

The Evaluation of Stack Metal Emissions from Hazardous Waste Incinerators: Assessing Human Exposure through Noninhalation Pathways

Richard M. Sedman¹, James M. Polisini², and John R. Esparza³

¹Oregon Department of Environmental Quality, Waste Management and Cleanup Division, Portland, Oregon; ²California Department of Fish and Game, Sacramento, California; ³California Department of Toxic Substances Control, Sacramento, California

Potential public health effects associated with exposure to metal emissions from hazardous waste incinerators through noninhalation pathways were evaluated. Instead of relying on modeling the movement of toxicants through various environmental media, an approach based on estimating changes from baseline levels of exposure was employed. Changes in soil and water As, Cd, Hg, Pb, Cr, and Be concentrations that result from incinerator emissions were first determined. Estimates of changes in human exposure due to direct contact with shallow soil or the ingestion of surface water were then ascertained. Projected changes in dietary intakes of metals due to incinerator emissions were estimated based on changes from baseline dietary intakes that are monitored in U.S. Food and Drug Administration total diet studies. Changes from baseline intake were deemed to be proportional to the projected changes in soil or surface water metal concentrations. Human exposure to metals emitted from nine hazardous waste incinerators were then evaluated. Metal emissions from certain facilities resulted in tangible human exposure through noninhalation pathways. However, the analysis indicated that the deposition of metals from ambient air would result in substantially greater human exposure through noninhalation pathways than the emissions from most of the facilities. — *Environ Health Perspect* 102(Suppl 2):105–112 (1994).

Key words: risk assessment, exposure, multimedia, incinerator, metals, lead, cadmium, soil, plant uptake

Introduction

The permitting of hazardous waste incinerators in California necessitates the assessment of the risks associated with stack emissions (1). Recent guidelines issued by state and local agencies in California advocate the assessment of all exposure media that could be substantially affected by stack emissions (2–4). In addition to exposure to airborne toxicants, a multimedia assessment of risk can include an evaluation of human exposure to toxicants that could migrate into surface and groundwater, shallow soils, crops, livestock, and aquatic species.

The authors thank Andrew Ranzieri and Richard Miller of the California Air Resources Board for assistance with the air dispersion modeling; Ramzi Mahmood, John Hart, Guido Franco and Dick Erickson for their valuable suggestions, and Jan Radimsky and Watson Gin for their valuable support for this project.

The views expressed here are not the official policy of the Oregon Department of Environmental Quality, the California Department of Toxic Substances Control or the California Department of Fish and Game.

Address correspondence to Dr. Richard M. Sedman, Oregon Department of Environmental Quality, Waste Management and Cleanup Division, 611 SW Sixth Avenue, Portland, Oregon 97204. Telephone (503)229-6773.

Numerous methods are employed to estimate human exposure to toxicants in different media (5–9). These methods rely on models that predict the movement of toxicants in the environment. In a typical assessment, air dispersion modeling is first employed to estimate the concentrations of toxicants in air that result from the stack emissions of an incinerator. The consequent levels of toxicants in soil or surface water due to the deposition of airborne particulates are then determined. The uptake of toxicants from soil or surface waters by various crops, livestock, and aquatic species is estimated. Conventional measures of human exposure to affected media including the quantities of various foods consumed are then employed to estimate potential human exposure to the toxicants.

A previous investigation of the risks associated with stack emissions from hazardous waste incinerators was limited to exposure due to the inhalation of toxicants (10,11). The current study focuses on evaluating the potential health effects associated with As, Cd, Pb, Hg, Cr, and Be emissions from the stacks of hazardous waste incinerators due to human exposure

through noninhalation pathways. The methodology employed in this study is not reliant on models that predict the movement of toxicants in the environment. Nor are estimates of human exposure to toxicants in food based on rates of metal uptake in combination with consumption rates of various affected food commodities. The following approach estimates human exposure to metals based on changes from baseline levels of exposure.

Methods

The result of trial burns at nine hazardous waste incinerators were employed to evaluate potential human health effects by noninhalation routes of exposure (11). The evaluation was conducted for each incinerator by assuming that it was located in a rural agricultural setting (Kern County, CA). In addition, potential effects associated with the levels of metals in ambient air were evaluated.

Estimates of human exposure to metals due to the movement from soil into affected foods are based on estimating the changes from baseline dietary intake measured in U.S. Food and Drug Administration's (FDA) total diet studies

(Table 1). The mean intakes from 1979, 1980, and 1981/1982 studies were employed to establish baseline intakes of arsenic, cadmium, zinc and mercury in adults (12-14). Daily dietary intake of lead by toddlers is from a 1980/1982 study (15).

Changes in human dietary intake of metals that result from incinerator emissions are predicated on: a) the predicted increased concentration of metals in soil above ambient levels found in California's Central Valley (Kern County); and b) the predicted increased concentration of metals in surface waters. Changes in metal concentrations in surface waters or soil due to the deposition of particulates were determined for the 100 m x 100 m area of maximum air concentration established by air dispersion modeling (see Appendix).

