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Quantitative risk assessment of stack emissions from municipal waste combustors

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Abstract

Multi-pathway risk assessment (MRA) is a tool for evaluating the possibility of adverse effects to human health that may result from contaminant releases from municipal solid waste combustors (MWCs) and other sources of air pollution. An MRA attempts to model the movement of pollutants within the environment to various points at which they may be contacted by people, beginning with inhalation of contaminated air and continuing with indirect pathways such as food chain exposures that entail contaminant transfer and accumulation through several media. MRAs of MWCs date back to the mid-1980s, and are rooted in methods developed to assess the consequences of radionuclide releases from nuclear power generation. A detailed example of a typical MRA is presented that follows the classic four-stage risk assessment paradigm (Valberg et al., 1995). MRA methods, however, continue to be enhanced and refined by recent research. A number of current risk assessment topics are explored that are new to traditional MRAs and may serve to re-focus our collective efforts. The purpose of and demands on MRAs must be considered. A philosophical shift toward placing greater realism in MRAs underscores the importance of uncertainties inherent to risk estimates. Tools such as probabilistic risk assessment techniques must be developed and endorsed so that decision-makers may intelligently utilize the information provided by MRAs.

Keywords: Multipathway risk assessment; Municipal solid waste combustion; Dioxin; Mercury

1. Introduction

Quantitative risk assessment tools have been used for almost a decade to evaluate stack emissions from municipal waste combustors (MWCs). Frequently, a human health risk assessment is performed as part of the siting process for newly proposed

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facilities, and in many cases is a regulatory requirement of environmental impact assessment. Risk assessments are also sometimes conducted for operating facilities to assess the import of recent stack testing or the implications of design changes (such as control technology retrofits).

Most MWC risk assessments focus on stack emissions, although emissions of fugitive ash have been addressed in some cases. Even limited to stack emissions, however, risk assessments of MWCs can be quite complex, and methods continue to evolve as we augment our knowledge of the operating characteristics of MWC facilities and the behavior of pollutants released to the environment. Early studies considered inhalation risks only, but quickly expanded to consider a host of indirect exposure avenues that involve pollutant deposition from the air followed by incorporation into soils, surface waters, vegetation, and foodstuffs. By the mid-to-late 1980s, multi-pathway risk assessments (MRAs) were the norm [1]. Recent guidance documents developed by the US EPA [2, 3] have codified and expanded the scope of MRAs.

The burgeoning complexity of MRAs should not be construed as proof of their comprehensiveness or accuracy. Until recently, most assessments did not explicitly consider phenomena such as precipitation scavenging of pollutants or multi-phase partitioning of contaminants in environmental media. In part, limitations of scientific knowledge are the reasons for not treating such phenomena – comprehensive, validated models have not been developed for many processes. Since an MWC risk assessment comprises a series of models that begin at the point of stack emissions, considerable (and in some cases compounding) uncertainties are introduced throughout.

Uncertainty in risk assessment is both pervasive and unavoidable, and its importance is magnified by recent tendencies to move towards plausible exposure profiles and away from hypothetical worst-case scenarios. It must be stressed that risk assessments (1) are (at best) order-of-magnitude estimates, and (2) will maintain a conservative bias as long as toxicologic data are interpreted in the current manner.

A detailed discussion of uncertainty is beyond the scope of this paper (although additional perspectives are developed in Section 3.5). Instead, this paper focuses on the practical implementation of MRA methods, and is intended to complement the overview presented by Valberg et al. [4]. Section 2 summarizes a typical MRA of MWC stack emissions, and includes a description of modeling algorithms, assumptions, parameter selection, and predictions. Section 3 addresses recent advances and trends not incorporated in the example MRA in Section 2. Several sections focus on polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs). These compounds have been associated with stack-gas emissions of MWCs since the 1970s, and potential health effects due to PCDD/PCDFs are a continuing source of controversy.

2. Multi-pathway risk assessment of combustor stack emissions

Municipal waste combustors release various potentially toxic compounds. Some of the chemicals emitted are constituents of the waste that travel through the combustion

chamber and are not captured by pollution control equipment. These chemicals include metals such as mercury, arsenic, and cadmium, and organic compounds that escape combustion or are only partially oxidized. Other pollutants, such as polychlorinated dibenzo-*p*-dioxins and furans (PCDD/PCDFs), are byproducts formed in the combustion train.

Risk assessment is a formal, mathematical tool that can be used to evaluate potential hazards introduced by pollutant emissions. The classic, four-step process involves hazard identification, exposure assessment, dose-response assessment, and risk characterization [5]. In hazard identification, chemicals of concern are identified from stack test measurements or, in the case of a planned facility, from the operating experience of similar plants. For each chemical, emission rates are assigned based on measurements (if available) or on projections based upon plant capacity, waste composition, pollution control equipment, and performance testing of existing facilities. Exposure assessment marries estimates of pollutant concentrations in environmental media with the anticipated rates at which these media will be contacted by receptors. Independently, the toxicity of each chemical is characterized quantitatively by a dose-response assessment that relates the degree of harm to varying levels of exposure. Finally, the exposure and dose-response assessments are compared to characterize potential risks. The paper by Valberg et al. [4] provides additional details.

The following sections summarize a risk assessment of a modern MWC. A number of design parameters are borrowed from a facility recently proposed to be built near Albany, New York [6]. The plant is designed to burn 1500 tons per day (tpd) of solid waste. Pollution control equipment will include a spray dryer (to remove acid gas compounds) and a baghouse (to remove particulate matter).

The risk assessment methods follow draft guidance developed by the New York State Department of Health [7]. This guidance is representative of MRAs conducted in the late 1980s and early 1990s. State-of-the-art methods, however, have augmented and supplanted some aspects of the NYSDOH guidance [7], which does not consider a number of phenomena (such as pollutant partitioning between vapor and particle phases in air and vapor uptake by vegetation) that have been identified recently to be important in predicting the fate and transport of pollutants in the environment. Several of these phenomena are addressed separately in Section 3.

2.1. Pollutant emission rates

Stack-emission rates based on the performance of operating MWCs of potentially hazardous chemicals are listed in Table 1.[1] Designated as *maximum*, are estimates of

[1] The chemicals listed in Table 1 are the trace constituents found in stack-gas emissions of waste-to-energy plants that are frequently considered in long-term health risk assessments. Waste-to-energy plants also emit other pollutants (such as sulfur dioxide, nitrogen oxides, hydrogen chloride, and particulate matter) that are addressed by other analyses that are typically required to obtain operating permits.

Table 1
Contaminant emission rates from the example MWC

Contaminant	Stack gas concentration (μg/dscm)[a]	Maximum emission rate Q_s (g/s)[b]	Anticipated emission (percent of Q_s)
Antimony (Sb)	50	4.49e − 03	4%
Arsenic (As)	10	8.97e − 04	6%
Beryllium (Be)	0.5	4.49e − 05	2%
Cadmium (Cd)	20	1.80e − 03	4%
Chromium VI (Cr VI)	4	3.59e − 04	5%
Chromium III (Cr III)	36	3.23e − 03	5%
Copper (Cu)	50	4.49e − 03	7%
Lead (Pb)	200	1.80e − 02	3%
Manganese (Mn)	200	1.80e − 02	10%
Mercury (Hg)	130	1.17e − 02	22%
Nickel (Ni)	50	4.49e − 03	3%
Selenium (Se)	4	3.59e − 04	7%
Vanadium (V)	4	3.59e − 04	7%
Zinc (Zn)	500	4.49e − 02	11%
Formaldehyde (HCHO)	200	1.80e − 02	18%
PCBs[c]	0.4	3.59e − 05	5%
Total PAH[d]	50	4.49e − 03	8%
Total CPAH (as B(a)P TEQs)[e]	1	8.97e − 05	25%
2,3,7,8-TCDD TEQs[f]	0.00003	2.69e − 08	2%

[a] μg/dscm = micrograms per dry standard cubic meter ($T = 298$ K, $P = 1$ atm).
[b] Based upon the stack gas concentration listed and combustion of 1500 tons per day of solid waste.
[c] PCBs represent the sum of all chlorinated biphenyls.
[d] Total PAHs represent the sum of all polycyclic aromatic hydrocarbons.
[e] Total CPAH represents the sum of all carcinogenic polycyclic aromatic hydrocarbons weighted by toxic equivalency factors to benzo(*a*)pyrene.
[f] 2,3,7,8-TCDD TEQs represents the sum of all chlorinated dioxin and furan congeners weighted by toxic equivalency factors to 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin.

the highest stack-emission rates that may occur during normal plant operation. Anticipated emission rates, reflected by percentages in Table 1, are significantly lower. Anticipated and maximum emission rate estimates differ in the conservatism embodied in estimating them. The anticipated annual emission rates represent best estimates of the likely performance of the hypothetical MWC. In contrast, maximum annual emission rates are founded on a highly conservative interpretation of operating data, and are not likely to be exceeded during normal plant operation. Since these rates often serve as the basis for formal permit limits, a strong incentive exists to ensure that the maximum emission rate estimates are indeed conservative.

Consistent with long-standing convention of employing conservative assumptions, the risk estimates that follow are calculated with the maximum emission rates. Recent guidance, however, has endorsed the calculation of central tendency exposure estimates. In such cases, use of the anticipated emission rates is appropriate. Ratios of the two sets of emission rates (expressed as percentages) are listed in the rightmost column

of Table 1. Except for a few pollutants, anticipated rates are less than 10% of maximum rates, which serves to emphasize the conservative bias of risk estimates based upon the maximum rates.

Several of the trace organic compounds listed in Table 1 represent a group of different (but related) chemicals. The entry '2,3,7,8-TCDD TEQs' stands for '2,3,7,8-tetrachloro-dibenzo-*p*-dioxin toxic equivalents', a measure of the estimated total toxicity of the particular distribution of polychlorinated dibenzo-dioxins and furans (PCDDs and PCDFs) present in stack emissions. The various PCDD and PCDF congeners are assigned toxicities (toxic equivalency factors, or TEFs) in relation to the toxicity of 2,3,7,8-TCDD, and their occurrence in stack gas is weighted by these TEFs. Polycyclic aromatic hydrocarbons (PAHs) are assessed (and thus listed in the table) in two ways. For the assessment of non-carcinogenic health impacts, total PAHs (the mass sum of all PAHs) are considered. For the assessment of carcinogenic risks, carcinogenic PAHs (CPAH) are evaluated as B(a)P TEQs (or benzo(a)pyrene toxic equivalents) as discussed in US EPA [8]. PCBs comprise a set of chlorinated biphenyls that are distinguished by the number and arrangement of chlorine atoms in the biphenyl rings; because toxicologic data are limited, the PCBs are grouped together.

2.2. *Air dispersion modeling*

Along with the development of emission rates, air dispersion modeling is a key aspect of MRA since all exposure estimates depend upon its results. Most assessments employ steady-state gaussian plume (GP) models that are applied to hour-by-hour data and averaged to obtain long-term estimates of air pollutant concentrations. Descriptions of the bases and capabilities of numerous GP models are available [9, 10]. GP models have been part of US regulatory programs for about twenty-five years, and their common use has conferred familiarity with and acceptance of the models, but has also produced a tendency to neglect modeling uncertainties and limitations. As discussed further on, consideration of these uncertainties can be important in MRAs.

COMPDEP, a GP model developed by the US EPA [3], is selected to predict the impacts of stack emissions from the hypothetical MWC.[2] COMPDEP is designed for application to combustor stack emissions and is intended for general-purpose use in MRAs. COMPDEP provides estimates of airborne pollutant concentrations and depositions throughout mixed terrain study areas.[3] Input data required by

[2] COMPDEP combines aspects of the ISCST and COMPLEX1 models along with wet and dry deposition algorithms. COMPDEP is not recommended for regulatory application by the US EPA [11], but it has received considerable attention in multi-pathway risk assessment guidance [2].

[3] The US EPA [10] defines three categories of terrain (simple, intermediate, and complex) based upon the elevations of lands with respect to the combustor stack and plume. Simple terrain elevations are below the top of the stack, complex terrain elevations are above the plume centerline (which is generally above stack-top due to plume rise), and intermediate terrain elevations are between the stack-top and plume centerline.

Table 2
COMPDEP model input for the baseline example

Relevant modeling options	
• Rural dispersion coefficients, no stack-tip downwash, final plume rise, buoyancy induced dispersion, calm winds processing, calculate plume settling and dry deposition (see Section 2.3 for more detail)	
Source parameters	
• Single stack	
• Stack height	114.3 m (375 ft)
• Stack exit diameter	2.2 m
• Stack-gas exit velocity	10 m/s
• Nominal emission rate	1 g/s
Receptor locations	
• Polar coordinate modeling grid with source at center	
• Radials established at 10° intervals (36 per receptor ring)	
• Receptor rings at radii of 50, 100, 200, 400, 600, 800, 1000, 1200, 1400, 1600, 1800, 2000, 2500, 3000, 3500, 4000, 4500, 5000, and 6000 m from the source	
• Four separate input files required to accommodate 720 locations	
• Flat terrain – all receptors at the same elevation as stack base	
Meteorologic data	
• 1 year of hourly data (calendar year 1989)	
• Surface observations and mixing height data from Denver, CO (Stapleton Airport)	
• Anemometer height:	10 m

COMPDEP include general modeling options, source parameters, receptor locations, and meteorologic data.

A generic, simple terrain modeling study was designed for the baseline example. Input data assumed for the baseline example are summarized in Table 2. Modeling options selected are those recommended for regulatory compliance applications [10]. Source parameters are typical of a modern 1500-tpd MWC. Receptor locations are assigned to provide adequate spatial coverage in all directions from the source out to a distance of 6 km. Meteorologic data were obtained from the US EPA [13] as part of the example COMPDEP file. A wind rose depicting the frequency of wind speeds and wind directions is shown in Fig. 1. Southerly winds ranging from 4 to 11 knots are most predominant in the distribution, while the lowest frequency of winds originates from the west.

A nominal emission rate of 1 g/s is modeled. The highest ground-level concentration predicted by the COMPDEP model within the modeling grid is 0.012 $\mu g/m^3$ per g/s. This value occurs at a distance 2 km south of the site.[4] Concentrations of a similar magnitude (>0.01 $\mu g/m^3$ per g/s) are predicted over wide regions (distances ranging from 1.5 to 4 km) to both the north and south of the facility. Such a broadly defined maximum is not unusual in simple terrain applications, and has important implications for MRA. Modeling uncertainties are too great to permit confidence in the

[4] Intuitively, Fig. 1 suggests that the maximum χ/Q should occur north of the facility. The larger impact predicted to the south results from a preferential distribution of atmospheric stability classes.

