III. Health Effects Summaries

This section contains summaries of the key health effects data for the chemicals addressed in the risk assessment of combustion devices that burn hazardous wastes. All of the numbers presented in these summaries are subject to change if EPA obtains new data in the future indicating that the risk is higher or lower than that currently being considered. For more information on health effects, readers can refer to the references listed at the end of each summary.

A. 2,3,7,8-Tetrachlorodibenzo-p-Dioxin

1. Introduction

2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) belongs to the class of compounds, chlorinated dibenzo-p-dioxins, that are referred to as dioxins. 2,3,7,8-TCDD is a colorless solid with no known odor. It does not occur naturally nor is it intentionally manufactured by any industry, although it can be produced inadvertently in small amounts as an impurity during the manufacture of certain herbicides and germicides and has been detected in products of incineration of municipal and industrial wastes. The only current use for 2,3,7,8-TCDD is in chemical research (ATSDR, 1994).

2. Cancer Effects

An increase in lung cancer risks was observed among Japanese males exposed as a result of an oil poisoning accident. Human studies have also found an association between 2,3,7,8-TCDD and soft-tissue sarcomas, lymphomas, and stomach carcinomas, although for malignant lymphomas, the increase in risk is not consistent. The increase in risk is of borderline significance for highly exposed groups and is less among groups exposed to lower levels of 2,3,7,8-TCDD. Although there are problems with the studies of human effects, such as confounding factors, short followup period, and lack of exposure information, the overall weight-of-evidence from epidemiological studies suggests that the generally increased risk of cancer in humans is likely due to 2,3,7,8-TCDD (U.S. EPA, 1994b).

Information on the carcinogenicity of 2,3,7,8-TCDD following inhalation exposure of animals is not available. In animal studies of oral exposure to 2,3,7,8-TCDD, multisite tumors in rats and mice including the tongue, lung, nasal turbinates, liver, and thyroid have been reported (U.S. EPA, 1994b).

Estimates derived from the human data suggest a unit risk for lung cancer of 3 to $5x10^{-4}$ $(pg/kg\text{-}day)^{-1}$. For all cancers combined the unit risk estimate is 2 to $3x10^{-3}(pg/kg\text{-}day)^{-1}$ (U.S. EPA, 1994a). EPA has derived an oral cancer slope factor of 156,000 $(mg/kg/day)^{-1}$ for 2,3,7,8,-TCDD (U.S. EPA, 1994c).

EPA has assigned the dioxin compounds individual toxicity equivalence factors (TEFs). TEFs are estimates of the toxicity of dioxin-like compounds relative to the toxicity of TCDD, which is assigned a TEF of 1.0. Following is the list of TEFs for dioxin compounds (U.S. EPA, 1994b); the concept of toxicity equivalence is based on a unifying mechanism of action within this class of compounds. This mechanism of action has been identified and described by the scientific

community as a series of common biological steps that are necessary for most, if not all, of the observed effects of dioxin and related compounds in vertebrates, including humans.

COMPOUND	TEF
2,3,7,8-TCDD	1
OCDD, 1,2,3,4,5,7,8,9-	0.001
HxCDD, 1,2,3,7,8,9-	0.1
HpCDD, 1,2,3,4,6,7,8-	0.01
OCDF, 1,2,3,4,6,7,8,9-	0.001
HxCDD, 1,2,3,4,7,8-	0.1
PeCDD, 1,2,3,7,8-	0.5
TCDF, 2,3,7,8-	0.1
HpCDF, 1,2,3,4,7,8,9-	0.01
PeCDF, 2,3,4,7,8-	0.5
PeCDF, 1,2,3,7,8-	0.05
HxCDF, 1,2,3,6,7,8-	0.1
HxCDD, 1,2,3,6,7,8-	0.1
HxCDF, 2,3,4,6,7,8-	0.1
HpCDF, 1,2,3,4,6,7,8-	0.01
HxCDF, 1,2,3,4,7,8-	0.1
HxCDF, 1,2,3,7,8,9-	0.1

3. Noncancer Effects

a. Acute (Short-Term)

The acute effects in humans exposed through the spraying in Vietnam of herbicides that contained 2,3,7,8-TCDD include diarrhea, vomiting, skin rashes, fever, and abdominal pain (EPA, 1994b). Routes of exposure in these instances are not well defined and may include inhalation as well as oral and dermal exposures.

No information is available on effects in animals from acute inhalation exposure to 2,3,7,8-TCDD. In oral exposure studies, 2,3,7,8-TCDD is highly toxic to all laboratory animals tested even though there are large differences in species sensitivity. LD_{50} values range from 0.6 µg/kg in male guinea pigs to 5,500 µg/kg in hamsters. Other effects in animals from acute oral exposure include loss of body weight, hepatotoxicity, and decreased thymus weight (ATSDR, 1994). Information on the effects of acute dermal exposure in animals is limited, although dermal effects have been reported (U.S. EPA, 1994b).

b. Chronic (Long-Term)

No studies are available on the inhalation toxicity of 2,3,7,8-TCDD in humans, although such exposure may have occurred in populations exposed to chemicals contaminated with 2,3,7,8-TCDD (ATSDR, 1994). Oral exposure of humans to chemicals contaminated with 2,3,7,8-TCDD has resulted in chloracne, immunotoxicity, hyperpigmentation, hyperkeratosis, possible hepatotoxicity, aching muscles, loss of appetite, weight loss, digestive disorders, headaches, neuropathy, insomnia, sensory changes, and loss of libido (ATSDR, 1994).

Chloracne is the only substantiated effect in humans produced by dermal exposure to compounds contaminated with 2,3,7,8-TCDD (ATSDR, 1994).

No information on chronic inhalation and dermal exposure is available for animals. Oral exposure to 2,3,7,8-TCDD has resulted in dermatitis, extreme loss of body weight, and effects on the liver and immune system (ATSDR, 1994).

EPA has not established a Reference Concentration (RfC) or a Reference Dose (RfD) for 2,3,7,8-TCDD.

c. Reproductive and Developmental

Several studies have investigated the incidence of birth defects and reproductive effects in humans exposed to 2,3,7,8-TCDD through accidental releases or the spraying of 2,3,7,8-TCDD-contaminated herbicides. EPA has concluded that the data were not inconsistent with 2,3,7,8-TCDD adversely affecting development, but as a result of the limitations of the data, these studies could not prove an association with 2,3,7,8-TCDD exposure and the observed effect. The major limitations in these human studies were the concomitant exposure to other potentially toxic chemicals, the lack of any specific quantitative data on the extent of exposure of individuals within the study group, and the lack of statistical power of the studies (ATSDR, 1994).

No studies are available on the reproductive and developmental effects in animals caused by inhalation or dermal exposure to 2,3,7,8-TCDD (ATSDR, 1994). In oral exposure studies, 2,3,7,8-TCDD has produced fetal anomalies, including cleft palate and hydronephrotic kidneys in mice and internal organ hemorrhage in rats, and resulted in spontaneous abortions in monkeys and decreased fetal survival.

4. References for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin

ATSDR (Agency for Toxic Substances and Disease Registry). 1994. *Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo-p-dioxin*. U.S. Public Health Service. U.S. Department of Health and Human Services.

U.S. EPA (Environmental Protection Agency). 1994a. *Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*. Vol II. Draft. Office of Research and Development. Washington, DC.

U.S. EPA (Environmental Protection Agency). 1994b. *Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*. Vol III. Draft. Office of Research and Development. Washington, DC.

U.S. EPA (Environmental Protection Agency). 1994c. *Health Effects Assessment Summary Tables. Annual Update*. OHEA-ECAO-CIN-909. Environmental Criteria and Assessment Office, Office of Research and Development, Cincinnati, OH.

B. Antimony

1. Introduction

Antimony is found at very low levels throughout the environment. Soil usually contains very low concentrations of antimony (less than 1 ppm). However, higher concentrations have been detected at hazardous waste sites and at antimony processing sites. Food contains small amounts of antimony: the average concentration of antimony in meats, vegetables, and seafood is 0.2 to 1.1 ppb. Persons who work in industries that process antimony ore and metal or make antimony oxide may be exposed to antimony by breathing dust or by skin contact (ATSDR, 1992).

2. Noncancer Effects

The primary effects from chronic (long-term) exposure to antimony in humans are respiratory effects that include antimony pneumoconiosis (inflammation of the lungs due to irritation caused by the inhalation of dust), alterations in pulmonary function, chronic bronchitis, chronic emphysema, inactive tuberculosis, pleural adhesions, and irritation. Other effects noted in humans chronically exposed to antimony by inhalation are cardiovascular effects (increased blood pressure, altered EKG readings, and heart muscle damage) and gastrointestinal disorders (ATSDR, 1992).

The RfD for antimony is 0.0004 mg/kg/day, based on a lowest observed adverse effects level (LOAEL) of 0.35 mg/kg/day, an uncertainty factor of 1,000, and a modifying factor of 1. The RfD was based on a study that examined longevity, blood glucose, and cholesterol in rats. EPA has low confidence in the study on which the RfD was based because only one species was used, only one dose level was used, no observed adverse effect level (NOAEL) was determined, and gross pathology and histopathology were not well described; low confidence in the database due to lack of adequate oral exposure investigations; and, consequently, low confidence in the RfD (U.S. EPA, 1995).

EPA has not established an RfC for antimony (U.S. EPA, 1995).

3. References for Antimony

ATSDR (Agency for Toxic Substances and Disease Registry). 1992. *Toxicological Profile for Antimony*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System (IRIS) on Antimony*. Duluth, MN.

C. Arsenic

1. Introduction

Arsenic is a naturally occurring element in the earth's crust that is usually found combined with other elements. Arsenic combined with elements such as oxygen, chlorine, and sulfur is referred to as inorganic arsenic; arsenic combined with carbon and hydrogen is referred to as organic arsenic. In this health effects summary, arsenic refers to inorganic arsenic and its associated compounds. Organic arsenic compounds, such as arsine gas, are not discussed.

2. Cancer Effects

There is clear evidence that chronic exposure to inorganic arsenic in humans increases the risk of cancer. Studies have reported that inhalation of arsenic results in an increased risk of lung cancer. In addition, ingestion of arsenic has been associated with an increased risk of nonmelanoma skin cancer, and bladder, liver, and lung cancer. No information is available on the risk of cancer in humans from dermal exposure to arsenic (U.S. EPA, 1995).

Animal studies have not clearly associated arsenic exposure, via ingestion exposure, with cancer. No studies have investigated the risk of cancer in animals as a result of inhalation or dermal exposure.

EPA has classified inorganic arsenic in Group A - Known Human Carcinogen. For arsenic, the Group A classification was based on the increased incidence in humans of lung cancer through inhalation exposure and the increased risk of skin, bladder, liver, and lung cancer through drinking water exposure (U.S. EPA, 1995).

a. Inhalation Cancer Risk

EPA used the absolute-risk linear extrapolation model to estimate the inhalation unit risk for inorganic arsenic. Five studies on arsenic-exposed copper smelter workers were modeled for excess cancer risk. All five studies showed excess risks of lung cancer that were related to the intensity and duration of exposure and the duration of the latency period. The estimates of unit risk obtained from the five studies were in reasonably good agreement, ranging from 1.25×10^{-3} to 7.6×10^{-3} $(ug/m^3)^{-1}$. Using the geometric mean of these data, EPA calculated an inhalation unit risk estimate of 4.29×10^{-3} $(ug/m^3)^{-1}$ (U.S. EPA, 1995).

EPA has high confidence in the arsenic cancer risk estimate for inhalation exposure, because the studies examined a large number of people, the exposure assessments included air measurements and urinary arsenic measurements, and lung cancer incidence was significantly increased over expected values (U.S. EPA, 1995).

b. Oral Cancer Risk

To estimate the risks posed by ingestion of arsenic, EPA used the data obtained in Taiwan concerning skin cancer incidence, age, and level of exposure via drinking water. In 37 villages that had obtained drinking water for 45 years from artesian wells with various elevated levels of arsenic, 40,421 individuals were examined for hyperpigmentation, keratosis, skin cancer, and blackfoot disease (gangrene of the extremities caused by injury to the peripheral vasculature). The local well waters were analyzed for arsenic, and the age-specific cancer prevalence rates were found to be correlated with both local arsenic concentrations and age (duration of exposure). Based on these data, EPA (1995) proposed an oral cancer slope factor of 1.5 $(mg/kg/day)^{-1}$ with a corresponding unit risk estimate of 5×10^{-5} $(\mu g/L)^{-1}$ from oral exposure to arsenic in drinking water.

The Taiwan cancer data have the following limitations: (1) the water was contaminated with substances such as bacteria and ergot alkaloids in addition to arsenic; (2) total arsenic exposure was uncertain because of intake from the diet and other sources; (3) early deaths from blackfoot disease may have lead to an underestimate of prevalence; and (4) there was uncertainty concerning exposure durations. Due to these limitations, and also because the diet, economic status, and mobility of individuals in Taiwan are different from most U.S. citizens, EPA (1995) has stated "the uncertainties associated with ingested inorganic arsenic are such that estimates could be modified downwards as much as an order of magnitude, relative to risk estimates associated with most other carcinogens."

3. Noncancer Effects

The primary effect noted in humans from chronic exposure to arsenic, through both inhalation and oral exposure, are effects on the skin. The inhalation route has resulted primarily in irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis) while chronic oral exposure has resulted in a pattern of skin changes that include the formation of warts or corns on the palms and soles, along with areas of darkened skin on the face, neck, and back. Other effects noted from chronic oral exposure include peripheral neuropathy, cardiovascular disorders, liver and kidney disorders, and blackfoot disease. No information is available on effects in humans from chronic low-level dermal exposure to arsenic (ATSDR, 1993).

No studies are available on the chronic noncancer effects of arsenic in animals, from inhalation or dermal exposure. Oral animal studies have noted effects on the kidney and liver (ATSDR, 1993).

EPA has established an RfD for inorganic arsenic of 0.0003 mg/kg/day, based on a NOAEL (adjusted to include arsenic exposure from food) of 0.0008 mg/kg/day, an uncertainty factor of 3, and a modifying factor of 1 (U.S. EPA, 1995). This was based on two studies that showed that the prevalence of blackfoot disease increased with both age and dose for individuals exposed to high levels of arsenic in drinking water. This same population also displayed a greater incidence of hyperpigmentation and skin lesions. Other human studies support these findings, with several studies noting an increase in skin lesions from chronic exposure to arsenic through the drinking water. EPA has not established an RfC for inorganic arsenic.

EPA has medium confidence in the studies on which the RfD was based and in the RfD. The key studies were extensive epidemiologic reports that examined effects in a large number of people. However, doses were not well-characterized, other contaminants were present, and potential exposure from food or other sources was not examined. The supporting studies suffer from other limitations, primarily the small populations studied. However, the general database on arsenic does support the findings in the key studies; this was the basis for EPA's "medium confidence" ranking of the RfD (U.S. EPA, 1995).

4. References for Arsenic

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Arsenic (Update)*. U.S. Public Health Service; U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Arsenic*. Duluth, MN.

D. Barium

1. Introduction

Barium is a naturally occurring element that is found in the earth's crust. Barium enters the environment primarily through the weathering of rocks and minerals. The general population is exposed to barium through consumption of drinking water and foods, usually at low levels. Barium and its compounds are used in oil and gas drilling muds, automotive paints, stabilizers for plastics, and jet fuel (ATSDR, 1990).

2. Noncancer Effects

Chronic oral exposure to barium in experimental animals has resulted in increases in blood pressure. Other effects noted from chronic exposure include musculoskeletal effects, such as progressive muscle weakness, and neurological effects including numbness and tingling around the mouth and neck (ATSDR, 1990).

For oral exposure to barium, EPA calculated a reference dose of 0.07 mg/kg/day. This was based on several epidemiological studies that investigated the effects of elevated levels of barium in drinking water. In one study, no increases in systolic or diastolic blood pressure were seen in subjects who consumed drinking water containing barium at levels ranging from 0 to 10 mg/L for 10 weeks. A retrospective epidemiology study compared mortality and morbidity rates in populations ingesting elevated barium levels (2-10 mg/L) in drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Differences in mortality rates from all cardiovascular diseases were significantly higher in the communities with elevated barium. However, these differences were largely in the 65 and over age group and did not account for confounding variables such as population mobility or use of water softeners or medication (U.S. EPA, 1995).

EPA has calculated a provisional reference concentration of 0.0005 $\mu g/m^3$ for barium (U.S. EPA, 1994).