The food groups in the FDA study were segregated into commodities that could primarily be obtained from a 100 m x 100 m area. Potatoes, leafy vegetables, legumes, root vegetables, garden fruit and fruit were considered to be in this group of potentially affected food groups for adults (Table 1). Potatoes, vegetables, fruit and fruit juices are considered to be potentially impacted food groups for toddlers (Table 1). Projected increases in human dietary exposure to metals in food due to uptake from soil or surface water are determined as follows:

$$Metal_{Food} = (DI \times \Delta S) + (DF \times \Delta R)$$

Where

$Metal_{Food}$ = Increase in dietary intake of metals from affected foods.

DI = Baseline dietary intake of metal in affected agricultural food groups (FDA studies).

ΔS = Projected increase level of metal in soil above ambient levels in Kern County.

DF = Baseline dietary intake of metal from fish (excluding exposure to fish from fish pond), poultry, and meat (FDA studies).

ΔR = Projected increase of metal concentration in the Kern River above ambient levels.

Projected increases in human exposures to metals in affected drinking water obtained from the Kern River are determined as follows:

$$Metal_{Drinking\ Water} = \Delta River \times Water_{Baseline\ Exposure}$$

Table 1. Affected food groups and their contributions of metals to the total diet..

Metal	Contribution to total diet		Reference dose, $\mu\text{g}/\text{day}$
	Affected foods	Nonaffected foods	
Lead	15.2 ^a	30.4	62 ^c
Cadmium	11.4 ^b	17.8	70 ^d
Mercury	0.2 ^b	4.2	21 ^d
Arsenic	0.3 ^b	46.7	70 ^d

^aToddler diet; potatoes, vegetables, fruit and fruit juices (15). Expressed as daily dietary intake ($\mu\text{g}/\text{day}$). ^bAdult diet: potatoes, leafy vegetables, legumes, root vegetables, garden fruit and fruit (12-14). Expressed as daily dietary intake ($\mu\text{g}/\text{day}$). ^cReference Dose = 10 $\mu\text{g}/\text{dl}$ blood/0.16 (μg Pb/dl blood)/(μg Pb/day) dietary intake. The reference dose is based on 10 $\mu\text{g}/\text{dl}$ of lead in blood in children posing a minimal health risk (16,17) and the slope of 0.16 (μg Pb/dl blood)/(μg Pb/day) from the study of Ryu and coworkers (18). ^dU.S. EPA (19).

Where

$Metal_{Drinking\ Water}$ = Increase in exposure due to the consumption of drinking water.

$Water_{Baseline\ Exposure}$ = Ambient metal levels in river x 2 l/day.

Increased concentrations of metal in soil could result in increased human exposure due to the ingestion of surface soil. Employing a soil ingestion rate of 250 mg/day for a toddler and a lifetime ingestion rate of 70 mg/day (20), changes in human exposure due to soil ingestion are determined as follows:

$$Metal_{Soil} = Soil_{Baseline\ Exposure} \times \Delta Soil$$

Where

$Soil_{Baseline\ Exposure}$ = Ambient metal soil level x Soil ingestion rate

$Aggregate\ Human\ Exposure = Metal_{Food} + Metal_{Water} + Metal_{soil}$

Projected changes in metal concentrations in water of commercial fish pond that was assumed to be located in the area of maximum air concentration were also determined (see Appendix). The effect of incinerator emissions on human exposure due to the consumption of fish raised in an affected fish pond was determined as follows:

$$Metal_{Fish\ (pond)} = DF \times \Delta Pond$$

Where

$Metal_{Fish\ (pond)}$ = Increase in metal intake due to ingestion of fish from a fish pond.

$\Delta Pond$ = Projected increase in metal concentration in pond water above ambient levels in the Kern River.

In addition to the uptake of metals from soil into plants, increased levels of metals in crops can result from the deposition of particulates on foliage. Projected human exposure to metals due to the deposition of particulates on foliage and the subsequent dietary intake of affected foods was ascertained by the method of Moghissi and coworkers (21) employing default assumptions described by California Air Resources Board (21).

Results

The changes in the concentrations of metals in soil resulting from 30 years of deposition of particulates are displayed in Table 2. Stack emissions from four facilities resulted in a material increase (>1%) in projected cadmium or lead soil levels. Soil arsenic levels were materially increased by stack emissions at only one facility (Trade Waste Incinerator). Beryllium and chromium levels in soil remained essentially unchanged for all facilities (data not shown).

The projected increases in metal soil concentrations due to the deposition of metal laden particulates from ambient air were substantial. Lead and cadmium depositions from ambient air were substantially greater than that from stack emissions at seven of the nine facilities. Arsenic deposition from ambient air was substantially greater than that from stack emissions at all facilities where this metal was measured.

An increase in the concentration of a metal in soil would not necessarily be reflected by a proportional increase in human exposure to that metal. For two incinerators, projected aggregate human exposure to lead or cadmium increased by 1% or more above baseline levels of exposure (Table 3). Projected aggregate human exposure to arsenic or mercury were not substantially increased (>1%) from stack emissions of incinerators where these metals were measured.

