May 2002

Exposure Analysis for Dioxins, Dibenzofurans, and CoPlanar Polychlorinated Biphenyls in Sewage Sludge

Technical Background Document

DRAFT

Prepared for

ICF Consulting, Inc. The Office of Water, U.S. Environmental Protection Agency 401M Street, SW (5307W) Washington, DC 20460

Exposure Analysis for Dioxins, Dibenzofurans, and CoPlanar Polychlorinated Biphenyls in Sewage Sludge

Technical Background Document

DRAFT

May 2002

Prepared for

ICF Consulting, Inc. Office of Water, U.S. Environmental Protection Agency 401 M Street, SW (5307W) Washington, DC 20460

Prepared by

Center for Environmental Analysis RTI Research Triangle Park, NC 27709

Table of Contents

Sectio	on	Pa	ge
Acror	iyms and	d Abbreviations	xii
List o	f Figure	s	vii
List o	f Tables		ix
1.0	Introd	uction	-1
	1.1	Background 1	-1
	1.2	Summary of the Risk Assessment Process 1	-1
	1.3	Overview of Risk Assessment Methodology 1	-2
	1.4	Document Organization 1	-3
	1.5	References 1	-5
2.0	Hazar	d Identification/Dose-Response Assessment	2-1
	2.1	Adverse Effects in Humans and Animals	2-1
		2.1.1 Mechanism of Action	2-2
		2.1.2 Epidemiologic Studies—Cancer Endpoint	2-3
		2.1.3 Animal Studies—Cancer Endpoint	2-6
	2.2	Risk Characterization	2-6
	2.3	Dose-Response and Slope Factors 2	2-8
		2.3.1 Human and Animal Studies 2	2-8
		2.3.2 Toxicity Equivalency Factors	2-9
	2.4	References 2-	10
3.0	Risk A	Assessment Overview	3-1
	3.1	Human Health Risk Assessment	8-1
		3.1.1 Application of Biosolids to Agricultural Land	3-1
		3.1.2 Constituents of Concern	3-2
		3.1.3 Site Configuration and Environmental Setting 3	8-2
		3.1.4 Exposure Point Estimates 3	3-4
		3.1.5 Assessing Human Exposures 3	3-5
		3.1.6 Toxicity Assessment and Risk Characterization	8-6
	3.2	Probabilistic Method for Determining Exposure Point Concentrations 3	8-6
	References	8-7	

Table of Contents (continued)

Secti	on		Page
4.0	Input	t Data Characterization	4-1
	4.1	Input Data Development Procedure	4-1
	4.2	Characterization of Biosolids	4-1
		4.2.1 Concentrations of Dioxin and Furan Congeners	4-2
		4.2.2 Agricultural Application of Biosolids	4-3
	4.3	Site Characterization	4-3
		4.3.1 Conceptual Site Layouts	4-4
		4.3.2 Regional Environmental Setting	4-4
	4.4	References	. 4-21
5.0	Estin	nating Exposure Point Concentrations	5-1
	5.1	Source Partition Modeling of Constituent Releases	5-2
		5.1.1 Land Application Unit Partitioning Model Used for	
		Agricultural Fields	5-2
	5.2	Fate and Transport Modeling	. 5-11
		5.2.1 Dispersion and Deposition Modeling	. 5-12
		5.2.2 Estimation of Soil and Sediment Concentrations	. 5-19
	5.3	Calculation of Food Chain Concentrations	. 5-25
		5.3.1 Terrestrial Food Chain	. 5-25
		5.3.2 Aquatic Food Chain	. 5-39
	5.4	Infant Breast Milk Exposure	. 5-40
	5.5	References	. 5-44
6.0	Hum	an Exposure Assessment	6-1
	6.1	Receptors and Exposure Pathways	6-2
		6.1.1 Childhood Exposure	6-3
		6.1.2 Infant Exposure	6-4
		6.1.3 Exposure Pathways	6-4
	6.2	Exposure Factors	6-5
		6.2.1 Intake Factors	6-7
		6.2.2 Other Exposure Factors	. 6-22
	6.3	Dose Estimates	. 6-25
		6.3.1 Average Daily Dose	. 6-26
		6.3.2 Lifetime Average Daily Dose	. 6-26
	6.4	References	. 6-27

Table of Contents (continued)

Sec	tion		Page
7.0	Huma	n Health Risk Results	7-1
	7.1	Human Health Risk Characterization	7-1
		7.1.1 Lifetime Excess Cancer Risk	7-1
		7.1.2 Total Lifetime Excess Cancer Risk	7-2
		7.1.3 Risk Results	7-2
	7.2	Multipathway Risks	7-9
8.0	Analysis	of Variability and Uncertainty	8-1
	8.1	Variability	8-1
		8.1.1 Source Characterization and Emissions Modeling	8-2
		8.1.2 Fate and Transport Modeling	8-3
	8.2		8-9
		8.2.1 Scenario Uncertainty	8-9
		8.2.2 Model Uncertainty	. 8-11
		8.2.3 Variable Uncertainty	. 8-14
	8.3	References	. 8-15
9.0	Scree	ning Ecological Risk Assessment of Dioxins and PCBs in Land-Applied	9-1
	9 1	Introduction	9-1
	9.1	Problem Formulation	9_2
).2	9.2.1 Assessment Endpoint Selection	9_2
		9.2.1 Assessment Endpoint Selection	<i>J 2</i> 9_4
		9.2.2 Development of conceptual Wodel	9-15
	93	Analysis Methods	. <i>J</i> -15
	7.5	0.3.1 Phase 1 Maximum Potential Rick	0_16
		9.3.2 Phase 2 - Deterministic Screening	9_20
	94	Results and Risk Characterization	. <i>J</i> 20 9_27
	7.7	$9.4.1$ Interpreting Results from the SER Δ	9_28
		0.4.2 Silvicultural and Reclamation Site Applications	0_32
		9.4.2 Subscuttural and Reclamation Site Applications	0.32
	9.5	References	. 9-35
Anr	endix A	2001 National Sewage Sludge Survey – Congener Concentration Data	A-1
Anr	endix B	2001 National Sewage Sludge Survey – Sample Selection Strategy	B-1
Anr	endix C	Agricultural Parameters	C-1
Apr	endix D	Congener-Specific Parameters for Source Partitioning and Fate and	
rr		Transport Models	D-1
Apr	endix E	Site Data	E-1

Table of Contents (continued)

Section

Page

Appendix F	Source Model for Land Application Units F-1
Appendix G	Air Dispersion and Deposition Modeling Input FilesG-1
Appendix H	Direct and Indirect Exposure Equations
Appendix I	Variables for Aboveground Fate and Transport I-1
Appendix J	Human Exposure Factors J-1
Appendix K	Sensitivity Analysis Results
Appendix L	Screening Ecological Risk Assessment Data L-1
Appendix M	Climate Region Selections M-1

List of Figures

Numb	Number Page			
3-1	Agricultural Application Conceptual Site Model 3-3			
3-2	Map of 41 Climatic Regions 3-3			
5-1	Biosolids Application to Agricultural Field Source Module			
5-2	Emissions Mechanisms in the Local Watershed			
5-3	Example Depth-Averaged Soil Concentration Annual Time Series			
5-4	Biosolids Application to Agricultural Fields Media Concentration Module 5-26			
6-1	Human Exposure Pathways 6-1			
6-2	Distribution of Exposed Fruit Consumption Rates by Age Group 6-9			
6-3	Distribution of Exposed Vegetable Consumption Rates by Age Group 6-10			
6-4	Distribution of Root Vegetable Consumption Rates by Age Group 6-12			
6-5	Distribution of Beef Consumption Rates by Age Group 6-13			
6-6	Distribution of Milk Consumption Rates by Age Group 6-15			
6-7	Distribution of Poultry Consumption Rates by Age Group 6-17			
6-8	Distribution of Egg Consumption Rates by Age Group 6-18			
6-9	Distribution of Adult Fish Consumption Rates by Age Group 6-20			
6-10	Distribution of Breast Milk Consumption Rates by Age Group 6-21			
6-11	Distribution of Inhalation Rates by Age Group			
6-12	Distribution of Body Weights by Age Group 6-23			
6-13	Distribution of Exposure Duration for Child and Adult			
8-1	Convergence analysis			

List of Figures (continued)

Numb	er Pa	ge
9-1	Conceptual model for the biosolids SERA	-5
9-2	Terrestrial food web, including example receptors	-9
9-3	interface between terrestrial receptors and aquatic food web, including Example receptors	10

List of Tables

Numb	er Page
2-1	Summary of Combined Cohort and Selected Industrial Cohort Studies with High Exposure Levels
2-2	Toxic Equivalency Factors 2-9
4-1	Physical Characteristics of Biosolids 4-2
4-2	Median Farm Size for Each Climatic Region 4-5
4-3	Relation between Anderson Land Use codes and PCRAMMET Land Use Codes 4-8
4-4	Daytime Bowen Ratio by Land Use Season 4-10
4-5	Minimum Monin-Obukhov Length (Stable Conditions) 4-10
4-6	Albedo Values of Natural Ground Covers for Land Use Types and Seasons 4-11
4-7	Surface Roughness Length for Land Use Types and Seasons (Meters) 4-11
4-8	Summary of Soi8ld Properties Collected for Biosolids Risk Analysis 4-14
4-9	Hydrological Soil Parameters Correlated to Soil Texture 4-16
4-10	Depth to Root Zone Values 4-17
4-11	Field Capacity (FC) and Wilting Point (WP) Values 4-17
4-12	SCS Curve Number Values by SCS Hydrologic Soil Group 4-18
4-13	Default Flow Lengths by Slope 4-20
5-1	Calculated TCDD Half-Lives for Selected Risk Distribution Percentiles 5-10
5-2	Soil Half-Life Data Reported in the Draft Dioxin Reassessment Document 5-11
5-3	TCDD-TEQ Media Concentration for Ambient Air Variable Concentrations 5-18
5-4	TCDD-TEQ Media Concentration for Soil in Buffer, Cropland, Pasture, and
	Sediment Variable Concentrations
5-5	Terrestrial Food Chain Vegetation
5-6	TCDD-TEQ Media Concentratin for Exposed Fruits and Vegetables
	Variable Concentration 5-30
5-7	TCDD-TEQ Media Concentration for Belowground Vegetables
	Variable Concentrations 5-31
5-8	TCDD-TEQ Media Concentrations by Percentile for Beef
	Variable Concentrations 5-35
5-9	TCDD-TEQ Media Concentrations by Percentile for Milk
	Variable Concentrations 5-35
5-10	TCDD-TEQ Media Concentrations for Poultry Thigh Meat
	Variable Concentrations 5-38
5-11	TCDD-TEQ Media Concentrations for Eggs Variable Concentrations 5-38
6-1	Receptors and Exposure Pathways
6-2	Human Exposure Factor Input Parameters and Data Sources
6-3	Soil Ingestion Rates Used in this Risk Analysis
6-4	Exposed Fruit Consumption Data and Distributions

List of Tables (continued)

Numb	Page
6-5	Exposed Vegetable Consumption Data and Distributions
6-6	Root Vegetable Consumption Data and Distributions
6-7	Beef Consumption Data and Distributions 6-13
6-8	Dairy Products (Milk) Consumption Data and Distributions 6-14
6-9	Poultry Consumption Data and Distributions
6-10	Egg Consumption Data and Distributions 6-18
6-11	Fish Consumption Data and Distributions 6-19
6-12	Breast Milk Consumption Data and Distributions
6-13	Inhalation Rate Data and Distributions
6-14	Body Weight Data and Distributions 6-23
6-15	Exposure Duration Data and Distributions 6-24
7-1	Percentile Risk for Soil Ingestion Pathway
7-2	Percentile Risk for Exposed Produce Ingestion Pathway
7-3	Percentile Risk for Belowground Vegetable Ingestion Pathway
7-4	Percentile Risk for Poultry Ingestion Pathway
7-5	Percentile Risk for Egg Ingestion Pathway 7-6
7-6	Percentile Risk for Beef Ingestion Pathway 7-7
7-7	Percentile Risk for Milk Ingestion Pathway
7-8	Percentile Risk for Fish Ingestion Pathway
7-9	Percentile Risk for Air Inhalation Pathway 7-9
7-10	Percentile Risk for Breast Milk Ingestion Pathway
7-11	Multipathway Risks and Associated LADD for Adult and Child Farm Family
	Members—Baseline All Samples from 2001 NSSS 7-10
7-12	Multipathway Risks for Adult and Child Farm Family Members—300 TEQ
	Cutoff Limit for Samples from 2001 NSSS 7-10
7-13	Multipathway Risks for Adult and Child Farm Family Members—100 TEQ
	Cutoff Limit for Samples from 2001 NSSS
8-1	Results of Sensitivity Analysis by Pathway 8-10
9-1	Assessment Endpoints for the Ecological Receptors of Concern
9-2	Dioxin Congeners Assessed in the SERA 9-5
9-3	Wildlife Receptors for the Biosolids SERA
9-4	Values and Assumptions for the SERA
9-5	Phase 1 Receptors
9-6	Selected Diet Items for Phase 1 Receptors
9-7	Receptors Evaluated in Phase 2
9-8	Phase 1 Results
9-9	Screening Results from Phase 2

Acronyms and Abbreviations

ADD	Average daily dose
Ah	Aryl hydrocarbon
AhR	Ah receptor
AHF	Altered hepatocellular foci
AML	Arc Macro Language
BAF	Bioaccumulation factor
BEF	Bioaccumulation equivalence factor
BSAF	Biota-sediment accumulation factor
CI	Confidence intervals
CSF	Cancer slope factor
CV	Coefficients of variation
CWA	Clean Water Act
DW	Dry weight
ED	Effective dose
EFH	Exposure factors handbook
EGF	Epidermal growth factor
EPA	U.S. Environmental Protection Agency
ER	Estrogen receptor
FC	Field capacity
foc	Fraction organic carbon
GIRAS	Geographic retrieval and analysis system
GIS	Geographic information systems
GSCM	Generic soil column model
HQ	Hazard quotient
HUC	Hydrological unit code
I-P	Initiation-promotion
IARC	International agency for research on cancer
ISCST3	Industrial source complex, short-term model, version 3
IUPAC	International Union of Pure and Applied Chemists
LADD	Lifetime average daily dose
LOAEL	Lowest observed adverse effects level
LOEC	Lowest observed effects concentration
MAF	Moisture adjustment factor
MATC	Maximum allowable toxicant concentration
NOAEL	No observed adverse effects level

Acronyms and Abbreviations (continued)

NIOSH	National Institute of Occupational Safety and Health
NSSS	National sewage sludge survey
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo-p-dioxin
PCDF	Polychlorinated dibenzofuran
POTW	Publicly owned treatment works
QC	Quality control
RSD	Relative standard deviations
SAMSON	Solar and meteorological surface observation network
SCRAM	Support center for regulatory air models
SCS	Soil conservation service
SERA	Screening ecological risk assessment
SMR	Standardized mortality ratio
STATSGO	State soil geographic
Т3	Trophic level 3
T4	Trophic level 4
TCDD	Tetrachlorodibenzo-p-dioxin
TEF	Toxic equivalency factor
TEQ	Toxic equivalency quotient
UAC	Unit air concentration
UDPGT	Uridine diphosphate-glucuronyltransferases
USDA	U.S. Department of Agricultural
USGS	U.S. Geological Survey
USLE	Universal soil loss equation
WHO	World health organization
WP	Wilting point
WW	Wet weight
WWTP	Wastewater treatment plant

1.0 Introduction

1.1 Background

In February 1993, the U.S. Environmental Protection Agency (EPA) published the Standards for the Use or Disposal of Sewage Sludge (40 CFR Part 503). This regulation lists management practices and pollutant limits that protect public health and the environment from the reasonably anticipated adverse effects of pollutants in municipal biosolids (formerly referred to as "sewage sludge") when the biosolids are land-applied, placed on a surface disposal site, or fired in a biosolids incinerator. The Part 503 rule published in February 1993 is known as the Round One Biosolids Regulation. Section 405 of the Clean Water Act (CWA) requires EPA to publish a Round Two Biosolids Regulation, which will contain limits for pollutants not regulated in Round One.

Pollutants considered but not regulated under Round One were again considered under Round Two for potential regulation. Subsequently, EPA conducted preliminary exposure analyses in a Comprehensive Hazard Identification exercise to determine which of the 31 pollutants should be on the final pollutant list for potential regulation under Round Two (U.S. EPA, 1996). Based on the results of those analyses, three groups of pollutants were placed on the pollutant list for Round Two: polychlorinated dibenzo-p-dioxins (PCDDs, or dioxins), polychlorinated dibenzofurans (PCDFs, or furans), and coplanar polychlorinated biphenyls (PCBs).

This document describes the risk assessment conducted to determine the concentrations of dioxins, furans, and PCBs that can be present in biosolids and remain "protective" (below a specified level of risk) of human health. This risk-based concentration limit was generated by evaluating cancer risks for individuals (receptors) who may be exposed to these constituents if biosolids are applied to agricultural fields. The goal of this risk assessment was to estimate a national distribution of the incremental increase in individual lifetime risk of developing cancer due to exposure to dioxins, furans, and PCBs potentially present in the biosolids for farm families who apply biosolids as fertilizer or soil conditioner.

1.2 Summary of the Risk Assessment Process

For risk assessments, human health risks are generally assessed using a four-step process, as outlined in NRC (1994):

1. **Hazard Identification**. Identify the hazard posed by a pollutant by determining whether a pollutant may cause health hazards, quantifying environmental concentrations of the pollutant, describing the toxicity that may be caused by the

pollutant, and evaluating the conditions under which toxicity might be expressed in humans. Sources for this information include environmental monitoring data, as well as epidemiologic and animal studies.

- 2. Dose-Response Assessment. Establish the relationship between pollutant doses and the health effects in humans through data analysis (most often data from animal studies and occasionally from human studies) and modeling. Mathematical models may help determine the quantitative relationship between the dose of the pollutant and toxic responses; in particular, the potencies of suspected carcinogens have frequently been evaluated using such models.
- 3. **Exposure Assessment.** Use available data on constituent concentrations in materials of concern to estimate concentrations of constituents in environmental media and human contact with those media. Exposure assessments should consider fate and transport of material in the environment, routes of exposure, and pharmacokinetics of material once in the body. Data limitations on the environmental concentrations of interest often require the use of environmental modeling to provide relevant estimates of exposure, as they did in this risk assessment.
- 4. **Risk Characterization.** Integrate information from Steps 1, 2, and 3 to estimate the likelihood that any of the hazards associated with the pollutant will be manifested in exposed persons. In addition, EPA emphasizes the importance of clearly describing uncertainties in the risk assessment when characterizing risks.

The Draft Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds (U.S. EPA, 2000) describes Steps 1 and 2 of the process for this risk assessment. This document is referred to here as the Draft Dioxin Reassessment Document. The current document focuses on the last two steps of the process—exposure assessment and risk characterization.

1.3 Overview of Risk Assessment Methodology

The purpose of this analysis was to estimate the total concentrations of dioxins, furans, and PCBs that can be present in biosolids and remain protective of human health when biosolids are applied to agricultural land. The two final steps of the risk assessment process—exposure assessment and risk characterization—were conducted to arrive at the estimates.

Steps in the exposure assessment included

- Characterizing the management practices associated with the agricultural uses of biosolids,
- Describing the environmental settings where agricultural uses of biosolids may occur,

- Identifying scenarios under which contaminants in biosolids may be transported through the environment and/or the food chain to a human receptor, and
- Quantifying an individual's exposure to the contaminants resulting from the agricultural use of biosolids in the environment.

Steps in the risk characterization phase included

- Describing the individual's predicted risk from exposure to concentrations of constituents in environmental media, and
- Determining the risk-based concentrations for dioxins in biosolids that are protective of individual health when biosolids are applied to agricultural land.

EPA estimated protective constituent concentrations using a probabilistic analysis. A probabilistic risk analysis produces a distribution of risks for each receptor by allowing some of the parameters in the analysis to have more than one value. This type of analysis was ideal for this risk assessment because biosolids are generated nationwide and, therefore, may be used on agricultural fields anywhere in the United States. The probabilistic analysis not only captures the nationwide variability in biosolid application practices, it also captures the differences in the environmental settings (e.g., soils, meteorology) in which biosolids may be land-applied.

1.4 Document Organization

This document is organized into the following sections:

- Section 2, Hazard Identification/Dose-Response Assessment, summarizes the toxicological data supporting the health benchmark used in this analysis and the toxic equivalency factors (TEFs) used for the congeners evaluated in this risk assessment. These data are based on the Draft Dioxin Reassessment Document (U.S. EPA, 2000).
- Section 3, Risk Assessment Overview, describes the conceptual framework for the biosolids risk assessment. This section presents the conceptual framework for the human health risk assessment, including a description of biosolids and biosolids management practices, fate and transport modeling, exposure assessment, and calculation of protective biosolids concentrations, as well as a detailed explanation of the framework for the probabilistic analysis.
- Section 4, Input Data Characterization, presents the methodologies used to characterize the environmental setting, including delineation of the site layout and environmental setting (e.g., meteorology, climate, and soils). It also describes how the agricultural fields were characterized.
- Section 5, Estimating Exposure Point Concentrations, describes the models and methods used for source partition modeling, air dispersion and deposition

modeling, watershed and waterbody modeling, terrestrial food chain modeling, and aquatic food chain modeling.

- Section 6, Human Exposure Assessment, presents an overview of the human receptors, selected exposure pathways, and exposure scenarios considered for this assessment. It also presents exposure factors (i.e., values needed to calculate human exposure) used in the analysis and methods used to estimate dose, including lifetime average daily dose (LADD).
- Section 7, Human Health Risk Results, presents the methods used to characterize the risk posed to an individual. It describes the calculation methods used to generate risk-based constituent concentrations that are protective of human health.
- Section 8, Analysis of Variability and Uncertainty, discusses the methods that were used to account for variability and uncertainty in the risk assessment.
- Section 9, Screening Ecological Risk Assessment of Dioxins in Land-Applied Biosolids, describes the screening ecological risk assessment that was performed to investigate the potential for adverse ecological effects from dioxins in landapplied biosolids.

The following appendices provide supplemental technical information and supporting data:

- Appendix A, 2001 National Sewage Sludge Survey—Congener Concentration Data
- Appendix B, 2001 National Sewage Sludge Survey—Sample Selection Strategy
- Appendix C, Agricultural Parameters
- Appendix D, Congener-Specific Parameters for Source Partitioning and Fate and Transport Models
- Appendix E, Site Data
- Appendix F, Source Model for Land Application Units
- Appendix G, Air Dispersion and Deposition Modeling Input Files
- Appendix H, Direct and Indirect Exposure Equations
- Appendix I, Variables for Aboveground Fate and Transport
- Appendix J, Human Exposure Factors
- Appendix K, Sensitivity Analysis

- Appendix L, Ecological Assessment
- Appendix M, Climate Region Selections

1.5 References

- NRC. 1994. *Science and Judgement in Risk Assessment*. National Research Council, Committee on Risk Assessment of Hazardous Air Pollutants. Washington, DC: National Academy Press.
- U.S. EPA (Environmental Protection Agency). 1996. *Technical Support Document for the Round Two Sewage Sludge Pollutants*. EPA-822-R-96-003. Washington, DC: U.S. Government Printing Office.
- U.S. EPA (Environmental Protection Agency). 2000. *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*. EPA/600/P-00/001Bg. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. September.

2.0 Hazard Identification/Dose-Response Assessment¹

The constituents evaluated in this risk assessment are dioxins, furans, and PCBs contained in biosolids managed as a beneficial use on agricultural fields. All of these constituents were evaluated in the Draft Dioxin Reassessment Document (U.S. EPA, 2000), which concluded that "based on all available information, dioxins are potent animal toxicants with potential to produce a broad spectrum of adverse effects in humans." This risk assessment focuses on the potential of these biosolid constituents to act as human carcinogens. EPA characterizes 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as a Human Carcinogen based on weight of evidence and characterizes other dioxins, furans, and PCBs as Likely Human Carcinogens. The toxicity of all of the dioxin, furan, and PCB congeners considered in this analysis is based on the toxicity of the most highly characterized congener, 2,3,7,8-TCDD (U.S. EPA, 2000).

The cancer slope factor (CSF) for 2,3,7,8-TCDD used by EPA in this risk assessment is $1.56 \times 10^5 \text{ (mg/kg-d)}^{-1}$ (U.S. EPA, 1997). The CSF is defined as the upper bound on the slope of the dose-response curve in the low-dose region and is generally assumed to be linear. It is expressed as a lifetime excess cancer risk per unit exposure. The same slope factor is used to estimate cancer risks for both child and adult resident receptors. However, significant uncertainties exist concerning the estimation of lifetime cancer risks in children. This facter differs from the more recent CSF for TCDD proposed in the Draft Dioxin Reassessment Document (U.S. EPA, 2000). At the time this risk assessment was conducted, the decision was made to use the older value until a concensus was reached on a new value.

2.1 Adverse Effects in Humans and Animals

2,3,7,8-TCDD and related compounds have been reported to produce a wide variety of adverse effects in humans and animals, including cancer, reproductive and developmental effects, immunotoxicity, chloracne, diabetes, and several other less common health effects. This assessment will evaluate risk based only on the cancer endpoint because this is the only endpoint for which there are sufficient data to adequately support the assessment for all the dioxin-like congeners (U.S. EPA, 2000).

¹ This section summarizes and draws heavily from the material presented in *Part II: Health Assessment for* 2,3,7,8-*Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds* (U.S. EPA, 2000).

2.1.1 Mechanism of Action

The mechanisms of toxicity for dioxins are not completely understood but have been studied extensively, particularly for 2,3,7,8-TCDD. Many dioxins, furans, coplanar PCBs, and other structurally related halogenated aromatic hydrocarbons are believed to share a common mechanism of action related to similarities in their structures. The extraordinary potency of 2,3,7,8-TCDD in evoking a dose-related induction response, and the tissue specificity of enzyme induction led Poland and Glover (U.S. EPA, 2000, citing Poland and Glover, 1973) to postulate the existence of an induction receptor. This receptor, the Ah receptor (Ah for aromatic hydrocarbon), was identified in the cytosol of mouse liver cells (U.S. EPA, 2000, citing Poland et al., 1976) and in hepatic and extrahepatic tissues of a variety of laboratory animals, mammalian cell cultures, human organs and cell cultures, and tissues of nonmammalian species (U.S. EPA, 2000, citing Okey et al., 1994). 2,3,7,8-TCDD and structurally related compounds induce a wide range of biological responses, including alterations in metabolic pathways, body weight loss, thymic atrophy, impaired immune responses, hepatotoxicity, chloracne and related skin lesions, developmental and reproductive effects, and neoplasia. These responses are thought to be initiated by the binding of individual congeners (or ligands) with the aryl hydrocarbon (Ah) receptor. Of the many adverse responses observed both in humans and experimental animals after exposure to 2,3,7,8-TCDD, the ones that appear at the lowest dose (more sensitive) are developmental and reproductive effects, alterations in the immune response, and neoplasia.

Much evidence indicates that 2,3,7,8-TCDD acts via the intracellular protein, AhR, that functions in partnership with a second protein (known as the Ah receptor nuclear translocator, Arnt) to alter gene expression. In addition, receptor binding may result in release of cytoplasmic proteins, which alter the activity of cell regulatory proteins. Comparative data from animal and human cells and tissues suggest a strong similarity in response to dioxin-like chemicals across species. Biochemical and biological responses to dioxin exposure are sometimes considered adaptive, or reflective, of exposure but are within normal homeostatic limits and thus may not be considered adverse. However, many of these biochemical changes are potentially on a continuum of dose-response relationships, which leads to adverse responses. Given the possible mechanism of action, there are constraints on the possible models that account for dioxin's biological effects and on the assumptions used during the risk assessment. The linear relationship expected between ligand concentration and receptor binding may or may not be reflective of dose-response relationships for downstream events that require complex interactions. Biochemical and genetic analyses of these mechanisms suggest a novel regulatory system whereby a chemical signal can alter cellular regulatory processes.

The ability of 2,3,7,8-TCDD and other dioxin-like compounds to modulate a number of biochemical parameters is well recognized. Despite the ever-expanding list of these responses over the past 20 years and the work on the molecular mechanisms mediating some of these, there is still a considerable gap between our knowledge of the biochemical changes and the degree to which they are related to the more complex biological and toxicological endpoints.

TCDD-elicited activation of the Ah receptor has been clearly shown to mediate altered transcription oncogenes (cancer genes) and genes encoding growth factors, receptors, hormones, and drug-metabolizing enzymes. Based on the cumulative evidence available, it is presumed that

all of these processes are mediated by the binding of 2,3,7,8-TCDD to the AhR. The dioxin induces certain drug-metabolizing enzymes such as CYP1A1, CYP1A2, and CYP1B1 in different animal species including humans at body burdens as low as 1 to 10 ng TCDD/kg. These and other enzymes are responsible for the metabolism of a variety of exogenous and endogenous compounds. Several lines of experimental evidence suggest that these enzymes may be responsible for either enhancing or protecting against the toxic effects of a variety of agents, including known carcinogens, as well as endogenous substrates such as hormones. These effects are dependent upon the compounds and the experimental system examined. Several reports (U.S. EPA, 2000, citing Kadlubar et al., 1992; Esteller et al., 1997; Ambrosone et al., 1995; Kawajiri et al., 1993) provide evidence that higher levels of enzyme activity are associated with increased susceptibility to colorectal, endometrial, breast, and lung tumors. Changes in these enzymes by dioxin may play a role in chemical carcinogenesis. However, the exact relationship between the induction of these enzymes and any toxic endpoint observed following dioxin exposure has not been clearly established. Animal evidence supports the understanding that AhR plays a key role in tumor production.

The role of the epidermal growth factor (EGF) receptor in 2,3,7,8-TCDD-induced carcinogenicity has also been examined. EGF is a mitogen that stimulates the generation of mitotic signals in both normal and neoplastic cells, and its receptor and ligands have a variety of functions involved in cell transformation and tumorigenesis. It has been shown that 2,3,7,8-TCDD decreases the binding capacity of the plasma membrane EGF receptor for its ligand without changing the affinity constant (U.S. EPA, 2000, citing Abbott and Birnbaum, 1990; Hudson et al., 1985; Lin et al., 1991; Madhukar et al., 1984). The effects of 2,3,7,8-TCDD on the EGF receptor have been shown to require the Ah receptor (U.S. EPA, 2000, citing Lin et al., 1991).

The possible role of uridine diphosphate-glucuronyltransferases (UDPGT) on the carcinogenicity of 2,3,7,8-TCDD has also been studied. UDPGTs are thought to be a deactivation pathway for many environmental chemicals by increasing their water solubility, thereby facilitating excretion. 2,3,7,8-TCDD induces synthesis of at least one UDPGT isozyme (U.S. EPA, 2000, citing Lucier et al., 1986) by an Ah receptor-mediated mechanism (U.S. EPA, 2000, citing Bock, 1991). The results of Kohn et al. (1996) (U.S. EPA, 2000, citing Kohn et al., 1996) provide further support to the hypothesis that induction of UDPGT is an early event in the generation of thyroid tumors by 2,3,7,8-TCDD in the rat.

There is evidence that some carcinogenic responses to 2,3,7,8-TCDD are related to effects of 2,3,7,8-TCDD on the estrogen receptor (ER) and on estrogen metabolism. The responses appear to be tissue-specific. In rats, 2,3,7,8-TCDD increases liver tumor incidence, but decreases tumor incidence in mammary glands, the uterus, and the pituitary gland (U.S. EPA, 2000, citing Kociba et al., 1978a).

2.1.2 Epidemiologic Studies—Cancer Endpoint

Numerous studies have provided support for an association between exposure to dioxin and dioxin-like compounds and several types of cancer. Since the last formal EPA review of the human database relating to the carcinogenicity of TCDD and related compounds in 1988, a

number of new follow-up mortality studies have been completed. Among the most important of these are

- Studies of 5,172 U.S. chemical manufacturing workers by Fingerhut et al. (U.S. EPA, 2000, citing Fingerhut et al., 1991a) and Steenland et al. (U.S. EPA, 2000, citing Steenland et al., 1999) from the National Institute of Occupational Safety and Health (NIOSH) and an independent study by Aylward et al. (U.S. EPA, 2000, citing Aylward et al., 1996)
- A study of 2,479 German workers involved in the production of phenoxy herbicides and chlorophenols by Becher et al. (U.S. EPA, 2000, citing Becher et al., 1996, 1998) and by others in separate publications (U.S. EPA, 2000, citing Manz et al., 1991; Nagel et al., 1994; Flesch-Janys et al., 1995, 1998)
- A study of more than 2,000 Dutch workers in two plants involved in the synthesis and formulation of phenoxy herbicides and chlorophenols (U.S. EPA, 2000, citing Bueno de Mesquita et al., 1993) and subsequent follow-up and expansion by Hooiveld et al. (U.S. EPA, 2000, citing Hooiveld et al., 1998)
- A smaller study of 247 workers involved in a chemical accident cleanup by Zober et al. (U.S. EPA, 2000, citing Zober et al., 1990) and subsequent follow-up (U.S. EPA, 2000, citing Ott and Zober, 1996b)
- An international study of more than 18,000 workers exposed to phenoxy herbicides and chlorophenols by Saracci et al. (U.S. EPA, 2000, citing Saracci et al., 1991), with subsequent follow-up and expansion by Kogevinas et al. (U.S. EPA, 2000, citing Kogevinas et al., 1997).

Although uncertainty remains in interpreting these studies, because not all potential confounders have been ruled out, all indicate a potential association between exposure to dioxin and related compounds and increased cancer mortality. One of the strengths of these studies is that each has some exposure information that permits an assessment of dose response (U.S. EPA, 2000).

Results from several epidemiologic studies are summarized in Table 2-1. Observed numbers of cases, standardized mortality ratios (SMRs), and 95 percent confidence intervals (CI) are given for all cancers and for lung cancer, specifically. Although uncertainty remains concerning potential confounders in the studies, there is a strong inference regarding the carcinogenic potential of these constituents and the increased cancer mortality. Some of these studies have been judged adequate for use for fitting the dose-response models in the dioxin reassessment (U.S. EPA, 2000). In studies reviewed for the International Agency for Research on Cancer (IARC) monograph (U.S. EPA, 2000, citing IARC, 1997), the working group focused on the most exposed subcohorts with adequate latency and found that the most exposed groups had the highest incidence for all cancers combined and for lung cancer mortality. Although the increase was generally low (20 to 50 percent), it was highest in subcohorts with presumed

		All Cancers		Lung Ca		ancer
Reference ^a	Obs.	SMR	95% CI	Obs.	SMR	95% CI
International cohort						
Kogevinas et al. (1997) ^b	394	1.2	1.1–1.3	127	1.2	1.0–1.4
Industrial populations (high-exposure su	ıbcohor	ts)				
Fingerhut et al. (1991a) ^c (USA)	114	1.5	1.2–1.8	40	1.4	1.0–1.9
Becher et al. (1996) ^d (Germany)	105	[1.3]	[1.0–1.5]	33	[1.4]	[1.0–2.0]
Hooiveld et al. (1998) ° (Netherlands)	51	1.5	1.1–1.9	14	1	0.5–1.7
Ott and Zober (1996b) ^f (BASF accident)	18	1.9	1.1–3.0	7	2.4	1.0–5.0
Total ^g	288	[1.4]	[1.2–1.6]	[94]	[1.4]	[1.1–1.7]
<i>p</i> value		<0.00)1		< 0.0	1

Table 2-1. Summary of Combined Cohort and Selected Industrial Cohort Studieswith High Exposure Levelsa

CI = Confidence intervals.

Obs = Observed number of cases.

SMR = Standardized mortality ratios.

Adapted from IARC; Table 38 (U.S. EPA, 2000, citing IARC; Table 38, 1997); non-Hodgkin's lymphoma, soft tissue sarcoma, and gastrointestinal results not shown.

^a All references are as cited in U.S. EPA (2000).

^b U.S. EPA, 2000 citing Kogevinas et al. (1997): men and women >20 years since first exposure. These data include the cohorts of Fingerhut et al. (1991a,b), Becher et al. (1996), Hooiveld et al., (1998), the original IARC cohort (Saracci et al., 1991), and other cohorts.

^c Fingerhut et al. (1991a): men \geq 20 years latency and \geq 1 year exposure.

^d Becher et al. (1996): men, Cohort I and II, summed (Boehringer-Ingelheim, Bayer-Uerdingen cohorts).

^e Hooiveld et al. (1998): men and women, Factory A.

^f Ott and Zober (1996b): men, chloracne subgroup, ≥20 years latency. Data presented for lung cancer are all respiratory tract cancers combined.

^g Totals in square brackets are those calculated by the IARC Working Group.

heaviest exposure. This outcome is unlikely due to chance, and the increase in lung cancer is not explained by confounding exposure due to smoking. Positive dose-response trends in the German studies and increased risk in the longer-duration U.S. subcohort and the most heavily exposed Dutch workers support this view. These results are further substantiated by the increased mortality found in the Japanese rice oil poisoning accident where high levels of exposure to furans and PCBs were observed and were associated with increased incidence of lung and liver cancers. Although increases in cancer incidence at other sites (e.g., non-Hodgkin's lymphoma, soft tissue sarcoma, gastrointestinal cancer) have been reported, the data to associate them with exposure to dioxin-like chemicals are less compelling because of the limited numbers of observed tumors at any specific site (U.S. EPA, 2000).

2,3,7,8-TCDD and, by inference from more limited data, other dioxin-like compounds are potentially Multisite Carcinogens in the more highly exposed human populations that have been studied, primarily in adult males. 2,3,7,8-TCDD cancer experience for women may differ from that for men. Animal and mechanistic studies suggest different responses in males and females, but there are no data to adequately support this. Although the epidemiologic data are not sufficient by themselves to infer a causal association between exposure to TCDD and other dioxin-like chemicals and increased cancer in humans (U.S. EPA, 2000, citing IARC, 1997, and ATSDR, 1998) and although uncertainty remains, the epidemiologic data are generally consistent with results from studies of multiple laboratory animal species where dioxin-like compounds have clearly been identified as Multisite Carcinogens and Tumor Promoters. In addition, the findings of increased cancer incidence at multiple sites in occupationally exposed workers appear to be plausible given what is known about mechanisms of dioxin action. The epidemiological data, however, are insufficient to establish the shape of the dose-response curve below the range of observation in these occupationally exposed populations.

2.1.3 Animal Studies—Cancer Endpoint

Many animal studies have shown that 2,3,7,8-TCDD is a carcinogen based on long-term bioassays conducted in numerous species, including both sexes of rats and mice. According to the Draft Dioxin Reassessment Document, "TCDD is a nongenotoxic carcinogen because it is negative in most assays for DNA damage; however, it is a potent "promoter" and a weak initiator or noninitiator in two-stage initiation-promotion (I-P) models for liver and skin" (U.S. EPA, 2000). Multiple I-P studies show that induction of altered hepatocellular foci (AHF) is dose-dependent, exposure duration-dependent, and partially reversible. AHF induction is associated with liver cancer in rodents.

In addition to liver effects, TCDD is a potent cancer promoter in mouse skin (source of the CSF used). It is also characterized as a Multisite Carcinogen because it increases the incidence of tumors at sites distant from treatment sites. This association is substantiated by the fact that all long-term cancer bioassays have been positive in both sexes of both rats and mice.

2.2 Risk Characterization

Characterization of dioxin risks is based on an extensive amount of data. Characterization of the health hazard, modes of action, dose-response, and exposure all contribute to the dioxin risk. Subpopulations and developmental stages are included in this characterization.

EPA drew several solid conclusions regarding carcinogenicity based on its analysis (U.S. EPA, 2000):

- "Dioxin and related compounds can produce a wide variety of effects in animals and might produce many of the same effects in humans"
- "Dioxin and related compounds are structurally related and elicit their effects through a common mode of action"
- "EPA and the international scientific community have adopted toxic equivalency of dioxin and related compounds as prudent science policy"
- "Complex mixtures of dioxin and related compounds are highly potent, likely carcinogens."

Adequate evidence supports the belief that humans are likely to respond to exposure to dioxin with a broad spectrum of effects. These effects appear to begin with biochemical changes at or near background levels of exposure (concentrations measured in the ambient environment), increasing in severity as body burdens increase. Enzyme induction, changes in hormone levels, and altered cellular function may represent effects of unknown significance at the lowest exposure levels. Adverse effects, including cancer, may not be detectable until exposure reaches 10 to 100 times background levels. Humans most likely fall into the middle of the range of sensitivity among mammals, neither extremely sensitive nor extremely insensitive to the effects of dioxin.

Currently, there have been few cohorts with dioxin exposure high enough to raise body burdens significantly over background levels. In those studies, few clinically significant noncancer effects were detected.

Most, if not all, observed effects of dioxin can be described in a series of common biological steps. The initial step and the single largest determinant of toxicity, including tumor development, is binding of dioxin and related compounds to the AhR. Dioxin and dioxin-related compounds exist as complex mixtures in nature, and the biological activity of the mixture can be estimated using relative potency values, coupled with an assumption of dose additivity. This exposure has evolved to the use of Toxic Equivalency Quotients (TEQs) in risk assessment. With this approach, cumulative exposures of AhR-mediated chemicals can be translated with increasing confidence to human responses.

A weight-of-evidence evaluation concluded that mixtures of dioxin and related compounds are Strong Cancer Promoters and likely pose a cancer hazard to humans. The data for complex mixtures of dioxins, furans, and coplanar PCBs constitute "strong evidence" of carcinogenicity (U.S. EPA, 2000) and include epidemiological cancer observations and unequivocal positive responses in both sexes, multiple species, multiple sites, and different routes. Laboratory evidence supports the epidemiological results, suggesting dioxin exposure contributes to carcinogenic response, but it is insufficient to confirm a causal relationship. Human studies alone cannot demonstrate this causal relationship.

2.3 Dose-Response and Slope Factors

Current knowledge of the mechanisms of action of dioxin, receptor theory, and the available dose-response data are insufficient to establish a nonlinear procedure for estimating cancer potency. Both cancer and noncancer effects appear to result from qualitatively similar modes of action; thus, the potential for either type of effect is considered equal. A common metric for comparison is the effective dose (ED). In the observable range of 1 percent excess response, quantitative differences between cancer and noncancer EDs are relatively small.

2.3.1 Human and Animal Studies

Dioxins and other xenobiotics that operate through receptor-binding mechanisms will, according to theory, follow a linear dose-response binding. This theory is supported by empirical findings. The biochemical and transcription reactions for dioxins may also follow linear dose-response kinetics. More distal toxic effects could be linear or threshold (sublinear) depending on (1) the toxic mechanism, (2) the location on the dose-response curve, and (3) interactions with other processes. Too much data variability exists to clearly distinguish statistically between dose-response curve options and to determine whether dose-response follows linear, supra/sublinear, power curve, or threshold kinetics. Toxic effects at higher doses may be more likely to result from multiple cellular changes and thus be less likely to follow linear relationships. Empirical dose-response data from cancer studies—both epidemiological and bioassays—do not provide consistent or compelling support to either threshold or supralinear models. Thus, the default linear extrapolation policy is used.

Current human body burdens are already substantially along the dose-response curve. Margins of exposure between population levels of background exposure and the empirical 1 percent effect levels due to additional exposure for a number of biochemical and toxic effects are on the order of less than 1 to 2 orders of magnitude. Therefore, the extrapolation between observed effects and background levels is not large.

Because human data were available for cancer dose-response analysis and because EPA wanted to stay within the estimated range of responses, EPA chose a 1 percent excess risk as a point of departure (U.S. EPA, 2000). Restricting the analysis to log-linear models, human cancer effective doses at the 1 percent excess risk level $(ED_{01}s)$ were estimated to range from 5.7 to 250 ng/kg. In similar estimates based on animal studies, most $ED_{01}s$ ranged from 14 to 500 ng/kg.

Calculations of a CSF based on the extrapolation of lower ED_{01} to background response rates based on human data yielded a CSF estimate of approximately 1×10^{-3} per pg TCDD per kg body weight per day. Based on animal data, a similar CSF of 1.4×10^{-3} per pg TCDD/kg body weight/day was estimated (U.S. EPA, 2000). "The Agency, although fully recognizing the range and the public health conservative nature of slope factors that make up the range, suggests the use of the 1×10^{-3} per pg TEQ/kgBW/day as an estimator of upper bound cancer risk for both background intakes and incremental intakes above background" (U.S. EPA, 2000).

For this risk assessment, however, the current EPA-sanctioned CSF was used because of the draft nature of the most recent dioxin risk assessment document. EPA has used a CSF for 2,3,7,8-TCDD of 1.56×10^{-1} (pg/kg-d)⁻¹ and unit risk estimates of 3.3×10^{-5} (pg/m³)⁻¹ for inhalation exposure and 4.4×10^{-3} (pg/L)⁻¹ for drinking water exposure. These values are now under review and are subject to change; they are based on an oral study in which rats were exposed to 2,3,7,8-TCDD in their diet for 720 days, resulting in tumors of the respiratory system and liver (U.S. EPA, 2000; U.S. EPA, 2000, citing Kociba et al., 1978). The inhalation unit risk estimate was based on route-to-route extrapolation from the oral CSF, assuming 75 percent absorption (U.S. EPA, 1997, 2000).

2.3.2 Toxicity Equivalency Factors

Over the past decade, the scientific community, led by the World Health Organization (WHO), has developed a system of TEFs that relate the toxicity of each dioxin, furan, and PCB congener to the toxicity of 2,3,7,8-TCDD. The TEFs used in this analysis are those developed by the WHO in 1998 (U.S. EPA, 2000, citing Van den Berg et al., 1998) and recommended in the Draft Dioxin Reassessment Document (U.S. EPA, 2000).

These TEFs, presented in Table 2-2, were multiplied by the CSF of $1.56 \times 10^5 \text{ (mg/kg-d)}^{-1}$ currently recommended by EPA to determine the congener-specific CSF that was used to estimate congener-specific risks.

