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Evaluating the health impacts of incinerator emissions

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Abstract

Before a municipal solid waste incinerator (MSWI) can be operated, it is generally required that a health risk assessment be performed and that human health risks predicted fall below permissible levels. There are several components to the risk assessment paradigm, including: (1) determination of stack emissions for potentially toxic chemicals, (2) calculation of atmospheric dispersion and exposure point concentrations, (3) development of scenarios by which humans become exposed to airborne chemicals, (4) identification of dose-response functions for carcinogenic and noncarcinogenic effects, and (5) prediction of the probability of health impacts. Typical MSWI air contaminants of concern are metals (e.g., Ag, As, Be, Cd, Cr, Hg, Ni, Pb, Sb) and organic compounds (e.g., benzene, PCBs, B(a)P, polychlorinated dioxins/furans). MSWI risk assessments include both direct exposure pathways (air inhalation, incidental ingestion of soil), and indirect pathways (food-chain exposures such as human consumption of produce, beef, fish, and milk). To perform a risk assessment for direct and indirect routes of exposure, both atmospheric concentration and deposition rate are required; assumptions need to be made about toxicity as a function of route of exposure. Interpretation of risk-assessment results requires understanding how some of the conservative assumptions made in the risk-assessment process play out relative to real-world health hazards. Some attempts have been made to verify that predicted concentrations of airborne contaminants are reflected by measured levels, but in most cases the predicted air and soil concentrations fall below limits of detection and always within background variability. In summary, health risk assessments are useful for regulatory guidance, but it has not been possible to verify that health risks of MSWI emissions contribute measurably to population health risks.

Keywords: Solid waste processing; Combustion emissions; Air emissions; Multipathway risk assessment; Biomarkers of exposure; Resource recovery; PCDDs; Metals

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1. The concept of 'risk'

Currently, the majority of municipal solid waste is disposed of in sanitary landfills. However, because of increasing regulations and cost of using landfills, there has been more interest in alternatives to solid waste disposal. Municipal solid waste incinerators (MSWI) are becoming a reasonable means by which communities can dispose of their solid trash. Approval to operate a MSWI generally requires that a 'quantitative health risk assessment' be performed and that predicted numbers for health risks fall below permissible levels.

The term 'risk' has different meanings in different contexts. For a layperson it embodies the concepts of both severity of outcome and probability of outcome. For example, people do not consider death by asteroid impact very risky, primarily because the likelihood of such an occurrence is perceived to be very small. Similarly, death from a fall in the home is not appreciated as a significant risk because falls do not normally connote a lethal injury, and their severity seems to be within one's control. Death and injury from attack by strangers is widely feared as a high risk because of the apparent frequency of such occurrences as reported by the news media. Risk implies not only some adverse result, but also uncertainty. Risk changes as information becomes more specific – a golfer has greater risk of death by lightning than the population as a whole. Whether perceived as likely or not, the risk from an injury at home or being struck by lightening can be calculated, because these events actually happen. In contrast, assessment of risk attributable to low levels of environmental contaminants is a hypothetical exercise. For many environmental hazards, the very existence of human health risk is uncertain. Risk assessment in the context of a MSWI focuses on the emissions from the combustor stack and calculates the probability of incremental increases in health risks for the nearby population.

The US Environmental Protection Agency (USEPA) has adopted a policy that a lifetime cancer risk of one in ten thousand to one in a million represents an acceptable range of risk for the general population. Because of the cost of implementing control strategies to reduce 'unacceptable' risks, it is desirable to determine if exposures and risks predicted from risk assessment estimates can be verified. However, the 'bright lines' for acceptable risk set by regulatory agencies are at such low levels (e.g., 1 in 10^6) that actual observation may be difficult or impossible.

In the following sections, we discuss the risk assessment process used when seeking approval for the building of a MSWI. This procedure is used by the USEPA and other regulatory agencies in their evaluation of the potential health risks from such a facility. If the calculated risks are less than some regulatory guideline, the agencies may conclude that the building of a MSWI will not pose unacceptable health hazards to the community. However, the risk assessment procedure is based both on specific data inputs and numerous assumptions that are of limited accuracy. Interpretation of risk assessments requires understanding how assumptions made in the process will play out relative to real-world health hazards. In addition to discussing the risk assessment process, we show that, with modern-day MSWIs, it is not possible to verify whether or not MSWI emissions contribute measurable health risks to potentially exposed populations.

2. Sequential steps in performing a risk assessment

In 1983, the National Research Council of the National Academy of Sciences (NAS) published a book entitled *Risk Assessment in the Federal Government: Managing the Process* [1]. Here, risk assessment was defined as the 'characterization of the potential adverse health effects of human exposure to environmental hazards', along with 'characterization of the uncertainties inherent in the process of inferring risk'. The NAS report classified the risk assessment process into four broad components – *hazard identification, exposure assessment, dose-response assessment,* and *risk characterization.*

The first component of risk assessment, hazard identification, involves an evaluation of whether a particular chemical can cause an adverse health effect in humans. The hazard identification process in itself is a qualitative risk assessment that examines both the potential for exposure and the nature of the adverse effect expected. The types of information used in hazard identification include human, animal, and mechanistic evidence. In hazard identification, the risk assessor must evaluate the quality of the evidence, the severity of the effects, and whether the mechanisms of toxicity in animals are relevant to humans. The result is a scientific judgment that the chemical or process can, at some concentration, cause a particular adverse health effect in humans. Most often, risk assessors utilize toxicity information developed by the USEPA and cataloged by chemical in the Integrated Risk Information System (IRIS). When this information is lacking, risk assessors can either develop toxicity information themselves from data in the scientific literature, or evaluate the chemical qualitatively.

In exposure assessment, the second component of the risk assessment process, a determination is made of the amount of a chemical to which humans are exposed. Actual data are frequently very limited for exposure assessment, and considerable reliance is placed on modeling exposure-point concentrations. Measures of chemicals in environmental media, such as in stack emissions may be available; however, the extrapolation of those levels to a dose received by humans has many uncertainties. Models exist that can describe the movement of chemicals through a particular medium and assumptions can be made regarding inhalation, ingestion, or dermal contact rates and the bioavailability of the chemical. This information can then be used to derive an estimate of the dose taken up by humans.

Some experts argue that it is unnecessary to first determine if a chemical is hazardous. Their philosophy stems from the first definition of toxicology, given by Paracelsus (1493–1541) over 450 years ago: "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy". In other words, all chemicals have the potential to be hazardous, depending on the dose; therefore, exposure assessment could potentially supersede any consideration of hazard identification.

