619/SQRT(69))	=RiskNormal(0.3432,0.1719/SQRT(67))
SmMammal	=RiskNormal(1.4704,1.7955/SQRT(45))	=RiskNormal(0.1739,0.7002/SQRT(43))
MdMammal	=RiskNormal(1.2217,1.742/SQRT(126))	=RiskNormal(7.3354,31.1/SQRT(107))
Hrp	=RiskNormal(1.4929,1.9355/SQRT(7))	=RiskNormal(0.8184,0.2741/SQRT(6))
Kestrel	=BAFLIT.XLS!$D$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,D21:D28)	=BAFLIT.XLS!$E$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,E21:E28)
Owl	=BAFLIT.XLS!$D$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,D21:D29)	=BAFLIT.XLS!$E$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,E21:E29)
Shorebird	=RiskNormal(1.1369,1.234/SQRT(10))	=RiskNormal(0.0181,0.0087/SQRT(10))
Heron	=BAFLIT.XLS!$D$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,D21:D31)	=BAFLIT.XLS!$E$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,E21:E31)
Eagle	=BAFLIT.XLS!$D$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,D21:D32)	=BAFLIT.XLS!$E$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,E21:E32)

RMA IEA/RC 2.94.dr

Figure D.1-5 (page 2 of 2)

Calculation of Top Predator BMFs by the EPA Approach

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

BMFShell.XLS- Page 1 of 2

BMF Distributions Calculated by the Shell Approach

This table displays the expected values of BMF distributions derived by the Shell approach. BMFs for kestrel, owl, heron, and eagle were calculated using the RMA terrestrial food web model because tissue concentration data were unavailable for these top predators. The remaining BMFs were calculated by the Shell collocated distributions approach.

For kestrel, owl, heron, and eagle:

BMFShell(k) = BAFlit(k) * SUM(j)(FR(k, j)*BMFShell(j)) (equation 1- Shell)

Nomenclature

BMFShell = mean biomagnification factor predicted by the Shell collocated distributions approach or equation 1- Shell (tissue conc./estimated exposure soil conc.)

BAFlit = mean bioaccumulation factor distribution derived from literature values (predator tissue conc. / prey tissue conc.)

FR = mass fraction of total prey (dimensionless)

k = trophic box index variable for predator (dimensionless)

j = trophic box index variable for prey (dimensionless)

SUM(j)(f(j)) = summation over j of f(j)

Trophic Box	Ald/Dld	DDE/DDT	Endrin	Mercury
Soil	1	1	1	1
TerPlt	6.0E-02	9.2E-01	2.1E-01	1.6E-01
Worm	1.0E+00	1.1E+00	2.4E-01	4.0E-01
Insect	9.7E-02	9.9E-01	5.3E-02	1.3E-01
SmBird	2.7E-01	8.1E-01	1.3E-01	1.9E-01
SmMammal	5.9E-01	6.5E-01	2.7E-01	1.5E-02
MdMammal	2.7E-01	3.1E+00	3.6E-01	3.3E-01
Hrp	2.4E+00	2.5E+00	9.0E-01	7.8E-01
Kestrel	4.9E+00	1.4E+01	2.6E-01	6.8E-02
Owl	6.9E+00	1.7E+02	4.0E-01	2.4E-01
Shorebird	2.3E+00	6.0E+01	6.0E-01	1.6E-01
Heron	3.0E+00	1.8E+01	1.0E-01	7.2E-01
Eagle	4.4E+00	1.2E+02	4.0E-01	2.6E-01

RMA IEA/RC 6.94.dr

Figure D.1-6 (page 1 of 2)

BMFSHELL.XLS

Calculation of Top Predator BMFs by the Shell Approach

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

This table contains the BMF distributions derived by the Shell collocated distributions approach, as well as the formulas used to calculate kestrel, owl, heron and eagle BMFs. Th references shown in the formulas can be located on the spreadsheets BAFLIT.XLS, PREYFRAC.XLS, and page 1 of BMFShell.XLS.

Trophic Box	Ald/Dld	DDE/DDT
Soil	1	1
TerPlt	=RiskNormal(0.06,0.0127)	=RiskNormal(0.92,0.14)
Worm	=RiskNormal(1.0416,0.3818)	=RiskNormal(1.0691,0.3021)
Insect	=RiskNormal(0.0965,0.0247)	=RiskNormal(0.994,0.1957)
SmBird	=RiskNormal(0.2663,0.1765)	=RiskNormal(0.805,0.1647)
SmMammal	=RiskNormal(0.5861,0.1331)	=RiskNormal(0.6514,0.136)
MdMammal	=RiskNormal(0.2669,0.0478)	=RiskNormal(3.1135,0.4388)
Hrp	=RiskNormal(2.368,1.7418)	=RiskNormal(2.5092,1.6152)
Kestrel	=BAFLIT.XLS!$B$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,B21:B28)	=BAFLIT.XLS!$C$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,C21:C28)
Owl	=BAFLIT.XLS!$B$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,B21:B29)	=BAFLIT.XLS!$C$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,C21:C29)
Shorebird	=RiskNormal(2.2906,2.0268)	=RiskNormal(60.4588,49.3872)
Heron	=BAFLIT.XLS!$B$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,B21:B31)	=BAFLIT.XLS!$C$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,C21:C31)
Eagle	=BAFLIT.XLS!$B$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,B21:B32)	=BAFLIT.XLS!$C$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,C21:C32)

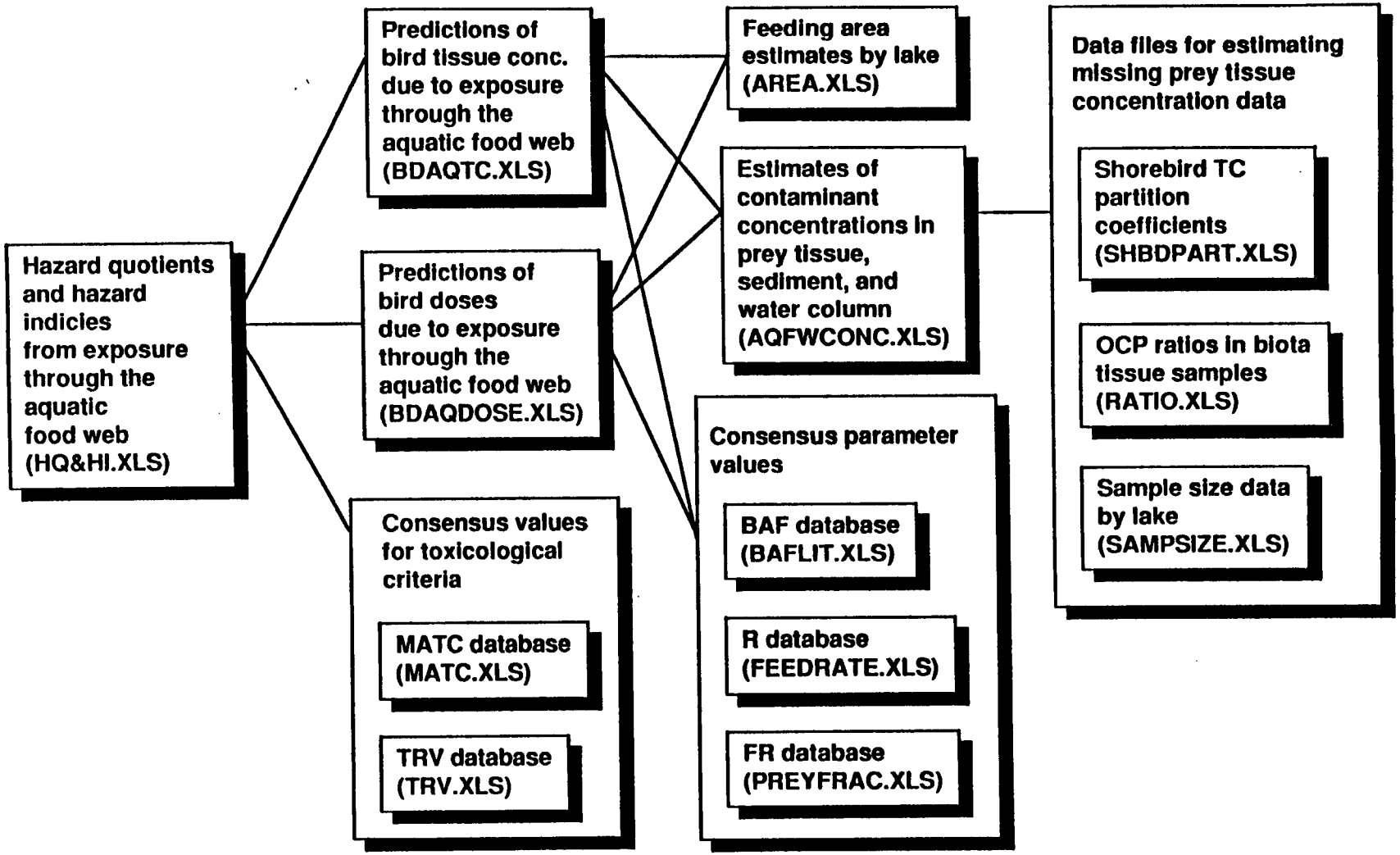
Trophic Box	Endrin	Mercury
Soil	1	1
TerPlt	=RiskNormal(1.3008,2.2946/SQRT(112))	=RiskNormal(0.3106,0.2283/SQRT(80))
Worm	=RiskNormal(1.0898,1.0001/SQRT(24))	=RiskNormal(0.8098,0.4959/SQRT(27))
Insect	=RiskNormal(0.3595,0.8454/SQRT(55))	=RiskNormal(0.2702,0.1852/SQRT(41))
SmBird	=RiskNormal(0.9062,1.7619/SQRT(69))	=RiskNormal(0.3432,0.1719/SQRT(67))
SmMammal	=RiskNormal(1.4704,1.7955/SQRT(45))	=RiskNormal(0.1739,0.7002/SQRT(43))
MdMammal	=RiskNormal(1.2217,1.742/SQRT(126))	=RiskNormal(7.3354,31.1/SQRT(107))
Hrp	=RiskNormal(1.4929,1.9355/SQRT(7))	=RiskNormal(0.8184,0.2741/SQRT(6))
Kestrel	=BAFLIT.XLS!$D$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K15,D21:D28)	=BAFLIT.XLS!$E$14*SUMPRODUCT(PREYFR.XLS!$K$7:$K$14,E21:E28)
Owl	=BAFLIT.XLS!$D$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,D21:D29)	=BAFLIT.XLS!$E$15*SUMPRODUCT(PREYFR.XLS!$L$7:$L$15,E21:E29)
Shorebird	=RiskNormal(0.5986,0.2869)	=RiskNormal(0.1617,0.0029)
Heron	=BAFLIT.XLS!$D$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,D21:D31)	=BAFLIT.XLS!$E$17*SUMPRODUCT(PREYFR.XLS!$N$7:$N$17,E21:E31)
Eagle	=BAFLIT.XLS!$D$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,D21:D32)	=BAFLIT.XLS!$E$18*SUMPRODUCT(PREYFR.XLS!$O$7:$O$18,E21:E32)

RMA IEA/RC 2.94.dr

Figure D.1-6 (page 2 of 2)

Calculation of Top Predator BMFs by the Shell Approach

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated



RMA IEA/RC 2.94.dr

Figure D.1-7

Aquatic Model Spreadsheet Linkages

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

Development of Shorebird Tissue Partitioning coefficients

Use

Tissue partitioning coefficients (TPC) are used to obtain terrestrial and aquatic contributions to observed TC.

TCobserved = TCobserved * TPC_Ter + TCobserved*TPC_Aqua

= TC_Ter + TC_Aqua

where TPCs are estimated in this spreadsheet

General Approach

TPCs depend on relative contribution of aquatic and terrestrial contaminant intake to total dose. Therefore TPCs depend on the food web and the relative exposure concentrations in the media. TPCs for the shorebird samples are developed for each of two areas where shorebird sampling occured. The TPCs for a given shorebird area are then applied to shorebird samples taken from that area.

Shorebird areas:

Area 1: Ladora and Lake Mary

Area 2: Upper and Lower Derby Lakes

Data used to estimate TPC

BMF

	TRPLT	INSCT
Ald/Dld	0.016	0.074
DDE/DDT	0.664	0.754
Endrin	0.143	0.102
Hg	0.035	0.011

FR

TRPLT	0.007
INSCT	0.728
Aqlnv	0.105
Sed	0.160

Average TCobserved for Aqlnv

(Average of observed tissue concentrations in the two lakes for each area)

	Area 1	Area 2
Ald/Dld	0.0154	0.0529
DDE/DDT	0.08	0.095
Endrin	0.025	0.025
Hg	0.0645	0.2667

Note: DDE/DDT average tissue concentration is the average of lake specific TC estimated based on the following ratios for other trophic boxes: (DDE + DDT) / (ALD + DLD) and (DDE + DDT) / Endrin . (Estimates are developed in RATIOS.XLS.)

Figure D.1-8 (page 1 of 3)

Calculation of Shorebird Tissue Partitioning Coefficients

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

Average Csoil

	Area 1	Area 2
Ald/Dld	0.0618	0.1701
DDE/DDT	0.0134	0.0273
Endrin	0.0041	0.0265
Hg	0.0501	0.0537

Average Csed

	Area 1	Area 2
Ald/Dld	0.0094	0.6782
DDE/DDT	0.0070	0.0108
Endrin	0.0008	0.0063
Hg	0.1339	0.0638

TPC Equations

$$TPC\_Ter = \frac{BAF\_k * SUM\_T (TC\_j\ FR\_j)}{BAF\_k * SUM\_T (TC\_j\ FR\_j) + BAF\_k * SUM\_A (TC\_j\ FR\_j)}$$

$$= \frac{T}{T + A}$$

$$TPC\_Aqua = \frac{A}{T + A}$$

where SUM_T and SUM_A refer to the summation over terrestrial and aquatic food items, respectively.

Calculation of T and A

T = avg Csed * (BMF_trplt * FR _trplt + BMF_insct*FR_insct)

A = avgTC_aqinv * FR_aqinv + avgCsed * FR_sed

Ald/Dld	
T_area 2	0.00333
T_area 1	0.00919
DDE/DDT	
T_area 2	0.00743
T_area 1	0.0151
Endrin	
T_area 2	0.000311
T_area 1	0.00200
Hg	
T_area 2	0.000413
T_area 1	0.000443

Ald/Dld	
A_area 2	0.00312
A_area 1	0.11406
DDE/DDT	
A_area 2	0.00951
A_area 1	0.0117
Endrin	
A_area 2	0.002759
A_area 1	0.00364
Hg	
A_area 2	0.028197
A_area 1	0.038204

RMA IEA/RC 2.94.dr

Figure D.1-8 (page 2 of 3)

Calculation of Shorebird Tissue Partitioning Coefficients

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E	F	G
79	**Calculation of TPCs**						
80	Ald/Dld				DDE/DDT		
81	Area 1	TPC Ter	0.517		Area 1	TPC Ter	0.438
82		TPC Aqua	0.483			TPC Aqua	0.562
83	Area 2	TPC Ter	0.075		Area 2	TPC Ter	0.563
84		TPC Aqua	0.925			TPC Aqua	0.437
85							
86	Endrin				Hg		
87	Area 1	TPC Ter	0.101		Area 1	TPC Ter	0.014
88		TPC Aqua	0.899			TPC Aqua	0.986
89	Area 2	TPC Ter	0.355		Area 2	TPC Ter	0.011
90		TPC Aqua	0.645			TPC Aqua	0.989

Figure D.1-8 (page 3 of 3)

SHBDPART.XLS

Calculation of Shorebird Tissue Partitioning Coefficients

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

Sediment (Csed)

Lake	Ald/Dld	Endrin
Upper Derby	0.10	12.43
Lower Derby	0.02	2.44
od & Gun Club	0.33	5.78
Ladora	0.33	11.27
Mary	0.56	10.68

Large Fish

Lake	Ald/Dld	Endrin
Upper Derby		
Lower Derby	1.68	11.89
Rod & Gun Club		
Ladora	1.22	5.75
Mary	3.84	7.60

Plankton

Lake	Ald/Dld	Endrin
Upper Derby		
Lower Derby	0.60	3.39
od & Gun Club		
Ladora	1.82	3.40
Mary	3.71	3.52

Waterbird

Lake	Ald/Dld	Endrin
Upper Derby	0.30	7.29
Lower Derby	0.46	24.02
Rod & Gun Club	0.20	10.02
Ladora	0.98	5.11
Mary	1.13	8.08

Aquatic Plant

Lake	Ald/Dld	Endrin
Upper Derby		
Lower Derby	2.32	4.52
od & Gun Club	1.96	2.50
Ladora	2.71	5.00
Mary	4.27	3.46

Shorebird

Lake	Ald/Dld	Endrin
Upper Derby		
Lower Derby	1.52	44.47
Rod & Gun Club		
Ladora		
Mary	13.60	65.81

Small Fish

Lake	Ald/Dld	Endrin
Upper Derby	0.37	3.81
Lower Derby	0.77	3.76
od & Gun Club		
Ladora	0.56	3.50
Mary	1.49	6.00

This spreadsheet contains chemical concentration and tissue concentration prediction ratios. Chemical concentration ratios for plankton and aquatic plants are used to predict tissue DDE/DDT concentrations in aquatic invertebrates, and chemical concentration ratios for small and large fish are used to predict tissue DDE/DDT concentrations in amphibians on the spreadsheet AQFWCONC.XLS. Cell entries are ratios of DDE/DDT tissue concentration to COC tissue concentration, where COC is given by the column heading, for the lake indicated by the row heading and the trophic box indicated by the table heading. Empty cells indicate missing data.

RMA IEA/RC 6.94.dr

Figure D.1-9

RATIOS.XLS

Organochlorine Pesticide Ratios in Average Aquatic Biota Tissue Samples

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E	F	G	H
1	**Sample Size Database**							
4								
5			**Sample Size**					
6		Plankton	Aquatic Plant	Aquatic Invert.				
7								
8	E. Upper Derby	0	0	0				
9	Upper Derby	0	0	0				
10	Lower Derby	19	29	10				
11	Rod & Gun Club	0	4	0				
12	Ladora	18	45	9				
13	Mary	13	23	0				
14								
15	This spreadsheet contains sample size data that are used as lake-specific sample average weighting factors in estimating							
16	RMA-average tissue concentrations on the spreadsheet AQFWCONC.XLS.							

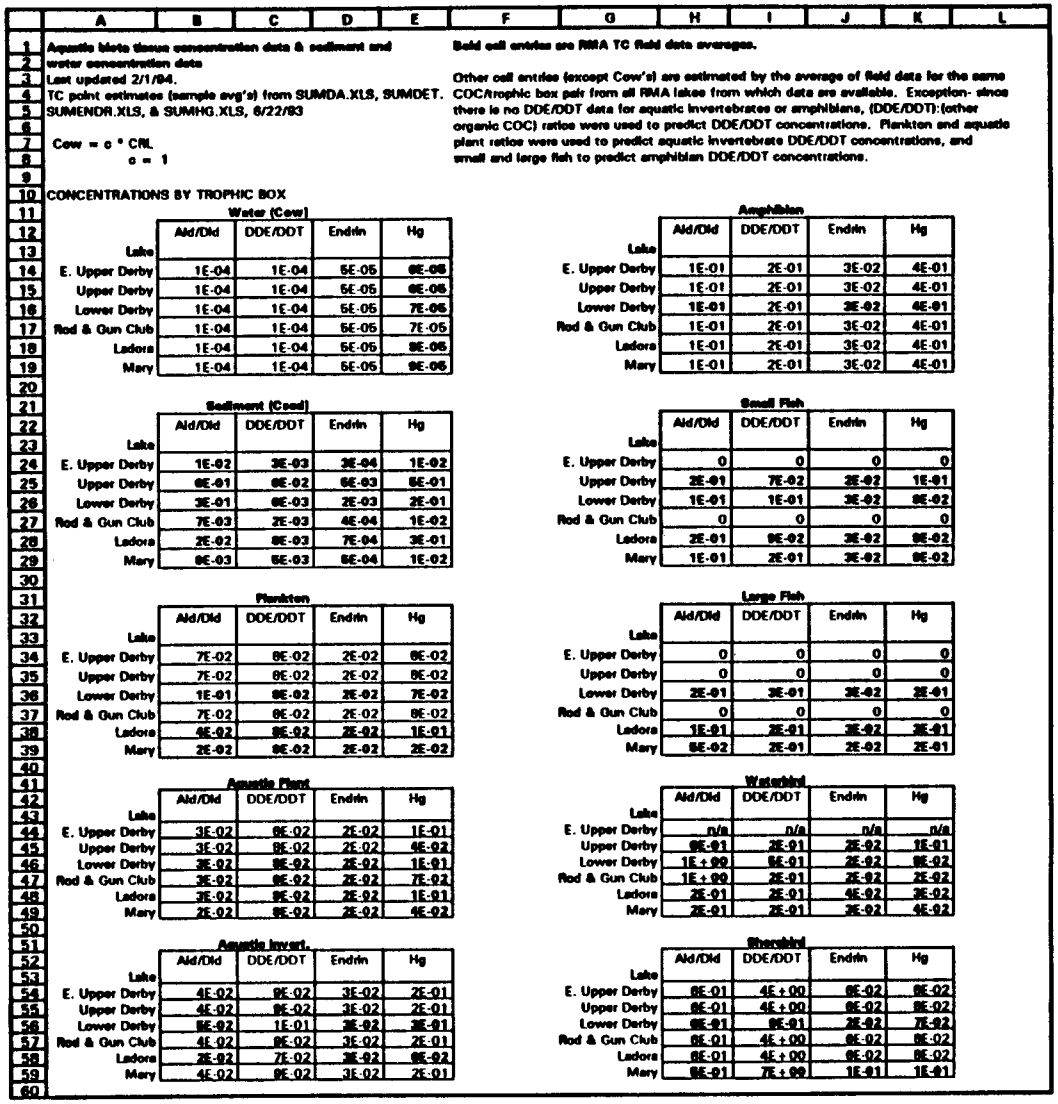
RMA IEA/RC 6.94.dr

Figure D.1-10

SAMPSIZE.XLS

Aqatic Biota Sample Size Database

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated



Aquatic biota tissue concentration data & sediment and water concentration data
Last updated 2/1/94.
TC point estimates (sample avg's) from SUMDA.XLS, SUMDET.XLS, SUMENDR.XLS, & SUMHG.XLS, 6/22/93

Cow = c * CRL
c = 1

Bold cell entries are RMA TC field data averages.

Other cell entries (except Cow's) are estimated by the average of field data for the same COC/trophic box pair from all RMA lakes from which data are available. Exception- since there is no DDE/DDT data for aquatic invertebrates or amphibians, (DDE/DDT):(other organic COC) ratios were used to predict DDE/DDT concentrations. Plankton and aquatic plant ratios were used to predict aquatic invertebrate DDE/DDT concentrations, and small and large fish to predict amphibian DDE/DDT concentrations.

CONCENTRATIONS BY TROPHIC BOX

Water (Cow)

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	1E-04	1E-04	5E-06	9E-06
Upper Derby	1E-04	1E-04	5E-06	9E-06
Lower Derby	1E-04	1E-04	5E-06	7E-06
Rod & Gun Club	1E-04	1E-04	5E-06	7E-06
Ladora	1E-04	1E-04	5E-06	9E-06
Mary	1E-04	1E-04	5E-06	9E-06

Sediment (Csed)

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	1E-02	3E-03	3E-04	1E-02
Upper Derby	6E-01	6E-02	6E-03	5E-01
Lower Derby	3E-01	9E-03	2E-03	2E-01
Rod & Gun Club	7E-03	2E-03	4E-04	1E-02
Ladora	2E-02	6E-03	7E-04	3E-01
Mary	9E-03	5E-03	5E-04	1E-02

Plankton

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	7E-02	6E-02	2E-02	6E-02
Upper Derby	7E-02	6E-02	2E-02	6E-02
Lower Derby	1E-01	6E-02	2E-02	7E-02
Rod & Gun Club	7E-02	6E-02	2E-02	6E-02
Ladora	4E-02	8E-02	2E-02	1E-01
Mary	2E-02	9E-02	2E-02	2E-02

Aquatic Plant

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	3E-02	9E-02	2E-02	1E-01
Upper Derby	3E-02	8E-02	2E-02	4E-02
Lower Derby	3E-02	8E-02	2E-02	1E-01
Rod & Gun Club	3E-02	9E-02	2E-02	7E-02
Ladora	3E-02	9E-02	2E-02	1E-01
Mary	2E-02	9E-02	2E-02	4E-02

Aquatic Invert.

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	4E-02	9E-02	3E-02	2E-01
Upper Derby	4E-02	9E-02	3E-02	2E-01
Lower Derby	8E-02	1E-01	3E-02	3E-01
Rod & Gun Club	4E-02	9E-02	3E-02	2E-01
Ladora	2E-02	7E-02	3E-02	6E-02
Mary	4E-02	9E-02	3E-02	2E-01

Amphibian

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	1E-01	2E-01	3E-02	4E-01
Upper Derby	1E-01	2E-01	3E-02	4E-01
Lower Derby	1E-01	2E-01	3E-02	4E-01
Rod & Gun Club	1E-01	2E-01	3E-02	4E-01
Ladora	1E-01	2E-01	3E-02	4E-01
Mary	1E-01	2E-01	3E-02	4E-01

Small Fish

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	0	0	0	0
Upper Derby	2E-01	7E-02	2E-02	1E-01
Lower Derby	1E-01	1E-01	3E-02	9E-02
Rod & Gun Club	0	0	0	0
Ladora	2E-01	9E-02	3E-02	9E-02
Mary	1E-01	2E-01	3E-02	9E-02

Large Fish

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	0	0	0	0
Upper Derby	0	0	0	0
Lower Derby	2E-01	3E-01	3E-02	2E-01
Rod & Gun Club	0	0	0	0
Ladora	1E-01	2E-01	3E-02	3E-01
Mary	5E-02	2E-01	2E-02	2E-01

Waterbird

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	n/a	n/a	n/a	n/a
Upper Derby	9E-01	2E-01	2E-02	1E-01
Lower Derby	1E+00	6E-01	2E-02	8E-02
Rod & Gun Club	1E+00	2E-01	2E-02	2E-02
Ladora	2E-01	2E-01	4E-02	3E-02
Mary	2E-01	2E-01	3E-02	4E-02

Shorebird

Lake	Ald/Dld	DDE/DDT	Endrin	Hg
E. Upper Derby	8E-01	4E+00	8E-02	8E-02
Upper Derby	8E-01	4E+00	8E-02	8E-02
Lower Derby	8E-01	9E-01	2E-02	7E-02
Rod & Gun Club	8E-01	4E+00	8E-02	8E-02
Ladora	8E-01	4E+00	8E-02	8E-02
Mary	8E-01	7E+00	1E-01	1E-01

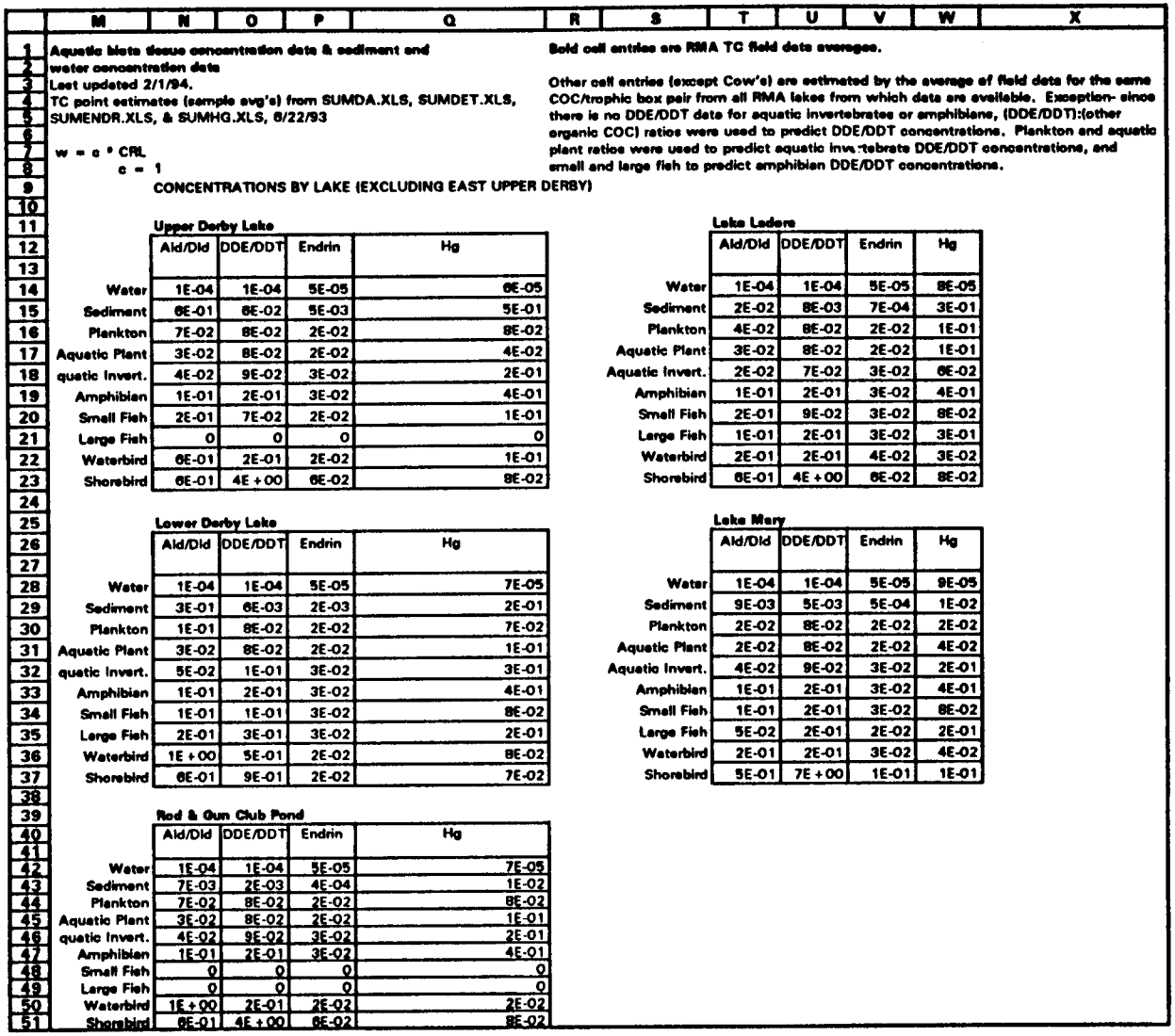
RMA IEA/RC 6.94.dr

Figure D.1-11 (page 1 of 2)

AQFWCONC.XLS

Aquatic Biota Tissue, Sediment, and Water Concentration Database

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated



Aquatic biota tissue concentration data & sediment and water concentration data
Last updated 2/1/94.
TC point estimates (sample avg's) from SUMDA.XLS, SUMDET.XLS, SUMENDR.XLS, & SUMHG.XLS, 6/22/93

w = c * CRL
c = 1

Bold cell entries are RMA TC field data averages.

Other cell entries (except Cow's) are estimated by the average of field data for the same COC/trophic box pair from all RMA lakes from which data are available. Exception- since there is no DDE/DDT data for aquatic invertebrates or amphibians, (DDE/DDT):(other organic COC) ratios were used to predict DDE/DDT concentrations. Plankton and aquatic plant ratios were used to predict aquatic invertebrate DDE/DDT concentrations, and small and large fish to predict amphibian DDE/DDT concentrations.

CONCENTRATIONS BY LAKE (EXCLUDING EAST UPPER DERBY)

Upper Derby Lake

	Ald/Dld	DDE/DDT	Endrin	Hg
Water	1E-04	1E-04	5E-05	6E-05
Sediment	6E-01	6E-02	5E-03	5E-01
Plankton	7E-02	8E-02	2E-02	8E-02
Aquatic Plant	3E-02	8E-02	2E-02	4E-02
quatic Invert.	4E-02	9E-02	3E-02	2E-01
Amphibian	1E-01	2E-01	3E-02	4E-01
Small Fish	2E-01	7E-02	2E-02	1E-01
Large Fish	0	0	0	0
Waterbird	6E-01	2E-01	2E-02	1E-01
Shorebird	6E-01	4E+00	6E-02	8E-02

Lake Ladora

	Ald/Dld	DDE/DDT	Endrin	Hg
Water	1E-04	1E-04	5E-05	8E-05
Sediment	2E-02	8E-03	7E-04	3E-01
Plankton	4E-02	8E-02	2E-02	1E-01
Aquatic Plant	3E-02	8E-02	2E-02	1E-01
Aquatic Invert.	2E-02	7E-02	3E-02	6E-02
Amphibian	1E-01	2E-01	3E-02	4E-01
Small Fish	2E-01	9E-02	3E-02	8E-02
Large Fish	1E-01	2E-01	3E-02	3E-01
Waterbird	2E-01	2E-01	4E-02	3E-02
Shorebird	6E-01	4E+00	6E-02	8E-02

Lower Derby Lake

	Ald/Dld	DDE/DDT	Endrin	Hg
Water	1E-04	1E-04	5E-05	7E-05
Sediment	3E-01	6E-03	2E-03	2E-01
Plankton	1E-01	8E-02	2E-02	7E-02
Aquatic Plant	3E-02	8E-02	2E-02	1E-01
quatic Invert.	5E-02	1E-01	3E-02	3E-01
Amphibian	1E-01	2E-01	3E-02	4E-01
Small Fish	1E-01	1E-01	3E-02	8E-02
Large Fish	2E-01	3E-01	3E-02	2E-01
Waterbird	1E+00	5E-01	2E-02	8E-02
Shorebird	6E-01	9E-01	2E-02	7E-02

Lake Mary

	Ald/Dld	DDE/DDT	Endrin	Hg
Water	1E-04	1E-04	5E-05	9E-05
Sediment	9E-03	5E-03	5E-04	1E-02
Plankton	2E-02	8E-02	2E-02	2E-02
Aquatic Plant	2E-02	8E-02	2E-02	4E-02
Aquatic Invert.	4E-02	9E-02	3E-02	2E-01
Amphibian	1E-01	2E-01	3E-02	4E-01
Small Fish	1E-01	2E-01	3E-02	8E-02
Large Fish	5E-02	2E-01	2E-02	2E-01
Waterbird	2E-01	2E-01	3E-02	4E-02
Shorebird	5E-01	7E+00	1E-01	1E-01

Rod & Gun Club Pond

	Ald/Dld	DDE/DDT	Endrin	Hg
Water	1E-04	1E-04	5E-05	7E-05
Sediment	7E-03	2E-03	4E-04	1E-02
Plankton	7E-02	8E-02	2E-02	8E-02
Aquatic Plant	3E-02	8E-02	2E-02	1E-01
quatic Invert.	4E-02	9E-02	3E-02	2E-01
Amphibian	1E-01	2E-01	3E-02	4E-01
Small Fish	0	0	0	0
Large Fish	0	0	0	0
Waterbird	1E+00	2E-01	2E-02	2E-02
Shorebird	6E-01	4E+00	6E-02	8E-02

RMA IEA/RC 6.94.dr

Figure D.1-11 (page 2 of 2)

AQFWCONC.XLS

Aquatic Biota Tissue, Sediment, and Water Concentration Database

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E
1	**BAFlit Database**				
2					
3			**BAFlit Means**		
4		Ald/Dld	DDE/DDT	Endrin	Mercury
5	**Trophic Box**				
6	Soil	1	1	1	1
7	TerPlt	6.49E-01	1.17E+00	6.49E-01	5.14E-01
8	Worm	4.84E+00	3.55E+00	1.32E+01	1.45E+00
9	Insect	1.91E+01	3.92E+00	1.03E+01	9.04E-01
10	SmBird	6.60E+00	1.84E+01	1.12E+00	8.00E-01
11	SmMammal	9.35E-01	7.10E-01	8.00E-02	2.46E+01
12	MdMammal	9.35E-01	7.10E-01	1.61E-01	2.46E+01
13	Hrp	3.58E+00	2.70E+00	3.58E+00	1.55E+00
14	Kestrel	1.05E+01	1.84E+01	1.12E+00	8.00E-01
15	Owl	2.11E+01	4.37E+01	1.12E+00	8.00E-01
16	Water Bird	1.60E+01	9.60E+01	1.12E+00	8.67E+00
17	Shorebird	1.33E+01	1.84E+01	1.12E+00	8.00E-01
18	Heron	1.60E+01	93.5000	1.12E+00	8.6719
19	Eagle	15.9000	3.98E+01	1.1168	8.00E-01
20					
21					
22			**BAFlit Distributions**		
23		Ald/Dld	DDE/DDT	Endrin	Mercury
24	**Trophic Box**				
25	Soil	1	1	1	1
26	TerPlt	=RiskLognorm2(-1.109,1.	=RiskLognorm2(-0.151,0.	=RiskLognorm2(-1.109,1.	=RiskTriang(0.001,0.45,
27	Worm	=RiskLognorm2(1.194,0.	=RiskLognorm2(0.956,0.	=RiskLognorm2(0.916,1.8	=RiskLognorm2(0.095,0.
28	Insect	=RiskTriang(6.5,8.8,42)	=RiskLognorm2(0.405,1.	=RiskLognorm2(1.988,0.8	=RiskLognorm2(-0.117,0.
29	SmBird	=RiskNormal(6.6,1.8)	=RiskUniform(7.7,29)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.3,2.
30	SmMammal	=RiskUniform(0.27,1.6)	=RiskUniform(0.44,0.98)	=RiskLognorm2(-2.526,0.	=RiskTriang(0.001,22.5,
31	MdMammal	=RiskUniform(0.27,1.6)	=RiskUniform(0.44,0.98)	=RiskLognorm2(-1.833,0.	=RiskTriang(0.001,22.5,
32	Hrp	=RiskTriang(0.73,3.6,6.4	=RiskNormal(2.7,0.5)	=RiskTriang(0.73,3.6,6.4)	=RiskLognorm2(0.405,0.
33	Kestrel	=RiskNormal(10.5,1.2)	=RiskUniform(7.7,29)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.3,2.
34	Owl	=RiskNormal(21.1,3.4)	=RiskLognorm(43.7,26.2)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.3,2.
35	Water Bird	=RiskNormal(16,5.1)	=RiskNormal(96,26.2)	=RiskLognorm2(0,0.47)	=RiskLognorm2(1.411,1.
36	Shorebird	=RiskNormal(13.3,4.2)	=RiskUniform(7.7,29)	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.3,2.
37	Heron	=RiskNormal(16,5.1)	=RiskNormal(93.5,20)	=RiskLognorm2(0,0.47)	=RiskLognorm2(1.411,1.
38	Eagle	=RiskNormal(15.9,3.9)	=RiskLognorm2(3.3,0.87	=RiskLognorm2(0,0.47)	=RiskTriang(0.001,0.3,2.

RMA IEA/RC 6.94.dr

Figure D.1-12

BAFLIT.XLS

Consensus BAF_{lit} Distributions Database

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C
1	**Feeding Rate Coefficient (R) Database**		
2	The feeding rate coefficient is an estimate of the normalized average rate		
3	of food consumption for a trophic box (grams food per gram body weight per day).		
4			
5	Trophic Box	Expected Value of R	Distributions
6	Water	na	na
7	Sediment	na	na
8	Plankton	1	1
9	Aquatic Plant	1	1
10	Aquatic Invert.	1	1
11	Amphibian	1.03E-01	=RiskNormal(0.10303,0.0332)
12	Small Fish	2.33E-02	=RiskNormal(0.02333,0.00416)
13	Large Fish	3.12E-03	=RiskNormal(0.00312,0.001)
14	Waterbird	7.60E-02	=RiskNormal(0.07603,0.0245)
15	Shorebird	9.97E-02	=RiskLognorm2(-2.4315,0.50189)
16	Heron	8.91E-02	=RiskNormal(0.08913,0.02689)
17	Eagle	8.91E-02	=RiskNormal(0.08913,0.02689)

RMA IEA/RC 6.94.dr

Figure D.1-13

FEEDRATE.XLS

Consensus Feeding Rate Coefficient Distributions Database

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E	F	G	H	I	J	K	L	M	N
1	Aquatic Prey Fraction Database												k = predator	
2													j = prey	
3		FR(k, j)												
4	Trophic Box	k =	1	2	3	4	5	6	7	8	9	10	11	12
5	Water	j = 1	1	1	1	1	1	0	0	0	0.02	0	0.07	0
6	Sediment	2	0	0	0	0	0	0	0.130	0	0.04	0.160	0	0
7	Plankton	3	0	0	0	0	0	0	0	0	0	0	0	0
8	quatic Plant	4	0	0	0	0	0	0	0.01	0	0.94	0	0	0
9	quatic Invert.	5	0	0	0	0	0	1	0.86	0.05	0	0.11	0.02	0
10	Amphibian	6	0	0	0	0	0	0	0	0	0	0	0.01	0
11	Small Fish	7	0	0	0	0	0	0	0	0.95	0	0	0.19	0
12	Large Fish	8	0	0	0	0	0	0	0	0	0	0	0.6	0
13	Waterbird	9	0	0	0	0	0	0	0	0	0	0	0	0.030
14	Shorebird	10	0	0	0	0	0	0	0	0	0	0	0	0
15	Heron	11	0	0	0	0	0	0	0	0	0	0	0	0
16	Eagle	12	0	0	0	0	0	0	0	0	0	0	0	0
17	SUM(j)(FR(k, j)		1	1	1	1	1	1	1	1	1	0.27	0.891	0.03
18														
19	This worksheet contains estimates of relative importance of aquatic prey trophic boxes in a predator's diet.													
20	The numbers are weight fractions. For example it is estimated that the diet for trophic box 7 (small fish) is													
21	comprised of 13% sediment, 0.1% plankton, 0.7% aquatic plants, and 86.2% aquatic invertebrates (by weight).													

RMA IEA/RC 6.94.dr

Figure D.1-14

PREYFRAC.XLS

Database of Consensus Values for Aquatic Prey Fractions

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E
1	**Assumed Lake Areas (square feet)**				
2	This spreadsheet contains estimates of lake surface and perimeter areas. Normalized lake perimeter areas are used				
3	to apportion shorebird's aquatic feeding among the lakes. Normalized water areas are used for waterbird, heron,				
4	and eagle. Perimeter area is defined as the area of a ring around the lake's edge with outer perimeter at the water's				
5	edge and inner perimeter three feet from the lake's edge. Water area is defined as the lakes's surface water area.				
6	Areas represent November, 1988 lake dimensions.				
7					
8					
9	**Assumed Lake Areas**				
10		shoreline area	water area		
11	Upper Derby Lake	27384	1862586		
12	Lower Derby Lake	27349	2962483		
13	Rod & Gun Club Pond	16675	1028901		
14	Lake Ladora	38584	2531880		
15	Lake Mary	9791	406194		
16					
17					
18	**Assumed Relative Sizes of Lake-Specific Feeding Areas**				
19					
20		Waterbird	Shorebird	Heron	Eagle
21		(normalized water are	(normalized perimeter are	(normalized water are	(normalized water areas)
22	Upper Derby	0.2118	0.2286	0.2118	0.2118
23	Lower Derby	0.3370	0.2283	0.3370	0.3370
24	Rod & Gun Club	0.1170	0.1392	0.1170	0.1170
25	Ladora	0.2880	0.3221	0.2880	0.2880
26	Mary	0.0462	0.0817	0.0462	0.0462

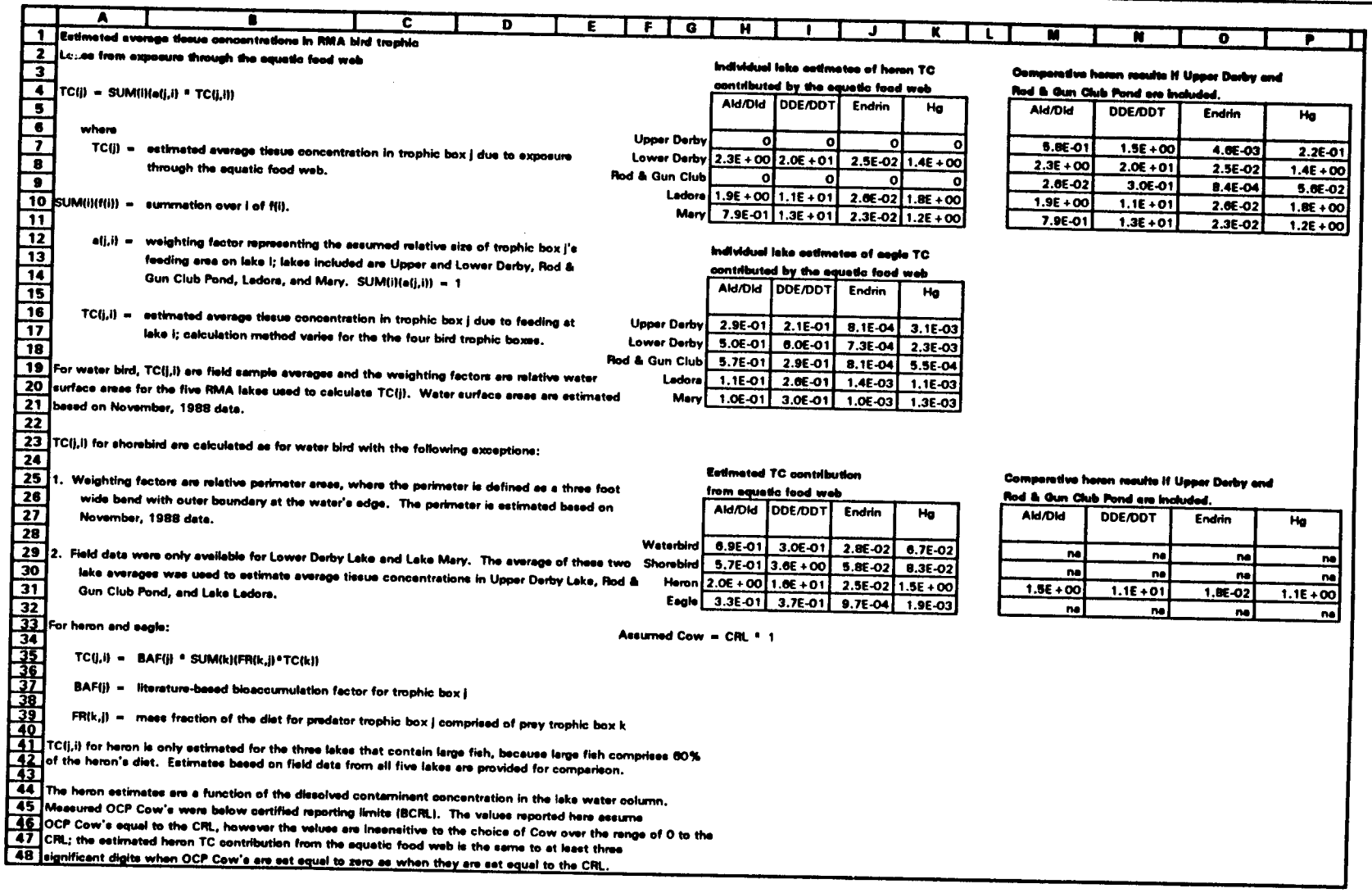
RMA IEA/RC 6.94.dr

Figure D.1-15

AREA.XLS

Database of Assumed Lake-Specific Feeding Areas for WaterBird, Shorebird, Heron, and Eagle

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated



Estimated average tissue concentrations in RMA bird trophic
Le:.ee from exposure through the equatic food web

TC(j) = SUM(i)(e(j,i) * TC(j,i))

where

TC(j) = estimated average tissue concentration in trophic box j due to exposure through the equatic food web.

SUM(i)(f(i)) = summation over i of f(i).

e(j,i) = weighting factor representing the assumed relative size of trophic box j's feeding area on lake i; lakes included are Upper and Lower Derby, Rod & Gun Club Pond, Ledora, and Mary. SUM(i)(e(j,i)) = 1

TC(j,i) = estimated average tissue concentration in trophic box j due to feeding at lake i; calculation method varies for the the four bird trophic boxes.

For water bird, TC(j,i) are field sample averages and the weighting factors are relative water surface areas for the five RMA lakes used to calculate TC(j). Water surface areas are estimated based on November, 1988 data.

TC(j,i) for shorebird are calculated as for water bird with the following exceptions:

1. Weighting factors are relative perimeter areas, where the perimeter is defined as a three foot wide band with outer boundary at the water's edge. The perimeter is estimated based on November, 1988 data.

2. Field data were only available for Lower Derby Lake and Lake Mary. The average of these two lake averages was used to estimate average tissue concentrations in Upper Derby Lake, Rod & Gun Club Pond, and Lake Ledora.

For heron and eagle:

TC(j,i) = BAF(j) * SUM(k)(FR(k,j)*TC(k))

BAF(j) = literature-based bioaccumulation factor for trophic box j

FR(k,j) = mass fraction of the diet for predator trophic box j comprised of prey trophic box k

TC(j,i) for heron is only estimated for the three lakes that contain large fish, because large fish comprises 60% of the heron's diet. Estimates based on field data from all five lakes are provided for comparison.

The heron estimates are a function of the dissolved contaminent concentration in the lake water column. Measured OCP Cow's were below certified reporting limits (BCRL). The values reported here assume OCP Cow's equal to the CRL, however the values are insensitive to the choice of Cow over the range of 0 to the CRL; the estimated heron TC contribution from the aquatic food web is the same to at least three significant digits when OCP Cow's are set equal to zero as when they are set equal to the CRL.

Individual lake estimates of heron TC contributed by the aquatic food web

	Ald/Did	DDE/DDT	Endrin	Hg
Upper Derby	0	0	0	0
Lower Derby	2.3E+00	2.0E+01	2.5E-02	1.4E+00
Rod & Gun Club	0	0	0	0
Ledora	1.9E+00	1.1E+01	2.6E-02	1.8E+00
Mary	7.9E-01	1.3E+01	2.3E-02	1.2E+00

Comparative heron results if Upper Derby and Rod & Gun Club Pond are included.

Ald/Did	DDE/DDT	Endrin	Hg
5.8E-01	1.5E+00	4.6E-03	2.2E-01
2.3E+00	2.0E+01	2.5E-02	1.4E+00
2.6E-02	3.0E-01	8.4E-04	5.6E-02
1.9E+00	1.1E+01	2.6E-02	1.8E+00
7.9E-01	1.3E+01	2.3E-02	1.2E+00

Individual lake estimates of eagle TC contributed by the aquatic food web

	Ald/Did	DDE/DDT	Endrin	Hg
Upper Derby	2.9E-01	2.1E-01	8.1E-04	3.1E-03
Lower Derby	5.0E-01	6.0E-01	7.3E-04	2.3E-03
Rod & Gun Club	5.7E-01	2.9E-01	8.1E-04	5.5E-04
Ledora	1.1E-01	2.6E-01	1.4E-03	1.1E-03
Mary	1.0E-01	3.0E-01	1.0E-03	1.3E-03

Estimated TC contribution from aquatic food web

	Ald/Did	DDE/DDT	Endrin	Hg
Waterbird	6.9E-01	3.0E-01	2.8E-02	6.7E-02
Shorebird	5.7E-01	3.6E+00	5.8E-02	8.3E-02
Heron	2.0E+00	1.6E+01	2.5E-02	1.5E+00
Eagle	3.3E-01	3.7E-01	9.7E-04	1.9E-03

Assumed Cow = CRL * 1

Comparative heron results if Upper Derby and Rod & Gun Club Pond are included.

Ald/Did	DDE/DDT	Endrin	Hg
ne	ne	ne	ne
ne	ne	ne	ne
1.5E+00	1.1E+01	1.8E-02	1.1E+00
ne	ne	ne	ne

RMA IEA/RC 6.94.dr

Figure D.1-16

BDAQTC.XLS

Calculation of Estimated Mean Tissue Concentrations of Bioaccumulative COCs in WaterBird, Shorebird, Heron, and Eagle due to Exposure Through the Aquatic Portion of the RMA Food Web

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

Estimated average doses in RMA bird trophic boxes from exposure through the aquatic food web

dose(j) = SUM(i)(e(j,i) * dose(j,i))

where

dose(j) = estimated average dose in trophic box j due to exposure through the aquatic food web.

SUM(i)(f(i)) = summation over i of f(i).

e(j,i) = weighting factor representing the assumed relative size of trophic box j's feeding area on lake i; lakes included are Upper and Lower Derby, Rod & Gun Club Pond, Ladora, and Mary. SUM(i)(e(j,i)) = 1

dose(j,i) = estimated average dose in trophic box j due to feeding at lake i; calculation method varies for the the four bird trophic boxes.

For water bird, dose(j,i) are field sample averages and the weighting factors are relative water surface areas for the five RMA lakes used to calculate dose(j). Water surface areas are estimated based on November, 1988 data.

dose(j,i) for shorebird are calculated as for water bird with the following exceptions:

1. Weighting factors are relative perimeter areas, where the perimeter is defined as a three foot wide band with outer boundary at the water's edge. The perimeter is estimated based on November, 1988 data.

2. Field data were only available for Lower Derby Lake and Lake Mary. The average of these two lake averages was used to estimate average doses in Upper Derby Lake, Rod & Gun Club Pond, and Lake Ladora.

For heron and eagle:

dose(j,i) = R(j) * SUM(k)(FR(k,j)*TC(k))

R(j) = feeding rate coefficient for trophic box j

FR(k,j) = mass fraction of the diet for predator trophic box j comprised of prey trophic box k

TC(k) = estimated average tissue concentration in prey trophic box k, lake i

Dose(j,i) for heron is only estimated for the three lakes that contain large fish, because large fish comprises 60% of the heron's diet. Estimates based on field data from all five lakes are provided for comparison.

The heron estimates are a function of the dissolved contaminant concentration in the lake water column. Measured OCP Cow's were below certified reporting limits (BCRL). The values reported here assume OCP Cow's equal to the CRL, however the values are insensitive to the choice of Cow over the range of 0 to the CRL; the estimated heron dose contribution from the aquatic food web is the same to at least three significant digits when OCP Cow's are set equal to zero as when they are set equal to the CRL.

Individual lake estimates of heron dose contributed by the aquatic food web

	Ald/Did	DDE/DD	Endrin	Hg
Upper Derby	0	0	0	0
Lower Derby	1.3E-02	1.9E-02	2.0E-03	1.4E-02
Rod & Gun Club	0	0	0	0
Ladora	1.0E-02	1.1E-02	2.1E-03	1.8E-02
Mary	4.4E-03	1.3E-02	1.8E-03	1.2E-02

Comparative heron results if Upper Derby and Rod & Gun Club Pond are included.

Ald/Did	DDE/DDT	Endrin	Hg
3.2E-03	1.4E-03	3.7E-04	2.3E-03
1.3E-02	1.9E-02	2.0E-03	1.4E-02
1.5E-04	2.8E-04	6.7E-05	5.7E-04
1.0E-02	1.1E-02	2.1E-03	1.8E-02
4.4E-03	1.3E-02	1.8E-03	1.2E-02

Individual lake estimates of eagle dose contributed by the aquatic food web

	Ald/Did	DDE/DD	Endrin	Hg
Upper Derby	1.6E-03	4.7E-04	6.5E-05	3.5E-04
Lower Derby	2.8E-03	1.3E-03	5.8E-05	2.6E-04
Rod & Gun Club	3.2E-03	6.5E-04	6.5E-05	6.1E-05
Ladora	5.9E-04	5.9E-04	1.1E-04	1.3E-04
Mary	5.7E-04	6.7E-04	8.3E-05	1.4E-04

Estimated dose contribution from aquatic food web

Trophic Box	Ald/Did	DDE/DD	Endrin	Hg
Waterbird	3.3E-03	2.4E-04	1.9E-03	5.9E-04
Shorebird	4.3E-03	2.0E-02	5.2E-03	1.0E-02
Heron	1.1E-02	1.5E-02	2.0E-03	1.6E-02
Eagle	1.9E-03	6.3E-04	7.8E-05	2.1E-04

Comparative heron results if Upper Derby and Rod & Gun Club Pond are included.

Ald/Did	DDE/DDT	Endrin	Hg
ne	ne	ne	ne
ne	ne	ne	ne
8.1E-03	1.0E-02	1.4E-03	1.1E-02
ne	ne	ne	ne

Assumed Cow = CRL * 1

RMA IEA/RC 6.94.dr

Figure D.1-17

BDAQDOSE.XLS

Calculation of Estimated Mean Doses of Bioaccumulative COCs in WaterBird, Shorebird, Heron, and Eagle due to Exposure Through the Aquatic Portion of the RMA Food Web

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E
1	**MATC (Tissue-Based Criterion) Database**				
2					
3					
4		Ald/Dld	DDE/DDT	Endrin	Hg
5	**Trophic Box**				
6	Waterbird	2.40E-01	1.80E-01	9.00E-02	1.00E-02
7	Shorebird	1.50E-01	1.38E+00	5.00E-02	1.00E-02
8	Heron	8.70E-01	1.50E+01	4.00E-02	1.00E-02
9	Eagle	4.10E-01	2.17E+00	3.00E-02	1.00E-02
10					
11	MATC = maximum allowable tissue concentration (µg chemical/g tissue).				

RMA IEA/RC 6.94.dr

Figure D.1-18

MATC.XLS

Database of Consensus MATC Values for WaterBird, Shorebird, Heron, and Eagle

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E
1	TRV (Dose-Based Criterion) Database				
2					
3					
4		Ald/Dld	DDE/DDT	Endrin	Hg
5	Trophic Box				
6	Waterbird	2.70E-02	4.00E-03	3.00E-03	1.00E-03
7	Shorebird	2.20E-02	8.00E-03	2.00E-03	1.00E-03
8	Heron	2.70E-02	4.00E-03	3.00E-03	1.00E-03
9	Eagle	2.00E-03	5.00E-03	1.00E-03	1.00E-03
10					
11	TRV = toxicity reference value (μg chemical/g tissue*day)				

RMA IEA/RC 6.94.dr

Figure D.1-19

TRV.XLS

Database of Consensus TRV Values for WaterBird, Shorebird, Heron, and Eagle

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	A	B	C	D	E	F
1	**Hazard quotients (HQs) and hazard indicies (HIs) from exposure through the aquatic**					
2	**food web.**					
3						
4		Ald/Dld HQ	DDE/DDT HQ	Endrin HQ	Hg HQ	HI
5	**Trophic Box**					
6	Waterbird	2.87	1.66	0.63	6.75	11.91
7	Shorebird	0.19	2.60	1.17	8.30	12.26
8	Heron	2.28	1.06	0.63	15.63	19.60
9	Eagle	0.93	0.17	0.03	0.21	1.34
10						
11						
12						
13						
14						
15	**Toxicological Criterion Selection**					
16						
17		Ald/Dld	DDE/DDT	Endrin	Hg	
18	**Trophic Box**					
19	Waterbird	MATC	MATC	TRV	MATC	
20	Shorebird	TRV	MATC	MATC	MATC	
21	Heron	MATC	MATC	MATC	TRV	
22	Eagle	TRV	MATC	MATC	TRV	

RMA IEA/RC 6.94.dr

Figure D.1-20 (page 1 of 2)

HQ & HI.XLS

Calculated Hazard Quotients and Hazard Indicies for WaterBird, Shorebird, Heron, and Eagle from Exposure to Bioaccumulative COCs through the Aquatic Portion of the RMA Food Web

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

	G	H	I	J	K	L
3		HQ and HI Formulas				
4		Ald/Did HQ	DDE/DDT HQ	Endrin HQ	Hg HQ	HI
5	Trophic Box					
6	Waterbird	=BDAQTC.XLS!H28/MATC.XLS!B6	=BDAQTC.XLS!I28/MATC.XLS!C6	=BDAQDOSE.XLS!J28/TRV.XLS!D6	=BDAQTC.XLS!K28/MATC.XLS!E6	=SUM(B6:E6)
7	Shorebird	=BDAQDOSE.XLS!H29/TRV.XLS!B7	=BDAQTC.XLS!I29/MATC.XLS!C7	=BDAQTC.XLS!J29/MATC.XLS!D7	=BDAQTC.XLS!K29/MATC.XLS!E7	=SUM(B7:E7)
8	Heron	=BDAQTC.XLS!H30/MATC.XLS!B8	=BDAQTC.XLS!I30/MATC.XLS!C8	=BDAQTC.XLS!J30/MATC.XLS!D8	=BDAQDOSE.XLS!K30/TRV.XLS!E8	=SUM(B8:E8)
9	Eagle	=BDAQDOSE.XLS!H31/TRV.XLS!B9	=BDAQTC.XLS!I31/MATC.XLS!C9	=BDAQTC.XLS!J31/MATC.XLS!D9	=BDAQDOSE.XLS!K31/TRV.XLS!E9	=SUM(B9:E9)

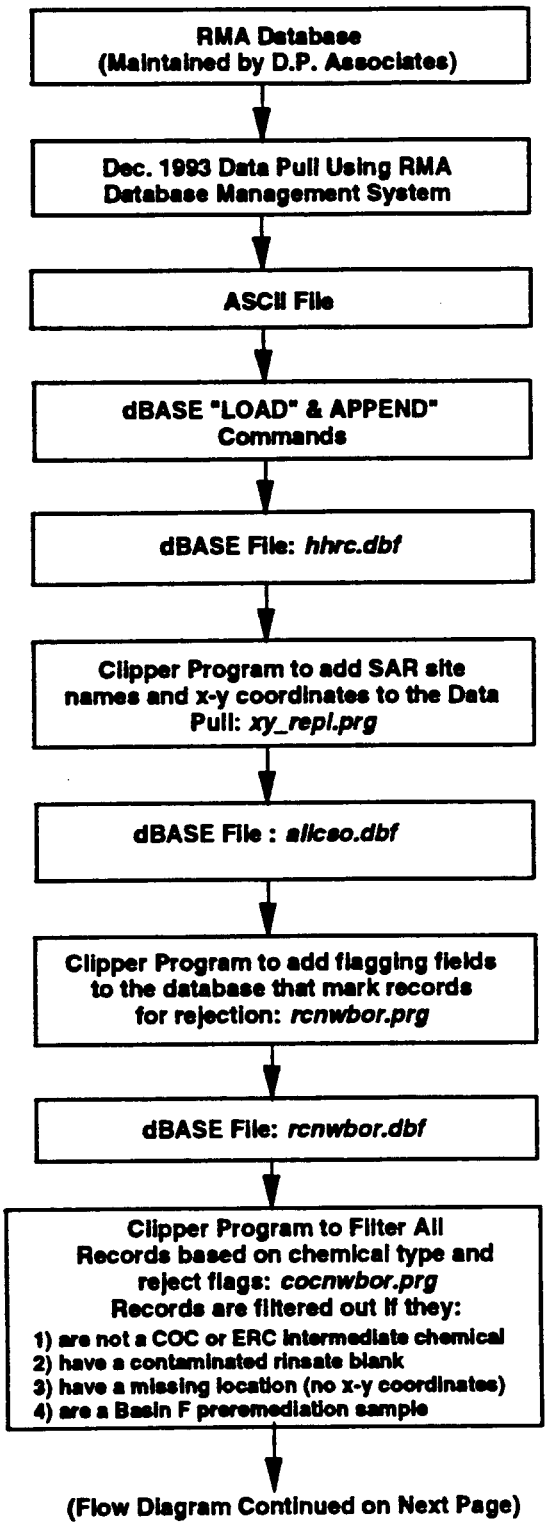
RMA IEA/RC 6.94.dr

Figure D.1-20 (page 2 of 2)

HQ & HI.XLS

Calculated Hazard Quotients and Hazard Indicies for WaterBird, Shorebird, Heron, and Eagle from Exposure to Bioaccumulative COCs through the Aquatic Portion of the RMA Food Web

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated



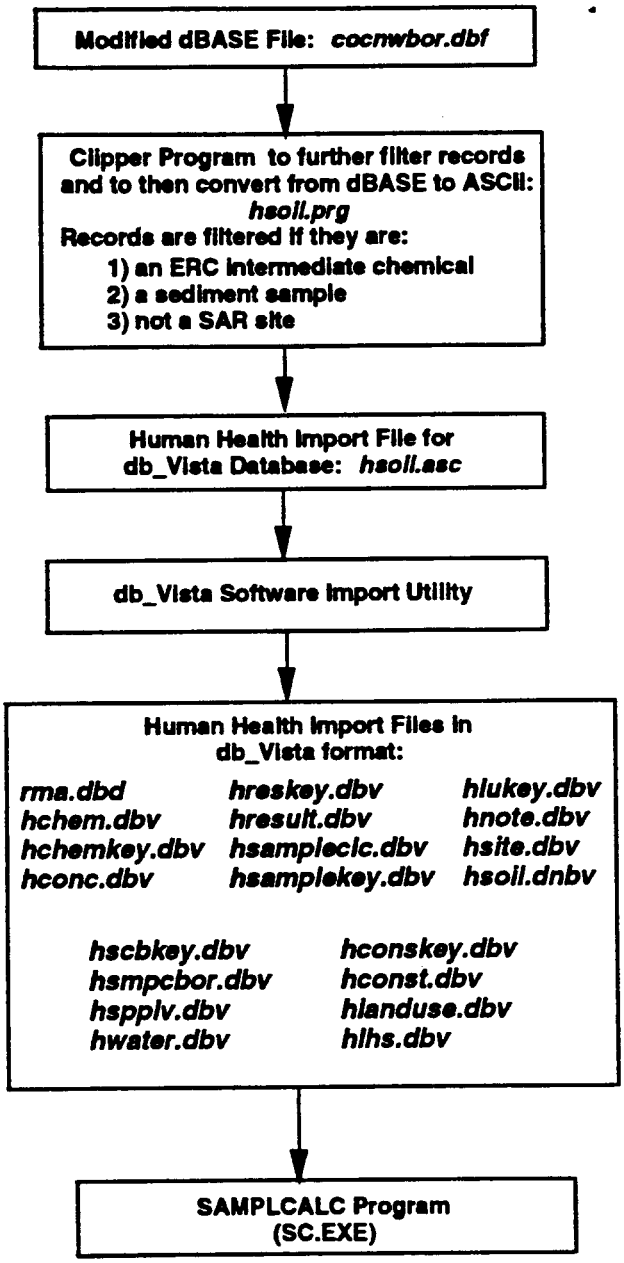
RMA IEA/RC D 2.94.jb

Figure D.1-21

Flow Diagram for the Database Preprocessing System for the Human Health Code
Page 1 of 2

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

(Flow Diagram continued from previous page)

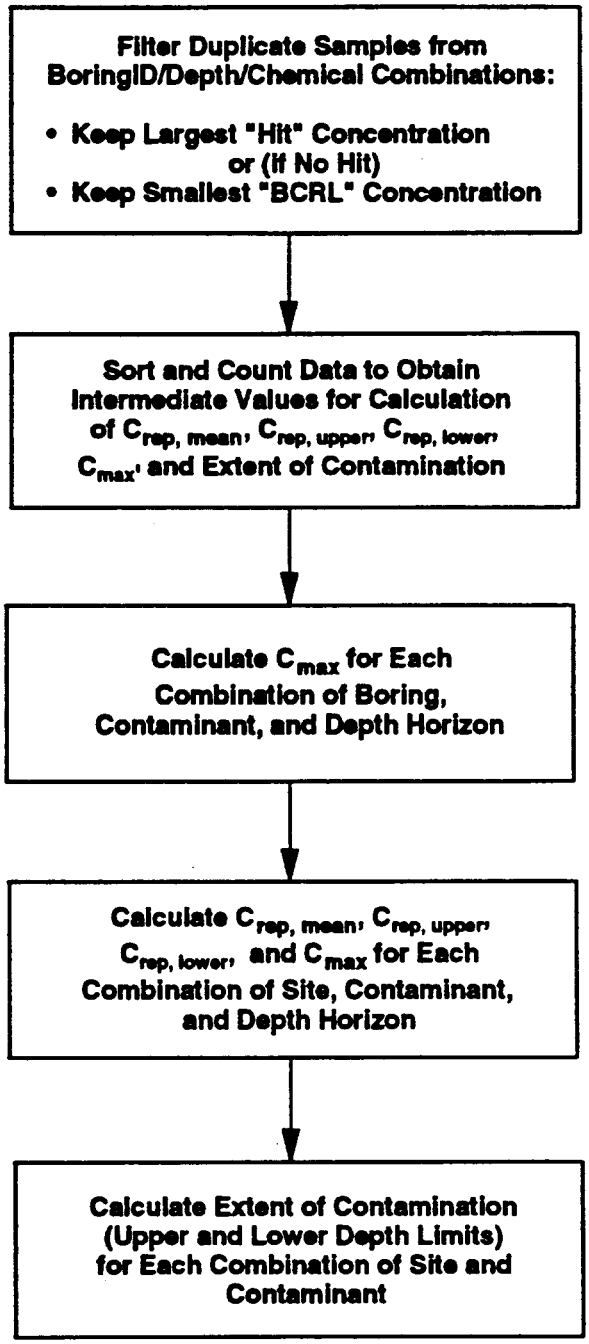


RMA IEA/RC D 2.94.jb

Figure D.1-21

Flow Diagram for the Database Preprocessing System for the Human Health Code
Page 2 of 2

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

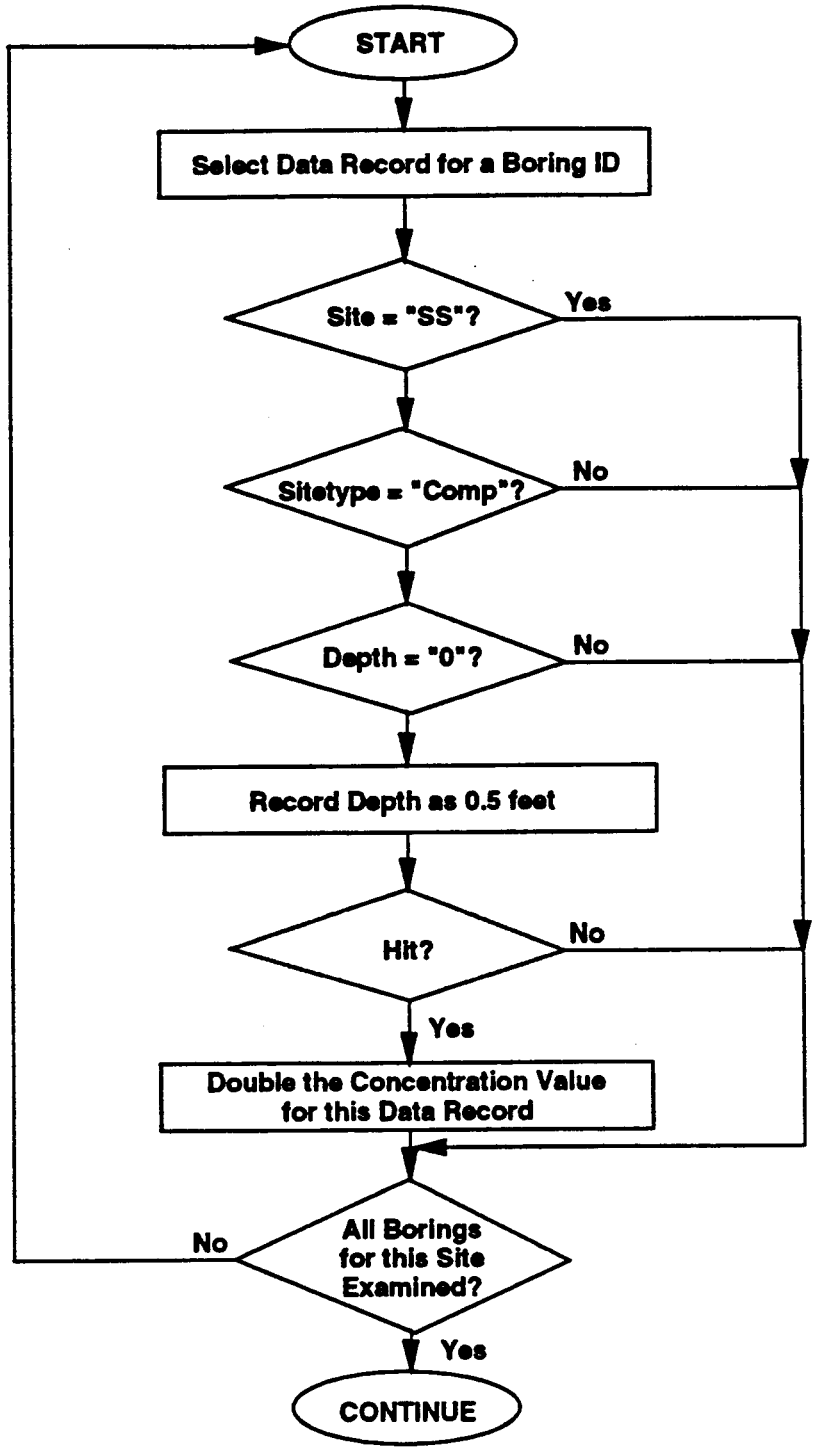


RMA IEA/RC D 2.94 jb

Figure D.1-22

Overview of HHRC SampleCalc Program (SC.EXE)

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

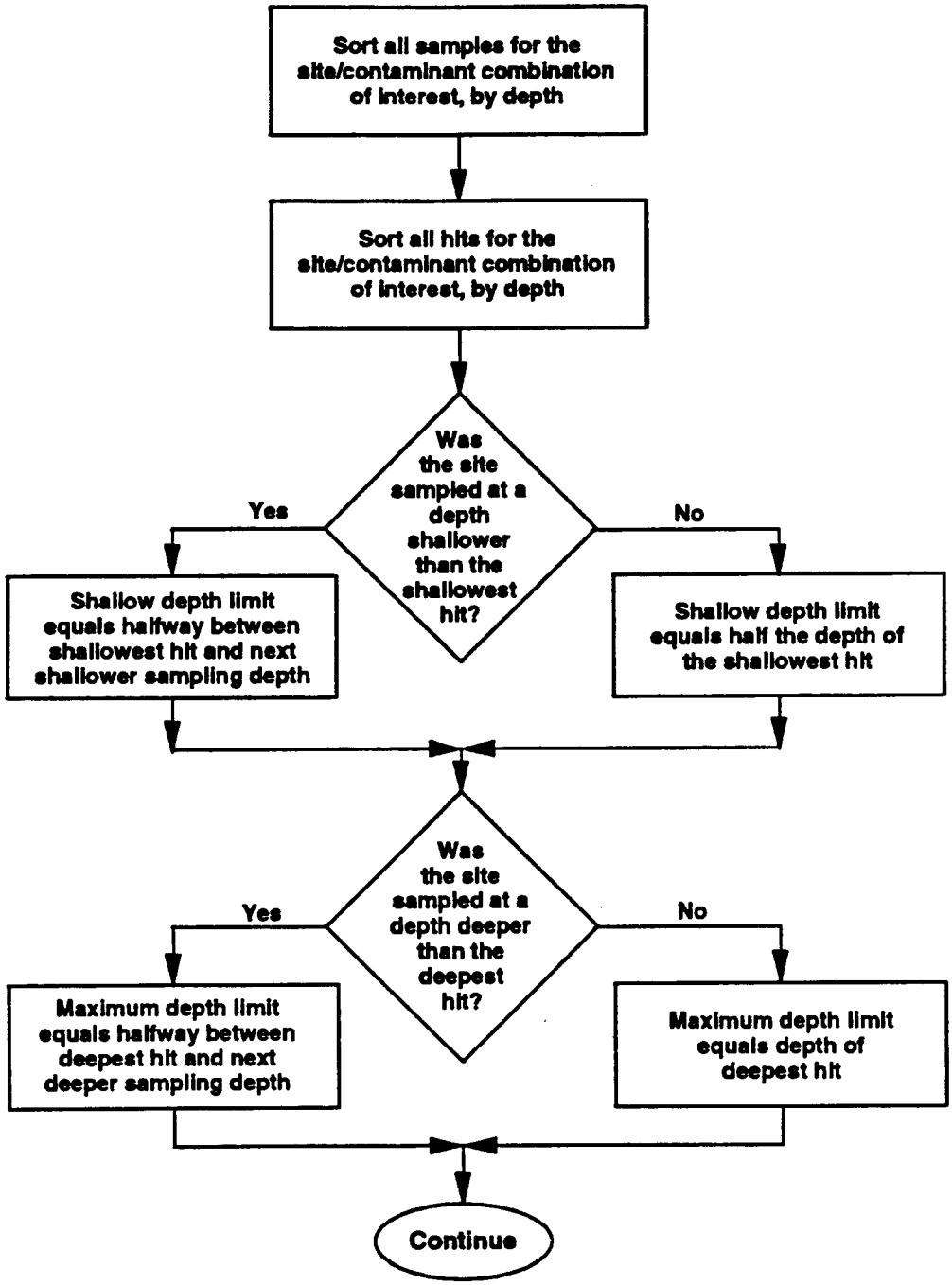


RMA IEA/RC D 2.94.jb

Figure D.1-23

SampleCalc Adjustment Method for Composite Borings

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated



RMA IEA/RC D 2.94.jb

Figure D.1-24

Estimating Vertical Extent of Contamination

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

ATTACHMENT D-1

HUMAN HEALTH RISK CHARACTERIZATION EQUATIONS

Direct PPLV Equations:

I Regulated/Casual and Recreational Visitors

Soil Ingestion (NonCarc)

(1)

$$\text{Intermediate Intake (II)} \left(\frac{\text{kg-day}}{\text{mg}}\right) = \sum_{i=1}^{\text{Max Age}} \frac{\text{Bodyweight}_i\text{(kg)}}{\text{Soil Ingestion Rate}_i\text{(mg/day)}}$$

(2)

$$\text{SPPPLV}_{\text{Ingestion (NonCarc)}}\ \text{(mg/kg)} = \text{DTING}_{\text{NonCarc}} \left(\frac{\text{mg}}{\text{kg-day}}\right) * \frac{\text{II} \dfrac{\dfrac{\text{(kg-day)}}{\text{mg}}}{\text{Max Age}} * 10^6\ \text{(mg/kg)}}{\text{RAF}_{\text{Oral (NonCarc)}} * \dfrac{\text{DW (day/yr)}}{\text{365 (day/yr)}} * \dfrac{\text{TM (hours/day)}}{\text{8 (hours/day)}}}$$