Congener	WHO—98			
Polychlorinated dibenzodioxins				
2,3,7,8-TCDD	1			
1,2,3,7,8- PeCDD	1			
1,2,3,4,7,8-HxCDD	0.1			
1,2,3,7,8,9-HxCDD	0.1			
1,2,3,6,7,8-HxCDD	0.1			
1,2,3,4,6,7,8-HpCDD	0.01			
1,2,3,4,6,7,8,9-OCDD	0.0001			
Polychlorinated dibenzofurans	·			
2,3,7,8-TCDF	0.1			
1,2,3,7,8-PeCDF	0.05			
2,3,4,7,8-PeCDF	0.5			
1,2,3,4,7,8-HxCDF	0.1			
	(continued)			

Table 2-2. Toxic Equivalency Factors

Polychlorinated dibenzofurans		
1,2,3,7,8,9-HxCDF		0.1
1,2,3,6,7,8-HxCDF		0.1
2,3,4,6,7,8-HxCDF		0.1
1,2,3,4,6,7,8-HpCDF		0.01
1,2,3,4,7,8,9-HpCDF		0.01
1,2,3,4,6,7,8,9-OCDD		0.0001
Polychlorinated biphenyls		
IUPAC #	Structure	WHO—98
77	3,3',4,4'-TCB	0.0001
81	3,4,4',5-TCB	0.0001
105	2,3,3',4,4'-PeCB	0.0001
114	2,3,4,4',5-PeCB	0.0005
118	2,3',4,4',5-PeCB	0.0001
123	2',3,4,4',5-PeCB	0.0001
126	3,3',4,4',5-PeCB	0.1
156	2,3,3',4,4',5-HxCB	0.0005
157	2,3,3',4,4',5'-HxCB	0.0005
167	2,3',4,4',5,5'-HxCB	0.00001
169	3,3',4,4',5,5'-HxCB	0.01
170	2,2',3,3',4,4',5-HpCB	-
180	2,2',3,,4,4',5,5'-HpCB	-
189	2,3,3',4,4',5,5'-HpCB	0.0001

 Table 2-2. (continued)

2.4 References

- ATSDR. 1998. *Toxicological Profile for Chlorinated Dibenzo-p-dioxins*. Agency for Toxic Substances and Disease Registry, Department of Human and Health Services. Atlanta, GA.
- U.S. Environmental Protection Agency (EPA). 1997. Chapter 8. Dose-response modeling for 2,3,7,8-TCDD. January 1997 Workshop Review Draft. EPA/600/P-92/001C8.
- U.S. Environmental Protection Agency (EPA). 2000. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. EPA/600/P-00/001Bg. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. September.

3.0 Risk Assessment Overview

This section describes the conceptual framework for the risk assessment conducted for dioxins, furans, and coplanar PCBs in biosolids. Section 3.1 presents the conceptual framework for the human health risk assessment. This includes a description of biosolids and the agricultural practices, fate and transport modeling, exposure assessment, and calculation of risk-based concentrations of these constituents in biosolids. Section 3.2 describes the framework for the probabilistic analysis.

3.1 Human Health Risk Assessment

The human health risk assessment for the evaluation of dioxins, furans, and coplanar PCBs is intended to evaluate nationwide risk to farmers and the children of farmers who apply biosolids to their crop lands and pastures and consume home-produced foods. The concentrations of the 29 dioxin, furan, and PCB congeners used in this risk assessment were derived from the 2001 National Sewage Sludge Survey (NSSS, see Appendix A).

Biosolids are solid, semisolid, or liquid residue generated during the treatment of domestic sewage in municipal wastewater treatment works. When biosolids are land-applied, surface-disposed, or fired in a biosolids incinerator, the applicable requirements in Part 503 of the CWA must be met. Part 503 contains both risk-based requirements and technology-based requirements. The EPA risk assessment approach used in this analysis was designed to produce a scientifically defensible evaluation of the concentrations of dioxins in biosolids that are protective of human health when biosolids are applied to agricultural land.

3.1.1 Application of Biosolids to Agricultural Land

Biosolids may be applied to agricultural land that may be used as crop land or as pasture for cattle. These applications occur nationwide; therefore, a probabilistic risk assessment was structured to capture the variability in climate, soil, and agricultural practices throughout the United States. The 48 contiguous states were subdivided into 41 climatic regions assumed to be sufficiently uniform to be represented adequately by climate data from any reporting meteorologic station within the bounds of the region. These geographic regions were also used as the basis for identifying a representative farm size and a distribution of soil types on the farms.

The data sources for characterizing the distribution of agricultural field sizes are

U.S. DOC (Department of Commerce). 1989. 1987 Census of Agriculture, Volume 1, Geographic Area Series State and County Data.

 U.S. DOC (Department of Commerce). 1994. 1992 Census of Agriculture Volume 1, Geographic Area Series State and County Data. Bureau of the Census, Washington, DC.

3.1.2 Constituents of Concern

Constituents of concern for the Round Two Biosolids Regulation are dioxins, furans, and coplanar PCBs. These pollutants are similar in many respects, including fate and transport and toxicology. The human health benchmarks for these constituents are related by a system of TEFs to the health benchmark for the most well-characterized of these compounds: 2,3,7,8-TCDD. Table 2-2 presents the constituents of concern evaluated in this risk assessment.

3.1.3 Site Configuration and Environmental Setting

A single conceptual site layout was used to define the relationship between the agricultural site and the human receptors evaluated in this risk assessment. The same site layout was used for the 41 geographical regions. The environmental characteristics of the regions provide the data used for the environmental characteristics of the sites.

3.1.3.1 <u>Conceptual Site Layout</u>. Figure 3-1 depicts the conceptual site layout for the agricultural application of biosolids. Farmers are assumed to apply biosolids to crop land where exposed fruits, vegetables, and root crops are produced and pasture land where beef and dairy cattle are grazed. The farmers are assumed to live on a small strip of land (the buffer area) between the crop or pasture land and the stream. The farmer raises free-range chickens in a yard that is also located in the buffer area. Beyond the buffer area is a third-order stream.¹ This order-size stream was chosen because it is the smallest size stream that is assumed to be fishable. The farmer, his lactating wife, their infant, and older children may come in contact with dioxin congeners via several routes of exposure.

3.1.3.2 <u>Regional Environmental Setting</u>. Biosolids are produced and managed in all states in the continental United States; therefore, environmental settings used in this risk assessment are developed to be representative of each geographical region in the United States. The primary objective in characterizing a regional environmental setting is to represent the variation in environmental conditions that results from the geographic diversity in the United States. Within each of the 41 representative climatic regions, a meteorological station was identified to represent the climatic and meteorological conditions for that geographic area. The 41 climatic regions used for modeling are shown in Figure 3-2.</u>

¹ A third-order stream is defined as the joining of two second-order channels. Second-order channels are formed from the joining of two first-order channels. First-order channels are the smallest finger-tip tributaries in a watershed. Thus, the actual dimensions of streams of each order may vary.



Figure 3-1. Agricultural application conceptual site model.



Figure 3-2. Map of 41 climatic regions.

The following characteristics are assumed to be associated with the 41 regions:

- Soil characteristics for land having agricultural use (crops or pasture)
- Representative meteorological and climatic data
- Agricultural field sizes (medium farm size for the region).

3.1.4 Exposure Point Estimates

A series of models was used to estimate concentrations of congeners in the environment with which individuals may come into contact. A source partition model was used to estimate environmental releases of each congener from the crop land, or pasture, where biosolids are applied. These estimated environmental releases provide input to the fate and transport models to estimate media concentrations for dioxins, furans, and coplanar PCBs in air, soil, above- and belowground produce, and surface water. A farm food chain model was used to estimate environmental concentrations of these congeners in home-produced produce, poultry, eggs, beef, and dairy products. These models are discussed in detail in Section 5.0. Aquatic bioconcentration factors were used to estimate concentrations and TEQ concentrations (both congener-specific and total).

3.1.4.1 <u>Source Partition Modeling</u>. Biosolids application to pastures is assumed to differ from biosolids application to crop land, and the differences affect the behavior of constituents in the environment. The source partition model requires information on farm area, biosolids characteristics (e.g., moisture content, congener concentrations), and environmental setting (e.g., precipitation, temperature, soil characteristics) to estimate environmental releases.

Crop Land. Biosolids applied to crop land are tilled into the soil; thus, the dioxins are thoroughly mixed with the top 20 cm of soil. The congeners are released to the air from the soil as vapors and particulates; the crops take the congeners in through the air; and congeners bound to the soil particles are eroded onto and through the residential property and chicken yard and into the nearby stream.

Pasture. Biosolids applied to the pasture are not tilled and, thus, are not actively mixed with the soil. However, over time the congeners penetrate into the soil and are assumed to be mixed in the top 2 cm of soil. The congeners are released to the air from the soil surface; grasses take up the congeners through air-to-plant transfer. The congeners bound to the biosolids and to the soil are eroded onto and through the residential property and chicken yard and into the nearby stream (where they are mixed with the soil estimated to be eroded from the adjacent crop land).

3.1.4.2 <u>Fate and Transport Modeling</u>. Fate and transport algorithms describe the mechanism by which the congeners move from the source through the environment. As described above, a source partition model was used to determine the amount and nature of congener released from the agricultural field. A multimedia approach was used to characterize the movement of the dioxins through the environment. This approach considered atmospheric concentrations, atmospheric deposition, soil concentrations, and sediment concentrations in the waterbody.

3.1.4.3 <u>Farm Food Chain Model</u>. A farm food chain model was used to estimate the concentration of congeners in aboveground produce, belowground produce, poultry, eggs, beef, and dairy products. Aboveground produce is affected via vapor transfer and deposition of dioxins in the air. Belowground produce is affected only by uptake of dioxins from tilled soil. The concentration of dioxins was also estimated for the forage and silage consumed by cattle. Forage is assumed to be grown on the untilled pasture, whereas silage is assumed to be harvested from the tilled fields. Agricultural field size was estimated as the median agricultural field size for each of the 41 climatic regions modeled, and it varied among the climatic regions. Dioxins that are ingested by animals were partitioned to the lipid fraction of each animal product.

3.1.4.4 <u>Aquatic Food Chain Model</u>. An aquatic food chain model was used to estimate the concentration of dioxins, furans, and coplanar PCBs in fish populations. These congeners are eroded from the agricultural fields where they are managed into the sediment of the adjacent stream where they can contaminate fish. The uptake into fish from the sediment is represented by congener-specific bioaccumulation constants called biota sediment accumulation factors (BSAFs). Trophic level 3 (T3) and 4 (T4) fish were considered in this analysis. Trophic level 3 fish are those that consume invertebrates and plankton. Trophic level 4 fish are those that consume other fish. Most of the fish that humans consume are T4 fish (e.g., salmon, trout, walleye, bass) and medium to large T3 fish (e.g., carp, smelt, perch, catfish, sucker bullhead, sauger).

3.1.5 Assessing Human Exposures

Individuals may come into contact with dioxins in biosolids applied to agricultural fields through a variety of pathways.

3.1.5.1 <u>Human Receptors</u>. Four individual receptors were evaluated in this assessment:

- Adult farmer (members of the farm family who begin exposure as adults)
- Child of farmer (members of the farm family who begin exposure in childhood)
- Infant of farmer (infant born to the farm family during the exposure period)
- Fisher (adult member of the farm family who fishes in the stream adjacent to the farm where biosolids are applied).

These receptors reflect the range of possible individual exposures for direct and indirect exposure pathways. Child exposures were evaluated based on an initial start age of 1 to 6 years. This age range was selected because this represents the highest consumption rate (intake/body weight) for most of the exposure pathways evaluated in this risk assessment. The child was assumed to age through a selected exposure duration; thus, because consumption rates vary over time, childhood exposures reflect a time-weighted consumption rate for the selected exposure duration.

3.1.5.2 <u>Exposure Pathways</u>. Environmental media and exposure pathways were modeled in this assessment for agricultural and fisher scenarios. Exposure pathways are either direct, such as inhalation of ambient air, or indirect, such as the farm food chain pathways. The exposure pathways considered in this assessment were

- Inhalation of ambient air
- Incidental ingestion of soil in the buffer
- Ingestion of above- and belowground produce grown on the crop land
- Ingestion of beef and dairy products from the pasture
- Ingestion of home-produced poultry and eggs from the buffer
- Ingestion of fish from the nearby waterbody.

3.1.6 Toxicity Assessment and Risk Characterization

The single risk characterization endpoint used in this risk assessment was the incremental individual lifetime risk of developing cancer. To characterize this risk from human exposure to dioxins, furans, and PCBs, TEFs were used with the CSFs developed for 2,3,7,8-TCDD and the congener-specific exposure assessment results. The toxicity of all other dioxin, furan, and PCB congeners was determined based on the relationship of each congener to the toxicity of 2,3,7,8-TCDD. The series of TEFs used in this analysis was developed by the WHO and published in 1998 and are recommended for use by the Draft Dioxin Reassessment Document (U.S. EPA, 2000). These TEFs are applied to the CSF for TCDD (1.56×10^5) to determine the congener-specific health benchmarks used in this risk analysis.

3.2 Probabilistic Method for Determining Exposure Point Concentrations

The primary objective of this assessment was to estimate risk using a probabilistic (Monte Carlo) approach. The probabilistic analysis produces a nationwide distribution of risk for each receptor type by varying parameter values over multiple iterations of the model.

An overview of the probabilistic analysis follows, and this analysis method is discussed in greater detail throughout this document. The results of this analysis are presented in Section 7.0.

The probabilistic analysis was performed using a Monte Carlo simulation. In a Monte Carlo simulation, the models are run for a fixed number of iterations, each producing a single result (e.g., a single estimate of cancer risk). For this assessment, 3,000 iterations were run in the Monte Carlo simulation; therefore, the output of the probabilistic analysis was a distribution of 3,000 values. This distribution represents the distribution of possible outcomes, which reflects the underlying variability in the data used in the analysis. These results were then used to identify risk at various percentile levels (e.g., 90th percentile risk value).

Some model input parameters used in the Monte Carlo simulation were drawn from statistical distributions. For others, variability was associated with variable locations; thus, location variability was explicitly considered in the setup of the data used for the probabilistic analysis. For location-dependent parameters, locations were first selected at random with equal

probability of occurrence based on the 41 climatic regions. These regions defined a set of related environmental conditions (e.g., soil type, hydrogeologic environment) that characterized the environmental setting. All location-specific parameters (e.g., rainfall) thus remained correlated while allowing variability within and among locations. Location-dependent parameters are discussed in Section 4.3.

3.3 References

- U.S. DOC (Department of Commerce). 1989. 1987 Census of Agriculture, Volume 1, Geographic Area Series State and County Data. Bureau of Census, Washington, DC.
- U.S. DOC (Department of Commerce). 1994. 1992 Census of Agriculture Volume 1, Geographic Area Series State and County Data. Bureau of the Census, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 2000. *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds.* EPA/600/P-00/001Bg. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. September.
4.0 Input Data Characterization

This risk assessment provides a national characterization of biosolids applied to agricultural fields. How this practice is characterized in terms of the physical dimensions of the farms, agricultural application practices, and climatic region is fundamental to the construction of scenarios for modeling.

The foundation for the Monte Carlo simulation is the data describing the scenario that defines each of the Monte Carlo iterations. Specifically for this analysis, 3,000 iterations were completed to define a distribution of risk for each pathway in the agricultural application scenario. Compiling the source data for this analysis required characterizing the environmental setting in which biosolids application to agricultural fields is likely to occur.

Section 4.1 presents an overview of the source data development procedure. Section 4.2 summarizes development of the biosolids management scenarios evaluated in this risk assessment. Section 4.3 presents the methodologies used to characterize the environmental setting, including delineation of the environmental setting (e.g., meteorology, climate, and soils). Section 4.4 describes the characterization of the agricultural field size.

4.1 Input Data Development Procedure

To capture the national variation in agricultural practices for the Monte Carlo analysis, a database of representations of agricultural practices was developed that contains all of the parameters needed to describe the application of biosolids to crop land or pasture. These source data, which provide the input data for the fate and transport modeling, are organized into two source data files, one for pastures and one for crop lands. The source data files contain information on climatic region and biosolid characteristics and descriptions of agricultural practices. Agricultural application rates, frequencies, and duration for the use of biosolids on crop land and pastures were selected to be consistent with common agronomic practices.

4.2 Characterization of Biosolids

Biosolids in this risk assessment were assumed to be characterized by a single set of physical and chemical parameter values. Thus, the physical characteristics of biosolids (e.g., bulk density, percent solids, and fraction organic carbon) required to estimate emissions using the source models used the biosolids characteristics provided by EPA. If biosolid-specific physical characteristics were not available from EPA for a specific parameter, silt soil parameters were used to represent biosolids. Table 4-1 provides the biosolids characteristics used in this analysis.

Characteristic	Parameter Value	Units	Source
Dry bulk density (BD)	1.6	g/cm ³	Technical Support Document for Land Application of Sewage Sludge (U.S. EPA, 1992)
Fraction organic carbon (foc)	0.4	Unitless	
Percent solid	Variable	Volume percent	2001 NSSS (U.S. EPA, 2001)
Porosity	0.4	Unitless	Based on Carsel & Parrish (1988)
Silt content	2.2 to 21 Uniform distribution	Mass percent	Table 13.2.2-1 AP-42 (U.S. EPA, 1995a)

4.2.1 Concentrations of Dioxin and Furan Congeners

The concentrations of dioxin and furan congeners in biosolids were obtained from the NSSS 2001 (U.S. EPA, 2001). This survey analyzed more than 100 samples of biosolids for the 17 dioxin and furan congeners and 12 PCBs of concern in this risk assessment. These analytical results are presented in Appendix A of this document. The biosolids samples were obtained from 94 municipal wastewater treatment facilities. The following steps outline how the variable concentrations were selected for use in the variable concentration Monte Carlo risk analysis:

- 1. **Identify one representative sample for each wastewater treatment plant** (WWTP) in the NSSS. The 100 analyzed samples in the NSSS represented 94 WWTPs. For the facilities with multiple types of biosolids, multiple samples were taken and analyzed. These multiple samples were combined based on the percent of the total biosolids volume represented by each sample to produce a single weighted average concentration for each congener. This process produced a single representative sample for each congener for each facility.
- 2. Select samples from the distribution. The frequency with which a facility was selected from the distribution of sample data was weighted according to the quantity of biosolids produced by the facility. The facilities were placed into one of four strata depending on the quantity of biosolids produced at that facility. The strata were given weights of 0.0035, 0.03902, 0.23027, or 0.71921. The weighting method is the same as that used for the 1988 NSSS samples and is described in detail in Appendix B.
- 3. **Use the concentrations in sample selected for all congeners in the sample.** When a facility was selected, that facility's sample was used. The concentrations for each congener in the sample were thus kept correlated throughout the analysis.

4. **For congener concentrations below the analytical detection limit, use a value of one-half the analytical detection limit.** When the congener concentrations were below the minimum detection limit, a concentration equal to one-half of the detection limit was assumed.

Each iteration of the Monte Carlo analysis evaluated one sample from a single facility from the distribution of dioxin, furan, and PCB samples. Thus, each total TEQ risk result represents the total risk from all 29 congeners (17 dioxin and furan congeners and 12 PCBs). For each iteration, the concentration of dioxins in the biosolids was assumed to remain constant for the entire period of application. Thus, some iterations in this analysis represented the repeated application of biosolids containing up to 700 ng/kg of dioxin TEQ to agricultural land. Comparisons of samples for the same facility from the EPA 1988 NSSS and the EPA 2001 NSSS indicate that high dioxin TEQ concentrations in sewage sludge (i.e., > 100 ppt TEQ) do not appear to remain constant over time. For this reason, the use of a constant high dioxin TEQ concentration in biosolids may somewhat overestimate the risk for those iterations.

4.2.2 Agricultural Application of Biosolids

Biosolids were assumed to be applied to agricultural land at appropriate agronomic rates. Agronomic rates vary according to soil type, crop type, biosolid characteristics, and climatic conditions. Currently, Section 503 rules limit application of biosolids based on loading of metals to the soil. For this risk assessment, the following assumptions were made about the application of biosolids. These assumptions reflect a distribution of agricultural practices common throughout the United States:

- Biosolids are applied at a rate of 5 to 10 metric tons per hectare per application (uniform distribution).
- Applications occur once every 2 years.
- Application continues for up to 40 years (20 applications).
- Crop land is tilled to a depth of 20 cm multiple times during the year.
- Pasture land is not tilled; thus, biosolids are assumed to be incorporated into only the top 2 cm of soil.

Application rates for biosolids were not varied with location in this analysis and were assumed to be uniform nationwide.

4.3 Site Characterization

The site characteristics used in this analysis were based on one conceptual site layout and regional characterization of environmental parameters. The conceptual site layout defines the area in the immediate vicinity of the farm applying biosolids and defines the geographic relationship among important features, such as the crop land, pasture, residence, chicken yard,

and stream. A single conceptual site layout was evaluated at each of the 41 climatic regions in the analysis.

4.3.1 Conceptual Site Layouts

This risk assessment was based on a conceptual site layout rather than on site-specific layouts. The conceptual site layout was designed to capture possible relationships between management practices for biosolids and individual receptors.

The conceptual or general site layouts are shown in Figure 3-1, which shows the agricultural field, the buffer area (i.e., an area between the agricultural field and the stream or the monofill), and the residence.

The agricultural field area was assumed to be the median area for farms in each climatic region. The agricultural field sizes were taken from the county-level data provided in the Census of Agriculture. The Census of Agriculture (U.S. DOC, 1989, 1994) provides periodic and comprehensive statistics about agricultural operations, production, operators, and land use. It is conducted every 5 years for years ending in 2 and 7. Its coverage includes all operators of U.S. farms or ranches (Division A, SIC 01-02) that sold or normally would have sold at least \$1,000 worth of agricultural products during the census year. In 1992, approximately 1.9 million operators produced \$162 billion in crops and livestock. Data for 1987 and 1992 were averaged. The median farm size was determined for all counties in each of the 41 climatic regions. From this distribution, the median farm size for each climatic region was determined. The agricultural field size used in this analysis are presented in Table 4-2. The agricultural field size was important in this analysis for the air dispersion and deposition and soil erosion pathways. The larger the source, the greater the off-site concentrations due to air deposition and erosion.

Adjacent to the agricultural field is a waterbody that is assumed to be 5.5 m wide and 0.21 m deep. These values are typical of a third-order stream (van der Leeden et al., 1990). The stream length is determined by the width of the agricultural field. Surface area of the stream is, therefore, determined by the fixed width (5.5 m) and the size of the agricultural field, which varies by climatic region as described above. The fishing scenario estimated risks to adult fishers who caught and consumed fish on a recreational basis from this waterbody.

4.3.2 Regional Environmental Setting

The regional environmental setting approach was developed as a way to include the variability associated with geographic locations throughout the United States. The boundaries of the climatic regions used in this analysis were drawn to circumscribe areas that could be represented by a single set of climatic data. The boundaries considered geographic boundaries, such as mountains, and other parameters that differentiate meteorological conditions (rainfall, temperature, windspeed). A description of the selection of the climatic regions and the representative meteorological stations is presented in Appendix M. However, once the boundaries of the climatic regions were drawn, other data associated with geographic location were linked to the climatic region designations. For example, soil characteristics also vary by

Climatic Region Name (Selected Met. Station)	Median Farm Size (Acres)
Seattle	40.10
Boise	194.40
Billings	1241.70
Burlington	159.20
Portland	98.20
Bismarck	923.80
Minneapolis	208.60
Salem	44.60
Muskegon	117.10
Chicago	177.60
Cleveland	109.20
Winnemucca	162.30
Casper	829.60
Hartford	50.00
San Francisco	39.80
Williamsport	127.10
Salt Lake City	143.50
Fresno	46.80
Lincoln	282.20
Philadelphia	39.00
Denver	738.00
Harrisburg	102.80
Norfolk	97.50
Huntington	86.70
Raleigh-Durham	85.40
Nashville	94.40
Asheville	55.40
Las Vegas	97.60
Little Rock	159.10
Tulsa	184.00
Albuquerque	464.30
Los Angeles	24.20
Charleston	80.40
Atlanta	105.90
Phoenix	339.70
Meridian	123.00
Shreveport	110.90
New Orleans	90.90
Houston	123.50
Miami	39.60
Tampa	67.00

Table 4-2. Median Farm Size for Each Climatic Region

geographic location, and this variability is reflected using a regional environmental setting approach. Another variable that is associated with location, but not directly linked to climate or soil conditions, is farm size. Farms in the more densely populated eastern part of the United States are much smaller than farms and ranches in the less densely populated western potion of the country. This variation was also included by using the regional environmental setting approach, which keeps correlated the conditions that are likely to occur together and prevents implausible combinations from being chosen during a random selection process. Using this approach, the climatic region was randomly selected, but all other data were selected to be consistent with conditions in that geographic location.

A meteorological station was selected to represent each of the 41 climatic regions. All meteorological stations within each climatic region were assumed to be representative of the entire region. The selected meteorological stations are listed in Table 4-2. Each climatic region was equally weighted in the probabilistic analysis.

4.3.2.1 <u>Meteorological Data</u>. Five years of representative meteorological data were processed for this analysis. The data gathered included surface data, upper-air data, and precipitation data. These observational data were used as Industrial Source Complex, Short-Term Model, version 3 (ISCST3), inputs.

Surface Data. Hourly surface meteorological data used in air dispersion modeling were processed from the Solar and Meteorological Surface Observation Network (SAMSON) CD-ROM (U.S. DOC and U.S. DOE, 1993). Variables included

- Temperature
- Pressure
- Wind direction
- Windspeed
- Opaque cloud cover
- Ceiling height
- Current weather
- Hourly precipitation.

Upper-Air Data. Twice-daily mixing-height data were calculated from upper-air data contained in the radiosonde data of the North America CD-ROM set (NCDC, 1997). This set contains upper-air data from 1946 through 1996 for most upper-air stations in the United States. The upper-air data were combined with the SAMSON data to create the mixing-height files. EPA's Support Center for Regulatory Air Models (SCRAM) bulletin board was also used to obtain mixing-height data (if available) when mixing-height data could not be successfully calculated from the radiosonde data.

Filling Missing Data. Missing surface data were identified using a program called SQAQC, which searched for incidents of missing data on the observation indicator, opaque cloud cover, temperature, station pressure, wind direction and speed, and ceiling height. Years that were missing 10 percent or more of the data were discarded (Atkinson and Lee, 1992). Verification (quality control or QC) checks were performed on the SQAQC program by applying

it to station data where the missing data were known and by intentionally degrading surface meteorological files and then running SQAQC to detect the missing values.

Missing surface data were filled in by a program called METFIX. This program fills in up to 5 consecutive hours of data for cloud cover, ceiling height, temperature, pressure, wind direction, and windspeed. For single missing values, the program follows the objective procedures developed by Atkinson and Lee (1992). For two to five consecutive missing values, other rules were developed because the subjective methods provided by Atkinson and Lee (1992) rely on professional judgment and could not be programmed. The METFIX program flagged files where missing data exceeded five consecutive values. In the few cases where this occurred and the missing data did not constitute 10 percent of the file, they were filled manually according to procedures set forth in Atkinson and Lee (1992). If more than 10 percent of the data were missing, the station was discarded and another station in the climatic region was selected.

All upper-air files were checked for missing data using a program called QAQC. QAQC produces a log file containing occurrences of missing mixing height. Verification (QC) checks were performed on the QAQC program by applying it to station data where the missing data were known and by intentionally degrading existing mixing height files and then running QAQC to detect the missing values.

Missing mixing heights were filled in by running the files through another program written to interpolate one to five consecutive missing values. According to Atkinson and Lee (1992), if there are one to five consecutive missing values, the values should be filled in subjectively using professional judgment. Again, programming these subjective procedures was not feasible, and the program used simple linear interpolation to fill in these values automatically. Information from Atkinson and Lee (1992) was used to determine which files should be discarded (i.e., files missing more than five consecutive missing values or missing 10 percent or more of the data). After the missing mixing heights were filled in for all upper-air files, they were checked once more for missing data using the QAQC program.

Other Meteorological Data. In addition to the surface and upper-air data, air modeling requires the input of the following meteorological parameters (U.S. EPA, 1995b):

- Minimum Monin-Obukhov length (m)
- Anemometer height (m)
- Roughness length (m), surface meteorological station
- Roughness length (m), area around facility
- Noontime albedo
- Bowen ratio
- Anthropogenic heat flux (W/m²)
- Fraction net radiation absorbed by the ground.

Anemometer height was collected from local climatic data summaries (NOAA, 1983). When anemometer height was not available, the station was assigned the most common anemometer height from the other stations. This value was 6.1 m. Land use information is required for determining a number of inputs. To obtain this information, a geographic information system (GIS) was used to determine the land use within a 3 km radius around each meteorological station by using Geographic Retrieval and Analysis System (GIRAS) spatial data with Anderson land use codes (Anderson et al., 1976). Table 4-3 shows how the Anderson land use codes were related to PCRAMMET land use codes.

A weighted average, based on the land use percentages for a 3 km radius around each meteorological station, was used to estimate the Bowen ratio, minimum Monin-Obukhov length, the noontime albedo, the roughness height at the meteorological station, and the fraction of net radiation absorbed by the ground.

The Bowen ratio is a measure of the amount of moisture at the surface around a meteorological station. The wetness of a location was determined based on the annual average precipitation amount. The range of values is provided in Table 4-4 as a function of land use type, season, and moisture condition. For this analysis, the annual average values were applied.

The minimum Monin-Obukhov length, a measure of the atmospheric stability at a meteorological station, was correlated with the land use classification, as shown in Table 4-5.

Noontime albedo values also were correlated with land use around a meteorological station, as shown in Table 4-6.

The surface roughness length is a measure of the height of obstacles to the wind flow. It is not equal to the physical dimensions of the obstacles but is generally proportional to them. Surface roughness length data are shown in Table 4-7 along with their corresponding land use. The roughness height was assumed to be the same at the meteorological station and at the farm site.

	Anderson Code and Description ^a	P	CRAMMET Type and Description ^b
51	Streams and canals	1	Water surface
52	Lakes	1	Water surface
53	Reservoirs	1	Water surface
54	Bays and estuaries	1	Water surface
41	Deciduous forest land	2	Deciduous forest
61	Forested wetland	2	Deciduous forest
42	Evergreen forest land	3	Coniferous forest
43	Mixed forest land	4	Mixed forest

 Table 4-3. Relation between Anderson Land Use Codes and PCRAMMET

 Land Use Codes

(continued)

And	lerson Code and Description ^a	RA	MMET Type and Description ^b
62	Nonforested wetland	5	Swamp (nonforested)
84	Wet tundra	5	Swamp (nonforested)
21	Cropland and pasture	6	Agricultural
22	Orchards-groves-vineyards-nurseries-ornamental	6	Agricultural
23	Confined feeding operations	6	Agricultural
24	Other agricultural land	6	Agricultural
31	Herbaceous rangeland	7	Rangeland (grassland)
32	Shrub and brush rangeland	7	Rangeland (grassland)
33	Mixed rangeland	7	Rangeland (grassland)
11	Residential	9	Urban
12	Commercial and services	9	Urban
13	Industrial	9	Urban
14	Transportation-communication-utilities	9	Urban
15	Industrial and commercial complexes	9	Urban
16	Mixed urban or built-up land	9	Urban
17	Other urban or built-up land	9	Urban
71	Dry salt flats	10	Desert shrubland
72	Beaches	10	Desert shrubland
73	Sandy areas not beaches	10	Desert shrubland
74	Bare exposed rock	10	Desert shrubland
75	Strip mines-quarries-gravel pits	10	Desert shrubland
76	Transitional areas	10	Desert shrubland
81	Shrub and brush tundra	10	Desert shrubland
82	Herbaceous tundra	10	Desert shrubland
83	Bare ground	10	Desert shrubland
85	Mixed tundra	10	Desert shrubland
91	Perennial snowfields	10	Desert shrubland
92	Glaciers	10	Desert shrubland

^a Anderson codes from Anderson et al. (1976). ^b PCRAMMET codes from U.S. EPA (1995b).

	Spring			Summer		Autumn		Winter		Annual Average					
Land Use Type	Dry	Wet	Avg.	Dry	Wet	Avg.	Dry	Wet	Avg.	Dry	Wet	Avg.	Dry	Wet	Avg.
Water surface	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	2.0	0.3	1.5	0.575	0.15	0.45
Deciduous forest	1.5	0.3	0.7	0.6	0.2	0.3	2.0	0.4	1.0	2.0	0.5	1.5	1.53	0.35	0.875
Coniferous forest	1.5	0.3	0.7	0.6	0.2	0.3	1.5	0.3	0.8	2.0	0.3	1.5	1.4	0.275	0.825
Swamp	0.2	0.1	0.1	0.2	0.1	0.1	0.2	0.1	0.1	2.0	0.5	1.5	0.65	0.2	0.45
Cultivated land (agricultural)	1.0	0.2	0.3	1.5	0.3	0.5	2.0	0.4	0.7	2.0	0.5	1.5	1.63	0.35	0.75
Grassland	1.0	0.3	0.4	2.0	0.4	0.8	2.0	0.5	1.0	2.0	0.5	1.5	1.75	0.425	0.825
Urban	2.0	0.5	1.0	4.0	1.0	2.0	4.0	1.0	2.0	2.0	0.5	1.5	3.0	0.75	1.6
Desert shrubland	5.0	1.0	3.0	6.0	5.0	4.0	10.0	2.0	6.0	10.0	2.0	6.0	7.75	2.5	4.75

Table 4-4. Daytime Bowen Ratio by Land Use and Season

Source: U.S. EPA, 1995b. Averages were computed for this effort.

(Stable Conditions)						
Urban Land Use Classification	Length (m)					
Agriculture (open)	2					
Residential	25					
Compact residential/industrial	50					
Commercial (19–40 story buildings)	100					
(> 40 story buildings)	150					

Table 4-5.	Minimum Monin-Obukhov Length
	(Stable Conditions)

Source: U.S. EPA, 1995b.

Section 4.0

Land Use Type	Spring	Summer	Autumn	Winter	Annual Average
Water surface	0.12	0.1	0.14	0.2	0.14
Deciduous forest	0.12	0.12	0.12	0.5	0.22
Coniferous forest	0.12	0.12	0.12	0.35	0.18
Swamp	0.12	0.14	0.16	0.3	0.18
Cultivated land (agricultural)	0.14	0.2	0.18	0.6	0.28
Grassland	0.18	0.18	0.20	0.6	0.29
Urban	0.14	0.16	0.18	0.35	0.21
Desert shrubland	0.3	0.28	0.28	0.45	0.33

Table 4-6. Albedo Values of Natural Ground Covers for Land Use Types and Seasons

Source: U.S. EPA, 1995b. Average values were computed for this analysis.

Land Use Type	Spring	Summer	Autumn	Winter	Annual Average
Water surface	0.0001	0.0001	0.0001	0.0001	0.0001
Deciduous forest	1.0	1.3	0.8	0.5	0.9
Coniferous forest	1.3	1.3	1.3	1.3	1.3
Swamp	0.2	0.2	0.2	0.05	0.16
Cultivated land (agricultural)	0.03	0.2	0.05	0.01	0.07
Grassland	0.05	0.2	0.01	0.001	0.04
Urban	1.0	1.0	1.0	1.0	1.0
Desert shrubland	0.3	0.3	0.3	0.15	0.26

Table 4-7. Surface Roughness Length for Land Use Types and Seasons (meters)

Source: U.S. EPA, 1995b. Average values were computed for this analysis.

During daytime hours, the heat flux into the ground is parameterized as a fraction of the net radiation incident on the ground. This fraction varies based on land use. A value of 0.15 was used for rural locations. Suburban and urban locations were given values of 0.22 and 0.27, respectively (U.S. EPA, 1995b).

Anthropogenic heat flux for a meteorological station can usually be neglected in areas outside of highly urbanized locations; however, in areas with high population densities or energy use, such as an industrial facility, this flux may not always be negligible (U.S. EPA, 1995b). For this analysis, anthropogenic heat flux was assumed to be zero for all meteorological stations.

4.3.2.2 <u>Meteorological Data</u>. Meteorological stations selected for purposes of air dispersion modeling also provided long-term climatic data that were necessary for fate and transport modeling. For each of the 41 stations, the following data were compiled:

- Mean annual wind direction
- Mean annual windspeed
- Average temperature
- Average annual runoff
- Universal Soil Loss Equation (USLE) rainfall/erosivity factor.

4.3.2.3 <u>Soil Characterization</u>. The fate and transport models used in the biosolids risk assessment require surface soil properties to model erosion and overland transport and properties of the entire soil column. A regional approach was also used to compile soil data for these modeling requirements. All land with agricultural use was used to characterize the soils within the 41 climatic regions. This regional characterization of soil types captured variability in soils in a manner that is generally representative of agricultural lands across the United States. A GIS was used to compile soil texture and other soil data within each climatic region. Then, database programs processed these data to create a distribution of input variables required by the models.

Data Sources. The primary data source for soil properties is the State Soil Geographic (STATSGO) database. STATSGO is a repository of nationwide soil properties primarily compiled by the U.S. Department of Agriculture (USDA) from county soil survey data (USDA, 1994). STATSGO includes a 1:250,000-scale GIS coverage that delineates soil map units, and an associated database containing soil data for each STATSGO map unit. (Map units are areas used to spatially represent soils in the database.)

In addition, two compilations of STATSGO data, each keyed to the STATSGO map unit GIS coverage, were used in the analysis as a convenient source of average soil properties:

- **USSOILS.** USSOILS (Schwarz and Alexander, 1995) averages STATSGO data over the entire soil column for each map unit.
- **CONUS.** CONUS (Miller and White, 1998) provides average STATSGO data by map unit and a set of 11 standardized soil layers.
- **GIRAS.** The GIRAS land use database (U.S. EPA, 1994) provides comprehensive land use data, in digital GIS format, for the contiguous 48 states.

Soil properties derived directly from STATSGO, CONUS, or USSOILS data include organic matter content, USLE K (erodibility) and S (slope) factors, and pH. A complete set of

hydrologic soil properties¹ was not available from STATSGO. To ensure consistent and realistic values, it was necessary to rely on established, nationwide relationships between hydrologic properties and soil texture or hydrologic soil group, both of which are available from STATSGO. Sources for these relationships include Carsel and Parrish (1988), Carsel et al. (1988), and Clapp and Hornberger (1978). These peer-reviewed references provide a consistent set of correlated hydrologic properties for each soil texture or hydrologic group.

Finally, two parameters—root zone depth and Soil Conservation Service (SCS) curve number (used for recharge calculations)—required site-based land use data, as well as soil texture or hydrologic soil group. The land use data were obtained for each of the 41 climatic regions from the GIRAS land use database (U.S. EPA, 1994). GIRAS provides comprehensive land use data, in digital GIS format, for the contiguous 48 states. Land use/land cover information in GIRAS was mapped and coded using the Anderson classification system (Anderson et al., 1976), which is a hierarchical system of land use characterizations. This nationwide coverage is based on late-1970s to early-1980s satellite images and aerial photography. The relationships used to convert the land use and soil data were obtained from Dunne and Leopold (1978) for root zone depth and USDA (1986) for the SCS curve number.

Methodology. The soil data collection methodology begins with GIS programs (in Arc Macro Language (AML)) that overlay the boundaries of the 41 climatic regions on the STATSGO map unit coverage to determine the STATSGO map units and their area within the regions. These data are then passed to data processing programs that derive predominant soil properties within each climatic region, either through direct calculations or by applying established relationships in lookup tables. In deriving soil model inputs, the biosolids soil data processing effort bases all collected soil properties on the predominant soil type (texture and hydrologic group) for the STATSGO map units having agricultural land use within each climatic region. Depending on modeling requirements, soil properties were derived for surface soils (top 20 cm), the entire soil column (to represent the vadose zone), or both, as shown in Table 4-8.

To ensure consistent, realistic properties, the soil data processing effort bases all collected soil properties on the predominant soil texture for each STATSGO map unit. For each STATSGO map unit within a meteorological station region, predominant texture was determined both for surface soils (top 20 cm) and the entire soil column (to represent the vadose zone) from CONUS data. For surface soils, the predominant texture is the thickest, weighted by depth, soil texture for the top three CONUS layers (20 cm). Where there was a tie, the texture of the top two layers was used as the predominant soil texture for that map unit. Twelve common soil textures were collected to develop hydrologic properties (Table 4-4). Map units that did not have one of the 12 common soil textures (e.g., those with water or organic matter) were excluded from the analysis. Soil column texture was obtained in a similar manner, except that all CONUS layers were used.

¹ Hydrologic soil properties required for modeling include bulk density, saturated water content, residual water content, field moisture content, wilting point, saturated hydraulic conductivity, soil moisture coefficient b, and soil moisture retention parameters alpha and beta.

Soil Variable	Units	Data Source
Properties Derived from Soil Texture		
USDA soil texture	Unitless	CONUS/STATSGO
Saturated hydraulic conductivity	cm/h	Relationship from Carsel and Parrish (1988)
Saturated water content	L/L	Relationship from Carsel and Parrish (1988)
Soil moisture coefficient b	Unitless	Relationship from Clapp and Hornberger (1988)
Soil bulk density	mg/L	Calculated from saturated water content
Root zone depth	cm	Relationship (with land use) from Dunne and Leopold (1978)
Properties Derived from Soil Hydrologic	Class	
SCS hydrologic class	Unitless	CONUS/STATSGO
Field capacity	% (vol.)	Relationship from Carsel et al. (1988)
Wilting point	% (vol.)	Relationship from Carsel et al. (1988)
SCS curve number	Unitless	Relationship (with land use) from USDA (1986)
Properties Obtained Directly from STATS	5G0	
Fraction organic carbon	g/g	STATSGO
Silt content	% (wt.)	STATSGO
USLE erodibility factor (K)	kg/m ²	STATSGO
USLE slope (S)	Degrees	STATSGO
Properties Derived from Slope		
USLE slope length (L)	m	Relationship from Lightle and Weesies (1998)
USLE length/slope factor (LS)	Unitless	Calculated from L and S per Williams and Berndt (1977)

Table 4-8. Summary of Soil Properties Collected for Biosolids Risk Analysis

To limit data collection to agricultural soils, GIS programs (in AML) were used to overlay the STATSGO map unit GIS coverage with the GIRAS land use GIS coverage and then determine the map units (and their respective areas) that occur in crop land and pasture land use (i.e., Anderson land use code 21) within each meteorological region. These data were then processed to create a set of the 12 soil textures, ranked by percentage of crop land and pasture land with each texture, for each region. These textures were used to derive soil properties for this analysis for each region/texture combination as described in the next section. These properties were then passed on to the model in Access database tables indexed by meteorological station and soil texture. Because certain soil properties were derived from SCS hydrologic soil groups, it was necessary to develop a hydrologic soil group that would be consistent with the soils of each texture within a region. To do so, a table of hydrologic soil groups by STATSGO map unit was created using STATSGO data for hydrologic soil groups by the component soils within the map unit. Based on the predominant texture for each map unit, hydrologic soil groups for the component soils with the same texture were averaged across each map unit (weighted by component percent) using the numeric conversion: group A = 1, group B = 2, group C = 3, and group D = 4. These values were then averaged again (weighted by map unit area) for each soil texture occurring in a region. After this regional average by texture was calculated, the numbers were converted back to letters using the same conversion, resulting in a hydrologic soil group for each texture occurring within a meteorologic region. Note that hydrologic soil group applies to the entire soil column and is not layer-specific.

Development of Soil Properties. Once the distribution of soil textures and their related hydrologic class was determined for each meteorological region, average soil properties were determined for each soil texture present in a region by relationships with soil texture or hydrologic class or by extracting the data for soils of each texture directly from STATSGO.

Soil Properties Based on Relationship with Predominant Texture—Several soil hydrologic properties were derived directly from predominant texture using database lookup tables relating mean properties to texture class (see Table 4-9). Tables 4-9 through 4-11 summarize the relationships used, which are described below.

- Saturated hydraulic conductivity (cm/h) was determined for both surface soil (*Ksat_top20*) and the entire soil column (*VadSATK*) using a national relationship from Carsel and Parrish (1988) (Table 4-9).
- Saturated water content (unitless) was determined for both surface soil (WCS_top20) and the entire soil column (VadWCS) using a relationship from Carsel and Parrish (1988) (Table 4-9).
- Bulk density (g/cm³) was calculated for surface soil (BD_top20) from saturated water content using the equation

$$\rho_{\rm b} = 2.65(1 - \phi) \tag{4-1}$$

where

 $\rho_b = \text{bulk density of the soil (U.S. EPA, 1997)}$ 2.65 = particle density in g/cm³ (assumed to be quartz) $\phi = \text{saturated water content.}$

Soil moisture coefficient (unitless) was determined for both the surface soil (*SMb_top20*) and the entire soil column (*SMb_sub*) using a relationship from Clapp and Hornberger (1978) (Table 4-9).

Soil Texture	Saturated Hydralic Conductivity Ksat ^a (cm/h)	Storated Water Content WCS ^a (L/L)	Bulk Density RHOB ^b (g/cm ³)	Soil Moisture Coefficient b SMb ^c
Clay (C)	0.20	0.38	1.643	11.4
Clay loam (CL)	0.26	0.41	1.5635	8.52
Loam (L)	1.04	0.43	1.5105	5.39
Loamy sand (LS)	14.59	0.41	1.5635	4.38
Silt (SI)	0.25	0.46	1.431	
Silt loam (SIL)	0.45	0.45	1.4575	5.30
Silty clay (SIC)	0.02	0.36	1.696	10.4
Silty clay loam (SICL)	0.07	0.43	1.5105	7.75
Sand (S)	29.70	0.43	1.5105	4.05
Sandy clay (SC)	0.12	0.38	1.643	10.4
Sandy clay loam (SCL)	1.31	0.39	1.6165	7.12
Sandy loam (SL)	4.42	0.41	1.5635	4.90

Table 4-9. Hydrological Soil Parameters Correlated to Soil Texture

^a Carsel and Parrish (1988).

^b Calculated from WCS using equation from U.S. EPA (1997).

^c Clapp and Hornberger (1978).

Depth to root zone (cm) was determined using a Dunne and Leopold (1978) table of rooting depth by vegetation type and soil texture (Table 4-10). For each soil texture, a minimum and a maximum root zone depth (for shallow and deep-rooted crops) were used to represent the range across crop land and pasture land use. Because Dunne and Leopold included only five soil textures, these five textures were mapped across the 12 basic textures used in this analysis as shown in Table 4-10.

Soil Parameters Based on Relationship with Hydrologic Group—The following soil parameters are all based on the average hydrologic soil group for each texture within a meteorological region. Mean values by hydrologic group were obtained using the following relationships:

- Soil moisture field capacity (volume %). A single field capacity value (*SMFC*) was obtained by hydrologic soil group by averaging the layered property values from Carsel et al. (1988). Table 4-11 presents the mean value for field capacity by hydrologic soil group and layer, as well as the average values used in this analysis.
- Soil moisture wilting point (volume %). A single wilting point value (*SMWP*) was obtained by hydrologic soil group by averaging the layered property values from Carsel et al. (1988). Table 4-11 lists the mean value for wilting point by hydrologic soil group and layer, as well as the average values used in this analysis.

USDA Soil Texture	Dunne & Leopold Texture	Shallow-Rooted Crops (DRZ_Min, cm)	Deep-Rooted Crops (DRZ_Max, cm)	
Sand	Fine sand	50	100	
Loamy sand	P '	50	100	
Sandy loam	Fine sandy loam	50	100	
Silt				
Silt loam	Silt loam	62	125	
Loam				
Sandy clay loam				
Silty clay loam	Clay loam	40	100	
Clay loam				
Sandy clay			67	
Silty clay	Clay	25		
Clay				

Table 4-10.	Depth to Root Zone	Values

Source: Dunne and Leopold (1978).