3. References for Barium

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Toxicological Profile for Barium and Compounds*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1994. *Health Effects Assessment Summary Tables. Annual Update.* OHEA-ECAO-CIN-909. Environmental Criteria and Assessment Office, Office of Research and Development, Cincinnati, OH.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Barium*. Duluth, MN

E. Beryllium

1. Introduction

Pure beryllium is a hard gray metal that does not occur naturally, but does occur as a chemical component of certain kinds of rocks, coal and oil, soil, and volcanic dust. Two kinds of mineral rocks, bertrandite and beryl, are mined commercially for the recovery of beryllium. Beryllium is also found combined with other elements such as fluoride, chlorine, sulfur, oxygen, and phosphorus (ATSDR, 1993).

2. Cancer Effects

Several human epidemiological studies have investigated the relationship between beryllium exposure in workers and lung cancer deaths. However, these studies are considered to be inadequate because they did not take a variety of confounding factors, such as smoking, into account (U.S. EPA, 1995).

Beryllium compounds have been shown to cause lung cancer from inhalation exposure in rats and monkeys, while oral exposure to beryllium in animals has not resulted in a statistically significant increased incidence of tumors (U.S. EPA, 1995).

EPA has classified beryllium in Group B2 - Probable Human Carcinogen. For beryllium, this classification was based on animal studies showing an increased risk of lung tumors, and inadequate human evidence (U.S. EPA, 1995).

a. Inhalation Cancer Risk

EPA used the relative risk extrapolation model, based on human data, to estimate the inhalation unit cancer risk for beryllium. EPA calculated an inhalation unit risk estimate of 2.4×10^{-3} $(\mu g/m^3)^{-1}$ (U.S. EPA, 1995).

This cancer risk estimate was based on an epidemiologic study having several confounding factors. The estimates of exposure levels and duration were also uncertain. EPA has also calculated an inhalation unit risk estimate based on several animal studies, which resulted in a similar estimate of risk; however, the quality of the available studies was poor, i.e., they were conducted at a single dose level or lacked control groups (U.S. EPA, 1995).

b. Oral Cancer Risk

EPA used the linearized multistage extrapolation model to estimate the risks posed by ingestion of beryllium. EPA calculated an ingestion slope factor of 4.3 $(mg/kg/day)^{-1}$ and a unit risk estimate (upper 95 percent confidence limit) of 1.2×10^{-4} $(\mu g/L)^{-1}$, based on nonstatistically significant increases in tumors in rats administered beryllium in the drinking water.

This oral cancer risk estimate was derived from a study that did not show a significant increase in tumorigenic response. The study was limited by the use of only one dose group and the occurrence of high mortality and unspecified type and site of the tumors (U.S. EPA, 1995).

3. Noncancer Effects

The major effect from chronic inhalation exposure in humans to beryllium is chronic beryllium disease (berylliosis), in which granulomatous lesions (noncancerous) develop in the lung. The onset of these effects may be delayed by 3 months to more than 20 years. Symptoms of chronic beryllium disease include irritation of the mucous membranes, reduced lung capacity, shortness of breath, fatigue, anorexia, dyspnea, malaise, and weight loss. Chronic beryllium disease may cause death in severe cases. No information is available on the chronic effects of beryllium in humans from oral exposure, and a skin allergy may result from chronic dermal exposure to beryllium (ATSDR, 1993).

Animal studies have also reported effects on the lung, such as chronic pneumonitis, from chronic inhalation exposure to beryllium. Effects on the adrenal gland and immune system are other effects noted in animals chronically exposed by inhalation. No effects were observed in the lung, heart, blood, liver, or kidney from chronic oral exposure to beryllium in animals. Chronic dermal exposure to beryllium in animals has resulted in effects on the immune system (ATSDR, 1993).

EPA has established an RfD for beryllium of 0.005 mg/kg/day, based on a NOAEL (adjusted) of 0.54 mg/kg/day, an uncertainty factor of 100, and a modifying factor of 1 (U.S. EPA, 1995). This was based on a study that reported no adverse effects in rats exposed to beryllium in the drinking water over their lifetime. The same study carried out in mice also reported no adverse effects, and another study in rats indicated a much higher dose level in the diet may be a no-effect-level (U.S. EPA, 1995). EPA has not established an RfC for beryllium.

EPA has low confidence in the RfD and low confidence in the study on which it was based. Only one dose level was used in the study, and EPA considered the study to be of low to medium quality. The low confidence ranking of the RfD reflects the need for more toxicity data by the oral route for beryllium (U.S. EPA, 1995).

4. References for Beryllium

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Beryllium*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Beryllium*. Duluth, MN.

F. Cadmium

1. Introduction

Cadmium is a soft silver-white metal that occurs naturally in the earth's crust and is usually found in combination with other elements such as oxygen, chlorine, or sulfur. The major uses of cadmium are to manufacture pigments and batteries and in the metal-plating and plastics industries. Most of the cadmium used in this country is obtained as a byproduct from the smelting of zinc, lead, or copper ores (ATSDR, 1993).

2. Cancer Effects

Several occupational studies have reported an excess risk of lung cancer from exposure to inhaled cadmium. However, the evidence is limited rather than conclusive due to confounding factors such as the presence of other carcinogens and smoking. Studies of human ingestion to cadmium are inadequate to assess its carcinogenicity (U.S. EPA, 1995).

Animal studies have reported lung cancer resulting from inhalation exposure to several forms of cadmium, while animal ingestion studies have not seen cancer from exposure to cadmium compounds (U.S. EPA, 1995).

EPA has classified cadmium as a Group B1, Probable Human Carcinogen. For cadmium, this classification was based on human studies showing a possible association between cadmium exposure and lung cancer, and animal studies showing an increased incidence of lung cancer (U.S. EPA, 1995).

EPA used the two-stage extrapolation model, based on data from an occupational study of workers exposed to cadmium, to estimate the inhalation unit risk estimate for cadmium. EPA calculated an inhalation unit risk estimate of 1.8×10^{-3} $(\mu g/m^3)^{-1}$ (U.S. EPA, 1995).

EPA used human data to develop the risk estimate for cadmium, since the data were derived from a relatively large cohort and the effects of arsenic and smoking were accounted for in the quantitative analysis of cadmium's effects. EPA also calculated an inhalation unit risk of 9.2×10^{-2} $(\mu g/m^3)^{-1}$ for cadmium based on animal data. This estimate was higher than that derived from human data and thus more conservative; however, EPA felt that available human data were more reliable because of species variations in response and the type of exposure (U.S. EPA, 1995).

3. Noncancer Effects

The kidney appears to be the main target organ in humans following chronic inhalation exposure to cadmium. Abnormal kidney function, indicated by proteinuria and a decrease in glomerular filtration rate, and an increased frequency of kidney stone formation are some of the effects noted. Respiratory effects, such as bronchitis and emphysema have also been noted in humans chronically exposed to cadmium through inhalation. Oral exposure to cadmium in humans also results in effects on the kidney, with effects similar to those seen following inhalation exposure. In humans, dermal exposure to cadmium does not appear to cause allergic reactions (ATSDR, 1993).

Animal studies have reported effects on the kidney, liver, lung, and blood from chronic inhalation exposure to cadmium. Chronic oral exposure to cadmium in animals results in effects on the kidney, bone, immune system, blood, and nervous system. No information is available on chronic dermal exposure to cadmium in animals (ATSDR, 1993).

EPA has established two RfDs for cadmium: one for cadmium ingested in drinking water and one for cadmium ingested in food. The RfD for cadmium in drinking water is 0.0005 mg/kg/day and the RfD for dietary exposure to cadmium is 0.001 mg/kg/day (U.S. EPA, 1995). These RfDs were based on a number of human studies that showed kidney effects (significant proteinuria) from chronic exposure to cadmium. Both RfDs were calculated based on the highest level of cadmium in the human renal cortex (200 μg/g) that was not associated with the critical effect, i.e., significant proteinuria (U.S. EPA, 1995). A toxicokinetic model was then used to determine the highest level of exposure associated with the lack of the critical effect, i.e., the NOAEL. This model allowed for the difference in absorption between drinking water and food to be taken into account. The NOAELs for water and food were calculated to be 0.005 mg/kg/day and 0.01 mg/kg/day, respectively. The RfDs were calculated by applying an uncertainty factor of 10 and a modifying factor of 1 to each NOAEL (U.S. EPA, 1995). EPA has not established an RfC for cadmium.

EPA has high confidence in the studies on which the RfD was based and in the RfD. The RfD was not based on a single study, but rather on data obtained from many studies on the toxicity of cadmium in humans and animals. These data permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism, and elimination. EPA stated that all of this information considered together gives high confidence in the database and in the RfD (U.S. EPA, 1995).

4. References for Cadmium

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Cadmium*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Cadmium*. Duluth, MN.

G. Chromium

1. Introduction

Chromium is a metallic element that occurs in the environment in two major valence states: trivalent chromium (chromium III) and hexavalent chromium (chromium VI). Chromium (VI) compounds are much more toxic than chromium (III) compounds; chromium (III) is an essential element in humans, with a daily intake of 50 to 200 µg/day recommended for an adult, while chromium (VI) is quite toxic. However, the human body can detoxify some amount of chromium (VI) to chromium (III) (ATSDR, 1993).

2. Cancer Effects

Epidemiologic studies of workers have clearly established that inhaled chromium is a human carcinogen, resulting in an increased risk of lung cancer. These studies were not able to differentiate between exposure to chromium (III) and chromium (VI) compounds. No information is available on cancer in humans from oral or dermal exposure to chromium (ATSDR, 1993; U.S. EPA, 1995a).

Animal studies have shown chromium (VI) to cause lung tumors via inhalation exposure. No studies are available that investigated cancer in animals from oral or dermal exposure to chromium (VI). Chromium (III) has been tested in mice and rats by the oral route, with several studies reporting no increase in tumor incidence. No studies are available on cancer in animals from inhalation or dermal exposure to chromium (III) (ATSDR, 1993; U.S. EPA, 1995a).

EPA has classified chromium (VI) in Group A - Known Human Carcinogen. Since the human studies could not differentiate between chromium (III) and chromium (VI) exposure, and only chromium (VI) was found to be carcinogenic in animal studies, EPA concluded that only chromium (VI) should be classified as a human carcinogen (U.S. EPA, 1995a). EPA has classified chromium (III) in Group D - Not Classifiable as to Human Carcinogenicity (U.S. EPA, 1995b).

EPA used the multistage extrapolation model, based on data from an occupational study of chromate production workers, to estimate the unit cancer risk for chromium (VI). EPA calculated an inhalation unit risk estimate of 1.2×10^{-2} $(\mu g/m^3)^{-1}$ (U.S. EPA, 1995a). EPA has not calculated a risk estimate from oral exposure to chromium (VI) or from inhalation or oral exposure to chromium (III).

EPA has confidence in the risk estimate for chromium (VI), based on the fact that the results of studies of chromium exposure are consistent across investigators and countries, and a

dose-response for lung tumors has been established. However, an overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population (U.S. EPA, 1995a).

3. Noncancer Effects

Chronic inhalation exposure to chromium (VI) in humans results in effects on the respiratory tract, with perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, pneumonia, asthma, and nasal itching and soreness reported. Chronic exposure to high levels of chromium (VI) by inhalation or oral exposure may also produce effects on the liver, kidney, gastrointestinal and immune systems, and possibly the blood. Dermal exposure to chromium (VI) may cause contact dermatitis, sensitivity, and ulceration of the skin (ATSDR, 1993).

Limited information is available on the chronic effects of chromium in animals. The available data indicate that, following inhalation exposure, the lung and kidney have the highest tissue levels of chromium. No effects were noted in several oral animal studies with chromium (VI) and chromium (III) (ATSDR, 1993).

EPA has established an RfD for chromium (VI) of 0.005 mg/kg/day, based upon a NOAEL (adjusted) of 2.4 mg/kg/day, an uncertainty factor of 500, and a modifying factor of 1 (U.S. EPA, 1995a). This was based on a study in rats that reported no adverse effects after exposure to chromium (VI) in the drinking water for 1 year. Other studies support these findings; one study reported no significant effects in female dogs given chromium (VI) in the drinking water for 4 years and a case study on humans reported no adverse health effects in a family of four who drank water for 3 years from a private well containing chromium (VI) at 1 mg/L (U.S. EPA, 1995a).

EPA has low confidence in the study on which the RfD for chromium (VI) was based and in the RfD. Confidence in the key study was ranked low due to the small number of animals tested, the small number of parameters measured, and the lack of toxic effects at the highest dose tested. The low ranking of the RfD was due to a lack of high-quality supporting studies and the fact that developmental and reproductive effects are not well studied (U.S. EPA, 1995a).

The RfD for chromium (III) is 1 mg/kg/day, based upon a NOAEL (adjusted) of 1,468 mg/kg/day, an uncertainty factor of 1,000, and a modifying factor of 1 (U.S. EPA, 1995b). This was based on no effects observed in rats fed chromium (III) in the diet for 2 years. EPA has low confidence in the study on which the RfD was based and in the RfD. The low ranking of the key study was due to the lack of explicit detail on study protocol and results, while the low

ranking of the RfD was due to the lack of supporting data and the lack of an observed effects level in the key study (U.S. EPA, 1995b). EPA has not established an RfC for chromium (III) or chromium (VI).

4. References for Chromium

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Chromium*. U.S. Public Health Service; U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995a. *Integrated Risk Information System on Chromium VI*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1995b. *Integrated Risk Information System on Chromium III*. Duluth, MN.

H. Lead

1. Introduction

Lead is a naturally occurring, bluish-gray metal that is found in small quantities in the earth's crust; it is present in a variety of compounds such as lead acetate, lead chloride, lead chromate, lead nitrate, and lead oxide (ATSDR, 1993).

Exposure to lead can occur through the air, drinking water, food, and soil. Most lead exposure occurs through a combination of the inhalation and oral routes, with inhalation generally contributing a greater proportion of the dose for occupationally exposed groups, and the oral route generally contributing a greater proportion for the general population. The effects of lead are the same regardless of the route of exposure (inhalation or oral) and are correlated with internal exposure as blood lead levels. For this reason, the discussion in this summary will not discuss lead exposure in terms of route, but will present it in terms of blood lead levels (ATSDR, 1993).

Children are at particular risk to lead exposure since they commonly put hands, toys, and other items in their mouths that may come in contact with lead-containing dust and dirt. In addition, lead-based paints were commonly used for many years and flaking paint, paint chips, and weathered paint powder may be a major source of lead exposure, particularly for children (ATSDR, 1993).

2. Cancer Effects

Human studies are inconclusive regarding lead and an increased cancer risk. Four major human studies of workers exposed to lead have been carried out; two studies did not find an association between lead exposure and cancer, one study found an increased incidence of respiratory tract and kidney cancers, and the fourth study found excesses for lung and stomach cancers. However, all of these studies are limited in usefulness because the route(s) of exposure and levels of lead to which the workers were exposed were not reported. In addition, exposure to other chemicals probably occurred (U.S. EPA, 1995).

Animal studies have reported kidney cancer in rats and mice exposed to lead via the oral route. No studies are available on cancer in animals exposed to lead via the inhalation or dermal routes (U.S. EPA, 1995).

EPA has classified lead in Group B2 - Probable Human Carcinogen. For lead, this classification was based on animal studies showing an increased risk of kidney tumors, and inadequate human evidence (U.S. EPA, 1995).

EPA has not calculated a unit cancer risk estimate for lead, due to the number of uncertainties that are unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, EPA (1995) has stated that "the current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk."

3. Noncancer Effects

The primary effect in humans from chronic exposure to lead are nervous system effects. Neurological symptoms have been reported in workers with blood lead levels of 40 to 60 μg/dL, and slowed nerve conduction in peripheral nerves in adults occurs at blood lead levels of 30 to 40 μg/dL. Children are particularly sensitive to the neurotoxic effects of lead. There is evidence that blood lead levels of 10 to 30 μg/dL or lower may affect the hearing threshold and growth in children. Chronic exposure to lead in humans can also affect the blood. Anemia has been reported in adults at blood lead levels of 50 to 80 μg/dL, and in children at blood lead levels of 40 to 70 μg/dL. Other effects from chronic lead exposure in humans include effects on blood pressure and kidney function and interference with vitamin D metabolism (ATSDR, 1993).

Animal studies have reported effects similar to those found in humans, with effects on the blood, kidneys, and nervous, immune, and cardiovascular systems noted (ATSDR, 1993).

EPA has not established an RfD or an RfC for lead. EPA (1995) believes that it is inappropriate to develop an RfD for lead, because there is a low degree of uncertainty about the health effects of lead, as compared to most other environmental toxicants. In addition, "it appears that some of these effects, particularly children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold."

The Centers for Disease Control has established a goal of 10 µg/dL as the level below which blood lead levels should be reduced in children (CDC, 1991).

EPA has established an operational level of lead in soil of 400 ppm. This is the level above which it is recomended that further evaluation and appropriate exposure reduction activities be undertaken (U.S. EPA, 1994).

