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Use of Probabilistic Methods to Understand the Conservatism in California's Approach to Assessing Health Risks Posed by Air Contaminants

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Many state and federal agencies have prepared risk assessment guidelines, which describe methods for quantifying health risks associated with exposure to vapors and particulates emitted from point and area sources (e.g., California Air Pollution Control Officers Association [CAPCOA] under the Air Toxics "Hot Spots" Act [Assembly Bill 2588] and the U.S. Environmental Protection Agency [EPA] under the Clean Air Act). In general, these guidelines recommend or require the use of upperbound "point" estimates for numerous exposure parameters. This methodology yields a single risk estimate, which is intended not to underestimate the true risk and may significantly overstate it. This paper describes a risk assessment of a facility's airborne emissions using a probabilistic approach, which presents a range and distribution of risk estimates rather than a single point estimate. The health risks to residents living near a food processing facility, as estimated using techniques recommended by California AB2588, are compared to the results of a probabilistic analysis. Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were identified as the emitted chemicals of concern. The point estimate method recommended by CAPCOA resulted in estimates that were greater than the 99.99th percentile risk predicted by the probabilistic analysis. As shown in other assessments of persistent airborne chemicals, secondary or indirect exposure pathways (i.e., ingestion of beef, ingestion of cow's milk, and ingestion of mother's milk) rather than inhalation, were the greatest contributors to risk. In this analysis, the probability distributions for the cancer potency factor and ingestion of cow's milk had the largest impact on the results of the 33 exposure factors considered.

Introduction

In 1987, the State of California passed Assembly Bill 2588 (AB2588), also known as the Air Toxics "Hot Spots" Information and Assessment Act, which established a statewide program to build an inventory of air toxics emissions for individual facilities. It required that risk assessments be conducted in accordance with regional air pollution control district (RAPCD) policy and that the public be notified of the potential health risks (California Code of Regulations, Title 17, Section 93300 et seq., 1987). The regulation identified geographic areas where the incremental concentration of airborne chemicals in a community, because of point source emissions, was greater than considered acceptable. If the results of a risk assessment indicated that, for some individuals, the health risks may exceed acceptable risks established by each RAPCD, potentially exposed individuals would be publicly notified. The law was significant because it required emitters to understand the hazard posed by nearly 400 different chemicals rather than the roughly 30 chemicals that have historically been regulated by the U.S. Environmental Protection Agency (EPA).

In 1991, 1992, and 1993, the State issued AB2588 risk assessment "guidelines" through the California Air Pollution Control Officers Association (CAPCOA) and required that all parties comply to ensure that consistent methodologies were used for all risk assessments.[1-3] The guidelines describe conservative methods that allow comparison of one facility to another, expedite the review of risk assessments by regulatory

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7163

agencies, and minimize the revision and resubmittal of risk assessments. Although this approach streamlines regulatory evaluation, it encourages a generic, nearly "worst case" approach that will usually overestimate the true risk for the typical person. Not only can this lead to unnecessary costs associated with emission reductions, it can also result in reporting risks to the public that are much higher than actual risks.

The AB2588 risk assessment guidelines contain algorithms for estimating chemical concentrations in environmental media, chemical uptake from each medium, and the risks associated with exposure to each chemical.[3] Default values, published by the State of California or EPA, are provided for environmental fate, toxicity, and exposure parameters. Virtually all of California's RAPCDs require that facility operators use 'default' assumptions when conducting risk assessments. In general, the default parameters are conservative estimates representing the upperbound value within the range of plausible values. As a result, the risk estimates are almost always overestimated.[4-8] Until recently, attempts to quantify the degree of conservatism in the risk estimates have not been routinely conducted.[9]

The objective of this case study was to quantify the conservatism in the exposure estimation and risk characterization inherent in the CAPCOA methodology ("regulatory" assessment) by comparing the resulting point estimate of risk with a probability-based risk distribution generated by a Monte Carlo analysis ("probabilistic" assessment). Probabilistic analyses have been used over the past three years to assess water pollutants, contaminated soil, and other media.[10-16] The probabilistic methodology offers several advantages over the point estimate approach. First, this approach provides an accurate estimate of the upperbound or maximum plausible health risk. Rather than compounding upperbound values for each exposure parameter to arrive at a point estimate of the upperbound or maximum exposure, this approach uses statistical data specific to the distributions for the individual parameters to mathematically characterize a range of potential risks. Second, the methodology can incorporate all valid data for an individual parameter into the risk calculation (with an option for relative weighing of data), rather than limit the assessor to a single study or single data point. These features provide the risk manager, who is frequently faced with requiring costly control devices or conducting extensive remediation programs, with invaluable information that can improve the quality of decision making.

This case study evaluated a food-processing facility located on a 1,200-acre parcel in a rural area of California, approximately 2.5 miles from the center of a city with a population of 60,000. The land surrounding the facility is used for cattle grazing as well as the commercial production of crops such as sugar beets, alfalfa, and flower seeds. In this analysis, probability distributions were developed for 33 exposure parameters relevant to the seven potential exposure pathways identified. Interdependency among certain exposure parameters was considered in our analysis. Specifically, covariance was identified for beef ingestion and body weight; dairy milk ingestion and body weight; and mother's milk ingestion and infant body weight. A probability distribution for the cancer potency factor (CPF) for 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) was developed for the analysis based on a recently published reassessment of the dose-response data.[17]

Although as many as 30 chemicals were emitted from the stack at this facility, an assessment showed that polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) were the only chemicals that could pose health risks of regulatory concern because of their relative toxicity and persistence in the environment. The PCDD/PCDF emissions resulted from the use of a coal-burning kiln for processing food products, and emissions were quantified by source testing. Because PCDD/PCDFs are listed in the AB2588 risk assessment guidelines as "multipathway" chemicals (i.e., chemicals emitted as particulates that undergo deposition and can enter the food chain), the assessment accounted for relevant non-inhalation exposure pathways.

Methodological Approach

For comparison purposes, the predicted uptake of PCDD/PCDF emissions and subsequent risk was estimated first using the point estimate approach and specific assumptions required by CAPCOA (regulatory assessment). This approach used all required exposure parameter values and the California CPF for 2,3,7,8-TCDD. The results were compared to a distribution of risk that was developed using a Monte Carlo analysis (probabilistic assessment). The probabilistic assessment relied upon distributions for all key exposure parameters and a distribution recently published for the cancer slope factor for 2,3,7,8-TCDD (that has been accepted by FDA).[17,18]

Figure 1 illustrates the approach to the comparative study and the interdependency of the exposure pathways. The regulatory and the probabilistic assessments were based upon the equations in the AB2588 risk assessment guidelines,[3] which were not specifically developed for risk assessment of particulate emissions from a combustor. The guidelines do not require the characterization of health risks to children ages one to 18—a refinement that would complicate the analyses.

The regulatory assessment was conducted using an Excel™ spreadsheet to estimate multipathway exposure using required regulatory algorithms.[3,19] The Monte Carlo analysis was conducted using the same Excel spreadsheet and the Excel add-on program @Risk™.[20] The validity of our dose estimates was verified by checking them against the regulatory assessment using the ACE2588 computer program, which was developed by the Santa Barbara Air Pollution Control District[21] specifically for use in multipathway exposure analyses for AB2588.

Dose-Response Assessment

Studies focusing on the structure-activity relationships of the various PCDD/PCDF congeners have shown that not all PCDDs and PCDFs are equally potent.[22,23] This complicates the risk assessment process as PCDDs and PCDFs are generally found in the environment as mixtures containing any number of 210 possible congeners. Accordingly, an interim approach for risk assessment has been adopted by EPA[24,25] for evaluating the health risks associated with PCDD/PCDFs. This method normalizes the potency of individual congeners relative to 2,3,7,8-TCDD, as this congener is the most potent one.[26] Only 2,3,7,8-substituted congeners are assigned TEFs because numerous structure-activity and structure-binding studies have established that non-2,3,7,8-substituted congeners are relatively inactive in biological systems.[22,27,28] To obtain a total 2,3,7,8-TCDD toxicity equivalent (TEQ) value for a given PCDD/PCDF mixture, each 2,3,7,8-congener concentration is multiplied by the appropriate TEF, and then the TEQs are summed. The 2,3,7,8-TCDD TEQ for the mixture is the sum of the individual 2,3,7,8-TCDD TEQ for each congener.