Hydrologic Group	Layer	FC	WP
А	1	9.4	3.1
	2	8.1	2.3
	3	5.9	2.1
	4	5.8	1.9
	Avg.	7.3	2.4
В	1	19.1	8.7
	2	18.8	9.3
	3	18.7	8.9
	4	17.5	8.4
	Avg.	18.5	8.8

	Table 4-11.	Field Capacity	(FC) and	Wilting	Point (W	VP) Value
--	--------------------	-----------------------	----------	---------	----------	-----------

(continued)

Hydrologic Group	Layer	FC	WP
С	1	22.5	10.4
	2	23.2	12.1
	3	22.9	11.9
	4	21.3	11.5
	Avg.	22.5	11.5
D	1	24.2	13.8
	2	26.3	17.0
	3	25.6	16.3
	4	24.4	15.1
	Avg.	25.1	15.6

 Table 4-11. (continued)

Source: Carsel et al. (1988).

SCS curve number (unitless). Minimum and maximum SCS curve number values (*CN_min* and *CN_max*) were determined for each regional soil texture based on a USDA (1986) table of curve numbers by cover type and hydrologic soil group, assuming a good condition pasture land use for *CN_min* and poor condition crop land use for *CN_max*. A lookup table (Table 4-12) with minimum and maximum SCS curve numbers by hydrologic soil group was used to assign the appropriate value for each regional soil texture according to its hydrologic soil group.

	SCS Cur	ve Number
SCS Hydrologic Soil Group	CN_Min (Pasture)	CN_Max (Crop Land)
А	39	72
В	61	81
С	74	88
D	80	91

Table 4-12.SCS Curve Number Values
by SCS Hydrologic Soil Group

Source: Derived from USDA (1986).

Parameters Collected Directly from STATSGO-Based Data Sources—Several variables were obtained directly from STATSGO (Schwarz and Alexander, 1995). Although they are not derived from soil texture, they were extracted and averaged based only on soil map units with the predominant texture to ensure consistent soil properties.

- USLE erodibility factor—top 20 cm (ton/acre). An area-weighted average erodibility factor for the top 20 cm of soil (*K_top20*) was calculated from STATSGO data by layer and component. STATSGO layer data were translated into *K* values using standardized CONUS layers and calculating a depth-weighted average value. Further, a component percent-weighted average *K* was calculated for each CONUS layer across all components contained in each map unit. The resulting table contains *K* values by map unit and standardized CONUS layer. To get one value for *K* by map unit for the top 20 cm of soil, a depth-weighted average for the top three CONUS layers was calculated. The final *K* value by meteorological region and soil texture was obtained by averaging the map units for each surface soil texture present within the meteorological region.
- Fraction organic carbon—top 20 cm (mass fraction). An area-weighted average fraction organic carbon for surface soils (*foc_top20*) was calculated for each region and soil texture using only the map units with the predominant surface soil texture of interest within the region. Percent organic matter for the top 20 cm of soil was obtained from STATSGO organic matter data by layer and component (Schwarz and Alexander, 1995) and converted to fraction organic carbon by dividing by 174 (100 × 1.74 g organic matter/g organic carbon) (U.S. EPA, 1997). Percent organic matter values were translated from STATSGO layer and component into standardized CONUS layers using the same methodology described for the USLE erodibility factor *K*. Then, a depth-weighted average percent organic matter was calculated for the top three CONUS layers (top 20 cm of soil).
- Silt content—top 20 cm (weight percent). An area-weighted average silt content for surface soils (*Ss_top20*) was derived from STATSGO data for each region and soil texture in the same manner described for USLE erodibility factor.

The USLE's length slope factor (*LS*) was derived from STATSGO slope data. Percent slope (*Theta*) was obtained by region and soil texture by using only the map units with the predominant texture of interest. An area-weighted average slope was calculated for each texture occurring in a region. Length (*Length*, ft) was then obtained from a Lightle and Weesies (1998) lookup table of default flow lengths by slope, using slope values rounded to the nearest integer (Table 4-13). All slopes less than 0.5 were given the length corresponding to 0.5 and all slopes greater than 24 were given the length corresponding to 24. The USLE length/slope factor *LS* (unitless) was then calculated using the equation from Williams and Berndt (1977):

$$LS = (L/72.6)^{m}(0.065 + 0.0454S + 0.0065S^{2})$$
(4-2)

where

L = flow lengthS = slope in percent

and

One basic type of source was evaluated in this assessment: land application of biosolids to crop land or pastures. It was necessary to determine the physical characteristics of the farm where biosolids were assumed to be applied and the operating parameters used for that application for the air dispersion modeling and source partition modeling. First, representative agricultural field sizes were identified. To identify a representative farm size for each climatic region, the median farm size in each county within a climatic region was determined from the Census of Agriculture (U.S. DOC, 1989, 1994). Then the median farm sizes for each county within each of the 41 climatic regions were ranked and the median farm size for each climate region was selected. The farm was assumed to devote one-half of its area to raising crops and one-half to pasturing cattle.

Slope	Length (ft)	Slope	Length (ft)
≤0.5	100	13	90
1	200	14	80
2	300	15	70
3	200	16	60
4	180	17	60
5	160	18	50
6	150	19	50
7	140	20	50
8	130	21	50
9	125	22	50
10	120	23	50
11	110	≥24	50
12	100		

 Table 4-13. Default Flow Lengths by Slope

Source: Lightle and Weesies, 1998.

Next 3,000-record source data files were constructed for use in the probabilistic analysis. These files were constructed by combining the environmental setting data, agricultural practice data, and the biosolids characterization data using the following steps:

- Select one of the 41 climatic regions (each region was assumed to be equally likely)
- Select data associated with the selected climatic region (farm size, soil data, meteorologic data)
- Select agricultural practice data independent of climatic region (application rate, frequency, and number of applications)
- Select biosolids characteristics (independent of climatic region and agricultural practice).

Two source data files were generated in this manner: one for crop land and one for pastures. Each of the 3,000 records in each of the source data files was identified by a model run identification number.

4.4 References

- Anderson, J.R., E.E. Hardy, J.T. Roach, and R.E. Witmer. 1976. A land use and land cover classification system for use with remote sensor data. Geological Survey Professional Paper 964. In: U.S. Geological Survey Circular 671. U.S. Geological Survey, Washington, DC.
- Atkinson, D., and R.F. Lee. 1992. Procedures for Substituting Values for Missing NWS Meteorological Data for Use in Regulatory Air Quality Models. U.S. Environmental Protection Agency, Research Triangle Park, NC.
- Carsel, R.F., and R.S. Parrish. 1988. Developing joint probability distributions of soil water retention characteristics. *Water Resources Research* 24(5):755-769.
- Carsel, R.F., R.S. Parrish, R.L. Jones, J.L. Hansen, and R.L. Lamb. 1988. Characterizing the uncertainty of pesticide leaching in agricultural soils. *Journal of Contaminant Hydrology* 2:111-124.
- Clapp, R.B., and G.M. Hornberger. 1978. Empirical equations for some soil hydraulic properties. *Water Resources Research* 14:601-604.
- Dunne, T., and L.B. Leopold. 1978. *Water in Environmental Planning*. New York: W. H. Freeman and Company.

- Lightle, D.T., and G. Weesies. 1998. Default slope parameters. Memorandum to Scott Guthrie (RTI) from D.T. Lightle and G. Weesies (USDA, Natural Resources Conservation Service), West Lafayette, IN. June 8.
- Miller, D. A., and R. A. White. 1998. A Conterminous United States Multilayer Soil Characteristics Dataset for Regional Climate and Hydrology Modeling. Website at http://www.essc.psu.edu/soil_info/index.cgi?soil_data&index.html.
- NCDC (National Climatic Data Center and Forecast Systems Laboratory). 1997. Radiosonde Data of North America, 1946-1996, Version 1.0, June 1997 (Updated).
- NOAA (National Oceanic and Atmospheric Administration). 1983. *Local Climatological Data. Annual Summaries for 1982: Part I - ALA - MONT and Part II - NEB - WYO*. National Climatic Data Center, National Environmental Satellite, Data, and Information Service, Asheville, NC.
- Schwarz, G.E., and R.B. Alexander. 1995. State Soil Geographic (STATSGO) Data Base for the Conterminous United States. Edition 1.1. Open-File Report 95-449. U.S. Geological Survey, Reston, VA. Website at http://water.usgs.gov/GIS/metadata/usgswrd/ussoils.html. September 1.
- USDA (U.S. Department of Agriculture). 1986. Urban Hydrology for Small Watersheds. TR-55 (210-VI-TR-55). Engineering Division, Soil Conservation Service, Washington, DC. pp. 2-5. June.
- USDA (U.S. Department of Agriculture). 1994. *State Soil Geographic (STATSGO) Data Base. Data Use Information.* Miscellaneous Publication No. 1492. Natural Resources Conservation Service, Fort Worth, TX. Available online: http://www.ftw.nrcs.usda.gov/stat_data.html.
- U.S. DOC (Department of Commerce). 1989. 1987 Census of Agriculture, Volume 1, Geographic Area Series State and County Data. Bureau of Census, Washington, DC.
- U.S. DOC (Department of Commerce). 1994. 1992 Census of Agriculture Volume 1, Geographic Area Series State and County Data. Bureau of the Census, Washington, DC.
- U.S. DOC and U.S. DOE (U.S. Department of Commerce National Climatic Data Center and U.S. Department of Energy National Renewable Energy Laboratory). 1993. Solar and Meteorological Surface Observation Network (SAMSON) 1961-1990. Version 1.0.
- U.S. EPA (Environmental Protection Agency). 1992. Technical Support Document for Land Application of Sewage Sludge. Office of Water, Office of Science and Technology. EPA 822/R-93-001a. November.
- U.S. EPA (Environmental Protection Agency). 1994. 1:250,000 Scale Quadrangles of Landuse/Landcover GIRAS Spatial Data in the Conterminous United States: Metadata.

Office of Information Resources Management, Washington, DC. Website at http://www.epa.gov/ngispgm3/nsdi/projects/giras.htm.

- U.S. EPA (Environmental Protection Agency). 1995a. Compilation of Air Pollutant Emission Factors, Volume 1: Stationary Point and Area Sources. 5th Edition. AP-42. PB95-196028INZ. Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1995b. *PCRAMMET User's Guide* (Draft). Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1997. EPA's Composite Model for Leachate Migration with Transformation Products. EPACMTP: User's Guide. Office of Solid Waste, Washington, DC.
- U.S. EPA, 2001. 2001 National Sewage Sludge Survey.
- van der Leeden, F., F.L. Troise, and D.K. Todd. 1990. *The Water Encyclopedia*. 2nd edition. Chelsea, Michigan: Lewis Publishers. p. 176.
- Williams, J.R., and H.D. Berndt. 1977. Determining the universal soil loss equation's lengthslope factor for watersheds. In: A National Conference on Soil Erosion - Soil Erosion: Prediction and Control, May 24-26, 1976, Perdue University, West Lafayette, IN, pp. 217-225, Soil Conservation Society of America, Ankeny, IA.

5.0 Estimating Exposure Point Concentrations

Exposure point concentrations are constituent concentrations at the location in the environment at which an individual may be exposed. To determine constituent concentrations in environmental media (e.g., air or soil) with which a receptor comes in contact, several computer-based models and sets of equations are used:

- Source partition models
- Fate and transport models
- Farm food chain equations
- Aquatic food chain equations.

The agricultural application of biosolids evaluated in this risk assessment is described in Section 4.0. Dioxins, furans, and coplanar PCBs found in biosolids are released from these agricultural applications into the environment. Releases to the atmosphere occur through volatilization or wind erosion of particles. Releases from the agricultural field may also occur through erosion of soil particles onto the residential plot and, subsequently, into a nearby stream. The constituents may move into the human food chain by contaminating fruits, vegetables, poultry, eggs, beef, milk, and fish consumed by humans.

This risk analysis was performed in a probabilistic format. Section 3.0 explains the risk assessment framework, including the structure of the probabilistic analysis. The current section describes the models and algorithms used for the risk analysis. In the probabilistic analysis, specified model input parameter values were varied in each of 3,000 iterations to generate a distribution of media concentrations.

The following subsections describe the models and equations used in this risk assessment and their application. Section 5.1 describes the source partition models used to predict environmental releases of constituents from the biosolids. Section 5.2 discusses the air dispersion and deposition modeling and methodologies used to estimate concentrations of constituent releases used in the human health risk analysis. Section 5.3 discusses the methodology for calculating food chain concentrations based on air, soil, and water concentrations.

Greater detail on the modeling performed for this risk analysis is provided in appendices to this document:

• Appendix F, Source Model for Land Application Unit. This appendix explains the source partition model used in this risk assessment.

- Appendix G, Air Dispersion and Deposition Modeling. This appendix provides details, including all input data files, used in the air dispersion and deposition modeling for this risk assessment.
- Appendix H, Direct and Indirect Exposure Equations. This appendix documents the algorithms used to calculate exposure point concentrations for the surface water sediment, terrestrial food chain, and aquatic food chain.
- Appendix I, Variables for Aboveground Fate and Transport. This appendix presents and references the input values or distributions used in the algorithms presented in Appendix H.

5.1 Source Partition Modeling of Constituent Releases

5.1.1 Land Application Unit Partitioning Model Used for Agricultural Fields

Fate and transport of chemicals within the agricultural fields and chemical emissions from these fields to surrounding media were simulated using adaptations of the Land Application Unit (LAU) model. The LAU model was used in two slightly different versions for this biosolids risk assessment—one version representing the crop agricultural field, and the second representing the pasture agricultural field. An overview of the LAU model is presented in the following sections, including a description of the LAU's "local watershed" concept, the important assumptions inherent in the LAU methodology, its hydrology and soil erosion methodologies, its fundamental fate and transport algorithmic "engine"—the Generic Soil Column Model (GSCM), its particulate emissions to the atmosphere estimation methods, and the differences between the LAU (crop) and LAU (pasture) versions and other modifications required to execute the LAU model for purposes of this analysis. The LAU module is described in Appendix F.

A complete listing of input parameters for both the crop and pasture source partition models is provided in Appendix C.

Figure 5-1 shows the data flow into and out of the source model.

5.1.1.1 Local Watershed. The agricultural land where biosolids are applied, whether a crop land or a pasture land, is considered an integral part of a "local watershed," as illustrated in Figure 5-2. A local watershed is defined here as that drainage area that contains only the agricultural field and its downslope contiguous land areas in which runoff occurs as overland flow (sheet flow) only. Thus, a local watershed extends downslope only to the point that runoff flows and eroded soil loads would enter a well-defined drainage channel, e.g., a ditch, stream, lake, or some other waterbody. The sheet-flow-only restriction is based on the assumption that any area downstream of the agricultural field is subject to contamination from the application of biosolids through overland runoff and soil erosion.

The "buffer" illustrated in Figure 5-2 shows the area where the farm family is assumed to live. For simplicity, it is assumed that the agricultural field extends from the drainage divide of the local



Figure 5-1. Biosolids application to agricultural field source module.





watershed downslope to the boundary with the buffer (where the family resides). The buffer, which is part of the local watershed, is also simulated by the LAU model; that is, the LAU model simulates the dynamic fate and transport of constituents within the agricultural field, from the agricultural field to the buffer, within the buffer, and from the buffer to the waterbody.

Also illustrated in Figure 5-2 are the mechanisms by which constituent emissions to surrounding media occur. In the agricultural field itself, emissions to the atmosphere occur via volatilization of gaseous phases and wind/vehicular erosion of particulate phases. Runoff and erosion processes transport surficial constituents downslope to contaminate the buffer area and the contiguous waterbody. Fate and transport processes are simulated in the buffer, similar to and concurrent with the agricultural field simulation.

5.1.1.2 <u>LAU Assumptions</u>. A number of assumptions inherent in the LAU model pertain primarily to how the computational engine, the GSCM, is applied to simulate application of biosolids to agricultural land. The GSCM is discussed in greater detail in Section 5.1.1.3. The LAU assumptions are summarized as follows:

- Biosolids are applied to the soil surface periodically at set intervals (e.g., biennially) and then either tilled into the soil to a depth of 20 cm in the case of crop agricultural field or mixed with the top 2 cm of soil in the case of pasture.
- Whether the biosolids are tilled into the soil or remain near the surface, the constituent concentration in the zone occupied by the biosolids is assumed to be uniformly distributed (completely mixed) after each application.
- The contaminant mass is concentrated in the solids portion of the biosolids and is repartitioned among the solid, aqueous, and gas phases in the soil column.
- Biosolids applications do not result in any buildup of the soil surface, nor does erosion significantly degrade the soil surface (i.e., the distance from the site surface [z = 0] to a fixed point below the surface is constant). As a result, there is no naturally occurring limit to the modeled total soil concentration. In other words, the modeled constituent concentration in the agricultural field could exceed the constituent concentration in the waste. Indeed, this is physically possible for highly immobile constituents if the waste matrix is organic (as is the case for biosolids) and decomposes, leaving behind the constituent to concentrate over multiple applications.
- The first-order loss rate due to wind erosion and other surface disturbances is applied to the surface layer of the soil only and is calculated each year as an annual average with consideration of losses from an active agricultural field due to wind erosion, vehicular activity, and tilling operations (for the crop field). The particulate emission loss rate from an inactive agricultural field or pasture includes wind erosion only.

$$\frac{\partial C_T}{\partial t} = D_E \frac{\partial^2 C_T}{\partial z^2} - V_E \frac{\partial C_T}{\partial z} - kC_T$$
(5-1)

5.1.1.3 <u>Generic Soil Column Model</u>. The GSCM solves the following partial differential equation in space and time

where

C _T	=	total (dissolved plus sorbed plus vapor-phase) chemical concentration (M/L ³)
t	=	time (T^{-1})
D_E	=	effective diffusivity (L^2/T)
Z	=	depth in the soil column (L)
V_{E}	=	effective solute advection velocity (L/T)
k	=	overall first-order loss rate constant (T ⁻¹).

The term on the left side of Equation 5-1 represents changes in C_T over time. The first term on the right side represents vertical transport due to diffusion. The second term represents vertical transport due to bulk advective movement of water passing downward through the soil column. The last term represents the cumulative loss of chemical due to decay, hydrolysis, volatilization, or erosion losses from the surface. The vertical extent over which Equation 5-1 is solved is the depth from the soil surface down to a depth of 20 cm. Boundary conditions are $C_T = 0$ at the soil surface and a zero-gradient boundary condition at the lower soil column boundary (20 cm). A zero-gradient boundary condition implies that the concentration, whatever it may be, on either side of the boundary is identical on both sides. Initial conditions reflect the concentration profile over the soil column depth, *z*, at the time the simulation begins.

The solution technique used for solving Equation 5-1 over space and time is a hybrid of analytical and numerical methods developed to achieve a balance between simulation accuracy and execution speed. It is more fully described in Appendix F. The GSCM operates on a daily time step, with outputs aggregated to annual average values. The output of the GSCM is a time series of annual average values. A brief narrative description of how the GSCM is used to simulate fate and transport of chemical in the crop and pasture agricultural fields follows.

At the start of the simulation (time 0), the soil column is clean; that is, the chemical concentration is 0 throughout the soil column modeled depth. (Both the crop and pasture modeled soil column depths are 20 cm.) The initial waste application then occurs, introducing chemical mass into the soil column. This mass is introduced by calculating the total chemical mass associated with the waste application, "mixing" that mass into a specific depth of the soil column, and calculating the resulting uniform chemical concentration over that mixed depth. For the crop model, the depth over which waste is mixed at the time of application is the full soil column depth, 20 cm, under the assumption that the waste is mechanically tilled into that depth. For the pasture model, the mixing depth is 2 cm, under the assumption that cattle activity and/or other bioturbation processes effectively mix the surface-applied biosolids to that depth.

Following incorporation of newly added waste into the soil column, the dynamic solution of Equation 5-1 then proceeds over space (the vertical soil column is disaggregated into 20 1-cm thick layers for purposes of simulating vertical gradients—concentration is uniform within a layer, but can vary among layers) and time. As previously mentioned, the fundamental time step is daily, so that fate and transport of chemical in the surficial soil layer can respond to daily rainfall and runoff events. For example, on a dry day, chemical in the surficial soil layer (and elsewhere) is lost or transported via the mechanisms of vertical advection (long-term average infiltration/leaching), vertical diffusion, and, as appropriate for the chemical being analyzed, volatilization, hydrolysis, and biodegradation. On a day with precipitation and runoff (not all precipitation events lead to runoff), in addition to these processes, chemical is lost from the surface layer to downslope land areas (the buffer and waterbody) due to lateral advective transport of any dissolved chemical that has diffused from the soil pore water into the runoff water and due to erosion of chemical sorbed to eroded soil particles.

This dynamic solution of Equation 5-1 on a daily time step continues until the time of the next waste application. At that time, the residual chemical concentration profile (just prior to the new application time) is retrieved, and the concentrations in the soil layers receiving waste (20 cm in the crop field, 2 cm in the pasture field) are then increased by the newly added chemical mass, and the simulation is begun again from this new initial condition. At the end of the agricultural field operating life, when biosolids cease to be added, the simulation continues to simulate depuration or reduction of chemical. The simulation ultimately terminates when either 99 percent of the peak chemical mass has been removed via the various fate and transport processes or 200 years has elapsed (from the first application), whichever comes first. (For dioxins, 200 years comes first because of the persistence of the chemicals.)

The following major assumptions were used in the development of the GSCM:

The contaminant partitions to three phases: sorbed (solid), dissolved (liquid), and gaseous. The total contaminant concentration in soil is calculated as follows:

$$C_T = \rho_b C_S + \theta_w C_L + \theta_a C_G$$
(5-2)

where

C _T	=	total contaminant concentration in soil
ρ_{b}	=	soil dry bulk density (M/L^3)
C_s	=	sorbed-phase contaminant concentration in soil (M/M of dry soil)
$\theta_{\rm w}$	=	soil volumetric water content (L^3 soil water/ L^3 soil)
CL	=	aqueous-phase contaminant concentration soil $(M/L^3 \text{ of soil water})$
θ_a^-	=	soil volumetric air content (L^3 soil air/ L^3 soil)
C _G	=	gas-phase contaminant concentration in soil (M/L ³ of soil air).

The contaminant undergoes reversible, linear equilibrium partitioning between the adsorbed and dissolved phases. The sorbed-phase contaminant concentration in soil is calculated as follows:

$$C_{S} = K_{d} C_{L}$$
(5-3)

where K_d is the linear equilibrium partitioning coefficient (L³/M). For organic contaminants:

$$K_d = foc \times K_{oc} \tag{5-4}$$

where *foc* is the organic carbon fraction in soil and K_{oc} is the equilibrium partition coefficient, normalized to organic carbon. (It is implicit in this linear equilibrium partitioning assumption that the sorptive capacity of the soil column solids is considered to be infinite with respect to the total mass of contaminant over the duration of the simulation, i.e., the soil column sorptive capacity does not become exhausted. This condition is assumed true for this case.)

The contaminant in the dissolved and gaseous phases is assumed to be in equilibrium and to follow Henry's law. The gas-phase contaminant concentration in soil is calculated as follows:

$$C_G = H' C_L \tag{5-5}$$

where H' is the dimensionless Henry's law coefficient.

- Material in the soil column (including bulk waste) can be approximated as unconsolidated homogeneous porous media whose basic properties (ρ_b , foc, θ_w , θ_a , and η , where η is the total soil porosity) are average annual values, constant in space.
- Contaminant mass may be lost from the soil column due to one or more first-order loss processes.
- The total chemical flux is the sum of the vapor flux and the flux of the dissolved solute, diffusive losses of the dissolved phase from the surficial soil pore water into overlying runoff water during a runoff event, loss of sorbed phase due to wind and water erosion from the surficial soil, and internal sinks due to biochemical decay and hydrolysis.
- The chemical is transported in one dimension through the soil column.
- The modeled soil column remains constant in volume and fixed in space with respect to the water table.

5.1.1.4 <u>Hydrology and Soil Erosion</u>. The hydrology model used in the agricultural field model provides estimates of daily soil moisture, runoff, potential evapotranspiration, actual evapotranspiration, and infiltration. The hydrology model is based on a daily soil moisture water balance performed on the root zone depth of the soil column. At the end of a given day, t, the soil moisture in the root zone of an arbitrary local watershed subarea, i, is updated as

$$SM_{i,t} = SM_{i,t-1} + P_t + RO_{i-1,t} - RO_{i,t} - ET_{i,t} - IN_{i,t}$$
 (5-6)

where

=	soil moisture (L) in root zone at end of day t for subarea i
=	soil moisture (L) in root zone at end of previous day for subarea i
=	total precipitation depth (L) on day t
=	storm runoff depth (L) on day t coming onto subarea i from $i-1$
=	storm runoff depth (L) on day t leaving subarea i
=	evapotranspiration (L) from root zone on day t for subarea i
=	infiltration (groundwater recharge) on day $t(L)$ for subarea i .

Precipitation is undifferentiated between rainfall and frozen precipitation; that is, frozen precipitation is treated as rainfall.

Runoff is calculated as a function of soil type, soil cover, precipitation, and antecedent soil moisture using the SCS "curve number" method (USDA, 1986). Potential evapotranspiration is estimated as a function of air temperature, latitude (solar declination), and day-of-year using the Hargreaves equation (Shuttleworth, 1993). Actual evapotranspiration is estimated as a function of potential evapotranspiration, soil moisture, soil wilting point, and soil field capacity (Dunne and Leopold, 1978). Finally, infiltration is estimated as that day's residual soil moisture (net of runoff and evapotranspiration) in excess of the soil's field capacity. Maximum infiltration rates are limited to the saturated hydraulic conductivity of the soil. If the calculated infiltration exceeds the saturated hydraulic conductivity, a feedback loop is triggered that increases runoff and/or evapotranspiration (if less than potential evapotranspiration) to maintain the daily water balance.

Soil erosion is estimated based on the USLE methodology (Wischmeier and Smith, 1978), modified for application of a daily storm event. The daily application of the USLE is achieved by linearly distributing the USLE's long-term average rainfall factor (R) to storm-event-specific R values. This allocation is made based on the fraction of the long-term R value that is contributed by each individual storm event, as measured by the storm event's contribution to long-term average precipitation depths. A sediment delivery factor is also used in the soil erosion methodology. The sediment delivery factor accounts for the empirical observation that less eroded soil leaves a watershed per unit surface area as the size of the watershed increases; that is, soil mobilized by precipitation and runoff may be trapped in surface depressions before it can exit the watershed.

5.1.1.5 <u>Particulate Emissions</u>. Wind erosion, vehicular activity, tilling operations, or other surface disturbances may result in suspension of surficial soil particles into the atmosphere. To the extent that those soil particles contain sorbed chemical, this process becomes a source of particulate chemical flux into the atmosphere. The agricultural field model includes equations to estimate particulate emissions of chemical sorbed onto particles of 30 μ m diameter or smaller. These equations are based on empirical relationships developed by EPA in 1986 (updated, U.S. EPA, 1995a) and Cowherd et al. (1985), which are summarized in U.S. EPA (1999b). These empirical relationships estimate emission fluxes of surficial soil particles resulting from various surface-disturbing activities. The contemporaneous sorbed chemical concentration on surficial soils, estimated by the GSCM, then provides the chemical concentration also sorbed onto the airborne particles. No chemical "enrichment" is assumed to occur; that is, the sorbed concentration on the 30- μ m particles is the same concentration as on the surficial soils. Land-disturbing activities for the agricultural fields (crops and pastures) are wind erosion, vehicular activity, and spreading (pasture) or tilling (crop).

5.1.1.6 <u>Effective Soil Half-life</u>. Although the source model used in this risk assessment to simulate chemical releases from and soil concentrations within the agricultural fields receiving biosolids has been extensively verified, it has not been validated. Verification is the process of confirming, through testing, sensitivity analysis, or benchmarking against other models, for example, that a model performs as it was intended by the modelers; that is, its functionality is verified. Validation is the more rigorous process of confirming that a model's predictions are in fact in reasonable agreement with phenomena observed in nature. Model validation requires extensive and appropriate data on observed emission rates and soil concentrations, as well as model calibration activities, neither of which were feasible for this modeling study primarily for the practical reason that there are no data available for all the components of this modeling effort.

Although strict validation to actual site emission rates and soil concentrations was not feasible, an analysis was performed to estimate overall chemical half-lives at selected percentiles of the risk distribution built up by the 3,000 probabilistic simulations. A chemical half-life is simply the period of time that it takes for a chemical to depurate (be reduced) from some initial concentration to one-half of that concentration. Thus, the half-life is an overall measure of the various mechanisms by which a chemical may be reduced: physical mechanisms such as runoff/erosion or leaching or biochemical mechanisms such as decay/degradation or volatilization. Chemicals that are "quick," i.e., highly volatile or quickly degraded by biochemical processes, have correspondingly short half-lives-on the order of weeks, days, or even smaller time spans. In contrast, other chemicals have exceedingly long half-lives; for example, some radioactive isotopes have half-lives on the order of thousands of years. A chemical's half-life may also be affected by the environment in which the chemical exists. For example, some chemicals may be rapidly degraded by aerobic biochemical processes, but much more slowly degraded by anaerobic processes. Thus, the same chemical would have a much longer half-life in an anaerobic landfill than it would in an oxygen-rich environment, such as a surficial soil.

An example analysis of half-life was performed for TCDD for the LAU (pasture) model for selected iterations of the risk distribution. The runs selected corresponded to the runs that produced beef risk values at the 10th, 25th, 50th, 75th, 90th, 95th, and 99th percentiles. Each of these

specific model runs, out of the 3,000 probabilistic runs, is defined by its own set of input parameters. The sets of input parameters corresponding to each of these runs were retrieved from the larger database, and the source model used to represent the LAU (pasture) was executed for each set. For each of these half-life runs, the time series of annual average depth-averaged (20 cm) soil concentrations was then analyzed. This analysis first determined the year that the soil concentration reached its peak (the last year of biosolids application to the pasture), and then simply counted the number of years required for the depth-averaged soil concentration to reach half that peak value. The time series of soil concentrations for the 95th percentile analysis is shown in Figure 5-3 as an example. In this example, TCDD concentration reaches its peak at year 34 and reaches one-half that peak concentration in year 82, for a half-life of 48 years. Calculated half-lives for all the selected percentiles are shown in Table 5-1.



Figure 5-3. Example depth-averaged soil concentration annual time series.

Table 5-1. Calculated TCDD Half-Lives for Selected Risk Distribution Percentiles

Risk Distribution Percentile	Calculated TCDD Half-Life (years)
10 th	20
20 th	38
50 th	39
75 th	26
90 th	35
95 th	48
99 th	37

Because each of the 3,000 sets of input parameters represents a somewhat different "environment" for TCDD, it was expected that the resulting half-lives for the selected percentiles would be variable, as indeed they are. They do not vary a great deal, however, and the conclusion of the analysis is that TCDD's half-life for this risk assessment is within the approximate range of 20 to 50 years. The Draft Dioxin Reassessment Document presents a review of the literature on the persistence of dioxin in soil (U.S. EPA, 2000; Section 2.6.1.3, Transport Mechanisms in Soil). These studies are summarized in Table 5-2. These observed half-lives seem to corroborate the range of half-lives resulting from the source model runs, thereby affording a measure of credibility to the modeled results.

Soil Half-Life	Study Parameters	Reference
25 to 100 years 9 to 15 years	Subsurface soil Top 0.1 cm	Paustenbach, D.J., R.J. Wenning, V. Lau, N.W. Harrington, D.K. Rennix, and A.H. Parsons, 1992. Recent developments on the hazards posed by 2,3,7,8-tetrachlorobenzo-p-dioxin in soil: implications for setting risk-based cleanup levels at residential and industrial sites. <i>J.</i> <i>Toxicol. and Environ. Health</i> 36:103-149.
Approximately 20 years	Sludge-amended soil sampled from a long-term field experiment	McLachlan, M.S., A.P. Sewart, I.R. Bacon, and K.C. Jones. 1996. Persistence of PCDD/Fs in a sludge-amended soil. <i>Environ. Sci. Technol.</i> 30(8):2567-2571.
10 to 12 years	Field studies on a military test area aerially sprayed with 2,4,5-T. Data for 2,3,7,8-TCDD incorporated in the soil	Young, A.L. 1983. Long-term studies on the persistence and movement of TCDD in a natural ecosystem. In: <i>Human and</i> <i>environmental risks of chlorinated</i> <i>dibenzodioxins and related compounds</i> . Tucker, R.E., A.L. Young, A.P. Gray. Eds. Plenum Press.

Table 5-2.	Soil Half-Life	Data Re	ported in t	he Draft Dioxin	Reassessment	Document
1 abic 5-2.	Son man-Line	Data NC	porticu m t		I Cassessment	Document

5.2 Fate and Transport Modeling

This section describes the methodology and the models that were used to predict the fate and transport of chemical constituents in the environment. The methodology is based on the methodology used in the Draft Dioxin Reassessment (U.S. EPA, 2000).

Once dioxin congeners are released, they can move through the air, soil, and food chain by natural processes. The purpose of the fate and transport modeling performed for this assessment is to estimate the concentration of dioxins in environmental media (i.e., air, soil, and food items) to which individuals may be exposed. To predict a contaminant's movement through these different media, several media-specific fate and transport models are employed. Fate and transport models typically used by EPA are either a series of computer-based algorithms or sets of equations that predict chemical movement due to natural forces. These fate and transport models integrate information on a site's geology, hydrology, and meteorology with chemical, physical, and biological processes that can take place in the environment. The result is a simulation of chemical movement in the environment and a prediction of the concentration of a constituent at a certain point called the "exposure point." The following fate and transport models were used for this analysis:

- Air dispersion and deposition model
- Watershed model
- Food chain model.

These three models and the general framework for performing the fate and transport modeling are described in the following sections. Section 5.2.1 discusses the air dispersion and deposition modeling. Section 5.2.2 describes the watershed model used to determine soil and water constituent concentrations. Detailed descriptions of the models and a comprehensive list of the input values used in them can be found in Appendices G and H, respectively. The calculations of the food chain model are based on these media concentrations and are presented in Section 5.3.

5.2.1 Dispersion and Deposition Modeling

Dispersion modeling is a computer-based set of calculations used to estimate ambient ground-level constituent concentrations associated with constituent releases from biosolids management practice. The dispersion model uses information on meteorology (e.g., windspeed and wind direction, temperature) to estimate the movement of constituents through the atmosphere. Movement downwind is largely determined by windspeed and wind direction. Dispersion around the centerline of the contaminant plume is estimated by empirically derived dispersion coefficients that account for movement of constituents in the horizontal and vertical directions. In addition, constituent movement from the atmosphere to the ground is also modeled to account for deposition processes driven by gravitational settling and removal by precipitation.

The air dispersion and deposition modeling conducted for this analysis produced output data that were used to calculate environmental media concentrations and food chain concentrations (see Section 5.3). The dispersion model outputs included air concentration of vapors and particles, wet deposition of vapors and particles, and dry deposition of particles. Dry deposition of vapors was also calculated, but outside the dispersion model.

5.2.1.1 <u>Industrial Source Complex Short-Term Dispersion Model</u>. A number of dispersion models are available for estimating the transport of constituent through the atmosphere, several of which are available on EPA's SCRAM Bulletin Board (http://www.epa.gov/scram001/). These dispersion models were developed for a variety of applications and each has its own strengths and weaknesses. The ISCST3 model was selected for air dispersion modeling in this analysis. Because this assessment required a model with the capability to model ground-level area sources, ambient air concentrations and deposition fluxes, vapors and particulates, and annual averaging times, ISCST3 was an appropriate model to use.
In addition, ISCST3 is supported by EPA's Office of Air Quality Planning and Standards and has been used extensively in regulatory applications.

ISCST3 (U.S. EPA, 1999a), a recommended dispersion model in EPA's *Guideline on Air Quality Models* (U.S. EPA, 1999c), is a steady-state, Gaussian plume dispersion model. A steady-state model is one in which the model inputs and outputs are constant with respect to time. That is, the system being modeled is assumed to be unchanging over time. The term "Gaussian plume" refers to the kind of mathematical solution used to solve the air dispersion equations. It essentially means that the constituent concentration is dispersed within the plume laterally and vertically according to a Gaussian distribution, which is another name for to a normal distribution. These assumptions and solutions hold for each hour modeled. The results for each hour are then processed to provide values for different averaging times depending on the user's needs (e.g., annual average).

ISCST3 is capable of simulating dispersion of pollutants from a variety of source types, including point, area, volume, and line sources. ISCST3 can account for both long- and short-term air concentration of particles and vapor and wet and dry deposition of particles and vapor. In addition to deposition, wet and dry plume depletion can be selected to account for removal of matter by deposition processes and to maintain mass balance. Receptor locations can be specified in polar or cartesian arrays or can be set to discrete points as needed. Flat or rolling terrain may be modeled, but only flat terrain may be used for area sources. ISCST3 considers effects on dispersion of environmental setting by allowing the user to choose dispersion parameters representing either an urban or rural setting.

5.2.1.2 <u>Configuration of ISCST3 for Air Dispersion and Deposition Modeling</u>. Results of air dispersion and deposition modeling represent the initial step in the fate and

transport of vapor and particle emissions in the environment. The ISCST3 model was used to estimate

- Air concentration of vapors
- Air concentration of particles
- Wet deposition of vapors and particles
- Dry deposition of particles.

Dry deposition of vapors was calculated outside of ISCST3, as explained below.

All air concentrations and deposition values developed by ISCST3 were unit values based on modeling default unit emission rates. Later in the exposure modeling process, the unit air concentrations $[(\mu g/m^3)$ per (unit emission rate of 1 g/s-m²)] and deposition rates $[(g/m^2)$ per (unit emission rate of 1 g/ s-m²)] were multiplied by chemical-specific emission rates to produce values used to calculate environmental media concentrations.

Modeling was conducted using 5 years of data obtained from each of 41 meteorological stations assumed to be representative of the climatic regions throughout the country (see Section 4.3 for a discussion of meteorological site selection). Modeling was conducted using the median farm size area in each of the 41 climatic regions as the source area.

Air Concentrations of Vapor and Particles. ISCST3 estimates air concentrations of particles and vapors based on a number of variables, including wet and dry deposition and plume depletion. The model accounts for downwind movement of the plume containing airborne vapors and particles. It also accounts for dispersion of vapors and particles around the centerline of the plume as the plume travels in a downwind direction. Removal of constituent mass from the plume occurs as a result of wet and dry deposition. Wet and dry deposition are important processes in indirect exposure modeling because they account for the movement of constituent mass from the atmosphere to soil, water, and vegetation. Deposition is discussed below. There is, however, a closely related process, known as depletion, that affects the calculation of air concentrations.

Depletion is essentially the mirror of deposition. That is, while deposition accounts for the amount of constituent that moves to the ground, depletion accounts for the amount of mass removed from the atmosphere by deposition. The ISCST3 model allows the user to model depletion and deposition separately (i.e., the user may select depletion, deposition, or both depletion and deposition). When depletion is included, the mass deposited on the ground due to wet or dry deposition is removed from the plume, thereby avoiding double counting (U.S. EPA, 1995d). In this analysis, air concentration of particles was modeled with both wet and dry depletion activated. For vapors, ISCST3 was used to model only wet deposition and depletion, and dry deposition of vapors was

Summary of ISCST3 Modeling

- The wet and dry depletion option was activated in the dispersion modeling for particles. Wet depletion was considered for vapors.
- Area source was modeled for biosolids management.
- Modeling was conducted using unit emission rates.
- The rural option was used in the ISCST3 modeling because the agricultural management practices being assessed are typically in nonurban areas.
- Flat terrain was assumed.

calculated outside the model. As a result of calculating dry deposition/depletion of vapors outside ISCST3, the mass balance for vapors was not maintained and uncertainty was introduced into the air modeling calculation, which would tend to overpredict vapor air concentrations.

Wet Deposition of Particles and Vapor. Wet deposition is the deposition of material on a surface from a plume as a result of precipitation. The amount of material removed by wet deposition from the plume is a function of the scavenging rate coefficient, which is based on particle size (U.S. EPA, 1995d). To perform these calculations, wet deposition, wet depletion, and dry depletion were all selected in the input run-stream file. Precipitation data from the SAMSON CD-ROM (U.S. DOC and U.S. DOE, 1993) were required to process the meteorological inputs for this analysis.

Dry Deposition of Particles. Dry deposition refers to the deposition of material on a surface (e.g., ground, vegetation) from a plume of material as a result of processes such as gravitational settling, turbulent diffusion, and molecular diffusion. Dry deposition is calculated as the product of air concentration and dry deposition velocity. To calculate dry deposition,

ISCST3 requires mass mean diameter, particle density, and mass fraction to be input into the source pathway for deposition calculations (U.S. EPA, 1995b). Dry deposition calculations also require the meteorological input file to contain surface friction velocity, hourly Monin-Obukhov length, and surface roughness length. Surface friction velocity and hourly Monin-Obukhov length were calculated in the PCRAMMET preprocessor (U.S. EPA, 1995c). More detail on the PCRAMMET preprocessor is provided in Appendix G.

Dry Deposition of Vapors. Dry deposition of vapors was calculated using a step external to the ISCST3 model because chemical-specific dry deposition modeling within ISCST3 was precluded by time considerations. Using a dry deposition algorithm for particles (from the ISCST3 user's manual), dry deposition of vapor was calculated by multiplying the vapor air concentration by a default deposition velocity of 0.5 cm/s (Koester and Hites, 1992). This approach assumes that vapors behave as fine aerosols and, therefore, are amenable to modeling using the dry deposition algorithm for particles.

To calculate the weighted dry deposition velocity, land use was obtained from 1:250,000scale quadrangles of land use and GIRAS spatial data obtained from the EPA website and placed in an ARC-INFO format (U.S. EPA, 1994). Land use was based on data from the mid-1970s to the early 1980s. The fraction of time in each stability class was based on 5-year hourly meteorological files used in ISCST3 modeling.

Averaging Time. For the dioxins in the biosolids risk assessment, all human health risks were evaluated based on benchmarks for chronic, long-term exposure. Therefore, the air concentrations and deposition values required for the human health and ecological risk assessment were long-term averages. Long-term averages calculated by the ISCST3 model were annual averages. However, because the ISCST3 model was run using 5 years of meteorological data, it actually averages the hourly concentrations over the entire 5-year period.

Rural vs. Urban. The rural vs. urban setting in ISCST3 allows the user to account for differences between rural and urban environments. In urban environments, the built environment (e.g., buildings, roads, and parking lots) alters the dispersion character of the atmosphere, particularly at night because of building-induced turbulence and reduced nighttime cooling. Thus, there is greater nighttime mixing of constituents in urban areas compared with rural areas. For purposes of ISCST3 modeling, the urban classification applies mainly to large cities; even small cities and suburban areas are classified as rural for ISCST3 purposes. For this analysis, the rural setting was used.

Placements of Points Where Air Concentrations Were Calculated. A grid of points where air concentration and deposition values were calculated was established using a Cartesian grid. Air concentration and deposition values were produced for each point on the grid (i.e., x, y coordinate) at fixed distances ranging from 0 to 20,000 m from the edge of the management site. For the agricultural application, the receptors were placed on the field and at the following distances from the edge of the field: 100, 200, 500, 1,000, 1,500, 2,000, 3,000, 5,000, 7,500, 10,000, 15,000, and 20,000 m.

Flat vs. Elevated Terrain. The ISCST3 model allows the user to account for elevated terrain by specifying an elevation for each point on the grid where air concentrations and deposition values are calculated. This feature, however, is not available for use with area sources. Because all sources modeled in this analysis were area sources, elevated terrain was not considered.

TOXICS vs. Regulatory Mode. The most recent version of ISCST3 (99155, U.S. EPA, 1999a) allows the user to select a regulatory default option or to select a TOXICS option. The regulatory default option uses the Romberg numeric integration solution to estimate air concentration from an area source. Based on the results of validation tests performed by EPA, EPA concluded that the Romberg algorithm performs very well in terms of efficiency and reasonableness (U.S. EPA, 1992). However, this algorithm takes a significant amount of time to execute for large area sources. To improve model run times, the TOXICS option was added to the area source model by EPA. The TOXICS option also uses a Romberg numeric integration solution to estimate air concentrations and deposition rates near the management site. Farther from the site, however, the TOXICS option uses a two-point Gaussian Quadrature routine instead of the Romberg solution to estimate air concentration and deposition. The two-point Gaussian Quadrature solution is computationally more efficient, which accounts for the shorter model run time. For this study, a sensitivity analysis was conducted to compare the estimated air concentrations calculated using the regulatory option with those calculated using the TOXICS mode. This analysis showed small differences between results obtained using either option (see Appendix G). Given the benefit of reduced run times, the TOXICS option was selected for this analysis.

Source Shape. Agricultural land was modeled as a ground-level area source. The ISCST3 model allows the user to model area sources as polygonal sources with 3 to 20 sides (U.S. EPA, 1999a). The ISCST3 was set up in this analysis to model an area source as a square. This option was chosen because there are no actual data on the shape of sources, and a square source is assumed most like agricultural land.

5.2.1.3 <u>Preparing ISCST3 Input Files</u>. Two types of input files are required to run ISCST3, a run-stream file and a meteorological file. The run-stream file is an ASCII file that contains the model option settings, source parameters, and receptor locations. The meteorological file contains hourly values of windspeed, wind direction, stability class, mixing height, ambient air temperature, and precipitation type and amount.

ISCST Run-Stream Files. The ISCST3 run-stream file is composed of six pathways that drive different model functions: the Control Pathway, Source Pathway, Receptor Pathway, Meteorology Pathway, Terrain Grid Pathway, and Output Pathway. Each of these pathways is described in Appendix G. The Terrain Grid Pathway is not presented because it is used only with point sources (i.e., for facilities with stacks), which were not modeled in this analysis.

Meteorological Files. The meteorological file was generated using the meteorological preprocessor PCRAMMET (U.S. EPA, 1995c). The preprocessor pairs hourly surface observations with upper-air measurements. For each of the 41 meteorological stations modeled, 5 years of surface and upper-air data were used. The preprocessor creates a file in binary format

that contains hourly windspeed, wind direction, atmospheric stability class, temperature, and mixing height. Land use data also were required by PCRAMMET in the vicinity of each meteorological station to derive air model inputs, such as Bowen ratio, surface roughness height, minimum Monin-Obukhov length, noontime albedo, and the fraction of net radiation absorbed by the ground. Appendix G discusses the derivation of each of these model inputs.

5.2.1.4 Source Areas Modeled. In

the modeling analysis, application to agricultural land was considered. Because the ISCST3 model is sensitive to the size of the

Key Meteorological Data for the ISCST3 Model

Wind Direction determines the wind direction of the greatest impacts.

Windspeed is inversely proportional to ground-level air concentration, so the lower the windspeed, the higher the concentration.

Stability Class affects the rate of lateral and vertical diffusion. The more unstable the air, the greater the diffusion.

Mixing Height determines the height to which chemical constituents can be diffused vertically.

area source, the relationship between air concentrations and size of the area source was analyzed. For relatively small area sources, air concentrations increase significantly as the size of the area source increases. For large area sources, this increase in air concentrations is not as significant. The median farm size for each climatic region was modeled.

5.2.1.5 <u>Spatial Averaging of Air Concentrations and Deposition</u>. A GIS model was used to calculate air concentration and deposition rates for the buffer area, agricultural field, regional watershed, and waterbodies. This crucial step combines the spatial characterization of the buffer area, agricultural field, and waterbody in the site layout with air modeling outputs for each climatic region/management practice combination.