Dose-response evaluation, the third component of the risk assessment process, involves the characterization of the relationship between the dose administered or received and the incidence or severity of an adverse health effect in the exposed population. Characterizing the dose-response relationship involves understanding the

importance of the intensity of exposure, of the concentration versus time relationship, whether a chemical has a threshold level, and the shape of the dose-response curve. The metabolism of a chemical at different doses, its persistence over time, and an estimate of the similarities in disposition of a chemical between humans and animals also are important aspects of a dose-response evaluation. Again, the quantitative toxicity factors that describe dose and response are most often obtained from IRIS. However, the validity of IRIS values is by no means universally recognized, and toxicity information in IRIS does not have regulatory status.

The last stage of the risk assessment process, risk characterization, involves a prediction of the probability and severity of health impacts in the exposed population. That is, the information from the dose-response evaluation (what dose is necessary to cause the effect?) is combined with the information from the exposure assessment (what dose is the population receiving?) to produce an estimate of the likelihood of observing the effect in the population being studied. Most risk assessments performed in the regulatory arena produce a single-number estimate of risk (e.g., lung cancer risk of 1 in a million). These estimates are generally designed to represent the risk to the maximally exposed individual (MEI), or the risk due to the reasonable maximal exposure (RME) in the potentially exposed population.

Substantial *variability* exists within any potentially exposed population in exposure rates, intake and uptake rates, and sensitivity to the effect. This variability is such that the risk to the most highly exposed and sensitive portion of the population may be much higher than the risks to the majority of the population. Information should be provided on both the risk to individuals and the aggregate risk of the exposed population. Point estimates of risk to a single individual in the population can be misleading when no information is provided to indicate whether that individual represents, say, $\sim 50\%$ or less than 0.1% of the potentially exposed population.

In addition to population variability, there is also significant *uncertainty* present in risk estimates due to the assumptions that need to be made in many of the risk assessment components. For example, the dose-response evaluation is generally highly uncertain. This is often due to the unknown factors inherent to extrapolating effects from animals to humans or from short-term to lifetime exposures. Information may not be available to characterize the active chemical species, the mechanism of effect, the effective dose, or absorption, metabolism, and excretion rates. For example, in a case-study of tetrachloroethylene in groundwater, it was determined that 65% of the variance in the risks resulted from uncertainty in the estimate of the chemical potency [2].

A risk assessment needs to address both sources of overall uncertainty, that is, both variability of exposure among different individuals and uncertainty in data and in model parameters.

3. Mechanics of the risk assessment

3.1. Chemicals of concern

Polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are the chemical compounds of greatest concern for MSWI's because of

the reported toxicity and carcinogenicity or organo-chlorides in animal studies. USEPA has recently released an extensive analysis of the health effects of dioxins and related compounds [3]. In addition to dioxins and furans, typical MSWI air contaminants include metals, such as arsenic, beryllium, cadmium, chromium, mercury, nickel, and lead, and organic compounds like benzene, polychlorinated biphenyls (PCBs), and benzo[a]pyrene.

3.2. Emissions

In order to assess exposure, the stack emission rates for potentially toxic chemicals must be determined. To obtain accurate data, it is important to consider whether the emissions for each chemical will be on an acute (infrequent releases of large quantities), intermittent (sporadic releases of varying amounts), or continual basis. The frequency of emissions can affect both quality of the sampling data and the choice of toxicity values, such as whether to examine acute or chronic exposures. Exposure-point modeling is used to calculate the atmospheric dispersion of the chemicals and to locate the point of maximum exposure concentration. Then, stack emission rates for each chemical are combined with the modeling results to derive the exposure-point concentrations of airborne chemicals. In order to perform a risk assessment for direct and indirect routes of exposure, one needs both atmospheric concentration and rate of contaminant deposition on soil and surface waters.

Generally, potential human exposure scenarios are calculated for an individual residing at the off-site point of maximum annual air concentrations, and who also receives maximum indirect exposure via the routes identified in the discussion to follow. Exposures are estimated based on the modeled average air concentrations and the modeled soil and surface water deposition rates. The location of maximum deposition may be different from the location of maximum ground level concentration. However, it is often assumed that the high-risk individual is also exposed via indirect pathways derived from maximum deposition at the appropriate locations (e.g., farmland or lakes). Fugitive emissions (e.g., windblown emissions from ash piles) and emissions from upset conditions at the incinerator should also be considered when calculating emissions.

3.3. Pathways

Risk assessment requires identification of the pathways via which people will be exposed to the potential chemicals of concern. All feasible direct pathways (direct contact with contaminated media, e.g., inhalation of polluted air, ingestion of impacted soil) and indirect pathways (contact with media to which contaminants have been transferred, e.g., eating contaminated fish) must be examined. Current USEPA guidance for incinerators identifies 16 specific compounds as the ones that need to be evaluated for indirect-pathway exposure [4].

3.3.1. Direct versus indirect exposure

Human exposure to chemicals present in MSWI emissions can occur directly, by inhalation of ambient air containing emissions, or indirectly, through consumption of food (plants and animals) produced near the facility. Typical exposure pathways are shown in Fig. 1. Location of food production relative to the combustion source is an important factor in human exposure to target chemicals through the food chain. Some of an individual's diet may come from food sources within the vicinity (i.e., gardens, farmland, cattle), whereas other foods may be imported from distant areas. In the context of MSWI exposure assessment, only receptors within approximately 30 miles of the source need to be considered [3]. MSWIs are likely to be in urban or suburban areas where dairies and large-scale farming are not typically located. Even for MSWIs in rural areas, detection of an impact on crops, milk, or meat requires that MSWI-attributable concentrations be above background concentrations. Backyard gardens likely contribute only a small fraction of ingested food, whereas the majority of the ingested food available is likely to be derived from distant sources. Lakes where fishing is common represent another food source that could be impacted by emissions, but again, may contribute only a small fraction of the food supply. USEPA guidance generally requires, however, that a 'subsistence fisherman' and a 'subsistence farmer' be considered in the risk assessment. The following are examples of the most common exposure pathways:

Direct:

(1) inhalation of airborne gases and particles*, (if the following media are contaminated only by deposition of airborne emissions, they could be considered 'indirect' pathways rather than 'direct')
(2) incidental ingestion of contaminated soil*,
(3) ingestion of contaminated drinking water,
(4) incidental ingestion of contaminated surface water during recreation,
(5) contact with contaminated sediment,
(6) dermal absorption during recreational activities on contaminated soil or in contaminated surface waters;

Indirect:

(1) ingestion of locally grown vegetables (both above-ground and root vegetables)*,
(2) ingestion of locally produced dairy products (primarily milk)*,
(3) ingestion of meat products from locally raised farm animals (primarily beef)*,
(4) ingestion of fish caught in contaminated lakes and streams (both finfish and shellfish)*,
(5) infant ingestion of mother's milk contaminated by mother's exposure to incinerator emissions (often not quantified due to uncertainly regarding relevant parameters).