Table 2. Projected changes in metal concentrations in soil as the result of 30 years of stack emissions.^a

Facility	Cadmium	Lead	Mercury	Arsenic
American Cyanamid	ND ^b	ND	ND	— ^c
Du Pont	3	1	ND	ND
Florida Solite	20	30	—	—
Mobay	1	<1	—	<1
Chevron	<1	<1	<1	<1
Ogden CA	<1	<1	—	—
SCA 1983	3	3	—	—
Rockwell	ND	<1	—	—
Trade Waste Incin.	60	30	ND	1
Ambient air ^d	20	40	^e	3

^aProjected increase (percent) in the metal concentration from existing levels in soil resulting from the deposition of metals from hazardous waste incinerators. ^bND = metal was not detected in stack emissions. ^cNo emissions data for metal. ^dPercent increase above ambient soil levels as a consequence of 30 years of deposition of metals from ambient air. ^eNo monitoring of mercury levels in ambient air.

Table 3. Projected aggregate human exposure resulting from the deposition of metals from hazardous waste incinerators.

Facility	Lead	Cadmium	Mercury	Arsenic
American Cyanamid	ND ^{a,b} (ND) ^c	ND(ND)	ND(ND)	— ^d
Du Pont	<0.3(<1)	0.4(<1)	ND(ND)	ND(ND)
Trade Waste Incin.	6(10)	6(9)	ND(ND)	<0.6(<1)
Florida Solite	6(9)	2(4)	—	—
Mobay	<0.3(<1)	<0.3(<1)	—	<0.6(<1)
Chevron	<0.3(<1)	<0.3(<1)	<0.04(<1)	<0.6(<1)
Ogden CA	<0.3(<1)	<0.3(<1)	—	—
SCA 1983	0.6(<1)	0.3(<1)	—	—
Rockwell	<0.3(<1)	ND(ND)	—	—
Ambient air ^e	8(13)	2(3)	^f	<0.6(<1)

^aProjected increase (µg/day) in aggregate human exposure (soil ingestion + food + water) to metals above baseline exposure levels. ^bND = metal not detected in stack emissions. ^cProjected percent of reference dose contributed by metal emissions from hazardous waste incinerators. Reference doses are displayed in Table 2. ^dNo emissions data for metal. ^ePercent increase in aggregate human exposure as a consequence of 30 years of deposition of metals from ambient air. ^fNo monitoring of mercury levels in ambient air.

The projected aggregate human exposure to lead and cadmium that resulted from the deposition of metals from ambient air was substantial (Table 4). Aggregate human exposure to metals in ambient air were projected to be substantially greater than that from stack emissions from seven of the nine facilities. Aggregate human exposure due to the deposition of arsenic from ambient air although not substantial (<1%), was greater than that resulting from stack emission from all incinerators where arsenic was measured.

An increase in human aggregate exposure to a metal would not necessarily result in a significant impact on human health. The level of exposure may remain well below levels associated with adverse health effects. Therefore, the projected aggregate human exposures resulting from the depo-

sition of metals were compared to the reference doses for the metals.

Projected aggregate human exposures to lead or cadmium did not exceed the benchmark level of 20% of the reference dose for any of the facilities (Table 3). None of the nine facilities contributed a significant portion of the reference dose for mercury or arsenic. The deposition of metals from ambient air also were not projected to contribute a substantial portion of the reference dose for any of the metals.

Projected human exposures to metals due to the consumption of aquatic organisms from an affected stream or fish pond were also evaluated. This analysis employed the highly conservative assumption that all dietary animal protein was obtained from fish affected by incinerator emissions. Projected increased levels of Cd, Hg, As, Pb, Be, or Cr in the Kern River and there-

fore increased human exposures due to the consumption of fish were negligible because voluminous water flow would markedly dilute any metal contributed by the stack emissions (data not shown).

Projected increases in the concentrations of metals in a fish pond located in the area of maximum air concentration and the subsequent increase in human exposure were only notable for the TWI and Florida Solite incinerators (Table 4). The consequent increase in human exposure would be tangible, although it would never exceeded 20% of the reference dose for any of the metals evaluated (Table 4). In contrast to most of the incinerators, deposition of lead and arsenic from ambient air was projected to increase human exposure due to the consumption of fish from a fish pond by 10 to 20% of the reference dose.

Projected human exposures due to foliar deposition of lead exceeded 20% of the reference dose for the TWI, Florida Solite and SCA 1983 facilities (Table 5). Foliar deposition of lead from ambient air is projected to result in humans being exposed to 50% of the reference dose. However, food does not contribute a significant portion of the reference dose of lead according to FDA total diet studies. Given that agricultural products are exposed to particulates deposited from ambient air, the projected exposure of humans to lead in food is not consistent with the dietary intakes measured in the FDA total diet studies.

Discussion

Methodology. While hazardous waste incinerators are designed to efficiently eliminate organic compounds, metals are usually detected in stack emissions. Recent studies that have demonstrated increase levels metal in soil and vegetation adjacent to metal smelters indicate that the emission of metals can have environmental impacts (22–24). Therefore, evaluations of potential human health effects associated with stack emissions generally address all media to which exposure can occur.

Methods that are typically employed for multimedia assessments of human exposure to environmental contaminants rely on models that predict the partitioning of chemicals into various environmental media (5–9). The partitioning of chemicals in various media are typically derived as some function (usually linear) of projected water or soil concentrations.

In this study, estimates of changes in human exposure to metals in food are

Table 4. Human exposure to metals emitted from hazardous waste incinerators due to consumption of fish from a fish pond.