The oral CPF for 2,3,7,8-TCDD adopted by the Cal-EPA[29] and used by CAPCOA[3] is 133,000 $(mg/kg\text{-}day)^{-1}$. This CPF was used in the regulatory (point estimate) assessment and represents a conservative estimate of the cancer potency in humans based on animal bioassays. The CPF is based on the 95th percentile upper confidence limit of the slope of the dose-

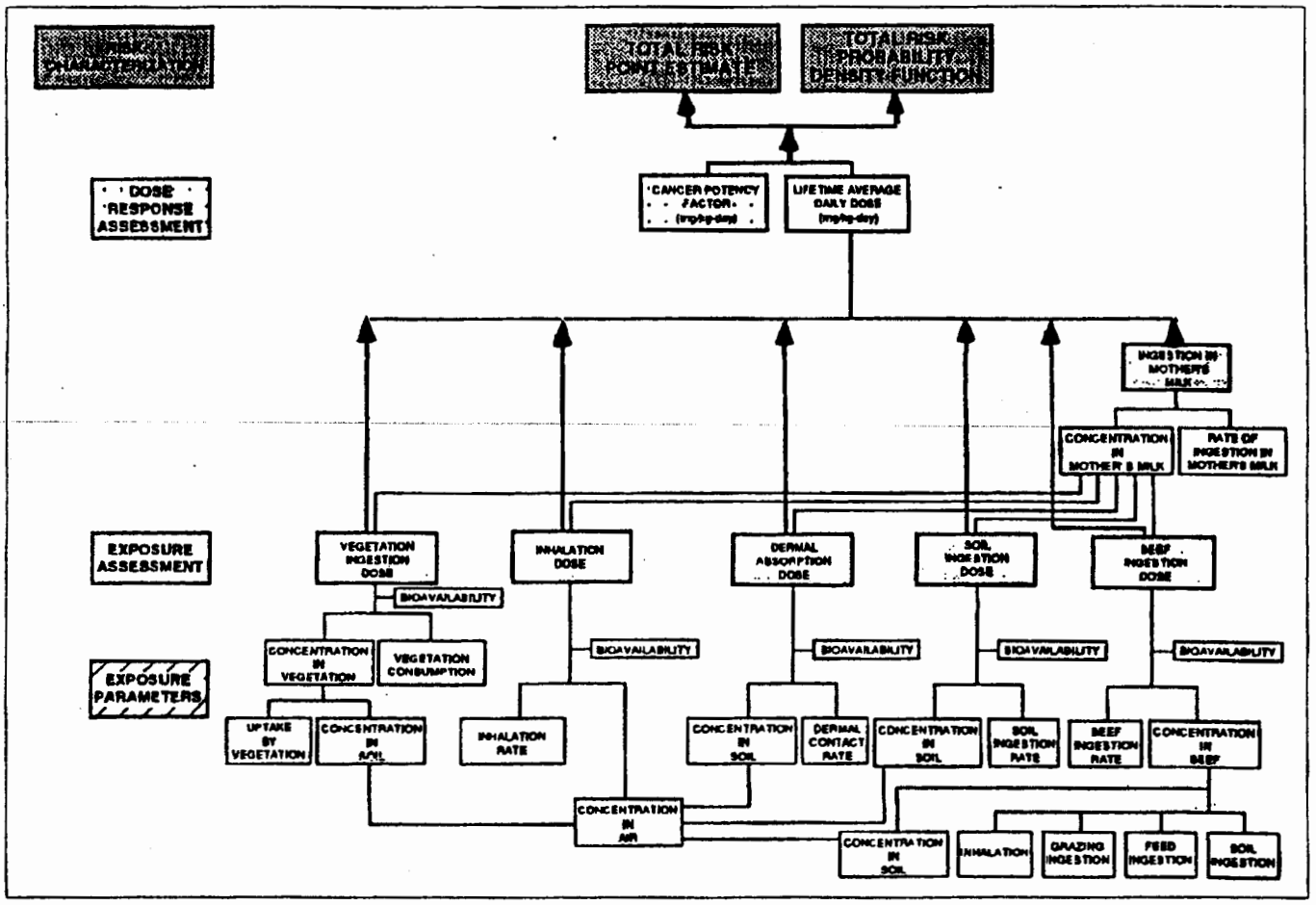

Figure 1. Components of the multipathway risk assessment.

response curve predicted with the linearized multistage (LMS) model. For these reasons and others, a lack of confidence in the CPF value has contributed to uncertainties in the health risks associated with exposure to PCDDs and PCDFs.[11,13,17] The current Cal-EPA CPF for 2,3,7,8-TCDD is based on the original (1970) histopathological evaluation criteria for tumor pathology rather than the current (1986) National Toxicology Program classification scheme for proliferative lesions in rat liver.[30]

The CPF for 2,3,7,8-TCDD has been evaluated using the results of the histopathological reevaluation.[18,31] Using survival-adjusted tumor incidence data, the LMS model, and scaling up to humans using body weight rather than surface area,[31] the CPF for 2,3,7,8-TCDD was estimated to be 2,700 (mg/kg-day)$^{-1}$ based on the incidence of hepatocellular carcinomas, and 9,700 (mg/kg-day)$^{-1}$ when hepatocellular carcinomas and adenomas were combined. When the data were not adjusted for survival rates, the estimates ranged from 1,500 (mg/kg-day)$^{-1}$ to 8,200 (mg/kg-day)$^{-1}$. The reevaluated CPF has been well received over the past two and one-half years since at least ten states have adopted it. The FDA (Docket No. 93N-0352) has proposed to adopt a value based on a 1 pg/kg-day (9 in 1,000,000) acceptable risk.[18] This is equivalent to a cancer potency slope factor of 9,000 mg/kg-day^{-1}, whereas the current EPA cancer slope factor is 156,000 mg/kg-day^{-1}.

For this assessment, we developed a distribution for the CPF[17] based on the current histopathology criterion using a version of the LMS model, which allows model output to be expressed as a distribution[32]; this distribution was used in the probabilistic assessment. Table I presents the CPF values predicted for specific percentiles. Figure 2 shows the probability distribution for the CPF, which approximates a normal distribution. The 50th and 95th percentiles of this distribution are approximately 7,000 and 9,700 (mg/kg-day)$^{-1}$, respectively.

Exposure Assessment

Components of the exposure assessment for AB2588 include quantification of emissions, environmental fate and transport modeling, identification of the exposed population (conservatively represented in this case by the maximum exposed individual), identification of exposure pathways, and the estimation of chemical uptake (dose) by the exposed population.

Concentrations in PCDDs/PCDFs in Airborne Emissions

The PCDD/PCDF emissions from the kiln were quantified and speciated during a stack test.[33] Annual emission rates were estimated using the results from the stack test, the amount of material processed during the stack test, and the total amount of material processed annually. Emission rates for PCDDs/PCDFs were converted to a 2,3,7,8-TCDD TEQ for input into the air dispersion model.

Environmental Fate and Transport

The concentrations of airborne PCDD/PCDF (2,3,7,8-TCDD TEQs) for locations of interest were estimated using the Industrial Source Complex Short Term (ISCST) air dispersion

Table I. Cumulative distribution for the cancer potency factor (CPF) for 2,3,7,8-TCDD based on the bioassay results in reference 17.

Percentile	CPF (mg/kg-day)$^{-1}$
0.176	0
0.437	1,000
1.05	2,000
2.42	3,000
5.39	4,000
11.8	5,000
23.3	5,830
27.1	6,000
51.7	7,000
74.2	8,000
88.8	9,000
96.0	10,000
98.8	11,000
99.7	12,000
99.9	13,000

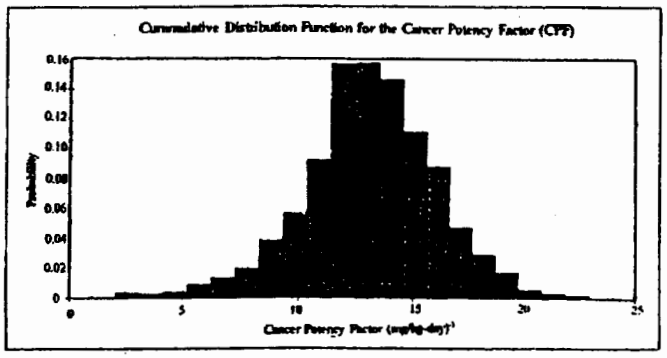

Figure 2. Probability distribution for the cancer potency factor for 2,3,7,8-TCDD, based on the results from the linearized multistage model MSTAGE (Crouch, 1992) and histopathological reevaluation of the Kociba cancer bioassay data (Keenan et al., 1991).

model and the annual average emission rate. The model accounts for meteorological conditions, source type, geography, and the effects of nearby buildings.