The above pathways may be evaluated following USEPA guidelines for assessing health risks associated with indirect exposure to combustion emissions [5]; the pathways indicated by an asterisk (*) are recommended for evaluation in current USEPA guidance [4]. The plausibility of each of the exposure pathways depends both on the

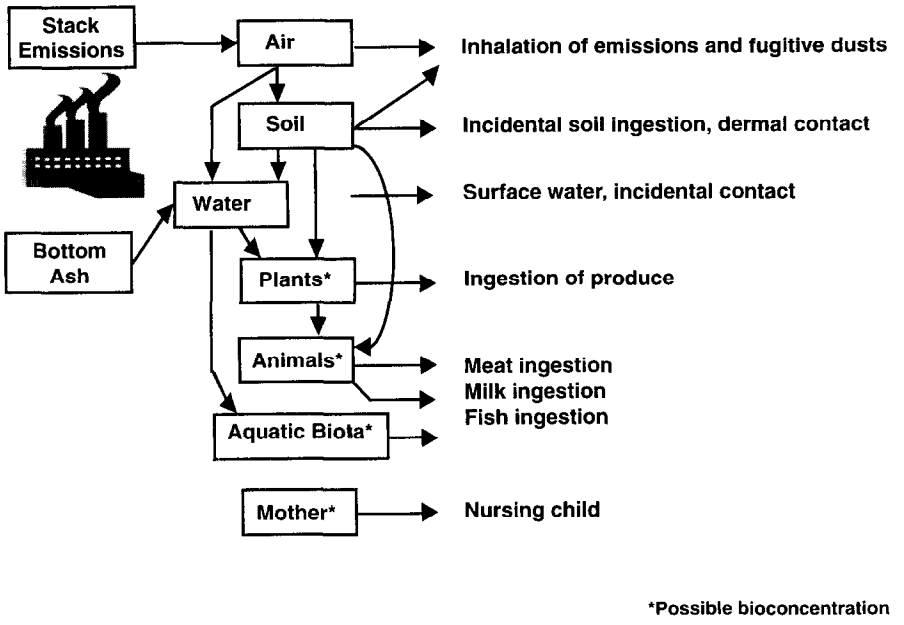

Fig. 1. Conceptual model of exposure pathways between the MSWI and potential human receptors.

proximity of receptors and on land use. Some chemicals, such as dioxins and furans, are known to bioaccumulate, so the equilibrium concentrations of dioxins in dairy products, meat, and fish needs to allow for a 30-yr operation of the MSWI. Thirty years is a frequently chosen exposure period in USEPA risk assessments based on an upper limit for duration of residency at one location. The procedures for calculating bioaccumulation have been outlined by the USEPA [6, 7].

The pathways in a risk assessment are site specific and require examination of both the geography of the land and the human habitations surrounding the site. For example, the presence of lakes, rivers, or ponds must be considered as areas where people could eat contaminated fish, or be dermally exposed to pollutants while swimming or wading. If there are residences downwind of the site, people could be exposed by atmospheric deposition on vegetables in their gardens, dermal contact with soil, or the incidental ingestion of soil. Fig. 1 is a diagram depicting the possible pathways for incinerator emissions. A complete investigation of pathways is important in order to determine that all possible exposure routes have been identified and to ensure that all necessary site samples have been collected.

Food chain exposure in the vicinity of a MSWI is often estimated to be the primary source of human exposure to a large class of organic airborne MSWI emissions, including PCDDs and PCDFs [8]. For MSWI emissions, two separate estimates of what percentage of intake each exposure pathway contributes are shown in Table 1. Exposure via the food chain is responsible for the greatest percentage of the exposure. PCDDs/PCDFs are ubiquitous and are naturally occurring, and their presence in the food chain can reflect sources other than MSWI emissions. In a

Table 1
Estimate of the average daily intake of PCDD/PCDFs from exposure to a typical municipal solid waste incinerator

Exposure pathway	Percent of daily intake	
	Source: [17]	Source: [Nessel]
Inhalation	45.6	30.0
Total ingestion	54.2	48.1
Soil and dust	4.6	0.7
Garden produce	5.3	14.4
Drinking water	—	0.2
Fish	26	6.2
Milk and beef	18.2	26.8
Mother's milk	—	21.5
Dermal absorption	0.2	0.4
Total intake	100	100

multi-pathway analysis of dioxin and furan intake it has been shown that PCDDs/PCDFs associated with MSWI emissions account for only 0.7% of the total daily dioxin intake and that background levels of PCDDs/PCDFs account for more than 99% of the total daily intake of a maximally exposed individual living near a typical MSWI [9].

3.4. Exposure scenarios

After the potential pathways between the MSWI source and human receptors have been identified, exposure scenarios can be developed for the populations of interest. In a residential area, child and adult residents would be considered, whereas for an industrial site, the exposure of workers would be considered, along with potential exposure to site trespassers. If a recreational area is downwind from the source, exposure to pollutants during recreation must be considered. Defining the characteristics of the vulnerable populations is critical for determining the appropriate exposure parameters to use when calculating the administered dose. The administered dose is calculated using the following formula:

$$[\text{Average daily dose}] = [\text{Chemical concentration}] \times \left[\frac{(\text{Intake rate}) \times (\text{Exposure frequency}) \times (\text{Exposure duration})}{(\text{Averaging time}) \times (\text{Body weight})}\right]$$

Examples of parameter values that change with scenario are the chemical concentration (air, groundwater, soil, etc.), intake rate (work rate versus normal rate), exposure frequency (365 d for residents versus 18 d for a trespasser), exposure duration (24 h/d for a resident versus 4 h/d for a recreational activity) and body weight (child versus adult). The development of scenarios by which humans become exposed

to airborne chemicals is critical to being able to quantify the dose affected populations receive. Appropriate exposure parameters are obtained from USEPA guidance (such as the *Risk Assessment Guidance for Superfund* or the *Exposure Factors Handbook*) or from recent literature. Use of both high-end and central-tendency values for exposure parameters are recommended by the USEPA.

Using different exposure scenarios can significantly affect the relative contribution of different exposure pathways, and thus, ultimately influence the predicted excess cancer risk. Nessel and coworkers [10] modeled three different populations and found an approximately 30-fold difference in lifetime cancer risk for PCDD/PCDF between the 'common case' and 'worse case' exposure groups.

