DRAFT

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Attachment C

GUIDANCE FOR PERFORMING SCREENING LEVEL RISK ANALYSES AT COMBUSTION FACILITIES BURNING HAZARDOUS WASTES

Office of Emergency and Remedial Response

Office of Solid Waste

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1. INTRODUCTION

This document provides guidance for performing a screening level analysis of direct and indirect human health risks from combustion emissions. The screening procedure is intended to give a conservative estimate of the potential risk in order to determine whether a more detailed site-specific assessment is warranted. The screening guidance provides information on the constituents, exposure scenarios, indirect pathways, and parameter values that are needed for estimating risk. The document is designed as a kind of "workbook" that is clear, concise, and simple to use. The document is divided into two parts: Part 1 presents a screening methodology for combustion units that are intended to operate for a long period of time such as 30 years. Part 2 presents modifications to this methodology for evaluating short-term operations including trial burns and remediation activities lasting months or several years.

The screening procedure is based on the guidance in the January, 1990 interim final report *Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (EPA/600/6-90/003 and referred to as the Indirect Exposure Document), the draft Addendum to the Indirect Exposure Document (dated November 10, 1993), and the draft implementation guidance entitled "Implementation Guidance for Conducting Indirect Exposure Analysis at RCRA Combustion Units" (dated April 22, 1994 and referred to as the Implementation Guidance). In the interest of simplicity, the procedure has been streamlined by reducing the number of algorithms that need to be evaluated, while retaining the degree of conservatism appropriate for a screening level analysis.

The screening guidance specifies the particular exposure scenarios that should be evaluated and provides default values for most input parameters. In addition, the screening guidance also allows the flexibility to use available site-specific information to modify certain assumptions. For example, site-specific land use information may be used to determine that certain assumptions regarding the exposure scenarios are implausible (e.g., that exposure occurs at the points of maximum air concentration and maximum deposition) and to make alternative assumptions (e.g., to identify locations at which the exposure scenarios used for the screening analysis are plausible). If the final estimated risk is below levels of concern, then there is good reason to conclude that further analysis of the risk from stack emissions is unnecessary.

The primary focus of the screening guidance is on indirect exposures. However, in order to characterize the risk from stack emissions it is necessary to characterize the risk from direct inhalation exposures as well. The screening guidance, therefore, includes a brief discussion of estimating risk from direct inhalation exposures. It is important to recognize that the constituents for which direct inhalation exposures are of primary concern may be different from (and generally more numerous than) those for indirect exposures.

The endpoints of the screening analysis are estimates of individual risk for several exposure scenarios. The exposure scenarios selected for the screening analysis are considered to be the most significant ones for combustion sources. For each scenario, the risk estimates

are based on combining exposures and risk for an individual constituent across several pathways. Where appropriate, risk from multiple constituents are also combined to provide estimates of overall risk for each exposure scenario.

As indicated in the text box, the document is divided into two parts; the first part addressing long-term releases and the second part addressing short-term releases from combustion sources. The following sections in Part 1 of the document give a general overview of the screening approach (Section 2), discuss the required air dispersion and deposition modeling and input parameters (Section 3), present the equations to use and gives default parameter values for calculating media concentrations for each of the pathways that are associated with indirect exposures (Section 4), and explain how to characterize risk for each of the exposure scenarios in the screening analysis (Section 5). Part 2 presents a discussion of changes to the Part 1 methodology that can be used to address short-term combustion operations. These sections correspond to the sections presented in Part 1 of the document. The parameter values used in this screening guidance, their derivation, chemical specific data and all data sources are provided in an Appendix.

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Part 1

Assessing Risk From Long-Term Emissions

Polycyclic Aromatic Hydrocarbons (PAH's)

Benzo(a)pyrene
Benz(a)anthracene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Chrysene
Dibenz(a,h)anthracene
Indeno(1,2,3-cd)pyrene

Based on comparative potency estimates provided in EPA's *Provisional Guidance for the Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (Office of Health and Environmental Assessment, 1993) emissions of these PAH's are converted to benzo(a)pyrene toxicity equivalents (BaP-TEQ). All PAH's are then modeled using the fate and transport properties of benzo(a)pyrene.

Polychlorinated Biphenyls (total PCB's)

total Polychlorinated biphenyls (all congeners)

All polychlorinated biphenyl congeners (209 congeners) are treated as a mixture having a single carcinogenic potency, as recommended in EPA's *Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs)* (U.S. EPA, 1988).