In an automated batch program, the ASCII files produced by ISCST3 were converted from a Cartesian array of values into an evenly spaced grid of concentration values distributed around the center of the site layout in the form of a GIS point coverage. To calculate the point estimate for a location, the program estimates the air concentration of vapors and particles from this GIS point coverage by selecting the grid point nearest that location. These values are used directly to determine human inhalation exposures.

To calculate the spatial averages for the buffer area, agricultural field, and waterbody polygons, the program individually overlays these areas with this point coverage and averaged the overlapping points. These mean concentration values and their associated identifiers are the output of the program and represent the average air concentrations and deposition values used in subsequent modeling steps to predict soils, water, and food chain concentrations. The distribution of average ambient air concentration estimated in the buffer using all sample concentrations used to estimate inhalation risks are presented in Table 5-3. This distribution also includes variability across the 41 climatic regions, as well as variability in agricultural practices.

Percentile	TCDD-TEQ Concentration in Ambient Air (ng/m ³)	Rural Air Background
50 th	2.3E-7	1.7E-5
75^{th}	4.8E-7	
90 th	9.5E-7	
95 th	1.4E-6	
99 th	3.1E-6	

Table 5-3. TCDD-TEQ Media Concentrationfor Ambient Air Variable Concentrations

The air concentration of dioxin congeners was influenced by several factors in the risk analysis that were ranked by the statistically based sensitivity analysis according to percentage of variation accounted for by the variable. This analysis has shown that the most important factors influencing the air concentrations of dioxins are

- Agricultural application rate (how many tons of biosolids are applied to the land per unit area)
- Number of years biosolids are applied to the land.

Other factors that are also important in this analysis are linked to the geographic location of the modeled farm:

Meteorological conditions (temperature, rainfall, windspeed, etc.)
 Soil conditions (soil foc, soil bulk density, etc.)
 Area of the farm where biosolids are assumed to be applied.

All of the factors linked to location influence the release of dioxin congeners to the air to varying degrees. The location that is linked to 26 of the 30 iterations in the highest 1 percent of the air concentrations is Phoenix, AZ. This is not unexpected in that this is an extremely hot location with large farm areas. The other locations represented in the highest 1 percent of air concentrations are Tampa, FL, and Fresno, CA, also hot locations. Other locations associated with the top 10 percent of air concentrations are mostly more southern areas (Houston, TX; Meridian, MS; Atlanta, GA; Charleston, SC; Shreveport, LA; and Las Vegas, NV) or areas with very large average farm sizes (i.e., western climate regions; Bismark, ND; Boise, ID; Boulder, CO; Casper, WY). This indicates that ambient temperature is an important climatic parameter.

5.2.2 Estimation of Soil and Sediment Concentrations

This section describes the components that make up the waterbody model and those portions of the watershed model that simulate fate and transport of chemicals that have been aerially deposited and eroded. There are two different types of watersheds-local watersheds, which contain the agricultural land where biosolids are applied that are subject to sheet flow runoff and erosion directly from the field, and regional watersheds. The regional watershed is the drainage area upstream of the modeled waterbody. Fate and transport of eroded chemical from the agricultural field downslope across the buffer area and into the waterbody is simulated by the source model as described in Section 5.1. The watershed models discussed below consider only chemical that is airborne from either the crop or pasture and subsequently deposited onto the residential buffer area of the local watershed¹ and the regional watershed. Thus, eroded chemical contaminating the buffer area or entering the waterbody from the local watershed consists of two components: (1) direct erosion from agricultural field and (2) aerially deposited and subsequently eroded. These two components are summed to determine total chemical in the buffer soils and load entering the waterbody. For the regional watershed, only the aerially deposited component is relevant. Discussions in this section are general in nature. Two appendices support the discussions with more detailed information: Appendix H contains the full set of equations used to calculate media concentrations, and Appendix D lists the physical/chemical properties used; the parameter values selected for fate, transport, and exposure modeling; and citations for the parameter values selected.

5.2.2.1 <u>Predicting Soil Concentrations</u>. Soil concentrations due to aerially deposited chemical were calculated for the buffer area of the local watershed and the entire regional watershed. Soil concentrations are determined by the deposition flux of chemical and loss mechanisms of that chemical from the soil. Soil losses accounted for in this analysis include only erosion. Other losses, such as biodegradation, volatilization, leaching, and dissolved loss in surface runoff, were assumed to be negligible for the chemicals considered in this analysis.

Soil concentrations in the regional watershed were calculated using the solution to a differential equation that expresses soil concentrations over time as a function of loadings and first-order losses, as presented in Equation 5-7. This equation is based on the soil concentration equation presented in the Draft Dioxin Reassessment Document (U.S. EPA, 2000) and was modified for this application to include aerially deposited loads of congeners as inputs. One of the fundamental underlying assumptions of the approach used for the regional watershed is that the soil compartment can be modeled as completely mixed. No losses other than erosion losses were assumed in this application, i.e., $K_s = 0$.

¹ Aerial deposition from a crop or pasture back onto itself is not considered, nor is aerial deposition from the crop onto the pasture, or vice versa; only deposition onto the buffer is considered.

C_{soil t}

$$C_{soil_{t}} = C_{soil_{t}} + \frac{Dep + Load}{SoilR + K \times Mass} \times 1 - e^{\left(\frac{SoilR}{Mass} + K_{s}\right) \times T}$$
(5-7)

Parameter	Definition	Value
C_{soil_t}	Total soil concentration	Calculated
C_{soil_i}	Initial soil concentration (mg/kg)	Calculated
Dep	Deposition term for soil (mg/yr)	Calculated in Table H-2.8
Load	Mass of contaminant loaded to soil (mg/yr)	Calculated in Table H-2.17
Soil R	Mass of soil removed from site (kg/yr)	Calculated in Table H-2.22
K _s	Soil loss constant (1/yr)	Calculation
Mass	Mass of soil (kg)	Calculated in Table H-2.20
Т	Time for which soil concentration is being calculated (yr)	

Source: Based on U.S. EPA (2000) with values for deposition load added into the equation. Note: Depending on the value of T, this equation is used to calculate Csoil _{t1}, Csoil _{t2}, Csoil _{td}. The value for T

The USLE was used to estimate soil erosion losses (X_e) as shown in Equation 5-8. The USLE is an empirically derived equation originally developed by the SCS of the USDA to estimate soil erosion losses from agricultural fields during soil conservation planning. The USLE is applied in the context of the Gross Erosion Sediment-Delivery Ratio method outlined in USDA (1978) and described in greater detail in the SCS *National Engineering Handbook* (USDA, 1971). Gross erosion is defined as the summation of erosion from all sources within a watershed, as estimated for sheet and rill erosion by USLE. The sediment delivery ratio adjusts gross erosion rates to account for terrain and cover features, which effectively reduce sediment erosion.

Constituent loadings to soil in the buffer area and the regional watershed area due to aerial deposition of vapors and particles were calculated using Equation 5-9.

Soil constituent concentration changes with each year of application of biosolids to the agricultural land. During the application period, the dioxin concentrations in soils resulting from aerial deposition steadily increase for such a persistent chemical. This temporal change, combined with the assumption that a receptor can begin his or her exposure duration at any time during the facility operation period, is accounted for in the soil concentration model by dynamically estimating the soil concentration at the beginning of the exposure duration and the soil concentration at the end of the exposure duration and determining the average concentration over the exposure period.

is determined in either Csoil $_{1F}$ or Csoil $_{2F}$.

X _e		
$X_e = R \times K \times LS \times C \times P \times \frac{907.18}{4047} $ (5-8)		
Parameter	Definition	Value
X _e	Loss due to erosion (kg/m ² /yr)	Calculated
R	USLE rainfall/erosivity factor (1/yr)	See Appendix E
К	USLE soil erodibility factor (short tons/acre)	See Appendix E
LS	USLE length-slope factor (unitless)	Calculated in Table H-2.20
С	USLE cover management factor (unitless)	See Appendix C
Р	USLE supporting practice factor (unitless)	See Appendix C
907.18	Conversion factor (kg/short tons)	

Source: U.S. EPA (1998).

4047

Dep

Conversion factor (m²/acres)

$$D_{ydv} = 0.31536 \times C_{yv} \times V_{dv}$$

$$Dep = 1000 \times Q \times Area \times \left[F_v \times \left(D_{ydv} + D_{ywv} \right) + \left(1 - F_v \right) \times \left(D_{ydp} + D_{ywp} \right) \right]$$
(5-9)

Parameter	Definition	Value
Dep	Deposition term for soil (mg/yr)	Calculated
0.31536	Unit conversion factor (m-g-s/cm-µg-yr)	
C _{yv}	Normalized vapor-phase air concentration (µg-s-m ² /gm ³)	See Appendix G
V _{dv}	Dry deposition velocity (cm/s)	See Appendix D
1000	Unit conversion (mg/g)	
Q	Emission rate from source (g/s-m ²)	Calculated by source model
Area	Area of deposition (m ²)	See Appendix E
F _v	Fraction of air concentration in vapor phase (unitless)	See Appendix D
\mathbf{D}_{ydv}	Normalized annual average dry deposition from vapor phase (s-m ² /m ² -yr)	Calculated
$\mathbf{D}_{\mathrm{ywv}}$	Normalized annual average wet deposition from vapor phase $(s-m^2/m^2-yr)$	See Appendix G
D _{ydp}	Normalized annual average dry deposition from particle phase (s- m^2/m^2 -yr)	See Appendix G
D _{ywp}	Normalized annual average wet deposition from particle phase $(s-m^2/m^2-yr)$	See Appendix G

Source: U.S. EPA (1998).

Table 5-4 presents the soil concentrations estimated in the buffer area, tilled cropland, pasture, and stream sediment using all the samples of dioxins, furans, and PCBs as variable congener concentrations in the model.

The individual congeners in each biosolids sample are modeled individually in the source partition model. However, the congeners remained linked throughout the modeling by their sample number. Thus, all media concentrations of the dioxin congeners can be summed using the TEF system to produce a single TEQ media concentration resulting from the application of biosolids represented by a specific sample. Therefore, the results of the probabilistic source partition model may be expressed as a distribution of TEQ soil concentration in the crop, pasture, and buffer soils. Table 5-4 presents TEQ soil concentrations that show specific percentiles from this distribution. The background concentration for rural soil is 2.5 ng/kg TCDD-TEQ, and the national average background concentration for sediment is 5.8 ng/kg TCDD-TEQ.

	TCDD-TEQ Concentration (ng/kg)				
Percentile	Buffer	Crop Land	Pasture	Sediment	Applied Biosolids
50 th	0.6	0.3	1.5	0.04	24
75 th	1.0	0.6	2.6	0.08	35
90 th	1.6	0.9	4.2	0.15	55
95 th	2.3	1.4	5.7	0.25	74
99 th	8.4	3.4	21.4	0.7	453

Table 5-4. TCDD-TEQ Media Concentration for Soil in Buffer, Crop Land, Pasture, and Sediment Variable Concentrations

The dioxin congener concentrations in the soils of the crop land, pasture, and residential area are influenced by the following factors in the risk analysis. These factors were identified by the statistically based sensitivity analysis described in detail in Section 8.1.2.6 and are ranked according to the percentage of variation they account for in the estimation of the soil concentrations:

The year during biosolids application that the farm family moves to the farm Agricultural application rate (tons of biosolids applied to the land per unit area) Number of years biosolids are applied to the agricultural area.

Other less important factors in this analysis as ranked by the sensitivity analysis are linked to the geographic location of the modeled farm:

Soil conditions (soil foc, soil bulk density, etc.) Meteorological conditions (temperature, rainfall, windspeed, etc.) Area of the farm where biosolids are assumed to be applied.

The greater the loading of biosolids to the soil during the period of time the farm family is exposed, the greater the soil media concentration to which the family is exposed. The farm family is assumed to move to the farm where biosolids are applied during the period of biosolids application. The later the farm family moves to the farm, the greater their estimated exposure because dioxins are persistent and accumulate during the period of application and then remain at high levels for many years after applications cease.

All of the factors linked to location influence the concentration of dioxin congeners in the soil. The locations linked to the highest 1 percent of the soil concentrations are more varied than the locations associated with the highest 1 percent of air concentrations. The highest 1 percent of soil concentrations are linked to cooler locations with smaller average farm sizes. There is no single location that is linked to more than 5 of the top 30 soil concentrations. The locations and the number of iterations in the top 30 for the location are Little Rock, AR (5); Burlington, VT (4); Salem, OR (4); Seattle, WA (4); Muskegon, MI (3); Boise, ID (3); Bismark, ND (2); Winnemucca, WI (2); Minneapolis, MN (1); Salt Lake City, UT (1); and Chicago, IL (1).

5.2.2.2 Predicting Surface Water Concentrations. The waterbody in this analysis is a stream located downslope of the waste management unit. For modeling purposes, the stream is shaped as a rectangle 5.5 m wide and as long as the width of the agricultural fields. It was assumed that the stream is 5.5 m because this width is the median of a third-order fishable stream (van der Leeden et al., 1990). A third-order stream refers to a type of stream segment classification. In this classification scheme, a first-order stream segment is one with no tributaries. That is, a first-order stream segment receives all of its flow from runoff from the surrounding watershed soils. A second-order stream segment occurs when two first-order stream segments come together. A third-order stream segment occurs when two second-order stream segments come together, but not when a second-order and a first-order stream segment combine. The third-order stream segment, therefore, has the combined flow of at least two second-order stream segments. The third-order stream was selected because it reasonably represents the smallest waterbody that would routinely support recreational fishing of consumable fish.

Constituents can enter the waterbody by one of four pathways:

- Constituents in the air above the waterbody can be deposited directly onto the waterbody's surface. This occurs for airborne particles via dry and wet deposition due to gravitational settling and scavenging by precipitation, respectively.
- Vapors can also deposit directly onto the waterbody's surface via scavenging by precipitation (i.e., wet deposition).
- Constituents on the soils in the local watershed can enter the waterbody through runoff and erosion.

Constituents on the soils in the upstream regional watershed can also enter the waterbody through runoff and erosion.

Thus, the total chemical load to the waterbody is the sum of

- a. Direct atmospheric inputs
- b. Eroded load from the local watershed (which itself is the sum of chemical eroded directly from the agricultural fields plus aerially deposited and eroded load from the buffer)
- c. Eroded load from the regional watershed.

Once in the waterbody, constituents are assumed to be uniformly mixed in a single stream segment. There is water flow in and out of the stream segment, which is predicted by the Regional Watershed Model as described next. Water flowing into the upstream boundary of the waterbody is assumed to have a constituent concentration determined by the application of the soil concentration algorithms described in Section 5.2.2.1 applied to the soils in the regional watershed. The waterbody is modeled based on the waterbody model described in the Draft Dioxin Reassessment Document (U.S. EPA, 2000). The equations used are presented in Appendix H; they partition the chemical mass into chemical sorbed to suspended solids in the water column and chemical sorbed to sediment solids. The soluble fractions are assumed to be zero.

Regional Watershed Model and Waterbody Streamflow. Because the chemicals of concern in this analysis have a strong tendency to be both persistent and accumulative in soils and sediments, it was considered essential to include in the analysis inputs to the waterbody that result from aerial deposition over the upstream watershed and subsequent erosion. As discussed, that upstream watershed is termed here the regional watershed to distinguish it from the local watershed, i.e., the hillside area containing the agricultural fields or landfill. Chemicals deposited onto the regional watershed will be transported in their particulate form on eroded soils into the waterbody network that drains the regional watershed and hence downstream into the modeled waterbody where fishing is assumed to occur. (Not all soils that are eroded from the regional watershed complete the journey downstream to the modeled waterbody. A sediment delivery ratio is included in the calculations that estimates the fraction of mobilized soil that actually arrives at the modeled waterbody as a function of regional watershed area.) A schematic diagram illustrating the regional watershed and its relationship to the agricultural fields and the local watershed is shown in Figure 3-1.

The regional watershed was modeled to provide estimates of two inputs to the modeled waterbody: streamflow and chemical loads associated with eroded soil. Suspended solids concentrations in the waterbody were not modeled, but were assumed to be a constant of 10 mg/L, in accordance with the Draft Dioxin Reassessment Document (U.S. EPA, 2000). Chemical loads associated with eroded soils were estimated using the same equations used for aerially deposited chemical in the local watershed, as discussed in Section 5.2.2.1. Methods used for estimating streamflow are discussed below.

Streamflow in the modeled waterbody consists of surface runoff from the upstream regional watershed, a baseflow component, and surface runoff from the local watershed (for agricultural fields only). Surface runoff from the upstream regional watershed was estimated using the hydrology algorithm from the source partition model. Because the hydrology algorithm is not stand-alone, executing it required running the source partition model in a mode termed the "LAU as Regional Watershed" (see Appendix F). In this mode, the model is run to estimate the surface water runoff.

Baseflow represents the component of streamflow that is not direct surface runoff. Baseflow was estimated as a function of regional watershed area and U.S. Geological Survey (USGS) Hydrological Unit Code (HUC) number using regional regression models. These regression models predict HUC-specific 30Q2 low flows as a function of watershed area. The 30Q2 flow is a statistical estimate of the 30-day average low flow expected to occur, on average, every other year (2-year return period). The 30Q2 low flow was assumed (for this analysis) to be a reasonable representation of stream baseflow.

The third component of streamflow is direct surface runoff from the local watershed, i.e., the tributary hillside containing the agricultural fields. This runoff is a modeled output of the model used for the agricultural fields; however, the monofill is assumed to have no runoff. Thus, surface runoff for the agricultural field local watershed was available and was used as a contributor to total streamflow. For the landfill (monofill) scenario, streamflow is composed only of surface runoff from the upstream regional watershed and baseflow.

5.3 Calculation of Food Chain Concentrations

Constituents can pass from contaminated air, soil, and surface water to reach individuals through the food chain. For example, constituents that are entrained in air may be deposited on plants growing in the agricultural field or home garden. Constituents from the air and soil may accumulate in fruits and vegetables that are consumed by people. In addition, beef and dairy cattle may feed on forage and silage that are grown in biosolids-amended soil. The beef and dairy products may be subsequently consumed by people. Free-range chickens may also consume contaminated soil. Similarly, constituents that erode into surface water may accumulate in fish, which are subsequently consumed by a recreational fisher.

This section presents the methodology used to calculate contaminant concentrations for each of the food chain pathways considered. An approach was developed for a terrestrial food chain to calculate concentrations of produce, poultry, eggs, beef, and milk that are consumed by the adult and child farmer evaluated in this assessment. In addition, an approach was developed for an aquatic food chain to calculate concentrations in fish that may be consumed by a recreational fisher.

5.3.1 Terrestrial Food Chain

The terrestrial food chain is designed to predict the accumulation of a contaminant in the edible parts of aboveground vegetation from direct deposition of contaminants in air. Concentrations are predicted for three main categories of food crops presumed to be eaten by

humans: exposed fruits, exposed vegetables, and root vegetables. The term "exposed" refers to the fact that the edible portion of the produce is exposed to the atmosphere. Examples of the three categories include tomatoes (exposed vegetable), apples (exposed fruit), and potatoes (root vegetables). Figure 5-4 shows the data flow into and out of the food chain model.



Figure 5-4. Biosolids application to agricultural fields media concentration module.

In addition, the terrestrial food chain estimates the contaminant concentration in farm crops for cattle. Vegetation consumed by cattle includes grain, forage, and silage. Forage is considered exposed vegetation. Silage is calculated as exposed vegetation; however, an empirical correction factor for silage takes into account that silage is partly protected and partly exposed.

Table 5-5 summarizes the mechanisms by which vegetation can be exposed to contaminants. The two mechanisms are deposition of particle-bound contaminants to exposed plant tissues and vapor-phase deposition of contaminants to exposed plant tissues. Exposed vegetation is subject to contamination via particulate deposition and vapor-phase deposition, while protected vegetation is not contaminated because the edible portion of the vegetation is not in direct contact with air.

Type of Vegetation	Particulate Deposition	Vapor-Phase Deposition
Human ingestion		
Exposed vegetables	\checkmark	\checkmark
Exposed fruit	1	1
Beef and dairy cow ingestion		
Forage	\checkmark	\checkmark
Silage	1	 Image: A set of the set of the

Table 5-5. Terrestrial Food Chain Vegetation

5.3.1.1 <u>Aboveground Vegetation</u>. Aboveground vegetation is subject to contamination via deposition of particle-bound contaminants and vapor transfer of contaminants. Equation 5-10 is used to calculate the concentration of contaminant in aboveground vegetation.

Deposition of Particle-Bound Contaminants. Airborne particle-bound contaminants are deposited by wet and dry deposition; thus they affect only exposed vegetation. As described earlier, the air dispersion model ISCST3 was used to calculate the wet and dry deposition rates for the particle-bound contaminants. Not all airborne particles will settle on a plant's edible surface. Some will fall to the ground; others will fall on other surfaces that will undergo weathering processes, such as wind removal, water removal, and growth dilution; and most will end up in the soil or eroded soil. Thus, only a fraction of the total deposition rate per area is used to estimate the amount of airborne particles that contacts the edible portion of the plant.

The calculation of vegetative concentration due to deposition also takes into account the length of time plants are exposed to contaminants. One determination of the length of exposure is the growing season. For instance, the time from when a tomato begins to grow until it is harvested equals its length of exposure to deposition. The productivity level of the plant or biomass is also a factor. The biomass is determined by the amount of standing crop for the average farm. The biomass is needed to take into account the dilution of constituent by biomass growth. Equation 5-11 is used to calculate the concentration of congeners due to direct deposition.

$$P_{veg}$$

$$P_{veg}_{ww} = \left(P_{d_{veg}} + P_{v_{veg}} + P_{r_{veg}}\right) \times \frac{(100 - MAF)}{100}$$

$$P_{veg}_{DW} = \left(P_{d_{veg}} + P_{v_{veg}} + P_{r_{veg}}\right)$$
(5-10)

Parameter	Definition	Value
P _{veg}	Vegetation concentration (mg/kg)	Calculated
P_{veg_ww}	Vegetation concentration [wet weight (mg/kg-WW)]	Calculated
P_{veg_DW}	Vegetation concentration [dry weight (mg/kg-DW)]	Calculated
P_{d_veg}	Vegetative concentration due to direct deposition (mg/kg - DW)	Calculated in Table H-3.12
P_{v_veg}	Vegetative concentration due to air-to-plant transfer (mg/kg - DW)	Calculated in Table H-3.14
P _{r_veg}	Aboveground vegetation concentration due to root uptake, zero for this analysis (mg/kg - DW)	0
MAF	Plant tissue-specific moisture adjustment factor to convert DW concentration into WW (percent)	

Source: U.S. EPA (1998).

Note: For exposed vegetation, MAF is 92; for exposed fruit, MAF is 85. Dry weight (DW) is used for silage and feed. Wet weight (WW) is used for exposed vegetation and exposed fruit.

\boldsymbol{P}_d

$P_d = \frac{\left(D_p \times R_p\right)}{\left(V_{p_1} \times V_{p_2}\right)} \tag{5-1}$			
Parameter	Definition $(Y_p \times K_p Par)$	Value	
\mathbf{P}_{d}	Vegetation concentration due to air deposition (mg/kg DW)	Calculated	
D_p	Deposition term for plants (mg/m ² -yr)	Calculated	
R _p	Interception fraction - aboveground vegetables (fraction)	See Appendix I Exposed fruits and vegetables 0.48 Forage 0.35 Feed 0.62	
Yp	Crop yield (kg DW/m ²)	See Appendix I Exposed fruits and vegetables 1.17 Forage 0.15 Feed 0.63	
K _p Par	Plant surface loss coefficient, particulate (1/yr)	18.07	

Source: U.S. EPA (2000).

Vapor-Phase Transfer of Contaminants. The concentration of contaminants due to vapor-phase transfer depends on the constituent being considered. Evidence shows that wet deposition is negligible and contact of vapor phase with the plant surface is the primary mechanism of plant uptake; therefore, a different equation is used based on the vapor-phase air concentration of the constituent. Equation 5-12 is used to calculate the concentration of congeners in aboveground vegetation due to air-to-plant transfer.

Vapor-phase transfer for high log K_{ow} constituents, such as dioxins, furans, and coplanar PCBs, uses a congener-specific air-to-plant biotransfer factor to estimate the concentration of contaminants in vegetation. The air-to-plant biotransfer factor is defined as the ratio of contaminant concentration in exposed plant parts to the vapor-phase concentration of contaminant in air. The biotransfer factors have been measured for these constituents (U.S. EPA, 2000). In addition, an empirical correction factor (VG_{AG}) is recommended by EPA (U.S. EPA, 1997) to be applied to the calculation of concentrations in each type of vegetation. The factor is used to adjust the air-to-plant bioconcentration factors that are developed using the different types of vegetation considered in this analysis. This factor also is applied to take into account the difference between outer-surface and whole-plant concentrations. This is important for lipophilic organic chemicals that tend to remain on the outer portion of the plant surface because washing and peeling fruits and vegetables reduces the outer surface residues. Because silage is assumed to be partly protected and partly exposed, the correction factor for silage takes into account that some of the vegetation is not contaminated as a result of vapor deposition onto plant surfaces. Table 5-6 presents the percentile concentrations estimated in aboveground fruits and vegetables using all biosolids samples of dioxins, furans, and PCBs as variable congener concentrations in the model.

$$P_{\nu} = \frac{\left(C_{\nu a p o r} \times B_{\nu} \times V G_{a g} \times 1000\right)}{1200}$$
(5-12)

Parameter	Definition	Value
P _v	Plant concentration due to vapor (mg/kg DW)	Calculated
C _{vapor}	Concentration of vapor (mg/m ³)	Calculated
$\mathbf{B}_{\mathbf{v}}$	Air-to-plant biotransfer factor ($\mu g/g DW plant/\mu g/g air$)	Constituent-specific
VGAG	Empirical correction factor for aboveground vegetables (unitless)	Exposed fruits and vegetables 0.1 Forage 1.00 Feed 0.5
1000	Conversion factor (g/kg)	1,000
1200	Rho - the density of air (g/m^3)	1,200

Source: U.S. EPA (1998).

	TCDD-TEQ Concentration (ng/kg)	
Percentile	Fruits	Vegetables
50 th	0.00010	0.00005
75 th	0.00024	0.00013
90 th	0.00046	0.00024
95 th	0.00069	0.00037
99 th	0.0018	0.00096

Table 5-6. TCDD-TEQ Media Concentration for Exposed Fruits and Vegetables Variable Concentrations

The exposed aboveground vegetation media concentrations of dioxins are driven by the air concentrations of vapors; therefore, the factors that increase the vapor concentrations increase the aboveground vegetation concentrations also.

5.3.1.2 <u>Belowground Vegetation</u>. In belowground plants, roots can take in contaminants from the soil that may accumulate in the edible portion of the plant. For organdies, the calculation is a function of the root concentration factor, which is used to estimate the amount of constituent moving from the soil into the root vegetable. Equation 5-13 gives the equation for calculating the concentration of congeners in root vegetables.

 P_{r_bg}

$$P_{r_{bg}} = \frac{\left(C_{soil} \times RCF \times VG_{bg}\right)}{(Kd)}$$
(5-13)

Parameter	Definition	Value
P _{r_bg}	Concentration in root vegetables (mg/kg)	Calculated
C _{soil}	Concentration of contaminant in soil (mg/kg)	Calculated
RCF	Root concentration factor $(\mu g/g - WW plant)/(\mu g/mL soil water)$	Congener-specific
VG _{bg}	Empirical correction factor for belowground vegetables (unitless)	0.25
Kd	Soil water partition coefficient	Calculated

Source: U.S. EPA (1998).

In addition, an empirical correction factor (VG_{BG}) is applied to the concentrations in belowground vegetables. The correction factor is applied to adjust the root concentration factor so that it is appropriate for bulky belowground root crops. This factor adjusts for the concentration gradient from the outside of the root vegetable to the center. Another factor also accounts for constituent losses due to cleaning and cooking and the tendency of lipophilic contaminants to remain in the outer portions of the root (U.S. EPA, 1997). Table 5-7 presents the percentile concentrations estimated in belowground fruits and vegetables using all biosolids samples of dioxins, furans, and PCBs as variable congener concentrations in the model.

Percentile	TCDD-TEQ Concentration in Belowground Vegetables (ng/kg)	
50 th	0.0071	
75^{th}	0.014	
90 th	0.028	
95 th	0.040	
99 th	0.090	

Table 5-7. TCDD-TEQ Media Concentrationfor Belowground VegetablesVariable Concentrations

The dioxin congener concentrations in root crops are influenced by the following factors in the risk analysis. These factors were identified by the statistically based sensitivity analysis (see Appendix K) and are presented according to the percentage of variation they account for in the estimation of the root concentrations:

Soil foc

The year during biosolids application that the farm family moves to the farm Agricultural application rate (tons of biosolids applied to the land per unit area) Number of years biosolids are applied to the agricultural area.

Another factor important in this analysis is linked to the geographic location of the modeled farm:

Soil bulk density.

The greater the loading of biosolids to the soil during the period of time the farm family is exposed, the greater the soil media concentration. The farm family is assumed to move to the farm where biosolids are applied during the period of biosolids application. The later the farm family moves to the farm, the greater their estimated exposure because dioxins are persistent and

accumulate during the period of application and then remain at high levels for many years after applications cease.

All of the factors linked to location influence the behavior of dioxin congeners in soil to varying degrees. The locations that are linked to the highest 30 estimations of the root concentrations are Las Vegas, NV (21), and Phoenix, AZ (9). The soils in these locations are very arid and have low organic content. These are the properties identified by the sensitivity analysis.

5.3.1.3 Animal Tissue Concentration. The animal products considered in this risk analysis are beef and milk from beef and dairy cattle, respectively, and poultry and eggs. The contaminant concentrations in beef tissue and milk were estimated based on the amount of contaminant the cattle were assumed to have consumed through ingestion. Specifically, the diet for cattle was assumed to comprise a specific fraction of soil, forage, and silage. The animals were assumed to ingest soil, with which they come in contact during grazing or other activities on untilled soils. Different diet fractions were used for beef and dairy cattle, depending on the amount of feed they consume and the activity patterns of the animals. For example, beef cattle are assumed to spend more time grazing and, therefore, have a higher incidental ingestion rate of soil and forage. The animal concentrations also depend on biotransfer factors, which are the ratio of the contaminant concentration in animal tissue to the daily intake of contaminant by the animal. Congener-specific biotransfer factors derived for milk were suggested for use for both milk and beef in the Draft Dioxin Reassessment Document (U.S. EPA, 2000). Chemical concentrations in feed and soil are multiplied by their respective diet fraction and by constituentspecific biotransfer factors and then summed to obtain the concentration of individual constituents in tissue. Equation 5-14 is used to calculate the concentration of congeners in beef. Equation 5-15 is used to calculate the concentration in milk.

Table 5-8 presents the percentile concentrations estimated in beef using all biosolids samples of dioxins, furans, and PCBs as the variable congener concentrations in the model. Table 5-9 presents the percentile concentrations estimated in milk using all biosolids samples of dioxins, furans, and PCBs as the variable congener concentrations in the model.

 A_{beef}

$$A_{beef} = C_{fat} \times 0.2$$

(5-14)

$$C_{fat} = (BCF_{cattle} \times FF) \times (DF_{beef_{soil}} \times B_s \times C_{soil} + DF_{beef_{forage}} \times P_{forage} + DF_{beef_{feed}} \times P_{feed})$$

Parameter	Definition	Value
A _{beef}	Concentration in beef (mg/kg)	Calculated
C _{fat}	Concentration of dioxin (2,3,7,8-TCDD) in beef fat (mg/kg)	Calculated
0.2	Fraction of fat in beef (unitless)	
BCF _{cattle}	Bioconcentration ratio of contaminant as determined from cattle vegetative intake (pasture grass or feed)	See Appendix D Congener-specific
FF	Feedlot factor for beef fat calculation (<=1 for beef fat and = 1 for milk fat) (unitless)	See Appendix I 1.0
DF _{beef soil}	Fraction of cattle diet that is soil (unitless)	0.04
B _s	Bioavailability of contaminant on the soil vehicle relative to the vegetative vehicle (unitless)	0.65
C _{soil}	Average contaminant soil concentration (mg/kg)	Calculated in Tables H-2.3, H-2.4
$\mathrm{DF}_{\mathrm{beef}_\mathrm{forage}}$	Fraction of cattle diet that is pasture grass (unitless)	0.48
P _{forage}	Average concentration of contaminant on pasture grass (mg/kg)	Calculated
$\mathrm{DF}_{\mathrm{beef}_\mathrm{feed}}$	Fraction of cattle diet that is feed (unitless)	0.48
P _{feed}	Average concentration of contaminant in feed (mg/kg)	Calculated

Source: U.S. EPA (2000).

 A_{milk}

$$A_{milk} = C_{fat} \times 0.04$$

(5-15)

$$C_{fat} = \left(BCF_{cattle} \times FF\right) \times \left(DF_{dairy_{soil}} \times B_s \times C_{soil} + DF_{dairy_{forage}} \times P_{forage} + DF_{dairy_{feed}} \times P_{feed}\right)$$

Parameter	Definition	Value
A _{milk}	Concentration in milk (mg/kg)	Calculated
C _{fat}	Concentration of dioxin (2,3,7,8-TCDD) in milk fat (mg/kg)	Calculated
0.04	Fraction of fat in milk (unitless)	
BCF _{cattle}	Bioconcentration ratio of contaminant as determined from cattle vegetative intake (pasture grass or feed) (unitless)	See Appendix D Congener-specific
FF	Feedlot factor for beef fat calculation (≤ 1 for beef fat and = 1 for milk fat) (unitless)	1.0
DF _{dairy_soil}	Fraction of cattle diet that is soil (unitless)	0.02
B _s	Bioavailability of contaminant on the soil vehicle relative to the vegetative vehicle (unitless)	See Appendix I 0.65
C _{soil}	Average contaminant soil concentration (mg/kg)	Calculated in Tables H-2.3, H-2.4
DF _{dairy_forage}	Fraction of cattle diet that is pasture grass (unitless)	0.08
P _{forage}	Average concentration of contaminant on pasture grass (mg/kg)	Calculated
DF _{dairy_feed}	Fraction of cattle diet that is feed (unitless)	0.90
P _{feed}	Average concentration of contaminant in feed (mg/kg)	Calculated

Source: U.S. EPA (2000).

Percentile	TCDD-TEQ Concentration in Beef (ng/kg)
50 th	0.088
75^{th}	0.16
90 th	0.28
95 th	0.40
99 th	0.86
Background	0.29

Table 5-8. TCDD-TEQ Media Concentration by Percentile for Beef Variable Concentrations

Table 5-9. TCDD-TEQ Media Concentration by Percentile for Milk Variable Concentrations

Percentile	TCDD-TEQ Concentration in Milk (ng/kg)
50 th	0.0081
75 th	0.015
90 th	0.027
95 th	0.038
99 th	0.094
Background	0.047

The contaminant concentrations in poultry and eggs were estimated based on the amount of contaminant the chickens were assumed to have consumed through ingestion. Specifically, the diet for chickens was assumed to comprise a specific fraction of soil and feed. The animals were assumed to ingest soil with which they came in contact during free-range activities in the contaminated chicken yard. The chicken diet was assumed to contain 20 percent soil. The chicken feed was assumed to be uncontaminated. Bioaccumulation factors specific for chickens and eggs and based on ingestion of contaminated soils are from the Draft Dioxin Reassessment Document (U.S. EPA, 2000). Equations 5-16 and 5-17 are used to calculate the concentration of congeners in poultry and eggs, respectively.

Table 5-10 presents the percentile concentrations estimated in poultry using all biosolids samples of dioxins, furans, and PCBs as variable congener concentrations in the model. Table 5-11 presents the percentile concentrations estimated in eggs using all biosolids samples of dioxins, furans, and PCBs as variable congener concentrations in the model.

 $A_{poultry} = C_{fat} \times 0.1$

(5-16)

$$C_{fat} = BCF_{poultry} \times \left(DF_{poultry_{soil}} \times B_s \times C_{soil} + DF_{poultry_{forage}} \times P_{forage} + DF_{poultry_{feed}} \times P_{feed} \right)$$

Parameter	Definition	Value
A _{poultry}	Concentration in poultry (mg/kg)	Calculated
C _{fat}	Concentration of dioxin (2,3,7,8-TCDD) in chicken fat (mg/kg)	Calculated
0.1	Fraction of fat in poultry (unitless)	
BCF _{poultry}	Bioconcentration ratio of contaminant developed for chicken vegetative intake (unitless)	See Appendix D Congener-specific
$DF_{poultry_soil}$	Fraction of chicken diet that is soil (unitless)	See Appendix I 0.05
B _s	Bioavailability of contaminant on the soil vehicle relative to the vegetative vehicle (unitless)	See Appendix I 0.65
C _{soil}	Average contaminant soil concentration (mg/kg)	Calculated in Tables H-2.3, H-2.4
$DF_{poultry_forage}$	Fraction of chicken diet that is incidental vegetation while free ranging (unitless)	See Appendix I 0.05
P _{forage}	Average concentration of contaminant on free-range vegetation (mg/kg)	Calculated
$DF_{poultry_feed}$	Fraction of chicken diet that is feed (unitless)	See Appendix I 0.85
\mathbf{P}_{feed}	Average concentration of contaminant in feed (mg/kg)	0

Source: U.S. EPA (2000).

 A_{eggs}

$$A_{eggs} = C_{fat} \times 0.1$$

(5-17)

$$C_{fat} = BCF_{egg} \times \left(DF_{poultry_{soil}} \times B_s \times C_{soil} + DF_{poultry_{forage}} \times P_{forage} + DF_{poultry_{feed}} \times P_{feed} \right)$$

Parameter	Definition	Value		
$\mathbf{A}_{\mathrm{eggs}}$	Concentration in eggs (mg/kg)	Calculated		
C _{fat}	Concentration of dioxin (2,3,7,8-TCDD) in egg fat (mg/kg)	Calculated		
0.1	Fraction of fat in eggs (unitless)			
BCF _{egg}	Bioconcentration ratio of contaminant developed for chicken vegetative intake (unitless)	See Appendix D Congener-specific		
$DF_{poultry_soil}$	Fraction of chicken diet that is soil (unitless)	See Appendix I 0.10		
B _s	Bioavailability of contaminant on the soil vehicle relative to the vegetative vehicle (unitless)	See Appendix I 0.65		
C _{soil}	Average contaminant soil concentration (mg/kg)	Calculated in Tables H-2.3, H-2.4		
$DF_{poultry_forage}$	Fraction of chicken diet that is incidental vegetation while free ranging (unitless)	See Appendix I 0.05		
P _{forage}	Average concentration of contaminant on free-range vegetation (mg/kg)	Calculated		
$DF_{poultry_feed}$	Fraction of chicken diet that is feed (unitless)	See Appendix I 0.85		
P _{feed}	Average concentration of contaminant in feed (mg/kg)	0		

Source: U.S. EPA (2000).

Table 5-10. TCDD-TEQ Media Concentration for Poultry Thigh Meat Variable Concentrations

Percentile	TCDD-TEQ Concentration in Poultry Thigh Meat (ng/kg)
50 th	0.021
75^{th}	0.036
90 th	0.060
95 th	0.088
99 th	0.18
Background	0.16

Table 5-11. TCDD-TEQ Media Concentration for Eggs Variable Concentrations

Percentile	TCDD-TEQ Concentration in Eggs (ng/kg)
50 th	0.026
75 th	0.046
90 th	0.075
95 th	0.11
99 th	0.32
Background	0.13

All of the factors linked to location for the poultry and egg ingestion pathway influence the soil concentration of dioxin congeners in the residential buffer. The ingestion of soil in the buffer area is the exposure pathway for the free-range chickens raised and eaten by the farm family. The geographic locations linked to the highest 30 estimations of dioxin concentrations in poultry meat and eggs are linked to colder locations with soils with higher soil foc, which binds dioxins to the soil particles. These locations are not confined to a single area of the country, but are more dispersed as indicated by the following locations associated with the top 1 percent of the poultry and egg concentration estimates (listed in order of decreasing frequency of occurrence):

- Burlington, VT (6)
- Salem, OR (6)
- Chicago, IL (5)
- Little Rock, AR (2)
- Portland, ME (2)
- Williamsport, PA (2)
- Muskegon, MI (2)
- Minneapolis, MN (2)
- Atlanta, GA (1)
- Cleveland, OH (1)
- Seattle, WA (1).

5.3.2 Aquatic Food Chain

An aquatic food chain model was used to estimate the concentration of constituent that may accumulate in fish. It is assumed for this analysis that fish is a food source for a recreational fisher. T3 and T4 fish were considered in this analysis. T3 fish are those that consume invertebrates and plankton. T4 fish are those that consume other fish. Most of the fish that humans eat are T4 fish (e.g., salmon, trout, walleye, bass) and medium to large T3 fish (e.g., carp, smelt, perch, catfish, sucker, bullhead, sauger).

The concentration of constituent that accumulates in fish is calculated using the concentration calculated for the sediment in the waterbody adjacent to the buffer. Fish tissue concentrations are dependent on a BSAF. These factors are used to estimate the amount of constituent being transferred from the sediment into the fish tissue. Specifically, they reflect the ratio between the tissue concentration in fish and the appropriate sediment concentration. BSAFs only take into account partitioning from the sediment to the fish and do not consider accumulation through the food chain. The fish concentrations calculated for human receptors are generally lower than whole fish concentrations. Human receptors usually consume only the filet portion of the fish, which has a lower lipid content. Because constituents tend to accumulate in the fatty tissue, the concentration in the filet portion of the fish is lower than the concentration in the sediment only on the variation in the sediment concentration in the sediment only on the variation in the sediment concentration also applies to fish concentration.

	C_{fish}		
$C_{fish} = C_{fish_{lipid}} \times LF$ $C_{fish_{lipid}} = BASF \times C_{sed}$ (5-18)			
Parameter	Definition	Value	
C _{fish}	Concentration in fish (mg/kg)	Calculated	
C _{fish_lipid}	Concentration of contaminant in fish lipid (mg/kg)	Calculated	
LF	Lipid fraction (unitless)	T3, 0.0182 T4, 0.031	
BASF	Biota sediment accumulation factor (unitless)	See Appendix D Congener-specific	
C _{sed}	Concentration in sediment settling to bottom (mg/kg)	Calculated in Table H-2.2	

Source: U.S. EPA (1998).

5.4 Infant Breast Milk Exposure

The concentrations of dioxins in breastmilk are modeled using a steady-state first-order kinetics model obtained from U.S. EPA (1998). This approach allows infant exposures to both lipophilic and nonlipophilic constituents to be modeled based on projected constituent concentrations in maternal breast milk. Lipophilic compounds, such as dioxins, are assumed to accumulate in the lipid fraction of breast milk, and the concentrations in breast milk are equal to concentrations in maternal body fat. Nonlipophilic constituents are assumed to accumulate in the aqueous phase of breast milk and to be proportional to the concentrations in maternal blood plasma. Dioxins are assumed to accumulate exclusively in the lipid phase of breast milk. The equation for estimating the dioxin concentration in milk fat is presented in Equation 5-19.

The concentration of dioxins in maternal milk fat is dependent on the maternal exposure and the biological half-life for the contaminant. A range of 5–7 years was identified for biological half-life (U.S. EPA, 1998); for this analysis, an upper bound of the range (7 years) was used. This assumption will result in a longer time to steady state and in a higher dioxin concentration in maternal fat and, thus, breast milk. This is a protective assumption.

In this risk assessment, the maternal body burdens are assumed to have reached steady state. Reductions in maternal body burden resulting from losses from breast-feeding are not considered. These assumptions may introduce error if the constituent being modeled has a relatively long half-life

C_{milkfat}

$$C_{milkfat} = \frac{ADD_{mat} \times f_{am} \times f_{f}}{(ln2)/t_{1/2}^{b} \times f_{fm}}$$
(5-19)

Parameter	Definition	Value
C _{milkfat}	Concentration in maternal milk fat (mg/kg)	Calculated
ADD _{mat}	Average daily dose consumed by the mother (mg/kg-day)	Calculated
f _{am}	Fraction of ingested contaminant absorbed by the mother (unitless)	1.0
f_{f}	Fraction of contaminant stored in maternal fat (unitless)	0.9
t _{1/2} ^b	Biological half-life of contaminant in lactating women (days) (used to calculate biological elimination constant for the contaminant in nonlactating women)	2,555 days (7 y)
\mathbf{f}_{fm}	Fraction of mother's weight that is fat (unitless)	0.3

Source: U.S. EPA (1998).

and the maternal exposure duration used for the mother prior to the start of lactation is relatively short. In this analysis, maternal body burdens approach steady-state concentrations; thus, the amount of error introduced by not considering losses due to breast-feeding are expected to be small. These losses have been shown to be greatest during the initial stages of maternal exposure, when body burden levels are low and the breast milk loss mechanism is more significant.

5.5 References

- Cowherd, C., G.E. Muleski, P.J. Englehart, and D.A. Gillette. 1985. *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites*. EPA/600/8-85/002. U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Washington, DC. February.
- Dunne, Thomas, and Luna B. Leopold. 1978. *Water in Environmental Planning*. W.H. Freeman and Company, New York.
- Koester, C.J., and R.A. Hites. 1992. Wet and dry deposition of chlorinated dioxins and furans. *Environmental Science and Technology* 26:1375-1382.
- Paustenbach, D.J., R.J. Wenning, V. Lau, N.W. Harrington, D.K. Rennix, and A.H. Parsons.1992. Recent developments on the hazards posed by 2,3,7,8-tetrachlorobenzo-p-dioxin in

soil: implications for setting risk-based cleanup levels at residential and industrial sites. *J. Toxicol. and Environ. Health* 36:103-149..

- McLachlan, M.S., A.P. Sewart, I.R. Bacon, and K.C. Jones. 1996. Persistence of PCDD/Fs in a sludge-amended soil. *Environ. Sci. Technol.* 30(8):2567-2571.
- Shuttleworth, W. James. 1993. Chapter 4: Evaporation. In: *Handbook of Hydrology*, David R. Maidment (ed.). McGraw-Hill, Inc., New York, NY. pp. 4-4.
- USDA (Department of Agriculture). 1971. Chapter 6: Sediment sources, yields, and delivery ratios. In: *National Engineering Handbook, Section 3: Sedimentation*, Soil Conservation Service, Washington, DC. pp. 6-1 to 6-14.
- USDA (Department of Agriculture). 1978. Predicting Rainfall Erosion Losses: A Guide to Conservation Planning. Agriculture Handbook No. 537. Science and Education Administration, Washington, DC.
- USDA (Department of Agriculture). 1986. Urban Hydrology for Small Watersheds. TR-55. U.S. Department of Agriculture, Engineering Division, Soil Conservation Service, Washington, DC. pp. 2-5. June.
- U.S. DOC and U.S. DOE (U.S. Department of Commerce National Climatic Data Center and U.S. Department of Energy National Renewable Energy Laboratory). 1993. Solar and Meteorological Surface Observation Network (SAMSON) 1961-1990. Version 1.0.
- U.S. EPA (Environmental Protection Agency). 1992. Comparison of a Revised Area Source Algorithm for the Industrial Source Complex Short Term Model and Wind Tunnel Data. EPA Publication No. EPA-454/R-92-014. Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1994. 1:250,000 Scale Quadrangles of Landuse/Landcover GIRAS Spatial Data in the Conterminous United States: Metadata. National GIS Program, Office of Information Resources Management, Washington, DC. Website at http://www.epa.gov/ngispgm3/nsdi/projects/giras.htm
- U.S. EPA (Environmental Protection Agency). 1995a. Compilation of Air Pollutant Emission Factors, Volume 1: Stationary Point and Area Sources. 5th Edition. AP-42. PB95-196028INZ. Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1995b. Draft User's Guide for the Industrial Source Complex (ISC3) Dispersion Models. Volume I: User Instructions (Revised). EPA-454/B-95-003a. Emissions, Monitoring, and Analysis Division, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1995c. *PCRAMMET User's Guide* (Draft). Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1995d. User's Guide for the Industrial Source Complex (ISC3) Dispersion Models. Volume II: Description of Model Algorithms.