3.5. Dose-response evaluation

3.5.1. Uncertainty in dose response

The next component of a risk assessment uses the calculated dose to predict the probability of adverse health effects. The application of toxicological data to assess risk from dose according to the current paradigm is based on making several assumptions:

(1) Toxicity is dependent on lifetime average daily dose, and not on dose rate or on dose distribution over a lifetime.
(2) Expressing dose on a per-kg-body-weight basis (i.e., mg of chemical intake per kg body weight per day) correctly expresses the susceptibility of individuals with differing body weights.
(3) Toxicity for low-level exposures can be accurately extrapolated from results seen for high-level exposures in animal experiments or in occupational settings.
(4) The USEPA's weight-of-evidence classification correctly identifies substances as carcinogenic or not carcinogenic in humans.
(5) Cancer risk depends linearly on dose all the way to zero dose and assumes there exists no threshold dose below which incremental lifetime cancer risk is zero.
(6) Risk due to simultaneous exposure to several substances is equal to the sum of each acting independently.
(7) Toxicity depends only on the dose of the substance in question, with no dependence on co-factors, metabolic status, gender, genetic predisposition, or age at first or last exposure.

Although some of these assumptions are clearly flawed and are discussed below, extrapolation of toxicity information is necessary for predicting health outcomes for MSWI emissions. Once these assumptions have been made, and if quantitative toxicity data are available, the risk posed by carcinogens and noncarcinogens for creating deleterious health effects can be determined. The USEPA has developed several sets of toxicity values to provide quantitative estimates of chemical toxicity. For carcinogenic effects the USEPA develops cancer slope factors (CSFs) and unit risks (UR), and for noncarcinogenic effects the USEPA develops oral reference doses (RfDs) or inhalation reference concentrations (RfCs). These numbers are cataloged in IRIS. Because carcinogens are considered to have no threshold dose level, cancer risks are assessed differently from noncancer risks.

3.5.2. Carcinogens

For carcinogens, which USEPA identifies by a weight-of-evidence classification of the chemical, the average daily dose and the cancer slope factor are multiplied together to find the lifetime cancer risk posed by the chemical. Cancer slope factors are estimates of carcinogenic potency and are used to relate average daily dose of a substance over a lifetime exposure to the lifetime probability of excess tumors. The USEPA estimates CSFs using mathematical extrapolation models, most commonly the linearized multistage model, and the CSF is presented as the risk per intake (mg/kg-day). When adequate human epidemiology data are available, the central estimates (i.e., the maximum likelihood estimates) of model parameters are used to generate a CSF; when only animal data are available, the CSF is derived from the largest possible linear slope that is consistent with the animal data (within the 95% upper confidence limit).

The lifetime probability of contracting cancer due to exposure to site-related chemicals is calculated as follows:

$$\text{(Lifetime probability of cancer)} = \text{(Cancer potency factor (mg/kg-day)}^{-1}\text{)} \times \text{(Average daily intake (mg/kg-day))}$$

The USEPA states that 10^{-4} (1 in 10 000) to 10^{-6} (1 in 1 000 000) represents a range of permissible predicted lifetime risks for carcinogens. Chemicals for which the risk factor falls below 10^{-6} may be eliminated from further consideration as a chemical of concern. Draft Office of Solid Waste (OSW) guidance recommends being consistent with an incremental target risk level of 10^{-5}, applicable to the sum of cancer risks from all compounds over all pathways (one excess cancer after lifetime exposure of 100 000 people to incinerator emissions) [4]; this value was used for the original regulations covering boilers and industrial furnaces (BIF) (*Federal Register*, 21 February 1991, pp. 7134–7240). However, in the BIF rules, indirect exposure was not assessed, so an argument could be made that a higher level of risk (e.g., 10^{-4}) might be permissible if the sum of risks from all pathways is considered.

The dioxins and furans are examples of a family of contaminants that can lead to significant cancer risks in MSWI risk assessments. Although USEPA's CSF for dioxin is currently under review, the allowable daily intake for a lifetime incremental risk of 10^{-5} is about 10 pg/day for a 70 kg individual who is exposed over 30 yr. As shown earlier, the majority of dioxin intake comes via indirect pathways. This distribution of intake derives from assumptions about bioaccumulation and from the assumption that crop uptake occurs from soil concentrations based on a tilled depth of 20 cm and 30 yr of deposition at the point of maximum deposition. The apportionment of risks by pathway depends on air modeling, which determines how air concentrations at the maximum-impact point (off-site) relate to soil and surface water contamination. Thus, in order for a proposed MSWI to meet regulatory guidelines, emission rates would have to be such that the calculated average daily intake of dioxin in potentially exposed individuals would be less than 10 pg/day for the average-sized adult, from all pathways. Other regulatory agencies, e.g., USFDA, allow average daily lifetime intakes of 910 pg/day [8].

It is common practice to characterize the dioxin and furan emissions in terms of Toxicity Equivalency Factors (TEF) [7]. Stack emissions contain many types of dioxin and furan congeners. Each congener exhibits different toxicity effects, and the approach most commonly used is to express the toxicity of all congeners in terms of the most potent congener, 2,3,7,8-TCDD. The term 'TEF-equivalents', refers to the amount of 2,3,7,8-TCDD that would have the same toxicity as the mixture. Hence, the weight fraction of each congener is adjusted according to the TEF for that congener, where the TEF of 2,3,7,8-TCDD is unity.

3.5.3. Non-carcinogens

The calculated average daily dose also is used to determine the risk presented by noncarcinogenic chemicals. The toxicity value of concern here is an RfD or an RfC, which represent an estimate of the lifetime-daily dose or air concentration that is likely to be without an appreciable risk of deleterious effects, even for sensitive subpopulations. RfDs are reference oral doses and RfCs are reference concentrations in air. Chronic RfDs/RfCs are specifically developed to be protective for long-term exposure to a chemical, whereas subchronic RfDs, where available, are designed for shorter-term exposures. RfDs and RfCs are usually expressed as chronic intake levels. RfDs are presented in mg/kg-day (mg substance per kg body weight), while RfCs are presented in mg/m^3 (mg substance per cubic meter of air). The hazard quotient (HQ) for a noncarcinogen is the estimated daily intake divided by the reference dose for that chemical.

$$HQ_i = (\text{Hazard quotient, chemical } i) = (\text{Average daily intake of } i\ (\text{mg/kg-day})) \div (\text{Reference dose for chemical } i\ (\text{mg/kg-day}))$$

As a screening procedure, the HQs of different chemicals can be added to generate an overall hazard index (HI). If the HI is less than 1, the noncarcinogenic effects are not of concern. If the HI is greater than 1, individual HQs should be examined to determine if any specific chemicals are of potential concern. Also, individual pathways can be summed to find the total HI presented by each exposure pathway. For a more detailed analysis, chemicals acting by separate pathways to produce different outcomes should not have HQs added, but rather, only HQs for chemicals acting via a common pathway should be summed. Recent USEPA guidance suggest that a HQ of 0.25 be used as an acceptable target level rather than 1, to allow for other possible sources of environmental contamination aside from the MSWI in question.