Nitroaromatics

1,3-Dinitro benzene
2,4-Dinitro toluene
2,6-Dinitro toluene
Nitrobenzene
Pentachloronitrobenzene

Phthalates

Bis (2-ethylhexyl) phthalate
Di(n)octyl phthalate

Other Chlorinated Organics

Hexachlorobenzene
Pentachlorophenol

raise cattle for both beef and milk consumption and grow crops for home consumption. Site-specific information could be used to modify these assumptions.

Table 2.1. Consumption Rates and Fraction Contaminated Used in Exposure Scenarios

Contaminated food or media	Exposure Scenario							
	Subsistence Farmer		Subsistence Fisher		Adult Resident		Child Resident	
	Rate	Frac.	Rate	Frac.	Rate	Frac.	Rate	Frac
Beef (g/day)	57	1	NA	NA	NA	NA	NA	NA
Milk (g/day)	181	1	NA	NA	NA	NA	NA	NA
Fish (g/day)	NA	NA	60*	1	NA	NA	NA	NA
Above-ground fruits and vegetables (g DW/day)	28	1	28	0.25	28	0.25	6*	0.25
Soil (mg/day)	100	1	100	1	100	1	200	1
Air (m^3/day)	20	1	20	1	20	1	4.3	1

Notes: DW = dry weight NA = not applicable * = provisional value for interim use only
All values from the Exposure Factors Handbook (U.S.EPA, 1990a).
Units shown are for consumption rate; all fractions contaminated are dimensionless.

Consumption rates for contaminated beef, milk, and above-ground fruits and vegetables, are representative of a typical subsistence farmer, rather than the general population. Exposures to crops include consumption of above-ground fruits and vegetables. The incidental soil ingestion rate and the inhalation rate are typical for adults.

Subsistence Fisher

In the subsistence fisher scenario, an adult fisher is exposed via consumption of contaminated fish and homegrown fruits and vegetables, incidental ingestion of soil, and direct inhalation of vapors and particles. Fish consumption rates are intended to be representative of a typical subsistence fisher, rather than the general population. However, limited data are available on rates of fish consumption by subsistence fishers. Therefore, the consumption rate given in Table 2.1 is provisional and is intended for interim use only. Consumption rates for above-ground fruits and vegetables and the incidental soil ingestion and inhalation rates are typical for adults.

Adult Resident

In the adult resident scenario, an adult is exposed via consumption of homegrown fruits and vegetables, incidental soil ingestion, and direct inhalation of vapors and particles.

2.5 Indirect Exposure Pathways

For screening purposes, indirect exposures include ingestion of above-ground fruits and vegetables, beef and milk, freshwater fish and soil. Contaminants in combustion emissions may reach these media or foods by many pathways. The pathways that provide the highest media or food concentration have been selected for use in the screening analysis. Different pathways give the highest concentrations for different constituents. For example, soil erosion gives the highest water concentration for some constituents, while runoff gives the highest water concentration for other constituents. However, the calculations for each pathway are to be completed for all of the constituents, as discussed in Section 4.

For the indirect exposure pathways analysis, a combination of two parameters that have the greatest impact on media or food concentrations are set at "high end" values, while other parameters are set at typical or "central tendency" values. These parameters are the concentration of the contaminant at the point of maximum combined deposition and the duration of exposure. In addition, the pathways resulting in the highest media concentration and the subsistence farmer and fisher scenarios contribute to the conservative nature of the screening guidance. Tables in Section 4 and Section 5 provide all parameter values that need to be used in the screening analysis.

The indirect exposure pathways selected for screening analyses are described in the following paragraphs.

Above-ground Fruits and Vegetables

Above-ground fruits and vegetables are ingested by humans and cattle. Cattle ingestion of above-ground plants is discussed below in the sections for beef and milk. For human ingestion of above-ground fruits and vegetables, the following two pathways of contaminant transport are included: deposition of particle phase contaminants directly onto plant surfaces and direct transfer of vapor phase contaminants into plant material. One or the other of these pathways may dominate or be inapplicable for specific constituents. Constituent-specific guidance is provided in Section 4.

Beef

For the concentration of a contaminant in beef, three pathways are included: (1) ingestion of contaminated forage and hay, (2) ingestion of contaminated grain and silage, and (3) ingestion of contaminated soil. Forage and hay are grown in fields and can become contaminated by deposition of contaminants directly onto plant surfaces; by direct transfer of vapor phase contaminants into forage plant material; and by uptake of contaminants in the soil. The cows consume the contaminated forage and the contaminants can bioaccumulate in the muscle tissue. Grain and silage also make up some fraction of the cow's diet and can become contaminated through root uptake of contaminants in the soil. It is assumed that grains are protected from direct deposition and vapor phase transfers. In addition, a small fraction of a cow's diet can be contaminated soils, when cows are free ranging. Thus, the

- Emissions from the combustion source for each constituent generally represent high end values. The *Implementation Guidance for Conducting Indirect Exposure Analysis at RCRA Combustion Units* provides guidance for determining metals and organic emissions.