Soil Ingestion (Carc)

(3)

$$\text{Intermediate Intake (II)}\ \frac{\text{(kg-day)}}{\text{mg}} = \sum_{i=1}^{\text{Max Age}} \frac{\text{Bodyweight}_i\ \text{(kg)}}{\text{Soil Ingestion Rate}_i\ \text{(mg/day)}}$$

(4)

$$\text{SPPPLV}_{\text{Ingestion Carc}}\ \text{(mg/kg)} = \text{DTING}_{\text{Carc}}\ \left(\frac{\text{mg}}{\text{kg-day}}\right) * \frac{\dfrac{\text{II}\ \dfrac{\text{(kg-day)}}{\text{mg}}}{\text{Max Age}} * 10^6\ \text{(mg/kg)}}{\text{RAF}_{\text{Oral (Carc)}} * \dfrac{\text{DW (day/yr)}}{\text{365 (day/yr)}} * \dfrac{\text{TE (yr)}}{\text{70 (yr)}} * \dfrac{\text{TM (hours/day)}}{\text{8 (hours/day)}}}$$

Soil Inhalation (NonCarc)

(5)

$$\text{Intermediate Intake (II)} \left(\frac{\text{kg-hr}}{\text{m}^3}\right) = \sum_{i=i}^{\text{Max Age}} \frac{\text{Bodyweight}_i \text{ (kg)}}{\text{Breathing Rate}_i \text{ (m}^3\text{/hr)}}$$

(6)

$$\text{SPPPLV}_{\text{Inhalation (NonCarc)}} \left(\frac{\text{mg}}{\text{kg}}\right) = \text{DTINH}_{\text{NonCarc}} \left(\frac{\text{mg}}{\text{kg-day}}\right) * \frac{\frac{\text{II} \left(\frac{\text{kg-hr}}{\text{m}^3}\right)}{\text{Max Age}} * 10^6 \text{ (mg/kg)}}{\text{CSS} \left(\frac{\text{mg}}{\text{m}^3}\right) * \text{TM} \left(\frac{\text{hr}}{\text{day}}\right) * \text{FR} * \frac{\text{DW (day/yr)}}{365 \text{ (day/yr)}}}$$

Soil Inhalation (Carc)

(7)

$$\text{Intermediate Intake (II)} \left(\frac{\text{kg-hr}}{\text{m}^3}\right) = \sum_{i=i}^{\text{Max Age}} \frac{\text{Bodyweight}_i \text{ (kg)}}{\text{Breathing Rate}_i \text{ (m}^3\text{/hr)}}$$

(8)

$$\text{SPPPLV}_{\text{Inhalation (Carc)}} \text{ (mg/kg)} =$$

$$\text{DTINH}_{\text{Carc}} \left(\frac{\text{mg}}{\text{kg-day}}\right) * \frac{\frac{\text{II}\left(\frac{\text{kg-hr}}{\text{m}^3}\right)}{\text{Max Age}} * 10^6 \text{ (mg/kg)}}{\text{CSS} \left(\frac{\text{mg}}{\text{m}^3}\right) * \text{TM} \left(\frac{\text{hr}}{\text{day}}\right) * \text{FR} * \frac{\text{DW (day/yr)}}{365 \text{ (day/yr)}} * \frac{\text{TE (yr)}}{70 \text{ (yr)}}}$$

Soil Dermal Contact (NonCarc)

(9)

$$\text{Intermediate Intake (II)} \left(\frac{\text{kg-day}}{\text{mg}}\right) = \sum_{i=i}^{\text{Max Age}} \frac{\text{Bodyweight}_i \text{ (kg)}}{\text{Skin Surface Area}_i \text{ (cm}^2\text{)} * \text{Soil Covering}_i \left(\frac{\text{mg}}{\text{cm}^2\text{-day}}\right)}$$

(10)

$$\text{SPPLV}_{\text{Dermal (NonCarc)}} \left(\frac{\text{mg}}{\text{kg}}\right) = \text{DTING}_{\text{NonCarc}} \left(\frac{\text{mg}}{\text{kg-day}}\right) * \frac{\frac{\text{II} \left(\frac{\text{kg-day}}{\text{mg}}\right)}{\text{Max Age}} * 10^6 \left(\frac{\text{mg}}{\text{kg}}\right)}{\text{RAF}_{\text{Dermal (NonCarc)}} * \frac{\text{DW (day/yr)}}{365 \text{ (day/yr)}}}$$

Soil Dermal Contact (Carc)

(11)

$$\text{Intermediate Intake (II)} \left(\frac{\text{kg-day}}{\text{mg}}\right) = \sum_{i=i}^{\text{Max Age}} \frac{\text{BodyWeight}_i \text{ (kg)}}{\text{Skin Surface Area}_i \text{ (cm}^2\text{)} * \text{Soil Covering}_i \left(\frac{\text{mg}}{\text{cm}^2\text{-day}}\right)}$$

(12)

$$\text{SPPPLV}_{\text{Dermal (Carc)}} \text{ (mg/kg)} = \text{DTING}_{\text{Carc}} \left(\frac{\text{mg}}{\text{kg-day}}\right) * \frac{\frac{\text{II}\left(\frac{\text{kg-day}}{\text{mg}}\right)}{\text{Max Age}} * 10^6 \text{ (mg/kg)}}{\text{RAF}_{\text{Dermal (Carc)}} * \frac{\text{DW (day/yr)}}{365 \text{ (day/yr)}} * \frac{\text{TE (yr)}}{70 \text{ (yr)}}}$$

II Commercial, Industrial, Biological Worker

Soil Ingestion (NonCarc)

(13)

$$SPPLV_{Ingestion\ (NonCarc)}\left(\frac{mg}{kg}\right) = \frac{DTING_{NonCarc}\left(\frac{mg}{kg\text{-}day}\right) * BW(kg) * 10^6\left(\frac{mg}{kg}\right)}{DING\left(\frac{mg}{day}\right) * RAF_{Oral\ (NonCarc)} * \frac{DW\ (day/yr)}{365\ (day/yr)}}$$

Soil Ingestion (Carc)

(14)

$$SPPPLV_{Ingestion\ (Carc)}\left(\frac{mg}{kg}\right) = \frac{DTING_{Carc}\left(\frac{mg}{kg\text{-}day}\right) * BW\ (kg) * 10^6\left(\frac{mg}{kg}\right)}{DING\left(\frac{mg}{day}\right) * RAF_{Oral\ (Carc)} * \frac{DW\ (day/yr)}{365\ (day/yr)} * \frac{TE\ (yr)}{70\ (yr)}}$$

Soil Inhalation (NonCarc)

(15)

$$SPPPLV_{Inhalation\ (NonCarc)}\left(\frac{mg}{kg}\right) = \frac{DTINH_{NonCarc}\left(\frac{mg}{kg\text{-}day}\right) * BW(kg) * 10^6\left(\frac{mg}{kg}\right)}{CSS\left(\frac{mg}{m^3}\right) * FR * BR\left(\frac{m^3}{hr}\right) * \frac{DW(day/yr)}{365(day/yr)} * TM\frac{(hr)}{day}}$$

Soil Inhalation (Carc)

(16)

$$SPPPLV_{Inhalation\ (Carc)}\left(\frac{mg}{kg}\right) = \frac{DTINH_{Carc}\left(\frac{mg}{kg\text{-}day}\right) * BW\ (kg) * 10^6\left(\frac{mg}{kg}\right)}{CSS\left(\frac{mg}{m^3}\right) * FR * BR\left(\frac{m^3}{hr}\right) * \frac{DW\ (day/yr)}{365\ (day/yr)} * TM\ \frac{(hr)}{day} * \frac{TE\ (yr)}{70\ (yr)}}$$

Dermal Contact (NonCarc)

(17)

$$SPPPLV_{\text{Dermal (NonCarc)}} \left(\frac{mg}{kg}\right) =$$

$$\frac{DTING_{\text{NonCarc}} \left(\frac{mg}{kg\text{-day}}\right) * BW\ (kg) * 10^6 \left(\frac{mg}{kg}\right)}{\text{Skin Surface Area } (cm^2) * \text{Soil Covering } \left(\frac{mg}{cm^2\text{-day}}\right) * RAF_{\text{Dermal (NonCarc)}} * \frac{DW\ (day/yr)}{365\ (day/yr)}}$$

Dermal Contact (Carc)

(18)

$$SPPPLV_{\text{Dermal (Carc)}} \left(\frac{mg}{kg}\right) =$$

$$\frac{DTING_{\text{Carc}} \left(\frac{mg}{kg\text{-day}}\right) * BW\ (kg) * 10^6 \left(\frac{mg}{kg}\right)}{\text{Skin Surface Area } (cm^2) * \text{Soil Covering } \left(\frac{mg}{cm^2\text{-day}}\right) * RAF_{\text{Dermal (Carc)}} * \frac{DW\ (day/yr)}{365\ (day/yr)} * \frac{TE\ (yr)}{70\ (yr)}}$$

Open Space Vapor

(19)

$$Fcrit_{NonCarc}\ \left(\frac{mg}{m^2\text{-day}}\right) =$$

$$\frac{DTINH_{NonCarc}\ \left(\frac{mg}{kg\text{-day}}\right) * BW\ (kg)}{DINH\ \left(\frac{m^3}{hr}\right) * \left(\frac{X_d\ \left(\frac{mg}{m^3}\right)}{F_o\ \left(\frac{mg}{m^2\text{-day}}\right)}\right) * FR * \frac{DW\ (day/yr)}{365\ (day/yr)} * TM\ \left(\frac{hr}{day}\right)}$$

(20)

$$Fcrit_{Carc}\ \left(\frac{mg}{m^2\text{-day}}\right) =$$

$$\frac{DTINH_{Carc}\ \left(\frac{mg}{kg\text{-day}}\right) * BW\ (kg)}{DINH\ \left(\frac{m^3}{hr}\right) * \left(\frac{X_d\ \left(\frac{mg}{m^3}\right)}{F_o\ \left(\frac{mg}{m^2\text{-day}}\right)}\right) * FR * \frac{DW\ (day/yr)}{365\ (day/yr)} * TM\ \left(\frac{hr}{day}\right) * \frac{TE\ (yr)}{70\ (yr)}}$$

Calculation of FAVN:

(21)

$$D\ (\frac{cm^2}{sec}) = D_i\ (\frac{cm^2}{sec}) * \left(\frac{P_a^{10/3}}{P_T^{\ 2}}\right) * \frac{H\ (\frac{atm-m^3}{mole})}{R\ (\frac{atm-m^3}{mole-°K}) * T\ (°K)}$$

(22)

$$C_B\ (\frac{g}{cm^3}) = C_{Soil}\ (\frac{mg}{kg}) * \rho\ (\frac{g}{cm^3}) * 10^{-6}\ (\frac{kg}{mg})$$

(23)

$$C_W\ (\frac{g}{cm^3}) = \frac{C_B\ (\frac{g}{cm^3})}{\left(Koc\ (\frac{l}{kg}) * foc * \rho\ (\frac{g}{cm^3})\right) + \theta}$$

(24)

$$t_d\ (\text{sec}) = \frac{(h\ (\text{cm})^2 - d\ (\text{cm})^2)}{2D\ (\frac{\text{cm}^2}{\text{sec}})} * \frac{C_B\ (\frac{\text{g}}{\text{cm}^3})}{C_W\ (\frac{\text{g}}{\text{cm}^3})}$$

(25)

$$t_e\ (\text{sec}) = TE\ (\text{yrs}) * 3.15E + 07\ (\frac{\text{sec}}{\text{yr}})$$

(26)

$$C_g\ (\frac{\text{g}}{\text{cm}^3}) = \frac{H(\frac{\text{atm-m}^3}{\text{mole}}) * C_W\ (\frac{\text{g}}{\text{cm}^3})}{R\ (\frac{\text{atm-m}^3}{\text{mole-°K}}) * T\ (\text{°K})}$$

(27)

$$C_{sg}\ (\frac{g}{cm^3}) = \frac{P_{VP}\ (mm\ Hg) * MW\ (\frac{g}{mole})}{R\ (\frac{atm-m^3}{mole-°K}) * T\ (°K) * 760\ (\frac{mm\ Hg}{atm}) * 10^6\ (\frac{cm^3}{m^3})}$$

If $C_g > C_{sg}$ (soil air space is saturated) then:

set $C_g = C_{sg}$ and recalcualte $C_W \rightarrow C_{W,Sat}$

(28)

$$C_{W,Sat}\ (\frac{g}{cm^3}) = \frac{C_{sg}\ (\frac{g}{cm^3}) * R\ (\frac{atm-m^3}{mole-°K}) * T\ (°K)}{H\ (\frac{atm-m^3}{mole})}$$

(29)

$$K = 10^3\ (\frac{mg}{g}) * 10^4\ (\frac{cm^2}{m^2}) * 8.64\ E{+}4\ (\frac{sec}{day}) = 8.64\ E{+}11\ (\frac{mg-cm^2-sec}{g-m^2-day})$$

If $t_d > t_e$, then:

calculate FAVN Long Term

(30)

$$FAVN = FAVN_{LT}\left(\frac{mg}{m^2\text{-day}}\right) = \frac{K * C_B\left(\frac{g}{cm^3}\right)}{t_e(sec)} * \left[\left[(d(cm)^2 + 2 * t_e(sec) * \frac{C_w\left(\frac{g}{cm^3}\right)}{C_B\left(\frac{g}{cm^3}\right)} * D\left(\frac{cm^2}{sec}\right)\right]^{1/2} - d(cm)\right]$$

if $\frac{C_w}{C_B} \approx 0$, **then:**

calculate the limiting value of FAVN Long Term as $C_B \rightarrow \infty$

(30a)

$$FAVN = FAVN_{LT}\left(\frac{mg}{m^2\text{-day}}\right) = \frac{K * D\left(\frac{cm^2}{s}\right) * C_w}{d\ (cm)}$$

otherwise calculate FAVN Mass Balance

(31)

$$FAVN = FAVN_{MB}\left(\frac{mg}{m^2\text{-day}}\right) = \frac{K * (h\ (cm) - d\ (cm)) * C_B\left(\frac{g}{cm^3}\right)}{t_e\ (sec)}$$

(32)

$$SPPPLV_{NonCarc}\left(\frac{mg}{kg}\right) = \frac{Fcrit_{NonCarc}\left(\frac{mg}{m^2\text{-day}}\right)}{FAVN\left(\frac{mg}{m^2\text{-day}}\right)} * C_{soil}\left(\frac{mg}{kg}\right)$$

(33)

$$SPPPLV_{Carc}\left(\frac{mg}{kg}\right) = \frac{Fcrit_{Carc}\left(\frac{mg}{m^2\text{-day}}\right)}{FAVN\left(\frac{mg}{m^2\text{-day}}\right)} * C_{soil}\left(\frac{mg}{kg}\right)$$

Enclosed Space Vapor Models

(34)

$$VAR\ (m) = \frac{VOL\ (m^3)}{AREA\ (m^2)} = \frac{\ell\ (m) * w\ (m) * 3\ (m)}{\ell\ (m) * w\ (m) + 4\ (m)\ (\ell\ (m) + w\ (m))}$$

(35)

$$TAC\ (day) = \frac{3\ (m) * \ell\ (m) * w\ (m) * 10^6\ (\frac{cm^3}{m^3})}{8.64\ E{+}4\ (\frac{sec}{day}) * Q_A\ (\frac{cm^3}{sec})}$$

(36)

$$Fcrit_{NonCarc}\ (\frac{mg}{m^2\text{-}day}) =$$

$$\frac{DTINH_{NonCarc}\ (\frac{mg}{kg\text{-}day}) * BW\ (kg) * VAR\ (m)}{DINH\ (\frac{m^3}{hr}) * FR * TAC\ (day) * \frac{DW\ (day/yr)}{365\ (day/yr)} * TM\ (\frac{hr}{day}) * FRHRS}$$

(37)

$$Fcrit_{Carc}\ \left(\frac{mg}{m^2\text{-day}}\right) =$$

$$\frac{DTINH_{Carc}\ \left(\frac{mg}{kg\text{-day}}\right) * BW\ (kg) * VAR\ (m)}{DINH\ \left(\frac{m^3}{hr}\right) * FR * TAC\ (day) * \frac{DW\ (day/yr)}{365\ (day/yr)} * \frac{TE\ (yr)}{70\ (yr)} * TM\ \left(\frac{hr}{day}\right) * FRHRS}$$

(38)

$$C_B\ \left(\frac{g}{cm^3}\right) = C_{soil}\ \left(\frac{mg}{kg}\right) * \rho\ \left(\frac{g}{cm^3}\right) * 10^{-6}\ \left(\frac{kg}{mg}\right)$$

(39)

$$C_W\ \left(\frac{g}{cm^3}\right) = \frac{C_B\ \left(\frac{g}{cm^3}\right)}{\theta + \left(Koc\ \left(\frac{l}{kg}\right) * foc * \rho\ \left(\frac{g}{cm^3}\right)\right)}$$

(40)

$$C_g \left(\frac{g}{cm^3}\right) = \frac{C_W \left(\frac{g}{cm^3}\right) * H\left(\frac{atm\text{-}m^3}{mole}\right)}{R \left(\frac{atm\text{-}m^3}{mole\text{-}°K}\right) * T\ (°K)}$$

(41)

$$C_{sg} \left(\frac{g}{cm^3}\right) = \frac{P_{vp}\ (mm\ Hg) * MW \left(\frac{g}{mole}\right)}{R \left(\frac{atm\text{-}m^3}{mole\text{-}°K}\right) * T\ (°K) * 760 \left(\frac{mm\ Hg}{atm}\right) * 10^6 \left(\frac{cm^3}{m^3}\right)}$$

If $C_g > C_{sg}$ (soil air space is saturated) then:

set $C_g = C_{sg}$ and recalculate $C_W \rightarrow C_{W,Sat}$

(42)

$$C_{W,Sat} \left(\frac{g}{cm^3}\right) = \frac{C_{sg} \left(\frac{g}{cm^3}\right) * R \left(\frac{atm\text{-}m^3}{mole\text{-}°K}\right) * T\ (°K)}{H \left(\frac{atm\text{-}m^3}{mole}\right)}$$

(43)

$$H' = \frac{H\ (\frac{atm\text{-}m^3}{mole})}{R\ (\frac{atm\text{-}m^3}{mole\text{-}^\circ K}) * T\ (^\circ K)}$$

(44)

$$\frac{Rg = \left[Koc\ (\frac{l}{kg}) * foc * \rho\ (\frac{g}{cm^3})\right] + \theta}{H'}$$

(45)

$$A\ (cm^2) = \left(\left[4(m) * (\ell(m) + w(m))\right] + \ell(m) * w(m)\right) * 10^4\ (\frac{cm^2}{m^2})$$

(46)

$$V_s\ (cm^3) = \left[8(m^2) * (\ell(m) + w(m)) + 1(m) * \ell(m) * w(m)\right] * 10^6(\frac{cm^3}{m^3})$$

(47)

$$T_z(\text{sec}) = \frac{Rg * Vs\ (cm^3)}{f * Q_A\ (\frac{cm^3}{sec})}$$

(48)

$$T_d(\text{sec}) = \left[\frac{Rg * Vs\ (cm^3)}{f * Q_A\ (\frac{cm^3}{sec})}\right] * \ln\left[\frac{f * Q_A\ (\frac{cm^3}{sec}) * 100\ (cm)}{A\ (cm^2) * D\ (\frac{cm^2}{sec})}\right]$$

(49)

$$t_d\ (\text{sec}) = \frac{h'^2(m^2) - d'^2(m^2)}{2D\ (\frac{cm^2}{sec})} * \frac{C_B\ (\frac{g}{cm^3})}{C_W\ (\frac{g}{cm^3})}$$

(50)

$$K = 10^3 \left(\frac{mg}{g}\right) * 10^4 \left(\frac{cm^2}{m^2}\right) * 8.64E4 \left(\frac{sec}{day}\right) = 8.64E11 \left(\frac{mg\text{-}cm^2\text{-}sec}{g\text{-}m^2\text{-}day}\right)$$

If d'(cm) ≤ 100.0 (cm) and 0 (sec) < T_d (sec) < t_e (sec), then:

d'(cm) = 100 (cm)

(51)

$$FAVN(2) \left(\frac{mg}{m^2 - day}\right) = K * \frac{T_z\ (sec)}{T_d\ (sec)} * \left[\frac{f * Q_A \left(\frac{cm^3}{sec}\right)}{Rg * A\ (cm^2)}\right] * C_B \left(\frac{g}{cm^3}\right) * \left[1 - e \left(\frac{-T_d\ (sec)}{T_z\ (sec)}\right)\right]$$

If t_d < t_e then:

(52)

$$FAVN(1) = FAVN(1)_{MB} \left(\frac{mg}{m^2 - day}\right) = \frac{K * (h'(cm) - d'(cm)) * C_B \frac{g}{cm^3}}{(t_e\ (sec) - T_d\ (sec))}$$

Otherwise:

(53)

$$FAVN(1) = FAVN(1)_{LT}\ (\frac{mg}{m^2\text{-day}}) =$$

$$K * \frac{C_B\ (\frac{g}{cm^3})}{(t_e\ (sec) - T_{d\ (sec)})} * \left[\left(d'(cm)^2 + \frac{2 * (t_e\ (sec) - T_d\ (sec)) * D(\frac{cm^2}{sec}) * C_W(\frac{g}{cm^3})}{C_B\ (\frac{g}{cm^3})}\right)^{\frac{1}{2}} - d'(cm)\right]$$

If $\frac{C_W}{C_B} \approx 0$, **then:**

calculate the limiting value of $FAVN(1)_{LT}$ as $C_B \rightarrow \infty$

(53a)

$$FAVN(1) = FAVN(1)_{LT} = \frac{K * D\ (\frac{cm^2}{s}) * C_W\ (\frac{g}{cm^3})}{d'(cm)}$$

(54)

$$FAVN\ (\frac{mg}{m^2\text{-}day}) = (\frac{T_d\ (sec)}{t_e\ (sec)}) * FAVN(2)\ (\frac{mg}{m^2\text{-}day}) + (1 - \frac{T_d\ (sec)}{t_e\ (sec)}) * FAVN(1)\ (\frac{mg}{m^2\text{-}day})$$

If $d'(cm) \leq 100(cm)$ and $T_d(sec) \geq t_e\ (sec)$ then:

(55)

$$FAVN(2)\ (\frac{mg}{m^2\text{-}day}) = K * \frac{T_z\ (sec)}{t_e\ (sec)} * \left(\frac{f * Q_a\ (\frac{cm^3}{sec})}{Rg * A\ (cm^2)}\right) * C_B\ (\frac{g}{cm^3}) * \left[1 - e\ (\frac{-t_e\ (sec)}{T_z\ (sec)})\right]$$

(56)

$$FAVN\ (\frac{mg}{m^2\text{-day}}) = FAVN(2)\ (\frac{mg}{m^2\text{-day}})$$

If d'(cm) > 100 (cm) or T_d (sec) $\leq$ 0, then:

d'(cm) = 100 (cm)

If $t_d > t_e$ then:

(57)

$$FAVN(1) = FAVN(1)_{LT}\ (\frac{mg}{m^2\text{-day}}) =$$

$$K * \frac{C_B\ (\frac{g}{cm^3})}{t_e\ (sec)} * \left[\left(d'(cm)^2 + \frac{2 * t_e\ (sec) * D\ (\frac{cm^2}{sec}) * C_W\ (\frac{g}{cm^3})}{C_B\ (\frac{g}{cm^3})}\right)^{\frac{1}{2}} - d'(cm)\right]$$

If $\frac{C_W}{C_B} \approx 0$, then:

calculate the limiting value of $FAVN(1)_{LT}$ as $C_B \rightarrow \infty$

(57a)

$$FAVN(1) = FAVN(1)_{LT} = \frac{K * D\ (\frac{cm^2}{s}) * C_W\ (\frac{g}{cm^3})}{d'(cm)}$$

Otherwise calculate FAVN(1) mass balance:

(58)

$$FAVN(1) = FAVN(1)_{MB} = \frac{K * (h'(cm) - d'(cm)) * C_B\ (\frac{g}{cm^3})}{t_e\ (sec)}$$

(59)

$$FAVN\ (\frac{mg}{m^2\text{-}day}) = FAVN(1)\ (\frac{mg}{m^2\text{-}day})$$

(60)

$$SPPPLV_{NonCarc}\ (\frac{mg}{kg}) = \frac{Fcrit_{NonCarc}\ (\frac{mg}{m^2\text{-}day})}{FAVN\ (\frac{mg}{m^2\text{-}day})} * C_{soil}\ (\frac{mg}{kg})$$

(61)

$$SPPPLV_{Carc}\ (\frac{mg}{kg}) = \frac{Fcrit_{Carc}\ (\frac{mg}{m^2\text{-}day})}{FAVN\ (\frac{mg}{m^2\text{-}day})} * C_{soil}\ (\frac{mg}{kg})$$

APPENDIX E

ELABORATION ON UNCERTAINTY IN THE HUMAN HEALTH AND ECOLOGICAL RISK EVALUATIONS

TABLE OF CONTENTS

TABLE OF CONTENTS
(continued)

TABLE OF CONTENTS
(continued)

TABLE OF CONTENTS
(continued)

LIST OF TABLES

LIST OF FIGURES

Figure

LIST OF ATTACHMENTS

LIST OF ACRONYMS AND ABBREVIATIONS

ANL	Argonne National Laboratory
Army agent	Army chemical warfare agent
BAF	bioaccumulation factor
BC	probabilistic biota criteria
BCF	bioconcentration factor
BCRL	below certified reporting limit
BMF	biomagnification factor
C_{max}	maximum site concentration
CHO	chinese hamster ovary
CI	confidence interval
CMP	Comprehensive Monitoring Program
COC	contaminant of concern
CPMS	chlorophenylmethyl sulfide
$CPMSO_2$	chlorophenylmethyl sulfone
C_{rep}	representative site concentration
CRL	certified reporting limit
DBCP	dibromochloropropane
DBC	probabilistic biota criteria (dose-based)
DDE	dichlorodiphenyldichloroethene
DDT	dichlorodiphenyltrichloroethane
DNA	deoxyribonucleic acid
DT	critical toxicity value
EA	Endangerment Assessment
EPA	U.S. Environmental Protection Agency
ERC	ecological risk characterization
ESC	exposure area soil concentration
<ESC>	estimated exposure area soil concentration
EVR	expected value robust
FFA	Federal Facility Agreement
ft	foot/feet
GC/MS	gas chromatography/mass spectrometry
HCCPD	hexachlorocyclopentadiene
HEAST	EPA Health Effects Assessment Summary Tables
HHEA	Human Health Exposure Assessment
HHRC	human health risk characterization
HI	hazard index
HQ	hazard quotient
IARC	International Agency for Research on Cancer
IEA/RC	Integrated Endangerment Assessment/Risk Characterization
IRIS	EPA Integrated Risk Information System
K_{oc}	soil-water equilibrium partitioning coefficient normalized to organic carbon
LCL	lower confidence limit
MATC	maximum allowable tissue concentration
mg/kg	milligrams per kilogram
MKE	Morrison-Knudsen Engineering

LIST OF ACRONYMS AND ABBREVIATIONS

(continued)

µg/g	micrograms per gram
NTP	National Toxicology Program
OAS	Organizations and State
PBC	probabilistic biota criteria (tissue based)
PMRMA	Program Manager for the Rocky Mountain Arsenal
PPLV	preliminary pollutant limit value
ppm	parts per million
PSCo	Public Service Company of Colorado
RAF	relative absorption factor
RfD	reference dose
RI	Remedial Investigation
RMA	Rocky Mountain Arsenal
SAR	Study Area Report
SC	soil concentration
Shell	Shell Oil Company
SMR	Standardized Mortality Ratio
SPPPLV	single pathway preliminary pollutant limit value
TC	biota tissue concentration
the Army	U.S. Department of the Army
TRV	toxicity reference value
UCL	upper confidence limit
UF	uncertainty factor
USATHAMA	U.S. Army Toxic and Hazardous Materials Agency
USFWS	U.S. Fish and Wildlife Service
WHO	World Health Organization

E.1 RATIONALE FOR UNCERTAINTY ANALYSIS

Sources of uncertainty and variability were identified and analyzed for both the human health risk characterization (HHRC) and ecological risk characterization (ERC) completed as part of the Rocky Mountain Arsenal (RMA) Integrated Endangerment Assessment/Risk Characterization (IEA/RC). Model parameter distributions were developed based on empirical data, and in instances where empirical data are lacking, best professional judgment was incorporated. In addition, when uncertainty in the empirical data for a given parameter warranted conservative assumptions, these assumptions were incorporated into the parameter distribution.

The following three examples illustrate how explicit consideration of uncertainty has benefitted RMA risk characterization in a number of ways. First, analysis of uncertainty about exposure soil concentrations helps explain the quantitative and qualitative differences between biomagnification factors (BMFs) reported in the scientific literature and those computed for RMA, and facilitates the choice of the appropriate BMFs needed to characterize ecological risk at RMA. Second, analysis of the spatial distribution of biota tissue concentration predictions helps explain the lack of correlation between tissue and home-range soil concentration databases. Third, consideration of HHRC model parameter uncertainty clarifies the conservative bias introduced by compounding conservative exposure point estimates and provides the basis for selecting preliminary pollutant limit values (PPLVs) that accurately reflect risk management objectives.

The impacts of process and parameter uncertainties on RMA risks were investigated in numerous ways as necessitated by the complexity of RMA ecosystems and ambiguities in relevant databases. This appendix, which explains these methods and uncertainties, is organized as follows. Sections E.2, E.3, and E.4 discuss uncertainties associated with the chemical database, exposure point concentrations, and land use, respectively. Sections E.5, E.6, E.7, and E.8 discuss uncertainties associated with the human health exposure scenarios, toxicity estimates, exposure parameters, and PPLVs, respectively. The uncertainty distributions for the PPLVs arise from the

parameter distributions and from the random-sampling process involved in Latin Hypercube sampling. This uncertainty was compared among chemicals and exposed populations to assess which parameter uncertainties drive the PPLV uncertainties. Confidence intervals for the 5th percentile PPLV were also developed.

Sections E.9, E.10, E.11, E.12, and E.13 discuss the analyses conducted during the ERC. Sensitivity and convergence analyses were de-emphasized in these discussions because model structural uncertainties dominate the overall uncertainty about ecological risk. Section E.9 discusses uncertainty with the RMA soil and tissue databases used in the ERC. Section E.10 describes uncertainties associated with toxicity threshold values, maximum allowable tissue concentrations, or MATCs, and toxicity reference values, or TRVs. Section E.11 discusses uncertainty in estimating parameter distributions. Section E.12 discusses uncertainty associated with the development of BMFs. Finally, Section E.13 identifies uncertainty associated with ecological measurement endpoints.

E.2 UNCERTAINTIES ASSOCIATED WITH THE CHEMICAL DATABASE

E.2.1 DATA COLLECTION AND ANALYSIS

E.2.1.1 Phase I/Phase II Sampling Programs

The primary soil sampling and analysis program at RMA, the Remedial Investigation (RI) program, was conducted in two phases. The Phase I program was designed to identify the presence of potential contaminants within designated sites and surrounding areas. The objective of the Phase II program was to more precisely estimate the areal and vertical extent of contaminated soil within designated sites. Phase I and Phase II programs used different analytical techniques for some of the organic chemicals. Phase I employed gas chromatography/mass spectrometry (GC/MS) methods, and Phase II employed more precise GC methods. The Phase I GC/MS methods were capable of screening a larger range of compounds than GC methods, but with typically higher detection limits than GC methods alone. Accordingly, the Phase II GC

methods were employed to more precisely quantify Phase I detections and improve determination of the extent of contamination for the analytes detected during that Phase.

In a few cases, Phase I samples required dilution to facilitate analysis, and the dilution may have masked the presence of some compounds by raising the effective detection level and, therefore, the certified reporting limit (CRL). These situations were examined on a case-by-case basis and comparisons were made with data from surrounding areas. If necessary, an expanded suite of Phase II analyses and/or additional GC/MS analyses was used to ensure that all target analytes were evaluated.

Analytical procedures or results such as the following warranted an extension of the Phase I program:

- Concentrations were detected in only a few of many samples at a particular site. (This led to uncertainty that these measurements were true detections.)

- Concentrations detected were very close to background concentration levels or the method detection limit.

- Adherence to the Phase I protocol was not complete.

- A field sampling or laboratory analysis error during the Phase I program possibly affected the analytical results.

- Further characterization of the site was necessary using investigative methods other than soil sampling and analysis, such as geophysics.

High CRLs may over- or underestimate contaminant concentrations depending on site-specific data and statistical methods used to determine concentration levels. In the HHRC, high CRL

values were removed during data analysis to avoid overestimating representative concentrations or underestimating maximum concentrations at a site. In the ERC, a protocol for replacing data that were below the CRL by using information from surrounding borings (Appendix Section C.1) was used to minimize this source of uncertainty. Appendix Section E.12.4.1 provides a detailed critical discussion of the BCRL replacement protocol.

E.2.1.2 Soil Boring Density

The density of Phase I borings at each designated site was determined through the use of empirical relationships based on past site experience. A boring-density curve was developed using knowledge of soil investigation data from other hazardous waste sites throughout the country, historical data concerning contaminant disposal practices, and the estimated areal extent of each site. The curve was used to estimate the total number of Phase I and II soil borings and to allow preliminary estimates of required effort and schedule. Closer borehole spacing was utilized at smaller sites, allowing for multiple sample collection at even the smallest sites. Exceptions to the initial program design were allowed for site-specific reasons.

Non-source areas, defined as the areas within each of the 1-square-mile sections of RMA that are not included as part of the designated sites within that section, were sampled on a nearly uniform grid. Borehole spacing was based on the occurrence of designated sites within the non-source area (i.e., a closer borehole spacing was chosen for non-source areas with a greater number of designated sites in the section due to the greater potential for contamination in these areas). Once the grid was established, actual bore locations were adjusted during field inspections to ensure that samples were collected from areas most likely to contain or concentrate contaminants (e.g., depressions and scarred areas). Additional data were collected as required to meet Endangerment Assessment and Feasibility Study needs.

E.2.1.3 Boring Locations

RMA borings were associated with existing sites by mapping their spatial coordinates onto a site boundary map. While approximately 10 percent of the borings in the October 1991 RMA Environmental Database (DPA 1991) lacked spatial coordinates, most of these were added by March 1993 when the database was downloaded for use in the IEA/RC. The absence of spatial coordinates prevents borings from being assigned to the appropriate sites, thereby affecting the representative and maximum concentration values, indirect PPLVs, and the total boring and boring exceedance counts.

E.2.1.4 Certified Reporting Limits and Detection Limits

For each of the analytical methods, analyte concentrations can be quantified within a specified concentration range. The lower limit of this range was designated by U.S. Army Toxic and Hazardous Materials Agency (USATHAMA) as the lower CRL. This value is sometimes greater than the method detection limit. Confidence in the results reported at or near the method detection limit for these cases is lower than for the CRL. The CRL specifically incorporates method variability estimates and calibration function uncertainties and serves to reduce the uncertainties inherent in the analytical methods. Use of the CRL increases confidence in the analytical results at an early stage in the program, reduces the necessity of applying more global assumptions to manage uncertainties at a later stage, and provides a range of concentrations below the CRL at which analyte can be detected. While the uncertainty associated with analyte concentrations detected in this range is large, the raw data can be used to supplement reported results where there may be ambiguities or contradictory analyses. Finally, given the necessity of using multiple laboratories for analyses during the RI program, use of the CRLs improved the level of confidence in results from different laboratories.

E.2.1.5 Composite Samples

In non-source areas, the 0- to 1-foot (ft) depth interval was selected to detect the presence of contaminants such as organochlorine pesticides or trace metals that may have been surficially

disposed or transported by wind. The 4- to 5-ft depth interval was selected to screen for the presence of deeper contamination. The 0- to 1-ft sample and the 4- to 5-ft sample from each boring were composited for chemical analysis. This approach provided an efficient and cost-effective method to screen the nearly 20 square miles of non-source areas, although there was a loss of analytical sensitivity since the CRLs were effectively doubled. That is, if a sample with no contamination was composited with a contaminated sample, the concentration of the contaminated sample would have to be twice the CRL in order for any detection to be quantified.

To adjust for the potential underestimation of contaminant concentrations in composited samples, detected concentrations and the CRL were doubled. The overall bias due to this conservative treatment of composite "hits" (i.e., measured values greater than the CRL) is unknown.

E.2.2 ARMY CHEMICAL WARFARE AGENTS

As the RI Phase I program progressed, analytes were added to the initial target list based on recommendations from parties involved in the investigation. These additional analytes included U.S. Army chemical warfare agent (Army agent) degradation compounds and organonitrogen compounds. In areas with potential agent presence, samples were sent to the RMA laboratory for initial screening using specific analyses for Army agents. If analytical results indicated that no agents were present, the samples were shipped to a commercial laboratory for the remaining analyses. It was not reported, however, which specific samples were screened for agents, nor were all potential agent detections systematically confirmed.

Army agents are not quantitatively evaluated in the IEA/RC because they were not identified as contaminants of concern (COCs) in the Human Health Exposure Assessment (HHEA) screening assessment (Ebasco 1990). The lack of sufficient data also precludes their quantitative evaluation. For example, some agent detections were reported to be analytical artifacts. However, due to the high toxicity of Army agents (mustard, Lewisite, Sarin, etc.) and their degradation products, known or suspected areas of agent contamination on RMA (e.g., most of

Section 36 and the Toxic Storage Yard) are addressed in the qualitative risk assessment (Section 3.3.2). Additionally, any areas identified as containing agent or agent-related products will be evaluated in the Feasibility Study and remediated as required to mitigate potential risks.

E.2.3 TENTATIVELY IDENTIFIED COMPOUNDS

Phase I sampling detected a number of compounds that could not be clearly identified. Twenty tentatively identified compounds were considered significant enough to include with target analytes for the data presentations, discussions, and evaluations in the Study Area Reports (SARs) and in the Summary Report. The 20 tentatively identified compounds were included with the target analytes for data presentation, discussions, and evaluations in the SARs and in the RISR, and were included in the source-by-source exposure assessments and the Endangerment Assessment; however, they were not designated as specific target analytes because the RI sampling program (Phase I and II) was completed prior to the determination that their occurrences were of sufficient significance to warrant inclusion in the referenced documents. With the exception of 1,1,2,2-tetrachloroethane, none of the compounds was added as an additional COC for probabilistic PPLV development in the IEA/RC. As described in Appendix A of the HHEA report, factors accounting for their exclusion included infrequent detection, low concentration, and/or co-occurrence with other target chemicals (EBASCO 1990). The lower limit of detection for tentatively identified compounds was assumed to correspond to 10 percent of the internal standard for the GC/MS method used. For the purposes of the RI reports, a value of 0.3 micrograms per gram (µg/g) was used. Potential uncertainties associated with the under- or overestimation of risks based on the presence of additional tentatively identified compounds are not considered significant in relation to any of the previously discussed uncertainties associated with other aspects of the analysis.

E.3 UNCERTAINTIES ASSOCIATED WITH EXPOSURE POINT CONCENTRATIONS

E.3.1 UNCERTAINTIES ASSOCIATED WITH RMA CHEMICAL DATABASE

As discussed in Section 5.1, uncertainties associated with several aspects of the RMA chemical database limit the precision of exposure point concentration estimates. Quantitative estimates of these limitations and their influence on risk estimate uncertainty were often unavailable.

E.3.2 CALCULATION METHOD UNCERTAINTY

Uncertainties associated with the exposure point concentrations (C_{max} [maximum site concentration] and C_{rep} [representative site concentration]) are also associated with the estimation method used to approximate site concentration values used to calculate risk. In accordance with the U.S. Environmental Protection Agency (EPA) guidance, representative soil concentrations were estimated using the arithmetic mean ($C_{rep\ mean}$). The uncertainty in these estimates was characterized by also reporting the 95 percent upper and lower confidence limits (95 UCL and 95 LCL, respectively) on the mean. The confidence limits were estimated using the bootstrap resampling method described in Noreen (1989) and outlined in this section.

The bootstrap method was selected instead of the more common method of estimating confidence limits based on Student's t distribution, because t-based estimation led to computation of negative lower confidence limits (LCLs) for many of RMA's more highly skewed data sets. The bootstrap method is commonly used when the underlying distribution type is uncertain or variable, because it does not require assumptions about either the type of distribution or its degree of skewness.

In the bootstrap method, a hypothetical sample of size N (the number of original data points) is drawn from the N data points with replacement. Under replacement, each time a sample is drawn, it is replaced back into the data set, and therefore can be drawn multiple times. (If replacement was not used, the new sample would simply be a randomly ordered arrangement of the original sample.) A mean is then calculated from this hypothetical sample. The sampling with replacement process is repeated 1,000 times, and each time the mean is calculated. The

resulting 1,000 means are then sorted. The 95 percent lower confidence limit, 95 LCL, is obtained by calculating the midpoint between the 50th and 51st of these 1,000 ordered means. The probability that the underlying population mean is less than the 95 LCL calculated using this procedure is 0.05 (50/1,000). Similarly, the upper confidence limit, 95 UCL, is calculated as the midpoint between the 950th and 949th ordered mean. Therefore, the probability that the population mean is greater than this procedure's 95 percent upper confidence limit, 95 UCL, is also 0.05.

The theory behind the bootstrap estimate of confidence intervals is as follows. The original sample is assumed to be representative of the underlying population, and is then treated as though it actually were that population. Each sample with replacement represents a plausible outcome of drawing a random sample with N data points from the population. The observed sample could have turned out to have the values of any one of the 1,000 resamples with equal probability, and therefore could have implied any one of the 1,000 means. The bootstrap method uses the variability in the statistic of choice (in this case, the sample mean) to describe the uncertainty in using this statistic to estimate the true mean. If the statistic is unbiased, and the sample is truly representative of the population, then the bootstrap confidence intervals will also be unbiased and accurate. The bootstrap method cannot produce negative confidence limits for non-negative samples because the confidence limits are selected from the array of possible sample means, none of which will be less than zero for a non-negative sample.

Figure E.3-1 displays sample arithmetic means and bootstrap-method confidence limits for several limiting C_{rep} calculations (small sample size, high below certified reporting limits [BCRL] fraction), and compares them with C_{max}, the maximum measured concentration.

E.3.3 CONSIDERATIONS FOR CONTAMINANT FATE AND ATTENUATION AT RMA

The disappearance of a contaminant from RMA soils is governed by several factors. These include biodegradation, chemical degradation, photodegradation, volatilization, dust dispersion, biological uptake, surface water migration, and infiltration. The most ubiquitous contaminants at RMA are aldrin and dieldrin; as with every persistent organic chemical, it is well recognized that given sufficient time and proper conditions these chemicals will disappear from environmental media. The following discussion presents a chronology of the evaluations performed by the U.S. Army (Army) and Shell Oil Company (Shell) to address this issue, including a summary of review comments by a panel of experts assembled by the Army. This discussion is followed by a position paper provided by Shell, entitled "Dieldrin Soil Loss Position," which is provided in Attachment E.3-1.

In response to Shell's comments on the Army's failure to consider the fate of contaminants in RMA soils, the Army proposed that fate and attenuation of COCs in soils be considered as part of the Risk Characterization (Shell letter to Program Manager for the Rocky Mountain Arsenal (PMRMA) dated May 30, 1990 [Shell 1990], Comment # 12; PMRMA letter to EPA dated June 22, 1990 [PMRMA 1990], page 7). Subsequent to a number of meetings with Shell (February 27, 1991 and March 3, 1991), the Army developed a position paper regarding degradation of organic contaminants as applied to RMA. This position paper was discussed at an Endangerment Assessment Technical Subcommittee meeting on February 28, 1991. At that meeting, the Army requested written comments from the Parties. Comments were received from EPA (EPA 1991a). No comments were received from the State of Colorado. The Army requested specific information from Shell to augment its evaluation (EBASCO 1991). Shell provided verbal comments to the Army and agreed to provide additional data that would support inclusion of a fate and persistence term in the exposure and risk computations.

In an external review of the issue of contaminant fate and attenuation at RMA (ANL 1991), the following conclusions were offered: (1) although there is much information about the persistence

and kinetics of aldrin/dieldrin, considerable uncertainties remain; (2) the loss rates reported in the literature cannot be extrapolated to RMA since they were measured only in the initial period after the pesticides reached the soil; (3) for soils in which aldrin/dieldrin persisted for some time, it was not possible to distinguish between site characteristics associated with continuing but slow loss and those for which a loss could not be detailed; (4) the slope of lines describing chemical persistence did not appear to be statistically different from zero, hence indicating no loss; and (5) poor precision in chemical analyses and small concentration-level changes precluded a meaningful assessment of loss rate. The report, however, indicated that dissipation by volatilization might give values that could be useful in predicting future trends, if well-mixed soil samples at RMA were incubated under ambient conditions and aldrin/dieldrin volatilization was measured. Regarding metabolite product formation, it was concluded that some metabolites and products would persist in the field. Nonetheless, only a few studies definitively support this conclusion.

The external review and the aldrin/dieldrin loss issue were discussed in subsequent meetings between the Army, Shell, and Argonne National Laboratory (ANL). As a result of these meetings, Shell prepared a revised summary document, Dieldrin Soil Loss Position, and submitted it to the Army on January 10, 1992. This position paper is provided in Attachment E.3-1. Shell's document provided a summary of the literature and field studies conducted at RMA and considered the review comments and suggestions made by ANL. The Shell analysis concluded that literature studies show a steady decline in dieldrin concentration after the use of aldrin has declined or ceased, although the responsible transformation processes, including biodegradation and chemical transformation, could not be quantified at RMA due to the lack of site-specific data. Literature references and experimental results for volatilization of dieldrin from soils were also provided by Shell, along with a preliminary model to describe volatilization from soils. Shell's volatilization loss monitoring studies, conducted at RMA during summer 1988 and October 1991, suggested that surficial moisture content, ambient temperature, and surficial soil dieldrin concentration were the dominant parameters affecting the volatilization of dieldrin from surficial

soils. The Shell model did not account for diffusion and re-sorption to soils as the dieldrin (in vapor phase) migrated through the soils toward the surface. This process may be neglected in assessment of surficial soils, but for the deeper soil horizons, diffusion and resorption is expected to significantly reduce the loss due to volatilization. Another limitation of the model is that it did not address the expected temperatures or moisture content of the soils at depth. Although the study noted that volatilization rates are a function of organic carbon content, the model did not include factors to address the variability in organic carbon concentrations at RMA.

A final review of this issue concluded that site-specific data are not currently available to quantify the loss rates for biodegradation and chemical transformation and that the literature information cannot be extrapolated to RMA. Based on the data gaps described above, inclusion of a fate and attenuation term to account for biodegradation or chemical transformation in the risk computation performed at RMA was not deemed appropriate by the Army.

E.4 UNCERTAINTIES ASSOCIATED WITH LAND USE

Uncertainty exists regarding the likelihood that the land uses evaluated will in fact occur under a future development scenario at RMA. The following discussion addresses the potential for these scenarios to occur at RMA.

Land use at RMA currently is limited to commercial, industrial, recreational, and open space (i.e., nature preserve/wildlife refuge) uses. These land uses are described in Appendix Section B.2 and Section 3.1 according to their classification within either an Open Space or Economic Development scenario. The detailed assessment of current land uses and the evaluation of future land uses for RMA was presented in the HHEA report (Volume 1) (EBASCO 1990).

The land-use assessments that appear in Volume I of the HHEA report (EBASCO 1990) were based on extensive information obtained from several governmental agencies overseeing and directing land-use within their respective jurisdictions surrounding RMA. The agencies were the

Adams County Planning Department, Commerce City Planning Department, City and County of Denver Planning Offices, and Denver Airport Planning Offices. The United States has exercised exclusive federal jurisdiction and has controlled land use at RMA since 1942. Although RMA is within the boundaries of Adams County, no planning agencies, with the exception of the Denver Airport Planning Team, have specifically addressed land-use planning for RMA. However, the predictions about potential future land use, which were developed based on these information sources, are thought to be reasonable.

As discussed in Section XLIV of the Federal Facility Agreement (FFA) (EPA 1989b) and in Volume I of the HHEA report (EBASCO 1990), there are restrictions on ownership, use, and transfer of property at RMA now and into the future. Consistent with the FFA, certain future land uses at RMA are not considered foreseeable by the United States. These land uses include residential and agricultural development.

The FFA indicates that significant portions of RMA will be available for public benefit (including, but not limited to, wildlife habitats and parks). On October 9, 1992, RMA was designated as a National Wildlife Refuge to be managed by the U.S. Fish and Wildlife Service (USFWS). Limited areas at RMA may also be developed for commercial and industrial uses. Given these projections, two land-use scenarios were identified that formed the basis for defining target receptor populations. (1) open space, which includes nature preserve, wildlife refuge, and recreational park scenarios, and (2) economic development, which encompasses commercial and industrial scenarios.

Because the final mix of future RMA land uses is still under evaluation by the Army, each of the five foreseeable land-use options was evaluated in the development of PPLVs and the estimation of RMA-wide risks. It is recognized, however, that only select areas at RMA may actually be applicable to any given land-use option.

Evaluation of potential risk to biota has been based on the assumption that appropriate habitat exists for most species across RMA and will be available under future land-use options. Because not all areas have appropriate habitat or will be available, even given the National Wildlife Refuge status accorded RMA, this assumption introduces uncertainties that are likely to be conservatively biased. For the bald eagle, great blue heron, shorebird, and strictly aquatic trophic boxes, areas of assumed use were restricted, somewhat minimizing this source of uncertainty.

E.5 HUMAN HEALTH: UNCERTAINTIES ASSOCIATED WITH EXPOSURE SCENARIOS

E.5.1 EXPOSED POPULATIONS/SUBPOPULATIONS

In light of current land uses and the potential for a mix of land uses under a future economic development scenario, exposures of the populations/subpopulations associated with each of the land-use options were conservatively evaluated in estimating current and future risk. One source of conservatism is that the HHRC assumed no access restrictions. However, the Army restricts general access, as it does to the contaminated areas. Therefore, exposures of the magnitude assumed in this analysis, particularly the boring-by-boring analysis, are unlikely to occur. Based on available information, all populations and subpopulations were considered likely to frequent RMA to some varying degree. Each evaluated population or subpopulation is discussed below.

E.5.1.1 Regulated/Casual Visitors

Current visitors to RMA engage in a variety of activities. Buses and organized wildlife observation groups, known as regulated visitors, tour RMA regularly, although official statistics on the visitation frequency are not available. People designated as casual visitors are known to visit RMA on an irregular basis to observe and photograph wildlife, walk for leisure, hike, or picnic. Given the local interest in preserving RMA in its natural state, these activities, and therefore the visitor populations and subpopulations, are expected to continue at RMA in the future.

The risk evaluations for the regulated/casual visitor were predicated on a local neighborhood regulated/casual visitor subpopulation. Members of this subpopulation could have a higher

visitation frequency than other groups due to their proximity to RMA. No information is currently available to substantiate the numbers of individuals comprising such a subpopulation or their relative use frequency of RMA (or of any one "park") since a local survey has not yet been conducted. It seems reasonable to assume, however, that such a subpopulation currently exists and can reasonably be thought to exist under a future scenario as well. The allowable future land uses for RMA will be determined in the Fish and Wildlife Service's Refuge Management Plan, which is currently under development.

E.5.1.2 Recreational Visitors

Current recreational activities at RMA include catch-and-release fishing and dispersed activities such as walking, jogging, bicycling, cross country skiing, picnicking, and other miscellaneous activities. In the future, these activities would be expected to continue in a manner compatible with wildlife preservation. Developed recreational facilities (e.g., athletic fields) do not currently exist at RMA, and the likelihood of their occurrence in the future is unknown. Local planners, however, have indicated a sufficient quantity of athletic fields in the Commerce City area.

The risk evaluations for the recreational visitor were predicated on a local neighborhood recreational visitor subpopulation. Members of this subpopulation could have a potentially higher visitation frequency due to their proximity to RMA. This subpopulation was thought to be comprised of individuals who would use RMA on a frequent basis to engage in walking, jogging, bicycling, or other miscellaneous activities for which actual participation frequency data were available. No information is currently available to substantiate the numbers of individuals comprising such a subpopulation, nor the accuracy of the assumed relative use frequency of RMA (or of any one "park"), since a local survey was not conducted in support of the evaluations. It seems reasonable to assume, however, that such a subpopulation currently exists and can reasonably be thought to exist under a future scenario as well. The allowable future land uses for RMA will be determined in the Fish and Wildlife Service's Refuge Management Plan, which is currently under development.

E.5.1.3 Refuge Workers

USFWS currently has offices at RMA and oversees the majority of wildlife-viewing tours conducted on post. In addition, USFWS biologists are involved in a variety of different activities including wildlife surveys, census-taking, trap and release wildlife sampling, habitat enhancement activities (e.g., tree planting), and other wildlife-management practices. Under a future development scenario in which RMA is predominantly a wildlife refuge, the activities of USFWS staff would be expected to be similar to those of the current staff.

In the risk evaluations for a refuge worker population, a biological worker subpopulation with a potentially higher frequency of soil contact due to predominantly outdoor activities was identified, based on survey data of current wildlife refuges considered most analogous to RMA. Individuals within this subpopulation would spend greater than one-half of their working day engaged in activities that could bring them in greater contact with soils. The number of individuals surveyed in this category was small, but confirms the presence of this type of an individual among the ranks of USFWS staff.

Because RMA is likely to be designated for significant open space land use, enhanced wildlife-oriented activities are expected to increase under a future development scenario. Therefore, it seems reasonable to assume that a biological worker population would also exist, especially given their current presence on analogous facilities.

E.5.1.4 Industrial Workers

Although there is currently limited use being made of the Rail Classification Yard and associated warehouses at RMA, workers at RMA maintain the groundwater boundary treatment facilities, and facilities engineering staff are engaged as laborers and equipment operators. In the future, RMA boundary treatment system personnel are expected to continue operations and maintenance of the system, and the likelihood of the facilities engineering staff continuing to maintain a presence at RMA in the future is considered reasonably good. The likelihood of continued use of the Rail Classification Yard and warehouses in the future, however, is unknown. Additionally,

there are personnel employed at the water treatment facility located at the west entrance of RMA who will continue to work at RMA into the foreseeable future. The presence of other types of industrial workers at RMA in the future will depend on the land-use decisions made by the Army, including the potential for any new industrial leases at RMA.

E.5.1.5 Commercial Workers

Currently the PMRMA staff occupy Building 111 at RMA. These individuals are engaged in administrative functions that are almost exclusively conducted indoors. Though not considered "commercial" workers, their exposures parallel those identified for a commercial worker population. The PMRMA staff is expected to maintain a presence at RMA for an indeterminate period, and the United States Post Office is also expected to maintain its distribution center at RMA well into the future.

E.5.2 EXPOSURE PATHWAYS ASSOCIATED WITH HUMAN RECEPTORS

E.5.2.1 Exposure Pathways Considered

Exposure pathways were evaluated in consideration of the actual or projected activities for each population or subpopulation as discussed in Appendix Section B.2 and Section 3.1. Direct soil exposure pathways were considered applicable to all populations and subpopulations given the potential for contact with soil during the course of their activities. For the commercial worker population—assumed to have a predominantly indoor exposure regime—soil contact pathways were considered reasonable under the assumption that indoor dust had an origin in outdoor soils.

Outdoor (open space) soil vapor inhalation was considered reasonable for all of the visitor populations, the biological worker subpopulation, and the industrial worker population in recognition of the fact that a substantial portion of their activities are projected to be spent outdoors. Enclosed space vapor inhalation was considered reasonable for commercial workers and industrial workers. Enclosed space vapor inhalation was not considered for biological workers due to their predominantly outdoor exposure regime. The combination of both direct soil and indirect soil vapor exposure pathways evaluated was considered realistic.

The presence of basement structures on RMA in the future, which would be necessary for the enclosed space vapor exposure pathway to be applicable, may not be realistic given a number of physical constraints that could prohibit or severely limit these types of structures. These constraints include the following: (1) the presence of the Bald Eagle Management Area for which development would likely be restricted or severely limited, (2) the occurrence of significant areas where the depth to groundwater would prohibit the construction of below-surface structures, and (3) the presence of a 100-year flood plain in significant areas of RMA.

The combination of soil exposure pathways is thought to represent a conservative estimate of routes through which receptors may encounter contaminants.

E.5.2.2 Exposure Pathways Not Considered

Several exposure pathways were not evaluated in the IEA/RC as discussed below. These pathways were typically associated with land uses not considered likely at RMA, or with limitations on the uses of environmental media as specified in the FFA. Consumptive exposure pathways (e.g., vegetable, meat, fish, and dairy ingestion) associated with agricultural and rural residential uses at RMA were not evaluated due to land-use and environmental media restrictions specified in the FFA. Had consumptive exposure pathways been evaluated as part of a reasonable future land use at RMA, it is expected that risks — cancer risks predominantly — would have been greater than those currently projected, due to the known bioaccumulative properties of a number of the COCs, particularly the organochlorine pesticides. Nevertheless, risks potentially associated with the evaluation of consumptive exposure pathways would be highly uncertain due to a general lack of contaminant transfer coefficients in both environmental and biological media.

Dermal contact with soils was not evaluated for the metal COCs due to their known low absorption potential (see Appendix Section B.1). Therefore, the risk estimates projected for the metal COCs may be underestimated. The underestimation would not be significant, however, due to their low bioavailability from a soil matrix.

Groundwater ingestion exposures at RMA were not evaluated due to restrictions (as specified in the FFA) on the use of groundwater beneath RMA. Evaluation of ingestion exposures for the groundwater medium would, however, likely result in an increased risk from that projected above for soil exposures. Vapor inhalation exposures from groundwater were previously evaluated during the HHEA (EBASCO 1990) and were shown to contribute to open and enclosed space vapor inhalation exposure for several chemicals.

Ingestion of fish from RMA lakes was not evaluated due to restrictions (as specified in the FFA) on this use. The exclusion of this pathway in the risk evaluations could underestimate exposures and risks for recreational anglers. Potential exposures associated with occasional dermal contact with surface water by this visitor was evaluated in the HHEA report (EBASCO 1990) and shown to pose risks within acceptable levels for both carcinogenic and noncarcinogenic chemicals.

E.5.3 SPATIAL EXPOSURE CONSIDERATIONS

The approach used to evaluate exposures and potential risks at RMA sites was predicated on the simplifying assumption that any individual of a population or subpopulation may spend his or her entire exposure period at each site. This is not a realistic exposure scenario for an area as large as RMA. In reality, some individuals (e.g., biological workers) will likely spend portions of their exposure period at a number of different sites at RMA at a number of different activities and will actually receive only a fraction of the assumed exposure. Therefore, the assumption that each individual of each population has a complete exposure at every site represents an overestimate of risks for a large number of sites at RMA.

E.5.4 ADDITIVITY OF CANCER RISKS AND THE HAZARD INDEX

In the IEA/RC, both cancer risks and noncancer hazard quotients (HQs) are assumed to be additive, consistent with current risk assessment procedures (EPA 1989a). There are several limitations associated with this assumption, which are summarized below for cancer and noncancer systemic toxicity endpoints. Due to these limitations, the potential to over- or underestimate risk cannot be firmly established.

E.5.4.1 Additivity of Cancer Risks

Cancer risks were summed consistent with procedures discussed in EPA (1989a, pg. 8-12). In summing cancer risks, the underlying assumption is that there is an independence of action (i.e., effect to organ, tissue, etc.) by the chemicals involved and that there are no synergistic or antagonistic chemical interactions. In other words, all carcinogenic chemicals are assumed to produce the same effect (i.e., cancer). If the assumption is incorrect, cumulative site risks projected may be over- or underestimated. The assumption of risk additivity for carcinogens poses limitations. For example, the cancer slope factors represent upper 95th percentile values that are not strictly additive. Therefore, the total site cancer risk may become artificially more conservative as cancer risks from a number of different carcinogens are summed. An additional limitation with summing cancer risks over multiple chemicals is the equal weight given to chemicals with differing weights of evidence for human carcinogenicity (i.e., Group A versus Group C carcinogens) and slope factors derived from animal data vs. those with slope factors based on human data.

E.5.4.2 Additivity of Noncarcinogenic Hazard Quotients

Noncancer effects from multiple chemicals were also assumed to be additive consistent with current risk assessment procedures (EPA 1989a, pg 8-14). In summing the HQs at each site, the underlying assumption is that there is an independence of action and that dose additivity is appropriate. Because little or no information on antagonistic or synergistic effects is available for the RMA COCs, the assumption of additivity was incorporated in the HI determinations. Another limitation associated with the approach used was that HIs were not segregated by major effect. Because only a single COC appeared to contribute to the majority of the risk at most sites, however, this simplifying step may not introduce large degrees of uncertainty into the noncarcinogenic hazard evaluations.

E.6 HUMAN HEALTH: UNCERTAINTIES ASSOCIATED WITH TOXICITY ESTIMATES

Results of the importance analysis described in Appendix Section B.3 for the dose-response (DT) parameter indicated variability in computed PPLVs for a number of chemicals. Although the importance analysis supported the use of probabilistic distributions rather than fixed values for DT, a distribution was not developed for the dose-response parameter. DT was designated a fixed parameter to maintain consistency with established EPA DT values used in Superfund risk assessments. However, a large degree of uncertainty is known to be associated with the DT values. The major sources of uncertainty include the following:

- Extrapolation of DT information from effects observed at high doses administered in a laboratory setting to effects observed at relatively low doses expected from human contact with the chemical in environmental media. This could lead to over- or underestimation of risk.

- Use of short-term DT studies to predict the effects of long-term (chronic) exposures and vice-versa. This could lead to over- or underestimation of risk.

- Use of animals to predict the effects of contaminant exposures on humans where adequate human data are lacking. This could lead to over- or underestimation of risk.

- Use of DT data from laboratory animals (homogeneous populations) and healthy humans to predict the effects observed in a general population, which includes individuals with a wide range of sensitivities (e.g., pre-existing diseases that increase susceptibility to contaminants). This could lead to underestimation of risk, especially for sensitive human subpopulations.

Specific uncertainties associated with the toxicity estimates for the human health COCs are discussed below for carcinogens and noncarcinogens. With the exception of those for dibromochloropropane (DBCP), the uncertainties described below for each COC were obtained

from EPA's Integrated Risk Information System (IRIS) database (EPA 1992). Information regarding the estimates for aldrin/dieldrin were drawn from a number of sources, each of which is referenced below. Estimates for DBCP were obtained from EPA's Health Effects Assessment Summary Tables (HEAST) (EPA 1991b) because this chemical has been temporarily withdrawn from IRIS and is under review by EPA.

E.6.1 CARCINOGENIC CHEMICALS OF CONCERN

The carcinogenic COCs with the greatest number of exceedances of 5th percentile PPLVs for a 10^{-4} or 10^{-6} reference risk level were aldrin, dieldrin, arsenic, DBCP, chlordane, and chromium. Risks for all other carcinogenic COCs were generally insignificant relative to other COCs; accordingly, these chemicals are not discussed.

The uncertainty associated with the carcinogenicity assessment for the COCs contributing to estimated site cancer risks is described below. The uncertainties include the basis for the weight of evidence classification assigned by the EPA in relation to the adequacy of human and animal data, the number of animal species demonstrating the effect, the weight of evidence for carcinogenic activity in humans, and other supporting documentation.

Aldrin/Dieldrin

The carcinogenicity assessments for aldrin and dieldrin are considered together since the uncertainties for the two pesticides overlap to a significant degree. Aldrin is quickly metabolized to dieldrin in the body, and human epidemiologic studies in pesticide-manufacturing workers are based on exposures to both chemicals, thus making it impossible to separate the effects of the two chemicals.

The carcinogenicity assessments of aldrin and dieldrin are complicated by several areas of uncertainty. The lack of evidence of carcinogenicity in species other than mice and the occurrence of carcinogenic effects in mouse livers from chemicals that are not carcinogens in humans have been cited as indicating that mice may be particularly susceptible to the effects of

organochlorines and that, therefore, mouse carcinogenicity data are not applicable to any other species. Additionally, new epidemiologic studies that were not available at the time of EPA's carcinogenicity assessment may provide important information regarding the carcinogenicity of aldrin and dieldrin in humans.

It should be noted that the EPA designation of probable human carcinogen (Group B2) for aldrin and dieldrin is not consistent with the assessments of their carcinogenic potential in humans conducted by other agencies and organizations including the International Agency for Research on Cancer (IARC), the World Health Organization (WHO), and the National Toxicology Program (NTP) of the Department of Health and Human Services. IARC indicates that dieldrin is not classifiable as to its carcinogenicity in humans. The IARC classification is equivalent to EPA Group C-possible human carcinogen based on limited evidence in animals and absence of human data.

EPA's Carcinogenicity Assessment of Aldrin and Dieldrin (1987) reviewed the studies that were available at the time in order to develop the weight-of-evidence classification. An evaluation of their findings and conclusions and a discussion of the areas of uncertainty are included in this summary of the carcinogenicity assessment.

Aldrin is designated as a probable human carcinogen (Group B2) for ingestion and inhalation exposures. EPA (1987) determined that there were three studies of mice that were adequate in regard to number of animals studied, length of study in relation to the animal's life expectancy, and range of doses studied. These three studies found significant increases in liver cancer in three strains of mice fed aldrin in their diet. The slope factors calculated from the data sets were within a factor of 2 (with the final slope factor computed as the geometric mean of the three slope factors). Only one of seven rat studies was considered adequate by EPA, and it showed an increased incidence of thyroid and adrenal tumors. The incidences of thyroid and adrenal tumor development in various dosing groups were not consistently significant when compared with matched controls, therefore the authors concluded that the tumors were not associated with

treatment. The EPA review of the data concluded that the results were "equivocal" (EPA 1987). Two dog studies were considered unacceptable by EPA due to small numbers of animals and the short duration of the test relative to the animal's lifespan.

Dieldrin is designated as a probable human carcinogen (Group B2) for ingestion and inhalation exposures. This designation is based on an increased incidence of benign and malignant liver tumors in 7 strains of mice administered dieldrin orally in 11 studies that were considered adequate by EPA. The final slope factor was computed as the geometric mean of 13 slope factors estimated for male and female mice. The slope factors for the 13 data sets were within a factor of 8. There were only three studies of rats that were considered adequate; liver changes were noted, but no tumors were identified.

One hamster study, three dog studies, and two monkey studies were all considered unacceptable due to inadequate numbers or the short-term nature of the study relative to the life spans of the animals. Some of these studies found evidence of liver effects in treated groups but several did not. The hamster study (Cabral et al. 1979) detected liver cell hypertrophy in treated groups and hepatomas in two animals. The incidence of hepatomas was not significantly different from controls. Two dog studies (Treon and Cleveland 1955; Fitzhugh et al. 1964) revealed increased liver weights and fatty degeneration of the liver and kidneys in treated animals. A third dog study using lower doses found no organ changes (Walker et al. 1969). No tumors were found in the dog studies. The study of monkeys fed high doses of dieldrin for six years found no evidence of tumors or liver cell damage (Wright et al. 1978). Mice and rats exhibit specific, observable changes to the liver cells after exposure to chlorinated insecticides. A study of three strains of mice exposed to dieldrin found that a small number of animals showed these changes as early as four months after exposure began (Meierhenry et al. 1983).

The hamster study had high premature mortality in both treatment and control groups and was therefore considered inadequate to determine carcinogenicity. The dog and monkey studies were considered inadequate because the study time was short compared to the animals' life spans (two-

year study out of a ten-year life span for dogs and six-year study out of a twenty-year life span for monkeys). Mouse studies have found initial evidence of tumor development between 40 and 120 weeks in treatment groups (Walker et al. 1972; Thorpe and Walker 1973; Tennekes et al. 1982). This timeframe indicates the wide variability in time to tumor development within the animal's natural life span of approximately 130 weeks.

Therefore, other than the mouse studies, there have been only four studies (all of which were of rats) of the carcinogenic potential of aldrin and dieldrin that were considered by EPA to be adequate for its carcinogenicity assessment. A possible carcinogenic response was found in one of those studies, though not for liver tumors. Because there are significant data gaps in studies of other species, it is unknown whether aldrin and dieldrin are carcinogenic to any animals but mice.

The increased incidence of liver tumors in mice treated with phenobarbital and other chemicals, which has not been observed in humans or most other species, has been cited as evidence that mice have a particular oncogenic susceptibility to some chemicals and are thus not good surrogates for other species and humans in particular (Shell 1992a, b). This finding has been cited as evidence that aldrin and dieldrin are promoters rather than initiators of cancer and that mice are uniquely susceptible to the carcinogenic effects of aldrin and dieldrin (Tennekes et al. 1982). However, animal studies use genetically homogeneous populations in controlled situations so that the animals are not exposed to any other carcinogens or co-carcinogens. When extrapolating to humans, the large variations in genetic susceptibility to cancer and in the prevalence of other risk factors for cancer must be considered. For liver cancer in particular, several conditions unique to humans may increase their susceptibility. These risk factors include cirrhosis of the liver (due to alcoholism or other causes) and a chronic carrier state for hepatitis B virus, risk factors that are not uncommon in the United States (there are an estimated 1 to 1.25 million hepatitis B carriers in the United States) (MMWR 1991).

The mechanism by which aldrin and dieldrin exert their carcinogenic effects in mouse livers is unknown, but some evidence suggests that they may act as promoters (Wade et al. as cited in EPA 1987). Promoters do not cause cancer by themselves, but may interfere with communication between cells that inhibits the growth of latent tumor cells (Casarett and Doull 1986). Thus promoters may enhance the development of preneoplastic lesions. This promoting effect can occur even if the contact with the promoter occurs after the exposure to the carcinogen (Casarett and Doull, 1986). Sensitive subpopulations with preexisting abnormal liver cells who are exposed to a promoter may be at increased risk of developing liver cancer. If it is determined that aldrin and dieldrin are promoters, then it is possible that exposure to them in the presence of preexisting abnormal liver cells could increase the rate of development of liver cancer. No studies have been conducted to test the hypothesis that aldrin and dieldrin could increase the risk of liver cancer in sensitive human subpopulations. Because of the small numbers of people who have been exposed to measurable doses of these chemicals, this type of study would be difficult to conduct. However, because of this uncertainty, EPA has incorporated an added layer of protection for these sensitive human subpopulations by assuming that humans are as sensitive as the most sensitive species.

EPA's assessment of mutagenicity studies for aldrin (EPA 1987) concludes that aldrin is probably not mutagenic but major data gaps exist. EPA indicated that no conclusions can be drawn regarding chromosomal aberrations but found aldrin presumptively genotoxic in human cells based on unscheduled DNA synthesis. However, the validity of the positive studies was questioned due to technical deficiencies. Aldrin was not genotoxic in bacteria, yeast, and rat liver cells.

EPA's analysis of dieldrin (EPA 1987) found that the results of thirteen studies indicate that dieldrin is not mutagenic. There was one inconclusive presumptive positive study of gene mutations in bacteria but EPA concluded that technical deficiencies of the study precluded acceptance of the results as valid. There was also inconclusive evidence of mutagenicity in hamster cells but this study also suffered from technical deficiencies. Despite these technical

deficiencies, EPA classified dieldrin as a presumptive mutagen for one bacteria and for hamster cells. Dieldrin was found to cause chromosomal aberrations in some human and mouse cells. It was determined to be presumptively genotoxic in human cells based on unscheduled DNA synthesis but technical deficiencies made the results inconclusive. There was no evidence for genotoxicity in yeast or in rat and mouse liver cells. A single study of epigenetic toxicity (effects that occur from a mechanism other than a direct effect on genetic material) suggests that dieldrin may possess promotional activity as evidenced by interference with cellular communication.

The World Health Organization reviewed the mutagenicity studies in its 1989 Environmental Health Criteria document (WHO 1989) and concluded that neither aldrin nor dieldrin exhibited mutagenic potential. WHO also cited studies indicating that aldrin and dieldrin interfere with intercellular communication.

Based on the lack of strong evidence for mutagenicity in any of the studies and the questionable validity of the few studies that were presumptively positive, it is likely that aldrin and dieldrin do not have significant genotoxic properties. However, until the major data gaps are addressed, a firm conclusion cannot be drawn regarding the genotoxicity of aldrin and dieldrin.

In its carcinogenicity assessments of aldrin and dieldrin, EPA considered human studies with pesticide-manufacturing workers to be inadequate due to lack of exposure quantification, confounding exposures, small number of workers studied, short duration of exposure and latency period, inclusion of unexposed individuals in the cohort, and a lack of survivorship data for a portion of the cohort. Since EPA, WHO, and IARC developed their carcinogenicity assessments, two new epidemiologic studies have been published on the carcinogenicity of aldrin and dieldrin in pesticide-manufacturing workers. These studies report results in terms of Standardized Mortality Ratio (SMR), which is the ratio, expressed as a percentage, of the number of deaths from cancer observed in the exposed population to the cancer deaths expected in the general population with age and time differences taken into account.

A study at the aldrin/dieldrin plant of the Shell Petrochemical complex in Pernis, The Netherlands (de Jong 1991) evaluated mortality through 1987 in 570 workers employed for at least 1 START year between 1954 and 1970. These workers represented 2,858 person-years of exposure. Dieldrin blood levels were available for workers after 1963, and these results were extrapolated to those exposed before 1963 to estimate exposure. Workers were divided into low, moderate, or high exposure categories.

De Jong (1991) found no increase in deaths from neoplasms in any exposure category. However, a statistically significant increase in deaths from rectum cancer was observed in the low exposure group (2 observed vs. 0.2 expected, SMR 957.6 percent, $p<.05$). One case of liver cancer was reported in a worker with moderate exposure (0.6 expected, SMR 170.6 percent, 95 percent confidence interval (CI) 2.2-927.3). This worker was reported to have chronic alcoholism by his former physician. The author concluded that the results indicate neither an increased risk for cancer in general nor an increase in specific neoplasms that could be attributed to exposures to aldrin and dieldrin. De Jong also evaluated mortality from total cancers and site-specific cancers for workers in the three exposure groups and compared the mortality rates to age- and gender-specific cancer mortality rates in the general population. The only significant increase in cancer mortality was for rectal cancers in the low exposure group.

The major flaw of this study was its lack of statistical power to detect increased relative risks for site-specific cancers, particularly liver cancer. Statistical power is the probability that a study will be able to find a difference between the exposed group and the control group if it truly exists. This probability is based on the numbers of people in each group, and the frequency of the disease in the general population. A study that has a low statistical power (usually because there were not enough people studied to find an increase in the numbers of rare cancers) is unlikely to detect a difference even if it exists. Although the study had greater than 99 percent power to find a relative risk of 2.0 for total cancer deaths, it had less than 14 percent power to detect a relative risk of 2.0 for liver cancer. Even if the relative risk were as high as 3.0, the study had less than 35 percent statistical power to find the increased risk. Since liver cancer is

relatively rare in humans, a large number of person-years of exposure may be required to detect an increased risk. Although no increased risk of total cancers was identified in the de Jong study, the lack of statistical power to detect an increased risk of liver cancer limits the conclusions that can be drawn from this study.

Sielken and Stevenson (1992) used the blood dieldrin levels from the de Jong study (1991) to derive a cancer potency factor using the same DT models that were used in the mouse studies. The result was a negative cancer potency factor, implying that the probability of cancer at a dose of 1 micrograms per kilogram per day (μg/kg-day) was less than the background level of cancer with no exposure. However, this analysis was performed only for deaths caused by any cancer and not for site-specific cancers. Sufficient data were not available to develop a DT curve for liver cancer.

Another epidemiologic study (Brown 1992) evaluated mortality in production workers employed for at least 6 months prior to 1964 at four organochlorine pesticide plants in the United States. The following discussion is limited to Plant 3, where aldrin and dieldrin were the predominant products and organophosphates and DBCP were produced part of the time. Vital status was examined through 1987 for 1,153 workers representing 34,479 person-years of follow-up. No historical exposure measurements were available; however, type of job, length of employment, and time from employment to death were determined for each case.

Mortality for all cancers was less than expected (SMR 86 percent, 95 percent C.I. 0.67-1.08), but there was a statistically significant increase in deaths from hepatobiliary (liver, gallbladder, and bile duct) cancers (5 observed, SMR 393 percent, 95 percent C.I. 1.27-9.20). When compared to the county rate rather than the national rate, the SMR was 486 percent (95 percent C.I. 1.57-11.36). Three of the deaths occurred in workers with less than 2 years employment and all occurred at least 15 years after exposure. Three of the deaths had occurred since investigators first examined this cohort through 1976 (Ditraglia et al. 1981). There were three biliary tract cancers, one gallbladder cancer, and one liver cancer.

Several factors complicate the interpretation of these results. Exposures to aldrin and dieldrin were not quantified, so a DT relationship could not be determined. Potentially confounding exposures to other chemicals also occurred, further complicating the evaluation. The cancers were extrahepatic (gallbladder and biliary tract) as well as intrahepatic (liver), although it is uncertain whether these findings reflect a carcinogenic response that is analogous to the intrahepatic tumors reported in mice.

In view of the uncertainties discussed above, it is reasonable to base the human potential for developing liver cancer from exposure to aldrin and dieldrin on the most sensitive species because of the sensitive subgroups that exist in the human population. There have not been enough studies of other species that were considered adequate by EPA to conclude that carcinogenic effects of aldrin and dieldrin are limited to the mouse. Due to the lack of statistical power, the epidemiologic data from the de Jong 1991 study is inadequate to detect an increased risk of liver cancer in humans. The study by Brown (1992) suggests an increased risk of hepatobiliary cancer for workers exposed to aldrin and dieldrin but is limited by a lack of exposure data. Because of these persistent data gaps, the lack of evidence of carcinogenicity in other species and the absence of a carcinogenic effect in the de Jong study are not compelling enough to justify classification of aldrin and dieldrin as noncarcinogens at this time.