3.6. Uncertainty in modeling

Because the risk assessment paradigm is both complex and multi-factorial, there are many steps during which uncertainty may arise. Uncertainty is not synonymous with variability, which is the natural range of values for a particular parameter. Variability applies to parameters such as incinerator feed rate, individual body weight, individual breathing rate and food consumption rate, for which a known range of particular values exist.

Uncertainty is the error caused by a lack of knowledge regarding model parameters. That is, a 'correct' value likely exists, but we are not certain what that value is. For example, incinerator exhaust temperatures, particle deposition velocities, and fly-ash characteristics will influence the vegetative and soil concentrations of MSWI-derived dioxins in an uncertain way [8]. Farming practices can significantly affect the concentrations of dioxins in milk and meat fats by as much as five-fold, and yet, in most situations, we have no knowledge as to what farming practices are being used [8]. Another source of uncertainty derives from insufficient emissions data. The set of samples may be too small to reflect the appropriate annual fluctuations, variation with upset conditions, or background quantities. A small number of samples also can lend uncertainty to the chemical analysis by failing to accurately characterize the emissions. For example, focus on transient 'turn on' or 'upset' conditions could skew the emissions concentrations to appear much larger than expected under normal operating conditions. Finally, many values used in the models are simply a 'best guess,' and inherent in their selection is recognition of the fact that there will be many situations and values that lie outside the chosen range.

3.7. Toxicity uncertainty

Risk assessments are based on toxicological assumptions whose validity is uncertain. Extrapolating from animal laboratory data to develop human toxicity values requires numerous assumptions. Not only is accuracy lost in transforming values between species, but also exposure of laboratory animals to exaggerated doses is necessary to obtain positive results. The data from studies involving massive doses in animals are extrapolated to represent the effects of low, chronic doses in humans. There are some chemicals that only cause adverse effects when administered at high doses; exposures to levels below such a threshold do not cause adverse health effects. For carcinogens, such as dioxin, that produce tumors based on nongenotoxic mechanisms, a different approach in cancer modeling should be used when extrapolating from high to low doses. The linearized multistage model used by regulatory agencies will overestimate the cancer risk if used with nongenotoxic carcinogens [11].

In transforming doses between species, a conversion factor is generally used that scales toxicity according to dose equivalence per unit body weight or surface area [12]. However, a simple ratio cannot capture the complexities involved in scaling doses. Conversion is complicated by the tremendous life span and metabolic differences among species. The high metabolic rate and shorter lifespan of rodents might cause toxic symptoms that the slower metabolic rate and longer life of humans may not incur, and vice versa. Even among potential human receptors, expressing dose on a per kilogram basis does not take into account the wide variation among humans in metabolic rates and susceptibility, even for individuals of the same body weight.

Another toxicological issue is determination of target-tissue dose. Most chemicals have 'target tissue' effects, that is, they cause damage or tumors in only one or two organs. Target-tissue dosing is the question of how much of a dose reaches the specific tissue it affects. In order to quantify this, the distribution of the chemical in the body and its absorption rate must be determined. However, these factors are chemical

specific, depend on the animal species, and depend on the media via which the chemical is consumed. Using standard bioavailability values in the risk assessment can create uncertainty in risk results.

Estimating the health effects of chemicals is further complicated by the possibility of antagonistic and synergistic interactions between chemicals. In an antagonistic response, the presence of one chemical inhibits the harmful actions of another, thereby reducing the overall adverse effect. Conversely, in a synergistic relationship, one chemical can intensify the deleterious effects of another, thereby increasing the total health risk. Current practice is to assume an additive relationship, which is reasonable given our lack of knowledge.

3.8. Role of variability and Monte Carlo characterization of risk distributions

The parameters quantifying contaminant intake can take on a range of values. When actual data are not available, 'default' values are used when modeling environmental fate, exposure parameters, and toxicity. Default values are chosen to be very conservative and are taken from the upper limits of a range of possible values. USEPA guidance proposes a 'reasonable maximum exposure' (RME) scenario, which incorporates multiple conservative parameter choices to generate a highly conservative estimate of risk [13]. The RME approach sets a large number of intake parameters (such as soil ingestion rate) at their upper (95th percentile) limits. The maximally exposed individual is assumed to be simultaneously exposed to upper-bound concentrations of all contaminants in all media, even though it may be impossible for one individual to be so exposed. Hence, the risks calculated could be unrealistic and not apply to any current or future resident. If this is the only point estimate of risk that is calculated, there is no way assess how representative it is. Using conservative point estimates is a poor way to address the problem of parameter 'variability', and a more enlightening approach is to combine *distributions* of input parameters rather than just point values. If all the input variables have log normal distributions, this can be done analytically. For combining distributions of disparate character, 'Monte Carlo' estimation of health risks can be used.

Monte Carlo analysis is used to produce risk estimates that help account for variability in exposure, and to give a median as well as the upper (and lower) bound of the exposure distribution (i.e., above the 95th percentile). In the Monte Carlo approach, all the intake parameters: chemical concentrations, exposure variables, ingestion rates, etc., are assumed to be distributed around some mean value, with both high values and low values possible, as determined by the natural distribution of that parameter for the site and for the exposed population. A distribution of risk is generated from multiple individual calculations ('point estimates') of risk, where for each point estimate a value is picked from each intake parameter distribution according to its probability of occurrence [14]. This process builds up an entire probability distribution of risk in the exposed population, as shown in Fig. 2. That is, at the end of such a process, one can define the risk experienced by the one person in 20 who is most exposed (the upper 95th percentile of risk). This risk level is likely to be far lower than the risk calculated by compounding a series of RME values. That is, when one

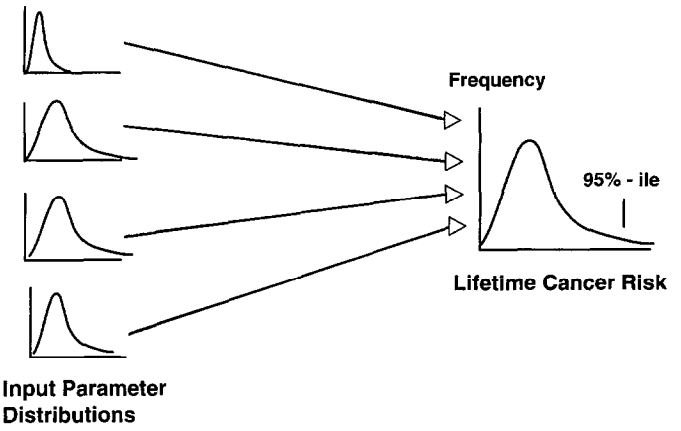

Fig. 2. In Monte Carlo analysis, the probability distributions of the intake parameters are combined to yield a probability distribution for risk. This avoids the problem of giving a single, upper-bound estimate of risk without knowing the fraction of the populace to which it applies.

combines several distributions, the 95th percentile of the resultant distribution is *not* the product of the 95th percentile values of the individual distributions.