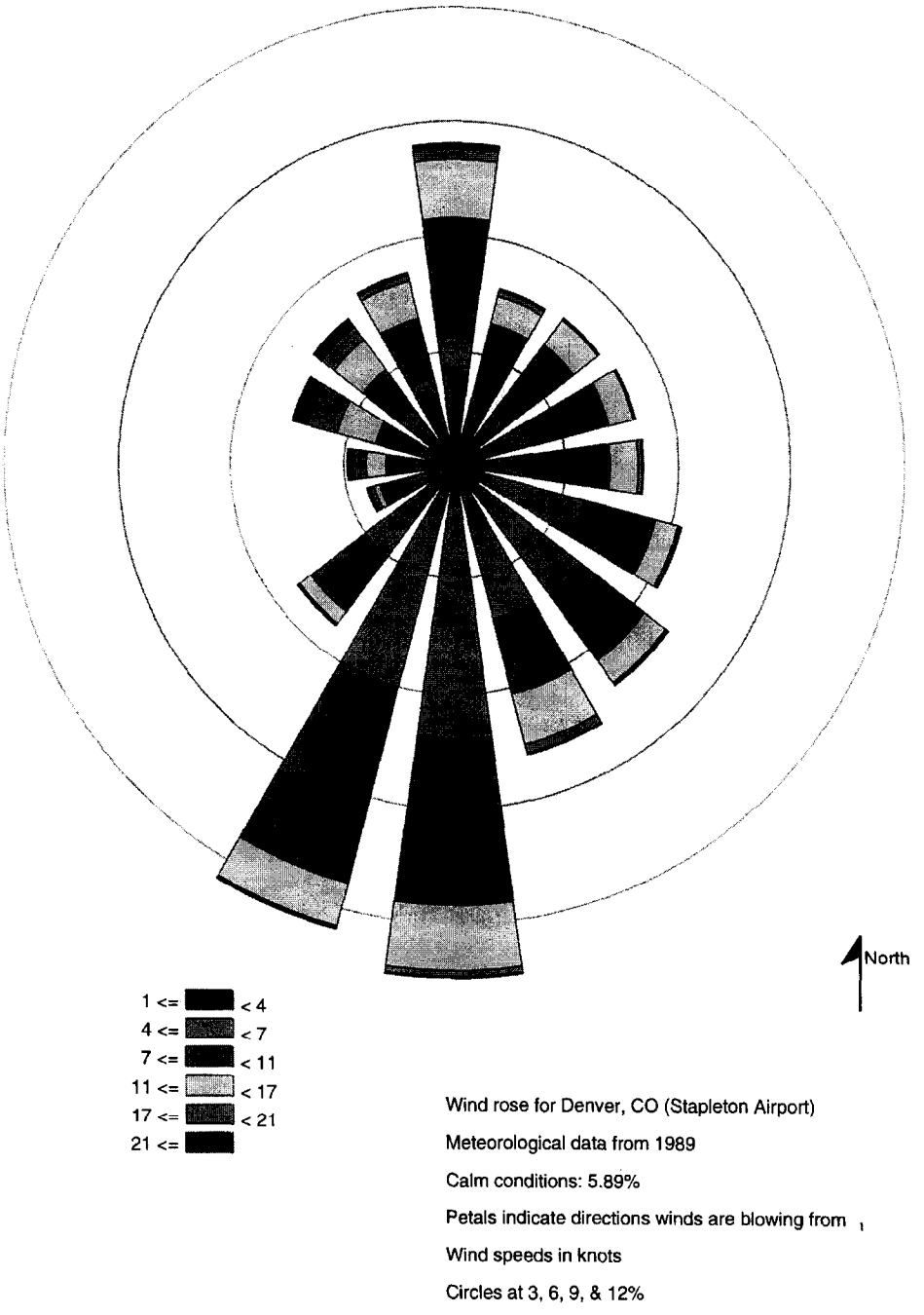

Fig. 1. Wind rose for 1989 hourly meteorologic observations at Denver, CO. The length of each petal is proportional to the frequency of winds *originating* from each sector.

Table 3
Maximum predicted air pollutant concentrations for various modeling scenarios

Scenario	Stack height (m)	Nearby building dimensions	Terrain	Maximum χ/Q_n($\mu g/m^3$ per g/s) and location relative to the facility
Baseline	114.3	None	Simple	0.012, 2 km due south
Shorter stack	45.7	None	Simple	0.035, 2.5 km, 10° east of north
Shorter stack with building	45.7	Height: 36.6 m (12 ft) Width: 61.0 m (200 ft)	Simple	0.34, 0.2 km, 10° east of north
Baseline with complex terrain	114.3	None	Mixed simple and complex	0.58, 1.4 km, 10° east of north

precise location of the predicted maximum, and it is necessary to examine a larger region in identifying the areas in which the highest exposures to facility-related pollutants may occur.

Air pollutant concentrations are predicted by:

$$c_a = Q_s \frac{\chi}{Q_n}, \tag{1}$$

where c_a is the modeled pollutant concentration in air ($\mu g/m^3$), χ/Q_n is the concentration in air modeled for the nominal 1 g/s emission rate ($\mu g/m^3$ per g/s), and Q_s, the pollutant-specific emission rate (g/s).

Table 6 lists the values of c_a based upon the maximum χ/Q_n of 0.012 $\mu g/m^3$ per g/s and the maximum Q_s values (Table 1).

χ/Q_n is sensitive to a number of model parameters, and deviations from the baseline example can lead to significantly higher values. Predictions for three alternate (but not atypical) modeling scenarios are presented in Table 3. The "Shorter stack" scenario models a lower stack height of 45.7 m (150 ft), which is more typical of older MWCs. In this case, the maximum χ/Q_n is predicted to be 0.035 $\mu g/m^3$ per g/s, or about three times that of the baseline case. The 'Shorter stack with building' adds a building 120-ft high by 200-ft wide next to the stack that aerodynamically affects the near-source dispersion of the plume. This effect is modeled with the building downwash algorithms contained in COMPDEP. A maximum χ/Q_n of 0.34 $\mu g/m^3$ per g/s results, and this impact is predicted very close (200 m) to the source. The final scenario, labeled 'Baseline with complex terrain', reverts to the taller (375 ft) stack but introduces terrain around the facility that rises 200 m in all directions between radii of 0.5 km and 1.5 km from the source.[5] A maximum χ/Q_n of 0.58 is modeled at a location 1.4 km from the stack where the receptor elevation is 180 m (about 66 m above stack-top).

The 'Shorter stack with building' and 'Baseline with complex terrain' scenarios illustrate the potential importance of building downwash and complex terrain to

[5] The terrain adjustment option is employed in the COMPDEP model with a minimum ground-to-plume centerline distance of 10 m.

predictions of the COMPDEP model and other US EPA GP models. These options can increase maximum χ/Q_n values by an order of magnitude. In order to obtain the lowest possible model predictions, MWCs should (1) avoid stack heights lower than the Good Engineering Practice height [10] and (2) not (if possible) be situated in complex terrain.

2.3. Deposition rate modeling

Pollutants in the stack gas of an MWC exist in both gaseous and particle-bound phases. Prior to exiting the stack, some of the gases will condense on the particulates. After leaving the stack, the particulates will disperse in the atmosphere, eventually depositing on land, plants, and water. Estimates of pollutant deposition are used in assessing risks associated with a variety of indirect exposure pathways involving ingestion and dermal absorption of contaminants that are incorporated in soils, surface waters, and foodstuffs.

This section summarizes methods used to calculate the amount of pollutants from the stack that deposit on the earth's surface. Dry deposition of pollutants occurs when (1) particles are transported through gravitational settling and turbulence and attach to surfaces, and (2) gases absorb or adsorb to surfaces. Wet deposition occurs when pollutants are scavenged by rain or snow. Consistent with NYSDOH guidance [7], however, only dry deposition is considered in the baseline example. The implications of wet deposition are discussed in Section 3.1.

All pollutants are treated as particle-bound, even though evidence suggests that there are significant vapor-phase concentrations of chemicals such as mercury and PCDD/PCDF (special considerations for these pollutants are discussed in Sections 3.2 and 3.3, respectively). Contaminants with relatively high vapor pressures (at stack gas temperatures) are modeled assuming they have condensed onto the surface of particulate matter emitted from the stack. These pollutants are designated as surface-weighted (SW), and include all of the organic compounds, lead, mercury, and selenium. The remainder of the metals are categorized as volume-weighted (VW) to reflect their assumed distribution as uniform throughout particulate matter.

Dry deposition for the baseline example is predicted by the COMPDEP model, which embodies algorithms developed by Sehmel [14] and the California Air Resources Board (CARB) [15]. The rate of deposition (D_p) is proportional to the contaminant concentration in air at ground level (c_a), with the constant of proportionality called the deposition velocity (v_d):

$$D_p = c_a v_d. \tag{2}$$

In the CARB [15] model, deposition velocities (v_d) vary with particle size (which in part determines the gravitational settling velocity), the Monin–Obukhov length (a function of atmospheric conditions), surface friction velocity (u_*), and surface roughness height (z_0). The particle-averaged deposition velocity is calculated as a weighted value for a given particle size distribution. The COMPDEP model estimates deposition velocities hour-by-hour, and sums the hourly values given by Eq. (2) to produce annual deposition estimates. Additional parameters required for deposition modeling

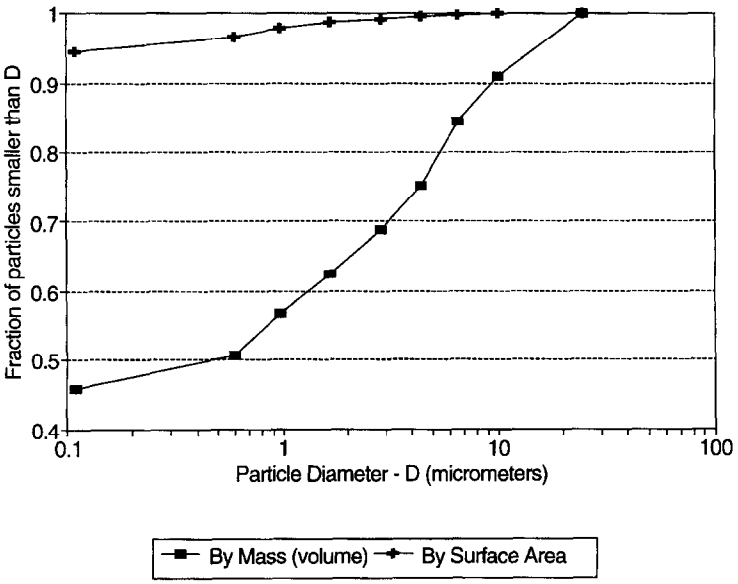

Fig. 2. Particle size distributions assumed for the MRA example, as derived from data collected at an operating MWC [59].

include the particle density and the surface roughness length (z_0). Representative values of 1.8 g/cm^3 and 0.3 m are assumed for these parameters, respectively. Also required are particle size distributions for VW and SW pollutants. Modeled distributions are depicted in Fig. 2, and are derived from stack testing data collected at an operating MWC [16]. The facility is equipped with a spray dryer/baghouse, and emits the bulk of particulate matter (46% by mass) as very small particles (<0.1 μm in diameter).

The highest dry deposition rates predicted by the COMPDEP models are 0.0031 g/m^2-yr and 0.00041 g/m^2-yr for the VW and SW categories, respectively. Both of these values are based upon a nominal emission rate of 1 g/s, and pollutant-specific values may be obtained by multiplying by the emission rates listed in Table 1. The location of these highest rates is coincident with the location of the highest predicted concentration in air (2 km south of the facility).

The effective annual deposition velocities (obtained by dividing the deposition rates by the maximum χ/Q_n) are 0.8 cm/s and 0.1 cm/s, respectively, for VW and SW pollutants. These values reflect the predominance of small particles within the particle size distribution (Fig. 2). Baghouses are efficient at removing all but the smallest of particles. Facilities equipped with different particulate control equipment are likely to release larger (on average) particles, for which the modeling is apt to predict higher effective deposition velocities (and hence deposition rates). Also, the deposition modeling is sensitive to the assumed distribution of small particles. As a practical matter, sampling equipment cannot differentiate particles smaller than about 0.1 μm in diameter. Particle deposition velocity is minimized at roughly this same diameter.

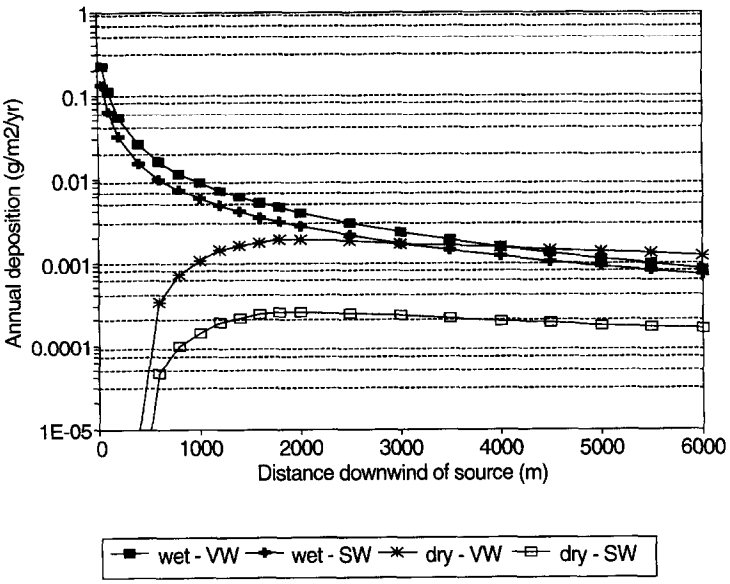

Fig. 3. Particle deposition rates (g/m^2-yr per unit [g/s] emission rate) predicted by the COMPDEP model. VW and SW categories distinguish volume- and surface-weighted pollutants, respectively.

For smaller particles (<0.1 μm), deposition velocities increase due to Brownian diffusion effects. Consequently, deposition rates can be significantly influenced by the diameter(s) selected to represent the smallest particle size category, for which measurements are unavailable [17].

2.4. *Estimated contaminant mass fractions in soil*

Contaminants released from an MWC will deposit onto soils. The contaminant mass fractions in surface soils are estimated using a simple accumulation model in which pollutants are assumed to deposit, mix, and remain within a fixed soil depth. The average mass fraction attained over a period of constant pollutant deposition is given by:

$$m_s = \frac{D\tau}{2\rho_s z}, \tag{3}$$

where m_s is the contaminant mass fraction in soil (mg/kg), D, the rate of contaminant deposition to the soil (mg/m^2-yr), τ, the length of the emission period (years), ρ_s, the soil bulk density (kg/m^3), and z the soil mixing depth (m).

The processes responsible for distributing contaminants throughout the soil include (1) transport by infiltrating rainwater; (2) mechanical mixing by a variety of fauna, including earthworms, ants, termites, and burrowing animals; and (3) biological decay and tree uprooting [18]. For cultivated lands, plowing and hoeing are assumed to mix contaminants throughout a soil layer that roughly corresponds to the root penetration distance of plants. Two soil mixing depths (z) are used to account for differences

in soil conditions in cultivated and uncultivated lands. Consistent with US EPA [3] guidance, the tilled soil mixing depth is assumed to be 0.20 m and the untilled soil mixing depth is assumed to be 0.01 m. A typical bulk soil density (ρ_s) of 1500 kg/m^3 is used as recommended by NYSDOH [7], and a 70-year deposition period is assumed.