EPA-454/B-95-003b. Emissions, Monitoring, and Analysis Division, Office of Air Quality Planning and Standards, Research Triangle Park, NC.

- U.S. EPA (Environmental Protection Agency). 1997. The Parameter Guidance Document. A Companion Document to the Methodology for Assessing Health Risks Associated with Multiple Pathways Exposure to Combustion Emissions. (Internal draft) NCEA-2038. National Center for Environmental Assessment, Cincinnati, OH.
- U.S. EPA (Environmental Protection Agency). 1998. Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions. Update to Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions. EPA-600/R-98/137. National Center for Environmental Assessment, Cincinnati, OH.
- U.S. EPA (Environmental Protection Agency). 1999a. Addendum. User's Guide for the Industrial Source Complex (ISC3) Dispersion Models. Volume I: User Instructions for the Revised ISCST3 Model (Dated 99155). Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. EPA (Environmental Protection Agency). 1999b. Source Modules for Nonwastewater Waste Management Units (Land Application Units, Waste Piles, and Landfills): Background and Implementation for the Multimedia, Multipathway, and Multireceptor Risk Assessment (3MRA) for HWIR99. Office of Solid Waste, Washington, DC. October.
- U.S. EPA (Environmental Protection Agency). 1999c. Appendix W to Part 51--Guideline on Air Quality Models. *Code of Federal Regulations* 40 CFR 51.
- U.S. EPA (Environmental Protection Agency). 2000. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. EPA/600/P-00/001Bg. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. September.
- van der Leeden, F., F.L. Troise, and D.K. Todd. 1990. *The Water Encyclopedia*. 2nd edition. Chelsea, Michigan: Lewis Publishers. p. 176.
- Wischmeier, W. H., and D. D. Smith. 1978. Predicting rainfall erosion losses. A guide to conservation planning. In: *Agricultural Handbook*. 537 Edition. U.S. Department of Agriculture, Washington, DC.
- Young, A.L. 1983. Long-term studies on the persistence and movement of TCDD in a natural ecosystem. In: *Human and environmental risks of chlorinated dibenzodioxins and related compounds*. Tucker, R.E., A.L. Young, A.P. Gray. Eds. Plenum Press.

6.0 Human Exposure Assessment

This section describes the human exposure assessment that was conducted for this risk assessment. An exposure assessment is the determination or estimation of the magnitude, frequency, duration, and route of exposure to contaminants that an individual may experience. The term "exposure," as defined by EPA's *Guidelines for Exposure Assessment* (U.S. EPA, 1992), is the condition that occurs when a contaminant comes into contact with the outer boundary of the body. The exposure of an individual to a contaminant is what completes an exposure pathway (i.e., the course a constituent takes from the agricultural land amended with biosolids to an exposed individual). Once the body is exposed, the constituent can cross the outer boundary and enter the body. The amount of contaminant that crosses and is available for adsorption at internal exchange boundaries is referred to as the "dose" (U.S. EPA, 1992). Each exposure pathway, as illustrated in Figure 6-1, includes an exposure point and exposure route.

The biosolids agricultural application risk assessment evaluated the risk to farmers and their families and adult fishers.



Figure 6-1. Human exposure pathways.

Although all fishers and farmers are exposed to dioxins in biosolids, not all individuals experience the same exposure. Different individuals will have a different magnitude, frequency, duration, and route of exposure. Steps were taken in this analysis to capture the variability in individual exposures by taking into account differences in physiological characteristics and daily activity patterns. One step was to vary the values (i.e., exposure factors) used to calculate exposure/intake for fishers, farmers, and their infants and children. Section 6.1 presents an overview of the selected exposure pathways and exposure scenarios considered for this assessment. Section 6.2 presents particular exposure factors (i.e., values needed to calculate human exposure) used in the analysis. Section 6.3 describes the methods used to estimate dose, including average daily dose (ADD) and LADD.

6.1 **Receptors and Exposure Pathways**

Four types of human receptors were assumed to be representative of the individuals who might be exposed to dioxin-like compounds in biosolids: an infant of a farmer, a child of a farmer, an adult farmer, and an adult recreational fisher. These receptors reflect the range of possible individual exposures for direct and indirect exposure pathways. The routes of exposure differ for the farmer and fisher. For example, for this assessment, it is assumed that a farmer consumes produce grown on the farm, as well as animal products (i.e., beef, dairy, poultry, and eggs).

Table 6-1 lists each receptor along with the specific exposure pathways that apply to that receptor. The adult and child farmer are exposed via the inhalation of air and the ingestion of soil, homegrown above- and belowground produce, beef, dairy, poultry, and egg products. The fisher is assumed to be a recreational angler who catches and consumes fish from the nearby waterbody. Infants of farmers are exposed via the ingestion of breast milk only. For very lipophilic constituents that have low volatility, such as dioxins and PCBs, infant exposures from breast milk were assumed to be much greater than exposures through other potential infant pathways, i.e., inhalation or incidental soil ingestion. Therefore, only the breast milk pathway was evaluated in this risk analysis for infants. Infants were considered separately from other childhood exposures.

Receptor	Inhalation of Ambient Air	Ingestion of Soil	Ingestion of Above- and Belowground Produce	Ingestion of Beef and Dairy Products	Ingestion of Poultry and Egg Products	Ingestion of Fish	Ingestion of Breast Milk
Adult farmer	1	1	1	1	1		
Child farmer	1	1	1	1	1		
Infant farmer							1
Adult fisher						1	

Table 6-1. Receptors and Exposure Pathways

6.1.1 Childhood Exposure

Children are an important subpopulation to consider in a risk assessment. They are likely to be more susceptible to exposures, compared with adults, because they may eat more food and drink more fluids per unit of body weight. This higher intake-rate-to-body-weight ratio can result in a higher ADD than adults experience.

As children mature, however, their physical characteristics and behavior patterns change. To capture these changes in the analysis, the life of a child was divided into several age ranges: ages 1 to 5, 6 to 11, 12 to 19, and 20 to 70 (adult). Each age range has distributions of the values, called "exposure parameters," that are required to calculate exposure to an individual. The exposure parameter distributions for each age range reflect the physical characteristics and behavior patterns of that age range. Data from the *Exposure Factors Handbook* (EFH) were used to derive distributions appropriate for each age range (U.S. EPA, 1997a,b,c). The distributions for the 20- to 70-yr-old cohort were used for adult receptors.

Development of the child exposure parameters consisted of three steps:

- Define the start age of the child.
- Select the exposure duration of the child.
- Calculate time-weighted exposure parameters.

To capture the higher intake-rate-to-body-weight ratio of children, a start age between the ages of 1 and 6 was selected for all children. For the probabilistic analysis, a start age between these ages was selected randomly for each iteration.

To select the exposure duration for each of the 3,000 iterations in the analysis, a distribution was chosen to define the exposure duration based on the start age. For example, if the start age was 2, the distribution for cohort 1 (children between ages 1 and 5) was used to define exposure duration. However, if the start age was 6, the distribution developed for cohort 2 (children between ages 6 and 11) was used to define exposure duration.

After the start age and the exposure duration were defined for a given iteration, all the other exposure parameters needed to calculate exposure to a child were developed using the distributions associated with each of the age groups through which the child would age. In this process, an exposure parameter selected from each age group was time-weighted and combined with values from the other age groups to create a single time-weighted exposure parameter. For example, the beef ingestion rates selected from each age group were time-weighted according to the number of years the child remained in the age group and were combined to generate a single time-adjusted beef ingestion rate for the child. The same was done for all the parameters (e.g., body weight, inhalation rate, fruit ingestion rate) required to assess exposure. Equation 6-1 is used to combine each child's exposure parameters into one time-weighted exposure parameter:

$$EP_{TW} = \frac{(EP_1 \times ED_1) + (EP_2 \times ED_2) + (EP_3 \times ED_3) + (EP_4 \times ED_4)}{ED}$$
(6-1)

where

EP _{TW}	=	time-weighted exposure parameter (e.g., ingestion rate of milk, body
		weight)
EP_1	=	exposure parameter for ages 1 to 5
ED_1	=	time spent in age group 1
EP_2	=	exposure parameter for ages 6 to 11
ED_2	=	time spent in age group 2
EP ₃	=	exposure parameter for ages 12 to 19
ED_3	=	time spent in age group 3
EP_4	=	exposure parameter for ages 20 to 70
ED_4	=	time spent in age group 4
ED	=	total exposure duration of the receptor (sum of $ED_1 + ED_2 + ED_3 + ED_4$).

In some cases, the time-weighted exposure parameter methodology resulted in a higher ADD for children than for adults. However, even in those cases where the ADD was higher for children than for adults, the LADD (used for assessing long-term cumulative endpoints, such as cancer) was lower for children than for adults. The reason for this is that total exposure duration is usually shorter for children than for adults, while the same 70-year average lifetime is assumed for averaging the LADD for both children and adults.

6.1.2 Infant Exposure

Infants are an important subpopulation to consider in this risk assessment because they may be exposed to dioxin-like compounds via the ingestion of breast milk. The characterization of risks to infants of farmers and home gardeners was considered separately from the characterization of risks to older children (i.e., aged 1 year or older).

6.1.3 Exposure Pathways

Human receptors may come into contact with dioxins, furans, and PCBs present in environmental media by a variety of pathways. In general, exposure pathways are either direct, such as inhalation of ambient air, or indirect, such as the farm food chain pathways. The exposure pathways considered in this assessment were inhalation of ambient air and ingestion of soil, aboveground produce, belowground produce (i.e., root crops), beef, dairy products, poultry, eggs, fish, and breast milk (infants only).

6.1.3.1 <u>Inhalation of Ambient Air</u>. Both vapors and particles can be inhaled in ambient air by a receptor. Both adults and children (except infants) were affected via direct inhalation.

6.1.3.2 <u>Ingestion of Soil</u>. Both adults and children (except infants) were exposed to soil based on incidental ingestion, mostly due to hand-to-mouth behavior. Soil ingested was modeled as the top 1 cm of soil, untilled for children. The adult farmer was assumed to ingest soil from the tilled crop land.
6.1.3.3 <u>Ingestion of Above- and Belowground Produce</u>. Ingestion of the following categories of produce was used in this risk assessment: exposed fruit, exposed vegetables, and root vegetables. For aboveground produce, the term "exposed" refers to the fact that the edible portion of the plant is exposed to the atmosphere. It was assumed that farmers grow a portion of their fruits and vegetables on land amended with biosolids and that these fruits and vegetables become contaminated via soil and air. Belowground produce refers to root crops grown by the farmer. The soil root crops were grown in was assumed to be tilled, so dioxins, furans, and PCBs were mixed throughout the root zone.

6.1.3.4 <u>Ingestion of Beef and Dairy Products</u>. Beef and dairy cattle were assumed to be exposed to dioxins, furans, and PCBs via differing intake rates of contaminated soil, forage, and feed. Adult and child farmer receptors were assumed to consume beef and drink milk from cattle that grazed in the pasture amended with biosolids.

6.1.3.5 <u>Ingestion of Poultry and Egg Products</u>. Chickens were assumed to be exposed to dioxins, furans, and PCBs via intake rates of contaminated soil while free-range feeding. Adult and child farmer receptors were assumed to consume poultry and eggs from the chicken.

6.1.3.6 <u>Ingestion of Fish</u>. Fish are exposed to dioxins, furans, and PCBs via uptake of contaminants from surface waters. Adult fishers were assumed to consume fish caught in local waterbodies.

6.1.3.7 <u>Ingestion of Breast Milk</u>. Adult women farmers were assumed to be exposed to dioxins, furans, and PCBs via the consumption of contaminated food items and soil and inhalation of contaminated ambient air until they reach a steady-state concentration. Infants of farmer receptors were assumed to consume breast milk from exposed adult receptors for the first year of life.

6.2 Exposure Factors

Table 6-2 lists the exposure factors used in this risk assessment, along with their data sources and whether they were represented by a distribution or a fixed value in the Monte Carlo analysis. Exposure factors are used to calculate the dose of a chemical based on contact with contaminated media or food, the duration of that contact, and the body weight of the exposed individuals. The primary data source of human exposure model inputs used in this risk assessment was EPA's EFH (U.S. EPA, 1997a,b,c). The EFH summarizes data on human behaviors and characteristics related to human exposure from relevant key studies and provides recommendations and associated confidence estimates on the values of exposure factors. EPA carefully reviewed and evaluated the quality of the data before their inclusion in the EFH. EPA's evaluation criteria included peer review, reproducibility, pertinence to the United States, currency, adequacy of the data collection period, validity of the approach, representativeness of the population, characterization of the variability, lack of bias in study design, and measurement error (U.S. EPA, 1997a,b,c).

Parameter	Variable Type	Data Source
Body weight (adult, child, infant)	Distribution	U.S. EPA (1997a)
Inhalation rate (adult, child)	Distribution	U.S. EPA (1997a)
Ingestion rate: soil (adult, child)	Fixed (constant)	U.S. EPA (1997a)
Consumption rate for farmer: exposed vegetables (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: root vegetables (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: exposed fruit (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for recreational fisher: fish (adult)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: beef (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: milk (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: poultry (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: eggs (adult, child)	Distribution	U.S. EPA (1997b)
Consumption rate for farmer: breast milk (infant)	Distribution	U.S. EPA (1997b)
Exposure duration (adult, child)	Distribution	U.S. EPA (1997c)
Exposure frequency (adult, child)	Fixed (constant)	U.S. EPA policy
Fraction contaminated: soil	Fixed (constant)	U.S. EPA policy
Fraction contaminated for recreational fisher: fish	Fixed (constant)	U.S. EPA (1997b)
Fraction homegrown for farmer: exposed vegetables	Fixed (constant)	U.S. EPA (1997b)
Fraction homegrown for farmer: root vegetables	Fixed (constant)	U.S. EPA (1997b)
Fraction homegrown for farmer: exposed fruit	Fixed (constant)	U.S. EPA (1997b)
Fraction contaminated (home-raised) for farmer: beef	Fixed (constant)	U.S. EPA (1997b)
Fraction contaminated (home-raised) for farmer: dairy	Fixed (constant)	U.S. EPA (1997b)
Fraction contaminated (home-raised) for farmer: poultry	Fixed (constant)	U.S. EPA (1997b)
Fraction contaminated (home-raised) for farmer: eggs	Fixed (constant)	U.S. EPA (1997b)
Fraction of T3 fish consumed	Fixed (constant)	U.S. EPA (1997b)
Fraction of T4 fish consumed	Fixed (constant)	U.S. EPA (1997b)
Food preparation and cooking losses: exposed vegetables	Fixed (constant)	U.S. EPA (1997b)
Food preparation and cooking losses: root vegetables	Fixed (constant)	U.S. EPA (1997b)
Food preparation and cooking losses: exposed fruit	Fixed (constant)	U.S. EPA (1997b)
Food preparation and cooking losses: beef	Fixed (constant)	U.S. EPA (1997b)
Food preparation and cooking losses: poultry	Fixed (constant)	U.S. EPA (1997b)
Human lifetime (used in carcinogenic risk calculation)	Fixed (constant)	U.S. EPA policy

Table 6-2. Human Exposure Factor Input Parameters and Data Sources

For probabilistic risk analyses, probability distribution functions were developed from the values in the EFH (U.S. EPA, 1997a,b,c). Appendix K presents the exposure factors used in the probabilistic analysis. Appendix K also describes the rationale and data used to select the parametric models (i.e., gamma, lognormal, and Weibull) for those exposure factors that were varied and the maximum and minimum exposure parameter values used in the analysis.

6.2.1 Intake Factors

This section presents the basis for the intake rates used for soil and food items in the probabilistic analysis. Adult and child receptor intake rates for soil and food items were derived from data in the EFH (U.S. EPA, 1997a,b).

6.2.1.1 <u>Soil Ingestion</u>. Ingestion of contaminated soil is a pathway common to all receptors. Because most available data are from studies measuring soil ingestion in children under the ages of 5 or 6, the adult soil ingestion rate was used for children older than age 5. Thus, soil ingestion rates used in the probabilistic analysis were not varied for any age group. The constant rates used for soil ingestion in this analysis are presented in Table 6-3.

Receptor	Soil Intake Rate (mg/d)
Child	100
Adult	50

Table 6-3. Soil Ingestion Rates Usedin This Risk Analysis

6.2.1.2 <u>Fruit and Vegetable Ingestion</u>. Ingestion of contaminated homegrown fruits and vegetables is a potential pathway of exposure for adult farmers and home gardeners and their children. Consumption rate data of homegrown exposed fruit, exposed vegetables, and root vegetables by these receptors were obtained from the EFH. Examples of exposed fruits are apples, peaches, pears, and berries. Aboveground exposed vegetables include tomatoes, green leafy vegetables (e.g., lettuce, cabbage, kale), cucumber, summer squash, peppers, broccoli, okra, and snap beans. Common root vegetables include carrots, onions, potatoes, and beets (U.S. EPA, 1997b).

Because farmers grow much but generally not all of their food, the fraction of the farmers' diets that may be contaminated was considered. Specifically, the EFH provides recommendations on the percent of the total diet of farmers that is homegrown. In addition, produce consumption rate data were adjusted to account for food preparation and cooking losses.

Table 6-4 presents exposed fruit consumption data used in the Monte Carlo analysis. Data for consumption of homegrown exposed fruit were obtained from Table 13-61 of the EFH (U.S. EPA, 1997b). Data (in g WW/kg-d) were presented by age groups and for farmers and home gardeners (adults). For the 1- to 5-yr-old age group, data were only available for those aged 3 to 5 years; therefore, these data were used for the entire 1- to 5-yr-old age group. Percentile data were used to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select the most appropriate model. The fraction of exposed fruit intake that is home-produced is 0.328 for households that farm and 0.116 for households that garden (Table 13-71, U.S. EPA, 1997b). Figure 6-2 presents these distributions graphically. The distributions were truncated at the maximum value shown in the table and graph.

Table 6-4.	Exposed Fruit	Consumption	Data and	Distributions
------------	----------------------	--------------------	----------	---------------

					Distril	Distributions										
Age Cohort	N	Data Mean	Data SDev	P01	P05	P10	P25	P50	P75	P90	P95	P99	Distribution	Pop- Estd Mean	Pop- Estd SDev	Max
1-5	49	2.6	3.947			0.373	1	1.82	2.64	5.41	6.07		Gamma	2.25	1.89	16
6-11	68	2.52	3.496		0.171	0.373	0.619	1.11	2.91	6.98	11.7		Lognormal	2.78	5.12	36
12-19	50	1.33	1.457		0.123	0.258	0.404	0.609	2.27	3.41	4.78		Lognormal	1.54	2.44	18
Adult Farmer	112	2.32	2.646	0.072	0.276	0.371	0.681	1.3	3.14	5	6.12	15.7	Lognormal	2.36	3.33	31

N = Number of samples; P01-P99 = Percentiles; Pop-Estd = Population-estimated; SDev = Standard deviation; Minimum is assumed = 0



Figure 6-2. Distribution of exposed fruit consumption rates by age group.

Exposed Vegetable Consumption. Table 6-5 presents exposed vegetable consumption data and distribution. Data for consumption of homegrown exposed vegetables were obtained from Table 13-63 of the EFH (U.S. EPA, 1997b). Data (in g WW/kg/d) were presented for those aged 1 to 2, 3 to 5, 6 to 11, 12 to 19, 20 to 39, and 40 to 69 years, as well as farmers and home gardeners. Weighted averages of percentiles, means, and standard deviations were calculated for the 1- to 5-yr-old age group (combining groups of those aged 1 to 2 years and 3 to 5 years). Percentile data were used to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select the most appropriate model. The fraction of exposed vegetable intake that is home-produced is 0.42 for households that farm and 0.233 for households that garden (Table 13-71, U.S. EPA, 1997b). Figure 6-3 presents these distributions graphically. The distributions were truncated at the maximum value shown in the table and graph.

						EFH Da		Di	Distributions							
Age Cohort	N	Data Mean	Data SDev	P01	P05	P10	P25	P50	P75	P90	P95	P99	Distribution	Pop- Estd Mean	Pop- Estd SDev	MAX
1-5	105	2.453	2.675		0.102	0.37	0.833	1.459	3.226	6.431	8.587		Gamma	2.55	2.58	21
6-11	134	1.39	2.037		0.044	0.094	0.312	0.643	1.6	3.22	5.47	13.3	Lognormal	1.64	3.95	27
12-19	143	1.07	1.128		0.029	0.142	0.304	0.656	1.46	2.35	3.78	5.67	Gamma	1.08	1.13	11
Adult farmer	207	2.17	2.316		0.184	0.372	0.647	1.38	2.81	6.01	6.83	10.3	Lognormal	2.38	3.5	26

 Table 6-5. Exposed Vegetable Consumption Data and Distributions

N = Number of samples; P01-P99 = Percentiles; Pop-Estd = Population-estimated; SDev = Standard deviation; Minimum is assumed = 0



Figure 6-3. Distribution of exposed vegetable consumption rates by age group.

Root Vegetable Consumption. Table 6-6 presents root vegetable consumption rates and distributions. Homegrown root vegetable consumption data were obtained from Table 13-65 of the EFH (U.S. EPA, 1997b). Data (in g WW/kg/d) were presented for those aged 1 to 2, 3 to 5, 6 to 11, 12 to 19, 20 to 39, 40 to 69 years, and adult farmers and home gardeners. Weighted averages of percentiles, means, and standard deviations were calculated for the 1- to 5-yr-old age group (combining groups of those aged 1 to 2 and 3 to 5 years). Percentile data were used to fit

parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select the most appropriate model. The fraction of root vegetable intake that is home-produced is 0.173 for households that farm and 0.106 for households that garden (Table 13-71, U.S. EPA, 1997b). Figure 6-4 presents these distributions graphically. The distributions were truncated at the maximum value shown in the table and graph.

6.2.1.3 <u>Beef and Dairy Ingestion</u>. The farmer (adult and child) is assumed to ingest beef and dairy products from cattle raised on pastures amended with biosolids. As with fruits and vegetables, it was necessary to consider the fraction of the total beef and dairy in the farmer's diet that consists of products raised on the amended pasture. In addition, beef consumption rate data were adjusted to account for food preparation and cooking losses.

						EFH Da		Distributions								
Age Cohort	N	Data Mean	Data SDev	P01	P05	P10	P25	P50	P75	P90	P95	P99	Distribution	Pop- Estd Mean	Pop- Estd SDev	MAX
1-5	45	1.886	2.371		0.081	0.167	0.291	0.686	2.653	5.722	7.502		Lognormal	2.31	6.05	41
6-11	67	1.32	1.752		0.014	0.036	0.232	0.523	1.63	3.83	5.59		Weibull	1.38	2.07	15
12-19	76	0.937	1.037		0.008	0.068	0.269	0.565	1.37	2.26	3.32		Weibull	0.99	1.19	9
Adult farmer	136	1.39	1.469	0.111	0.158	0.184	0.365	0.883	1.85	3.11	4.58	7.47	Lognormal	1.45	2.06	15

Table 6-6. Root Vegetable Consumption Data and Distributions

N = Number of samples; P01-P99 = Percentiles; Pop-Estd = Population-estimated; SDev = Standard deviation; Minimum is assumed = 0



Figure 6-4. Distribution of root vegetable consumption rates by age group.

Beef Consumption. Table 6-7 presents beef consumption data and distributions. Home-produced beef consumption data were obtained from Table 13-36 of the EFH (U.S. EPA, 1997b). Data (in g WW/kg-d) were presented for farmers and those aged 6 to 11, 12 to 19, 20 to 39, and 40 to 69. Percentile data were used to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select the most appropriate model.

Data were not available for those aged 1 to 2 and 3 to 5. For beef consumption for 1- to 5-yr-olds, the lognormal model was used because, among the other age groups, it was the best-fitted model in all but one case. The population-estimated mean and standard deviation for 6- to 11-yr-olds were used for 1- to 5-yr-olds for the analysis (normalized for body weight) and are supported by data in Table 11-3 of the EFH (per capita intake for beef, including store-bought products), which indicate that those aged 1 to 2, 3 to 5, and 6 to 11 have the highest consumption rate of beef on a gram/kilogram/ day basis. Figure 6-5 presents these data graphically. The distribution of beef consumption rates was truncated at the maximum value indicated in the table and graph. The fraction of beef intake that is home-produced is 0.485 for households that farm (Table 13-71, U.S. EPA, 1997b).

					EFH	Data (g WW/ł		Distributions							
Age Cohort	N	Data Mean	Data SDev	P01	P05	P10	P25	Р50	P75	P90	P95	P99	Distribu- tion	Pop- Estd Mean	Pop- Estd SDev	MAX
1-5		ND	ND										Lognormal	3.88	4.71	36
6-11	38	3.77	3.662		0.663	0.753	1.32	2.11	4.43	11.4	12.5		Lognormal	3.88	4.71	36
12-19	41	1.72	1.044		0.478	0.513	0.896	1.51	2.44	3.53	3.57		Gamma	1.77	1.12	10
Adult farmer	182	2.63	2.644	0.27	0.394	0.585	0.896	1.64	3.25	5.39	7.51	11.3	Lognormal	2.5	2.69	23

Table 6-7. Beef Consumption Data and Distributions

N = Number of samples; P01-P99 = Percentiles; Pop-Estd = Population-estimated; SDev = Standard deviation; Minimum is assumed = 0





Beef consumption rate data were adjusted to account for food preparation and cooking losses. A mean net cooking loss of 27 percent accounts for dripping and volatile losses during cooking (averaged over various cuts and preparation methods). A mean net postcooking loss of 24 percent accounts for losses from cutting, shrinkage, excess fat, bones, scraps, and juices. These data were obtained from Table 13-5 of the EFH (U.S. EPA, 1997b).

Dairy Products (Milk) Consumption. Table 6-8 presents summary statistics on consumption of dairy products. Home-produced dairy product consumption rate data were obtained from Table 13-28 of the EFH (U.S. EPA, 1997b) for farmers, all ages combined, and individual age groups. No age-specific data for children were available for home-produced dairy products consumption. Per capita intake data for dairy products (including store-bought products), however, were available from the EFH and from CSFII (USDA, 1997) for those aged 1 to 2, 3 to 5, 6 to 11, and 12 to 19; the data in the EFH were based on the 1989–1991 CSFII, so the more recent 1994–1996 CSFII raw data were used. Therefore, data for the general population were used to calculate adjustment factors to develop distributions for the nonadult age groups for consumption of home-produced dairy products. Figure 6-6 presents these distributions graphically. The distributions were truncated at the maximum value as shown in the table and graph.

					Data (g	WW/kg	g-d)		Distributions					
Source	Age Cohort	Data Mean	Data SDev	P05	P10	P25	P50	P75	P90	P95	Distribution	Pop- Estd Shape	Pop- Estd Scale	Max
CSFII (gen)	All	6.81	10.8	0.199	0.392	1.14	3.25	7.59	16.9	26.1				
CSFII (gen)	1-5	27.4	22.3	1.12	4.39	12.2	22.3	37.1	55.9	70.1				
CSFII (gen)	6-11	14	10	0.826	2.16	6.48	12.3	19.2	27.3	33.5				
CSFII (gen)	12-19	6.2	5.87	0.264	0.484	1.88	4.55	8.88	13.5	17.8				
CSFII (gen)	20-69	3.23	3.3	0.162	0.303	0.854	2.22	4.48	7.45	9.88				
HP	1-5										Gamma	0.961	61.80	482
HP	6-11										Gamma	0.961	31.40	245
HP	12-19										Gamma	0.961	13.90	109
EFH (HP)	20_39	7.41	6.12	0.396	0.446	1.89	6.46	12.1	15.4	19.5	Gamma	0.961	8.01	
EFH (HP)	All	14	15.28	0.446	0.508	3.18	10.2	19.5	34.2	44	Gamma	0.78	18.26	
EFH (HP)	Adult farmer	17.1	15.8	0.736	3.18	9.06	12.1	20.4	34.9	44	Gamma	1.38	11.85	116

Table 6-8. Dairy Products (Milk) Consumption Data and Distributions

CSFII = CSFII (USDA, 1997); gen = general population data; EFH = U.S. EPA (1997b); HP = home-produced data; P05-P95 = Percentiles; Sdev = standard deviation; Pop-Estd = population-estimated; Minimum is assumed = 0



Figure 6-6. Distribution of milk consumption rates by age group.

Percentile data (USDA, 1997) were used to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select gamma as the most appropriate model in all cases. Tables J-19 and J-20 (Appendix J) provide the data used to develop the distributions and adjustment factors. It was assumed that the relative standard deviations (RSD) for consumption rates were the same for all age groups; the similarity of coefficients of variation (CV) suggests that this is a reasonable approximation for the general population. The other assumption used to develop distributions for the child age groups for the consumption of home-produced dairy products was that the mean intake rates have the same fixed ratio for all the age groups of a given food type. That is, the ratio of the mean amount consumed of home-produced dairy products divided by the mean amount of dairy products consumed in the general population is the same for any two age groups. These two assumptions, of constant RSD and constant mean ratio, were used to infer the parameters of the gamma distributions for the home-produced foods from those of the general population (i.e., mean, standard deviation, shape, and scale).

The fraction of dairy product intake that is home-produced is 0.254 for households that farm (Table 13-71, U.S. EPA, 1997b).

6.2.1.4 <u>Poultry and Egg Ingestion</u>. The farmer (adult and child) is assumed to ingest poultry and egg products from chickens raised on the farm using biosolids as a soil amendment. As with fruits and vegetables, it was necessary to consider the fraction of the total poultry and eggs in the farmer's diet that consists of products raised on the farm. In addition, poultry consumption rate data were adjusted to account for food preparation and cooking losses.

Poultry Consumption. Table 6-9 presents summary statistics on consumption of poultry. Home-produced poultry consumption rate data were obtained from Table 13-55 of the EFH (U.S. EPA, 1997b) for farmers, all ages combined, and individual age groups 20 to 39 and 40 to 69; statistics for the 20- to 69-yr-old age group were calculated as simple averages of the statistics for the 20- to 39- and 40- to 69-yr-old age groups. No age-specific data for children were available for home-produced poultry consumption. Per capita intake data for poultry (including store-bought products), however, were available for those aged 1 to 2, 3 to 5, 6 to 11, and 12 to 19 years old from the EFH and from CSFII (USDA, 1997); the data in the EFH were based on the 1989-1991 CSFII, so the more recent 1994–1996 CSFII raw data were used. Therefore, data for the general population were used to calculate adjustment factors to develop distributions for the nonadult age groups for consumption of home-produced poultry. Figure 6-7 presents these distributions graphically. The distributions for poultry consumption were trancated at the maximum value indicated in the table and graph.

			Data (g WW/kg-d)								Di	stributio	ons	
Source	Age Cohort	Data Mean	Data SDev	P05	P10	P25	P50	P75	P90	P95	Distribution	Pop- Estd Shape	Pop- Estd Scale	MAX
CSFII (gen)	All	0.688	0.942	0.018	0.034	0.111	0.334	0.917	1.76	2.47				
CSFII (gen)	1-5	1.43	1.73	0.025	0.056	0.192	0.736	2.2	3.63	4.66				
CSFII (gen)	6-11	0.884	1.15	0.019	0.036	0.116	0.365	1.29	2.42	3.22				
CSFII (gen)	12-19	0.645	0.795	0.019	0.034	0.103	0.346	0.896	1.71	2.23				
CSFII (gen)	20-69	0.57	0.712	0.017	0.032	0.105	0.303	0.804	1.4	1.92				
HP	1-5										Gamma	1.69	1.92	21
HP	6-11										Gamma	1.69	1.21	14
HP	12-19										Gamma	1.69	0.87	10
EFH (HP)	20-69	1.34	1.088	0.299	0.352	0.524	0.962	2.03	2.545	3.765	Gamma	1.69	0.80	
EFH (HP)	All	1.57	1.178	0.303	0.418	0.637	1.23	2.19	3.17	3.83	Gamma	1.83	0.85	
EFH (HP)	Adult farmer	1.54	1.375	0.228	0.303	0.595	1.06	2.18	3.47	4.83	Gamma	1.38	1.16	11

 Table 6-9. Poultry Consumption Data and Distributions

CSFII = (USDA, 1997); gen = general population data; EFH = U.S. EPA (1997b); HP = home-produced data; P05-P95 = Percentiles; Sdev = standard deviation; Pop-Estd = population-estimated; Minimum is assumed = 0



Figure 6-7. Distribution of poultry consumption rates by age group.

Percentile data (USDA, 1997) were used to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select gamma as the most appropriate model in all cases. Tables J-19 and J-20 (see Appendix J) provide the data used to develop the distributions and adjustment factors. Constant RSD and constant mean ratio were assumed, and these data were used to infer the parameters of the gamma distributions for the home-produced foods from those of the general population (i.e., mean, standard deviation, shape, and scale). The fraction of poultry intake that is home-produced is 0.156 for households that farm (Table 13-71, U.S. EPA, 1997b).

Egg Consumption. Table 6-10 presents summary statistics on consumption of eggs. Home-produced egg consumption rate data were obtained from Table 13-43 of the EFH (U.S. EPA, 1997b) for farmers, all ages combined, and individual age groups 20 - 39 and 40 - 69; statistics for the 20- to 69-yr-old age group were calculated as simple averages of the statistics for the 20- to 39- and 40- to 69-yr-old age groups. No age-specific data for children were available for home-produced egg consumption. Per capita intake data for eggs (including store-bought products), however, were available from the EFH and from CSFII (USDA, 1997) for those aged 1 to 2, 3 to 5, 6 to 11, and 12 to 19; the data in the EFH were based on the 1989–1991 CSFII, so the more recent 1994–1996 CSFII raw data were used. Therefore, data for the general population were used to calculate adjustment factors to develop distributions for the nonadult age groups for consumption of home-produced eggs. Figure 6-8 presents these distributions graphically. The distribution of egg consumption rates was truncated at the maximum value shown in the table and graph.

					Data	(g WW/k		Distributions						
Source	Age Cohort	Data Mean	Data SDev	P05	P10	P25	P50	P75	P90	P95	Distribution	Pop- Estd Shape	Pop- Estd Scale	MAX
CSFII (gen)	All	1.01	1.04	0.133	0.253	0.422	0.724	1.22	1.99	2.82				
CSFII (gen)	1-5	2.41	1.94	0.101	0.328	1.16	1.88	3.23	5.03	6.15				
CSFII (gen)	6-11	1.44	1.25	0.125	0.302	0.641	1.08	1.87	2.95	3.45				
CSFII (gen)	12-19	0.962	0.708	0.092	0.328	0.469	0.821	1.22	1.71	2.24				
CSFII (gen)	20-69	0.792	0.663	0.145	0.248	0.389	0.633	1.01	1.52	1.88				
HP	1-5										Gamma	1.88	0.839	10
HP	6-11										Gamma	1.88	0.493	6
HP	12-19										Gamma	1.88	0.334	4
EFH (HP)	20-69	0.611	0.442	0.106	0.183	0.308	0.465	0.829	1.31	1.645	Gamma	1.88	0.336	
EFH (HP)	All	0.731	1.114	0.15	0.175	0.268	0.466	0.902	1.36	1.69	Gamma	1.81	0.357	
EFH (HP)	Adult farmer	0.898	1.128	0.165	0.177	0.272	0.666	1.19	1.65	1.85	Gamma	1.64	0.488	13

Table 6-10. Egg Consumption Data and Distributions

CSFII = CSFII (USDA, 1997); gen = general population data; EFH = U.S. EPA (1997b); HP = home-produced data; Sdev = standard deviation; Pop-Estd = population-estimated; Minimum is assumed = 0





Percentile data (USDA, 1997) were used to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select gamma as the most appropriate model in all cases. Tables J-19 and J-20 (see Appendix J) provide the data used to develop the distributions and adjustment factors. It was assumed that the relative standard deviations for consumption rates were the same for all age groups; the similarity of coefficients of variation suggests that this is a reasonable approximation for the general population. The other assumption used to develop distributions for the child age groups for the consumption of home-produced eggs was that the mean intake rates have the same fixed ratio for all the age groups of a given food type. That is, the ratio of the mean amount consumed of home-produced eggs divided by the mean amount of eggs consumed in the general population is the same for any two age groups. These two assumptions, of constant RSD and constant mean ratio, were used to infer the parameters of the gamma distributions for the home-produced foods from those of the general population (i.e., mean, standard deviation, shape, and scale).

The fraction of egg intake that is home-produced is 0.146 for households that farm (Table 13-71, U.S. EPA, 1997b).

6.2.1.5 <u>Fish Ingestion</u>. Fish ingestion rates were based on an adult recreational angler who catches and eats some fish from a stream affected by contaminants released from biosolids. All fish are assumed to be home-caught and contaminated for households that fish.

Fish Consumption. Table 6-11 presents fish consumption data and distribution. Fish consumption data were obtained from Table 10-64 of the EFH (U.S. EPA, 1997b). Data (in g/d) were available for adult freshwater anglers in Maine. The Maine fish consumption study was one of four recommended freshwater angler studies in the EFH (U.S. EPA, 1997b). The other recommended fish consumption studies (i.e., Michigan and New York) had large percentages of anglers who fished from the Great Lakes, which is not consistent with the modeling scenarios used in this risk analysis. The anglers in the Maine study fished from streams, rivers, and ponds; these data are more consistent with modeling scenarios for this risk analysis. Although the Maine data have a lower mean than the Michigan data, the Maine data compared better with a national USDA study. Also, the Maine study had percentile data available, which were necessary to develop a distribution. Figure 6-9 presents fish consumption rate distribution for adults. The distribution of fish consumption rates was truncated at the maximum value shown in the table and graph.

Table 6-11. Fish Const	umption Data and Distribution	ns
------------------------	-------------------------------	----

				EFH I	Data (g/	'd)			Distributions					
Age Cohort	N	Data Mean	Data SDev	P50	P66	P75	P90	P95	Distribution	Pop-Estd Mean	Pop-Estd SDev	MAX		
Adult	1,053	6.4		2	4	5.8	13	26	Lognormal	6.48	19.9	1500		

 $N = Number \ of \ samples; \ P50-P95 = Percentiles; \ Pop-Estd = Population-estimated; \ SDev = Standard \ deviation.$



Figure 6-9. Distribution of adult fish consumption rates.

Percentile data were used to fit parametric models (gamma, lognormal, and Weibull) and measures of goodness of fit were used to select lognormal as the most appropriate model. The fraction of fish intake that is locally caught is 0.325 for adult fishers (Table 13-71, U.S. EPA, 1997b). The fractions of consumed T3 and T4 fish were 0.36 and 0.64, respectively (Table 10-66, U.S. EPA, 1997b).

6.2.1.6 <u>Breast Milk Ingestion</u>. Ingestion of contaminated breast milk is a potential pathway of exposure for infants of farmers. Consumption rate data were obtained from the EFH.

Breast Milk Consumption. Table 6-12 presents breast milk consumption data for infants. The data mean and upper percentile for breast milk consumption in 1- to 12-month-olds were 688 and 980 mL/d, respectively (Table 14-16, U.S. EPA, 1997b). The triangular model was used for breast milk consumption (12-month-olds) because no percentile or related data were available; other distributions (e.g., lognormal) resulted in overestimation of the upper percentile. Figure 6-10 presents this distribution graphically. The EFH population mean for breast milk consumption was 688 mL/d and was assumed to equal the mode. The distribution of breast milk consumption rates was truncated at the maximum value shown in the table and graph.

 Table 6-12. Breast Milk Consumption Data and Distribution

Age Cohort	Data Mean (mL/d)	Data SDev	Upper Percentile	Distribution	Pop-Estd Mode (mL/d)	Pop-Estd SDev (mL/d)	Max
<1	688	ND	980	Triangular	688	688	1380

Pop-Estd = population-estimated; SDev = Standard deviation; ND = No data.



Figure 6-10. Distribution of breast milk consumption rates.

6.2.1.7 <u>Inhalation Rates</u>. The EFH reports inhalation values by age, gender, activity pattern, and outdoor workers; however, it does not provide high-end values in most cases. The inhalation rate is the same for all adults, whether farmer, or fisher, while child receptors use a single child inhalation rate.

Inhalation Rate. Table 6-13 presents inhalation rate data and distribution. No percentile data were available for the inhalation rate, and the default lognormal model was assumed. In an analysis of inhalation data, Myers et al. (U.S. EPA, 2000) found that, for those younger than 3 years, CV was close to 70 percent; for other age groups, it was close to 30 percent. The lognormal distribution was fitted by using CV = 50 percent [(30+70)/2] for the 1- to 5-yr-old age group and CV = 30 percent for the 6- to 11-yr-olds, 12- to 19-yr-olds, and adult age groups. Figure 6-11 presents this distribution graphically. The distribution of inhalation rates was truncated at the minimum and maximum values shown in the table and graph.

Age Cohort	Distribution	Population- Estimated Mean (m³/d)	Population- Estimated SDev (m ³ /d)	Min	Max
1-5	Lognormal	7.55	3.78	1	40
6-11	Lognormal	11.75	3.53	1	45
12-19	Lognormal	14.0	4.2	1	55
Adult	Lognormal	13.3	3.99	1	50

Table 6-13. Inhalation Rate Data and Distribution

SDev = Standard deviation.



Figure 6-11. Distribution of inhalation rates by age group.

6.2.2 Other Exposure Factors

6.2.2.1 <u>Body Weights</u>. Distributions of body weight were developed for adult (farmer and fisher), child (farmer), and infant (farmer) receptors based on data from the EFH.

Table 6-14 presents body weight data and distribution. Body weight data were obtained from Tables 7-2 through 7-7 of the EFH (U.S. EPA, 1997a). Data (in kg) were presented by age and gender. Weighted averages of percentiles, means, and standard deviations were calculated for infants (<1 year old), 1- to 5-yr-olds, 6- to 11-yr-olds, 12- to 19-year olds, and adult age groups; male and female data were weighted and combined for each age group. These percentile data were used as the basis for fitting distributions. These data were analyzed to fit parametric models (gamma, lognormal, and Weibull) using maximum likelihood estimation. Measures of goodness of fit were used to select the most appropriate model. Figure 6-12 presents these distributions graphically. The body weight distributions are truncated at the maximum values shown in the table and graph.

			EFH Data (kg)							Distributions							
Age Cohort	N	Data Mean	Data SDev	P05	P10	P15	P25	P50	P75	P85	P90	P95	Distribution	Pop- Estd Mean	Pop- Estd SDev	Min	Max
<1	356	9.102	1.287	7.053	7.451	7.852	8.252	9.151	9.752	10.4	10.65	11.15	Gamma	9.09	1.23	2	26
1-5	3,762	15.52	3.719	12.5	13.1	13.45	14.03	15.26	16.67	17.58	18.32	19.45	Lognormal	15.5	2.05	4	50
6-11	1,725	30.84	9.561	22.79	24.05	25.07	26.44	29.58	33.44	36.82	39.66	43.5	Lognormal	30.7	5.96	6	200
12-19	2,615	58.45	13.64	43.84	46.52	48.31	50.94	56.77	63.57	68.09	71.98	79.52	Lognormal	58.2	10.2	13	300
20+	12,504	71.41	15.45	52.86	55.98	58.21	61.69	69.26	78.49	84.92	89.75	97.64	Lognormal	71.2	13.3	15	300

Table 6-14. Body Weight Data and Distributions

 $N = Number \ of \ samples; \ P05-P95 = Percentiles; \ Pop-Estd = Population-estimated; \ SDev = Standard \ deviation.$



Figure 6-12. Distribution of body weights by age group.

6.2.2.2 <u>Exposure Duration</u>. Exposure duration refers to the amount of time that a receptor is exposed to a contaminant source. For this risk analysis, exposure duration was assumed to correspond to the receptor's residence time in the same house. Exposure durations were determined using data on residential occupancy from the EFH (U.S. EPA, 1997c). Separate distributions were developed for both adult and child adult farmers. Children of farmers were assumed to have the same exposure duration as rural resident children because no age-specific data were available for residential occupancy for farmers.

Exposure duration for all adult and child receptors was capped at a total lifetime of 100 years.

Table 6-15 presents exposure duration data and distributions. Exposure duration was assumed to be equivalent to the average residence time for each receptor. Exposure durations for adult residents and children (resident and farmer) were determined using data on residential occupancy from the EFH, Table 15-168 (U.S. EPA, 1997c). The data represent the total time a person is expected to live at a single location, based on age. The table presented male and female data combined. For adult residents, age groups from 21-yr-olds to 90-yr-olds were pooled. For children, the 3-yr-old age group was used for the 1- to 5-yr-olds. Figure 6-13 represents these distributions graphically.

Table 6-15.	Exposure	Duration	Data	and	Distributions
-------------	----------	----------	------	-----	---------------

EFH Data	ı	Distributions						
Age Cohort	Data Mean (yr)	Distribution	Pop-Estd Shape (yr) ^a	Pop-Estd Scale (yr)	Min	Max		
Child (1- to 5-yr-olds)	6.5	Weibull	1.32	7.059	1	100		
Adult farmer	18.75	Gamma	0.607	29.76	1	100		

Pop-Estd = Population-estimated.

^a Distributions used in risk assessment.





In an analysis of residential occupancy data, Myers et al. (U.S. EPA, 2000) found that the data, for most ages were best fit by a Weibull distribution. The Weibull distribution as implemented in Crystal Ball[®] is characterized by three parameters: location, shape, and scale. Location is the minimum value and, in this case, was presumed to be 0. Shape and scale were determined by fitting a Weibull distribution to the pooled data, as follows: to pool residential occupancy data for the age cohorts, an arithmetic mean of data means was calculated for each age group. Then, assuming a Weibull distribution, the variance within each age group (e.g., 6-yr-olds) was calculated in the age cohort. These variances in turn were pooled over the age cohort using equal weights. This is not the usual type of pooled variance, which would exclude the variation in the group means. However, this way the overall variance reflected the variance of means within the age groups (e.g., within the 6-yr-old age group). The standard deviation was estimated as the square root of the variance. The coefficient of variation was calculated as the ratio of the standard deviation divided by the Weibull mean. For each cohort, the population-estimated parameter uncertainty information (e.g., shape and scale) was calculated based on a Weibull distribution, the calculated data mean for the age cohort, and the CV.

