Human Health and Ecological Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes

Response to Public Comments

July 1999

Office of Solid Waste
U.S. Environmental Protection Agency
Washington, DC 20460

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List of Acronyms

ADD	average daily dose
Ah	aryl hydrocarbon
APA	Administrative Procedure Act
BIF	boilers and industrial furnaces
BTF	beyond-the-floor
BSAF	biota-sediment accumulation factor
CAA	Clean Air Act
CCC	Chlorine Chemistry Council
CKD	cement kiln dust
CMA	Chemical Manufacturer's Association
CO	carbon monoxide
CPF	cancer potency factor
dscf	dry standard cubic foot
EFH	*Exposure Factors Handbook*
EPA	Environmental Protection Agency
GLCs	ground-level concentrations
HAPs	hazardous air pollutants
HCs	hydrocarbons
HCl	hydrogen chloride
HEAST	Health Effects Assessment Summary Tables
HQ	hazard quotient
HWCs	hazardous waste combustors
HWF	hazardous waste feed
HWIs	hazardous waste incinerators
IADD	Intake Average Daily Dose
IEM	Indirect Exposure to Combustor Emissions
IDLH	Immediately Dangerous to Life and Health
IRIS	Integrated Risk Information System
LMS	linear multistage model
LWAKs	lightweight aggregate kilns
MACT	maximum achievable control technology
MEI	maximally exposed individual
MOE	margin of exposure
µg	micrograms
NAAQS	National Ambient Air Quality Standards
NESHAP	National Emission Standards for Hazardous Air Pollutants
ng/dscm	nanogram per dry standard cubic meter
NIOSH	National Institute for Occupational Safety and Health

NOAEL	no observed adverse effect level
NODA	Notice of Data Availability
OSHA	Occupational Safety and Health Administration
PICs	products of incomplete combustion
PM	particulate matter
PM_{10}	particulate matter less than 10 microns in diameter
ppm	parts per million
QSAR	quantitative structure-activity relationship
RCRA	Resource Conservation and Recovery Act
RfC	reference air concentration
RfD	oral reference dose
SAB	Science Advisory Board
SVM	semi-volatile metal
TEF	toxicity equivalency factors
TEQ	toxicity equivalency concentration
TSS	total suspended solids
Vdv	dry deposition velocity

Section 1
Introduction

Hazardous waste combustors (HWCs) emit hazardous air pollutants (HAPs) that are listed under Section 112(b) of the Clean Air Act (CAA). In an effort to control emission from these HAPs, the U.S. Environmental Protection Agency (EPA) proposed National Emission Standards for Hazardous Air Pollutants (NESHAP) pursuant to Section 112 (d) of the CAA that establish emission standards based on application of maximum achievable control technology (MACT). These MACT standards are technology-based standards; they are not risk-based. The facilities affected, however, are also regulated under the Resource Conservation and Recovery Act (RCRA), as amended. Specifically, Sections 3004(a) and 3004(q) require EPA to develop standards that are protective of human health and the environment. To meet the current MACT requirements under the CAA and to satisfy RCRA requirements, EPA conducted a risk assessment to evaluate human health and ecological risks associated with emissions from HWCs.

On April 19, 1996, EPA proposed a rule to revise standards for HWCs—incinerators, hazardous waste–burning cement kilns, and lightweight aggregate kilns (61 FR 17358). The rule was proposed under joint authority of the CAA, as amended, and RCRA, as amended. The methodology used in EPA's risk analysis for the proposed rule was presented in the February 20, 1996, document *Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document.* EPA received comments from the public on the proposed rule until August 19, 1996 (U.S. EPA Docket Number F-96-RCSP-FFFFF).[1] In addition, a notice of data availability was published on August 23, 1996, requesting comment on the report of an external peer review of the risk analysis for the proposed rule (61 FR 43501). EPA received comments on the external peer review until September 23, 1996.[2]

As a result of these public comments, as well as comments submitted by the peer-review panel, EPA modified its risk analysis for the proposed rule in a number of important areas that include:

- Using stratified random sampling to select an additional 66 facilities to represent the universe of hazardous waste facilities (for a total of 76 facilities)
- Expanding combustor categories to include cement kilns, lightweight aggregate kilns, commercial incinerators, large on-site incinerators, small on-site incinerators, incinerators with waste heat recovery boilers, and area sources
- Using facility-specific and site-specific data for site characterization

[1]The comment period for the proposed rule originally ended June 18, 1996, but was extended to August 19, 1996.

[2] EPA's responses to the peer review comments are found in the document *Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments (July 1999) (U.S. EPA,1999b).*

- # Projecting facility-specific emission reductions under MACT controls
- # Conducting a multipathway exposure analysis for all chemical constituents for which adequate emissions data were available
- # Conducting multimedia fate and transport modeling of three mercury species
- # Evaluating risks from inhalation of fine particulate matter (PM)
- # Using Monte Carlo simulation to evaluate variability in exposure parameters for key exposure pathways
- # Using U.S. Census data and Census of Agriculture data to identify the number and location of human receptors, allowing both individual and population-based risks to be better characterized
- # Expanding the number of age groups to four for all receptor populations
- # Evaluating risks to special subpopulations
- # Performing an ecological screening analysis for multiple ecological receptors
- # Characterizing uncertainty in the risk analysis

Details of the modifications to the risk assessment methodology used for the final rule are presented in the document, *Human Health and Ecological Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Document* (July 1999). This document describes the methods used in the risk analysis, documents the data used, and characterizes the human health and ecological risks from HWCs. It also discusses uncertainty associated with data and models.

EPA received 108 submissions from public commenters. (A list of comments by docket number is given in Appendix A.) In these submissions, 39 public commenters addressed risk assessment issues. A number of the commenters submitted additional comments on issues addressed by the peer-review panel. As these comments were received and summarized, individual comments from within the entire submittals were tagged with a unique identification number within the summary database.

This document presents comments from public commenters on the risk assessment for the proposed rule and gives EPA's responses to these comments. It is organized to present comments and responses for each commenter (arranged in alphabetical order) in Sections 2 through 40. Each commenter has a docket identification number included in the section heading. If a commenter made more than one comment on the risk assessment, all such comments are included in the commenter's section (each tagged with the unique comment identification number). Public comments on topics other than the risk assessment are not included here. These include comments on the MACT standards and their rationale, cost-benefit analysis, and EPA's permitting policy concerning site-specific risk assessments. Such comments are addressed in separate response to comment documents.

Section 2
American Cyanamid Company
RCSP-0131

Comment 528: The commenter cites a statement in the preamble to the proposed rule that EPA does not have sufficient reliable data with respect to mercury and nondioxin product of incomplete combustion (PICs) to be able to assess, on a national basis, the magnitude of the risks that can routinely be expected from burning hazardous waste in HWCs. From this the commenter concludes that EPA does not currently know the health effects due to exposure to combustion products from hazardous waste incinerators. Since the EPA has no evidence that the current standards are unprotective, it is premature to set further, more restrictive emission standards. The EPA should wait until a risk assessment is done on these emissions to determine the safe exposure levels. Until the EPA is able to do this, it is setting standards for substances based on very conservative estimates that may or may not be true. The commenter recommends that EPA perform a risk assessment to determine the health effects from exposures to emission products from HWCs.

Response 528: *The establishment of NESHAP for sources that emit HAPs is required by Sections 112(c) and 112(d) of the CAA. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards. However, Sections 3004(a) and (q) of RCRA mandate that standards governing the operation of HWC facilities be protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate whether the MACT standards satisfy this requirement in order to determine what, if any, RCRA standards for emissions from these sources may be needed.*

The risk assessment for the final rule included an analysis of risks from mercury. Although EPA assessed the risks from chlorinated dioxins and furans, risks from other organics that may be present as PICs could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited, are highly variable, and are therefore inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of the rule, permitting authorities

will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

Section 3
American Industrial Health Council
RCSP-0239

Comment 622: The commenter supports the use of peer review in the development of environmental regulations. In recent comments on the draft report of the President's Commission on Risk Assessment and Risk Management, the commenter agreed with the Commission's finding regarding the importance of peer review in regulatory decision-making. The commenter believes that the risk analysis peer review offers a valuable critique of the risk assessment for the HWC rule.

> ***Response 622:*** *EPA is committed to the use of the peer review process for major scientific studies and technical analyses used in decision-making. EPA has incorporated comments from peer reviewers in the risk analysis for the final rule and has prepared a response to comments document on the peer review.*

Comment 268: EPA's risk assessment evaluates many exposure scenarios for specific subpopulations, such as subsistence farmers, who may have potentially higher exposures than the typical populations surrounding hazardous waste facilities. However, the peer review of the HWC risk assessment review is critical of EPA's analysis because it does not address the likelihood of the exposures occurring or the number of people who might be affected or the uncertainty introduced. The commenter cites a comment from the peer review, which indicates that, because risk estimates for special subpopulation exposure scenarios are highly sensitive to the assumed location of exposure, it is critical that the actual location of special subpopulations be accurately identified. The commenter recommends that the evaluation of potentially highly exposed subpopulations include a discussion of plausibility and likelihood that these populations exist in the situations being evaluated. EPA should reconsider this aspect of the risk assessment. The commenter recommends that high-end exposures be used only in site-specific screening analyses unless evidence is available to document that such exposures actually exist.