Arsenic

Arsenic is designated by EPA as a human carcinogen (Group A) based on increased lung cancer mortality through inhalation exposure and increased skin cancer incidence in several populations consuming drinking water. The inhalation exposure study population observed was large, and exposure assessment data supporting the carcinogenicity assessment was adequate and included ambient and urinary arsenic measurements for two smelters. The range of slope factors computed using data from the two exposure areas, which were smelters, was within a factor of 6, indicating increased variability between the data sets. The final slope factor was computed as the geometric mean of the data sets. There are no animal data on carcinogenicity because various chemical forms of arsenic administered by different routes to several species have not yielded consistent

results. There are several supporting *in vitro* studies that have indicated mutagenic activity associated with sodium arsenate including sister chromatic exchange in Chinese hamster ovary (CHO) cells, and human peripheral lymphocytes. Additionally, transformations have been observed in Syrian hamster embryo cells.

Dibromochloropropane

DBCP is designated by EPA as a probable human carcinogen (Group B2) based on inadequate evidence in humans and positive results in animal studies. Detailed information regarding the slope factors for DBCP are pending on the IRIS database (EPA 1992). The information that follows was taken from the EPA HEAST (1991b), the Drinking Water Criteria Document for DBCP (EPA 1985), and the NTP (1982). Data demonstrating the potential carcinogenic activity in humans are not available. DBCP was carcinogenic in several animal bioassays via the oral (gastric gavage), inhalation, and dermal routes of exposure. Gastric gavage in mice and rats produced significant increases in squamous-cell carcinomas of the forestomach in both sexes of both species, and adenocarcinomas in female rats (EPA 1985). In an inhalation study, rats had increased incidence of nasal cavity tumors and lung tumors (EPA 1985). Dermal exposure resulted in an increased incidence of skin and lung tumors (EPA 1985). Tumors were also observed in the tongue, pharynx, nasal cavity, and adrenal cortex. Positive evidence of chromosome aberrations and sister chromatic exchange have been observed in CHO cells (NTP 1982). Somatic cell mutations and chromosomal aberrations have also been reported in *Drosphila melanogaster* (EPA 1985). No effects on dominant lethal frequency were observed in mice that received intraperitoneal and subcutaneous injections of DBCP (NTP 1982).

Chlordane

Chlordane is designated by EPA as a probable human carcinogen (Group B2) for ingestion and inhalation exposures based on benign and malignant tumors induced in four strains of male and female mice and in a single strain of male rats. Adequate numbers of animals were treated in the studies, and DT effects were reported in all studies. The geometric mean of slope factors for the mice was used as a basis for the final slope factor.

Human studies with pesticide manufacturing workers were considered inadequate due to limitations with sample size, follow-up duration, confounding exposures, or lack of statistically significant increased cancer mortality. Positive results have been reported in Chinese hamster lung cells and mouse lymphoma cells with and without exogenous metabolism. Chlordane is not mutagenic in bacteria, and it does not induce DNA repair in bacteria, rodent hepatocytes, or human lymphoid cells. It is genotoxic in yeast, human fibroblasts, and fish. Five compounds structurally related to chlordane—aldrin, dieldrin, heptachlor, heptachlor epoxide, and chlorendic acid—have induced malignant liver tumors in mice. Chlorendic acid produced liver tumors in rats.

Chromium

Chromium is designated by EPA as a known human carcinogen (Group A) following inhalation based on results of occupational epidemiological studies of chromium-exposed workers and established dose-response relationships between chromium exposure and lung cancer. This classification is for chromium VI since only chromium VI has been found to be carcinogenic in animal studies. Results of the studies of chromium exposure are consistent across investigators and countries. The assumption that the ratio of chromium III to chromium VI is 6:1 may lead to a seven-fold underestimation of risk, whereas the use of 1949 hygiene data, which may underestimate worker exposure, may result in an overestimation of risk. Further overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population because it is generally accepted that the proportion of smokers is higher for industrial workers than for the general public.

Epidemiological studies of chromate production facilities in the United States and West Germany have established an association between chromium exposure and lung cancer. Three other occupational studies also found an association between chromium exposure and lung cancer. Other occupational epidemiological studies were found to be inconclusive or negative as to lung cancer risk. Data supporting this classification of chromium includes positive mutagenic activity in bacterial assays for hexavalent chromium. In addition, chromium VI was mutagenic in yeasts

and in V79 cells. Chromium III and VI compounds were found to decrease the fidelity of DNA synthesis *in vitro*, while chromium VI compounds inhibit replicative DNA synthesis in mammalian cells and produce unscheduled DNA synthesis. Chromate has been shown to transform both primary cells and cell lines. Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors. No long-term studies of ingested chromium VI exist. There appears to be significant *in vivo* conversion of chromium VI to chromium III and chromium III to chromium IV. Chromium III is an essential trace element.

E.6.1.1 Implication on Projected Risks

As indicated in the EPA Guidelines for Carcinogen Risk Assessment (EPA 1986b), the cancer slope factors generated from the linearized multistage extrapolation procedure lead to what is considered a "plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the [cancer] risk. The true value of the risk is unknown, and may be as low as zero." With the exception of arsenic and benzene, whose slope factors were generated using a different extrapolation model, all of the slope factors for carcinogenic COCs used to estimate PPLVs and potential cancer risks were derived using the linearized multistage DT model and are thus upper bound estimates. Therefore, cancer risks associated with these chemicals are not likely to be underestimated, but may be substantially overstated.

Additionally, with the exception of arsenic, benzene, and chromium, the weight of evidence for carcinogenic COCs are predicated on animal data that may not be representative of the potential carcinogenic response induced by the chemical in humans. Debate surrounding the applicability of the animal models employed to derive the slope factor for aldrin and dieldrin continues in the scientific community. Nearly all risk assessments involve the extrapolation of biomedical data collected in animal models to the human condition. Such extrapolation, by nature, involves considerable uncertainty. EPA's classification of aldrin and dieldrin as "probable human carcinogens" indicates that EPA scientists consider the animal data adequate but have no human data to support this classification.

As indicated above, extrapolation of animal data to humans is conducted such that the estimated risk to humans is not likely to be underestimated. In the specific case of aldrin/dieldrin, the cancer slope factor is derived from studies conducted in several strains of mice. Efforts to induce cancer in animals other than the mouse have been unsuccessful, thus raising the question of the relevance of the slope factor to other species. Epidemiological studies conducted to date to assess the carcinogenicity of aldrin/dieldrin in humans have been of insufficient statistical power to make definitive statements about the carcinogenicity of these compounds in humans. As a result, estimates of aldrin/dieldrin carcinogenicity reported in the IEA/RC should be interpreted as plausible upper bound estimates of cancer risk for aldrin/dieldrin. The actual cancer risk is likely to be below the estimates provided and may be as low as zero. If aldrin/dieldrin were assumed not to be human carcinogens, total estimated cancer risks would likely be reduced significantly at selected sites (see, for example, Figures 3.2-11 and 3.2-12 from Section 3 of the IEA/RC report).

E.6.2 NONCARCINOGENIC COCs

As indicated in Section 3.2, site concentrations of several chemicals were shown to exceed an HI (systemic effects) of 1.0 for the most exposed subpopulation, the biological worker. Several COCs (chloroacetic acid, isodrin, and mercury) exhibited an HI exceedance at one site. The remaining noncancer COCs exhibited no exceedances for this subpopulation. (The cancer endpoint drives the PPLV for chemicals with both cancer and noncancer endpoints; therefore, the noncancer endpoint is not discussed.) Similar trends were exhibited for other populations or subpopulations.

A potentially significant degree of uncertainty is associated with the EPA noncarcinogenic DT values used in this assessment. A review of the critical effects for each RfD and the magnitude of the uncertainty factors (UFs) incorporated in their derivation is presented in Table B.1-10 (Appendix Section B.1). The confidence the EPA places in the study used to develop each RfD, as well as the confidence in the toxicological database underlying each chemical and the derived

RfD value are summarized in Table B.1-11 (Appendix Section B.1). Comments on the basis for the confidence ratings are also summarized (as available in IRIS) for each COC in Table B.1-11.

The following sections review the confidence in chemical-specific RfD values and the UFs were assigned by the EPA during the development of RfDs. Based on these determinations, each COC was assigned to a general category of uncertainty (low, medium, high). Other factors such as route-to-route extrapolation and interim RfDs developed in the assessment are also discussed in relation to their potential uncertainties.

E.6.2.1 COCs With High Uncertainties

Uncertainty was considered high for those COCs with a low confidence in the established RfD, regardless of the magnitude of the assigned UF. The COCs fitting these parameters for the ingestion route include chlordane (UF=1,000), chromium VI (UF=500), dieldrin (UF=100), and hexachlorocyclopentadiene (HCCPD) (UF=1,000). No COCs were identified within this category for the inhalation route.

Risk estimates for those COCs with RfDs extrapolated from the ingestion route to the inhalation route are also considered highly uncertain. Oral DT data were commonly extrapolated for most COCs in the IEA/RC vapor and particulate inhalation exposure evaluations due to the general lack of verified reference concentrations for most chemicals. The resulting risk evaluations, which incorporate these pathways, are therefore considered uncertain as well.

Route-to-route extrapolation was also required to evaluate potential risks from the dermal exposure pathway. The uncertainties are expected to be particularly high for all COCs[1] with regard to the dermal exposure pathway (except metals) since oral DT values were routinely used as the basis for evaluating dermal exposures and risks due to a lack of dermal DT data. The potentially large uncertainties associated with these extrapolations is very important to note due

[1] Metals are an exception since dermal exposures and risks were not quantified.

to the dominant nature of this exposure pathway for a majority of the COCs evaluated at RMA. The impacts on risks that result from extrapolation of oral to dermal RfDs are chemical-specific and may over- or underestimate risks. The oral DT data used to evaluate toxicity associated with the dermal exposure pathway (a standard practice in risk assessment) is considered unrepresentative of the toxicity likely to be associated with this pathway for most chemicals due to the generally more efficient absorption that occurs from the gastrointestinal tract. Absorption data from the critical toxicity studies (oral basis) was lacking, as was good dermal absorption data. Therefore, corrections for absorption efficiency between oral and dermal pathways in the relative absorption factor (RAF) parameter were largely incomplete for most chemicals. RAFs for the dermal pathway were highly variable for a large proportion of COCs due to conservative absorption assumptions incorporated in the development of dermal RAFs.

Uncertainty was also considered high for those COCs for which verified EPA RfDs or HEAST values were not available. For many of these chemicals, older allowable daily intakes were used, or interim values established based on available toxicological data (see Appendix Section B.1). Chemicals within this category included isodrin and lead. Confidence in the isodrin DT values and associated risks are especially low since they were established with very high UFs from acute lethality data using an extrapolation procedure based on the work of Layton et al. (1987).

The ingestion and inhalation values used for lead are based on older allowable daily intake values published in EPA (1986a). The lead DT values may well underestimate the lead PPLV and, therefore, noncarcinogenic risks associated with lead, in light of more recent information suggesting that the threshold for toxic effects associated with lead exposures may be much lower than previously estimated.

E.6.2.2 COCs With Moderate Uncertainties

Uncertainty was considered moderate for those COCs with a medium confidence in the established RfD and UFs ranging between a factor of 100 and 1,000. The COCs that fit these parameters for the ingestion route include aldrin (UF=1,000), carbon tetrachloride (UF=1,000),

chlorobenzene (UF=1,000), chloroform (UF=1,000), DDT (UF=100), 1,1-dichloroethylene (UF=1,000), endrin (UF=100), methylene chloride (UF=100), tetrachloroethylene (UF=1,000), and toluene (UF=1,000).

E.6.2.3 COCs With Low Uncertainties

Uncertainty was considered low for those COCs that have a high confidence in the established RfD and UFs ranging between 1 and 100. Only the oral DT value for cadmium (UF=10) falls within these parameters. It should be noted that two oral DT values associated with two different media—food and water—are available for cadmium. The more conservative value for water was used in the IEA/RC soil exposure evaluations. The use of the water medium as a basis for the oral DT for cadmium is expected to result in an overestimation of the exposures and risks from soil.

E.6.2.4 COCs With Unknown Degrees of Uncertainties

Uncertainty for those COCs with no verified EPA RfDs (HEAST values) is unknown since an evaluation of confidence in the database, RfD, or critical study was not available. The COCs with ingestion DT values in this category include chloroacetic acid (UF=10,000), dicyclopentadiene (UF=1,000), mercury (UF=1,000), and arsenic (UF=3). For the inhalation route the following COCs are included: chlorobenzene (UF=10,000), chromium VI (UF=300), dicyclopentadiene (UF=10,000), hexachlorocyclopentadiene (HCCPD) (UF=1,000), mercury (UF=30), methylene chloride (UF=100), and toluene (UF=300).

E.6.2.5 Implication on Projected Risk

Fewer numbers of sites displayed exceedances for the following COCs: aldrin, carbon tetrachloride, chloroacetic acid, chlordane, dicyclopentadiene, dieldrin, HCCPD, isodrin, and arsenic. The low confidence associated with the DT value for and isodrin places a high degree of uncertainty on the HIs quantified for this COC. With the exception of arsenic, all the COCs associated with a few or isolated HI exceedances have very large UFs (1,000). These potentially large uncertainties may be very important for the large number of sites displaying HI exceedances

within a factor of 10. Within this category, exceedances may not be significant in light of the potentially greater uncertainty associated with the DT values. Conversely, the high uncertainties posed by the above-listed chemicals may not be as significant for the large number of sites with HI exceedances in excess of a factor of 100 since at this magnitude the observed exceedances are not likely to be artifacts of uncertainty alone. The low UF incorporated in the arsenic RfD increases the confidence in the isolated HI exceedances indicated.

E.7 HUMAN HEALTH: UNCERTAINTIES ASSOCIATED WITH EXPOSURE PARAMETERS AND VAPOR MODELS

The variability and uncertainty in the PPLVs were estimated by developing probabilistic distributions for each of the models' parameters (see Appendix Section B.3). As defined here, variability in the parameter distribution refers to the real variation in possible parameter values, which may be spatial (e.g., soil density), temporal (e.g., dust loading), physiological (e.g., body weight, skin surface areas) or due to the effects of other factors such as behavior. Uncertainty is that part of the parameter distribution resulting from random sampling variation and other sources of potential error. Uncertainty increases the overall spread of the distribution and may also result in bias, both intentional (e.g., conservative assumptions) and unintentional (unknown). The types of variability, uncertainty, and bias most relevant to the PPLV model parameter distributions are described below in Sections E.7.1 and E.7.2. Additionally, the uncertainties associated with the PPLV vapor models are described in Section E.7.3.

A summary of the major uncertainties associated with the PPLV equation parameters for human health is presented in Tables E.7-1 through E.7-3. This tabular summary identifies aspects of parameter development for the human health evaluations corresponding to the types of uncertainties discussed in the subsections immediately following. More detailed information on the distribution development process for each parameter is provided in Appendix Section B.3, and a detailed description of the vapor models is provided in Appendix Section B.1.

E.7.1 PARAMETER VARIABILITY

In general, all of the PPLV model parameters are inherently variable. The variation may be spatial, temporal, physiological, or due to the dependence of the parameter on other possibly unknown variable factors such as environmental conditions (e.g., temperature) or behaviors (e.g., activity patterns). For example, the time-dependent variables (e.g., exposure days per year) for human health evaluations are known to vary substantially for different populations and within a given population due to different individual habits and activities, although the nature of the dependence on individual traits is not precisely characterized. The data set for a probabilistic parameter ideally represents the effects of these factors in roughly the same proportions as they are expected to occur at RMA. Variability in those human health parameters that were fixed (see Appendix Section B.3) is not represented in the probabilistic PPLVs.

High variability in model parameters results in high variability in the PPLV distribution, indicating that the 5th percentile PPLV, which protects 95 percent of the population would be much lower (i.e., orders of magnitude) than the value of the PPLV that protects only 50 percent of the population. Conversely, low parameter variability results in low PPLV variability, indicating that the criteria required to protect different levels of exposure is similar.

E.7.2 PARAMETER UNCERTAINTY

The distribution or fixed value assigned to a PPLV parameter is only an estimate of the true distribution or fixed value. The parameter is influenced by several types of uncertainty, described below, that include data representation error, extrapolation error, measurement error, uncertainty due to small data sets, and uncertainty associated with parameter correlation.

E.7.2.1 Data Representativeness

In general, it was difficult to ensure that all of the data were closely representative of the exposure conditions at RMA. Soil density, soil moisture, and fraction of organic carbon were characterized based on data collected from representative RMA-wide locations and incorporated into specific distributions for use in estimating subsurface soil vapor flux for human vapor

exposure evaluations. Nevertheless, RMA-specific conditions could not be directly incorporated into the chemical-specific parameter distributions in all instances. For example, vapor pressure data were screened to exclude values corresponding to experimental temperatures that were not relevant to the assumed RMA soil temperature (RMA soil temperature data were fixed based on ambient temperature data). As a result of the screening process, however, the remaining vapor pressure data were limited and so cannot be said to accurately represent the temperature regime at RMA.

There was substantial uncertainty about the representativeness of data for parameters describing human exposures (e.g., soil intake parameters, time-dependent exposure parameters). In general, however, conservative assumptions were made. Ages and activities associated with the open space visitor land-use options were characterized using available empirical data and professional judgment. Although survey data were used to characterize time and activity patterns for the refuge worker population and biological worker subpopulation in order to improve the confidence in the analysis, the representativeness of the resulting distributions for current or future exposed populations on RMA remains uncertain. The data sets compiled for these populations or subpopulations may under-represent exposures for some portion of the future RMA population and over-represent for some other portion. For example, because of a relatively low number of studies and subjects sampled, soil ingestion and soil covering data for children may represent a population with somewhat different attributes such as economic class, climate and soil type, and activity tendencies than the children being evaluated at RMA. As described in Appendix Section B.3, an effort was made in all cases to collect data to focus on specific RMA populations in order to reduce bias as much as possible. In some cases, the parameter distributions were assigned to reflect subpopulations of differing exposure potential (e.g., local neighborhood recreational visitors vs. biological workers) in order to ensure PPLVs were protective. It is not possible to determine with certainty whether data representativeness in the current evaluations imparted a conservative or nonconservative bias to the results. It is expected, however, that in the case of most parameters, distributions, and therefore PPLVs and site risks, were estimated with conservative biases and are therefore likely to overestimate risks.

E.7.2.2 Extrapolation Error

When no data directly describing a parameter were available, the parameter distribution was extrapolated based on additional information and professional judgment. In some cases, a chemical-specific parameter was extrapolated from one chemical to another and assumed to have similar properties. For example, the soil intake parameters for human health were sometimes extrapolated across age groups if such an extrapolation was deemed reasonable. The distributions for many of the population-specific, time-dependent exposure parameters were derived from "surrogate" data representing a different variable and a different or general population. For example, the jogging frequency of the visitor subpopulation at RMA was estimated from data on national jogging frequency and adjusted by an assumed distribution for the fraction of jogs that such visitors would undertake at RMA.

An attempt was made to clearly document all assumptions made in the extrapolations or applications of professional judgment. The impact of the potential extrapolation error on the PPLV distributions and resulting site risks cannot be determined with certainty, but likely results in a combination of over- and underestimation of site exposures and risk.

E.7.2.3 Data Measurement Error

The individual data points within each parameter distribution embody some degree of experimental measurement error. For example, the chemical-specific parameters (e.g., the organic carbon-based partition coefficient, or K_{oc}) are influenced by errors in analytical measurement techniques. Soil covering data for human health are influenced by the accuracy in removing soil from the hands of experimental subjects, while soil ingestion data are influenced by time lags between the measurement of soil ingestion and the soil tracer output in feces (which sometimes resulted in impossible negative soil ingestion values). The impact of measurement error on the PPLV distributions and site risks is unknown.

E.7.2.4 Small Data Sets

Because distribution shape and descriptors (e.g., means) cannot be accurately estimated from a small data set, the data were supplemented by professional judgment to facilitate assignment of a distribution type. If the sample is truly random, the estimation of distributional descriptors from small data sets is unbiased (i.e., the overestimates balance the underestimates); however, the degree of overestimation or underestimation varies more widely with smaller data sets than with larger data sets. As described in Appendix Section B.3, several methods were used to circumvent problems with small data sets or to provide a conservative estimate in the case of high uncertainty.

The impact of small data sets on the PPLVs and risk estimates is not precisely known. However, the conservative estimates derived for some parameters with small data sets will potentially result in conservative estimates of the PPLVs and site risks for some chemicals.

E.7.2.5 Correlation Between Parameters

Potential correlations between different model parameters were investigated throughout the distribution development process. For example, the strong correlation between total porosity and soil density was incorporated into the Latin hypercube sampling procedure as explained in Appendix Section B.3. In addition, the dependence (correlation) of water content on total porosity was also incorporated into the Latin Hypercube sampling of the model.

Because correlation data were not available for all the parameters for which distributions were developed, the parameters were assumed to be independent of each other. For example, because Henry's Law constant and vapor pressure are both dependent on temperature, other factors likely confound and diminish the potential correlation due to temperature (e.g., atmospheric pressure, interaction with the soil matrix and water). For time-dependent variables, it was reasoned for both visitor and worker populations that the three parameters would either be uncorrelated or possibly slightly correlated but in an unknown direction (i.e., positive or negative). In this instance, the number of hours of exposure per day for a worker might be positively or negatively

related (or unrelated) to the number of days of exposure per week, and the number of days of exposure per year would probably not be related to the number of years of exposure per lifetime.

The impact of correlation on the PPLVs ultimately depends on the direction of the correlation and the relationship of the parameters. If a variable in the numerator of the PPLV is positively correlated with a variable in the denominator of the PPLV, the PPLV distribution will be less variable, i.e., the 5th percentile PPLV will increase and the 95th percentile PPLV will decrease.

E.7.2.6 Correlation Over Time

In the human health evaluations, each PPLV was calculated based on a single value drawn from each of the parameter distributions. The resulting PPLV reflects an implicit assumption that exposure conditions are constant over time for a given individual. For example, the PPLV for the soil ingestion pathway was calculated by assuming that a constant soil ingestion value randomly selected from a probability distribution of possible soil ingestion values occurs every hour over the lifetime of hourly exposures. Most significantly, in all pathways the calculation of hours per lifetime assumed a constant number of daily exposure hours selected randomly from a probability distribution of possible daily hourly exposures for each exposure day, as well as a constant number of exposure days, again selected randomly from a probability distribution of the same for each year.

The elimination or reduction of maximal correlation in the exposure parameters over time would substantially reduce the variability in the PPLV distribution: the 5th percentile PPLV would increase and the 95th percentile would decrease. This reduction in variability would occur because soil intake rates would vary for different days, the number of daily exposure hours would vary each day, and the number of exposure days would vary each year. Because an individual's exposure would be calculated based on a range of values for each parameter, extreme exposures (e.g., all days/lifetime are associated with low soil ingestion or all days/lifetime are associated with high soil ingestion days/year) would become improbable. The lowered variability and

uncertainty would not necessarily indicate that the PPLVs were more accurate since a correlation of zero may be unrealistic for some individuals.

It is possible that exposures (contaminant intakes) would be variable over time for an unknown proportion of individuals within a population. For these individuals, the current assumption of a correlation of 1.0 over time would be conservative and result in an underestimation of the 5th percentile PPLV as well as an overestimation of the true variability and uncertainty associated with the PPLV. However, it is equally possible that certain individuals could encounter similar exposures daily on a long-term basis based on repetitive activities. For example, some industrial activities may entail consistently higher soil exposures than other industrial activities, while some recreational visitors may tend to engage in consistently similar activities for long periods of time as a matter of personal preference. Because the PPLV distribution is sensitive to this type of correlation, the appropriate correlation coefficient requires careful investigation prior to incorporation into the PPLV models. Additional discussions on the appropriateness of revising this assumption in future versions of this assessment are ongoing.

E.7.3 VAPOR MODEL UNCERTAINTY

For the vapor models, the single pathway preliminary pollutant limit value (SPPPLV) is defined as the ratio of the time-averaged vapor flux for the pathway to the critical vapor flux (F_{crit}) for the receptor of concern. This definition assumes the existence of a linear relationship between the time-averaged vapor flux (FAVN) and the soil concentration (C_{soil}). This simplifying assumption holds true for the empirical relationship employed in modeling convective flux for the enclosed space model (FAVN2) (Jury et al. 1991). However, nonlinear flux/soil concentration relationships are employed in modeling the time-averaged diffusive flux (FAVN1) (EPA 1988) for both the open and the enclosed space vapor models. The use of these relationships leads to concentration-dependent (and therefore site-dependent) indirect PPLV estimates for all receptors in which diffusive vapor flux has a significant influence. Since the flux modeled in the open space vapor model is entirely diffusive and all receptors but the

commercial worker have open space vapor inhalation exposure pathways, only the commercial worker receptor has a non-site-specific indirect PPLV estimate.

The degree to which the vapor inhalation SPPPLV for a particular combination of chemical, site, depth horizon, receptor, and exposure pathway depends upon C_{soil} can be assessed by replacing C_{soil} with the SPPPLV for that combination, recalculating the flux, and taking the ratio of the SPPPLV-based SPPPLV to the C_{soil}-based SPPPLV. This can be shown mathematically as:

$$SPPPLV_{C_{soil}} = \frac{F_{crit}}{FAVN_{C_{soil}}} * C_{soil}$$

$$SPPPLV_{SPPPLV} = \frac{F_{crit}}{FAVN_{SPPPLV}} * SPPPLV_{C_{soil}}$$

$$\frac{SPPPLV_{SPPPLV}}{SPPPLV_{C_{soil}}} = \frac{\frac{F_{crit}}{FAVN_{SPPPLV}} * SPPPLV_{C_{soil}}}{SPPPLV_{C_{soil}}} = \frac{F_{crit}}{FAVN_{SPPPLV}}$$

Since the SPPPLV value is defined to be the soil concentration that would produce the critical flux (F_{crit}), the FAVN equation calculated using the SPPPLV as the soil concentration ($FAVN_{SPPPLV}$) should equal the critical flux. Correspondingly, the ratio of the critical flux to the $FAVN_{SPPPLV}$ flux should equal 1.0 if the relationship between the soil concentration and the flux were truly linear.

The further this ratio is from 1.0, the more heavily the SPPPLV is influenced by the estimate of C_{soil}, and therefore the stronger the link is between uncertainties in estimating chemical concentrations and uncertainties in the human health criteria for the vapor-inhalation pathways.

The HHRC model calculates these flux ratios, and the PPLV Results Module provides a menu option for saving all indirect (vapor model) SPPPLVs and their corresponding flux ratios to ASCII text files for diagnostic use.

The enclosed-space vapor inhalation model is based on an empirical model of convective vapor flux that was developed from studies of radon gas buildup in basements. Experts in this field have also recommended its use for modeling convective vapor fluxes form organic contaminants in soil. However, in situations with a significant contribution to convective flux from the presence of non-aqueous phase liquids, use of this model without modification could lead to underestimation of vapor inhalation risk.

As noted in Section E.5.1, the enclosed-space vapor inhalation pathway was not modeled for those portions of RMA in which construction of basements could be prohibited or severely limited, which include the Bald Eagle Management Area, areas of shallow groundwater, and the 100-year flood plain. This could lead to underestimation of vapor inhalation exposure at RMA if either of the following specific situations were to occur: (1) construction of basements in these areas, or (2) construction of above-grade structures whose air-circulation systems established a significant negative internal air pressure.

E.7.4 INTENTIONAL AND UNINTENTIONAL BIASES

Uncertainty in the data and PPLV models imply that an unbiased approach will overestimate risk part of the time and underestimate risk another portion of the time. However, it is important that risk assessments err on the side of overestimating rather than underestimating risks for given receptors. To avoid potential underestimation of risk, the goal of protectiveness required that uncertainty be countered with an increase in bias. For influential parameters in the model, the greater the uncertainty, the higher the degree of conservative bias that may be warranted. Conversely, to decrease the bias of the analysis, uncertainty must be reduced by incorporating the full extent of information provided by available empirical data and other information, including the incorporation of professional judgment.

Because uncertainty could not be eliminated for some parameters, conservative estimates for these parameters were developed. For example, recreational and casual/regulated visitors were assigned a distribution for exposure frequency that reflected conservative assumptions on visitation frequency and data interpretation to ensure protectiveness for a potentially more exposed subpopulation of neighborhood visitors. Conservatism was also incorporated into the estimation procedure for some very small data sets as described in Appendix Section B.3. Because of the conservatism incorporated into several parameters distributions for human health, the resulting PPLV distributions and site risk estimates are expected to have a conservative bias.

For parameters where data were deemed adequate, the estimation procedure was unbiased. However, unbiased estimation formulas do not guarantee an unbiased result. Unintentional biases may potentially result from any of the sources of uncertainty described above. Because such bias results from a lack of understanding of the parameter, those parameters resulting from processes that are complex and not well understood (e.g., those dependent on human behavior, ingestion of soil by adults and children) are most susceptible to unintentional biases. The impact of unintentional bias cannot be known, but may potentially increase or decrease the expected conservatism of the analysis.

E.8 HUMAN HEALTH: UNCERTAINTIES ASSOCIATED WITH PPLVs

The variation in the HHRC model parameters is reflected in the spread of the PPLV distribution. Because the uncertainty and/or variability in many key probabilistic parameters is higher for particular chemicals or for exposed populations, the resulting PPLV distributions corresponding to these chemicals and land uses have a wider spread. This section pertains to the overall variability of the PPLV distributions.

Distribution variability can be measured in a number of ways. For the purposes of comparability, the distribution variability was measured by calculating the interquantile multiplier (see Section E.8.1). Confidence intervals were also estimated (see Section E.8.2) to quantify the random sampling error associated with estimating the PPLVs.

E.8.1 COMPARISON OF DIRECT PPLV DISTRIBUTIONS

The interquantile multiplier is the 50th percentile criteria divided by the 5th percentile criteria. For example, the interquantile multiplier in the human evaluations for the commercial worker for toluene was 1.3, indicating that the 50th percentile cumulative direct PPLV was 1.3 times higher than the 5th percentile PPLV. A simple, percentile-based measure was chosen because the distributions were highly skewed (lognormality was not assumed) and tended to have one or more extreme values that would unduly influence a parametric measure such as the standard deviation or coefficient of variation. The interquantile multiplier describes variability relative to the location of the 5th percentile of the distribution, and therefore indicates the relative magnitude of variation rather than the absolute distance (e.g., a range from 10^{-4} to 10^{-2} has a relative range factor of 100, even though its absolute distance is only .0099.) In addition, the interquantile multiplier, as defined above, focuses on the variability of the lower half of the PPLV distributions, which is the portion that influences the Monte Carlo confidence intervals (discussed below) and the implications of choosing criteria based on other percentiles near the 5th percentile. A high interquantile multiplier indicates that there was high variability between the 5th and 50th percentile PPLVs.

The cumulative direct PPLVs include contributions from the soil ingestion, dermal absorption, and soil inhalation pathways. Table E.8-1 summarizes the interquantile multipliers for the direct cumulative PPLV distributions for each exposed population. For each exposed population or subpopulation, the multiplier is listed for the carcinogenic and noncarcinogenic chemicals exhibiting the lowest PPLV, the largest variability, and the lowest variability. A discussion of the influence of parameter uncertainty on the cumulative direct PPLVs is presented in the subsections that follow.

E.8.1.1 Comparison of Carcinogens and Noncarcinogens

In general, PPLV distributions for carcinogens were more widely varied than those for noncarcinogens. This trend was due to incorporation of the exposure duration parameter, which was used only in the SPPPLV equations for carcinogenic chemicals.

E.8.1.2 Comparison of Exposed Populations/Subpopulations

The PPLV distributions for the recreational visitor were substantially more varied than those for the other populations. The increased variability in the recreational visitor population was largely due to the increased variability in the time-dependent variable distributions assigned to the neighborhood visitor subpopulation. In particular, exposure frequency ranged from a few days to more than 181 days per year for the recreational visitor, while the biological, commercial, and industrial worker populations had an exposure frequency ranging within 200 to 240 days per year. Additionally, the distributions assigned to exposure duration (years per lifetime) for the two visitor populations ranged from greater than zero to 50 years (at the 97.5th percentile), while the worker exposure duration distributions ranged from greater than zero to about 20 years (at the 97.5th percentile).

The distributions of soil intake parameters did not strongly influence the relative variability of PPLVs for different land uses. Although soil ingestion and dust loading parameter distributions were the most varied for the biological worker, they were generally not dominant in influencing the direct cumulative PPLV variability, which was relatively low for all chemicals for this subpopulation. The between-population differences in the variability of time-dependent parameter distributions were larger than those for the soil intake parameters. Therefore, the time-dependent parameter distributions were the largest factor in determining which populations had the most varied PPLVs. In addition, the time-dependent variables influenced all exposure pathways, whereas soil intake parameters were unique for each exposure route.

E.8.1.3 Comparison of Chemicals

As shown in Table E.8-1, the most and least varied PPLVs were not consistent for different exposed populations. However, some chemicals consistently resulted in highly varied PPLVs, while others were consistently the lowest. For each population, the organic chemical that resulted in the most varied PPLVs were the following: aldrin, dieldrin, dichlorodiphenyltrichloroethane (DDT), dichlorodiphenyldichloroethene (DDE), tetrachloroethylene, chlordane, benzene,

trichloroethylene, and 1,2-dichloroethane. The inorganic chemicals that resulted in the most varied PPLVs were cadmium and chromium. While cadmium and chromium had relatively wide PPLV distributions, they also had narrow oral relative absorption factor $(RAF)_{RFD}$ distributions (fixed at 1.0) for noncarcinogenic endpoints and were dominated by the soil inhalation pathway. The chemicals that resulted in the least varied PPLVs were the noncarcinogens isodrin, chlorobenzene, chloroacetic acid, and toluene.

The variability in the PPLVs shown in Table E.8-1 was due to the variability in the oral and dermal RAF distributions. The relationship between the RAF parameters and the cumulative direct PPLVs is not linear due to the reciprocal contribution of the three direct pathways to obtain the PPLVs as described in Appendix Section B.1. Additionally, no chemical-specific parameters were used for the particulate inhalation pathway. Therefore, the contribution of the particulate inhalation SPPPLVs to the cumulative PPLV varied by chemical and also added to the variability in the PPLVs.

E.8.2 CONFIDENCE INTERVALS FOR ESTIMATED PPLVS

The PPLV distributions were estimated using Latin hypercube sampling, a type of Monte Carlo simulation. In general, the Monte Carlo simulations produce a random sample of the true output distribution, and are therefore influenced by the variations inherent in random sampling variation. If two different random samples of N simulations are drawn, the two distributions of model output will differ, with the difference between the distributions decreasing as N gets large. Due to limitations associated with computer memory and the difficulty associated with pooling multiple 100-sample Latin hypercube sampling runs, the simulation sample size was set at 100. The confidence interval for the 5th percentile of the PPLV distributions estimated by 100 random samples was calculated using the non-parametric quantile test (Morgan and Henrion 1991; Connover 1980) and is discussed below. This test assumes that the 100 samples are completely random and therefore overestimates the uncertainty of the Latin Hypercube sampling, which entails stratified random sampling (Morgan and Henrion 1991). Therefore, the 95 percent confidence intervals given in this section are somewhat wider than the true intervals. Moreover,

as is discussed below, Latin hypercube sampling size influences the estimated PPLV confidence intervals.

The PPLV confidence intervals must be interpreted with caution. The confidence intervals reflect the random sampling error associated with estimating the PPLV percentiles using 100 Monte Carlo samples and based on the selected parameter distributions. The uncertainty in estimating PPLV percentiles increases as the variability or uncertainty represented in the parameter distributions increases. Therefore, the estimated confidence intervals incorporate the uncertainty and variability reflected in the defined parameter distributions. For example, the uncertainty in K_{oc} values given by experimental and literature data was taken into account in the distributions, and therefore in the confidence intervals, for the PPLVs. However, the confidence intervals do not explicitly incorporate any error or bias in defining the input parameter distributions. In particular, the distributions for some parameters reflect intentional conservative bias, and the 5th percentile PPLV and confidence intervals are similarly biased. Therefore, the resultant confidence intervals are expected to give a conservatively biased representation of the probable range for the true 5th percentile PPLV (i.e., the value that would arise from a simulation where all parameter distributions are known with certainty). Confidence intervals for the PPLVs for a sample size of 100 are discussed below in Section E.8.2.1. The influence of larger Latin hypercube sampling sizes on PPLVs is described in Section E.8.2.2.

E.8.2.1 PPLV Confidence Intervals for a Sample Size of 100

To obtain confidence intervals using the non-parametric quantile test, a sample size of 100 was ordered from smallest to largest so that the 100th PPLV (PPLV[100]) was the largest sample. For a sample size of N=100, the test implies that there is a 95 percent certainty, given the selected parameter distributions, that the true quantile p (e.g., p=0.05) of a population lies within the following confidence intervals (based on actual sample values):

$$\text{Lower Bound} = \text{PPLV}\left[\text{floor}\left(100*p - 1.96\sqrt{100*p*(1-p)}\right)\right] \quad (1)$$

$$\text{Upper Bound} = \text{PPLV}\left[\ \text{ceiling}\left(100 * p + 1.96\sqrt{100 * p * (1-p)}\right)\ \right] \qquad (2)$$

The multiplier 1.96 is the deviation enclosing 95 percent of the standard unit normal. Floor and ceiling refer to rounding down or up, respectively, to the next integer. This integer was then used to select the ordered PPLV sample values to define the lower and upper bounds.

For N=100 and p=0.05, the integer for the lower bound equals the floor of 0.73, which is 0, indicating that the lower 95 percent confidence limit for the 5th percentile cannot be calculated exactly when N=100. However, an approximate lower bound was obtained by rounding to the nearest integer, 1, rather than rounding down. The upper 95 percent confidence interval was approximated by the 10th ordered PPLV.

Tables E.8-2 and E.8-3 present the 95 percent confidence intervals for the 5th percentile cumulative direct PPLV for each chemical for the biological worker and industrial worker, respectively. Based on the selected parameter distributions, the true 5th percentile is most likely to fall near the estimated 5th percentile and is least likely to fall toward the boundaries of the interval. The estimated confidence intervals were roughly symmetrical. The percentage deviation from the 5th percentile, defined as 100 * (one-half the width of the confidence interval/5th percentile PPLV), is also presented in Tables E.8-2 and E.8-3. For example, the carcinogenic aldrin confidence interval for the biological worker is ± 45 percent (Table E.8-2). Based on the developed parameter distributions, this indicates that there is 95 percent confidence that the true 5th percentile cumulative direct PPLV for aldrin would be within ± 45 percent of the estimated 5th percentile cumulative direct PPLV.

For carcinogenic PPLVs, the biological worker subpopulation (described in Section E.8.1 as having the least variability in PPLV distributions has the smallest confidence intervals, with three-fourths of the chemicals exhibiting a percentage deviation less than approximately 49 percent. The percentage deviations for all chemicals ranged from 33-52 percent (Table E.8.2).

The industrial worker population, which had higher variability in PPLV distributions compared to the biological worker, also had larger confidence intervals, with percentage deviations ranging from 49 to 129 percent (Table E.8-3) with three-fourths of the chemicals exhibiting a percentage deviation less than 69 percent.

For chemicals with noncarcinogenic endpoints, the percentage deviations were generally lower. For the biological worker, deviations ranged from 12 percent to 45 percent (Table E.8-2), and three-fourths of chemicals had deviations below 30 percent. For the industrial worker, deviations ranged from 15 to 53 percent (Table E.8-3), with three-fourths of the chemicals below 31 percent. Because risk is proportional to 1/PPLV, the confidence intervals for the 95th percentile risk can be calculated from the intervals presented for the 5th percentile PPLV. A higher PPLV based on the upper confidence intervals would equate to a reduction in estimated site risks, while a lower PPLV based on the lower confidence interval would result in an increase in estimated site risks.

E.8.2.2 Influence of Larger Latin Hypercube Sampling Sizes on PPLVs

Larger Latin hypercube sampling sizes imply narrower confidence intervals (i.e., the PPLV sample values chosen for the bounds are closer together). This decrease in confidence interval width could be calculated directly by running the PPLV model with larger Latin hypercube sample sets. However, since the amount of time required to do this is unreasonably long, a reasonable approximation of the larger sample confidence intervals was made using the 100-sample set. This approximation was made for sample sizes of N=200 and N=500 and may be explained as follows.

The lower and upper bounds for a Latin hypercube sampling size of N=200 are given by the 4th- and 16th-ordered cumulative direct PPLVs out of 200 PPLVs (as calculated using equations (1) and (2)). Since the 100-sample set is one-half the size of the 200-sample set, the 4th- and 16th-ordered direct PPLVs out of 200 are estimated by the 2nd- and 8th-ordered PPLVs out of 100. Similarly, the lower and upper bounds for an Latin hypercube sampling size of N=500,

given by the 15th- and 35th-ordered cumulative direct PPLVs out of 500, are estimated by the 3rd- and 7th-ordered cumulative direct PPLVs out of 100.

These approximate larger sample confidence limits cannot be used to imply more certainty in the PPLVs than is currently warranted with the 100-sample set. However, the comparison of their width to that of the intervals, based on N=100, indicates the approximate effect of increasing the sample size. The widths of the 100 sample confidence intervals were compared to the approximated widths of the larger confidence intervals for the biological worker using equation (3) below:

$$\textbf{Percent Decrease in Confidence Interval} = (1 - CI_N / CI_{100}) * 100 \qquad (3)$$

where: CI_{100} = Confidence interval of the 100 sample set

CI_N = Confidence interval of the larger sample set

An approximated sample size of N=200 reduced the observed confidence interval widths by 3 to 78 percent of the widths observed at N=100, depending on the chemical. Using an approximated sample size of N=500 for the same exposed population reduced the confidence intervals by 14 to 82 percent of the 100-sample widths.

As stated above, the confidence intervals based on a sample size of N=100 were less than about ± 49 percent for three-fourths of the carcinogenic cumulative direct PPLV distributions for the biological worker. Doubling this sample size reduces the confidence intervals so that most are below about ± 38 percent, but entails a substantial cost in computational speed, memory requirements, and data storage space. Increasing the sample size five times to N=500 again reduces the confidence intervals so that most are below 26 percent, but this also entails the greater tradeoffs in processing time and memory.

E.9 ECOLOGICAL HEALTH: UNCERTAINTIES ASSOCIATED WITH EXPOSURE

E.9.1 EXPERIMENTAL DESIGN

Despite the relative abundance of site-specific field data to characterize ecological risk at RMA, the need to work with data from sampling programs designed for other purposes impeded the estimation of exposure soil concentrations and biomagnification factors (BMFs). The bulk of the soil data available for calculating <ESC>s and BMFs was collected in the RI sampling program. The intent of RI sampling was to characterize soil contaminant concentrations across RMA. More intense sampling was performed in areas with higher and more variable contaminant concentrations, and less intense sampling was performed in areas with relatively low homogenous contaminant concentrations (EBASCO 1992). The bulk of the tissue data was collected in the Comprehensive Monitoring Program (CMP). The CMP was designed to monitor tissue concentrations in "problem areas" on RMA, i.e., those areas with relatively high, variable contaminant concentrations (RLSA 1990). Because the soil and tissue data were not collected to estimate BMFs, there was no effort to design soil sampling to best estimate exposure soil concentrations for individuals from which tissue samples were taken, or even to collocate tissue and soil samples. In addition, the sampling focused on those areas of RMA most likely to be changed by site remediation, providing little data to assess biomagnification on the areas of RMA that most closely resemble anticipated future conditions.

E.9.2 TARGET SPECIES

Target species sampled on RMA were chosen from species that best represent the uptake of contaminants from environmental media and the subsequent contaminant transfer, via food consumption, through food chains to top predators. Most of these target species were selected and sampled during the Biota RI (ESE 1989) and Biota CMP (RLSA 1990). The remainder were later selected and sampled during the ERC specifically to complete missing links in existing food chains or to add entire new food chains to the RMA food-web model.

Many target species were chosen based on known food habits. For example, the killdeer was chosen as the most available representative of shorebirds because it is known for its habit of

probing sediments for food; the prairie dog was chosen because of its extensive exposure to soil and because it is an important food item for the bald eagle; and grasshoppers were chosen because they are herbivores and because they are a choice food item for American kestrels. The selected target species were assigned to the more general trophic boxes of the RMA food web to represent that general feeding category. For example, deer mice were chosen as a target species and placed in the trophic-level box named small mammals and black-tailed prairie dogs and desert cottontails were chosen as target species and placed in the trophic-level box named medium mammals. Several assumptions and uncertainties accompanied the creation of the food web as described below.

To evaluate the impact of the choice of target species on exposure-scenario uncertainties, the data were used for two distinct purposes to characterize risk to target species, and to characterize risk to a species that preys on the target species' trophic box.

E.9.2.1 Characterizing Risk to the Target Species

A potential source of error in the ERC is the use of BMFs defined at the trophic level to characterize risk at the species level. If a trophic box has more than one target species, and the different target species have different mean BMFs, then the trophic box mean BMF calculated from the target species data will fall between the target species' BMFs, biasing target species tissue concentration predictions. There are a number of obvious sources of species variability (i.e., differences across species), both physiological and behavioral. Species variability in the mechanisms that collectively cause biomagnification does not necessarily cause species variability in BMFs. For example, a higher average rate of chemical ingestion in one target species may be offset by a lower average rate of depuration in another target species in the same trophic box, so that the net result is similar mean BMFs in the two target species despite the species variability in behavior and physiology. Individual variability within a species, resulting from such differences as age, sex, and seasonal feeding patterns, is a source of variability in measured tissue concentrations, but not in BMF, since BMF is a tropic-box average.

E.9.2.2 Characterizing Risk to a Predator

The impact of the selection of target prey species on exposure scenario uncertainties is determined by the degree to which the trophic box mean BMF calculated from the target species data accurately estimates the average BMF in the portion of the predator's diet that comes from the target species' trophic box. In other words, since the contribution of a trophic box to its predator's risk is determined by the average prey tissue concentration, the loss of information about species variability when a trophic box mean BMF is calculated is not a concern. In fact, uncertainty about the trophic box mean BMF is a better measure of uncertainty in risk to the predator; species mean BMF variability is likely to overestimate uncertainty in risk to the predator. In the ERC, target species data averaging to calculate a trophic box mean BMF assigns relative weights to target species based on their estimated importance as prey. Incorrect specification of weights is a potential source of bias in predator risk estimates.

E.9.3 EXPOSURE PATHWAYS ASSOCIATED WITH ECOLOGICAL RECEPTORS

Exposure pathways—the means and routes of contaminant uptake and transfer—were selected to include the predominant pathways of exposure believed to exist at RMA. Those selected for the food-web model include food consumption, dermal exposure to surface water by all aquatic trophic boxes, ingestion of water by some terrestrial trophic boxes, and sediment and soil ingestion by some aquatic and terrestrial trophic boxes. Exposure pathways excluded from the food-web model include inhalation of contaminant vapors and particulates and dermal exposure to contaminants from soil contact. These exposure pathways are implicitly contained in the calibrated BMF because the measured tissue concentrations used to calibrate are the result of cumulative exposure by all pathways.

E.9.4 EXPOSURE CONCENTRATIONS

This section discusses uncertainty in <ESC>s used to calculate terrestrial risk. Aquatic risk was estimated directly from observed tissue concentrations and therefore was not based on exposure concentrations in aquatic media. Terrestrial tissue concentration, dose, and risk are theoretically dependant on exposure soil concentration: i.e., the concentration in soils which is bioavailable

and accessed by an individual during exposure activity. The exposure soil concentration is, for all practical purposes, unmeasurable in the field, and therefore it is represented by exposure area soil concentration, i.e., the average soil concentration in a specified depth profile within a circular species specific exposure area. These definitions lead to two types of uncertainty in applying ESC to estimate risk. "Representation uncertainty" refers to the uncertainty in representing the exposure soil concentration by exposure area soil concentration. "Estimation uncertainty" refers to the uncertainty in estimating the exposure area soil concentration based on available data. Sources contributing to representation and estimation uncertainties are discussed below.

The empirical constant used to relate exposure area soil concentration to tissue concentration is termed the biomagnification factor (BMF). BMF is therefore defined based on the variable exposure area soil concentration and not on actual exposure soil concentration. A BMF value determined purely from literature data will describe the relationship between tissue concentration and a different quantity than ESC, and therefore may create a bias if it is used with ESC to predict risk for the Arsenal.

E.9.4.1 Representation Uncertainty

Representation uncertainty explains the difference between true exposure concentration for an individual and the exposure area concentration for a typical (mean) individual. Unfortunately, representation uncertainty is for all practical purposes unquantifiable and irreducible, because the detailed information on individual organisms (and their prey) required for its calculation cannot be obtained. The following sources of error result in discrepancies between modeled and actual exposure conditions and thus contribute to representation uncertainty.

(1) Representation of bioavailabilty: Potential discrepancy exits between soil concentrations measured in the soil sample units (e.g. 1 foot cores, 0-2" surficial samples) and the actual available soil concentration, which is affected by chemical bioavailability in the soil and individual exposure activities such as digging and consumption of prey items exposed to different soil depths.

(2) Representation of spatial/temporal pattern of exposure: Potential discrepancy exists between the assumed circular exposure range with uniform areal weighting and the actual spatial/temporal exposure pattern for a given individual.

The potential for error in representing spatial/temporal exposure pattern is believed to be highest for species with large exposure ranges such as the mourning dove. Actual use of large exposure ranges is likely to be non-uniform and to depend on prey distribution. The definition of exposure area soil concentration assumed uniform areal weighting due to the lack of information on the present and future distributions of prey density across RMA. The exposure range assumptions, while simple, represent a substantial improvement over past assumptions regarding exposure.

E.9.4.2 Estimation Uncertainty

Estimation uncertainty explains the differences between the true exposure area soil concentration (ESC) in a given area or for a given individual, and the value (<ESC>) estimated based on available data. The estimation uncertainty is thought to be a smaller contributor to overall ESC uncertainty than representation uncertainty. The following sources of error contribute to this estimation uncertainty.

(1) Estimation of spatial distributions of RMA soil concentrations: Potential discrepancy exists between the interpolated estimates of soil concentration, based on the RMA soil samples, and the true distribution of soil concentrations.

BCRL data and sparse sampling contribute to this uncertainty. As described in Appendix Section C.1.4, the inverse distance squared algorithm is used to replace BCRLs with estimates and to interpolate soil concentration data onto a 100 foot grid, from which <ESC> was then calculated. The inverse distance squared algorithm is a common, easily understood method that assumes a spatial structure in the soil concentration data. More sophisticated methods for analyzing and modeling spatial structure were considered and rejected by the EA Technical Subcommittee for a variety of reasons including potential

for misuse, lack of significant clustering in the soil data, and analytical difficulties in incorporating BCRL measurements in more sophisticated spatial data analyses.

(2) Estimation of exposure range size: Potential discrepancy exists between the assumed exposure range radius and the radius most representative of a given species.

Representation error is involved in representing individual exposure activities as being uniformly distributed over a constant (mean) circular exposure range. In contrast, estimation error is involved in estimating the appropriate radius for this mean exposure range. Exposure range radii were derived from analysis of literature studies, RMA observations, and professional judgement. The size of exposure range has a substantial impact on the variation in risk estimates. Smaller exposure ranges imply higher variability in risk estimates because individuals of the species are expected to be exposed to more extreme (high and low) mean concentrations than individuals of a species with a large exposure range.

(3) Estimation of exposure range center: Potential discrepancy exists between the assumed exposure range center for a given biota sample and the center of the actual exposure range which best represents this sample individual's exposure.

In estimating ESC for a given sample, each sample is assumed to have an exposure range centered at its sample collection location. In contrast the sample individual may have been found near the edge of its actual exposure range, and thus was exposed to possibly different levels contaminants than assumed. Errors in estimating the center of the exposure range will result in the largest ESC errors in areas where the concentrations are changing rapidly, so that a small change in exposure range results in a large change in ESC. Biota samples were often taken from such areas of transition and therefore this error contributes substantially to the overall uncertainty in ESCs associated with specific biota samples. This uncertainty pertains only to use of ESC to estimate BMF. It does

not influence ESC when used as the exposure concentration for estimating risk because, in this case, ESC is characterized for a given location and not a specific individual.

E.9.5 ADDITIVITY OF HAZARD QUOTIENTS

Toxicological effects from multiple chemicals are assumed to be additive, consistent with the risk assessment procedures used for human health. This assumes independence of action, i.e., no net synergistic or antagonistic effects, since these effects are poorly understood. HQs for COCs were added to estimate total risk.

E.10 ECOLOGICAL HEALTH: UNCERTAINTIES ASSOCIATED WITH TOXICITY ESTIMATES

Maximum allowable tissue concentration (MATC) and toxicity reference value (TRV) uncertainty was incorporated quantitatively by use of UFs (see Appendix Sections C.2.4 and C.2.6) and is discussed qualitatively here.

The UFs were applied to add a margin of safety to the toxicity measures. The UF protocol included factors to account for four categories of uncertainty: intertaxon variability, study duration, toxicity effect levels (study endpoints), and other modifying factors (including nine subcategories) that were multiplied to arrive at the total estimated uncertainty. This use of multiplicative strings of UFs assumes that everything will go wrong at once (Suter 1993), and so has a high potential to be overly conservative.

MATCs, TRVs, and UFs were selected by the Organizations and State (OAS) by consensus. However, some of the values are better characterized as compromise rather than consensus values. In particular, there was disagreement about the MATC of 0.19 for dieldrin in mammals, with some parties preferring a lower and others a higher MATC. In addition, it is noted that the pre-UF MATC and TRV for small birds are considered highly uncertain. Consequently, the MATC and TRV uncertainty factors are high, and the final MATC and TRV (0.14 mg/kg-bw/day

and 0.003 mg/kg-bw/day, respectively) may be unrealistically low. The small bird DDE/DDT risk characterization was based on the TRV of 0.003 mg/kg-bw/day.

In addition to the uncertainty incorporated in the UFs are potentially unrecognized or unquantifiable sources of uncertainty. These include the following:

- Representativeness of toxicity endpoint tissue concentration data from one species relative to other species in the trophic box
- Differences in metabolic rate, body size, and physiology between test and target species
- Differences in feeding habits and behavioral patterns in test vs. target species
- Differences in life stage of the organisms tested vs. those exposed
- Seasonal differences in response to toxicants (e.g., "fat" versus "lean" times)
- Difficulty in adequately estimating exposure concentrations (including environmental variability in time and space)
- The possibility that exposed organisms may avoid, or be attracted to, contaminated media (e.g., pesticide-debilitated prey) and so may not show effects seen in laboratory tests (Suter 1993)
- Inability to quantify the other stresses that biota may face (e.g., climate, food supplies, background levels of toxicants, habitat disturbance, and other anthropogenic causes)
- The possibility that exposure pathways, in addition to ingestion, are significant

- The fact that there are no standard measures of effect, patterns of dosing, durations of exposure, etc., so comparison across studies/ecosystems is obscured or confounded

E.11 ECOLOGICAL HEALTH: UNCERTAINTIES ASSOCIATED WITH EXPOSURE PARAMETERS

Sources of parameter uncertainty that contribute to overall uncertainty about risk include data representation error, extrapolation error, measurement error, uncertainty due to small data sets, and uncertainty associated with parameter correlation. These sources of uncertainty are discussed below.

E.11.1 DATA REPRESENTATIVENESS

The assimilation fraction, depuration, and bioconcentration factor values reported in the literature were measured in laboratories under temperatures and other conditions that may not reflect the conditions in RMA lakes. Data appropriate for specific RMA populations were used whenever possible to reduce this bias. Further, the use of biota samples from RMA to calculate observed biomagnification factors allowed calibration of the terrestrial food-web model to site-specific conditions and provided a means of minimizing error in data representation. It is not possible to determine the extent to which data representativeness in the current evaluations imparted uncertainty to the results.

E.11.2 EXTRAPOLATION ERROR

When no data directly describing a parameter were available, the parameter distribution was extrapolated based on additional information and best professional judgment. For the ecological parameters, extrapolations due to a lack of data were primarily between trophic boxes. For example, aldrin/dieldrin depuration values in the literature for pheasant, chicken, pigeon, and turkey were used as the depuration value for bald eagle, great blue heron, water bird, and shorebird. The assimilation fraction used for most trophic boxes was based on best professional judgment.

All extrapolation assumptions are documented in Appendix Section C.2. The impact of extrapolation error on the input parameter distributions and resulting site risks cannot be determined quantitatively. However, since a goal of such extrapolations was to avoid underestimation of risk, they are expected to impart a conservative bias to the results.

E.11.3 DATA MEASUREMENT ERROR

The individual data points within each parameter distribution also embody some degree of experimental measurement error. For example, the chemical-specific parameters (e.g., K_{oc}) used in estimating water concentrations were influenced by errors in analytical chemistry techniques. The bioconcentration factor or bioaccumulation factor values reported from laboratory studies assume a steady state that may or may not have been achieved. The impact of measurement error on the BMF distributions and risk estimates is unknown.

E.11.4 SMALL DATA SETS

Small sample sizes contributed to uncertainty in distribution mean and standard deviation, as well as distribution form.

For the food-web model input parameters, the need to develop parameter values for a multiplicity of trophic boxes at times resulted in a small number of data points for any one trophic box. Further detail is provided in the parameter-specific sections of Appendix Section C.2. The impact of small data sets on the risk estimates is not known with certainty and could lead to overestimates of risks for some chemicals and under-estimates for others.

E.11.5 CORRELATION BETWEEN PARAMETERS

Another source of uncertainty is the potential correlation between different model parameters. For example, the assimilation fraction, depuration rate, and bioconcentration factor may be correlated because these parameters may depend on some of the same physiological attributes of a given individual. No data were available to quantify this type of correlation among input parameters.

E.11.6 INTENTIONAL AND UNINTENTIONAL BIASES

To avoid potential underestimation of risk, the goal of protectiveness required that uncertainty about risk be countered with a conservative bias in risk estimation. For influential parameters in the model, the greater the uncertainty, the higher the degree of conservative bias that may be warranted. For example, UFs were used to ensure that the toxicity criteria (MATC and TRV) were protective. Conversely, to decrease bias, uncertainty must be reduced by incorporating the full extent of information provided by available empirical data and other information, including the incorporation of best professional judgment (e.g., the selection of literature data for various model parameters).

E.12 ECOLOGICAL HEALTH: UNCERTAINTIES ASSOCIATED WITH BIOMAGNIFICATION PARAMETERS

E.12.1 INTRODUCTION

Biomagnification factor estimation is one of three major steps in the analysis required to calculate the terrestrial HQ, which estimates average risk to a biota population from exposure to a soil contaminant. These three tasks are:

- Estimation of exposure area soil concentrations (ESCs)
- Estimation of biomagnification factors (BMFs)
- Estimation of maximum allowable tissue concentrations and toxicity reference values (MATCs and TRVs)

The focus of Appendix E.12 is on analysis of uncertainty in BMF estimates. Uncertainty in estimating ESCs is also discussed in Appendix E.12 (Section E.12.4.2) because <ESC> is an input to the calculation of BMF by the Army, EPA, and Shell approaches. Information about the selection of exposure ranges used to calculate <ESC>s is presented in Appendix Section C.2.5. Uncertainty in estimating MATCs and TRVs is discussed in Appendix, Sections C.2.4 and C.2.6, respectively, as well as Appendix Section E.10.

The biomagnification factor appears in both tissue- and dose-based formulas for estimating the hazard quotient:

$$HQ_i(x,y,t) = \frac{\langle ESC \rangle_i(x,y,t) \cdot BMF_i}{MATC_i}$$

$$HQ_i(x,y,t) = \frac{R_i \cdot BMF_i \cdot \langle ESC \rangle_i(x,y,t)}{BAF_i \cdot TRV_i}$$

where: $HQ_i\ (x,y,t)$ = Hazard quotient for species i at point (x,y) at RMA, based on estimated exposure area soil concentration at that point at time t

$\langle ESC \rangle_i(x,y,t)$ = Estimated exposure area soil concentration for an individual of species i living at point (x,y) at RMA based on interpolated contaminant soil concentrations across its home range at time t

BMF_i = Biomagnification factor

$MATC_i$ = Maximum allowable tissue concentration

TRV_i = Toxicity reference value

R_i = Feed rate coefficient

BAF_i = Bioaccumulation factor

Appendix Section C.1.4.1 provides a detailed definition of <ESC>. Sections C.2.2, C.2.3, C.2.4 and C.2.6 define BAF, MATC, R, and TRV, respectively.

Six methods have been investigated for estimating biomagnification factors at RMA. These methods are:

- Three alternative approaches for computing biomagnification factors from RMA tissue and soil concentration data
- Two alternative methods of computing biomagnification factors from a food-web model
- Calculating biomagnification values from tissue and soil concentration data reported in the literature (for aldrin/dieldrin and DDE/DDT only)

All six of these BMF estimation techniques involve quantifiable uncertainty. The results of the alternative estimation techniques vary considerably.

The variability in BMF estimates calculated by different methods is significantly greater than the uncertainty within any particular method, as can be seen in the box plots in Figure E.12-1. These plots, which are for owl aldrin/dieldrin BMF distributions, are characteristic of other trophic boxes. Expected values and standard deviations of aldrin/dieldrin mean BMF estimates by all six methods for all trophic boxes are reported in Table E.12-1, and comparable results for DDE/DDT, endrin, and mercury, respectively, are given in Tables E.12-2 through E.12-4.

The distributions shown in Figure E.12-1 are calculated by the four principal estimation techniques used in the BMF uncertainty analysis: the Army and Shell collocated distributions approaches, the modified paired data approach (also referred to as the EPA approach), and the calibrated prey BMFs referred to as the Army approach. The Army approach combined the Army collocated distributions BMF with a calibration procedure to generate the Army calibrated BMFs, which are deterministic coefficients. A fifth approach, food-web modeling using prey BMFs derived from literature values (the litcalc approach) gives a sample range of 4 to 671 for the owl aldrin/dieldrin BMF, with a mean of 55 and sample median of 30. The ranges of BMF estimates presented in Tables E.12-1 through E.12-4 imply a correspondingly broad range of tissue concentration and ecological risk predictions.

The remainder of Appendix E.12 is organized as follows. Sections E.12.2 and E.12.3 provide pertinent background information. Section E.12.2 summarizes the nomenclature used in this appendix, including statistical terminology and the nomenclature used to describe BMFs calculated by alternative methods. Section E.12.3 provides a brief conceptual overview of sources of variability and uncertainty in BMFs. Sections E.12.4 and E.12.5 report the results of BMF uncertainty analysis. Section E.12.4 describes uncertainty about BMFs calculated from field data. It focuses on the key statistical issues in estimating BMFs from RMA field data, including interpretation of below certified reporting limit (BCRL) data, grouping of chemicals (i.e., aldrin/dieldrin and DDE/DDT), and the problem of small sample sizes. This section also discusses sources of uncertainty in ESC, including spatial interpolation of soil concentration data; uncertainty due to species aggregation; uncertainty about tissue and estimated exposure area soil concentration correlation; and uncertainty about the shapes of tissue and estimated exposure area soil concentration distributions. Section E.12.4 also discusses the issue of irreducible uncertainty in any of the three field BMF methods. Section E.12.5 discusses the specific advantages and disadvantages of the key statistical assumptions that differentiate the Army, EPA, and Shell BMF methods.

E.12.2 NOMENCLATURE

E.12.2.1 Alternative Definitions of BMF

Any parameter that quantifies the amplification of a contaminant's concentration from the initial source (e.g., sediment, soil, or water) to a specified target biota receptor or trophic box is referred to as a BMF. Several alternative BMFs have been used in the ERC. This section introduces the nomenclature required to understand why alternative BMF definitions are possible, and why they give different results:

- Contaminant—These may be individual or grouped chemicals. The contaminants for which BMFs are computed in the ERC are aldrin/dieldrin, DDE/DDT, endrin, and mercury.

- Database—The ERC uses literature and RMA databases to compute BMFs. Databases include chemical concentration data for the initial source of contamination, prey tissue (intermediate source), and in some cases, target tissue. In addition, the databases include parameters describing uptake from the initial source to intermediate sources, between intermediate sources, and from intermediate sources to the target. Different data sets may arise from the same set of measurements when different interpretations of the measurements are applied (e.g., different methods for interpreting BCRL data.

- Estimation technique—Derivation of a BMF from a particular database requires statistical estimation of parameters describing the measured population from the sample represented in the database. The use of alternative estimation techniques on the same database can lead to alternative BMFs. For example, BMFs calculated using alternative collocated distributions techniques yield different BMFs from the same database.

- Target—The target is the population for which the BMF is being estimated. It may be all individuals of a species or group of related species within a particular geographical and temporal boundary, some subset of individuals (e.g., those within a specified age class), or some aggregation of individuals. BMFs used in the ERC are mean BMFs; they characterize average biomagnification in a group of individuals rather than biomagnification in each individual. In other words, a mean BMF characterizes biomagnification in an "average" individual. Three interchangeable forms of notation are used for mean BMFs. Mean BMFs are sometimes explicitly noted through the use of an overbar ($\overline{BMF}$) or the prefix "mean" (mean BMF). The overbar and mean prefix are used interchangeably because not all computing environments support the overbar. Finally, unless otherwise noted, all BMFs are mean BMFs; the overbar and mean prefix are often excluded since mean BMF is the default.

Alternative BMFs are defined through the use of suffixes or subscripts on the root variable name BMF, and through the context in which the BMF is used. Suffixes and subscripts are used

interchangeably because not all computing environments support subscripts. The contaminant, initial source, and target are determined from the context. The suffix or subscript identifies the database. If multiple estimation techniques are applied to the same database, the estimation technique is typically assigned a name that is used in the context of the BMF (e.g., the text might specify "BMF_{obs} calculated by the Shell collocated distributions approach").

Specific BMF nomenclature used in the ERC includes the following:

- BMF_{obs}—This is a mean BMF calculated from RMA field data using one of three approaches, Army, EPA, or Shell. The development of BMF_{obs} is discussed in Section C.1.6.1.

- BMF_{EPA}—This is a mean BMF calculated from paired tissue and soil concentration data using the EPA modified paired data approach, described in Section C.1.6.1.2.

- BMF_{Shell}—This is a mean BMF calculated from collocated tissue concentration and <ESC> distributions by the Shell collocated distributions approach, described in Section C.1.6.1.2.

- $BMF_{lit/model}$—This is a mean BMF calculated using a literature-derived bioaccumulation factor (BAF) for the target population and BMFs for the target's prey derived from a database that includes literature and field data. The development of $BMF_{lit/model}$ is discussed in Section C.1.6.2.

- $BMF_{lit/calc}$—This is a mean BMF calculated using a literature-derived bioaccumulation factor (BAF) for the target population and BMFs for the target's prey derived from a literature-only database. It differs from $BMF_{lit/model}$ only in that a different database is used to calculate prey BMFs.

- BMF_{model}—This is the Army's calibrated mean BMF, calculated using the BMF_{obs} and $BMF_{lit/model}$ databases. The development of BMF_{model} is discussed in Section C.1.6.3.

- BMF_{lit}—This is a BMF taken directly from the literature. These BMFs may or may not represent a mean value.

E.12.2.2 Statistical Terminology

Definitions of statistical terms used throughout the discussion of uncertainties associated with biomagnification parameters include the following:

- Latin hypercube sampling—A stratified sampling technique used in Monte Carlo analysis. Stratified sampling techniques tend to force convergence of a sampled distribution in fewer samples.

- Mean—The expected value of a random variable. The mean represents the center of its probability distribution in the sense that it is the average of all possible values of the variable, weighted by their probabilities of occurrence.

- Monte Carlo analysis—A technique by which a model is solved many times with different input values, with the intent of getting a representation of all possible scenarios that might occur in an uncertain situation. The term Monte Carlo sampling refers to simple random sampling of the distributions of model input parameters.

- Pearson correlation coefficient—An estimator of the strength of the linear relationship between two parameters. The Pearson correlation coefficient for the variables x and y is defined as the covariance of x and y divided by the product of the standard deviations of x and y. The Pearson correlation coefficient is used to describe correlation between ln(TC) and ln(<ESC>) distributions to calculate BMF_{obs}. It is the most commonly used description of correlation.

- Point estimate—A single-valued estimate of an unknown.

- Population—The set of all objects or individuals from which a sample is drawn or variable parameter or to which and inference is made.

- Standard deviation—A measure of the spread in a probability distribution. Sample standard deviation is a measure of the spread in a sample. It is approximately the square root of the average squared distance of a sample value from the sample average.

- Standard error—The standard deviation of the distribution of a sample mean. Estimated standard error is sample standard deviation divided by the square root of the sample size.

- Uncertainty—A qualitative or quantitative description of one's current beliefs about the range and relative likelihoods of possible values for a parameter that actually has a single, true value. If a parameter is truly variable (i.e., its "true" value varies), then uncertainty is the remaining ambiguity about the parameter's true value after the variability has been accounted for. An over-confident assessor can be certain, and also be wrong, about the

true value of a parameter, i.e., if the assessor incorrectly assigns a probability of 1 to a parameter value.

- Variability—A qualitative or quantitative description of how a multivalued parameter's true value changes over time and/or location.

- Variance—A measure of the spread in a probability distribution. It is specifically the average squared distance of a realization of the random variable from its expected value. The variance is the square of the standard deviation.

E.12.3 SOURCES OF VARIABILITY AND UNCERTAINTY IN BIOMAGNIFICATION PARAMETERS

The term "biomagnification parameter" encompasses bioaccumulation, bioconcentration, and biomagnification factors (BAFs, BCFs, and BMFs, respectively). The development of biomagnification parameters is discussed in Appendix Section C.2.2. Bioaccumulation is defined as the amplification of a contaminant's concentration from the immediate source (either prey or ingested sediment, soil, or water). Bioconcentration refers to the portion of biomagnification in an aquatic trophic box due to direct uptake from water. Biomagnification is the total amplification of a contaminant's concentration from the initial source (e.g., sediment, soil, or water) to a specified target species or trophic level. Multiple biomagnification parameter estimates exist for each trophic box/COC combination in the ERC model. Even within a particular trophic box/COC, biomagnification parameter comparisons are difficult because of differences in sampling populations, measurement techniques, and reporting. The remainder of this section focuses on BMFs since the BMFs are used to estimate risk. BAFs and BCFs are a portion of the data used to calculate BMFs. The development of sources of uncertainty in literature-derived BAF and BCF distributions is documented in Appendix Section C.2.2. Variability across studies and inter-species variability are the dominant sources of uncertainty in BAF and BCF distributions for RMA food-web model trophic boxes.

Ultimately, the uncertainty in the estimated BMFs depends on the uncertainty in the RMA data and their interpretation. The literature that is not site-specific becomes more useful as the uncertainty in the site data increases. However, in order to be considered reasonably dependable, estimates based heavily on such data must in some way pass a reality check that ideally would depend on the ability to predict site-specific phenomena. This is because the numerical value of the BMF depends on its definition as the empirical constant relating ESC to biota tissue concentrations at RMA. For example, if soil concentration in the top 1 ft of soil tends to be lower than in the top 1 inch, then the BMF for the top 1 ft of soil is lower than the BMF corresponding to the top 1 inch of soil.

The RMA databases were used to investigate the relationship between biota tissue concentration (TC) and estimated exposure area soil concentration (<ESC>) in two basic ways:

- RMA Collocated Data—A model was constructed to predict the ESC associated with each TC data point, resulting in a paired data set that was used in both the paired and collocated distribution approaches for estimating BMF.

- RMA Mapped Data—The maps of the TC data and the predicted TC values implied by the RMA-wide soil data set were compared. The predicted TC map represents the model of TC=BMF*<ESC> for a particular BMF value, (i.e., the map shows the predicted TC for a hypothetical individual that experiences the estimated ESC at a given location.

The RMA collocated data include assumptions (i.e., the prediction of ESC for each TC value) that both allow and affect the estimation of BMF. Therefore, the RMA collocated data were used to estimate BMF_{obs}. The comparison of RMA mapped data does not force the TC data to be associated with any given <ESC> value and therefore cannot be used to estimate BMF. In the map comparisons, the relatively accurate measurements represented in the TC data are displayed without any assumptions, rather than combined with an uncertain ESC estimate to get a BMF. The question is then left open as to which <ESC> values occurring near an individual TC sample,

represent a possible exposure for an individual. For example, TC samples with relatively high and low concentrations are sometimes found close together at the edge of a hot spot. In cases where the high and low TCs are inversely associated with soil concentrations (i.e., the high TC is located just outside the hot spot and the low TC is located just inside the hot spot), no simple ESC-based model can predict these TCs from their collocated <ESC>s. However, the mapped data provide additional information that supports the appropriateness of an ESC-based model: the model predicts a sharp gradient in ESC at the edge of the hot spot and therefore is able to predict the observed occurrence of both high and low TCs in this area. Therefore, the mapped data provide a reality check for predicted BMFs, regardless of their dependence on the RMA collocated data and/or literature values. The mapped data were used in the Army method to calibrate the BMF.

E.12.4 SOURCES OF UNCERTAINTY IN SITE-SPECIFIC BMFs

The following section discusses uncertainties in site-specific BMFs. Sections E.12.4.1 through E.12.4.4 cover uncertainty about TC distributions, <ESC> distributions, uncertainty due to species aggregation, and correlation of TC and <ESC> distributions, respectively. Section E.12.4.5 discusses limitations in the ability to predict tissue concentrations in individual biota from mean BMFs.

E.12.4.1 Uncertainty About TC Distributions

In developing distributions for TC for each trophic box, uncertainty was introduced through the following processes: interpretation of BCRL data, summation of the two chemical pairs aldrin/dieldrin and DDE/DDT, and estimation based on small sample sizes.

E.12.4.1.1 Interpretation of BCRL data

Table E.12-5 presents information regarding sample size, number of BCRL data, and selected BCRL replacement method for each trophic box. The proportion of BCRL data for most of the trophic box/chemical combinations was high. In such cases the interpretation of the BCRL data through different statistical methods could have a significant impact on the fitted distribution;

therefore, the uncertainty due to BCRL data was relatively large. The arithmetic estimators used to fit lognormal distribution parameters were chosen because they reduced the impact of uncertainty in BCRL data.

Several statistical methods for censored data (data sets which include below detection values) were considered for application to the TC data. The "robust method" of Gilliom and Helsel (1986) was selected because it handled multiple CRLs in a probabilistic manner and was robust to departures from an assumed distribution shape (lognormal in this case). This method assigns replacement values for the BCRL data based on the extrapolation of the lower part of a lognormal distribution that is estimated based on the measured concentrations (hits) and the proportions of data labeled as below different CRLs. A second method, the replacement by 1/2 CRL, was also used. Shell would have preferred that BCRLs be incorporated directly into the maximum likelihood estimation of the TC distributions in the Shell approach, rather than generating specific TC replacement values by the robust method.

It is widely recognized that the 1/2 BCRL method (i.e., replacement of the BCRL observation with a value equal to half its CRL) tends to have a positive bias for estimates of the mean and negative bias for estimates of standard deviation when the proportion of BCRL data is high. The robust method, which estimates replacement values for the BCRL data, was observed to produce extremely low (near zero) replacement values in cases where there were relatively high hits mixed with a large portion of BCRL data. Gilliom and Helsel (1986) found that when the robust method was combined with the arithmetic estimator of the mean, the bias was relatively low (0 to 17 percent of the true mean), even when the underlying distribution was very skewed and sample sizes were as small as 10. The bias was positive or negative depending on the distribution, sample size, and percentage of censoring (i.e., percentage of BCRL data in a data set; censoring levels of up to 80 percent were tested). When combined with the arithmetic sample standard deviation, the robust method tended to produce a downward-biased estimate of the true standard deviation, i.e., 3 to 46 percent depending on the distribution, sample size, and censoring level (Gilliom and Helsel 1986).

The authors of the robust method warn that this and all other methods are unreliable when the proportion of BCRL data exceeds 20 percent. However, estimations were required even under this circumstance. The method of replacing BCRL data with 1/2 CRL was used in cases where the number and proportion of hits were so low that a distribution-based interpolation was considered extremely unreliable. The following rules were used to determine which method should be used:

Cases Analyzed With the Robust Method
—Greater than 3 data points AND
—Greater than 10 percent hits (detected concentrations)
OR
—Greater than 5 data points AND
—Greater than 5 percent hits

Cases Analyzed by Replacement with 1/2 CRL
—Above conditions not met

The robust method replaces the N BCRL data with N different estimated concentrations that are not associated with a particular sample. Therefore, a modification of this method, called the expected value robust (EVR) method was used in cases where the association of individual TC samples and ESC estimates had to be maintained if the data set had 10 BCRL data points with 3 different CRLs, the EVR method first calculated 10 estimated concentrations and then used these estimations to calculate 3 expected concentrations that could be associated with the 3 CRLs, i.e., the lowest expected concentration was assigned to the data with the lowest CRL, the middle to the data with the middle CRL, and the highest to the data of the highest CRL. These calculations are further specified below.

The estimation of an expected value for each CRL can be defined in different ways. The following method was chosen because it resulted in 3 expected values with a variance that was

as large as possible and therefore as close as possible to the variance of the original 10 estimates. The expected values for the CRLs ordered from low to high (A_1, A_2, A_3) were calculated by averaging the lowest N_1 estimates, the next N_2 estimates, and then the highest N_3 estimates, respectively, where N_j is the number of BCRL data points with CRL_j. The average of these 3 averages, weighted by $N_j/10$ is equal to the average of the 10 original estimates.