Exposure duration for adult farmers was determined using data on residential occupancy from the EFH, Tables 15-163 and 15-164 (U.S. EPA, 1997c). The data represent the total time a person is expected to live at a single location, based on household type. Age-specific data were not provided. For residence duration of farmers (U.S. EPA 1997c, Tables 15-163 and 15-164), the gamma model was used because it was the best-fitted model in five age groups and was the second-best-fitted model in two cases (based on data in U.S. EPA 1997c, Tables 15-167 and 15-168). A population mean of 18.07 years and a population standard deviation of 23.19 years were calculated for adult farmers.

6.2.2.3 <u>Exposure Frequency</u>. Exposure frequency is the frequency at which the receptor is exposed to the contaminated source during the exposure duration. Exposure frequency is not expected to vary much, so distributions were not developed. All receptors were assumed to be exposed to the contaminant source 350 d/yr. This value is based on an assumption that individuals are away from their homes (e.g., on vacation) approximately 2 weeks out of the year.

6.2.2.4 <u>Lifetime and Averaging Time</u>. Averaging time is the period of time over which a receptor's dose is averaged. When evaluating carcinogens, total dose is averaged over the lifetime of the individual, assumed to be 70 years for exposure durations of equal to or less than 50 years. For exposures greater than a lifetime of 70 years, the lifetime averaging time was assumed to be the lifetime of the individual evaluated in the risk assessment. For example, if an adult is assumed to have an exposure duration of 70 years (adult exposure period starts at age 20), that person is assumed to have a total lifetime (averaging time) of 90 years.

6.3 Dose Estimates

The purpose of the exposure assessment is to estimate the dose to each receptor by combining intake values with media concentrations. Estimates of exposure are based on the potential dose (e.g., the dose ingested or inhaled) rather than the applied dose (e.g., the dose delivered to the gastrointestinal tract) or the internal dose (e.g., the dose delivered to the target

organ). This is generally consistent with the exposure metric used in most epidemiologic and toxicologic studies that serve as the basis for establishing the toxicological benchmarks used for risk assessment (see Section 9.2).

Doses from individual pathways (e.g., soil, exposed vegetables) were calculated by multiplying the contaminant concentration with the respective intake rate on a per kilogram body weight basis. Doses received from the various ingestion pathways (e.g., soil, food) were then summed over the period of time in which exposure occurred, resulting in an ADD received from ingestion exposure. The ADD was used for the calculation of maternal body burden. For cancer effects, where the biological response is described in terms of lifetime probabilities, even though exposure may not occur over the entire lifetime, dose is presented as an LADD. The LADD was used to assess cancer risks from each exposure route (i.e., inhalation and ingestion).

6.3.1 Average Daily Dose

For the purposes of this risk analysis, ADD was defined as

$$ADD = C \times IR \tag{6-2}$$

where

ADD	=	average daily dose (mass constituent/body weight mass/time)
С	=	concentration (mass/volume or mass/mass)
IR	=	intake rate (mass/body weight mass/time or volume/body weight mass/time).

Contaminant concentration represents the concentration of a chemical in a medium that contacts the body. Intake rate for the respective ingestion pathway was applied. For several food parameters, intake rates were provided in milligram per kilogram body weight per day. However, intake rates for fish and soil were adjusted by body weight in order to be on a milligram per kilogram body weight per day basis.

Pathway-specific ADDs, designated as ADD_is , were calculated for individual ingestion pathways (e.g., soil, exposed vegetables). The summation of the ADD_is results in an ADD for the ingestion pathway (ADD_{ingest}) , which was used to calculate maternal body burdens and assess risk to infants of farmers and home gardeners resulting from the ingestion of breast milk.

6.3.2 Lifetime Average Daily Dose

The LADD, used for assessing risks for carcinogenic effects, was defined as

$$LADD = \frac{C \times IR \times ED \times EF}{AT \times 365}$$
(6-3)

where

LADD	=	lifetime average daily dose (mass constituent/body weight mass/time)
С	=	average concentration (mass/mass or mass/volume)
IR	=	intake rate (mass/body weight mass/time or volume/body weight mass/time)
ED	=	exposure duration (yr)
EF	=	exposure frequency (d/yr)
AT	=	averaging time (yr)
365	=	unit conversion factor (d/yr).

The contaminant concentration represents the concentration of a chemical in a medium that contacts the body. Intake rate depends on the route of exposure; for example, it might be an inhalation rate or an ingestion rate. Exposure frequency is the number of days per year the receptor is exposed to the contaminated source during the exposure duration.

For cancer effects, biological responses are described in terms of lifetime probabilities, even though exposure may not be lifelong. Here, the exposure duration (the length of time of contact with a contaminant) was used to average the ADD over a lifetime (70 years or more). The media concentrations used in the analysis for assessing the LADD (e.g., soil concentration) were generally averaged explicitly over the duration of exposure. This provides a more exact estimate of the LADD. An $LADD_{ingest}$ was calculated for ingestion exposures and an $LADD_{inh}$ was calculated for inhalation exposures.

6.4 References

- USDA (U.S. Department of Agriculture). 1997. 1994-96 Continuing Survey of Food Intakes by Individuals, CD-ROM. U.S. Department of Agriculture, Agricultural Research Service, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1992. Guidelines for exposure assessment. Final guidelines. *Federal Register* 57 FR 22888-22893. Washington, DC. May 29.
- U.S. EPA (Environmental Protection Agency). 1997a. *Exposure Factors Handbook, Volume I, General Factors*. EPA/600/P-95/002Fa. Office of Research and Development, Washington, DC. August.
- U.S. EPA (Environmental Protection Agency). 1997b. *Exposure Factors Handbook, Volume II, Food Ingestion Factors*. EPA/600/P-95/002Fa. Office of Research and Development, Washington, DC. August.

- U.S. EPA (Environmental Protection Agency). 1997c. *Exposure Factors Handbook, Volume III, Activity Factors*. EPA/600/P-95/002Fa. Office of Research and Development, Washington, DC. August.
- U.S. EPA (Environmental Protection Agency). 2000. Development of Statistical Distributions for Exposure Factors.

7.0 Human Health Risk Results

The final step of the risk assessment process is to characterize the risk posed to receptors (e.g., farmers and fishers). In this step, the preceding components of the risk assessment—estimates of toxicity (the health benchmarks) and exposure assessments—are summarized and integrated into quantitative expressions of risk. For this risk assessment, estimates of dose and toxicity were used to calculate individual excess lifetime carcinogenic risk estimates for all dioxin, furan, and PCB congeners in biosolids as a total TEQ. Section 7.1 describes the risk calculations completed for this analysis. Section 7.2 describes multipathway risks.

7.1 Human Health Risk Characterization

The goal of this risk assessment was to estimate a national distribution of the incremental increase in individual lifetime risk of developing cancer due to exposure to dioxins, furans, and PCBs potentially present in the biosolids for farm families who apply biosolids as fertilizer or soil conditioner. The probabilistic analysis showed that biosolids are equally likely to be applied to farms nationwide (i.e., no region is more likely to have this practice than another). The farmer is assumed to apply biosolids at agronomic rates once every other year for a maximum of 40 years (maximum 20 additions). The biosolids are assumed tilled into crop land but not tilled into pasture land The farmer is assumed to consume a significant portion of his diet from homegrown items produced on the biosolids-amended land. This scenario does not represent the general population but intentionally reflects the risks to highly exposed individuals within the subpopulation of farmers who apply biosolids.

7.1.1 Lifetime Excess Cancer Risk

Cancer risk was characterized using lifetime excess cancer risk estimates to represent the excess probability of an individual developing cancer over a lifetime as a result of exposure to dioxins, furans, or PCBs in biosolids. Lifetime excess cancer risk estimates use the LADD as the measure of exposure and are the product of the LADD, expressed as a toxicity equivalent of 2,3,7,8-TCDD for a specific receptor (i.e., adult farmer), and the CSF for 2,3,7,8-TCDD, as shown in Equation 7-1. Lifetime excess cancer risk estimates are calculated independently for each route of exposure and for the receptor, assuming multipathway exposures:

Lifetime excess cancer risk= LADD x CSF
$$(7-1)$$

where

LADD = lifetime average daily dose (mg/kg BW/d) CSF = cancer slope factor (mg/kg BW/d)⁻¹.

7.1.2 Total Lifetime Excess Cancer Risk

Congener-specific individual incremental increases in lifetime excess cancer risks were generated for each receptor for each inhalation and ingestion pathway exposure. These pathwayspecific lifetime excess cancer risks for each congener were then summed to generate a total risk due to exposure to all dioxin, furans, and PCBs in biosolids; this total risk for all congeners combined is presented in this section. This total risk is estimated for multipathway exposures, as well as for individual exposure pathways for each of the 3,000 iterations in the probabilistic analysis.

7.1.3 Risk Results

The results of the risk analysis yielded distributions of risk for each receptor for each potential exposure pathway individually and a distribution of risk for each receptor considering multipathway exposures. When each pathway is considered individually, the percentiles of the risk distribution describe the risk from only that single pathway. For example, the risk distribution for the aboveground vegetable ingestion pathway considers only the factors that are included in that pathway, including all factors that increase the concentration of dioxin-like congeners in aboveground vegetation and ingestion rates for this dietary item. The inputs to the risk analysis iteration that yield the 90th percentile risk from the ingestion of aboveground vegetation pathway are highly unlikely to occur in the same iteration as the set of inputs (individual) that yield the 90th percentile risk for the beef ingestion pathway. In addition, the multipathway analysis considered the risk from all pathways simultaneously; thus, the individual with the 90th percentile risk for a single pathway.

Risk represents the combination of the exposure point media concentration and the receptor-dependent exposure factors. The distributions of media concentrations used in the risk calculations and the representative percentiles from the distributions are presented in Section 5.0 The distributions of the exposure factors used in this risk assessment are presented in Section 6.0.

A statistical sensitivity analysis was performed using the inputs and outputs to the probabilistic analysis risk to identify and rank the most influential factors in calculating the risk for each pathway. For all pathways, exposure duration and consumption rate are the two most important factors in the risk calculation. Of the factors that affect the loading of dioxins in biosolids to the soil, the most important factors appear to be the number of applications of biosolids made to the soil prior to the start of the exposure and the rate at which the biosolids are applied. The later in the period of application the family lives on the farm, the higher the average soil concentration during the exposure period because the total soil loadings to which the farmers are exposed are higher. The number of additions of biosolids and the rates at which those additions are made determine the total loading of dioxins in biosolids to the soil. The sensitivity

analysis used to identify these parameters is described in detail in Section 8.1.2.6. The following sections present the percentiles for the incremental increase in individual lifetime risk to the adult and child farmers for each pathway in the probabilistic analysis. All dioxin-like congener concentrations were modeled individually in the exposure analysis.

7.1.3.1 <u>Soil Ingestion</u>. Soil is assumed ingested incidentally by adult farmers and their children. The adult farmer is assumed to ingest soil from the crop land where he is assumed to have the greatest opportunity for exposure. The children are assumed to be exposed to soil concentrations at the residence location (buffer area). The buffer soil is assumed to receive erosion, runoff, and air deposition from both the crop land (tilled) and pasture (untilled) areas. Thus, the soil concentration in the buffer is higher than the soil concentration in the crop area, but lower than the concentration modeled for the pasture area. All soil assumed ingested by the adult farmer and child is assumed to be from their own farm where biosolids are applied (contaminated fraction = 1). The data on soil ingestion rates for both adults and children are limited; therefore, the soil ingestion rate is assumed constant in this analysis. From age 1 to 7, the child is assumed to ingest 100 mg/d (this does not include pica behavior), and all individuals over age 7 (older children and all adults) are assumed to ingest 50 mg/d. Because ingestion rates are assumed constant for soil, risks are driven by the exposure duration and soil concentration. The LADD for soil ingestion is presented below:

$$LADD = I_{soil} \times C_{soil} \times ED \times 365 \times 10^{-6}$$
(7-2)

where

LADD	=	lifetime average daily dose (mg $_{constituent}/kg_{BW}/d$)
I _{soil}	=	intake of soil (mg _{soil} /day)
C _{soil}	=	concentration of in soil (mg/kg soil)
ED	=	exposure duration (yr)
365	=	d/yr
1×10 ⁻⁶	=	kg/mg
BW	=	body weight (kg).

The most important factor in the soil ingestion risk is the length of time that the individual is exposed. The soil concentration to which the individual is exposed is driven by the total loading of the soil with dioxins at the time the individual is exposed. The factors that affect this exposure concentration are the number of applications that have occurred before or during the time of exposure and the rate at which the biosolids are applied to the land. Of these factors, the most important factor, as shown by the sensitivity analysis, is the start year of exposure, which is an indication of how many applications have occurred before the individual moves to the farm (i.e., the later in the process the farmer starts his exposure, the higher the average concentration to which he is exposed, because even if additions of biosolids cease, concentrations fall slowly due to the extended half-life of dioxin in soil). Table 7-1 presents the risks for the soil ingestion pathway for adult farmers and their offspring who begin their exposure in childhood.

	Lifetime Indi	vidual Risk
Percentile	Adult	Child
50 th	8E-9	2E-8
75^{th}	2E-8	4E-8
90 th	5E-8	7E-8
95 th	8E-8	1E-7
99 th	2E-7	2E-7

Table 7-1.	Percentile	Risk fo	r Soil	Ingestion	Pathway
-------------------	------------	---------	--------	-----------	---------

7.1.3.2 Exposed Produce. Exposed produce is assumed to be grown on tilled crop land that is amended with biosolids. The fruits and vegetables receive exposure to dioxin-like congeners only through the air pathway (there is no root uptake of dioxin-like congeners to aboveground vegetation). The concentrations in the produce result primarily from vapor uptake through the leaves and fruit as represented by the air-to-plant transfer factors. The home-produced exposed vegetables are assumed to represent a fraction (fruit = 0.328; vegetables = 0.42) of the total amount of exposed produce the farmer and his offspring consume throughout the year. The exposure factors (exposure duration and intake rate) are the major driving components in the risk equation for this pathway as well. In addition, because this pathway is driven by the air-to-plant transfer of dioxins, the factors that increase the tendency of constituents to volatilize are also important. These factors, such as ambient temperature as noted in Section 5. Table 7-2 presents the risks for the exposed produce ingestion pathway.

	Lifetime In	dividual Risk		
Percentile	Adult	Child		
50 th	1E-9	8E-10		
75 th	5E-9	2E-9		
90 th	2E-8	6E-9		
95 th	3E-8	1E-8		
99 th	1E-7	3E-8		

 Table 7-2.
 Percentile Risk for Exposed Produce Ingestion Pathway

7.1.3.3 <u>Belowground Vegetables</u>. Belowground vegetables are assumed produced on tilled crop land that is amended with biosolids. The farm family is assumed to consume these

home-produced root vegetables as a fraction (0.173) of their total intake of root vegetables. Root vegetables absorb dioxin-like congeners directly from the soil. The exposure factors (exposure duration and intake rate) are most important to the risk calculation; however, for root vegetables, soil parameters are more important than for other pathways. The soil parameter that is identified in the sensitivity analysis is the soil foc, which is a measure of the soil organic carbon content. Because the concentration in the soil is the media concentration that drives this pathway, the factors that reduce losses of dioxins to the environment through volatilization or erosion increase the concentration in the soil. Table 7-3 presents the risks for the belowground vegetable ingestion pathway.

	Lifetime Individual Risk				
Percentile	Adult	Child			
50 th	2E-8	1E-8			
75 th	6E-8	3E-8			
90 th	2E-7	9E-8			
95 th	4E-7	2E-7			
99 th	1E-6	4E-7			

Table 7-3.	Percentile	Risk for	Belowground	Vegetable	Ingestion	Pathway
			0	0	0	•

7.1.3.4 Poultry. Free-range chickens are assumed to be raised by the farm family near the residence. The chickens are assumed to be confined to the buffer area and, thus, are assumed to consume soil only from that area. The chicken feed is assumed to be purchased from an uncontaminated source. The concentration of dioxin-like congeners in poultry is calculated based on the lipid concentration of chicken thigh meat. The farm family is assumed to consume homegrown chickens as a fraction (0.156) of their total poultry consumption. The exposure factors (exposure duration and intake rate) are again the most important factors in the risk calculation. The media concentration that drives this pathway is the soil concentration in the buffer area. This soil receives dioxins predominantly from erosion from the crop land and pasture amended with biosolids with some contribution from air deposition from these areas. Thus, the factors that lead to higher concentrations in the soil also lead to higher concentrations in the poultry. These factors include higher application rates and a greater number of applications at the time of exposure. Table 7-4 presents the risks for the poultry ingestion pathway.

7.1.3.5 <u>Eggs</u>. The free-range chicken scenario used in the poultry scenario was also assumed for the egg ingestion scenario. The farm family is assumed to consume home-produced eggs as a fraction (0.146) of their total egg consumption. The factors important to the poultry ingestion pathway are identical to the egg ingestion pathway. Table 7-5 presents the risks for the egg ingestion pathway.

	Lifetime Individual Risk		
Percentile	Adult	Child	
50 th	3E-8	3E-8	
75 th	1E-7	8E-8	
90 th	3E-7	2E-7	
95 th	5E-7	2E-7	
99 th	1E-6	6E-7	

Table 7-4. Percentile Risk for Poultry Ingestion Pathway

 Table 7-5. Percentile Risk for Egg Ingestion Pathway

	Lifetime Individual Risk		
Percentile	Adult	Child	
50 th	4E-8	4E-8	
75 th	1E-7	9E-8	
90 th	3E-7	2E-7	
95 th	6E-7	2E-7	
99 th	2E-6	7E-7	

7.1.3.6 <u>Beef</u>. Beef cattle are assumed to be raised on the pasture that is top-dressed with biosolids. The cattle are assumed to graze in the amended pasture obtaining 48 percent of their diet from forage, 48 percent from silage, and 4 percent from incidental ingestion of surficial soil while grazing in the pasture. The cattle consumed by the farm family are assumed *not* finished in a feed lot. Exposure factors (exposure duration and intake rate) and soil loading factors (application rate and number of applications) also drive this pathway. The majority of the concentration in the beef is due to the concentration of dioxins in the pasture grass where the cattle forage. The concentration in the forage is due to air-to-plant transfer of vapors. Thus, higher loadings of biosolids to the soil, especially in areas where it is hot and dry to promote volatilization of constituents from the soil, increase the risk from the beef ingestion pathway. The farm family that raises beef cattle is assumed to obtain 49 percent of the beef it consumes from home-raised cattle. Table 7-6 presents the risks for the beef ingestion pathway.

	Lifetime Individual Risk		
Percentile	Adult Child		
50 th	6E-7	6E-7	
75 th	2E-6	1E-6	
90 th	6E-6	5E-6	
95 th	1E-5	5E-6	
99 th	3E-5	1E-5	

Table 7-6. Percentile Risk for Beef Ingestion Pathway

7.1.3.7 <u>Milk</u>. Dairy cattle are also assumed to be raised on a pasture that is top-dressed biennially with biosolids. The cattle are assumed to graze in the amended pasture; however, dairy cattle are assumed to obtain only 8 percent of their diet from forage, 90 percent from silage, and 2 percent from incidental ingestion of surficial soil in the pasture. The same factors that drive the risk from the beef pathway also drive the risks from the dairy pathway, although the dairy cattle are assumed to eat less forage. The silage that dairy cattle are assumed to consume is grown on crop land that is amended with biosolids. Thus, the silage is assumed to receive dioxins through air-to-plant transfer to the nongrain portion of the silage (0.5). The concentration of dioxins in silage is less than that in forage; however, it is still a significant source of dioxin in milk. The farm family that raises dairy cattle is assumed to obtain 25 percent of its total milk consumption from home-raised cattle. Table 7-7 presents the risks for the milk ingestion pathway.

	Lifetime Individual Risk		
Percentile	Adult	Child	
50 th	3E-7	5E-7	
75 th	1E-6	1E-6	
90 th	3E-6	3E-6	
95 th	6E-6	5E-6	
99 th	2E-5	1E-5	

 Table 7-7. Percentile Risk for Milk Ingestion Pathway

7.1.3.8 <u>Fish</u>. Edible fish are assumed to be caught from a stream adjacent to the farm where biosolids are applied. The stream, therefore, receives runoff, erosion, and air deposition from the amended crop land and pasture. The eroded soil from the farm is transported across the buffer directly to the stream. The stream also receives direct air deposition of particles and

vapors from the crop land and pasture. In addition, particles and vapors from the crop land and pasture are transported and deposited on the much larger area of the regional watershed from which they are also eroded to the modeled stream. The fish that live in the stream are assumed to include T3 and T4 fish (i.e., edible species). These fish are assumed to be caught and consumed by a recreational fisher. This fisher may also be the farmer who applies the biosolids or an individual from a nearby town who has no other pathways of exposure. The recreational fisher is assumed to catch all the home-caught fish he consumes from this single stream adjacent to the biosolids-amended field. Table 7-8 presents the risks for the fish ingestion pathway.

	Lifetime Individual Risk	
Percentile	Adult	
50 th	8E-10	
75 th	4E-9	
90 th	2E-8	
95 th	4E-8	
99 th	2E-7	

Table 7-8. Percentile Risk for Fish Ingestion Pathway

7.1.3.9 <u>Ambient Air</u>. The ambient air concentration that the farm family is assumed to breathe is the average air concentration estimated over the residential buffer. The vapor and particulate air concentrations are estimated independently in the air modeling, but are summed in the inhalation risk estimates. The risks for this pathway are driven predominantly by exposure factors (exposure duration and inhalation rate), also. Other factors that influence this pathway are the loading to the soil and the soil and climate properties that lead to greater air emissions from the amended soil. Table 7-9 presents the risks for the air inhalation pathway.

7.1.3.10 <u>Breast Milk</u>. The lactating woman is an adult member of the farm family and, therefore, is assumed to consume all types of home-produced food with the consumption rates and fractions homegrown presented in the preceding sections for each of the following dietary items: exposed produce, root vegetables, poultry, eggs, beef, and milk. The mother is assumed to have reached a steady-state concentration of dioxins in lipids before lactation begins. The maternal concentrations of dioxins are then modeled to partition each congener to the lipid fraction of breast milk, which is subsequently assumed ingested by an infant. The infant is assumed to consume no homegrown dietary items directly and, thus, obtains exposures only through the ingestion of breast milk during the first year of life. Table 7-10 presents the risks for the breast milk ingestion pathway.

	Lifetime Individual Risk		
Percentile	Adult	Child	
50 th	7E-10	9E-10	
75^{th}	2E-9	2E-9	
90 th	7E-9	5E-9	
95 th	1E-8	8E-9	
99 th	3E-8	2E-8	

 Table 7-9. Percentile Risk for Air Inhalation Pathway

Table 7-10. Percentile Risk for Breast Milk Ingestion Pathway

	Lifetime Individual Risk	
Percentile	Infant	
50 th	2E-9	
75 th	4E-9	
90 th	8E-9	
95 th	1E-8	
99 th	3E-8	

7.2 Multipathway Risks

The structure of the probabilistic analysis is based on the modeling of 3,000 individual exposure scenarios for adults and children. This means that for each individual (iteration) in the analysis, a set of intake rates is chosen from the distribution for each pathway for an adult receptor and a child receptor. The intake rates for dietary items are not correlated in any way. Insufficient data are available to enable correlation. This process allows the evaluation of risk from each pathway independently, and it also allows the evaluation of multipathway risks. The distribution of multipathway risks is the distribution of the sum of the risks across pathways for each of the 3,000 individual adults and children in the analysis. Thus, for example, the 90th percentile multipathway risk to an adult receptor may not correspond to the 90th percentile risk for any single pathway, but it is selected from the distribution of 3,000 risks summed across all pathways. Multipathway risks were also evaluated for the adult farmer and his child. The multipathway results presented in Tables 7-11 through 7-13 are from the distribution of the total risk. Table 7-11 presents the multipathway risks to the adult and child members of the farm family and includes the lifetime average daily dose (LADD) that produced these risks.

	Adult		Child	
Percentile	Risk	Daily Exposure, pg TEQ/kg-d	Risk	Daily Exposure, pg TEQ/kg-d
50 th	1 × 10 ⁻⁶	0.009	1×10^{-6}	0.009
75 th	4×10^{-6}	0.03	3×10^{-6}	0.02
90 th	1×10^{-5}	0.06	$7 imes 10^{-6}$	0.04
95 th	2×10^{-5}	0.1	1×10^{-5}	0.06
99 th	$4 imes 10^{-5}$	0.3	2×10^{-5}	0.1

Table 7-11. Multipathway Risks and Associated LADD forAdult and Child Farm Family Members—Baseline All Samples from 2001 NSSS

In order to see the effect of setting a cutoff limit based upon the TEQ of the biosolids sample, several additional evaluations of multipathway risk were made with biosolids samples having TEQ values above a specified value removed. The cutoff limits examined in this way were 300 ng/kg TEQ and 100 ng/kg TEQ. Table 7-12 presents the multipathway risks to the adult and child members of the farm family using only the biosolid samples with a total TEQ concentration below 300 ng/kg TEQ. Table 7-13 presents the multipathway risks to the adult and child members of the farm family using only the biosolid samples with a total TEQ concentration below 100 ng/kg TEQ.

No decrease in total risk is observed with the elimination of the samples with the highest concentrations. This is because there are so few samples in these concentration ranges.

Percentile	Adult	Child
50 th	1E-6	1E-6
75 th	4E-6	3E-6
90 th	1E-5	7E-6
95 th	2E-5	1E-5
99 th	4E-5	2E-5

Table 7-12. Multipathway Risks for Adult and Child Farm Family Members—300 TEQ Cutoff Limit for Samples from 2001 NSSS

Table 7-13. Multipathway Risks for Adult and Child Farm Family Members—100 TEQ Cutoff Limit for Samples from 2001 NSSS

Percentile	Adult	Child
50 th	1E-6	1E-6
75 th	4E-6	3E-6
90 th	1E-5	6E-6
95 th	2E-5	1E-5
99 th	4E-5	2E-5
8.0 Analysis of Variability and Uncertainty

This section discusses the methods that were used in the risk assessment for dioxins, furans, and PCBs in biosolids to account for variability and uncertainty. Variability and uncertainty are fundamentally different. Variability represents true heterogeneity in characteristics such as body weight differences within a population or differences in contaminant levels in the environment. It accounts for the distribution of risk within the exposed population.

Variability arises from true heterogeneity in characteristics, such as body weight differences within a population or differences in contaminant levels in the environment.

Uncertainty represents lack of knowledge about factors, such as the nature of adverse effects from exposure to constituents, that may be reduced with additional research.

Uncertainty, on the other hand, represents lack of knowledge about factors, such as adverse effects from contaminant exposure, that may be reduced with additional research to improve data or models.

This discussion describes the treatment of variability and uncertainty in reference to some parameters used to describe human exposures and risk. Treatment of variability using a Monte Carlo simulation forms the basis for the human health risk distributions, which in turn are the basis for calculating a protective concentration for dioxins, furans, and PCBs in biosolids. Previous sections of this document describe how distributions were generated and point values estimated for input parameters. They also describe how these values were used in the models and in calculations to produce a national-level TEQ concentration in biosolids that is protective of human health. Uncertainty necessitated the use of assumptions and default values in this study. This discussion focuses on how this treatment of variability and uncertainty affects the results.

8.1 Variability

Variability is often used interchangeably with the term uncertainty, but the two are not synonymous. Variability is tied to variations in physical, chemical, and biological processes and cannot be reduced with additional research or information. Although variability may be known with great certainty (e.g., age distribution of a population may be known and represented by the mean age and its standard deviation), it cannot be eliminated and needs to be treated explicitly in the analysis. Spatial and temporal variability in parameter values used to model exposure and risk account for the distribution of risk in the exposed population.

For example, the meteorological parameters used in dispersion modeling, such as windspeed and wind direction, are measured hourly by the National Weather Service at many

locations throughout the United States, and statistics about these parameters are well documented. Although the distributions of these parameters may be well known, their actual values vary spatially and temporally and cannot be predicted exactly. Thus, the concentration calculated by a dispersion model for a particular receptor for a particular time period will provide information on average conditions that may over- or underpredict actual concentrations. Much of the temporal variation is accounted for by using models such as ISCST3 that calculate concentrations hourly and sum these hourly values to provide annual concentration estimates. Additionally, using meteorological data from multiple monitoring stations located throughout the United States can account for some but not all spatial variability.

In planning this analysis, it was important to specifically address as much of the variability as possible, either directly in the Monte Carlo analysis or through disaggregation of the data into discrete elements of the analysis. For example, use of a refined receptor grid accounts for spatial variability in concentrations on and around the agricultural field where biosolids are applied. Variability in agricultural practices is accounted for by using distributions that represent the range of possible agricultural practices.

Spatial variability in environmental setting was accounted for by using 41 different climatic regions throughout the contiguous 48 states. Because biosolids are generated nationwide, the application of biosolids to agricultural fields may occur nationwide; thus, this analysis characterized environmental conditions that influence the fate and transport of constituents in the environment using regional data based on climatic conditions.

The risk assessment components discussed include

- Source characterization and emissions modeling
- Fate and transport modeling
- Exposure modeling.

8.1.1 Source Characterization and Emissions Modeling

The specific agricultural fields where biosolids were applied were not known; however, EPA assumed that biosolids could be applied to any agricultural land. For this analysis, agricultural field areas were varied according to climatic regions. The median farm size for each climatic region was used to represent the regional variability of farm size. However, uncertainty about farm size within a climatic region remained. Distributions were used to capture nationwide variability in agricultural practices. The variation in median farm size based on regions and the nationwide distribution of agricultural practice parameters was used in the probabilistic analysis to characterize the national variation in farm areas and operating characteristics.

Source partition modeling was performed for 41 different climatic regions, which allowed variation in location-dependent parameters (e.g., soil, temperature, precipitation) to be considered explicitly in the modeling. Variation in these parameters influenced variation in predicted air emissions rates. These meteorological data sets were combined with the surface area of the

agricultural field to provide unit air concentrations (UACs), which were used with emissions data to estimate air concentrations for crop land and pastures.

In the Monte Carlo analysis, the agricultural field characteristics, environmental conditions from 41 climatic regions, and parameter values for biosolids characteristics were combined to produce the 3,000 iterations of the source partition model calculations. The source model calculations generated the distribution of environmental releases used in the fate and transport modeling.

8.1.2 Fate and Transport Modeling

The parameter values required to model contaminant fate and transport were obtained from regional databases. The treatment of regional variation in location-dependent parameters used in fate and transport modeling is discussed in the following sections.

8.1.2.1 <u>Air Dispersion Modeling</u>. To capture geographic variation, dispersion modeling was conducted using meteorological data sets from 41 different meteorological stations throughout the contiguous 48 states. This provided regional representation of the variability in meteorological data. Obviously, 41 meteorological stations do not represent every site-specific condition that could exist in the 48 states. However, in selecting the climatic regions, consideration was given to representing different Bailey's ecological regions and to not excluding from the analysis those areas with unique dispersion characteristics (e.g., coastal areas). Thus, it is believed that these 41 climatic regions are a reasonable representation of the variability in meteorological conditions for the United States.</u>

8.1.2.2 <u>Soil and Water Modeling</u>. Soil characteristics were based on the location of the 41 climatic regions used in the modeling. Soil characteristics for all nonurban soil within the climatic region were used to determine the soil characteristics for watershed modeling. This approach captured the national distribution of soil types and accounted for regional variation in soil characteristics.

Waterbody characteristics were not varied in the fate and transport modeling. However, in addition to variation in soil type and precipitation, watershed modeling also took into account regional variation in agricultural field size and regional watershed size, which can affect constituent loading to the waterbody via runoff and erosion. Otherwise, regional variations in waterbody were not accounted for in this analysis.

8.1.2.3 <u>Terrestrial and Aquatic Food Chain</u>. To the extent that agricultural field size and variation in regional watershed areas affects runoff and erosion of constituents into the waterbodies modeled in this assessment, the variation had an effect on runoff and erosion loadings to the waterbody. Otherwise, no regional variations were considered for the aquatic food chain modeling.

8.1.2.4 Exposure Modeling. Individual physical characteristics, activities, and behavior are quite different. As such, the exposure factors that influence the exposure of an individual, including inhalation rate, ingestion rate, body weight, and exposure duration, are quite variable.

To include this variability explicitly in the analysis, statistical distributions for these variables were used for each receptor in the analysis: adult, child, and infant in the farm family and a recreational fisher. For adults, a single exposure factor distribution was used for males and females. For child exposures, one age group (ages 1 to 6) was used to represent the age at the start of exposure, because this age group is considered to be most sensitive for most health effects. The infant was evaluated only for breast milk ingestion during the first year of life. Exposure parameter data from the EFH (U.S. EPA, 1997a, b, c) were used to establish statistical distributions of values for each exposure parameter for each receptor.

8.1.2.5 <u>Summary of Variability Considerations</u>. In summary, a protective biosolids concentration was developed that includes specific consideration of the variability in

- Agricultural field size and biosolids characteristics
- Agricultural practices
- Regional-specific environmental conditions
- Exposure factors for each receptor.

Taken together, these provide nationally applicable risk-specific TEQ concentration for dioxins, furans, and PCBs in biosolids.

8.1.2.6 <u>Sensitivity Analysis</u>. A statistically based sensitivity analysis was performed to rank the variable parameters in the analysis according to their contribution to the variability of the resulting risk for each pathway. This methodology is referred to as a response surface regression approach because it uses models characteristic of those used in a response surface experiment. Response surface methodology involves a statistical approach to designing experiments and an associated model estimation methodology. The terminology "response surface" derives from the fact that a regression model involving a number of continuous independent variables can be viewed as providing an estimated surface of the results in space. Often a goal of response surface experimentation is to ascertain the combination(s) of input variable values that will yield a minimum or a maximum response. The complexity of the model (e.g., whether it contains only first- and second-order terms or terms of higher degree) determines the general shape of the contours and the degree to which the "true" surface can be approximated.

In this analysis, a regression analysis was applied to a linear equation to estimate the relative change in the output of a probabilistic simulation relative to the changes in the input variable values. This methodology is one of the recommended methods for conducting a sensitivity analysis based on the results of a Monte Carlo analysis described in Appendix B of *RAGS 3A - Process For Conducting Probabilistic Risk Assessment - Draft* (1999) (U.S. EPA, 1999).

Sensitivity analyses historically were conducted by evaluating how much change in risk occurred as a result of varying an individual input variable from a median or mean value to a 90th percentile or high-end value. When the risk depends on the aggregate impact of a number of input variables, however, such an approach may not necessarily identify the most important one. This may occur for several reasons:

- The ranges chosen for the various input variables may not be defined consistently.
- Various input variables may interact with one another (i.e., the effect of input X_1 on an outcome Y depends on the level of other inputs X_2 , X_3 , etc., so that the observed effect of X_1 depends on what values were chosen for the other variables as well).
- Nonlinear effects may obscure the effect of the input variable (e.g., if only low and high values of an input variable are examined but the relationship between the risk and the input variable is of a quadratic nature, then the importance of the input variable may be overlooked).

To address such issues, statistical regression methods were used to perform the sensitivity analyses. Although regression methods have distinct advantages over previous approaches, certain limitations remain. Regression methods are not capable of determining the sensitivity of model results to input variables that are not varied in the analysis (e.g., assumptions) or are not otherwise included within the scope of the analysis (e.g., model-derived variables). If, for some reason, the most important variables are not varied or their variability is improperly characterized, the sensitivity analysis may not identify them as being important.

The sensitivity analysis was conducted on a data set generated during modeling of each pathway. For example, a set of input variables $(X_1, X_2, ..., X_p)$ was used in the modeling simulation.

The result of interest is the individual risk calculated for each pathway as a result of exposure to all dioxin-like congeners as expressed as a TEQ. In this case, the *Xs* are parameters associated with agricultural practices, site, environmental conditions, and exposure parameters.

The regression approach uses the various combinations of X values that were used during the simulation and the resulting risk values as input data to a regression model. Functions of the results variables (denoted as Ys) were treated as dependent variables; for example, Y denoted the logarithm of the risk. Functions of the Xs were treated as independent variables. The goals of the approach were

- 1. To determine a fairly simple polynomial approximation to the simulation results that expressed the *Ys* as functions of the *Xs*
- 2. To optimize this "response surface" and assess the importance of the various *Xs* by performing statistical tests on the model parameters
- 3. To rank the *Xs* based on their relative contribution (in terms of risk) to the final response surface regression model.

These goals were realized using a second-order regression model. Such a model takes the following form:

$$\hat{Y} = \hat{\beta}_0 + \sum_{k=1}^{p} \hat{\beta}_k x_k + \sum_{k=1}^{p} \hat{\beta}_{kk} x_k^2 + \sum_{k=1,j=k+1}^{p-1} \hat{\beta}_{kj} x_k x_j$$
(8-1)

where the βs are the least squares regression estimates of the model parameters.

The statistical significance of the parameters associated with the first-order, squared, and cross-product terms were tested and all nonsignificant terms were removed from the model. The parameters in this reduced model were then reestimated and the process of testing was repeated. This was done to capture the most important independent variables (Xs) that influence the dependent variables (Ys).

Once the final regression model was developed, the input parameters (Xs) were ranked based on percentage of risk accounted for by that parameter. The percent risk was calculated using the following equation:

$$Percent Risk = \frac{[FMSS - RMSS]}{[FMSS + ERSS]}$$
(8-2)

where

- FMSS = model sum of squares for the final model
- RMSS = model sum of squares for a model in which all terms involving x_u are removed (i.e., a reduced model)
- ERSS = model error sum of squares.

The two parameters responsible for the largest percentage of the risk are the two parameters set to high-end values in the deterministic analysis.

The major steps in the sensitivity analysis are identified below, along with details on the reasons for these steps.

- Perform any necessary manipulations to the data set. To perform the sensitivity analysis, the data set must contain only one record for each Monte Carlo iteration, and all variables in the data set must be numeric.
- Remove any variables that are constants. Any variable that was constant across all Monte Carlo iterations does not have any effect on the resulting risk and was removed from the data set prior to the start of the regression analysis.
- Perform transformations (log, square root, etc.) to the continuous input variables, if necessary, so that all input variables will have approximately symmetric distributions. Transforming the input variables so that each one has

an approximately symmetric distribution is necessary to make the standardization of the variables meaningful (i.e., so the mean is near the midpoint of the extremes, and the mean and standard deviation are not highly related).

- Check the correlations of the transformed input variables. Remove any input variables that are highly correlated with other input variables in the data set. Regression analysis measures the linear relationship between the terms in the model and the response variable. If two or more input variables are highly correlated with one another, then there is a strong linear relationship between those input variables. Keeping all highly correlated variables in the model will reduce the significance of each of the correlated input variables because each one is essentially explaining the same linear relationship with the response variable (i.e., the effect of one such variable may mask the effect of another).
- Standardize the transformed variables. Standardizing the input variables (i.e., subtracting the mean and dividing by the standard deviation) allows the regression results to be independent of the magnitude of the value of the input variables. The larger value input variables could cause the regression results to seriously underestimate the effects of the smaller value input variables on the changes in environmental concentration and risk. The combination of transforming and standardizing the input variables creates more optimal conditions for regression analysis.
- Use response surface regression methods to test for the main effects, squared terms, and cross products that have the greatest effect on the log(environmental concentration). Develop a model for log(environmental concentration) based on the results of the regression analysis. After the response surface regression results are obtained, the significance of each term on environmental concentration is evaluated. First, any second-order terms that are determined not to have a significant effect on the environmental concentration are dropped from the model. Any first-order term that is part of a significant secondorder term will remain in the model, regardless of the level of significance of that first-order term. For example, if the second- order term $X1 \times X2$ has a significant effect on the environmental concentration and remains in the model, then both of the first-order terms, X1 and X2, will also remain in the model. Any first-order terms that are determined not to be significant and not to have any significant second-order terms are dropped from the model. The regression analysis is then conducted on the reduced model. This process is repeated until all of the secondorder terms in the model have significant effects on the environmental concentration and no more terms can be removed. The iterative process of dropping insignificant terms and reevaluating the model allows only the input variables with the most effect on the environmental concentration to remain in the model.
- Use the model for log(environmental concentration) as part of the model for the log(risk). The equation that must be evaluated is

 $risk = \frac{environmental \ concentration \ \times \ risk \ factor \ \times \ exposure \ duration \ \times \ intake}{body \ weight}$ (8-3)

Taking the log of both sides of the above equation results in

log(risk) = log(environmental concentration) + log(risk factor) +log(exposure duration) + log(intake) - log(body weight)(8-4)

The log(environmental concentration) in the above equation is replaced with the final model of input variables from the regression analysis in the previous analysis step. Regression analysis is performed on the new model for log(risk).

Test for the effect of each variable on log(risk) and use the *p*-values to rank the variables by the amount of effect each variable has on log(risk). Because the final model will most likely contain first- and second-order terms involving the same input variables, *F*-tests need to be performed to evaluate the effect of each input variable in the final model on the log(risk). The *F*-tests of each variable will be of the form

$$F = \frac{[FMSS - RMSS] / [FMDF - RMDF]}{FRSS / FRDF}$$
(8-5)

where

FMSS	=	model sum of squares for full model containing all significant terms
RMSS and RMDF	=	model sum of squares and degrees of freedom, respectively, for reduced model
FMDF	=	model degrees of freedom for full model
FRSS and FRDF	=	residual sum of squares and degrees of freedom, respectively, for full model.

The full model refers to the model containing all significant terms in the final log(risk) model. The reduced model refers to the full model minus all terms containing the input variable *X* whose significance is being tested. The *F*-tests evaluate the effect of variable *X* on the risk by evaluating the differences when variable *X* is in the regression model (full model) and when all model terms containing variable *X* are removed (reduced model). If a substantial increase in the residuals results from ignoring terms involving the variable *X*, then *F* will be "large," implying that these factors can be considered important, in the sense that

they require different regression coefficients for the Xs. The ordering of the p-values from such tests can then be used to rank the importance of the various factors on the risk. The most important four parameters for each pathway identified by the sensitivity analysis are presented in Table 8-1. Detailed results of the sensitivity analysis are presented in Appendix K.

8.2 Uncertainty

Uncertainty is a description of the imperfection in knowledge of the true value of a particular parameter. In contrast to variability, uncertainty is reducible by additional information-gathering or analysis activities (e.g., better data, better models). EPA typically classifies the major areas of uncertainty in risk assessments as scenario uncertainty, model uncertainty, and parameter uncertainty. Scenario uncertainty refers to missing or incomplete information needed to fully define exposure and dose. Model uncertainty is a measure of how well the model simulates reality. Parameter uncertainty is the lack of knowledge regarding the true value of a parameter used in the analysis.

Although some aspects of uncertainty were directly addressed in this analysis, much of the uncertainty associated with this analysis could only be addressed qualitatively. Significant sources of uncertainty are presented in this section. If the analysis directly addressed uncertainty, the approach used is described. If the analysis did not directly address uncertainty, a qualitative discussion of its importance is provided.

8.2.1 Scenario Uncertainty

Sources of scenario uncertainty include the assumptions and modeling decisions that are made to represent an exposure scenario. The entire hypothetical farm scenario is a source of uncertainty in this analysis. The analysis is based on a single conceptual site model that assumes biosolids are applied to a farm that is half crop land and half pasture and that the farm family lives adjacent to those areas where biosolids are applied. These are reasonable assumptions; however, much uncertainty is associated with the scenario. The lack of information or resources to define and model actual exposure conditions introduced uncertainty into this analysis.

Professional judgment, data availability, and, in some cases, an evaluation of the results of a sensitivity analysis are used to decide which parameters to include in describing exposure conditions and behaviors. Scenario uncertainties that are important to understand in interpreting the results of this study are discussed in the following subsections.

8.2.1.1 <u>Biosolids Characteristics</u>. Few data were available on the physical and chemical characteristics of biosolids. To address this lack, assumptions on specific biosolids characteristics were based on general knowledge of biosolids. In this analysis, except for constituent concentration, which was measured, general biosolids characteristics, including default assumptions for bulk density, moisture, and porosity, were used.

Pathway	Sensitivity Variables	Percent of Risk Accounted for by Variable
Air	Exposure duration	66
	Inhalation rate	4
	Soil moisture retention exponent b	2
	Application rate	2
Soil	Exposure duration	78
	Average year that the farm family moves in	3
	Application rate	2
	Body weight	1.5
Aboveground vegetables and fruit	Exposure duration	Fruit 56, Veg. 59
	Consumption rate	Fruit 11, Veg. 6
	Average year that the farm family moves in	Fruit 2, Veg. 3
	Soil moisture retention exponent b	Fruit 2, Veg. 2
Root vegetable	Exposure duration	49
	Consumption rate	30
	Soil fraction organic carbon (foc)	10
	Average year that the farm family moves in	4
Poultry	Exposure duration	55
	Consumption rate	33
	Average year that the farm family moves in	2
	Application rate	1
Egg	Exposure duration	60
	Consumption rate	28
	Average year that the farm family moves in	2
	Application rate	1
	Exposure duration	60
Deef	Consumption rate	26
Beel	Application rate	1
	Average year that the farm family moves in	1
Milk	Exposure duration	54
	Consumption rate	32
	Average year that the farm family moves in	1
	Application rate	1
Fish	Consumption rate	47
	Exposure duration	34
	Average year that the farm family moves in	1
	Application rate	1

Table 8-1. Results of Sensitivity Analysis by Pathway

8.2.1.2 <u>Characteristics and Location of Waterbodies</u>. One aspect of the site layout of particular relevance to aquatic food chain modeling is the location and characteristics of the waterbodies. The size of the waterbody impacts constituent concentration predicted for that waterbody. The waterbody characteristics selected were for a third-order stream, intended to represent a small but fishable waterbody. This small size would tend to ensure that calculated waste concentrations would be protective of routes of exposure from surface water. The location of the waterbody was assumed to be at the edge of the agricultural field.</u>

8.2.1.3 <u>Receptor Populations Evaluated</u>. The land use for the application of biosolids to agricultural fields is assumed to be agricultural. As such, human receptors evaluated include an adult farmer, the child and infant of the farmer, and a resident who is a recreational fisher at a nearby waterbody. Risk estimates presented in this document address hypothetical chronic exposures for these receptors and are designed to provide a realistic range of potential scenarios.

8.2.1.4 Exposure Uncertainty. Exposure modeling relies heavily on default assumptions concerning population activity patterns, mobility, dietary habits, body weights, and other factors. As described earlier in the variability section, the probabilistic analysis for the adult and child exposure scenario addressed the possible variability in the exposure modeling by using distributions of values for exposure factors. There are some uncertainties, however, in the data that are used. Although it is possible to study various populations to determine various exposure parameters (e.g., age-specific soil ingestion rates or intake rates for food) or to assess past exposures (epidemiological studies) or current exposures, risk assessment is about prediction. Therefore, long-term exposure monitoring in this context is infeasible. The EFH (U.S. EPA, 1997a,b,c) provides the current state-of-the-science concerning exposure assumptions, and it is used throughout this document. To the extent that actual exposure scenarios vary from the assumptions in this risk assessment, risks could be underestimated or overestimated. For example, there could be farmers and children who have higher exposures than those predicted; however, it is more likely that actual exposures for most of these individuals would fall within the predicted range and, moreover, would be similar to what was modeled.