- Air concentration and deposition from the locations of the maximum air concentration and maximum combined wet and dry deposition are used as the point of departure. However, alternative locations may be considered. Additional guidance for identifying alternative locations is provided in Section 3.

- The exposure duration for each exposure scenario is set to a high end value. The values for exposure duration are given in Section 6.

Use of these assumptions with the exposure scenarios described in Section 2.3, together with simplifying conservative assumptions in the exposure pathways analysis, will ensure that the results represent high end or bounding estimates of risk. If there actually are subsistence farmers, subsistence fishers, or residents in the area of concern, the risk estimates will represent high end estimates of risk. However, if there are not subsistence farmers, subsistence fishers, or residents in that area, the risk estimates will represent bounding estimates of risk for the general population.

Additivity of Pathways Within an Exposure Scenario

The exposures from the indirect pathways should be combined for each scenario and constituent. Therefore, for the subsistence farmer scenario, exposures from ingestion of beef, milk, above-ground fruits and vegetables, and incidental soil ingestion should be added together for each constituent. For the subsistence fisher, exposures from ingestion of fish, above-ground fruits and vegetables, and soil should be added together for each constituent. In the adult and child resident scenarios, exposures from ingestion of above-ground fruits and vegetables and incidental soil ingestion should be added together. However, adult exposure and child exposure are considered separately and should not be combined. The end result is one oral exposure (dose) for each scenario and constituent. Given these exposures, a carcinogenic risk and, for non-cancer effects, a hazard quotient is calculated for each scenario and each constituent. (Note that a hazard quotient cannot be calculated for lead, as no health effects benchmark has been established for lead. Therefore, only soil concentrations are calculated for lead.)

Exposures to noncarcinogens from the direct exposure pathway should not be added to those from the indirect pathways. This is because the risk from the direct exposure pathway, which results from the inhalation route of exposure, is determined separately from the risk from the indirect pathways, which result from the oral route of exposure. However, for carcinogens, the risk from direct exposures to a constituent is added to the risk from indirect exposures to the constituent for each exposure scenario.

5. RISK CHARACTERIZATION

Characterization of risk is the final step of the screening analysis. In this step, for each exposure scenario the health effects criteria or benchmarks are used in conjunction with dose estimates which are calculated for each exposure pathway to arrive at the risk assessment endpoints. The assessment endpoints of the screening analysis are as follows: a) the increased probability of cancer in an individual over a lifetime, referred to as the excess lifetime individual cancer risk (or simply, individual cancer risk) arising from both oral and inhalation routes of exposure; b) for oral exposures, a measure of an individual's exposure to chemicals with noncancer health effects relative to the reference dose (RfD), referred to as the hazard quotient; c) for inhalation exposures, a hazard quotient relative to the reference concentration (RfC) in air; and d) where appropriate, a hazard index which represents the combined hazard quotients for those chemicals with the same noncancer health effects. Population risk is not an assessment endpoint for the screening analysis. Although oral and inhalation routes of exposure are handled separately in the screening analysis, the individual risks associated with exposures to carcinogenic chemicals are combined for the oral and inhalation routes of exposure.

Indirect Exposures

For indirect exposures, a series of tables is provided for each exposure scenario. The tables are used for estimating individual cancer risk and hazard quotients for the various chemicals and for combining the cancer risks and hazard quotients across pathways and chemicals as appropriate. Each equation is presented on a separate table. The table provides the mathematical form of the equation, identifies the parameters in the equation, and provides the parameter values (or, if calculated, the tables from which the values are obtained). It should be noted that not all equations are used for all chemicals. Specifically, calculations of individual cancer risks, hazard quotients, and

Section 5.1	**Subsistence Farmer Tables 5.1.1. - 5.1.9.**
Section 5.2	**Subsistence Fisher Tables 5.2.1. - 5.2.9.**
Section 5.3	**Adult Resident Tables 5.3.1. - 5.3.8.**
Section 5.4	**Child Resident Tables 5.4.1. - 5.4.8.**
Section 5.5	**Direct Inhalation Exposures**
Section 5.6	**Infant Exposure Through Breast Milk**
Section 5.7	**Exposure from Contaminated Chicken and Eggs**
Section 5.8	**Overall Direct and Indirect Cancer Risk**

hazard indices address different (albeit overlapping) lists of chemicals. There are four sets of tables presented in the first four sections as indicated in the text box. In addition, equations are provided for direct inhalation exposures, infant exposure through breast milk and exposure through ingestion of contaminated chicken and eggs. Finally, equations are provided for characterizing overall direct and indirect risk for each of the four scenarios.