As an illustration, consider the point estimate of risk calculated by using high, or upper-end, values for each parameter, for example: chemical concentration, ingestion rate, bioavailability, exposure time, exposure frequency, exposure duration, etc. The probability of any one individual experiencing a maximal value in all of these exposure parameters is very small. If the probability of any one upper-end value occurring is 1 in 20 (e.g., at the upper 95th percentile for that parameter), then if five such values are used to calculate health risk, the probability of that combination of exposure parameters occurring is about $(1/20)^5$, or 3 chances in 10 million. So, whatever risk is calculated by such an RME procedure applies to an extremely tiny portion of the population, and likely to no one at all. Because of the use of extreme values for exposure parameters, the RME health risk is much greater but far less likely than the risk that would be calculated by a Monte Carlo approach for the upper 95th percentile risk in the exposed population.

Monte Carlo results are based on a range of estimated intakes and provide an average expected risk value along with the probability that any given risk value will occur. USEPA has recognized that Monte Carlo simulations can be used to address uncertainties in risk calculations that may arise from insufficient data and from natural variabilities in concentration, uptake, bioavailability, and susceptibility [15]. Disadvantages of the Monte Carlo approach are the increased complexity of the calculations, and the requirement for the additional data describing the distributions of intake parameters.

3.9. Impact of risks estimates

The extensive tier of calculations, considerations, and problems of risk assessment boils down to a simple ratio expressing a person's chance of developing cancer (or

Table 2
Chances per lifetime of dying from selected causes (USA)

Primary determinants of life expectancy:	
Cardiovascular disease	1 in 2
Cancer	1 in 4
Causes that shorten life expectancy:	
Motor vehicle accident	1 in 100
Murder	1 in 300
Fire	1 in 800
Electrocution	1 in 5000
Asteroid/comet impact	1 in 20 000
Passenger aircraft crash	1 in 20 000
Venomous bite or sting	1 in 100 000
Food poisoning by botulism	1 in 3 000 000
Lifetime drinking water containing TCE at EPAs maximum contaminant limit	1 in 10 000 000

Source: [16].

noncancer health effects) from a MSWI site. This individual risk should also be viewed from a population perspective, i.e., what total number of additional cancers are being predicted, given the size of the affected population?

The EPA sets the level of lifetime cancer risk requiring corrective action to be 1 in 100 000. As shown in Table 2 [16], other common risks to life are much greater; even exotic risks such as being killed by an asteroid or comet impact are more likely (1 in 20 000) than the USEPAs regulatory limit. Even these comparisons understate the magnitude of EPA's conservatism. The accidental risks on Table 2 are actuarial, best estimates of risk based on historical data. Risks calculated in a risk assessment are hypothetical, upper limits to risk. The data in Table 2 also make it clear the virtual impossibility of measuring the real-world impact of 'acceptable' levels of risk set by regulators (e.g., 1 in 10 000 to 1 in 1 000 000). Of course, these comparisons do not diminish the importance of investigating and reducing the risk posed by industrial sources. Statistics on the chance of mortality from various causes merely serve as a reminder that for all individuals, all competing causes of mortality must ultimately add up to unity.

On another level, such statistics also call into question the financial practicality of striving to reduce lifetime risks to a one-in-a-million level. To ensure that the building and operation of a MSWI do not introduce an incremental risk greater than an additional cancer in the lifetime of 100 000 persons, protective or corrective measures may be implemented. These measures carry some cost. To put these costs into perspective, the costs per health benefit can be compared for risk avoidance strategies of different hazards by expressing the dollar amount as cost per premature death averted. Table 3 [17] illustrates how the millions of dollars spent in hazardous waste risk reduction actually serve to protect only a few people at enormous expense, on a cost-per-life-saved basis. Although not directly related to incinerator emission standards, the comparisons in Table 3 [17] can be helpful in appreciating

Table 3
Relative costs of selected regulations

Regulation	Year issued	Cost per premature death averted, $ in millions
Car seat belt standards	1984	0.1
Aircraft floor emergency lighting standard	1984	0.6
Children's night clothes flammability ban	1973	0.8
Side-impact standards for buses	1989	2.2
Coke ovens occupational exposure limit	1976	63.5
Arsenic occupational exposure limit	1978	106.9
Asbestos ban	1989	110.7
Hazardous-waste land disposal ban	1988	4190.4
Hazardous-waste listing for wood-preservative chemicals	1990	5 700 000

Source: [17].

how disparate the costs of averted risks can be. The society as a whole needs to make decisions on how to allocate available resources for improving public health.

4. Validation of MSWI risk assessments

One method to determine the actual health benefit of MSWI emission control would be to verify that the concentrations predicted by risk assessments are in fact the levels that reach vulnerable populations. Verification of a particular risk assessment exercise can be done by either (1) measuring an impact on a health outcome, (2) detecting the chemicals of concern or surrogates in biological samples, or (3) measuring the actual levels of specific chemicals in the environmental media. Because of the numerous intervening steps and associated assumptions, verification at the level of media concentrations still allows for considerable uncertainty. Ideally, one would like to verify risk assessment predictions for the outcome of greatest concern, that is, the impact on health. Unfortunately, the impact of MSWI emissions on public health cannot be detected. The predicted cancer risks, even if they are in the 1×10^{-4} range, are impossible to discern in the background individual lifetime risk for dying of cancer, which is about 0.25. Thus, one must rely on biomonitoring or measurements of environmental levels of MSWI contaminants in order to validate a risk assessment.