Exposure Scenario

A residential scenario was evaluated. For purposes of simplicity and conservatism, health risks were characterized for the location of maximum impact, i.e., the residence that might receive the highest airborne concentration of particulates.

Based on the facility setting and relevant site-specific data, exposure to PCDD/PCDF emissions was evaluated for inhalation of airborne particulates for the following pathways: soil ingestion; dermal contact with soil; consumption of home-grown produce; consumption of locally produced beef and milk; and consumption of mother's milk by infants (Figure 1). Beef and dairy cattle were observed near the facility and represent the only potential exposure via feed stock. Goats, chickens, and pigs were not observed within 10 kilometers of the facility. Because home-grown produce represents a much greater potential for exposure to most airborne chemicals than does commercial produce,[34] only consumption of home-grown vegetables from residential gardens was considered. Commercial agriculture from any one area is generally expected to constitute only a negligible portion of a person's total diet.

Exposure Parameter Distributions

Probability distributions were developed for the critical exposure parameters for each of the exposure pathways. Based on a preliminary sensitivity analysis, the following five exposure pathways were identified as most significant: particulate inhalation, beef consumption, ingestion of dairy milk, ingestion of home-grown vegetables, and mother's milk ingestion. This is consistent with the results of other multipathway dioxin risk assessments.[34,35]

The primary studies cited in the AB2588 risk assessment guidance were used to develop probability distributions when data were adequate. When data were inadequate, a review of

Table II. General human exposure parameters used to estimate uptake via all pathways for the regulatory and probabilistic assessment. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution		
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors	Reference
EF	Exposure frequency (fraction of a year; used in inhalation, soil ingestion, and dermal absorption pathways)	1	Uniform	Min.: 0.58 Max.: 1	CAPCOA, 1993 EPA, 1990
ED	Exposure duration (years)	26 for mother in mother's milk pathway; 44 for adult exposed via mother's milk pathway.	Discrete	Years / Percentile 0-1 / 7.5% 1-3 / 16.9% 3-13 / 40.2% 13-18 / 11.0% 18-23 / 7.9% 23-33 / 9.5% > 33 / 7.0%	CAPCOA, 1993 EPA, 1990
ABW	Average body weight (kg)	70	Normal	Mean: 64.2 S.D.: 13.19	CAPCOA, 1993 Snyder, 1984
AT	Averaging time (days)	25,550	Static value	N/A	CAPCOA, 1993

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum

Table III. Equations and exposure parameters used to estimate uptake via the inhalation pathway for both the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution			
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors		Reference
RR	Respiration rate (m^3/D)	20	Not critical to uncertainty	N/A		CAPCOA, 1993
BIOinh	Fraction of particulates retained in lung	1	Uniform	Min: 0.46	Max: 1.0	ICRP, 1969

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum
GLC = Ground-level concentration based on air dispersion modelling

Equation:
Dose-inh = RR*GLC*BIOinh*ED*EF/(ABW*AT*1,000).

Table IV. Equations and exposure parameters used to estimate uptake via the soil ingestion pathway for both the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution			
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors		Reference
GLC	Ground-level concentration (mg/m^3)	Use air modeling results	N/A	N/A		N/A
Dep-rate	Vertical rate of deposition (m/day)	4,320	Lognormal	Mean: 300	S.D.: 3	CAPCOA, 1993 McMahon and Denison, 1979 McKone and Ryan, 1989
$t_{1/2}$	Half-life of contaminant in soil (D)	4,380	Uniform	Min: 3,285	Max: 18,250	Young, 1983
SD	Soil mixing depth (m)	0.15 (agricultural setting)	Uniform	Min: 0.1	Max: 0.15	CAPCOA, 1993
BD	Soil bulk density (kg/m^3)	1,333	Uniform	Min: 1,000	Max: 1,800	Brady, 1974
Is	Lifetime average soil ingestion rate (mg/D)	110	Not critical to uncertainty	N/A		CAPCOA, 1993
GI	Gastrointestinal absorption factor	1.0	Not critical to uncertainty	N/A		CAPCOA, 1993
BIOing	Bioavailability	0.43	Uniform	Min: 0.39	Max: 0.49	CAPCOA, 1993 Shu, et al., 1988

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum
Cs = Average soil concentration (µg/kg)
DEP = Deposition (mg/m^2/D)
Ks = Soil elimination constant
Tt = End of evaluation period [25,500 (D)]
To = Beginning of evaluation period [0(D)]

Equations:
Cs = DEP*X/(Ks*SD*BD*Tt)
DEP = GLC*Dep-rate
$X = [(e^{(-Ks*Tt)} - (e^{(-Ks*To)})/Ks] + Tt$
Ks = 0.693/$t_{1/2}$
Dose-soil inges = Cs*Is*GI*BIOing*EF*ED*1e-06/(ABW*AT*1000)

the scientific literature was conducted to identify appropriate distributions. Of the 49 exposure parameters contributing to site-specific risks, distributions for 33 parameters were developed. Sixteen parameters were judged as particularly significant in this assessment: (1) particulate deposition rate, (2) crop yield, (3) vegetable interception fraction, (4) vegetable weathering constant, (5) beef cattle diet-to-meat uptake factor, (6) soil ingestion by grazing, (7) dairy cattle diet-to-milk uptake factor, (8) inhaled particulate retention factor, (9) produce consumption rate, (10) beef consumption rate, (11) percentage of beef from local sources, (12) dairy milk consumption rate, (13) percentage local dairy milk, (14) mother's milk consumption rate, (15) exposure frequency, and (16) exposure duration. The other 17 distributions were used in the calculation but did not have an appreciable impact on the results and therefore could have been addressed by using a point estimate approach.

For the regulatory and probabilistic assessments, doses were estimated using the recommended CAPCOA equations.[3]

Table V. Equations and exposure parameters used to estimate uptake via the dermal absorption pathway for the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution		
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors	Reference
SA	Surface area of exposed skin (cm^2)	4,656	Not critical to uncertainty	N/A	CAPCOA, 1993
SL	Soil loading on skin ($mg/cm^2/day$)	0.5	Not critical to uncertainty	N/A	CAPCOA, 1993
ABS	Fraction of contaminant absorbed across skin	0.02	Not critical to uncertainty	N/A	CAPCOA, 1993

N/A = Not applicable
Cs = Concentration in soil (See soil ingestion pathway.)

Equation:
Dose-dermal = Cs*SA*SL*ABS*EF*ED/(ABW*AT*1e+09)

Table VI. Equations and exposure parameters used to estimate uptake via the vegetable ingestion pathway for both the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution			
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors		Reference
IF	Interception fraction	Crop specific Leafy=0.2 Vine=0.1 Pasture grasses not provided, 0.5 estimated	 Uniform Uniform Uniform	 Min: 0.15 Min: 0.034 Min: 0.14	 Max: 0.39 Max: 0.073 Max: 0.84	CAPCOA, 1993 (except pasture grasses) Baes et al., 1984
$t_{1/2}$	Half-life of contaminant on plant(D)	14	Truncated lognormal	Mean: 10 Min: 2.8	S.D.: 3 Max: 34	CAPCOA, 1993 Baes et al, 1984
Y	Yield (kg/m^2)	Crop specific Leafy=2 Vine=2 Pasture grasses = 2	 Uniform Uniform Uniform	 Min: 2 Min: 2 Min: 0.005	 Max: 4.4 Max: 4.4 Max: 0.63	CAPCOA, 1993 Baes et al., 1984
T	Growth period (D)	Crop specific Leafy=90 Vine=90 Pasture grasses = 90	 Uniform Uniform Uniform	 Min: 45 Min: 45 Min: 14	 Max: 90 Max: 90 Max: 45	CAPCOA, 1993 Clement, 1988 Baes et al., 1984
BIO	Bioavailability	1	Point estimate	N/A		CAPCOA, 1993
UF2	Uptake factor based on soil concentration	N/A	N/A	N/A		CAPCOA, 1993
Ifp	Rate of plant consumption (kg/D)	Vine = 0.25 Leafy = 0.01	Normal (covaried with body weight based on a normal distribution for kg ingestion per kg body weight per day)	Ifp = ABW* (ingestion/body weight-day) Mean: 0.004 kg/kg-day S.D.: 0.00038		CAPCOA, 1993 Clement, 1980 USDA, 1985 McKone and Ryan, 1989
Lp	Fraction of home-grown plants	Site specific Vine: 0.25 based on APCO Leafy: 0.25 based on APCO	Uniform	 Min: 0.4 Min: 0.04	 Max: 0.75 Max: 0.2	Santa Barbara Air Pollution Control District, EPA, 1989
Glp	Gastrointestinal absorption factor	1.0	Not critical to uncertainty	N/A		CAPCOA, 1993