For each of the four exposure scenarios, an estimate is made of the dose (or intake) of each contaminant from all oral routes of exposure. Thus, for the subsistence farmer, the daily intake of each contaminant is calculated for soil ingestion (Table 5.1.1), above-ground produce ingestion (Table 5.1.2), beef and milk ingestion (Table 5.1.3), and drinking water ingestion (Table 5.1.4). The total daily oral intake of a contaminant is calculated by adding together the intake from each pathway (Table 5.1.5). For each carcinogen, the excess lifetime individual cancer risk is calculated using the cancer slope factor and total daily intake (Table 5.1.6). For each chemical with noncancer health effects, a hazard quotient (HQ) is calculated using the RfD and the total daily intake (Table 5.1.7). For the carcinogens, cancer risks are added across chemicals (Table 5.1.8). For noncancer health effects, hazard quotients are added across chemicals only when they target the same organ. Therefore, the hazard quotients from chemicals that target the same organ are added together to calculate an overall hazard index for liver effects (Table 5.1.9) or neurotoxic effects (Table 5.1.10).

Lead

Childhood exposures to lead in soil are assessed by comparing the estimated soil lead level at the location of maximum combined (wet and dry) deposition (or an alternative location, as discussed in Section 3.4, Exposure Locations) to the soil health-based level given in the Implementation Guidance. Childhood and adult exposures to airborne lead are assessed by comparing the maximum estimated air concentration (or the highest air concentration from an alternative location, as discussed in Section 3.4, Exposure Locations) to the air health-based level given in the Implementation Guidance. No hazard quotient is calculated and no other exposure pathways are considered for lead.

Infant Exposure Through Breast Milk

The dioxin exposure assessment document released by the Office of Research and Development in April 1994, presents procedures for calculating infant exposures to dioxins and other lipophilic compounds through ingestion of human breast milk. The procedures are based on the intake of the contaminant by the mother. The exposure to an infant from breast feeding can be presented as an average daily dose (ADD) or a lifetime average daily dose (LADD). The ADD to the infant over a one year averaging time is predicted to be much higher (e.g. 30 to 60 times higher) than the ADD for the mother. However, if a 70 year averaging time is used, then the LADD to the infant is below the lower end of the range for the mother's LADD. Research is incomplete however in the area of calculating risk for infant exposures to dioxin-like compounds in breast milk. One method of risk characterization, and the method used in this document, is comparison of the ADD to the average adult background level for dioxin exposure, 0.5 pg/kg/day. Algorithms for calculating the ADD for infant

exposure are presented as Equations 5.6.1 and 5.6.2.

Exposure Through Ingestion of Contaminated Chicken Meat and Eggs

Some subsistence farmers raise chickens for meat and eggs instead of or in addition to cows. Recent studies by Stevens et al. (1992) and Petreas et al. (1991) have shown the bioconcentration factors for PCDDs and PCDFs in chicken meat and eggs are relatively high and can result in exposures from ingestion of contaminated chicken meat and eggs that are of the same order of magnitude as through ingestion of contaminated beef and dairy. Based on these preliminary data, a method is presented in Section 5.7 for calculating exposures to PCDDs/PCDFs from contaminated chicken meat and eggs, for the subsistence farmer scenario.

The remainder of this section is organized as follows. As indicated in the previous text box, the tables for characterizing risk from indirect exposures for the four exposure scenarios are given in Section 5.1 through Section 5.4. Characterizing risk from direct inhalation exposures is discussed for all four exposure scenarios in Section 5.5. Characterizing the risk to breast-fed infants is discussed for the three adult exposure scenarios in Section 5.6 and characterizing exposure due to ingestion of contaminated chicken meat and eggs in Section 5.7 Finally, characterizing overall cancer risk from both direct and indirect exposures is discussed in Section 5.8.