8.2.1.5 <u>Natural Background Exposures</u>. Dioxins are present in the environment as a result of the application of biosolids and from other sources. Thus, receptors potentially receive a "background" exposure that may be greater than the exposure resulting from release of dioxins from biosolids. For national analyses such as this assessment, the inclusion of background concentrations as part of the analysis is not feasible because of the variability of background concentrations for each constituent. Not including the exposure an individual may already have to a constituent of concern (i.e., exposure to background concentrations) does not change the "incremental" increase in risk to an individual due to possible exposures to constituents in biosolids.

8.2.2 Model Uncertainty

Model uncertainty is associated with all models used in all phases of a risk assessment because models and their mathematical expressions are simplifications of reality that are used to approximate real-world conditions and processes and their relationships. Computer models are simplifications of reality, requiring exclusion of some variables that influence predictions but cannot be included in models either because of their complexity or because data are lacking on a particular parameter. Models do not include all parameters or equations necessary to express reality because of the inherent complexity of the natural environment and the lack of sufficient data to describe the natural environment. Because this is a probabilistic assessment that predicts what may occur with the management of biosolids under assumed scenarios, it is not possible to compare the results of these models (sometimes referred to as model validation) to any specific situation that may exist. The risk assessor needs to consider the importance of excluded variables on a case-by-case basis, because a given variable may be important in some instances and not in others. A similar problem can occur when a model that is applicable under average conditions is used for conditions that differ from the average. In addition, in some instances, choosing the correct model form is difficult when conflicting theories seem to explain a phenomenon equally well. In other instances, EPA does not have established model forms from which to choose to address certain phenomena, such as facilitated transport.

Models used in this risk assessment were selected based on science, policy, and professional judgment. These models were selected because they provide the information needed for this analysis and because they are generally considered to be state-of-the-science. Even though the models used in the risk analyses are used widely and have been accepted for numerous applications, they each retain significant sources of uncertainty. Evaluated as a whole, the sources of model uncertainty in this analysis could result in either an overestimation or underestimation of risk. For example, exposure modeling relies heavily on default assumptions concerning population activity patterns, mobility, dietary habits, body weights, and other factors. There are some uncertainties associated with some of the data used for these parameters. Although it is possible to study various populations to determine various exposure parameters (e.g., age-specific soil ingestion rates or intake rates for food) or to assess past exposures (epidemiological studies) or current exposures, risk assessment is about prediction. Therefore, long-term exposure monitoring in this context is infeasible. The EFH (U.S. EPA, 1997a,b,c), which provides the current state-of-the-science concerning exposure assumptions, was used in this risk assessment. To the extent that actual exposure factors vary from the assumptions in this risk assessment, risks could be underestimated or overestimated.

Another issue in model uncertainty is the number of iterations necessary to achieve convergence of the analysis, especially at the higher ends of the distribution. In order to determine the convergence of this analysis, the results from various portions of the iterations were selected from the total number of iterations. The percentile values for the 90th, 95th, and 99th percentiles of the smaller number of iterations were compared to the 90th, 95th, and 99th percentiles for 10,000 iterations. Convergence at the 95th percentile was achieved with 2,500 to 3,000 iterations. Thus, for this analysis, 3,000 iterations was assumed to be sufficient to estimate a reliable distribution of risks, including risks at and above the 95th percentile. Figure 8-1 presents the convergence analysis of risk values.

8.2.2.1 <u>Air Dispersion Modeling</u>. The ISCST3 model was used to calculate the dispersion of particle and vapor emissions from a waste management unit. This model has many capabilities needed for this assessment, such as the ability to model area sources. For dispersion modeling of this type, ISCST3 is considered a fairly accurate model with error within about a



Figure 8-1. Convergence analysis.

factor of 2. It does not include photochemical reactions or degradation of a chemical in the air, which results in additional model uncertainty. Deposition and associated plume depletion are important for particulates and vapors and were explicitly incorporated into this analysis. Currently, algorithms specifically designed to model the dry deposition of gases have not been verified for the specific compounds in question (primarily volatile organics). In place of algorithms, a transfer coefficient was used to model the dry deposition of gases. A concern with this approach is that the deposition is calculated outside of the model. As a result, the mass is deposited on the ground from the plume and is not subtracted from the air concentrations estimated by ISCST3. This results in a slight nonconservation of mass in the system.

Other uncertainties introduced into the analysis in dispersion modeling are related to agricultural field shape. A square shape was selected because it minimizes the error introduced by not knowing the orientation of the agricultural field to wind direction.

8.2.2.2 <u>Human Health Benchmarks</u>. Toxicological benchmarks are designed to be conservative (that is, to potentially overestimate risk) because of the uncertainties and challenges associated with condensing toxicity data into a single quantitative expression.

<u>Cancer Slope Factor</u>. The CSF for TCDD was derived as the 95 percent upper confidence limit of the slope of the dose-response curve using a linear, no-threshold, dose-response model. The CSF, is, therefore, an upper-bound estimate of the cancer risk per unit dose and, for this reason, may overstate the magnitude of the risk. In addition, the use of CSFs in projecting excess individual cancer risk introduces uncertainty stemming from a number of factors, including

- Limited understanding of cancer biology
- Variability in the response of animal models
- Differential response in animal models versus humans
- Difference between animal dosing protocols and human exposure patterns.

A key step in CSF development is high- to low-dose extrapolation. Depending on the model used to fit the data, extrapolations to the low-dose range can vary by several orders of magnitude, reflecting the potential uncertainty associated with the CSF. In addition, uncertainty is introduced in the analysis of dioxins, furans, and PCBs because the TEF scheme is used to relate the toxicity of all congeners to the toxicity of TCDD. There are no other data for use for congener-specific toxicity endpoints.

Human Health Benchmarks and Children. EPA recognizes that significant uncertainties exist regarding the estimation of lifetime cancer risks in children. EPA estimated the risk of developing cancer from the estimated LADD and the slope of the dose-response curve. A CSF is derived from either human or animal data and is taken as the upper bound on the slope of the dose-response curve in the low-dose region, generally assumed to be linear, expressed as a lifetime excess cancer risk per unit exposure. Individuals exposed to carcinogens in the first few years of life may be at increased risk of developing cancer.

8.2.3 Variable Uncertainty

Variable uncertainty occurs when (1) there is a lack of data about the values used in the equations, (2) the data that are available are not representative of the particular instance being modeled, or (3) variable values cannot be measured precisely and/or accurately because of limitations in measurement technology. Random, or sample, errors are a common source of parameter uncertainty that is especially critical for small sample sizes. More difficult to recognize are nonrandom or systematic errors that result from bias in sampling, experimental design, or choice of assumptions.

8.2.3.1 <u>Agricultural Field Variables</u>. Source characterization required making assumptions about agricultural practices on farms where biosolids may be applied. There is much uncertainty associated with the actual practices employed on farms where biosolids are actually employed. It is not known what area is amended with biosolids and what crops or animals are raised on the amended land or what specific practices are employed. The variables used in this analysis represent the data available on potential agricultural practices. For this reason, substantial uncertainty concerning the variable values for agricultural practices remains.

8.2.3.2 <u>Watershed Universal Soil Loss Equation (USLE) Variables</u>. A combination of region-specific and national default variables was used along with USLE to model soil erosion losses from watersheds to waterbodies. The USLE calculations are particularly sensitive to site-specific values; thus, uncertainty is associated with using regional and national parameter values. Many of the ULSE parameters were based on the regional meteorological and regional soil data used in other parts of the analysis. These include soil erodibility factor (K), rainfall erosivity, and slope. Other variables were based on national default values (e.g., cover and management factors) or default relationships with other factors (e.g., length was determined as a function of slope).

8.2.3.3 <u>Exposure Factors</u>. For most exposure factors addressed, data analyses involved fitting distributions of data summaries from the EFH (U.S. EPA, 1997a, b, c), in most cases by fitting distributions to selected percentiles. It is assumed that little information is lost by fitting

to percentiles versus fitting to raw data. However, some believe that such analyses should always be based on raw data, synthesizing all credible sources.

Three standard two-parameter probability statistical distributions (gamma, lognormal, and Weibull) were used for this analysis. These distributions are special cases of a three-parameter distribution (generalized gamma) that allows for a likelihood ratio test of the fit of the two-parameter models. Other statistical distributions are possible (e.g., U.S. EPA, 2000), but the technique used in this analysis offered considerable improvement over using a lognormal model in all cases, and it was appropriate for this analysis. In support of this conclusion, a comparison of results showed that the three-parameter generalized gamma distribution did not significantly improve on goodness of fit over the two-parameter distributional forms in 58 of 59 cases at the 5 percent level of significance.

Although they offer significant improvement in objectivity over visual estimation, goodness-of-fit tests used to determine which statistical distribution to use for a particular parameter are themselves subject to some uncertainty that should be considered in their application to exposure factors. One area of concern is uncertainty about how the survey statistics in the EFH (U.S. EPA, 1997a, b, c) were calculated. All of the statistics that have been used to assess goodness of fit assume a random sample, which may or may not be a valid assumption for EFH data. Specifically, many of the EFH data sources are surveys that, in many cases, do not involve purely random samples. Rather, they use clustering and stratification, primarily for economic reasons.

8.3 References

- U.S. EPA (Environmental Protection Agency). 1994. *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry*. EPA/600/8-90-066F. Washington, DC: U.S. Government Printing Office.
- U.S. EPA (Environmental Protection Agency). 1997a. *Exposure Factors Handbook, Volume I, General Factors*. EPA/600/P-95/002Fa. Washington, DC: U.S. Government Printing Office.
- U.S. EPA (Environmental Protection Agency). 1997b. *Exposure Factors Handbook, Volume II, Food Ingestion Factors*. EPA/600/P-95/002Fa. Washington, DC: U.S. Government Printing Office.
- U.S. EPA (Environmental Protection Agency). 1997c. *Exposure Factors Handbook, Volume III, Activity Factors*. EPA/600/P-95/002Fa. Washington, DC: U.S. Government Printing Office.
- U.S. EPA (Environmental Protection Agency). 1999. *Risk Assessment Guidance for Superfund, Volume 3, Part A, Process for Conducting Probabilistic Risk Assessment.* EPA Office of Solid Waste and Emergency Response.

U.S. Environmental Protection Agency (EPA). 2000. *Options for Development of Parametric Probability Distributions for Exposure Factors*. EPA/600/R-00/058. Washington, DC: U.S. Government Printing Office.

9.0 Screening Ecological Risk Assessment of Dioxins and PCBs in Land-Applied Biosolids

9.1 Introduction

This section describes the screening ecological risk assessment (SERA) that was performed to investigate the potential for adverse ecological effects from dioxins in land-applied biosolids. Screening-level ecological risk assessments are designed to provide a high level of confidence in determining a low probability of adverse effects to ecological receptors (U.S. EPA, 2001a). The SERA was not designed or intended to provide definitive estimates of risk; rather, the SERA provides insight into the potential for ecological risk. The SERA was designed to be consistent with EPA's Guidelines for Ecological Risk Assessment (U.S. EPA, 1998).

The SERA was conducted in two phases. In Phase 1, an initial screen was conducted to determine whether the dioxin concentrations in land-applied biosolids warranted an ecological risk assessment. The purpose of this screen was not to identify ecological receptors for further analysis; rather, the screen was designed to provide a simple, efficient indicator of the potential for adverse ecological effects at a high-end exposure. In Phase 2, a deterministic screening assessment was performed on a representative suite of ecological receptors that are typical of terrestrial and waterbody margin habitats.

The risk metric chosen for the SERA is the hazard quotient (HQ), the ratio of the exposure (in units of dose) to an ecological benchmark. Media concentrations (e.g., sediment, soil) from the human health risk assessment modeling simulations were used to predict exposure doses, and HQs were calculated on a TCDD TEQ basis. Calculation of HQs has a binary outcome: either the dose is below the protective ecological benchmark (HQ<1), or it is equal to or greater than the benchmark (HQ \ge 1). However, the screening HQ results should be interpreted within the context of the SERA design. For example, a high level of conservatism built into the SERA may support a conclusion of low potential for adverse ecological effects at an HQ between 0.1 and 1. Conversely, an HQ that is within a factor of 10 of the target HQ of 1.0 may provide sufficient justification for further analysis. The HQ results presented in this section are intended only to provide useful information for the decision-making process; the screening HQ results cannot be used to predict the probability or ecological significance of adverse effects.

Sections 9.2, 9.3, and 9.4 describe the SERA methodology as suggested in EPA's guidelines: problem formulation, analysis, and risk characterization.

9.2 **Problem Formulation**

The problem formulation process consists of (1) selection of assessment endpoints, (2) development of a conceptual model, and (3) development of an analysis plan (U.S. EPA, 1998). The selection of endpoints and development of the conceptual model are discussed in Sections 9.2.1 and 9.2.2, and development of the analysis plan is briefly described in 9.2.3.

9.2.1 Assessment Endpoint Selection

Assessment endpoints are defined as "explicit expressions of the actual environmental value that is to be protected" (U.S. EPA, 1998). The assessment endpoints serve as critical links between the ecological risk assessment and the management goal. For the biosolids SERA, the management goal was the following:

• Evaluate and characterize the potential for adverse ecological effects on wildlife that may be affected by land application of biosolids.

The assessment endpoints (the values to be protected) are viable wildlife populations and ecological communities. Specifically, the assessment endpoints were defined in terms of characteristics relevant to population viability—reproductive and developmental success—for which toxicity data were identified for the mammalian and avian species¹ included in the SERA. However, population-level risks were not directly assessed. A population-level assessment would require information on a variety of parameters, such as survival, fecundity, immigration, and predator-prey relations. Although models were identified to evaluate the effects of chemical stressors on wildlife species populations, the data needed to support them are not readily available for a national-level assessment, and such an approach was considered beyond the scope of this screening-level analysis. Consequently, the SERA evaluated organism-level endpoints considered highly relevant to the variability of wildlife populations.

Although the assessment endpoints shown in Table 9-1 include both species populations and communities, the ecotoxicological data were considered insufficient to develop environmental quality criteria relevant to the soil, sediment, and surface water communities. The receptors selected under each assessment endpoint reflect the desire to represent:

- Significance of the receptor to the ecosystem
- Position of the receptor along a continuum of trophic levels
- Susceptibility of the receptor through media and food exposure pathways
- Toxicological sensitivity of the receptor to dioxins and PCBs.

¹ Populations of reptiles and amphibians (i.e., herpetofauna) were not included in the SERA because suitable data on chronic toxicity could not be identified.

Table 9-1. Assessment Endpoints for the Biosolids SERA

Ecological Significance	Assessment Endpoint	Representative Receptors	Characteristic(s)	Measure of Effect
 Includes upper trophic level consumers Socially valued (e.g., endangered species) Top recipients of bioaccumulative 	Viable mammalian wildlife populations	e.g., deer mouse, meadow vole, red fox	Reproductive and developmental success	Chronic or subchronic NOAEL or MATL for developmental and reproductive effects
 Represents species with large foraging ranges Represents species with longer life spans 	Viable avian wildlife populations	e.g., red-tailed hawk, belted kingfisher	Reproductive and developmental success	Chronic or subchronic NOAEL or MATL for developmental and reproductive effects
	Viable amphibian and reptile wildlife populations	e.g., green frog, eastern newt, northern water snake, eastern box turtle	Reproductive and developmental success	Chronic or subchronic NOAEL or MATL for developmental and reproductive effects
 Represents base food web in terrestrial systems Habitat vital to decomposers and soil aerators Proper soil community function related to nutrient cycling 	Sustainable soil community structure and function	e.g., nematodes, soils mites, springtails, annelids, arthropods	Growth, survival, and reproductive success	Soil quality criteria
 Highly exposed receptors from constant contact with contaminated media Act as vectors to transfer contaminants to terrestrial species 	Sustainable aquatic community structure and function	e.g., fish (salmonids), aquatic invertebrates (daphnids)	Growth, survival, and reproductive success	Water quality criteria
 Provide habitat for reproductive life stages (e.g., eggs, larval forms) Habitat for key invertebrate species Act to process nutrients and decompose organic matter 	Sustainable benthic community structure and function	e.g., protozoa, flat worms, ostracods	Growth, survival, reproductive success	Sediment quality criteria

NOAEL - No observed adverse effects level MATL - Maximum allowable toxicant level This approach assumes that, if individuals are protected from adverse reproductive and developmental effects associated with dioxins and PCBs in biosolids, protection at a higher level of organization (in this case, wildlife populations) may be inferred. In the case of mammals and birds, studies identifying reproductive and developmental effects in laboratory species were extrapolated to benchmarks for representative wildlife species. As noted above, these endpoints do not reflect true population benchmarks because they do not consider other factors relevant to population dynamics, such as emigration, immigration, carrying capacity, and predator-prey interactions. Nevertheless, the selection of endpoints on reproductive fitness allows for inference on the possible impacts on wildlife populations.

9.2.2 Development of Conceptual Model

The conceptual model for the assessment describes the exposure scenarios and the relationships between the ecological receptors and the stressors of concern. The conceptual model is developed through analysis of the (1) environmental behavior of constituents, (2)identification of exposure pathways of concern, (3) identification of habitats and receptors of concern, and (4) characterization of ecological effects. Therefore, the conceptual model integrates information related to the constituents to be modeled (e.g., environmental behavior such as bioaccumulation), ecotoxicological effects data for constituents of concern, receptors and ecosystems potentially at risk, and relevant pathways of exposure. Because land application of biosolids may occur throughout the United States, virtually any type of ecosystem and ecological receptor may be exposed to dioxins and PCBs in biosolids. For screening purposes, the conceptual model included ecological receptors that are representative of either waterbody margin habitats in freshwater systems (e.g., streams, lakes, or ponds) or terrestrial habitats (e.g., forests, crop lands). Previous sections of this report provide extensive details on the agricultural application of biosolids and how these exposure scenarios are developed. To avoid duplicating the discussions in Sections 3.0 through 5.0, the description of the conceptual model is intentionally brief with respect to the exposure scenario, application rates of biosolids, and other pertinent information on the site layout. As appropriate, references to previous sections have been provided to allow the reader to quickly identify additional details on the fate and transport modeling of chemical constituents in biosolids.

Because dioxins and PCBs are persistent, bioaccumulative organics, the conceptual model includes both direct and indirect (i.e., food chain) exposures for ecological receptors. Constituents released from an agricultural application of biosolids may be transported to surface waterbodies through erosion and runoff and, frequently, are buried in the bed sediment. In addition, constituents may be dispersed and deposited directly onto plants, soils, and surface waterbodies by wet and dry deposition mechanisms. Soils and sediments have been shown to be sinks for environmental releases of dioxin and dioxin-like compounds; therefore, direct contact with these contaminated media may pose potential risks to ecological receptors (e.g., benthic dwellers). The dioxin-like constituents in biosolids have been shown to bioaccumulate in the food chain, and receptors in higher trophic levels may be particularly at risk through food chain exposures. Figure 9-1 presents a graphic representation of the conceptual model.



Figure 9-1. Conceptual model for the biosolids SERA.

9.2.2.1 Environmental Behavior of Chemicals of Concern. The SERA addresses the 29 dioxin and PCB congeners modeled in the human health risk assessment, shown in Table 9-2.

Polychlorinated dibenzodioxins		
CAS #	Congener	
1746016	2,3,7,8-TCDD	
40321764	1,2,3,7,8- PeCDD	
39227286	1,2,3,4,7,8-HxCDD	
19408743	1,2,3,7,8,9-HxCDD	
35822469	1,2,3,6,7,8-HxCDD	
3268879	1,2,3,4,6,7,8-HpCDD	
51207319	1,2,3,4,6,7,8,9-OCDD	

Table 9-2. Dioxin Congeners Assessed in the SERA

(continued)

Polychlorinated dibenzofurans			
	CAS #	Congener	
51207319		2,3,7,8-TCDF	
57117416		1,2,3,7,8-PeCDF	
57117314		2,3,4,7,8-PeCDF	
	70648269	1,2,3,4,7,8-HxCDF	
	57117449	1,2,3,7,8,9-HxCDF	
	72918219	1,2,3,6,7,8-HxCDF	
	60851345	2,3,4,6,7,8-HxCDF	
	67562394	1,2,3,4,6,7,8-HpCDF	
	55673897	1,2,3,4,7,8,9-HpCDF	
	39001020	1,2,3,4,6,7,8,9-OCDF	
	Polychlorinated biphenyls		
IUPAC #	CAS #	Structure	
77	32508133		
	32398133	3,3',4,4-TCB	
81	70362504	3,3',4,4-TCB 3,4,4',5-TCB	
81 105	70362504 32598144	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB	
81 105 114	70362504 32598144 74472370	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB	
81 105 114 118	32598133 70362504 32598144 74472370 31508006	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB	
81 105 114 118 123	32598133 70362504 32598144 74472370 31508006 65510443	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB 2',3,4,4',5-PeCB	
81 105 114 118 123 126	32598133 70362504 32598144 74472370 31508006 65510443 57465288	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB 2,3',4,4',5-PeCB 3,3',4,4',5-PeCB	
81 105 114 118 123 126 156	32398133 70362504 32598144 74472370 31508006 65510443 57465288 38380084	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB 2,3,4,4',5-PeCB 3,3',4,4',5-PeCB 2,3,3',4,4',5-HxCB*	
81 105 114 118 123 126 156 157	32598133 70362504 32598144 74472370 31508006 65510443 57465288 38380084 52663726	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB 2,3,4,4',5-PeCB 3,3',4,4',5-PeCB 2,3,3',4,4',5-HxCB* 2,3,3',4,4',5'-HxCB*	
81 105 114 118 123 126 156 157 167	32598133 70362504 32598144 74472370 31508006 65510443 57465288 38380084 52663726 32774166	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB 2,3,4,4',5-PeCB 3,3',4,4',5-PeCB 2,3,3',4,4',5-HxCB* 2,3,3',4,4',5'-HxCB* 2,3',4,4',5,5'-HxCB	
81 105 114 118 123 126 156 157 167 167	32598133 70362504 32598144 74472370 31508006 65510443 57465288 38380084 52663726 32774166 39635319	3,3',4,4'-TCB 3,4,4',5-TCB 2,3,3',4,4'-PeCB 2,3,4,4',5-PeCB 2,3',4,4',5-PeCB 2,3,4,4',5-PeCB 3,3',4,4',5-PeCB 2,3,3',4,4',5-HxCB* 2,3,3',4,4',5,5'-HxCB 3,3',4,4',5,5'-HxCB	

Table 9-2. (continued)

 \ast These two congeners are co-eluting and are therefore modeled as a single congener.

Overall, the mobility and fate of dioxins and PCBs is closely tied to the movement of sediments, particulates, and soils via erosion. For example, in surface water, dioxin is associated primarily with suspended organic matter, which eventually settles into sediments. Concentrations in sediments range from 6.0E-05 to 7.6E-03 mg/kg sediment, with the latter being related to sediments in areas of high industrial activity. In addition to the movement of dioxin via abiotic means, dioxin is also mobile through biotic means. Concentrations in fish range from below detection, 5.0E-07, to 1.0E-04 mg/kg fish tissue (whole body, wet weight). Over time, concentrations in sediment and biota decrease as dioxins and PCBs are slowly metabolized or transported elsewhere through sediment movement. Similar chemical behavior is observed in terrestrial systems; however, dioxin is adsorbed to organic content in the soil and is somewhat less mobile (Eisler, 1986). The accumulation of dioxins from the soil into plants has been shown to be negligible (U.S. EPA, 2000).

The environmental behavior of chemical contaminants in biosolids is, to some degree, determined by application and management practices. For example, concentration profiles for dioxins and PCBs applied on a daily basis would likely be very different than the profiles developed for biannual applications. In Section 3.1.3, Figure 3-1 is accompanied by a detailed explanation of the conceptual site model used in the model simulations, and Section 4.0 provides a complete characterization of agriculturally applied biosolids, from the physical characteristics of the biosolids to the properties of the environmental setting (e.g., soil properties). Because biosolids applications occur nationwide, the model simulations produce distributions of media concentrations that capture the variability in climate, soil, and agricultural practices across the contiguous United States. To support the modeling simulations described in Sections 4.0 and 5.0, the 48 states were subdivided into 41 climatic regions (see Figure 3-2), and each region was represented by climate data from any reporting meteorological station within the bounds of the region. This implicitly assumes that the meteorological conditions in any region are sufficiently uniform so as to be represented by a single station. As described in Section 4.3.2, these geographic regions were also used as the basis for identifying a representative farm size and a distribution of soil types on the farm. For convenience, several key assumptions on common agricultural practices are presented below.

- Biosolids are applied at a rate of 5 to 10 metric tons per hectare per application (application rates for biosolids were assumed to be uniform nationwide).
- Applications occur once every 2 years.
- Application continues for up to 40 years (20 applications).
- Crop land is tilled to a depth of 20 cm multiple times during the year.
- Pasture land is not tilled; thus, biosolids are assumed to be incorporated into only the top 2 cm of soil.

9.2.2.2 <u>Habitats Potentially at Risk</u>. For agricultural application of biosolids, the SERA addresses two generalized habitat types: a terrestrial habitat and a water body margin habitat associated with freshwater systems (e.g., streams, ponds). These habitat types provide a

framework for identifying exposure pathways of concern and define the context for receptor species selection.

The terrestrial habitat consists of crop fields and pastures where biosolids are applied. The conceptual layout described in detail in Section 4.3.1 is based on the assumption that farmers apply biosolids to adjacent crop fields and pasture. Thus, ecological receptors may be exposed to contaminants in plants, prey, and soil in the crop field and pasture by feeding and foraging in these areas. The waterbody margin habitat consists of nearby surface waterbodies and their adjacent terrestrial margin. The waterbodies receive chemical loads through runoff and erosion from the agricultural field. The buffer area shown in Figure 3-1 is located between the fields and a nearby surface waterbody; for the purposes of the SERA, it constitutes the terrestrial margin associated with the waterbody. Receptors may be exposed to terrestrial plants and prey and to soil in the buffer area as part of the margin habitat; in addition, receptors may take fish, other aquatic biota, sediment, and drinking water from the receiving waterbody.

In summary, the representative terrestrial and margin habitats in the SERA are intended to capture the key elements of freshwater and agricultural field systems. However, the actual exposures received by wildlife will be strongly influenced by a variety of habitat characteristics. In margin habitats, the waterbody size, flow rate, bed sediment composition, and the presence of aquatic vegetation will significantly affect the ecological exposures. Similarly, in terrestrial systems, factors such as regional location, vegetative cover type, soil characteristics, and adequacy of food sources will determine the applied dose to wildlife. Although these habitat characteristics are not explicitly addressed in the SERA, receptors assigned to the two representative habitats are intended to address significant exposure pathways and represent scenarios appropriate for a screening-level analysis.

Figures 9-2 and 9-3 show simplified food webs for exposure in terrestrial and margin habitats. The trophic levels and feeding guilds shown in the figures are defined as follows:

	Trophic Levels	Feeding Guilds
T1:	Species is prey to other receptors, but is not a predator.	Herbivore: Consumes only plant matter.
T2:	Species is both predator and prey to other receptors.	Omnivore: Can be expected to consume both plant and animal matter, although can at times feed exclusively on one or the other.
T3:	Top predators; species are generally assumed not to be prey to other receptors.	Carnivore: Feeds exclusively on animals.



Figure 9-2. Terrestrial food web, including example receptors.

9-9

Terrestrial Receptors Dependent on Aquatic Habitats

Simplified Aquatic Food Web



Figure 9-3. Interface between terrestrial receptors and aquatic food web, including example receptors.

The food webs were developed based on generally accepted concepts about food webs and natural community dynamics (Anderson, 1997; Begon and Mortimer, 1981; Caduto, 1990; Davis and Simon, 1995; Kadlec and Knight, 1996; Sample et al., 1997; Schoener, 1989; Schoenly and Cohen, 1991; Suter, 1993; Tanner, 1978; U.S. EPA, 1993a, 1994). Species-specific information was taken from the references listed in Appendix L, Table L-5. The food webs serve two purposes: they illustrate potential exposure pathways, and they facilitate the selection of receptor species for each habitat type.

9.2.2.3 <u>Selection of Receptors of Concern</u>. Ecological receptors typical of the terrestrial and margin habitats were considered on the basis of (1) trophic levels, taxa, and feeding guilds (e.g., herbivores, carnivores), (2) potential for exposure to dioxins in land-applied biosolids, (3) toxicological sensitivity and (4) geographical distribution (e.g., avoid narrow ecological niches). Receptors with a high potential for exposure were defined as those documented to feed and forage in agricultural fields or in margin habitats. Because dioxin congeners are known to bioaccumulate in fish, small mammals, and soil and sediment invertebrates, receptors whose diets include these items were also assumed to have a high potential for exposure.

Of the representative receptors considered for inclusion in the SERA, adequate ecotoxicological data were identified only for mammals and birds. Therefore, the only assessment endpoints in Table 9-1 that were quantitatively screened were mammalian and avian wildlife populations. The primary exposure route of interest for mammals and birds is ingestion, and exposure is expressed in terms of ingestion dose. The wildlife species included in the SERA are shown in Table 9-3. The SERA did not include aquatic and terrestrial plants, aquatic invertebrates, or amphibians, because of their demonstrated tolerance to TCDD in laboratory studies (U.S. EPA, 2001b).

The representative species selected for the Phase 2 analysis were not limited to keystone or indicator species. Indicator species imply that a level of significance to total ecosystem structure or function can be ascertained; however, in a screening-level assessment, this can not be determined with a high level of confidence. The receptors were selected because (1) these species represent a full range of trophic levels and feeding guilds relevant to dioxin exposures through the food web; (2) life-history data, such as dietary habitats and distribution in the contiguous United States were available; and (3) toxicological data were identified, suggesting that the species was sensitive to dioxins and PCBs (e.g., mammals are highly sensitive to dioxins). Although this approach tends to "overrepresent" certain taxa, such as *Mustella*, the SERA was intended to provide information across a number of receptors, not just the most highly exposed and sensitive species.

9.2.2.4 <u>Identification of Exposure Pathways of Concern</u>. Dioxin and PCB congeners are persistent, bioaccumulative, and hydrophobic compounds that have been shown to biomagnify in the food web. Typically, these congeners are stored in the fat tissues of organisms and are minimally metabolized over time. Consequently, animals foraging in the terrestrial and

Species	Scientific Name	Feeding Guild ¹	Trophic Level ²	Habitats
American kestrel	Falco sparverius	С	T2	terrestrial
American robin	Turdus migratorius	0	T2	terrestrial
American woodcock	Scolopax minor	0	T2	terrestrial
Bald eagle	Haliaeetus leucocephalus	С	Т3	margin
Beaver	Castor canadensis	Н	T1	margin
Belted kingfisher	Ceryle alcyon	0	T2	margin
Black bear	Ursus americanus	0	Т3	terrestrial
Canada goose	Branta canadensis	Н	T1	terrestrial
Cooper's hawk	Accipiter cooperi	С	T3	terrestrial
Coyote	Canis latrans	0	Т3	terrestrial
Deer mouse	Peromyscus maniculatus	0	T2	terrestrial
Eastern cottontail rabbit	Sylvilagus floridanus	Н	T1	terrestrial
Great blue heron	Ardea herodias	Ο	T2	margin
Green heron	Butorides virescens	Ο	T2	margin
Herring gull	Larus argentatus	0	T2	margin
Least weasel	Mustela nivalis	С	T2	terrestrial
Lesser scaup	Aythya affinis	Ο	T2	margin
Little brown bat	Myotis lucifugus	Ι	T2	terrestrial
Long-tailed weasel	Mustela frenata	С	T2	terrestrial
Mallard	Anas platyrhynchos	0	T2	margin
Meadow vole	Microtus pennsylvanicus	Н	T1	terrestrial
Mink	Mustela vison	С	T2	margin
Muskrat	Ondatra zibethicus	Н	T1	margin
Northern bobwhite	Colinus virginianus	0	T2	terrestrial
Osprey	Pandion haliaetus	С	T3	margin
Prairie vole	Microtus ochrogaster	Н	T1	terrestrial

Table 9-3.	Wildlife Rece	ptors for	the Bios	solids SERA
------------	---------------	-----------	----------	-------------

(continued)

Species	Scientific Name	Feeding Guild ¹	Trophic Level ²	Habitats
Raccoon	Procyon lotor	0	T2	terrestrial, margin
Red fox	Vulpes vulpes	0	Т3	terrestrial
Red-tailed hawk	Buteo jamaicensis	С	Т3	terrestrial
River otter	Lutra canadensis	С	T2	margin
Short-tailed shrew	Blarina brevicauda	0	T2	terrestrial
Short-tailed weasel	Mustela erminea	С	T2	terrestrial
Tree swallow	Tachycineta bicolor	0	T2	terrestrial
Western meadowlark	Sturnella neglecta	О	T2	terrestrial
White-tailed deer	Odocoileus virginianus	Н	T1	terrestrial

 Table 9-3. (continued)

¹ Feeding guild: C = carnivore, H = herbivore, I = insectivore, O = omnivore.

² Trophic level: T1 = prey, not a predator; T2 = both a predator and prey; T3 = a top predator, not prey.

margin habitats may be exposed through the food chain, as well as through direct ingestion of contaminated soil, surface water, and sediment. Inhalation was not considered to be a significant route of exposure for dioxins and PCBs and was not included in the SERA.

Receptor species are exposed through the ingestion of

- Aquatic prey, such as fish, mussels, and snails
- Terrestrial prey from the waterbody margin or field, such as vegetation and small mammals
- Soil from the contaminated field or buffer
- Water from contaminated waterbodies
- Sediment from contaminated waterbodies.

In addition, receptors that live in close contact with contaminated media (e.g., benthic invertebrates) may receive significant exposures to dioxins and PCBs. The primary routes of exposure for these receptors include ingestion of contaminated plants and prey, as well as direct contact. However, as indicated in Section 9.2.1, sufficient toxicological data were not identified to develop environmental quality criteria for soil, sediment, or surface water. As a result, direct contact with contaminated media was not included in the SERA.

9.2.2.5 <u>Characterization of Ecological Effects</u>. As indicated in the previous section, the focus for the SERA is on mammalian and avian receptors. Therefore, the effects characterization in this section discusses the relevant studies reviewed in selecting the most appropriate toxicological data to develop the ecological benchmarks for mammals and birds.

The effects characterization is based on a review of recently published sources, other literature citations, and EPA publications. In particular, the *Dose-Response Assessment from Recently Published Research of the Toxicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds to Aquatic Wildlife-Laboratory Studies* was reviewed to identify appropriate benchmark studies (NCEA, 2001).

9.2.2.5.1 *Mammals.* TCDD exposures have been associated with a variety of reproductive and developmental effects in mammals. For example, Khera and Ruddick (1973) assessed the postnatal effect of TCDD on pregnant Wistar rats and observed a dose-related decrease in the average litter size and pup weight at birth in all but the 0.125 ug/kg-day dose. Bowman et al. (1989a, 1989b) studied the reproductive effects of Rhesus monkeys exposed to diets containing 5 ppt and 25 ppt TCDD for 7 and 24 months. The female monkeys exposed to 25 ppt had a significantly lower Index of Overall Reproductive Success (IORS), while the 5 ppt group did not differ from the control. Hochstein et al. (1988) administered TCDD dietary concentrations of 0, 0.001, 0.01, 0.1, 1.0, 10, and 100 ppb to mink for 125 days. While no significant adverse effects were observed on mink fed dietary concentrations of 0.1 ppb or less, mortality was noted in groups fed 1 and 10 ppb.

Murray et al. (1979) exposed three generations of Sprague-Dawley rats to diets containing 0, 0.001, 0.01, or 0.1 μ g TCDD/kg-day. At the 0.01 μ g/kg-day dose, Murray et al. (1979) observed no effect on fertility among the f_0 rats, but a significant reduction in fertility was observed among the f_1 and f_2 rats. Thus, through three successive generations, the reproductive capacity of rats ingesting TCDD was clearly affected at dose levels of 0.01 and 0.1 μ g/kg-day, but not at 0.001 μ g/kg-day. This study was selected for benchmark derivation because it consists of a multigenerational exposure scenario that demonstrates a clear dose-response for reproductive effects attributable to TCDD.

The 125-day test performed by Hochstein et al. (1988) was not considered appropriate for deriving a benchmark because the study was subchronic rather than chronic and the perceived endpoints focus more on mortality than reproductive effects. The Murray et al. (1979) study was chosen over the Khera and Ruddick study (as cited in U.S. EPA, 1995) because of a lower reported NOAEL for rats. The reproduction study by Bowman et al. (1989a, 1989b) on Rhesus monkeys (which produced a lower NOAEL) was not selected because the Murray et al. (1979) study incorporated a multigenerational exposure regime and contained stronger dose-response information.

9.2.2.5.2 *Birds.* TCDD toxicity has been demonstrated in the embryos of many bird species, including domestic chickens (Brunstrom and Lund, 1988), great blue herons (Hart et al., 1991), ring-necked pheasants (Nosek et al., 1993) and double-crested cormorants (Powell et al., 1997). Sublethal responses include subcutaneous edema (Hart et al., 1991), induction of hepatic microsomal ethoxyresorufin-*O*-dealkylase, depressed embryonic growth, brain asymmetry (Custer et al., 1997), short beaks, fatty liver, heart abnormalities, and poorly developed stomachs (Henshel et al., 1997). Egg mortality has also been found in many studies (e.g., Nosek et al., 1993; Powell et al., 1997). Exposure in these studies was usually by injection, either into the yolk, albumin, or air cell.

Effects on adult birds appear to have been much less studied. Nosek et al. (1992) injected ring-necked pheasants (*Phasianus colchicus*) with various doses of TCDD, once a week for 10 weeks. Mortality, egg production, and embryo mortality were recorded. Embryo mortality was increased by exposure of adults to TCDD, with 100 percent egg mortality at a cumulative dose of $10 \ \mu g \ kg^{-1}$ body weight. However, even at the highest dose, some eggs were produced. Adult mortality only occurred at the highest dose. The weekly dose to the pheasants for 10 weeks by intraperitoneal (ip) injection is at an equivalent rate of 0.14, 0.014, and 0.0014 μ g TCDD/kg-day (weekly dose was divided by 7 for the equivalent daily dose and adjusted for intake and body weight). Cumulative egg production was significantly reduced among pheasants exposed to 0.14 µg TCDD/kg-day, but not among those pheasants exposed to the two lower doses. Based the limited data on adult birds, the study by Nosek was considered to be the most appropriate benchmark study because the endpoint is highly relevant to population viability and because there is substantial support in EPA publications to use these data to evaluate ecological risks to birds. Assuming 100 percent absorption from ip injection, the ip exposure route may overestimate the absorption rate of TCDD via oral ingestion by a factor of 1 to 5 depending upon diet composition (Abt, 1993).

9.2.3 Analysis Plan

The analysis plan is the third critical product of the problem-formulation phase. In essence, the analysis plan provides a blueprint for evaluating the potential for adverse ecological effects for the assessment endpoints, receptors, and exposure pathways of concern. The analysis plan can be broken down into two sections: an exposure analysis and an ecological response analysis. As summarized in the introduction, the analysis consisted of a two-phased approach. Phase 1 was designed as a bounding analysis to assess the potential for ecological effects at highend exposures. Therefore, the exposure analysis is based on an evaluation of the 50^{th} and 90^{th} percentile, and maximum TEQ concentrations in biosolids. This phase was a highly conservative "trigger" that is based on the lowest available ecological benchmarks (i.e., NOAELs) and considers only a few highly exposed ecological receptors (e.g., American robin eating 100 percent diet of earthworms). Phase 2 was designed to provide a conservative screen of the potential hazard to an expanded list of mammalian and avian receptors intended to represent general terrestrial and waterbody margin habitats. For the exposure analysis, fate and transport algorithms described in detail in Section 5.0 provide an appropriate tool to estimate concentrations of dioxins and PCBs in the environmental media and terrestrial plants attributed to each habitat. For the ecological response analysis, the critical ecotoxicological data presented above are used to estimate less conservative ecological benchmarks (i.e., the geometric mean of the NOAEL and lowest observed adverse effects level (LOAEL)) for receptors representing the terrestrial and margin habitats. The exposure dose predicted using the modeled concentrations of dioxins and PCBs is compared to the benchmark to generate HQs for the entire list of mammals and birds in the generalized terrestrial and margin habitats.

9.3 Analysis Methods

The analysis phase of the SERA began with a highly conservative approach to determine whether any of the habitats, receptor categories, and exposure routes might be of concern. In this phase, the concentrations of dioxins and PCBs in biosolids were used to predict maximum possible exposure doses to several highly exposed receptors. Risks were estimated using conservative ecological benchmarks. The congener concentrations in biosolids were obtained from the NSSS 2001 (U.S. EPA, 2001c). As previously suggested, the intent of the Phase 1 "trigger" was simply to determine whether any further ecological risk analysis was warranted. Phase 2 consisted of a less conservative analysis based on representative exposure scenarios. The values and data sources used for the key input variables in the SERA are shown in Table 9-4 for both phases of the SERA.

The concentrations of dioxins and PCBs in various media were derived using a conceptual site model (see Figure 3-1) that simulates the application of biosolids based on the available data on biosolids management. The release, fate and transport, and estimation of media concentrations are presented in detail in Section 5.0. The following sections present the methods used in each phase of the analysis.

9.3.1 Phase 1 – Maximum Potential Risk

The Phase 1 analysis was a highly conservative assessment of the maximum possible risks for highly exposed receptors. Exposure was based on the 50th and 90th percentiles and maximum concentrations in biosolids; HQs were calculated using NOAELs for reproductive endpoints on individual organisms. The Phase 1 receptors shown in Table 9-5 were selected to maximize exposure. The ingestion route of exposure was assessed using receptor species whose diets consist largely of animals known to accumulate dioxins and PCBs from soil and sediments. These receptors are widely distributed across a large portion of the United States and, based on their diet, represent high-end exposures for birds and mammals in terrestrial and waterbody margin habitats.

Phase 1 risk estimates were generated using a simple spreadsheet model with the following steps. These steps are further discussed below.

- 1. For each receptor, select diet item to maximize exposure.
- 2. Calculate congener-specific concentrations in diet items.
- 3. Calculate congener-specific exposure dose for each receptor.
- 4. Apply TEFs to congener-specific dose estimates; sum to obtain TCDD TEQ.
- 5. Calculate HQ using receptor-specific TCDD benchmark.

9.3.1.1 <u>Development of Benchmarks for Phase 1</u>. For the SERA, exposure for all 29 congeners in the assessment was expressed in terms of 2,3,7,8-TCDD toxicity equivalence, and risk estimates were based on NOAELs. Benchmark studies for TCDD for mammals and birds were identified in the literature, and species-specific scaled benchmarks were calculated for each mammal and bird receptor. In identifying appropriate studies to develop benchmarks, several study selection criteria were adopted to ensure that (1) the endpoint was highly relevant to the viability of populations, (2) the dose-response information was sufficient to support development of a MATL, and (3) the study had been reviewed and approved by other EPA and federal agencies.</u>

Parameter	Phase 1 – Maximum Potential Risk	Phase 2 – Deterministic Screening
Congeners addressed	All	All
Receptors	Four highly exposed mammals and birds	35 representative mammals and birds
Dietary composition	Diets reflecting maximum exposure	Representative diets
Biouptake factors	Fixed values	Fixed values
Percentage of diet taken from contaminated area	100%	100%
Ecological benchmarks	NOAELs	MATL, calculated as the geometric means of NOAELs and LOAELs
Media concentrations used to estimate exposure	50 th and 90 th percentiles and maximum biosolids concentrations	90 th percentile modeled media concentrations (see Section 5.0)

Table 9-4. Values and Assumptions for the SERA

Table 9-5. Phase 1 Receptors¹

Receptor	Description	Pathway	Habitat
Osprey	Piscivorous bird that uses variety of margin habitats (e.g., wetlands, streams); diet consists entirely of fish.	Ingestion	Margin
American robin	Bird found in variety of terrestrial habitats; diet consists largely of earthworms.	Ingestion	Terrestrial
Belted kingfisher	Bird that primarily uses small ponds and streams; diet consists largely of fish.	Ingestion	Margin
Mink	Mammal that uses variety of margin habitats; diet consists largely of fish and invertebrates.	Ingestion	Margin

¹ Sources for species-specific dietary composition data are listed in Appendix L, Table L-5.

Using the benchmark study identified during the problem formulation (see Section 9.2.2.5), a scaled benchmark was calculated for each receptor species. For mammals, a scaling factor of 1/4 was used in accordance with the default methodology proposed by EPA for carcinogenicity assessments and reportable quantity documents for adjusting animal data to an equivalent human dose (U.S. EPA, 1992). For birds, research suggests that the cross-species scaling equation used for mammals is not appropriate (Mineau et al., 1996). Using a database that characterized acute toxicity of pesticides to avian receptors of various body weights, Mineau et al. (1996) concluded that applying mammalian scaling equations may not predict sufficiently protective doses for avian species. Mineau et al. recommended that a scaling factor of 1 provided a better dose estimate for birds. Therefore, a scaling factor of 1 was applied for avian receptors

Appendix L (page L-3) provides a detailed description of the development of ecological benchmarks. Table L-3 presents the benchmark values used in Phase 1.

9.3.1.2 <u>Estimating Exposure for Phase 1</u>. Exposure was estimated as an applied dose based on species-specific body weights, ingestion rates, and dietary composition. For Phase 1, exposure was maximized by assuming that

- Environmental concentrations are equal to biosolids concentrations
- Each receptor's diet consists entirely of a single food item that significantly bioaccumulates TCDD²
- The entire diet comes from contaminated media.

The dietary item for the ingestion pathway for each receptor is shown in Table 9-6.

9.3.1.2.1 Concentrations in Diet Items. The first step in estimating exposure dose is the calculation of congener-specific concentrations in each receptor's selected diet item. Concentrations in worms are a function of the soil concentration, the soil-to-worm bioaccumulation factor (BAF), and the congener-specific bioaccumulation equivalency factor (BEF), as given in Equation 9-1. Concentrations in terrestrial prey items (e.g., worms) were calculated using a BAF for TCDD. The soil-to-worm BAF for TCDD was identified in the literature; all BAFs and their respective sources are presented in Appendix L, Table L-1. Congener-specific BEFs were not available for soil-based uptake; therefore, a default BEF of 1 was assumed for all congeners.

² For example, although the robin's diet could consist of anywhere from less than 10 percent to 100 percent soil invertebrates, it was conservatively assumed that the robin's entire diet consists of earthworms for Phase 1.