4. References for Lead

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Lead*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

CDC (Centers for Disease Control). 1991. *Preventing Lead Poisoning in Young Children*. U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Lead*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1994. *Memorandum on guidance on residential lead-based paint, lead-contaminated dust, and lead-contaminated soil*. From Lynn R. Goldman, Assistant Administrator for Prevention, Pesticides and Toxic Substances.

I. Nickel

1. Introduction

Nickel is a silvery-white metal that is usually found in nature as a component of silicate, sulfide, or arsenide ores. The most predominant forms of nickel in the atmosphere are nickel sulfate, nickel oxides, and the complex oxides of nickel. Each form of nickel exhibits different physical properties. Most nickel is used to make stainless steel; other uses include the manufacture of batteries, electroplating baths, textile dyes, coins, spark plugs, and machinery parts (ATSDR, 1993).

2. Cancer Effects

Human studies have reported an increased risk of lung and nasal cancers among nickel refinery workers exposed to nickel refinery dust. Nickel refinery dust is defined as the "dust from pyro- metallurgical sulfide nickel matte" refineries and is a mixture of many nickel compounds, including nickel subsulfide. It is not certain which compound is carcinogenic in the nickel refinery dust (U.S. EPA, 1995c). No information is available on the carcinogenic effects of nickel in humans from oral or dermal exposure (ATSDR, 1993; U.S. EPA, 1995c).

Animal studies have reported lung tumors from inhalation exposure to the following nickel compounds and mixtures: nickel refinery dusts, nickel subsulfide, and nickel carbonyl. Oral animal studies have not reported tumors from exposure to nickel acetate in the drinking water. No information is available on the carcinogenic effects of nickel in animals from dermal exposure (ATSDR, 1993; U.S. EPA, 1995a, b, and c).

a. Nickel Refinery Dust

EPA has classified nickel refinery dust in Group A - Known Human Carcinogen. For nickel refinery dust, the Group A classification was based on an increased risk of lung and nasal cancer in humans through inhalation exposure and increased lung tumor incidences in animals (U.S. EPA, 1995c).

EPA used the additive and multiplicative extrapolation method, based on human data, to estimate the unit cancer risk for nickel refinery dust. EPA calculated an inhalation unit risk estimate of 2.4×10^{-4} $(\mu g/m^{3})^{-1}$ (U.S. EPA, 1995c). EPA used four data sets, all from human exposure, to calculate the unit risk estimates for nickel refinery dusts. A range of incremental unit risk estimates were calculated from these data sets that were consistent with each other (U.S. EPA, 1995c).

3. Noncancer Effects

Contact dermatitis is the most common effect in humans from exposure to nickel, via inhalation, oral, and dermal exposure. Cases of nickel contact dermatitis have been reported following occupational and nonoccupational exposure, with symptoms of itching of the fingers, wrists, and forearms. Chronic inhalation exposure to nickel in humans also results in respiratory effects. These effects include direct respiratory effects such as asthma due to primary irritation or an allergic response and an increased risk of chronic respiratory tract infections (ATSDR, 1993).

Animal studies have reported effects on the lungs, kidneys, and immune system from inhalation exposure to nickel, and effects on the respiratory and gastrointestinal systems, heart, blood, liver, kidney, and decreased body weight from oral exposure to nickel. Dermal animal studies have reported effects on the skin (ATSDR, 1993).

EPA has established an RfD for nickel (soluble salts) of 0.02 mg/kg/day, based on a NOAEL (adjusted) of 5 mg/kg/day, an uncertainty factor of 300, and a modifying factor of 1 (U.S. EPA, 1995d). This was based on a study in rats that showed decreased body and organ weights from chronic (2-year) exposure to nickel in the diet. Several other studies showed similar results, with decreased body and organ weights after exposure to nickel chloride via gavage and through the drinking water. EPA has not established an RfC for any nickel compound.

EPA has medium confidence in the RfD for nickel (soluble salts) and low confidence in the study on which it was based. The study on which the RfD was based was properly designed and provided adequate toxicologic endpoints; however, high mortality occurred in the controls. The database provided adequate supporting subchronic studies; this was the basis for EPA's medium confidence level in the RfD (U.S. EPA, 1995d).

4. References for Nickel

ATSDR (Agency for Toxic Substances and Disease Registry). 1993. *Toxicological Profile for Nickel*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995a. *Integrated Risk Information System on Nickel Carbonyl*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1995b. *Integrated Risk Information System on Nickel Subsulfide*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1995c. *Integrated Risk Information System on Nickel Refinery Dust*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1995d. *Integrated Risk Information System on Nickel, Soluble Salts*. Duluth, MN.

J. Selenium

1. Introduction

Selenium is a naturally occurring substance in the earth's crust and is commonly found in sedimentary rock combined with other substances such as sulfide minerals or with silver, copper, lead, and nickel minerals. Selenium is an essential element for humans and animals and exposure occurs daily through food intake. lt is used in the electronics industry; the glass industry; in pigments used in plastics, paints, enamels, inks and rubber; in pharmaceuticals manufacturing; and as a constituent of fungicides (ATSDR, 1994).

2. Noncancer Effects

No information is available on the chronic effects of selenium in humans from inhalation exposure. Ingestion of high levels of selenium in food and water has led to discoloration of the skin, deformation and loss of nails, hair loss, excessive tooth decay and discoloration, lack of mental alertness, and listlessness. Chronic dermal exposure has resulted in skin rashes and contact dermatitis (ATSDR, 1994).

No data are available on the chronic effects in animals from inhalation exposure. Livestock chronically exposed through consumption of high levels of selenium develop "alkali disease." No studies were located on the chronic effects of dermal exposure in animals (ATSDR, 1994).

EPA has established an RfD for selenium of 0.005 mg/kg-day based on an adjusted NOAEL of 0.015 mg/kg/day, an uncertainty factor of 3, and a modifying factor of 1 (U.S. EPA, 1995). The RfD is based on an epidemiologic study that reported clinical selenosis in a population in China.

EPA confidence in the study is medium; confidence in the database and RfD are high. (U.S. EPA, 1995).

EPA has not established an RfC for selenium. (U.S. EPA, 1995).

3. References for Selenium

ATSDR (Agency for Toxic Substances and Disease Registry). *1994. Toxicological Profile for Selenium.* U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency) 1995. *Integrated Risk Information System on Selenium*. Duluth, MN.

K. Silver

1. Introduction

Silver is a naturally occurring element. It is often found deposited as a mineral ore in association with other elements. It is acquired as a byproduct during the retrieval of copper, lead, zinc, and gold ores. It is used in photographic materials, electrical products, silver paints, batteries, sterling ware, and jewelry (ATSDR, 1990).

2. Noncancer Effects

The only clinical condition that is known in humans to be associated with long-term exposure to silver is argyria, a gray or blue-gray discoloring of the skin. Argyria was common around the turn of the centrury when many pharmacologic preparations contained silver. It is much less common today. Today, case reports in humans have reported that repeated dermal contact with silver may in some cases lead to contact dermatitis and a generalized allergic reaction to silver (ATSDR, 1990).

EPA has established an RfD for silver of 0.005 mg/kg/day based on an adjusted LOAEL of 0.014 mg/kg/day, an uncertainty factor of 3, and a modifying factor of 1 (U.S. EPA, 1995). The RfD is based on a report summarizing 70 cases of argyria following use of silver medication.

EPA has medium confidence in the critical study used as the basis for the RfD because it is an old study and describes only patients who developed argyria; no information is presented on patients who received injections of silver and did not deveop argyria. EPA has low confidence in the database because the studies used to support the RfD were not controlled studies, and low-to-medium confidence in the RfD because the RfD is based on a study using intravenous administration and thus necessitated a dose conversion with inherent uncertainties (U.S. EPA, 1995).

EPA has not established an RfC for silver (U.S. EPA, 1995).

3. References for Silver

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Toxicological Profile for Silver*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Silver*. Duluth, MN.

L. Thallium

1. Introduction

Thallium is a metallic element that exists in the environment combined with other elements, such as oxygen, sulfur, and the halogens. Thallium is quite stable in the environment, since it is neither transformed nor biodegraded. It is released to the environment from coal burning and smelting, and its major use is in the semiconductor industry where it is used in the production of switches and closures (ATSDR, 1990).

2. Noncancer Effects

Thallium compounds can affect the respiratory, cardiovascular, and gastrointestinal systems, liver, kidneys, and the male reproductive systems in humans. Temporary hair loss has also been associated with ingestion of thallium in humans. Developmental effects were not noted in children born to mothers who had been exposed to thallium during pregnancy (ATSDR, 1990).

EPA has established an RfD for thallium (thallium sulfate, thallium chloride, and thallium carbonate) of 0.00008 mg/kg/day based on an adjusted NOAEL of 0.25 mg/kg/day, an uncertainty factor of 3,000, and a modifying factor of 1 (U.S. EPA, 1995a, b, and c). The RfD is based on a subchronic toxicity study of thallium sulfate in rats.

EPA has low confidence in the critical study used as the basis for the RfD because of uncertainties in the results and because supporting studies show adverse health effects at doses slightly higher than the NOAEL; low confidence in the database because there is only one subchronic study and some anecdotal human data , and consequently low confidence in the RfD (U.S. EPA, 1995a, b, and c).

EPA has not established an RfC for thallium (U.S. EPA, 1995a, b, and c).

3. References for Thallium

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Toxicological Profile for Thallium*. U.S. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

U.S. EPA (Environmental Protection Agency). 1995a. *Integrated Risk Information System (IRIS) on Thallium Sulfate*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1995b. *Integrated Risk Information System (IRIS) on Thallium Chloride*. Duluth, MN.

U.S. EPA (Environmental Protection Agency). 1995c. *Integrated Risk Information System (IRIS) on Thallium Carbonate*. Duluth, MN.

M. Hydrochloric Acid

1. Introduction

Hydrochloric acid is an aqueous solution of hydrogen chloride gas and is commercially available in several concentrations and purities. Because of impurities, commercial varieties of hydrochloric acid are generally yellow. Hydrochloric acid is used in the refining of metal ore, as a laboratory reagent, and in the removal of scale from boilers (Merck, 1989).

2. Noncancer Effects

In humans, cases of gastritis, chronic bronchitis, dermatitis, and photosensitization have been reported among individuals exposed occupationally to hydrochloric acid (U.S. EPA, 1995). No other data are available specifically on the effects of long-term human exposure via inhalation, ingestion, or dermally.

In animals, the only study of the effects of long-term inhalation of hydrochloric acid reported epithelial or squamous hyperplasia of the nasal mucosa, larynx, and trachea. In a 90-day inhalation study, decreased body weight gains, minimum to mild rhinitis, nasal cavity lesions, and eosinophilic globules in the epithelial lining of the nasal tissues were reported in test animals (U.S. EPA, 1995). No studies are available on the long-term effects on animals from low-level oral or dermal exposures to hydrochloric acid.

EPA has established an RfC for hydrochloric acid of 0.02 mg/m^3 based on a LOAEL (human equivalent concentration) of 6.1 mg/m^3, and an uncertainty factor of 300, (U.S. EPA, 1995). The RfC is based on a chronic rat inhalation study that reported an increased incidence of hyperplasia of the nasal mucosa as well as the laryngeal-tracheal segments in the group exposed to hydrochloric acid. A 90-day inhalation study using mice and rats showed minimum to mild rhinitis in the rats and eosinophilic globules in the epithelial lining of the nasal tissue in mice. EPA has not established an RfD for hydrochloric acid.

EPA has low confidence in the chronic study on which the RfC was based because it used only one dose and limited toxicologic measurements. Confidence in the database is also low because the supporting data consisted of two subchronic bioassays and the database does not provide any additional chronic or reproductive studies. Therefore, EPA's confidence in the RfC for hydrochloric acid is also low (U.S. EPA, 1995).

4. References for Hydrochloric Acid

The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals (11th edition). 1989. S. Budavari (ed). Merck & Co., Inc., Rahway, NJ.

U.S. EPA (Environmental Protection Agency). 1995. *Integrated Risk Information System on Hydrogen Chloride*. Duluth, MN.

IV. Risk Characterization

The objective of the human health risk assessment is to characterize the risks associated with emissions from hazardous waste burning incinerators, cement kilns, and lightweight aggregate kilns. Such a characterization includes both the quantification of risk, using several numerical descriptors, and an assessment of the uncertainties that underlie the risk estimates. The risk descriptors used in this analysis include both central tendency and high-end estimates of individual risk. High-end estimates of individual risk are intended to represent above the 90th percentile of the distribution of individual risk in a specific population of interest but not higher than the individual with the highest risk. Central tendency estimates are intended to represent risks near the 50th percentile of the distribution of individual risk or, in other words, the risk to a more typically exposed individual.

A. Approach to Characterizations

Both central tendency and high-end characterizations of risk were developed. All fate and transport and exposure variables were initially set to central tendency (near 50th percentile) values. For the purpose of estimating high-end individual cancer risk, the exposure duration was varied to high-end values. Resident and child values were obtained from the Exposure Factors Handbook (U.S. EPA, 1990a). The central tendency and high-end exposure durations for residents were also assumed to be valid for fishers. Due to the nature of farming, both typical and subsistence farmers were assumed to reside at the same location for longer periods of time than residents. In addition, for estimating high-end baseline risks associated with both cancer and noncancer effects, contaminated fractions and emissions were also set to high-end values. Exposure parameter values used are presented in Table IV.1. By setting only one or two variables to high end, it is possible to estimate risk in the upper range of the distribution without exceeding the true distribution. For the purpose of determining bounding estimates of oral exposures for a given constituent, in addition to setting one or two exposure parameters to high-end values as for the high-end risk estimates, the location of maximum impact was used rather than the actual location of exposure for the exposure scenario having the highest risk. The bounding estimates represent estimates that, although unlikely, cannot be ruled out given the large number of hazardous waste facilities and the relatively small sample size represented by the example facilities. For evaluating risks from inhalation exposures, an individual was assumed to reside at the location of maximum predicted ambient air concentration. Intake rates were set to mean values for all exposure scenarios evaluated.

All exposure variables and the sources of the values used in this assessment are presented in Appendix E. Individual risk estimates were calculated using standard EPA risk equations. Equations used in the assessment are presented in Appendix C.

Table IV.1 Central Tendency and High-End Exposure Parameter Values

Exposure Parameter		Central Tendency	High End	Source
Exposure Duration				
Child		6 years		U.S. EPA (1990a)
Residents and fishers		9 years	30 years	U.S. EPA (1990a)
Farmers		20 years	40 years	Assumption
Contaminated Fraction				
Subsistence farmers		1.0		Assumption
Typical farmers	Dairy	0.40	0.75	U.S. EPA (1990a) (beef values assumed for pork & poultry)
	Beef, pork or poultry	0.44	0.75	
	Vegetables	0.25	0.40	
Home gardeners		0.25	0.40	U.S. EPA (1990a)

1. Emission Characterizations

Emission rates of the individual constituents (in units of mass per time) were calculated from the stack gas concentration of the chemical, the facility's volumetric flow rate, and the facility's operating hours. To estimate risks resulting from current conditions, baseline emission concentrations were developed from an EPA database (U.S. EPA, 1995f). Two levels of baseline emission concentrations were developed -- central tendency and high end. In addition, emission rates that would result from applying the proposed MACT regulatory alternative levels, and the MACT regulatory alternative levels that are presented for comment, were considered as part of this analysis. Tables IV.2 through IV.4 present the baseline dioxin/furan congener emissions concentrations used in the risk assessment. Table IV.5 provides the TEQ emission concentrations for the baseline and the MACT regulatory alternatives. The same congener distribution was used for the MACT regulatory alternatives as for the baseline. Tables IV.6 through IV.8 provide the baseline and MACT regulatory alternative stack gas concentrations for the metals and hydrogen chloride.