E.12.4.1.2 Summation of Pairs of Chemicals

Two pairs of chemicals, aldrin and dieldrin (aldrin/dieldrin) and DDE and DDT (DDE/DDT), were summed and treated as single chemicals in the development of BMFs and calculation of risk. When both of the values were either detections or replacement values for BCRL data, the values were simply summed. If both values were NA, the data from the sample could not be used. However, the combination of a detection or BCRL value with an NA required special treatment, as discussed below for TC and ESC data. The only such case that arose in the R data was the occurrence of a BCRL for aldrin with an NA for dieldrin. Because the ratio of detected aldrin to dieldrin concentrations was much less than 1.0 for most or all TC samples, it was inferred that the dieldrin concentration was likely to be much higher than the aldrin concentration. Therefore, if a (BCRL, NA) pair was to be used, the concentration of dieldrin would have to be extrapolated upward from the concentration of aldrin (e.g. $C_{dieldrin} = R * C_{aldrin}$, where R is much greater than 1.0). Such an extrapolation was highly uncertain both because the aldrin value was a BCRL and because the extrapolated dieldrin concentration would then dominate the sum of aldrin and dieldrin for that sample (i.e., $C_{aldrin} + R * C_{aldrin}$ would be much greater than C_{aldrin}). Therefore, all TC samples with aldrin and dieldrin concentrations of BCRL and NA, respectively, were eliminated from the analysis.

E.12.4.1.3 Estimation Based on Small Sample Sizes

Small sample sizes (e.g., $N \leq 10$) increased the uncertainty in estimating TC distributions in some cases (see Table E.12-5).

E.12.4.2 Uncertainty About <ESC> Distributions

Two types of ESC uncertainty were discussed in E.9.4. Representation uncertainty refers to the uncertainty in representing the exposure soil concentration by the ESC. This type of uncertainty is for all practical purposes unquantifiable and irreducible, because it involves the detailed information on individual organisms (and their prey) which cannot be characterized for all parts of RMA. Estimation uncertainty refers to the uncertainty in estimating the ESC based on available data. Estimation uncertainty can be reduced, for example, by selection of statistical methods, increasing the amount of soil concentration data, or obtaining more accurate literature estimates of parameters such as the exposure range radius expected for a given species. For example, the representation error is involved in representing individual exposure activities as being uniformly distributed over a constant (mean) circular exposure range, while estimation error is involved in estimating the appropriate radius for this mean exposure range. Specific components contributing to representation and estimation uncertainty are discussed in E.9.4 and are summarized below. The remainder of this section elaborates on estimation components for which substantial effort was made to minimize uncertainty.

Representation Uncertainty

—Depth of soil exposure due to varied activities (e.g., digging, feeding)

—Bioavailability of contaminants in different soils

—Spatial/temporal pattern of exposure (includes variation in individual age, life histories, and behavior)

Estimation Uncertainty

—Interpretation of BCRL soil data

—Interpolation of RMA soil data

—Estimation of exposure range radius (literature data and judgement)

—Estimation of exposure range center for a given sample individual

—Summing concentrations for pairs of chemicals (e.g., aldrin + dieldrin)

—Small sample sizes (i.e., estimation of distributions from low numbers of ESC estimates associated with TC samples)

E.12.4.2.1 Interpretation of BCRL Data

The estimation of ESC data required that soil concentrations be interpolated over the portions of RMA where TC samples were taken. Although an attempt was made to estimate the most likely concentrations for the BCRL data uncertainty is still present in the data sets after they have been processed by the BCRL interpolation method. The BCRL interpolation method, described in Appendix Section C.1.4.1.1, was developed to allow concentrations measured in the proximity of a BCRL sample to affect the estimation of a replacement value for this sample. In many cases, the surrounding concentrations provided useful information about the concentration, with the result that the uncertainty in interpreting this BCRL value was greatly reduced. For example, very high BCRL values were replaced with low or high concentration estimates if the surrounding samples indicated this was an area of low or high concentration, respectively. In other cases, the surrounding samples were also high BCRL values and so did not provide information that allowed estimation of a likely value for the BCRL. In these cases, the BCRL was replaced with NE, indicating no estimation was made. Table E.12-6 presents the number of BCRL samples that did not receive an estimate for all biota chemicals.

E.12.4.2.2 Interpolation of Soil Concentrations

The calculation of <ESC> values for samples required soil concentrations (SC) to be interpolated for areas in and around the TC samples. This was accomplished by interpolating SC values onto a grid with 100-ft spacing. In general, the actual SC samples were not randomly located within exposure ranges and therefore could not be used to estimate the mean concentration unless some sort of interpolation was used. The interpolation greatly reduced the uncertainty in estimating ESC values from SC samples because it provided statistically based estimates of SC values in each potential exposure range and allowed equal areas to receive equal weight in the average. However, uncertainty is still present after the interpolation, especially in cases where the sampling density is sparse relative to the variability in concentrations. In cases where there are

hot spots, the magnitude of the concentrations and the boundaries of the hot spots are uncertain since the rapid changes in concentration may not be adequately characterized by the few samples in such areas.

Tissue samples were often collected in areas of rapid transition between very high concentrations and low concentrations. This was because tissue samples could not be collected from areas where concentrations were so high as to preclude the presence of a given species, however, samples with some exposure to these hot spots (e.g., on the edge) were desirable. It should be emphasized that the estimates interpolated from SC samples are highly uncertain in such areas of rapid concentration transition; therefore, the ESC estimates calculated for these areas are also very uncertain.

E.12.4.2.3 Estimation of Exposure Range Radius

The development of exposure range radii for each species is discussed in Appendix Section C.2.5. In some cases (e.g., heron, eagle) the exact outline of a noncircular exposure range was specified. Professional judgement and consensus discussions were used to select the exposure range values that provided the most appropriate representation of the expected areal exposure for a given species. This process included the critical evaluation of many published literature studies (e.g., for methodology and regional relevance) as well as interviews with local and regional experts. Uncertainty in the final consensus value varies with species and is described in Appendix Section C.2.5.

E.12.4.2.4 Summing of Pairs of Chemicals

As discussed above for the TC data, a measured concentration or BCRL was sometimes available for only one of the paired chemicals to be added together. In order to provide an estimate of the summed value for such a sample, the following method was used.

For the ESC concentrations, aldrin/dieldrin and DDE/DDT pairs had occurrences of (NA, value) but no occurrences of (value, NA). The ratios of aldrin/dieldrin and DDE/DDT in the ESC data

had the median values of 0.224 and 0.865, respectively. For DDE/DDT, the mean and the upper 95 percent confidence limit for the mean were also well below 1.0. For aldrin/dieldrin, the mean was 2.64, pulled upward by the skewed shape of the distribution (the mean was the 90th percentile). Although the ratios for aldrin/dieldrin and, especially, DDE/DDT varied, and although the extrapolations were therefore uncertain, it was decided that an uncertain extrapolation of aldrin and DDE was more informative than discarding the sample. In contrast to a steep upward extrapolation (R >> 1), where the extrapolation dominates the sum, a downward or slightly upward extrapolation introduces a reasonably low amount of uncertainty into the sum (i.e., if the true extrapolation constant is either 0.0, 1.0, or 2.0, the value of the sum is either $C_{dieldrin}$, 2 * $C_{dieldrin}$, or 3 * $C_{dieldrin}$, which is a relatively small range of uncertainty.) Therefore, in cases where only aldrin or DDE values were missing, the concentrations were estimated as C_{aldrin} = 0.224 * $C_{dieldrin}$ and C_{DDE}= 0.865 * $C_{dieldrin}$, (i.e., the median of the ratios calculated from all pairs with hits for both aldrin and dieldrin).

E.12.4.2.5 Estimation with Small Sample Sizes

Small sample sizes increased the uncertainty in estimating ESC distributions in some cases (see Table E.12-5), which reflects samples sizes used for <ESC> as well as TC.

E.12.4.3 Uncertainty Due to Species Aggregation

Three trophic boxes included representative species with substantial differences in exposure range radii: small mammals, medium mammals, and small birds. In these cases, the uncertainty in comparing species-combined TC and <ESC> distributions is increased because the species with small exposure ranges tend to be exposed to more varied <ESC> distributions than species with large exposure ranges.

When data were sufficient, BMFs were first calculated for each species and then combined to get a weighted-average BMF for the trophic box. This occurred for aldrin/dieldrin, for which the number of hits for each species was sufficient to estimate species-specific TC and <ESC> distributions. However, for most chemicals, the number and proportion of hits was not sufficient

to distinguish between BMFs for different species, and therefore, samples from all species were combined into one data set and were not weighted to reflect the relative sample sizes and to reflect the relative abundance of the species in the predators' diet.

E.12.4.4 Correlation of TC and <ESC> Distributions

The <ESC> for each sample exhibited a near-zero correlation with TC samples. As can be seen from Table E.12-7, some relatively high sample correlations occur; however, the degree of correlation is not consistent either within a given trophic box or within a given chemical. This lack of consistency indicates that these observed (sample) correlations generally may provide very poor estimates of the true correlations between actual exposure and tissue concentration.

E.12.4.5 Limitations in the Ability to Predict Individual Tissue Concentrations With BMFs

Tissue concentration measurements and predictions are drawn from two different populations: measurements from the population of individuals at RMA and predictions from the estimated distribution on the mean tissue concentration. Therefore, the tissue concentrations predicted by the food-web model will not equal observed tissue concentrations even if one assumes ideal field and literature data (i.e., soil concentrations accurately characterized, biota samples each collected from the center of the samples exposure range, exposure range radii representative of the mean individual for a given species, prey fractions accurately characterized). As discussed in Section E.9.4, these factors contribute to estimation error and their elimination does not reduce the representation error inherent in the media based risk estimation approach. Representation error arises because the model does not account for physiological and behavioral variability among target species individuals, so it cannot predict individual variability in contaminant biomagnification. In addition, the model does not account for variation in soil concentrations associated with the different depths to which biota are actually exposed. The contribution of individual variability to the overall error in predicting TC cannot be quantified with the current RMA data.

While predicted and observed tissue concentrations are expected to differ, the model should not consistently over- or underpredict observed tissue concentrations. This provides a qualitative check on the plausibility of alternative BMF values. If, for a given target species, TC field data constituted a random sample of the RMA population, one could compare the sample average to the mean of RMA-wide TC predictions as a quantitative test of the empirical performance of alternative BMFs. However, statistical comparisons of means are not robust to violations of the random sampling assumption, and so cannot be applied RMA tissue concentration data.

E.12.5 COMPARISON OF ALTERNATIVE BMFs

As discussed in Section E.12.4, three BMF_{obs} estimates were developed for each trophic box and chemical, those based on the Army, Shell, and EPA approaches.

The three methods are presented in Appendix Section C.1.6.1. The methods utilize the same basic TC and <ESC> data sets; however, these data sets are processed differently with respect to data screening, BCRL handling (slight differences), correlation treatment, and distribution parameter estimation. The differences in these methods, which are detailed in Appendix Section C.1.6.1, are discussed below with respect to the associated uncertainties.

TC and <ESC> Data Sets

Although the EPA method screened the TC and <ESC> data, the data sets used for all three methods were subject to approximately the same sources of error and uncertainty that were described in Sections E.12.4.1 and E.12.4.2. BCRL data were handled identically for the <ESC> calculations, and very similarly for the TC calculations (see Appendix Section C.1.6.1 for BCRL replacement methods). Uncertainty in summing the two pairs of chemicals (aldrin/dieldrin, and DDE/DDT) was identical in the three approaches. Uncertainty in modeling exposure ranges and in interpolating soil concentrations onto a grid were also the same for the three methods. The uncertainty due to small sample sizes was increased for the EPA method in a few cases where the data screening resulted in very low sample sizes (e.g., ground squirrel). The three methods also had the same sources of uncertainty in aggregating species within trophic boxes. The

differences in BCRL handling for TC were as follows. For the Army and Shell approaches the robust method was used for mercury and endrin while the expected value robust method (EVRM) was used for aldrin/dieldrin and DDE/DDT so that concentrations in individuals could be added together. For the EPA approach, the EVR method was used for all chemicals in order to maintain the pairing between TC and <ESC>.

Correlation

All three BMF calculation approaches require estimation of the correlation between the populations of TC and ESC estimates used to derive BMF_{obs}. The three BMF approaches differ in their correlation assumptions. The remainder of this section discusses uncertainty about correlation, and describes the advantages and disadvantages of the correlation assumptions for the three BMF approaches.

As discussed in E.12.4.4, the TC_{obs} and <ESC> data sets had correlations which tended to be low or near zero, with inconsistent and seemingly arbitrary medium and high correlations for some trophic box/COC combinations. In addition, screening the paired data sets did not increase correlation. The low and variable sample correlations between the TC_{obs} and <ESC> data sets is due to both representation and estimation uncertainty, as defined in Section E.9.4. Because of representation uncertainty, the true correlation between population mean tissue concentration (TC) and exposure area soil concentration (ESC) is expected to be less than one, and variable across trophic box/COC combinations. Consequently, the sample correlation between TC_{obs} and <ESC> data sets is also expected to be less than one, and variable across trophic box/COC combinations. In addition, because of estimation uncertainty in both TC_{obs} and <ESC>, the sample correlation between TC_{obs} and <ESC> data sets is expected to be even lower than the true correlation between TC and ESC. The correlation between the populations of TC_{obs} and <ESC> estimates used to derive BMF_{obs} probably lies somewhere between the sample correlation between TC_{obs} and <ESC> data sets and the true correlation between TC and ESC (except for the EPA BMF approach, where the TC_{obs} and <ESC> data sets <u>are</u> the populations used to derive

BMF_{obs}, so the sample correlation between the screened, paired (TC_{obs}, <ESC>) data is the correlation between the populations used to derive the EPA BMF_{obs}).

An important source of uncertainty about correlation is uncertainty in estimates of exposure range center, as described in Section E.9.4.2. The error in the assumption that organisms are sampled at the center of their home ranges is referred to as "location error." Location error weakens the observed relationship between TC_{obs} and <ESC>, lessens the observed correlation, and makes it more difficult for all three approaches to estimate BMF_{obs}. Paired (TC_{obs}, <ESC>) data contain location error, but the pairs (TC_{pred}, <ESC>) do not. Because the BMF_{obs} is used to calculate TC_{pred} from <ESC>, the data used to calculate BMF_{obs} should be corrected for location error. However, the location error is unknown, so BMF_{obs} must be estimated either from paired data containing location error, or from distributions that are based on the data, but do not preserve the pairing between TC_{obs} and <ESC>. The EPA method uses paired data that contain location error. As a result, it underestimates the correlation of TC and ESC, which introduces a positive bias of unknown magnitude into the EPA BMF_{obs}. Location error also impacts the Army and Shell approaches. However, the Army and Shell approaches do not preserve the pairing between the sample TC_{obs} value and its predicted <ESC>, so they are not impacted by individual differences between predicted and estimated actual <ESC>s. (A sample TC_{obs}'s predicted <ESC> is the <ESC> centered on the location where the tissue concentration sample is collected. The estimated actual ESC is the <ESC> that is concentric with the home range of the sampled organism.) Instead, the Army and Shell approaches are impacted by the overall difference between the distribution of predicted <ESC>s and the distribution of actual <ESC>s.

The Army method assumed that the correlation between the estimated ln(TC) and ln<ESC> distributions would likely range from 0.3 to 0.7, and would tend to be an intermediate value near 0.5. A ln(TC), ln(ESC) correlation less than 0.3 was considered unlikely given that there is a documented mechanism of uptake; i.e., TC responds to ESC. A ln(TC), ln(ESC) correlation greater than 0.7 was considered unlikely, even if estimation error in the data was insignificant, because of variability in physiological and behavioral attributes of individuals. The advantages

of this assumption are as follows. First, the Army's mean ln(TC), ln(ESC) correlation (0.5) was generally intermediate to the correlations resulting from the other methods. The EPA estimates depend on the correlations in the paired, screened data, which were near zero, while the Shell assumption about BMF and <ESC> being uncorrelated and the estimates of $\sigma_{\ln(\langle ESC \rangle)}$ and $\sigma_{\ln(TC)}$ implied correlations between ln(TC) and ln(<ESC>) which were generally higher than 0.5. (The resulting Army BMF estimates would also be intermediate if the same parameter estimation method had been applied in all methods, which was not the case.) This is discussed further in the Parameter Estimation section below. A second advantage is that the Army correlation distribution for each trophic box/chemical combination was not subject to the inconsistencies appearing in the observed correlations and therefore did not result in greatly inconsistent estimates of uncertainty in the mean BMF. The disadvantages of the Army approach are as follows. First, any estimate of correlation which appears to be reasonably reliable (high N and moderate to high correlation) is not used for the associated trophic box/chemical, increasing the potential for bias in that case. A second disadvantage is that the appropriateness of the triangular distribution for representing the correlation between the estimated ln(TC) and ln(<ESC>) distributions is unknown and may vary from case to case.

The Shell method assumed a 0 correlation between <ESC> and BMF, which implied different values for BMF, and for the TC and <ESC> correlation, for different bootstrap samples of the paired data. The advantage of this approach is that it is designed to produce the BMF distribution (or constant, in some cases) which is best suited to predict the group of observed TCs from the group of estimated ESCs under the assumption of zero correlation between <ESC> and BMF. In the IEA/RC, risks are estimated (i.e., TC and dose are predicted) under the assumptions of zero correlation between BMF and <ESC> and between BMF and TC_{pred}. The disadvantage of the Shell correlation assumption is that the appropriateness of Shell's assumption that BMF and <ESC> are uncorrelated (and similarly, the appropriateness of the Army's assumption that $\rho_{\ln(TC),\ \ln(\langle ESC \rangle)}$ ~ triangular(0.3, 0.5, 0.7), which is meant to capture the implicit assumption that BMF and TC_{pred} are uncorrelated) is unknown and may vary from case to case. Even though the BMF is assumed to be uncorrelated with <ESC> during the prediction of TCs and risks, the estimation of BMF

is still dependent on the TC and <ESC> data, unless these data are correlated to a specific degree (defined by $\rho_{ln(TC),\ ln(ESC)} = \sigma_{ln(ESC)}/\sigma_{ln(TC)}$).

The EPA estimation of BMF depended on the correlation between TC and its predicted <ESC> in the screened paired data sets. The screening was intended to discard portions of the TC and <ESC> distributions which were extreme and therefore had the potential to cause non-linearities in the TC-<ESC> relationship or to substantially increase the noise in this relationship, in both cases causing a reduction in the correlation in the paired data. The estimates of correlation using the screened data are given in Table E.12-8 which can be compared to Table E.12-7. For most trophic boxes, the screening resulted in correlations which were small or not meaningful. The few substantial changes in correlation due to screening were fairly evenly divided between increases (e.g., small mammal aldrin/dieldrin, insect aldrin/dieldrin, worms endrin and Hg) and decreases (e.g., small mammal aldrin/dieldrin and DDE/DDT, and shorebird endrin). The advantage of the EPA correlation assumption is that it utilizes the information contained in the pairing of the data. However, this information is subject to location error and other types of estimation error. The disadvantage of the EPA correlation assumption is that it has a positive bias of unknown magnitude due to location error.

An analysis was performed of the sensitivity of the Army BMF_{obs} to the assumed correlation of TC and <ESC>. The correlation that appropriately relates the observed TC and <ESC> distributions is highly uncertain due to the lack of confidence in adequate collocation of the individual pairs of TC and <ESC>. The sensitivity of the mean BMF to the assumed correlation was investigated by recalculating the mean BMF for all trophic boxes and chemicals based on three different assumed correlations: $r = 0.0$, $r = 0.5$, and $r = 1.0$. These results, shown in Table E.12-9, indicate that in all but three cases magnitude over the mean BMF varied less than an order of magnitude over the full range of possible correlations (i.e., 0.0 to 1.0). The mean BMF under a correlation of 0.0 was commonly a factor of 1 to 3 times higher than the mean BMF at a correlation of 0.0.

Parameter Estimation

The Army method uses arithmetic sample estimators, i.e., the mean and variance of the untransformed TC and ESC data, and then converts the arithmetic estimators using standard formulas to estimate the parameters of the lognormal distribution. The Shell method uses logarithmic sample estimators, i.e., the mean and variance of the natural logarithm of TC and ESC data, which can be converted using standard formulas to get estimates of the mean and standard deviation of the lognormal distribution. The EPA method uses arithmetic estimators applied to the BMF estimate associated with each TC sample. The pros and cons of these different estimators are as follows.

The advantage of the arithmetic estimators is that they are insensitive to data errors among the lowest concentrations in the data set. This reduced sensitivity is an advantage because TC and <ESC> data sets tend to have a high frequency of BCRL data. For TC, the robust method for handling BCRLs data estimates replacement values for the BCRL data based on the assumption of lognormality. These replacement estimates are sometimes several orders of magnitude lower than the CRLs and are intended to be used with arithmetic estimators that are relatively insensitive to an error in the replacement value within the range of values close to zero. The main advantage of the robust method for handling BCRL data is that the replacement values for BCRLs estimated under the assumption of lognormality can be transformed back to linear space and the arithmetic estimators applied, thereby reducing the reliance on the assumption that the entire data set is lognormal (Gilliom and Helsel 1986). Therefore, the use of arithmetic estimators was considered part of the robust estimation methodology. This same rationale for using arithmetic estimators, even when the data sets are skewed was applied in the HHRC in estimating C_{rep} from data sets with frequent occurrence of BCRL data. The disadvantage of the arithmetic estimators is that, while they are statistically unbiased estimators of the mean and standard deviation of the observed data, they do not have the lowest variance of all estimators when used on distributions known to be lognormal.

The log-based estimators used for the Shell method are lower in variance than the arithmetic estimators and are generally recommended over the arithmetic estimators for distributions known to be lognormal and to be highly skewed (Gilbert 1987). The log-based estimators are less sensitive to random sampling error in the highest concentrations in the data set. As stated above, the disadvantage of these estimators is that the standard deviation of the log-transformed data, and therefore the estimates of mean and standard deviation in linear space, are relatively sensitive to any errors made in interpreting the BCRL data.

An analysis was performed of the joint sensitivity of the Army BMF_{obs} to BCRL replacement method and correlation of TC and <ESC>. The combined influence of different assumptions for BCRL treatments and assumed correlations can be shown by calculating BMFs using these different assumptions for an example trophic box/chemical combination. Table E.12-10 shows the mean BMFs calculated under different assumptions for the small bird/endrin data, which had a sample size of 83 (excluding missing data) and 15.7 percent hits. This case was selected because it represented a borderline case where the robust method was selected even though substantial uncertainty existed due to the low percentage of hits.

All BMFs shown in Table E.12-10 had identical distributions for <ESC>, while the distributions for TC varied with the BCRL method. BMF varied as a function of the different TC distributions and the correlation assumed.

A decrease in r from 1.0 to 0.0 resulted in a 3.9- to 4.7-fold increase in BMF, with larger increases occurring when the BCRL data were analyzed using the robust or expected value robust (EVR) method. For a given value of r, the two robust methods gave estimates of the mean BMF that were only slightly lower than the 1/2 CRL method. As discussed above, low sensitivity to the BCRL method reflects the use of the arithmetic estimators. The low sensitivity to the correlation indicates that, in this case, the TC and <ESC> component distributions have a relatively low variance. With higher variances in the component distributions, r would have a larger impact on the mean BMFs.

E.13 UNCERTAINTIES ASSOCIATED WITH ECOLOGICAL MEASUREMENT ENDPOINTS

On the preceding pages, the areas of potential ecological risk identified for various trophic boxes on the basis of toxicological endpoints were given credence and perspective by discussion of the uncertainty in their calculation. The presence of potential ecological risk was given further perspective by considering it together with available data on ecological endpoints (Appendix Section C.5). The available data on ecological status and health used to evaluate ecological endpoints are also subject to uncertainty. In this context, uncertainty results from:

(1) The short term nature of many of the studies relative to the cycles of natural variability

(2) Estimation of quantitative ecological parameters at levels of precision that may not be biologically and/or statistically significant

(3) Study designs that did not precisely and quantitatively correlate ecological parameters with parameters related to contaminant concentrations,

(4) Study designs that did not precisely quantify all parameters that might have positively or negatively affected the ecological data

For example, low prairie dog colony density on RMA relative to reference areas could be interpreted as resulting from contamination. However, colonies in the compared areas might have been at different stages of maturity; level of maturity is known to affect colony density. Longer term studies would have revealed the cyclical nature of density in the colonies of both areas and eliminated uncertainty source #1 above. Study designs that quantitatively correlated the density of young produced by individual pairs with the contaminant concentration in their exposure range might have revealed no correlation of density of young with contamination, leading study conclusions elsewhere. This would have minimized uncertainty sources #2 and #3 above. The collection of additional data on the colony, including its level of maturity, would have eliminated uncertainty source #4 above.

Long term highly replicated studies that measure numerous parameters precisely are in the realm of research studies. Note, for example, the statement in Heinz et. al (1990, page 553) that "...we cannot begin to model the effects of pesticides on bird populations until we understand all the other major factors that affect bird populations in a given ecosystem. The authors go on the note the success of a model based on 40 years of data that did consider major ecological factors affecting populations. Such research studies do not lend themselves in either timing or cost to a schedule appropriate for remediation. Therefore, the uncertainties present in the ecological status and health studies are acknowledged, and ongoing studies are planned to collect data to reduce the uncertainties identified.

E.14 REFERENCES

ANL (Argonne National Laboratories). 1991. White Paper on Aldrin-Dieldrin Loss from Soil. In: Risk Characterization Integrated Endangerment Assessment Report. Appendix A.3-A.5. Preliminary Draft. Rocky Mountain Arsenal, AMXRM-PM, Commerce City, Colorado, 80022.

Brown, D.P. 1992. Mortality of workers employed at organochlorine pesticide manufacturing plants--an update. Scand. J. Work Environ. Health, 18:155-61.

Bureau of the Census. 1987. Current Population Study, Tenure with Current Employer by Age and Sex. Prepared for the Bureau of Labor Statistics. Washington, D.C. January 1981, 1983, 1987.

BNA (Bureau of National Affairs). Personnel Policies Forum. Job Absentee and Turnover Survey. Quarterly and Annual Surveys from 1974 to current. Western Region Absence and Turnover data, 1978 to current. Published by Bureau of National Affairs, Washington, D.C. 1974-1990.

Conover, W.J. 1980. Practical Non-Parametric Statistics. John Wiley & Sons, Inc.

de Jong G. 1991. Long Term Health Effects of Aldrin and Dieldrin. Toxicol. Lett. (Suppl): 115-141.

Ditraglia, D., D.P. Brown, N. Namekata, and N. Iverson. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Work Environ. Health, 7: 140-6.

DPA (D.P. and Associates). 1991. Rocky Mountain Arsenal Environmental Database.

RTIC 88357R01
EBASCO (EBASCO Environmental). 1988. Rocky Mountain Arsenal Chemical Index with Applicable, Relevant and Appropriate Requirements (ARARs). Volumes I—III.

RTIC 90277R01
EBASCO. 1990. Human Health Exposure Assessment for Rocky Mountain Arsenal. Volumes I—VIII, Version 4.1. September 1990.

EBASCO. 1991. Letter from EBASCO to PMRMA dated March 20, 1991.

RTIC 92017R01
EBASCO. 1992 Remedial Investigation Summary Report. Final Report. January.

EPA (U.S. Environmental Protection Agency). 1985. Drinking Water Criteria Document for Dibromochloropropane. ECAO-CIN-410.

EPA. 1986a. Air Quality Criteria for Lead. June 1986 and Addendum, September 1986. Office of Health and Environmental Assessment. EPA 600/8-83-028F. NTIS PB 87-142378.

EPA. 1986b. Guidelines for Carcinogenic Risk Assessment. 51 Federal Register. Pp. 33992. September 24, 1986.

EPA. 1987. Carcinogenicity Assessment of Aldrin and Dieldrin. EPA/600/6-87/006: 4-46-4-104.

RTIC 91176R01V.1
EPA. 1988. SEAM (Superfund exposure assessment manual). EPA540/1-88/001. Office of Remedial Response, Washington, D.C.

RTIC 91309R06V.1
EPA. 1989a. Risk Assessment Guidance for Superfund. Volume 1. Human Health Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response. EPA/540/1-89/002.

RTIC 89068R01
EPA. 1989b. Federal Facility Agreement for the Rocky Mountain Arsenal. U.S. Department of the Army. Agency for Toxic Substances and Disease Registry.

EPA. 1991a. Letter from EPA to PMRMA dated March 5, 1991.

EPA. 1991b. HEAST (Health Effects Assessment Summary Tables). Office of Health and Environmental Assessment. January 1991.

EPA. 1992. IRIS (Integrated Risk Information System). Online database. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

ESE (Environmental Science and Engineering). 1989. Biota Remedial Investigation Final Report, Version 3.3 Prepared for the Office of the Program Manager. Contract No. DAAK 11-84-D0016. Task 9, Denver, CO.

Forsyth, D.J., T.J. Peterle, and L.W. Bandy. 1983. Persistence and transfer of ^{36}C-DDT in the soil and biota of an old-field ecosystem: A six-year balance study. Ecology 64:1620-1636.

Gilbert, R.O. 1987. Statistical Methods for Environmental Pollution Monitoring. New York. Van Nostrand Reinhold.

Gilliom, R.J. and D.R. Helsel. 1986. Estimation of Distribution Parameters for Censored Trace Level Water Quality Data. (1. Estimation Techniques.) Water Res. 22(2): 135-146.

Jury, William A., William W. Nazaroff and Vern C. Rogers. 1991. Enclosed-space Vapor Models: Technical Panel Report. Prepared for Rocky Mountain Arsenal and Shell Oil Company by University of California, Riverside, California.

Korschgen, L.J. 1970. Soil-food chain-pesticide-wildlife relationships in aldrin treated fields. J. Wildl. Manage. 35:494-500.

Layton, D.W., B.J. Mallor, D.H. Rosenblatt, and M.J. Smith. 1987. Deriving Allowable Daily Intakes for System Toxicants Lacking Chronic Toxicity Data. Regulatory Toxicology and Pharmacology. 7:96-112.

MMWR (Morbidity and Mortality Weekly Report). 1991. Hepatitis B Virus: A Comprehensive Strategy for Eliminating Transmission in the United States Through Universal Childhood Vaccination. 40(RR-13):1-19.

Morgan, M.G. and M. Henrion. 1991. Uncertainty: A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis. Cambridge University Press.

Mountain States Employers' Council, Inc. 1981-1990. Metro Denver Turnover Surveys, Annual Surveys from 1981 to current, incorporated into Colorado Turnover Survey data after 1989.

NTP (National Toxicology Program). 1982. Carcinogenesis Bioassay of 1,2-Dibromo-3-chloropropane (CAS No. 96-12-8) in F344 rats and B6C3F1 Mice (inhalation study). Tech. Rep. Ser. No. 206. Pub. No. 82-1762, p. 188.

Noreen, Eric W. 1989. Bootstrap resampling. Chapter 4 in Computer Intensive Methods for Testing Hypotheses: An Introduction. pp. 63-82, John Wiley and Sons, New York.

PMRMA (Program Manager for RMA). 1990. Letter from PMRMA to EPA dated June 22, 1990, page 7.

PSCo (Public Service Company of Colorado). 1989. Residential Energy Use Survey.

RTIC 90220R01
RLSA (R.L. Stollar and Associates Inc.). 1990. Comprehensive Monitoring Program Final Biota Annual Report for

RLSA. 1989. Technical Report, Version 1.5. Prepared for the U.S. Army Program Manager for Rocky Mountain Arsenal.

Shell (Shell Oil Company). 1990. Letter from Shell to PMRMA dated May 30, 1990, comment #12.

Shell. 1992a. Uncertainty analysis of the human carcinogenic potential of aldrin and dieldrin.

Shell. 1992b. The carcinogenic potential of aldrin and dieldrin.

Sielken, R.L. and D.E. Stevenson. 1992. Comparisons of human cancer potency projections for dieldrin based on human data with those based on animal data. In: D'Amato, Slaga, et al. (eds). Relevance of Animal Studies to the Evaluation of Human Cancer Risk. Wiley-liss, Inc., New York. pp 295-319.

Suter, G.W., II. (ed.). 1993. Ecological Risk Assessment. Lewis Publishers, Chelsea, Michigan.

Tennekes, H.A., L. Edler, and H.W. Kunz. 1982. Dose-response analysis of the enhancement of liver tumor formation in CF-1 mice by dieldrin. Carcinogenesis 3:8: 941-5.

Table E.7-1 Uncertainties Potentially Influencing Assigned Distributions for Soil Intake Parameters

Soil Covering		Soil Ingestion		Dust Loading	
Population and Age Class	Uncertainties	Population and Age Class	Uncertainties	Population and Age Class	Uncertainties
REGULATED/CASUAL VISITOR 0 to < 1	• judgment distribution	REGULATED/CASUAL VISITOR 0 to < 1	• assumed minimal (1 milligram/day)	REGULATED/CASUAL AND RECREATIONAL VISITOR All ages	• assumed outdoor ambient exposure • representation of activities by ambient outdoor dust loading conditions • data measurement error
1 to < 7	• data measurement error • extrapolation of sample patch to entire surface area • data representation of age distribution and activities	1 to < 7	• judgment 95th percentile (EPA default) • data median (literature) • data measurement error • data representation of age and activities		
7 to < 18	• data measurement error • extrapolation of sample patch to entire surface area • data representation of age and activities	7 to < 75	• judgment 95th percentile (EPA default) • shape extrapolated from literature distribution for child		

Table E.7-1 Uncertainties Potentially Influencing Assigned Distributions for Soil Intake Parameters

Soil Covering		Soil Ingestion		Dust Loading	
Population and Age Class	Uncertainties	Population and Age Class	Uncertainties	Population and Age Class	Uncertainties
18 to < 75	• data measurement error • extrapolation of sample patch to entire surface area • data representation of age and activities				
RECREATIONAL VISITOR 0 to < 1	• judgment distribution	0 to < 1	• assumed minimal (1 milligram/day)		
1 to < 7	• data measurement error • extrapolation of sample patch to entire surface area • data representation of age and activities	1 to < 7	• judgment 95th percentile (EPA default) • data median (literature) • data measurement error • data representation of age and activities		

Table E.7-1 Uncertainties Potentially Influencing Assigned Distributions for Soil Intake Parameters

Soil Covering		Soil Ingestion		Dust Loading	
Population and Age Class	Uncertainties	Population and Age Class	Uncertainties	Population and Age Class	Uncertainties
7 to < 18	• data measurement error • extrapolation of sample patch to entire surface area (data representativeness) • representation of age and activities (study representativeness)	7 to < 75	• judgment 95th percentile (EPA default) • shape extrapolated from literature distribution (child)		
18 to < 75	• data measurement error • extrapolation of sample patch to entire surface area (data representativeness) • representation of age and activities (study representativeness)				

Table E.7-1 Uncertainties Potentially Influencing Assigned Distributions for Soil Intake Parameters

Soil Covering		Soil Ingestion		Dust Loading	
Population and Age Class	Uncertainties	Population and Age Class	Uncertainties	Population and Age Class	Uncertainties
COMMERCIAL WORKER	• theoretical estimate of mean, judgment range	COMMERCIAL WORKER	• judgment 50th and 95th percentile	COMMERCIAL WORKER	• assumed indoor exposure • dust loading data measurement error • outdoor/indoor attenuation data measurement error
INDUSTRIAL WORKER	• judgment 95th percentile (EPA default) • distribution shape extrapolated from biological/maintenance worker	INDUSTRIAL WORKER	• judgment 95th percentile • shape extrapolated from literature distribution (child)	INDUSTRIAL WORKER	• assumed ambient outdoor exposure • representation of activities by ambient conditions • data measurement error
BIOLOGICAL/ MAINTENANCE WORKER	• data representation of time spent in activities • data representation of soil covering to projected activities • judgment estimate of indoor soil covering distribution	BIOLOGICAL WORKER	• data representation of time spent in activities • judgment based activity specific distributions	BIOLOGICAL WORKER	• data representation of time spent in activities

Table E.7-2 Uncertainties Potentially Influencuing Assigned Distributions for Time Dependent Exposure Parameters

POPULATION	TM (HOURS/DAY)	DW (DAYS/YEAR)	TE (YEARS/LIFETIME)
REGULATED/CASUAL VISITOR	• representativeness of chosen activities for neighborhood population • representativeness of data-based mean for activity-specific distributions • judgment-based distribution shape • representativeness of participation rate in multiple daily activities • representativeness of national means for percent participation in each activity and duration of each activity	• no data specific to visitation of RMA neighborhood subpopulation • intentional conservative estimation bias • judgment-based distribution for number of activity days/year • judgment-based distribution for fraction of activity days occurring at RMA	• representativeness of PSCo data for neighborhood subpopulation (PSCo 1989) • positive bias (overestimation) due to analysis method,which under-represents low TE values in population • negative bias (underestimation) due to moves within same county
RECREATIONAL VISITOR	• representativeness of chosen activities for neighborhood population • representativeness of data-based mean for activity-specific distributions • judgment-based distribution shape • representativeness of participation rate in multiple daily activities • representativeness of national means for percent participation in each activity and duration of each activity	• intentional conservative estimation bias • representativeness of chosen activities for neighborhood subpopulation • representativeness of western region and national means for percent participation in activity • representativeness of national distribution of number of jogging days per week and assumption of 52 weeks per year for neighborhood subpopulation • judgment-based distribution for number of activity days/year for some activity-specific distributions • judgment-based distribution for fraction of activity days occurring at RMA	• representativeness of PSCo data for neighborhood subpopulation • positive bias (overestimation) due to analysis method, which under-represents low TE values in subpopulation • negative bias (underestimation) due to moves within same county
COMMERCIAL/INDUSTRIAL WORKER	• representativeness of national data on hours spent at work	• incorporation of judgment estimates for vacation time and holidays • representativeness of western region data on job absence rates (Bureau of National Affairs, 1974-1990)	• representativeness of Mountain States Employer's council mean job turnover data used to obtain distribution mean (MSEC 1981-1990) • representativeness of national data on occupational turnover used to obtain distribution shape
BIOLOGICAL WORKER	• representativeness of on-site work schedule of interviewed personnel at three refuges	• representativeness of on-site work schedule of interviewed personnel at three refuges	• representativeness of job tenure history of interviewed personnel at three refuges (Bureau of the Census, 1987) • censored data (current tenure was longer than reported at time of survey)

Table E.7-3 Uncertainties Potentially Influencing Assigned Distributions for Chemical-Specific Parameters[1]

Henry's Law Constant (K_H)[2]		Soil to water partition Coefficient Normalized to Organic Carbon K_{oc} (Kd*)		Vapor Pressure (V_P)[2]	
Chemical Group	Uncertainties	Chemical Group	Uncertainties	Chemical Group	Uncertainties
Aldrin Endrin 1,1,2,2-Tetrachloroethane DDT DDE Chlordane HCCPD	• representation of RMA temperature regime • experimental measurement error • $\leq$ 6 data points	Aldrin Endrin 1,2-Dichloroethane Methylene Chloride	• experimental measurement error • $\leq$ 6 data points	Endrin Chlorobenzene Chlordane	• experimental measurement error • representation of RMA temperature regime • $\leq$ 6 data points
Isodrin	• representation of RMA temperature regime • experimental measurement error • no data, extrapolation across chemicals	Isodrin 1,1-Dichloroethylene HCCPD Dicyclopentadiene Dibromochloropropane	• experimental measurement error • $\leq$ 2 data points • extrapolation across chemicals	1,1-Dichloroethylene 1,1,2,2-Tetrachloroethane DDE HCCPD	• experimental measurement error • representation of RMA temperature regime • $\leq$ 6 data points • intentional conservative bias in estimation of SD
Dicyclopentadiene Dibromochloropropane Chloroacetic Acid	• representation of RMA temperature regime • experimental measurement error • no data, extrapolation based on vapor pressure and solubility	Chloroacetic Acid	• $\leq$ 2 data points • extrapolation from other partitioning information	Isodrin Chloroacetic Dicyclopentadiene Dibromochloropropane	• experimental measurement error • representation of RMA temperature regime • 2 data points • judgment range

Table E.7-3 Uncertainties Potentially Influencing Assigned Distributions for Chemical-Specific Parameters[1]

Henry's Law Constant (K_H)[2]		Soil to water partition Coefficient Normalized to Organic Carbon K_{oc} (Kd*)		Vapor Pressure (V_p)[2]	
Chemical Group	Uncertainties	Chemical Group	Uncertainties	Chemical Group	Uncertainties
Dieldrin Toluene Benzene Chloroform 1,2-Dichloroethane 1,1-Dichloroethylene Methylene Chloride Carbon Tetrachloride Tetrachloroethylene Chlorobenzene Trichloroethylene	• representation of RMA temperature regime • experimental measurement error	Dieldrin Toluene Benzene Chloroform Carbon Tetrachloride 1,1,2,2-Tetrachloroethane Tetrachloroethylene Chlorobenzene Trichloroethylene DDT DDE Chlordane Arsenic* Cadmium* Chromium* Lead* Mercury*	• experimental measurement error	Aldrin Dieldrin Toluene Benzene Chloroform 1,2-Dichloroethane Methylene Chloride Carbon Tetrachloride Tetrachloroethylene Trichloroethylene DDT	• experimental measurement error • representation of RMA temperature regime

[1] See text for discussion of types of uncertainties.
[2] K_H[2] and V_p[2] not defined for metals.
3 Kd(distribution coefficient) used for organic COCs lacking K_{oc} data
DDE - dichlorodiphenyldichloroethene
DDT - dichlorodiphenyltrichloroethane
HCCPD - hexachlorocyclopentadiene
RAF - relative absorption factor

Table E.8-1. Interquantile Multipliers for Cumulative Direct PPLV Distributions 1/

	Biological Worker			Recreational Visitor			Commercial Worker		
Contaminant	5% PPLV (mg/kg)	50% PPLV (mg/kg)	Multiplier	5% PPLV (mg/kg)	50% PPLV (mg/kg)	Multiplier	5% PPLV (mg/kg)	50% PPLV (mg/kg)	Multiplier
Aldrin	0.72	4.27	5.96	3.29	94.30	28.66	4.71	38.90	8.26
Benzene	11.80	34.30	2.91	13.00	326.00	25.08	226.00	1530.00	6.77
Carbon Tetrachloride	2.51	7.69	3.06	2.69	67.50	25.09	51.40	305.00	5.93
Chlordane	3.72	19.70	5.30	10.90	235.00	21.56	26.60	253.00	9.51
Chloroacetic Acid*	101.00	219.00	2.17	234.00	1310.00	5.60	1880.00	2600.00	1.38
Chlorobenzene*	966.00	2190.00	2.27	2550.00	12800.00	5.02	16800.00	25000.00	1.49
Chloroform	48.20	191.00	3.96	89.10	1660.00	18.63	1110.00	7480.00	6.74
DDE	12.50	71.30	5.70	30.50	810.00	26.56	126.00	822.00	6.52
DDT	13.50	64.90	4.81	36.00	1010.00	28.06	95.80	901.00	9.41
DBCP	0.20	0.72	3.60	0.25	6.21	24.64	4.51	28.90	6.41
1,2-Dichloroethane	3.23	10.70	3.31	3.75	91.40	24.37	70.70	399.00	5.64
1,1-Dichloroethylene	0.52	1.57	3.04	0.73	15.20	20.74	10.20	68.30	6.70
Dicyclopentadiene*	3690.00	8120.00	2.20	29100.00	209000.00	7.18	58300.00	133000.00	2.28
Dieldrin	0.41	2.45	5.92	1.96	48.10	24.54	2.54	22.70	8.94
Endrin*	232.00	642.00	2.77	865.00	6720.00	7.77	1120.00	3410.00	3.04
Hexachlorocyclopentadiene*	1060.00	2220.00	2.09	6160.00	40500.00	6.57	16700.00	33200.00	1.99
Isodrin*	52.40	148.00	2.82	215.00	1560.00	7.26	251.00	776.00	3.09
Methylene Chloride	35.30	127.00	3.60	45.80	1190.00	25.98	778.00	5320.00	6.84
1,1,2,2-Tetrachloroethylene	1.45	5.16	3.56	9.61	45.50	4.73	33.10	197.00	5.95
Tetrachloroethylene	5.43	19.20	3.54	6.26	186.00	29.71	130.00	751.00	5.78
Toluene*	9460.00	20400.00	2.16	21100.00	90200.00	4.27	138000.00	176000.00	1.28
Trichloroethylene	28.40	103.00	3.63	39.80	883.00	22.19	627.00	4620.00	7.37
Arsenic	4.17	26.40	6.33	36.80	902.00	24.51	26.00	244.00	9.38
Cadmium	50.10	310.00	6.19	217.00	13600.00	62.67	1870.00	21900.00	11.71
Chromium	7.52	47.20	6.28	32.80	2160.00	65.85	326.00	4210.00	12.91
Lead*	2170.00	7220.00	3.33	26500.00	218000.00	8.23	7060.00	24000.00	3.40
Mercury*	574.00	1800.00	3.14	5490.00	68100.00	12.40	1350.00	5960.00	4.41

* Denotes a noncarcinogen. No asterisk denotes PPLV based on carcinogenic slope factors for both oral and inhalation pathways.
Interquantile Multiplier = 50% PPLV / 5% PPLV.
1/ PPLVs and confidence limits represent a cancer risk level of 1E-6 for carcinogens and a hazard index of 1.0 for noncarcinogens.

Table E.8-1. Interquantile Multipliers for Cumulative Direct PPLV Distributions 1/

	Industrial Worker			Regulated/Casual Visitor		
Contaminant	5% PPLV (mg/kg)	50% PPLV (mg/kg)	Multiplier	5% PPLV (mg/kg)	50% PPLV (mg/kg)	Multiplier
Aldrin	3.02	15.20	5.03	11.60	110.00	9.48
Benzene	10.40	104.00	10.00	57.60	621.00	10.78
Carbon Tetrachloride	2.33	19.40	8.33	13.20	128.00	9.70
Chlordane	7.58	50.30	6.64	53.90	330.00	6.12
Chloroacetic Acid*	77.10	167.00	2.17	813.00	2840.00	3.49
Chlorobenzene*	845.00	1610.00	1.91	6950.00	28800.00	4.14
Chloroform	48.40	458.00	9.46	323.00	3080.00	9.54
DDE	18.70	195.00	10.43	177.00	1280.00	7.23
DDT	36.10	220.00	6.09	151.00	1290.00	8.54
DBCP	0.24	1.89	8.01	1.17	12.40	10.60
1,2-Dichloroethane	3.39	29.90	8.82	17.40	188.00	10.80
1,1-Dichloroethylene	0.52	4.53	8.69	2.82	29.40	10.43
Dicyclopentadiene*	6650.00	16600.00	2.50	61100.00	217000.00	3.55
Dieldrin	1.40	8.42	6.01	6.45	57.30	8.88
Endrin*	318.00	681.00	2.14	2990.00	12800.00	4.28
Hexachlorocyclopentadiene*	1780.00	3800.00	2.13	14700.00	61200.00	4.16
Isodrin*	73.90	155.00	2.10	643.00	2670.00	4.15
Methylene Chloride	44.30	351.00	7.92	206.00	2040.00	9.90
1,1,2,2-Tetrachloroethylene	1.49	13.20	8.86	1.94	90.40	46.60
Tetrachloroethylene	5.87	53.30	9.08	35.70	364.00	10.20
Toluene*	7220.00	14600.00	2.02	64800.00	174000.00	2.69
Trichloroethylene	29.00	279.00	9.62	178.00	1840.00	10.34
Arsenic	26.00	138.00	5.31	79.10	938.00	11.86
Cadmium	212.00	2340.00	11.04	855.00	12400.00	14.50
Chromium	32.30	356.00	11.02	129.00	1890.00	14.65
Lead*	4460.00	16800.00	3.77	47700.00	237000.00	4.97
Mercury*	1240.00	4350.00	3.51	9850.00	68200.00	6.92

* Denotes a noncarcinogen. No asterisk denotes PPLV based on carcinogenic slope factors for both oral and inhalation pathways.
Interquantile Multiplier = 50% PPLV / 5% PPLV.
1/ PPLVs and confidence limits represent a cancer risk level of 1E-6 for carcinogens and a hazard index of 1.0 for noncarcinogens.

Table E-8-2. Monte Carlo 95 percent Confidence Intervals for the 5th Percentile Biological Worker Cumulative Direct PPLV 1/

	CARCINOGENIC				NONCARCINOGENIC			
Chemical	PPLV (mg/kg)	Lower Limit (mg/kg)	Upper Limit (mg/kg)	Percentage Deviation	PPLV (mg/kg)	Lower Limit (mg/kg)	Upper Limit (mg/kg)	Percentage Deviation
Aldrin	0.72	0.37	1.01	45	71.05	54.60	80.01	18
Benzene	11.82	4.26	15.23	46	NA	NA	NA	NA
Carbon Tetrachloride	2.51	0.92	2.92	40	36.32	22.16	42.13	27
Chlordane	3.72	2.18	4.86	36	55.17	43.83	65.14	19
Chloroacetic Acid	NA	NA	NA	NA	101.32	71.52	115.18	22
Chlorobenzene	NA	NA	NA	NA	966.54	689.13	1211.56	27
Chloroform	48.24	25.20	71.25	48	441.30	315.35	540.92	26
DDE	12.50	7.56	19.15	46	NA	NA	NA	NA
DDT	13.49	6.28	18.58	46	408.97	293.03	456.30	20
Dibromochloropropane	0.20	0.10	0.28	46	9.76	5.81	11.90	31
1,2-Dichloroethane	3.23	1.31	4.33	47	NA	NA	NA	NA
1,1-Dichloroethylene	0.52	0.25	0.65	38	452.20	279.56	519.07	26
Dicyclopentadiene	NA	NA	NA	NA	3688.29	2639.71	4257.36	22
Dieldrin	0.41	0.24	0.56	39	57.64	34.08	66.99	29
Endrin	NA	NA	NA	NA	232.30	185.68	308.29	26
Hexachlorocyclopentadiene	NA	NA	NA	NA	1059.11	829.47	1232.90	19
Isodrin	NA	NA	NA	NA	52.45	44.48	57.25	12
Methylene Chloride	35.26	19.26	53.19	48	3106.31	1859.00	3736.84	30
1,1,2,2-Tetrachloroethane	1.45	0.63	1.89	44	NA	NA	NA	NA
Tetrachloroethylene	5.43	2.61	8.02	50	547.27	326.18	601.91	25
Toluene	NA	NA	NA	NA	9543.13	5913.03	10873.22	26
Trichloroethylene	28.37	18.02	36.59	33	NA	NA	NA	NA
Arsenic	4.17	1.98	5.51	42	476.17	301.11	525.33	24
Cadmium	50.07	34.37	85.96	52	528.99	317.52	682.86	35
Chromium	7.52	5.25	13.01	52	38.70	25.55	43.20	23
Lead	NA	NA	NA	NA	2166.23	1172.88	2572.67	32
Mercury	NA	NA	NA	NA	573.97	189.81	707.99	45

DDE: Dichlorodiphenyldichlorethene

DDT: Dichlorodiphenyltrichloroethane

NA: Not Available

1/ PPLVs and confidence limits represent a cancer risk level of 1E-6 for carcinogens and a hazard index of 1.0 for noncarcinogens.

Table E.8-3 Monte Carlo 95 percent Confidence Intervals for the 5th Percentile Industrial Worker Cumulative Direct PPLV 1/

	CARCINOGENIC				NONCARCINOGENIC			
Chemical	PPLV (mg/kg)	Lower Limit (mg/kg)	Upper Limit (mg/kg)	Percentage Deviation	PPLV (mg/kg)	Lower Limit (mg/kg)	Upper Limit (mg/kg)	Percentage Deviation
Aldrin	3.02	0.76	3.44	44	119.67	106.51	145.70	16
Benzene	10.42	3.95	17.63	66	NA	NA	NA	NA
Carbon Tetrachloride	2.33	0.67	3.82	68	29.55	18.52	31.99	23
Chlordane	7.58	2.05	11.54	63	62.29	38.77	65.83	22
Chloroacetic Acid	NA	NA	NA	NA	77.10	47.86	87.13	25
Chlorobenzene	NA	NA	NA	NA	845.03	513.93	872.05	21
Chloroform	48.42	22.03	84.13	64	373.21	276.04	421.68	20
DDE	18.71	8.01	56.18	129	NA	NA	NA	NA
DDT	36.14	6.29	44.60	53	470.11	304.67	538.85	25
Dibromochloropropane	0.24	0.07	0.35	57	7.99	5.26	8.38	20
1,2-Dichloroethane	3.39	0.98	5.61	68	NA	NA	NA	NA
1,1-Dichloroethylene	0.52	0.17	0.89	70	327.64	269.37	370.04	15
Dicyclopentadiene	NA	NA	NA	NA	6645.81	5415.80	8340.52	22
Dieldrin	1.40	0.38	2.25	67	105.77	62.61	127.09	30
Endrin	NA	NA	NA	NA	317.82	236.88	341.35	16
Hexachlorocyclopentadiene	NA	NA	NA	NA	1781.93	1393.71	2053.08	19
Isodrin	NA	NA	NA	NA	73.92	42.40	86.45	30
Methylene Chloride	44.31	13.55	65.29	58	2256.68	1434.00	2614.70	26
1,1,2,2-Tetrachloroethane	1.48	0.50	2.71	74	NA	NA	NA	NA
Tetrachloroethylene	5.87	1.83	10.77	76	404.98	280.85	453.69	21
Toluene	NA	NA	NA	NA	7272.92	5186.52	8247.76	21
Trichloroethylene	28.96	13.12	49.54	63	NA	NA	NA	NA
Arsenic	25.97	5.09	35.58	59	866.57	394.02	1311.67	53
Cadmium	211.82	107.82	344.76	56	1050.69	617.83	1361.39	35
Chromium	32.30	16.19	52.51	56	73.04	55.34	90.08	24
Lead	NA	NA	NA	NA	4462.22	2765.26	5445.26	30
Mercury	NA	NA	NA	NA	1235.99	597.55	1333.45	30

DDE: Dichlorodiphenyldichlorethene

DDT: Dichlorodiphenyltrichloroethane

NA: Not Available

1/ PPLVs and confidence limits represent a cancer risk level of 1E-6 for carcinogens and a hazard index of 1.0 for noncarcinogens.

Table E.12-1 Aldrin/Dieldrin Mean BMFs Calculated by Alternative Methods

	BMF_{obs} by the Army Collocated Distributions Approach (Pre-Calibration)		BMF by the Army Calibration Procedure	BMF_{obs} by the Shell Collocated Distributions Approach		BMF_{obs} by the (EPA) Modified Paired Data Approach		$BMF_{lit/model}$		$BMF_{litcalc}$	
Trophic Box	Mean BMF	standard err.[1]	Mean BMF	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	
Soil	1	0	1	1	0	1	0	1	0	1	0
Terrestrial Plant	1.6E-02	2.0E-03	1.6E-02	6.0E-02	1.3E-02	1.8E-01	3.3E-02	6.5E-01	8.5E-01	6.5E-01	8.5E-01
Worm	2.3E-01	3.1E-02	2.3E-01	1.0E+00	3.8E-01	2.5E+00	5.4E-01	4.9E+00	4.4E+00	4.9E+00	4.5E+00
Insect	7.4E-02	1.1E-02	7.4E-02	9.7E-02	2.5E-02	4.2E-01	8.2E-02	3.1E-01	1.3E-01	1.2E+01	2.2E+01
Small Bird	2.1E-01	9.0E-03	2.1E-01	2.7E-01	1.8E-01	6.8E-01	1.2E-01	9.1E-01	2.5E-01	6.3E+01	1.1E+02
Small Mammal	6.1E-01	5.1E-02	2.7E-01	5.9E-01	1.3E-01	3.0E+00	1.4E+00	4.9E-02	1.2E-02	2.2E+00	2.8E+00
Medium Mammal	3.8E-01	4.9E-02	3.8E-01	2.7E-01	4.8E-02	1.9E+00	5.8E-01	1.7E-01	6.6E-02	1.3E+00	2.0E+00
Herptile	2.4E+00	1.8E-01	2.4E+00	2.4E+00	1.7E+00	7.7E+00	5.7E+00	9.6E-01	3.1E-01	6.4E+01	1.3E+02
Kestrel	5.0E+00	7.1E-01	2.6E+00	4.9E+00	1.1E+00	2.3E+01	1.1E+01	2.6E+00	3.1E-01	1.2E+02	2.2E+02
Owl	8.9E+00	1.7E+00	8.0E+00	6.9E+00	1.4E+00	4.1E+01	1.2E+01	8.0E+00	1.3E+00	2.3E+01	9.0E+01
Shorebird	3.6E+00	2.0E-01	3.6E+00	2.3E+00	2.0E+00	6.2E+00	2.1E+00	7.2E-01	2.3E-01	5.5E+01	1.9E+02
Heron	3.0E+00	9.8E-01	2.9E+00	3.0E+00	2.0E+00	8.6E+00	6.4E+00	2.9E+00	9.3E-01	1.2E+02	1.4E+02
Eagle	6.1E+00	1.6E+00	6.1E+00	4.4E+00	1.3E+00	2.8E+01	1.1E+01	6.1E+00	1.5E+00	6.3E+01	4.2E+01

Notes:
For the three BMF_{obs} methods, kestrel, owl, heron, and eagle BMFs were calculated with the food-web model, because there are no available field data.
For these four trophic boxes:

$$BMF_{obs(k)} = BAF_{lit(k)} * SUM_{(j)(FR(k,j)} * BMF_{obs(j)}$$

where: $BMF_{obs(k)}$ is the BMF for predator trophic box k
$BAF_{lit(k)}$ is the literature-derived BAF distribution for trophic box k
$SUM_{(j)(\cdot)}$ is the summation function over the argument j
$FR_{(k,j)}$ is the mass fraction of predator k's food from prey trophic box j
$BMF_{obs(j)}$ is the BMF for prey trophic box j

[1] The standard error is the standard deviation of the uncertainty distribution for the mean BMF.

Notes (cont'd):

The following aldrin/dieldrin BMF point estimates have been derived by EPA on the basis of tissue and soil concentration data reported in the literature (Korschgen 1970).

Trophic Box	Species	BMF_{lit}
Insect	*Poecilus chalcites* (ground beetle)	3.1E+01
Insect	*Harpalus pennsylvanicus* (ground beetle)	3.5E+00
Insect	*Gryllus assimilus* (cricket)	7.4E-01
Small Mammal	*Peromyscus maniculatus* (white-footed mouse)	3.2E+00
Herptile	*Thamnophis sirtalis & Pituophis sayi* (garter snake (2 individuals) and bull snake (1 individual))	4.0E+01
Herptile	*Bufo americanus* (toad)	1.5E+01

Table E.12-2 DDE/DDT Mean BMFs Calculated by Alternative Methods

	BMF_{obs} by the Army Collocated Distributions Approach		BMF by the Army Calibration Procedure	BMF_{obs} by the Shell Collocated Distributions Approach		BMF_{obs} by the (EPA) Modified Paired Data Approach		$BMF_{lit/model}$		$BMF_{litcalc}$	
Trophic Box	Mean BMF	standard err.[1]	Mean BMF	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]
Soil	1	0	1	1	0	1	0	1	0	1	0
Terrestrial Plant	6.6E-01	4.0E-02	6.6E-01	9.2E-01	1.4E-01	5.2E+00	7.9E-01	1.2E+00	1.1E+00	1.2E+00	1.1E+00
Worm	1.4E+00	1.7E-01	1.4E+00	1.1E+00	3.0E-01	7.8E+00	4.1E+00	3.5E+00	2.9E+00	3.5E+00	5.1E+00
Insect	7.5E-01	2.0E-02	7.5E-01	9.9E-01	2.0E-01	3.9E+01	5.8E-01	2.6E+00	5.9E+00	3.9E+00	7.7E+00
Small Bird	5.4E-01	4.0E-02	5.4E-01	8.1E-01	1.6E-01	3.3E+00	5.3E-01	1.5E+01	5.0E+00	1.8E+01	6.9E+01
Small Mammal	4.6E-01	6.0E-02	4.6E-01	6.5E-01	1.4E-01	2.8E+00	4.9E-01	4.9E-01	1.1E-01	7.1E-01	9.3E+01
Medium Mammal	4.0E+00	1.2E-01	4.9E-01	3.1E+00	4.4E-01	6.0E+00	4.7E-01	4.9E-01	1.1E-01	7.1E-01	7.9E-01
Herptile	2.5E+00	9.0E-02	1.3E+00	2.5E+00	1.6E+00	6.3E+00	4.0E+00	1.3E+00	2.8E-01	2.7E+00	4.7E+01
Kestrel	9.9E+00	3.4E+00	9.9E+00	1.4E+01	5.2E+00	5.5E+01	2.0E+01	9.9E+00	3.4E+00	1.8E+01	1.9E+02
Owl	1.8E+02	2.4E+02	3.2E+01	1.7E+02	2.3E+02	3.4E+02	4.5E+02	3.2E+01	5.2E+01	6.4E+01	2.3E+02
Shorebird	5.9E+01	5.2E+00	4.8E+01	6.0E+01	4.9E+01	1.5E+02	7.8E+01	1.0E+01	3.5E+00	1.8E+01	9.3E+01
Heron	1.8E+01	3.9E-00	1.1E+01	1.8E+01	9.7E+00	4.2E+01	2.5E+01	1.1E+01	3.2E+00	9.4E+01	2.9E+02
Eagle	1.2E+02	1.2E+02	1.9E+01	1.2E+02	1.2E+02	2.2E+02	2.5E+02	1.9E+01	2.1E+01	4.0E+01	4.0E+01

Notes:

For the three BMF_{obs} methods, kestrel, owl, heron, and eagle BMFs were calculated with the food-web model, because there are no available field data. For these four trophic boxes:

$$BMF_{obs(k)} = BAF_{lit(k)} * SUM_{(j)(FR(k,j)} * BMF_{obs(j)}$$

where: $BMF_{obs(k)}$ is the BMF for predator trophic box k
$BAF_{lit(k)}$ is the literature-derived BAF distribution for trophic box k
$SUM_{(j)(\cdot)}$ is the summation function over the argument j
$FR_{(k,j)}$ is the mass fraction of predator k's food from prey trophic box j
$BMF_{obs(j)}$ is the BMF for prey trophic box j

[1] The standard error is the standard deviation of the uncertainty distribution for the mean BMF.

Notes (cont'd):

The following DDE/DDT BMF point estimates have been derived by EPA on the basis of tissue and soil concentration data reported in the literature (Forsyth et al. 1983).

Trophic Box	Species	BMF_{lit}
Insect	*Tracheoniscus rathkei* (isopods)	9.8E+00
Insect	*Nemobius allardi* (cricket)	8.9E+00
Insect	*Gryllus pennsylvanicus* (cricket)	7.5E+00
Insect	*Melanoplus femur-rubrum* (red-legged grasshopper)	7.3E+00
Insect	*Photuirs spp.* (firefly larvae)	4.3E+00
Insect	*Carabidae* (ground beetles)	5.7E+00
Small Mammal	*Microtus pennsylvanicus* (meadow vole)	6.2E+00
Small Mammal	*Blarina brevicauda* (short-tailed shrew)	1.7E+01
Small Mammal	*Sorex cinereus* (masked shrew)	8.9E+00
Herptile	*Thamnophis sirtalis* (garter snake)	5.0E+00

Table E.12-3 Endrin Mean BMFs Calculated by Alternative Methods

	BMF_{obs} by the Army Collocated Distributions Approach		BMF by the Army Calibration Procedure	BMF_{obs} by the Shell Collocated Distributions Approach		BMF_{obs} by the (EPA) Modified Paired Data Approach		$BMF_{lit/model}$		$BMF_{litcalc}$	
Trophic Box	Mean BMF	standard err.[1]	Mean BMF	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]
Soil	1	0	1	1	0	1	0	1	0	1	0
Terrestrial Plant	1.4E-01	1.7E-02	1.4E-01	2.1E-01	3.4E-02	1.3E+00	2.2E-01	6.5E-01	9.1E-01	6.5E-01	9.1E-01
Worm	4.0E-01	5.5E-02	4.0E-01	2.4E-01	1.4E-01	1.1E+00	2.0E-01	1.3E+01	3.1E+01	1.3E+01	3.1E+01
Insect	1.0E-01	1.9E-02	1.0E-01	5.3E-02	2.2E-02	3.6E-01	1.1E-01	1.5E+00	1.4E+00	6.7E+00	1.3E+01
Small Bird	1.1E-01	1.3E-02	1.1E-01	1.3E-01	3.1E-02	9.1E-01	2.1E-01	2.1E-01	1.1E-01	7.2E+00	1.2E+01
Small Mammal	1.7E-01	2.2E-02	1.7E-01	2.7E-01	6.3E-02	1.5E+00	2.7E-01	1.3E-02	1.3E-03	1.1E-01	1.7E-01
Medium Mammal	1.6E-01	1.5E-02	3.3E-02	3.6E-01	6.0E-02	1.2E+00	1.6E-01	3.3E-02	4.0E-03	1.1E-01	1.3E-01
Herptile	1.0E+00	7.0E-02	1.0E+00	9.0E-01	5.4E-01	1.5E+00	7.3E-01	6.0E-01	2.0E-01	7.4E+00	1.4E+01
Kestrel	1.9E-01	1.1E-01	1.9E-01	2.6E-01	1.6E-01	1.3E+00	6.9E-01	1.9E-01	1.1E-01	2.5E+00	4.4E+00
Owl	2.1E+01	1.1E-01	8.8E-02	4.0E-01	2.4E-01	1.4E+00	7.7E-01	8.8E-02	4.7E-02	3.1E-01	7.3E-01
Shorebird	9.9E-01	2.2E-02	9.9E-01	6.0E-01	2.9E-01	1.1E+00	3.9E-01	8.4E-02	4.2E-02	5.5E+00	1.4E+01
Heron	1.1E-01	5.4E-02	1.1E-01	1.0E-01	6.1E-02	1.6E-01	1.0E-01	1.1E-01	5.4E-02	54E-01	8.7E-01
Eagle	2.0E-01	1.0E-01	6.7E-02	4.0E-01	1.9E-01	1.3E+00	6.8E-01	6.7E-02	3.3E-02	1.7E-01	1.8E-01

Notes:

For the three BMF_{obs} methods, kestrel, owl, heron, and eagle BMFs were calculated with the food-web model, because there are no available field data. For these four trophic boxes:

$$BMF_{obs(k)} = BAF_{lit(k)} * SUM_{(j)(FR(k,j)} * BMF_{obs(j)}$$

where: $BMF_{obs(k)}$ is the BMF for predator trophic box k
$BAF_{lit(k)}$ is the literature-derived BAF distribution for trophic box k
$SUM_{(j)(\cdot)}$ is the summation function over the argument j
$FR_{(k,j)}$ is the mass fraction of predator k's food from prey trophic box j
$BMF_{obs(j)}$ is the BMF for prey trophic box j

[1] The standard error is the standard deviation of the uncertainty distribution for the mean BMF.

Table E.12-4 Mercury Mean BMFs Calculated by Alternative Methods

	BMF_{obs} by the Army Collocated Distributions Approach		BMF by the Army Calibration Procedure	BMF_{obs} by the Shell Collocated Distributions Approach		BMF_{obs} by the (EPA) Modified Paired Data Approach		$BMF_{lit/model}$		$BMF_{litcalc}$	
Trophic Box	Mean BMF	standard err.[1]	Mean BMF	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]	Mean BMF	standard err.[1]
Soil	1	0	1	1	0	1	0	1	0	1	0
Terrestrial Plant	3.5E-02	3.0E-04	3.5E-02	1.6E-01	2.5E-02	3.1E-01	2.6E-02	4.8E-01	2.0E-01	4.8E-01	2.0E-01
Worm	6.2E-01	8.8E-02	6.2E-01	4.0E-01	8.2E-02	8.1E-00	9.5E-02	1.4E+00	1.3E+00	1.4E+00	1.3E+00
Insect	1.1E-02	1.0E-03	1.1E-02	1.3E-01	3.4E-02	2.7E-01	2.9E-02	3.2E-02	5.8E-03	4.4E-01	2.2E-01
Small Bird	1.7E-01	4.0E-04	1.1E-01	1.9E-01	3.4E-02	3.4E-01	2.1E-02	1.1E-01	6.3E-02	4.6E-01	9.7E-40
Small Mammal	2.5E-03	1.0E-03	5.5E-01	1.5E-02	2.2E-02	1.7E-01	1.1E-01	1.4E+00	5.8E-01	1.2E+01	7.5E+00
Medium Mammal	2.8E-01	5.5E-02	2.8E-01	3.3E-01	5.2E-02	7.3E+00	3.0E+00	2.6E+00	1.1E+00	1.3E+01	7.9E+00
Herptile	6.0E-01	1.1E-02	6.0E-01	7.8E-01	1.2E-01	8.2E-01	1.1E-01	6.8E-01	3.5E-01	1.3E+01	1.1E+01
Kestrel	4.1E-02	2.3E-02	3.2E-01	6.8E-02	4.1E-02	1.8E-01	1.2E-01	3.2E-01	2.5E-01	6.3E+00	5.5E+00
Owl	2.1E-01	1.2E-01	2.6E-01	2.4E-01	1.3E-01	4.8E+00	3.6E+00	2.6E-01	1.5E-01	9.3E+00	9.1E+00
Shorebird	1.2E+00	2.0E-04	1.2E+0	1.6E-01	2.9E-03	1.8E-02	2.8E-03	6.4E-03	3.5E-03	2.5E-01	1.8E-01
Heron	6.2E-01	9.9E-01	6.8E-01	7.2E-01	1.2E+00	7.6E-01	1.2E+00	6.8E-01	1.1E+00	8.6E+00	1.1E+01
Eagle	2.3E-01	1.4E-01	2.3E-01	2.6E-01	1.4E-01	5.4E+00	4.2E+00	2.3E-01	1.4E-01	9.2E+00	2.2E+39

Notes:

For the three BMF_{obs} methods, kestrel, owl, heron, and eagle BMFs were calculated with the food-web model, because there are no available field data. For these four trophic boxes:

$$BMF_{obs(k)} = BAF_{lit(k)} * SUM_{(j)(FR(k,j)} * BMF_{obs(j)}$$

where: $BMF_{obs(k)}$ is the BMF for predator trophic box k
$BAF_{lit(k)}$ is the literature-derived BAF distribution for trophic box k
$SUM_{(j)(\cdot)}$ is the summation function over the argument j
$FR_{(k,j)}$ is the mass fraction of predator k's food from prey trophic box j
$BMF_{obs(j)}$ is the BMF for prey trophic box j

No mercury BMFs were derived directly from the scientific literature

[1] The standard error is the standard deviation of the uncertainty distribution for the mean BMF.