Facility	Lead	Cadmium	Mercury	Arsenic
American Cyanamid	ND ^{a,b}	ND	ND	— ^c
DuPont	<1(<1) ^d	<1(<1)	ND	ND
Trade Waste Incin.	11(10)	<1(<1)	ND	3(5)
Florida Solite	8(8)	1(<1)	—	—
Mobay	<1(<1)	<1(<1)	—	<1(<1)
Chevron	<1(<1)	<1(<1)	<1(<1)	<1(<1)
Ogden CA	<1(<1)	<1(<1)	—	—
SCA 1983	1(1)	<1(<1)	—	—
Rockwell	1(<1)	ND	—	—
Ambient air ^e	10(10)	<1 (<1)	f	14(20)

^aProjected increase above baseline human exposure (µg/day). ^bND = metal not detected in stack emissions. ^cNo emissions data for metal. ^dIncrease in exposure as a percent of reference dose. Reference doses displayed in Table 1. ^eHuman exposure resulting from 1 year of deposition of particulates from ambient air. ^fNo monitoring of mercury levels in ambient air.

Table 5. Projected human exposure to metals resulting from the deposition of particulate emissions on foliage.^a

Facility	Lead	Cadmium	Mercury	Arsenic
American Cyanamid	ND ^b	ND	ND	— ^c
Du Pont	1	<1	ND	ND
Trade Waste Incin.	40	3	ND	1
Florida Solite	30	1	—	—
Mobay	<1	<1	—	<1
Chevron	<1	<1	<1	<1
Ogden CA	<1	<1	—	—
SCA 1983	20	1	—	—
Rockwell	<1	ND	—	—
Ambient ^d air	50	1	e	3

^aResults expressed as percent of reference dose. Reference doses displayed in Table 1. ^bND = metal not detected in stack emissions. ^cNo emissions data for metal. ^dPercent of the reference dose as a consequence of the 90 days of deposition of metals from ambient air. ^eNo monitoring of mercury levels in ambient air.

predicated on changes from baseline levels of dietary intake measured in the FDA total diet studies. In essence, it is assumed that the baseline levels of exposure to metals in food measured in the FDA studies are a consequence of existing ambient levels of metals in soil or surface water. Therefore, changes in soil or surface water concentrations are reflected by proportional increases in human exposures due to the consumption of affected foods.

Unlike previous efforts, the methodology employed in this study does not employ multimedia models with their attendant uncertainties. Information concerning the movement of toxicants between various media is not needed nor are estimates of the intakes of individual foods. Baseline human exposures, empirically derived in the FDA total diet studies, provide the basis for estimating the impact of incinerator emissions.

Potential human dietary intakes of metals were estimated based on projected

changes from baseline exposures determine in FDA total diet studies. Food groups obtained from large tracts of land were judged unlikely to be significantly affected by the emissions of a hazardous waste incinerator, given that an incinerator would impact a localized area. Grain products, oils, fats, and sugar, commodities whose processing and distribution result in the blending of large quantities were judged highly unlikely to significantly contribute to human exposure to metals from incinerator emissions. Livestock and poultry also were not selected as an impacted food group because they must graze over large areas or consume feed grains that are derived from large tracts of land.

A food group was judged to be affected by incinerators stack emissions if most of an individuals dietary intake from the food group could reasonably be obtained from a localized 10,000 m² area. The evaluation is based on all food in the impacted group being obtained from the 10,000 m² area of

highest air concentrations of metals as determine by air dispersion modeling.

Effects on a fish pond were evaluated separately and not included in aggregate human exposure. Individuals that obtained a major portion of their dietary intake from a single agricultural plot would be unlikely to obtain all animal protein from commercially distributed fish species raised in an adjacent fish pond. Since the FDA studies combine metal intake from fish with intakes from poultry and livestock, metal intakes from all these groups was attributed to that obtained from fish from a fish pond.

The most recent total diet studies in which results were reported for individual food groups were employed to establish baseline levels of dietary exposure to metals. Beginning in 1982, results are no longer reported for various food groups. Therefore, the results of the latest total diet study could not be incorporated into this study. The baseline levels of exposure to lead are based on the lead intake of toddlers because this age group appears to be highly sensitive to the adverse effects of lead. Baseline intakes of cadmium, arsenic and mercury are based on adult values, since sensitive toxic effects are associated with long-term low levels of exposure.

The key tenet, that increases in dietary intake of metals are directly proportional to changes in soil or surface water metal levels, is supported by metal uptake studies in plants and animals. Numerous studies have evaluated plant or animal uptake of radioisotopes (25–27), uptake of metals following application of sewage sludge (28–31), and uptake from soils affected by incinerators or from other sources (33–35). Although other factors such as pH and cation exchange capacity, as well as competition from other ions appear to influence plant uptake of metals from soil, the assumption that for metals, plant uptake and therefore human intake is proportional to changes in soil metal concentrations, is supported by most studies. At high concentrations, uptake may be less than proportional to changes in soil concentration.

The bioconcentration of toxicants into animal tissues is addressed by this method. The levels of exposure monitored by the FDA total diet studies are a consequence of any bioconcentration of toxicants in animals. Changes in concentration of a toxicant in soil or surface water are predicted to result in proportional to changes from baseline dietary exposure established in the FDA total diet study.