Table IV.2. Baseline Stack Gas Emission Levels of Dioxin and Furan Congeners at 7% Oxygen - Incinerators

CAS No.	Name	TEF	50th %ile Dioxin Congeners		90th %ile Dioxin Congeners	
			Emissions Concentration	2,3,7,8-TCDD TEQ	Emissions Concentration	2,3,7,8-TCDD TEQ
			ng/dscm		ng/dscm	
Incinerators						
1746016	2,3,7,8-TCDD	1	2.39e-02	2.39e-02	2.90e-01	2.90e-01
3268879	OCDD, 1,2,3,4,5,7,8,9-	0.001	4.60e-01	4.60e-04	4.56e+00	4.56e-03
19408743	HxCDD, 1,2,3,7,8,9-	0.1	3.91e-02	3.91e-03	6.15e-01	6.15e-02
35822469	HpCDD, 1,2,3,4,6,7,8-	0.01	1.44e-01	1.44e-03	2.26e+00	2.26e-02
39001020	OCDF, 1,2,3,4,6,7,8,9-	0.001	4.29e-01	4.29e-04	1.25e+01	1.25e-02
39227286	HxCDD, 1,2,3,4,7,8-	0.1	3.35e-02	3.35e-03	3.77e-01	3.77e-02
40321764	PeCDD, 1,2,3,7,8-	0.5	2.58e-02	1.29e-02	2.93e-01	1.47e-01
51207319	TCDF, 2,3,7,8-	0.1	1.29e-01	1.29e-02	5.38e+00	5.38e-01
55673897	HpCDF,1,2,3,4,7,8,9-	0.01	1.02e-01	1.02e-03	2.96e+00	2.96e-02
57117314	PeCDF, 2,3,4,7,8-	0.5	1.12e-01	5.60e-02	4.94e+00	2.47e+00
57117416	PeCDF, 1,2,3,7,8-	0.05	8.76e-02	4.38e-03	3.05e+00	1.53e-01
57117449	HxCDF, 1,2,3,6,7,8-	0.1	1.59e-01	1.59e-02	7.07e+00	7.07e-01
57653857	HxCDD, 1,2,3,6,7,8-	0.1	4.08e-02	4.08e-03	5.43e-01	5.43e-02
60851345	HxCDF, 2,3,4,6,7,8-	0.1	1.42e-01	1.42e-02	6.03e+00	6.03e-01
67562394	HpCDF,1,2,3,4,6,7,8-	0.01	5.04e-01	5.04e-03	2.82e+01	2.82e-01
70648269	HxCDF, 1,2,3,4,7,8-	0.1	2.73e-01	2.73e-02	1.52e+01	1.52e+00
72918219	HxCDF, 1,2,3,7,8,9-	0.1	3.49e-02	3.49e-03	6.03e-01	6.03e+00
			Total TEQ =	1.91e-01	Total TEQ =	6.99e+00

Table IV.3. Baseline Stack Gas Emission Levels of Dioxin and Furan Congeners at 7% Oxygen – Cement Kilns

CAS No.	Name	TEF	50th %ile Dioxin Congeners		90th %ile Dioxin Congeners	
			Emissions Concentration	2,3,7,8-TCDD TEQ	Emissions Concentration	2,3,7,8-TCDD TEQ
			ng/dscm		ng/dscm	
Cement Kilns						
1746016	2,3,7,8-TCDD	1	2.10e-02	2.10e-02	3.95e-01	3.95e-01
3268879	OCDD, 1,2,3,4,5,7,8,9-	0.001	6.37e-01	6.37e-04	5.32e+00	5.32e-03
19408743	HxCDD, 1,2,3,7,8,9-	0.1	1.34e+00	1.34e-01	4.01e+00	4.01e-01
35822469	HpCDD, 1,2,3,4,6,7,8-	0.01	6.35e-01	6.35e-03	1.22e+01	1.22e-01
39001020	OCDF, 1,2,3,4,6,7,8,9-	0.001	5.96e-02	5.96e-05	6.14e-01	6.14e-04
39227286	HxCDD, 1,2,3,4,7,8-	0.1	9.08e-02	9.08e-03	2.26e+00	2.26e-01
40321764	PeCDD, 1,2,3,7,8-	0.5	7.51e-02	3.76e-02	1.69e+00	8.45e-01
51207319	TCDF, 2,3,7,8-	0.1	5.68e-01	5.68e-02	1.18e+01	1.18e+00
55673897	HpCDF,1,2,3,4,7,8,9-	0.01	4.29e-02	4.29e-04	6.33e-01	6.33e-03
57117314	PeCDF, 2,3,4,7,8-	0.5	3.02e-01	1.51e-01	6.97e+00	3.49e+00
57117416	PeCDF, 1,2,3,7,8-	0.05	1.57e-01	7.85e-03	3.22e+00	1.61e-01
57117449	HxCDF, 1,2,3,6,7,8-	0.1	9.69e-02	9.69e-03	1.98e+00	1.98e-01
57653857	HxCDD, 1,2,3,6,7,8-	0.1	1.17e-01	1.17e-02	2.92e+00	2.92e-01
60851345	HxCDF, 2,3,4,6,7,8-	0.1	1.42e-01	1.42e-02	4.42e+00	4.42e-01
67562394	HpCDF,1,2,3,4,6,7,8-	0.01	1.15e-01	1.15e-03	2.10e+00	2.10e-02
70648269	HxCDF, 1,2,3,4,7,8-	0.1	2.05e-01	2.05e-02	4.67e+00	4.67e-01
72918219	HxCDF, 1,2,3,7,8,9-	0.1	2.76e-02	2.76e-03	4.49e-01	4.49e-02
			Total TEQ =	4.85e-01	Total TEQ =	8.29e+00

Table IV.4. Baseline Stack Gas Emission Levels of Dioxin and Furan Congeners at 7% Oxygen – Lightweight Aggregate Kilns

CAS No.	Name	TEF	50th %ile Dioxin Congeners		90th %ile Dioxin Congeners	
			Emissions	2,3,7,8-TCDD TEQ	Emissions	2,3,7,8-TCDD TEQ
			ng/dscm		ng/dscm	
Lightweight Aggregate Kilns						
1746016	2,3,7,8-TCDD	1	4.46e-03	4.46e-03	5.62e-03	5.62e-03
3268879	OCDD, 1,2,3,4,5,7,8,9-	0.001	3.27e-01	3.27e-04	5.00e-01	5.00e-04
19408743	HxCDD, 1,2,3,7,8,9-	0.1	1.44e-02	1.44e-03	2.14e-02	2.14e-03
35822469	HpCDD, 1,2,3,4,6,7,8-	0.01	9.78e-02	9.78e-04	1.21e-01	1.21e-03
39001020	OCDF, 1,2,3,4,6,7,8,9-	0.001	8.61e-02	8.61e-05	1.10e-01	1.10e-04
39227286	HxCDD, 1,2,3,4,7,8-	0.1	6.92e-03	6.92e-04	1.23e-02	1.23e-03
40321764	PeCDD, 1,2,3,7,8-	0.5	8.93e-03	4.47e-03	1.36e-02	6.80e-03
51207319	TCDF, 2,3,7,8-	0.1	2.12e-02	2.12e-03	3.17e-02	3.17e-03
55673897	HpCDF,1,2,3,4,7,8,9-	0.01	1.70e-02	1.70e-04	1.88e-02	1.88e-04
57117314	PeCDF, 2,3,4,7,8-	0.5	2.15e-02	1.08e-02	3.02e-02	1.51e-02
57117416	PeCDF, 1,2,3,7,8-	0.05	2.36e-02	1.18e-03	3.24e-02	1.62e-03
57117449	HxCDF, 1,2,3,6,7,8-	0.1	2.02e-02	2.02e-03	2.47e-02	2.47e-03
57653857	HxCDD, 1,2,3,6,7,8-	0.1	9.47e-03	9.47e-04	1.61e-02	1.61e-03
60851345	HxCDF, 2,3,4,6,7,8-	0.1	3.04e-02	3.04e-03	3.69e-02	3.69e-03
67562394	HpCDF,1,2,3,4,6,7,8-	0.01	6.33e-02	6.33e-04	1.05e-01	1.05e-03
70648269	HxCDF, 1,2,3,4,7,8-	0.1	4.39e-02	4.39e-03	5.46e-02	5.46e-03
72918219	HxCDF, 1,2,3,7,8,9-	0.1	1.03e-02	1.03e-03	1.48e-02	1.48e-03
			Total TEQ =	3.87e-02	Total TEQ =	5.34e-02

Table IV.5. Dioxin TEQ Stack Gas Concentrations (7% O_2) Used in Calculation of Risk

Facility Type	2,3,7,8-TCDD-TEQ Concentration ng/dscm	
	Central Tendency	High End
Baseline		
Incinerators	0.191	6.99
Cement kilns	0.485	8.29
Lightweight aggregate kilns	0.0387	0.0534
Proposed floor - Existing and New Sources		
Incinerators	0.2	4.0
Cement kilns	0.2	1.4
Lightweight aggregate kilns	0.2	
Proposed BTF - (also BTF Options 1,2, & 3) Existing and New Sources		
Incinerators	0.2	
Cement kilns	0.2	
Lightweight aggregate kilns	0.2	
Alternative Floor - (also Alternative BTF) - Existing Sources		
Incinerators	0.12	
Cement kilns	0.14	
Lightweight aggregate kilns	0.14	

Table IV.6. Baseline Stack Gas Concentrations of Metals in µg/dscm and Hydrogen Chloride in ppmv at 7% Oxygen

	Baseline Emission Concentration					
	Incinerator		Cement Kiln		Lightweight Aggregate Kiln	
	50th %ile	90th %ile	50th %ile	90th %ile	50th %ile	90th %ile
Lead	90.7	1800	109	1480	15.4	522.
Nickel	31.5	296	12.3	62.3	38.1	227.
Silver	2.94	27	3.5	27.2	1.31	6.87
Thallium	3.86	33.7	6.07	66.9	1.02	2.91
Antimony	14.5	583	6.5	115	9.45	53.6
Arsenic	5.02	57.9	2.84	18.7	2.58	19.7
Barium	24.5	232	70.3	620	10.4	84.4
Beryllium	0.406	4.56	0.38	2.55	0.435	2.11
Cadmium	10.3	158	12.6	113	7.18	63.1
Chromium (VI)	3.07	54.1	1.62	13.2	1.28	6.64
Chromium (III)	19	117	8.78	32.5	23.52	56.46
Selenium	2.51	18.5	10.6	90.4	0.498	4.29
Hydrogen chloride	5.78	97.8	9.76	99.2	173	3,830

Table IV.7. Regulatory Option Emission Concentrations of Metals in µg/dscm at 7% Oxygen

Facility Type	Semivolatile Metals Cadmium Lead	Low-volatility Metals Antimony Arsenic Beryllium Chromium
Proposed Floor (also Proposed BTF and BTF Options 1, 2, & 3) - Existing Sources		
Incinerators	120	110
Cement kilns	34	67
Lightweight aggregate kilns	7.4	230
Alternative Floor (also Alternative BTF) - Existing Sources		
Incinerators	22	28
Cement kilns	92	19
Lightweight aggregate kilns	29	36
Proposed Floor - New Sources		
Incinerators	120	110
Cement kilns	34	26
Lightweight aggregate kilns	4	36
Proposed BTF - New Sources		
Incinerators	35	35
Cement kilns	35	26
Lightweight aggregate kilns	35	35
CEM Compliance Option - New Sources		
Incinerators	40	80
Cement kilns	40	80
Lightweight aggregate kilns	40	80

Table IV.8. Hydrogen Chloride Emission Concentrations in ppmv at 7% Oxygen

Facility Type	Central Tendency	High End
Baseline		
Incinerators	5.78	97.8
Cement kilns	9.76	99.2
Lightweight aggregate kilns	173	3830
Proposed Floor - (also BTF Option 1, 2, and 3) - Existing Sources		
Incinerators	96	
Cement kilns	270	
Lightweight aggregate kilns	1400	
Proposed BTF - Existing Sources		
Incinerators	93	
Cement kilns	270	
Lightweight aggregate kilns	210	
Proposed Alternative Floor - Existing Sources		
Incinerators	8.6	
Cement kilns	11	
Lightweight aggregate kilns	1300	
Proposed Alternative BTF - Existing Sources		
Incinerators	8.6	
Cement kilns	11	
Lightweight aggregate kilns	25	
Proposed Floor - New Sources		
Incinerators	97	
Cement kilns	270	
Lightweight aggregate kilns	36	
Proposed BTF - New Sources		
Incinerators	25	
Cement kilns	25	
Lightweight aggregate kilns	25	

2. Site Variations

Risks between the different facility types and between the different subpopulation scenarios are not directly comparable because of the variations between the sites. Through the selection of a variety of sites, the many different site-specific fate and transport parameters are varied in a plausible manner. The following list identifies some of the major variations in the exposure methodology that result from the variations between the sites. Section II discusses how these parameters were used. Appendix A contains the site-specific values used in the analysis.

- Variations in emissions are incorporated through the facility operating parameters
- Variations in air pathway fate and transport are incorporated through the use of local meteorologic data
- Variations in exposure locations (and contamination levels) are incorporated through the use of site-specific land uses and waterbodies
- Variations in fraction contaminated are site-specific based on local land production and processing capacity.

B. Results

This section presents an overview of the results. Risk estimate results were generated for each facility, for each exposure pathway, and for the summation of all indirect ingestion pathways. Results were generated for each of the case studies and emission and exposure levels (i.e., central tendency and high end) separately. They are summarized in the tables as the range of the risks from the lowest to the highest over the set of facilities and exposure levels. A range of results is arrived at due to the site variations discussed above. The individual facility results were compiled by facility type into summary tables that present the range of risks by facility type. For inhalation exposures, risk estimates were generated for the maximum exposed individual (MEI). For oral exposures, the summary tables include the risks from all indirect ingestion pathways over all scenarios and for each scenario individually. For inhalation exposures, the summary tables contain the risk to the MEI. These summary tables are presented in Appendix B along with tables that summarize soil lead levels and infant dioxin exposures through the breast milk pathway.

1. Dioxin Results

Dioxin and furan congeners were modeled independently for emissions and fate and transport; however, for calculating risks, dioxin congener concentrations in media are converted to 2,3,7,8-TCDD TEQs. Risks for three levels of exposure were calculated for each emission level -- central tendency, high end, and bounding levels.

Lifetime excess cancer risks for dioxins exceed 10^{-5} for many of the subsistence scenarios for high-end baseline emissions. Only the lightweight aggregate kilns, with low baseline stack emissions of dioxin compounds, show baseline risk estimates below 1 in a million.

Table IV.9 presents the dioxin risks over all the subsistence scenarios for each facility type, for the central tendency and high-end levels of exposure, for both the baseline and the MACT regulatory alternative emissions levels. Table IV.10 presents the risks for the typical farmer scenario. The risks for the typical resident scenario are similar to those of the typical farmer. Table IV.11 provides more detail on the highest baseline risks for each scenario, demonstrating that one single scenario does not always result in the highest risks. As presented in Table IV.12, baseline breast milk exposures for infants do not exceed background exposures.

Inhalation risks for the MEI presented in Table IV.13, are lower than the risks from dioxin ingestion for all emissions and exposure levels. The scenarios that amplify the animal ingestion pathways were those that resulted in the higher risks from dioxins. Because of the bioaccumulation potential of dioxin in tissue for all of the animals modeled, the animal ingestion pathways are responsible for the higher risk estimate. Ingestion of beef, dairy, poultry, and fish have all shown similar levels of risk for the subsistence scenarios.

Site conditions that amplify the risks through the animal ingestion pathways include the proximity of the subsistence farmers and fishers to the facility (or the point of maximum impact by the facility) and the environment in which the facility is located (e.g., the meteorologic conditions that decrease dispersion or increase deposition). Site factors that increase the impact to waterbodies would amplify the fish pathways. Included are the size of the watershed and waterbody, the proximity to the facility's maximum impact, and factors such as the impervious area, which affects runoff of the contaminant into the waterbody.

The typical farmer's risk estimates range up to 5×10^{-6} for the high-end baseline estimates. The risks are lower than the subsistence scenario's risks because the general population's animal products ingestion is modeled as having a lower level of contamination (reflecting an average contamination level out to 20 kilometers from the sites) and because the fraction contaminated for the general population is assumed to be lower than that in the subsistence scenarios.

Due to the variability between the locations modeled, almost every pathway is the driving pathway in the general population risk estimates at one location or another. For instance, in areas where local beef production is high, the risk estimates from the beef pathway may be an order of magnitude above all others. At a location with low levels of agricultural activity, the soil ingestion pathway becomes the driver. Direct inhalation risks were

greater than indirect risks for one location because the location had low local production of agricultural products.

Table IV.9. Dioxin/Furan Individual Risk Estimates over All Subsistence Scenarios

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	2E-9	2E-6	2E-7	9E-5
Cement kilns	1E-8	2E-6	4E-7	9E-5
Lightweight aggregate kilns	2E-9	3E-7	9E-9	4E-7
Proposed Floor - Existing and New Sources				
Incinerators	3E-9	2E-6	1E-7	5E-5
Cement kilns	4E-9	1E-6	6E-8	2E-5
Lightweight aggregate kilns*	1E-8	2E-6	3E-8	
Proposed BTF - Existing and New Sources				
Incinerators*	3E-9	2E-6	6E-9	
Cement kilns	4E-9	1E-6	8E-9	2E-6
Lightweight aggregate kilns*	1E-8	2E-6	3E-8	
Alternative Floor - Existing Sources				
Incinerators*	2E-9	1E-6	4E-9	
Cement kilns	3E-9	7E-7	5E-9	1E-6
Lightweight aggregate kilns*	8E-9	1E-6	2E-8	

* For the scenario which gave the highest risk (the Subsistence Dairy Farmer Child), there is no high-end characterization. (See Table IV.1.)