Table E.12-5 Sample Size, Number of BCRLs, and Selected Replacement Method for Each Trophic Box

Trophic Box	Number of Data Points	Number of NA	Number of Values	Number of Hits	Percent Hits	BCRL Method
ALDRIN						
Herptile	7	0	7	2	28.57%	1/2 CRL
Insect	98	3	95	9	9.47%	R
Medium Mammal	156	0	156	0	0.00%	1/2 CRL
Shorebird	10	0	10	0	0	1/2 CRL
Small Bird	83	0	83	1	1.20%	1/2 CRL
Small Mammal	93	0	93	6	6.45%	R
Terrestrial Plant	236	4	232	6	2.59%	1/2 CRL
Worm	74	3	71	3	4.23%	1/2 CRL
DIELDRIN						
Herptile	7	0	7	7	100.00%	NA
Insect	98	3	95	61	64.21%	EVR
Medium Mammal	156	2	154	107	69.48%	*
Prairie Dog	128	2	126	96	76.19%	EVR
Desert Cottontail	28	0	28	11	39.29%	EVR
Shorebird	10	0	10	10	100%	NA
Small Bird	83	0	83	57	68.67%	*
Vesper Sparrow	5	0	5	3	60.00%	EVR
Meadowlark	10	0	10	9	90.00%	EVR
Mourning Dove	68	0	68	45	66.18%	EVR
Small Mammal	93	3	90	66	73.33%	*
Deer Mouse	90	3	87	63	72.41%	EVR
Ground Squirrel	3	0	3	3	100.00%	NA
Terrestrial Plant	236	5	231	63	27.27%	EVR
Worm	74	3	71	47	66.20%	EVR

* method applied to separate species

R = robust estimate, EVR = expected value robust estimate, 1/2 CRL = replacement by 1/2 CRL

Table E.12-5 Sample Size, Number of BCRLs, and Selected Replacement Method for Each Trophic Box

Trophic Box	Number of Data Points	Number of NA	Number of Values	Number of Hits	Percent Hits	BCRL Method
ENDRIN						
Herptile	7	0	7	2	28.57%	1/2 CRL
Insect	98	3	95	13	13.68%	R
Medium Mammal	156	0	156	2	1.28%	1/2 CRL
Shorebird	10	0	10	5	50%	R
Small Bird	83	0	83	13	15.66%	R
Small Mammal	93	1	92	2	2.17%	1/2 CRL
Terrestrial	236	4	232	6	2.59%	1/2 CRL
Worm	74	7	67	12	17.91%	R
DDE						
Herptile	7	0	7	1	14.29%	1/2 CRL
Insect	98	3	95	2	2.11%	1/2 CRL
Medium Mammal	156	47	109	2	1.83%	1/2 CRL
Shorebird	10	0	10	10	100%	NA
Small Bird	83	0	83	2	2.41%	1/2 CRL
Small Mammal	93	0	93	5	5.38%	1/2 CRL
Terrestrial Plant	236	22	214	3	1.40%	1/2 CRL
Worm	74	2	72	12	16.67%	EVR
DDT						
Herptile	7	0	7	1	14.29%	1/2 CRL
Insect	98	3	95	3	3.16%	1/2 CRL
Medium Mammal	156	47	109	1	0.92%	1/2 CRL
Shorebird	10	0	10	50	50%	EVR
Small Bird	83	0	83	0	0.00%	1/2 CRL
Small Mammal	93	0	93	3	3.23%	1/2 CRL
Terrestrial Plant	236	22	214	4	1.87%	1/2 CRL
Worm	74	2	72	8	11.11%	EVR

R = robust estimate, EVR = expected value robust estimate, 1/2 CRL = replacement by 1/2 CRL

Table E.12-5 Sample Size, Number of BCRLs, and Selected Replacement Method for Each Trophic Box

Trophic Box	Number of Data Points	Number of NA	Number of Values	Number of Hits	Percent Hits	BCRL Method
MERCURY						
Herptile	7	1	6	3	50.00%	R
Insect	98	32	66	3	4.55%	1/2 CRL
Medium Mammal	156	18	138	1	0.72%	1/2 CRL
Shorebird	10	0	10	9	90%	R
Small Bird	83	0	83	0	0.00%	1/2 CRL
Small Mammal	93	1	92	7	7.61%	R
Terrestrial Plant	236	68	168	0	0.00%	1/2 CRL
Worm	74	1	73	38	52.05%	R

R = robust estimate, EVR = expected value robust estimate, 1/2 CRL = replacement by 1/2 CRL

Table E.12-6 Fraction of BCRL Samples Not Estimated During Spatial Interpolation

	BCRL not estimated/total BCRL	Percent
Aldrin	2,500/7,534	33.2
Dieldrin	1,563/6,659	23.5
Endrin	2,676/7,766	34.5
DDE	3,700/8,278	44.7
DDT	2,163/8,073	39.2
Arsenic	1,222/5,568	21.9
Mercury	2,049/6,473	31.7
Chlordane	4,374/8,110	53.9
DBCP	5,379/7,121	75.5
DCPD	5,565/7,065	78.8
CPMS	6,171/7,251	85.1
$CPMSO_2$	5,589/7,174	77.9
Cadmium	2,653/6,411	41.4
Copper	62/952	6.5

Trophic Box	Ald/Dld	DDE/DDT	Endrin	Mercury
{⟨ESC⟩, TC} Sample Correlation Coefficients				
Herptile	-0.27	-0.27	0.43	-0.24
Insect	0.32	0.15	0.19	-0.04
Medium Mammal	-0.04	-0.02	-0.06	-0.01
Shorebird	-0.24	-0.30	0.82	0.05
Small Bird	0.00	-0.03	-0.06	0.05
Small Mammal	0.37	0.54	0.00	0.04
Terrestrial Plant	0.18	0.01	-0.08	-0.01
Worm	0.03	-0.03	-0.02	0.58
{LN(⟨ESC⟩), LN(TC)} Sample Correlation Coefficients				
Herptile	-0.23	-0.43	0.49	-0.13
Insect	0.52	0.25	0.18	0.16
Medium Mammal	0.17	0.12	0.17	-0.01
Shorebird	-0.31	-0.46	0.68	0.05
Small Bird	0.21	-0.05	0.11	-0.01
Small Mammal	0.78	0.39	0.12	0.09
Terrestrial Plant	0.24	-0.01	-0.05	-0.09
Worm	0.58	0.07	0.28	0.35

Trophic Box	Ald/Dld	DDE/DDT	Endrin	Mercury
{ESC, TC} Sample Correlation Coefficients				
Herptile	-0.23	-0.23	0.37	-0.20
Insect	0.59	0.17	0.19	-0.06
Medium Mammal	0.12	-0.02	0.14	-0.02
Shorebird	-0.21	-0.70	0.74	0.09
Small Bird	0.00	-0.05	-0.06	0.06
Small Mammal	0.63	0.20	0.04	0.02
Terrestrial Plant	-0.13	-0.05	-0.09	0.01
Worm	0.13	0.19	0.66	0.50
{LN(ESC), LN(TC)} Sample Correlation Coefficients				
Herptile	-0.20	-0.37	0.42	-0.11
Insect	0.53	0.37	0.26	0.16
Medium Mammal	0.15	0.11	0.31	0.01
Shorebird	-0.28	-0.41	0.61	0.08
Small Bird	0.22	-0.07	0.13	-0.04
Small Mammal	0.58	0.29	0.20	0.09
Terrestrial Plant	0.00	-0.02	-0.05	-0.15
Worm	0.58	0.24	0.59	0.50

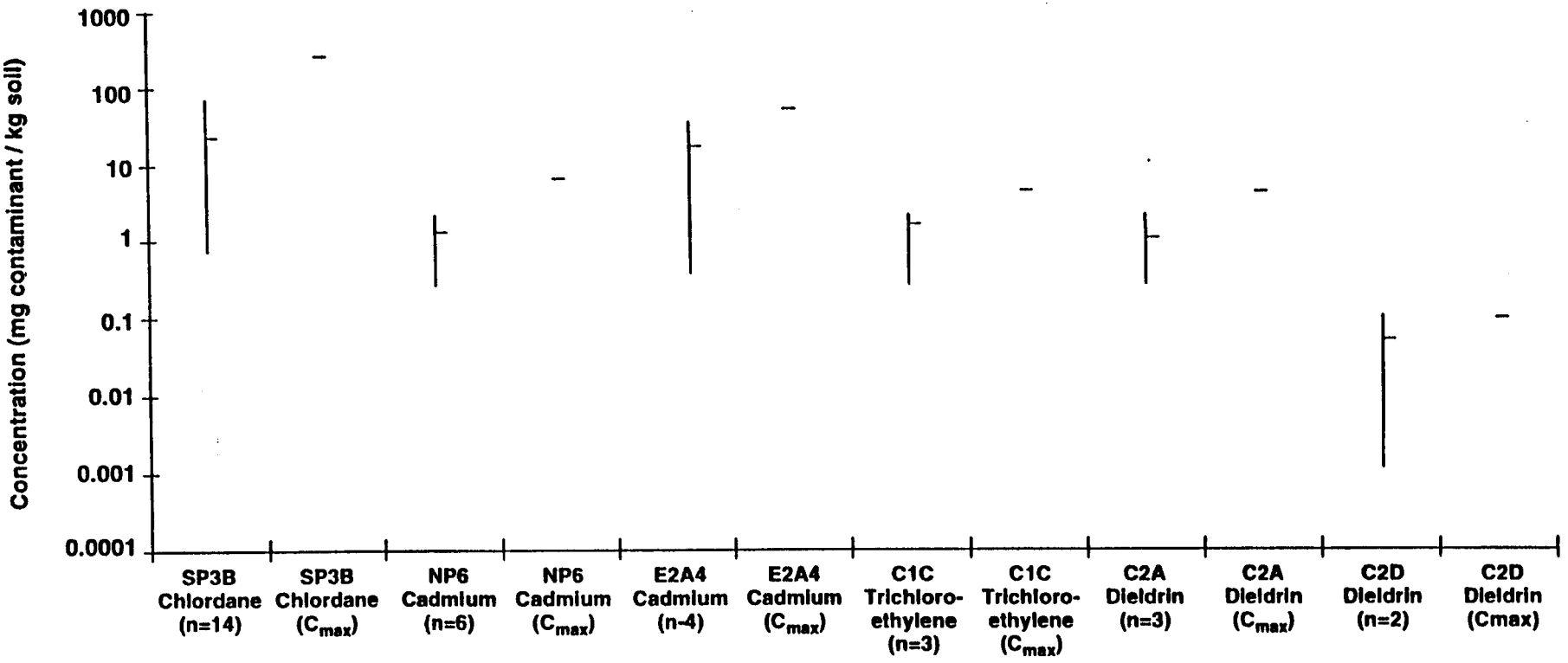
Table E.12-9 Comparison of Army BMF_{obs} Calculated Based on Different {⟨ESC⟩, TC} Correlations

	Mean BMF(R)			Ratio of Mean BMFs
	R=0	R=0.5/1	R=1	R=0/R=1
ALDRIN/DIELDRIN				
Herps	3.78E+00	2.39E+00	1.61E+00	2.5
Insct	1.88E-01	7.31E-02	2.84E-02	6.6
MdMml				
Prairie Dog	1.14E+00	5.09E-01	2.28E-01	5.0
Cotton Tail	2.51E-02	1.28E-02	6.54E-03	3.8
ShBrd	2.3631087	1.595262022	1.07691234	2.2
SmBrd				
Vesper Sparrow	4.84E-01	3.44E-01	2.45E-01	2.0
Meadow Lark	1.08E+00	7.72E-01	5.49E-01	2.0
Mourning Dove	6.79E-02	3.61E-02	1.92E-02	3.5
SmMml				
Deer Mouse	9.41E-01	4.23E-01	1.90E-01	5.0
Ground Squirrel	2.99E-01	2.47E-01	2.05E-01	1.5
TrPlt	3.31E-02	1.69E-02	7.67E-03	4.3
Worms	5.25E-01	2.30E-01	1.01E-01	5.2
DDE/DDT				
Herps	3.1896821	2.5253817	1.999432	1.6
Insct	0.92321	0.7535694	0.6151004	1.5
MdMml	4.7820064	3.9503738	3.2633693	1.5
ShBrd	100.782225	58.3256522	33.7547788	3.0
SmBrd	0.8933909	0.5362075	0.3218283	2.8
SmMml	1.0391939	0.4528421	0.1973318	5.3
TrPlt	1.0145343	0.6623243	0.4323891	2.3
Worms	2.9412893	1.3618282	0.6305317	4.7
ENDRIN				
Herps	1.5576912	1.02345183	0.67243984	2.3
Insct	0.3173195	0.09978466	0.03137841	10.1
MdMml	0.2793259	0.16037308	0.09207712	3.0
ShBrd	0.55621548	0.392515403	0.27699398	2.0
SmBrd	0.2292344	0.10991613	0.05270394	4.3
SmMml	0.3670129	0.16342657	0.07277194	5.0
TrPlt	0.2908392	0.141778	0.06911381	4.2
Worms	0.920067	0.39536027	0.16988953	5.4
MERCURY				
Herps	0.6658768	0.596291393	0.53397779	1.2
Insct	0.01658519	0.010746537	0.00696332	2.4
MdMml	0.90299897	0.27778749	0.08545512	10.6
ShBrd	0.02008474	0.018612284	0.01724778	1.2
SmBrd	0.17018094	0.167492798	0.16484711	1.0
SmMml	0.01127593	0.002441864	0.0005288	21.3
TrPlt	0.03696061	0.034816747	0.03279724	1.1
Worms	1.45628472	0.614608262	0.25938837	5.6

1/ Approximately equal to the ARMY BMF_{obs} (variation due to rounding)5.6

Table E.12-10 Sensitivity of Army BMF_{obs} to Different Correlations and BCRL Methods for Small Bird, Endrin

BCRL Method	R=0.1	R=0.5	R=1.0
1/2 CRL	0.25	0.13	0.06
Robust	0.23	0.11	0.05
Expected Value Robust	0.21	0.10	0.05

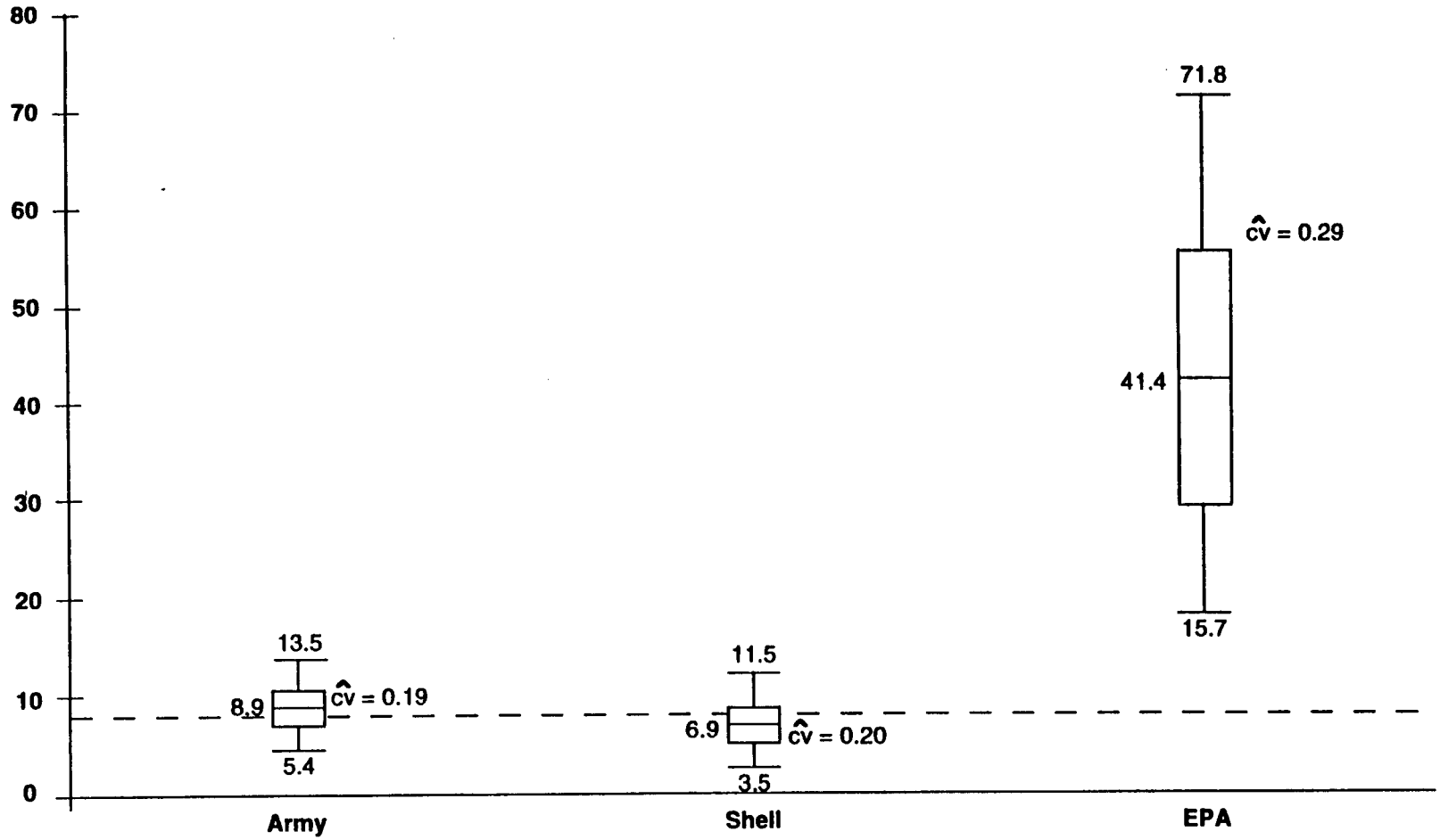


RMA IEA/RC 6.94 dr

Figure E.3-1

Soil Concentration: Sample Arithmetic Means, (Bootstrap Method) 95 percent Upper and Lower Confidence Limits, and C_{max}

Rocky Mountain Arsenal
Prepared by: Enserch Environmental Corporation



RMA IEA/RC 2.94.dr

* The ranges spanned by the bars correspond to the ranges of 100 case Latin Hypercube samples from the BMF distributions. The numbers above and below the bars are the maximum and minimum sample values. The ends of the boxes correspond to 25th and 75th percentiles of the sample distributions. The bar within the box indicates the sample median. The numbers to the left of the boxes are the expected values of the mean BMF distributions. The dashed line corresponds to the Army's calibrated owl aldrin/dieldrin BMF value of 8.0. Also indicated is the sample coefficeint of variation ($\hat{cv}$ = sample standard deviation/mean).

Figure E.12-1

Mean BMF distributions for owl, aldrin/dieldrin computed using prey BMFs calculated by Army and Shell collocated distribution methods, and the (EPA) paired data approach*

Rocky Mountain Arsenal
Prepared by: Ebasco Services Incorporated

ATTACHMENT E.3-1

SUMMARY OF SHELL'S JANUARY 1992

"DIELDRIN SOIL LOSS POSITION"

DIELDRIN SOIL LOSS

EXECUTIVE SUMMARY

This report is the culmination of a concerted effort to gain a greater understanding of dieldrin disappearance from soils based on available pertinent literature and RMA site-specific information.

Based on presently available site-specific data, the dieldrin loss rate due to volatilization at RMA is 1.6% per year. This value is based only on losses by volatilization; however, published data from other sites show that other mechanisms, such as biodegradation and chemical transformation may substantially increase the rate of loss.

A study by Freeman et al. (1975) and Glotfelty et al. (in prep.) in Coshocton, Ohio indicates a total disappearance rate of about 7% per year consistently over the duration of the whole study (dieldrin ranging from 3.6-0.38 ppm) with volatilization accounting for only 1.5 to 2.5% per year. This value is consistent with the value reported for dieldrin in the RMA Offpost RI/FS. Other published reports for losses by all mechanisms in agricultural applications in temperate climates indicate average dieldrin disappearance rates ranging from 5 to 26% per year over the study duration.

The results from analysis of data from dieldrin application studies and long time data from aldrin application studies are in very good agreement, dieldrin loss rates ranging from 5 to 24% per year and 6 to 26% per year, respectively. In both cases, the loss of dieldrin from soils is well characterized by a pseudo-first order decay.

A continuous loss of dieldrin was observed, even at low concentrations (less than 0.1 ppm) and after long elapsed time since application, in most (>80%) of the studies reviewed.

Moreover, dieldrin loss from soils by volatilization was measured at RMA at concentrations ranging from 0.1 to 2 ppm. Therefore, it is likely that dieldrin soil concentrations diminish with time with no threshold or plateau effects. Based on site-specific volatilization data, the dieldrin loss rate by this single mechanism at RMA is at least 1.6% per year.

I. INTRODUCTION

The RMA On-Post Risk Characterization/Integrated Endangerment Assessment addresses the potential exposure pathways for humans and biota for contaminants of concern in soils. Dieldrin is generally considered to be one of the most pervasive of the organochlorine pesticides at RMA. An extensive review of pertinent literature has been conducted to evaluate the natural loss of dieldrin from soils. The purposes of this paper are to discuss the relevance to RMA of information found in the literature and present the results of RMA site-specific dieldrin loss data.

A realistic estimate of future dieldrin concentrations in RMA soils may be useful in RMA Feasibility Studies. The current methodology for RMA On-Post Risk Characterization and Off-Post Endangerment Assessment does not consider reductions in dieldrin soil concentrations over time. However, basis the published literature and site-specific studies, it can be concluded that dieldrin soil concentrations are not constant, but diminish with time; therefore, the carcinogenic risks due to the presence of dieldrin correspondingly diminish with time.

Additional information regarding this subject may be found in a report prepared by the Shell Oil Company titled "Dieldrin Soil Loss Position." The report was transmitted to the Army via a letter to Kevin T. Blose from W. J. McKinney dated July 1, 1992 and may be found in the Administrative Record for RMA.

II. DIELDRIN FIELD LITERATURE SURVEYS

The literature on dieldrin loss rates from soils have several distinct differences. The first major distinction is the chemical initially applied, aldrin- or dieldrin-only. This is important since the formation rate of dieldrin from aldrin must be taken into account when aldrin is present (Decker et al., 1965). The soil moisture and temperature, the soil type, method of application, and application depth are also important factors in determining the loss rate of dieldrin from soils (Kushwaha et al., 1979). Of these, the most significant is the soil condition as affected by the local climate and seasonal variations. The warm moist conditions in tropical areas are expected to enhance both volatilization and biodegradation relative to temperate areas (Agnihotri et al., 1977). With these distinctions in mind, the literature was reviewed and separated into four groups: dieldrin-only application in temperate climates, dieldrin-only application in tropical climates, aldrin application in temperate climates, and aldrin application in tropical climates. The literature reviewed, including a review article by Scheunert et al. (1989), and a laboratory study by Kushwaha and Gupta (1980), are listed in Table 1.

Table 1. Published Literature Reviewed

Study Authors	Chemical Applied	Climate	Study Duration	Number of Sampling Events
Beyer and Gish, 1980	Dieldrin	Temperate	11 years	14
Cliath and Spencer, 1971	Dieldrin	Temperate	2 years	3
Freeman et al., 1975; Glotfelty et al., in prep.	Dieldrin	Temperate	21 years	12-14
Gilbert and Lewis, 1982	Dieldrin	Temperate	4 years	6
Guenzi et al., 1971	Dieldrin	Temperate	6 months	5-6
Harris et al., 1977; Miles et al., 1978	Dieldrin	Temperate	4 years	4
Nash and Woolson, 1967; Nash and Harris, 1973	Dieldrin	Temperate	16 years	8-9
Stewart and Fox, 1971	Dieldrin	Temperate	10 years	2
Wingo, 1966	Dieldrin	Temperate	8 years	6-7
Agnihotri et al., 1977	Dieldrin	Tropical	6 months	9
Agnihotri et al., 1984	Dieldrin	Tropical	4 months	5
Bess and Hylin, 1970	Dieldrin	Tropical	7 years	2
El Zorgani, 1976	Dieldrin	Tropical	7 weeks	8
Talekar et al., 1977; Talekar et al., 1983;	Dieldrin (annually)	Tropical		
Scheunert, 1989	Dieldrin/Aldrin	Review		
Wang et al., 1988	Dieldrin/Aldrin (residues only)	Tropical		
Ball, 1983	Aldrin (to beetles)	Temperate		
Kushwaha and Gupta, 1980	Aldrin	Laboratory		
Beck et al., 1962	Aldrin	Temperate	110 years	2
Bruce and Decker, 1966	Aldrin	Temperate	4 years	4
Decker et al., 1965	Aldrin	Temperate	4 years	11-22

Table 1 (continued)

Study Authors	Chemical Applied	Climate	Study Duration	Number of Sampling Events
Elgar, 1966; Elgar, 1975	Aldrin	Temperate	4 years	6-7
Harris et al., 1977; Miles et al., 1978	Aldrin	Temperate	4 years	4
Korschgen, 1971	Aldrin	Temperate	6 years	11
Lichtenstein and Schulz, 1960; Lichtenstein and Schulz, 1965; Lichtenstein et al., 1970	Aldrin	Temperate	8 years	5-7
Onsager et al., 1970	Aldrin	Temperate	3 years	7
Scheunert et al., 1977	Aldrin	Temperate	1 year	1
Stewart and Fox, 1971	Aldrin	Temperate	10 years	2
Nash and Woolson, 1967; Nash and Harris, 1973	Aldrin	Temperate	16 years	8-9
Wilkinson et al., 1964	Aldrin	Temperate	9 years	1
Agnihotri et al., 1974	Aldrin	Tropical	100 days	6
Agnihotri et al., 1977	Aldrin	Tropical	6 months	9
Agnihotri et al., 1984	Aldrin	Tropical	4 months	5
Bess and Hylin, 1970	Aldrin	Tropical	7 years	2
El Zorgani, 1976	Aldrin	Tropical	7 weeks	8
Gupta and Kavadia, 1979; Kushwaha and Gupta, 1979	Aldrin	Tropical	2 years	5-8
Kathpal et al., 1981	Aldrin	Tropical	8.5 months	3
Kushwaha et al., 1978	Aldrin	Tropical	250 days	8
Kushwaha et al., 1981	Aldrin	Tropical	221 days	3
Singh et al., 1985	Aldrin	Tropical	9 months	6
Singh et al., 1991	Aldrin	Tropical	682 days	7

Dieldrin-Only Application Studies

All dieldrin-only application study data were converted to percent of initial dieldrin remaining. When sufficient data existed, the data were analyzed using several different methods. Results are presented here for analyses using two models: pseudo-first order decay and a two-stage process allowing for varying rates of decay. Overall, the analyses indicate that there is no basis to prefer the more complex two stage model over a simple pseudo-first order decay when applied to dieldrin-only application studies. The data is adequately described using a pseudo-first order decay process (Jefferys and Berger, 1992).

Study data from temperate climates, with dieldrin concentrations ranging from 100 ppm to less that 0.1 ppm, indicate an average dieldrin disappearance rate of between 5 and 24% per year. Results from the study with the longest duration (Freeman et al., 1975; Glotfelty et al., in prep.) are shown in Figure 1. These data indicate a disappearance rate of about 5% per year consistently over the duration of the whole study with volatilization accounting for 1.5 to 2.5% per year. An interesting observation is that there appears to be two distinct groups within the temperate studies as summarized in Figure 2. The studies of Freeman et al. (1975) and Glotfelty et al. (in prep.), and Nash and Woolson (1967) and Nash and Harris (1973), show loss rates ranging from 5 to 8% per year. While, the studies of Beyer and Gish (1980); Gilbert and Lewis (1982); and Wingo (1966), show loss rates ranging from 14 to 24% per year (excluding the high concentration study of Beyer and Gish which exhibits a loss rate of 6% per year). These differences are most likely due to differences in the soil and climatic conditions for each of the studies.

Data from tropical areas obtained by Agnihotri et al. (1977) and Agnihotri et al. (1984) indicate dieldrin loss rates of greater than 50% in six months; whereas, the El Zorgani (1976) data showed dieldrin loss rate of 90% in seven weeks. However, the extraction method used by El Zorgani (hexane-only) fails to recover dieldrin consistently (Salanitro, 1992) and thus the validity of this study is questionable.

The dieldrin-only application study in temperate climate of Stewart and Fox (1971) and in tropical climate of Bess and Hylin (1970) present only two data points. Their data, taken initially and seven or ten years after application, show a decline in dieldrin concentration of 11-17% per year and 45-47% per year, respectively. The dieldrin-only application studies of Cliath and Spencer (1971); Guenzi et al. (1971); and Harris et al. (1977) and Miles et al. (1978), failed to present enough data for a mathematical description to be made; however, their data show a decline in dieldrin concentration over time. The dieldrin-only application studies of Talekar et al. (1977 and 1983) applied dieldrin annually; therefore, no conclusions were made from the data.

Aldrin Application Studies

All aldrin application study data were converted to percent of initial aldrin remaining. Aldrin application studies having sufficient data were analyzed using two models: pseudo-first order decay for long time data and series reaction (e.g., mother-daughter relationship) for all data.

The analyses indicated that a mother-daughter relationship exists between aldrin and dieldrin. However, after long periods of time, the dieldrin disappearance rate could be approximated with a pseudo-first order decay model. Results from the aldrin application study of the longest duration with sufficient dieldrin data in a temperate climate (Lichtenstein and Schulz, 1960; 1965; and Lichtenstein et al., 1970) are given in Figure 3. Data from this study and other aldrin application studies in temperate climates (Decker et al., 1965; Elgar, 1966; 1975; Nash and Woolson, 1967 and Nash and Harris, 1973; and Onsager et al., 1970), give a dieldrin disappearance rate ranging from 15 to 45% per year based on the complete mother-daughter analysis. In addition, analysis of the data at long time indicate dieldrin loss rates ranging from 6 to 26% per year which is in good agreement with the dieldrin-only application studies.

The aldrin application studies in tropical climates (Agnihotri et al., 1977 and Singh et al., 1991) showed similar results as aldrin application studies in temperate climates with the exception that the dieldrin disappearance rate was greatly enhanced (to greater than 90% per year).

The temperate study of Ball (1983) presents data on aldrin application to western corn rootworm beetles and indicates a dieldrin loss rate of 7.4% per year based on a decline in the LD_{50} values measured between 1962 and 1981. The temperate study of Bruce and Decker (1966) and the tropical studies of Agnihotri et al. (1974); Agnihotri et al. (1984); and Singh et al. (1985) fail to present enough date for a mathematical description to be made. Wang et al. (1988) present data on aldrin and dieldrin residue in soil; however, the chemical application history is unavailable. The studies by El Zorgani (1976); Gupta and Kavadia (1979) and Kushwaha et al. (1979); Kathpal et al. (1981); Kushwaha and Gupta (1980; Kushwaha et al. (1978); and Kushwaha et al. (1981), used hexane (only) in their extractions which fails to adequately analyze dieldrin (Salanitro, 1992); therefore, no conclusions were drawn from these studies.

Several aldrin application studies (Beck et al., 1962; Bess and Hylin, 1970; Harris et al., 1977; and Miles et al., 1978; Scheunert et al., 1977; Stewart and Fox, 1971; and Wilkinson et al., 1964), fail to present enough data (e.g., only a single dieldrin data point) to estimate the dieldrin loss rate. The study data presented by Korschgen (1971) indicate an appearance of aldrin several years into the study; therefore, no conclusions were drawn from the data. Scheunert (1989) presents a review of available aldrin and dieldrin literature.

Conclusions of Literature Studies

In the studies considered in this review, continuing losses are evident at dieldrin levels ranging from 100 to less than 0.1 ppm in soils over many years. The dieldrin loss was attributed to a combination of natural processes, including biotransformation, chemical transformation, and volatilization. There is no evidence of any threshold concentration, i.e.,

minimum substrate, bound residues, etc., at which there is a cessation of the dieldrin loss process. In addition, studies in temperate climates by Cliath and Spencer (1971); Stewart and Fox (1971); and Scheunert et al. (1977), showed that dieldrin remained in the treated zone with little downward vertical migration.

In summary, a critical review of the literature indicates average disappearance rates for dieldrin in soil of 5 to 26% per year over the study duration under temperate conditions. Freeman, et al. (1975) and Glotfelty et al. (in prep.) found a disappearance rate of about 7% per year with a documented volatilization rate of 1.5 to 2.5% per year. The 7% per year disappearance rate was consistent over the entire duration of the study, even when disregarding earlier portions of data. Other mechanisms, such as, biodegradation and chemical transformations, are probably responsible for the remaining 4.5 to 5.5% per year not accounted for by volatilization.

III. RMA SITE-SPECIFIC INFORMATION

At the request of the Army to provide RMA site-specific data that demonstrates the loss of dieldrin from soils, a dieldrin volatilization loss study was conducted in October 1991. In addition, RMA soils were analyzed for transformation intermediates of aldrin and dieldrin to validate that degradation occurs at RMA.

Dieldrin Volatilization

Test sites were selected for a range of dieldrin concentrations (from 0.1 to 2 ppm) in the surficial soils. Each of the sites was sampled at the locations indicated in Figure 4. The concentrations of aldrin and dieldrin were measured in several 0-2 inch samples per site, and the averages (based on 3 samples) are given in Table 2.

Volatilization loss monitoring studies conducted at RMA during the summer of 1988 and October 1991 indicate that surficial soil moisture content, ambient temperature, and surficial soil dieldrin concentration appear to be the parameters that most significantly affect the volatilization loss of dieldrin from surficial soils at RMA. Diurnal cycle monitoring studies indicate that volatilization loss rates increase with increases in temperature, while measurements from different locations indicate that volatilization loss rates also increase with surficial soil dieldrin concentration. Surficial soil moisture content effects were assessed by comparing volatilization loss rates before and after precipitation events. Dramatic increases in volatilization loss rates were measured immediately after precipitation events.

This increased rate decreased during the period in which the soils dried out.

Table 2. Summary of Analytical Results from the Analyses of RMA Soil Samples

Site No.	Aldrin ppm	Dieldrin ppm	Photodieldrin ppm	Diacid ppm
1*	0.3	2	0.8	0.2
2*	0.08	0.5	0.3	0.09
3*	0.03	0.15	0.08	0.003
4	0.08	0.4	0.2	0.008

Practical quantitative limit = 0.001 to 0.005 ppm x dilution factor

*Indicates the location of volatilization loss monitoring tests.

An empirical long-term average volatilization loss rate prediction has been calculated based on results from RMA volatilization loss monitoring studies. Calculations mimic loss rate

cycles as measured in the RMA field studies, i.e., volatilization loss rates remain at a "base" level corrected for temperature during dry periods, then rapidly increase (also corrected for temperature) following precipitation events. A decline in the predicted volatilization rate follows over a specified time period as the soil dries and the base volatilization rate is again achieved. Temperature corrections are based on thermodynamic considerations and published data on the relationship between dieldrin vapor pressure and temperature. Hourly meteorological data (temperature, precipitation) for a one year period (October 1989-September 1990) are combined with RMA site-specific measured values to calculate hourly average volatilization loss rates, which are then used to determine an average annual volatilization loss rate.

Average annual volatilization loss rates were calculated for a RMA site containing about 1.4 ppm dieldrin concentrations in surficial soils (0-2 inch depth). The calculated annual average volatilization rate was 0.61 ng/cm^2-day. Based on soil sampling data to a depth of 12 inches, this corresponds to a 1.6% per year dieldrin loss rate at RMA. Demonstrated losses due to volatilization support the position that the dieldrin concentration will continuously decline in the future.

Dieldrin Transformation

Dieldrin transformation can occur by several pathways including biodegradation and chemical transformation processes. For example, dieldrin loss may occur when it is contacted with clays under appropriate conditions. There is no evidence of any threshold concentration, i.e., minimum substrate, bound residues, etc., at which there is a cessation of the dieldrin loss process. The potential transformation intermediates most observed in literature studies were photodieldrin, aldrin-trans-diol, and dihydrochlordene dicarboxylic acid. Analytical standards of these chemicals were obtained and analytical protocols developed.

RMA soil samples from the volatilization loss study sites shown in Figure 4 were analyzed for the aforementioned transformation intermediates. The data in Table 2 show that

photodieldrin is the predominant transformation intermediate as is also reported in the literature. The diacid was also found but in lesser concentrations. The diol was not found in RMA soils and laboratory studies showed that it appears to transform rapidly in the presence of RMA soil.

The photodieldrin and diacid concentrations ranged up to 60% of the dieldrin concentration at any of the sites. It is likely that dieldrin intermediates are subject to the same mechanisms (e.g., degradation) as dieldrin and, therefore, are likely to diminish with time.

In addition, a recent publication of Atkinson (1988) finds the atmospheric lifetime of dieldrin to be about one day. The actual calculations gives ten hours, but due to uncertainties and the fact that the OH concentration vanishes after sunset, one day appears reasonable.

IV. CONCLUSIONS

The various literature studies show that there is a steady decline in dieldrin concentrations after the use of aldrin and/or dieldrin has declined or ceased. This conclusion is strongly supported by the results of the RMA site-specific experimental work, which conclusively demonstrate the loss of dieldrin by volatilization. Other natural transformation processes including biodegradation and chemical transformation are observed in literature studies and are presumed to also occur in RMA soils. However, RMA site-specific data is not currently available to quantify the loss rates of these processes.

MKE BIBLIOGRAPHY FOR THE DIELDRIN SOIL LOSS PROJECT

Agnihotri, N.P., S.Y. Pandey, and H.K. Jain, "Persistence of BHC and Aldrin in Soil and Translocation in Mung (Phaseolus Aureus L.) and Lobia (Vigna Sinensis Siva)", Indian J. Ent., 36(4), 261-267, 1974.

Agnihotri, N.P., S.Y. Pandey, H.K. Jain, and K.P. Srivastava, "Persistence of Aldrin, Dieldrin, Lindane, Heptachlor, and p,p'-DDT in Soil", J. Ent. Res., 1(1), 89-91, 1977.

Agnihotri, N.P., H.K. Jain, and K.P. Srivastava, "Loss of Aldrin, Dieldrin, Heptachlor and Lindane in Soil", Indian Journal of Agricultural Chemistry, 17(1), 33-37, 1984.

Atkinson, R. Proceedings of the NATO Advanced Workshop on the Long Range Atmospheric Transport of Natural and Contaminant Substances, January 10-17, 1988, edited by A. Knap, NATO ASI series. Series C, Mathematical and Physical Sciences: No. 297.

Ball, H.J. "Aldrin and Dieldrin Residues in Nebraska Soils as Related to Decreasing LD_{50} Values for the Adult Western Corn Rootworm, 1962-1981", J. Environ Sci. Health, B18(6), 735-744, 1983.

Beck, E.W., L.H. Dawsey, D.W. Woodham, D.B. Leuck, and L.W. Morgan, "Insecticide Residues on Peanuts Grown in Soil Treated with Granular Aldrin and Heptachlor", J. Econ. Ent., 55(6), 953-956, 1962.

Bess, H.A. and J.W. Hylin, "Persistence of Termiticides in Hawaiian Soils", J. Econ. Entomol., 63(2), 633-638, 1970.

Beyer, W.N. and C.D. Gish, "Persistence in Earthworms and Potential Hazards to Birds of Soil Applied DDT, Dieldrin and Heptachlor", J. Appl. Ecol., 17, 295-307, 1980.

Bruce, W.N. and G.C. Decker, "Insecticide Residues in Soybeans Grown in Soil Containing Various Concentrations of Aldrin, Dieldrin, Heptachlor, and Heptachlor Epoxide", J. Agr. Food Chem., 14(4), 395-398, 1966.

Cliath, M.M. and W.F. Spencer, "Movement and Persistence of Dieldrin and Lindane in Soil as Influenced by Placement and Irrigation", Soil Sci. Soc. Amer. Proc., (35), 791-795, 1971.

Decker, G.C., W.N. Bruce, and J.H. Bigger, "The Accumulation and Dissipation of Residues Resulting from the Use of Aldrin in Soils", J. Econ. Entomol., 58(2), 266-271, 1965.

Elgar, K.E. "Analysis of Crops and Soils for Residues of the Soil Insecticides Aldrin and Telodrin", J. Sci. FD Agric., 17, 541-545, 1966.

Elgar, K.E. "The Dissipation and Accumulation of Aldrin and Dieldrin Residues in Soil", Environ. Qual. Safety, 3, 250-257, 1975.

El Zorgani, G.A. "Persistence of Organochlorine Insecticides in the Field in the Gezira Soil Under Cotton", Bulletin of Environmental Contamination & Toxicology, 15(3), 378-382, 1976.

Freeman, H.P., A.W. Taylor, and W.M. Edwards, "Heptachlor and Dieldrin Disappearance from a Field Soil Measured by Annual Residue Determinations", J. Agric. Food Chem., 23(6), 1101-1105, 1975.

Gilbert, W.S. and C.E. Lewis, "Residues in Soil, Pasture and Grazing Dairy Cattle After the Incorporation of Dieldrin and Heptachlor into Soil before Sowing", Australian Journal of Experimental Agriculture & Animal Husbandry, 22, 106-115, 1982.

Glotfelty, D.E., A.W. Taylor, C.J. Schombert, W.F. Spencer, and W.M. Edwards, "Long-term Volatilization Release of Dieldrin from Ohio Soil", in prep.

Guenzi, W.D., W.E. Beard, and F.G. Viets, "Influence of Soil Treatment on Persistence of Six Chlorinated Hydrocarbon Insecticides in the Field", Soil Sci. Soc. Amer. Proc., 35, 910-913, 1971.

Gupta, H.C.L., and V.S. Kavadia, "Dissipation of Aldrin Residues in Clay Loam Soil Under the Cover of Root Crops", Ind. J. Plant Protection, 7(1), 43-49, 1979.

Harris, C.R., R.A. Chapman, and J.R.W. Miles,"Insecticide Residues in Soils on Fifteen Farms in Southwestern Ontario, 1964-1974", J. Environ. Sci. Health, B12(3), 163-177, 1977.

Jefferys, W.H. and J.O. Berger, "Ockham's Razor and Bayesian Analysis", American Scientist, 80, 64-72, 1992.

Kathpal, T.S.; P.R. Yadav, and K.S. Kushwaha, "Residues of Some Organochlorine Insecticides in Soils Under Different Agro-Climatic Conditions of India", Indian J. Ent., 43(4), 420-427, 1981.

Korschgen, L.J. "Disappearance and Persistence of Aldrin after Five Annual Applications", Journal of Wildlife Management, 35(3), 494-500, 1971.

Kushwaha, K.S., H.C.L. Gupta, and V.S. Kavadia, "Effect of Temperature on the Degradation of Aldrin Residues in Sandy Loam Soil", Annuals of Arid Zone, 17(2), 200-206, 1978.

Kushwaha, K.S., H.C.L. Gupta, V.S. Kavadia, and B.P. Srivastava, "Influence of Different Factors on the Persistence and Degradation of Aldrin Residues in Soils", Annuals of Arid Zone, 18(4), 225-231, 1979.

Kushwaha, K.S., and H.C.L. Gupta, "Effect of Autoclaving, Moisture, and Rate of Application on the Degradation and Conversion of Aldrin into Dieldrin in Soil", Indian J. Ent., 42(2), 166-169, 1980.

Kushwaha, K.S., H.C.L. Gupta, and V.S. Kavadia, "Dissipation of Aldrin Residues from Soil Under Maize/Bajra-Fallow Rotation", Ind. J. Entomol., 43(1), 76-79, 1981.

Lichtenstein, E.P., and K.R. Schulz, "Breakdown of Lindane and Aldrin in Soils", J. of Econ. Entomol., 53(i), 118-124, 1960.

Lichtenstein, E.P., and K.R. Schulz, "Residues of Aldrin and Heptachlor in Soils and Their Translocation into Various Crops", J. Agric. Food Chem., 13, 57-63, 1965.

Lichtenstein, E.P., K.R. Schulz, T.W. Fuhremann, and T.T. Liang, "Degradation of Aldrin and Heptachlor in Field Soils During a Ten-Year Period: Translocation into Crops", J. Agric. Food Chem., 18(1), 100-106, 1970.

Miles, J.R.W., C.R. Harris, and P. Moy, "Insecticide Residues in Organic Soil of the Holland Marsh, Ontario, Canada, 1972-75", J. Econ. Entomol., 71(1), 97-101, 1978.

Nash, R.G., and E.A. Woolson, "Persistence of Chlorinated Hydrocarbon Insecticides in Soils", Science, 157(25), 924-927, 1967.

Nash, R.G., and W.G. Harris, "Chlorinated Hydrocarbon Insecticide Residues in Crops and Soil", J. Environ. Quality, (2)2, 269-273, 1973.

Onsager, J.A., H.W. Rusk, and L.I. Butler, "Residues of Aldrin, Dieldrin, Chlordane, and DDT in Soil and Sugarbeets", J. Econ. Entomol., 63(4), 1143-1146, 1970.

Salanitro, J.P., "Summary of Dieldrin Degradation in Cultures and Soil: Contract and Shell Studies", Shell Development Company, Westhollow Research Center, Technical Progress Report RMA 2-92, 1992.

Scheunert, I., J. Kohli, R. Kaul, and W. Klein, "Fate of [^{14}C] Aldrin in Crop Rotation under Outdoor Conditions", Ecotox. Environ. Safety, 1, 365-385, 1977.

Scheunert, I. "Fate and Effects of Aldrin/Dieldrin in Terrestrial Ecosystems in Hot Climates", Ecotoxicology and Climate, Bordeau, P., Hines, J.A., Klein, W. and Krishna Murti, C.R. eds., John Wiley & Sons Ltd., 299-316, 1989.

Singh, G., T.S. Kathpal, K.S. Kushwaha, and G.S. Yadav, "Persistence of Aldrin in Flooded Soil Under the Cover of Rice", Ecotoxicol. Environ. Safety, 9, 294-299, 1985.

Singh, G., T.S. Kathpal, W.F. Spencer, J.S. Dhankar, "Dissipation of Some Organochlorine Insecticides in Cropped and Uncropped Soil", Environmental Pollution, 70, 219-239, 1991.

Stewart, D.K.R., and C.J.S. Fox, "Persistence of Organochlorine Insecticides and Their Metabolites in Nova Scotian Soils", J. Econ. Entomol., 64(2), 367-371, 1971.

Talekar, N.S., L.T. Sun, E.M. Lee, and J.S. Chen, "Persistence of Some Insecticides in Subtropical Soil", J. Agric. Food Chem., 25(2), 348-352, 1977.

Talekar, N.S., J.S. Chen, and H.T. Kao, "Long-Term Persistence of Selected Insecticides in Subtropical Soil: Their Absorption by Crop Plants", J. Econ. Ent., 76(2), 207-214, 1983.

Wang, Y.S., W.T. Chiu, F.P. Chang, and Y.L. Chen, "Decline of Chlorinated Hydrocarbon Insecticides Residues in the Tea-garden Soils of Taiwan", Proc. Natl. Sci. Counc. B. ROC, 12(1), 9-13, 1988.

Wilkinson, A.T.S., D.G. Finlayson, and H.V. Morley, "Toxic Residues in Soil 9 Years After Treatment With Aldrin and Heptachlor", Science, 143, 681-682, 1964.

Wingo, C.W. "Persistence and Degradation of Dieldrin and Heptachlor in Soil and Effects on Plants", Research Bulletin 914, University of Missouri, College of Agriculture, October 1-27, 1966.

Figure 1
Freeman et al., 1975
Glotfelty et al., in preparation
1966 Study

□ Data
——— Single-phase Model
- - - - Multi-phase Model

Single-phase Model		
	Value	Error
m1	60	5
m2	0.05	0.01
RMSE	0.204	NA
R	0.865	NA

Multi-phase Model		
	Value	Error
m1	36	140
m2	0.14	0.53
m3	25	150
m4	0.01	0.23
RMSE	0.236	NA
R	0.851	NA

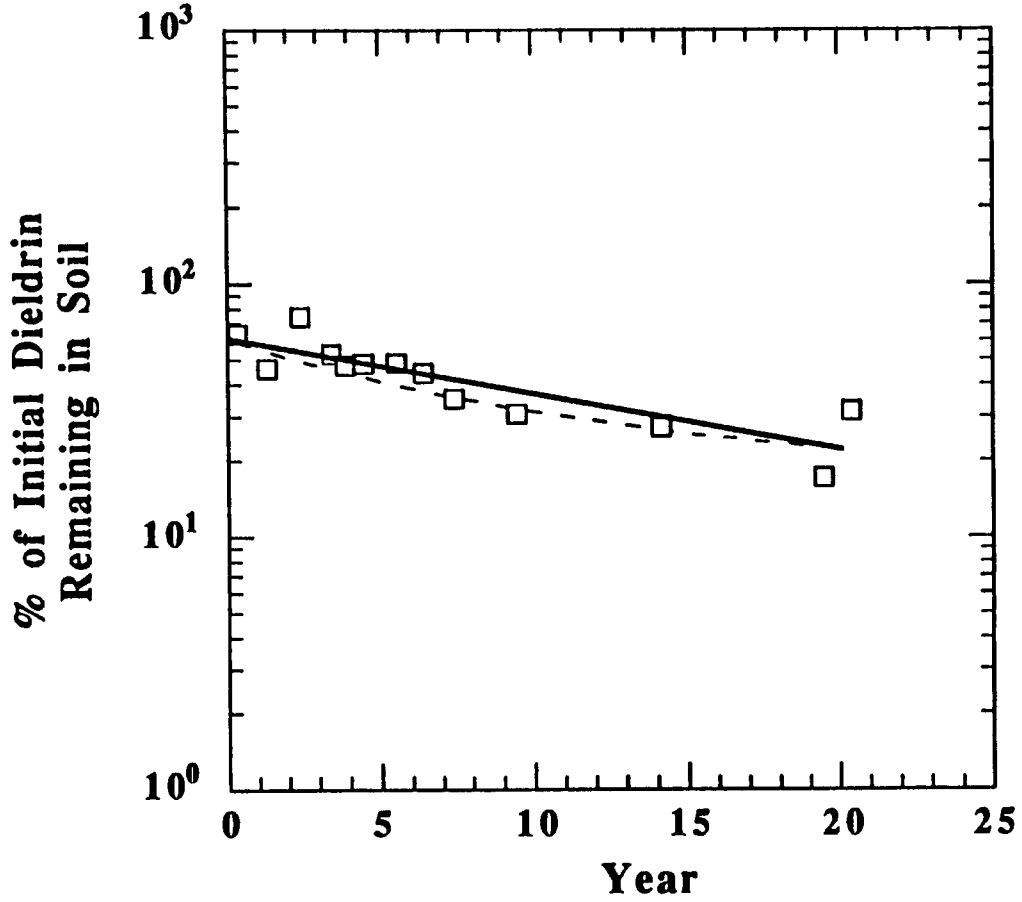
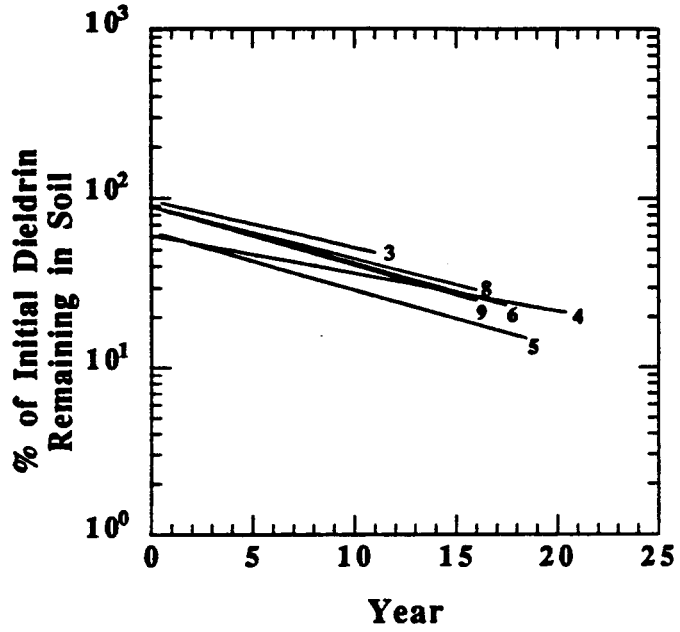
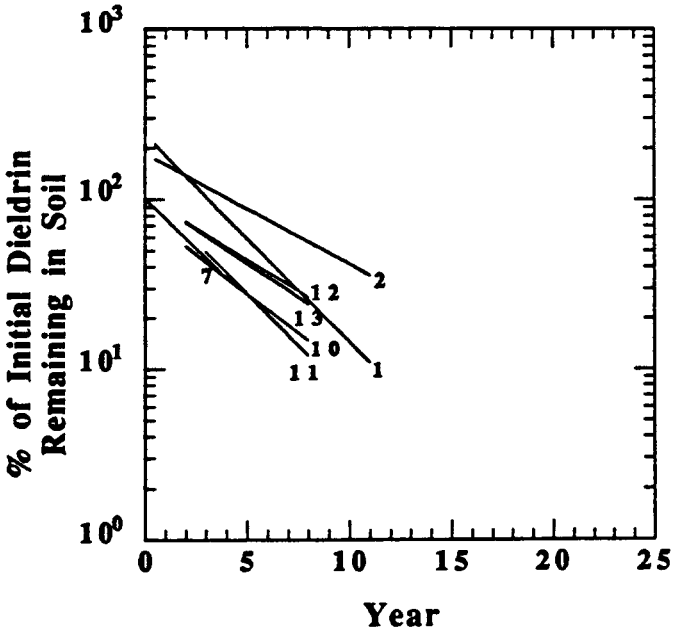


Figure 2
Dieldrin Only Application Studies
in Temperate Climates
(Regressed Data Single-phase Model)



(a)

- Beyer and Gish, 1980
  1 0.6 kg/ha Study
  2 2.2 kg/ha Study
  3 9.0 kg/ha Study
- Freeman et al., 1975;
  Glotfelty et al., in preparation
  4 1966 Study
  5 1968 Study
  6 1969 Study
- Gilbert and Lewis, 1982
  7 Soil Plot #1 Study



(b)

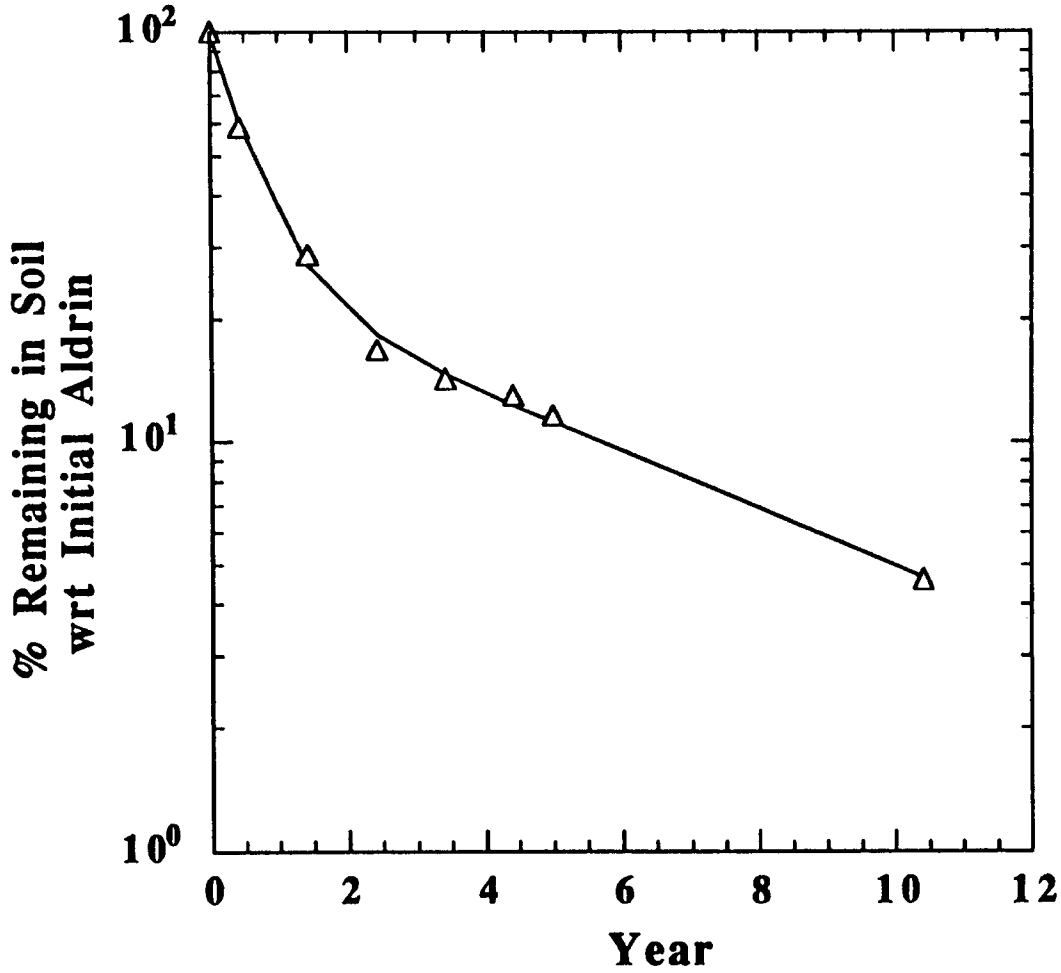
- Nash and Woolson, 1967
  Nash and Harris, 1973
  8 25 ppm Study
  9 100 ppm Study
- Wingo, 1966
  10 10 lb/ac (non cultivated) Study
  11 10 lb/ac (cultivated) Study
  12 100 lb/ac (non cultivated) Study
  13 100 lb/ac (cultivated) Study

Figure 3
Lichtenstein and Schulz, 1960; 1965
Lichtenstein et al., 1970
Single Application Study

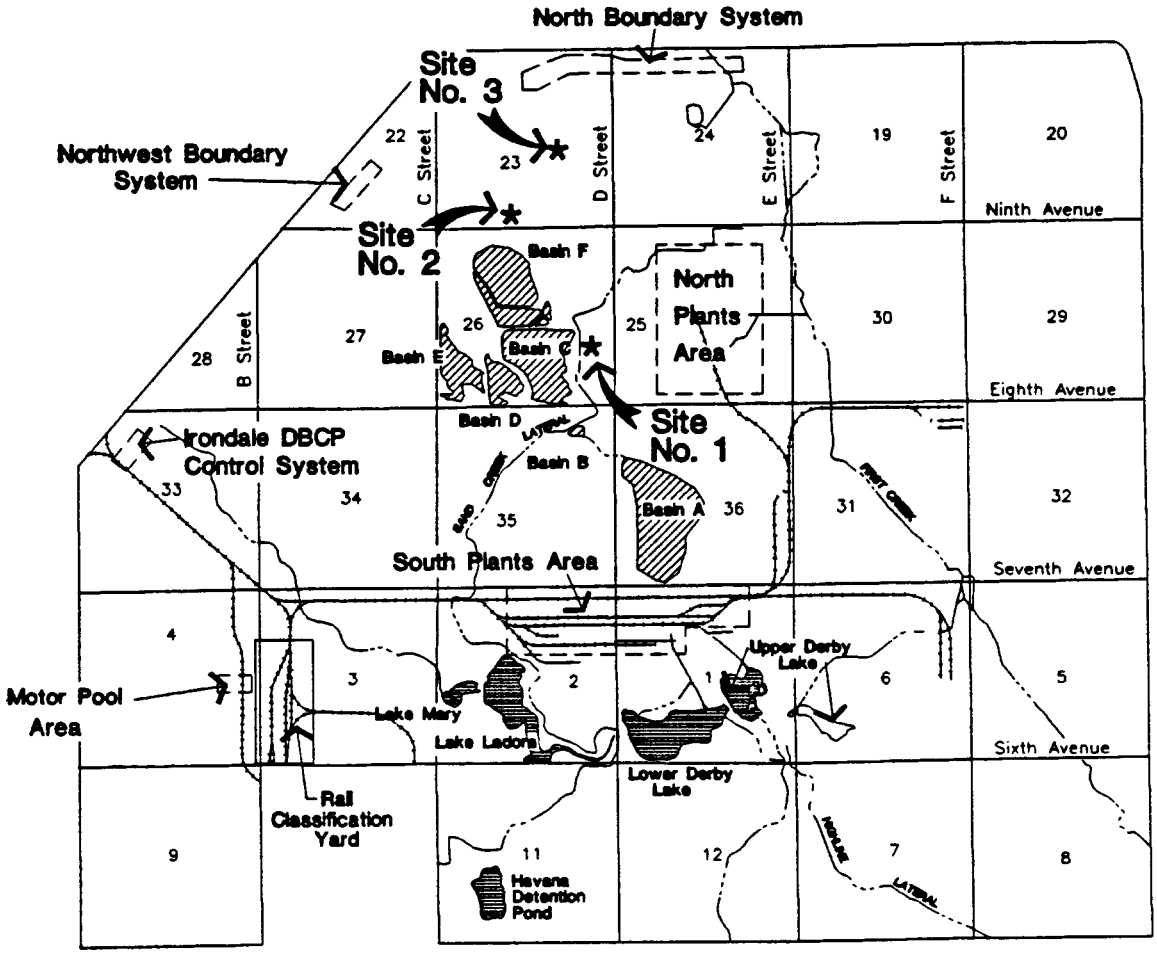
△ **Aldrin + Dieldrin Data**

——— **Aldrin + Dieldrin Model**

Aldin + Dieldrin Model		
	Value	Error
m1	99	6
m2	0.16	0.01
m3	0.38	0.07
m4	1.3	0.14
RMSE	0.065	NA
R	0.999	NA



north



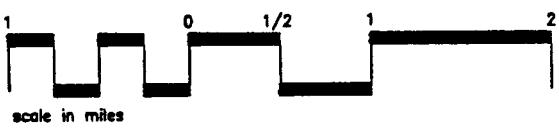


Figure 4

Dieldrin Degradation Soil Sample Locations

APPENDIX F

RESPONSES TO COMMENTS ON THE AUGUST 1993 PROPOSED FINAL IEA/RC

SHELL OIL COMPANY

SHELL'S MAJOR COMMENTS ON THE PROPOSED FINAL IEA/RC ECOLOGICAL RISK CHARACTERIZATION

GENERAL COMMENTS

Comment 1: Presentation of Uncertainty. Appropriate interpretation of ecological risk requires balanced consideration of both the quantitative risk results and their associated uncertainties. This balance is best achieved through careful co-placement of relevant discussions of risk and uncertainty. The proposed final IEA/RC report contains these discussions, but it does not effectively integrate them into the main body of the report. Both the executive summary and Section 4 of the report emphasize the quantitative risk results but barely mention the uncertainty. This disparity is especially troublesome for the aquatic risk characterization where there is a reliance on some very uncertain methodologies. Shell recommends that the final IRA/RC ecological risk characterization provide better integration of summary discussions of risk and uncertainty in the main body of the report.

Response: Shell recommends that the final IEA/RC ecological risk characterization provide better integration of summary discussions of risk and uncertainty in the main body of the report.

Appropriate summary discussions of uncertainty in risk estimates will be incorporated into the main body of the revised draft final IEA/RC report. As in the previous version, information about risk levels will be presented in the form of HQ maps; however the revised report will contain HQ maps calculated for each of the three BMF estimation techniques used in the IEA/RC ecological risk characterization. Presentation of the three sets of maps will provide information about uncertainty in risk estimates. Section 5 will be revised to strengthen the interpretation of qualitative and quantitative uncertainty analysis results.

Comment 2: Reference Source Citations. The Proposed Final IEA/RC ecological risk characterization is a product of numerous innovative ideas for solving difficult technical problems. Sources of technical solutions were varied and included published articles, reports, and scientific panel recommendations. Not all of these sources, however, were properly identified and described within the IEA/RC report. Specifically, the Shell document entitled "Ecological Risk Characterization for the Onpost Operable Unit of RMA" prepared January 1993 by EA Engineering, MKE and R.L. Sielken, Inc. was a source for the spatial exposure component; the March 1993 Society of Toxicology abstract entitled "A Co-located Distributions Approach to Estimating the Bioaccumulation Factor for Ecological Risk Assessment" authored by R.L. Sielken, Jr., C. Valdez, P.A., Clifford, D. Ludwig, and M.I. Banton, was a source of the Shell co-located distribution methodology for calculating field BMF; and the expert panel provided guidance on evaluating field data and uncertainty associated with toxicity benchmarks. Shell recommends that these and all other significant sources/contributors be acknowledged in the Final IEA/RC report.

Response: The Army appreciates the contributions from staff members of the OAS and their respective contractors in the preparation of the IEA/RC. Because of the complex nature of this program and the processes involved (i.e., consensus decisions), the Army feels that it is fair to not acknowledge specific contributions from OAS personnel and contractors unless they are authors of a paper published in a recognized journal or federal document which served as source material for the IEA/RC.

Comment 3: Ecological Risk Exceedance. The expression of complex ecological risk results is a challenge for the risk assessor. Figures can provide a unique perspective on risk; however, it is essential that such presentations be both technically sound and accurate. Shell has concerns about the accuracy of several of the figures presented in the ecological risk sections of the Proposed Final IEA/RC report. Specifically, there are a number of figures that depict the spatial extent and number of trophic box exceedances and serve as basis for risk conclusions, yet are apparently misleading. A major concern is that the number of trophic box exceedances given for a specific RMA area is not entirely accurate. This is because the "generic predator trophic box" is represented on the maps as four different top predator species (eagle, American kestrel, great horned owl and great blue heron), each of which is counted as an individual "trophic box." In addition, there are a number of trophic boxes (including earthworm, insect, herptile and terrestrial plant) for which risk could not be quantified because of a lack of suitable toxicity benchmark values (MATC and TRV). Thus, the total trophic box tally presented in the figures are incomplete for these trophic boxes. Shell recommends that the Army consider revising these figures and associated discussion to more accurately reflect the RMA ecological risk results.

Response: Because the five different top predator food webs were specifically selected to reflect differing exposures to the RMA ecosystem, their treatment as separate trophic boxes is appropriate. However, the Army agrees that clarification is needed in the IEA/RC as to the trophic boxes actually depicted. An insert will be added to the text indicating that risk was evaluated only for specified selected trophic boxes. Further, an introductory page to maps in both the main text and Appendix C.3 will include this and other information to facilitate map interpretation. This map introduction will note that this may result in the simultaneous presence of risk to several top predator trophic boxes in the same areas of RMA.

Comment 4: Treatment of Terrestrial Mercury Risk. It is well known that risk from mercury exposure is highly dependent on the form of this metal in the environment. In the Proposed Final IEA/RC report, risk to the terrestrial RMA ecological receptors presumes that the environmental form of mercury is entirely organic. Although the conservative nature of this assumption is acknowledged qualitatively in the discussions, its relative impact on quantitative risk is not provided. Shell believes the impact of this assumption is likely profound given the low levels of organic mercury generally reported in soils and the historical indication that the likely source of mercury on RMA was inorganic mercury chloride which was used as

a Lewisite catalyst. Therefore, Shell recommends that the Army provide perspective in the Final IEA/RC report on the relative quantitative effect that the assumption of organic mercury contributes to the overall terrestrial ecological risk.

Response: Confusion has arisen over the assumptions used, and their consequences, in assessing risk from mercury contamination. While the toxicity-limiting parameters MATC and TRV are based exclusively on studies using methylmercury, the soil and tissue samples collected from RMA that result in the "observed BMF" are based on the analysis of total mercury. The equations below illustrate the relative contributions (emphasis noted by bold subscript) of inorganic and organic forms of mercury in the computation of the final "model BMF".

$$\frac{TC_{\mathbf{inorganic+organic}}}{SC_{\mathbf{inorganic+organic}}} = observed\ BMF_{\mathbf{organic/inorganic}} \qquad (1)$$

$$\ldots\ BAF_{\mathbf{organic}} * FR \ldots = litmodel\ BMF_{\mathbf{organic}} \qquad (2)$$

Calibration of the BMFs from equations (1) and (2) results in the final model BMF that includes contributions from organic and inorganic forms of mercury.

It is acknowledged that the original mercury contaminant was mercuric chloride and that literature sources dictate that inorganic mercury should be the predominant form of mercury in the soil. Literature sources also indicate that most of the mercury detected in animal tissue is in organic form, usually methylmercury. Inorganic forms of mercury *are* bioavailable and *are* toxic to animals, but less so than organic forms of mercury. In addition, inorganic mercury in soil can be transformed (i.e., methylated) to organic mercury through biotic and abiotic mechanisms. Therefore, the use of methylmercury as the basis for the MATC and TRV parameters is a reasonable approach in the estimation of risk from mercury contamination. The resultant protective criterion, whether tissue-based or dose-based, is computed from a toxicity parameter for organic mercury divided by a BMF for both forms of mercury *that has been calibrated to the site conditions*. Additional text will be included in the IEA/RC addressing the basis for the likely overestimation of mercury risks.

Comment 5: Additivity of Chemicals. In general, exposure to multiple chemicals can only result in interactive effects in biological systems, if there are concurrent dispositional, functional or anatomical interactions among these chemicals. The characterization of total risk to the RMA ecological receptors in the Proposed Final IEA/RC, however, assumes additive interaction effects for all the RMA COCs and intermediate chemicals, regardless of the realistic likelihood that these chemical interactions may take place. Shell believes this approach is overly conservative, scientifically indefensible and thus inappropriate for ecological risk characterization at RMA. Shell recommends that an assumption of additivity be

restricted in the Final IEA/RC report only to these chemicals that are likely to actually interact within biological organisms.

Response: The Army acknowledges that different chemicals have different modes of action and that the addition of all chemicals, regardless of their mode of action, to calculate risk is a conservative approach. However, the IEA/RC notes the various possible types of interactions among the ecological COCs, and the conservatism introduced through the addition of all COCs considered, regardless of their interactive mechanisms. The Army believes this approach is appropriate because while chemical interactions may be additive, independent, synergistic or antagonistic, these interactions are not well documented. Further, the additive approach used by the Army is consistent with EPA risk assessment guidance for human health (EPA 1989a) and with the approach being used in the HHRC portion of the IEA/RC.

Comment 6: Treatment of the Shell Co-Located BMF. Several misleading and/or incorrect statements have been made in the document regarding the Shell co-located distributions approach (the "Shell method"). With respect to how the Shell method deals with the correlation between TC and ESC, statements made on pages E-81 and 82 are wrong and misleading. The Shell method and corresponding statistical equations do not require that the correlation between TC and ESC be estimated at all. The Shell method also makes no other assumptions about correlation and does not require, utilize or estimate any other correlation values. An advantage of this approach is that the estimates of BMF made by the Shell method are consistent with the way in which BMFs are utilized.

In discussion on pages E-83 and 84, it is not clear that the Shell method of parameter estimation focuses on the primary target, the mean BMF, and is designed to provide the best available estimates of this parameter. Finally, presentation of the three BMF_{obs} calculation approaches (page C.1-26, third paragraph) does not indicate that these methods characterize uncertainty differently. The Shell method includes the variability in obtaining the sample of TC and ESC values and their corresponding sample means and standard deviations. The Army method does not. The EPA method characterizes variability of a hypothetical statistical construction of a BMF rather than an actual BMF.

Response: It is impossible to take the ratio of two probability density functions without making some sort of implicit or explicit assumption about the degree of correlation between those distributions. The correlation assumption is implicit in the Shell method. The Army believes its description of the treatment of correlation in the Shell method is accurate, but is prepared to revise its statements if Shell provides information explaining the error in the Army's description.

In discussion on pages E-83 and E-84, it is not clear that the Shell method of parameter estimation focuses on the primary target, the mean BMF, and is designed to provide the best available estimates of this parameter.

The three BMF methods are all designed to provide the best available estimate of the mean BMF from the currently available tissue and soil concentration database.

Presentation of the three available BMF_{obs} calculation approaches (page C.1-26, third paragraph) does not indicate that these methods characterize uncertainty differently. The Shell method includes the variability in obtaining the sample of TC and ESC values and their corresponding sample means and standard deviations. The Army method does not. The EPA method characterizes variability of a hypothetical statistical construct of a BMF rather than an actual BMF.

The Shell method recognizes that pairs of values (TC, ESC) obtained during the sampling and ESC estimation procedure are only one random realization of the underlying collocated distribution. Shell uses bootstrap resampling (with an assumed correlation of TC and ESC) to estimate the variability of TC and ESC distributions that would result from different possible (TC, ESC) sampling realizations. This variability provides a measure of the uncertainty in BMF. However, the objective of the analysis to estimate BMF and its uncertainty, not to estimate variability in (TC, ESC) samples. There are ways to satisfy the objective that do not explicitly estimate the variability in (TC, ESC) samples. The Army method, for example, estimates uncertainty in BMF by treating the correlation between the TC and ESC distributions estimated from the observed sampling realization as uncertain. Uncertainty in the BMF calculated by the Army method depends on the uncertainty in the correlation of TC and ESC, as well as on the TC and ESC distributions estimated from the observed sampling realization. The EPA method utilizes a third statistical approach to estimate uncertainty in BMF. It assumes that paired (TC, ESC) samples represent an independent, random sample of BMF. BMF uncertainty is estimated based on the BMF sample variance and (TC, ESC) sample correlation.

Comment 7: BMF Performance Criteria. The proposed maps are expected to contain lots of information including spatial relationships; however, that information may be too easily dismissed if it is not readily comparable for different BMF estimators. Quantitative summaries of the information in the maps should help convey the implications about alternative estimators of the mean BMF that should follow from a careful study of the maps. Quantitative evaluations should make the implications of the maps more concrete and make the maps harder to dismiss or misuse. Quantitative characterizations of the maps may reduce the danger of purely subjective characterizations or misleading conclusions based on only very small subsets of the available information.

Response:

The Army is working within the constraints of Council dispute resolution guidance to report the three sets of BMF results with impartiality. Tissue concentration prediction maps will not be included in the revised draft final IEA/RC, because they do provide information about the relative performance of BMFs calculated by the three alternative estimation methods as predictors of tissue concentration field data on the Arsenal. The Parties to the BMF dispute disagree about

the relevance of a BMF performance evaluation (based on current conditions) to possible post-remediation conditions. A supplemental field study will seek to improve confidence in risk estimates and, if necessary, update BMF estimates. A number of dispute issues pertaining to the reliability of risk estimates remain unresolved.

Comment 8: Characterization of the Boring-by-Boring Risk Analysis. Shell continues to be concerned about the emphasis placed on the results of the boring-by-boring analysis in the IEA/RC. In the Human Health IEA/RC Dispute Resolution Agreement, it was agreed that the boring-by-boring analysis would be characterized as a "worst-case chronic exposure scenario and a boundary risk estimate rather than a high-end, plausible risk estimate (i.e., RME) required for Superfund risk assessment." When the boring-by-boring evaluation is discussed in the third paragraph of page 5 of the Executive Summary, there is no discussion of the evaluation as a "worst-case" scenario, but instead isolated exceedances of a 10-4E cancer risk are represented as a single boring—a physical impossibility for a 30-year chronic human exposure. Even more detailed discussions on page 3-28 fail to characterize the interpretation as "worst-case or boundary" risk estimate. While the current Gray Cover IEA/RC presents an improved discussion of the uncertainties and limitations of the boring-by-boring risk assessment, the overall tone and language of the document continues to place inappropriate emphasis on these results. The use of such an analysis for risk assessment is technically incorrect and is inconsistent with Superfund guidance, prior dispute resolution agreements made by the parties and representations made by the Army in the Brown Cover IEA/RC and Comments. Shell believes that the current representation of the boring-by-boring risk assessment presents an unsupportable exposure case that is an unsatisfactory presentation of a dispute resolution issue.

In addition, Shell's review of the results of the Gray Cover IEA/RC and the Draft DAA, indicates that the EA and FS groups may not be using the same soil contaminant database in their respective calculations. Since information developed in the IEA/RC is of critical importance for guiding the designation of areas for response actions in the FS, it is imperative that the same databases are used by both groups.

Response: The conservatism of the boring-by-boring analysis from a risk/exposure standpoint is acknowledged repeatedly in the IEA/RC. Despite this conservatism, this type of evaluation is useful in that, unlike the site risk analysis, spatial information is retained (rather than being "buried" in a single estimator). Site-specific risk calculations basically aggregate contaminant concentrations into individual upper bound estimates of site concentrations and often assume spatial covariance of elevated levels of all contaminants. While this situation could well occur in simple situations (e.g., at sites with a single source of multiple contaminants), it is rarely observed at complex sites containing multiple sources such as RMA. Additionally, as acknowledged previously by Shell, sites as currently defined may not represent appropriate averaging zones for future exposures. Given the lack of information regarding the distribution

of specific future land uses at RMA, the bore-by-bore analysis is ultimately required, particularly for evaluating results on a contaminant-specific basis.

Conceptually, the ultimate objective of risk characterization is to integrate site characterization data into a decision making tool which can both identify the need for remedial action(s) and be used in the evaluation of alternative remedies. Given the limitations of the site risk analysis stated above, this objective can only be achieved by evaluating HHRC results on both a site-specific and a boring-by-boring basis.

The March 1994 (Gray II) HHRC results were calculated using data pulled from the soil contaminant database on December 30, 1993 (ALLCSO6.DBF).

APPENDIX F

RESPONSES TO COMMENTS ON THE MARCH 1994 PROPOSED FINAL IEA/RC

SHELL OIL COMPANY

Shell Oil Company
Comments on the Proposed Final IEA/RC
Version 3.1

GENERAL COMMENTS

Human Health Issues:

Characterization of Receptor Populations:

Comment 1: Page 3-1, second paragraph. This section focuses on the maximally exposed receptor population (commercial and industrial workers) or subpopulation (biological worker subpopulation of the refuge worker, local neighborhood subpopulation of the regulated/casual visitors, and local neighborhood subpopulation of the recreational visitors). Therefore, this section should emphasize that the quantitative cancer risks or hazard indices for maximally exposed subpopulations provided do not accurately characterize the cancer risks and hazard indices for the entire populations. The results for maximally exposed subpopulations at best provide upper bounds for the entire populations.

Response: The referenced paragraph clearly states that the risk assessment focuses on the maximally exposed receptor populations or subpopulations, and that estimated risks for such populations/subpopulations would be highest for a given land-use scenario.

Comment 2: The number of individuals in each subpopulation is not characterized nor is the size of the subpopulation relative to the whole population characterized.

Response: The number of individuals in each subpopulation can not be quantified given that the subpopulations evaluated in the IEA/RC are hypothetical, and based on projections regarding future access and use of the Arsenal under a future-use wildlife refuge/recreational park scenario. Furthermore, the size of a population is not required to estimate risks; rather, this information is used in estimating the cancer burden.

Comment 3: The last sentence in this paragraph should be modified to replace the words "all five receptor populations" by "all five maximally exposed receptor populations/subpopulations". This would help avoid the mistaken impression that Appendix B provides any quantitative results for the entire

populations of refuge workers, regulated/casual visitors, or recreational visitors. Of course, Appendix B should have contained those quantitative results. The absence of the population results should be explained.

Response: The last sentence in the second paragraph on page 3-1 will be revised as requested by Shell. However, the characterization of the populations and subpopulations provided in Appendix B is considered to sufficiently document the assumptions and approach used in the HHRC, and thus will not be revised.

Comment 4: The assumptions used in deriving the biological worker scenario present a physical impossibility: An individual working out of doors 253 days per year with 3,200 square centimeters of skin exposed. The text should be revised to indicate that the biological worker scenario is ultra-conservative and unique to RMA.

Response: The existing text and characterization is considered appropriate, and thus will not be revised. The 3,200 cm^2 value assumed to represent the amount of skin exposed for the biological worker is a time-weighted average that takes into account the varying amount of skin exposed throughout the year by using 50th percentile male and female skin surface area estimates. This value was derived assuming that the biological worker's head, neck, hand, forearms, and a portion of the upper arms are exposed in the summer, spring, and fall. However, only the head and neck are assumed to be exposed in the winter months. Additionally, for an individual who works outdoors, exposure 253 days/year is considered reasonable as an upperbound estimate.

Comment 5: The upper bound nature of the risk characterizations based on maximally exposed subpopulations should be a part of Section 3 and be presented before and during any discussion of the magnitude of these risks.

Response: The existing discussion of maximally exposed populations and the associated conservatism in the analysis provided in the HHRC is considered sufficient, and thus will not be revised.

TDV Distributions:

Comment 6: Attachment B.3-4, page Att.B.3-4-1, first two paragraphs. These two paragraphs make reference to Table 1. There are two errors associated with Table 1 and the two corresponding paragraphs. First, the initial

paragraph labels the distributions as Shell's distributions when the distributions for the "Subpopulation of Most Concern" are really the Army's distributions not Shell's distributions. Second, the distributions and figures labelled "Subpopulation of Most Concern" should be updated to match the Army's current distributions for these subpopulations.

As noted, the frequency distributions of lifetime duration of exposure (hours/lifetime) in Table 1 for Regulated/Casual Visitors: Subpopulation of Most Concern and Recreational Visitors: Subpopulation of Most Concern are not Shell's distributions at all. They are EBASCO's neighborhood distributions using the EBASCO distributions for TM, DW, and TE from May 6, 1993. Sielken, Inc. had calculated these distributions using EBASCO parameter distributions, so that the lifetime duration distributions corresponding to EBASCO's more restrictive definition of the "neighborhood subpopulation" (i.e., the distributions labelled "Subpopulation of Most Concern") could be compared with Shell's lifetime duration distributions for the more general neighborhood (i.e., the distributions labelled "General Neighborhood Population"). The idea of comparing these two subpopulations (i.e., EBASCO's "Subpopulation of Most Concern" and Shell's "General Neighborhood Population") is fine; however, to avoid misleading the reader the distributions should be identified as the Army's distribution and Shell's distribution, rather than both being labelled as Shell's distributions.

Response: The cited paragraphs and the corresponding table will be revised to eliminate any references to Shell's distributions when referring to the "Subpopulation of Most Concern."

Comment 7: The numbers in Table 1 and Figures 1 and 2 for the distributions for "Subpopulation of Most Concern" should be updated to correspond to the Army's final distributions for TM, DW, and TE. These would correspond to the numbers in Table B.3-31 except for the fact that the numbers in that table are also in error.

Response: The values in Table 1 and Figures 1 and 2 will be revised to correspond with those listed in Table B.3-31. Table B.3-31 will be revised to eliminate incorrect values as indicated below (in the responses to Comments 8 and 9).

Comment 8: Table B.3-31. The numerical values for the percentiles in the distributions for TM*DW*TE (hours/lifetime) for Neighborhood Regulated/Casual Visitor and Neighborhood Recreational Visitor do not match the lognormal

distributions for TM*DW*TE corresponding to the parameter values given in Table B.3-30. The errors do not appear to be simply round-off errors but large errors, particularly for the percentiles less than the 50th percentile. Also, the percentiles are not self-consistent. For example, for the Neighborhood Recreational Visitors, if the lognormal distribution had a 95th percentile equal to 4646.31 as shown in Table B.3-31 and a 50th percentile equal to 516.28 as shown, then the parameters on the logarithmic scale would be 6.246649 for the mean and 1.335671 for the standard deviation. These parameters would imply that the 20th percentile was 167.76 and not the tabled value of 207.25. The source of the errors in Table B.3-31 is not known. The numbers can be calculated exactly (without simulation) from the parameter values and should be consistent even if there is some rounding error in the parameter values listed in Table B.3-30.

The distributions of TM*DW*TE corresponding to the parameters in Table B.3-30 all have 100th percentile equal to plus infinity, so the 100th percentile entries in Table B.3-31 must refer to something other than the 100th percentiles.

Response: Table B.3-31 will be revised to correct the discrepancies noted in this comment. The "100th percentile" row will be replaced with a 99th percentile row. The TM*DW*TE percentile values currently shown in Table B.3-31, which do not accurately reflect the use of these distributions in the HHRC computer code, will be replaced.

TM*DW*TE percentiles for the Neighborhood Regulated/Casual Visitor and Neighborhood Recreational Visitor populations will be calculated directly from the lognormal distributions that result from multiplying the lognormal distributions for TM, DW and TE. Parameters for these lognormal TM*DW*TE distributions will be calculated using equations 3.3.40 and 3.3.41 of Benjamin and Cornell (1970). Equation 13.24 and Table A.1 of Gilbert (1987) will be used in estimating the percentiles.

These corrections to Table B.3-31 do not require changes to the HHRC code, nor to the results reported elsewhere in the IEA/RC. This table is provided solely for the reader's reference, and is not used as the basis for any quantitative analyses. The incorrect values noted by Shell and discussed above resulted from an incomplete simulation of the TM*DW*TE distribution, which was performed outside the HHRC code.

Comment 9: Table B.3-31. The numerical values for the percentiles in the distribution for TM*DW*TE (hours/lifetime) for Biological/Maintenance Worker do

not come close to matching 10,000 simulations of the distribution for TM*DW*TE corresponding to the parameter values given in Table B.3-30 and the added restriction that simulated values of TE less than 1.29 be replaced by 1.29. Starting at the 90th percentile, the lower the percentile the greater the discrepancy.

Response: Table B.3-31 will be revised to correct the discrepancies noted in this comment. The TM*DW*TE percentile values currently shown in Table B.3-31, which do not accurately reflect the use of these distributions in the HHRC computer code, will be replaced.

TM*DW*TE distribution percentiles for the Commercial/Industrial Worker and Biological/Maintenance Worker populations will be obtained using the input-parameter distribution files created for the HHRC sensitivity analysis reported in Section B.5. This approach is necessary because TM*DW*TE distributions for these populations cannot be calculated readily using analytical methods. [Rather, they required multiplication by a normal distribution.] As noted in the response to Comment 3, these corrections to Table B.3-31 do not require changes to the HHRC computer code, nor to the results reported elsewhere in the IEA/RC.