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum
Cf = Concentration in vegetation (µg/kg)

Dep = Deposition (µg/m²/D)
Cdepv = Concentration due to direct deposition (µg/kg)
Cs = Average concentration in soil (mg/kg)
Ctrans = Concentration due to root uptake (µg/kg)
k = Weathering constant $(D)^{-1}$

Equations:
Cf = Cdepv*BIO + Ctrans
Cdepv = [Dep*IF/(k*Y)]* (1-e^-kT)
Ctrans = Cs *UF2
k = 0.693/$t_{1/2}$
Dose-plt inges = Cfp*Ifp*Glp*Lp*ED/(ABW*AT*1000)

Table VII. Equations and exposure parameters used to estimate uptake via the beef ingestion pathway for both the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameter			Distribution			
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors		Reference
RR	Inhalation rate (m^3/D)	80	Not critical to uncertainty	N/A		CAPCOA, 1993
%G	Percent diet by grazing	Site specific (90 used)	Uniform	Min: 90	Max: 95	CAPCOA, 1993 Jensen, 1991
Fib	Feed ingestion rate (kg/D)	8	Normal	Mean: 8	S.D.: 2.2	CAPCOA, 1993 NRC, 1965 McKone and Ryan, 1989
L	Percent of feed locally grown (not from pasture)	Site specific (0 used)	N/A	N/A		CAPCOA, 1993 Jensen, 1991
%Sf	Soil ingested as a percent of feed	1.0	Not critical to uncertainty	N/A		CAPCOA, 1993 Jensen, 1991
%Sp	Soil ingested as a percent of pasture grass ingested	5	Uniform	Min: 1.1	Max: 17.5	CAPCOA, 1993 McDowell, 1985
Fib	Transfer coefficient from feed to meat (D/kg)	0.4	Discrete	(0.1, 0.4)		CAPCOA, 1993 Jensen, 1981 Clement, 1988 Fries and Paustenbach, 1990
Ifb	Rate of beef consumption (kg/D)	0.100	Normal (covered with body weight based on a normal distribution for kg ingestion per kg body weight per day	Ifb = ABW*(Ingest/body weight-day) Mean: 0.003	S.D.: 0.00062	CAPCOA, 1993 USDA, 1982 Clement, 1988
Lb	Fraction of beef locally produced	Site specific, estimated value 0.10	Uniform	Min: 0.065	Max: 0.163	Jensen, 1991 Ensminger, 1976
Glb	Gastrointestinal absorption factor	1.0	Not critical to uncertainty	N/A		CAPCOA, 1993

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum
CLC = Ground-level concentration (µg/m^3)
Cf = Concentration in feed (µg/kg) (See concentration in vegetable pasture grasses.)
Sf = Soil ingestion rate (kg/D)

Equations:
Cfa = (Inhalation + Feed ingestion + Grazing ingestion + Soil ingestion)*Fib
Inhalation = RR*GLC
Feed ingestion = (1-%G)*Fib*L*Cf
Grazing ingestion = %G*Cf*Fib
Soil ingestion = Sf*Cs
Sf = [(1-%G)*%Sf*Fi] + %G*%Sp*Fi
Dose-beef inges = Cfb*Ifb*Glb*Lb*ED/(ABW*AT*1000)

The bases for the 33 exposure parameter distributions used to estimate dose in the probabilistic assessment are presented in Tables II-IX and in Appendix A.

Risk Characterization

The Monte Carlo simulation was used to calculate the pathway-specific Lifetime Average Daily Dose (LADD), the total LADD, and the incremental lifetime cancer risk (ILCR). The ILCR was calculated using the following equation[3,36]:

$$\text{Risk (ILCR)} = \text{CPF} \times [\text{LADD}_{inh} + \text{LADD}_{soil\ inges.} + \text{LADD}_{dermal} + \text{LADD}_{veg.\ inges.} + \text{LADD}_{beef\ inges.} + \text{LADD}_{dairy\ milk} + \text{LADD}_{m.milk}]$$

The results of the probabilistic assessment are presented in Table X and Figures 3a and 3b. They indicate that the 50th and 95th percentile LADDs for the location receiving the highest deposition of particulates from the facility were 2.3×10^{-11} mg/kg-day and 2.9×10^{-10} mg/kg-day, respectively. Using the point estimate approach, the estimated LADD was 9.0×10^{-10} mg/kg-day (Figure 3a). In short, the CAPCOA methodology predicted an intake of TCDD three-fold higher than the 95th percentile dose and about 40-fold greater than the 50th percentile dose.

When the probability distribution for the CPF for 2,3,7,8-TCDD is considered in the probabilistic assessment, the predicted 50th percentile ILCR is 1.5×10^{-7}, and the predicted 95th percentile ILCR is 2.0×10^{-6}. The ILCR estimated in the

Table VIII. Equations and exposure parameters used to estimate uptake via the dairy milk ingestion pathway for both the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution			
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors		Reference
RR	Inhalation rate (m^3/D)	80	Not critical to uncertainty	N/A		CAPCOA, 1993
%G	Percent diet by grazing	Site specific (90 used)	Uniform	Min: 90	Max: 95	Jensen, 1991
FI	Feed ingestion rate (kg/D)	16	Point estimate	N/A		CAPCOA, 1993
L	Percent of locally grown feed (not from pasture)	Site specific (0 used)	N/A	N/A		CAPCOA, 1993 Jensen, 1991
%St	Soil ingested as a percent of feed	1	Not critical to uncertainty	N/A		Jensen, 1991
%Sp	Soil ingested as a percent of pasture ingested	5	Uniform	Min: 1.1	Max: 17.5	CAPCOA, 1993 McDowell, 1985
Flm	Transfer coefficient from feed to milk (D/L)	0.04	Discrete	(0.09, 0.032, 0.008)		CAPCOA, 1993 Jensen, 1982 Clement, 1988 Fries and Paustenbach, 1990
Ifm	Consumption of dairy milk (L/day)	0.30	Normal (covaried with body weight based on a normal distribution for kg ingestion per kg body weight per day	Ifb = ABW* (ingestion/body weight-day) Mean: 0.0033 kg/kg-day	S.D.: 0.00037	CAPCOA, 1993 USDA, 1982
Lm	Fraction of dairy milk locally produced	0.21	Uniform	Min: 0.067	Max: 0.21	Jensen, 1991 Ensminger, 1976
Glm	Gastrointestinal absorption factor	1.0	Not critical to uncertainty	N/A		CAPCOA, 1993

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum
GLC = Ground-level concentration (μg/m^3)
Cf = Concentration in feed (μg/kg) (See vegetation pathway-pasture grasses)
SI = Soil ingestion rate (kg/D)

Equations:
Cfa = (Inhalation + Feed ingestion + Grazing ingestion + Soil ingestion)*Flm
Inhalation = RR*GLC
Feed ingestion = (1-%G)*Flm*L*Cf
Grazing ingestion = %G*Cf*Flm
Soil ingestion = SI*Cs
SI = [(1-%G)*%St*FI] + %G*%Sp*Flm
Dose-dairy milk inges = Cfm*Ifm*GI*L*ED/(ABW*AT*1000)

regulatory assessment is 8 x 10^{-5}, a value about 500-fold higher than 50th percentile risk. The inclusion of a probability distribution for the CPF in the probabilistic assessment reduced the estimated 95th percentile risk by approximately 40-fold. The ILCRs estimated by the regulatory and probabilistic assessments are compared in Figure 4.

As shown in both the regulatory and probabilistic assessments, the indirect (secondary) exposure pathways are primarily responsible for potential health risks associated with PCDD/PCDF emissions (Figures 5a and 5b). These results are in agreement with the analyses of Stevens and Gerbec[35] and of Fries and Paustenbach[34] and are consistent with the recent EPA analysis.[25] It should be noted that pathway contributions are different for the deterministic and probabilistic assessments due to the variability in the percentile value represented by the point estimate.