4.1. Biomonitoring in populations exposed to MSWI emissions

One of the most important steps in the risk-assessment process is the determination of potential human exposure. Typical exposure estimation involves combining predicted concentrations for target chemicals with certain assumptions about environmental fate of these chemicals and activity patterns of the receptors. Subsequently, the results of the exposure assessment are combined with toxicity information to provide a quantitative estimate of risk. Rather than relying on models to estimate

exposure, biological monitoring data may allow actual measurement of exposure and accurate assessment of likely health outcomes. Biological monitoring involves analyzing human biological samples (i.e., blood, urine, or hair) for the presence of target chemicals or marker metabolites. Detection of a target chemical or metabolite of the chemical in a biological sample indicates that exposure has occurred. Biological monitoring has been useful in assessing occupational exposures to airborne chemicals because the workplace typically involves exposure to a single or only a few chemicals at relatively high concentrations (in contrast to typical environmental concentrations), and exposure activity patterns are well known [18]. There are several advantages to biological measurements. They may (1) define environmental exposures more accurately and precisely, (2) identify associated health effects better, and (3) improve the determination of susceptibility to target pollutants. Such data could lead to better characterization of actual human health risks.

Although biomonitoring has the potential to be a useful approach for accurately assessing exposures, there are disadvantages as well. The predictive value of biomonitoring is comprised by the following factors:

(1) Even after bioconcentration, the level of the expected MSWI impact in human receptors may be below quantification limits.
(2) If several chemicals can result in the same biomarker, the presence of this biomarker may not provide useful information about target chemical exposure.
(3) Because biomarkers integrate all routes and sources of exposure, it is not possible to distinguish whether the presence of the biomarker is due to exposure to the chemical in ambient air, water, or food.
(4) Variations in the exposed population, such as health status and individual lifestyle, give rise to differences in biomarker accumulation and decrease the value of a biomonitoring program.
(5) The timing of sample collection in relation to exposure is critical to the successful measurement of a biomarker.

The ideal biomarker is one that is chemical-specific, well measurable in trace quantities, is measurable in easily sampled biological media or by noninvasive techniques (i.e., blood, urine, hair or nails), and is well correlated with a previous exposure. As specifically related to MSWI emissions, a biomarker would ideally be associated with a chemical that is unique to the emissions and could be easily monitored in the stack, and is associated only with inhalation exposure.

The inorganic tracer chemicals for MSWI emissions include antimony, arsenic, beryllium, cadmium, chromium, lead, mercury, nickel, and tin. Organic tracer chemicals include benzo(a)pyrene, polychlorinated biphenyls, and dioxins. Overall, none of these chemicals is ideal for biomonitoring because each one exists naturally in the environment, meaning that exposures may occur naturally via air, water, soil, and food. Although the organic compounds are not naturally occurring, they are inadvertently produced as an impurity in the manufacture of many chemicals or as a byproduct of many combustion processes and are thus, considered to be ubiquitous in the environment. Exposures related to MSWI emissions, therefore, cannot be distinguished from other natural exposures to the same compounds. For instance,

food is the primary source of human exposure to both tin and nickel. The daily intake of nickel in food is estimated to be approximately 108–468 μg/day [19], which is much higher than the daily nickel intake that would be anticipated from any MSWI emissions. Consequently, if biomonitoring were performed for either of these chemicals, their presence in the body would most likely be indicated, but exposure resulting from tin or nickel in MSWI emissions could not be differentiated from the much larger exposures to either chemical in food.

Although a linear relationship between air concentrations of lead and blood lead levels has generally been seen for air concentrations as low as 0.1 $\mu g/m^3$ [20], the maximum predicted air concentration of lead due to MSWI emissions, as provided in Table 4, is three orders of magnitude below this value (0.001 $\mu g/m^3$). Mercury would not be a useful biomarker for MSWI emissions either, because blood and urine measurements are not well correlated to mercury exposures at air concentrations of less than 0.5 mg/m^3 [21] and the maximum predicted mercury concentration in air was 4.2×10^{-7} mg/m^3. Urine measurements of total arsenic are accepted as the most reliable indicator of recent arsenic exposure and have been used in identifying above-average exposures in populations living near industrial point sources of arsenic. Researchers have found a linear relationship between urinary arsenic and inhaled arsenic exposures up to 150 $\mu g/m^3$ as follows: $C_{air}(\mu g/m^3) = 0.3C_{urine}(\mu g/l)$ [22]. This equation can be used to predict that the urine concentration resulting from exposure to the maximum predicted arsenic concentration in air of 5.2×10^{-6} $\mu g/m^3$, would be 1.7×10^{-5} μg/l. This value is well below the analytical detection limit of about 0.1–1 μg/l for urinary arsenic [22].

Although many people believe that incinerators are the major source of human exposure to dioxins, detection of trace amounts of PCDDs/PCDFs in virtually all human adipose (fat) tissue is evidence that dioxins are ubiquitous in the environment and would not be a good marker for MSWI emissions [23, 24]. Other sources of dioxins and furans include numerous combustion processes, both large (e.g., power plants) and small (e.g., fireplaces, home heating, and automobile exhaust); various

Table 4
Maximum predicted air concentrations and minimum detection limits

Chemical	Maximum 1 h average predicted concentration ($\mu g/m^3$)	Maximum annual-average predicted concentration ($\mu g/m^3$)	Minimum detection limits in air ($\mu g/m^3$)
Arsenic	4.8×10^{-5}	5.2×10^{-6}	4.6×10^{-3}–4.7×10^{-3}
Chromium	0.039	0.0042	0.0065–0.0069
Beryllium	1.5×10^{-4}	1.6×10^{-5}	2.2×10^{-4}
Cadmium	1.3×10^{-4}	1.4×10^{-5}	9.0×10^{-4}–1.4×10^{-3}
Lead	0.0090	0.001	0.0061
Nickel	0.043	0.0046	0.0038–0.0077
Mercury	0.0039	0.00042	(Not reported)
PCDDs	1.4×10^{-6}	1.5×10^{-7}	1.0×10^{-6}–5.0×10^{-6}

Source: [28].

industrial processes are sources of dioxins (e.g., copper smelting, steel mills, and herbicide and germicide production and use, and the manufacturing of paper, pulp, and pressure treated wood). Techniques for analyzing dioxins in fat tissue (the location of dioxin accumulation) are not commonly available and other methodologies are not sensitive enough to readily detect dioxins in body fluids [25].

Because modern-day MSWIs are not the only source of chemical contaminants and their contribution to body burdens of toxic chemicals is minor, biomonitoring is not a useful tool to evaluate exposure to MSWI emissions. Supporting this conclusion is a Canadian study of the impact of emissions from the Greater Vancouver municipal waste incinerator on regional soil and vegetation [26]. The monitoring program in Greater Vancouver was designed to look at background conditions, operational conditions for a period of two years following start-up, and long-term conditions. Soil and vegetation samples were collected in 1987, prior to start-up, and were then collected for three consecutive years (1988–1990). The results indicated that the start-up and operation of the facility had no measurable impact on the levels of elements (arsenic, cadmium, nickel, lead, selenium and mercury) and organic compounds (polyaromatic hydrocarbons) on surface soil or vegetation in the vicinity of the facility.