> ***Response 268****: The risk assessment for the final rule was modified to account for the number of individuals exposed and their location. The analysis used U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. However, it was not possible from census data to identify and locate individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios were retained in the risk analysis for the final rule. Although it is*

not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. For this purpose, EPA assumed that subsistence farming takes place within certain prescribed distances of HWCs (i.e., within 2, 5, 10, and 20 kilometers). EPA also assumed that subsistence fishing takes place at each body of water that was modeled in the risk analysis. EPA recognizes that these assumptions may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

The same food intake rates were used for subsistence farmers as were used for farmers engaged in farming for commerce. These rates were derived from data collected on consumption of home-produced foods from the U.S. Department of Agriculture's Nationwide Food Consumption Survey (as cited in the 1997 Exposure Factors Handbook*, U.S. EPA, 1997a). For subsistence fishing, EPA used fish intake rates representative of Native American tribes from the Columbia River basin, who obtain a significant portion of their dietary intake from fish. The use of exposure factors derived from such studies ensures the plausibility of the exposure scenarios EPA used to characterize risks to individuals who may be engaged in subsistence activities.*

***Comment 269*:** The commenter believes that analyses other than screening should incorporate all reasonable, available, and applicable data. In this instance, EPA should reevaluate the appropriateness of, and the rationale for, the defaults used. Moreover, the risk analysis should incorporate site-specific or regional data where appropriate for risk assessment beyond screening analyses and develop guidance or criteria for generating or selecting such data.

Response 269: *EPA agrees with the commenter that site-specific data are generally preferred to the use of default values. In the risk assessment for the final rule, EPA used site-specific information whenever possible. Facility data were used for characterizing emissions and the conditions of stack release. Air modeling employed meteorological data from the nearest weather station and site-specific terrain and land use data. Exposed populations were located using a combination of block-level U.S. Census data and county-level Census of Agriculture data. Bodies of water selected for analysis were characterized using stream gauging and other site-specific data. Watersheds were delineated using site-specific topographic data and were further characterized using site-specific soils and land use data.*

However, assessing human exposures requires that certain assumptions be made about the pathways of exposure. For example, EPA assumed that dairy farm households consume home-produced milk. Contact rates, such as consumption rates for home-produced foods, are based on national surveys of populations that resemble the receptor population being assessed. Such data were used to establish national default

values for human intake rates in lieu of site-specific or regional data. EPA believes it is necessary and appropriate to use national-level data such as these for characterizing exposures in the HWC risk analysis.

Comment 624, 1076: In their comments, some of the peer reviewers questioned the air-plant-animal pathway used in the analysis of dioxin/furan emissions. There is a paucity of field data confirming this pathway and certainly many unknowns concerning the mathematical representation of this largely speculative exposure route. The commenter cites peer reviewers' comments that the greatest weakness in modeling the deposition of airborne emissions is the lack of field data from a representative cross-section of locations to thoroughly validate the vapor particle partitioning, deposition, and air-to-plant transfer models. The commenter also cites peer reviewers' comment that empirical relationships used to describe intermedia transfer factors are derived from highly limited data sets and that data gaps and model-related uncertainties introduce large uncertainties in the risk estimates. The commenter strongly recommends that this air-plant-animal exposure pathway not be used until the pathway is better validated and multiple uncertainties are reduced.

The commenter notes that air-to-plant transfer factors are calculated using an algorithm derived from studies in azalea plants by Bacci et al. (1990, 1992) on a very limited number of chemical constituents. Only 10 organics were examined in the first study and an additional four in the second. Thus, the air-to-plant transfer algorithm proposed for use in the methodology reflects a correlation equation based on only a handful of chemicals. The commenter questions the scientific validity of using this approach to calculate transfer for the multitude of chemicals which fall into very diverse chemical classes addressed in indirect risk assessments.

Response 624, 1076: *EPA believes there is ample data to suggest that the air-to-plant-to-animal pathway is an important human exposure pathway. In its review of the 1994 draft dioxin reassessment, EPA's Science Advisory Board agreed with this assessment, saying that the air-to-plant-to-animal pathway is most probably the primary way in which the food chain is impacted and humans are exposed. While there continue to be uncertainties in the model, evidence suggests that dioxin levels in meat and dairy products are largely attributable to deposition of air emissions to plants used as feed for livestock. EPA has performed model validation studies that indicate the model is a reasonable predictor of dioxins in beef. Although additional studies could help further refine the models, EPA believes there are currently sufficient evidence and data available to evaluate exposures from the air-plant-animal pathway and that it is important to assess the risks attributable to such exposures.*

EPA did not use the Bacci algorithm in the HWC risk analysis. Instead, air-to-leaf factors were derived from empirical data on the uptake of chlorinated dioxins and furans in pasture grass. See Lorber (1995) "development of an air-to-leaf vapor phase transfer factor for dioxins and furans."

Comment 644: The commenter notes that many of the HWC risk assessment peer reviewers' comments were directed toward specific assumptions made in the dioxin/furan risk analysis. The reviewers noted that the risk contractor generally followed EPA default assumptions but questioned the appropriateness of the assumptions and the lack of explanation of the defaults selected. In particular, the commenter cites a reviewer who indicated that despite all refinement in the construction of case-study-specific exposure pathways, the critical variables— i.e., contaminant concentrations and exposure assumptions (frequency and duration of exposure)—are high-end conservative default EPA assumptions, respectively. The commenter cites another reviewers who indicated that calculations include numerous standard "exposure factors," many of which do not represent the central tendency of the normal human variance, and other assumptions that are far from the median. Finally, the commenter cites a third peer reviewer who noted that the case study approach to characterizing individual risk does reduce the number of generic, default, worst-case assumptions that have to be made in the risk assessment process. However, this approach is still conservative in that the methodologies and assumptions used to characterize chemical emissions, fate and transport, exposure, and toxicity all lead to the overestimation of potential risk.

Response 644: *Regarding the peer review completed for the proposed rule, EPA gave much consideration to the comments of the peer-review panel. In response to these comments, EPA made a number of changes to the risk assessment in order to improve the overall representativeness of the assessment.*

For the final rule, EPA did not use a case study approach and instead used stratified random sampling to select a subset of facilities from the HWC universe for risk characterization. Each of these selected facilities was modeled using site-specific data when available. Using stratified sampling in selecting facilities allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe they are designed to (e.g., confidence intervals). Such statements could not be made using the case study approach that was used at proposal.

In the risk assessment for the final rule, EPA used site-specific parameter values whenever possible. These included facility-specific parameters such as emissions used to estimate media concentrations and concentrations in agricultural products and site-specific parameters such as soils and land use. However, for agricultural parameters EPA used default values derived as explained in the risk assessment Background Document (RTI, 1999). Human exposure factors such as fish ingestion rates were set to central tendency values except in those instances where EPA used Monte Carlo simulation. In setting exposure factors, EPA used mean values obtained from the 1997 Exposure Factors Handbook *(U.S. EPA,1997a). Although mean values are higher than the 50th percentile, mean values are necessary for estimating population risk and, therefore, were used throughout the risk analysis. EPA agrees that combining several high-end*

parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. This is why, for the final rule, EPA did not use high-end values. In all instances, EPA either used central tendency values or conducted a full distributional analysis to account for the full range of exposure.

The commenter is referred to the risk assessment background document for the final rule for a discussion of the analytical framework and a complete description of the methods used for modeling exposures. Also, EPA has prepared a separate response to comments document entitled Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *that addresses the peer review comments (U.S. EPA, 1999b). The commenter is referred to this document for EPA's response to the issues raised by the peer reviewers.*

Section 4
American Lung Association
RCSP-0126

***Comment 71*:** EPA's narrative on the health benefits of the proposed standards and the health risk assessment do not address the benefits of PM emissions or the health impact of PM emissions from waste combustion sites. This is a deficiency that is most significant for cement plants. PM exposures around cement plants occur as a result of main stack emissions, fugitive plant emissions, and fugitive cement kiln dust (CKD) handling and disposal emissions. The commenter cites a February 7, 1995, *Federal Register* notice as evidence of possible risk to human health due to the fine particulate nature of inhaled dust. The notice indicates that windblown dust from uncontrolled CKD waste management units could potentially exceed EPA's health-based fine particulate (10 microns or less) National Ambient Air Quality Standard (NAAQS) at nearby residences. From this, the commenter concludes that it is reasonable to assume that there are some geographical receptor sites around cement plants and their dust (CKD) disposal piles that presently violate the current NAAQS for PM_{10}, solely from the influence of CKD pile fugitive emissions. It is also reasonable to assume that stack emissions of PM_{10} can make an additional contributions to PM exposures over and above the contribution from CKD piles. Given this situation and the potential of main stack particulate emissions to increase particulate ambient concentrations around cement plants, the risk analysis should have included considerations of PM exposure in its consideration of the health risk assessment and the human health benefits of the proposed standard.

Exposure to inhalable PM has been associated with increased morbidity and mortality, increased rates of asthma attack, increased rates of hospitalization of respiratory disease patients, and aggravation of other pre-existing respiratory diseases at ambient concentrations that are well below EPA's current NAAQS for PM_{10}. American Thoracic Society, the medical arm of the American Lung Association, the commenter cites the Epidemiologic studies have consistently provided evidence of adverse health effects of these air pollutants. Particulate and sulfur oxide (SO) pollution were strongly implicated in the acute morbidity and mortality associated with the severe pollution episodes in Donora (Pennsylvania), London, and New York in the 1940s, 1950s, and 1960s. New evidence associates even current ambient levels of PM_{10} (30 to 150 micrograms/m^3) with increases in daily cardiorespiratory mortality and in total mortality, excluding accidental and suicide deaths. These associations have been shown in many different communities, as widely different in particle composition and climate as Philadelphia, St. Louis, Utah Valley, and Santa Clara County, California. It has recently been shown in a long-term prospective study of adults in the United States that chronic levels of higher PM-10 pollution are associated with increased mortality after adjusting for several individual risk factors. Daily fluctuations in PM_{10} levels have also been shown to be related to acute respiratory hospital admissions in children, to school and kindergarten absences, to decrements in peak-flow rates in normal children, and to increased medication use in children and adults with asthma.