The probabilistic analysis suggested that the 95th percentile concentration of TCDD TEQ in soil is 0.2 ppt. This soil concentration did not produce significant human exposure via the direct exposure pathways (such as soil ingestion, dermal contact, and inhalation of fugitive dust emissions). Interestingly, the range of predicted soil concentrations (in the low parts per trillion range) plausibly resulting from the emissions are characteristic of background levels of the PCDD/

Table IX. Equations and exposure parameters used to estimate uptake via the mother's milk ingestion pathway for both the regulatory and probabilistic assessments. The type of distribution, relevant descriptors, and the basis for the PDFs used are presented.

Parameters			Distribution			
Abbreviation	Description	CAPCOA Value	Type	Relevant Descriptors		Reference
$t_{1/2}$	Half-life of contaminant in mother (D)	2,117	Discrete	(1,630; 2,120)		Poiger and Schlatter, 1986
f1	Fraction of contaminant that partitions to mother's fat	0.9	Uniform	Min: 0.8	Max: 0.9	Smith, 1987
f3	Percent fat in mother's milk	4.0	Uniform	Min: 2.0	Max: 4.0	Clement, 1988
f2	Percent mother's weight that is fat	33	Normal	Mean: 29.5	S.D.: 1.12	Butte et al, 1991
DERm	Daily mother's milk ingestion rate (kg/D)	0.9	Normal (covaried with body weight based on average kg intake per kg body weight per day)	DERm=ABW*ingestion/body weight-day Average ingestion per kilogram 87.3 kg/kg-day		Butte, 1984 Darling, 1993
F	Frequency of exposure (D/YR)	365	Uniform	Min: 180	Max: 365	LaLeche, 1991
YR	Breast feeding period (YR)	1	Point estimate	N/A		CAPCOA, 1991
ABW_{infant}	Average body weight (kg)	6.5	Normal	Mean: 8.6	S.D.: 0.0129	EPA, 1989

N/A = Not applicable
SD = Standard deviation
Min = Minimum
Max = Maximum
Cm = Concentration in mother's milk (mg/kg)
EmI = Average maternal intake (mg/kg milk/D) (from other exposure pathways)

Equations:
$Cm = EmI * t_{1/2} * f1 * f3/(f2 * 0.693)$
$Dose\text{-}m.milk = Cm * DERm * F * YR/(25000 * ABW_{infant})$

PCDFs.[25,37,38] The predicted level in soils adjacent to the facility was compared with the typical regulatory action levels in residential settings and the typical background levels in rural areas. Results support the conclusion that PCDD/PCDF emissions from incinerators with proper air pollution control devices will rarely pose a significant health concern due to chemical uptake into food and animal pathways.

The results of exposure estimates can often be validated using field data. For example, in the regulatory assessment, consumption of dairy milk represented the greatest single contribution to the total cancer risk (47%), while about 40% of the dose was associated with ingestion of mother's milk. However, studies have shown that the uptake of PCDD/PCDF by grazing animals is less than would be expected; much of the PCDD/PCDF is never absorbed by the cows because of the strong chemical adsorption to soil, biodegradation in the ruminant gut, and, to a lesser extent, photodecomposition.[34] With the availability of highly sensitive analytical methods, laboratory analysis of meat and dairy milk is recommended in cases where significant animal body burdens are predicted by risk assessments, which rely upon default transfer coefficients.

Conclusions

This assessment illustrates the degree of conservatism in the current California regulatory approach for the assessment of toxic air contaminants. In our case study, the point estimate (regulatory) approach predicted a cancer risk that would be deemed significant (greater than 10^{-5}); the 95th percentile cancer risk estimate, characterized by the probabilistic assessment, is well below this value. Most point estimates suggested by CAPCOA were at the 95th percentile of the distribution. Thus the result yielded risks greater than the 99th percentile. Based on the point estimate approach, this facility would be required to notify the public that a "significant" health risk was present and would likely be required to implement several emission controls.

Our assessment indicates that the indirect (secondary) exposure pathways (beef, dairy milk, vegetable, and mother's milk consumption) compose approximately 95% of the total dose. Estimates of the dose caused by these pathways have the highest degree of uncertainty because of the number of parameters that are considered and the compounded use of conservative assumptions. The uptake of PCDD/PCDF estimated by the probabilistic method could, in fact, be less than estimated because when the data were inadequate to assign a specific distribution, the data were conservatively represented as a uniform distribution. Therefore, future work should emphasize better characterization of secondary pathway parameters. A quantitative assessment of uncertainty of the emission estimates and air dispersion modeling parameters would more completely characterize these results, but such work goes beyond this analysis.

This paper shows how the application of probabilistic techniques can provide a more complete characterization of a potential health hazard than that presented using a point estimate approach, and offers substantial insight into quantifying the conservatism in typical risk assessment methods. Fortunately, both EPA and the Food and Drug Administration have begun to consider assessments that rely upon probabilistic methods, even though the results of assessments based on the point estimate approach must usually accompany the submission. It is anticipated that state and local agencies will soon be receptive.[39] For example, in 1992 EPA issued a memorandum encouraging the agency to consider the results of probabilistic risk assessments.[40] To encourage acceptance of this methodology by regulatory agencies, standardized, scientifically verified parameter distributions must be developed for health risk assessments.

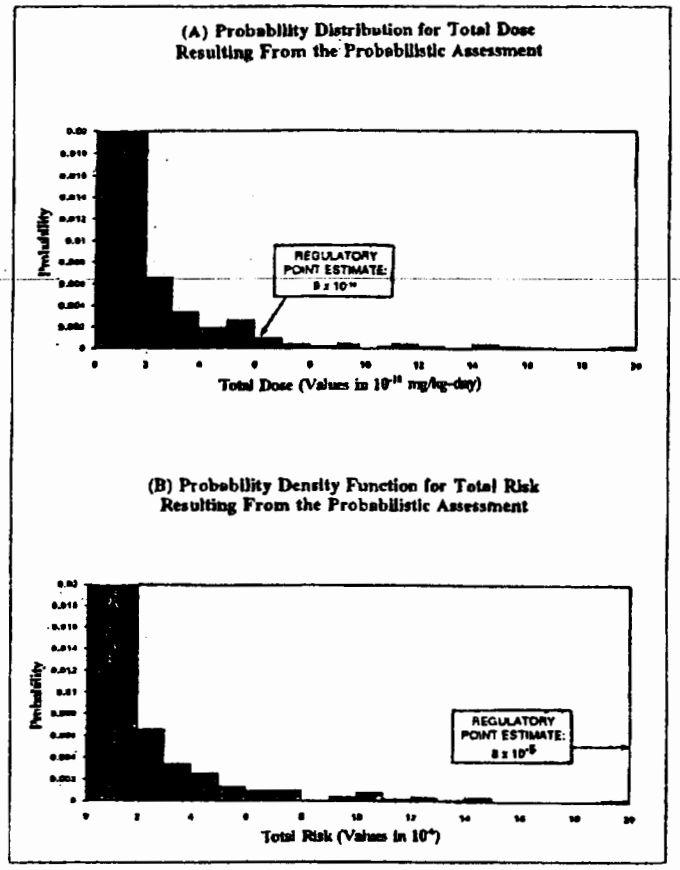

Figure 3. Probability distributions representing (A) total dose (uptake) (mg/kg-day) and (B) total risk for the maximum exposed individual living within 10 kilometers of the facility resulting from particulate emissions. The point estimate resulting from the regulatory assessment is indicated on each chart.

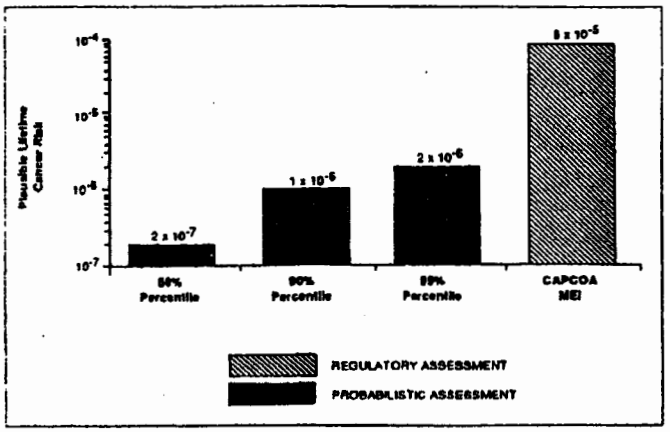

Figure 4. A comparison of the plausible increased lifetime cancer risk (ILCR) at the 50th, 90th, and 95th percentiles as predicted in the probabilistic assessment compared with the point estimate of risk predicted using California's recommended approach for compliance to AB2588. This degree of difference in estimated risk between a regulatory point estimate approach and a Monte Carlo approach can be expected for nearly all assessments of PCDD/PCDFs in aerial emissions from incinerators, combustors, and resource recovery units.