Receptor	Diet Item Selected to Maximize Exposure
Osprey	Fish
American robin	Worms
Belted kingfisher	Fish
Mink	Fish

 Table 9-6.
 Selected Diet Items for Phase 1 Receptors

$$C_{worm\,i} = C_{soil\,i} \times BAF \times BEF_i \tag{9-1}$$

where

 $C_{\text{worm }i}$ = Total concentration of congener *i* in earthworms (mg/kg WW)

 $C_{soil i}$ = Soil concentration for congener *i* (mg/kg)

- BAF = Bioaccumulation factor for TCDD reflecting biouptake from soil into worms (mg/kg WW_{worm} / mg/kg soil)
- BEF_i = Bioaccumulation equivalence factor for congener *i* (unitless; default value of 1 was used)

The concentration in fish was calculated as a function of sediment concentration normalized for organic carbon and a congener-specific BSAFs for uptake of dioxins from sediment to fish, as shown in Equation 9-2. Congener-specific BSAFs were recommended in EPA's Draft Dioxin Reassessment Document (U.S. EPA, 2000); all BSAFs and their respective sources are presented in Appendix L, Table L-2.

$$C_{fishli} = C_{oc_{sadiment i}} \times BSAF_{li}$$
(9-2)

where

- $C_{\text{fish }li}$ = Lipid-based concentration of congener *i* in fish (mg/kg_l)
- $C_{oc_sediment i}$ = Sediment concentration normalized for organic carbon for congener *i* (mg/kg_{oc})
- $BSAF_{li}$ = Biota-sediment accumulation factor for congener *i* reflecting biouptake from sediment into fish lipid (kg_{oc}/kg_l).

9.3.1.2.2 *Receptor Dose.* As given by Equation 9-3, the ingestion exposure dose for Phase 1 receptors was calculated based on species-specific body weights and ingestion rates.

$$Dose_{i} = \frac{(IR_{diet} \times C_{diet i})}{BW}$$
(9-3)

where

Dose _i	=	Phase 1 exposure dose for congener i (mg/kg-d)
$C_{diet i}$	=	Concentration of congener i in fish or earthworms (mg/kg WW)
IR _{diet}	=	Species-specific ingestion rate (kg WW/d)
BW	=	Species-specific adult body weight (kg).

Body weights and ingestion rates were taken from the EPA's *Wildlife Exposure Factors Handbook* (U.S. EPA, 1993a). Average adult body weights and adult ingestion rates were used throughout the assessment.

Congener-specific exposure estimates were multiplied by their respective TCDD TEFs to derive a total dose for each receptor in terms of TCDD equivalence (i.e., TEQs). The summation of congener-specific doses is given by Equation 9-4:

$$Dose_{TEO} = \Sigma Dose_i \times TEF_i$$
 (9-4)

where

 $\begin{array}{rcl} \text{Dose}_{\text{TEQ}} & = & \text{Total dose in toxicity equivalence (mg/kg-d)} \\ \text{Dose}_i & = & \text{Dose for congener } i \ (\text{mg/kg-d}) \\ \text{TEF}_i & = & \text{Toxicity equivalence factor for congener } i. \end{array}$

The TEFs were taken from the WHO consensus TEFs for mammals, fish, and birds (U.S. EPA, 2001b), and are presented in Appendix L, Table L-9.

9.3.1.3 <u>**Risk Calculations for Phase 1**</u>. The risk metric for the Phase 1 screen was the HQ, calculated as the ratio of the TEQ exposure dose to the species-specific ecological benchmarks based on allometric scaling of the NOAELs. The exposure doses were calculated using the 50th, 90th, and maximum TEQ concentrations in biosolids. The toxicological studies used in benchmark derivation are described in Section 9.2.2.5 on effects characterization. The assumptions, scaling equations, and factors (i.e., factor of 1/4 for mammals and 1 for birds) are presented in Appendix L.

9.3.2 Phase 2 – Deterministic Screening

The second phase of the analysis was a deterministic screening of an expanded list of mammals and birds intended to represent a broad range of feeding guilds and trophic levels that are typical of the terrestrial and margin habitats. Phase 2 included all receptors shown in Table 9-3 and addressed receptors typical of crop fields, pastures, and surface waterbodies. The
dietary preferences for these receptors were based on information presented in the *Wildlife Exposure Factors Handbook* (U.S. EPA, 1993a), as well as information from the open literature. Consequently, the receptor diet is intended to reflect the documented variability in dietary preferences rather than to maximize exposure (as in Phase 1). The receptor-specific benchmarks were calculated as the geometric mean of the LOAEL and NOAEL—referred to as the MATL—to provide a less conservative benchmark for adverse ecological effects. The exposure and potential ecological risk (expressed as an HQ) is estimated for each receptor in each habitat type assuming that 100 percent of the diet originates on the contaminated area. The analysis consisted of the following major steps:

- 1. Assign representative receptors to each habitat type.
- 2. Establish dietary composition for each receptor based on habitat assignment.
- 3. Calculate congener-specific concentrations in each diet item.
- 4. Calculate total exposure dose for each congener for each receptor based on the ingestion of contaminated food and media.
- 5. Apply TEFs to congener-specific exposure doses to derive total TCDD equivalent exposure.
- 6. Calculate HQs for each receptor in each habitat.

9.3.2.1 <u>Development of Benchmarks for Phase 2</u>. Appropriate studies (e.g., on reproductive fitness) were identified in the literature and in EPA sources, and species-specific benchmarks were calculated using the scaling algorithms described in Appendix L. Although the same studies were used to derive benchmarks in both phases of the SERA, the Phase 2 screen used a less conservative measure of effect, the MATL. As the geometric mean between the NOAEL and LOAEL, the MATL is intended to represent a de minimis level of effect for a wildlife species population. Because the benchmarks are based on effects to individual organisms, a less conservative measure was considered appropriate for the assessment endpoint of population viability. A NOAEL is highly conservative in that it suggests that any toxicological response to a chemical stressor is considered unacceptable. Given the conservative nature of the Phase 2 screen (e.g., 100 percent of diet is contaminated), making inferences about a wildlife population based on a NOAEL for individual organisms would have been overly conservative and inconsistent with management goals for the SERA. Appendix L includes a detailed description of the benchmark development methods and presents the species-specific benchmark values used in Phase 2.

9.3.2.2 <u>Characterization of Exposure</u>. The 90th percentile congener-specific concentrations in soil, sediment, surface water and terrestrial plants were used to calculate exposure doses. The 90th percentile concentrations were derived from the fate and transport modeling described in Section 5.0 by (1) adjusting the concentrations predicted by the model by the TEFs for mammals and birds, respectively, (2) arranging the TEQ concentrations for mammals and birds in rank order by the soil concentration in the field and pasture for terrestrial

habitats, and the sediment concentration for margin habitats, and (3) selecting the 90th percentile set of concentrations based on the TEQ rank order for mammals and birds, respectively, for the terrestrial habitat (driven by soil TEQ concentration) and the margin habitat (driven by sediment concentration).

The concentration profiles (for media and plants) generated in the model simulations are maximum annual average concentrations. To derive these concentrations, the source model (see Section 5.1) generated 3000 Monte Carlo realizations of a 200-year time series, and the maximum annual average concentration was picked off of these distributions and rank ordered as described in the preceding paragraph. Consequently, "the 90th percentile" represents the 90th percentile from a distribution of maximum annual average concentrations and provides a conservative upper bound of the modeled concentrations.

Exposures were calculated for receptors assigned to the terrestrial (i.e., field/pasture) and margin (i.e., pond/lake/stream) habitats depending on foraging and feeding habits indicated in the ecological exposure factor database. Table 9-7 presents the list of receptors according to their feeding guild, trophic level, and habitat that were evaluated in the Phase 2 screen. Appendix L, Tables L-4 and L-5 show the data sources used for habitat assignments, as well as other exposure factor data for each receptor.

9.3.2.2.1 Estimation of Exposure Dose. Exposure doses were estimated in Phase 2 using the same basic approach that was used in Phase 1; however, the receptor diet in Phase 2 was constructed from the exposure factor database, and modeled concentrations in environmental media and terrestrial plants were used rather than the congener concentrations in biosolids. Ingestion exposure doses were calculated in three steps: (1) development of species-specific diets, (2) calculation of concentrations in each category of food (e.g., vegetation, small mammals, small birds), and (3) summation of total exposure dose. For the terrestrial habitat, incidental ingestion of soil (e.g., associated with the ingestion of terrestrial prey, preening, and other behaviors) was assumed to come from the agricultural field and pasture. For the margin habitat, the incidental ingestion of sediment—rather than soil—was evaluated because wildlife assigned to this habitat consume primarily aquatic biota (e.g., fish, sediment invertebrates). These steps are described in the following sections.

Receptor Diets

Dietary composition for Phase 2 was based on species-specific data on foraging and feeding behavior and reflected a year-round adult diet. The receptor diets were constructed to represent variability in feeding habits, rather than to artificially maximize exposure using the range (defined by the minimum and maximum) for each item in the diet. Diet items are grouped in 17 categories, including different types of vegetation (e.g., fruits, forage, grain, roots) and several categories of prey (e.g., small birds, small mammals, invertebrates, fish). For example, the American robin's dietary percentage ranges are as follows (Terres, 1980; U.S. EPA, 1993a; Stokes and Stokes, 1996):

Species	Feeding Guild	Trophic Level	Terrestrial Habitat	Margin Habitat
American kestrel	С	T2	•	
American robin	0	T2	•	
American woodcock	0	T2	•	
Bald eagle	С	Т3		•
Beaver	Н	T1		•
Belted kingfisher	0	T2		•
Black bear	0	Т3	•	
Canada goose	Н	T1	•	
Cooper's hawk	С	Т3	•	
Coyote	0	Т3	•	
Deer mouse	0	T2	•	
Eastern cottontail rabbit	Н	T1	•	
Great blue heron	0	T2		•
Green heron	0	T2		•
Herring gull	0	T2		•
Least weasel	С	T2	•	
Lesser scaup	0	T2		•
Little brown bat	Ι	T2	•	
Long-tailed weasel	С	T2	•	
Mallard	0	T2		•
Meadow vole	Н	T1	•	
Mink	С	T2		•
Muskrat	Н	T1		•
Northern bobwhite	0	T2	•	
Osprey	С	Т3		•

Table 9-7. Receptors Evaluated in Phase A	Table 9-7.	Receptors	Evaluated	in	Phase	2
---	------------	-----------	------------------	----	-------	---

(continued)

Species	Feeding Guild	Trophic Level	Terrestrial Habitat	Margin Habitat
Prairie vole	Н	T1	●	
Raccoon	0	T2	●	•
Red fox	0	Т3	●	
Red-tailed hawk	С	Т3	●	
River otter	С	T2		•
Short-tailed shrew	0	T2	•	
Short-tailed weasel	С	T2	●	
Tree swallow	0	T2	●	
Western meadowlark	0	T2	•	
White-tailed deer	Н	T1	•	

Table 9-7. (continued)

Feeding guild: C = carnivore, H = herbivore, I = insectivore, O = omnivore.

Trophic level: T1 = prey, not a predator; T2 = both a predator and prey; T3 = a top predator, not prey.

Diet Item	Dietary Percentage Range
Soil invertebrates (other than earthworms)	8 to 93
Fruits	7 to 92
Earthworms	15 to 27
Forage	0 to 24

For the Phase 2 analysis, each receptor's diet was constructed using the midpoint of dietary percentages for each diet item, beginning with the item with highest midpoint value and proceeding through the diet items until a full diet (100 percent) was accumulated. Thus, the robin's diet would consist of 50.5 percent soil invertebrates and 49.5 percent fruits, based on the following dietary percentage midpoints:

Diet Item	Dietary Percentage Midpoint
Soil invertebrates	50.5
Fruits	49.5
Worms	21
Forage	12

The dietary composition used for each receptor species is presented in Appendix L, Tables L-6 and L-7.

Concentrations in Diet Items

Dietary concentrations were calculated separately for terrestrial-based food items (e.g., soil invertebrates, small mammals) and for aquatic-based food items (e.g., fish, sediment invertebrates).

<u>Terrestrial items</u>. Terrestrial items in the diet include vegetation and small prey, and the prey concentrations for each congener are based on soil-to-organism BAFs and the soil concentration. Concentrations in vegetation occur through particle deposition and vapor transfer (U.S. EPA, 2000), and plant concentrations were calculated based on these two transport mechanisms using the methods described in Section 5.0 (U.S. EPA, 2001a). For the ecological assessment, concentrations in all types of vegetation were calculated on a wet weight (WW) basis, as described in Appendix H, Table H3.16.

Concentrations in prey items (e.g., small mammals and birds) were calculated as described in Section 9.3.1.2.1 for Phase 1. BAFs for terrestrial prey items are empirical values that reflect prey tissue concentrations as a function of soil concentrations. That is, the BAF does not represent a biotransfer from one compartment in the food chain to another. In addition, a diet fraction variable was added to the calculation to account for each diet item's contribution to the total diet (Equation 9-5).³

$$C_{diet i} = \Sigma C_{soil i} \times BAF_{ij} \times BEF_i \times DietFrac_j$$
(9-5)

where

$C_{diet i}$	=	Total concentration of congener i in diet (mg/kg WW)
$C_{\text{soil }i}$	=	Soil concentration for congener i (mg/kg)
BAF _{ij}	=	Bioaccumulation factor for congener <i>i</i> for food item <i>j</i> (mg/kg WW/ mg/kg soil)
BEF _i	=	Bioaccumulation equivalence factor for congener i (unitless; default value of 1 was used)
DietFrac,	=	Fraction of item <i>j</i> in diet.

<u>Aquatic items</u>. Aquatic items in the diet include T3 and T4 fish, aquatic plants, and benthic invertebrates, primarily filter feeders. Concentrations in these items were calculated as described in Section 9.3.1.2.1 for Phase 1. The dietary fraction was added to the calculation (as for Phase 2 terrestrial prey) to account for each diet item's contribution to the diet (Equation 9-6).

³ Because soil-to-plant uptake of dioxin-like congeners is negligible, plant concentrations were taken from the multimedia modeling simulation and used directly to calculate $C_{diet i}$.

$$C_{diet \ li} = \Sigma \ C_{oc_{sediment \ i}} \times BSAF_{lij} \times DietFrac_{j}$$
(9-6)

where

$\mathbf{C}_{ ext{diet }li}$	=	Lipid-based concentration of congener i in diet (mg/kg)
$C_{oc_sediment i}$	=	Sediment concentration normalized for organic carbon for congener i (mg/kg _{oc}).
BSAF _{lij}	=	Biota-sediment accumulation factor reflecting biouptake from sediment into lipid tissue of item j (kg _{oc} /kg _l)
DietFrac _j	=	Fraction of item <i>j</i> in diet (unitless).

Total Exposure Dose

Each receptor's exposure dose was calculated as a function of its respective ingestion rate, body weight, and the concentrations in the various diet items.⁴ In addition to prey and plant items, soil and sediment ingestion, as a fraction of total diet, were also accounted for in both the terrestrial and margin habitats.⁵ In addition, exposure through drinking water ingestion was included in predicting the exposure dose for receptors in the margin habitat. For completeness, Equation 9-7 presents the total exposure dose calculation for mammals and birds assigned to the margin habitat. For the terrestrial habitat, the last term representing water ingestion is simply omitted from the equation.

$$Dose_{i} = \frac{(IR_{diet} \times C_{diet i}) + (C_{soil/sed i} \times IR_{diet} \times S_{frac}) + (C_{water i} \times IR_{water})}{BW}$$
(9-7)

where

Dose _i	=	Exposure dose for congener i (mg/kg-d)
IR _{diet}	=	Species-specific ingestion rate (kg WW/d)
$C_{diet i}$	=	Total concentration of congener i in diet (mg/kg WW)
C _{soil/sed i}	=	Concentration of congener i in soil or sediment (mg/kg)
$\mathbf{S}_{\mathrm{frac}}$	=	Fraction of soil or sediment in the diet (unitless)
C _{water i}	=	Concentration of congener i in surface water (mg/L)
IR _{water}	=	Species-specific water ingestion rate (L/d)
BW	=	Species-specific average adult body weight.

⁴ Concentrations in lipid tissue of aquatic organisms were converted to whole-body concentrations by adjusting for lipid content in prey.

⁵ The assimilation efficiency for dioxin-like chemicals was conservatively assumed to be 1.0 for each congener. Therefore, it was not shown explicitly in this equation.

Congener-specific doses were summed to derive a single TEQ dose for each receptor in each habitat, as shown in Equation 9-8:

$$Dose_{TEQ} = \Sigma Dose_i \times TEF_i$$
 (9-8)

where

Dose _{TEO}	=	Total dose in toxicity equivalence (mg/kg-d)
Dose _i	=	Dose for congener i (mg/kg-d)
TEF_i	=	Toxicity equivalence factor for congener <i>i</i> .

TEFs were taken from the WHO consensus TEFs for mammals, fish, and birds (U.S. EPA, 2001b) and are presented in Appendix L, Table L-9.

9.3.2.3 <u>Risk Calculations for Phase 2</u>. As with Phase 1, the risk metric for the Phase 2 screen was the HQ, calculated as the ratio of the TEQ exposure dose to the species-specific ecological benchmarks based on allometric scaling of the MATLs. The exposure doses were calculated using the 90th percentile TEQ concentrations in environmental media for mammals and birds, respectively. The toxicological studies used in deriving the species-specific MATLs were described in Section 9.2.2.5 on effects characterization. The assumptions, scaling equations, and factors (i.e., factor of 1/4 for mammals and 1 for birds) are presented in Appendix L.

9.4 Results and Risk Characterization

The two phases of the SERA were designed to provide insight into the potential for adverse ecological effects, and the results from each phase support different conclusions and decisions. Phase 1 was a highly conservative screen intended to serve as the "trigger" for a more refined screening assessment. The HQ results from Phase 1 were used only to indicate that further analysis was warranted. Phase 2 of the SERA was a less conservative screen of the potential for adverse effects on wildlife associated with terrestrial and waterbody margin habitats that may be affected by the agricultural application of biosolids. Although both phases of the SERA were deterministic, the Phase 2 risk estimates were based on less conservative assumptions regarding the environmental media concentrations, receptor-specific dietary preferences, and ecological benchmarks. The HQ results from Phase 2 are point estimates of risk to a wide variety of mammals and birds, and were intended to inform the ongoing assessment of the ecological risks associated with the agricultural application of biosolids.

In the Phase 1 analysis, the HQ values varied from a low of 2 (osprey) for the 50th percentile concentration, to a high of 209 (mink) for the maximum concentration. The highest HQs are associated with biosolids concentrations that were used as a surrogate for sediment (i.e., exposures in margin habitats for osprey, belted kingfisher, and mink). As suggested in the problem formulation, a target HQ of 1 for the Phase 1 screen was used as a "trigger" to determine whether further analysis was warranted. Simply put, HQs greater than 1 in the first phase of the

SERA indicated that the second phase of the SERA was necessary. The results of the Phase 1 analysis⁶ are presented in Table 9-8.

	HQ			
Receptor	50 th percentile [TEQ] in biosolids	90 th percentile [TEQ] in biosolids	Maximum [TEQ] in biosolids	
Osprey	2	11	31	
American robin	5	15	166	
Belted kingfisher	4	25	72	
Mink	36	26	209	

Table 9-8	Phase 1	Results
-----------	---------	---------

As shown in Table 9-9, no HQ values exceeded the target HQ of 1; values range from a minimum of 0.0035 (Canada goose) to a maximum of 0.36 (short-tailed shrew). The median HQ for the receptors assigned to margin habitats was 0.015, and the median HQ for receptors assigned to terrestrial habitats was 0.044, suggesting that the potential risks to terrestrial receptors may be slightly higher than risks to receptors in margin habitats. Although the risk results from Phase 2 did not exceed the target HQ of 1, the HQ values for 8 receptors were within a factor of 10 of the target HQ (7 terrestrial receptors and 1 margin receptor).

9.4.1 Interpreting Results from the SERA

As described in Section 9.2, the SERA was designed to evaluate the potential for adverse effects to mammals and birds selected to represent species in general terrestrial and waterbody margin habitats. By inference from the measures of effect (e.g., reproductive fitness), the SERA is intended to provide insight into the potential effects on wildlife populations, capturing the most significant exposure pathways associated with dioxin and PCB releases into the environment. Consequently, the SERA addresses only wildlife species of mammals and birds.

For Phase 1 of the SERA, the exceedances of the target HQ clearly indicated that Phase 2 should be conducted. Although the HQ results from Phase 2 are suggestive of a low potential for adverse ecological effects, these results are intended only to inform the ongoing evaluation of potential ecological risks associated with biosolids application. The conservative assumptions

⁶ For the mink, the 50th percentile TEQ biosolids concentration results in an HQ that is 1.4 times higher than the HQ for the 90th percentile TEQ biosolids concentration. Although the 90th percentile biosolids are more toxic to mammals than the 50th percentile biosolids, the congeners in the 50th percentile biosolids include higher concentrations of more bioaccumulative congeners. As a result, the predicted hazard associated with fish ingestion is actually higher for the less toxic sludge. That is, the applied dose in fish reflects a stronger potential to bioaccumulate the 50th percentile congener mixture than the 90th percentile congener mixture.

Receptor Species	Terrestrial Habitats	Margin habitats
American kestrel	3.5E-02	not assigned
American robin	1.2E-02	not assigned
American woodcock	1.8E-01	not assigned
Bald eagle	not assigned	0.0028
Beaver	not assigned	0.025
Belted kingfisher	not assigned	0.009
Black bear	8.1E-02	not assigned
Canada goose	0.0035	not assigned
Cooper's hawk	2.9E-02	not assigned
Coyote	2.2E-01	not assigned
Deer mouse	3.0E-02	not assigned
Eastern cottontail rabbit	0.044	not assigned
Great blue heron	not assigned	0.0035
Green heron	not assigned	0.0063
Herring gull	not assigned	0.0088
Least weasel	1.6E-01	not assigned
Lesser scaup	not assigned	0.021
Little brown bat	6.2E-02	not assigned
Long-tailed weasel	2.2E-01	not assigned
Mallard	not assigned	0.01
Meadow vole	0.017	not assigned
Mink	not assigned	0.023
Muskrat	not assigned	0.081
Northern bobwhite	1.3E-02	not assigned
Osprey	not assigned	0.0036
Prairie vole	2.3E-02	not assigned

Table 9-9. Screening Results from Phase 2

(continued)

Receptor Species	Terrestrial Habitats	Margin Habitats
Raccoon	4.4E-02	0.13
Red fox	1.7E-01	not assigned
Red-tailed hawk	1.9E-02	not assigned
River otter	not assigned	0.026
Short-tailed shrew	3.6E-01	not assigned
Short-tailed weasel	1.8E-01	not assigned
Tree swallow	2.8E-02	not assigned
Western meadowlark	1.7E-02	not assigned
White-tailed deer	0.061	not assigned

 Table 9-9. (continued)

(e.g., 100 percent of the diet is contaminated) built into the SERA do provide some basis for interpreting the HQ results; however, the Phase 2 screening analysis was not intended to replace a formal ecological risk assessment. Rather, it represents one step in the evaluation of ecological risk. For example, threatened and endangered species and habitats were not included in the analysis because a more site-specific approach would be required to address the co-occurrence of these receptors and their critical habitat with biosolids application sites. Consequently, the screening results do not indicate whether endangered species are at risk. In addition, the potential for adverse ecological effects (as indicated by the HQ results) should not be confused with the ecological significance. Screening results can only suggest the potential for ecological damage; they do not demonstrate actual ecological effects, nor do they indicate whether those effects will have significant implications for ecosystems and their components.

The results from the Phase 2 screening were compared to the results from an ecological risk assessment of TCDD in pulp and paper sludge (Meyn et al., 1997). The assessment conducted by Meyn et al. evaluated many of the same receptors (e.g., red-tailed hawk, shrew) as those considered in the biosolids SERA, and included four scenarios: agricultural fields (row crops), pasture, silviculture, and mine reclamation. The authors used a Monte Carlo approach to characterize the potential risks to wildlife, and determined that shrews were the wildlife species associated with the highest risks from exposure to TCDD in sludge. This finding is consistent with the results from the biosolids SERA; however, the hazard quotients predicted in Meyn et al. were substantially higher than those predicted in the biosolids SERA. The 50th percentile hazard quotients for row crops and pastures were 60 and 200, respectively, for shrews.

The difference between the results presented by Meyn et al and those presented in Phase 2 can be attributed primarily to differences in the soil concentrations predicted by the respective models, as well as the choice of benchmarks. The 90^{th} percentile TCDD concentrations for the

row crop and pasture scenarios in the Meyn study were 54 ng/kg and 72 ng/kg, respectively, as compared to the 90th percentile TEQ soil concentrations predicted for the biosolids SERA shown in Table 5-4 (0.9 ng/kg and 4.2, respectively). The benchmarks used by Meyn et al., were NOAELs divided by an interspecies uncertainty factor (UF) of 10; the MATLs used in the SERA were not adjusted to address interspecies differences in sensitivity. Considering for the differences in soil concentrations and benchmarks, the risk results from the biosolids SERA for are consistent with risk estimates presented by Meyn et al., (1997). For example, increasing the HQ results by a factor of 25 to account for differences in soil concentrations⁷, and a factor of 10 to account for the interspecies uncertainty factor, would result in an HQ of approximately 90 for the shrew. This HQ is between the HQ values at the 50th percentile calculated for the row crop and pasture scenarios for shrews presented in Meyn et al., (1997). It should be noted that the 50th percentile hazard quotients for the silviculture and mine reclamation scenarios were also 60 and 200.

As indicated in the problem formulation, the toxicological data identified during the effects characterization were considered inadequate to evaluate risks to certain receptors, such as reptiles. In addition, the toxicity data were insufficient to develop environmental quality criteria for water column, sediment, and soil communities. However, substantial data were available on adverse effects to fish exposed to dioxin and PCBs. Therefore, adverse effects concentrations for dioxins were compared with the 90th percentile TEF-adjusted surface water concentration (3.5E-12 mg/L) to investigate the potential risks to fish populations. A variety of sources were reviewed to identify appropriate effects concentrations for comparison, as well as to determine which fish species and endpoints were considered most sensitive based on the available data. The *Dose-Response Assessment from Recently Published Research of the Toxicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds to Aquatic Life–Laboratory Studies* (NCEA, 2001) was reviewed along with other relevant reports, such as

- Workshop Report on the Application of 2,3,7,8-TCDD Toxicity Equivalence Factors to Fish and Wildlife (U.S. EPA, 2001b)
- A Compendium of Environmental Quality Benchmarks (MacDonald et al., 1999)
- Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities (U.S. EPA, 1999)
- Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision (Suter and Tsao, 1996)
- Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife (U.S. EPA, 1993b).

 $^{^{7}}$ The factor of 25 was estimated by calculating a simple arithmetic average for the agricultural field and pasture for Meyn study (63 ng/kg) and the biosolids SERA (~2.5 ng/kg), respectively, and dividing.

The environmental quality criteria and effects concentrations for 2,3,7,8-TCDD for fish and aquatic life range from 2.0E-11 mg/L from a proposed water quality criterion for Ontario, Canada, (MacDonald et al., 1999) to a value of 3.8E-09 mg/L proposed as a screening-level benchmark for fish in the ecological risk assessment protocol for Hazardous Waste Combustion facilities (U.S. EPA, 1999). The Canadian value likely reflects wildlife exposures, as well, so it was of limited value in the SERA because wildlife exposures were estimated in the Phase 2 screening. Suter and Tsao (1996) presented a screening value of 1.0E-08 mg/L for fish, based on EPA Region IV Water Management Division, Water Quality Standards Unit's Screening List. EPA proposed a low-risk water concentration of 6E-10 mg/L and a high-risk water concentration of 1.0E-09 mg/L for fish (U.S. EPA, 1993b); the follow-on dose-response report (NCEA, 2001) provided additional data in support of EPA's proposed benchmark concentrations.

The preponderance of studies on adverse effects to fish report data on reproductive and developmental endpoints. However, EPA determined that the critical life stage for several fish species was embryo development, that salmonid fish were the most sensitive group tested, and that lake trout were the most sensitive species in that group (NCEA, 2001). Toxicity was not observed in adult female lake trout exposed to 2,3,7,8-TCDD dissolved in water even at concentrations at which the oocytes were nonviable. The environmental concentration for low risk to fish proposed in the *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife* (U.S. EPA, 1993b) was based on the survival of lake trout sac fry exposed as eggs. This concentration reflects data on the most sensitive fish species (lake trout) at the most sensitive life stage and, therefore, was considered to be an appropriate benchmark for screening purposes. EPA's analysis of ecotoxicological data suggests that fish are more sensitive to TCDD than are aquatic invertebrates or amphibians (U.S. EPA, 2001b).

The comparison of the low-risk-effects concentration (6E-10 mg/L) with the 90th percentile TEF-adjusted surface water concentration for 2,3,7,8-TCDD (3.5E-12 mg/L) resulted in a screening HQ of 0.0058. Subsequent research (NCEA, 2001) indicates that the observations regarding the most sensitive species (salmonids) and endpoint (survival of sac fry) presented by EPA (U.S. EPA, 1993b) are still valid. Based on these results, fish were not considered to be among the most sensitive receptors evaluated in the SERA.

9.4.2 Silvicultural and Reclamation Site Applications

In addition to agricultural applications, biosolids are applied as a soil amendment to silvicultural operations and to land reclamation projects. In general, reclamation applications of biosolids are not well characterized. These applications can consist of spreading biosolids on reformed land surfaces as an amendment to support revegetation or as fill material deposited in excavations. In the former case, some tilling may occur with landscaping operations; for the latter case, tilling is unlikely. In either case, the dioxins and PCBs would be expected to bind to soil particles and exhibit fate and transport behavior similar to that in pastures; that is, the biosolids will not be tilled into the soil. While the application rates and frequency are not necessarily comparable, ecological exposures are likely to occur in a manner similar to that for agricultural fields. The terrestrial vertebrates evaluated in Phase 2 of the SERA are likely to be similar to receptors found at reclamation sites for terrestrial and margin habitats.

For silvicultural application of biosolids, the application rates and frequency are not well characterized; however, it appears that biosolids are probably applied once per site. The concentration profile for soils may be similar to pastures with the exception of reforestation projects where site preparation for new plantings could include tilling of biosolids into the soil. The concentration profile for reforestation projects would tend to be more similar to the agricultural field applications evaluated in the SERA that involve tilling. Twelve of the avian and mammalian species listed in Table 9-3 for the terrestrial habitat are also be expected to feed and forage in forests; therefore, the screening results for the generalized terrestrial habitat are not represented in the agricultural scenario, the major trophic elements are substantially represented.

The HQ results from Phase 2 of the SERA have limited applicability to silvicultural and reclamation site applications. The application of biosolids to the surface may form a litter layer with substantially higher concentrations of dioxins and PCBs than the concentrations estimated in the model simulations for agricultural fields and pastures. The invertebrate community feeding on the biosolids layer may accumulate relatively high congener concentrations resulting in exposures for mammals and birds that are similar to those evaluated in the Phase 1 screen. Extrapolating from the information presented in Phases 1 and 2 of the SERA, the hazards to receptors feeding on soil invertebrates in a silvicultural application could potentially fall within a range of concern (i.e., an HQ above 1). Although the biosolids SERA provides some indication of the potential for adverse ecological effects associated with the silvicultural and reclamation scenarios, further evaluation will be required to characterize the potential ecological risks.

9.4.3 Uncertainty

In discussing the uncertainties associated with Phase 2 of the SERA, it is important to consider the management goal as the context for identifying key uncertainties and deciding whether these uncertainties are acceptable. Uncertainties do not necessarily diminish the value of the information presented in the SERA. For example, given the goals of the Phase 2 screening assessment, uncertainties that tend to bias the risk results to produce more conservative estimates of the potential for adverse ecological effects may be considered acceptable. Consequently, this discussion is focused on the most significant sources of uncertainty and describes the most likely impact of those uncertainties on the screening risk estimates. The results of the SERA are not intended to provide a final or conclusive statement regarding the ecological risks associated with the agricultural application of biosolids.

Ecological effects associated with background concentrations of dioxins and PCBs are not considered. The screening results reflect the incremental risk to ecological receptors from exposure to dioxins and PCBs in biosolids. However, there is some evidence to suggest that ecological damages may be associated with background concentrations of dioxin-like compounds. Table 5.4 presents soil and sediment concentrations of TCDD-TEQs (based on human health TEFs) for comparison with background concentrations in rural soil (2.5 ng/kg) and sediment (5.8 ng/kg). Based on this information, the aggregate risk (i.e., background and biosolids-related) from TCDD exposure may be higher than the incremental risk attributable to biosolids application.

- The SERA evaluates only dioxins and PCBs, implying that other stressors are insignficant. The SERA did not address other chemical constituents in biosolids, nor did it address other potential stressors (chemical or other) to which wildlife may be exposed. As a result, the predicted screening risks (as represented by the HQ results) may underestimate the potential for adverse ecological effects in a multistressor environment.
- The agricultural application of biosolids does not adequately represent the silviculture and reclamation scenarios. As discussed above, there is considerable uncertainty in extrapolating from the agricultural application of biosolids to other scenarios. This uncertainty suggests that further evaluation of silvicultural and reclamation practices may be required to evaluate the potential for adverse ecological effects in those scenarios.
- The temporal scale for the assessment is driven by the modeling system and is based on annual concentrations averaged across the area of interest. Because annual concentrations are used in calculating exposure doses, potentially significant peaks in exposure are not explicitly addressed (e.g., the concentration profile following an application of biosolids). To some degree, the data on bioaccumulation and toxicity support the use of annual averages because they are based on long-term, steady-state situations. Nevertheless, risks to wildlife may be underestimated if peak exposures occur at sensitive lifestages.
- The spatial scale of the assessment assumes that 100 percent of the diet originates from the contaminated area. For certain receptors (e.g., deer mouse), this assumption is consistent with the relationship between the species home range and the size of the agricultural field and pasture in the conceptual site model. However, for species with much larger home ranges (e.g., coyote), this assumption tends to overestimate the potential hazard associated with biosolids application.
- Margin habitats are broadly defined in terms of streams, ponds, and lakes. Defining the margin habitat broadly, rather than simply modeling a small farm pond, has implications with regard to receptor selection, as well as the applicability of exposure estimates. For example, concentrations of dioxins and PCBs in a lake ecosystem attributable to biosolids are likely to be very small and, possibly, negligible. In contrast, the concentration in sediment of a small farm pond may increase substantially from erosion and runoff of soil-bound congeners. The surface water model does not distinguish between these types of waterbodies, and, as a result, the sediment and surface water concentrations would likely overestimate potential exposures for lakes and moderate-sized streams.
- The measure of effect is at the level of the individual organism; therefore, effects at the population level must be inferred from the endpoint. Although the endpoints chosen for benchmark development for mammals and birds are highly relevant to population viability, they cannot be used to directly evaluate the

potential risks to wildlife species populations. Population-level models have been used by ecologists for decades to evaluate population impacts associated with a variety of stressors. However, parameterizing a population model requires a substantial investment in resources, and there are many difficult decisions to be made regarding the appropriate level of effect for the population (e.g., is a 10 percent reduction in the reproductive fitness of the shrew population acceptable?). Currently, it is not possible to determine whether inference to populations from endpoints relevant to population viability tends to over- or underestimate the potential for adverse ecological effects.

- Ecological benchmarks in Phase 2 are based on a statistical rather than biological derivation, and data are insufficient to provide defensible adjustment factors to account for interspecies variability. Although it is widely recognized that using NOAELs in screening analyses tends to produce results that are difficult to interpret, there is no consensus on the most appropriate measure of effect for a SERA. Moreover, the available toxicological data provide little support to develop adjustment factors to account for interspecies variability. Indeed, there is some evidence to suggest that the ring-necked pheasant may be one of the more tolerant avian species (Giesy et al., 1995). As a result, there is significant uncertainty associated with deriving the species-specific ecological benchmarks.
- It is not possible to verify that reproductive and developmental endpoints are, in all cases, sufficient to protect the assessment endpoints for wildlife populations. The endpoints for certain wildlife populations (i.e., mammals, birds) were almost exclusively taken from reproductive and developmental studies. Although reproductive and developmental endpoints have been recognized by the SAB as relevant to population viability, they are not always the critical effect associated with a chemical stressor. The assumption that effects that are relevant to population viability do not occur at lower environmental concentrations limits confidence in the screening HQ results.
- Uncertainty is inherent in the TEF/TEQ methodology. Although EPA has determined that the TEF/TEQ methodology used in this analysis reduces the uncertainty associated with risk estimates for AhR agonists relative to those based on a single compound (e.g., TCDD), there may be effects associated with these chemicals that are unrelated to AhR and, therefore, are not accounted for (U.S. EPA, 2001b). Furthermore, EPA points out that use of the TEFs is most appropriate for taxa and endpoints used in developing the TEF values. Uncertainties are introduced with increasing taxonomic and endpoint extrapolation.
- The selection of terrestrial BAFs is based on regression analyses and empirical data and does not include all of the prey categories. The terrestrial BAFs were based on empirical data and regression analyses (see Appendix L), and information on small mammals was used to represent terrestrial vertebrates of

different sizes. In addition, measures of central tendency—rather than high-end values—were considered appropriate in selecting input values. Although the uncertainty associated with representing terrestrial vertebrates using data on small mammals has not been quantified, using a high-end value for bioaccumulation may have produced HQ results that exceeded the target HQ of 1.

- The default BEF of 1 assumes that all congeners are accumulated in terrestrial prey at a rate similar to 2,3,7,8-TCDD. Lacking congener-specific adjustment factors for bioaccumulation, no adjustment was made to account for differences in congener-specific accumulation in terrestrial animals. Based on the BEFs for bioaccumulation in aquatic organisms, the default factor likely overestimates the tissue concentrations in terrestrial prey.
- The congener-specific BSAFs were recommended for use in risk assessment in the Dioxin Reassessment (U.S. EPA, 2000). The U.S. EPA Office of Research and Development recommends congener-specific values for BSAFs. These recommendations were based on a review of numerous studies from different types of waterbodies and many species of fish. Although there is some uncertainty in applying empirical values to estimate tissue concentrations in fish, this is considered to be a relatively minor source of uncertainty given the exhaustive review conducted by EPA.

9.5 References

- Abt (Abt Associates, Inc.). 1993. *Revision of Assessment of Risks to Terrestrial Wildlife from TCDD and TCDF in Pulp and Paper Sludge*. Prepared for U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics, by Abt Associates, Inc., Bethesda, MD.
- Anderson, A.N. 1997. Using ants as bioindicators: Multiscale issues in ant community ecology. *Conservation Ecology* 1:8.
- Begon, M., and M. Mortimer. 1981. *Population Ecology: A Unified Study of Animals and Plants*. Sunderland, MA: Sinauer Assoc., Inc.
- Bowman, R.E., S.L. Schantz, M.L. Gross, and S.A. Ferguson. 1989a. Behavioral effects in monkeys exposed to 2,3,7,8-TCDD transmitted maternally during gestation and four months of nursing. *Chemosphere* 18(1-6):235-242.
- Bowman, R.E., S.L. Schantz, N.C.A. Weerasinghe, M.L. Gross, and D.A. Barsotti. 1989b. Chronic dietary intake of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at 5 or 25 parts per trillion in the monkey: TCDD kinetics and dose-effect estimate of reproductive toxicity. *Chemosphere* 18(1-6):243-252.
- Brunstrom, B., and J. Lund. 1988. Differences between chick and turkey embryos in sensitivity to 3,3',4,4'-tetrachlorobiphenyl and in concentration/affinity of the hepatic receptor for

2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Comparative Biochemistry and Physiology* C 91:507-512.

Caduto, M.J. 1990. Pond and Brook. Hanover, NH: University Press of New England.

- Custer, T.W., R.K. Hines, M.J. Melancon, D.J. Hoffman, J.K. Wickliffe, J.W. Brickham, J.W. Martin, and D.S. Henshel. 1997. Contaminant concentrations and biomarker responses in great blue heron eggs from 10 colonies on the Upper Mississippi River, USA. *Environmental Toxicology and Chemistry* 16:260-271.
- Davis, W.S., and T.P. Simon (eds). 1995. *Biological Assessment and Criteria: Tools for Water Resource Planning and Decision Making*. Boca Raton, FL: Lewis Publishers.
- Eisler, R. 1986. Dioxin hazards to fish, wildlife, and invertebrates: a synoptic review. In *Contaminant Hazard Reviews, Report No. 8.* U.S. Fish and Wildlife Service, U.S. Department of the Interior, Laurel, MD.
- Giesy, J.P., W.W. Bowerman, M.A. Mora, D.A. Verbrugge, R.A. Othoudt, J.L. Newsted, C.L. Summer, R.J. Aulerich, S.J. Bursian, J.P. Ludwig, G.A. Dawson, T.J. Kubiak, D.A. Best, and D.E. Tillitt. 1995. Contaminants in fishes from Great Lakes-influenced sections and above dams of three Michigan rivers: Implications for health of bald eagles. *Archives of Environmental Contamination and Toxicology* 29:309-321.
- Hart, L.E., K.M. Cheng, P.E. Whitehead, R.M. Shah, R.J. Lewis, S.R. Ruschkowski, R.W. Blair, D.C. Bennett, S.M. Bandiera, R.J. Norstrom, and G.D. Bellward. 1991. Dioxin contamination and growth and development in great blue heron embryos. Journal of Toxicology and Environmental Health 32:331-344.
- Henshel, D.S., B. Hehn, R. Wagey, M. Vo, and J.D. Steeves. 1997. The relative sensitivity of chicken embryos to yolk- or air-cell-injected 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Environmenal Toxicology and Chemistry* 16:725-732.
- Hochstein, J.R., R.J. Aulerich, and S.J. Bursian. 1988. Acute toxicity of 2,3,7,8tetrachlorodibenzo-*p*-dioxin to mink. *Archives of Environmental Contamination and Toxicology* 17:33-37.
- Kadlec, R.H., and R.L. Knight. 1996. Treatment Wetlands. CRC Press, Boca Raton, Florida
- Khera, K.S., and J.A. Ruddick. 1973. Polychlorodibenz-p-dioxins: perinatal effects and the dominant lethal test in wistar rats. In *Chlorodioxins - Origin and Fate. A Symposium Sponsored by the Division of Pesticide Chemistry at the 162nd Meeting of the American Chemical Society*, E.H. Blair (ed.), 8th Edition. pp. 70-84. American Chemical Society, Washington, DC. September 16-17, 1971.

- MacDonald, D.D., T. Berger, K. Wood, J. Brown, T. Johnsen, M.L. Haines, K. Brydges, M.J. MacDonald, S.L. Smith, and D.P. Shaw. 1999. A Compendium of Environmental Quality Benchmarks. Prepared for Environment Canada, Vancouver, B.C., Canada.
- Meyn, O., M. Zeeman, M.J. Wise, and S.E. Keane. 1997. Terrestrial wildlife risk assessment fo TCDD in land-applied pulp and paper mill sludge. *Environmental Toxicology and Chemistry* 16:1789-1801.
- Mineau, P., B.T. Collins, and A. Baril. 1996. On the use of scaling factors to improve interspecies extrapolation of acute toxicity in birds. *Regul. Toxicol. and Pharmacol.* 24:24-29.
- Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. Schwetz. 1979. Three-generation reproduction study of rats given 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in the diet. *Toxicology and Applied Pharmacology* 50:241-252.
- NCEA (National Center for Exposure Analysis). 2001. "Dose-Response Assessment from Recently Published Research of the Toxicity of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds to Aquatic Wildlife—Laboratory Studies." NCEA-C-0649. July 2001.
- Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and reproductive effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in ring-necked pheasant hens. *Journal of Toxicology and Environmental Health* 35:187-198.
- Nosek, J.A., J.R. Sullivan, S.R. Craven, A. Gendron-Fitzpatrick, and R.E. Peterson. 1993. Embryotoxocity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in the ring-necked pheasant. *Environmental Toxicology and Chemistry* 12:1215-1222.
- Powell, D.C., R.J. Aulerich, J.C. Meadows, D.E. Tillitt, J.F. Powell, J.C. Restum, K.L. Stromborg, J.P. Giesy, and S.J. Bursian. 1997. Effects of 3,3',4,4',5-pentachlorobiphenyl (PCB 126), 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), or an extract derived from field-collected cormorant eggs injected into double-crested cormorant (*Phalacrocorax auritus*) eggs. *Environmental Toxicology and Chemistry* 16:1450-1455.
- Sample, B.E., M.S. Alpin, R.A. Efroymson, G.W. Suter, and C.J.E. Welsh. 1997. Methods and Tools for Estimation of the Exposure of Terrestrial Wildlife to Contaminants. Prepared for the U.S. Department of Energy, Office of Environmental Policy and Assistance, Air, Water, and Radiation Division. Prepared by Oak Ridge National Laboratory, Oak Ridge, TN.

Schoener, T.W. 1989. Food webs from small to large. *Ecology* 70(6):1559-1589.

Schoenly, K., and J. Cohen. 1991. Temporal variation in food web structure: 16 empirical cases. *Ecological Monographs* 61(3):267-298.

- Stokes, D.W., and L.Q. Stokes. 1996. *Stokes Field Guide to Birds*. Boston, MA: Little, Brown, & Co.
- Suter, G.W., II. 1993. Ecological Risk Assessment. Chelsea, MI: Lewis Publishers.
- Suter, G.W., II, and C.L. Tsao. 1996. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision. ES/ER/TM-96/R2. Prepared for the U.S. Department of Energy, Washington, DC.
- Tanner, J.T. 1978. *Guide to Study of Animal Populations*. Knoxville, TN: University of Tennessee.
- Terres, J.K. 1980. *The Audubon Society Encyclopedia of North American Birds*. New York: Alfred A. Knopf.
- U.S. EPA (Environmental Protection Agency). 1992. Draft Report: A Cross-Species Scaling Factor for Carcinogen Risk Assessment Based on Equivalence of mg/kg^{3/4}/day. *Federal Register* 57 FR 24152, June 5, 1992.
- U.S. EPA (Environmental Protection Agency). 1993a. *Wildlife Exposure Factors Handbook*. EPA/60/P-92-003C. Office of Research and Development, U.S. Environmental Protection Agency, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1993b. Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife. EPA/600/R-93/055. Office of Research and Development, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1994. Application of Trophic Level Concept to Analysis of Environmental Contaminant Transfer through Terrestrial Food Webs: Issues and Comparisons with Aquatic Trophic Levels. Submitted by M. McVey, ICF Inc., Fairfax, VA, to Office of Science and Technology, Office of Water, U.S. Environmental Protection Agency, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8-TCDD, and PCBs.* EPA-820-B-95-008. Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1998. Risk Assessment Forum. Guidelines for Ecological Risk Assessment – Final. EPA/630/R-95/002F.
- U.S. EPA (Environmental Protection Agency). 1999. Screening-Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Office of Solid Waste, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 2000. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. EPA/600/P-

00/001Bb. Exposure Assessment and Risk Characterization Group, National Center for Environmental Assessment, Office of Research and Development, Washington, DC. September.

- U.S. EPA (Environmental Protection Agency). 2001a. "The Role of Screening-Level Risk Assessments and Refining Contaminants of Concern in Baseline Ecological Assessments." EPA ECO Update, Publication 9345.0-14. EPA/540/F-01/014. Office of Solid Waste and Emergency Response, U.S. EPA, Washington, DC. June.
- U.S. EPA (Environmental Protection Agency). 2001b. "Workshop Report on the Application of 2,3,7,8-TCDD Toxicity Equivalence Factors to Fish and Wildlife." EPA/630/R-01/002. Risk Assessment Forum, U.S. EPA, Washington, DC. August.
- U.S. EPA (Environmental Protection Agency). 2001c. 2001 National Sewage Sludge Survey.