Little information concerning the forms of the metals emitted from the stacks of hazardous waste incinerators is available. The analytical methodology employed in these studies precludes identifying the forms of the metals (36). However, studies of municipal waste incinerators (37) and coal combustors (38) indicate oxides, chlorides, sulfides, and the elemental forms of metals are emitted.

The methods employed to estimate human exposure by noninhalation pathways essentially ignores the forms of the metals given little is known about the metals. Nevertheless, essentially no movement of deposited metals is anticipated since the soil retention capacity should immobilize the small increase of soil metals that result from stack emissions (39). Deposited metals would be expected to equilibrate with native ions in the soil and behave as native metals. Toxicological criteria employed to evaluate human exposure to native forms of the metals are therefore used to address additional exposures that result from incinerator emissions.

Results. While a variety of metals were monitored in the hazardous waste incinerator stack emissions testing, this study focused on metals with low reference doses or with relatively high stack emissions. The results of this study indicate that in certain circumstances, incinerator emissions could tangibly contribute to aggregate human exposure to specific metals. Projected aggregate human exposures to lead and cadmium were materially increased by two facilities, Trade Waste Incinerator and Florida Solite. Projected human aggregate exposure to mercury, arsenic, beryllium, and chromium were not substantially altered by emissions from any of the facilities.

An increase in soil metal concentration, and therefore increased aggregate human exposure to a metal from affected foods, does not necessarily result in an exposure that would adversely impact human health. To investigate this possibility, the contribution of aggregate human exposure associated with incinerator stack emissions was compared to the reference dose for each metal. Twenty percent of the reference dose was selected as a conservative benchmark of a significant increase of exposure, because it is used to apportion the reference dose of metals in the development of drinking water standards (40). While metal emissions from certain incinerator tangibly contributed to aggregate human exposure, no facility contributed a significant portion of the reference dose.

The projected human exposure to fish from fish obtained from a fish pond did not exceed 20% of the reference dose for any metal. Exposure to fish obtained from the Kern River resulted in minimal human exposure to metals because large volumes of water flowing through the river would markedly dilute metals contributed by a hazardous waste incinerator.

For three incinerators, predicted human exposure resulting from foliar deposition of lead was substantial. However, the contribution of ambient air to human exposure due to foliar deposition was also substantial. This finding is not consistent with the dietary intake of metals measured in the FDA total diet studies. The method employed in this as well as other studies to evaluate exposure due to foliar deposition would appear to overestimate the level of human exposure to metals.

Limitations and Uncertainty. While the methodology employed in this study avoids many of the uncertainties associated with environmental modeling, considerable uncertainty remains in the projections of possible human exposure due to incinerator emissions.

The projected aggregate human exposures rely on estimates on dietary intake of metals measured in the FDA total diet studies. These studies are not designed to precisely monitor the intake of toxicants but rather to track changes in exposure from year to year. In addition, the evaluation is also predicated on the presumption that metals measured in the FDA total diet study result from uptake from soil or surface water. The total diet studies monitor the intakes of metals in prepared foods. A portion of metals detected in food may result from food processing, from packaging or during its preparation. Therefore, the methodology would overpredict the human exposure consequences of increasing the concentrations of metals in soils and surface waters.

The evaluation is based on the assumptions that all dietary intake of foods that could be affected are obtained from a 100 × 100 m plot, the area of maximum air concentration. In addition, all animal protein is assumed to be obtained from fish obtained from a river affected by the incinerator emissions. Given the diversity of the food supply, the analysis provides a highly conservative estimate of exposure. No reduction in the metal concentration in soil due to erosion or due to movement into deeper soils was assumed. One study indicated that the level of metal contamination

in surface soil from the fallout from smelter declined following the closure of the smelter (41).

This study modeled air emission of each incinerator using meteorology monitored at one rural location. A previous study indicated that locating these same facilities in a urban location would result in significantly higher concentrations of metals in air (11). While the findings of this study indicate that the stack emissions from most incinerators would not significant contribute to human exposure to metals through noninhalation pathways, siting the facility elsewhere may result in a different findings.

Therefore, an evaluation of the potential impacts of hazardous waste incinerators on public health should address noninhalation pathways of exposure. The methodology employed in this study should facilitate these assessments.

Appendix

The increase in human exposure due to the consumption of affected foods is derived as a function of the change in soil and water concentrations. Available data is employed to establish the ambient levels of metals in the environment. The impact of stack emissions on ambient levels is then determined.

The means of the levels of metals in soil samples collected in Kern County were taken to be the ambient levels in these areas (Table A1). The means of the levels of arsenic, cadmium, lead and total suspended particulates detected in air monitoring studies conducted by the California Air Resources Board from 1987 through 1988 in Bakersfield (Kern County) were taken as the ambient levels of metals in air (Table 1). Mercury levels in air were not determined in these studies. The ambient levels of lead, mercury, arsenic and cadmium in the Kern River were obtained from the California Department of Water Resources, Water Data Information System (Table A1).

The results of nine trial burns at hazardous waste incinerators where metals were monitored in stack emissions were employed in this study. A summary of the various incineration technologies, air pollution control equipment and waste streams processed at each trial burn is found elsewhere (46). The method employed to model the stack emission has been described elsewhere (47).