The commenter cites a study by Dockery and Pope that recently reviewed the epidemiologic literature. This review suggests that the epidemiologic studies of adverse morbidity measures are coherent with the mortality studies showing quantitatively similar adverse effects of acute exposures to particulate pollution. (Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society, 1996, "Health Effects of Outdoor Air Pollution," *American Journal of Respiratory & Critical Care Medicine* 153:3-50.)

The proposed rule would drop permissible PM emissions from 0.08 grains/dscf to 0.03 grains/dscf for many of the cement plants regulated by the rule. If comments of the ALA are reflected in the final rule, permissible particulate emissions would be reduced even further. Accordingly, the risk analysis should have evaluated the PM_{10} exposure issue in both its health risk assessment and in derivation of the benefit of the proposed rule.

Response 71: *EPA agrees with the commenter concerning the health effects of fine PM. EPA has recently revised its ambient air quality standards for PM and has put in place a national strategy for implementing the new standards. In addition, EPA is developing a regulatory program for the safe management of CKD (see 60 FR 7366). For HWCs, EPA has set technology-based emission standards using the MACT process, as explained in the preamble to the final rule. These include standards for PM. In developing the final rule, EPA examined the potential for reducing risks from ambient PM by controlling HWC stack emissions. In particular, EPA estimated the reductions in risk associated with a number of health endpoints, including:*

- *Mortality from long-term exposure and from short-term exposure*
- *Hospital admissions for all respiratory infections, congestive heart failure, and ischemic heart disease*
- *Chronic bronchitis*
- *Acute bronchitis, lower respiratory symptoms, and upper respiratory symptoms*
- *Work loss days, minor restricted activity days, and restricted activity days*

The results of the analysis indicate that risk reductions are expected for a number of health endpoints, including reductions in mortality, hospital admissions, chronic bronchitis, and respiratory symptoms, although the reductions are modest relative to those achieved by controls on other sources of PM.

Comment 431: The inhalation pathway shown in the health risk assessment does not account for re-entrainment of solid-phase HAPs as a contribution to community exposures. Re-entrainment of solid-phase pollutants involves subsequent reintroduction of solid-phase, dry deposited HAPs by the action of wind scouring. Re-entrainment of HAPs should be evaluated as a contributor to direct inhalation exposures.

Response 431: *EPA believes that direct inhalation risks from HWC stack emissions are much greater than any risks associated with inhalation of re-entrained PM resulting from wind erosion. This consistently has been shown to be the case in studies that have examined re-entrainment of stack emissions deposited to soils. Therefore, for the final rule EPA did not evaluate this exposure pathway. However, this does not mean EPA has reached the same conclusion regarding re-entrainment of PM from other sources, such as fugitive dust emissions from the management of CKD. EPA is currently evaluating the risks from exposures to CKD in a separate rulemaking effort (see 60 FR 7366).*

Comment 433: The risk analysis assumes only a 30-year exposure for direct inhalation. However, waste burning may continue at some sites for longer periods given the history of cement production at many existing sites; there is no reason to assume that an upper-bound risk assessment should not include a 70-year lifetime risk.

Response 433: *The duration of exposure to a contaminant is directly related to the period of time an individual resides at a given site. This period of time is often considerably shorter than the operating life of a facility. Information on population mobility and residence time indicate that for most sectors of the U.S. population, the likelihood of anyone residing at the same location for 70 years is relatively small. For example, Census Bureau data show that over half of all households have lived in their current residence for 9 years or less and only 1 percent have lived in their current residence for more than 50 years.*

For the final rule, EPA relied on data reported in the 1997 Exposure Factors Handbook *(EFH) (U.S. EPA,1997a) on total time in the same residence to determine the duration of exposure. For nonfarm households, EPA used a residence time of 6.5 to 13.5 years, depending on an individual's age. These estimates were taken from a study that used a mathematical model to simulate the probability over time that an individual would either move away or die. The model was based on survey data obtained from the Census Bureau and vital statistics data from the National Center for Health Statistics (as cited in the 1997 EFH). EPA selected mean values from the distribution of total residence time for each age group. For farm households, EPA fitted a distribution to data on total residence time specific to farm households, as reported in the 1997 EFH. From the fitted distribution, it was estimated that 50 percent of adult farmers have a total residence time of 9 years or less, 75 percent a residence time of 19 years or less, 90 percent have a residence time of 40 years or less, and 95 percent have a residence time of 62 years or less. The same percentiles (i.e., 50th, 75th, 90th, and 95th) for farm children ages 0-5 were estimated to be 3, 7, 15, and 23 years, respectively. EPA used these results to estimate the duration of exposure for adults living in farm households that are exposed to dioxins through beef and milk. Due to a lack of data on residence times for children*

living in farm households, farm children were assumed to have the same duration of exposure as children in nonfarm households.

Section 5
Arkansas Department of Pollution Control and Ecology
RCSP-0191

Comment 434: The partial risk assessments conducted as part of this proposed rule are incomplete. EPA cannot decide internally which guidance should be used for risk assessment determinations. At best, the risk analysis performed is a moving target, at worst, it is unrealistic.

Response 434: *The risk analysis for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level. Therefore, the risk methodology may differ from what is typically used in a permitting context. Specifically, EPA did not follow the 1994 draft guidance for performing screening level analyses at hazardous waste combustion facilities. EPA is confident that the revised risk assessment provides a thorough assessment of risks from HWC emissions for which sufficient data were available. These include emissions of chlorinated dioxins and furans, mercury, lead, cadmium, arsenic, chromium, a variety of other metals, hydrogen chloride, chlorine gas, and PM.*

The risk assessment for the final rule is based on methodology presented in the 1990 Indirect Exposure Document, Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IEM) *(U.S. EPA,1990) and the 1993 Addendum to the 1990 Indirect Exposure Document (U.S. EPA,1993a). These methods were further updated with respect to dioxins and mercury. For dioxins, the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist of the procedures published in the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. For mercury, the assessment followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(U.S. EPA,1997c) and used the IEM-2M surface water model developed especially for that report. Human exposure factors were obtained from the 1997* Exposure Factors Handbook *(U.S. EPA,1997a), including consumption rates for home-produced foods and recreationally caught fish. Risks from both cancer and noncancer effects were assessed. For lead, EPA used the Integrated Exposure Uptake Biokinetic Model (IEUBK) (U.S. EPA, 1994c) to estimate blood lead concentrations in children. Risk estimates were made for selected percentiles of the cumulative distribution of individual risks and for population risk.*

In addition to assessing human health risks, EPA also conducted a screening-level ecological assessment. In this approach, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils,

sediments, and surface water. These criteria were then compared to model-predicted media concentrations in order to assess the potential for ecological risk.

Comment 435: EPA fails to speciate mercury while it is well known that certain mercury compounds have minimal health impacts. Guidance indicates that mercury is not to be included in risk assessments.

Response 435: *EPA recognizes that mercury speciation is a critical factor in determining exposure and risks from mercury emissions. Environmental exposures to elemental mercury are most likely to occur through inhalation, whereas exposures to methylmercury are most likely to occur through consumption of fish. Exposure to divalent mercury can occur through a variety of dietary sources. For the final rule, EPA assessed the human health and ecological risks associated with elemental, divalent, and methylmercury. EPA's analysis shows that the most significant exposures come from methylmercury. This is due primarily to deposition and runoff of divalent mercury to surface water and subsequent methylation and bioaccumulation in fish.*

Comment 436: The statement, "With respect to mercury, EPA suspects that there may be significant individual risks..." is totally subjective and without merit. If EPA has data that show this to be the case then put that position forth.

Response 436: *The health effects of mercury are well documented. Clinically observed neurotoxicity has been observed following exposure to mercury. Generally the most subtle indicators of mercury toxicity are neurological changes that include losses of motor skills and sensory ability. Humans, plants, and animals are routinely exposed to mercury, potentially resulting in a variety of ecological and human health impacts. EPA's 1997* Mercury Study Report to Congress *(U.S. EPA,1997c) concluded that exposure to methylmercury through consumption of fish is most important to human health. For the final rule EPA evaluated human health and ecological risks associated with exposure to elemental mercury, divalent mercury, and methylmercury. With respect to methylmercury, the human health evaluation shows that exposures are projected to be below EPA's reference dose. However, the analysis is subject to considerable uncertainty, including the fact that background exposures are not taken into account. Exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing.*

Comment 440: EPA indicates that 11 facility locations (5 cement kilns, 4 incinerators and 2 lightweight aggregate kilns) were evaluated to determine dioxin risks. Further site-specific characteristics such as meteorological conditions, topography stack heights and gas flow rates were also considered. However, stack gas concentrations were derived from national emissions data. This

flies in the face of reason. National emissions data, as mentioned earlier, is severely suspect in its accuracy. To determine potential risk using partial site-specific data and partial generic data invalidates the entire exercise.

> ***Response 440:*** *EPA agrees that site-specific emissions data are preferable to national-level data. For the final rule, the number of facilities analyzed was expanded from 11 to 76. For each facility for which emission measurement data were available, site-specific stack gas concentration data from trial burn and certificate of compliance tests were used in conjunction with stack gas flow rates to estimate emissions. However, in those instances where emissions measurements were not available, stack gas concentrations were imputed from a pool of emissions measurements for other, similar facilities. EPA believes that this approach makes the best use of the available data and improves the representativeness of the analysis.*

Section 6
Cement Kiln Recycling Coalition
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Comment 80, 1066, 1083: The commenter feels that EPA has seriously overestimated risks posed by cement kilns burning hazardous waste, and the commenter objects to EPA's use of its "seriously deficient" indirect exposure models. The commenter states that application of these deficient models is exacerbated by EPA's selection of "an astounding array of extremely conservative assumptions." The commenter states that the manner in which EPA has chosen its case studies and constructed exposure scenarios leads to overly conservative results that characterize the upper tail of the risk distribution by selecting scenarios that go far beyond high-end exposures. The commenter feels that given the conservative choices of default parameters and subsistence subpopulations upon which risk calculations are based, EPA should acknowledge and compensate for the overly conservative nature of the methodology.