Acknowledgments

The authors would like to thank Susanne Furtado and Lisa Kent for their assistance in preparing this manuscript.

Appendix A – Exposure Parameters and Associated Probability Distributions

Inhalation of Particulates

For the regulatory and probabilistic assessments, exposure via inhalation is estimated using the algorithm[3] and exposure parameters presented in Tables II and III. In accordance with

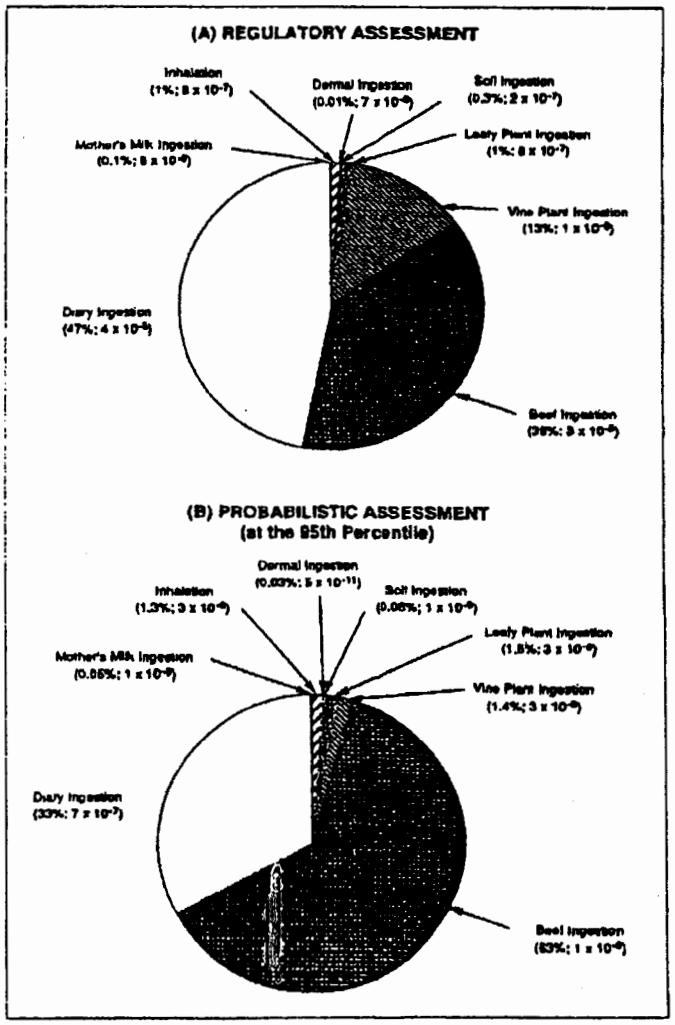

Figure 5. The pie charts represent the fractional contributions to risk for each exposure pathway. Pathway-specific risks resulting from the regulatory assessment are depicted in (A); those from the probabilistic assessment are depicted in (B).

the approach suggested by CAPCOA,[3] it was assumed that 100% of inhaled particulates are retained in the lung (BIO_{inh}); however, only a fraction of inhaled particulates are retained in the lung because a large portion of the mass of suspended particulates is exhaled and/or swallowed.

The probabilistic assessment accounted for particulate retention in the lung and transport to the stomach. For example, it is known that 24–37% of all inhaled particles having a mean diameter of 2 μm are retained in the lung.[41] For the particulates inhaled but not retained in the lung, approximately 25% are exhaled and 38–51% are eventually swallowed.[41] To accurately estimate uptake via ingestion of inhaled particulates, the oral bioavailability factor must be considered. Based on published work,[42] a value of 43% was used, which is consistent with most published assessments of PCDD/PCDF. The percentage of inhaled particulates retained in the lung or ingested following inhalation was also accounted for and was characterized by a uniform distribution with a range of 46–59%. This percentage accounts for the fraction retained in the lung, the fraction swallowed, and the oral bioavailability applicable to the swallowed fraction. To ensure that the CAPCOA[3] value was included as a plausible value within this range, a uniform distribution of 46–100% was used in the probabilistic analysis.

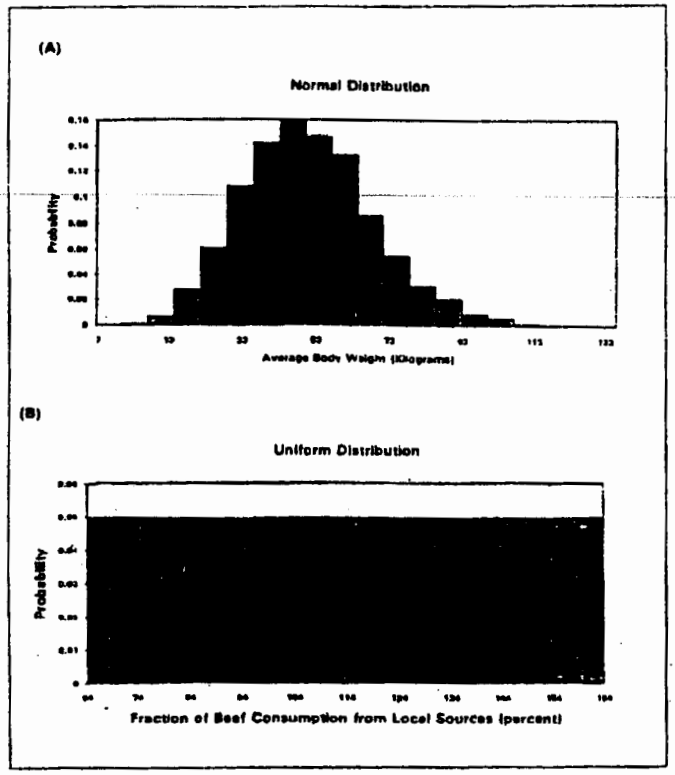

Figure 6. Probability distributions for (A) body weight (Snyder, 1984), and (B) fraction of diet composed of locally grown beef (Jensen, 1991; Ensminger, 1976).

Table X. The calculated plausible increased lifetime average daily dose (LADD) and the associated plausible cancer risk posed by dioxin emissions from a combustor for the maximally exposed person based on the probabilistic assessment.

Percentile	Total Dose	Total Risk
5	1.3×10^{-12}	7.4×10^{-9}
10	2.5×10^{-12}	1.5×10^{-8}
15	3.9×10^{-12}	2.5×10^{-8}
20	5.4×10^{-12}	3.5×10^{-8}
25	7.5×10^{-12}	4.8×10^{-8}
30	9.9×10^{-12}	6.2×10^{-8}
35	1.3×10^{-11}	7.8×10^{-8}
40	1.6×10^{-11}	8.8×10^{-8}
45	1.9×10^{-11}	1.2×10^{-7}
50	2.3×10^{-11}	1.5×10^{-7}
55	2.7×10^{-11}	1.8×10^{-7}
60	3.3×10^{-11}	2.3×10^{-7}
65	4.0×10^{-11}	2.7×10^{-7}
70	5.0×10^{-11}	3.4×10^{-7}
75	6.2×10^{-11}	4.3×10^{-7}
80	8.0×10^{-11}	5.5×10^{-7}
85	1.1×10^{-10}	7.5×10^{-7}
90	1.6×10^{-10}	1.1×10^{-6}
95	2.9×10^{-10}	2.0×10^{-6}

The CAPCOA[3] guidelines require the use of an exposure duration (ED) of 70 years. In our probabilistic assessment, the range and distribution for ED was developed based on a survey conducted by the U.S. Census Bureau and previously evaluated by EPA.[43,44] Several published assessments have adopted similar distributions.[13,15] The percentage of persons living in current residences for specified ranges of time was also considered. These data were presented as a cumulative distribution ranging from a minimum residency of one year (7.5th percentile) to greater than 33 years (93rd percentile). The same range and distribution for exposure duration was used for all exposure pathways. Based on the probabilistic assessment, the 50th and 95th percentile doses resulting from inhalation were 7.09×10^{-13} and 3.77×10^{-12} mg/kg-day, respectively, compared to 5.69×10^{-12} mg/kg-day estimated using the point estimate approach (e.g., the point estimate was similar to the 95th percentile value).