Contaminant mass fractions in soil are estimated for tilled and untilled soils at the point of maximum impact. The predicted mass fractions are reported in Table 6, and are based upon the maximum deposition estimates developed in Section 2.3.

Contaminants may be removed from surface soils as a result of leaching. In addition, biodegradation, hydrolysis, and photolysis serve to destroy some chemicals. The estimates of soil mass fraction based on Eq. (3) are conservative since these loss mechanisms are not taken into account. First-order removal models have been developed to account for these processes [2]. Care must be exercised in using these models, however. For pollutants with extended half-lives in soil, steady-state mass fractions may correspond to dozens or even hundreds of years of deposition in the framework of the no-loss model [Eq. (3)].

2.5. *Pollutant concentrations in surface water and fish*

Potential contamination of surface waters that results from MWC emissions is generally estimated for lakes, ponds, and waterways located near the region of greatest projected impacts. More distant (less impacted) surface waters may be of importance if they have unusual physical characteristics (e.g., a low dilution rate) or are of special commercial or political significance. In general, impacts will be higher in small lakes and ponds with limited drainage areas than in rivers that drain wide regions.

For the purpose of the baseline example, a small lake is assumed to be located in the region of maximum impact. The maximum projected deposition rates (Section 2.3) are assumed to apply throughout the watershed. Contaminant loading to the lake is assumed to occur via two pathways: (1) direct deposition to the surface of the lake and (2) deposition to the soil in the lake's watershed followed by surface soil erosion and run-off to the water body. The total waterborne concentration (c_w) for a compound is estimated as the total mass of the compound entering the water body per unit time divided by the rate of water flow through the water body:

$$c_w = \frac{m_s R_s + D_w A_w}{V_w}, \tag{4}$$

where c_w is the total waterborne concentration (mg/l), m_s, the average contaminant mass fraction in watershed run-off soils (mg/kg), R_s, the sediment loading to the water body (kg/yr), D_w, the deposition rate to the surface of the water body (mg/m^2-yr), A_w, the surface area of the water body (m^2), and V_w, the water volume flow rate through water body (l/yr).

Estimates of total waterborne concentrations predicted by Eq. (4) for a hypothetical lake are presented in Table 6. The parameters used to derive these estimates are taken from a case study of a small reservoir utilized for swimming and fishing [6]. A water

surface area (A_w) of 2.1×10^5 m^2, a dilution flow (V_w) of 2×10^9 l/yr, and a total watershed area of 1040 acres are available from a specific study of lakes in the region [19].

The rate of soil erosion is estimated by applying the Universal Soil Loss Equation (USLE) [20, 21] over the watershed:

$$R_s = \sum_{i=1}^{n} [A_i(RKL_SCP)_i S_d], \tag{5}$$

where n is the number of subareas in the reservoir watershed area, A_i, the acreage of the subarea i (acres), R, the rainfall factor, which expresses the erosion potential of average annual rainfall in the locality, K, the soil-erodibility factor for subarea i (tons/acre-yr-R unit), L_S, the topographic factor depending on slope-length and slope-steepness for subarea i (dimensionless), C, the cover factor for subarea i (dimensionless), P, the erosion control practice factor (dimensionless), and S_d, the sediment delivery ratio (dimensionless).

In this case, application of the USLE involved the evaluation of individual portions of the watershed. Agricultural, wooded, and open lands were considered separately, and appropriate values of C and P were selected for each category. Area-specific values of K and L_S were obtained from the local branch of the Soil Conservation Service. A sediment delivery factor of 0.26 was selected based upon the area of the watershed [22].

The presence of steep slopes and a significant fraction of agricultural land led to a fairly high estimate of soil erosion. A net erosion rate R_s of 885 t/yr was estimated for the watershed, with 82.5% of the total originating from agricultural lands. The percentage is used to weight the tilled and untilled pollutant mass fractions in soil (m_s) within Eq. (4).

Contaminant mass fractions in fish (m_f) are assumed to be proportional to the *dissolved* contaminant concentrations in water (c_{wd}):

$$m_f = B_{cf} c_{wd}, \tag{6}$$

where B_{cf} is the bioconcentration factor (in l/kg). For metals, the dissolved component is assumed to be essentially equal to the total concentration, as estimated by Eq. (4) and listed in Table 6 under the 'surface water' column. Organic compounds, however, can be extremely hydrophobic and strongly sorb to suspended organic material. For such compounds, the total surface water loading (c_w) can be partitioned into dissolved (c_{wd}) and sorbed (c_{ws}) components:

$$c_w = c_{wd} + c_{ws}. \tag{7}$$

At equilibrium, the dissolved and sorbed components will be related to each other:

$$c_{ws} = K_{oc} O c_{wd}, \tag{8}$$

where K_{oc} is the equilibrium partitioning coefficient between water and organic carbon particles (l/kg) and O is the suspended organic content within the water column (kg/l).

Combining Eqs. (6)–(8), the pollutant mass fractions in fish (m_f in mg/kg) can be related to the total water concentration (c_w) as:

$$m_f = \frac{B_{cf} c_w}{1 + K_{oc} O}, \tag{9}$$

where the $K_{oc}O$ term is included for organic chemicals only.

The suspended organic content O varies among surface waters. Generally, suspended solids concentrations range from 1 to 100 mg/l, with flowing streams exhibiting higher values than quiescent ponds and lakes. The organic fraction of suspended solids also depends on the nature of the water body. Ranges of values are discussed in the literature [4], and local measurements are sometimes available as part of water quality evaluations. An O value of 0.25 mg/l derived from local observations is assumed for the reservoir of the baseline example [6].

Chemical-specific values of K_{oc} are available in the literature, as are values of B_{cf}. The latter parameter, however, may depend greatly on particular species of fish and surface water characteristics (e.g., freshwater or saltwater), and considerable judgement may be needed to select appropriate values. Default values for these parameters are listed in Table 5. Table 6 presents the contaminant mass fractions in fish (m_f) calculated using Eq. (9) with the parameters described above and the total contaminant concentrations in the reservoir water predicted by Eq. (4).

2.6. Estimated contaminant mass fractions in locally-raised foods

Vegetation may assimilate airborne contaminants through two mechanisms: (1) direct deposition to the exposed surfaces of plants and (2) deposition onto the soil followed by uptake through the root systems. Both of these mechanisms are evaluated when estimating contaminant mass fractions in homegrown produce and animal feeds that may result from operation of an MWC. In addition, pollutants may accumulate in livestock that are raised on feed grown in the vicinity of an MWC.

The methodologies used to predict the mass fractions of pollutants in vegetation are described in NYSDOH [7], which itself derives from a number of references (most notably [23, 24]). Particle-borne contaminants released from an MWC will deposit on vegetation in the surrounding area. The potential for local vegetation to intercept and incorporate settling particles depends on a variety of factors, including rate of contaminant deposition, the fraction of deposition intercepted (which depends principally upon the cross-sectional area covered by the vegetation), the rate at which degradation and weathering processes remove contaminants, the length of the growing season, and the yield of the particular crop. The model used to estimate contaminant mass fraction in vegetation due to atmospheric deposition takes account of these factors:

$$m_{vd} = D_p^a r \frac{1 - e^{-k_v t_v}}{y k_v} \tag{10}$$

where m_{vd} is the estimated contaminant mass fraction in produce or feed due to direct deposition of particulate matter (mg/kg dry weight or wet weight, according to the

convention used for y), D_p^a, the annual rate of contaminant deposition (mg/m^2-yr), r, the vegetation-specific intercept fraction, y, the crop yield (kg/m^2), k_v, the effective removal (or degradation) rate from plant surfaces (yr^{-1}), and t_v, the length of the growing season (yr).

Root uptake is the second mechanism whereby plants may assimilate contaminants that originate from an MWC. Particulate-bound contaminants are assumed to deposit into surface soils as discussed in Section 2.4. Compounds in soil may become incorporated into vegetation via uptake through the roots. The degree of uptake will vary with the type of vegetation and the availability of the contaminant in the soil. The contaminant mass fraction in vegetation due to root uptake is estimated:

$$m_{vr} = m_s R_u d_w, \tag{11}$$

where m_{vr}, is the estimated contaminant mass fraction in the produce or feed due to root uptake (mg/kg dry weight if d_w is omitted, wet weight if d_w is included), m_s, the contaminant mass fraction in soil (mg/kg), R_u, the root uptake factor [(mg/kg plant dry weight) per (mg/kg soil)], and d_w, the dry- to wet-weight conversion factor (included only for produce for human consumption) [(mg/kg ww)/(mg/kg dw)].

Contaminant mass fractions in homegrown produce are calculated in terms of wet weight, since rates of produce consumption by humans are typically reported on a wet-weight basis. Feed consumption by cows is typically reported on a dry-weight basis. Thus, the contaminant mass fractions for feeds are calculated in terms of dry weight.

The total contaminant mass fraction in vegetation (m_v, in mg/kg) is the sum of the components due to root uptake (m_{vr}) and direct deposition (m_{vd}):

$$m_v = m_{vr} + m_{vd}. \tag{12}$$

A number of parameters used in Eqs. (10) and (11) will vary with the particular vegetable, fruit, or crop of interest. In theory, the contaminant mass fractions in each type of produce or feed crop would be evaluated individually. A paucity of empirical data, however, necessitates the categorization of vegetables, fruits, and crop feeds into a limited number of groups.

Based on the data available in the literature and draft guidance [7], produce grown for human consumption can be divided into three categories: leafy produce, exposed produce, and protected produce. Leafy produce (e.g. spinach, broccoli, and lettuce) is characterized by the relatively large interception fractions of their edible portion, which qualitatively suggests that direct deposition should be of greater importance relative to non-leafy vegetation. Exposed produce (e.g. tomatoes, bell peppers, and strawberries) includes non-leafy fruits and vegetables for which the edible portion is grown above-ground. Last, protected produce (e.g. oranges, carrots, and potatoes) includes fruits and vegetables that are not exposed to direct deposition – the edible portion is found below ground or protected by an inedible casing.

Mass fractions of pollutants in vegetables and field crops are also estimated at the location of highest projected impact. All mass fractions in vegetation are based upon

Table 4
Vegetable and soil modeling parameters[a]

Parameter	Vegetables			Feed crops				Soil
	Leafy	Exposed	Protected	Hay	Pasture grass	Corn silage	Grain	
Root uptake factor [21] – values listed in Table 5	B_v	B_r	B_r	B_v	B_v	B_v	B_r	NA
Intercept fraction r	0.1192	0.0319	NA	0.3203	0.2922	0.6021	NA	NA
Yield y (kg/m^2) – in wet weight for leafy/exposed; in dry weight for others	1.5	1	NA	0.1341	0.12	1.1982	NA	NA
Length of growing season t_v (yr)	0.42	0.42	NA	0.125	0.15	0.3846	NA	NA
Dry-to-wet weight conversion factor d_w	0.066	0.126	0.222	NA	NA	NA	NA	NA
Washoff coefficient k_v (1/yr)[b]	18	18	NA	18	18	18	NA	NA
Feed consumption rates for lactating cows (kg dry weight/day)	NA	NA	NA	5	2.5	5	5	0.2
Feed consumption rates for steers and non-lactating cows (kg dry weight/day)	NA	NA	NA	2.5	1.25	2.5	2.5	0.2

NA: not appropriate.
[a] Values for all parameters are taken from NYSDOH [7] unless otherwise noted.
[b] Derived from a weathering half-life of 14 days.

tilled soil mass fractions (as described in Section 2.4). The root uptake factors (R_u) are specific to compound and vegetation type. The values of R_u are taken from NYSDOH [7] guidance where available, or from Baes et al. [23]. Recommended default values [7] for vegetation-specific parameters (r, y, k_v, t_v, d_w, and R_u) are summarized in Table 4. Frequently, agricultural agencies can supply information on local agricultural practices and statistics, and these data may be used to supplant default values (along with additional relationships described in Baes et al. [23]).

Chemical specific parameters used in the risk assessment are listed in Table 5. Estimated contaminant mass fractions in leafy, exposed, and protected produce grown at the maximum impact location are listed in Table 6. Excepting mercury and zinc, modeled contaminant levels in leafy and exposed vegetables are greater than those in protected produce. This tendency reflects the fact that, for above-ground vegetation, direct atmospheric deposition is a larger contributor of contamination than uptake from soil. In most cases, atmospheric deposition accounts for 90% or more of total modeled contamination. Atmospheric deposition is most pronounced for leafy vegetables due to the high surface-to-volume ratio. Root-zone uptake is of somewhat greater importance to exposed produce, but atmospheric deposition still contributes the majority of vegetable contamination for four pollutants (zinc, copper, mercury, and cadmium).

Table 5
Chemical specific parameters used in the risk assessment

Contaminant	K_{oc} (l/kg)	B_{ef} (finfish) (l/kg)	Root uptake factor B_v for leafy vegetables	Root uptake factor B_r for fruits, tubers, and roots	Milk biotransfer factor F_m (d/kg)	Beef Biotranfer Factor F_b (d/kg)
Antimony (Sb)	NA	1	0.2	0.03	1.00e − 04	1.00e − 03
Arsenic (As)	NA	44	0.04	0.006	6.00e − 05	2.00e − 03
Beryllium (Be)	NA	19	0.01	0.0015	9.00e − 07	1.00e − 03
Cadmium (Cd)	NA	81	0.55	0.15	1.00e − 03	5.50e − 04
Chromium (Cr)	NA	16	0.0075	0.0045	1.50e − 03	5.50e − 03
Copper (Cu)	NA	200	0.4	0.25	1.50e − 03	1.00e − 02
Lead (Pb)	NA	49	0.045	0.009	2.50e − 04	3.00e − 04
Manganese (Mn)	NA	100	0.25	0.05	3.50e − 04	4.00e − 04
Mercury (Hg)	NA	5500	0.9	0.2	4.50e − 04	1.50e − 02
Nickel (Ni)	NA	47	0.06	0.06	1.00e − 03	6.00e − 03
Selenium (Se)	NA	16	0.025	0.025	4.00e − 03	1.50e − 02
Vanadium (V)	NA	5500	0.0055	0.003	2.00e − 03	2.50e − 03
Zinc (Zn)	NA	47	1.5	0.9	1.00e − 02	1.00e − 01
Formaldehyde	3.6	NA	NA	NA	NA	NA
PCBs	1.00e + 06	100 000	0.017	0.017	1.10e − 02	5.20e − 02
PAHs	5.50e + 06	200	0.012	0.012	9.00e − 03	2.90e − 02
2,3,7,8-TCDD TEQs	4.00e + 06	5000	0.013	0.013	6.00e − 02	2.20e − 01