Table IV.10. Dioxin/Furan Individual Risk Estimates for the Typical Farmer Scenario

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	2E-9	1E-8	1E-7	1E-6
Cement kilns	1E-8	1E-7	4E-7	5E-6
Lightweight aggregate kilns	1E-9	3E-9	3E-9	9E-9
Proposed Floor - Existing and New Sources				
Incinerators	2E-9	1E-8	7E-8	6E-7
Cement kilns	4E-9	5E-8	6E-8	9E-7
Lightweight aggregate kilns	6E-9	2E-8	1E-8	3E-8
Proposed BTF - Existing and New Sources				
Incinerators	2E-9	1E-8	3E-9	3E-8
Cement kilns	4E-9	5E-8	8E-9	1E-7
Lightweight aggregate kilns	6E-9	2E-8	1E-8	3E-8
Alternative Floor - Existing Sources				
Incinerators	1E-9	9E-9	2E-9	2E-8
Cement kilns	3E-9	3E-8	5E-9	7E-8
Lightweight aggregate kilns	4E-9	1E-8	9E-9	2E-8

Table IV.11 Maximum High-End Baseline 2,3,7,8-TCDD-TEQ Individual Risk Results by Scenario

Facility Type	Subsistence Beef Farmer	Subsistence Dairy Farmer	Subsistence Pork Farmer	Subsistence Poultry Farmer	Subsistence Fisher	Subsistence Dairy Farmer - Child	Typical Resident	Direct Inhalation for MEI
Incinerators	8E-5	6E-5	5E-5	5E-5	1E-5	9E-5	8E-7	8E-7
Cement kilns	3E-5	2E-5	6E-6	5E-5	9E-5	2E-5	3E-6	7E-7
Lightweight aggregate kilns	4E-7	3E-7	2E-8	2E-7	1E-7	4E-7	7E-9	1E-8

NOTE: Risks for scenarios not shown in this table fell between the subsistence scenarios and the typical residents.

Table IV.12. Ratio of Infant 2,3,7,8-TCDD-TEQ Exposure Through Breastmilk to Background (50 pg/kg/d) over All Subsistence Scenarios

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	0.00002	0.02	0.0008	0.6
Cement kilns	0.00006	0.08	0.0004	0.9
Lightweight aggregate kilns	0.00001	0.002	0.00001	0.003
Proposed Floor - Existing and New Sources				
Incinerators	0.00002	0.02	0.0004	0.3
Cement kilns	0.00002	0.03	0.00007	0.2
Lightweight aggregate kilns	0.00006	0.008		
Proposed BTF - Existing and New Sources				
Incinerators	0.00002	0.02		
Cement kilns	0.00002	0.03		
Lightweight aggregate kilns	0.00006	0.008		
Alternative Floor - Existing Sources				
Incinerators	0.00001	0.009		
Cement kilns	0.00002	0.02		
Lightweight aggregate kilns	0.00007	0.007		

Table IV.13. Dioxin/Furan Inhalation Individual Risk Estimates for the Maximum Exposed Individual (MEI)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	2E-9	6E-9	2E-7	8E-7
Cement kilns	1E-9	1E-8	7E-8	7E-7
Lightweight aggregate kilns	1E-9	2E-9	7E-9	1E-8
Proposed Floor - Existing and New Sources				
Incinerators	2E-7	6E-7	6E-7	2E-6
Cement kilns	1E-8	1E-7	4E-8	4E-7
Lightweight aggregate kilns	1E-7	2E-7	6E-7	9E-7
Proposed BTF - Existing and New Sources				
Incinerators	2E-9	6E-9	6E-9	2E-8
Cement kilns	4E-10	4E-9	2E-9	2E-8
Lightweight aggregate kilns	5E-9	1E-8	3E-8	4E-8
Alternative Floor - Existing Sources				
Incinerators	1E-9	4E-9	3E-9	1E-8
Cement kilns	3E-10	3E-9	1E-9	1E-8
Lightweight aggregate kilns	4E-9	7E-9	2E-8	3E-8

2. Metals and Hydrogen Chloride Results

The metals show hazard quotients for ingestion of noncarcinogens that do not exceed unity (see Tables IV.14 and IV.15). Tables IV.16 and IV.17 present the metal inhalation risks. For each metal that has both ingestion and inhalation health benchmarks, the ingestion risks were always higher. Chromium (VI) had the highest inhalation risks of the metals, with a maximum baseline risk of 2×10^{-6}. Baseline cancer risk estimates for ingestion of metals only reach 1 in a million for arsenic ingestion with a maximum ingestion high-end baseline risk estimate of 4×10^{-6}. Soil lead levels are below a soil lead level of concern of 400 ppm as shown in Table IV.18.

The MACT regulatory alternative option for metals results in higher risks than the 90th percentile baseline estimates for both inhalation and ingestion. This is a result of the manner in which the MACT metal limits are defined (U.S. EPA, 1995g). The MACT regulatory alternative options set limits for groups of metals--the semivolatile metals and the low-volatility metals. These groupings and limits are based on the metal's physical properties and not their toxicity.

The MACT regulatory alternative metal limits are meant to be imposed on the entire group of metals. For the risk assessment, the group limit was used for each metal's emissions calculations. This is based on the premise that any one of the metals may be the only metal of that group present in the emissions and, in that case, would be allowed to be emitted up to the group limit. An increase in the individual risks from the baseline could occur when the MACT regulatory alternative standards are applied, given the way the metal standards are defined.

Hydrogen chloride direct inhalation risks have been modeled for all facility types for baseline and the MACT regulatory alternative options. Cement kiln and incinerator hazard quotients never exceeded a hazard quotient of 1 for the high-end baseline emissions (see Table IV.19). Maximum high-end baseline hydrogen chloride hazard quotients reached 4 for the lightweight aggregate kilns and dropped to 1 with the proposed floor option.

Table IV.14. Individual Ingestion Risk Estimates over All Special Subpopulation Scenarios - Metals with Regulatory Options

Facility Type	Central Tendency		High End	
	Low	High	Low	High
ANTIMONY				
Baseline				
Incinerators	HQ = 0	HQ = 0.005	HQ = 0	HQ = 0.2
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0.004
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Proposed Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0.04		
Cement kilns	HQ = 0	HQ = 0.003		
Lightweight aggregate kilns	HQ = 0	HQ = 0.002		
Alternative Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0.009		
Cement kilns	HQ = 0	HQ = 0.001		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed Floor - New Sources				
Incinerators	HQ = 0	HQ = 0.04		
Cement kilns	HQ = 0	HQ = 0.001		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed BTF - New Sources				
Incinerators	HQ = 0	HQ = 0.01		
Cement kilns	HQ = 0	HQ = 0.001		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
CEM Compliance Options - New Sources				
Incinerators	HQ = 0	HQ = 0.03		
Cement kilns	HQ = 0	HQ = 0.003		
Lightweight aggregate kilns	HQ = 0	HQ = 0.001		

Table IV.14. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
ARSENIC				
Baseline				
Incinerators	6E-11 / HQ = 0	2E-7 / HQ = 0.004	2E-9 / HQ = 0	4E-6 / HQ = 0.05
Cement kilns	3E-11 / HQ = 0	3E-8 / HQ = 0.001	7E-10 / HQ = 0	5E-7 / HQ = 0.003
Lightweight aggregate kilns	3E-11 / HQ = 0	4E-8 / HQ = 0.001	8E-10 / HQ = 0	3E-7 / HQ = 0.007
Proposed Floor - Existing Sources				
Incinerators	1E-9 / HQ = 0	4E-6 / HQ = 0.09	4E-9	8E-6
Cement kilns	7E-10 / HQ = 0	6E-7 / HQ = 0.02	2E-9	2E-6
Lightweight aggregate kilns*	3E-9 / HQ = 0	3E-6 / HQ = 0.08	9E-9	
Alternative Floor - Existing Sources				
Incinerators	3E-10 / HQ = 0	9E-7 / HQ = 0.02	1E-9	2E-6
Cement kilns	2E-10 / HQ = 0	2E-7 / HQ = 0.007	7E-10	5E-7
Lightweight aggregate kilns*	4E-10 / HQ = 0	5E-7 / HQ = 0.01	1E-9	
Proposed Floor - New Sources				
Incinerators	1E-9 / HQ = 0	4E-6 / HQ = 0.09	4E-9	8E-6
Cement kilns	3E-10 / HQ = 0	2E-7 / HQ = 0.009	1E-9	7E-7
Lightweight aggregate kilns*	4E-10 / HQ = 0	5E-7 / HQ = 0.01	1E-9	
Proposed BTF - New Sources				
Incinerators	4E-10 / HQ = 0	1E-6 / HQ = 0.03	1E-9	3E-6
Cement kilns	3E-10 / HQ = 0	2E-7 / HQ = 0.009	1E-9	7E-7
Lightweight aggregate kilns*	4E-10 / HQ = 0	4E-7 / HQ = 0.01	2E-9	
CEM Compliance Option - New Sources				
Incinerators	1E-9 / HQ = 0	3E-6 / HQ = 0.06	3E-9	6E-6
Cement kilns	8E-10 / HQ = 0	8E-7 / HQ = 0.03	3E-9	2E-6
Lightweight aggregate kilns*	1E-9 / HQ = 0	1E-6 / HQ = 0.03	3E-9	

***** For the scenario which gave the highest risk for central tendency (the Subsistence Dairy Farmer Child), there is no high-end characterization.

Table IV. 14. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
BERYLLIUM				
Baseline				
Incinerators	3E-11 / HQ = 0	5E-9 / HQ = 0	6E-10 / HQ = 0	5E-8 / HQ = 0
Cement kilns	5E-11 / HQ = 0	2E-8 / HQ = 0	6E-10 / HQ = 0	1E-7 / HQ = 0
Lightweight aggregate kilns	4E-11 / HQ = 0	8E-9 / HQ = 0	5E-10 / HQ = 0	4E-8 / HQ = 0
Proposed Floor - Existing Sources				
Incinerators	8E-9 / HQ = 0	1E-6 / HQ = 0.001		
Cement kilns	9E-9 / HQ = 0	4E-6 / HQ = 0.002		
Lightweight aggregate kilns	2E-8 / HQ = 0	4E-6 / HQ = 0.002		
Alternative Floor - Existing Sources				
Incinerators	2E-9 / HQ = 0	3E-7 / HQ = 0		
Cement kilns	3E-9 / HQ = 0	1E-6 / HQ = 0.001		
Lightweight aggregate kilns	3E-9 / HQ = 0	7E-7 / HQ = 0		
Proposed Floor - New Sources				
Incinerators	8E-9 / HQ = 0	1E-6 / HQ = 0.001		
Cement kilns	4E-9 / HQ = 0	2E-6 / HQ = 0.001		
Lightweight aggregate kilns	3E-9 / HQ = 0	7E-7 / HQ = 0		
Proposed BTF - New Sources				
Incinerators	2E-9 / HQ = 0	4E-7 / HQ = 0		
Cement kilns	4E-9 / HQ = 0	1E-6 / HQ = 0.001		
Lightweight aggregate kilns	3E-9 / HQ = 0	6E-7 / HQ = 0		
CEM Compliance Option - New Sources				
Incinerators	6E-9 / HQ = 0	9E-7 / HQ = 0		
Cement kilns	1E-8 / HQ = 0	5E-6 / HQ = 0.002		
Lightweight aggregate kilns	8E-9 / HQ = 0	1E-6 / HQ = 0.001		

Table IV.14. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
CADMIUM				
Baseline				
Incinerators	HQ = 0	HQ = 0.001	HQ = 0	HQ = 0.02
Cement kilns	HQ = 0	HQ = 0.001	HQ = 0	HQ = 0.01
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0.003
Proposed Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0.01		
Cement kilns	HQ = 0	HQ = 0.004		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Alternative Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0.003		
Cement kilns	HQ = 0	HQ = 0.01		
Lightweight aggregate kilns	HQ = 0	HQ = 0.001		
Proposed Floor - New Sources				
Incinerators	HQ = 0.001	HQ = 0.01		
Cement kilns	HQ = 0	HQ = 0.004		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed BTF - New Sources				
Incinerators	HQ = 0	HQ = 0.004		
Cement kilns	HQ = 0	HQ = 0.005		
Lightweight aggregate kilns	HQ = 0	HQ = 0.001		
CEM Compliance Option - New Sources				
Incinerators	HQ = 0	HQ = 0.004		
Cement kilns	HQ = 0	HQ = 0.005		
Lightweight aggregate kilns	HQ = 0	HQ = 0.001		

Table IV.14. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
CHROMIUM (III)				
Baseline				
Incinerators	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Proposed Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Alternative Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed Floor - New Sources				
Incinerators	HQ = 0	HQ = 0		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed BTF - New Sources				
Incinerators	HQ = 0	HQ = 0		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
CEM Compliance Option - New Sources				
Incinerators	HQ = 0	HQ = 0		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		

Table IV. 14. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
CHROMIUM (VI)				
Baseline				
Incinerators	HQ = 0	HQ = 0	HQ = 0	HQ = 0.001
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Proposed Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0.003		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0.001		
Alternative Floor - Existing Sources				
Incinerators	HQ = 0	HQ = 0.001		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed Floor - New Sources				
Incinerators	HQ = 0	HQ = 0.003		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
Proposed BTF - New Sources				
Incinerators	HQ = 0	HQ = 0.001		
Cement kilns	HQ = 0	HQ = 0		
Lightweight aggregate kilns	HQ = 0	HQ = 0		
CEM Compliance Option - New Sources				
Incinerators	HQ = 0	HQ = 0.002		
Cement kilns	HQ = 0	HQ = 0.001		
Lightweight aggregate kilns	HQ = 0	HQ = 0.001		

Table IV.15. Subpopulation Scenarios -Metals without Regulatory Options

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Barium				
Incinerators	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0.003
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Nickel				
Incinerators	HQ = 0	HQ = 0	HQ = 0	HQ = 0.002
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Selenium				
Incinerators	HQ = 0	HQ = 0	HQ = 0	HQ = 0.003
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0.004
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0
Silver				
Incinerators	HQ = 0	HQ = 0.001	HQ = 0	HQ = 0.005
Cement kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0.002
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0	HQ = 0.001
Thallium				
Incinerators	HQ = 0	HQ = 0.02	HQ = 0	HQ = 0.1
Cement kilns	HQ = 0	HQ = 0.01	HQ = 0	HQ = 0.1
Lightweight aggregate kilns	HQ = 0	HQ = 0.001	HQ = 0	HQ = 0.002

Table IV.16. Inhalation Individual Risk Estimates for Maximally Exposed Individual - Metals with Regulatory Options

Facility Type	Central Tendency		High End	
	Low	High	Low	High
ARSENIC				
Baseline				
Incinerators	4E-9	2E-8	2E-7	6E-7
Cement kilns	6E-10	7E-9	1E-8	1E-7
Lightweight aggregate kilns	9E-9	2E-8	2E-7	4E-7
Proposed Floor - Existing Sources				
Incinerators	9E-8	4E-7	4E-7	1E-6
Cement kilns	1E-8	2E-7	4E-8	4E-7
Lightweight aggregate kilns	8E-7	2E-6	2E-6	5E-6
Alternative Floor - Existing Sources				
Incinerators	2E-8	1E-7	1E-7	3E-7
Cement kilns	4E-9	5E-8	1E-8	1E-7
Lightweight aggregate kilns	1E-7	3E-7	4E-7	7E-7
Proposed Floor - New Sources				
Incinerators	9E-8	4E-7	4E-7	1E-6
Cement kilns	5E-9	6E-8	1E-8	1E-7
Lightweight aggregate kilns	1E-7	3E-7	4E-7	7E-7
Proposed BTF - New Sources				
Incinerators	3E-8	1E-7	1E-7	4E-7
Cement kilns	5E-9	6E-8	1E-8	1E-7
Lightweight aggregate kilns	1E-7	3E-7	4E-7	7E-7
CEM Compliance Option - New Sources				
Incinerators	6E-8	3E-7	3E-7	8E-7
Cement kilns	2E-8	2E-7	4E-8	4E-7
Lightweight aggregate kilns	3E-7	6E-7	8E-7	2E-6