Response 80, 1066, 1083: *It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from HWC facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the Addendum to the 1990 Indirect Exposure Document (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA,1994a,b) and the* Mercury Study Report to Congress *(MRTC) (U.S. EPA,1997c), as well as other sources of information.*

For the final rule, EPA made a number of changes to the risk assessment to address commenters' concerns and to improve the representativeness of the analysis. In particular, EPA used central tendency exposure factors for estimating exposures, except for a few risk-driving pathways for which an exposure factor variability analysis using Monte Carlo simulation was performed. In addition, the risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and concentrations in agricultural products. Also, U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in several different types of commercial farming as an occupation or fishing as a recreational sport and persons engaged in farming or fishing for subsistence. All of these modifications were designed to produce exposure estimates that are more

representative of the different receptor populations being modeled with respect to both behavior and location. EPA believes that these factors provide considerable assurance that the exposure estimates are reasonable.

Comment 96: The commenter identified numerous errors in key risk evaluation parameters such as inappropriately varied oxygen correction factors, antiquated dioxin cancer slope factors, and an erroneous mercury reference dose (RfD). Together, these parameters have resulted in an overstatement of baseline and floor level risks posed by these cement plants. The commenter believes EPA greatly exaggerates the risk reduction benefits and overstates the cost effectiveness of the proposed standards.

Response 96*: For the final rule, EPA corrected all errors known to have occurred in the risk analysis for the proposed rule. At proposal, stack gas flow rates (dscfm) were not corrected to 7 percent oxygen for use in calculating mass emissions. The emissions estimates used in the risk assessment for the final rule were based on emission measurement and stack flow rate data that were all corrected to standard conditions of temperature, oxygen, and moisture. Therefore, this error has been corrected. For the final rule, the RfD for methylmercury of 1 x* 10^{-4} *mg/kg-d cited in IRIS (U.S. EPA, 1998a) and EPA's 1997* Mercury Study Report to Congress *(U.S. EPA,1997c) was used. This is the same reference dose that was discussed in the preamble to the proposed rule.*

With regard to the slope factor for 2,3,7,8-TCDD, it is EPA's policy that, until the dioxin reassessment is concluded, EPA will use the cancer slope factor from the 1985 health assessment (Health Assessment Document for Polychlorinated Dibenzo-p-dioxins, *U.S. EPA, 1985). The estimate from that assessment is 156,000 [mg/kg/day]*$^{-1}$*. For the dioxin reassessment, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d). This study lends considerable support to the cancer potency estimate in the 1985 health assessment, as well as the estimate in the 1994 draft reassessment (see* Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds, *U.S. EPA,1994d,e).*

Comment 174: The commenter notes that although EPA seeks control of mercury emissions from HWCs because of their contribution to total national emissions, in the proposed rule and supporting analyses EPA presents limited information on the risks posed by mercury emissions or the benefits of abating these emissions. The commenter states that although EPA presents several varieties of risk and benefit analyses to support the proposed rule, most of these address pollutants other than mercury. Further, the commenter notes that regarding mercury specifically, EPA focuses on human health risks from consumption of fish that have bioaccumulated mercury and cites as evidence the numerous widespread state bans or advisories on fish consumption that involve mercury. The commenter feels that EPA provides no information and makes no claims regarding ecological or

economic damages from mercury and, in discussing the potential human health risks from mercury, provides no information about likely risks from HWC emissions in their immediate vicinity. Instead, EPA adopts only a nationally aggregated perspective, evaluating the nationwide impacts of total mercury emissions from all sources. The commenter states that EPA provides no evidence linking mercury emissions from any HWC to resulting risks at any specific location and points out that mercury is not included in EPA's modeling of the health risks from emissions from HWC facilities.

Response 174: *For the final rule, EPA conducted a quantitative analysis of both the human health risks resulting from exposure to mercury released from HWC facilities as well as an analysis of the potential for adverse impacts to ecological receptors following mercury exposure (i.e., a screening-level ecological analysis).*

The mercury analysis completed for the final rule followed the general modeling approach developed for the 1997 Mercury Study Report to Congress *(U.S. EPA,1997c) and used the IEM-2M surface water model developed especially for that report. The modeling approach tracked three species of mercury (divalent, elemental, and methylmercury) in projecting concentrations for different media compartments. With respect to methylmercury, the human health evaluation shows that exposures are projected to be below EPA's reference dose. However, the analysis is subject to considerable uncertainty, including the fact that background exposures are not taken into account. Exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing.*

The screening-level ecological analysis completed for the final rule evaluated the potential for adverse impacts to ecological receptors. With this approach, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water. These criteria were then compared to model-predicted media concentrations in order to assess the potential for ecological risk. With respect to methylmercury, the ecological analysis shows that surface water concentrations are projected to be below EPA's wildlife criteria for the protection of aquatic wildlife, suggesting a low potential for ecological risk. However, as with the human health evaluation, the analysis is subject to considerable uncertainty, including the fact that background levels were not considered. Therefore, the potential for ecological risk may be higher than the quantitative analysis would suggest.

***Comment 1016*:** The commenter cites the part three of the preamble to the proposed rule, which states that the current state of knowledge concerning the behavior of mercury in the environment does not allow for a meaningful quantitative risk assessment of emission sources that is precise enough to support regulatory decisions at the national level. Specifically, there is insufficient information with

respect to speciation of the mercury into various forms in emissions and with respect to the deposition and cycling of mercury species in the environment to conduct a defensible national quantitative assessment of mercury deposition, erosion to surface waters, and bioaccumulation in fish. The commenter notes that although EPA does not mention it in the preamble, another critical reason for the lack of any quantitative risk analysis for mercury is the great uncertainty regarding the health effects of mercury and disagreement regarding appropriate reference dose for ingestion of the substance. The uncertainty is sufficient to have caused EPA several times to delay submission of the *Mercury Study Report to Congress* pending completion of review of important health effects studies.

Response 1016: *EPA is concerned about exposure to mercury from HWC emissions because mercury is a known neurological toxicant in humans. However, at the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA,1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA,1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from hazardous waste combustors. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

*For the final rule, the concentrations of mercury species in HWC emissions were determined from emissions tests at HWC facilities. Specifically, trial burn and certificate of compliance emissions measurements from the standard stack gas sampling train, EPA Method 29, were used for estimating emissions of mercury species. The EPA Method 29 sampling train was originally developed for quantifying total metals in the stack gas. However, due to the manner in which the mercury is captured (and subsequently analyzed) in the sampling train, it has been proposed that Method 29 measurements can be used to estimate three general forms for mercury: condensed solid-phase mercury (primarily HgO); soluble vapor-phase ionic mercury forms (such as $HgCl_2$); and elemental mercury (Hg*E*). The condensed mercury and water-soluble mercury vapor represent the divalent forms of mercury in the particulate and vapor phases, respectively. This approach was taken in the 1997 MRTC and was also used for the HWC risk assessment.*

The risk assessment for the final rule also followed the same general modeling approach developed for the 1997 MRTC and used the IEM-2M surface water model and the methylmercury reference dose developed especially for that report. The 1997 MRTC was critically reviewed by EPA's Science Advisory Board. Although the SAB had some

concerns with the surface water modeling in the draft report (which EPA believes were addressed in the final report), the subcommittee felt that EPA's reference dose for methylmercury was well supported by the available data while recognizing that important human studies were still ongoing.

EPA recognizes that mercury speciation is a critical factor in determining exposure and risks from mercury emissions. Environmental exposures to elemental mercury are most likely to occur through inhalation, whereas exposures to methylmercury are most likely to occur through consumption of fish. Exposure to divalent mercury can occur through a variety of dietary sources. For the final rule, EPA assessed the human health and ecological risks associated with elemental, divalent, and methylmercury. EPA's analysis shows that the most significant exposures come from methylmercury. This is due primarily to deposition and runoff of divalent mercury to surface water and subsequent methylation and bioaccumulation in fish.

***Comment 388*:** EPA's risk assessment procedures are undocumented, unreliable and flawed. The commenter objects to any use of indirect exposure risk assessments to make legally binding decisions in light of the highly preliminary nature of EPA's guidance on this issue (e.g., EPA's 1993 *Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*); the determination by EPA's Science Advisory Board that current Agency guidance is inadequate; the lack of public notice and opportunity for comment under the Administrative Procedure Act prior to application of indirect risk assessment methodologies; and the need for rulemaking on this critical issue are resolved. The commenter continues to object to EPA's use of indirect exposure risk assessments, both in the context of site-specific permitting decisions and in the context of national standard setting. The commenter refers to comments it submitted in response to a November 22, 1993, *Federal Register* notice on the 1993 Addendum. The comments (to which EPA has never responded) highlight numerous serious deficiencies that render EPA's current protocols wholly inappropriate for legally binding decision-making.

Moreover, as a legal matter, EPA would be violating the Administrative Procedure Act (APA) by issuing and using indirect exposure protocols without first subjecting them to rulemaking procedures. As the D.C. Circuit has ruled, where EPA develops a so-called "guidance" document that actually specifies scientific procedures and protocols to make binding and enforceable decisions, it must follow notice and comment rulemaking procedures (McLouth Steel v. EPA, 838 F.2d 1317 [D.C. Cir. 1988]). While EPA has solicited comment on a "draft guidance document" called an Addendum, this document, which is labeled "DRAFT — DO NOT QUOTE OR CITE" on every page, is not by any stretch of the imagination a proposed rule. As the comments submitted on the November 22, 1993, notice show, the Addendum does not even begin to provide a clear path as to which procedures are to be used in which particular circumstances. At this stage, the very nature of the document makes it inappropriate to be considered a "rule." Moreover, simply noticing the Addendum for public comment in the *Federal Register* did not suffice to meet the APA requirements for proposing a rule. Thus, EPA

cannot validly claim that soliciting public comments on the Addendum in 1994 could satisfy the APA and McLouth requirements for rulemaking. This is particularly true where EPA has never responded to the numerous public comments.