Table 6
Modeled exposure point concentrations and mass fractions

Contaminant	Air ($\mu g/m^3$)	Untilled soil (mg/kg)	Surface water (mg/l)	Leafy veg. (mg/kg ww)	Exposed veg. (mg/kg ww)	Protected veg. (mg/kg ww)	Milk conc. (mg/kg)	Beef conc. (mg/kg)	Fish conc. (mg/kg)
Antimony (Sb)	5.39e – 05	3.25e – 02	1.51e – 05	8.29e – 05	3.08e – 05	1.08e – 05	2.54e – 06	1.60e – 05	1.51e – 05
Arsenic (As)	1.08e – 05	6.49e – 03	3.02e – 06	1.31e – 05	5.17e – 06	4.32e – 07	2.63e – 07	5.69e – 06	1.33e – 04
Beryllium (Be)	5.39e – 07	3.25e – 04	1.51e – 07	6.25e – 07	2.50e – 07	5.41e – 09	1.92e – 10	1.39e – 07	2.87e – 06
Cadmium (Cd)	2.16e – 05	1.30e – 02	6.06e – 06	4.83e – 05	2.22e – 05	2.17e – 05	1.34e – 05	4.41e – 06	4.91e – 04
Chromium VI (Cr VI)	4.31e – 06	2.60e – 03	1.21e – 06	4.98e – 06	2.05e – 06	1.30e – 07	2.55e – 06	6.11e – 06	1.93e – 05
Chromium III (Cr III)	3.88e – 05	2.34e – 02	1.09e – 05	4.48e – 05	1.84e – 05	1.17e – 06	2.30e – 05	5.50e – 05	1.74e – 04
Copper (Cu)	5.39e – 05	3.25e – 02	1.51e – 05	1.04e – 04	7.59e – 05	9.02e – 05	4.69e – 05	1.89e – 04	3.02e – 03
Lead (Pb)	2.16e – 04	1.72e – 02	8.01e – 06	3.51e – 05	1.41e – 05	1.72e – 06	2.93e – 06	2.27e – 06	3.93e – 04
Manganese (Mn)	2.16e – 04	1.30e – 01	6.06e – 05	3.54e – 04	1.40e – 04	7.23e – 05	3.73e – 05	2.65e – 05	6.06e – 03
Mercury (Hg)	1.40e – 04	1.12e – 02	5.21e – 06	5.44e – 05	2.26e – 05	2.49e – 05	6.36e – 06	1.23e – 04	2.87e – 02
Nickel (Ni)	5.39e – 05	3.25e – 02	1.51e – 05	6.79e – 05	3.70e – 05	2.16e – 05	2.28e – 05	8.79e – 05	7.10e – 04
Selenium (Se)	4.31e – 06	3.44e – 04	1.60e – 07	6.78e – 07	3.15e – 07	9.54e – 08	9.23e – 07	2.25e – 06	2.56e – 06
Vanadium (V)	4.31e – 06	2.60e – 03	1.21e – 06	4.96e – 06	2.02e – 06	8.65e – 08	3.40e – 06	2.77e – 06	6.65e – 03
Zinc (Zn)	5.39e – 04	3.25e – 01	1.51e – 04	2.22e – 03	2.09e – 03	3.25e – 03	5.89e – 03	3.27e – 02	7.10e – 03
Formaldehyde	2.16e – 04	1.72e – 02	8.01e – 06	3.26e – 05	1.31e – 05	NA	NA	NA	NA
PCBs	4.31e – 07	3.44e – 05	1.60e – 08	6.69e – 08	2.98e – 08	6.49e – 09	2.51e – 07	7.72e – 07	1.28e – 03
Total PAH	5.39e – 05	4.30e – 03	2.00e – 06	8.30e – 06	3.59e – 06	5.73e – 07	2.55e – 05	5.36e – 05	1.68e – 04
Total CPAH	1.08e – 06	8.59e – 05	3.99e – 08	1.66e – 07	7.17e – 08	1.14e – 08	5.10e – 07	1.07e – 06	3.36e – 06
2,3,7,8-TCDD TEQs	3.23e – 10	2.58e – 08	1.20e – 11	4.98e – 11	2.17e – 11	3.72e – 12	1.02e – 09	2.44e – 09	2.99e – 08

Eqs. (10)–(12) are also used to predict contaminant mass fractions (not shown) in locally grown cattle feed (hay, corn silage, grain, and pasture grass).[6] Farm animals are assumed to consume a mixture of these products along with a small amount of soil. The pollutant content of beef from cattle or milk from dairy cows (and other foodstuffs derived from livestock) may be related to the total daily intake of the compounds by these animals. Contaminant mass fractions in beef (m_b) and milk (m_m) are related to dietary intake (I_d) by biotransfer factors (F_b and F_m for beef and milk, respectively):

$$m_b = F_b I_d, \quad \text{beef,} \tag{13}$$

$$m_m = F_m I_d, \quad \text{milk,} \tag{14}$$

where m_b and m_m are the contaminant mass fractions (mg/kg) in beef and milk, respectively, I_d is the daily contaminant intake by the animal (mg/day), and F_b and F_m the biotransfer factors (days/kg) for beef and milk, respectively.

As for other pathways, feed for beef cattle and dairy cows is assumed to be grown in the region most heavily impacted by emissions from the MWC. The dietary intake of the animal is simply the sum of contaminants derived from each source of feed, calculated as:

$$I_d = R_h m_h + R_c m_c + R_g m_g + R_p m_p + R_s m_s, \tag{15}$$

where the terms R_h, R_c, R_g, R_p, and R_s are the ingestion rates of hay, corn silage, grain, pasture grass, and soil (kg dry weight/day), and m_h, m_c, m_g, m_p, and m_s are contaminant mass fractions in hay, corn silage, grain, pasture grass, and soil (mg/kg dry weight).

Dietary ingestion rates differ for lactating cows and non-lactating cows and cattle. Default recommendations for feed consumption rates for dairy cows and beef cattle are taken from NYSDOH [7]. These intake rates are provided in Table 4. Contaminant mass fractions in beef and cow's milk predicted by Eqs. (13) and (14) are listed in Table 6.

2.7. Exposure estimates

Sections 2.1–2.6 have described a series of fate and transport models that begin with stack emissions from the MWC and proceed to estimate pollutant concentrations in various environmental media. These exposure point concentrations are summarized in Table 6, and they constitute one of two data sets needed to estimate the rates at which people may be exposed to pollutants released from the MWC. The second set is simply the levels and frequencies at which people inhale, ingest, and contact the media affected by the MWC.

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[6] Again, atmospheric deposition provides the bulk of modeled contaminant mass fractions in the three crops (hay, corn silage, and pasture grass) for which both deposition and root uptake from soil are considered. Root uptake is significant for corn silage, but contributes the larger fraction of contamination for only a few chemicals (zinc, mercury, cadmium, copper, and manganese).

2.7.1. General considerations

Choosing exposure assumptions can be difficult and is (in our opinion) one of the critical elements of an MRA. Efforts have been made to standardize the process of exposure assessment, but the best approach remains to tailor the exposure assessment to the particular characteristics of the area surrounding the MWC and those who inhabit it. Risk assessors should visit the study area if possible and contact relevant agencies and individuals to gather information regarding the habits and activities of local populations. Unusual avenues that could lead to pollutant exposure should be explored. As an example, for an MWC plant proposed for Florida, it could be important to assess the consumption of farm-raised alligator meat, especially given the alligator's position at the top of a food chain [25].

As an initial step, specific population(s) of interest must be identified and exposure profiles constructed. Most studies consider hypothetical scenarios that focus on an individual's exposure to pollutants released from an MWC. Some considerations are:

- *Demographics*
  Different types and ages of individuals may be required to characterize the population(s) at greatest risk. Frequently, exposures to adults and children are considered separately, since adulthood provides the longest period of exposure and childhood accentuates some exposure routes (such as the incidental ingestion of soil) and potential sensitivities due to higher ratios of intake to body weight.
- *Degree of exposure*
  Risk assessments contain numerous uncertainties that are typically compensated by conservative assumptions designed to bias risk estimates high. There has been a recent philosophic shift toward the use of less conservatism. Most risk assessments of MWCs conducted in the late 1980s centered on extreme scenarios such as a maximally exposed individual (MEI). An MEI was constructed to receive (in theory) a level of exposure not likely to be exceeded by any person living in the vicinity of the MWC, a level that would be extremely improbable. More recent guidance, however, has recommended the use of reasonable maximum exposure (RME) scenarios that endeavor to devise plausible, high-end exposure estimates [3]. In reality, the difference between MEI and RME scenarios may be one of semantics, since concepts such as plausible, maximum, and high-end are too often subjective. Psychologically, however, the shift from an MEI to an RME implies a movement from the unlikely to the plausible, and ascribes a greater sense of realism to the risk estimates.
- *Land use*
  Land use in the vicinity of an MWC can influence exposure assessment by placing constraints on where exposure to pollutants is likely or even possible. At the most conservative extreme, worst-case exposures can be evaluated at the location of maximum projected impacts irrespective of land use. This practice is consistent with conservative MEI-type analyses, and accounts for any changes in land use that may transpire. A first-order consideration of land use demands that exposure pathways be reasonable with respect to current land use. For example, exposure pathways that depend on surface water are often evaluated at the locations of actual lakes and streams, and do not hypothesize water bodies at the maximum

impact point. The shift towards RME scenarios places even greater emphasis on current land use. Traditionally, food-chain pathways have dominated risk estimates in MWC assessments. Consequently, construction of an RME scenario may entail a detailed examination of farming, including the specific locations and sizes of agricultural lands, and local farming practices.

- *Exposure pathways*

  The goal of an MRA is to identify all routes through which humans may be exposed to air pollutants released from an MWC. The number of potentially important pathways has grown with time as risk assessments have evolved. Multiple exposure routes are typically considered [4]. The most direct pathway is that of inhalation. Indirect pathways, which first require pollutant deposition of pollutant's to the earth's surface, include the ingestion of soil (incidental), water, vegetables, fish, meat, and dairy products. Dermal exposure to pollutants in soil and water is also possible, although such routes are typically of lesser importance. Also, nursing may be a significant source of exposure for lipophilic organic pollutants.

The many subjective facets of exposure assessment require the exercise of considerable professional judgement. As RME assessments incorporate more realism, the shedding of conservatism places greater importance on the uncertainties inherent in risk estimates.

2.7.2. Exposure estimates

The quantitative process of estimating exposure is straightforward. Excepting the inhalation pathway, exposure is normally estimated as the rate of pollutant contact per unit body weight:[7]

$$Dose = \frac{Concentration \times Contact\ \ Rate \times Frequency}{Body\ \ weight}, \tag{16}$$

where *Dose* is the rate of exposure, *Concentration* the level of pollutant in a particular environmental media, *Contact rate* the amount (per time) of the media contacted, *Frequency* a measure of how often (and over what period) exposure occurs, and *Body weight* the weight of the individual. Doses are typically calculated in units of mg pollutant per day per kg body weight per day (mg/kg-d). For some exposure routes, the individual terms in Eq. (16) may encompass multiple parameters. For example, in estimating dermal pollutant intake during swimming, the contact rate is calculated as the product of (1) the surface area of the skin, (2) a chemical-specific permeability, and (3) the density of water.

Exposure parameters are generally selected as a mix of typical and high-end values to provide an overall conservative bias. While situation-specific values are always preferable, they are seldom available and often impractical to develop. Default values have been established for many parameters, and some conventions have evolved. For

[7] Exposures are generally expressed in forms that parallel toxicologic data. As discussed in Section 2.8, toxicologic data available to evaluate inhalation exposure are typically concentrations, while oral and dermal data are intake rates per body weight.

Table 7
Exposure parameters[a]

Parameter	Adult	Child
Body weight (kg)	70	13.2
Inhalation rate (m^3/d)	20	8.6
Consumption rate of leafy produce (kg/d)	0.039	0.023
Consumption rate of exposed produce (kg/d)	0.089	0.053
Consumption rate of protected produce (kg/d)	0.137	0.082
Consumption rate of beef (kg/d)	0.051	0.02
Consumption rate of milk (kg/d)	0.283	0.418
Consumption rate of fish (kg/d)	0.032	0.013[b]
Drinking water consumption rate (l/d)	2	1
Consumption rate of soil (mg/d)	100	200
Fraction of week soil contact occurs	0.286	0.714
Fraction of year soil contact occurs	0.417	0.500

[a] Values from NYSDOH [7] unless otherwise noted.
[b] Value scaled from the adult ingestion rate by the child:adult ratio of beef ingestion.

example, an average adult body weight of 70 kg is routinely used in dose calculations, and a 30-year exposure period is typical [26].

Still, exposure profiles are subject to considerable discretion, and the art of exposure assessment is to choose a combination of assumptions that satisfies the goals of the assessment and is appropriate for the populations of interest. Implications of parameter variability and uncertainty are difficult to test with deterministic methods, but probabilistic techniques such as those described in Section 3.5 can directly incorporate these effects and are gaining in sophistication and utility.

A typical set of exposure assumptions is used in the example. Specific parameter values, which are adopted from regulatory guidance, are listed in Table 7. Exposure profiles for both an adult and a $2\frac{1}{2}$ year old child are developed, and represent MEI-type exposure levels. Table 8 summarizes the exposure estimates for adults developed from the exposure parameters of Table 7 and the exposure point concentrations listed in Table 6.

Exposure estimates for the $2\frac{1}{2}$-year old child are higher than adult rates for all routes considered. Within a given exposure route, the ratio of doses estimated for a child and an adult is constant for all chemicals since doses are derived from the same exposure point and concentrations.[8] For brevity, only the ratios of exposures are listed at the bottom of Table 8. Dose estimates for a $2\frac{1}{2}$-year old child are at least 2 to 3 times greater than those for an adult, and the differences between adult and child exposures are greatest for the soil and cow's milk ingestion pathways (exposure ratios

[8] Differences result from contact rates, frequencies, and body weights, as indicated in Eq. (16). Exposure rates for the $2\frac{1}{2}$-year old child may be calculated from the information in Table 8 by multiplying the chemical-specific doses estimated for adults by the child-to-adult ratios listed at the bottom of the table.