Estimating the Change from Existing Soil Metal Concentrations

Estimates of the potential impact of emissions from the stacks of hazardous waste

incinerators on lead, cadmium, mercury and arsenic levels in soil are based on the projected maximum annual average air concentration in a 100 m × 100 m area adjacent to the facility (assuming each facility is located in Kern County) using air dispersion modeling. The estimates are based on thirty years of continuous stack emissions at rates observed during the trial burns. Thirty years is the estimated life of a hazardous waste incinerator. In addition, the evaluation is based on a particulate deposition velocity of 0.02 m/sec (48).

The change in concentrations of metals in soil were determined based on the blending of deposited particulates with existing soils. Blending occurs as the result of erosion and tillage. An erosion rate of 1 ton per acre per year (W Sheldon, personal communication) and an annual tillage to a depth of 15 cm (49) were employed in the determination. No soil was assumed to move into or out of the maximally affected 10,000 m² area.

An iterative procedure was employed to estimate the effect of deposited particulates on the concentration of metals in the top 15 cm of soil. On any given day, a change in metal concentrations occurs in the layer of soil that is eroded (Figure 1). The blending of eroded soil with deposited particulates results in a daily incremental change in concentration as follows:

$$c_{i+1} = ((c_i \times er) + (d \times cp)) / (er + d)$$

Where

i = time (days).

c_{i+1} = concentration of metal in erodible layer on day *i*+1 (µg/kg).

c_i = concentration of metal in erodible layer of soil on day *i* (µg/kg).

d = mass of particulates deposited each day (kg).

cp = concentration of metal in particulates (µg/kg).

er = mass of soil eroded each day (kg).

The mass of metals in the tillable soil layer (the upper 15 cm of soil) changes daily due to the deposition of particulates and the loss of the equivalent mass of soil at the bottom of the 15 cm soil column. The mass of metal within the tillable soil layer on day *i*+1 is therefore equal to:

$$m_{i+1} = m_i - ((c_i \times er) + (d \times c_{old})) + (c_{i+1} \times (d + er))$$

Table A1. Ambient levels of metals in air, soil and water in Kern County, California^a

Metal	Air, ng/m ³	Soil, ppm	Water, ppm
Arsenic	2.6 ^b	4.9 ^c	0.01 ^e
Mercury	—	0.1 ^c	0.0003 ^e
Cadmium	0.5 ^b	0.3 ^d	0.006 ^e
Lead	66 ^b	16 ^c	0.01 ^e

^aValues are the arithmetic means of data from the referenced studies. Consult the individual studies for detection limits and the number of samples in which metals were not detected. ^bFrom California Air Resources Board-Bakersfield Chester Street Station (42). ^cUS Geological Service (43). ^dUS Geological Service (44). ^eKern River (45).

Where

m_i = mass of metal in 1 m² by 15 cm of soil on the *i*th day (µg).

c_{old} = the level of metal in soil following the last tillage event (µg/kg).

Once a year, tillage in top 15 cm soil layer results in the blending of tilled soil. After tillage, the resultant metal concentrations are equal to the mass of metals divided by the mass of soil in a 1 m² × 15 cm volume.

$$C_i = M_i / 216.5 \text{ kg soil}$$

The percent increase of metal concentration above ambient levels is derived as follows:

$$\text{Percent increase} = C_{final} / C_{ambient} \times 100$$

Where

C_{final} = concentration of metal in soil following 30 years of deposition (µg/kg)

C_{ambient} = ambient level of metal in soil prior to deposition (µg/kg)

Estimating Changes in Metal Concentrations in Ambient Waters

Using the approach described by the California Air Resources Board (48), all deposited metals were assumed to be uniformly dissolved in the volume of water equivalent to the annual flow rate (50) reported for the Kern River (37746 ft³/sec, 1987-1988). Potential effects on a commercial fish farm were evaluated for a six foot deep "standard" fish pond

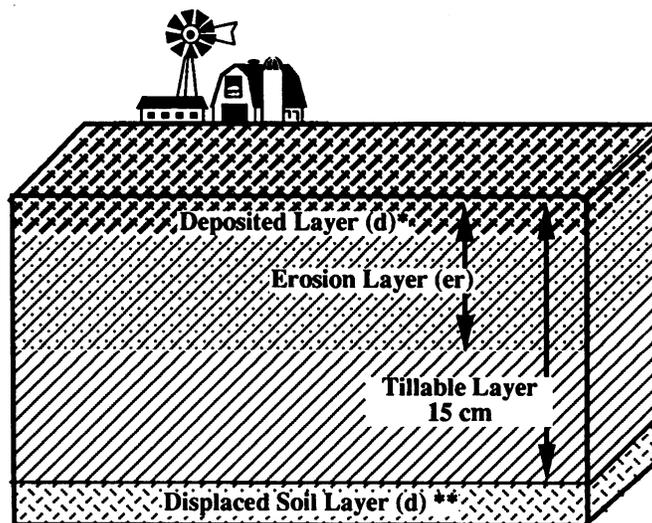


Figure 1. Impact of particulate deposition on soil metal concentrations. The various parameters employed to determine the impact of particulate emissions on the concentration of metals in soil include: (*d*) the layer of soil deposited each day, (*er*) the layer of soil eroded each day, the layer of soil that is tilled annually, and (*d*) the layer of soil that is displaced below the tillable layer each day. Once a year, tillage in the top 15 cm soil layer results in the blending of tilled soil. After tillage, the resultant metal concentrations are equal to the mass of metals divided by the mass of soil in a 1 m² × 15 cm volume.

with a 5% monthly evaporation rate (51). The effect of stack emissions on the levels of metals in the fish pond are determined using the maximum annual average air concentration of each metal projected by the air dispersion model, a deposition rate of 2 cm/sec and the annual replacement of fresh water in the ponds with water from the Kern River.