Table 16. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
BERYLLIUM				
Baseline				
Incinerators	2E-10	7E-10	7E-9	3E-8
Cement kilns	5E-11	5E-10	1E-9	1E-8
Lightweight aggregate kilns	9E-10	1E-9	1E-8	2E-8
Proposed Floor - Existing Sources				
Incinerators	5E-8	2E-7	2E-7	7E-7
Cement kilns	9E-9	9E-8	3E-8	3E-7
Lightweight aggregate kilns	5E-7	5E-7	1E-6	2E-6
Alternative Floor - Existing Sources				
Incinerators	1E-8	5E-8	4E-8	2E-7
Cement kilns	3E-9	3E-8	7E-9	7E-8
Lightweight aggregate kilns	7E-8	8E-8	2E-7	3E-7
Proposed Floor - New Sources				
Incinerators	5E-8	2E-7	2E-7	7E-7
Cement kilns	3E-9	3E-8	1E-8	1E-7
Lightweight aggregate kilns	7E-8	8E-8	2E-7	3E-7
Proposed BTF - New Sources				
Incinerators	2E-8	6E-8	5E-8	2E-7
Cement kilns	3E-9	3E-8	1E-8	1E-7
Lightweight aggregate kilns	7E-8	8E-8	2E-7	3E-7
CEM Compliance Option - New Sources				
Incinerators	4E-8	1E-7	1E-7	5E-7
Cement kilns	1E-8	1E-7	3E-8	3E-7
Lightweight aggregate kilns	2E-7	2E-7	4E-7	8E-7

Table IV.16. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
CADMIUM				
Baseline				
Incinerators	4E-9	1E-8	2E-7	7E-7
Cement kilns	1E-9	1E-8	4E-8	4E-7
Lightweight aggregate kilns	1E-8	2E-8	3E-7	5E-7
Proposed Floor - Existing Sources				
Incinerators	5E-8	1E-7	2E-7	5E-7
Cement kilns	3E-9	3E-8	1E-8	1E-7
Lightweight aggregate kilns	1E-8	2E-8	4E-8	6E-8
Alternative Floor - Existing Sources				
Incinerators	9E-9	2E-8	3E-8	1E-7
Cement kilns	7E-9	7E-8	3E-8	3E-7
Lightweight aggregate kilns	4E-8	8E-8	1E-7	2E-7
Proposed Floor - New Sources				
Incinerators	5E-8	1E-7	2E-7	5E-7
Cement kilns	3E-9	3E-8	1E-8	1E-7
Lightweight aggregate kilns	6E-9	1E-8	2E-8	3E-8
Proposed BTF - New Sources				
Incinerators	1E-8	3E-8	4E-8	2E-7
Cement kilns	3E-9	3E-8	1E-8	1E-7
Lightweight aggregate kilns	5E-8	1E-7	2E-7	3E-7
CEM Compliance Option - New Sources				
Incinerators	2E-8	4E-8	5E-8	2E-7
Cement kilns	3E-9	3E-8	1E-8	1E-7
Lightweight aggregate kilns	6E-8	1E-7	2E-7	3E-7

Table IV.16. (continued)

Facility Type	Central Tendency		High End	
	Low	High	Low	High
CHROMIUM (VI)				
Baseline				
Incinerators	7E-9	3E-8	4E-7	2E-6
Cement kilns	1E-9	1E-8	3E-8	3E-7
Lightweight aggregate kilns	1E-8	2E-8	2E-7	4E-7
Proposed Floor - Existing Sources				
Incinerators	3E-7	1E-6	8E-7	4E-6
Cement kilns	4E-8	4E-7	2E-7	2E-6
Lightweight aggregate kilns	2E-6	4E-6	7E-6	1E-5
Alternative Floor - Existing Sources				
Incinerators	6E-8	3E-7	2E-7	1E-6
Cement kilns	1E-8	1E-7	4E-8	4E-7
Lightweight aggregate kilns	3E-7	6E-7	1E-6	2E-6
Proposed Floor - New Sources				
Incinerators	3E-7	1E-6	8E-7	4E-6
Cement kilns	2E-8	2E-7	6E-8	6E-7
Lightweight aggregate kilns	3E-7	6E-7	1E-6	2E-6
Proposed BTF - New Sources				
Incinerators	8E-8	3E-7	3E-7	1E-6
Cement kilns	2E-8	2E-7	6E-8	6E-7
Lightweight aggregate kilns	3E-7	5E-7	1E-6	2E-6
CEM Compliance Option - New Sources				
Incinerators	2E-7	8E-7	6E-7	3E-6
Cement kilns	5E-8	5E-7	2E-7	2E-6
Lightweight aggregate kilns	6E-7	1E-6	2E-6	5E-6

Table IV.17. Inhalation Individual Risk Estimates for the Maximally Exposed Individual Metals without Regulatory Options

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Nickel - Baseline				
Incinerators	2E-9	5E-9	5E-8	2E-7
Cement kilns	2E-10	2E-9	3E-9	3E-8
Lightweight aggregate kilns	8E-9	1E-8	2E-7	3E-7
Barium - Baseline				
Incinerators	HQ = 0	HQ = 0	HQ = 0.001	HQ = 0.003
Cement kilns	HQ = 0	HQ = 0.001	HQ = 0.001	HQ = 0.006
Lightweight aggregate kilns	HQ = 0	HQ = 0	HQ = 0.001	HQ = 0.002

Table IV.18. Soil Lead Ratios

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	0.000006	0.0006	0.0002	0.01
Cement kilns	0.00001	0.006	0.0001	0.06
Lightweight aggregate kilns	0.000001	0.00008	0.00004	0.006
Proposed Floor - Existing Sources				
Incinerators	0.000008	0.0008		
Cement kilns	0.000003	0.002		
Lightweight aggregate kilns	0.0000004	0.00004		
Alternative Floor - Existing Sources				
Incinerators	0.000001	0.0001		
Cement kilns	0.000008	0.005		
Lightweight aggregate kilns	0.000002	0.0002		
Proposed Floor - New Sources				
Incinerators	0.000008	0.0008		
Cement kilns	0.000003	0.002		
Lightweight aggregate kilns	0.0000002	0.00002		
Proposed BTF - New Sources				
Incinerators	0.000002	0.0002		
Cement kilns	0.000003	0.002		
Lightweight aggregate kilns	0.000002	0.0002		
CEM Compliance Option - New Sources				
Incinerators	0.000003	0.0003		
Cement kilns	0.000004	0.002		
Lightweight aggregate kilns	0.000002	0.0002		

Table IV.19. Hydrochloric Acid Inhalation Individual Risks Estimate for Maximally Exposed Individual

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	HQ = 0.001	HQ = 0.003	HQ = 0.01	HQ = 0.05
Cement kilns	HQ = 0	HQ = 0.004	HQ = 0.004	HQ = 0.04
Lightweight aggregate kilns	HQ = 0.1	HQ = 0.2	HQ = 2	HQ = 4
Proposed Floor - Existing Sources				
Incinerators	HQ = 0.02	HQ = 0.05		
Cement kilns	HQ = 0.01	HQ = 0.1		
Lightweight aggregate kilns	HQ = 0.8	HQ = 1		
Proposed BTF - Existing Sources				
Incinerators	HQ = 0.02	HQ = 0.05		
Cement kilns	HQ = 0.01	HQ = 0.1		
Lightweight aggregate kilns	HQ = 0.1	HQ = 0.2		
Proposed Alternative Floor - Existing Sources				
Incinerators	HQ = 0.001	HQ = 0.004		
Cement kilns	HQ = 0	HQ = 0.005		
Lightweight aggregate kilns	HQ = 0.7	HQ = 1		
Proposed Alternative BTF - Existing Sources				
Incinerators	HQ = 0.001	HQ = 0.004		
Cement kilns	HQ = 0	HQ = 0.005		
Lightweight aggregate kilns	HQ = 0.01	HQ = 0.02		
Proposed Floor - New Sources				
Incinerators	HQ = 0.02	HQ = 0.05		
Cement kilns	HQ = 0.01	HQ = 0.1		
Lightweight aggregate kilns	HQ = 0.02	HQ = 0.04		
Proposed BTF - New Sources				
Incinerators	HQ = 0.004	HQ = 0.01		
Cement kilns	HQ = 0.001	HQ = 0.01		
Lightweight aggregate kilns	HQ = 0.01	HQ = 0.02		

C. Uncertainty/Limitations

Uncertainty can be introduced into a health risk assessment at every step in the process. It occurs because risk assessment is a complex process, requiring the integration of

- Release of pollutants into the environment
- Fate and transport of pollutants in a variety of different and variable environments by processes that are often poorly understood or too complex to quantify accurately
- Potential for adverse health effects in humans as extrapolated from animal bioassays
- Probability of adverse effects in a human population that is highly variable genetically, in age, in activity level, and in life style.

Even using the most accurate data with the most sophisticated models, uncertainty is inherent in the process.

1. Background

Finkel (1990) classified all uncertainty into four types (parameter uncertainty, model uncertainty, decision-rule uncertainty, and variability) which are summarized in Table IV.20. The first two, parameter uncertainty and model uncertainty, are generally recognized by risk assessors as major sources of uncertainty.

Parameter uncertainty occurs when parameters appearing in equations cannot be measured precisely and/or accurately either because of equipment limitations or because the quantity being measured varies spatially or temporally. Random, or sample errors, are a common source of parameter uncertainty that is especially critical for small sample sizes. More difficult to recognize are nonrandom or systematic errors that result from bias in sampling, experimental design, or choice of assumptions.

Model uncertainty is associated with all models used in all phases of a risk assessment. These include the animal models used as surrogates for testing human carcinogenicity, dose-response models used in extrapolations, as well as the computer models used to predict the fate and transport of chemicals in the environment. The use of rodents as surrogates for humans introduces uncertainty into the risk factor since there is considerable interspecies variability in sensitivity. Computer models are simplifications of reality, requiring exclusion of some variables

that influence predictions but cannot be included in models due either to increased complexity or to a lack of data on that

IV.20. Sources of Uncertainty in Risk Assessment*

General Type	Specific Source of Uncertainty	Comments/Examples
Parameter uncertainty	Measurement errors	• include limitations of equipment, methodology, and human error • some processes impossible to measure exactly
	Random errors	• sampling errors • can be minimized by increasing sample size
	Systematic errors	• nonrandom errors • result of inherent flaw in data gathering processes • minimize by external peer review
Model uncertainty	Surrogate variables	• e.g., use of animal bioassays to determine effect on humans
	Excluded variables	• may result from model simplification or failure to recognize an important variable
	Abnormal conditions	• e.g., failure to recognize importance of episodic meteorological events
	Incorrect model form	• e.g., choice of dose-response model for carcinogens
Decision-rule uncertainty		• more important for risk management, but need to recognize that value judgments affect choice of model and interpretation of results
Variability		• those important for health risk assessment include sources of pollutant releases, environmental factors, genetic variability, and lifestyle differences • even if variability is known (therefore, not in itself uncertain) it still contributes to overall uncertainty of the risk assessment

*Adapted from Finkel, 1990.

parameter. The risk assessor needs to consider the importance of excluded variables on a case-by-case basis, because a given variable may be important in some instances and not in others. A similar

problem can occur when a model that is applicable under average conditions is used for a case where conditions differ from the average. Finally, choosing the correct model form is often difficult because conflicting theories seem to explain a phenomenon equally well.

The third type, decision-rule uncertainty, is probably of more concern to risk managers. This type of uncertainty arises, for example out of the need to balance different social concerns when determining an acceptable level of risk. Finkel (1990) provides a complete discussion of decision-rule uncertainty.

Variability, the fourth source of uncertainty, is often used interchangeably with the term "uncertainty," but this is not strictly correct. Variability may be tied to variations in physical and biological processes and cannot be reduced with additional research or information, though it may be known with greater certainty (e.g., age distribution of a population may be known and represented by the mean age and its standard deviation). "Uncertainty" is a description of the imperfection in knowledge of the true value of a particular parameter or its real variability in an individual or a group. In general, uncertainty is reducible by additional information-gathering or analysis activities (better data, better models), whereas real variability will not change (although it may be more accurately known) as a result of better or more extensive measurements (Hattis and Burmaster, 1994).

The degree to which all types of uncertainty need to be quantified and the amount of uncertainty that is acceptable varies with the intent of the analysis. If a screening level analysis is desired, a high degree of uncertainty is often acceptable, provided that conservative assumptions are used to bias potential error toward protecting human health. A region-wide or nationwide study will be less uncertain than a localized site-specific one in determining average risks across the region or nation, since in the former case it may be possible to use the average of a parameter value over many sites (which often can be estimated better than a site-specific value). However, the general analysis may be highly uncertain in defining the range of possible risks which are influenced by site-specific conditions. In general, the more detailed or accurate the risk characterization, the more carefully uncertainty needs to be considered.

2. Characterize Uncertainty

A deterministic approach was used to estimate individual risk, for both cancer and noncancer health effects. However, this approach considered both the uncertainty and variability in many of the input parameters associated with the source, the environmental setting, and lifestyle difference in order to develop a range of risk estimates that are believed to capture the range of individual risk spanning a central tendency estimate to a high end estimate on the risk distribution. Some aspects of the uncertainty in these risk estimates can be addressed semiquantitatively, others qualitatively and others cannot be addressed.

Following the sources of uncertainty described in Table IV.20, parameter uncertainty, model uncertainty, decision-rule uncertainty, and variability will be discussed in this section. Since decision rule uncertainty has an major effect on the boundaries of the analysis, as well as providing the science policy on which the analysis is based, it will be discussed first. Within each of these categories of uncertainty, the following specific topics will be addressed, as appropriate: receptors, environmental settings/factors, facility characteristics and emissions, environmental fate and transport, and effects.

a. Decision rule uncertainty

There are a number of policy and risk management decisions that have an influence on the uncertainty of the risk analysis but will not be quantitatively addressed here. First, and possibly the most important aspect for the baseline risk estimates, is the selection of constituents to be included in the analysis. The constituents modeled in the analysis include the dioxin and furan congeners for which TEFs were available, hydrogen chloride for inhalation risks, and the metals listed below for both inhalation and indirect ingestion risks.

Lead (soil concentration)	Antimony
Arsenic	Barium
Beryllium	Cadmium
Chromium (III and VI)	Thallium
Nickel	Silver
Selenium	

The selection of the constituents for modeling was an EPA policy decision. Non-dioxin particles of incomplete combustion (PICS) were not included in the analysis at this time because adequate data on the emissions of PICs from combustors burning hazardous waste do not exist. Many PICs are highly lipophilic and tend to bioaccumulate in the food chain thus presenting potentially high risk through the consumption of contaminated food. The effect of excluding these constituents from the analysis is to potentially underestimate the baseline risk. However, some of the various MACT regulatory alternative options for dioxins would also provide some control for these PICs. If we assume that the MACT controls will also be effective in controlling non-dioxin PICs, then the effect of excluding these PICs from the analysis is to underestimate risk reductions and the benefits associated with these reductions.

Mercury was not evaluated quantitatively because in EPA's judgement, the current indirect exposure methodology and data are not adequate to address the complex

environmental fate and transport processes of mercury. The effect of excluding mercury from the quantitative analysis is to potentially underestimate the risks.

A second area of decision rule uncertainty is the selection of a deterministic approach based on example cases. The approach used in this analysis provides a range of risk estimates that vary based on emission rates, environmental factors, and receptors. The range in risk estimates is believed to encompass a central tendency estimate of risk to the general population in the vicinity of combustion facilities burning hazardous waste, as well as the risk to more highly exposed subpopulations. The analytical approach itself is an attempt to quantify parameter uncertainty and parameter variability. However, because this is a deterministic approach, the precision of each risk estimate, in terms of where on the risk distribution a particular estimate is, cannot be specified. Since each of the point estimates are made independent of a probability of occurrence, it is difficult to state precisely what percentile of the risk distribution is represented by the estimate. Nevertheless, the approach of providing central tendency, high end and bounding estimates for a suite of human receptors exposed via direct and indirect pathways in a variety of environmental settings provides a robust characterization of risk.

A third area of decision-rule uncertainty includes the use of standard EPA default values in the analysis. These include inhalation and consumption rates, body weight, and lifetime, which are standard default values used in most EPA risk assessments. Inhalation and consumption rates are highly correlated to body weight for adults. Using a single point estimate for these variables instead of a joint probability distribution ignores a variability that may influence the results by up to a factor of two or three.