Response 388: *It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from hazardous waste combustion facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA,1990) and was updated in 1993 with the draft Addendum (U.S. EPA,1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA,1994a,b) and the December 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA,1997c), as well as other sources of information.*

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board. EPA intends to respond to comments on draft Addendum that have been received from the public, as well as from the SAB, in an appropriate forum.

*The risk assessment for the proposed rule was fully documented in the risk assessment Background Document (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). As part of the rulemaking package, the risk assessment was subject to full notice and comment procedures consistent with the Administrative Procedure Act. A notice of proposed rulemaking was published in the* Federal Register *on April 19, 1996. The public comment period extended from April 19, 1996, to August 19, 1996, a period of more than 120 days. A public docket was established for the notice of proposed rulemaking and all comments received on the proposed rule were placed in the public docket (U.S. EPA Docket Number F-96-RCSP-FFFFF). In addition, the risk assessment for the proposed rule was externally peer reviewed. A notice of data availability was published in the* Federal Register *on August 23, 1996, requesting comment on the report prepared by the peer reviewers. A 30-day public comment period was established. Comments received during the public comment periods were considered by EPA in the risk assessment for the final rule. EPA has prepared this response to comments document for the final rule that explains how EPA considered each of the comments received on the risk assessment at proposal.*

As explained in the preamble, the final rule sets an emission standard for existing cement kilns based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources. The risk analysis does not play a part in establishing the actual emission standards themselves, which implement the technology-based requirements of CAA section 112(d). Thus, the commenter's notice and comment concerns about portions of the risk methodology, which EPA regards as unfounded, do not relate to the emission standards themselves.

***Comment 190*:** EPA's protocol for evaluating site-specific multipathway risk relies on unreasonable scenarios and invalidated models. EPA has not responded to key comments from the SAB regarding the methodologies and assumptions used to develop the MACT regulations. The risk analysis used for the MACT rule relies heavily on two documents which have been reviewed by the SAB: (1) *Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (the Addendum, U.S. EPA, 1993) and (2) the draft dioxin reassessment. The SAB, a scientific expert peer-review panel established to guide the EPA, made a number of critical comments concerning both of these documents which have not been addressed by EPA in the MACT risk assessment. Because EPA has not yet acknowledged the SAB comments and criticisms regarding the models and assumptions (in the Addendum and in the dioxin reassessment) used to support the MACT regulation, the validity of the science used by EPA is questionable.

The risk assessment relies on a methodology rejected by the Science Advisory Board. *The Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (U.S. EPA, 1990) and its Addendum (U.S. EPA, 1993) were both evaluated by the SAB as late as December 1993 and rejected as "not ready for release." In particular, among other issues, the SAB was concerned about the uncertainties associated with the model. The commenter cites a July 29, 1994, letter from the Chair of the SAB to the EPA Administrator that says the committee's principal conclusion is that the Addendum is not ready for release as an EPA methodology for routine regulatory assessment of indirect exposure from stationary combustors due to the substantial scientific uncertainties in the models. The commenter says that in developing the MACT rule, the EPA has relied upon the risk model as an accurate quantitative instrument. Given the conservative choices of default parameters and subsistence sub-populations upon which risk calculations are based, EPA should acknowledge and compensate for the overly conservative nature of the methodology.

Response 190: *It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from hazardous waste combustion facilities. As explained in the risk assessment for the proposed rule, EPA used the indirect exposure methodology as outlined in the 1990 document* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emission *(U.S. EPA,1990). This document was updated in*

1993 with the draft Addendum (U.S. EPA,1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure with information gathered in conjunction with the dioxin reassessment (e.g., Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA,1994a,b), as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."*

EPA acknowledges the uncertainty implicit in the use of the models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board. EPA intends to respond to comments on draft Addendum that have been received from the public, as well as from the SAB, in an appropriate forum.

EPA modified the exposure methodology used at proposal to improve the representativeness of the analysis. For the final rule, EPA used central tendency exposure factors for estimating exposures, except for a few risk driving pathways for which an exposure factor variability analysis using Monte Carlo simulation was performed. In addition, the risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and concentrations in agricultural products. Also, U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing as a recreational sport and persons engaged in farming or fishing for subsistence. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative.

Comment 0170-1: The commenter notes that EPA has asked that another external peer review of the MACT risk assessment methodology be undertaken, without heeding and incorporating past scientific reviews of underlying documents, and that EPA has only recently made the "new" review available to the public.

The commenter cites the proposed rule in which EPA states that it is also conducting an external peer review of its risk analysis conducted in support of the proposed rule and indicates that the

results of the review will be included in the public record for this rule and will be considered in developing the final rule. The commenter feels that EPA has allowed insufficient time for adequate review of these materials as part of these comments. SAB reviews of the risk methodology upon which the analysis in the proposed rule is based have questioned various parameters in the methodology. EPA has not yet either formally addressed these comments in the MACT risk assessment methodology or revised the methodology as suggested by the SAB but rather has chosen to go to another scientific forum. Given that EPA has not fully incorporated the scientific comments of past peer-review panels, it is not clear how the comments and criticisms of a new panel will be used. Thus, it is incumbent upon EPA to allow sufficient time for the public to review and comment on any new information of this nature.

Response 0170-1*: The risk assessment for the proposed rule was fully documented in the risk assessment background document (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). As part of the rulemaking package, the risk assessment was subject to full notice and comment procedures consistent with the Administrative Procedure Act. A notice of proposed rulemaking was published in the* Federal Register *on April 19, 1996. The public comment period extended from April 19, 1996, to August 19, 1996, a period of more than 120 days. A public docket was established for the notice of proposed rulemaking and all comments received on the proposed rule were placed in the public docket (U.S. EPA Docket Number F-96-RCSP-FFFFF). In addition, the risk assessment for the proposed rule was externally peer reviewed. A notice of data availability was published in the* Federal Register *on August 23, 1996, requesting comment on the report prepared by the peer reviewers. A 30-day public comment period was established. EPA believes that the 120-day comment period for the notice of proposed rulemaking, followed by the additional 30-day comment period on the peer review, provided ample opportunity for the public to comment on the risk assessment for the proposed rule. Indeed, EPA received numerous comments from this and other commenters.*

Comments received during the public comment periods were considered by EPA in the risk assessment for the final rule. EPA has prepared this response to comments document for the final rule that explains how EPA considered each of the comments received on the risk assessment at proposal. A response to comments document was also prepared on the report of the external peer review panel on the risk assessment for the proposed rule.

EPA intends to address the comments from the SAB on the 1993 Addendum to the indirect exposure methodology and the 1994 draft dioxin reassessment in the appropriate forum. EPA has responded to the SAB's concerns regarding the cancer slope factor for dioxin with Chapter 8 of the 1997 report Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds *(U.S. EPA, 1997d). In*

addition, EPA responded to the comments from the SAB review of the Mercury Study Report to Congress *and finalized the report in 1997 (U.S. EPA, 1997c).*

Comment 0170-2: The commenter states that EPA has relied on site-specific quantitative risk assessment to evaluate the proposed rule, but does not want to be held to the specific cancer risks identified with different options. EPA is proposing emission standards for HWCs under joint authority of the CAA and RCRA. The commenter cites the proposed rule, in which EPA stated that "the overall thrust of the proposed rule is to have the CAA standards supplant independent RCRA standards wherever possible (i.e., to have the CAA standards, wherever possible, also serve to satisfy the RCRA mandate so that additional RCRA regulation is unnecessary)." Under RCRA 3004(a) and (q), EPA must promulgate standards "as may be necessary to protect human health and the environment." The commenter notes that to determine whether the proposed MACT standards are consistent with EPA's mandate under RCRA, EPA conducted two types of analyses: site-specific risk assessments for individuals exposed to dioxin emissions living near 11 model combustion facilities and a more qualitative evaluation of risks associated with dioxin and mercury at the national level. With regard to site-specific risk assessments, EPA states, "…stack gas concentrations used in the modeling of the example facilities were derived from national emissions data. Therefore, while the example facilities are useful for providing information to evaluate national standards on a national basis, they are not site-specific assessments of any individual facility and cannot be regarded as such" (61 FR 17370). Thus, the Agency relies upon the site-specific risk assessments as a generic risk assessment to determine the protective ness of the proposed limitations. The commenter cites Table IV.4.C.1, *Individual Cancer Risk Estimates for Cement Kilns*, which lists a maximum risk from dioxins of 2×10^{-5} (e.g., 2 cases in 100,000) and notes that the Agency decided in this case to adopt beyond-the-floor controls. The commenter states that the Agency appears to justify the need for BTF emission standards based on floor-level risks of statistical excess cancer cases that range from 4×10^{-9} (4 cases in 1 billion) to 2×10^{-5} (2 cases in 100,000). Given that Agency has determined that a risk of 1×10^{-5} is protective of human health for the purposes of RCRA 3004(q)(1) and that Agency-calculated risks for hazardous waste-burning cement kilns are generally below this level, it would appear that the Agency has quantitatively correlated emissions with risk on a national scale. The commenter cites the proposed rule in which EPA states that a target risk level of 1×10^{-5} was used for the high-end individual risk.

The commenter notes that in the proposed rule, EPA states that it is not appropriate to quantitatively correlate emissions with risk on a national scale, although it believes this type of information is useful for qualitatively evaluating the potential impact of the proposed MACT rule (61 FR 17371). Following this logic, based on a less than 1 percent contribution of total TEQ dioxins from cement kilns using waste-derived fuels, the potential impact of the proposed rule on reduction of dioxin is very small (based on current emissions of 0.17 kg dioxin TEQ/yr and total dioxin TEQ emissions of 20 kg/yr).