REFERENCES

1. California Public Resources Code, Section 21000 *et seq.*, 1991.
2. California Air Resources Board. Health Risk Assessment Guidelines for Nonhazardous Waste Incinerators. California Air Resources Board, Sacramento, CA, 1990.
3. South Coast Air Quality Management District. Multi-Pathway Health Assessment Input Parameters Guidance Document. South Coast Air Quality Management District, El Monte, CA, 1988.
4. California Air Pollution Control Officers Association. Air Toxics Manual. California Air Pollution Control Officers Association, Berkeley, CA, 1989.
5. Belcher GD, Travis CC, Bruins RJF. The food chain as a source of human exposure from municipal waste combustion: an uncertainty analysis. DE90-003671, Oak Ridge National Laboratory, Oak Ridge, TN, 1989.
6. Baes CF, Sharp RD, Sjoreen A.L, Shor RW. A review and analysis of parameters for assessing transport of environmentally released radionuclides through agriculture. ORNL-5786. Oak Ridge National Laboratory, Oak Ridge, TN, 1984.
7. Fries GF, Paustenbach DJ. Evaluation of the possible transmission of 2,3,7,8-tetrachlorodibenzo-p-dioxin-contaminated incinerator emissions to humans via foods. *J Toxicol Environ Health* 29:1-43 (1990).
8. U.S. Environmental Protection Agency. Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions. PB90-187055, U.S. EPA, Cincinnati, OH, 1990.
9. Stevens JB, Gerbec EN. Dioxin in the agricultural food chain. *Risk Anal* 8:329-335 (1988).
10. Sedman RM, Esparza JR. The evaluation of organic emissions from hazardous waste incinerators. *Environ Health Perspect* 94:169-180 (1991).
11. Sedman RM, Esparza JR. The evaluation of the public health risks associated with semivolatile, metal and dioxin emissions from hazardous waste incinerators. *Environ Health Perspect* 94:181-187 (1991).
12. Gartrell MJ, Craun JC, Podrebarac DS, Gunderson EL. Pesticides, selected elements, and other chemical in adult total diet samples, October 1980-March 1982. *J Assoc Off Anal Chem* 69:146-161 (1986).
13. Gartrell MJ, Craun JC, Podrebarac DS, Gunderson EL. Pesticides, selected elements, and other chemicals in adult total diet samples, October 1979-September 1979. *J Assoc Off Anal Chem* 68:862-875 (1985).
14. Gartrell MJ, Craun JC, Podrebarac DS, Gunderson EL. Pesticides, selected elements, and other chemicals in adult total diet samples, October 1979-September 1980. *J Assoc Off Anal Chem* 68:1184-1197 (1985).
15. Gartrell MJ, Craun JC, Podrebarac DS, Gunderson EL. Pesticides, selected elements, and other chemical in infant and toddler total diet samples, October 1980-March 1982. *J Assoc Off Anal Chem* 69:123-145 (1986).
16. Centers for Disease Control. Preventing lead poisoning in young children. Center for Disease Control, Atlanta, GA, 1991.
17. Agency for Toxic Substances and Disease Registry. The nature and extent of lead poisoning in children in the United States: a report to Congress. Agency for Toxic Substances and Disease Registry, Atlanta, GA, 1988.
18. Ryu JE, Ziegler EE, Nelson SE, Foman SJ. Dietary intake of lead and blood lead concentration in early infancy. *Am J Dis Child* 137:886-981 (1983).
19. U.S. EPA, Environmental criteria and integrated risk information system (IRIS). U.S. Environmental Protection Agency, Washington, DC, 1992.
20. Sedman RM, Mahmood RJ. Estimating soil ingestion in children and adults based on recent mass balance studies. *J Air Waste Manage Assoc* 44:141-144 (1994).
21. Moghissi AA, Marland RE, Congel FJ, Eckerman KF. Methodology for environmental human exposure and health risk assessment. In: Dynamics, Exposure and Hazard Assessment of Toxic Chemicals (Haque R, ed). Ann Arbor, MI: Ann Arbor Science; 1980, 471-488.
22. Brunekreef B, Veenstra SJ, Biersteker K, Boleij JSM. The Arnhem lead study. *Environ Res* 25:441-448 (1981).
23. Roberts TM, Gizyn W, Hutchinson TC. Lead contamination of air, soil vegetation and people in the vicinity of secondary lead smelters. In: Trace Substances in Environmental Health-VIII (Hemphill DD, ed). Proceedings of University of Missouri's 8th Annual Conference on Trace Substances in Environmental Health, Columbia, Missouri, 1974;155-166.
24. Bache CA, Gutenmann WH, Rutzke M, Chu G, Elfving DC, Lisk DJ. Concentration of metals in grasses in the vicinity of a municipal refuse incinerator. *Arch Environ Contam Toxicol* 20:538-542 (1991).
25. Mosbaek H, Tjell JC, Sevel T. Plant uptake of airborne mercury in background areas. *Chemosphere* 17:1227-1236 (1988).
26. Sheppard MI, Sheppard SC. The plant concentration ratio concept as applied to natural U. *Health Phys* 48:494-500 (1985).
27. Tracy BL, Prantl FA, Quinn JM. Transfer of ²²⁶Ra, ²¹⁰Pb and uranium from soil to garden produce: assessment of risk. *Health Phys* 44:469-477 (1983).
28. Bingham FT. Bioavailability of Cd to food crops in relation to heavy metal content of sludge-amended soil. *Environ Health Perspect* 28:39-43 (1979).
29. Chang AC, Page AL, Warneke JE. Long-term sludge applications on cadmium and zinc accumulation in swiss chard and radish. *J Environ Qual* 16:217-221 (1987).
30. Hinesly TD, Hansen LG, Bray DJ. Use of sewage sludge on agricultural and disturbed lands. EPA-600/2-84-127, U.S. EPA, Cincinnati, OH, (1984).
31. Spósito G, LeClaire JP, LeVesque CS, Senesi N. Methodologies to predict the mobility and availability of hazardous metals in sludge-amended soils. PB84-171099, U.S. EPA, Washington, (1984).
32. Van Wijnen JH, Stijkel A. Health risk assessment of residents living on harbour sludge. *Int Arch Occup Environ Health* 61:77-87 (1988).
33. Bingham FT, Strong JE, Spósito G. Influence of chloride salinity on cadmium uptake by swiss chard. *Soil Sci* 135:160-165 (1983).
34. Preer JR, Sekhon HS, Stephens BR, Collins MS. Factors affecting heavy metal content of garden vegetables. *Environ Pollut* 1:95-104 (1980).
35. Spittler TM, Feder WA. A study of soil contamination and plant lead uptake in Boston urban gardens. *Community Soil Sci Plant Analysis* 10:1195-1210 (1979).
36. Harris J, Larsen D, Rechsteiner C, Thurn K. Sampling and analysis methods for hazardous waste combustion. EPA 600/8-84-002, Arthur D. Little Inc. Prepared for EPA Office of Research and Development, Research Triangle Park, NC, 1984.
37. Cahill CA, Newland LW. Comparative efficiencies of trace metal extraction from municipal incinerator ashes. *Int J Environ Anal Chem* 11:227-239, 1982.
38. Quann RJ, Sarofim AF. Vaporization of refractory oxides during pulverized coal combustion. In: 19th International Symposium on Combustion at the Technion-Israel Institute of Technology, Haifa, Israel, August 8-13, 1982, The Combustion Institute, Pittsburgh, PA, 1982.