Ingestion of Soil

Uptake of PCDD/PCDF via ingestion is dependent upon the concentration of 2,3,7,8-TCDD TEQ in soil and the quantity of soil ingested. The CAPCOA[3] guidelines provided the equation to estimate the steady-state concentration of 2,3,7,8-TCDD TEQ in soil caused by aerial deposition for both the regulatory and probabilistic assessments. This equation and the parameters we used are presented in Tables II and IV. When the source of aerial contamination is from a single point, the contaminant concentration of the soil can be reasonably predicted at specified distances by accounting for dispersion and deposition. The deposition velocity (Dep-rate) is dependent on particle size.[45,46] Particles less than 5 μm in diameter (kiln emissions were approximately 2 μm) have been characterized as having a deposition velocity between 0.003 and 1 cm/sec (2.6 and 864 m/day, respectively), a rather broad range.[45] A statistical review of these data and those reported in another study[34] suggested that the deposition velocity of particulates less than 5 μm is lognormally distributed, with a geometric mean of 300 m/day and a standard deviation of 3.[46] This value includes both wet and dry deposition processes because both of these processes contribute to the removal of atmospheric PCDD/PCDF.[47] Because the McKone and Ryan analyses[46] are generally accepted, these values were used in the probabilistic assessment. The CAPCOA default value of 5 cm/sec (4320 m/day) was used in the regulatory assessment.

For the soil ingestion pathway, the CAPCOA[3] default exposure values (with the exception of soil concentration, exposure frequency, and exposure duration) were used to estimate uptake via soil ingestion (e.g., 100 mg/day) in both the regulatory and probabilistic assessments and are presented in Table IV. Based on the probabilistic assessment, the uptakes via ingestion of soil for the 50th and 95th percentile persons were

estimated at 1.83×10^{-14} and 1.69×10^{-13} mg/kg-day, respectively, compared to 1.58×10^{-12} mg/kg-day based on the regulatory approach.

Dermal Update

Uptake via dermal absorption is dependent upon the concentration of the chemical in soil, adherence of soil-to-skin (soil loading), exposed skin surface area, and the fraction of chemical absorbed. The estimated concentration of 2,3,7,8-TCDD TEQ in soil was discussed in the previous section. The daily uptake via dermal contact was estimated using the equation presented in Table V. As dermal absorption did not significantly contribute to total risk, the CAPCOA default values (with the exception of exposure frequency and duration) were used to estimate uptake via dermal absorption in both the regulatory and probabilistic assessments (Tables II and V). However, a more precise estimate could be made using recently published analyses.

Based on the probabilistic assessment, the 50th and 95th percentile doses were estimated at 8.07×10^{-16} and 7.38×10^{-15} mg/kg-day, respectively, compared to 5.62×10^{-14} mg/kg-day estimated using the point estimate approach.

Ingestion of Vegetables

Exposure via ingestion of vegetables is a function of the concentration of chemicals present in or on the vegetable and the rate of consumption. In accordance with the CAPCOA guidelines,[3] the equations and parameter values presented in Table VI were used to estimate the concentration of 2,3,7,8-TCDD TEQ in vegetables (and pasture grasses for the beef and dairy ingestion pathway).

The fraction of deposited particulates retained on a vegetable surface (interception fraction, IF) is dependent upon the crop type and crop yield.[25,34] CAPCOA has characterized vegetable/crops into three groups: leafy vegetables (e.g., lettuce, spinach, cabbage), vine vegetables (e.g., tomatoes, squash), and pasture grasses. Leafy vegetable crops, with an average yield (Y) or density of 2 to 4.4 kg/m^2, intercept 15–39% of deposited particles settling onto edible plant parts.[35] Vine vegetable crops, with an average yield of 2–4.4 kg/m^2, intercept 3.4–7.3% of deposited particles.[34,48] Pasture grasses (cattle feed) yield 0.005–0.63 kg/m^2, which correlates with interception factors of 11–84%.[48] These ranges for yield and interception fraction were used in the probabilistic assessment for leafy vegetables, vine vegetables, and grazing pastures, respectively. The interception fraction values for leafy and vine vegetables and pasture grasses are lower than the values cited in EPA's 1992 reassessment of exposure to TCDD.[25] The crop yield for pasture grass and hay presented by Fries and Paustenbach[34] cited by the EPA[25] indicates that a value of 2 kg/m^2 is appropriate. Stevens and Gerbec[35] have suggested higher crop yields for leafy and vine crops at 8.6 and 12 kg/m^2. Because inadequate data existed to accurately assign specific distributions, uniform distributions were assumed for both the interception fraction and crop yield.

The weathering constant for vegetables (k) is the decay constant for the disappearance of the chemical from vegetable surfaces. This value is a function of the half-life of the deposited chemical on the surface of the plants (e.g., $k = 0.693/t_{1/2}$).[3] The regulatory default value of 0.05/day (based on a half-life of 14 days) was based on numerous values for the disappearance of radionuclides from plant surfaces as a result of weathering and wash-off.[48] Prior published assessments suggest that the half-life of most particulates on plant surfaces (based on radionuclide data) ranges from 2.8 to 34 days.[34,48] The half-life of PCDDs/PCDFs on plants, however, may be dependent on additional processes including volatilization, biodegradation, and photo-oxidation.[34,48] Based on the work of Baes et al.,[48] in the probabilistic assessment a lognormal distribution was used for the weathering constant with a geometric mean equal to 0.0693 (days)$^{-1}$ and a standard deviation equal to 0.231.

The uptake of PCDD/PCDF by plants from soil is not significant, due in large measure to its low water solubility.[34] Field data support this hypothesis. Therefore, this factor (C_{trans}) was not included in this assessment. Exposure via ingestion of vegetables impacted by PCDD/PCDF deposition was estimated using the equation and parameters presented in Tables II and VI. Default consumption rates (If_p) for vegetables suggested by CAPCOA were obtained from the U.S. Department of Agriculture (USDA).[49] The USDA values are 0.25 and 0.01 kg/day for vine and leafy vegetables, respectively. Using data from these studies, the EPA[44] estimated the fraction of all vegetables consumed (L_p) for rural, suburban, and city households from homegrown gardens.

Based on the probabilistic assessment, the 50th and 95th percentile uptake of PCDD/PCDF for leafy and vine vegetables combined was 1.37×10^{-12} and 8.27×10^{-12} mg/kg-day, respectively, compared to 8.29×10^{-11} mg/kg-day estimated using the point estimate approach. In short, the point estimate approach suggested an uptake twofold greater than the 95th percentile value.

Ingestion of Beef

The concentration of PCDD/PCDF in cows that graze on contaminated soil and inhale contaminated air is a function of the animals' exposure via inhalation, ingestion of grain, ingestion of pasture grass, and soil ingestion while foraging.[34] In Table VII, an equation recommended by CAPCOA[3] estimates a steady-state tissue concentration of TCDD in grazing cows.

At this site, cattle grazed on natural and irrigated pasture within two kilometers of the facility, and their diets were supplemented with protein-enriched feed. The supplements did not originate from areas that would have been influenced by aerial emissions from the facility. Only particulates deposited on the surface of the grass and soil were considered as ingested by grazing cattle.

The CAPCOA[3] has identified a chemical food-to-beef transfer coefficient (Fi_b) of 0.4 day/kg for PCDD/PCDF based on its interpretation of the data for 2,3,7,8-TCDD. This value is based on a study conducted by Jensen et al.[50] in which cattle were fed diets containing 24 parts per trillion (ppt) 2,3,7,8-TCDD for 28 days. The cattle were returned to a normal diet for another 36 weeks followed by tissue analysis. TCDD residues in the liver, kidney, and muscle were lower than the TCDD diet level. However, the level found in the fat tissue was approximately four times higher than the diet level.[50] Results suggest that the concentration of TCDD in muscle is proportional to the percentage of fat in that organ. Therefore, assumptions regarding the steady-state body-burden and 25% meat fat resulted in the estimated transfer coefficient of 0.4 day/kg.[50] The Jensen et al.[50] data were reexamined by Fries and Paustenbach[34] who, by correcting for the oral bioavailability of 2,3,7,8-TCDD and the high administered doses, recommended a diet-to-meat transfer coefficient of 0.1 day/kg for cattle.[34] The EPA[25] cited the data presented by Fries and Paustenbach[34] and used a diet-to-meat transfer coefficient of 0.15 day/kg for evaluating the potential health risks for 2,3,7,8-TCDD. The 0.1 day/kg value is now generally considered more valid for the purposes of the probabilistic analysis. However, the transfer coefficient values of 0.1 and 0.4 day/kg were assigned equal probability.