Response 0170-2*: Although EPA used site-specific parameter values whenever possible for the final rule, including site-specific estimates of emissions, the risk analysis continues*

to rely on a mix of facility-specific, site-specific, and national-level default data. Facility-specific data included stack parameters (e.g., stack height, exit velocity, stack diameter) in addition to emissions. Site-specific data included the use of GIRAS spatial land-use data to support erosion modeling to waterbodies, waterbody-specific parameters (e.g., depth, flow rate) to support waterbody modeling, and meteorological data obtained from the nearest meteorological station to support air modeling. Site-specific data were also used to determine the number and location of exposed populations.

By evaluating a suite of 76 facilities selected to be representative of the universe of facilities covered by the rule, the analysis increased the probability of capturing the range of facility-specific and site-specific conditions occurring at HWC facilities located across the nation. However, the analysis remains a national-level analysis. The analysis generated frequency distributions of individual risks from which various percentiles were reported (e.g., 50th, 90th, 95th, and 99th percentiles). In addition, aggregate population risks were estimated for a variety of health effect endpoints for the final rule. EPA used such information to assess the general protectiveness of the MACT standards with regard to human health and the environment and to assess the benefits of the rule. However, the emission standards themselves are technology-based and are not derived from the results of EPA's risk analysis.

Comment 195a: EPA improperly relies on the draft dioxin reassessment, many of its parameters, and its discussion of health benefits of reducing dioxin emissions. The dioxin reassessment remains a draft document and has had extensive comments by the public and the SAB with no resolution. EPA should not propose any standards until EPA has completed the reassessment, which has had unprecedented peer review. Once completed, EPA should then use the reassessment as the basis for future regulation and standards. If EPA must develop standards despite not having a final dioxin reassessment science document, then it is unclear why EPA: (i) did not at least incorporate SAB recommendations that EPA is using to rewrite the draft dioxin reassessment and (ii) used the draft dioxin reassessment selectively (relying on old methodology which was also criticized by the SAB). It is difficult to understand why the extensive comments of the SAB on the draft dioxin reassessment were not incorporated into the proposed MACT rule given that SAB's, report was officially issued in September 1995 (*A Second Look at Dioxin, Review of the Office of Research and Development's Reassessment of Dioxin and Dioxin-Like Compounds by the Dioxin Reassessment Review Committee,* EPA-SAB-EC-95-021, September 1995). It would appear that EPA had ample time to review this report and incorporate its key findings prior to the publication of the proposed rule in the *Federal Register* on April 19, 1996. Recognition of SAB comments and suggestions is particularly important for those specific issues that would dramatically change the cost benefit of beyond-the-floor decisions (e.g., a reduced cancer slope factor for dioxins).

CKRC also submitted comments to EPA on the draft dioxin reassessment which remain valid given the EPA's choice to rely upon the draft dioxin reassessment. Given that EPA relies upon the draft

dioxin reassessment for key aspects of the proposed rule, EPA must specifically address and justify any departures and conclusions from the draft dioxin reassessment. Moreover, EPA should repropose the MACT rule if the final dioxin reassessment changes the basis for this rule.

***Response 195a*:** *EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA, or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards.*

However, EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job."

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

***Comment 195b*:** EPA appears to have selectively used portions of the draft dioxin reassessment that support its preconceived notions of the risks of dioxins. The commenter cites a statement in the risk assessment Background Document (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document*, February 1996) that indicates the draft reassessment provided a source of physical and chemical properties and additional exposure methodology equations

used in this analysis and another statement in the preamble to the proposed rule (61 FR 17371) that indicates that much of the information used to derive the individual risk estimates for dioxins was taken from the draft reassessment (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds, Review Draft, Volume II of III,* August 1994).

The commenter says it is troublesome that EPA used the draft dioxin reassessment documents in a selective fashion. For example, although EPA did not use the reduced cancer slope factor cited in the reassessment (156,000 $[mg/kg/day]^{-1}$ to 100,000 $[mg/kg/day]^{-1}$), EPA appears to have used much of the risk assessment methodology (including many default parameters) cited in the reassessment for determining site-specific risks. It is inconsistent to use the draft dioxin reassessment as an acceptable source for certain values but not for others (e.g., the risk assessment methodology but not the dioxin cancer potency factor). Indeed, had EPA used the potency factor cited in the draft dioxin reassessment, EPA could not have justified BTF controls on the basis of risk. This suggests that EPA has selectively used those parameters that reflect a worst-case scenario for the health risks of dioxins.

> ***Response 195b:*** *EPA has not relied on the conclusions of the 1994 dioxin health assessment (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds, *U.S. EPA, 1994d,e) because this document remains a draft document. However, EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available, and EPA continues to rely on it for the final rule. This includes the information contained in the 1994 dioxin exposure assessment (*Estimating Exposure to Dioxin-like Compounds, *(external review draft), U.S. EPA, 1994a,b).*
>
> *In response to comments received from the SAB, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (as described in* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d). This study lends considerable support to the earlier cancer potency estimates. However, EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment should be used (*Health Assessment Document for Polychlorinated Dibenzo-p-dioxin, *U.S. EPA, 1985). That estimate is 156,000 $[mg/kg\text{-}d]^{-1}$.*
>
> *EPA's emissions standards are not based on the dioxin reassessment. The final rule sets emission standards for existing sources based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule.*

***Comment 196*:** EPA has established "high-end" risk estimates in a manner different from the procedures outlined in the preamble, ignoring its own long-standing guidance regarding exposure. The "high-end" estimates far exceed the 90th percentile. The commenter cites statements in the preamble to the proposed rule (61 FR 17371) that EPA projected both "high-end" and "central tendency" estimates of risks to the individuals of concern in the analysis and to derive high-end risk estimates, EPA set the emission levels at the 90th percentile of the distribution of available dioxin concentrations and, for most exposure scenarios, set one exposure parameter to a high-end value while keeping all other parameters at central tendency values.

The commenter says that EPA has created regulatory controls at or above the 99.9th percentile. This results from a focus on the high-end risk scenarios posited for different subsistence individuals and by choosing predominantly conservative modeling values. That is, by setting virtually all key parameters (i.e., those which have the greatest affect on the dose) to "high-end" values, the overall risk or dose modeled is far above the 90th percentile. Thus, it is not valid to suggest that the analysis is reasonable because a few parameters were kept at central tendency values. The commenter attaches two papers that discuss the problem with EPA's "high-end" risk modeling ("The Benefits of Probabilistic Exposure Assessment: Three Case Studies Involving Contaminated Air, Water, and Soil," and "Uncertainty and Variation in Indirect Exposure Assessments: An Analysis of Exposure to Tetrachlorodibenzo-p-Dioxin from a Beef Consumption Pathway").

EPA's use of "high-end" risk at the 99.9th percentile also appears to be at odds with the EPA's own guidance. The commenter cites a statement in EPA's Guidelines for Exposure Assessment that conceptually, high-end risk means risk above about the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest risk. The commenter says that implicit in this guidance is the fundamental concept that EPA should try to focus on reasonable "high-end" situations. EPA appears to have violated the spirit of this guidance by setting many key parameters to high-end percentile values (e.g., above the 90th percentile) for certain high-end scenarios. Indeed, this guidance explicitly states that bounding estimates and worst case scenarios should not be termed high-end risk estimates.

The commenter cites statements in EPA's *Guidance for Risk Characterization for Risk Managers and Risk Assessors* that high-end estimates focus on estimates of exposure or dose in the actual population. "Bounding estimates," on the other hand, purposely overestimates the exposure or dose in an actual population for the purpose of developing a statement that the risk is "not greater than." A "worst case scenario" refers to a combination of events and conditions such that, taken together, produces the highest conceivable risk. Although it is possible that such an exposure, dose, or sensitivity combination might occur in a population of interest, the probability of an individual receiving this combination of events and conditions is usually small, and often so small that such a combination will not occur in a particular, actual combination.

Response 196*: For the final rule, EPA modified the approach taken in the risk assessment at proposal for evaluating high-end exposures. Instead of setting high-end exposures deterministically, EPA generated population-weighted cumulative distributions of risks to individuals. To accomplish this, EPA selected a total sample of 76 facilities to represent various categories of HWCs. Of these, 66 were selected at random and 10 had previously been selected at proposal. Each facility was assigned a sample weight based on its selection probability. A study area was defined for each facility as the area surrounding the facility out to a distance of 20 kilometers. Each study area was divided into 16 sectors. Media concentrations were estimated for each facility sector using facility-specific (e.g, stack emissions) and site-specific (e.g., land use) information. Sector-specific exposures were then estimated from the media concentrations and age-specific exposure factors derived from the 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a). Mean exposure factor values were used in order to estimate the mean, or arithmetic average, risk to individuals within a sector. These mean risk values were then aggregated across an HWC category taking into account sector-specific population estimates and facility-specific sampling weights. Population estimates were derived from U.S. Census and Census of Agriculture data.*

For estimating risks from exposures to dioxins and furans via consumption of beef and milk, EPA performed an exposure factor variability analysis. This involved the generation of a distribution of risks at the sector level that reflected the variability in exposure factors between individuals. A cumulative risk distribution was generated from the sector level distributions using Monte Carlo analysis. A similar analysis was conducted for exposures to methylmercury through ingestion of fish.

From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, and 99th percentiles. Using this approach, it was possible for EPA to more precisely characterize the high end of the risk distribution while avoiding the need to set specific parameters to high-end values deterministically. This approach is fully consistent with EPA's risk characterization guidance.