Table 8
Exposure rate estimates for adults from ingestion pathways (mg/kg-d)

Contaminant	Soil	Leafy vegetables	Exposed vegetables	Protected vegetables	Cow's milk	Beef	Fish	Drinking water
Antimony (Sb)	5.53e − 09	4.62e − 08	3.92e − 08	2.12e − 08	1.03e − 08	1.16e − 08	7.00e − 09	4.32e − 07
Arsenic (As)	1.10e − 09	7.32e − 09	6.58e − 09	8.46e − 10	1.06e − 09	4.14e − 09	6.16e − 08	8.63e − 08
Beryllium (Be)	5.53e − 11	3.48e − 10	3.18e − 10	1.06e − 11	7.76e − 13	1.01e − 10	1.33e − 09	4.32e − 09
Cadmium (Cd)	2.22e − 09	2.69e − 08	2.82e − 08	4.25e − 08	5.43e − 08	3.21e − 09	2.27e − 07	1.73e − 07
Chromium VI (Cr VI)	4.42e − 10	2.77e − 09	2.60e − 09	2.54e − 10	1.03e − 08	4.45e − 09	8.96e − 09	3.45e − 08
Chromium III (Cr III)	3.98e − 09	2.50e − 08	2.34e − 08	2.29e − 09	9.29e − 08	4.01e − 08	8.06e − 08	3.11e − 07
Copper (Cu)	5.53e − 09	5.81e − 08	9.64e − 08	1.77e − 07	1.90e − 07	1.38e − 07	1.40e − 06	4.32e − 07
Lead (Pb)	2.93e − 09	1.96e − 08	1.79e − 08	3.37e − 09	1.18e − 08	1.66e − 09	1.82e − 07	2.29e − 07
Manganese (Mn)	2.22e − 08	1.97e − 07	1.78e − 07	1.42e − 07	1.51e − 07	1.93e − 08	2.81e − 06	1.73e − 06
Mercury (Hg)	1.90e − 09	3.03e − 08	2.88e − 08	4.87e − 08	2.57e − 08	8.95e − 08	1.33e − 05	1.49e − 07
Nickel (Ni)	5.53e − 09	3.78e − 08	4.70e − 08	4.24e − 08	9.22e − 08	6.41e − 08	3.29e − 07	4.32e − 07
Selenium (Se)	5.84e − 11	3.78e − 10	4.01e − 10	1.87e − 10	3.73e − 09	1.64e − 09	1.18e − 09	4.57e − 09
Vanadium (V)	4.42e − 10	2.76e − 09	2.57e − 09	1.69e − 10	1.37e − 08	2.02e − 09	3.08e − 06	3.45e − 08
Zinc (Zn)	5.53e − 08	1.24e − 06	2.66e − 06	6.35e − 06	2.38e − 05	2.38e − 05	3.29e − 06	4.32e − 06
Formaldehyde	2.93e − 09	1.82e − 08	1.66e − 08	NA	NA	NA	NA	2.29e − 07
PCBs	5.84e − 12	3.73e − 11	3.79e − 11	1.27e − 11	1.02e − 09	5.63e − 10	5.92e − 07	4.57e − 10
Total PAH	7.31e − 10	4.62e − 09	4.56e − 09	1.12e − 09	1.03e − 07	3.90e − 08	7.80e − 08	5.71e − 08
Total CPAH	1.46e − 11	9.24e − 11	9.11e − 11	2.24e − 11	2.06e − 09	7.80e − 10	1.56e − 09	1.14e − 09
2,3,7,8-TCDD TEQs	4.38e − 15	2.77e − 14	2.75e − 14	7.27e − 15	4.13e − 12	1.78e − 12	1.39e − 11	3.42e − 13
Ratio of exposure rates ($2\frac{1}{2}$-year old child to adult)	31.8	3.13	3.16	3.17	7.83	2.08	2.08	2.65

of 32 and 8, respectively). The ratios of these two routes are affected by large differences in consumption rates and (in the case of soil ingestion) frequency of exposure.

2.8. Dose-response assessment

By convention, the various adverse effects on health that may be caused by exposure to the contaminants released from an MWC are grouped into two broad categories: cancer, and all other adverse effects. Risks of these two sorts of harm are assessed in somewhat different fashions. For non-cancer health effects, different doses confer different degrees of risk of various adverse health effects. In general, risk is not attendant upon every measurable dose; at some sufficiently low level of exposure or dose, virtually all compounds carry no measurable or predicted risk. The break-point or threshold in a given dose-response curve is that non-zero dose at which the risk of an adverse response cannot be distinguished from zero. As a qualitative and quantitative matter, essentially all chemicals have been shown or are thought to have thresholds for most adverse effects.

Somewhat different analyses are applied to chemicals that are known or suspected to cause cancer in humans. The tenet of dose-response is still utilized – that is, for a given chemical carcinogen, it is assumed that high doses are more carcinogenic than moderate doses, which in turn are more carcinogenic than low doses. The critical difference is that however small a dose one considers, one continues to assume that there is some correspondingly small, non-zero risk of a response. In other words, one assumes that there is no threshold in the dose-response curve for chemical carcinogens – that if a chemical is a carcinogen at some (typically very high) dose, it will be a carcinogen at all doses.

By necessity, most of our information about what sorts of chemicals cause cancer derives not from observations on humans, but from studies in laboratory rodents. Currently, there are more than 700 chemicals shown to cause cancer in rats or mice following exposure to very high levels for essentially all of their lives. Only about 40 of these chemicals (or mixtures of chemicals) are also known to cause cancer in humans following high-level exposures. This leaves over six hundred compounds that cause cancer in laboratory rodents but are not known to do so in humans – either because epidemiologists have not identified populations exposed to the compounds at large enough levels for long enough periods of time to allow a meaningful study of patterns of morbidity and mortality, or because, to the extent that exposed human populations have been studied, no excess risks of cancer have appeared. It is nonetheless prudent and conventional to regard these many hundreds of compounds *as if* they are probably carcinogenic in humans, regardless of the human data or lack thereof. These compounds have thus come to be termed "probable human carcinogens".

2.8.1. Estimates of dose-response for effects other than cancer

For end points other than cancer, we compare the doses derived from the MWC with two measures of 'acceptable doses', depending on whether the dose is oral or

inhaled.[9] For oral and dermal doses, we identify or calculate a quantity called a reference dose (R_{fD}). An R_{fD} is an estimate of the daily dose of a chemical to which people (including sensitive subgroups) can be exposed for their entire lifetimes and not incur appreciable risk of health effects. A reference dose can be identified only for chemicals that demonstrate a threshold of toxicity and typically includes safety factors ranging from 30 to 1000. For inhalation exposure, a reference concentration (R_{fC}, in mg/m^3) is identified and represents an airborne concentration (modified by safety factors) of a chemical that may be tolerated for a lifetime without causing non-carcinogenic health effects. If doses or concentrations projected to result from operations of the proposed MWC are less than the appropriate reference doses or concentrations, no significant risk of harm is expected.

US EPA and other agencies have estimated R_{fD}s and R_{fc}s for exposure to various chemicals, and these values are listed in Table 9. R_{fD}s and R_{fc}s are periodically updated to incorporate the findings of new studies, and databases must be checked for current values. The US EPA has not developed toxicity values for some chemicals. In these cases, risk-based standards may be available from state agencies, or it may be possible to extrapolate R_{fD}s and R_{fc}s across exposure modes or derive them from the results of toxicological studies.[10]

Lead is a notable exception that often requires special evaluation because, unlike other chemicals, it seems not to possess a threshold for non-cancer toxicity. Consequently, use of R_{fD}s and R_{fc}s is inappropriate. Alternatively, changes in blood lead concentrations caused by exposure to environmental media and foods containing lead can be evaluated with empirically derived slope factors, as described in Section 2.9.4.

2.8.2. Estimates of cancer potency and unit risk

Ten contaminants or groups of contaminants herein considered are known to be carcinogens, either in rodents or humans or both. Each has been assigned an estimated carcinogenic potency by the US EPA. The potency estimates derive either from the results of chronic bioassays in rodents or, less often (but preferably), from the results of epidemiologic studies. The dose-response factors, also termed 'potency slope factors' or 'unit risks', are used to derive quantitative upper-bound estimates of carcinogenic risk for each chemical. Potency slope factors and unit risks are expressed on a risk per unit exposure basis. For oral and dermal doses, the potency slope factor P is expressed in units of kg-day/mg; the numerical value of P for a particular chemical corresponds to the estimated incremental risk of cancer conferred by an exposure of 1 mg/kg-d for life.

Incremental cancer risks for inhalation are quantified by unit risk values (U). Expressed in units of $m^3/\mu g$, the numerical value of U for a particular chemical is

[9] If dermal exposure is also considered, dermal R_{fD}s can be derived from R_{fD}s established for other modes of exposure (usually oral) by accounting for relative differences in absorption between the modes.

[10] Efforts have concentrated on the development of oral R_{fD}s. The US EPA has developed inhalation R_{fC}s for only a few chemicals. As such, R_{fC}s must be sought from alternative sources and sometimes derived from toxicologic studies, as indicated in the footnotes of Table 9.

Table 9
Toxicological data for chemicals of concern[a]

Contaminant	Reference concentration R_{fC} (mg/m^3)	Reference dose R_{fD} (mg/kg-d)	Unit risk (m^3/μg)	Potency (kg-d/mg)
Antimony (Sb)	1.40e − 03[b]	4.00e − 04	NA	NA
Arsenic (As)	1.05e − 03[b]	3.00e − 04	4.30e − 03	1.75e + 00
Beryllium (Be)	1.00e − 05[c]	5.00e − 03	2.40e − 03	4.30e + 00
Cadmium (Cd)	2.00e − 05[d]	5.00e − 04	1.80e − 03	ND
Chromium VI (Cr VI)	1.00e − 04[d]	5.00e − 03	1.20e − 02	ND
Chromium III (Cr III)	1.00e − 04[d]	1.00e + 00	NA	NA
Copper (Cu)	ND	3.70e − 02	NA	NA
Lead (Pb)	NA	NA	ND	ND
Manganese (Mn)	3.00e − 04[d]	5.00e − 03	NA	NA
Mercury (Hg)	3.00e − 04[d]	3.00e − 04	NA	NA
Nickel (Ni)	2.00e − 05[d]	2.00e − 02	2.40e − 04	ND
Selenium (Se)	1.75e − 02[b]	5.00e − 03	NA	NA
Vanadium (V)	2.00e − 04[d]	7.00e − 03	NA	NA
Zinc (Zn)	5.00e − 02[d]	3.00e − 01	NA	NA
Formaldehyde	3.00e − 02[e]	2.00e − 01	1.30e − 05	ND
PCBs	2.45e − 04[b]	7.00e − 05	2.20e − 03	7.70e + 00
Total PAH	4.00e − 02[f]	3.00e − 02	NA	NA
Total CPAH [benzo(*a*)pyrene]	NA	NA	2.09e − 03	7.30e + 00
2,3,7,8-TCDD TEQs	3.50e − 09[b]	1.00e − 09[g]	3.34e + 01	1.56e + 05

ND: no data, NA: not appropriate.

[a] Unless noted otherwise, obtained from the US EPA's integrated Risk Information System [54] or Health Effects Assessment Summary Tables [55].

[b] Extrapolated from the oral R_{fD} asuming an inhalation rate of 20 m^3/d and a body weight of 70 kg.

[c] Derived from data in NYSDEC [56].

[d] Derived from Ambient Air Criteria documents developed by the New York State Department of Health [57].

[e] Developed from a short-term standard developed by the New York State Department of Health.

[f] Developed from information in NTP [58], treating PAHs as naphthalene.

[g] ATSDR [28].

interpreted as an estimate of the excess risk of cancer incurred from a lifetime of exposure to 1 μg/m^3 of that chemical in the air.

Potency and unit risk values are listed in Table 9. As for R_{fD}s and R_{fC}s, values of P and U may change periodically and databases must be checked to ensure the use of the most current values.

2.9. Risk characterization

This section presents quantitative estimates of chronic human health risks that may ensue from operation of the example MWC. Risks of non-cancer (Section 2.9.1) and carcinogenic (Section 2.9.2) health effects simply combine estimates of exposure and

toxicity for the chemicals of concern. Such estimates are the typical output of a quantitative health risk assessment. In addition, two specialized risks are considered. First, excess cancer risks to nursing infants are estimated in Section 2.9.3. Second, potential effects of childhood exposure to lead are considered in Section 2.9.4.

Although numerous chemicals, exposure pathways, and health effects are considered in this example, the analysis is by no means exhaustive, and additional analyses may be warranted in some cases. For example, only chronic risks are considered herein. Risks from short-term exposure to air pollutants may also be of concern.[11]

2.9.1. Estimates of non-carcinogenic risks to children

Estimates of the risks of adverse health effects other than cancer are assessed for a 2½-year old child to evaluate worst-case risks, since children tend to have higher ratios of pollutant intake to body weight (as is the case consistently in Table 8). Non-cancer risks are calculated as hazard ratios and are evaluated halfway through an assumed 70-year operational period of the MWC. Generically, a hazard ratio is defined as an estimated exposure concentration or dose divided by a reference concentration or dose that corresponds to a level at which no adverse health effects are anticipated. The formulae for computing hazard ratios are differentiated by the mode of exposure. An inhalation hazard ratio $H_{c,i}$ is defined as the ratio of the modeled concentration of a contaminant in air (c_a) to its reference concentration (R_{fC}), where the subscripts 'c' and 'i' are used again to signify that the hazard ratio is defined for a particular combination of chemical and exposure route (in this case, inhalation). Similarly, a hazard ratio for oral exposure is designated by the symbol $H_{c,i}$, and is calculated as the ratio of exposure (e_0) to a reference dose. As a screening criterion, a total hazard index H_{total} is defined as the arithmetic sum of all of the individual hazard ratios over all chemicals and exposure routes:[12]

$$H_{total} = \sum_{chemicals} \left(H_{c,i} + \sum_{oral\ pathways} H_{c,0} \right) = \sum_{chemicals} \left(\frac{c_a}{R_{fC}} + \sum_{oral\ pathways} \frac{e_0}{R_{fD}} \right). \quad (17)$$

The value of H_{total} that results from the example MWC is 0.2.[13] The chemicals and exposure routes that constitute the bulk of the total hazard index are indicated in Fig. 4. Mercury exposure from the ingestion of contaminated fish is the principal contributor to the total hazard index. Mercury and PCDD/PCDFs account for 82% of the total hazard index. Similarly, two pathways – fish ingestion and cow's milk

[11] Typically, a proposed MWC plant must undergo rigorous permitting studies that include the evaluation of short-term air pollutant impacts.

[12] A combined hazard index of this sort is typically presented. Should H_{total} exceed values of concern, hazard indices for individual health endpoints may be calculated.

[13] Risk estimates (and the underlying estimates of exposure point concentrations and doses) are presented to 2–3 significant figures for the benefit of readers who wish to reproduce the calculations. It must be borne in mind that these estimates have an *accuracy* that corresponds to (at best) one significant digit, and even then, only as estimates of an upper bound of risk.

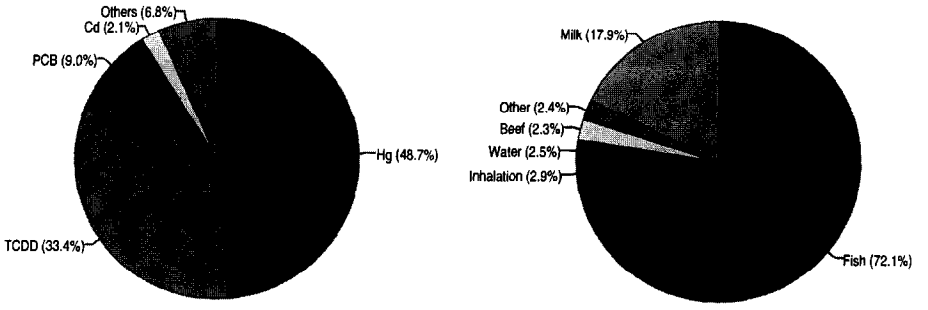

Fig. 4. Apportionment of the total hazard index for non-cancer risks due to MWC emissions.

ingestion – contribute 90% of the risk total when examined across exposure pathways. Inhalation, the most obvious route of exposure to MWC emissions, is only a minor portion (3%) of the total hazard index, which stresses the importance of indirect exposure pathways.