A fourth area of decision-rule uncertainty is the use of Agency-verified cancer slope factors, reference doses and reference concentration. These health benchmarks are used as single point estimates throughout the analysis. These benchmarks have both uncertainty and variability associated with them. However, the Agency has developed a process for setting verified health benchmark values to be used in all Agency risk assessments. With the exception of the dioxin toxicity equivalency methodology, all health benchmarks used in this analysis are verified through the Agency's work groups and available on the Agency's Integrated Risk Information System. No estimation of the uncertainty in the use of the Agency's verified health benchmarks or the dioxin toxicity equivalency methodology will be made here.

b. Model Uncertainty

The models used in this assessment were selected based on science policy. Thus, the air dispersion and deposition model and the indirect exposure models were selected because they provide the information needed for this analysis and are considered by the Agency to be state-of-the-science. This choice of models could also be considered under decision rule uncertainty. The air dispersion model used in the analysis, ISCSTDFT, is released in draft version and has not been widely applied in the present form. Few data are available on atmospheric deposition rates for chemicals other than criteria pollutants, making the selection of input parameters related to deposition and validation of modeled deposition rates difficult. Because dry deposition of vapor phase materials is evaluated external to the air dispersion model, the plume is not depleted and, therefore, mass balance is not maintained. The effect of this would be to overestimate deposition but the magnitude of the overestimation is unknown. Mass balance is maintained for other forms of deposition (i.e., wet deposition and particle phase dry deposition). Long range transport of pollutants into and out of the areas considered was not modeled. The result is the underestimation of risk attributable to each facility.

Also, although the facilities selected were representative with respect to the range in size and geographic location, their selection was influenced by availability of appropriate meteorologic data; therefore, the 11 facilities cannot be considered statistically representative of all hazardous waste combustion units. Furthermore, small on-site incinerators are not represented by the case studies. Therefore, it is expected that the individual risk estimates overstate the risk for these types of facilities, but the extent of overestimation is unknown.

c. Parameter Uncertainty and Variability

It is often difficult to separate variability from true uncertainty in the various input parameters used in the analysis. Therefore, both variability and uncertainty will be discussed jointly for the parameter input values and, where possible, the difference between the two will be identified.

The summary of risk results presented in Table IV.21 also provide a characterization of the parameter uncertainty and variability in the risk analysis. Twelve receptor scenarios, including the general population and special sub-populations, were modeled for the four incinerators, five cement kilns, and two light-weight aggregate kilns considered. The range in risk estimates for incinerators for baseline dioxin/furan emissions varies from the lowest central tendency risk estimate of 7×10^{-10} to a bounding risk estimate of 1×10^{-4}. For cement kilns that range is from 8×10^{-10} to 2×10^{-4}, and for lightweight aggregate

kilns the range is from 3×10^{-10} to 2×10^{-6}. The bounding estimates are considered to be the highest risk possible on the distribution of risk for a given receptor scenario. The true central tendency risk is unknown but represented by a range of estimates using central tendency emission estimates for different facilities, different environmental settings and different receptors. High end estimates are thought to be above the 90th percentile of the risk distribution for each receptor but not as high as a bounding estimate. The precision of the percentile estimate is unknown given the deterministic approach used in the analysis. Summary tables of the range of results for the other chemicals modeled are contained in Appendix B. Within the range of risk estimates, the magnitude of the parameter uncertainty and variability in the analysis is presumed to be captured. The differences result from the selection of values used, after a systematic evaluation of the data, to arrive at estimates of

- Central tendency and high-end emissions that includes both variability and uncertainty in the emission rates

- Facility characteristics that are representative of the variation in facilities within a category (including stack parameters and facility size)

- Environmental factors based on site specific data including meteorology, the size and locations of the waterbodies, and land use for determining locations of receptors

- Exposure variables including central tendency and high-end estimates of exposure durations and contaminated fractions for each receptor/environmental setting combination (e.g. recreational fisher/waterbody combination).

c.1. Emissions Characterizations

The emissions estimates were developed based on a systematic evaluation of available emissions data to obtain a 50th and 90th percentile estimate of emissions from stack sampling for trial burns and compliance tests (U.S. EPA, 1995f). These emission estimates do tend to overestimate the emissions of some constituents that would be expected under normal operating conditions, since chemical spikes were used in order to aid in the determination of removal efficiencies. However, the emission estimates used in the modeling do not reflect higher emissions that might result from upset conditions, and in this manner may result in an underestimate of risk. In addition, this analysis shows that dioxin/furan emissions from combustors burning hazardous waste are of potential concern through indirect pathways. Although spiked chemicals used in trial burns may have some influence on dioxin/furan formation, it is unlikely that the spikes have a large influence on the formation of these constituents in stack gasses.

IV.21 Range of 2,3,7,8-TCDD-TEQ Individual Risk Results Over All Scenarios

Facility Type	Central Tendency			High End			Bounding
	Low	High	Median	Low	High	Median	
Baseline							
Incinerators	7E-10	2E-6	3E-8	8E-8	9E-5	1E-6	1E-4
Cement Kilns	8E-10	2E-6	1E-7	4E-8	9E-5	4E-6	2E-4
LWAK	3E-10	3E-8	9E-9	1E-9	4E-7	2E-8	2E-6
Proposed Floor - Existing and New Sources							
Incinerators	7E-10	2E-6	3E-8	4E-8	4E-5	7E-7	6E-5
Cement Kilns	3E-10	1E-6	5E-8	7E-9	2E-5	7E-7	3E-5
LWAK	2E-9	2E-6	5E-8	5E-9	1E-6	7E-8	7E-6
Proposed BTF - Existing and New Sources							
Incinerators	7E-10	2E-6	3E-8	2E-9	2E-6	3E-8	3E-6
Cement Kilns	3E-10	1E-6	5E-8	9E-10	2E-6	9E-8	5E-6
LWAK	2E-9	2E-6	5E-8	5E-9	1E-6	7E-8	7E-6

c.2. Facility Characterizations

An attempt was made to select facilities for modeling that were representative of the universe of facilities burning hazardous wastes. The ranges in facility sizes and stack characteristics affect the range of risk results directly through the amount of chemical emitted and the dispersion of the chemical over the area modeled. For hazardous waste incinerators, the range in facility sizes used in the analysis is from the 30th percentile to the 90th percentile of the 11 facilities for which the size data were available at the initiation of this analysis. Eleven facilities represents 6 percent of all incinerators burning hazardous waste. For cement kilns that burn hazardous wastes, the sizes used in this analysis range from the 20th percentile to the 80th percentile of the 16 facilities for which the size data were available at the initiation of this analysis. Sixteen facilities represent 50 percent of all cement kiln facilities burning hazardous waste. For the lightweight aggregate kilns, the two selected ranked as the largest and at the 20th percentile for the facility size of the six facilities for which size data was available. Thus, the variability among facilities was incorporated into the analysis, however, the uncertainty in this variability has not been quantified.

Stack characteristics were not available for all of the facilities considered during the selection of facilities to model. Therefore, it is not possible to determine where the stack characteristics of the facilities selected for this analysis fall on the distribution of all facilities. However, the stack characteristics are less important for indirect exposures than for direct inhalation exposures because the deposition rates are influenced markedly by the wet deposition where the maximum impact occurs close to the stack, independent of the stack characteristics. Because of the relationship between the stack characteristics, the meteorological conditions, and the receptor placement as a function of site-specific land use, the uncertainty resulting from the stack characteristics of the selected facilities in relation to the entire group cannot be quantified independent of these other variables.

c.3. Environmental Factors

Two complex terrain sites, one incinerator and one cement kiln, were modeled for the analysis and as expected their risks were somewhat higher than comparable simple terrain locations. Thus the range of estimates of risk do attempt to capture terrain variations, however it is possible that a location may be found where terrain impacts may result in more extreme risks. Complex terrain can have a marked effect on the risk estimates, greater than a factor of 5.

The need to use appropriate meteorological data introduces uncertainty into the analysis, due to the inability to acquire onsite meteorological data, which is the most representative for each site. This introduces an uncertainty that would vary from location to location, and that cannot be quantified here. For most of the facilities, nearby National Weather Service data provided an approximation of the meteorological conditions at the site. However, for some of the facilities this was not an adequate approximation, due to the difference in the orientation of terrain features between the facility and the location where the available data were collected. Different meteorologic conditions can influence the risk results by up to an order of magnitude given the same facility characteristics and surrounding land uses.

Many parameter values are used in modeling the fate and transport of the pollutant for terrestrial and aquatic foodchain exposures. In order to capture the uncertainty in the range of risk estimates, site specific inputs for a number of different cases were used. The sites were selected to give a range of different settings to capture the parameter uncertainty and variability in the risk estimates. Uncertainties also arise from the attempt to model a complex system with algebraic equations. The equation selection was made by EPA and presented in the Indirect Exposure Document (EPA, 1990b) and its Addendum (EPA, 1993a). The uncertainty resulting from the use of the fate and transport equations will not be discussed here.

Soil concentrations varied as a result of the site-specific air impacts and the movement of the constituents through the soil. Soil concentrations are a function of the flux from the air, time, mixing depth, and soil losses. Uncertainties in the estimates of the air-to-soil flux have been discussed above with regard to the deposition model used in the analysis. An additional item in the calculation of the air-to-soil flux is the partitioning of the constituent between the particle and vapor phases. To avoid double counting the semi-volatile compounds, a partitioning between the vapor and particle bound fractions was approximated. The uncertainties resulting from the estimation of the partitioning would be reflected in changes between the impacts calculated from the vapor and particle pathways. However, since the flux of both phases are ultimately considered in the media concentrations (wet and dry deposition of both vapors and particles are modeled) the range of uncertainty is lessened.

Losses of contaminants deposited on soil are due to leaching, soil erosion, runoff, degradation, and volatilization. For each of the loss mechanisms, a variety of defaults and site-specific estimates of parameter values are used. As an example, those associated with leaching are discussed below. Parameters used in the leaching equations include EPA recommended values such as the mixing depth, single point

best judgement approximations such as soil bulk density and volumetric water content, chemical specific parameters such as the soil-water partition coefficient, and site specific parameters such as the annual average precipitation, runoff, and evapotranspiration. The range of soil concentrations for the various constituents across cases is, in part a characterization of the variability of these parameters, as well as, the variability among facility characteristics and deposition processes.

Water concentrations are a function of the contaminant flux from the air to the watershed and waterbody; soil concentrations and erosion and runoff estimates; and site-specific watershed parameters. Uncertainties in the estimates of the air-to-soil flux and in the estimation of soil concentrations have been discussed. By selecting three or four specific waterbodies for each case, that are modeled with site-specific parameters, the range of estimates in water concentrations should capture the uncertainty in these estimates. The waterbodies selected reflect those that are close to the facilities to capture the upper end of the distribution and waterbodies that are located at greater distances, which were identified as drinking water sources. Additional point estimates of parameter values were necessary to estimate water concentrations. Systematic evaluation of the available distributions of parameter values were undertaken to select values which represent central tendency estimates.

Plant concentrations are functions of air to soil and air to plant fluxes. Approximations of biotransfer values are the main source of uncertainty involved in the estimation of plant tissue concentrations from both root and direct uptake from the air. These equations used to estimate transfer factors were developed from experimental data for limited types of vegetation. Correction factors have been applied to the transfer factors for lipophilic compounds such as dioxins and furans. These correction factors reduce the range of uncertainty associated with the use of the transfer factors. Both the transfer factors and the correction factors used are EPA-recommended values discussed in the Addendum (EPA, 1993a), the Dioxin Exposure Assessment (EPA, 1994c), and Lorber (1995). The uncertainty involved in their use will not be quantified here.

Animal tissue concentrations for terrestrial animals are a result of the intermediate plant and soil concentrations, the animal exposure factors, and additional plant-to-animal biotransfer and bioconcentration factors. Animal- and chemical-specific factors were available for some animal-chemical pairs; however, additional uncertainty was introduced through the application of beef transfer factors to pork and through failure to consider exposures of poultry for chemicals other than dioxin. It is not possible to

quantify the pork uncertainty, but, for the poultry pathway, the effect of neglecting the exposures and resulting risks for these chemicals would be negligible when compared to other exposure pathways.

The animal exposures are unique to this analysis. Site-specific land use data were used to arrive at a range of estimates of exposure locations for the animals. In this range of estimates, and through the use of animal exposures for typical farms within 20 kilometers of the facility and more highly exposed farms located at the farm with the maximum point of deposition, the uncertainty involved in the estimation of animal exposures was captured.

Fish concentrations are a function of the water concentrations discussed above and the transfer and bioaccumulation factors. Two types of transfer factors are used, depending the chemical and on how the factor was experimentally derived. Fish biota to sediment accumulation factors (BSAF) were used to estimate fish tissue concentrations of dioxins and furans. Fish bioconcentration factors (BCF) were used for the metals with the exception of mercury, which was not quantitatively assessed. Uncertainties in these factors are a primary source of uncertainty for the fish pathways. The uncertainty associated with these bioaccumulation and bioconcentration factors depends on a variety of factors such as lipid content of various fish and stream conditions. This uncertainty can range up to two orders of magnitude for the BSAF for 2,3,7,8-TCDD (EPA, 1993c).

c.4. Exposure Factors

Most exposure assumptions used in the analysis were standard EPA default assumptions. There will be no attempt to quantify the uncertainty involved in the use of standard EPA default assumptions in this document. Examples of standard EPA default assumptions are body weight, inhalation rate, lifetime, and exposure frequency.

For exposure duration and intake rates, a systematic evaluation was conducted to find values which would best represent the range of exposures likely. Central tendency and high-end values were used to capture the range of uncertainty involved in quantifying exposure duration. Central tendency intake rates were used predominantly; however, the range of uncertainty was addressed somewhat through the variation of intake rates among scenarios. For instance, child scenarios were developed to address a child's increased consumption rates of milk and soil. Two fisher scenarios were developed with increased fish intake rates. Several farmer scenarios were developed to capture

the potential range of consumption of a variety of farm commodities including beef, pork, poultry, milk and produce. Consumption of various commodities can be up to a factor of 100 or more between scenarios.

c.5. Overall Uncertainty

Based on the approach used in this analysis, the overall uncertainty in risk estimates due to parameter uncertainty and variability can be interpreted from Table IV.21 for dioxin/furan emissions. For both central tendency and high-end estimates the range of risks for incinerators and cement kilns is approximately three order of magnitude. This would primarily reflect the differences between the cases. The differences include facility characteristics and environmental factors. Lightweight aggregates have a smaller range of risk estimates, however, only two cases were included in the analysis for this source categories. The difference between the median value for central tendency estimates and the median value for high-end estimates is less than two orders of magnitude for incinerators and cement kilns. This comparison is more reflective of the differences in high-end parameter values such as emission rates and exposure factors. Again for lightweight aggregate kilns, the difference between the central tendency and high-end median values is much less--a factor of three.

V. Ecological Risk

A. Technical Approach

To assess the possible effects of emissions from hazardous waste burning combustion facilities on freshwater aquatic organisms and associated wildlife, chronic water quality criteria were identified and compared to estimated surface water concentrations. Water quality criteria were identified in three primary sources: (1) the National Ambient Water Quality Criteria documents, (2) the *Final Water Quality Guidance for the Great Lakes Systems: Final Rule* (60 *FR* 15366), and (3) *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision* (Suter and Mabrey, 1994).

National Ambient Water Quality Criteria, or NAWQC, are developed according to the methods presented in the *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses* (Stephan et al., 1985). The chronic NAWQC are water concentrations that are intended to prevent significant toxic effects in chronic exposures to freshwater organisms. The NAWQC are applicable or relevant and appropriate requirements (ARARs) and, therefore, provide a strong basis for assessing the effect of a particular chemical on freshwater aquatic life. Briefly, the development of an NAWQC requires acute toxicity data representing seven taxonomic families (e.g., a fish from the family salmonids) and chronic toxicity data for at least three of the seven families, including an acutely sensitive freshwater species (see Figure V.1)

Figure V.1 Data Requirements for FCV Calculation

- The family Salmonidae in the class Osteichthyes
- One other family (preferably a commercially or recreationally important warmwater species) in the class Osteichthyes (e.g., bluegill, channel catfish)
- A third family in the phylum Chordata (e.g., fish, amphibian)
- A planktonic crustacean (e.g., a cladoceran, copepod)
- A benthic crustacean (e.g., ostracod, isopod, amphipod)
- An insect (e.g., mayfly, dragonfly, damselfly, stonefly, midge)
- A family in a phylum other than Arthropoda or Chordata (e.g., Rotifera, Annelida, Mollusca)

The chronic NAWQC are typically based on one of three benchmarks for aquatic life: the Final Chronic Value (FCV), the Final Residue Value (FRV) for constituents that bioaccumulate, or the Final Plant Value (FPV). The lowest concentration among the FCV, FRV, and FPV is referred to as the Criterion Continuous Concentration (CCC) and becomes the NAWQC for a particular chemical. However, because the FPV has never been the basis of a NAWQC and the FRV is only used for chemicals that bioaccumulate, the basis for the CCC is usually the FCV. The constituents for which chronic NAWQC were available included: lead, nickel, antimony, arsenic, cadmium, chromium III and VI, and selenium.