5.1 Subsistence Farmer Scenario

This section provides the equations needed for characterizing risk from indirect exposures for the subsistence farmer scenario. The following equation tables are included:

Table 5.1.1. Soil Intake for Subsistence Farmer Scenario

Chemicals	
2378 TCDD-TEQ 1.3-dinitrobenzene 2.4-dinitrotoluene 2.6-dinitrotoluene antimony arsenic barium benzo(a)pyrene toxicity equivalents beryllium bis (2-ethylhexyl) phthalate cadmium chromium	di-n-octyl phthalate hexachlorobenzene mercury nickel nitrobenzene pentachloronitrobenzene pentachlorophenol selenium silver thallium total PCBs

Equation

$$I_{soil} = Sc \cdot CR_{soil} \cdot F_{soil}$$

Parameter	Description	Value
I_{soil}	Daily intake of	
Sc	Soil concentration (mg/kg)	calculated (see Table 4.1.1)
CR_{soil}	Consumption rate of soil (kg/day)	0.0001
F_{soil}	Fraction of consumed soil contaminated (unitless)	1

Table 5.1.2. Above-Ground Produce Intake for Subsistence Farmer Scenario

Chemicals	
2378 TCDD-TEQ 1,3-dinitrobenzene 2,4-dinitrotoluene 2,6-dinitrotoluene antimony arsenic barium benzo(a)pyrene toxicity equivalents beryllium bis (2-ethylhexyl) phthalate cadmium chromium	di-n-octyl phthalate hexachlorobenzene mercury nickel nitrobenzene pentachloronitrobenzene pentachlorophenol selenium silver thallium total PCBs

Equation

$$I_{ag} = (Pd + Pv + Pr) \cdot CR_{ag} \cdot F_{ag}$$

Parameter	Description	Value
I_{ag}	Daily intake of contaminant from above-ground produce (mg/day)	
Pd	Concentration in above-ground produce due to deposition (mg/kg)	calculated (see Table 4.2.6)
Pv	Concentration in above-ground produce due to air-to-plant transfer (mg/kg)	calculated (see Table 4.2.7)
Pr	Concentration in above-ground produce due to root uptake (mg/kg)	calculated (see Table 4.2.8)
CR_{ag}	Consumption rate of above-ground produce (kg/day)	0.028
F_{ag}	Fraction of above-ground produce contaminated (unitless)	1

Table 5.1.3. Beef and Milk Intake for Subsistence Farmer Scenario

Chemicals	
2378 TCDD-TEQ	di-n-octyl phthalate
1.3-dinitrobenzene	hexachlorobenzene
2,4-dinitrotoluene	mercury
2,6-dinitrotoluene	nickel
antimony	nitrobenzene
arsenic	pentachloronitrobenzene
barium	pentachlorophenol
benzo(a)pyrene toxicity equivalents	selenium
beryllium	silver
bis (2-ethylhexyl) phthalate	thallium
cadmium	total PCBs
chromium	

Equation

$$I_{beef} = A_{beef} \cdot CR_{beef} \cdot F_{beef}$$

$$I_{milk} = A_{milk} \cdot CR_{milk} \cdot F_{milk}$$

Parameter	Description	Value
I_{beef}	Daily intake of contaminant from beef (mg/day)	
A_{beef}	Concentration in beef (mg/kg)	calculated (see Table 4.4.4)
CR_{beef}	Consumption rate of beef (kg/day)	0.057
F_{beef}	Fraction of beef contaminated (unitless)	1
I_{milk}	Daily intake of contaminant from milk (mg/day)	
A_{milk}	Concentration in milk (mg/kg)	calculated (see Table 4.4.5)
CR_{milk}	Consumption rate of milk (kg/day)	0.18
F_{milk}	Fraction of milk contaminated (unitless)	1

Table 5.1.4. Drinking Water Intake for Subsistence Farmer Scenario

Chemicals	
2378 TCDD-TEQ 1,3-dinitrobenzene 2,4-dinitrotoluene 2,6-dinitrotoluene antimony arsenic barium benzo(a)pyrene toxicity equivalents beryllium bis (2-ethylhexyl) phthalate cadmium chromium	di-n-octyl phthalate hexachlorobenzene mercury nickel nitrobenzene pentachloronitrobenzene pentachlorophenol selenium silver thallium total PCBs

Equation

$$I_{dw} = C_{dw} \cdot CR_{dw} \cdot F_{dw}$$

Parameter	Description	Value
I_{dw}	Daily intake of contaminant from drinking water (mg/day)	
C_{dw}	Dissolved contaminant concentration in drinking water (mg/L)	calculated (see Table 4.4.24)
CR_{dw}	Consumption rate of drinking water (L/day)	1.4
F_{dw}	Fraction of drinking water contaminated (unitless)	1