2.9.2. Estimates of carcinogenic risks to adults

Upper-bound estimates of the incremental lifetime cancer risks that may ensue from operation of the proposed MWC are calculated as the product of lifetime (70 years) exposure and carcinogenic slope factors. Table 8 summarizes daily exposure estimates to adults resulting from emissions from the MWC. The carcinogenic potencies and unit risk factors are given in Table 9. Risks are calculated for each exposure to a known or suspected carcinogen. As for non-cancer effects, the formulae used to calculate excess cancer risks differ depending upon the mode of exposure.

For inhalation exposure, incremental cancer risk ($R_{c,i}$) is estimated as the product of the time-averaged exposure point concentration in air (c_a) and the unit risk factor (U). Risks from oral pathways ($R_{c,0}$) are estimated as the product of the oral potency slope (P) and the estimated exposure (e_0). Since we assume that the risk estimates are additive, an estimate of the total incremental risk of cancer (R_{total}) is the sum of the individual estimates derived for each combination of chemical and exposure pathway:

$$R_{total} = \sum_{chemicals} (R_{c,i} + \sum_{oral\ pathways} R_{c,0}) = \sum_{chemicals} (c_a U + \sum_{oral\ pathways} e_0 P). \quad (18)$$

An R_{total} of 8×10^{-6} is estimated for the example MWC.[13] Fig. 5 depicts the distributions of chemicals and exposure pathways that comprise the excess cancer risk estimate. As for the total hazard index, the fish ingestion pathway dominates (84%) the excess cancer risk estimate. Ingestion of cow's milk and beef, which are also multi-step food-chain pathways, contribute lesser percentages (8% and 4%, respectively), but are still larger contributors than the direct inhalation route. On a chemical basis, PCBs and 2,3,7,8-TCDD TEQs provide 94% of the total excess cancer risk estimate, primarily due to their tendency to bioconcentrate in fish.

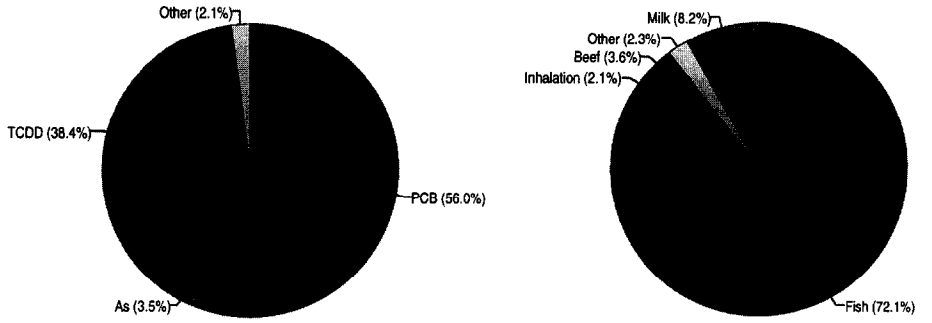

Fig. 5. Apportionment of estimated lifetime excess cancer risk due to MWC emissions.

2.9.3. Excess cancer risk to infants

A nursing infant can experience a significant portion of his lifetime cancer risk by nursing from a mother who herself is exposed to pollutants that originate from an MWC. The risk increment can be important because the per-body-weight exposure of an infant is large. Typically, such risk arises from exposure to highly lipophilic compounds that are concentrated within the fat of mother's milk. Two groups of chemicals – PCBs and 2,3,7,8-TCDD TEQs – are considered as examples.

The exposure rate during the period of nursing depends on the mass fraction of contaminant in mother's milk, the consumption rate of mother's milk, and body weight of the infant. Contaminant mass fractions in mother's milk are estimated with a steady-state model [27]:

$$m_{\mathrm{mm}} = \frac{d_{\mathrm{tot}}\alpha_{\mathrm{m}}\alpha_{\mathrm{f}}t_{1/2}}{\ln(2)\alpha_{\mathrm{w}}}, \tag{19}$$

where m_{mm} is the contaminant mass fraction in mother's milk (mg/kg), d_{tot} the total maternal absorbed dose of contaminant (mg/kg-day), α_{m} the weight fraction of mother's milk that is fat (0.04), α_{f} the fraction of contaminant that distributes to maternal fat (0.8), α_{w} the fraction of weight of mother that is fat (0.3), and $t_{1/2}$ the half life for contaminant in the mother's body (2555 d for TCDD, 1825 d for PCBs).

Default parameter values listed in parentheses are taken from NYSDOH [7]. The total maternal absorbed dose (d_{tot}) is estimated from adult exposure estimates developed in the baseline example. Absorption fractions that convert applied exposures to absorbed doses are obtained from toxicological profiles [28, 29]; route-specific values are listed in Table 10. The infant's exposure (e_{mm}, mg/kg-day) during the period of nursing is given by:

$$e_{\mathrm{mm}} = \frac{m_{\mathrm{mm}}\gamma_{\mathrm{mm}}}{W} \tag{20}$$

where γ_{mm} is the consumption rate of mother's milk (0.8 kg/day), and W the infant's body weight (8 kg).

Table 10
Estimated exposure and incremental cancer risk due to nursing

Chemical	Applied maternal exposure (mg/kg-d)		Absorption fractions		Absorbed maternal dose (mg/kg-d)	Dose estimate to infant (mg/kg-d) [from Eq. (20)]	Incremental risk of cancer[b]
	Inhalation (from Table 6)[a]	Oral intake (summed over all routes in Table 8)	Inhalation	Oral			
PCBs	1.23e − 10	5.94e − 07	0.80	0.95	5.64e − 07	1.58e − 05	1.74e − 06
2,3,7,8-TCDD TEQs	9.23e − 14	2.01e − 11	0.37	0.55	1.11e − 11	4.36e − 10	9.72e − 07

[a] The concentrations listed in Table 6 are converted to an applied dose with standard assumptions of a 20 m^3/d respiration rate and a 70 kg body weight.
[b] Estimated for one year of nursing over a 70-year lifetime.

Incremental cancer risks are calculated by averaging the nursing exposure rates over a lifetime and multiplying by the cancer potency estimates listed in Table 9. Incremental cancer risks of 2×10^{-6} and 1×10^{-6} are estimated for PCBs and TCDD TEQs, respectively. These values total almost half of the excess cancer risk estimated for adulthood exposure (8×10^{-6}), emphasizing the potential importance of the nursing pathway.

As demonstrated by this example and Table 8, exposure rate estimates are higher for children than for adults. Lifetime average exposure estimates, if properly integrated from birth to death, would be higher than the adult-based doses listed in Table 8. Timing considerations, however, often render the juxtaposition of exposure periods unlikely. For example, two generations must live at the worst-case location for an MEI and receive high-end exposure to MWC plant emissions both as a nursing infant and throughout the remainder of her/his life.

2.9.4. Lead

Because the toxicity of lead seems not to exhibit a threshold, or a dose below which no effect is likely, the importance of exposures to lead cannot be evaluated using reference doses or concentrations. Instead, one can use data describing the changes in the level of lead in the bloodstream that occur following an exposure to lead, and compare predictions to typical blood lead concentrations and concentrations associated with particular health effects. The magnitudes of coefficients, or slopes, relating environmental concentrations of lead to equilibrium blood lead increments have been extensively reviewed [30, 2]. Conservative estimates of these slopes[14] are presented in

[14] More detailed rationale for the development of blood-lead slope factors is available from the authors.

Table 11
Projected increase in levels of blood lead due to MWC emissions

Exposure pathway	Slope factor	Exposure rate	Blood lead increment (μg/dl)
Inhalation	2 (μg/dl)/(μg/m^3)	0.000216 μg/m^3[a]	0.00043
Ingestion of surface soil, vegetables, cow's milk, beef, fish, and water	0.16 (μg/dl)/(μg/d)	0.0143 μg/d[b]	0.00228
Total blood lead increment			0.00271

[a] From Table 6.
[b] Calculated from estimates of dose (Table 8, adjusted for the 2½ year-old child) and a 13.2 kg body weight (Table 7).

Table 11 along with predicted increases in blood lead concentrations resulting from the exposure levels predicted for the MWC example.

The total projected increase in the concentration of lead in the blood of the child MEI is less than 0.003 μg/dl. This increase represents less than 0.1% of the average blood level (4 μg/dl) in US children [31]. The Centers for Disease Control does not consider children with levels below 10 μg/dl to be poisoned [32]. An increase of this magnitude would not be measurable and would not perceptibly alter a child's health.

3. Recent topics in multi-pathway risk assessment

The MRA presented in Section 2, while extensive, fails to include a number of phenomena that can profoundly affect risk estimates. The implications of some of these findings are discussed in this section.

3.1. Wet deposition

Particle-bound pollutants are removed from the atmosphere by both dry and wet deposition. The latter process, in which particles are scavenged from the air by precipitation, is not considered in the Section 2 risk assessment, and has been excluded from many other assessments of MWCs. Recent guidance [2, 3], however, promotes the calculation of wet deposition since it contributes to the mass of pollutants that cycles through indirect exposure pathways.

The pattern of wet deposition differs from that of dry deposition since, among other things, scavenging occurs vertically throughout the air pollutant plume, and not just at ground-level. Consequently, models predict the highest rate of wet deposition to occur in the immediate vicinity of the stack, as opposed to dry deposition, for which the highest rates occur some distance from the facility where the airborne plume is predicted to reach the ground.

The COMPDEP model contains algorithms to estimate wet deposition.[15] These algorithms were applied to the example MWC, and the results (along with those of dry deposition) are depicted in Fig. 3. Average deposition rates are presented for each distance from the source, and separate curves are presented for VW and SW pollutants (see Section 2.3).[16] As expected, modeled wet deposition rates are highest near the MWC, and decrease in an exponential manner characteristic of a first-order removal process. Dry deposition rates are highest a distance of about 2 km downwind of the source. At locations close to the source (0–1000 m), the rate of wet deposition can be orders of magnitude greater than that of dry deposition.[17] At a distance of 2 km from the source, wet and dry deposition rates become equal for VW pollutants. For SW pollutants, however, wet deposition remains a factor of five greater than dry deposition to the boundary (6 km) of the study domain.

Failure to account for wet deposition has mixed implications in an MRA. Potentially high rates of pollutant deposition near a source may be incorrectly ignored. Such rates could be of primary importance to small water bodies close to the MWC. Impacts over an entire watershed are less significant, however. For VW pollutants, the area-weighted rate of wet deposition integrated over the 6 km radius study is about $1\frac{1}{2}$ times the dry deposition rate.[18]

For SW of pollutants, omission of wet deposition may be of greater significance, since the ratio area-weighted deposition rates (wet/dry) is almost a factor of ten. The importance of wet deposition may, however, be moderated by fate and transport considerations. In many MRAs, terrestrial food chain pathways dominate risk estimates. As demonstrated in Section 2.6, these pathways depend heavily on pollutant deposition to the surface of vegetation. Since only a fraction of pollutants deposited by wet deposition are likely to remain on plant surfaces (precipitation may actually serve to wash off pollutants already present), the overall amount that wet deposition will add to risk estimates is likely to be less than that implied by the total deposition rate.

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[15] The wet deposition module of COMPDEP requires the specification of scavenging coefficients, which depend on particle size and rainfall intensity. These coefficients were developed for the particle distribution indicated in Fig. 2 from the data collected by Radke et al. [32], and range from 8×10^{-5} to 1.2×10^{-3} h/mm-s. They are then multiplied by average precipitation rates for the three precipitation intensity classes designated by the US EPA [3] (1, 3.8, and 15 mm/h, respectively, for light, moderate, and heavy rains) for use in COMPDEP.

[16] Average deposition rates are calculated as the mean of the 36 values modeled at each radial distance. Maximum point estimates are about 1.6 and 2.9 times higher than the peak dry and wet deposition rates depicted in Fig. 3.

[17] Since these calculations were performed at a semi-arid location (the total rainfall at Denver, CO was 15.5 in in 1989), this disparity can even be greater at other sites where annual rainfall is greater.

[18] As a somewhat different matter, it should be noted that the fraction of particles removed from the airborne plume over the 6-km-radius study area totalled only about 1% (wet and dry deposition combined). In cases such as this, however, plume depletion is a relatively insignificant source of uncertainty, and traditional double-counting techniques in which pollutant deposition is estimated as a zero-depletion, add-on calculation are of sufficient accuracy. DEPST, a developmental model developed by the US EPA, contains sophisticated algorithms for accounting for plume depletion, and is indicative of a greater trend to develop physically correct models. In our opinion, efforts could be better expended to areas of greater uncertainty in multipathway risk assessment.

Unfortunately, this fraction is not well known, and at this time is a source of considerable uncertainty.

A final caution concerns the reliability of wet deposition algorithms. Although conceptually simple, wet deposition models have not been studied extensively, and the empirical scavenging coefficients that serve as the basis of most models have been questioned. Specifically, it is thought that wet deposition algorithms such as those employed by the COMPDEP model may overestimate particle deposition by one or more orders of magnitude [34]. In our opinion, validation studies of wet deposition models are necessary.

3.2. *Modeling of mercury*

The perception of mercury as a burgeoning threat to human health has been heightened by increasingly frequent reports of elevated concentrations in freshwater fish. Mercury's behavior in the environment is extremely complex, and simple models may be inadequate to assess mercury concentrations in fish that may result from mercury emissions from MWCs or other sources. The US EPA, as required by the Clean Air Act Amendments of 1990, is sponsoring a detailed examination of mercury, and findings are expected to be available soon.

As a result, we expect that MWC risk assessments will treat mercury emissions much differently than demonstrated in the baseline example. Therein, mercury is treated as an SW pollutant because of its high vapor pressure relative to most metals. Mercury is notoriously difficult to capture in typical pollution control devices, and evidence suggests that mercury escapes from MWCs primarily in the vapor phase [35]. Subsequently, its environmental transport may be unrelated to the fate of small particles (as modeled in Section 2.3).

There are also uncertainties regarding the chemical species of mercury that are relevant to releases from MWCs. Detailed stack gas testing of an MWC has demonstrated high levels of oxidized (Hg II) species (possibly $HgCl_2$ or HgO) [35], while elemental mercury predominates in the atmosphere [36]. This difference is part of mercury's complex and incompletely understood behavior in the environment.

Implications of species and phases for modeling of mercury deposition are unclear. If most mercury is released from MWCs as vapor, the ability of particle deposition modeling typical of traditional MRAs to predict the correct magnitude of dry deposition (irrespective of the correct physical mechanism) may be tied to atmospheric speciation. If elemental mercury dominates MWC emissions (as it does in the general distribution of species measured in the atmosphere), a dry deposition velocity of 0.1 cm/s may be appropriate [37]. Alternatively, if the principal species released from MWCs is $HgCl_2$ (which is thought to be a relatively reactive gas), the dry deposition velocity may be of the order of 1 cm/s [38]. The order of magnitude difference between these values roughly reflects the difference between SW and MW particle deposition velocities in the baseline example (Section 2.3). Since mercury is typically treated as an SW pollutant in traditional MRAs (as it is in the baseline example), deposition rates may reflect the lower end of potential estimates.