The uptake of PCDD/PCDF by beef cattle is primarily caused by incidental soil ingestion (SI) associated with normal grazing.[34] The soil ingestion rate, as a fraction of pasture grazing (%Sp), ranges from 1.1–17.5% of the dry mass consumed.[51] A uniform distribution was assigned to this parameter. The value recommended by CAPCOA[3] for soil ingestion rate is 5% of dry mass intake. Table VII presents the parameters used to estimate the concentration of 2,3,7,8-TCDD TEQ in beef.

Table VII also presents the equation and pathway-specific exposure parameters used to estimate uptake of PCDD/PCDF via ingestion of beef as recommended by CAPCOA.[3] The relationship between food intake and body weight was accounted for in the probabilistic assessment. McKone and Ryan[46] estimated a meat intake per unit body weight for adults based on the data of Yang and Nelson[52] and the assumption that food intake is directly related to body weight to the two-thirds power. A mean of 0.0033 kilogram meat per kilogram body weight per day and standard deviation of 0.00037 for adults, as calculated by McKone and Ryan,[46] were used in the probabilistic assessment for the rate of beef intake.

The fraction of consumed beef that is locally raised beef (L_b) can be estimated by dividing the amount of local beef production by the number of people in the area of its distribution. The grazing area in the facility's zone of impact is approximately 10 square kilometers. This area is capable of supporting 494–1,236 head of cattle, assuming that the grazing land is irrigated pasture.[53] On average, 43% of a cow's weight is converted to commercially sold beef.[54] Assuming the average weight of beef cattle is 500 kg, this yields 107,000–267,000 kg of beef available to be sold locally. Assuming that all the beef is distributed in local markets, it can be estimated that a typical resident consumes 0.0049–0.0122 kg/day of local meat based on the total amount of locally grown beef. These values constitute 6.5–16.3% of beef that is locally raised (Figure 6). Since the basis for this range is the number of cattle that can be supported on 10 square kilometers, it is impossible to specify the distribution type. Accordingly, a uniform distribution was assumed with a range of 6.5–16.3%.

In our probabilistic assessment, the 50th and 95th percentile doses for beef ingestion were 1.14×10^{-11} and 1.98×10^{-10} mg/kg-day, respectively, compared to 2.26×10^{-10} mg/kg-day estimated using the regulatory approach. Like several other exposure parameters, the point estimate value was similar to the 95th percentile value.

Ingestion of Dairy Milk

Human uptake of PCDD/PCDF via ingestion of dairy milk depends on the chemical concentration in the milk and the consumption rate. The concentration of PCDD/PCDF in dairy milk can be estimated using a formula that accounts for chemical uptake by the cow and the transfer coefficient from contaminated intake to milk (Fi_m)[3]. The formula presented in Table VIII is similar to the one used to estimate the concentration of PCDD/PCDF in beef.

It is uncommon for lactating dairy cattle to graze on pasture grass in the United States.[34] However, to be conservative, we assumed that dairy cattle at this site grazed on some natural and irrigated pasture within two kilometers of the facility. The diet of virtually all dairy cattle is supplemented with protein-enriched feed grown outside the facility's zone of impact.

CAPCOA[3] suggests a transfer coefficient from diet-to-milk (Fi_m) of 0.04 day/liter for PCDD/PCDF ingested by dairy cattle. This value is based on an average of three estimates derived from two feeding studies,[50,55] and one estimate based on the octanol:water partition coefficient for 2,3,7,8-TCDD.[56] Jensen et al.[50] suggested a diet-to-milk uptake factor of 0.008 day/liter from their study in which three cows were administered TCDD as a contaminant in 2,4,5-T over a 77-day period. Fries and Marrow[55] suggested an uptake factor of 0.09 day/liter based on Jensen's study[50] of the transfer of 2,3,7,8-TCDD from feed to milk by adjusting for the differences in fat content of milk (4%). Based on the octanol:water partition coefficient for organic compounds, Travis and Arms[56] estimated an uptake factor of 0.032 day/liter. As suggested by CAPCOA,[3] the regulatory assessment used the average of the three values. Fries and Paustenbach[34] recommended using 0.01 and 0.02, as did EPA in its 1992 reassessment of exposure to TCDD.[25] For the probabilistic assessment, all three values were assigned equal probabilities by using a "discrete-uniform" distribution. Table VIII presents the parameters used to estimate the concentration of 2,3,7,8-TCDD TEQ in cow's milk.

The mean milk consumption rate (IF_m) in the United States is 0.33 liters per day, with a standard deviation of 0.24 liters per day.[49] The CAPCOA default value of 0.30 liters per day is taken from a 1982 USDA study.[3] For the probabilistic assessment, the milk consumption rate was linked to body weight. Data for milk consumption rates reported by Yang and Nelson[52] were applied assuming that food intake scales with body weight to the two-thirds power (based on McKone and Ryan), such that one could calculate a mean and standard deviation of 0.003 and 0.00062 kilogram milk per kilogram body weight per day, respectively. These data were used in the probabilistic assessment.

To estimate the percentage of locally produced milk of the total milk consumed (L_m), the quantity of milk produced by the local dairy was averaged over the local population. In this study, the local dairy maintains about 80 cows and produces 1,200–4,400 liters of milk per day (15–55 liters per cow). By distributing that production rate over the local population of 60,000, a per capita consumption of locally produced milk was estimated to be 0.02–0.07 liters per day. To derive the fraction of locally produced milk consumed daily, these values were divided by the average milk consumption rate. The fraction of locally produced milk consumed was assigned a uniform range with a minimum of 6.7% and maximum of 21%. As recommended by CAPCOA,[3] the site-specific maximum value of 21% was used in the regulatory assessment. Parameter values used in both assessments are presented in Tables II and VIII. Based on the probabilistic assessment, the 50th and 95th percentile doses for dairy milk ingestion were 4.63×10^{-12} and 8.71×10^{-11} mg/kg-day, respectively, compared to 2.85×10^{-10} mg/kg-day estimated using the point estimate approach.

Ingestion of Mother's Milk

Uptake of PCDD/PCDF into mother's milk depends on daily intake rates and specific partitioning into milk. The algorithm recommended by CAPCOA[3] to estimate uptake, based on the work of Smith,[57] is presented in Table IX.

The concentrations of PCDD/PCDF as 2,3,7,8-TCDD TEQ in human mother's milk can be estimated based on the relative partitioning between various body fluid compartments (e.g., total body fluid, plasma, fat, milk). Although some of the key physiological factors that govern PCDD/PCDF partitioning into mother's milk (e.g., maternal fat content, partition coefficients, breast milk composition) have not been studied in sufficient detail to accurately characterize parameter distributions, their variance is anticipated to be relatively small.

The equation and parameters presented in Table IX were used to estimate the uptake via ingestion of mother's milk. Various values for mother's milk consumption rates (DERm) have been reported, most of which fall within the range of

0.6-0.9 kg/day, as described by Butte et al.[58,59] Recently, Dewey et. al.[60] reported breast milk intake rates that are correlated with infant body weight. An analysis of breast milk ingested per kilogram body weight per day indicates that intake rates are significantly different at three, six, nine, and twelve months of age. The average of these values was used and linked to a normal distribution of infant body weight based on data by EPA.[44] The maximum value of 0.9 kg/day was used in the regulatory assessment as recommended by CAPCOA.[3] An additional parameter influencing mother's milk consumption is the total time that an infant will nurse (YR) (i.e., exposure duration). In the probabilistic assessment, exposure frequency (EF) was characterized by using the value suggested by CAPCOA[3] of 365 days per year as the maximum value in a uniform distribution with a minimum of 180 days per year.[61] This is a conservative range because most of the population will have much lower values (e.g., zero for the infant population that is not breastfed). Based on the probabilistic assessment, the 50th and 95th percentile doses for ingestion of mother's milk were 1.17×10^{-14} and 1.30×10^{-13} mg/kg-day, respectively, compared to 6.01×10^{-13} mg/kg-day estimated using the point estimate approach.

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