Comment 10: Page B.3-135. The derivation of DW (days/year) for the maximally exposed subpopulation of Regulated/Casual Visitors has changed. The previous draft report (Brown Cover, September, 1992) assumed, on the basis of professional judgement with no supporting data, that the number of activity days per year (at all locations, not just at RMA) varied between 5 and 52 and has a triangular distribution with min=5, mode=26, and max=52. Both the previous draft (Version 3.0, August 1993) and the current draft report (Version 3.1, March, 1994) assume, again on the basis of professional judgement with no supporting data, that the number of activity days varies between 5 and 104 and has a triangular distribution with min=5, mode=26, and max=104. This is a considerable change. Presumably, the idea was to keep the min=5 and the most likely value (the mode) at 26 but extend the tail of the original distribution to include larger values (up to 104). If this were in fact appropriate, then it should have been done by switching from a triangular distribution (min=5, mode=26, max=52) to a lognormal distribution (min=5, mode=26, and 99th percentile = 104) rather than arbitrarily staying with a triangular distribution. By staying with a triangular distribution rather than shifting to a lognormal distribution, the change not only increased the tail of the distribution (i.e., the frequency of values between 52 and 104 days) but also made a big change in the central portion of the distribution. The median (50th percentile) changed from 27.3 to 41.9 which is more than a 50% increase.

Also, the mean changed from 27.67 to 45 which is more than a 60% increase.

Response: The analysis on page B.3-135 clearly acknowledges the lack of data and makes a few very simple, straightforward assumptions in estimating the number of activity days per year for the neighborhood population. Shell's proposed alternatives would add an unnecessary and easily misunderstood level of complexity to the analysis. Furthermore, incorporation of Shell's proposed alternatives would imply a level of knowledge regarding activity days for this receptor population that simply does not exist.

The analysis in the Brown Cover report assumed a triangular distribution with min = 5, mode = 26, and max = 52. This approach assumes the following:

(1) In the absence of relevant data, it is most reasonable to use a simple, straightforward distribution shape such as the triangular.

(2) Each individual in the neighborhood has at least 5 activity days per year.

(3) The most likely number of activity days per year in the neighborhood is 26, corresponding to 1 activity day every other week.

(4) The largest number of activity days per year is 52, corresponding to 1 activity day per week.

The only change that was made for the Gray Cover report was to double the assumed maximum number of activity days per year—from 52 to 104. The Army considers this revision to be valid and appropriate. Basically, it assumes that the member of the neighborhood population with the maximum number of activity days has two activity days per week instead of one. The rationale supporting this change was to retain the original approach used in defining the distribution (given the lack of data), but to allow for the possibility of more than one activity day per week (a reasonable assumption). The increase in the mean and the median values was expected. However, contrary to the implication in Shell's comment, the previous median of 27.13 activity days per year was not necessarily data-driven; rather, it was simply the result of the original assumptions used in the analysis.

Changing to a lognormal distribution, truncated or not, would be no less arbitrary than retaining the current assumption of a triangular distribution for activity days per year; it would not enhance the credibility of the model. The Army is not aware of any rationale based on recreational activity data that would support use of the lognormal distribution over use of the triangular distribution for this parameter, and Shell's comment did not provide such a rationale.

As Shell correctly noted in this comment, the distribution for the number of activity days is multiplied by the fraction of these days spent at RMA to obtain the DW distribution. In the analysis reported on page B.3-135, samples from the triangular distribution for the number activity days are multiplied by samples from a uniform distribution for the fraction of these days spent at RMA. A simple, untruncated, two-parameter lognormal distribution for DW is then fitted to the resulting values. Given the lack of relevant data, and the factors discussed above, revision of this analysis is not warranted. Consequently, the existing DW distribution will be retained in the IEA/RC.

Comment 11: If the objective of increasing the frequency in the upper tail of the distribution were appropriate, then lognormal distributions could have been used which would have preserved the original professional judgements of a minimum value near 5 and a most likely value (mode) near 26. In particular, a lognormal distribution with location parameter = 5, mean (ln scale) = 3.0445 and standard deviation (ln scale) = 0.6665 has min=5, mode=26, and 99th percentile = 104. Similarly, a lognormal distribution with location parameter = 0, mean (ln scale) = 3.2581 and standard deviation (ln scale) = 0.59591 has 0.28th percentile = 5, mode=26, and 99th percentile = 104. If the objective of the change were appropriate, then either of these latter two lognormal distributions would have been preferable. The latter two lognormal distributions would have both had medians=26. The latter two lognormal distributions would have had means equal to 26.22 and 32.58 respectively instead of the mean of 45 in the draft's triangular distribution for days.

Of course, the lognormal alternatives for the number of activity days (at all locations, not just RMA) would still need to be multiplied by the fraction of these days spent at RMA in order to get the DW distribution.

Response: See response to Comment 10 (above).

Characterization of Upper Bound Risks:

Comment 12: Executive Summary ES-2, first paragraph in Section 2. The human cancer risks are characterized using upper bounds on the chemical's cancer potency rather than best estimates of the cancer potency. Hence, the best estimates of human cancer risks are not being presented but rather quantified using upper bounds. The third sentence should acknowledge the upper bound nature of the human cancer risk characterization and the text "The cancer risks are expressed as a probability (e.g., 1 in 10,000) and represent excess lifetime cancer risks" should be changed to "The cancer risks are expressed as an upper bound on the cancer probability (e.g., 1 in 10,000) and represent an upper bound on the excess lifetime cancer risks".

Response: While the Army agrees that the toxicity criteria are likely a source of overestimation of risks, it is inappropriate to single out one parameter from the risk equation (i.e., toxicity criteria) in the Executive Summary and use it as the basis for a discussion of the potential for the risks to be overestimated. However, use of upperbound slope factors and other PPLV equation parameters and their potential impacts on the risk estimates is discussed in Appendix E.

Comment 13: The human risks are being characterized for the most exposed subpopulation rather than the whole population. Thus, human risks are being characterized using upper bounds on the exposure to the chemical rather than more complete distributional characterizations of the exposure in the entire exposed population. Hence, the best estimates of human risks are not being presented but rather quantified using upper bounds. Thus, after the third sentence "The cancer risks ..." and the fourth sentence "Noncarcinogenic risks are " there should be a new sentence inserted along the lines of "The human risks are being characterized for the most exposed subpopulation rather than the whole population." The words "the most exposed subpopulation" could even be more accurately described by something like "a hypothetical most exposed subpopulation".

The upper bound nature of the risk characterizations should be a part of the executive summary and be presented before and during any discussion of the magnitude of these risks.

Response: In response to this comment, the following sentence will be inserted after the third sentence in the cited paragraph (page ES-2, para. 1): "To ensure that risks would not be underestimated, risks were characterized for a subpopulation of visitors and wildlife refuge workers (i.e., biological workers) considered to have a high potential for exposure to the

contaminants." This language deviates slightly from that proposed by Shell, but is considered more accurate.

Uncertainty Discussion:

Comment 14: The uncertainty discussion in Appendix E on aldrin and dieldrin is speculative and unbalanced, containing errors of interpretation and fact. Shell understood that the issue of presenting a balanced summary of the toxicology of aldrin and dieldrin was resolved by the agreement to include the Shell prepared Toxicity Profiles in the HHEA. As then, Shell believes it is vital that any discussion should be as complete and accurate as possible. The discussion on aldrin and dieldrin is unfortunately neither, and in fact conflicts with the Army's own toxicity profile. Some of the principal concerns are discussed below.

The thyroid and adrenal tumors in the rat study occurred at the low dose and were considered by the authors to be unrelated to treatment. There is only evidence for a mouse liver response for both aldrin and dieldrin.

While the hamster, dog and monkey studies may have been inadequate for cancer bioassays, the pathology information should be viewed as potentially highly relevant to the mouse liver cancer response. The effects on mouse liver are immediate and very pronounced whereas monkeys fed at maximum non-lethal doses for 6 years showed no evidence of liver damage.

The discussion on page E-24 (2nd complete paragraph) is speculative and unfounded. The possibility of a promoting effect on people with risk factors is hypothesized but no studies are cited in which such interactions were studied. For instance, an NCI-SRI study on the interaction between aflatoxin and dieldrin in rats concluded that dieldrin neither increased liver tumors nor increased the incidence of aflatoxin-induced tumors. If there is in fact evidence for interactions, it should be cited and Shell should be given the opportunity to review and comment on it.

The Army concluded correctly in the Toxicity Profile that neither aldrin nor dieldrin appeared to be mutagenic. However, in the discussion on page E-24 the opposite is implied. The validity of each positive study has been questioned on grounds of inadequate experimental design, technical problems and particularly, cytotoxic dose levels. Many chemicals in sufficiently high dosage cause chromosomal damage. All in vivo studies have been negative and the World Health Organization (WHO 1989)

concluded that "numerous in vitro and in vivo mutagenic studies have demonstrated that neither aldrin nor dieldrin have mutagenic potential". Other negative data is not discussed. For example, the lack of strand breaks in mice or chromosome studies on Pernis workers.

The discussion on epidemiology data is incomplete and misleading. For example, the Pernis liver tumor case occurred in a worker who had been at sea for 40 years before starting work at Pernis and was a chronic alcoholic (de Jong 1991: p. 137). It is difficult to see how a study can be rejected on the grounds of inadequate exposure quantification when we have the unique situation of longitudinal data on actual blood levels (an accurate indicator of exposure) from 1964 to the present day. It is also of note that the cohort did not exhibit any unexpected health problems or rate of absenteeism.

Sielken and Stevenson also looked at mortality in addition to total tumors (Sielken and Stevenson 1993). This was also negatively correlated with dose.

Response:

Response to Paragraph 2: To clarify the authors' and EPA's evaluations of this study the following insert will be added to the second paragraph on page E-23 after the sentence "Only one of seven rat studies was considered adequate by EPA, and it showed an increased incidence of thyroid and adrenal tumors":

> The incidence of thyroid and adrenal tumor development in various dosing groups were not consistently significant when compared with matched controls, therefore the authors concluded that the tumors were not associated with treatment. The EPA review of the data concluded that the results were "equivocal" (EPA 1987).

Response to Paragraph 3: To discuss the pathology results and to address the comment regarding the timeframe of tumor development, the following insert will be added to the third paragraph on page E-23 after the sentence "One hamster study, three dog studies, and two monkey studies were all considered unacceptable due to inadequate numbers or the short-term nature of the study relative to the life spans of the animals":

> Some of these studies found evidence of liver effects in treated groups but several did not. The hamster study (Cabral et al. 1979) detected liver cell hypertrophy in treated groups and hepatomas in two animals. The incidence of hepatomas was not significantly different from controls. Two dog studies (Treon and Cleveland 1955; Fitzhugh et al., 1964) revealed increased liver weights and

fatty degeneration of the liver and kidneys in treated animals. A third dog study using lower doses found no organ changes (Walker et al. 1969). No tumors were found in the dog studies. The study of monkeys fed high doses of dieldrin for six years found no evidence of tumors or liver cell damage (Wright et al. 1978). Mice and rats exhibit specific, observable changes to the liver cells after exposure to chlorinated insecticides. A study of three strains of mice exposed to dieldrin found that a small number of animals showed these changes as early as four months after exposure began (Meierhenry et al. 1983).

The hamster study had high premature mortality in both treatment and control groups and was therefore considered inadequate to determine carcinogenicity. The dog and monkey studies were considered inadequate because the study time was short compared to the animals' life spans (two-year study out of a ten-year life span for dogs and six-year study out of a twenty-year life span for monkeys). Mouse studies have found initial evidence of tumor development between 40 and 120 weeks in treatment groups (Walker et al. 1972; Thorpe and Walker 1973; Tennekes et al. 1982). This timeframe indicates the wide variability in time to tumor development within the animal's natural life span of approximately 130 weeks.

Response to Paragraph 4 (Comment 14): The possibility that aldrin and dieldrin are promoters rather than direct-acting carcinogens had been put forth as an explanation for the effects of these chemicals on mouse livers which may have a genetic predisposition to developing tumors. Although there have been no studies of aldrin/dieldrin exposure in humans with underlying risk factors, the point of the discussion was that it is still possible that, if these chemicals are promoters, humans who have preexisting abnormal liver cells may be at increased risk of developing liver cancer if they are exposed to these chemicals. Until more is known about the method by which aldrin and dieldrin exert their carcinogenic effects on mouse livers, EPA has elected to base its carcinogenicity assessment on the most sensitive species. Shell did not provide a reference for the NCI-SRI study that they cited, and the Army was unable to locate it.

To reiterate the uncertainty about the mechanism of carcinogenic effects the following text will be deleted from page E-24 after the sentence "These risk factors include cirrhosis of the liver (due to alcoholism or other causes) and a chronic carrier state for hepatitis B virus, which are not uncommon in the United States (there are an estimated 1 to 1.25 million

hepatitis B carriers in the United States) (MMWR 1991)":

> If aldrin and dieldrin are promoters, then exposure to them in the presence of another risk factor could increase the rate of development of liver cancer. EPA has incorporated an added layer of protection for sensitive human subpopulations in its carcinogenicity assessment by assuming that humans are as sensitive as the most sensitive species.

The following text will be inserted in its place (new paragraph):

> The mechanism by which aldrin and dieldrin exert their carcinogenic effects in mouse livers is unknown, but some evidence suggests that they may act as promoters (Wade et al as cited in EPA 1987). Promoters do not cause cancer by themselves, but may interfere with communication between cells that inhibits the growth of latent tumor cells (Casarett and Doull 1986). Thus promoters may enhance the development of preneoplastic lesions. This promoting effect can occur even if the contact with the promoter occurs after the exposure to the carcinogen (Casarett and Doull 1986). Sensitive subpopulations with preexisting abnormal liver cells who are exposed to a promoter may be at increased risk of developing liver cancer. If it is determined that aldrin and dieldrin are promoters, then it is possible that exposure to them in the presence of preexisting abnormal liver cells could increase the rate of development of liver cancer. No studies have been conducted to test the hypothesis that aldrin and dieldrin could increase the risk of liver cancer in sensitive human subpopulations. Because of the small numbers of people who have been exposed to measurable doses of these chemicals, this type of study would be difficult to conduct. However, because of this uncertainty, EPA has incorporated an added layer of protection for these sensitive human subpopulations by assuming that humans are as sensitive as the most sensitive species.

Response to Paragraph 5 (Comment 14): The following paragraph will be deleted from page E-24 after the sentence "EPA has incorporated an added layer of protection for sensitive human subpopulations in its carcinogenicity assessment by assuming that humans are sensitive as the most sensitive species":

> Supporting evidence of potential carcinogenic activity for aldrin was observed in the chromosomal aberrations in mouse, rat, and human cells and in unscheduled Deoxyribonucleic acid (DNA)

synthesis in rat and human cells. For dieldrin, positive responses have been observed in chromosomal aberration studies with mouse and human cells, as well as in mutation studies in hamsters, and in studies of unscheduled DNA synthesis in rat and human cells. Negative responses were observed for dieldrin in gene conversion and mutation assays with a variety of bacteria. Compounds structurally related to aldrin and dieldrin--chlordane, heptachlor, heptachlor epoxide, and chlorendic acid have induced malignant liver tumors in mice. Chlorendic acid produced liver tumor in rats.

The following text will be inserted in its place to reflect the information regarding mutagenicity in EPA's carcinogenicity assessment:

EPA's assessment of mutagenicity studies for aldrin (EPA 1987) concludes that aldrin is probably not mutagenic but major data gaps exist. EPA indicated that no conclusions can be drawn regarding chromosomal aberrations but found aldrin presumptively genotoxic in human cells based on unscheduled DNA synthesis. However, the validity of the positive studies was questioned due to technical deficiencies. Aldrin was not genotoxic in bacteria, yeast, and rat liver cells.

EPA's analysis of dieldrin (EPA 1987) found that the results of thirteen studies indicate that dieldrin is not mutagenic. There was one inconclusive presumptive positive study of gene mutations in bacteria but EPA concluded that technical deficiencies of the study precluded acceptance of the results as valid. There was also inconclusive evidence of mutagenicity in hamster cells but this study also suffered from technical deficiencies. Despite these technical deficiencies, EPA classified dieldrin as a presumptive mutagen for one bacteria and for hamster cells. Dieldrin was found to cause chromosomal aberrations in some human and mouse cells. It was determined to be presumptively genotoxic in human cells based on unscheduled DNA synthesis but technical deficiencies made the results inconclusive. There was no evidence for genotoxicity in yeast or in rat and mouse liver cells. A single study of epigenetic toxicity (effects that occur from a mechanism other than a direct effect on genetic material) suggests that dieldrin may possess promotional activity as evidenced by interference with cellular communication.

The World Health Organization reviewed the mutagenicity studies in its 1989 Environmental Health Criteria document (WHO 1989) and concluded that neither aldrin nor dieldrin exhibited mutagenic potential. WHO also cited studies indicating that aldrin and dieldrin interfere with intercellular communication.

Based on the lack of strong evidence for mutagenicity in any of the studies and the questionable validity of the few studies that were presumptively positive, it is likely that aldrin and dieldrin do not have significant genotoxic properties. However, until the major data gaps are addressed, a firm conclusion cannot be drawn regarding the genotoxicity of aldrin and dieldrin.

Response to Paragraph 6 (Comment 14): The de Jong study (1991) was not rejected on the basis of inadequate exposure quantification. The de Jong study had inadequate numbers of subjects to conclude with statistical certainty that there was no significant increase in the incidence of liver cancer in the exposed workers. When a rare health effect is being evaluated in an epidemiologic study, large numbers of subjects are required to have enough statistical power to conclude that the exposure did not increase the incidence of the rare health effect. The de Jong study had only a 35% chance of detecting an increased incidence of liver cancer even if the workers actually had a risk three times higher than the general population.

The worker who died from liver cancer was reported by his former family physician to have chronic alcoholism. This information will be added to the uncertainty section by inserting the following text on page E-25, before the sentence "The authors concluded that the results do not indicate an increased risk for cancer in general nor for specific neoplasms that could be attributed to exposures in aldrin and dieldrin":

This worker was reported to have chronic alcoholism by his former family physician.

Response to Paragraph 7 (Comment 14): The Sielken and Stevenson study cited by Shell does not address the issue of mortality at the Pernis plant, and may therefore be an incorrect citation. The intended citation may be the 1992 study entitled "Comparisons of Human Cancer Potency Projections for Dieldrin Based on Human Data with those Based on Animal Data" that was previously discussed on page E-25. The following response is based on that study.

It does not appear from the article that Sielken and Stevenson actually evaluated mortality. Rather, they reported on de Jong's analysis of SMRs. Therefore, to include the information on de Jong's mortality analysis, the following text will be inserted on page E-25, after the sentence "The authors concluded that the results do not indicate an increased risk for cancer in general nor for specific neoplasms that could be attributed to exposures to aldrin and dieldrin":

De Jong also evaluated mortality from total cancers and site-specific cancers for workers in the three exposure groups and compared the mortality rates to age- and gender-specific cancer mortality rates in the general population. The only significant increase in cancer mortality was for rectal cancers in the low exposure group.

Ecological Risk Assessment Issues:

Treatment of BCRLs and Spatial Interpolation:

Comment 15: The combination of the Army method to treat BCRLs and the Inverse Distance Weighting method to interpolate soil concentrations leads to an exaggerated and biased estimate of soil concentrations, particularly at the periphery of RMA, that is not supported by the available data. This leads to an artifactual inflation of risk estimates for all compounds, but particularly for compounds like DDT/DDE, mercury and arsenic that have relatively few high hits and a large number of relatively high BCRLs. For example, the attached maps (using mercury soil analyses, MK, March 16, 1994, Mercury Concentration 0-1 foot depth and IEA/RC (Version 3.1) Figure 4.5-5) show the disproportionate effect of the Army methods relative to the Theissen polygon approach to mapping actual soil concentrations. It should be noted that the attached Theissen polygon maps do not include a BCRL replacement method because there was not time to prepare one during this review, but Shell has discussed appropriate methods with the Army previously, such as the Maximum Likelihood Estimate (MLE).

Response: Virtually all of the apparent difference between Figure 4.5-5 from the IEA/RC version 3.1 and the mercury soil concentration map provided by Shell is explained by differences in the way soil concentration ranges are defined and color-coded on the maps. Both maps indicate essentially the same areas in exceedence of 1 ppm, 10 ppm, and 100 ppm. There are three small areas (center of Section 4, SW corner of Section 19, and NW corner of Section 23/NE corner of Section 22 where Shell's map indicates insufficient data to estimate mercury soil concentration, but the Army map shows that soil concentration estimates were made (all in the 0-1 ppm range). These three areas are small, and not deemed worthy of further investigation at this time. Elsewhere, the two maps appear to concur on areas of insufficient data. Therefore, the maps are in virtual agreement, and do not show the difference in soil concentration estimates that Shell has described. The Army and Shell have, as Shell notes, previously discussed BCRL replacement methods, including maximum likelihood estimation (MLE). The Army believes that the consideration of spatial information is a necessary condition for a reliable BCRL replacement

method. The BCRL replacement employed in the IEA/RC does consider spatial information. The MLE method that has been discussed with Shell is non-spatial.

Comment 16: Shell predicted that the Army methods would overestimate ecological risks substantially at RMA, but was unable to convince the Army that this was indeed the case. Shell declined to invoke dispute over this issue so that the IEA/RC could be brought to a timely and final conclusion. The overestimation of risk predicted by Shell have come to pass, as evidenced by the risks projected for DDT/DDE based on concentrations that in many areas are equivalent to national background levels and the large areas of mercury risk driven by a handful of mercury hotspots, while biota show little if any actual risk, based upon measured tissue concentrations.

Response: The large areas of mercury risk noted by Shell generally are not driven by mercury hotspots. There is not an observed overestimation bias in HQ estimates made by the Army approach. The Army's tissue concentration predictions are consistent with the tissue concentration field data collected at RMA.

Comment 17: There is little that can be done at this late stage in the process to correct the overestimation of risks that are due to the Army BCRL and spatial interpolation methods except to attempt to alert the risk manager and the public that the ecological risks projected over many parts of the Arsenal are an artifact of these methods and are unlikely to exist. It would be helpful if the Army included language in the executive summary and Chapter 4 to the effect that conservative methods were selected and a compounding effect resulted in an overestimation of these risks. In the meantime, data collected by USFWS and others at RMA related to ecological risk will be evaluated and reviewed to refine the ecological risks at RMA and to develop a more realistic and more defensible risk assessment at RMA in the future.

(See also Specific Comments Section relating to this issue).

Response: This comment provides no evidence to substantiate the cause and effect relationship that Shell claims exists between the Army's BCRL replacement and spatial interpolation methods, and overestimation of ecological risk attributable to mercury soil concentrations. The comment also does not provide evidence that the Army's soil concentration estimates are too high, or even that they exceed the estimates provided by Shell in the mercury soil concentration map referred to in this comment. Finally, this comment cites no evidence that the Army's mercury analysis systematically overestimates risks.

Overestimation of Mercury Risks:

Comment 18: Risks due to mercury were calculated based on the unsupported assumption that all mercury in the soil is in the form of the highly toxic and bioaccumulative methylmercury. It is likely that only a small fraction of the mercury present at the RMA is in the methylated form, and so the risks have been greatly overestimated. The Army should provide, at a minimum, a brief discussion of the quantitative impact of their assumption on the risks predicted from mercury and the total risk estimates.

Response: The analysis presented in the IEA/RC is not based on the total mercury assumption, and the statement that risks are overestimated because only a fraction of mercury is in the methylated form is incorrect. The biomagnification factors (BMFs) developed by all three approaches (Army, EPA, and Shell) are coefficients relating COC concentrations measured in the soil to COC concentrations measured in biota tissue, so the BMFs, if correct, do correctly account for limited bioavailability of soil contaminants, even though the processes (e.g., COC speciation) affecting bioavailability are not explicitly incorporated in risk calculations. The risk calculations account for mercury speciation as long as the fraction of bioavailable mercury in the soils to which the tissue samples used to derive BMF were exposed is representative of the true fraction of bioavailable mercury in RMA soils.

Additivity of HIs/HQs:

Comment 19: It is not prudent to add HQs for chemicals that do not have the same primary target organ or mechanism of action. The Army needs to make it clear that it is assuming that the hazards from different chemicals are additive, and that this is an assumption or policy decision and not a scientific fact.

A discussion about the appropriateness of the hazard index (HI) measure should also be given. The fact that the additivity of hazard quotients is consistent with EPA risk assessment guidance for human health, as cited on page SHELL-4 of Appendix F, does not make the HI correct, scientifically defensible, or noncontroversial.

EPA guidance for human health risk assessment also states that while "...application of the hazard index equation to a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism, although appropriate as a screening-level approach,

could overestimate the potential for effects." The guidance goes on to state that "[i]f the HI is greater than unity as a consequence of summing several hazard quotients of similar value, it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group" (EPA; RAGS 1989: Section 8, page 8-15).

For example, if the HQ for each of several different chemicals falls below a specified threshold, then there is no risk (according to the definition of threshold) from exposure to any of those chemicals. However, when the HI is computed, the addition of the different HQs may end up exceeding a threshold HI, implying that even thought each of the chemicals does not pose a risk by itself, when you add them all together they do pose a risk.

If these risks are combined, there should be explicit recognition that these risks are likely to be overestimated and the issue should be acknowledged early in the body of the text.

Response: The Army acknowledges Shells' comments regarding the assumption of additivity of hazard quotients (HQs). The following text will be added to the end of Section 4.1.2 of the Final IEA/RC:

> "The assumption that the hazards from the different COCs are additive is uncertain. For example, risks may be less than implied by the additivity assumption if the COCs do not induce the same type of effects or do not act by the same mechanism; or more than implied by the additivity assumption if the COCs induce synergistic effects. EPA risk assessment guidance for human health calls for additivity of hazard quotients, but also states that while '...application of the hazard index equation to a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism, although appropriate as a screening-level approach, could overestimate the potential for effects.' The guidance goes on to state that '(i)f the HI is greater than unity as a consequence of summing over several hazard quotients of similar value, it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group' (EPA RAGS 1989: Section 8, page 8-15). The segregation of COCs by effect or mechanism of action to derive separate HIs was not done for the ERC because of limited toxicological data on the COCs for the species or trophic boxes of concern or appropriate surrogate animals. Therefore, the Army considered it prudent to sum the individual HQ values and derive species or trophic box HIs, albeit this process probably resulted in an overestimation of potential risks."

Calibration of Army Method:

Comment 20: The Army did not "calibrate" its BMF method; it simply adjusted the BMFs to conform with the observed tissue concentrations, something the Shell method does directly. The calibration procedure in the Army approach is not well defined or reproducible (page C.1-45). In addition, the squared perpendicular distances between a regression line and a scatter plot of the TC and ESC pairs was described as being used because it provided "more reasonable" estimates of the BMFs. There is no discussion as to how the BMFs obtained using this ad-hoc criterion without statistical basis are more reasonable. That is, the BMFs are more reasonable than what?, and on what basis?, etc. The Army calibrated BMFs are, in general, difficult or impossible to reproduce because most of the calibration procedure is based on "professional judgement," without clear explanation of the standards used in the subjective assignment of weights. The whole calibration procedure should be more detailed, more objective (less based on "professional judgement"), and more scientifically defensible (less reliant on ad-hoc procedures) so that it could be reproduced and evaluated. An alternative would be to delete the calibration from the document.

Response: The Army's calibration procedure is well defined and reproducible. It is stressed that even though the procedure is well-defined and reproducible, different analysts might choose somewhat different numerical values for a BMF. This is a consequence of the fact that the data are incomplete, and the underlying sampling populations do not permit the application of formal statistical methods to derive a theoretically-based "best" estimate of BMF. This is stated in the second sentence of the description of the tissue concentration map evaluations, on page C.1-45 of the IEA/RC version 3.1. Any procedure that denies the residual uncertainty about BMF and the role of judgment in choosing BMF is arbitrary and provides false precision. As such, the most defensible approach is to define a reproducible procedure for analyzing the data that explicitly calls for the application of judgment. The Army's procedure does this.

The first paragraph on page C.1-45 of the IEA/RC version 3.1 will be modified as follows:

The text beginning with "Instead, professional judgment indicated that..." and continuing until the end of the paragraph will be deleted and replaced by "Instead, a set of eleven candidate BMFs were considered using the tissue concentration map evaluation protocol described below.

The set of BMFs were bounded by $BMF_{lit/model}$ and BMF_{obs} and given by the equation:

$$BMF = w \cdot BMF_{obs} + (1 - w) \cdot BMF_{lit/model} \qquad (40)$$
$$w = \{0, 0.1, 0.2, ..., 0.9, 1.0\}$$

Calculated BMF (Shell Method):

Comment 21: The Army presentation of the Shell method for deriving BMF estimates (page E-82 and Comment 6: page SHELL-4, Appendix F) is inaccurate in principle as explained below.

The principal error is that the Shell method does not ever use an estimated correlation between TC and ESC in its derivation of BMF estimates. The formulas (20), (21), and (22) on page C.1-31, which correctly identify the Shell method's estimate of the mean BMF_{obs}, do not contain any estimated correlations. Therefore, the implementation of Shell's method does not require any correlation to be estimated.

The assumption that BMF and ESC are uncorrelated implies that the corresponding correlation between lnTC and lnESC is equal to the variance of lnESC divided by the variance of lnTC; however, Shell's method does not require that the value of this correlation be estimated.

Response: Shell has commented that the Army presentation of the Shell method for deriving BMF estimates is inaccurate in principle. There are, however, two errors in Shell's supporting arguments that negate the premise. These are addressed below.

First, Shell claims that "the implementation of Shell's method does not require any correlation to be estimated." To prove its claim, Shell notes that "(t)he formulas (20), (21), and (22) on page C.1-31, which correctly identify the Shell method's estimate of the mean BMF_{obs}, do not contain any estimated correlations." IEA/RC equation 21, however, is derived from IEA/RC equation 13. It can be seen from equation 13 that $\hat{\sigma}^2_{ln(BMF)}$ is a function of the estimated correlation (r) of ln(TC) and ln(ESC), and from equation 22 that the mean BMF_{obs} is a function of $\hat{\sigma}^2_{ln(BMF)}$ and, therefore, of r. The first argument in the MAX function of equation 21 is a special case of equation 13, derived by setting $r = \hat{\sigma}_{ln(ESC)}/\hat{\sigma}_{ln(TC)}$ (the derivation of this estimator is provided below in equations 1-6). Thus, even though the correlation term does not explicitly appear in the Shell formulas for calculating BMF parameter estimates, it is implicit in the

formulas. The correlation coefficient is not calculated to solve the Shell formulas because it was already calculated to derive the formulas.

The second error in Shell's supporting arguments lies in the logic used to evaluate the assumed ln(TC), ln(ESC) correlation. As Shell states in its comment, the Shell method of estimating the mean BMF is based on the assumption that because BMF is constant, the correlation between BMF and ESC is zero. Because BMF is constant, ln(BMF) is constant as well, so the correlation between ln(BMF) and ln(ESC) is also zero. As Shell has noted, this implies that the correlation between $\ln(TC_{pred})$ and ln(ESC) is given by $\sigma_{\ln(ESC)}/\sigma_{\ln(TC_{pred})}$. This follows from the following derivation.

If $BMF = \frac{TC_{pred}}{ESC}$, then $\ln(BMF) = \ln(TC_{pred}) - \ln(ESC)$. It can then be shown that:

$$\sigma^2_{\ln(TC_{pred})} = \sigma^2_{\ln(BMF)} + \sigma^2_{\ln(ESC)} + 2\cdot\rho_{\ln(BMF),\ln(ESC)}\cdot\sigma_{\ln(BMF)}\cdot\sigma_{\ln(ESC)} \quad (1)$$

which reduces to:

$$\sigma^2_{\ln(TC_{pred})} = \sigma^2_{\ln(BMF)} + \sigma^2_{\ln(ESC)} \quad (2)$$

Because $\rho_{\ln(BMF),\ \ln(ESC)} = 0$.

In addition, it can be shown that:

$$\sigma^2_{\ln(BMF)} = \sigma^2_{\ln(TC_{pred})} + \sigma^2_{\ln(ESC)} - 2\cdot\rho_{\ln(TC_{pred}),\ \ln(ESC)}\cdot\sigma_{\ln(TC_{pred})}\cdot\sigma_{\ln(ESC)} \quad (3)$$

Solving equation 2 for $\sigma^2_{\ln(BMF)}$ and substituting into equation 3 gives:

$$\sigma^2_{\ln(TC_{pred})} - \sigma^2_{\ln(ESC)} = \sigma^2_{\ln(TC_{pred})} + \sigma^2_{\ln(ESC)} + 2\cdot\rho_{\ln(TC_{pred}),\ \ln(ESC)}\cdot\sigma_{\ln(TC_{pred})}\cdot\sigma_{\ln(ESC)} \quad (4)$$

and solving equation 4 for $\rho_{\ln(TC),\ \ln(ESC)}$ gives:

$$\rho_{\ln(TC_{pred}),\ \ln(ESC)} = \frac{\sigma_{\ln(ESC)}}{\sigma_{\ln(TC_{pred})}} \qquad (5)$$

which leads to the correlation assumption used to derive the first argument of the MAX function in IEA/RC equation 21 from IEA/RC equation 13:

$$r_{\ln(TC_{pred}),\ \ln(ESC)} = \frac{\hat{\sigma}_{\ln(ESC)}}{\hat{\sigma}_{\ln(TC_{pred})}} \qquad (6)$$

The error in Shell's logic is that the derivation just presented is built on the relationship between BMF, ESC, and predicted TC (TC_{pred}), rather than observed TC (TC_{obs}). The denominators in equations 5 and 6 are functions of TC_{pred}, yet tissue concentration field data (TC_{obs}) are used by Shell to estimate r. The correlation of tissue concentration predictions and exposure area soil concentration is expected to be higher than the correlation of tissue concentration data and ESC, because the data contain individual variability whereas the predictions do not.

Comment 22: Having identified the principal error in the existing paragraph, Shell suggests that the paragraph be rewritten as follows:

> The Shell method of estimating the mean BMF assumes a 0 correlation between BMF and ESC. The Shell method characterizes the uncertainty in its estimated mean BMF in terms of how much this estimate changes in response to variability in the TC and ESC data. A bootstrap estimate of the mean BMF is computed for each of 1000 different bootstrap samples of paired TC and ESC data. The impact of experimental variability on the estimated mean BMF is then characterized by the variability in the bootstrap estimates of the mean BMF. An advantage of Shell's method of estimating the mean BMF is that the method is designed to produce the BMF distribution (or constant, in some cases) which is best suited to predict the distribution of observed TCs from the distribution of observed ESCs when BMF and ESCs are assumed to be uncorrelated. Thus, Shell's method finds the estimate of the mean BMF that will perform the best when the tissue concentration predictions use the same BMF with all ESCs (that is, it is assumed that BMF and ESC are uncorrelated). This is an advantage because that is exactly how the tissue concentrations are predicted from the

ESCs in the rest of this report. A second advantage of Shell's method is that it does not require the correlation between TC and ESC to be estimated. A third advantage is that the effect on the estimated mean BMF of sampling variability in the database of TC and ESC values is characterized.

A disadvantage of Shell's method is that the uncertainty in the estimated mean BMF due to the assumption that BMF and ESC are uncorrelated is not quantified. A weakness of Shell's method (and all three methods of TC prediction used in this report) is that the observed estimates of the correlations between TC and ESC are often less than what would be expected under the assumption that BMF and ESC are uncorrelated.

(See also Specific Comments Section for additional suggestions and corrections regarding this issue).

Response: The Shell comment that the Army's presentation of its method is inaccurate does not appear to apply to the methodology section (C.1.5.1.2), for reasons given in the response to Comment 21. The Army also notes that in describing the Shell methodology (page C.1-31), the IEA/RC text does not state that Shell's method requires that the correlation between TC and ESC be estimated. Shell's comment that the Army's presentation of its method is inaccurate is addressed for other sections (C.1.5.1.3 and E.12.5) in response to a number of Shell's specific comments.

SPECIFIC COMMENTS

Comment 23: Table of Contents and Section 4.0. Change "characterization" in the section heading to "Ecological Risk Characterization".

Response: The word "characterization" will be replaced with "Ecological Risk Characterization" in the Section 4.0 heading and table of contents.

Comment 24: Page ES-7, last sentence. (Executive Summary in Volume I). Figure 5 is true only if the Army approach generates the true BMFs.

Response: The last sentence on page ES-7 will be revised to read:

"Figure E.S-5 depicts a soil remediation scenario, based on the Army approach, that would eliminate the potential risk (i.e., result in HQ $\leq$ 1.0 everywhere at RMA) to the great horned owl from aldrin/dieldrin. Note that the potential remediation area depicted

in Figure E.S-5 is not based on total risk, but only on risk to the great horned owl from exposure to aldrin/dieldrin, as predicted by the Army approach. The Army approach is presented because it is, in this case, the intermediate result regarding areal extent of risk."

Comment 25: Page 2-7, second paragraph. This paragraph states that the frequency of dieldrin detections onpost was 60 percent compared to less than 6 percent in offpost samples. This comparison is misleading in that the majority of the onpost samples were collected in areas of known contamination whereas the offpost samples were collected on a more random basis. Since the discussion of the frequency of dieldrin detections is not informative to the FS process, but may be confusing to the casual reader, the sentence containing this discussion should be deleted.

Response: The referenced sentence (sentence 4 of paragraph 2) will be deleted from the IEA/RC to avoid using a potentially misleading comparison.

Comment 26: Page 3-10, first paragraph in subsection 3.1.5. The fourth sentence mistakenly states that DT is the CSF divided by 10^{-6} instead of 10^{-6} divided by the CSF.

Response: The fourth sentence in this paragraph will be revised to state that DT is 10^{-6} divided by the CSF.

Comment 27: Page 3-11, first sentence in second complete paragraph. PPLVs should be identified as soil concentrations that "may have an upper bound on the risk" instead of "may pose a risk".

Response: The first sentence will be revised as follows: "PPLVs are defined....as soils concentrations unlikely to pose a cancer risk greater than a specified risk level (e.g., 10^{-4} or 10^{-6})."

Comment 28: Page 3-13, second sentence in the second paragraph. The 50th percentile represents the "median PPLV" and not necessarily the "most likely estimate (MLE) PPLV". Also, the abbreviation MLE in statistical literature implies the "maximum likelihood estimate" which in layman's terms is also the "most likely estimate" but the statistical abbreviation is not understood to be for "most likely estimate".

The text also refers to the "confidence that the cumulative PPLV will be protective at the specified risk level". The text should be modified to read that the "confidence that the cumulative PPLV will have an upper bound

on the cancer risk less than the specified risk level".

Response: The second sentence in the second paragraph will be revised as follows: ",....and the 50th percentile represents the median PPLV estimate (i.e., there is 50 percent confidence that the cumulative PPLV will not exceed the specified risk level)."

Comment 29: Page 3-14. Equation (5) should not be described as being for "Site risk for contaminant i" but rather for "An upper bound on the site risk for contaminant i". In addition, the upper bound is assuming that the dose-response relationship is linear which is not fact but an assumption--generally regarded as a conservative assumption.

Furthermore, when the upper bounds on the risk at the site are summed over chemicals, the confidence level of the resulting upper bound is not clear even if the chemicals acted independently. On the other hand, if the maximum likelihood estimates of the cancer risks for different independent chemicals were summed, then the summation would be the maximum likelihood estimate of the total risk and, hence, have a clear interpretation. Sums of maximum likelihood estimates have a more meaningful (well understood) interpretation than the sums of upper bounds.

Response: *Response to Paragraph 1:* For ease of interpretation, Equation (5) will be retained in its present format. The text revisions described in the above two responses will clarify the concept of the protectiveness associated with the risk estimate.

Response to Paragraph 2: While it is accurate to indicate that the sum of several maximum likelihood estimates will result in a value that is also a maximum likelihood estimate, it is unclear how use of this in the risk assessment will result in a more meaningful interpretation of risk.

Comment 30: Chapter 4. In general, the role of the food web model seems to be overstated and overemphasized, particularly with respect to the dose-based approach to ecological risk assessment. The dose-based approach characterizes the risk to a receptor directly in terms of its dietary components and not indirectly through a food web starting with the soil. Thus, the dose-based approach only uses the last portion of the food web model and not the whole model. Of course, if the ecological risk assessment were restricted to the dose-based approach the food web model would not be needed at all, just knowledge of the individual dietary components. The wording in the report should not overemphasize the role or importance of the food web model.

Response: Shell's comment regarding the dose based approach is incorrect; the dose-based approach is as dependent on the food web model as the tissue based approach. In the dose based approach, the predicted COC dose [μg COC/g predator*day] is calculated from the estimated exposure area soil concentration by the food web model equation:

$$\text{dose} = R \cdot \langle ESC \rangle \cdot \sum_{\substack{\text{prey} \\ \text{trophic} \\ \text{boxes}}} FR_i \cdot BMF_i$$

where R is the predators feeding rate coefficient [g prey/g predator * day], <ESC> is the estimated exposure area soil concentration [μg COC/g soil], FR_i is the mass fraction of the predator's diet comprised of prey trophic box i, and BMF_i is the BMF for prey trophic box i [(μg COC/g prey)/(μg COC/g soil)].

Comment 31: Page 4-2, section 4.1.1, first paragraph. Assumption required to match TRVs to ecological endpoints--that "risks posed to target receptors...are assumed to create risk to the integrity of the ecosystem..." is faulty and not in keeping with current ecological understanding. The hazard quotient approach employed in this risk assessment quantifies possible risk to individuals. Populations and communities exhibit properties of compensation and redundancy which buffer the ecosystem from the illness or loss of individual organisms. No information in the risk assessment, and no information available in the general ecological literature, supports this assumption as a general case. Language should be added to address this point.

Response: The Army acknowledges Shell's comment. The question of whether risks posed to target receptors create risk to the integrity of the system is not well-posed, in that "risk to the integrity of the system" is not adequately defined. Therefore, it cannot be argued that "risks posed to target receptors create risk to the integrity of the system" is a generally valid assumption, nonetheless, it is an assumption underlying any ecological risk assessment based on toxicologically-based measurement endpoints.

Comment 32: Page 4-6, fourth line of third paragraph. (Volume I). ESC is wrongly defined as "Exposure soil concentration" when it is defined elsewhere as "estimated exposure area soil concentration". See page xi and page 4-7.

Response: The first two sentences of the third paragraph on page 4-6 will be revised to read:

"Actual exposure conditions for biota populations are difficult to measure because such measurement requires detailed information about individual organisms' behaviors as well as similar information about prey and the average soil concentration of bioavailable contaminant (the "exposure soil concentration") to which the organisms are directly and indirectly exposed. Exposure soil concentrations were estimated by estimated spatially averaged soil concentrations within "exposure areas," i.e., well defined areas selected to correlate with the foraging range of the target biota receptor for which the risks were estimated."

Comment 33: Page 4-8, section 4.3. Assumption that RMA risk estimates implicitly include dermal and inhalation exposure is only true for MATC-based evaluations. Dose-based risks do not incorporate these routes. However, as stated here, it is true that these routes likely represent *de minimis* component of exposure, and are unlikely to greatly impact risk assessment results.

Response: The second to last sentence on page 4-8 will be deleted.

Comment 34: Page 4-9. First paragraph. (Volume I). ESC is defined as "estimated average exposure area soil concentration" when it is defined elsewhere as "estimated exposure area soil concentration". See page xi and page 4-7.

Response: The correct nomenclature is as follows:

> ESC is defined as the exposure area soil concentration.
> <ESC> is defined as the estimated exposure area soil concentration.

The nomenclature will be corrected in the next version of the IEA/RC. Exposure soil concentration is not assigned an abbreviated name, and will be written out in full.

Comment 35: Page 4-12, third to last line. The sentence would read better if the words "over which measured soil concentrations" are deleted.

Response: The last sentence on page 4-12 will be revised to read:

> "For example, if the exposure assessment were to change such that the depth profile or some other aspect of the soil sampling protocol changed, then the <ESC> value would change and the BMF value would have to be recalculated accordingly so that the HQ could be correctly calculated."

Comment 36: Page 4-13, the last words of the last paragraph ("by improving information on site specific BMF") should be dropped. Phase I of the supplemental study, as Shell understands it, is not to improve information on site specific BMF.

Response: The phrase "by improving information on site specific BMF" will be dropped from the end of the last paragraph on page 4-13.

Comment 37: Page 4-14, end of first sentence. The current wording "by the dissociated TC/ESC pairs and taking their ratio, based on assumptions about the correlation of TC and ESC." is confusing with respect to TC/ESC representing a pair and not BMF and with respect to "taking their ratio" meaning the ratio of TC and ESC or the ratio of the TC and ESC distributions. A better sentence would result if those words are replaced by "by dissociating the pairs of TC and ESC values and deriving the BMF from these distributions and assumptions about the correlations between TC, ESC, and BMF."

Response: The end of the first sentence on page 4-14 (starting with the words "by the dissociated..." will be replaced with:

> "by dissociating the pairs of TC_{obs} and <ESC> values and deriving the BMF from these distributions and assumptions about the correlation between TC_{obs}, <ESC>, and BMF."

Comment 38: Page 4-14, the fifth sentence of the first paragraph that reads "The Shell approach and the EPA approach use the field BMF directly" should be replaced by the sentence "The Shell method incorporates the likelihood of the observed field data directly into the BMF estimation rather than in a separate calibration step."

Also, there is no observed "field BMF".

Response: The fifth sentence of the first paragraph on page 4-14 will be replaced with the sentence:

> "Shell and EPA chose not to incorporate a step parallel to the Army's calibration step in their approaches, although there is nothing about the Shell and EPA approaches that would prohibit including such a step."

Comment 39: Page 4-14, third line of the second paragraph. The portion of the sentence that reads "true correlated TC and ESC data distributions" should be replaced by either of the next two following wordings: a) "true co-located TC and ESC data distributions", or b) "true co-location or pairing between TC and the ESC actually biologically related to that TC".

Response: The sentence as written is valid and will be retained in its current form; the proposed revisions change its meaning.

Comment 40: Page 4-14, line 9 of the second paragraph. The end of the sentence should be deleted, because there is no heavy professional judgement or data extrapolation involved in the Shell method. That is, delete ", which depend more heavily on data extrapolations and best professional judgement."

Response: The sentence as written is valid and will be retained in its current form; the Shell and Army approaches rely on the extrapolated data in the <ESC> maps, on BCRL replacement values, and on $\{TC_{obs}$, <ESC>$\}$ correlation assumptions.

Comment 41: Page 4-15, first complete sentence of first paragraph does not need to overemphasize the calibration of the Army's approach. This sentence could be dropped.

Response: The first complete sentence of the first paragraph on page 4-15 will be dropped.

Comment 42: Page 4-16, equation 14. The right hand side of the equation should be divided by TRV_j.

Response: The right hand side of equation (1) should be divided by TRV_j; this correction will be made.

Comment 43: Page 4-17. The sentence that starts on the first line is incorrect as it is. It should read "Only Equation (14) was used for the ...."

Response: The sentence that starts on the first line of page 4-17 will be revised to read:

> "Only equation (14) was used for the nonbioaccumulative COCs and chlordane."

Comment 44: Page 4-21, first full paragraph, second sentence. This paragraph should be revised to indicate that 18 inches of clean soil were placed over the former Basin "F" area.

Response: The second sentence of the first full paragraph on page 4-21 will be revised to read:

> "Because of the Basin F IRA, a layer of 18 inches of clean soil was placed over and now covers the former basin, thus eliminating exposure to contaminants at this location."

Comment 45: Page 5-11, line 12. Change the words "contributed to a lack of correlation" to "helped diminish the correlation" A lack of correlation sounds like 0 correlation when it is meant to be low correlation as indicated by the wording in the next sentence.

Response: On page 5-11, line 12, the words "contributed to a lack of correlation" will be changed to "helped diminish the correlation."

Comment 46: Page 6-5, line 8. A better description of the HI would be given if the words"... represents the highest level of ..." are changed to "... represents a lower bound on the highest level of ..." Shell believes it is very important to emphasize the difference between a lower bound on the dose corresponding to an increase in added risk and an estimate of that dose.

Response: The sentence starting on line 7 of page 6-5 will be revised to read:

> "For the purposes of the ERC, an HQ or HI of 1.0 has been defined as the best estimate of the highest level of chronic exposure that is unlikely to result in adverse effects on the average individual of specific populations or subpopulations exposed chronically in the field."

Comment 47: Page 6-10,seventh line. "Uncertainties in conversions of concentrations between sediments and water". This appears to be related to the previous use of the WASP Model and may no longer be relevant or applicable.

Response: Shell's comment is correct; the last bulleted item will be deleted.

Comment 48: Page 6-11, third sentence in the first complete paragraph. The use of the linearized multistage cancer dose-response model is inaccurately characterized as involving an "appropriate degree of conservatism". The

word "appropriate" would be more appropriately replaced by "frequently a substantial" or "frequently a significant".

Furthermore, there are numerous articles published in the peer-reviewed literature that identify numerous flaws in the cancer risk characterizations based on the linearized multistage model. The section should identify the existence of several scientific concerns about the relevance of linearized multistage model based characterizations of human cancer risks. In addition, there are peer-reviewed articles proposing alternative cancer risk characterizations for some of the principle COCs (e.g., aldrin and dieldrin). The report needs to acknowledge the alternatives and state the basis for the characterizations being used.

In the last sentence on page 6-11, to be consistent with the EPA guidelines and the discussion in 5.1.2.1 of the report, the wording "reasonable characterization of the risk for populations receiving the highest exposure" should be changed to "plausible upper limit to the risk for populations receiving the highest exposures. Such a limit is consistent with some proposed mechanisms of carcinogenesis but does not necessarily give a realistic prediction of risk. The true value of the risk is unknown and may be as low as zero."

Response: *Response to Paragraph 1:* The word "appropriate" will be removed from the third sentence of the first complete paragraph.

Response to Paragraph 2: The first complete paragraph on page 6-11 acknowledges that the use of the linearized multistage model is a major source of uncertainty in the risk assessment. It is unnecessary to provide a review of the scientific literature confirming this finding in this section of the IEA/RC.

Response to Paragraph 3: Given the theme of this paragraph, identification of uncertainties associated with carcinogenic mechanisms is not appropriate. However, to increase the clarity of the report, the last sentence on page 6-11 will be revised as follows: "It is important to acknowledge, however, that the methods used are consistent with current practice in risk assessment, as well as the latest EPA guidance on risk assessment (1989)."

Comment 49: Page 6-12, first paragraph. (Volume I). ESC is wrongly defined as "exposure area soil concentration" when it is defined elsewhere as "estimated exposure area soil concentration". See page xi, page 4-7.

Response: See the response to Shell Comment 34. ESC nomenclature will be corrected in the next version of the IEA/RC. Exposure soil concentration

is not assigned an abbreviated name, and will be written out in full.

Comment 50: Page 6-13. Immediately before section 6.4 a paragraph on the assumption of additivity of HQs similar to the third paragraph of page 6-11 should be included.

Response: An eighth bulleted item will be added to the list on page 6-12:

- HQ additivity assumption

Comment 51: Page 6-14,seventh line. The line should be modified to read "... an HI of 1.0 represents a lower bound on the highest level of chronic exposure ..."

Response: The sentence starting on line 6 of page 6-14 will be revised to read:

> "An HI of 1.0 has been defined as the best estimate of the highest level of chronic exposure that is unlikely to result in adverse effects on the average individual of specific populations or subpopulations exposed chronically in the field."

Comment 52: Page 6-17. The first sentence should read "Figures 6.4-1 through 6.4-3 depict a soil ...".

Response: The first sentence on page 6-17 will be revised to read:

> "Figures 6.4-1 through 6.4-3 depict soil remediation scenarios that would eliminate the potential aldrin/dieldrin risk (i.e., reduce the aldrin/dieldrin HQ to 1.0 or less) to the great horned owl, based on the three approaches' great horned owl BMFs."

Comment 53: Page B.1-24, Equations 25 and 26. The exposure frequency is inconsistent with the other portions of this document in that it assumes an exposure period of 365 days per year. Since no one will be in an enclosed space at RMA every day of the year for 70 years the following revisions should be made:

> Exposure frequency from DW/365 to DW/253
>
> Exposure duration from TE/70 to TE/30

Response: The referenced equations (25 and 26) are accurate as written and take into account Shell's concern. For these equations, the exposure frequency is

"DW" (days/year) and the exposure duration is "TE" (years). The DW and TE values used in the assessment are listed in Table B.1-5. DW is divided by 365 days/year in the equations in order to obtain a fraction of the year over which exposure is assumed to occur. Similarly, TE is divided by 70 years/lifetime for carcinogens to obtain a fraction of the years of a person's life over which exposure is assumed to occur.

Comment 54: Page C.1-10, first paragraph. "The weights are inversely proportional to the inverse of the squared..." should be changed to "The weights are inversely proportional to the squared..." or to "The weights are proportional to the inverse of the squared..."

Response: The phrase "inverse of the squared" will be deleted from line 2, page C.1-10.

Comment 55: Page C.1-17. The third sentence of the last paragraph states that "... under the basic premise of the Dirichlet tessellation approach, an estimate for a BCRL data point would be based only on the value of its nearest neighbor". This statement seems incorrect or at least misleading. The description of the Dirichlet tessellation approach is similar to (or is another name for) the Theissen polygon approach that Shell used. The Shell Theissen polygon based procedure does not replace a soil concentration BCRL "...based solely on the value of the closest neighbor..." but rather a soil concentration BCRL is replaced on the basis of the distribution of soil concentrations found in the area where the BCRL occurred. Thus, either the 2nd and 3rd sentences in the paragraph should be dropped or followed by a new sentence "A Theissen polygon approach could also have been implemented with BCRLs replaced by the conditional expected values from distributions of soil concentrations in the same area."

Response: The third sentence of the last paragraph on page C.1-17 will be followed by a new sentence:

> "A Theissen polygon approach also could have been implemented with BCRLs replaced by the conditional expected values from distributions of soil concentrations in the same area."

Comment 56: Page C.1-17. Unless the Army has developed a method to modify their inverse distance-squared method and software to incorporate "less than" information, the last sentence on this page is not defensible and should be deleted.

Response: The last sentence on page C.1-17 will be revised to read:

> "The inverse distance squared method and software was modified to incorporate "less than" information, and provide estimates of BCRL data points."

Comment 57: Page C.1-25, the fourth line of the first paragraph reads "At each sample ....". It should read "At each tissue sample ....".

Response: The fourth line of the first paragraph on page C.1-25 will be revised to read "At each tissue sample....

Comment 58: Page C.1-25, fifth line of the second paragraph, before the sentence that starts "In cases where....", the following sentence should be inserted: "A preferred, but more complicated implementation of Shell's method would have incorporated BCRLs directly into the maximum likelihood estimation rather than generating specific TC replacement values."

Response: The second paragraph on page C.1-25 will be retained as written. The Army does not believe that the implementation referred to in the proposed insert can be accurately described as either preferred or more complicated, from a statistical point of view.

Comment 59: Page C.1-26. On the third line under the section on Statistical Terminology the portion of the sentence should read ".... describing the sample mean of X," since the mean of X is a constant.

Response: The phrase "describing the mean" in the third line on page C.1-26 will be replaced with "describing the estimated mean."

Comment 60: Page C.1-26. On the last item of the terminology, the subscripts for μ and σ should be $\bar{BMF}$, with a capital M rather than a lower case m. Also, the last line has in parenthesis "(described under Method 2)." This should be dropped or replaced by "(described under Shell's and Army's methods)."

Response: The BmF will be replaced with BMF in the subscripts for μ and σ on the last item of the terminology, and the parenthetical phrase will be deleted.

Comment 61: Page C.1-28. After Equation 13 it reads "where r = correlation (ln(TC), ln(ESC))." It should read "where r = *assumed* correlation (ln(TC), ln(ESC))." This change will make the presentation of the Army's method and the Shell's method balanced when referring to assumptions.

Response: The equation following equation 13 will be revised to read:

"r = assumed (ln(TC), ln(<ESC>)) correlation."

Comment 62: Page C.1-29, the last sentence of the paragraph following Equation 14, the sentence starts as "Each r sample was used in equations (11) and (12) to....". The sentence should read "Each r sample was used in equations (13) and (14) to...."

Response: The equation numbering will be corrected throughout Appendix C.1.

Comment 63: Page C.1-29. In Equation (17) the subscript for σ should be BMF_{obs}.

Response: The subscript for σ will be revised to $\overline{BMF}_{obs}$.

Comment 64: Page C.1-30, the line immediately above Equation 18, reads "as in equations (16) and (17):". It should read as in Equations (18) and (19):".

Response: The equation numbering will be corrected throughout Appendix C.1.

Comment 65: Page C.1-31, paragraph immediately after Equation (21). The reference to equations in this paragraph should be as follows: (10) should be replaced by (12), (19) should be replaced by (21) in both instances, and (11) should be replaced by (13).

Response: The equation numbering will be corrected throughout Appendix C.1.

Comment 66: Page C.1-31, the first line of the paragraph after Equation 22, makes reference to Equation (19). The equation number (19) should be replaced by (21).

Response: The equation numbering will be corrected throughout Appendix C.1.

Comment 67: Page C.1-31, Part a. should start "Draw N pairs {Tissue Concentration, ESC} of ....."

Response: Under part a at the bottom of page C.1-31, {Concentration, ESC} will be replaced with {TC, <ESC>}.

Comment 68: Page C.1-31, Part b is incomplete. Part of the sentence in the previous draft seems to have been inadvertently dropped and should be added to end the sentence: "parameter distributions from the pooled TC and ESC data, respectively, using estimators on the natural logarithmic scale:".

Response: Part b at the bottom of page C.1-31 will be replaced with:

> "b. Dissociate these pairs {TC, <ESC>}, then calculate the mean and standard deviation for the TC and <ESC> distributions. "

Comment 69: Page C.1-32, Part c. "... equations (18), (19) and (20)." should be changed to "... equations (20), (21) and (22)."

Response: The equation numbering will be corrected throughout Appendix C.1.

Comment 70: Page C.1-32. Equation 28 should have "$(\overline{BMF}_{obs})_i$" on the numerator instead of "$\overline{BMF}_i$".

Response: Equation 28 on page C.1-32 will be corrected to read:

$$\hat{\mu}_{\overline{BMF}_{obs}} = \frac{\sum_{i=1}^{1000} \left(\overline{BMF}_{obs}\right)_i}{1000}$$

Comment 71: Page C.1-32. Equation 29 should be "Std. Dev. $(\overline{BMF}_{obs})$" instead of "Std. Dev. $(\overline{BMF})$".

Response: Equation 29 on page C.1-32 will be corrected to read:

$$\hat{\sigma}_{\overline{BMF}_{obs}} = \text{Std.Dev.}\left(\overline{BMF}_{obs}\right)$$

Comment 72: Page C.1-33. The second line should have "$(\overline{BMF}_{obs})_i$" instead of "$\overline{BMF}$".

Response: The equation on the second line of page C.1-33 will be corrected to read:

$$\overline{BMF}(_{obs})_i = \text{i th bootstrap estimate.}$$

Comment 73: Page C.1-38, the fourth sentence on the last paragraph is misleading. The sentence states: "Although both estimators are unbiased for lognormally distributed data, one or both of the estimators were apparently biased for the RMA data due to nonlognormality and the high proportions of BCRL data points." The Shell method provides unbiased estimates for μ_{lnTC}, σ_{lnTC}, μ_{lnESC}, σ_{lnESC} and biased estimates for μ_{TC}, σ_{TC}, μ_{ESC}, σ_{ESC}. The Army method provides unbiased estimates for μ_{TC}, σ_{TC}, μ_{ESC}, σ_{ESC} and biased estimates for μ_{lnTC}, σ_{lnTC}, μ_{lnESC}, σ_{lnESC} -- the latter being acknowledged in Appendix E on page E-85.

Response: The first through sixth sentences of paragraph 3, page C.1-37 will be replaced with:

> "EPA's approach assumes that the data pairing an observed tissue concentration (TC_{obs}) with a "predicted <ESC>" (possibly containing "location error") provides appropriate information on the relationship between TC_{obs} and "estimated actual ESC" (<ESC> without location error). Location error is the error associated with the assumption that tissue samples were taken at the center of the sampled organism's home range. "Predicted <ESC>" is the <ESC> estimate centered at the location where an organism is sampled. "Estimated actual ESC" is the <ESC> concentric with the organism's (unknown) home range.
>
> "The Army and Shell approaches assume that the predicted <ESC> and TC_{obs} data are inaccurately paired in the sense that the predicted <ESC> paired with a TC_{obs} contains location error, and therefore the relationship between the TC_{obs} and actual <ESC> distributions cannot be estimated based on the paired sample data

which typically have correlations near zero. If this is the case, then the estimation of the mean BMF will be biased upward. To avoid this suspected bias, the Army and Shell approaches make assumptions regarding the correlation between the variables TC_{obs}, <ESC>, and BMF.

"As shown below, any assumption regarding the correlation of TC_{obs} and <ESC> has implications regarding the correlation of <ESC> and BMF, and visa versa. The Army approach restricts the correlation between ln(TC_{obs}) and ln(actual <ESC>) to what the Army assumed to be a plausible range of values. The assumption used in the Army approach implies that BMF and <ESC> are correlated. The rationale for assuming non-zero correlation between BMF and <ESC> is that the IEA/RC estimates risk under the constraint that the true mean BMF and TC_{pred} have zero correlation. (The correlation of BMF and <ESC> implies non-zero correlation between BMF and TC_{obs}. Non-zero correlation between BMF and TC_{obs} is needed to obtain zero correlation between BMF and TC_{pred}, because of errors in the measured values TC_{obs} as estimates of the spatially distributed population mean tissue concentration.) The assumption used in the Shell approach assumes that BMF and <ESC> are uncorrelated and that the estimates of BMF obtained from the available <ESC> data are appropriate for estimating population mean tissue concentrations at RMA. The rationale for assuming zero correlation between BMF and <ESC> is that the IEA/RC estimate risk under the assumption that the true mean BMF and <ESC> have a zero correlation."

The following will be inserted at the end of the correlation section:

"The Army and Shell approaches for calculating the variance of ln(BMF_{obs}), and from this the mean BMF_{obs}, both make assumptions about the correlations between TC_{obs}, <ESC>, and BMF_{obs}. They are derived using standard statistical theory from two forms of the same general equation, the first relating TC_{obs}, <ESC>, and BMF_{obs}:

$$BMF_{obs} = \frac{TC_{obs}}{\langle ESC \rangle}$$

$$\ln(BMF_{obs}) = \ln(TC_{obs}) - \ln(\langle ESC \rangle)$$

$$\sigma^2_{\ln(BMF_{obs})} = \sigma^2_{\ln(TC_{obs})} + \sigma^2_{\ln(\langle ESC \rangle)} - 2 \cdot \rho_{(\ln(TC_{obs}),\ln(\langle ESC \rangle)} \cdot \sigma_{\ln(TC_{obs})} \cdot \sigma_{\ln(\langle ESC \rangle)} \qquad (1)$$

and the second relating BMF_{obs}, TC_{pred}, and <ESC>:

$$
\begin{aligned}
TC_{pred} &= BMF_{obs} \cdot \langle ESC \rangle \\
\ln(TC_{pred}) &= \ln(BMF_{obs}) + \ln(\langle ESC \rangle) \\
\sigma^2_{\ln(TC_{pred})} &= \sigma^2_{\ln(BMF_{obs})} + \sigma^2_{\ln(\langle ESC \rangle)} + 2 \cdot \rho_{\ln(BMF_{obs}),\, \ln(\langle ESC \rangle)} \cdot \sigma_{\ln(BMF_{obs})} \cdot \sigma_{\ln(\langle ESC \rangle)} \\
\sigma^2_{\ln(BMF_{obs})} &= \sigma^2_{\ln(TC_{pred})} - \sigma^2_{\ln(\langle ESC \rangle)} \; 2 \cdot \rho_{\ln(BMF_{obs}),\, \ln(\langle ESC \rangle)} \cdot \sigma_{\ln(BMF_{obs})} \cdot \sigma_{\ln(\langle ESC \rangle)}
\end{aligned}
\tag{2}
$$

If one assumes that:

$$\sigma^2_{\ln(TC_{obs})} = \sigma^2_{\ln(TC_{pred})} = \sigma^2_{\ln(TC)}$$

and

$$\rho_{\ln(TC_{obs}),\, \ln(\langle ESC \rangle)} = \rho_{\ln(TC_{pred}),\, \ln(\langle ESC \rangle)} = \rho_{\ln(TC),\, \ln(\langle ESC \rangle)}$$

then equations (1) and (2) are equivalent and therefore indicate that any assumption regarding the values of the correlation between BMF_{obs} and <ESC> implies a formula for the correlation between <ESC> and TC_{obs}, and visa versa. In particular, the Shell method assumes:

$$\text{Relationship(i)} \quad \rho_{\ln(BMF_{obs}),\, \ln(\langle ESC \rangle)} = 0$$

which implies:

$$\text{Relationship(ii)} \quad \rho_{\ln(TC),\, \ln(\langle ESC \rangle)} = \frac{\sigma_{\ln(\langle ESC \rangle)}}{\sigma_{\ln(TC)}}$$

The equivalency of the implications of these relationships can be seen by substituting them into the respective forms of the equation for the variance of $\ln(BMF_{obs})$, that is, relationship (i) is applied using the form given in equation (2) and the relationship (ii) is applied using the form given in equation (1). With these substitutions made, both equations simplify to:

$$\sigma^2_{\ln(BMF_{obs})} = \sigma^2_{\ln(TC)} - \sigma^2_{\ln(\langle ESC \rangle)} \tag{3}$$

subject to the constraint that

$$\sigma^2_{\ln(BMF_{obs})} \geq 0 \qquad (4)$$

Equations (3) and (4) result in equation (21) used in the Shell approach. The variance of $\ln(BMF_{obs})$ can be calculated from either formula by plugging in the assumed variance of zero for $\rho_{\ln(BMFobs),\ \ln(<ESC>)}$ into equation (2) or by estimating $\rho_{\ln(TC),\ \ln(<ESC>)}$ using relationship (ii) and applied using the form given in equation (1). The motivation behind Shell's approach is that if BMF_{obs} and <ESC> are assumed to be independent when risk and TC predictions are made, then BMF_{obs} and <ESC> should be treated as independent when the mean BMF_{obs} is estimated.

The Army method assumes values for the correlation between $\ln(TC_{obs})$ and $\ln(<ESC>)$ and therefore simultaneously implies a relationship for the correlation between $\ln(<ESC>)$ and $\ln(BMF_{obs})$:

$$\rho_{\ln(BMF_{obs}),\ \ln(\langle ESC\rangle)} = \frac{\rho_{\ln(TC_{obs}),\ \ln(\langle ESC\rangle)} \cdot \sigma_{\ln(TC_{obs})} - \sigma_{\ln(\langle ESC\rangle)}}{\sigma_{\ln(BMF_{obs})}}$$

The motivation behind the Army's approach is that if BMF and TC_{pred} are defined to be independent, then the correlation between BMF_{obs} and <ESC> should be non-zero. This is because BMF_{obs} is calculated from <ESC> and TC_{obs}, and if BMF_{obs} and TC_{obs} have non-zero correlation, then BMF_{obs} and <ESC> also must have non-zero correlation in order for BMF_{obs} and TC_{pred} to be independent."

Comment 74: Page C.1-39. Because Shell's approach and EPA's approach are two very different approaches, they should not be lumped together in the text. The third paragraph that describes Shell's approach and EPA approach should be split into two paragraphs, one discussing Shell's approach and another paragraph describing EPA's approach.

Response: The third paragraph on page C.1-39 will be split into two paragraphs at the end of the third sentence (which ends "...transformed parameters.")

Comment 75: Page C.1-39. The paragraph describing Shell's approach should be: "The Shell approach uses logarithmic sample estimators; i.e., the mean and variance of the log-transformed tissue concentration and ESC. These estimators are the maximum likelihood parameter estimates of the TC and ESC lognormal distributions. These MLEs were used because the

parameters of the BMF distributions are functions of the mean and variance of the log-transformed tissue concentration and ESC (μ_{lnTC}, σ_{lnTC}, μ_{lnESC}, σ_{lnESC}) rather than μ_{TC}, σ_{TC}, μ_{ESC}, σ_{ESC}."

Response: The description of Shell's approach in the third paragraph of Page C.1-39 will be retained in its current form except nomenclature will be corrected by replacing ESC with <ESC>.

Comment 76: Pages C.1-41 and C.1-48. Equations numbers on the equations are wrong. The text would refer to the correct equation numbers if the mistake made after Equation 33 were corrected. That is, after Equation 33, the next equation number should be 34, then 35, and so on, instead of 36 and 37 as it is now. This correction would also correct the problem of having equation numbers 38 and 39 repeated.

Response: The equation numbering will be corrected throughout Appendix C.1.

Comment 77: Page C.5-3, Section C.5.2.1. The information in this section is relatively accurate; however, descriptions of Arsenal plant communities and animal habitats have been merged with regional descriptions. The result is a slight exaggeration of diversity, (e.g. although "hawthorn may form dense thickets" in riparian areas regionally, hawthorn is not common on RMA).

Response: The section noted is organized in three parts, the first describes the regional vegetation setting for RMA, the second presents the types and percentages of communities/habitats present on RMA, and the third describes these communities/habitats floristically. To address Shell's concern and clarify the section, the transitions between these sections have been strengthened.

Comment 78: Page C.5-5, second paragraph. Habitat modification projects have been joint efforts by USFWS, Army and Shell. The Army should also be given credit on line 1.

Response: Line 1 on page C.5-5 has been modified as suggested.

Comment 79: Page C.5-17, fourth paragraph, line 7-8. Although an effort "to locate and account for all dead animals" on RMA has not been conducted, nor would such a search be practical over such a large area, USFWS has conducted search transects which have indicated no mortality above that expected (See minutes of the 1/11/94 meeting of the Natural Resources Conservation Committee).

Response: The last sentence in the section on morbidity has been revised in response to Shell's comment and a comparable comment by the USFWS as follows: "For example, at RMA the number of dead animals located may be inflated over normal numbers as a result of a large, observant worker population (particularly in the vicinity of Building 111, the Administration Building). However, there are three ongoing USFWS surveys that are adding data on morbidity: (1) monitoring of dead animal occurrence in the vicinity of Building 111; (2) monitoring the number of dead animals encountered during a morning Dawn Patrols of RMA roads; and (3) quarterly surveying of dead animals encountered in walking transects across several RMA sections. The walking surveys, in particular, should provide more definitive information on the presence of carcasses in uncultivated habitat."

Comment 80: Page C.5-46, third paragraph, line 6-7 and page C.5-47, first paragraph, second line. It should be noted that, by definition, the feeding history of fortuitous samples is not known and, although the concentrations detected are consistent with known exposure pathways, the real source of tissue contamination in these samples is undetermined.

Response: The last sentence on page C.5-46 has been revised as follows in response to this comment: "When interpreting this information, two important considerations should be kept in mind: (1) the exposure of highly mobile raptors is unknown and their tissue concentrations could reflect exposure off RMA, and (2) the brain to whole-body ratio of dieldrin tends to be highly variable, ranging between 0.1 and 2 based on a survey of the general literature."

Given this revision, the second line at the top of page C.5-47 has not been revised.

Comment 81: Page C.5-60, third paragraph, last line. The citation MKE 1989a is incorrect. ESE 1989 may be the correct citation.

Response: ESE (1989) is the correct citation. The text has been revised accordingly.

Comment 82: Page C.5-25, third paragraph, line 5. Recommend deleting the word "possible" to describe Sections 25 (which includes North Plants) and 36 (which includes Basin A) with relation to contamination. Both of these Sections are universally agreed to be source areas.

Response: In the second sentence of the last paragraph on page C.5-25, the word "possible" has been changed to "likely". A qualifying word was retained to address the point (made by EPA earlier) that the actual soil

concentrations to which the collected prairie dogs were exposed are unknown.

Comment 83: Page C.5-27, third paragraph, line 11. Delete "in part" as this phrase appears twice.

Response: The second occurrence of the phrase "in part" has been deleted.

Comment 84: Page C.5-32, second paragraph, second to last line. The word "here" should be changed to "there."

Response: The suggested correction has been made.

Comment 85: Page C.5-39, second paragraph, second to last line. The word "to" should be changed to "do."

Response: The suggested correction has been made.

Comment 86: Page E-72. Add the following sentence at the end of the second paragraph: "A preferred, but more complicated implementation of Shell's approach would have incorporated BCRLs directly into the maximum likelihood estimation of the TC distribution rather than generating specific TC replacement values."

Response: The following sentence will be added to the end of the second paragraph on page E-72:

> "Shell would have preferred that BCRLs be incorporated directly into the maximum likelihood estimation of the TC distribution in the Shell approach, rather than generating specific TC replacement values by the robust method."

Comment 87: Page E-81. Add the following sentence at the end of the first paragraph: "A preferred, but more complicated implementation of Shell's approach would have incorporated BCRLs directly into the maximum likelihood estimation of the TC distribution rather than generating specific TC replacement values."

Response: Shell's preference for directly incorporating BCRLs into MLE estimation of the TC distribution will be noted in the revision to the second paragraph on page E-72; see the Army's response to Shell comment 86.

Comment 88: Page E-82. The first sentence should be replaced by "The Army method assumed that the correlation between the natural logarithm of TC and the natural logarithm of ESC would likely range from 0.3 to 0.7, and would tend to be an intermediate value near 0.5."

Response: The first sentence on page E-82 will be changed to read:

> "The Army method assumed that the correlation between the estimated ln(TC) and ln(<ESC>) distributions would likely range from 0.3 to 0.7, and would tend to be an intermediate value near 0.5."

In the second sentence, "Correlations less than 0.3 were" will be replaced with:

> "A ln(TC), ln(ESC) correlation less than 0.3 was."

In the third sentence, "Correlations greater than 0.7 were" will be replaced with:

> "A ln(TC), ln(ESC) correlation greater than 0.7 was."

In the fifth sentence, "the Army midpoint correlation" will be replaced with:

> "the Army's mean ln(TC), ln(ESC) correlation."

Comment 89: Page E-82. The ninth line, namely " Shell maximized the TC and ESC correlation ..." should be replaced by "Because tissue concentration predictions are made using the same value of BMF irrespective of the ESC value, Shell estimated BMF under the corresponding assumption that BMF and ESC are uncorrelated. If BMF and ESC are uncorrelated, then the corresponding correlation between TC and ESC is equal to the variance of lnESC divided by the variance of lnTC. If this corresponding correlation were estimated, then the estimated value would be large when the estimated variance of lnESC is large compared to the estimated variance of lnTC."

Shell did not "maximize" the correlation between TC and ESC. If Shell was trying to "maximize" the correlation "to the extent 'allowed' by the data", it could have assumed that the correlation was always 1.0. However, this was not what was done. Furthermore, this would have been inconsistent with Shell's assumption that BMF and ESC were uncorrelated.

Response: The sixth sentence of the first paragraph on page E-82 containing "EPA used the observed paired data correlations near 0 while Shell maximized the TC and ESC correlation...will be replaced with:

> "The EPA estimates depend on the correlations in the paired, screened data, which were near zero, while the Shell assumption about BMF and <ESC> being uncorrelated and the estimates of $\sigma_{ln(ESC)}$ and $\sigma_{ln(TC)}$ implied correlations between ln(TC) and ln(<ESC>) which were generally higher than 0.5."

Comment 90: Page E-82. The sentence that starts on the 14th line reads "The disadvantages of the Army method is ...". It should read "The disadvantages of the Army method are ...".

Response: The sentence that starts on the 14th line of page E-82 will be corrected to read "The disadvantages of the Army method are..."

Comment 91: Page E-82. The following sentence should be added to the end of the first paragraph: "A third disadvantage is that the Army method estimates BMFs that are correlated to (dependent on) the magnitude of the ESC, however, the BMFs and the ESCs are assumed to have 0 correlation (be independent) when used to estimate ecological risks."

Response: The proposed addition to the end of the first paragraph on page E-82 does not make the distinction between the correlation of <ESC> and TC_{pred} and the correlation of <ESC> and TC_{obs}. Nonetheless, the Army acknowledges the need to provide some clarification to the Correlation subsection of Section E.12.5, COMPARISON OF ALTERNATIVE BMFs. The following revisions will be made.

The two paragraphs under the correlation heading on page E-81 will be replaced with the three paragraphs that follow:

> "All three BMF calculation approaches require estimation of the correlation between the populations of TC and ESC estimates used to derive BMF_{obs}. The three BMF approaches differ in their correlation assumptions. The remainder of this section discusses uncertainty about correlation, and describes the advantages and disadvantages of the correlation assumptions for the three BMF approaches.
>
> "As discussed in E.12.4.4, the TC_{obs} and <ESC> data sets had correlations which tended to be low or near zero, with inconsistent

and seemingly arbitrary medium and high correlations for some trophic box/COC combinations. In addition, screening the paired data sets did not increase correlation. The low and variable sample correlations between the TC_{obs} and <ESC> data sets is due to both representation and estimation uncertainty, as defined in Section E.9.4.. Because of representation uncertainty, the true correlation between population mean tissue concentration (TC) and exposure area soil concentration (ESC) is expected to be less than one, and variable across trophic box/COC combinations. Consequently, the sample correlation between TC_{obs} and <ESC> data sets is also expected to be less than one, and variable across trophic box/COC combinations. In addition, because of estimation uncertainty in both TC_{obs} and <ESC>, the sample correlation between TC_{obs} and <ESC> data sets is expected to be even lower than the true correlation between TC and ESC. The correlation between the populations of TC_{obs} and <ESC> estimates used to derive BMF_{obs} probably lies somewhere between the sample correlation between TC_{obs} and <ESC> data sets and the true correlation between TC and ESC (except for the EPA BMF approach, where the TC_{obs} and <ESC> data sets <u>are</u> the populations used to derive BMF_{obs}, so the sample correlation between the screened, paired (TC_{obs}, <ESC>) data <u>is</u> the correlation between the populations used to derive the EPA BMF_{obs}).