Furthermore, if (as it appears) a large part of the mercury from MWCs is released in soluble, oxidized forms (such as $HgCl_2$), wet deposition could be the most significant removal mechanism.[19] By ignoring wet deposition, traditional MRAs may fail to account for the correct physical removal mechanism. An order of magnitude comparison may give some sense of the consequences of mercury deposition modeling. Measured ratios of total mercury concentrations in precipitation to those in air are of the order of 100 ng/l per ng/m^3 [36]. Assuming that (1) inorganic, oxidized mercury (e.g., $HgCl_2$) is the only form effectively scavenged by precipitation, and (2) the fraction of atmospheric mercury in this form is 20% [39], an effective scavenging ratio (expressed as a ratio of concentrations) for $HgCl_2$ is roughly 500 ng/l per ng/m^3. This value can be compared to scavenging ratios predicted by the COMPDEP model in the baseline example for SW particle deposition. At a distance of 6 km from the source (where the pollutant concentration is uniform in the vertical direction), COMPDEP predicts effective scavenging ratios of 90 and 400 ng/l per ng/m^3 for dry and wet deposition, respectively. The latter value suggests that, even if incorrect from a physical perspective, the modeling of mercury as particle-bound pollutant yields a wet deposition scavenging ratio comparable to values measured[20] for gaseous $HgCl_2$. The effective scavenging ratio based upon dry deposition, however, is about a factor of five lower, which suggests that consideration of only dry (particle-bound) deposition in traditional MRAs may underestimate the magnitude of mercury deposition if, in fact, the dominant removal mechanism is precipitation scavenging of $HgCl_2$.

The challenges of mercury extend far beyond atmospheric deposition, however. Once introduced to surface waters, mercury partitions between dissolved and particle-bound phases, and among various inorganic (oxidized or elemental) and organic (methylated) chemical species. The nature and dynamics of its distribution within the water column and sediments depend highly on the characteristics of the water body (pH, suspended solids, organic content, etc.). Furthermore, the use of a generic bioconcentration factor of 5500 l/kg (as described in Section 2.5)[21] to estimate the relationship between mercury concentrations in surface water and fish may be completely inappropriate, since the degrees of bioconcentration and bioaccumulation in the aquatic food chain may differ greatly between surface waters. In some cases, mercury speciation may be an important consideration. For example, methylmercury bioaccumulation factors of 100 000 l/kg and greater have been measured for predator fish [41]. Ideally, bioaccumlation modeling should be tailored to surface waters of interest. Sophisticated surface water models are available [42], but their reliability may depend on the availability of extensive input data.

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[19] The potential importance of wet deposition is supported by the early results of research aimed at measuring wet deposition rates downwind of an MWC [40].

[20] Note that the typical measured value of 500 ng/l per ng/m^3 has been adjusted to reflect mercury speciation.

[21] The bioconcentration factor of 5500 derives from a general guidance document [30] and has been widely applied in risk assessments. Typically, it is applied to total mercury concentrations in surface waters. Under field conditions, however, there may be little correlation between total mercury levels in the water column and mercury concentrations in fish.

Uncertainties compound further when human interactions with mercury are considered. There are numerous sources of exposure,[22] different species of mercury are harmful via different exposure pathways, and metabolic conversions further complicate toxicity. Many issues must be explored further to understand the behavior and consequences of mercury released from MWCs.

3.3. *Fate and transport modeling of polychlorinated dioxins and furans (PCDD/PCDFs)*

PCDD/PCDFs are a group of compounds that have been treated traditionally as a single compound – 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (2,3,7,8-TCDD) – in risk assessments. Typically, a 2,3,7,8-TCDD toxic equivalent (TEQ) emission rate is synthesized as a weighted (with respect to the toxicity of 2,3,7,8-TCDD) sum of all PCDD/PCDF emissions. Various congener weighting methods have been developed; toxic equivalency factors currently endorsed by the US EPA [43] are listed in Table 12.

2,3,7,8-TCDD TEQs are typically carried through fate and transport modeling as if all PCDDs and PCDFs behave similarly to 2,3,7,8-TCDD. The environmental behavior of individual PCDD/PCDF congeners varies substantially, however. Serious errors may be introduced by the 2,3,7,8-TCDD approach, especially for MWC emissions, for which PCDD/PCDF distributions are frequently dominated by congeners that have transport properties that differ widely from those of 2,3,7,8-TCDD.[23]

Recent US EPA [3] guidance emphasizes the need to consider each PCDD/PCDF congener separately, and data to perform congener-specific fate and transport assessments are rapidly becoming available.[24] Such an analysis, however, requires a large number of parameters, and considerable research is still needed to characterize all of the media-to-media transfers of interest.

A second important development in PCDD/PCDF fate and transport modeling involves the distinction between vapor and particle-bound phases in air. Recent experiments have suggested that vegetation readily assimilates vapor phase PCDD/PCDFs. For some congeners, a small vapor fraction may contribute the majority of the pollutant that becomes incorporated in leafy tissues [3]. Consequently, the failure to consider vapor-phase absorption may cause PCDD/PCDF mass fractions in vegetation to be underestimated. Since vegetation (feed crop) modeling is integral to food chain pathways such as cow's milk and beef consumption, and these routes frequently dominate MRAs, overall risk estimates may be understated in traditional assessments that consider only particle-bound deposition.

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[22] Even seemingly obvious routes, such as exposure due to off-gassing from dental amalgams, are not completely understood.

[23] Stack emissions of WTE plants typically contain larger quantities of the more highly chlorinated (hexa- thru octa-) congeners, even when weighted by toxic equivalency factors.

[24] A lack of physicochemical data for congeners other than 2,3,7,8-TCDD was a principal reason for using the single chemical approach in fate and transport modeling. Until recently, much of the research on PCDD/PCDF focussed on 2,3,7,8-TCDD.

Table 12
Congener-specific analysis of PCDD/PCDF content in beef and cow's milk

Congener[a]	Toxic equivalency factor[b]	Emission rate (g/s)[c]	Vapor fraction[b]	Vapor uptake ratio[b]	Fat to feed bioconcentration factor[b]	Mass fraction in beef or milk fat (mg/kg)
2,3,7,8-TCDD	1	1.30e − 09	0.55	1.00e + 05	4.32	1.02e − 09
1,2,3,7,8-PeCDD	0.5	4.01e − 09	0.26	6.30e + 05	4.16	8.78e − 09
1,2,3,4,7,8-HCDD	0.1	3.78e − 09	0.07	2.30e + 06	2.02	3.99e − 09
1,2,3,6,7,8-HCDD	0.1	6.62e − 09	0.04	6.90e + 05	1.74	1.26e − 09
1,2,3,7,8,9-HCDD	0.1	8.86e − 09	0.02	6.90e + 05	2.24	1.34e − 09
1,2,3,4,6,7,8-HpCDD	0.01	1.04e − 07	0.02	1.00e + 07	0.36	2.41e − 08
OCDD	0.001	2.81e − 07	0.01	2.40e + 09	0.52	1.09e − 05
2,3,7,8-TCDF	0.1	1.19e − 08	0.71	1.50e + 05	0.94	3.77e − 09
1,2,3,7,8-PCDF	0.05	5.73e − 09	0.42	5.30e + 05	3.1	1.25e − 08
2,3,4,7,8-PCDF	0.5	1.13e − 08	0.3	3.80e + 05	0.73	3.07e − 09
1,2,3,4,7,8-HCDF	0.1	4.83e − 08	0.06	5.90e + 05	2.34	1.51e − 08
1,2,3,6,7,8-HCDF	0.1	2.25e − 08	0.06	1.40e + 06	2	1.28e − 08
1,2,3,7,8,9-HCDF	0.1	4.51e − 09	0.11	8.30e + 05	2	2.75e − 09
2,3,4,6,7,8-HCDF	0.1	3.32e − 08	0.07	8.30e + 05	1.78	1.20e − 08
1,2,3,4,7,8,9-HpCDF	0.01	1.74e − 07	0.04	6.80e + 05	0.41	7.76e − 09
1,2,3,4,7,8,9-HpCDF	0.01	3.58e − 08	0.02	6.80e + 05	0.99	2.37e − 09
OCDF	0.001	2.55e − 07	0.01	1.70e + 08	0.2	2.70e − 07
2,3,7,8-TCDD TEQ sum						2.43e − 08

[a] In each abbreviation, the last three letters indicate a chlorinated dibenzo-dioxin (CDD) or furan (CDF) congener. The preceding letter(s) T, Pe, H, Hp, and O, are short for tetra, penta, hexa, hepta, and octa, respectively, and indicate the number (4 to 8, in order) of chlorine atoms in the congener. Numbers indicate the positions of chlorine atoms on the two carbon rings of the dioxin or furan congener.

[b] From US EPA [43].

[c] Derived from stack testing of the Hempstead Waste-To-Energy Plant, Hempstead, New York [59].

A congener-specific PCDD/PCDF analysis that considers vapor phase transport can be developed for contrast with the baseline example. For simplicity, only fate and transport modeling of beef and cow's milk are considered, since exposure to these media frequently dominates risk estimates in MRAs [1].[25] For consistency, values of feed intakes and exposure parameters used in the baseline example are maintained. The analysis differs by first estimating congener-specific exposure levels (i.e., tracking each congener through fate and transport modeling), and then calculating a toxic equivalent 2,3,7,8-TCDD exposure.

PCDD/PCDF congener emission rates are estimated from emission testing of an operating MWC, and are the same data used to construct the 2,3,7,8-TCDD TEQ emission rate used in the baseline example [6]. These rates are listed in Table 12, which includes all of the congeners that the US EPA [43] considers 'dioxin-like.' The same measurements serve as the basis of the calculations that follow.

Modeling methods and parameters are developed to be consistent with recent US EPA [43] guidance. The total concentration of a congener in air (c_a) is assumed to comprise vapor (c_{av}) and particle-bound (c_{ap}) components:

$$c_a = c_{av} + c_{ap}. \tag{21}$$

Vapor-particle partitioning of PCDD/PCDFs has been both modeled and measured. Available data were analyzed by the US EPA [43] to derive vapor-particle distribution estimates for each congener, which are listed in Table 12. The fraction of each congener present in the vapor phase decreases with the degree of chlorination. More than half of tetra-chlorinated dioxins and furans are believed to be present as vapor, while octa-chlorinated species are almost completely bound to particles.

A total contaminant mass in a crop feed (m_{vt}) is predicted based upon the vapor and particle-bound components, which are modeled separately through the food chain:

$$m_{vt} = m_v^p(c_{ap}) + m_v^v(c_{av}), \tag{22}$$

where $m_v^p(c_{ap})$ is the contaminant mass fraction in vegetation that results from particle deposition, which is derived from the particle-bound contaminant concentration in air (c_{av}), and $m_v^v(c_{av})$, the contaminant mass fraction in vegetation due to vapor uptake, which is related to the vapor-phase contaminant concentration in air (c_{av}).

Modeling of m_v^p proceeds in the same fashion as described in Sections 2.3, 2.4, and 2.6, except that the particle-bound concentration (c_{ap}) is used in place of the total concentration (c_a). Particles containing PCDD/PCDFs are assumed to deposit to soil

[25] Recent guidance [13] also contains sophisticated algorithms for surface water modeling, and a congener-specific method is also recommended for estimating the bioconcentration of PCDD/PCDFs and PCBs in fish.

and on the surface of vegetation. Crops translocate contaminants from soil via root systems and assimilate those deposited on leaves and stems.[26]

Estimation of m_v^v is accomplished through an empirical relationship that assumes the equilibrium partitioning of airborne and sorbed pollutants:

$$m_v^v = \frac{B_{vv} c_{av} f_p}{\rho_a}, \tag{23}$$

where B_{vv} is the equilibrium ratio of the contaminant mass fraction in vegetation to that in air (vapor phase), f_p, the adjustment factor to account for the tendency for PCDD/PCDFs to remain concentrated in the exposed surfaces of vegetation (as explained below), and ρ_a, the density of air (1.19 kg/m^3 at standard conditions).

The vapor absorption of PCDD/PCDFs has been studied in azalea leaves [44] and grass [45]. Values of B_{vv} interpreted from these studies by the US EPA [43] are presented in Table 12. US EPA [43] also recognizes that PCDD/PCDFs do not translocate significantly in vegetation, and congeners tend to remain and concentrate in the surfaces where they are absorbed. Since empirical $B_{vv}s$ are derived from studies of leaves and grass that have high surface-to-volume ratios, crops that have significant portions protected from air are likely to assimilate less contamination per volume. Of the four crops considered, pasture grass is likely to mimic the vegetation studied, and is assigned a f_p value of unity. Hay, silage, and grain have increasingly larger protected portions. For these crops, f_v values of 0.5, 0.1, and 0.01 are assigned in consideration of discussion and recommendations contained in US EPA [43].

US EPA [43] does not distinguish between cows and steers, but rather predicts contaminant concentrations in animal fat that apply to both beef and cow's milk. Similar to the traditional approach, congener-specific contaminant mass fractions in fat are assumed to be proportional to the contaminant content of the animal's diet. The form of the correlation, however, is based upon the average contaminant mass fraction in feed, and not the total rate of contaminant intake (which considers the amount of feed consumed):

$$m_f = F_f m_{vta}, \tag{24}$$

where m_f is the contaminant mass fraction in beef fat or milk fat (mg/kg), m_{vta} the average contaminant mass fraction in feed (mg/kg), and F_f the dimensionless bioconcentration factor.

Values of m_{vt} are first computed for each crop using Eq. (22), and then m_{vta} is constructed using the intake rates listed in Table 4.[27] Congener-specific values of F_f,

[26] US EPA [43] dismisses translocation from soil as negligible compared to direct deposition onto plant surfaces. For consistency, however, root uptake is maintained in this example.

[27] The average feed concentration m_{vta} also considers incidental soil ingestion. For this purpose, mass fractions in soil are derived from particle deposition modeling (vapors are assumed not to deposit appreciably to soil). For simplicity, feed intake rates based for the steer are used for weighting. Use of the higher intake rates for the cow would produce slightly different results because the assumed soil ingestion rate does not scale similarly to the feed rates.

obtained from US EPA [43], are derived from a study by McLachlan et al. [46] and are listed in Table 12.

Modeled congener mass fractions in milk/beef fat are reported in Table 12. Weighted by toxic equivalency factors, the summed 2,3,7,8-TCDD TEQ mass fraction is 2.43×10^{-8} mg/kg. This value can be compared to mass fractions predicted in the baseline example, which models only the transport of 2,3,7,8-TCDD, and does not embody more recent data concerning bioconcentration factors. Assuming fat contents of 20% and 3% for beef and cow's milk, respectively, the traditional approach (Table 6) predicts 2,3,7,8-TCDD TEQ mass fractions of 1.22×10^{-8} mg/kg in beef fat and 3.40×10^{-8} mg/kg in milk fat. Interestingly, detailed, congener-specific modeling predicts a mass fraction within the range of the values predicted by the less sophisticated, traditional approach. The similarity of these results is probably fortuitous, however. The range of congener-specific mass fractions predicted in Table 12 illustrates the need to consider congeners separately, although much more work is needed to establish modeling parameters and characterize uncertainties.