39. Sims R, Sorensen D, Sims J, Mclean J, Mahmood R, Dupont R, Jurinak J, Wagner K. Contaminated surface soils in-place treatment techniques. Park Ridge, NJ:Noyes Publications, 1986; 242-244.
40. National primary and secondary drinking water regulations: synthetic organic and inorganic chemicals. Fed Reg 55:30370 (1990).
41. Roberts TM, Goodman GT. The persistence of heavy metals in soils and natural vegetation following closure of a smelter. In: Trace Substances in Environmental Health, Vol VII (Hemphill DD, ed). Proceedings of University of Missouri's 7th Annual Conference on Trace Substances in Environmental Health, Columbia, MO, 1973;117-125.
42. Air quality data, October-November-December, 1983. Sacramento, CA:California Air Resources Board 1988.
43. Boerngen JG, Shacklette HT. Chemical analysis of soil and other surficial materials of the conterminous United States. US Geological Survey, 81-197.
44. Holmgren GGS, Mayer MW, Chaney RL, Daniels RB. Cadmium, lead, zinc and nickel in agricultural soil of the United States. Lincoln, Nebraska:US Soil Conservation Service, 1987.
45. Water data information system. Sacramento, CA:California Department of Water Resources,1991.
46. Sedman RM, Esparza JR. The evaluation of organic emissions from hazardous waste incinerators. Environ Health Perspect 94:169-180 (1991).
47. Sedman RM, Esparza JR. The evaluation of the public health risks associated with semivolatile, metal and dioxin emissions from hazardous waste incinerators. Environ Health Perspect 94:181-187 (1991).
48. Health risk assessment guidelines for nonhazardous waste incinerators. Sacramento, California:California Air Resources Board,1990.
49. Fries GF. Potential polychlorinated biphenyl residues in animal products from application of contaminated sewage sludge to land. J Environ Qual 11:14-20(1981).
50. U.S. Geological Service, Water Resources Data for California, Water Year 1987, Volume 3. Water Data Report CA-87-3, USGS, 1987.
51. Boyd CE. Water quality in warm water fish ponds, Auburn University Agricultural Experimental Station Opelika, AL:Craftmaster Printers, 1979; 359.