"An important source of uncertainty about correlation is uncertainty in estimates of exposure range center, as described in Section E.9.4.2. The error in the assumption that organisms are sampled at the center of their home ranges is referred to as "location error." Location error weakens the observed relationship between TC_{obs} and <ESC>, lessens the observed correlation, and makes it more difficult for all three approaches to estimate BMF_{obs}. Paired (TC_{obs}, <ESC>) data contain location error, but the pairs (TC_{pred}, <ESC>) do not. Because the BMF_{obs} is used to calculate TC_{pred} from <ESC>, the data used to calculate BMF_{obs} should be corrected for location error. However, the location error is unknown, so BMF_{obs} must be estimated either from paired data containing location error, or from distributions that are based on the data, but do not preserve the pairing between TC_{obs} and <ESC>. The EPA method uses paired data that contain location error. As a result, it underestimates the correlation of TC and ESC, which introduces a positive bias of unknown magnitude into the EPA BMF_{obs}. Location error also impacts the Army and Shell approaches. However, the Army and Shell approaches do not preserve the pairing between the sample TC_{obs} value and its predicted <ESC>, so they are not impacted by individual differences between predicted and estimated actual <ESC>s. (A sample TC_{obs} 's

predicted <ESC> is the <ESC> centered on the location where the tissue concentration sample is collected. The estimated actual ESC is the <ESC> that is concentric with the home range of the sampled organism.) Instead, the Army and Shell approaches are impacted by the overall difference between the distribution of predicted <ESC>s and the distribution of actual <ESC>s."

The following will be inserted at the end of the second sentence of second paragraph on page E-82:

"under the assumption of zero correlation between <ESC> and BMF. In the IEA/RC, risks are estimated (i.e., TC and dose are predicted) under the assumptions of zero correlation between BMF and <ESC> and between BMF and TC_{pred}."

The remainder of the second paragraph on page E-82, starting at "The disadvantages of this approach..." will be replaced with:

"The disadvantage of the Shell correlation assumption is that the appropriateness of Shell's assumption that BMF and <ESC> are uncorrelated (and similarly, the appropriateness of the Army's assumption that $\rho_{ln(TC),\ ln(ESC))} \sim$ triangular(0.3, 0.5, 0.7), which is meant to capture the implicit assumption that BMF and TC_{pred} are uncorrelated) is unknown and may vary from case to case. Even though the BMF is assumed to be uncorrelated with <ESC> during the prediction of TCs and risks, the estimation of BMF is still dependent on the TC and <ESC> data, unless these data are correlated to a specific degree (defined by $\rho_{ln(TC),\ ln(ESC))} = \sigma_{ln(ESC))}/\sigma_{ln(TC)}$)."

This description of the disadvantage of the Shell method is comparable to the description of the disadvantages of the Army method at the end of the first paragraph on page E-82. Minor modifications to the description of the disadvantages of the Army method will be made as follows:

"The disadvantages of the Army approach are as follows. First, any estimate of correlation which appears to be reasonably reliable (high N and moderate to high correlation) is not used for the associated trophic box/chemical, increasing the potential for bias in that case. A second disadvantage is that the appropriateness of the triangular distribution for representing the correlation between the

estimated ln(TC) and ln(<ESC>) distributions is unknown and may vary from case to case."

The first sentence in the second paragraph on page E-83 will be replaced with:

"The EPA estimation of BMF depended on the correlation between TC and its predicted <ESC> in the screened paired data sets."

The last three sentences in the first (partial) paragraph on page E-84 will be replaced with:

"The advantage of the EPA correlation assumption is that it utilizes the information contained in the pairing of the data. However, this information is subject to location error and other types of estimation error. The disadvantage of the EPA correlation assumption is that it has a positive bias of unknown magnitude due to location error."

Comment 92: Page E-84, the first two sentences of the Parameter Estimation section should be as follows: "The Army method uses arithmetic sample estimators, i.e., the mean and variance of the untransformed TC and ESC data, and then converts the arithmetic estimators using standard formulas to estimate the parameters of the lognormal distribution. The Shell method uses logarithmic sample estimators, i.e., the mean and variance of the natural logarithm of TC and ESC data, to estimate the parameters of the lognormal distribution."

Response: The first two sentences of the Parameter Estimation section, page E-84, will replaced with:

"The Army method uses arithmetic sample estimators, i.e., the mean and variance of the untransformed TC and ESC data, and then converts the arithmetic estimators using standard formulas to estimate the parameters of the lognormal distribution. The Shell method uses logarithmic sample estimators, i.e., the mean and variance of the natural logarithm of TC and ESC data, which can be converted using standard formulas to get estimates of the mean and standard deviation of the lognormal distribution."

Comment 93: Page E-85, the last sentence of the first paragraph should be replaced by "The disadvantage of the arithmetic estimators is that, while they are statistically unbiased estimators of the mean and standard deviation of the

observed data, they are biased estimators of the parameters of the lognormal distribution and do not have the lowest variance of all estimators when used on distributions known to be lognormal."

Response: The last sentence of the first paragraph on page E-85 will be replaced by:

> "The disadvantage of the arithmetic estimators is that, while they are statistically unbiased estimators of the mean and standard deviation of the observed data, they do not have the lowest variance of all estimators when used on distributions known to be lognormal."

Comment 94: Page E-85, first sentence in the second paragraph. Replace the clause "Although they are not the minimum variance unbiased estimators for the lognormal distribution," by "The Shell method provides minimum variance unbiased estimates for the parameters of the lognormal distributions (i.e., μ_{lnTC}, σ_{lnTC}, μ_{lnESC} and σ_{lnESC}) but not minimum variance unbiased estimates for μ_{TC}, σ_{TC}, μ_{ESC} and σ_{ESC} (Gilbert 1987). However, the formulas for the estimates of the parameters in the lognormal distribution for BMF are in terms of the estimates of μ_{lnTC}, σ_{lnTC}, μ_{lnESC} and σ_{lnESC}, rather than in terms of estimates of μ_{TC}, σ_{TC}, μ_{ESC} and σ_{ESC}." and then continue with "Hence, the log based estimators used for the Shell method are unbiased and lower in variance than ...."

Response: The phrase "Although they are not minimum variance unbiased estimators for a lognormal distribution, " will be deleted from the start of the first sentence in the first full paragraph on page E-85.

Comment 95: Table E12-1. The BMF for medium mammal listed under the BMF by the Army Calibration Procedure is 38. That number seems incorrect. We think that the correct number should be between 0.17 and 0.38.

Response: The medium mammal aldrin/dieldrin BMF listed in the "BMF by the Army Calibration Procedure" column of Table E.12-1 should be 0.38. Table E.12-1 will be corrected accordingly.

Comment 96: Table E12-1. The sample mean for the $BMF_{lit/model}$ (6.5)for terrestrial plant seems incorrect. We think that the correct number should be 0.65.

Response: The sample mean for the terrestrial plants aldrin/dieldrin $BMF_{lit/model}$, listed as 6.5 in Table E.12-1, should be 0.65. Table E.12-1 will be corrected accordingly.

Comment 97: Table E12-2. The BMF for medium mammal listed under the BMF by the Army Calibration Procedure is 49. That number seems incorrect. We think that the correct number should be between 0.49 and 4.0.

Response: The medium mammal DDE/DDT BMF listed in the "BMF by the Army Calibration Procedure" column of Table E.12-2 should be 0.49. Table E.12-2 will be corrected accordingly.

Comment 98: Appendix F, Response to Comment 6. Several of the above comments are also comments on this response. The comments are collected again here. Explaining the error in the Army's description of the treatment of correlation in the Shell method:

The principal error is that the Shell method does not ever use an estimated correlation between TC and ESC in its derivation of BMF estimates. The formulas (20), (21), and (22) on page C.1-31, which correctly identify the Shell method's estimate of the mean BMF_{obs}, do not contain any estimated correlations. Therefore, the implementation of Shell's method does not require any correlation to be estimated.

The assumption that BMF and ESC are uncorrelated implies that the corresponding correlation between lnTC and lnESC is equal to the variance of lnESC divided by the variance of lnTC; however, Shell's method does not require that the value of this correlation be estimated.

Response: The remaining Shell comments are a synopsis of issues pertaining to the Army's description of the Shell BMF approach in the Proposed Final IEA/RC, Version 3.1 and in Shell Comment 6 on the Proposed Final IEA/RC, Version 3.0.

In its Comment 6 on the Proposed Final IEA/RC, Version 3.0 (Appendix F, page SHELL-4 of the Proposed Final IEA/RC, Version 3.1), Shell states that:

> "The Shell method and corresponding statistical equations do not require that the correlation between TC and ESC be estimated at all. The Shell method also makes no other assumptions about correlation and does not require, utilize, or estimate any other correlation values."

The Army addresses this statement in its response to Shell Comment 21. The correlation coefficient need not be estimated when the Shell formulas are implemented, but it is needed to derive the Shell formulas. Shell acknowledges this in its statement that "(t)he assumption that BMF and ESC are uncorrelated implies that the corresponding correlation between

lnTC and lnESC is equal to the variance of lnESC divided by the variance of lnTC; however, Shell's method does not require that the value of this correlation be estimated."

The Army is not concerned with whether any of the three approaches requires calculation of the estimated correlation coefficients for BMF and <ESC> or TC_{obs} and <ESC>. The BMF values calculated by each of the three approaches are dependent on either implicit or explicit underlying correlation assumptions.

Comment 99: Having identified the principal error in the existing paragraph, we suggest that the paragraph be rewritten as follows:

> The Shell method of estimating the mean BMF assumes a 0 correlation between BMF and ESC. The Shell method characterizes the uncertainty in its estimated mean BMF in terms of how much this estimate changes in response to variability in the TC and ESC data. A bootstrap estimate of the mean BMF is computed for each of 1000 different bootstrap samples of paired TC and ESC data. The impact of experimental variability on the estimated mean BMF is then characterized by the variability in the bootstrap estimates of the mean BMF. An advantage of Shell's method of estimating the mean BMF is that the method is designed to produce the BMF distribution (or constant, in some cases) which is best suited to predict the distribution of observed TCs from the distribution of observed ESCs when BMF and ESCs are assumed to be uncorrelated. Thus, Shell's method finds the estimate of the mean BMF that will perform the best when the tissue concentration predictions use the same BMF with all ESCs (that is, it is assumed that BMF and ESC are uncorrelated). This is an advantage because that is exactly how the tissue concentrations are predicted from the ESCs in the rest of this report. A second advantage of Shell's method is that it does not require the correlation between TC and ESC to be estimated. A third advantage is that the effect on the estimated mean BMF of sampling variability in the database of TC and ESC values is characterized. A disadvantage of Shell's method is that the uncertainty in the estimated mean BMF due to the assumption that BMF and ESC are uncorrelated is not quantified. A weakness of Shell's method (and all three methods of TC prediction used in this report) is that the observed estimates of the correlations between TC and ESC are often less than what would be expected under the assumption that BMF and ESC are uncorrelated.

Response: In its response to Shell Comment 21, the Army has described two errors in Shell's supporting arguments that negate the premise that the Army's presentation of the Shell method for deriving BMF estimates, specifically regarding Shell's correlation assumptions, is inaccurate in principle. Therefore, the premise for modifying the last paragraph of the Army's response to Comment 6 (Appendix F, page SHELL-5) is unfounded.

Comment 100: The Army response to Comment 6, suggests that "The three BMF methods are all designed to provide the best available estimate of the mean BMF from the currently available tissue and soil concentration database." However, from a statistical perspective the Army's approach to parameter estimation is indirect whereas Shell's approach is direct. That is, Shell's approach directly estimates the parameters in the equations for the parameters of the BMF distribution whereas the Army's approach estimates secondary parameters that can then be used to estimate the actual parameters in the equations for the parameters of the BMF distribution. Thus, the Army's approach requires an extra step and uses the data to estimate secondary parameters rather than the parameters actually needed to find the BMF parameters.

> The Shell method provides unbiased estimates for μ_{lnTC}, σ_{lnTC}, μ_{lnESC}, σ_{lnESC} and biased estimates for μ_{TC}, σ_{TC}, μ_{ESC}, σ_{ESC}. The Army method provides unbiased estimates for μ_{TC}, σ_{TC}, μ_{ESC}, σ_{ESC} and biased estimates for μ_{lnTC}, σ_{lnTC}, μ_{lnESC}, σ_{lnESC}—the latter being acknowledged in Appendix E on page E-85.

and

> The Shell approach uses logarithmic sample estimators; i.e., the mean and variance of the log-transformed tissue concentration and ESC. These estimators are the maximum likelihood parameter estimates of the TC and ESC lognormal distributions. These MLEs were used because the parameters of the BMF distributions are functions of the mean and variance of the log-transformed tissue concentration and ESC (μ_{lnTC}, σ_{lnTC}, μ_{lnESC}, σ_{lnESC}) rather than μ_{TC}, σ_{TC}, μ_{ESC}, σ_{ESC}.

Response: The Army acknowledges Shell's position regarding parameter estimation for TC and <ESC> distributions. The Army has provided a clear and objective comparison of the two approaches on pages C.1-38, C.1-39, and E-84 through E-86 of the Proposed Final IEA/RC, Version 3.1. Modifications based on Shell's Version 3.1 comments are given in the Army's responses to Shell Comments 74, 75, 92, 93, and 94.

Comment 101: The Army response to Comment 6, suggests that "Shell uses bootstrap resampling (with an assumed correlation of TC and ESC) to estimate the variability of TC and ESC distributions that would result from different possible (TC,ESC) sampling realizations." The bootstrap resampling of the (TC,ESC) pairs does not use or require any "assumed correlation of TC and ESC".

Response: Shell is correct; the bootstrap resampling of (TC, <ESC>) pairs does not use or require any assumed correlation of TC and <ESC>. The correlation assumption enters into the Shell approach when the bootstrap resampling results are used to estimate the variance of $\ln(BMF_{obs})$. The formula for the estimated variance of $\ln(BMF_{obs})$ does depend on the Shell correlation assumption.

Comment 102: The Army response to Comment 6, incorrectly suggests that the purpose of the bootstrap resampling is to "estimate the variability in (TC,ESC) samples" instead of "to estimate BMF and its uncertainty". The purpose of the bootstrap resampling is to estimate the BMF for each bootstrap sample and to characterize the portion of the uncertainty in the estimate of BMF due to sampling variability in the (TC,ESC) database. As indicated above:

> The Shell method characterizes the uncertainty in its estimated mean BMF in terms of how much this estimate changes in response to variability in the TC and ESC data. A bootstrap estimate of the mean BMF is computed for each of 1000 different bootstrap samples of paired TC and ESC data. The impact of experimental variability on the estimated mean BMF is then characterized by the variability in the bootstrap estimates of the mean BMF.

Response: Shell is correct in its characterization of the purpose of bootstrap resampling.

Comment 103: The last line of the Army response to Comment 6, incorrectly suggests that in the EPA method "BMF uncertainty is estimated based on the sample variance and (TC,ESC) sample correlation." The last portion of that sentence, namely " and (TC,ESC) sample correlation," is incorrect and should be deleted.

Response: The Army's statement that (EPA) "BMF uncertainty is based on the sample variance and (TC, ESC) sample correlation" could be more precisely worded to read "BMF uncertainty is based on the sample variances of the screened TC and <ESC> data sets, and on the correlation

in the equations for the EPA approach; the correlation assumption is implicit.

References:

Cabral, J.R.P, R.A. Hall, S.A. Bronczyk, and P. Shubik. 1979. A carcinogenicity study of the pesticide dieldrin in hamsters. Cancer Lett. 6:241-6.

Casaratt and Doull 1986. Toxicology, Third edition. Macmillian Publishing Company, New York.

de Jong, G., 1991. "Long Term Health Effects of Aldrin and Dieldrin". Elsevier, Amsterdam.

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2:551-62.

Meierhenry, E.F., B.H. Reuber, M.E. Gershwin, L.S. Hsieh, and S.W. French. 1983. Dieldrin-induced mallory bodies in hepatic tumors of mice of different strains. Hepatology 3:90-95.

MMWR (Morbidity and Mortality Weekly Report) 1991. Volume 40, No. RR-4. April 19, 1991. Hepatitis B. Virus: A Comprehensive Strategy for Eliminating Transmission in the United States through Childhood Vaccination.

RTIC 89173R02
ESE (Environmental Science and Engineering) 1989. Biota Remedial Investigation, Rocky Mountain Arsenal. Final Report. Version 3.2.

RTIC 91309R06v.1
EPA (U.S. Environmental Protection Agency) 1989. Risk Assessment Guidance for Superfund. Volume I. Human Heath Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response. EPA/540/1-89/002.

Sielken, R.L. and D.E. Stevenson, 1992. Comparisons of Human Cancer Potency Projections for Dieldrin Based on Human Data with Those Based on Animal Data. In: D'Amato, slaga, et al. (ads). Relevance of Animal Studies to the Evaluation of Human Cancer Risk. Wiley-liss, Inc. New York. pp. 295-319.

Tennakas, H.A., L. Elder, and H.W. Kunz. 1982. Dose-response analysis of the enhancement of liver tumor in CF-1 mice by dieldrin. Carcinogenesis 3:8:941-5.

Thorpe E. and A.I.T. Walker. 1973. The toxicology of dieldrin (HEOD). Part II. Comparative long-term oral toxicology studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC, and gamma-BHC. Food Cosmet. Toxicol. 11:433-41.

Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. Agric. Food Chem. 3:402-8.

Walker, A.I.T., D.E. Stevenson, J. Robinson, E. Thorpe, and M. Roberts. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two year oral exposures of rats and dogs. Toxicol. Appl. Pharmacol. 15:345-73.

Walker, A.I.T., E. Thorpe, D.E. Stevenson DE. 1972. the toxicology of dieldrin (HEOD). Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11:415-32.

WHO (World Health Organization) 1989. Environmental Health Criteria 91: Aldrin and Dieldrin. Geneva.

Wright, A.S., C. Donninger, R.D. Greenland, K.L Stemmer, M.R. Zavon. 1978. The effects of prolonged ingestion of dieldrin on the livers of male rhesus monkeys. Ecotoxicol. Environ. Safety 1:477-502.

(There was no reference given for the Wade et al. citation in EPA 1987.)

References Cited - TDV Distributions

Benjamin, J. R., and C. A. Cornell 1970. Probability, Statistics and Decision for Civil Engineers. McGraw-Hill, New York, pp. 269-270.

Gilbert, Richard O. 1987. Statistical Methods for Environmental Pollution Monitoring. Van Nostrand Reinhold, New York, pp 174-175 and 254.

U.S. FISH AND WILDLIFE SERVICE

U.S. Fish and Wildlife Service
Proposed Modifications to the Text
of the Integrated Endangerment Assessment
Risk Characterization (IEA/RC)

Comment 1: Page ES-4. The last sentence of the first paragraph of Section 3.0 seems to imply that remediation of the Arsenal is required because of refuge management. In fact, the Arsenal requires remediation because of environmental contamination at the site. The Service agrees that remediation techniques should be selected as to be consistent with the ultimate use of the Arsenal as a Refuge, but that is a matter for the Feasibility Study, not the IEA/RC. Also, the "Refuge Act of 1992" is properly referred to as the "Rocky Mountain Arsenal National Wildlife Refuge Act of 1992." The Service suggests the following text changes:

> "...when selecting environmental remedies and for the future management of RMA as a National Wildlife Refuge as authorized by the Rocky Mountain Arsenal National Wildlife Refuge Act of 1992."

Response: The text in the Executive Summary referencing the Rocky Mountain Arsenal Wildlife Refuge Act has been changed to incorporate the text recommended by the USFWS.

Comment 2: Page 2-2, Section 2.1.2. The third paragraph of this section discusses bird species present at the Arsenal. The Service responded to similar comments from the Colorado Department of Health in a letter dated February 23, 1994, and the appropriate section is provided below.

> The Service is presently devising a bird list based on documented sightings. This work is being conducted by Hugh and Irling Kingery, two well known and respected birders. This bird list is divided into two groups; birds seen consistently in the proper habitat in the correct season (153 species) and those birds (accidental) which have been documented less than three times (73 species), for a total of 226 species. The Health and Diversity chapter references a list of 176 species including accidentals and gives it a ranking of 40% of all Colorado birds. Holt and Lane (1988) list 440 species in Colorado based on the same references cited by this chapter. That makes the Arsenal's total documented bird species 56.5% of the total Colorado birds and 34.8% of the total if the accidentals are subtracted.

The Service realizes that this information may have been presented too late for inclusion in this version of the IEA/RC, but suggests appropriate text changes to incorporate the more-accurate data.

Response: The first two sentences of the last paragraph on page 2-2 have been revised as follows: "One hundred seventy-five species of birds were identified on RMA by MKE (1989a) (Appendix Attachment C.5-1), which is approximately 40 percent of the 440 bird species recorded in the State of Colorado (Bailey and Niedrach 1965, Chase et al. 1982, Holt and Lane 1988). An ongoing study of bird presence being supported the USFWS has thus far revealed 153 species of birds seen consistently in the proper habitat in the correct season and 73 species of birds documented fewer than three times. These two groups of bird species, thus, respectively comprise 35 and 17 percent of the total Colorado species. Each of these data sources shows that the species richness of RMA avifauna is high relative to that of the region." This text will also be added to the first paragraph in Section C.5.2.2.2.

Comment 3: Page C.5-17, Morbidity. The last sentence of the introductory paragraph states that "no specific effort has been made to locate and account for all dead animals." While the Service would agree that any effort to account for all dead animals at a site the size of the Arsenal would have a very low probability of success, the Service is nevertheless performing three specific studies examining the occurrence of dead animals at the Arsenal. First, the Service monitored the occurrence of dead animals in the vicinity of Building 111 during FY93. The results of this effort have been presented in the Service's FY93 Annual Report. Second, the Service is monitoring the number of dead animals encountered during our morning Dawn Patrols of the Arsenal. This effort documents the number of animals seen per mile of road driven during the daily patrol. This is currently underway and will be continuing indefinitely. The Service is also performing quarterly walking transects of several sections of the Arsenal. This effort involves a specified number of staff walking 1 mile transects evenly spaced across a section (square mile). All dead animals encountered in each transect are recorded. This effort is repeated across several selected sections during a day, and repeated four times in a week changing direction walked each time. In conclusion, while only limited information has been presented regarding specific efforts to study the occurrence of dead animals at the Arsenal, there are at least three efforts currently underway and continued efforts will undoubtedly occur throughout remediation. Please delete or alter the phrase "... no specific effort has been made to account for all dead animals."

Response: The last sentence in the section on morbidity has been revised in response to USFWS's comment and a comparable comment by Shell as follows: "For example, at RMA the number of dead animals located may be inflated over

normal numbers as a result of a large, observant worker population (particularly in the vicinity of Building 111, the Administration Building). However, there are three ongoing USFWS surveys that are adding data on morbidity: (1) monitoring of dead animal occurrence in the vicinity of Building 111; (2) monitoring the number of dead animals encountered during a morning Dawn Patrols of RMA roads; and (3) quarterly surveying of dead animals encountered in walking transects across several RMA sections. The walking surveys, in particular, should provide more definitive information on the presence of carcasses in uncultivated habitat."

Comment 4: Page E-14. While the definitions of Recreational/Casual and Recreational visitors and their activities presented in sections E.5.1.1 and E.5.1.2 may be appropriate for the stated use, it should not be mis-interpreted by the reader that all of these activities (walking, jogging, bicycling, cross-country skiing, picnicking, etc.) are currently allowed or that any or all of these uses will continue in the future. Allowable public uses of the Arsenal will be determined in the Service's Refuge Management Plan currently under development. The Service will consider information developed in the IEA/RC and any other risk management decisions on future public use when formulating allowable activities at the Refuge.

Response: Sections E.5.1.1 and E.5.1.2 will be clarified to convey that the current and future recreational land uses upon which this risk assessment are based are hypothetical and may not necessarily be allowable public uses. A sentence will be added to the end of the second paragraph in Sections E.5.1.1 and E.5.1.2 that states: "The allowable future land uses for RMA will be determined in the Fish and Wildlife Service's Refuge Management Plan, which is currently under development."

REFERENCES

Bailey, A.M. and R.J. Neidrach. 1965. Birds of Colorado. Denver Museum of Natural History, Denver, CO.

Chase, C.A., S.J. Bissel, H.E. Kingery, W.D. Graul, and M.M. Dillon. 1982. Colorado Bird Distribution Latilong Study. Denver Museum of Natural History, Denver,CO.

MKE (Morrison-Knudsen Engineering) 1989. Wildlife Resources of Rocky Mountain Arsenal.

STATE OF COLORADO

Disputes of the State of Colorado Regarding
Second Proposed Final Integrated Endangerment
Assessment/Risk Characterization

GENERAL COMMENTS

I. Mischaracterization of Party Consensus:

Comment 1: In responses to several technical comments previously submitted by the State, the Army replies that State representatives participated in meetings in which the issues were discussed, and consensus reached. These responses mistakenly imply that the State agreed with these decisions at the time they were made. Such is not the case. In fact, although allowed to present its views, the State was summarily dismissed from meetings in which the decisions were actually made, and such decisions often did not address the State's expressed concerns which, therefore, remain. Examples include responses to State General Comments 3, paragraph 2; 4, paragraph 8; ERC General Comments 16, paragraphs 4 and 8; 18, paragraph 3; 19, paragraph 1; 20, paragraph 1; 22, paragraph 1, 23, paragraph 3; 24, paragraph 1; 25, paragraph 1; ERC Specific Comments 58, paragraph 1; 64, paragraph 1.

Response: The Army's responses were intended to indicate that the State was an active participant in the Technical Subcommittee meetings held to discuss and formulate approaches to be used in the ecological risk assessment. The State was only asked to leave when consensus decisions were made because the State elected not to be a signatory to the Federal Facility Agreement, thus limiting their role when final decisions were made. The consensus decisions reflect the agreements between the signatory parties. The State had ample opportunity to present information at the meetings, to react to information presented by other parties, and to make its positions clear to the other parties prior to the consensus deliberations. If the consensus decisions did not reflect the State's concern, that means that the other four parties did not agree with or fully support the State's position or concern. The fact that the State has these concerns is noted.

II. Exclusion of Basin F Wastepile Risk:

Comment 2: In this version of the IEA the Army includes old Basin F in the risk assessment but has excluded risks from the Basin F Wastepile. The present consolidation of highly concentrated wastes in the wastepile should be included in the risk assessment.

Response: The following text describing the potential risks associated with the Basin F Wastepile has been added to the IEA into Section 3.35 of the Qualitative Risk Assessment (current Section 3.3.5 will be renumbered to 3.3.6).

Potential risks associated with the Basin F Wastepile were not quantified because of the difficulty in determining a meaningful exposure point concentration. It is known that materials with concentrations that would exceed 10^{-3} carcinogenic risk or an HI of 1,000 are in the Basin F Wastepile; the quantities and locations of these materials are not known. Therefore, given the difficulty in determining exposure point concentrations, the Basin F Wastepile is referred to the FS for consideration in final remediation. Risks identified for the Basin F Wastepile on Figures 3.2-17 and 3.2-18 are based on a qualitative assessment of samples collected from the original wastes.

HUMAN HEALTH RISK CHARACTERIZATION

I. Qualitative Uncertainty Analysis and Implications for Risk Assessment:

Comment 3: The current IEA/RC report persists in emphasizing that many uncertainties associated with its exposure assumptions are, in fact, substantially conservative. The Army states (pages 6-13/14) "....the conservatism in the human health risk estimates must be taken into account when evaluating remedial alternatives. Care should be taken in defining remedial priorities among sites on areas with HIs that vary only slightly from the reference level of 1.0 (e.g., HI<10) for potential risk. Moreover, when considering the remediation of sites that pose a cancer risk, it would be fully justifiable, in light of the known conservatism of the baseline cancer risk estimates, to achieve risk reductions to any level within the EPA target range of 10^{-6} to 10^{-4} lifetime excess cancer risk".

The State emphasizes that three major flaws are thus introduced into the report:

(a) premature significant risk management decisions (for details see State's General Comment 3, IEA/RC, March,1994).

(b) violation of the National Contingency Plan (NCP), which mandates that "a cumulative risk level of 10^{-6} is used as the initial protectiveness goal...." (for details see State's HHRC Comment 3, IEA/RC, March,1994).

(c) claim of conservatism in risk assessment based upon speculations rather than any conclusive data. In fact, the State maintains that there are significant areas of underestimation as outlined below.

Response: a) Inclusion of the risk management perspective should not be considered a risk management decision. Rather, it is included in this section to provide a context within which human health (and ecological) risks can be interpreted. The text does not imply that a remedial decision has been made. However, in response to the State's comment, paragraph 3 on page 6-14 will be removed from the next version of the IEA/RC.

b) The referenced text is in compliance with the National Contingency Plan (NCP), which states, "EPA's risk range of 10^{-4} to 10^{-6} represents EPA's opinion on what are generally acceptable levels...Preliminary remediation goals for carcinogens are set at a 10^{-6} excess cancer risk as a point of departure, but may be revised to a different risk level within the acceptable risk range based on the consideration of appropriate factors..." (55 FR 8717). Also, the PPLVs presented in this document are derived using a 10^{-6} risk level as an initial protectiveness goal.

c) One of the largest areas of uncertainty in the risk assessment stems from the toxicity factors used in the evaluation, and substantial data are available to support this statement. Most of the cancer slope factors used in the assessment are 95th percent upper confidence limit of the dose-response curve derived using the EPA's linearized multistage model, which typically results in a conservative estimation. Further, the reference doses upon which the noncarcinogenic PPLVs are based are estimated by EPA by applying conservative "uncertainty factors" to a NOAEL (no observed adverse effects level). Thus, depending upon the chemical, often two to five orders of magnitude of uncertainty can potentially be reflected in the reference dose.

Underestimations in Risk Assessment Claimed by the Army as Sources of Conservatism:

(i) Dose-response models used for carcinogens and noncarcinogens (p.6-11).

Comment 4: The State acknowledges that RfDs and cancer slope factors are major sources of uncertainty in the assessment of risks. In general, the potential of such values to over- or underestimate overall risk cannot be firmly established because of insufficient information and chemical specific variations. However, analysis of the toxicity criteria of major risk drivers, such as aldrin/dieldrin would indicate the trend towards over- or underestimation of overall risk. For instance, the acute toxicity value of aldrin/dieldrin provided by the EPA (1.0E-04 mg/kg/day) is 25 fold higher than the State recommended value (4.0E-06 mg/kg/day); thereby, underestimating risk by a factor of 25 (for details of RfD see State's HHRC Comment 7, Attachment 2, IEA/RC, March,1994).

Thus, the acute PPLV of dieldrin which is the lowest for recreational visitor (3.0 ppm) is underestimated by 25 folds (i.e., new PPLV = 0.12 ppm). Therefore,

any overly conservative RfDs/cancer slope factors for other non-driver COCs cannot be considered significant in relation to 25 fold underestimation of risk posed by aldrin/dieldrin which are the most relevant compounds

Response: The acute/subchronic reference dose for aldrin and dieldrin was developed by toxicologists from EPA's Office of Research and Development specifically for the RMA site. On the basis of the expertise of EPA's scientists, their value is considered to be, at a minimum, protective of human health. Modifying factors were applied in developing the 1 x 10^{-4} mg/kg/day criterion to account for uncertainties in the underlying data.

(ii) Use of probabilistic chronic PPLV approach vs. deterministic RME chronic PPLV approach.

Comment 5: The EPA guidance indicates a preference for the use of deterministic values in evaluating risk to achieve a goal of streamlined assessment (EPA, 1989). Thus, the Army has significantly reduced the built-in conservatism in the RME deterministic approach of the EPA and, in fact, introduced underestimation (up to 100 fold) as demonstrated by the comparison between deterministic and probabilistic chronic PPLVs of aldrin/dieldrin (Table 1).

Table 1. Biological/Industrial worker deterministic carcinogenic chronic PPLV (HHEA, 1992) vs. Industrial/Biological worker probabilistic carcinogenic chronic PPLV (IEA, 1994) of aldrin/dieldrin.

COC	Deterministic PPLV (ppm) Ind/Biol	Probabilistic PPLV Ind/Biol	Underestimation of Probabilistic PPLV Ind/Biol
Aldrin	0.02/ 0.02	3.0/ 0.7	x160/ x40
Dieldrin	0.02/ 0.02	1.4/ 0.4	x70/ x20

Response: The State's assertion that risks may be underestimated up to 100 fold (based on a comparison between deterministic and probabilistic chronic PPLVs for aldrin/dieldrin) is erroneous. This statement assumes that the assumptions used in the deterministic analysis are most valid, an assumption that can not be supported. On the contrary, the probabilistic evaluation is considered to represent a more rigorous scientific and statistical approach to evaluating potential human health risks for this site. Also when estimating a parameter or distribution, incorporation of a value that is less than a more conservative "default" value does not necessarily imply underestimation. Rather, it may reflect an attempt to incorporate more defensible, realistic data into the assessment.

(iii) Non-conservative assumptions are incorporated in the development of key parameters (e.g. dermal RAFs and soil ingestion).

Comment 6: The Army has not provided any evidence to support its conclusion that the uncertainties associated with the key parameters were always addressed in a conservative manner. The State has never agreed with the Army's claim that probabilistic distributions for soil ingestion (for details see HHRC Comment 5, p.3, August 1993 and dermal RAFs (for details see Issue 1, State's comment on Dispute Resolution Agreement, February, 11, 1994) were developed from the best available scientific evidence and judgment as briefly discussed below.

Response: Responses to the State's specific comments regarding the conservativism of the exposure assumptions used in the HHRC are provided below.

Comment 7: Soil Ingestion. Recent developments in assessing soil ingestion in children by Dr. E. J. Calabrese (see Attachment 1) demonstrate that the Army's distributions of soil ingestion are not protective of all children. According to Calabrese's distributions, the median of the estimate daily average soil ingestion values over one year is 75 mg/day and the upper 95% value is 1750 mg/day (vs. 200 mg/day as upper 95% of the Army). Thus potential underestimation of upper 95% value may range from 1 to 8 fold.

Response: The soil ingestion distribution used in the HHRC was developed based on an extensive review of the available data, and has been agreed to by both EPA and Shell. The median value, 50 mg/day, is based on a statistical evaluation of the available soil ingestion literature. The 95th percentile value, 200 mg/day, is recommended as a reasonable maximum exposure parameter for childhood exposures in EPA's Standard Default Exposure Factors guidance document (1991).

The soil ingestion distribution proposed by the State incorporates pica behavior. However, as stated in the HHRC, pica behavior is not considered in the quantitative risk evaluation, an approach that is consistent with that recommended by EPA in the Risk Assessment Guidance for Superfund (1989). Regarding the State's reference to recent developments identified by Dr. Calabrese (1989), these findings are not conclusive, have yet to be peer reviewed, and thus will not be incorporated in the HHRC.

Comment 8: Dermal RAFs of driver COCs aldrin/dieldrin. The State has already provided a critical assessment of the Army's development of dermal RAFs for aldrin/dieldrin (see State's Comment on Shell's Dispute dated February 11, 1994). State's analysis shows that considerable uncertainty surrounds the Army's estimates of dermal RAFs for aldrin/dieldrin and results in the probable underestimation of risk. We believe that the distribution for aldrin should be 0.005 to 0.5 (vs. 0.0006 to 0.0052 according to the Army), and for dieldrin

should be 0.005 to 0.8 (vs. 0.012 to 0.01 according to the Army). Alternatively, the State suggested the use of EPA's default value of 0.10. Thus, in comparison to the State values, the Army's upperbound values are 80 - 100 fold too low, and according to the EPA default value, the Army's upperbounds are 10-20 fold underestimated.

Response: The dermal RAFs for aldrin and dieldrin were developed after extensive review of all available data (as documented in Appendix B.3.3), and have been endorsed by both EPA and Shell. Regarding the State's comparison to default values, EPA recommends use of the upperbound default value only when chemical-specific information is not available (which is not the case for this analysis). Again, the Army disagrees with the State's apparent assumption that an upperbound default value is most valid or appropriate for use in risk assessment.

(iv) Risks from non-COCs (i.e.,tentatively identified and unidentified chemicals).

Comment 9: For details see State's General Comment 7, IEA/RC, March, 1994.

Response: The approach used by the Army is consistent with EPA guidance and typical risk assessment approaches. A TIC's quantitation is always considered inaccurate and its assigned identity may also be inaccurate. The 27 COCs quantitatively evaluated in the HHRC are likely to contribute most to the risks at RMA. These COCs were selected as a subset of all chemicals potentially present at the site to streamline the risk assessment and focus the analysis on the chemicals of most concern.

(v) The use and application of vapor-inhalation modeling.

Comment 10: For details see State's HHRC Comments 17, and 37 to 42, IEA/RC, March, 1994.

Response: The State's specific comments concerning the HHRC vapor modeling are addressed below in the responses to Comment 15 through 18.

To summarize these major underestimations for the most conservative deterministic and probabilistic PPLVs:

Comment 11: Deterministic acute PPLV for recreational visitor (3.0 ppm for Aldrin). Sources of Major Underestimations: Acute RfD (25 fold), dermal RAF (up to 20 fold based on EPA's default values), soil ingestion by children including Pica behavior (up to 8 fold).

Response: The Army maintains that the deterministic acute PPLV for the recreational visitor is conservative on the basis of the assumptions employed and the use of the EPA-derived toxicity factors. Furthermore, both EPA and Shell have

endorsed the underlying RfD and exposure assumptions that were used to calculate this value.

Comment 12: Probabilistic carcinogenic chronic PPLV for biological worker (0.7 ppm for Aldrin). Overall underestimation, including dermal RAF, due to probabilistic approach would be up to 40 fold.

Response: The Army maintains that the chronic deterministic carcinogenic PPLV for the biological worker is health-protective, given the reasons stated in the responses to Comments 5, 7, and 8 (above).

II. Failure to Acknowledge Unquantified Risks: Premature Risk Management Decisions:

Comment 13: The State disagrees with the IEA/RC's pervasive failure to quantify many potential site risks, and, at the same time, its continued incorporation of premature risk management decisions that curtail the risk evaluation. This fundamental defect stems in part from the Army's inappropriate reliance on land use restrictions contained in the RMA Federal Facility Agreement. The IEA should acknowledge potential unquantified risks so that these can be fully considered by the risk manager during the feasibility study process, where risk management decisions are properly made. Because these potential risks would likely be present but for specific restrictions, and are not merely speculative, they should be identified so that the risk manager is aware that, in the event the restrictions are removed, these risks will need to be assessed.

The State's dispute covers each of the specific examples outlined below. None of these issues is new; the State refers the Army to its previous comments on the IEA/RC for in-depth discussion.

Response: The HHRC quantifies potential risks associated with all anticipated future land uses at the Arsenal. However, given the land use restrictions outlined in the RMA Federal Facility Agreement (FFA), surface water and groundwater pathways were excluded from quantitative evaluation. As with any risk assessment, if the expected land use were to change, potential exposure scenarios (and corresponding risks) would be reassessed.

A. Groundwater:

Comment 14: 1. Exclusion of Groundwater Exposure Pathway. The FFA prohibits potable use of groundwater on RMA; it does not prohibit non-potable uses and activities which may result in vapor inhalation, incidental ingestion, and dermal contact. Such pathways are reasonably foreseeable and must be acknowledged by the IEA/RC, and presented to the FS working-group. See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 4.

The Army's response to this comment acknowledges that, since only a few such pathways may exist, they were not quantified, and that additional restrictions are within the discretion of the U.S. to impose as necessary. However, the U.S. will be unable to determine the necessity of restrictions if the risks are not presented, at least qualitatively, in the IEA/RC.

Corollary to this problem is the Army's reliance on premature use restrictions to ignore other exposure pathways such as ingestion of water; this approach contravenes CERCLA, the NCP, EPA Risk Assessment Guidance for Superfund. The FFA restrictions on potable uses of RMA groundwater have been relied upon in the IEA to exclude characterization of risks from inorganic constituents in the groundwater, despite sometimes high levels of such compounds in the aquifers. See State Comments on the August, 1993 IEA/RC, General Comment No. 11. Similarly, risks from n-nitrosodimethylamine (NDMA) in groundwater have not been quantified in the IEA/RC, though the compound is present in groundwater at RMA and is toxic to humans at 1.4 parts per trillion. Nor are risks associated with the organic contaminants presented in the report. The failure to include at least a qualitative discussion of these risks contravenes EPA's statements during resolution regarding the Human Health Exposure Assessment. See letter of May 15, 1989 from EPA Region VIII Administrator James J. Scherer to Lewis D. Walker and Gary Dillard, State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 4, and State Comments on the August, 1993 IEA/RC, General Comment No. 13.

Response: As stated above, a groundwater exposure pathway was not quantitatively evaluated because potable uses of this medium are not anticipated (given the land use restrictions stated in the FFA).

B. Vapor Exposures:

Comment 15: 1. Groundwater Vapor. A potential exposure pathway exists, after soil remediation from contaminated groundwater in sites where soil contamination saturates the soil gas. Without acknowledgement of this risk, the risk manager will not have sufficient information upon which to base ultimate decisions which must be protective of public health. See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 23.

Response: As noted in the Army's response to the referenced comment on the August 1993 IEA/RC, both the HHEA and the IEA/RC quantify the potential exposure due to existing contamination conditions. Estimating exposure during or after remediation is beyond the scope of the IEA/RC and will not be attempted in the IEA/RC.

Comment 16: 2. Enclosed Space Vapor Inhalation. The IEA fails to consider risks from

indoor vapor inhalation exposure for structures without basements- See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 17. The Army correctly notes that consideration of basement exposure might be more conservative; however, this response ignores the fact that, for many areas where vapor exposure may occur in above-grade structures, basement vapor exposure was not quantified. Even if basements are not constructed in these areas, risk from abovegrade vapor exposure potentially may occur and should be addressed, at least qualitatively, in the IEA/RC.

Additionally, the Army's enclosed space vapor inhalation model is applied to sites where non-aqueous phase liquids (NAPLs) are present in soils, not just in groundwater. See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 17. The presence of NAPLs entrained in unsaturated soils could lead to a significant underestimation of contaminated vapor flux, and therefore underestimate the risk from exposure to NAPL vapors. This is especially a problem given the general fall of groundwater levels over the last ten years. The IEA should, at least qualitatively, acknowledge these potential risks.

Response: A brief qualitative discussion of the potential for: 1) enclosed-space vapor inhalation in above-grade structures; and 2) presence of nonaqueous phase liquids in RMA soils to contribute to uncertainty in vapor inhalation model results will be added to Section E.7.3 ("Vapor Model Uncertainty").

Comment 17: 3. Time per Basement Air Change. The State maintains its objection that the rate of basement ventilation flow (Qa) distribution is unrealistic and not conservative. See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 22. While it is true, as the Army responds, that the selection of a 23 second air exchange from the distribution is unlikely, it is also true that, even for the smallest basement, 23 seconds is unrealistic. The Army's distributions should be limited to realistic values.

Response: As noted in the Army's response to the referenced State comment on the August 1993 IEA/RC, the Army acknowledges that the model allows a very small probability of combining the largest flow rate with the smallest basement volume. However, this highly infrequent combination does not significantly affect the model results. There is no need to revise the model.

Comment 18: 4. Additivity of Vapor Exposures. Many contaminated sites at RMA are contiguous. However, exposures to the simultaneous vapor emissions from several sites are omitted from the risk evaluation. See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 24. These risks should be recognized, at least qualitatively, in the IEA.

Response: As noted in earlier responses to this comment, the Army still considers the site-

by-site evaluation presented in the report to be a reasonable and conservatively biased first approximation to the vapor inhalation exposure risk. It is still the Army's position that no further analysis is appropriate at this point, in view of the following: 1) it is unlikely that structures with basements will be constructed at RMA, and 2) exposure via the open space vapor inhalation pathway does not contribute significantly to the total risk.

Comment 19: C. Surface Water and Sediment. Human exposures to both surface water and sediments are not addressed by the IEA. Fishing and swimming in particular are activities that would expose people to both contaminated sediment and surface water, through vapor inhalation, dermal absorption, or ingestion (intentional or not). See State Comments on the August, 1993 IEA/RC, General HHRC Comment Nos. 25 and 26. While the State disagrees with the exposure parameters chosen for the HHEA as being underconservative, the Army had nonetheless identified quantifiable risks in that document. The Army's response to Comment 26, that the Army could place additional restrictions on uses of surface water should such restrictions be deemed necessary, ignores the fact that without at least a qualitative discussion in the IEA/RC the necessity of such restrictions cannot be determined. The text should recognize that potential risks from these media are omitted from the quantitative evaluation and include them at least qualitatively.

Response: The Army considers the previous evaluation of exposure to surface water contamination by wading fishermen to be sufficient. This potential exposure route was examined in the HHEA (Appendix A, September 1990) and found to be insignificant. The estimated cancer risk was 1×10^{-7} and the hazard index was 0.023.

Although swimming is not explicitly prohibited by the FFA, potential exposures associated with this activity do not warrant quantitative evaluation because such exposures are likely to be very rare, and contamination of surface water at the Arsenal is not evident.

Comment 20: D. Consumption Pathways. The Army's reliance on FFA land use restrictions which preclude onpost hunting does not prevent consumption of migratory RMA animals by offpost residents. See State Comments on the August, 1993 IEA/RC, General HHRC Comment No. 26. The Army's response, that tissue levels found in onpost animals may be the result of offpost exposure, sidesteps the issue of addressing these risks based upon tissue levels calculated from exposure to RMA media. The IEA should present sufficient information to the risk manager, such as a comparison of MATtissueCs to FDA maximum allowable values, to ensure that remedies ultimately selected will not result in tissue concentrations in migrating wildlife that, if consumed, could pose a threat to the consumer.

Response: A consumption pathway was not evaluated because the FFA specifically prohibits hunting and fishing (for consumption) activities at the Arsenal. The evaluation of potential risks associated with consumption of contaminated fish and game caught in offpost areas is not within the scope of the onpost IEA/RC.

Comment 21: E. Contaminant Fate and Transport. In response to State Comment No. 14, the Army agreed to replace the term "disappearance" with either "fate and transport" or "migration and fate"; however, the changes have not been made in the document (see, for example, the title of Section E.3.3, "CONSIDERATIONS FOR CONTAMINANT DISAPPEARANCE AT RMA" and the frequent use of the term "disappearance" in that section). Please revise the text accordingly.

Response: The term "disappearance" used in Section E.3 will be replaced with "fate and attenuation" in the next version of the IEA/RC. These changes were inadvertently not included in the March 1994 version of the IEA/RC.

III. Designation of No-Action Sites:

Comment 22: The Army has included a designation of "no-action sites" in this document, despite the State's repeated objections. Such designations are inappropriate prior to risk management analysis and should be explained or removed. The State has not concurred with these designations.

Response: "No-action site" is a term defined as part of the feasibility study (FS) process; it is used in the IEA/RC only when referring explicitly to designations assigned by the FS, as in the qualitative risk assessment (Section 3.3). These references are made solely to facilitate review and provide a link to the FS. The use of this term in the IEA/RC does not connote a risk management decision.

ECOLOGICAL RISK CHARACTERIZATION

I. Underestimation of Ecological Risks:

Comment 23: Repeatedly in the IEA/RC, the Army asserts that it has made conservative assumptions in the ecological risk assessment in order to ensure protectiveness. For instance, in Section E.11, ECOLOGICAL HEALTH: UNCERTAINTIES ASSOCIATED WITH EXPOSURE PARAMETERS, Subsection E.11.6 INTENTIONAL AND UNINTENTIONAL BIASES, the Army states:

> [t]o avoid potential underestimation of risk, the goal of protectiveness required that uncertainty about risk be countered with a conservative bias in risk estimation. For influential parameters in the model, the greater the uncertainty, the higher the degree of conservative bias that may be warranted.

However, the IEA/RC contains numerous instances where the Army has failed to recognize uncertainty and, therefore, failed to ensure protectiveness. While there may be considerable uncertainty and little data available, the following examples should be addressed, at least qualitatively, in the IEA/RC.

Response: Prior to addressing the State's specific comments regarding underestimation of ecological risks, a general response to the opening paragraph of the State's comment is in order. The Army considers the State's basic premise that "the IEA/RC contains numerous instances where the Army has failed to recognize uncertainty, and therefore, failed to ensure protectiveness" to be incorrect and specific comments that rely on this premise to be founded on faulty reasoning. However, the Army is aware that this is not the sole basis for the State's specific comments, and has attempted to address the State's other concerns in responding to specific comments regarding underestimation of ecological risks.

The State's general comment suggests that uncertainty about a parameter of interest (e.g., the hazard index) should be estimated by breaking it down into contributing factors, generating an uncertainty estimate for each, and recombining the contributing factors to estimate uncertainty about the parameter of interest. This type of uncertainty analysis, referred to here as "bottom up" analysis, has five basic drawbacks.

First, in some cases it is more difficult to estimate uncertainty about contributing factors than it is to estimate uncertainty about the parameter of interest itself, in which case uncertainty estimates based on the contributing factors are less reliable than uncertainty estimates based on direct measurements. For example, the contaminant concentration in the tissue of a fish can be described in terms of a set of contributing factors including concentrations in various environmental media (e.g., sediment, suspended particulates, pore water, water column, and prey tissues), mass transfer, kinetic, and thermodynamic coefficients, and knowledge of the fish's behavior and physiology—or the tissue concentration itself can be measured. The reliability of the direct measurement is not governed by the uncertainty in the estimates of the contributing factors. Therefore, one can not necessarily argue that failure to recognize uncertainty (for example, about dissolved contaminant concentration in the water column) leads to failure to ensure protectiveness.

Second, bottom up analysis of uncertainty creates its own uncertainty, because the relationship between the contributing factors and the parameter of interest (i.e., model uncertainty) generally is not precisely known. Using the previous example, one might be uncertain about the form of a dieldrin adsorption isotherm, or the kinetics of mercury speciation in detritus, but relatively certain about the average concentrations of dieldrin or mercury in a fish's tissues.

Again, failure to understand or characterize uncertainty does not necessarily imply uncertainty about risk or failure to ensure protectiveness.

Third, uncertainty about a parameter of interest may be low, even when uncertainty about the contributing factors is high, due to the nature of the relationship between the contributing factors and the parameter of interest. For example, uncertainty about an average soil concentration over an area (the parameter of interest) may be low even if uncertainty about soil concentrations at specific locations within the area (the contributing factors) is high. Therefore, when risk is related to average exposure, uncertainty about risk may be low, in which case failure to characterize uncertainty about local soil concentrations does not necessarily imply uncertainty about risk, or failure to ensure protectiveness.

The fourth drawback of bottom up uncertainty analysis is that it is inherently open-ended. A parameter of interest can always be broken down into more or different components, so it is always possible to argue that the analysis is incomplete. Moreover, the parameter can always be related to contributing factors that are poorly understood and difficult to measure. Consequently, if one is not mindful of these drawbacks, it can always be argued not only that an uncertainty analysis is incomplete, but also that uncertainty is large. Fifth, if contributing factor uncertainty is recommended and used without a "reality check" on whether the resultant parameter of interest value is meaningful the results may be nonsensical. Therefore, the State's comment that "the IEA/RC contains numerous instances where the Army has failed to recognize uncertainty, and therefore, failed to ensure protectiveness" is false and has biased the State's review of the ecological risk characterization.

A. Data Gaps: Unquantified Risks:

Comment 24: 1. Mammalian Carnivores. The IEA food-web does not consider mammalian predators, despite indications that these animals may be more susceptible to contaminants than other species. See State Comments on the August, 1993 IEA/RC, General ERC Comment No. 34. It is noteworthy that in this instance, in particular, the Army does not incorporate observations of RMA animals, e.g., badger fortuitous sample and tissue data. The Army's failure to quantify potential risks to carnivorous mammals should be acknowledged in the text.

Response: The Army did not "fail" to quantify potential risks to carnivorous mammals; COC doses to and body burdens in carnivorous mammals were not selected as measurement endpoints for the RMA ecological risk characterization. The rationale for these statements is documented in the Army's Response to State Comment 34, Proposed Final IEA/RC, Version 3.1.

In addition, data on fortuitous samples are available in several locations. The Biota RI contains analytical data on fortuitous samples, including badgers and raptors, as well as necropsy data for raptors. These data were collected to help define the nature and extent of contamination, as well as to document adverse effects of contamination. Three years of analytical data on fortuitous species were collected by the Biota CMP. Descriptive information associated with specimens collected and in some cases necropsied is tabulated in Appendix C.5. Tables from both these studies are provided as Attachment C.5-2 in the IEA/RC. Finally, Attachment C.5-3 provides information on fortuitous samples collected by the U.S. Fish and Wildlife Service between 1990 and 1993. It is not within the scope of the ERC to rewrite the documents from other programs. The IEA/RC only reports their findings and provides citations as necessary.

Comment 25: 2. BCF Values for Birds. The IEA bases avian biomagnification factors (BMF) on bioaccumulation factors (BAF). However, bioconcentration factors (BCF) are also significant for birds. See State Comments on the August, 1993 IEA/RC, Specific ERC Comment No. 52. Waterfowl BCFs have been shown to be relatively high. This unquantified risk factor should be discussed, at least qualitatively, in the text.

Response: Water bird risk estimates are based on water bird tissue concentration data, not on water concentration data. Because the water bird risk estimates were not based on water concentration data, the waterfowl BCFs are not "risk factors" in the IEA/RC; they have zero impact on the water bird risk estimates. Therefore, the text revisions that were responsive to the State's prior comments on this issue (Army Response to State Comment 50, Proposed Final IEA/RC, Version 3.1) are no longer included in Appendix C.1, which has been extensively revised. Please note the discussion pertaining to waterfowl in Appendix Section C.1.4.2.

Comment 26: 3. Excluded Pathways. Exposure pathways excluded from the ecological risk assessment include dermal absorption from soil and vapor, particulate inhalation, and ingestion of surface water by terrestrial receptors. Despite the Army's assertions to the contrary, the State believes that these pathways are not covered by model calibration because the field data are inappropriate for calibration purposes. The IEA should explain that the model may underestimate overall risks because it does not include the contribution of these pathways.

Response: With the exception of the kestrel, owl, heron, and eagle trophic boxes, for which tissue concentration data were not collected, the tissue-based risk estimates derived by the Army do not exclude or favor any exposure pathway, nor utilize or depend on any exposure pathway model. Risks are estimated using an empirical relationship between estimated exposure area soil concentrations and observed tissue concentrations for terrestrial biota, and from tissue concentration

data for water birds. The tissue concentration data reflect total exposure, without regard to exposure pathway. Dose-based risk estimates do assume that ingestion of contaminated food is the dominant pathway of exposure.

For the kestrel, owl, heron, and eagle trophic boxes, risk estimates are derived as a function of the estimated average tissue concentration in the predator population's prey. In these cases, the risk estimates may favor a particular exposure pathway if the tissue-based approach is used to estimate risk and the relative importances of the exposure pathways were significantly different at RMA than in the studies (reported in the scientific literature) used to derive bioaccumulation factors. For example, if dermal adsorption of endrin were an important exposure pathway for owl at RMA, but not an important exposure pathway in the literature studies used to derive the BAF, then the owl/endrin risk estimate would be expected to be underestimated because the contribution of dermally adsorbed endrin to the RMA owl's risk was underestimated. This particular source of uncertainty about the BAF (uncertainty about the representativeness of the relative importance of exposure pathways in literature studies) is very likely to be negligibly small in comparison to overall BAF uncertainty, especially if, as the Army believes, prey consumption is the dominant pathway of top predators' exposure to bioaccumulative COCs.

Comment 27: 4. Failure to characterize acute exposure. The IEA/RC does not characterize acute exposure of biota resulting from short-term exposure to abiotic media, such as soil hot-spots, and prey located in highly contaminated areas. Rather, exposures are averaged over a home-range. This is particularly of concern with regard to federally protected species, which conceivably could be adversely affected as a result of such short-term exposures.

Response: The ecological risk characterization is based on chronic exposure and effects considerations. Chronic effects occur at lower exposure levels than acute effects. Therefore, risk management based on characterization of chronic risk will protect against the effects of acute exposure, and characterization of acute exposure will not yield additional protection.

Comment 28: 5. Ephemeral Waters. Sediments and soils contribute contamination to ephemeral waters through surface run-off; this potential risk has not been evaluated. Additionally, ephemeral waters may pose risks to biota during the time when the waters exist. (For example, some amphibians strictly use ephemeral surface waters for breeding. Birds and other animals may also ingest the water.) The IEA should at least acknowledge these omissions so the risk manager is aware of this problem.

Response: Exposure to ephemeral waters contributes to the RMA biota tissue concentration data used in the ecological risk characterization, and so is accounted for in the

ERC risk estimates. Further, many species at RMA are adapted to arid environments and drink minimal amounts of water. Moreover, ephemeral water concentrations are related to soil concentrations, so remediation of contaminated soils based on ERC risk estimates will reduce the risk from exposure to ephemeral waters. Consequently, the Army does not believe that an explicit characterization of risks from exposure to ephemeral waters will add any risk management value to the ERC.

Comment 29: B. Unprotective Quantified Risk: Failure to use 95th Percentile BMFs to Calculate Risk. As the Army has stated, important parameters require conservative bias in order to ensure the goal of protectiveness. The State maintains that the Army should use the 95th percentile of the BMF values due to significant underestimations and uncertainties in its calculations of risk. See State Comments on the August, 1993 IEA/RC, General ERC Comment No. 27.)

Response: The State argues for use of a 95th percentile BMF "due to significant underestimations and uncertainties in its calculations of risk." The Army believes that its calculations of risk are not significant underestimations, as evidenced by the absence of systematic error in the Army's biota tissue concentration predictions; reasonably conservative bias in the protocols for determining MATC and TRV values; reasonable conservatism in other risk characterization assumptions, including the additivity of COC-specific risks; and ecological health and diversity findings. Moreover, the Army reiterates that the State's review of the RMA ecological risk characterization has been based on a false premise that has created a bias toward overestimating uncertainty in the ERC results (see Response to Comment 23). Therefore, the Army maintains that the State has not provided a legitimate technical basis for using a 95th percentile BMF.

Additionally, the Army has noted in its response to the State's Comment 27, Proposed Final IEA/RC, Version 3.1, that, depending on the skewness in the individual variation in BMF, the mean BMF may correspond to a high percentile on the distribution of individual biomagnification factors, thereby being protective of a high proportion of the population.

Comment 30: Specific examples are:

* The selection of a BMF for a trophic box without consideration of whether other species within the trophic box exhibit higher BMFs.

Response: The ERC estimates population mean BMFs for representative species or groups of species for each trophic box. These species were selected because they are common, important food items, or species of special interest for some other reason. To the extent that BMFs are based on measured tissue data, they are

based on data for these representative sampled species and other RMA species could exhibit higher BMFs. To the extent that BMFs are based on parameters quantified from the literature they used the most representative value for the trophic box irrespective of species.

Comment 31: * The complete lack of data supporting the Army's assumptions that the BMFs of species considered in a trophic box are as high as BMFs of all species represented by that trophic box.

Response: The State's implication that species present at RMA are not protected by the mean trophic box BMF is not substantiated by the current data on tissue concentrations and species occurrence. The Army maintains that in the absence of contradictory information, the species selected are appropriate and adequate for the ERC. Note that 226 species of birds alone have been documented on RMA and there are no data in the literature regarding which of these has the highest BMF. Therefore, selection of representative species was mandatory and was based on logical and readily substantiated criteria. Further, the largest exceedence areas on RMA were associated with the top predators' trophic boxes, so risk management actions protective of the top predators would also protect prey trophic box species and individuals with higher BMFs than used in the ERC.

Comment 32: * The failure of the ERC model to recognize life-stages and genders experiencing high BMFs.

Response: The RMA ecological risk characterization is designed to assess population average risks, not risks to sensitive subpopulations, except in the case of the bald eagle. Please see also the Response to Comment 31.

Comment 33: * The unsubstantiated assumption that the tissue data relied upon by the Army represent steady-state.

Response: The ERC does not depend on an assumption that tissue concentration data represent steady state. Tissue concentration data are assumed to be representative of the tissue concentrations in the sampled organisms' trophic box at the locations where the tissue samples were taken.

Comment 34: * Failure to consider synergistic interactions among contaminants.

Response: The Army acknowledges the State's comment that the ERC does not account for potential synergistic effects, but notes that evidence has not been presented that would support a synergistic effects assumption. The following text will be added to the end of Section 4.1.2 of the Final IEA/RC:

"The assumption that the hazards from the different COCs are additive is uncertain. For example, risks may be less than implied by the additivity assumption if the COCs do not induce the same type of effects or do not act by the same mechanism; or more than implied by the additivity assumption if the COCs induce synergistic effects. EPA risk assessment guidance for human health calls for additivity of hazard quotients, but also states that while '...application of the hazard index equation to a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism, although appropriate as a screening-level approach, could overestimate the potential for effects.' The guidance goes on to state that '(i)f the HI is greater than unity as a consequence of summing over several hazard quotients of similar value, it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group' (EPA, RAGS, 1989, Section 8, page 8-15)." The segregation of COCs by effect or mechanism of action to derive separate HIs was not done for the ERC because of limited toxicological data on the COCs for the species or trophic boxes of concern or appropriate surrogate animals. Therefore, the Army considered it prudent to sum the individual HQ values to derive species or trophic box HIs, albeit this process probably results in an overestimation of potential risks.

Although the EPA guidance (RAGS) regarding HIs is not provided for ecological risk characterization, it does not recognize conservatism of the approach the Army has used.

Comment 35: To develop a more protective assessment, the Army should use the 95th percentile BMFs to attempt to compensate in part for these 35 factors. In fact, EPA has determined that protection of indigenous ecological communities necessitates protecting most individuals within sensitive populations. See 59 F.R. 2652-01, Notice of Availability and Request for Comment on Sediment Quality Criteria and Support Documents, Section VI.3. "Level of Protection." According to EPA, this is accomplished by protecting most individuals in the most sensitive genera (those genera in the 95th percentile of those tested). Id. In order to protect communities at RMA, the Army should base its risk assessment on the 95th percentile BMFs of the organisms incorporated into the ERC model.

Response: The State's conclusion that "(i)n order to protect communities at RMA, the Army should base its risk assessment on the 95th percentile BMFs of the organisms incorporated into the ERC model" does not consider the fact that to determine the 95th percentile of the distribution of individual BMFs would require intensive biota sampling that would itself endanger biota communities at RMA. Additionally, it does not consider that conservatism already has been introduced deliberately into the risk calculations through the toxicity threshold

value (TRV and MATC) selection process. Note that the selection of conservative TRVs and MATCs parallels the principle expressed by the State in its citation of 59 F.R. 2652-2701 that the criteria against which predicted levels of exposure are concerned should be conservatively selected. Finally, the Army rejects the State's presumption that publication of a Request for Comment on Sediment Quality Criteria in any way constitutes EPA determination that the RMA ecological risk assessment should be based on 95th percentile BMFs.

II. Exclusion of Dioxins, Furans, and PCBs:

Comment 36: Introduction. Historically, the Rocky Mountain Arsenal (RMA) was the site of manufacture of a wide array of pesticides, herbicides, and other chemical materials during the period of operation. Production activities and the materials associated with production are known to have the potential for formation of other objectionable chemical substances, either as accidental contaminants of feed stocks, or as recombination constituents, by-products, degradation products, or other associated reaction products. Among these objectionable contaminants potentially present as a result of the manufacturing process at RMA are polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans and related congeners.

Historic Sampling and Analytical Results. It has been repeatedly suggested that previous testing of samples from the RMA have [sic] failed to reveal the presence of dioxins, dibenzofurans, and related congeners. See State Comments on the August, 1993 IEA/RC, General ERC Comment No. 13. The present Integrated Endangerment Assessment (IEA, March 1994) indicates that Phase I sampling and analysis detected a number of compounds that could not be clearly identified (Appendix E, Section E.2.3, page E-7). The Report further states that 20 tentatively identified compounds (TICs) were incorporated into the Remedial Investigation (RI) sampling program. Neither dioxins nor dibenzofurans were included in this group of TICs.

Response: Review of the referenced comment—State's General ERC Comment No. 13 on the August, 1993 Proposed Final IEA/RC—does not provide information or insight into the State's assertion that

> "It has been repeatedly suggested that previous testing of samples from the RMA have [sic] failed to reveal the presence of dioxins, dibenzofurans, and related congeners."

The referenced comment does mention gaps in the characterization of risk from contaminants such as (among others) chlorodibenzo-p-dioxins. It is true that no TICs were specifically labeled as 'dioxins' nor included the term 'dioxin' in a compound name; however, dibenzofuran and octachlorodibenzofuran are

included in Table RISR A2.1-6, "Summary of Tentatively Identified Compounds in Phase I and Phase II Soil Samples", as TICs from the Phase I and/or Phase II soil samples (RISR, p. A2-36; EBASCO, 1992). It is significant that, while these compounds do have moderate to high carcinogenicity and/or toxicity, they did not satisfy the other criteria used as the basis for upgrading nontarget compounds to target status (RISR, P. A2-32; EBASCO, 1992). That is, they were not confidently identified according to analytical technique, they were not clearly related to RMA activities or fuel components, their frequency of occurrence and concentrations were low as evidenced by the rarity with which they were identified in the nontarget fraction of the analytical results (15 occurrences of TICs with the term "...furan..." anywhere in the assigned name, out of 18,184 records for which tentative identifications were made), and there was no apparent relationship or co-occurrence with target analytes.

Please note that the statement quoted by the state from the March 1994 Proposed Final IEA/RC (Appendix E, p. E-7):

> "The 20 tentatively identified compounds were incorporated into the RI sampling program following the contaminant identification phase of the Endangerment Assessment (EBASCO, 1988).",

is incorrect and has been revised in the final IEA/RC to provide the following information. The 20 TICs were included with the target analytes for data presentations, discussions, and evaluations in the SARs and in the RISR, and were included in the source-by-source exposure assessments and the Endangerment Assessment; however, they were not designated as specific target analytes for RI sampling because the RI sampling (Phase I and II) was completed prior to the determination that their occurrences were of sufficient significance to warrant inclusion in the referenced documents. For the reasons outlined above, dioxins and dibenzofurans were not included in this group of TICs.

Comment 37: The initial failure to identify chlorinated dibenzodioxins and dibenzofurans is not at all surprising since the analytical methodologies utilized were intended to assay the samples for volatile and semi-volatile organic compounds. Volume I of the Technical Plan prepared by Environmental Science and Engineering Inc. (1985) indicates that USEPA Methods 624 (waste water), 503 (drinking water) and 8240 (solid wastes) were used for volatile organic compounds, and USEPA Method 8270 (solid wastes) coupled with Method 3540 (extraction and cleanup) were used for semivolatile organic compounds. These methods are all scanning protocols which are inappropriate for identifying dioxins and dibenzofurans. These compounds cannot be routinely observed in scan mode, except at extraordinarily high concentrations. It should be noted that during this period of the sampling program (1986-1988), appropriate methodology for the analysis of

dioxins and dibenzofurans was available and in routine use by a number of laboratories throughout the country. This methodology employed USEPA Method 8280 for dioxin and dibenzofuran analysis. Occasionally, negative ionization mass spectroscopy was also employed for additional sensitivity.

Table 4.2-1 of the Technical Plan (Environmental Science and Engineering, 1985) suggests additional reasons why dioxin or dibenzofuran congeners would not have been observed. This table suggests that the "desired X" or target detection limit was 0.5 µg/g (parts per million) for all compounds of interest. This level is entirely inappropriate for dioxins and dibenzofurans. For example, the maximum concentration of dioxins observed in the soils by USEPA analysts causing evacuation and quarantine of the town of Times Beach, Missouri, was only 1.7 µg/g. Assessment of human and wildlife health impacts is normally undertaken based upon analytical results reported in at least the pg/g (parts per trillion) if not lower. These values are a minimum of six orders of magnitude below the desired analytical levels stated in the ES and E (1985) Technical Plan.

Response: Comment noted. Dioxins and dibenzofurans are not chemicals of concern at RMA and, therefore, not heavily sampled. Please see Responses to Comments 36 and 46.

Comment 38: Given the lack of appropriate analytical protocol for dioxins and dibenzofurans, and the excessive limits of detection the negative results with respect to these compounds were a foregone conclusion. Because the proper analytical question was never addressed, it is clear that the absence of evidence of the existence of these materials cannot be construed as evidence of their absence from the soils and biota of the RMA.

Response: Comment noted. Please see Response to Comment 36.

Comment 39: Contemporary Studies. In November of 1991, EBASCO Environmental Services et al. issued a document entitled, "Proposed Final Remedial Investigation Summary Report". This document contained an appendix designated "Appendix A - Environmental Setting, RI Approach, nature and Extent of Contamination - Text and Tables Version 3.1". This document describes in detail the sampling and analytical protocols and provides an overview of findings.

In this document, EBASCO et al., on behalf of the Army, describes steps utilized to identify and quantify contaminants the RMA (page A2-1):

> In order to manage, evaluate, interpret, and present the array of information collected by the RI in an accessible manner the many hundreds of analytes and their breakdown products that were identifiable by the certified analytical methods used at RMA were organized into

"target" and "nontarget analytes. "Target" analytes are those that were identified in advance of the field investigations, and were deemed to have a high likelihood of being present at RMA based on knowledge of the activities that occurred there ...

The "nontarget" analytes are compounds that were not expected to be present at RMA in large quantities and that were not analyzed for specifically by a U.S. Army Toxic and Hazardous Materials Agency (USATHAMA) certified method. However, some of the analytical methodologies employed for soil, water, liquid, and air samples permitted identification of nontarget analytes. Depending on the analytical method, detected concentration, matrix effects, background noise level, and other factors, nontarget analytes were identified at varying levels of confidence. The nontarget analytes that could be identified were called tentatively identified compounds (TICs).

The document notes that the tentatively identified compounds (TICs) were identified by gas chromatography/mass spectrometry (GC/MS), and that they differ from target analytes only in the degree of certification attached to the qualitative identification (page A2-2). The lack of certification obviated the use of certified reporting limits (CRLs). In this regard, the Army states (page A2-24):

TICs have no CRL because the method used for identification is not USATHAMA-certified. Therefore, the lower limit of detection was assumed to correspond to 10 percent of the internal standard for the GC/MS method used. For the purposes of this report, a value of 0.3 µg/g was used.

Response: There are two important points to be made with respect to this comment: First, the State incorrectly paraphrases the RISR text from page A2-2. Page A2-2 of the Final RISR (EBASCO, 1992) does not say that TICs differ from the target analytes only in the degree of certification attached to the qualitative identification. The text does say "The TICs differ from the target analytes in the degree of certification attached to the qualitative identification.", and cites four different references for detailed discussions of this fact. In addition, the text in the same paragraph containing the quoted sentence reads: "Many of the TICs are uniquely identified; others are only generally described. *All TICS are less confidently identified* than are the target analytes and comprise a subset of the nontarget analytes." (emphasis added); Second, the State appears to be attaching undue significance to the fact that the value of 0.3 µg/g was used as a "lower limit of detection" for TICs for the purposes of the RISR. The purpose of the RISR was to present a summary of the findings of the RI. In order to accomplish that objective, and in consistency with the approach taken previously

in the SARs, 0.3 µg/g was used as a generalized value reflecting a conservative estimation of the minimum concentration at which an attempt to both tentatively identify and quantify an unknown compound was justified. As has been explained elsewhere in the RMA literature (for example, "Draft Final Phase I - Introduction to the Contamination Assessment Reports", ESE and EBASCO, 1987; "Proposed Final RMA Chemical Index", Vol. II, Appendix C, EBASCO, 1988), unknowns were tentatively identified if their GC peak had an area response greater than 10 percent of the response for the internal standard for that particular GC/MS method. Because the standards varied by method, and because of the variations between the different laboratories used throughout the RI, any attempt to apply specific values reflecting the "lower limit of detection" to each TIC reported would have been exceedingly difficult, time consuming, and more importantly, misleading by encouraging precisely the kind of reasoning in which the State is presently engaged, namely in implying a degree of certainty and precision to the detection of TICs which is inappropriate and unwarranted.

Comment 40: The initial Phase I efforts of the Remedial Investigation were supplemented by Phase II which was designed to estimate the aerial [sic] and vertical extent of contamination initially identified in Phase I (page A2-25). The results of the two-phased sampling and analytical approach are presented in Table A2.1-6 (page A2-3 through A2-40). This table, entitled, "Summary of Tentatively Identified Compounds in Phase I and Phase II Soils Samples" positively confirms the identification of both dibenzofuran (page A2-36) and octachlorodibenzofuran (page A2-34) in the soils of the RMA. These findings are remarkable since the presumed detection limit was a value of 0.3 ppm. Because of the toxicity of many of the furan isomers, positive identification of dibenzofurans in the low parts per million level is quite disturbing.

As noted previously, levels of concern for effects on humans and wildlife from dioxins are typically encountered in the parts per trillion range. However, since appropriate analytical methodologies for the identification of these compounds were not employed, and since the detection limits were some six orders of magnitude or more above the usual levels of investigation for these compounds, there is no evidence to suggest that these materials do not exist at the RMA. To the contrary, there is a variety of analytical and historical use information available that suggest it is likely that dioxins are present in RMA soils as a result of manufacturing activities.

Response: It would be helpful if the State were more specific in identifying which congeners they are concerned about rather than making global statements. If the State's primary or sole concern is about 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), then it should clearly state so and focus the discussion on TCDD. There is much informatior documenting the toxicity of TCDD to both humans

and animals. The State fails to acknowledge that there is a wide range in toxicity for the various dioxin and dibenzofuran congeners. Some congeners may be toxic at the parts per trillion (ppt) level and others at the parts per million (ppm) level. There is also great variability in species sensitivity for the individual congener TCDD (a range of 0.6 ppb for the guinea pig to 5051 ppb for the hamster, with some aquatic species sensitive to concentrations in the low ppt). In addition to TCDD, 1,2,3,7,8-pentachlorodibenzo-p-dioxin and 1,2,3,4,7,8-hexachlorodienzo-p-dioxin appear to be the more toxic congeners with the other congeners being less toxic (2,8-dichlorodibenzo-p-dioxin's toxicity to the more sensitive guinea pig is >300,000 µg/kg (ppb)) (Eisler, 1986; Kociba and Schwetz, 1982; Masuda et al., 1987). These distinctions in toxicity are important because the State implies that all are as toxic as TCDD and this is not true. Because TCDD has been designated by EPA as a possible human carcinogen (a designation extensively debated by toxicologists), a slope factor is available for human health risk assessment; however, specific slope factors are not available for the other congeners. EPA assumes other congeners may be carcinogenic; therefore, it recommends using toxicity equivalency factors (TEFs) to convert the concentration of any dioxin or dibenzofuran concentrations to an equivalent concentration of TCDD. This approach for ecological risk assessment is not pertinent because carcinogenicity is not appropriate endpoint for assessing potential impacts to ecological populations.

Assuming the State is primarily concerned about TCDD, the most toxic of the dioxin and dibenzofuran congeners, it is highly unlikely that there are concentrations high enough on RMA to be of ecological significance (irrespective of whether the analytical data are sufficient to support this statement). A long-term study of ecosystem contamination with TCDD at Eglin Air Force Base (Young et al., 1987) showed minimal effect on the biota. The Eglin study area was aerially sprayed with 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,4-dichlorophenoxyacetic acid (2,4-D) herbicides during the period of 1962 through 1970 and the ecosystem study occurred during a 15-year period beginning in 1969. On the basis of soil analyses, the study showed that less than one percent of the approximately 2.8 kg of TCDD applied to the area persisted in the soil environment. The study also reported that the area consisted of a very rich and diverse biota population (approximately 341 species were observed and identified). Some of the species had detectable levels of TCDD in tissue (e.g., cotton rat liver, 10-210 ppt and beachmouse liver, 300-2900 ppt). According to the article, significant concentrations of TCDD have been present in the soils at the Eglin test site for at least 20 years and many generations of animals have existed on the test site. Several field studies have been conducted with the sole purpose of observing the wildlife and searching for dead or dying animals at the Eglin site. The field studies were negative. In trapping programs for animals, gross observations for defects, illnesses, and overall health status were made and nothing out of the "ordinary" (e.g.,

parasites) was observed. Complete necropsies and histopathological examinations were performed on 255 adult or fetal beachmice from the test site and control area. Initially, the tissues were examined on a blind study basis but subsequently reexamined to directly compare test site animals to controls. The test and control mice could not be distinguished histopathologically.

The purpose of reporting the findings of this study is to demonstrate that at a site receiving significant application of TCDD-containing chemicals there was minimal impact to wildlife. The Eglin site most likely contains higher concentrations of dioxins, especially TCDD, than would be expected to occur at RMA from incidental sources.

Although it is theoretically possible for chlorinated dioxins and dibenzofurans to be present at RMA on the basis of past manufacturing practices, it is highly likely that the congeners present would be the less toxic forms and not TCDD. This is because TCDD is primarily associated with specific manufacturing processes, such as for 2,4,5-T (a herbicide) and hexachlorophene (a disinfectant), and not processes associated with the operations at RMA.

The State fails to acknowledge the ubiquitous nature of dioxins and dibenzofurans, particularly in an industrial area such as that surrounding RMA. In fact, dioxin congeners can be found almost anywhere, including the surfaces of charcoal-broiled steaks and homes with fireplaces (Bumb et al. 1980).

Comment 41: Chlorinated dioxins are well-known [sic] as unintended and accidental contaminants of chlorinated benzenes. Table A2.1-6 lists the following chlorinated benzenes as having been positively identified in RMA soils:

1. Chlorobenzene
2. Dichlorobenzene
3. Trichlorobenzene
4. Tetrachlorobenzene
5. Pentachlorobenzene
6. Hexachlorobenzene

In short, all of the possible direct combinations of chlorinated benzene have been identified in RMA soils, with tetra-, penta-, and hexachlorobenzene all indicated by the Army as, "significant former nontarget compounds". In addition, related compounds, e.g., pentachloro (trichloroethenyl) benzene have also been identified. The confirmed presence of this wide array of chlorobenzenes in the soils of RMA suggests the very high possibility of the presence of chlorinated dioxins as co-occurring contaminants.