3.4. *Toxicity assessment of PCDD/PCDFs*

Trends within the US EPA and elsewhere that may increase the level of sophistication of exposure modeling have yet to be matched by increments in the sophistication of toxicity modeling. In particular, the US EPA's approach to determining the carcinogenic potency of chemicals of potential concern is essentially unchanged since the 1970s: it is in dire need of improvement. The extreme estimate of cancer potency currently proposed by US EPA [47] for dioxin – 100 000 $(\text{mg/kg-day})^{-1}$ – drives the results of many multi-pathway risk assessments for combustors and other sources of dioxin. If a more valid estimate of such potency is much lower – and we believe the scientific evidence is that it is, at exposure levels of interest – then the results of all site-specific risk assessments will be lowered accordingly.

The approach to cancer potency estimation for dioxin (as for other carcinogens) used by the US EPA is based on the assumption that all levels of exposure, however small, are carcinogenic. As it turns out, such an approach is not based on all of the actual data from the 1978 bioassay of dioxin in laboratory rats [48], and may not be necessary for the intended protection of the public health (at least with respect to cancer risk).

Since at least 1978, it has been known that dioxin causes cancer in laboratory rats. Also known since then is that dioxin also *prevents* certain kinds of cancer. Table 13 and Table 14 summarize the results of the cancer bioassay for dioxin that serves as the foundation of the US EPA's cancer potency estimate [48].[28]

[28] Some of the percentages in Table 13 and Table 14 may be incorrect. Kociba et al. [48] list the overall numbers of animals examined pathologically at each of the dose-levels, but they also indicate that not all animals were examined for all tissues and/or targets. Thus, we do not know the target-specific denominators on which each percentage depends. Since not all tissues were examined in all animals, and since some animals died early, some denominators may be substantially in error. As a first-level analysis, though, we may assume that the correct denominators are those listed by Kociba et al. [48] and incorporated in the tables given here.

Table 13
Tumor responses (%) seen in male Sprague Dawley rats fed 2,3,7,8-TCDD

	Dose (pg/kg-day)			
	0	1000	10000	100000
Increased response observed				
Stratified squamous cell carcinoma of hard palate or nasal turbinates	0	3	0	13
Stratified squamous cell carcinoma of tongue	0	2	2	7
Adenoma of adrenal cortex	0	0	4	10
Decreased response observed				
Subcutaneous fibroadenoma/fibroma/lipoma	12	2	10	12
Pituitary adenoma	31	12	22	26
Acinar adenoma of pancreas	17	14	10	4
Pheochromocytoma of adrenal	33	12	20	8
Interfollicular C-celladenoma of thyroid	10	0	0	8

These data reveal several interesting things. First, they show that female rats and male rats respond differently to the carcinogenic, and anti-carcinogenic, effects of dioxin. These differences are largely the result of dioxin's interactions with estrogen.

Second, even the tumors which are clearly increased at high levels of exposure – such as the liver tumors – are not increased at low levels of exposure, and may even be decreased (that is, prevented) at such levels.

What would seem to be required is a simple, unbiased analysis that will both (1) treat the rodent tumor data as they appear and (2) allow an appropriate degree of conservatism in translating the rodent data to human risk assessment.

One approach would be as follows. First, recognize the sex-dependence of dioxin-induced carcinogenesis, and so develop one set of potency estimates for females and another for males. As mentioned before, the clear estrogen-dependence of dioxin-induced carcinogenesis (and anti-carcinogenesis) makes the US EPA approach an unscientific oversimplification.

Second, analyze each tumor type on which dioxin has, or might have, an effect, whether positive, negative, or neutral. In practice, this would mean looking at each of the 38 tumor types in females and 43 tumor types in males that were listed by the original investigators [48]. US EPA [47], in contrast, restricts itself to the single dose-response curve that describes liver tumors (adenomas and carcinomas combined, as reassessed by Sauer [49]) in the female rats studied by Kociba et al. [48]. There is no technical reason to disregard all of the other results for all of the other tumor types.

Third, after generating dose-response relationships for each tumor type, combine them. So doing, one would answer the question, What is the *net* effect of exposure to dioxin on development of cancer? Mathematically, the most straightforward way to derive a net result is to add together the slopes of each dose-response relationship. Tumor types that are increased by exposure to dioxin will have a positive slope; tumor

Table 14
Tumor responses (%) seen in female Sprague Dawley rats fed 2,3,7,8-TCDD

	Dose (p/kg-day)			
	0	1000	10000	100000
Increased response observed				
Hepatocellular arcinoma/adenoma	4	3	33	58
Stratified squamous cell carcinoma of hard palate or nasal turbinates	2	0	4	21
Keratinizing squamous cell carcinoma of lung	0	0	0	14
Decreased response observed				
Benign tumor of uterus	33	24	22	15
Benign neoplasm of mammary gland	85	70	72	50
Carcinoma of mammary gland	9	8	8	0
Pituitary adenoma	50	36	26	25
Interfollicular adenoma of thyroid	16	2	4	8

types that are decreased by exposure to dioxin will have a negative slope; and tumor types that are unaffected by exposure to dioxin will have a slope of zero.

Such an analysis might show dioxin to be clearly and strongly carcinogenic (in rodents) at moderate levels (on the order of a few thousand picograms per kilogram body weight per day), but only weakly carcinogenic (and perhaps anti-carcinogenic) at both low doses (less than 1000 pg/kg-day) and high doses (greater than 100 000 pg/kg-day). Since 'low levels' covers both background exposures plus any plausible increments from sources such as MWCs, it follows that the potency developed for low levels is the one to use for the risk assessments of interest.

3.5. *Probabilistic risk assessment*

Early MRAs of MWCs focussed on worst-case (MEI) scenarios intentionally designed to overestimate risks. A conservative bias pervaded MRAs, largely to compensate for the numerous uncertainties inherent to the assumptions and parameters of the risk assessment [2]. Recent developments (such as those discussed in Section 3.3) have expanded the scope of risk assessments, and it is rather easy to compound a series of high-end assumptions to produce unacceptable (from a regulatory standpoint) risk estimates. Borrowing from guidance developed to assess hazardous waste disposal sites, reasonable maximum scenarios have been developed that mix conservative and average values of parameters in an attempt to estimate high-end, yet plausible, risks.

At the same time, however, recent guidance promotes the consideration of a range of exposures through the use of multiple risk-descriptors that could be construed to represent population segments. Also, the philosophical shift from the MEI to the RME is a manifestation of a deeper trend to confer added realism to risk estimates. It

is at present difficult, if not impossible, to gauge the meaning and accuracy of risk estimates from deterministic MRAs. A central tendency estimate intended to represent the average level of risk to a given population may actually lie far from the mean.[29,30] Comprehensive uncertainty analyses are lacking, in part because uncertainties and variabilities are numerous and sometimes nebulous, but also because regulatory agencies promote deterministic methods.

Algorithms to perform uncertainty analyses are readily available, however, and are gaining in popularity. Monte Carlo risk assessments have been endorsed in some regulatory circles [50] and input data are increasingly available [51]. On the surface, performing an uncertainty analysis is straightforward, and simply requires the determination of distributions for each parameter that quantifies uncertainty and variability, followed by sufficient random sampling to construct risk estimates. Two barriers hinder such analyses, however. First, data to characterize variability and uncertainty are limited or lacking for some parameters used in MRAs, especially for site-specific information. Second, the uncertainty analysis is straightforward so long as the models underlying the risk assessment are correct. The adequacy of many of the models that govern fate and transport modeling has not been determined. Thus, a full uncertainty analysis should account for uncertainties in the basic physical assumptions that serve as the foundation of model equations.

It is imperative that probabilistic techniques be pursued (along with the collection of data required for their application) since they afford the only avenue for developing full, interpretable risk distributions. Some interesting insights can be gained from tools already available. Crouch et al. [52] have carried out portions of a detailed protocol [53] to conduct a full Monte Carlo analysis of an MRA. Although developed for a hazardous waste combustor (and not specifically an MWC), the pathways and models considered are consistent with recent guidance [11] and are equally applicable to stack emissions from any combustion source. The analysis considered exposure to PCDD/PCDFs through the two pathways – beef and cow's milk ingestion – typically of greatest importance in MRAs of MWCs. The physical models themselves are assumed to be correct (they correspond closely to recent guidance [11], with some modifications). All 17 of the congeners listed in Table 12 were modeled from their release from the stack to their ultimate consumption in food, and 120 uncertainty or variability distributions were incorporated.

Results generated for high-end PCDD/PCDF exposure to a farmer living near the point of worst-case impacts (as projected by air dispersion modeling) are presented in

[29] When defining a central tendency measure, a specific population must be identified. For example the population could include all people living within a given radius about the WTE plant, or could be limited to farmers or other groups expected to experience exposure to contaminants released from a WTE plant.

[30] Central tendency risk estimates, as promoted in recent guidance, are misnamed. Proposed methods [11] suggest the development of average exposure levels. The dose-response data used to estimate risk levels from exposure estimates, however, still maintain a conservative bias, and will lead to conservative risk estimates (unless efforts are also made to develop best estimates of these parameters).

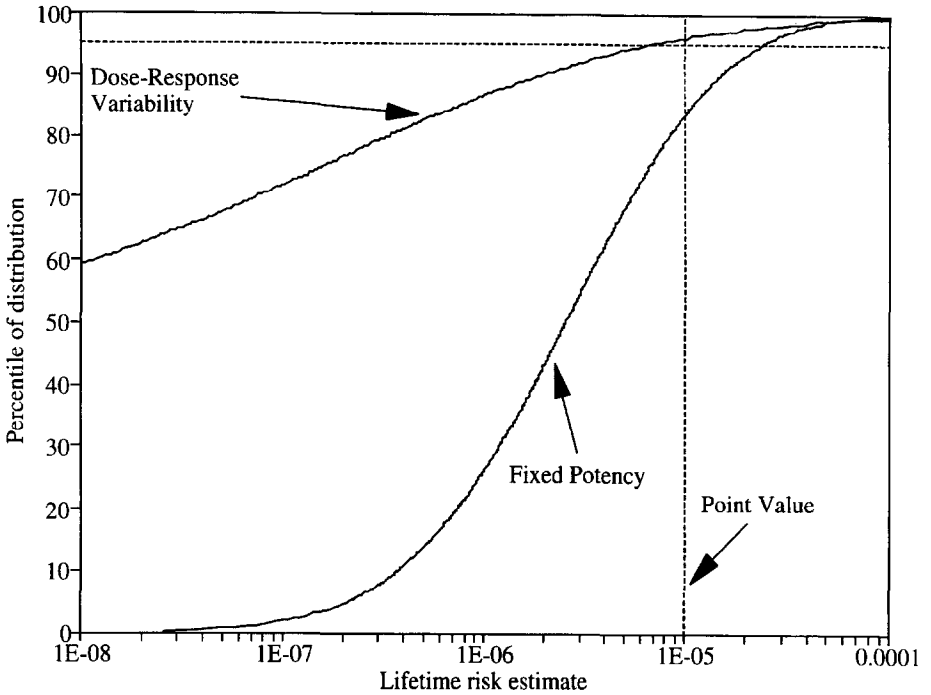

Fig. 6. Excess cancer risk distributions due to exposure to PCDD/PCDFs released from a combustion source [52].

Fig. 6.[31] Two risk distributions are depicted that are differentiated by the treatment of toxicologic data. The first curve, labeled 'Fixed Potency', models uncertainties and variabilities that affect exposure to PCDD/PCDFs, and risk estimates are generated with the fixed (conservative) estimate of cancer potency for 2,3,7,8-TCDD. The second distribution, labeled 'Dose-Response Variability', additionally incorporates variability and uncertainty associated with the cancer potency estimate.[32] For reference, a point risk estimate is also presented in Fig. 6. This risk estimate is derived using best estimates of the parameter values that are likely to be incorporated in a high-end (reasonable maximum) risk estimate [11].

The ranges of risk spanned by the distributions in Fig. 6 are considerable. The 'Fixed Potency' distribution, which considers only variabilities and uncertainties in exposure, spans more than two orders of magnitude over the 90% confidence interval.

[31] The exposure profile designed to approximate reasonable maximum exposure, as described in recent guidance [11].

[32] A distribution for the cancer potency slope factor for 2,3,7,8-TCDD is used, with uncertainties associated with dose-response data and interspecies extrapolation are incorporated. This uncertainty includes a 50% probability that low doses of 2,3,7,8-TCDD do not cause cancer in humans, which is implied within the standard US EPA method for calculating a confidence limit on the slope of the dose-response curve.

The 95th percentile risk value, a frequently cited regulatory level, is 2.3×10^{-5}, which is slightly more than twice the point risk estimate of 9.5×10^{-6}. The 50th percentile risk, which represents best estimate for a reasonable maximally exposed individual, is 2.5×10^{-6}, which is about four times smaller than the point risk estimate.[33] Including the cancer potency in the uncertainty analysis shifts the risk curve to the left (i.e., smaller risks at any given percentile) and flattens the distribution over an even greater range of risks 'Dose-Response Variability'. The 95th percentile risk of 6×10^{-6} is roughly two-thirds the point risk estimate, and is about four times smaller than the similar percentile value generated with a fixed cancer potency. This difference emphasizes the bias to overestimate risks introduced by the use of conservative dose-response factors. Clearly, attempts to generate central tendency estimates of risk (as opposed to exposure) must also account for uncertainties in toxicity data.

4. Conclusions

Multi-pathway risk assessments of combustion sources such as MWCs continue to evolve. Already complex algorithms, such as those described in Section 2, are becoming more intricate in detail and encompassing in scope. The use of deterministic methods to characterize multiple risk descriptors could place new and inappropriate demands on risk assessment. The danger of mischaracterizing high-end, central tendency, and other exposure levels can only be alleviated by the development of full probabilistic analyses. The Monte Carlo assessment discussed in Section 3.5 suggests that the results of deterministic risk assessments should be interpreted with caution, and never construed as absolute measures of risk. The span of the risk distribution demonstrates that, within reasonable confidence limits, point risk estimates may be accurate to within an order of magnitude. From a different perspective, deterministic risk estimates (in and of themselves) may be unreliable for standard setting, since minor, justifiable modifications to parameter values can vary values by more than a factor of ten. Point risk estimates may still be useful in a qualitative sense to rank the relative merits of similar facilities. Probabilistic methods, however, must be encouraged as the logical evolution of risk assessment, and should be accompanied by the development of risk management methods that can utilize the richness of information provided by Monte Carlo assessments and other techniques.

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[33] The analysis for this curve was performed for a location that was at the high end of the distribution, and for an individual (farmer) who would be most exposed. Such an individual/location combination corresponds to an RME individual, as defined by the EPA.

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