Table 5.1.5. Total Daily Intake for Subsistence Farmer Scenario

Chemicals	
2378 TCDD-TEQ 1.3-dinitrobenzene 2.4-dinitrotoluene 2.6-dinitrotoluene antimony arsenic barium benzo(a)pyrene toxicity equivalents beryllium bis (2-ethylhexyl) phthalate cadmium chromium	di-n-octyl phthalate hexachlorobenzene mercury nickel nitrobenzene pentachloronitrobenzene pentachlorophenol selenium silver thallium total PCBs

Equation

$$I = I_{soil} + I_{ag} + I_{beef} + I_{milk} + I_{dw}$$

Parameter	Description	Value
I	Total daily intake of contaminant (mg/day)	
I_{soil}	Daily intake of contaminant from soil (mg/day)	calculated (see Table 5.1.1)
I_{ag}	Daily intake of contaminant from above-ground produce (mg/day)	calculated (see Table 5.1.2)
I_{beef}	Daily intake of contaminant from beef (mg/day)	calculated (see Table 5.1.3)
I_{milk}	Daily intake of contaminant from milk (mg/day)	calculated (see Table 5.1.3)
I_{dw}	Daily intake of contaminant from drinking water (mg/day)	calculated (see Table 5.1.4)

Table 5.1.6. Cancer Risk for Individual Chemicals for Subsistence Farmer Scenario: Carcinogens

Chemicals	
arsenic beryllium benzo(a)pyrene toxicity equivalents bis (2-ethylhexyl) phthalate hexachlorobenzene	total PCBs pentachloronitrobenzene pentachlorophenol 2378 TCDD-TEQ

Equation

$$Cancer\ Risk = \frac{I \cdot ED \cdot EF \cdot CSF}{BW \cdot AT \cdot 365}$$

Parameter	Description	Value
Cancer Risk	Individual lifetime cancer risk (unitless)	
I	Total daily intake of contaminant (mg/day)	calculated (see Table 5.1.5)
ED	Exposure duration (yr)	40
EF	Exposure frequency (day/yr)	350
BW	Body weight (kg)	70
AT	Averaging time (yr)	70
365	Units conversion factor (day/yr)	
CSF	Oral cancer slope factor (per mg/kg/day)	chemical-specific (see Appendix 1)

Table 5.1.7. Hazard Quotient for Individual Chemicals for Subsistence Farmer Scenario: NonCarcinogens

Chemicals	
arsenic beryllium bis (2-ethylhexyl) phthalate 1.3-dinitrobenzene 2,4-dinitrotoluene 2,6-dinitrotoluene di-n-octyl phthalate hexachlorobenzene mercury nitrobenzene	pentachloronitrobenzene pentachlorophenol antimony barium cadmium chromium nickel thallium silver selenium

Equation

$$HQ = \frac{I}{BW \cdot RfD}$$

Parameter	Description	Value
HQ	Hazard quotient (unitless)	
I	Total daily intake of contaminant (mg/day)	calculated (see Table 5.1.4)
BW	Body weight (kg)	70
RfD	Reference Dose (mg/kg/day)	chemical-specific (see Appendix 1)

Table 5.1.8. Total Cancer Risk for Subsistence Farmer Scenario: Carcinogens

Chemicals	
arsenic beryllium benzo(a)pyrene toxicity equivalents bis (2-ethylhexyl) phthalate hexachlorobenzene	total PCBs pentachloronitrobenzene pentachlorophenol 2378 TCDD-TEQ

Equation

$$Total\ Cancer\ Risk = \sum_i Cancer\ Risk_i$$

Parameter	Description	Value
Total Cancer Risk	Total individual lifetime cancer risk for all chemicals (unitless)	
Cancer Risk$_i$	Individual lifetime cancer risk for chemical carcinogen i (unitless)	calculated (see Table 5.1.5)

Table 5.1.9. Hazard Index for Liver Effects for Subsistence Farmer Scenario: NonCarcinogens

Chemicals
bis (2-ethylhexyl) phthalate di-n-octyl phthalate hexachlorobenzene pentachloronitrobenzene pentachlorophenol
Equation
$HI_{liver} = \sum_i HQ_i$

Parameter	Description	Value
HI_{organ}	Hazard index for specific organ effects (unitless)	
HQ_i	Hazard quotient for chemical i with liver effects (unitless)	calculated (see Table 5.1.7)

Table 5.1.10. Hazard Index for Neurotoxic Effects for Subsistence Farmer Scenario: NonCarcinogens

Chemicals
2,4-dinitrotoluene 2,6-dinitrotoluene mercury
Equation
$HI_{neurotoxin} = \sum_i HQ_i$