When data are insufficient to develop an NAWQC, an alternative method developed by EPA in support of water quality criteria for the Great Lakes Water Quality Initiative, can be used. The method, referred to as Tier II in the final rule (60 *FR* 15366), allows for the calculation of a secondary chronic value (SCV) for data sets that do not fulfill all of the requirements of the NAWQC. Because of the importance of the FCV and SCV calculations for criteria development, a brief explanation is provided below.

The FCV is calculated in one of two ways. If acceptable chronic toxicity data are available on at least one species representing the seven different requirements in Figure V.1, the FCV is the concentration corresponding to a cumulative probability of 0.05 for these species. If the chronic toxicity data do not meet the seven general requirements, the FCV is calculated by: (1) calculating a final acute value (FAV) in the same manner described for chronic toxicity data, (2) estimating an acute-to-chronic ratio (ACR) as the ratio of at least three comparable (e.g., same species) acute and chronic toxicity studies, (3) dividing the FAV by two, and (4) dividing the FAV/2 by the ACR. It is important to note that this description is a simplification of the actual methods and does not address many of the nuances of study selection and data interpretation. For example, if multiple chronic studies are available on the same species, the geometric mean (i.e., Species Mean Chronic Value, or SMCV) is calculated because the distribution of sensitivities of individual species within a genus are more likely to be lognormal than normal (Stephan et al., 1985).

The SCV is calculated in essentially the same way. However, because the minimum data set only requires data from one to seven genera, the SCV is always calculated from a secondary acute value (SAV). The SAV is calculated in the same way as the FAV and divided by the adjustment factor (AF) appropriate to the data set as presented in Table V.1. The adjustment factors are based on the work of Host et al. (1991) and reflect the uncertainty in deriving criteria with limited data sets. This value (i.e., SAV/AF) is then divided by an ACR or the default ACR of 18 to estimate the SCV. The Tier II methodology was designed to generate SCVs that are below the FCVs (for a complete data set) with a 95 percent confidence limit. Secondary chronic values for constituents lacking NAWQC were identified in Suter and Mabrey (1994). The constituents for which SCVs were the only available benchmark included silver, thallium, barium, and beryllium.

Table V.1 Adjustment Factors (Daphnid Data Required)

Sample Size (number of FCV data requirements fulfilled)						
1	2	3	4	5	6	7
21.9	13.0	8.0	7.0	6.1	5.2	4.3

Dioxin has been shown to bioaccumulate appreciably in aquatic food chains and, therefore, presents significantly higher risks to wildlife that consume fish and other aquatic organisms than to aquatic biota. Consequently, water quality criteria based on the bioaccumulation potential of dioxin was identified in the *Final Water Quality Guidance for the Great Lakes System* (GLWQI hereafter) and supporting documents (e.g., *Water Quality Guidance for the Great Lakes System: Supplementary Information Document (SID)* - U.S. EPA, 1995i). The water quality criteria for Dioxin is based on the noncancer risk paradigm for human health risk assessment and require data on: (1) benchmarks for reproductive effects in mammals and birds, (2) measured or predicted bioaccumulation factors (BAFs),* and (3) the body weight and dietary habits of wildlife. Once these data are assembled, they are entered into the following equation:

$$WV = \frac{\dfrac{TD}{UF_A \times UF_S \times UF_L} \times Wt}{W + \sum (F_{TLi} \times BAF_{TLi}^{WL})} \qquad \text{(V-1)}$$

where

WV	=	wildlife value (i.e., criterion), mg/L
TD	=	test dose, mg/kg-day; either a NOAEL or LOAEL
UF_A	=	uncertainty factor for cross-species extrapolation
UF_S	=	uncertainty factor for subchronic to chronic exposures
UF_L	=	uncertainty factor for LOAEL-to-NOAEL extrapolation
Wt	=	average body weight of wildlife species, kg
W	=	average daily water intake, L/day
F_{TLi}	=	averaged daily food intake from trophic level i, kg/day
BAF^{WL}_{TLi}	=	bioaccumulation factor for wildlife food in trophic level i, L/kg.

* A complete description of how bioaccumulation factors were developed for the Great Lakes analysis may be found in *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors* (U.S. EPA 1995a).

The water quality criteria and sources for each of the constituents of concern are presented in Table V.2. It is important to note that for many of the metals of concern, water concentration is a function of water hardness. Hardness is influenced by variations in pH, alkalinity, dissolved carbon dioxide, and dissolved solids. For the purposes of the water quality criteria, hardness is represented by the total concentration of ions of the alkaline earth, expressed as parts per million of calcium carbonate. A hardness of 100 mg/L $CaCO_3$ is supported by EPA to calculate the NAWQC for those metals with hardness-dependent criteria. The range of NAWQC varies up to a factor of two as a function of the water hardness. For facility locations with different water hardness properties, site-specific criteria can be calculated at different hardness levels using the following formula (criteria in µg/L):

lead NAWQC = $e^{(1.273[\ln(\text{hardness})] - 4.705)}$
nickel NAWQC = $e^{(0.846[\ln(\text{hardness})] + 1.1645)}$
cadmium NAWQC = $e^{(0.7852[\ln(\text{hardness})] - 3.490)}$
chromium III NAWQC = $e^{(0.819[\ln(\text{hardness})] + 1.5161)}$

Table V.2. Chronic Water Quality Criteria for Constituents of Concern (mg/L)

Constituent	Water Quality Criteria	Source	Water Quality Criteria	CF	Basis
2,3,7,8-TCDD-TEQ	3.1E-12	GLWQI	NA	-	bioaccumulation
Lead	3.2E-03	NAWQC	2.5E-03	0.791	FCV
Nickel	1.6E-01	NAWQC	1.6E-01	0.997	FCV
Silver	3.6E-04	Suter & Mabrey, 1994	NA	-	SCV
Thallium	1.8E-02	Suter & Mabrey, 1994	NA	-	SCV
Antimony	3.0E-02	NAWQC	NA	-	FCV
Arsenic	1.9E-01	NAWQC	1.5E-01	1.000	FCV
Barium	3.8E-03	Suter & Mabrey, 1994	NA	-	SCV
Beryllium	5.1E-03	Suter & Mabrey, 1994	NA	-	SCV
Cadmium	1.1E-03	NAWQC	1.0E-03	0.909	FCV
Chromium VI	1.1E-02	NAWQC	1.1E-02	0.962	FCV
Chromium III	2.1E-01	NAWQC	1.8E-01	0.860	FCV
Selenium	5.0E-03	NAWQC	4.6E-03	0.922	FCV

GLWQI	=	Great Lakes Water Quality Initiative
FCV	=	Final Chronic Value
NA	=	Not Available
NAWQC	=	National Ambient Water Quality Criteria
SCV	=	Secondary Chronic Value

In addition to hardness considerations, it is also important to recognize the distinction between total and dissolved metals concentration. It is now EPA policy to use dissolved metal to set and measure compliance with water quality standards, because dissolved metal more closely approximates the bioavailable fraction of metal in the water column than does total recoverable metal (60 *FR* 15366). As a result, the Agency has developed conversion factors (CF) to estimate dissolved water quality criteria from total water quality criteria. The dissolved water quality criteria and conversion factors are also shown in Table V.2. Detailed information on the methods developed to convert total metal concentrations to dissolved concentrations may be found in *Final Water Quality Guidance for the Great Lakes System* (60 *FR* 15366) and supporting documents (e.g., *Water Quality Guidance for the Great Lakes System: Supplementary Information Document (SID),* U.S. EPA, 1995i or, *Derivation of Conversion Factors for the Calculation of Dissolved Freshwater Aquatic Life Criteria for Metals,* Stephan, 1995.

B. Results

Tables V.3 and V.4 present the ratio of total and dissolved water concentrations, respectively, to the predicted surface water concentrations for incinerators, cement kilns, and lightweight aggregate kilns. Only a subset of constituents could be evaluated on the basis of dissolved concentrations because conversion factors are available for only 8 of the 12 metals of concern. With the exception of dioxin, all constituents from the various combustor sources were present in the water column at concentrations significantly below the total water quality criteria. No dissolved water concentrations were significantly above the dissolved water quality criteria. Total water column concentrations of dioxin exceeded the water quality criterion for the maximum high-end scenarios for incinerators (HQ = 10) and cement kilns (HQ = 100) and for the maximum central tendency scenario for cement kilns (HQ = 9). It should be emphasized that the water quality criteria are based on exposures to piscivorous wildlife that feed primarily on fish from the contaminated surface waterbodies.

Table V.3 Ratios of Total Water Column Concentrations and Water Quality Criteria for Protection of Aquatic Organisms and Other Wildlife

Constituents	Total Water Criteria	Incinerators				Cement Kilns				Light Weight Aggregate Kilns			
		Central Tendency		High-End		Central Tendency		High-End		Central Tendency		High-End	
		Low	High	Low	High	Low	High	Low	High	Low	High	Low	High
2,3,7,8-TCDD -TEQ	3.1E-12	7.7E-3	4.2E-1	1.8E-1	1.3E + 1	1.2E-1	9.4	1.2	9.7	3.2E-3	1.5E-1	3.2E-3	1.5E-1
Lead	3.2E-3	2.5E-6	1.8E-4	5.0E-5	3.8E-3	1.7E-5	1.4E-3	2.3E-4	2.0E-2	1.2E-6	5.0E-5	4.1E-5	1.7E-3
Nickel	1.6E-1	2.9E-7	1.8E-3	2.8E-6	1.8E-2	2.3E-6	2.4E-4	1.2E-5	1.3E-3	5.7E-7	2.1E-4	3.4E-6	1.3E-3
Silver	3.6E-4	1.2E-5	1.0E-1	1.1E-4	9.4E-1	2.7E-4	3.1E-2	2.1E-3	2.4E-1	8.6E-6	3.3E-3	4.4E-5	1.7E-2
Thallium	1.8E-2	3.3E-7	1.2E-3	2.9E-6	1.1E-2	1.1E-5	1.1E-3	1.2E-4	1.2E-2	1.4E-7	4.9E-5	3.9E-7	1.4E-4
Antimony	3.0E-2	7.0E-7	6.0E-3	2.8E-5	2.4E-1	6.0E-6	6.7E-4	1.1E-4	1.2E-2	7.7E-7	2.8E-4	4.3E-6	5.7E-4
Arsenic	1.9E-1	4.1E-8	2.4E-4	4.5E-7	2.4E-3	4.5E-7	5.0E-5	2.8E-6	2.9E-4	3.3E-8	1.2E-5	2.4E-7	2.5E-4
Barium	1.0	1.9E-8	1.5E-6	1.8E-7	1.5E-5	3.1E-7	2.6E-5	2.8E-6	2.3E-4	1.9E-8	9.7E-7	1.6E-7	7.9E-6
Beryllium	5.1E-3	1.4E-7	1.1E-5	7.1E-7	7.5E-5	8.2E-7	7.3E-5	2.5E-6	1.9E-4	1.5E-7	1.2E-5	5.7E-7	4.1E-5
Cadmium	1.1E-3	1.5E-5	1.9E-2	2.4E-4	5.6E-1	4.2E-4	3.5E-2	3.7E-3	3.1E-1	1.5E-5	5.5E-3	1.4E-4	4.8E-2
Chromium VI	1.1E-2	4.1E-7	2.6E-3	7.3E-6	4.7E-2	4.4E-6	4.6E-4	3.5E-5	3.7E-3	2.8E-7	1.0E-4	1.5E-6	5.3E-4
Chromium III	2.1E-1	6.2E-9	4.4E-7	3.8E-8	2.7E-6	3.7E-10	3.1E-8	5.7E-8	7.6E-6	2.0E-8	9.0E-7	4.8E-8	2.1E-6
Selenium	5.0E-3	7.2E-7	5.8E-3	5.4E-6	4.4E-2	6.0E-5	6.6E-3	5.2E-4	5.6E-2	2.4E-7	8.8E-5	2.0E-6	7.6E-4

Table V.4. Ratios between Dissolved Water Concentrations and Water Quality Criteria for Protection Aquatic Organisms

Constituent	Dissolved Water Criteria	Incinerators				Cement Kilns				Light Weight Aggregate Kilns			
		Central Tendency		High-End		Central Tendency		High-End		Central Tendency		High-End	
		Low	High	Low	High	Low	High	Low	High	Low	High	Low	High
Lead	2.5E-3	8.3E-7	5.9E-5	1.6E-5	1.2E-3	5.5E-6	4.7E-4	7.5E-5	6.3E-3	3.8E-7	1.7E-5	1.3E-5	5.5E-4
Nickel	1.6E-1	2.9E-7	1.8E-3	2.8E-6	1.8E-2	2.3E-6	2.4E-4	1.2E-5	1.3E-3	5.7E-7	2.1E-4	3.4E-6	1.3E-3
Arsenic	1.5E-1	5.1E-8	3.1E-4	5.7E-7	3.1E-3	5.7E-7	6.3E-5	3.6E-6	3.7E-4	4.1E-8	1.5E-5	3.0E-7	3.1E-4
Cadmium	1.0E-3	1.7E-5	2.1E-2	2.6E-4	6.2E-1	4.6E-4	3.8E-2	4.1E-3	3.4E-1	1.7E-5	6.1E-3	1.5E-4	5.3E-2
Chromium VI	1.1E-2	4.3E-7	2.8E-3	7.6E-6	5.0E-2	4.6E-6	4.9E-4	3.7E-5	3.9E-3	3.0E-7	1.0E-4	1.5E-6	5.5E-4
Chromium III	1.8E-1	2.0E-10	1.5E-8	1.3E-9	9.2E-8	1.2E-11	1.0E-9	2.0E-9	1.7E-7	7.1E-10	3.0E-8	1.6E-9	2.4E-6
Selenium	4.6E-3	7.8E-7	6.3E-3	5.9E-6	4.8E-2	6.5E-5	7.2E-3	5.7E-4	6.1E-2	2.6E-7	9.6E-5	2.2E-6	8.3E-4

Table V.5 presents ratios for the various MACT options for dioxin. For cement kilns, the estimated total water column concentration exceeds the total water quality criteria for all MACT options. For incinerators and lightweight aggregate kilns, the beyond the floor options brought the total water column concentration below the total water quality criteria.

As previously stated, the dioxin water quality criterion for the protection of wildlife was developed for the Great Lakes System Final Rule. The dioxin criterion was based on an exposure scenario in which piscivorous mammals (i.e., mink and otter) in the Great Lakes ecosystem are exposed to contaminated fish and surface water. Although ingestion of contaminated drinking water is considered in the calculation, the contribution to risk is negligible compared to the food chain exposure. The wildlife values (WV) for these receptors are analogous to the reference dose (RfD) for noncancer effects in humans; adverse effects are assumed not to occur below the WV threshold. The WV for the mink and otter were derived from a multigenerational study on reproductive endpoints in Sprague-Dawley rats (Murray et al., 1979, as cited in U.S. EPA, 1993e). The reproductive effects observed in Murray et al. (1979) were: (1) significantly decreased litter sizes in the f_0 generation, (2) reduced gestational survival in the f_2 generation, (3) decreases in postnatal body weight in the f_2 and f_3 litters, and (4) reduced reproductive capacity in the f_1 and f_2 generations. Based on the results of this study at daily intake rates of 0, 0.001, 0.01, and 0.1 µg/kg-day, a NOAEL of 0.001 µg/kg-day and a LOAEL of 0.01 µg/kg-day were determined (U.S. EPA, 1993e). The criterion is the geometric mean of water quality concentrations calculated for each receptor assuming that the diet of the mink and otter consists almost exclusively of fish. The dietary habits of the mink and otter were used to select BAFs to match the trophic level of the fish assumed in their respective diets. For example, mink were assumed to ingest only trophic level 3 (smaller) fish. Based on the biological significance of these endpoints to reproducing populations of animals, and the notable bioaccumulation potential of TCDD and congeners, it is likely that exceedance of the water quality criteria may result in long-term ecological impacts on exposed wildlife.

Table V.5 Ratios of Total Water Column Concentrations for Various MACT Options for Dioxins

Facility Type	Central Tendency		High End	
	Low	High	Low	High
Baseline				
Incinerators	0.008	0.4	0.2	10
Cement kilns	0.1	9	1	100
Lightweight aggregate kilns	0.003	0.2	0.003	0.2
Proposed Floor - Existing and New Sources				
Incinerators	0.008	0.4	0.1	7
Cement kilns	0.05	4	0.2	20
Lightweight aggregate kilns	0.02	0.8		
Proposed BTF - Existing and New Sources				
Incinerators	0.008	0.4		
Cement kilns	0.05	4		
Lightweight aggregate kilns	0.02	0.8		
Alternative Floor - Existing Sources				
Incinerators	0.005	0.3		
Cement kilns	0.03	3		
Lightweight aggregate kilns	0.01	0.5		

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