Comment 198**:** EPA should review its choice of constants associated with physical-chemical properties. Although EPA has selected a set of chemical constants (e.g., Kow, Henry's Constant), it is not clear how EPA has chosen these values given that the literature is replete with values other than those chosen by EPA. Further, the values chosen by EPA are significantly different from the values presented in MacKay et al. (1992). The commenter presents a table that compares EPA values for the octanol-water partition coefficient (log Kow) for dioxin and furan homologues to values attributed to MacKay (no reference provided).Given that the peer-reviewed literature demonstrates this range of values, the commenter says EPA should explain how it has chosen the values cited in EPA's dioxin reassessment and how the uncertainty implicit in this range of values is to be incorporated into the implementation of the indirect risk methodology.

Response 198: *Knowledge of physical and chemical properties is essential to understanding and modeling the environmental transport and fate of dioxins. Congener-specific data for basic chemical properties, such as the octanol-water partition coefficient (K_{ow}) and Henry's Law Constant, are lacking. For the final rule, EPA selected chemical/physical properties for dioxin congeners from its 1994* Estimating Exposure to Dixoin-Like Compounds, Volume II: Properties, Sources, Occurrence and Background Exposures *(U.S. EPA, 1994a).*

In estimating chemical/physical properties for dioxins, EPA conducted a thorough search of published scientific literature in order to maximize and optimize the identification of measured values. For the purpose of identifying the most definitive of two or more physical/chemical property values reported in the literature for a given dioxin-like compound, a ranking methodology was developed to evaluate the degree of confidence in the reported values. A property value with a ranking of 1 was considered to have the highest level of confidence; a property value with a ranking of 6 was considered to have the lowest level of confidence. The ranking scheme considered five different factors, including:

- *Confirmation of measured values by at least one additional laboratory*
- *Method of measurement*
- *Adherence to Good Laboratory Practices*
- *Derivation based on known relationships developed for structurally similar chemicals and using directly measured physical/chemical properties*
- *Estimation based on known relationships and using estimated values or a combination of estimated and measured values.*

If two or more values obtained the same ranking, then the value that had been peer reviewed and identified as the most accurate by other EPA offices, other government agencies, or organizations supporting scientific databases was selected as the most definitive value for purposes of the 1994 document. If two or more values with the same ranking had not been peer reviewed, typically the most current value was chosen as the most definitive value. This decision was based on the assumption that the most current value would have been developed using the most recent scientific methods.

EPA acknowledges that other values characterizing the physical and chemical properties for dioxins exist in the literature; however, the values reported in the 1994 document have undergone extensive review and EPA believes them to be the most reliable values currently available. The reference mentioned by the commenter (MacKay et al., 1992) summarizes previously published fate/transport parameters including log Kow values. MacKay et al. presents many of the same log Kow values as does the dioxin reassessment

document. MacKay et al. (1992) however, does not include a prescribed approach for evaluating available log Kow values for selecting a preferred value. Although MacKay et al. (1992) does include a table of "selected" parameters including log Kow values, no explanation of how these particular values were chosen from the set of available values is provided. The dioxin reassessment, on the other hand, provides a detailed approach for ranking log Kow values and selecting a single value. Because the dioxin reassessment includes a comprehensive set of log Kow values (obtained from the open literature) and because it uses a clearly outlined approach to first rank available values and then select the most definitive value, log Kow values were selected from this document.

***Comment 199*:** EPA has, in a constant trend towards conservative estimation of risk, consistently chosen parameters that are not central tendency values. In some cases, parameters have been chosen in excess of values deemed "reasonable" by the SAB. Implicit in the characterization of the central tendency scenario is that the parameter values used in the risk assessment are "central tendency" (i.e., the center of the distribution). Although central tendency values need not always be the median of the distribution, it is not unreasonable that central tendency values be balanced about the 50th percentile. To evaluate this hypothesis and its implications for both the central tendency and high-end scenarios, the EPA's draft *Mercury Study Report to Congress* was reviewed. In this document, EPA utilized a Monte Carlo simulation model to determine a risk distribution for mercury. Many of the parameters used in EPA's simulation are the same for all risk models and independent of the constituent being examined (e.g., diet, climate).

To illustrate the choice of parameters, consider "Fw," the fraction adhering of wet deposition. This parameter is interesting for two reasons: it is a relatively obscure variable which is not associated with changes in scenario between central tendency and high-end and the SAB addressed this parameter. The commenter cites a statement in a July 1994 SAB draft report on its review of the draft *Addendum to the Methodology for Assessing Health Risk Associated with Indirect Exposure to Combustor Emissions* which says that Hoffman et al., 1992 ("Quantification of the Interception and Initial Retention of Radioactive Contaminants Deposited on Pasture Grass by Simulated Rainfall," *Atmospheric Environment* 26(18): 3313-3321) suggests that an assumption of 30 percent retention of particle-bound contaminants is a more supportable assumption than 100 percent, regardless of sorptive tendencies of the contaminant. The commenter says the MACT analysis uses a value of 60 percent, although an examination of EPA's chosen underlying distribution in its *Mercury Study Report to Congress* shows that the probability associated with this value is 71 percent. Thus, while a single value set at the 71st percentile may not be a "high-end" value, neither can it be described as a central tendency value. The commenter presents a table that compares parameter values described as "central tendency" in the risk analysis for the proposed rule with parameter distributions given in EPA's *Mercury Study Report to Congress.* The commenter says the value for Fw, coupled with the other parameter values shown in the table, leads to a central tendency scenario which should be more aptly described as "upper-end."

A parameter of similar interest is the mixing depth of the soil for untilled fields, Zd. The MACT risk analysis uses a value of 1 cm, the same value used by EPA in its *Mercury Study Report to Congress.* The probability associated with a Zd value of 1 cm is the 89th percentile. At this probability level, the value is beginning to meet all the criteria associated with the high-end scenario.

Referring to the above-mentioned table, the commenter says EPA has consistently chosen parameter values for the central tendency scenario which are upper to high-end values for the MACT risk analysis. The commenter presents a second table that illustrates the effect of adding together high-end variables. The table models the simple example of the results of adding together three identical, normally distributed variables and determining the resulting percentiles in the new added variable. As can be seen from these results, three high-end (i.e., 90th percentile) variables added together results in the 98.6th percentile of the new variable. Equally instructive is the sum of 80th percentile variables which yield a new distribution at the 91st percentile. Given that the MACT risk methodology has repeatedly used upper-end values, it is easy to see how such choices (generally representing modest increases from central tendency values) are magnified into extreme high-end risk calculations.

Response 199: *In the risk assessment for the final rule, EPA used site-specific parameter values whenever possible. These included facility-specific parameters such as emissions and site-specific parameters such as soils and land use. However, for agricultural parameters EPA used default values. These were derived as explained in the risk assessment Background Document (RTI, 1999). The default values for agricultural plants are generally consistent with the default values given in the 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c). The default values in the mercury study were set to mean values for those parameters for which it was possible to estimate the shape of the distribution (e.g., interception fraction and crop yield). For the MACT rule, different consumption rates were used for livestock depending on whether the livestock were raised commercially or for subsistence purposes. As a result, commercial beef and dairy cattle were assumed to consume more grain and less forage than subsistence cattle. Human exposure factors such as fish ingestion rates were set to central tendency values except in those instances where Monte Carlo simulation was used. In setting exposure factors, EPA used mean values obtained from the 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a). Although mean values are higher than the 50th percentile, mean values are necessary for estimating population risk and, therefore, were used throughout the risk analysis.*

With regard to the fraction of wet deposition that adheres to plant surfaces (Fw), the SAB commented on this parameter in its review of the draft Addendum. The SAB addressed the question of whether a wet retention fraction of 30 percent might be more appropriate than assuming 100 percent retention for contaminants that have high sorptive tendencies. The SAB agreed that 100 percent was not appropriate for such

contaminants, arguing that it is the particle as a whole and not the sorption of the individual contaminants that determines the degree of retention. Subsequently, EPA derived a wet deposition fraction based on Hoffman's interception fractions for particulates (Hoffman et al., 1992). To obtain a wet deposition fraction for particles, Hoffman's interception fractions for three different particle sizes were adjusted by an assumed interception fraction of 0.47 for forage. Based on the results for the three particle sizes, a value of 0.6 was chosen as a representative, central tendency value. This is the same value that was used in the 1997 MRTC. EPA acknowledges that there is some uncertainty in these estimates. The sources of uncertainty includes experimental errors, uncertainty of applying experimental results to other pollutants, variations in rainfall intensity, and uncertainty of long-term retention.

For the final rule, EPA used a soil mixing depth of 1 centimeter for untilled soil (pasture land) and a mixing depth of 20 centimeter for tilled soil (tilled agricultural land), the same values that were used at proposal. In the SAB review of the draft Addendum (U.S. EPA, 1993a), the SAB agreed that these were reasonable values to use These are also the same values used in the draft dioxin reassessment and the 1997 MRTC. As indicated in the mercury study, the soil mixing depth distributions are arbitrary and were only used to determine the sensitivity of the parameter relative to other parameters. Therefore, any attempt to use the distributions to estimate the percentile associated with a particular value is inappropriate.

EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. That is why, for the final rule, EPA did not use high-end values. In all instances, EPA either conducted a full distributional analysis or used central tendency values.

***Comment 221*:** The commenter states that EPA uses inflated central tendency parameters to exaggerate high-end subsistence scenarios. For example, the commenter says that subsistence beef farmers are located at higher air contaminant locations than the general population, eat 100 percent affected beef, live at the same location longer than the general population, and have cattle that eat more grass and food.