Parameter	Description	Value
HI_{organ}	Hazard index for specific organ effects (unitless)	
HQ_i	Hazard quotient for chemical i with liver effects (unitless)	calculated (see Table 5.1.7)

5.6 Breast Milk Exposure for Dioxins

To determine the average daily dose for a breast-feeding infant, the concentration of dioxin in the mother's milk must first be determined. Three mother scenarios are considered in the breastmilk exposure analysis, corresponding to the three adult exposure scenarios considered as part of this guidance (e.g., subsistence farmer, subsistence fisher, and adult resident). Table 5.6.1 provides equations for calculating the concentration of dioxin in maternal milk. Once the contaminant concentration in maternal milk is determined, the equation in Table 5.6.2 is used to determine the average daily dose for infant exposure in pg/kg/day.

Further research is required in the area of risk characterization of infant exposures. Many questions still exist about how to quantify a lifetime risk for exposure during this very short and developmentally critical period of time. The significance of the average daily dose calculation is unclear, especially considering that many dioxin-like compounds reach steady-state levels only during chronic exposures. As research provides new and better methods of characterizing breastmilk exposure they should be thoughtfully considered. Until that point, this guidance suggests that the average daily dose for one year of breastmilk exposure be compared to the average adult background exposure level for dioxin of 0.5 pg/kg/day, as suggested in the Dioxin Exposure Document.

Table 5.6.1. Concentration in Maternal Milk

Chemicals
All
Equation

$$C_{(milkfat)j} = \frac{m_j \cdot 10^9 \cdot h \cdot f_1}{0.693 \cdot f_2}$$

Parameter	Description	Value
C(milkfat)j	Concentration in maternal milk for exposure scenario j (j=1..3) (pg/kg of milkfat)	
m_1	Average maternal intake of dioxin for exposure scenario j (j=1..3) (pg/kg/day)	calculated (see Tables 5.1.5, 5.2.5, 5.3.4)
10^9	Conversion constant (pg/mg)	
h	Half-life of dioxin in adults (days)	2555
f_1	Proportion of ingested dioxin that is stored in fat (unitless)	0.9
f_2	Proportion of mother's weight that is fat (unitless)	0.3

Table 5.6.2. Average Daily Dose to the Exposed Infant

Chemicals
All
Equation
$$ADD_{(infant)j} = \frac{C_{(milkfat)j} \cdot f_3 \cdot f_4 \cdot IR_{milk} \cdot ED}{BW_{infant} \cdot AT}$$

Parameter	Description	Value
$ADD_{(infant)j}$	Average daily dose for infant exposed to contaminated breastmilk for exposure scenario j (j=1..3) (pg/kg/day)	
$C_{(milkfat)j}$	Concentration in maternal milk for exposure scenario j (j=1..3) (pg/kg of milkfat)	calculated (see Table 5.6.1)
f_3	Fraction of fat in breastmilk (unitless)	0.04
f_4	Fraction ingested contaminant which is absorbed (unitless)	0.9
IR_{milk}	Ingestion rate of breastmilk (kg/d)	0.8
ED	Exposure duration (year)	1
BW_{infant}	Body weight of infant (kg)	10
AT	Averaging time (year)	1

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5.7 Assessing Exposures from Consumption of Contaminated Eggs and Chicken Meat

Recent studies (Stevens et al., 1992 and Petreas et al., 1991) have demonstrated the bioconcentration of 2,3,7,8-substituted PCDD/PCDF isomers in chicken liver, adipose, thigh and eggs from exposure to contaminated soils in the diet. The bioconcentration factors (BCFs) derived in these laboratory studies are used here as a preliminary method for estimating the human health risk associated with exposure to contaminated chicken meat and eggs for subsistence farmers. The BCF were derived by dividing the tissue concentration (wet weight) by the feed concentration. The feed was spiked with 10 percent contaminated soil. Therefore, the basic assumptions for this assessment are that the chickens are free range and the intake of contamianted soil is approximately 10 percent of their diet. Based on a soil concentration and BCF for TCDD-TEQ, the exposure of the subsistence farmer to TCDD-TEQ through consumption of chicken meat and eggs can be estimated as follows:

1) Calculate average daily exposure from ingestion of eggs (I_{egg}):

$$S_c \cdot 0.1 \cdot BCF_{egg} \cdot 50 \cdot I = pg/day\ exposure$$

where:

S_c	=	concentration of TCDD-TEQ in soil (pg/g)
0.1	=	fraction of diet that is soil (dimensionless)
BCF_{egg}	=	Bioconcentration factor for TCDD-TEQ in eggs of range fed chickens
I	=	daily ingestion of eggs (1 egg per day)
50	=	g/egg