In summary, for modern incinerators, emissions are so low that concentrations of pollutants are unlikely to be measurable above background levels normally present in the environment. If so, it will not be possible to measure concentrations above background either in plants or in human tissue biomarkers. Furthermore, any calculation of risk from incinerator emissions is likely to be lower than a corresponding calculation of risks from background. Finally, in many cases, one can argue that the USEPA risk criterion cannot be met in the natural environment.

4.2. Feasibility of measuring environmental concentrations, a case study

Some attempts have been made to determine if levels of potential MSWI contaminants can be detected in the general background environment. Between 1987 and 1989, USEPA studied a MSWI in Rutland, VT, and sampled air, water, soil, and food for arsenic, beryllium, cadmium, chromium, lead, mercury, nickel, benzo(a)pyrene, polychlorinated biphenyls, and dioxins/furans [27]. The Rutland, VT, MSWI had a 165 ft stack with exhaust velocities of 50 ft/s. It burned 120 tons per day of municipal waste, and stack emission of particulate were 0.03 t/day. Dioxin emissions were 6.1 mg/day. The study did not demonstrate an impact from the MSWI because analytes were present at concentrations below the limits of detection or within background variability.

A subsequent analysis of the USEPA [27] Rutland, VT, MSWI compared maximum predicted concentrations (derived from an air dispersion model) to detection limits and ambient standards [28]. Table 4 shows that even at the point of maximum impact, present detection limits are not capable of identifying an air impact for this particular MSWI. That is, on an annual-average basis, the stack impact of most of the pollutants of interest cannot be measured in air; nickel is the only compound whose predicted stack impact is even marginally above the detection limit.

This finding is similar to the conclusion cited in USEPA [27] for annual-average pollutant concentrations for the Rutland, VT, MSWI.

Even if a monitor were at the location of the maximum 1 h average concentrations, the stack impacts of arsenic, beryllium, and cadmium still could not be measured in air, and the predicted 1 h average incinerator impacts of lead and PCDDs would be only marginally above their detection limits. Nickel and chromium are the MSWI air contaminants that might be measured on a short-term basis. Because the maximum impact point occurs only under certain weather conditions, even short-term monitoring would not be practically feasible.

Vermont Air Pollution Control Regulations contain 'Hazardous Ambient Air Standards', based on health considerations. The maximum annual average predicted concentrations calculated above are compared to these standards in Table 5. As shown in the table, the annual average concentrations of three compounds – chromium, nickel, and PCDDs – were predicted to exceed the Vermont standards at the point of maximum incinerator impact. However, the chromium standard is based on health standards for chromium VI, and the air modeling results are for total chromium. Thus, the percent of chromium VI in the incinerator emissions would need to be measured for an accurate comparison with the Vermont standard. For PCDDs, the predicted annual-average concentration was about 7.5 times higher than the standard, and for nickel, about 1.4 times higher than the value cited by the State of Vermont. Both levels are below technical detection limits.

To examine the feasibility of making soil measurements of pollutants traceable to the incinerator stack, maximum predicted chemical deposition rates attributable to the stack were used to calculate how long it would take, using relatively conservative assumptions, to significantly increase the average background soil concentrations in the region. The time that it would take to double soil concentrations is simply the time necessary to deposit enough pollutants, through emissions from the incinerator stack alone, to equal the existing background soil concentration, assuming no removal of deposited pollutants by rain, wind, or chemical conversion. For these

Table 5
Comparison of predicted maximum concentrations to Vermont standards

Metal/Chemical	Maximum annual-average predicted concentration ($\mu g/m^3$)	Hazard ambient air standards State of Vermont ($\mu g/m^3$)
Arsenic	5.2×10^{-6}	2.3×10^{-4}
Chromium	0.0042	0.000085[a]
Beryllium	1.6×10^{-5}	1.3×10^{-3}
Cadmium	1.4×10^{-5}	5.7×10^{-4}
Lead	0.001	—
Nickel	0.0046	0.0033
Mercury	0.00042	—
PCDDs	1.5×10^{-7} [b]	2×10^{-8} [b]

Source: [28].
[a] Expressed as chromium VI.
[b] Expressed as 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents.

Table 6
Deposition times to double soil concentration

Metal	Time required to double eastern soil concentration (yr)	Time required to double Rutland soil concentration (yr)
Arsenic	61 191	39 898
Beryllium	2275	468
Cadmium	NA	1882
Chromium	534	128
Lead	752	2569
Mercury	12	14
Nickel	168	161

Source: [28].

deposition rate calculations, the point of maximum deposition rate was examined, and a soil mixing depth of 1 in was used. Table 6 summarizes the resulting times corresponding to doubling existing concentrations in average eastern soils by dry deposition from the incinerator stack.

The analysis illustrates an important problem with trying to measure soil concentrations that can be attributed to MSWI emissions. Except for mercury, all chemicals of concern have relatively high background concentrations that require deposition times of over 100 yr to double the typical soil background concentration even at the point of maximum impact (i.e., the incinerator would have to operate continuously for over 100 yr to accumulate a significant impact over background). Mercury is the only compound that might possibly be traceable to stack emissions within the lifetime of the incinerator, and then only if sampling were conducted at the location of the maximum deposition rate. However, as monitoring for mercury has several analytical limitations and as it can be volatilized by microorganisms, it is uncertain that even a mercury impact could be detected in the predicted timeframe.

5. Summary

Risk assessment procedures can be used to relate MSWI stack emissions to the potential for adverse health effects in the surrounding community. Emission controls on MSWIs have become dramatically more stringent over the last 30 yr. Modern-day facilities, under proper operating conditions, emit levels of chemicals that are either undetectable, below ambient levels, or at least below regulatory ambient air standards. With current technical detection limits, risk-assessment related environmental concentrations, human exposure levels, and disease risks cannot be verified. With the possible exception of accidents and highly unusual MSWI operation conditions, one can conclude that modern-day MSWI facilities do not contribute measurably to health risks because the standards imposed by regulatory agencies (1 in 10^5 lifetime cancer risk) are impossible to verify using either biomarkers or population statistics. Although real stack emissions data are used in

MSWI risk assessments, it is unlikely that we will ever be able to verify whether a modern-day facility has a measurable impact on local environmental quality or community health.

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