Further, chlorinated phenols are well recognized as precursor and recombination products leading to chlorinated dioxin formation. While an exhaustive investigation of RMA soil for chlorinated phenol compounds was not undertaken, Table A2.1-6 indicates that at least one of these substances, trichlorophenol was positively identified (page A2-35). The origin of this material is unknown, i.e., as end-use product or raw material manufacture of other substances. However, the presence of chlorinated phenols in RMA soils raises additional concerns for the potential for dioxin contamination particularly when historical utilization of the RMA is examined.

Response: Although the chlorinated benzenes listed by the State have been positively identified in RMA soils, the concentrations are low. If any chlorinated dioxins were formed by these chemicals, the concentration resulting would be very small and unlikely to present a threat to wildlife at RMA.

Comment 42: Historical Use Patterns at RMA. In addition to the chlorination of benzene resulting in the formation of chlorinated dibenzodioxins as unintended by-products, accidental contamination of a number of other products, principally herbicides, is also well known. Dioxins have been associated with 2,4-D, 2,4,5-T and the defoliant Agent Orange. All of these compounds have been documented as having been present on RMA. Further, since the synthesis of DDT requires the use of chlorobenzene, there is a potential for accidental contamination of the raw material with dioxin. Chlorinated benzenes have been documented as being present in RMA soils, and as having been manufactured at the RMA. This same statement can be made for DDT.

Response: It is impossible for TCDD to be formed as an unintended by product of the manufacturing process for 2,4-D; however, other less toxic congeners are possible. TCDD as well as other dioxin congeners may be present in 2,4,5-T and Agent Orange. The fact that these compounds have been documented on RMA is not sufficient justification for further expenditure of tax dollars to look for dioxins, particularly if there is no documentation of a spill. If 2,4,5-T was used for weed control, it is highly likely, based on this limited use, that the concentration applied would be less than that associated with the Eglin Air Force site described in Response 40. Agent Orange was never used at RMA; it was stored in railroad boxcars and never unloaded.

TCDD is unlikely to be found in DDT; however, some of the lesser toxic dioxins are theoretically possible.

Comment 43: According to the 1991 EBASCO document entitled, "Proposed Final Remedial Investigation Summary. Version 3.1", both chlorinated benzenes and DDT were manufactured at RMA. Section 1.3.2 entitled, "Industrial Operations" (page 1-25) states that Colorado Fuel and Iron (CF&I) manufactured chlorinated

benzenes, chlorine, naphthalene, and caustic. The document further notes, CF&I undertook the manufacture of dichlorodiphenyltrichloroethane (DDT) at South Plants between 1946 and 1948. This assertion is supported by the EBASCO (1988) document, "Final Phase I Contamination Assessment Report - Sites 1-13 and 2-18 - South Plants Manufacturing Complex - Shell Chemical Company Spill Sites, Version 3.1." Page eight of this document notes that CF&I manufactured DDT between 1947 and 1948. Kuzneer and Trautmenn (1980) further support this assertion in their "History of Pollution Sources and Hazards at Rocky Mountain Arsenal", page 62, which notes that the chemical division of the Colorado Fuel and Iron Company (CF&I) was a leaseholder on the RMA from 1950-1953. They also maintained a research laboratory in Building B 313. These authors indicate that the products produced by CF&I included chlorobenzene, DDT, naphthalene, chlorine and fused caustic.

Response: Please see responses to Comments 40, 41, and 42.

Comment 44: The 1988 report entitled, "Draft Final Volume II - Structure Profiles - Structures Survey; Version 2.2" discusses a variety of building uses pertinent to the question of the potential for dioxins and dibenzofurans at RMA. Section 2.4b - Buildings 524-571B indicates the following information for specific structures:

Building 532

3.0 History:
The building was an auxiliary facility of the chlorinated paraffin plant. The Army leased this building, along with structures associated with a chlorinated paraffin plant to the Colorado Fuel & Iron Company in 1947 (Pick, 1947; Hastings, 1949).

Building 534A

3.0 History:
Constructed by CF&I, approval was requested and granted in August of 1947 to CF&I as drum storage facility.

Building 544

Table 544-2. Pesticides present in Building 544 area, 1975.

DDT powder	50 pounds
2,4,5-T	25 gallons
2,4,-D & 2,4,5-T	209 gallons

Table 544-3
2,4-D & 2,4,5-T . . . . . 50 gallons

The Ageiss Environmental, Inc. document of 1993 entitled, "Final Summary of Operator Knowledge of Rocky Mountain Arsenal Structures, Version 3.2" provides the following information highlights:

Inventory of Rocky Mountain Arsenal Structures

Building 471
Plant was used to manufacture DDT, chlorinated paraffin ... page 45/135

Building 512
"Unspecified pesticides" ... page 51/135

Building 515
Later building was used to produce DDT, monochlorobenzene ... page 54/135

Building 544
Used to store agent Orange and DDT ... page 69/135

Building 616
Stored DDT ... 74/135

Building 643
Stored DDT ... 82/135

Building 884, 885, and 886 (Igloo Storage)
Stored DDT contaminated materials ... page 110/135

Building 1607, 1608, 1609, 1610, and 1611
Stored DDT contaminated material ... pages 117, 118, 119, and 120/135

This information is further supported by sworn deposition. According to Knaus (1985) U.S. v Shell, No. 83-C-2379, Volume 1, DDT manufacture took place in Building 511 and/or 512. Knaus (1985) further states (Volume 7, page 986), "Chlorobenzene was utilized by CF&I in DDT manufacture and based upon plume concentrations, it appears leakage occurred from a storage tank". Adcock (1985), page 827 further supports the use of chlorobenzenes to produce DDT when he states (Volume 65, page 827) "CFI chlorinated benzene with chlorine then used it in the manufacture of DDT".

Response: Comment noted. Please see responses to Comments 41 and 42.

Comment 45: Polychlorinated Biphenyls (PCBs). The existence of polychlorinated biphenyls (PCBs) and related congeners has been documented in the soils at RMA. The EBASCO (1991) Appendix A of the Remedial Investigation Summary Report indicates that both an octachlorobiphenyl and a tetrachlorobiphenyl have been positively identified at levels greater than 0.3 parts per million (page A2-34). In addition, the related congeners biphenyl, perchlorobiphenyls, and terphenyls have been observed at levels greater than 0.3 ppm (pages A2-34, A2-36, and A2-40). This evidence is of concern, since to date, no effort has been undertaken to specifically analyze for chlorinated biphenyls (209 theoretically possible congeners), dioxins (75 possible congeners) or dibenzofurans (135 possible congeners). All of these substances are related chemically and toxicologically, and are frequently referred to as, "coplanar compounds".

Response: The State is correct in its observation that to date no effort has been made to specifically analyze for chlorinated biphenyls, dioxins, or dibenzofurans. However, the effort to identify unknowns throughout the RI has yielded a body of data which can provided some information, albeit with less confidence than for target analytes, regarding the general likelihood of occurrence of these compounds at RMA. In addition, data collected from the Soil Volume Refinement Program (SVRP) also provides some additional, limited data on the likelihood of these compounds occurring in RMA soils, including surficial soils. The findings of the SVRP will be presented in a final report to be released the end of June 1994. Preliminary data indicate that, of 22 samples collected from 14 surficial soil and two borehole locations and analyzed for eight PCB compounds, there were no detections of PCBs. In addition, pentachlorophenol, which the State noted (letter from Mr. Jeff Edson to Mr. Kevin Blose of PMRMA, June 11, 1992) was a waste product from Shell's manufacturing operations at RMA commonly contains impurities including chlorodibenzo-p-dioxins, was analyzed for in 65 samples, including 14 surficial soil samples. Preliminary data indicate there were no detections of this compound during the SVRP. Shell did not manufacture pentochlorophenol at RMA and, according to Shell, none of their manufacturing processes produced pentochlorophenol as an intermediate.

Comment 46: Summary. Various congeners and isomers of chlorinated dibenzofurans and polychlorinated biphenyls have been positively identified in the soils of RMA at levels above 0.3 parts per million. The chemical utilization and manufacturing processes at RMA suggest the likelihood of additional contamination with chlorinated dioxins. The potential for the occurrence of dioxins in RMA soils is further enhanced by the identification of storage of products known to contain substantial quantities of dioxin and dibenzofuran contaminants, including the herbicides 2,4-D and 2,4,5-T, and the defoliant Agent Orange. These facts, and the knowledge that past manufacturing activities and usage practices at RMA used and produced materials that either potentially contained or could potentially form chlorinated dioxins, dibenzofurans, and polychlorinated biphenyls provide sufficient information to warrant further investigation for the occurrence of these compounds in soils and biota tissue at RMA.

Response: The list of analytes to investigate at RMA was the result of an extensive effort which included review and comments from the parties. This effort produced a set of analyte selection criteria that were carefully followed. Additional sampling for chemicals that theoretically might be present from past RMA activities is not likely to yield useful additional information (i.e., identify new areas of contamination or risk). There is no information on RMA biota that indicated need for such additional sampling. The Army does not agree with the State that the past manufacturing and use practices at RMA warrant further investigation of polychlorinated dioxins, dibenzofurans, or biphenyls in soils or biota at RMA. Please see also the Responses to Comments 36 through 45.

III. Uncertainty Factor (UF) Protocol:

Comment 47: The Army has not responded in any meaningful way to the State's concerns regarding the inadequacy of the Army's proposed uncertainty factor (UF) values. It's responses essentially make the following points:

1. The State misplaces reliance on IRIS which pertains to human health, not ecological concerns, and fails to recognize that eco-risk assessments are only intended to protect populations, not individuals.

2. The State has not cited EPA guidance to support its positions.

3. The Army's UFs were based on meetings with the parties.

The State recognizes differences between human health and eco-risk goals and considerations; these differences are reflected in its proposed UFs. For example, its proposal regarding UFs for less than lifetime studies is based on ensuring protection of species through the reproductive phase. Additional examples of such considerations are replete throughout the State's proposal.

Response: The Army is clearly aware of the State's position regarding the setting of uncertainty factor (UF) values for use in ecological risk assessment and disagrees with its applicability at RMA. The UF values established for use in the ecological risk assessment are the product of several RMA EA Technical Subcommittee meetings at which the State was an active participant (the State was not part of the final selection process because it chose not to sign the FFA). The final UFs represent consensus values by the signatory parties. The Army disagrees with the State that the values are inadequate for the protection of biota at RMA. The process used to handle uncertainty about biota criteria (TRVs and MATCs) in the IEA/RC was developed (collectively by all Parties) to provide realistic estimates, without underestimating the criteria (i.e., without assigning criteria values that are too high). The process used to handle uncertainty about exposure attempts to provide unbiased estimates of average doses and tissue concentrations, through a probabilistic treatment of uncertainty and calibration to site-specific tissue concentration data.

In other words, the intent of the procedures for handling uncertainty is to likely under- or overestimate average doses and tissue concentrations equally, but less likely to under- than overestimate TRVs and MATCs. This implies the intent to err on the side of conservatism in the risk estimation methodology applied in the IEA/RC. Whether and to what extent actual risk estimates are conservatively biased can not be fully ascertained; but the question is tautological in the sense that if the bias in the results could be calculated, there would be no need to apply the conservative methodology.

Comment 48: Contrary to the Army's assertions, such differences do not justify the indiscriminate rejection of decades of thought and research upon which the IRIS methodology has been based. If these assumptions are to be abandoned, justification must be provided. The Army has not done so. This is especially true for the Army's unsupported decision to cap the cumulative UF at 400 Although the Army denies relying on risk management to compromise its risk assessment, it essentially acknowledges that its decision to cap was based on risk management considerations, such as detection limits. Its assertion that the cap was to prevent TRVs and MATissueCs from being lower than levels known to produce adverse effects reflects a fundamental lack of understanding of the reality being addressed--namely, that we do not know what that level is. If we did, we would not need UFs!

Response: The Army did not reject decades of thought and research that serves as the basis for the IRIS methodology to derive reference doses for humans. In fact, the Army carefully reviewed the IRIS methodology (which was the initial foundation for the development of uncertainty factors used with critical dose values to derive toxicity reference values [TRVs]) and associated literature. The

information obtained from review of the IRIS methodology was supplemented with information considered to be more pertinent for ecological assessment. All of this information was considered when the Army met with the parties to establish appropriate UF values for MATCs and TRVs. When the final UF values were selected, there was concern expressed (by several parties) that when all the values were multiplied together the total UF would result in a tissue concentration or dose that would be biologically unreasonable, hence the primary reason for the cap. This was not based on risk management considerations as alleged by the State.

The Army understands that the need for UF values is based on what is not known; however, what is known must also be considered. Please consider the following information as examples of the unreasonableness of the UF approach recommended by the State.

The maximum possible UF values using the State's recommended protocol (but not assuming any extrapolation among classes) would be 200,000,000. The comparable value for the Army's protocol without the cap would be 19,500. The UF cap was never invoked in the calculation of MATC value. The lowest final MATC values was 0.01 for mercury which resulted from a critical dose of 0.83 µg/g and a UF of 100. If the maximum UF values had been applied from the State and Army protocols, this final MATC values would have been 0.000000004 µg/g and 0.0000426 µg/g, respectively. If the capped maximum Army UF had been used, this final MATC would have been 0.0021 µg/g. However, the literature values for MATC and reported in previous versions of the IEA/RC range between 0.01 and 0.20 µg/g for background, 0.36 and 45 µg/g for no effects levels, 0.67 to 100 µg/g for levels resulting in signs of neurological effects, 4.8 and 165 µg/g for levels resulting in signs of toxicity, 0.01 to 3.3 µg/g for levels resulting in signs of reproductive effects, and 2.7 to 150 µg/g for levels resulting in lethality. None of the reported values are within three orders of magnitude of the maximum values. All but one of the reported values, even the background values, are an order of magnitude above the final MATC used in the IEA/RC.

In the derivation of TRVs, capped UF values were used only for copper in mammals, chlordane in birds, and DBCP in birds. The lowest TRV was 0.001 for mercury and for one endrin trophic box. This value would have been 0.00000000005 µg/g, 0.000000061 µg/g, and 0.0000025 µg/g for the maximum State, Army and capped Army UF values, respectively. Note that the two reported dose values were a NOAEL of 0.17 and a LOAEL of 0.047. None of these values are within six orders of magnitude of the maximum values. Both of these values are an order of magnitude above the final TRV used in the IEA/RC.

Note also that mercury concentrations in sample analyzed from RMA ranged from 0.0472 µg/g (beetles) to 0.807 µg/g (deer mouse) in live organisms collected intentionally from contaminated areas of RMA and from 0.0561 µg/g (earthworms) to 0.0641 µg/g (deer mouse) in control samples from RMA. Concentrations in fortuitous samples that were found dead from various causes ranges from 0.0477 µg/g (starling) to 0.4 µg/g (mourning dove). Therefore, the excessively low UF values promoted by the State would result in protective levels that are orders of magnitude below what is documented to be harmful, even if they could be measured analytically. The application of scientific judgement is more than warranted in this situation.

Comment 49: The Army has also rejected the State's proposal on the grounds that it is not supported by EPA guidance; such grounds equally justify the rejection of the Army's approach. In fact, as the Army is well aware, there is no specific EPA guidance on methodology to conduct a quantitative ecological risk assessment. The State's approach, however, is more consistent with historical positions taken by the agency. Discussions of such positions are contained in the State's proposed methodology. Given the lack of guidance, the selected approach should be based on the best scientific information available. This is consistent with the State's approach.

Response: Comment noted. The Army believes its approach is based on the best scientific information available and more appropriate for ecological risk assessment where the goal for most species is the protection of populations.

Comment 50: Lastly, as discussed elsewhere, the fact that the other parties agreed to alternate numbers and that the State was present at portions of those meeting, gives the State no comfort that those numbers are scientifically correct or legally supportable.

Response: The State's opinion is noted.

Comment 51: The State's suggestion that an ecosystem UF be incorporated appears to have been ignored. Its criticisms of the sink species approach to deriving protective criteria for bioaccumulative chemicals apparently have not been understood. The concern remains that the Army has not demonstrated that its target species are the most sensitive species currently or potentially located at the RMA. This fact introduces uncertainty regarding the protectiveness of the proposed criteria for other entire populations of animals. This uncertainty should be acknowledged in the report, and justifies conservative approaches toward the derivation of criteria based on the target species.

Response: Please see the Responses to Comments 30, 31, and 48. The Army believes additional uncertainty is unwarranted.

IV. Hazard Indices (HIs) for Biota:

Comment 52: The State appreciates the changes to the IEA/RC by the Army. However, the overall presentation in the report remains biased. Maps showing areas of risk based upon HI exceedances depict exceedances of HIs of 1 and 10. The text of the report states that, because the range of uncertainty regarding HIs spans at least one order of magnitude, some risks may occur at HIs as low as 0.1 while no risk may occur at HIs as high as 10; therefore, an unbiased presentation must include equally prominent representation of areas exceeding HIs of 0.1 or exclusion of the representations of areas exceeding HIs of 10.

Response: The language regarding the interpretation of the hazard quotients (HQs) and hazard indices (HIs) is the result of dispute resolution. The presentation of the risk areas was discussed during an EA Technical Subcommittee meeting held at RMA prior to the release of the Draft Final IEA/RC Version 3.1 report and the State did not register this concern at that time. No change will be made.

Comment 53: A significant omission in the IEA/RC is the failure to present HIs for biota receptors. Risks are illustrated only in terms of areas where a receptor exceeds an HI of 1 or 10, rather than the actual HI values. The State requests that HIs be presented on contour maps for each of the receptors and in tables showing the greatest HI for each receptor, as the Army does for aquatic pathways of the heron, shorebird, bald eagle, and water bird. Inclusion of maps and tables presenting actual HIs are [sic] important not only to understand the relative risks to receptors but also to assist in weighing the benefits of remediation against the harms of habitat destruction.

Response: There are potentially thousands of HI values represented on the contour maps; therefore, the State's request to present the HIs on the maps in unreasonable. However, the Army will be glad to meet with the State and show them how to access the thousands of HI values that are contained in the geographic information system (GIS)-based HQ/HI maps. Risk associated with the aquatic ecosystem could not be mapped; therefore, these risks were presented in tabular format. No change will be made.

V. TRVs (Dose Response) vs. Maximum Allowable Tissue Concentrations (MATissueCs):

Comment 54: The State's historical concerns regarding the use of the MATissueC approach have not been alleviated. This approach is still being used for almost all food webs for endrin, most food webs for DDT/DDE, and a few for aldrin/dieldrin and mercury. In addition, the State did not agree with the criteria suggested for choosing between tissue-based or dose-based studies. Therefore, the State cannot concur in the risk characterizations derived using tissue-based methodology.

Response: The State's comment is noted. MATCs were used only for bioaccumulative COCs and only when they provided a more certain toxicity threshold value. The Army's response to the State's historical concerns regarding the validity of the tissue-based risk characterization approach is provided in Appendix F of the Proposed Final IEA/RC, Version 3.1 (response to State Comment 18 on the Proposed Final IEA/RC, Version 3.0).

VI. Health and Status:

Comment 55: The State maintains its objections to the Army's biased use of mostly inappropriate studies in the Health and Status section. (See State Comments on the August, 1993 IEA/RC, General ERC Comment No. 30 and State ERC Attachment 5.) The State's position is expressed in the EPA evaluation of this section, which is incorporated herein by reference.

It is inappropriate to conclude Section 3.0, CHARACTERIZATION OF ECOLOGICAL RISK, in the EXECUTIVE SUMMARY, with the Army's view of Section C.5, ECOLOGICAL STATUS AND HEALTH. The conclusions which can be drawn about ecological status and health was one of the major disputes among the parties and was only resolved by agreeing to present each of the parties' views on an even playing field. We recommend that the EXECUTIVE SUMMARY be modified to remove the conclusory paragraph in Section 3.0 entirely or include the conclusions of the dissenting parties.

Response: The Army's responses to the State's August 1993 IEA/RC General ERC Comment No. 30 and State ERC Attachment 5 were provided in Appendix F, Volume IV of the March 1994 Proposed Final Integrated Endangerment Assessment/Risk Characterization. The response to Comment 30 can be found on page State-48. Similarly, the responses to the State's ERC Attachment 5 that contains general and specific Comments 69 through 86 can be found on pages State-102 to State-124. We refer the State to these responses, which are still valid. In addition, considerable detail has been added to the version of the IEA/RC currently being finalized. A draft of this version has been circulated among signatories to the Federal Facilities Agreement and as a courtesy, has also been provided to the State. This detail is fully responsive to comments from all the Organizations and the State.

The Army's comment on EPA's position paper included in the IEA/RC resolve EPA's dispute provides our response to EPA's evaluation of this appendix. To the extent this evaluation represents the State's position, the State may read the Army's comments as responsive; they are provided on the next page.

The Army believes it is fully appropriate to include information regarding Ecological Status and Health in the Executive Summary of the IEA/RC. Please

note that although the Army has expended extraordinary effort to produce a document that is acceptable to all the Organizations and the State, the IEA/RC is still authored by the Army. Further, the purpose of an Executive Summary is to provide synoptic information on all of the integral components of the report. The IEA/RC Executive Summary has done just that. There is no obligation to incorporate comments or responses in the Executive Summary, as they are not integral components of the report.

Finally, the Army would like to clarify the parties to the dispute on Ecological Status and Health. Shell initially disputed the absence of information on ecological status and health in the IEA/RC. They provided an early version of this information that they recommended be included in the IEA/RC. That early Shell draft was extensively revised and reworked by representatives from Shell, the Army, and the FWS and become Appendix C.5. In response to comments from the Organizations and the State on the revised version, two subsequent cycles of Appendix C.5 revision were performed. These two revision cycles were, in particular, responsive to EPA's comments on both the appendix and text inclusions regarding Status and Health information. EPA raised their comments to the status of dispute, not because the Army was unresponsive to their requests, but because EPA did not concur with the content of the responses. Thus, three of the four signatories to the Federal Facility Agreement and all of the parties that have had biological field experience on RMA are supportive of the appendix on Status and Health as it is written. The Army's comment on the EPA's position on ecological status and health is provided at the end of Appendix C.5.

VII. State's Position on the Estimation of BMF:

Comment 56: Pursuant to the "GUIDANCE CLARIFICATION FROM RMA COUNCIL", the State is providing its position on the estimation of BMF for inclusion in the final IEA/RC:

> The State of Colorado has reviewed the three approaches for estimating RMA-specific BMFs and strongly believes that EPA's method is the most scientifically defensible. It is the only approach which tests the fundamental hypothesis that the data collected at RMA can be used to relate measured biota-tissue concentrations to the soil concentrations to which the organisms are exposed. The other two methods impose an assumed correlation between soil and tissue concentrations despite the fact that the data show no such correlation. As explained in detail by the Army and EPA, the data-collection programs for soil and biota were not for the specific purpose of estimating contaminant uptake and therefore did not address the many factors which confound this relationship (for example, physiologic differences and specific knowledge about the

organisms' true exposure areas). The second phase of the Supplemental field Program, which at present has not been designed by the parties, would need to specifically address these confounding factors to explain and reduce the current lack of correlation between soil and tissue concentrations.

Response: First, the Army believes that in providing this comment the State misrepresents the RMA Council's Consensus Directive regarding the Approach for IEA/RC Report Finalization, which states that:

> "All three methods of development of the Biomagnification Factor (BMF) (U. S. Army, Shell Oil Company, and U. S. Environmental Protection Agency [EPA]) will be presented equally in the IEA/RC report. There be no preferential slant toward any single method. A range of risks will be characterized based on results of applying each of the three methods."

The State's comment that the EPA's method for estimating RMA-specific BMFs is the most "scientifically defensible" is inappropriate and its content is unsubstantiated. First the State has defined BMF differently than it is defined by the Army in the IEA/RC, so the State is commenting on a parameter that is not used in the ecological risk characterization. The Army defines BMF as an empirical coefficient, calculated by the Army, EPA, or Shell approach, to be used in the model:

$$TC_{pred} = BMF * \langle ESC \rangle \quad (1)$$

where:

TCpred is the predicted population mean tissue concentration at a specific RMA location,

<ESC> is the estimated exposure area soil concentration for the location where the population mean tissue concentration is being predicted, and

BMF is an empirical coefficient.

The State's comment is based on a specific biomagnification factor definition that is different from the BMF definition given above. The State's comment is based on the presumption that BMF is defined as the multiplicative factor by which the concentration of a contaminant in the tissues of an organism exceeds the true temporally and spatially averaged concentration of the contaminant in the soil to which an average individual in a biota population located at a particular RMA location is directly and indirectly (e.g., through its food)

exposed, implying the model:

$$TC_{pred} = \text{"BMF"} * \text{true average exposure soil concentration} \quad (2)$$

The term "BMF" in equation 2 is the term that the State is discussing in its comment; it is not derived or used in the IEA/RC. The State's comment implies that the purpose of bioaccumulation modeling in the ERC is to derive estimates of the terms on the right side of equation (2). The Army maintains that the purpose of bioaccumulation modeling is to derive mean tissue concentration and dose estimates to be compared to MATCs and TRVs in risk calculations. The Army believes, on the basis of comparisons of mean tissue concentration predictions to individual tissue concentration measurement from across RMA, reported in the IEA/RC, that the empirical model described in equation (1) above is more reliable and cost-effective than the theoretical model described in equation (2) for characterizing risks to biota. The Army's method makes full and consistent use of the thousands of RMA data points.

Therefore, the Army is fully confident that its approach to predicting RMA-wide population mean tissue concentrations is technically sound as a risk assessment methodology. The Army's methodology is thoroughly documented in the IEA/RC; to enter into a further discussion of its "defensibility" would be redundant.

This issue is discussed in greater detail in the "Army/EPA Joint Statement on Differences Between the Army and EPA BMF Approaches," which is presented in Appendix C.6.1 of the Final IEA/RC.

Comment 57: It is inappropriate, pursuant to the "GUIDANCE CLARIFICATION FROM RMA COUNCIL," to present only the risk characterization results of one of the party BMF estimation methods in Section 3.0, CHARACTERIZATION OF ECOLOGICAL RISK, in the EXECUTIVE SUMMARY. The BMF method used to characterize risk was one of the major disputes among the parties and was only resolved by agreeing to present each of the parties' own views, as well as the results of the EPA, Army, and Shell methods, on an even playing field. We recommend that the EXECUTIVE SUMMARY be modified to: (1) include summary maps which represents risks pursuant to the Army's, Shell's, and EPA's BMF approaches (for example, Figure 4.5-1), and (2) refer to the Supplemental Field Program as part of the resolution of the BMF dispute to attempt to estimate whether there is a risk to biota within the area of dispute and, if so, to estimate site-specific BMFs for RMA biota.

Response: The results for the three approaches are presented equally and impartially in both the main body of the IEA/RC (specifically, Figures 4.5-2 through 4.5-13 and 6.4-1 through 6.4-3) and in the Appendices (specifically Figures C.3-1

through C.3-85 and C.3-93 through C.3-115). The purpose of the maps included in Executive Summary Section 3.0 (Figures E.S.3 and E.S.4) is to illustrate how ERC results are presented in the report, and to support some qualitative IEA/RC findings that would not change if maps based on the Army or EPA approach were presented instead or in addition; specifically:

1. "based on the Shell approach (used because it is, in this case, the intermediate result relative to areal extent of risk) most of RMA presents a potential risk (HI > 1.0) from the combined COCs to two to four trophic boxes (receptors)" (page ES-5, third paragraph, second sentence); and

2. "one trophic box is almost always at potential risk (HI > 1.0 from aldrin/dieldrin, DDT/DDE, and endrin at any point at RMA," (page ES-5, third paragraph, third sentence).

Figure 5 is not discussed until Executive Summary Section 4.0, so the first sentence of the third paragraph on page ES-5 will be revised to read:

> "The results of the ecological risk assessment are best understood by examining Figures E.S.3 and E.S.4; note that the areas depicted on the maps reflect areas of potential risk and do not represent areas delineating the extent of contamination nor areas requiring cleanup."

Figure E.S.5 is specific to the Army approach, so the last sentence on page ES-7 which reads as follows,

> "Figure 5 depicts a soil remediation scenario that would eliminate the potential risk (i.e., HQ less than or equal to 1.0) to the great horned owl from aldrin/dieldrin"

will be revised to read:

> "Figure E.S.5 depicts a soil remediation scenario, based on the Army approach, that would eliminate the potential risk (i.e., result in HQ less than or equal to 1.0 everywhere at RMA) to the great horned owl from aldrin/dieldrin. Note that the potential remediation area depicted in Figure E.S.5 is not based on total risk, but only on risk to the great horned owl from exposure to aldrin/dieldrin, as predicted by the Army approach. The Army approach is presented because it is, in this case, the intermediate result regarding areal extent of risk."

The area depicted in Figure 5 is for illustrative purposes; it does not represent a prescribed remediation area. This is apparent in the fact that the map is

limited to a single COC and trophic box, whereas actual decisions about remediation areas will have to consider multiple COCs and trophic boxes.

VIII. Risks Based on Ambient Water Quality Criteria:

Comment 58: The IEA/RC fails to characterize risks based on exceedances of State and Federal Ambient Water Quality Criteria. In its responses to comments, as well as in the text of the IEA/RC, the Army states that CRLs were below AWQC. See State Comments on the August, 1993 IEA/RC, General ERC Comment No. 33. However, a review of CRLs utilized by the Army indicate that this is not the case.

Response: Even though some surface water detections do exceed AWQCs, these are a relatively small percentage of most of the sampled locations and many are in ephemeral pools that do not support well developed aquatic communities. The contaminants contributing to this potential risk are from soil. AWQCs may be considered a post remediation criterion to evaluate residual risk.

Comment 59: As Ecological Risk Attachment A shows, for may significant contaminants the CRLs have always been above Ambient Water Quality Criteria. The Army should resample RMA waters and employ EPA Method Detection Limits to determine whether these criteria are exceeded.

Response: The ERC characterizes risks to biota using TRVs and MATCs. Surface water concentrations in RMA lakes are not ERC measurement endpoints. Moreover, the ERC utilizes aquatic biota tissue concentration data as the base on the aquatic portion of the RMA food web model. Therefore the data collection effort proposed by the State would not serve the objectives of the ERC (i.e., they would not change estimates of biota tissue concentrations and doses).

Comment 60: As Ecological Risk Attachment B shows, for Zinc and Copper, water quality criteria have been exceeded. Establishing whether ARARs have been exceeded is the first-step in CERCLA risk assessment. This has not been accomplished at RMA.

Response: The State is incorrect in its assertion that "establishing whether ARARs have been exceeded is the first step in CERCLA risk assessment. This has not been accomplished at RMA." The Army has identified potential ARARs as part of its ongoing Feasibility Study (FS). After ARARs are finalized the Army will determine whether they have been exceeded, again, as part of the FS.

IX. Aquatic Risk Model Parameters:

Comment 61: The State believes that the Army has significantly underestimated risk to biota

from the aquatic portion of the food web. Our concerns are outlined below:

1. A review of Figure D.1-11, the Aquatic Biota Tissue, Sediment, and Water Concentration Database and the underlying data which support it (provided by EBASCO on 3 February 1994 at Seattle) indicates that approximately half (47.4%) of the numbers used to form concentrations of COCs by trophic box are unsupported by data. For example, although values are provided in the tables, there are no data for aquatic invertebrates in East Upper Derby Lake, Upper Derby Lake, Rod and Gun Club Lake, and Lake Mary. The accompanying note on Table D.1-11 states, "Plankton and aquatic plant ratios were used to predict aquatic invertebrate DDE/DDT concentrations." However, there are no plankton data whatsoever for East Upper Derby Lake, Upper Derby Lake, and Rod and Gun Club Lake, and no aquatic plant data for East Upper Derby Lake and Upper Derby Lake.

Please explain how the DDE/DDT values were calculated for plankton in these lakes in the absence of data. Please explain how the values for the other COCs were calculated for aquatic invertebrates in these lakes in the absence of data. The State reserves the right to dispute these values pending receipt of this information.

Response: First, the State's comments that "47.4 % of the numbers used to form concentrations of COCs by trophic box are unsupported by data" is meaningless for evaluating the risk characterization. Aquatic biota tissue concentrations are only used to predict heron and eagle doses, and neither heron nor eagle utilizes aquatic plants, aquatic invertebrates, or plankton as a significant component of its diet. Aquatic invertebrates are estimated to comprise 2.4% by mass of the heron diet and 0.0% of the eagle diet. Aquatic plants and plankton are estimated to comprise 0.0% by mass of the heron and eagle diets. These are conservative estimates in the sense that if the percentages are higher than estimated, the doses to heron and eagle are being overestimated, because the diets of heron and eagle are estimated to be comprised of higher trophic box biota that have higher COC body burdens; adjusting the aquatic plant, aquatic invertebrate, or plankton prey fractions upward would reduce heron and eagle risk estimates. The aquatic biota that contribute significantly to the ecological risk characterization are small and large fish and water birds. Data are available for these trophic boxes for all lakes they inhabited when the data were collected. Also note that both E. Upper Derby and Upper Derby Lakes are currently being managed as dry and inconsistently contained water between 1986 and 1990 when biota samples were collected.

Second, the State's comment ignores the methods that were developed and documented for estimating missing data. These methods are described case-by-case in Appendix Section C.1.4.2 of the Proposed Final IEA/RC, Version 3.1.

In addition, the State has copies of the electronic spreadsheets on which the actual calculations are performed. The specific calculation methods asked for by the State can be found in Appendix Section C.1.4.2, and the Army provided (at the time the electronic spreadsheets were provided to the Parties) a contact person to answer specific questions regarding these calculations. Since February 3, there have been no questions from the OAS regarding these calculations.

It is noted again that estimates of missing aquatic tissue concentration data contribute marginally or not at all to ERC risk estimates. In fact, at the February 3 Seattle meeting referred to in the State's comment, the Army performed a real-time analysis of sensitivity of risk estimates to uncertainty about the missing data algorithms, which demonstrated that heron hazard quotients were unaffected to at least the third significant digit. Therefore, the State's implication that the aquatic portion of the ecological risk characterization is based on numbers that are not supported by RMA data is unfounded.

Comment 62: 2. It appears that the data from the "Estimated TC contribution from the aquatic food web" table (Figure D.1-16) were used to calculate HQ, since:

For Heron Ald/Dld

$$HQ = \frac{\text{Estimated TC}}{\text{MATC}} \text{ or } \frac{1.330}{0.870} = 1.528 \text{ (see Figure D.1-20)}$$

For Heron DDE/DDT

$$HQ = \frac{\text{Estimated TC}}{\text{MATC}} \text{ or } \frac{0.107}{0.150} = 0.713 \text{ (see Figure D.1-20)}$$

However, the origin of the values in the "estimated TC contribution from the aquatic food web" is not evident. The table indicates a value of 1.33 ppm as the estimated TC contribution from aquatic sources to the heron. However, calculation from the actual data from individual lakes to the heron's aldrin/dieldrin TC yield 2.3 ppm for Lower Derby Lake, 1.9 ppm for Lake Ladora, and 0.79 ppm for Lake Mary. The mean of these three data points is 1.66 ppm, and the geometric mean is 1.51 ppm. Neither of these values agree

with the "estimated TC contribution from the aquatic food web". If the mean value of the actual lake data is applied to the HQ calculation:

$$HQ = \frac{\text{Mean Individual TC Estimates}}{MATC} = \frac{1.66}{0.870} = 1.91$$

If the geometric mean is used:

$$HQ = \frac{\text{Geom. Mean Individual TC Estimates}}{MATC} = \frac{1.511}{0.870} = 1.74$$

Both of these values are substantially higher than the HQ derived in the present IEA. Since the individual lake estimates are all higher for heron in each category, the HQs will all be increased and the resulting HI will be substantially greater. Please explain the origin of these values. The State reserves the right to dispute these values pending receipt of this information.

Response: The information requested by the State is presented in the left column of Figures D.1-16 (for tissue-based risk calculations) and D.1-17 (for dose-based risk calculations). The estimated average COC tissue concentrations and doses are calculated using a weighted average of the prey tissue concentrations in the RMA lakes, where the weighting factors represent the assumed relative sizes of the predator's feeding areas on the different lakes. In addition to the documentation provided on Figures D.1-16 and D.1-17, the State has copies of the electronic spreadsheets on which the actual calculations are performed, and the Army provided a contact person to answer specific questions regarding these calculations.

Comment 63: 3. Having estimated values for 11 of 24 Plankton categories, six of 24 Aquatic Plant categories, 16 of 24 Aquatic Invertebrate categories, and 21 of 24 Amphibian categories, the Army refuses to estimate COCs for Small Fish or Large Fish. The State regards this failure as inconsistent, and arbitrary. Either all categories should be estimated and an acknowledgement of this fact provided, or none should be estimated.

If the mean value for Upper Derby Lake and Lower Derby Lake are used across the small fish and large fish trophic boxes among COCs for which there are no

data, the resulting HQs for heron can be expected to increase by as much as 20 to 30 percent. Please explain the basis for the development of these values. The State reserves the right to dispute these values pending receipt of this information.

4. The State notes that in deriving the COC values for small fish in Lake Mary, Lower Derby Lake, and Upper Derby Lake, values for COCs in the brown bullhead and the black bullhead were apparently used. As the present IEA points out (page C.5-9), bullheads have been successfully eliminated from RMA lakes because of USFWS management practices. Because these fish accumulate contaminants poorly, the effect of inclusion of these species, which are no longer present in RMA lakes, tends to drive the average concentration of COCs downward. Similarly, the exclusion of carp, which are present in substantial numbers in RMA lakes, also tends to underestimate the COCs in fish of the RMA lakes. If bullheads are eliminated from the calculation and an estimated value for carp is included, the concentrations in fish increase dramatically. Though carp tend to be lake-bottom feeders, they surface and become available to be preyed upon by the eagle and heron during their spring mating season and when individual fish die. Because risk in heron is driven by fish values, even using conservative values for COCs in carp the HQs resulting in heron can increase by as much as 50 to 90 percent or more.

The State believes that the values used to represent fish in the Army's aquatic model seriously underestimate risk to biota dependent upon the aquatic systems at RMA and should be revised accordingly.

Response: The State fails to explain the basis for its comment that bullheads accumulate COCs poorly (note: dieldrin and endrin concentrations in bullheads were greater than or equal to those in pike). The Army has not "failed" to estimate COC tissue concentrations for small fish in East Upper Derby Lake and Rod and Gun Club Pond, or for large fish in East Upper Derby Lake, Upper Derby Lake, and Rod and Gun Club Pond. The fish species sampled were not found in these lakes. Upper Derby had no fish in it in 1988; by January 1990 it had large numbers of carp, which must have come from either Lower Derby Lake (for which the Army has numerous fish samples) or more probably from the Highline Canal or Uvalda Ditch (in which case they would not be representative of RMA). Composite samples of very small bullheads and of plankton were collected from Upper Derby in 1990, after the eagles had been observed feeding there. However, other aquatic species (except for waterfowl) were not sample there. We believe the fish species and locations were have sampled are most representative of well established aquatic habitats at RMA. It would thus have been inappropriate to simulate fish populations for the risk calculations. This is explained on page C.1-20 (Section C.1.4.2, Characterization of Exposure Concentration for Aquatic Food Webs) of the Proposed Final IEA/RC, Version

3.1. The ecological risk characterization assumes that heron and eagle catch fish in Lower Derby Lake and Lakes Ladora and Mary. Note that the wintering eagles would have left RMA before the carp breed, and that a large breeding carp would be too large to be preferred prey of herons. Further, Eastern Upper Derby and Upper Derby are filled by flood waters from the Highline Canal and Uvalda Ditch and are updrainage from Lower Derby Lake and Lake Ladora. Therefore, there is no underestimation bias in the algorithms for estimating heron and eagle doses and tissue concentrations. The State has copies of the electronic spreadsheets on which the actual calculations are performed, and the Army has provided a contact person to answer specific questions regarding these calculations.

Comment 64: 5. The State notes that the data supporting the values presented in the water bird table (Figure D.1-11) are all derived from tissue type categories 02, 03, and 04. Since tissue type category 01 represents wholebody and tissue type 07 represents a composite, any other category must represent some other type of tissue that was utilized. Please indicate the identities of tissue-type categories 02, 03, and 04. If these represent tissues other than wholebody values, please indicate how wholebody TC were calculated for the water bird category. The State reserves the right to dispute these values pending receipt of this information.

Response: The animal tissue sample type codes are as follows:

1 = whole body
2 = dressed carcass
3 = egg
4 = muscle tissue
5 = liver tissue
6 = brain tissue
7 = composite
10 = heart tissue
11= kidney tissue
12 = body fat
13 = solid stomach contents
15 = other

Most (62) of the water bird samples were of dressed carcasses. Mallard livers (10) were analyzed separately because they are sometimes eaten by humans. In the Biota RI some eggs (12) were also analyzed, and muscle and liver were analyzed from 17 specimens of several other water bird species.

Comment 65: 6. As with the trophic box Waterbird, the State notes that the Shorebird trophic box is based upon samples of killdeer which were labeled as tissue type 02. For approximately half of the aldrin/dieldrin values, all of the mercury values, and

half of the endrin values, the arithmetic mean of the RMA lake data was apparently used to calculate the values for the Shorebird table in Figure D.1-11. If no unspecified conversion for tissue type was made, it appears that several of the COC categories for shorebird are in error.

Analysis of a total of five shorebirds support the shorebird data table. Three of these birds came from Lake Mary and two came from Lower Derby Lake. All were analyzed as tissue type 02.

Response: Tissue sample type code 2 denotes dressed carcass.

Comment 66: The Army reports the following data for shorebird (Figure D.1-11):

Lakes	Ald/Dld	DDE/DDT	Endrin	Hg
East Upper Derby	0.6	4.0	0.06	0.08
Upper Derby	0.6	4.0	0.06	0.08
Lower Derby	0.6	0.9	0.02	0.07
Rod and Gun Club	0.6	4.0	0.06	0.08
Ladora	0.6	4.0	0.06	0.08
Mary	0.5	7.0	0.1	0.1

If no conversion for tissue type is required, the means of the available RMA data present an entirely different picture than that portrayed by the Army:

Lakes	Ald/Dld	DDE/DDT	Endrin	Hg
East Upper Derby	0	0	0	0
Upper Derby	0	0	0	0
Lower Derby	1.23	2.12	0.36	0.08
Rod and Gun Club	0	0	0	0
Ladora	0	0	0	0
Mary	1.09	12.77	0.118	0.07

With the exception of endrin in Lake Mary and mercury in Lakes Mary and Lower Derby, the true mean values of the data are nearly all double the values used by the Army. If these values are extrapolated across RMA lakes in the fashion used by the Army, the effects on tissue concentrations will be profound.

The State requests that the values in the table for shorebird be corrected and the estimated TC and HQs resulting from this correction be adjusted accordingly.

Response: The values in Figure D.1-11 for shorebird are correct. Shorebird is exposed to COCs through both the terrestrial and aquatic food webs. Therefore, it is necessary to partition the shorebird body burden among the source media. The numbers in Figure D.1-11 represent the portion of the total shorebird body burden attributed to COC exposure through the RMA lakes. The balance of the total shorebird body burden is attributed to terrestrial sources. Figure D.1-8 presents the shorebird partitioning coefficients and describes their development. The partitioning coefficients appear on page 3 of Figure D.1-8. The State has copies of the electronic spreadsheets on which the calculations shown in Figures D.1-8 and D.1-11 are performed, and the Army provided a contact person to answer specific questions regarding these calculations.

References

Bumb, R.R., W.B Crummett, S.S. Cutie, J.R. Gledhill, R.H. Hummel, R.O Kajel, L.L. Lamparksi, E.U. Luoma, D.L. Miller, T.J. Nestrick, L.A. Shadoff, R.H. Stehl, and J.S. Woods 1980. Trace Chemistries of Fire: A source of Chlorinated Dioxins. Science. Vol 210, No. 4468, pp 385-390.

Calabrase, E.J., R. Barnes, E.J. Stanek, M. Pastides, C.E. Gilbert, P. Veneman, X. Wang, A. Laszity, and P.T. Kostacki. 1989. How Much Soil Do Children Ingest: An Epidemiologic Study. Regul. Toxicol. Pharmacol. 10:123-137.

RTIC 92017R01
EBASCO (Ebasco Services Inc.). 1992. Remedial Investigation Summary Report. Final Report. January 1992.

RTIC 88357R01
1988. Rocky Mountain Arsenal Chemical Index with Applicable, Relevant and Appropriate Requirements. Volumes I-III.

Eisler, R. 1986. Dioxin Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review, Biological Report 85 (1.8). Contaminant Hazard Reviews Report No. 8. Fish and Wildlife Service, U.S. Department of the Interior. May 1986.

RTIC 91309R06v.1
EPA (U.S. Environmental Protection Agency). 1989. Risk Assessment Guidance for Superfund. Volume I. Human Heath Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response. EPA/540/1-89/002.

RTIC 92232R03
1991. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors." Office of Solid Waste and Emergency Response.

RTIC 88204R02
ESE (Environmental Science and Engineering). 1987. Remedial Investigation Program, Final, Phase I. Introduction to the Contamination Assessment Reports.

Kociba, R.J., and B.A. Schwetz. 1982a. Toxicity of 2,3,7,8-tetra-chlorodibenzo-p-dioxin (TCDD). Drug Metabol. Rev. 13: 387-406.

1982b. A review of the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the mouse. Toxicol. App. Pharmacol. 73: 69-77.

Masuda, Y., O. Hutzinger, F.W. Karasek, J. Nagayama, C. Rappe, S. Safe, and H. Yoshimura, eds. 1986. Chlorinated Dioxins and Related Compounds

1987. Chemosphere. Vol 16, Nos. 8/9.

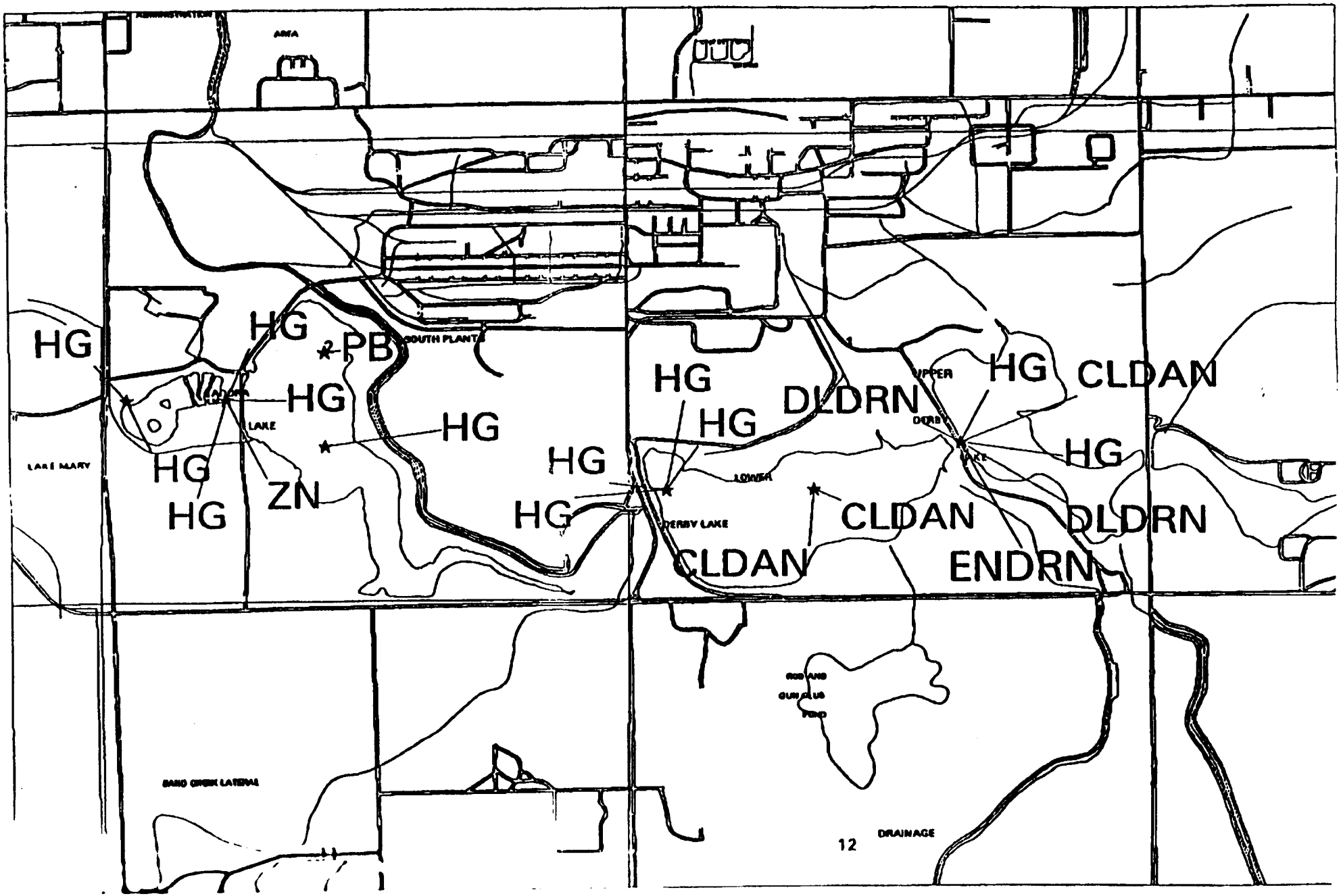
Young, A.L., L.G. Cockerham, and C.E. Thalken. 1987 Long-term Study of Ecosystem Contamination with 2,3,7,8-Tetrachlorodibenzo-p-dioxin. Chemosphere, Vol 16, Nos. 8/9, pp 1791-1815.

ırface water data.					
ılyte	Federal AWQC (ppb)	State Standard (ppb)	CRL range (ppb)	Comment	Problem ?
ldrin	0.0019	0.0019	0.05 - 26	Most CRLs are .05 ppb. CRLs have always been above standards.	yes
rin	1.5	1.5	0.05 - 13	Most CRLs are .05 ppb. CRLs have usually been below standards.	no
lrin	0.0023	0.0023	0.05 - 18	Most CRLs are .05 ppb. CRLs have always been above standards.	yes
E	1.05	1.05	0.046 - 14	Most recent CRLs are .049ppb. CRLs usually have been below standards.	no
T	0.001	0.001	0.049 - 18	CRLs have always been above standards.	yes
ordane	0.0043	0.0043	0.095 - 37	CRLs have always been above standards.	yes
enic	190	150	2.5 - 3.88	CRLs have always been below standards. (one minor exception).	no
al Mercury	0.012	0.01	0.2 - 2000	CRLs have always been above standards.	yes
lmium	1.1	1.134	4.09 - 8.4	CRLs have always been above standards.	yes
ɔper	11.8	11.824	6.2 - 26	CRL have gone up over time. Current above standards.	yes
ıd	3.2	3.89	18.6 - 74	CRLs have always been above standards.	yes
c	110	106	5.35 - 22	CRLs always below the standards.	no
omium III	210	206-776	4.44 - 24	Assumed data to be Cr III. CRLs always below standards. (one minor exception)	no
omium IV	11	11			

ECOLOGICAL RISK - ATTACHMENT A

Surface water Exceedances

Analyte	Federal AWQC (ppb)	State Standard (ppb)		Date	value	units	Stp_x	Stp_y	Section
Dieldrin	0.0019	0.0019							
			DLDRN	89109	0.0493	UGL	2187034	176932	01
			DLDRN	91091	0.0494	UGL	2187034	176932	01
Endrin	0.0023	0.0023							
			ENDRN	89109	0.0533	UGL	2187034	176932	01
Chlordane	0.0043	0.0043							
			CLDAN	89264	0.119	UGL	2185600	176400	01
			CLDAN	90102	0.211	UGL	2187034	176932	01
Total Mercury	0.012	0.01							
			HG	88166	0.158	UGL	2179692	177727	02
			HG	88166	0.215	UGL	2179692	177727	02
			HG	88166	0.262	UGL	2178434	177379	02
			HG	89262	0.179	UGL	2180600	177200	02
			HG	91091	0.128	UGL	2183945	176414	01
			HG	91091	0.128	UGL	2183945	176414	01
			HG	91091	0.169	UGL	2187034	176932	01
			HG	91091	0.209	UGL	2179692	177727	02
			HG	91091	0.249	UGL	2178434	177379	02
Copper	11.8	11.824							
			CU	87098	12.1	UGL	2222232	225495	
Lead	3.2	3.89							
			PB	89262	79.6	UGL	2180700	178200	02
Zinc	110	106							
			ZN	92258	1100	UGL	2179692	177727	02


HG
HG
PB
HG
HG
HG
HG
HG
HG
HG
ZN
HG
HG
DLDRN
HG
CLDAN
HG
CLDAN
DLDRN
CLDAN
ENDRN
LAKE MARY
LAKE
SOUTH PLANT
UPPER
DERBY
LOWER
DERBY LAKE
BANO CREEK LATERAL
DRAINAGE
12

RECENT DEVELOPMENTS IN ASSESSING SOIL INGESTION IN CHILDREN

Edward J. Calabrese

School of Public Health
University of Massachusetts
Amherst, MA 01003

SOIL INGESTION ISSUES AND RECOMMENDATIONS

A. Review of Published Studies

It has long been recognized that contaminated soil may present a potential public health concern because of groundwater contamination since groundwater is a significant drinking water source. Recently, regulatory and public health agencies have become concerned that consumption of contaminated soil by children may present a significant public health problem. For example, elevated levels of lead in soil and dust are suspected of contributing to elevated blood lead levels as a result of soil/dust ingestion (Spittler, 1986). Also widely discussed has been the dioxin contamination of Times Beach, Missouri. A major concern in the CDC assessment was the assumed consumption by children of soil containing dioxin.

The knowledge of how much soil and dust children ingest is critical for those involved with the issue of assessing public health risks from environmental contamination. Numerous site-specific clean up decisions, especially for those tightly bound to soil contaminants (e.g., dioxin, PCBs, lead, cadmium, others) are often driven by assumptions concerning estimates of soil and more recently, dust ingestion.

While early qualitative estimations of childhood soil ingestion have been made by several groups (Lepow et al., 1974; the National Research Council 1980; and Day et al., 1975), these attempts lack sufficient quantitative evaluation to allow confident estimation of actual soil ingestion. Subsequently, scientists at CDC developed an estimation for specific age groups based on unpublished behavioral observations of children aged 1 to 3.5 years. These children were estimated to ingest 10 g of soil/day (Kimbrough et al., 1984).

The first attempt to estimate human soil ingestion quantitatively was presented by Binder et al. (1986) using aluminum (Al), silicon (Si), and titanium (Ti) as soil tracer elements. These elements were selected because their concentration is high in soil but low in food products, and the

gastrointestinal tract absorption is low. The amount of soil ingested was calculated based on the fecal and soil concentrations of the tracer elements and the amount of fecal output. In their study involving 59 diapered children aged 1-3 years residing in Montana, the calculated mean soil ingestion estimates for the tracers Al and Si were 181 and 184 mg/day and ten times higher (1,834 mg/day) for Ti. The authors were unable to resolve the apparent conflict between estimates based on the various tracers.

Clausing et al. (1987) and Van Wijnen et al. (1990) have estimated the amount of soil consumed by children living in the Netherlands following a method similar to that of Binder et al. (1986). Clausing et al. (1987) reported mean soil ingestion values ranging from 127 mg to 1,084 mg/day, depending on the marker, with Ti yielding the highest estimate. The Binder et al. (1986) and Netherlands studies (Clausing et al., 1987; Van Wijnen et al., 1990) are indirect attempts to estimate soil ingestion and could be improved by measuring the concentration of tracers in food and other ingested products (e.g., medicines), of the participating subjects as well as the presence of tracers in diapers and other materials that contact the feces. Based on these and other limitations (see Calabrese and Stanek (1991) for a critical review), the Binder et al. (1986), Clausing et al. (1987), and Van Wijnen et al. (1990) studies are precluded from providing definitive evidence of soil ingestion.

Two additional soil ingestion studies on children have been published that include substantial improvements on the methodology used by the early investigators. The first study was on children aged 1-4 in western Massachusetts (Calabrese et al., 1989), while the second study was on children aged 2-7 in the state of Washington (Davis et al. 1990). Both studies accounted for tracer ingestion due to food consumption as part of their study protocol. Estimates of soil ingestion were made for three elements in the Davis et al., study, and eight elements in the Calabrese et al. study (Table 1).

Estimates of median soil ingestion were markedly lower in the Calabrese and Davis studies as compared with earlier

investigations. Estimates of median soil ingestion based on Al were less than 1/4th the earlier estimates while estimates based on Si and Ti were less than 1/2 and 1/7th the earlier estimates, respectively. Still, there was marked variation in the mean and median soil ingestion estimates for the individual studies. Estimates of median soil ingestion based on different tracers differed by over 300% in the Davis study, and by over 800% in the Calabrese study.

Due to the improvements in study design, the Davis and Calabrese studies provide the best estimates of soil ingestion to date. Since their conduct, much effort has been made to understand the reasons for the large variability in tracer specific estimates, and determine the most reliable estimate of soil ingestion (Stanek and Calabrese, 1991; Calabrese and Stanek, 1991). These investigations have identified the food/soil ratio for a tracer as a predictor of soil ingestion reliability. Basically, the larger the ratio, the less reliable the soil ingestion estimate for a given tracer due to a high signal to background noise ratio.

Those tracers (i.e., Ba, Mn) displaying the poorest performance in the adult validation study of Calabrese et al. (1989) in terms of precision of recovery were those with the highest food to soil ratios. Conversely, those tracers displaying very low food to soil ratio displayed considerably improved precision of recovery. Consequently, tracers with low food to soil ratios were estimated to have markedly lower (more sensitive) soil ingestion detection limits (see Stanek and Calabrese 1991; Calabrese and Stanek, 1991). Average daily ingestion of elements in food were larger in the Davis study than the Calabrese study (by 278%, 41% and 110%, for Al, Si, and Ti, respectively), thus implying that soil ingestion estimates are less reliable in the Davis study.

Table 1. Soil Ingestion Estimates in Children (mg/day)

	Binder et al.		Van Wijnen et al.		Davies et al.		Calabrese et al.	
	Mean	Median	Mean	Median	Mean	Median	Mean	Median
Al	181	121	--	--	40	25	153	29
Si	184	136	--	--	82	59	154	40
Ti	1834	618	--	--	246	81	218	55
Ba							32	<0
Mn							<0	<0
V							459	96
Y							85	9
Zr							21	16

Limiting Tracer Method (LTM)		
(Day Care Center)	103	111
(Campers)	213	160

The net impact of high food/soil ratios for a study has been large input-output misalignment errors. These errors have resulted in negative soil ingestion estimates for as many as 45% of study subjects depending on the specific tracer. While the negative misalignment errors can be easily recognized, the positive misalignment errors can only be guessed at due to the limitations in the study designs. Subsequent and on-going research in this area (see A.1. below, Recent Progress on Soil Ingestion) has served to further clarify such limitations.

In summary, estimates of soil ingestion to date are highly variable and of questionable reliability. Mean and median estimates for the study populations are inconsistent in given studies. The limited reliability has made subject-specific soil ingestion estimates, except for soil pica (> 1.0 gm/day) (Calabrese et al. 1991), impossible to construct.

The current studies also have significant limitations with respect to their generalizability. While the original UMass (Calabrese et al., 1989) study provided a higher reliability in soil ingestion estimates of the study participants due to improved precision of recovery estimates and lower soil ingestion detection limits than other reports, it is important to emphasize that the study has significant limitations with respect to its generalizability to other populations of children, especially those residing in urban areas and children of other Social Economic Status (SOES) and racial backgrounds. The non-random nature of the selected population affects its capacity to be generalized from an academic community in western Massachusetts to children in other communities. In addition, the study population was observed only between Monday and Thursday for two consecutive weeks. While suggesting a possible magnitude of inter-subject soil ingestion variation, the UMass as well as other soil ingestion studies provide no insight for seasonal, regional and ethnic variation in soil estimates, nor whether soil ingestion differentially occurs on weekends rather than week days. Another factor inadequately considered in these studies was the relationship of the extent of grass cover and how that was quantitatively related to soil ingestion.

The collective limitations of the present soil ingestion

data base to offer generalizations to other populations of children concerning quantitative estimates of soil ingestion present a serious challenge to regulatory/public health officials performing soil-based exposure assessments.

Despite the completion of four studies on soil ingestion in children, most of the initial benefit of these investigations has been in the development of improved understandings of how to design, conduct, analyze and interpret soil ingestion study data. Current methods now permit the development of study protocols that can adequately address critical issues of tracer selection, sample size, duration of study, and sources of positive error (e.g., input misalignment, unknown source input) and negative error (e.g., output misalignment, sample loss during analysis), such that highly reliable estimates of soil ingestion can be derived.

A.1. Recent Progress on Soil Ingestion

Since the publication of the original study on soil ingestion in 1989 (Calabrese et al., 1989), the University of Massachusetts (UMass) soil research group have extensively re-evaluated their original findings and have been able to advance understandings in several relevant areas:

(1) Clarification of the Causes of Intertracer Variation in Soil Ingestion Estimates. The major sources of error in the UMass soil ingestion study have recently been identified and quantified on a subject-tracer basis. The principal sources of error are (a) input/output misalignment[1] error due to both study design limitations and the presence of high background leve

[1] Input misalignment error occurs when an unusually high quantity of tracer is ingested on a day just prior to the start of the soil ingestion study, and this quantity is captured in fecal samples during the study. This "extra" amount of fecal tracer would be incorrectly attributed to soil ingestion (i.e., input misalignment error). An output misalignment error occurs when tracers ingested in food are not captured in fecal samples due to slow transit time and the study ends before the passage of food occurs. This is the principal cause of the high number of children in the Calabrese et al. (1989) and Davis et al. (1990) studies displaying negative soil ingestion values. In fact, 44 of 128 subject-weeks did not provide a fecal sample on the final day of the Calabrese et al. (1989) study, thereby contributing to incorrect lower and negative soil ingestion estimates.

of tracers in ingested food relative to ingested soil;

(b) unknown source error for several tracers. By comparing soil ingestion estimates on a subject by day basis, it has been possible to quantify the input and output error per element (Table 2). Furthermore, additional sources of element ingestion are evident. For example, Ti and V appeared to have been ingested from sources other than soil and food, thereby falsely inflating soil ingestion estimates for affected tracers. Table 3 illustrates the magnitude and type of positive and negative error within the UMass children soil ingestion study. This knowledge can readily be incorporated in the design and conduct of future studies so that more reliable estimates of soil ingestion can be obtained.

Table 2. **Positive/Negative Error (Bias) In Soil Ingestion Estimates In the UMass Mass-Balance Study.*** The Error has been identified by Source (Output/Input misalignment, sample loss, extraneous source) and Quantified by Tracer leading to improved new mean estimates of soil ingestion. (Values are given as mg of soil ingested/day.)

	Negative Error (Bias)		Positive Bias		Net Bias	Mean	Adjusted (Improved) Mean
Tracer	Output Error	Sample Loss	Input Error	Extraneous Error			
Al	6.3	--	8.0	3.2	+ 4.9	153	148.1
Si	7.8	--	9.5	15.1	+ 16.8	154	137.2
Ti	156.2	--	138.4	97.9	+ 80.1	218	137.5
V	24.9	--	83.3	243.6	+302	459	157
Y	48	--	1.6	1.7	- 44.8	85	129.8
Zr	25.3	69	0.0	0.9	- 97.4	21	118.4

*Values indicate impact on mean of 128-subject-weeks in mg of soil ingested per day.

The new range of 118.42 to 157 is 1.325-fold
The old range of 25 to 459 is 18.36-fold

Variation in the mean is reduced by 93% as a result of the quantification of the different sources of error.

If it were necessary to select a single estimate for soil ingestion we would recommend an estimate on a combined soil and dust element concentration, and consider the best estimate to be based on the median. The rejection of the arithmetic mean is based not only on its instability as a measure of central tendency of the population but that it does not have any precise meaning in the assessed population. For example, with variables that have skewed distributions, the mean does not represent any benchmark in terms of a percentile. This is in contrast with the median, which represents the 50th percentile.

Age Related Changes

It has been generally assumed by various state/federal regulatory and public health agencies that all human age groups ingest soil. It has been concluded, based principally on professional judgment, that children ingest more soil than adults and that children with high hand to mouth activity (i.e. ages 1-4) ingest more soil than children of other ages.

Analysis of the Calabrese et al. (1989) data revealed that soil ingestion increased linearly with age for all tracers. This was particularly evident for Ti while much less for Zr (Stanek et al., 1991). The slopes of these two most reliable tracers differ to such an extent (slope 7.36-Ti, 0.49-Zr) that it cannot be determined for the Calabrese et al. (1989) study population that soil ingestion increases as children increase in age from 1 to 4.

While it is believed that young children ingest more soil than other age groups, this assumption is not based on empirical data. Incidental or intentional soil ingestion in older children may be more or less but this study and others provide no quantitative information on the soil ingestion rates that answer this question.

In light of the inadequacies of the soil ingestion data base, how are age adjustments in soil ingestion to be made? It would appear logical that adults should ingest significantly less soil than young children. It would seem reasonable, in the absence of

reliable quantitative data, to assume that an "average" adult ingests from 25 to 10% of the "average" child based on diminished hand to mouth activity and other maturational and social factors.

Based on this rationale it is recommended that children 6-12 years of age be assumed to ingest 25% of the soil and dust ingestion value of a 1-6 year old child while those > 12 years of age be assumed to ingest 10% of the 1-6 year old child.

Rural vs Urban/Suburban Children

No quantitative data exist on the comparative soil ingestion rates of children from rural, urban and suburban areas. This remains an important data gap to be filled. At present, any attempt to make a distinction in soil ingestion rates would be speculative. There may be a number of potential factors affecting the differential rate of soil ingestion amongst rural, urban and suburban children such as time spent outdoors, degree of grass cover of outdoor play areas, quantity of dust in home and others. However, in the absence of adequate information on these variables, the present emphasis will focus on the extent of grass cover because of the obvious direct access to contact with soil. Under such circumstances, it is not unreasonable to suggest the incorporation of an uncertainty factor (UF) analogous to those used in risk assessment activities for non-carcinogens. Since this represents concern with inter-individual variation an uncertainty factor (UF) of approximately 1-10 could be selected depending on the degree of grass cover in areas where children play. If grass cover were extensive (>90%), an UF of 1 would be appropriate where the value derived from the study was based on extensive grass cover. However, if grass cover were more limited (50-90%) in areas of access, then a 5-fold factor would be recommended while a 10-fold factor would be used if grass cover were <50%. This approach may have site specific application but it is not recommended for national or statewide guidance.

Seasonality

The Calabrese et al. (1989) study was conducted in the fall (Sept./Oct) in Massachusetts. It may be speculated that ingestion of soil may be highest in the summertime and lowest in the winter in Massachusetts, based on the premise that children play longer hours outdoors in the summer with greater direct contact with soil. However, it may be argued that soil contact may actually be greater in the spring before the growth of grass becomes significantly thickened or during more rainy seasons such as spring and winter, depending on geographical locations. Thus, it is possible that seasonal effects may markedly vary according to a variety of factors and that soil ingestion may not be highest in the summer months in all locations. In addition, there may be seasonal variation in the tracking in of dust within the home with perhaps more mud being tracked into the home during the more rainy seasons. In the absence of information to clarify these uncertainties in the data base, no quantification of seasonal effect is recommended at this time.

Identification of Pica Children

The consumption of non-food items, especially by young children, is a very common activity; when this activity is excessively performed it becomes characterized as pica. The range of non-food items that such children may ingest is extremely variable, including: clothing, books/paper, crayons, soil, cigarettes, household furnishings and other items.

The prevalence of pica behavior appears to be highly variable, being contingent on the definition of pica and the population assessed amongst other factors. Table 4 reveals that the prevalence of pica behavior can range from 10% in Caucasian children to 66% in institutionalized psychotic children. It appears, therefore, that children 1 to 6 years old display a pica prevalence that is between 10 and 30% with no obvious significant variation between males and females. It should be noted, however, that these studies did not use a uniform definition of pica.

Table 4. Range of Pica Behavior Prevalence

Group Description	# Subjects	% of Pica	Reference
Retarded Children	30	50	Kanner, 1937
Black Children > 6 mo.	386	27	Cooper, 1957
White Children > 6 mo.	398	17	
Black, 1-6 years	486	32	Millican et al., 1962
White, 1-6 years	294	10	
Children, low income	859	55	Lourie et al., 1963
Children, high income		30	
Children, 1-6 (interview)	439	15	Barltrop, 1966
Children, 1-6 (mail survey)	227	50	
Institutionalized, psychotic 3-13 years	40	66	Oliver, 1966
Spanish American Children (California)	21	32	Bruhn & Pangborn, 1971
Children (Mississippi)	115	16	Vermeer & Frate, 1979

The identification of pica children presents a major initial stumbling block since there are no definitive criteria for this behavior. The present literature often represents subjective judgements based on individual perceptions of what comprises pica behavior, with limited standardized behavioral norms concerning whether children display pica behavior.

The four available large scale soil ingestion studies (Binder et al., Calabrese et al., Davis et al., and Van Wijnen et al.) were examined for evidence of pica soil behavior. These collective studies have provided daily soil ingestion on 517 children. If soil pica were subjectively defined in quantitative terms as consumption of greater than 1 gm of soil per day, then 10 individuals would be identified from these four studies as having displayed this behavior. This would amount to a soil-pica prevalence of 1.9% from the four available soil ingestion studies.

This soil tracer estimation of the prevalence of soil pica children of course rests on very limited data. The nine individuals in the Van Wijnen et al. study displayed the pica-like behavior (> 1000 mg/day) on only a single observation day over a 2-5 day period. The soil ingestion values of th e subjects was not adjusted downward for food ingestion of the tracer elements, thus leading to variable overestimates of soil ingestion. The one child pica subject in the Calabrese et al. study was observed over two separate 4 day periods and displayed soil-pica behavior only in the second of the two week period of observation. These data suggest that some children displaying soil pica behavior do so irregularly and thus would not be predicted to consistently ingest > 1 gram of soil per day. The Calabrese et al. (1989) data suggest that only 1 child of the 64 (0.64%) ingested greater than an average of 6.5 grams of soil per day over eight days. The duration of exposure for these estimates is most likely restricted to ages 1-6 (i.e. 5 years). While it is possible that soil pica may be observed in some children beyond age six, the prevalence of this behavior is expected to rapidly decrease as one ages.

Two relevant developments with respect to soil pica estimations have emerged. First, a study was recently completed that assessed the pica prevalence of over 500 children residing in rural, suburban and urban locations of western Massachusetts (Calabrese et al., 1993). Of particular significance to the issue of soil pica is that 11% of parents reported daily soil mouthing behavior for their children in the one year old age group (95% CI from 6-16%). A follow-up study of the specific soil ingestion behavior of 12 children identified by the survey questionnaire revealed that one of the children ingested on average about 1.5 gm/day over the seven consecutive day observation period. This observation is of particular significance since it provides additional quantitative evidence of soil pica but also indicates that soil pica is a consistent behavior in some children.

Secondly, the University of Massachusetts soil research unit has recently developed a methodology that permits the estimation of daily soil ingestion. In all past studies the investigators estimated the total soil ingestion quantity for the period of observation and then divided by the total number of days to derive a daily rate. The new methodology will permit estimates for each specific day. With this information it is now possible to estimate soil ingestion distributions for individual subjects for given time periods (e.g. 12 months). Calabrese and Stanek (1994) have recently provided individual soil ingestion distributions of 64 subjects of the Calabrese et al. (1989) study, based on up to eight individual daily ingestion values that have been corrected for positive and negative error using a log-normal distribution. The median of the estimate daily average soil ingestion values over one year is 75 mg/day while the upper 95% value is 1,750 mg/day (Table 5, 6). These findings for the upper 95% represent a striking departure from the recommendations of EPA of 200 mg/day. They are, however, consistent with the soil pica prevalence data as well as the quantitative estimates of soil ingestion seen in the soil pica child observed in Calabrese et al. (1993). The findings indicate that a substantial percentage of children are expected to ingest large amounts of soil on a daily basis. It also indicates that extreme soil pica exists in a smaller percentage of the

population. These findings suggest that soil ingestion has the capacity to affect both chronic and acute toxic responses.

Table 5. Soil Ingestion Estimates on 64 Subjects Over 365 Days Based on the Fitting of a Log-Normal Distribution Model to Daily Soil Ingestion Values (Calabrese and Stanek, 1994).

Range of median soil ingestion estimates of 64 subjects over 365 Days	1 - 103 mg/day
Median of the median soil ingestion estimates of 64 subjects over 365 days.	14 mg/day
Range of upper 95% soil ingestion estimates of 63 subjects over 365 days. (excluding the soil-pica child)	1 - 5,263 mg/day
Median of the upper 95% soil ingestion estimates of the 64 subjects over 365 days.	252 mg/day
Range of estimated total number of grams of soil ingested per year by 63 subjects.	.365 g - 828.16
Range of average daily soil ingestion values for the 63 subjects over 365 days. (excluding the soil-pica child)	1 - 2,268 mg/day
Median of the 64 subjects daily soil ingestion as average cover 365 days.	75 mg/day
The upper 95% of the daily soil ingestion average of 64 subjects over 365 days.	1,751 mg/day
The upper 90% of the daily soil ingestion average of 64 subject over 365 days.	1,190 mg/day

Table 6. Estimated Percent of Children with Soil Ingestion Exceeding Daily Rates for Given Time Periods Per Year (Calabrese and Stanek, 1994)

		Daily Rate of Soil Ingestion				
Estimate Number of Days/Year With Soil Ingestion	1-2 days	>200 mg 86%	500 mg 72%	>1 gm 63%	>5 g 42%	>10 g 33%
	7-10 days	72%	53%	41%	20%	9%
	35-40 days	42%	31%	16%	1.6%	1.6%

References

Barltrop, D. (1966). The prevalence of pica. *Am. J. Dis. Child.*,112:116-123.

Binder, S., Sokal, D. and Maughan, D. (1986). Estimating the amount of soil ingested by young children through tracer elements. *Arch. Environ. Health.* 41, 341-345.

Bruhn, C.M., and Pangborn, R.M. (1971). *J. Am. Diet. Assoc.*, 58:417-420.

Calabrese, E.J., Barnes, R., Stanek, E.J., Pastides, H., Gilbert C.E., Veneman, P., Wang, X., Lasztity, A. and Kostecki, P.T. (1989). How Much Soil Do Young Children Ingest: An Epidemiologic Study. *Reg. Toxic. and Pharm.* 10:123-137.

Calabrese, E.J., and Stanek, E.J. III. (1991). A guide to interpreting soil ingestion studies. II. Qualitative and quantitative evidence of soil ingestion. *Reg. Toxicol. and Pharm.* 13:278-292.

Calabrese, E.J., and Stanek, E.J. III. (1992). Distinguishing outdoor soil ingestion from indoor dust ingestion in a soil pica child. *Regulatory Toxicol. Pharm.*, 15:83-85.

Calabrese, E.J., and Stanek, E.J. (1994). How to interpret soil ingestion studies: Part 5, Development of annual soil ingestion distributional estimates of 64 children based on daily soil ingestion values. (Submitted).

Calabrese, E.J., and Stanek, E.J. III, Gilbert, C.E., and Barnes, R.M. (1990). Preliminary adult soil ingestion estimates; Results of a pilot study. *Reg. Toxicol. Pharm.*, 12:88-95.

Calabrese, E.J., Stanek, E.J. III, and Mundt, K. (1993). Final report on pica prevalence based on survey of children reporting for well-visits in western Massachusetts. (submitted).

Cooper, M. (1957). Pica. Springfield, IL: Charles C. Thomas. 109 pp

Davis, S., Waller, P., Buschbom, R., Ballou, J., White, P. (1989). Quantitative Estimates of Soil Ingestion in Normal Children Between the Ages of 2 and 7 Years: Population-based Estimates Using Aluminum, Silicon, and Titanium as Soil Tracer Elements. *Arch Env. Hlth.*, 45:112-122.

Harvey, P.G., Spurgeon, A., Morgan, G., Chance, J. and Moss, E. (1986) A method for quantifying hand-to-mouth activity in young children. *J. Child. Psychol.*

Kanner, L. (1937). Child Psychiatry, pp. 340-353. Springfield, IL: Charles C. Thomas.

Lourie, R.S., Layman, E.M., and Millican, F.K. (1963). The epidemiology of lead poisoning and children. *Arch. Pediat.*, 79:72-76.

Millican, F.K., Layman, E.M., Lourie, R.S., Rakahashi, L.Y., and Dublin, C.C. (1962). The prevalence of ingestion and mouthing of non-edible substances by children. *Clin. Proc. Child. Hosp.* (Wash.), 18:207-214.

Oliver, B.E., and O'Gorman, G. (1966). *Develop. Med. Child.* Neurol., 8:704-706.

Porter, J.W. U.S. EPA Office of Solid Waste and Emergency Response, Jan. 27, 1989). Memorandum to regional administrator, Region 1-X, regarding interim final guidance on soil ingestion rates.

Stanek, E.J. III, Calabrese, E.J., and Gilbert, C.E. (1990). Choosing a best estimate of children's daily soil ingestion. In: *Petroleum Contaminated Soil*. Vol. 3. P.T. Kostecki and E.J. Calabrese, (eds.). Lewis Pub., Chelsea, MI, pp. 341-348.

Stanek, E.J., III, and Calabrese, E.J. (1991). A guide to interpreting soil ingestion studies. I. Development of a model to estimate the soil ingestion detection level of soil ingestion studies. *Reg. Toxicol. and Pharm.*, 13:253-277.

Stanek, E.J. III, and Calabrese, E.J. (1992). Soil ingestion in children: Outdoor soil or indoor dust. *J. Soil Contamination*, 1(1):1-28.

Stanek, E.J. III, Calabrese, E.J., and Zheng, L. (1990). Soil ingestion estimates in children: Influence of age and sex. *Trace Substances In Environ. Health*, 24:43.

Van Wijnen, J.H., Clausing, P., and Brunekreef, B. (1989). Estimated Soil Ingestion By Children. *Env. Res.*, 51:147-162.

Vermeer, D.E., and Frate, D.A. (1979). Geophagia on rural Mississippi: environmental and cultural contexts and nutritional implications. *Am. J. Clin. Nutr.*, 32:2129-2135.