2) Calculate the average daily exposure from ingestion of chicken meat (I_{chick}):

$$S_c \cdot 0.1 \cdot BCF_{chick} \cdot I \cdot 100 = pg/day\ exposure$$

where:

S_c	=	concentration of TCDD-TEQ in soil (pg/g)
0.1	=	fraction of diet that is soil (dimensionless)
BCF_{chick}	=	bioconcentration factor for TCDD-TEQ in thigh meat of range fed chickens
I	=	daily ingestion of thigh meat (1 thigh per day)
100	=	g/thigh

The concentration in soil is based on the stack emissions and dispersion modeling and the behavior of the contaminant in soils as described in Equations 4.1.1 through 4.1.5 in Section 4 of this document. The exposure in pg/day, can be used in Equation 5.1.5 as I_{chick} and I_{egg}, in addition to or in place of I_{beef} and I_{milk}.

The bioconcentration factor is dependent on the distribution of 2,3,7,8-substituted PCDDs/PCDFs deposited on the soils. Since the estimated soil TCDD-TEQ concentration is based on the stack emissions of each isomer, the BCF should be calculated based on the percent contribution of each isomer in the stack emissions to the total TCDD-TEQ. Table 4.5.1 provides isomer specific BCFs for 2,3,7,8-substituted PCDDs/PCDFs. The total TCDD-TEQ in the stack emissions should be apportioned according to the contribution of each isomer's contribution to this total. The fraction of TCDD-TEQ contributed by each isomer is then used to develop a weighted BCF factor for egg or thigh meat by multiplying the fraction times the isomer specific BCF. In the absence of isomer specific emissions data, a BCF for TCDD-TEQ of 1.1 for thigh meat and 2.0 for egg yolk could be used. These default values are calculated as the average of the highest 3 isomer specific BCFs shown in Table 5.6.3 and are somewhat high values to use.

Table 5.6.3 Bioconcentration Factors for 2,3,7,8-Substituted PCDDs/PCDFs

Isomer	Thigh	Egg Yolk
2,3,7,8-TCDD[1]	1.11	1.27
1,2,3,7,8-PeCDD	1.11	1.27
1,2,3,4,7,8-HxCDD	0.85	1.46
1,2,3,6,7,8-HxCDD	0.99	1.62
1,2,3,7,8,9-HxCDD	0.50	1.05
1,2,3,4,6,7,8-HpCDD	0.22	0.98
OCDD	0.04	0.47
2,3,7,8-TCDF	0.92	0.46
1,2,3,7,8-PeCDF[1]	1.20	2.50
2,3,4,7,8-PeCDF	1.20	2.50
1,2,3,4,7,8-HxCDF	0.86	1.89
1,2,3,6,7,8-HxCDF	0.73	1.68
1,2,3,7,8,9-HxCDF[1]	0.73	1.68
2,3,4,6,7,8-HxCDF	0.39	0.54

Isomer	Thigh	Egg Yolk
1,2,3,4,6,7,8-HpCDF	0.18	0.68
1,2,3,4,7,8,9-HpCDF	0.16	0.49
OCDF	0.07	0.30

Source: R.D. Stephens et al., 1992.
No BCFs for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDF, or 1,2,3,7,8,9-HxCDF are presented in Stephens et al. due to low concentrations of these isomers. Values for these two isomers are taken from the most structurally similar isomer on the list.

5.8 Overall Direct and Indirect Cancer Risk

To determine the overall carcinogenic risk from all exposure pathways (except breastmilk), both direct inhalation and indirect exposure pathways, the total cancer risks for the indirect pathways (as calculated for each exposure scenario in Table 5.1.8, Table 5.2.8, Table 5.3.7, and Table 5.4.7) are added to the total cancer risk via inhalation. For each exposure scenario:

$$Overall\ Cancer\ Risk_j = Total\ Cancer\ Risk(inh)_j + Total\ Cancer\ Risk(oral)_j \qquad 5.8.1$$

where:

Overall Cancer Risk	=	Overall excess lifetime cancer risk via all routes of exposure (unitless), exposure scenario j (j=1..4)
Total Cancer Risk(inh)$_j$	=	Total excess lifetime cancer risk via inhalation (unitless, from Equation 5-4) exposure scenario j (j=1..4)
Total Cancer Risk(oral)$_j$	=	Total excess lifetime cancer risk via indirect (i.e., oral) exposures (unitless, from Tables 5.1.8, 5.2.8, 5.3.7, and 5.4.7), exposure scenario j (x=j=1..4)