The commenter notes that although EPA varied three parameters to construct its "high-end" and "central tendency" scenarios—exposure duration (ED), emission levels (Q), and (for two scenarios) the contaminated fraction (F)—there are other parameters that affect total media concentrations that EPA has not explicitly identified but has varied by scenario. Examples of variables that the commenter identifies as set to high-end values include:

The grass diet of subsistence farmer cattle versus the diet of average farmer cattle, as the daily diet of beef cattle changes from subsistence farmer to typical farmer: The cattle of the subsistence beef farmer eat a daily diet of forage, silage, grain, and soil totaling 12.27 kg/day, while the cattle of the typical beef farmer eat only 8.85 kg/day.

Maximizing the concentrations of dioxin/furans by choice of the location of the subsistence farm: EPA changes the location of the farm and the river/stream for the subsistence farmer to maximize dioxin concentrations. The commenter tabulates the effects of changing receptor location with respect to maximizing dioxin concentration and cattle diet and states that analysis of the sensitivity of the dioxin concentrations in beef to feed levels shows that the central tendency dioxin toxicity equivalency quotient (TEQ) concentration for the subsistence farmer increases nearly 100 percent from that calculated using the farm location of the typical farmer scenario.

Using the 90th percentile of emissions, high-end dietary practices, and farm location: The commenter presents a table to show the effect of combining multiple high-end values and notes that this holds true even while maintaining the closer location and therefore higher air concentrations of the subsistence beef farmer scenario. Analysis of the sensitivity of the dioxin concentrations in beef to feed levels shows that the central tendency dioxin TEQ concentration for the subsistence farmer increases more than 300 percent from that calculated using the feed rate, cattle diet, and farm location of the typical farmer scenario.

The commenter states that EPA has also chosen *a priori* default parameters that are different from other values recently published by EPA (e.g., HWIR, draft dioxin reassessment) and cause the risk model to calculate higher risks. The commenter feels that the following are two important illustrations of this point:

Soil mixing depth is an important parameter for estimating watershed soil concentrations that relate to the risk impacts resulting from ingestion and dermal contact with contaminated surface water and ingestion of fish. The commenter notes that the soil mixing depth in the draft dioxin reassessment is 10 cm, while the value used in the MACT risk analyses is 1 cm.

Lipid content of edible portions of fish is proportional to the dose of dioxin ingested from dioxin-contaminated fish. The commenter states that EPA has arbitrarily selected a lipid content of 7 percent for edible portions of fish based on limited data. The commenter cites the Great Lakes data compilation as indicating that the overall lipid content for edible portions of all game fish is approximately 5 percent. The commenter notes that, because lipid content is proportional to dose, a reduction in the lipid content from 7 percent to 5 percent would result in a 30 percent reduction in risk.

Response 221: *EPA has taken a number of steps to address comments and concerns raised by the public and the peer reviewers, including those identified by the commenter. EPA believes that these steps provide considerable assurance that the exposure estimates developed for the final rule are reasonable.*

The exposure estimates for the final rule are based on central tendency values (e.g., mean values) for all exposure parameters except in those instances in which an exposure parameter variability analysis was conducted using Monte Carlo simulation. The variability analysis was done for key risk-driving receptor population/constituent combinations including beef and dairy farmers for dioxin. Fate and transport modeling was conducted using site-specific data wherever possible. This is a modification of the approach used in the risk assessment at proposal in which certain parameter values were set to high-end values.

For the final rule, EPA relied on data reported in the 1997 Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a) on total time in the same residence to determine the duration of exposure. The data indicate that farm households have, on average, a longer residence time than nonfarm households. This is not surprising given that farmers are typically a less mobile population group than the general population. For farm households, EPA used a mean residence time of 6.5 to 17.3 years, depending on an individual's age.*

In the proposed rule, emissions were set to high-end values to characterize high-end exposures. This included use of the 90th percentile stack gas concentrations. This high-end characterization was not used in the final rule. Instead, facility-specific stack emissions were estimated based on a facility's trial burn or certificate of compliance tests. In those instances where emissions measurements were not available, stack gas concentrations were imputed from a pool of emissions measurements for other, similar facilities.

For the final rule, EPA assumed that beef farmers eat beef produced on the farm, similar to the approach taken at proposal. However, the intake rate of home-produced beef for the risk assessment for the final rule was based on consumption estimates specific to individuals who report consuming home-produced beef, as presented in the 1997 EFH. Previously, EPA had used beef consumption rates for the general population. EPA believes it is reasonable to assume that beef that is home-produced is produced by the farmer and therefore, is subject to dioxin contamination from local sources. As such, the contaminated fraction is 100 percent because all locally produced beef will contain some level of contamination from local sources depending on the location of the farm. Beef cattle raised commercially were assumed to be taken to slaughter at a younger age and a lower weight than beef cattle raised for subsistence; hence, feed consumption rates were

lower for commercially raised cattle than for cattle raised for subsistence. This is similar to the approach taken at proposal in that cattle raised by the typical farmer had a lower feed consumption rate than those raised for subsistence.

The risk assessment for the final rule was modified to account for the number of individuals exposed and their location. The analysis employed U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. However, it was not possible to identify and locate from census data individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios were retained in the risk analysis for the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. To assess the potential risks, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA recognizes that this assumption may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

For the final rule, EPA used a soil mixing depth of 1 centimeter for untilled soil (pasture land) and a mixing depth of 20 centimeter for tilled soil (tilled agricultural land), the same values that were used at proposal. In the Science Advisory Board review of the draft Addendum (U.S. EPA, 1993a), the Science Advisory Board agreed that these were reasonable values to use. These are also the same values used in the draft dioxin reassessment and the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c).*

EPA agrees with the commenter that the fish lipid content used in the proposed rule was too high to truly represent a central tendency value, as intended. Fish lipid content will vary greatly even within a given species depending on age, physiological state, availability of food, season, and other factors. For the final rule, EPA concluded that lipid content should represent a lipid fraction in the fillet for a wide variety of species across the United States. The fillet value was selected to better reflect the fact that humans consume the fillet portion of fish, while fish-eating wildlife consume the whole fish. The previously used fish lipid content of 7 percent represented the whole fish. For the human health analysis, therefore, EPA used a lipid content of 2.6 percent, which was estimated assuming 36 percent consumption of trophic level 3 fish (lipid content

2 percent) and 64 percent consumption of trophic level 4 fish (lipid concentration 3 percent). This is at the lower end of the range suggested by the commenter and is consistent with a lipid content of 3.1 percent recommended in the final rule of the Great Lakes Water Quality Initiative *for edible portions of trophic level 4 fish (60 FR 15373-15374).*

***Comment 222, 1064*:** The commenter notes that lipid content of edible portions of fish is proportional to the dose of dioxin ingested from dioxin-contaminated fish and that EPA arbitrarily has selected a lipid of 7 percent for edible portions of fish based on limited data. The commenter points out that large compilations of the lipid content of fish found in waters of the United States (that is, the Great Lakes and other water bodies) exist, and, based on the Great Lakes compilation, the overall lipid content for edible portions of all game fish is approximately 5 percent. Edible portions of salmonids from the Great Lakes have a lipid content of approximately 6.7 percent, while nonsalmonids have a lipid content of approximately 3 percent. Many nonsalmonid species have lipid concentrations in edible portions that range from 1 to 2 percent; indeed, only one nonsalmonid species (channel catfish) has a lipid content greater than 5 percent. Because lipid content is proportional to dose, a reduction in the lipid content from 7 percent to 5 percent would result in a corresponding 30 percent reduction is risk.

The commenter points out that the results of the fish lipid analysis are particularly important given recent studies showing fish lipid contents ranging from 3 percent to 6.7 percent. The value of 7 percent used in the MACT risk assessment represents a high-end value. Using a fish lipid content of 5 percent reduces the fish dioxin concentration to 1.15×10^{-6}, 70 percent of the value at 7 percent. The commenter feels that EPA has chosen high-end values for critical parameters and that these choices belie EPA's assertions that reasonable, high-end scenarios have been used to support the proposed rule.

Response 222, 1064: *EPA agrees with the commenter that the fish lipid content used in the proposed rule was too high to truly represent a central tendency value, as intended. The commenter is referred to EPA's response to the previous comment. For the final rule, EPA used a fish lipid content of 2.6 percent. This is a weighted average of the lipid contents in the edible portion of trophic level 3 and 4 fish.*

Comment 223: The commenter tabulates the effects of the use of high-end values rather than central tendency values for the subsistence fisherman scenario and notes that altering the input parameters for the aquatic food chain pathway can have a large impact on the final media concentration. In particular, the commenter evaluated the effects of altering the values of lipid concentration, soil mixing depth, sediment organic carbon fraction, total suspended solids, enrichment ratio, and bed sediment porosity. The commenter notes that altering the fish lipid content or the enrichment ratio, for example, reduces the dioxin TEQ concentration in fish to one-half to one-third of its standard value.

***Response 223**: EPA recognizes the sensitivity of the estimated exposures for the subsistence fishing scenario to the parameter values selected for use in the surface water model. As explained in the response to the previous comment, EPA reduced the fish lipid content for human consumption from 7 percent to 2.6 percent for the final rule. The previously used fish lipid content of 7 percent represented the whole fish, as opposed to the fillet portion typically consumed by humans. This change results in a considerable reduction in dioxin levels in fish and in human exposures that occur through the consumption of fish.*

Also, as indicated in response to the previous comment, EPA retained the 1 centimeter soil mixing depth for untilled soils in the risk assessment for the final rule. This is the same mixing depth that was used for watershed soils and is consistent with the value used in the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c).*

EPA acknowledges that total suspended solids (TSS) concentrations can vary widely For the proposed rule, a fixed default value was used for all waterbodies. However, due to the importance of this parameter in the partitioning of dioxins within the water column and between the water column and sediments, region-specific TSS values were used for the final rule depending on the type of waterbody (i.e., lakes vs. rivers and streams).

For the final rule risk analysis, EPA used a value of 0.045 for the fraction of organic carbon in suspended sediment, as compared to a value of 0.075 used for the proposed rule. This value was changed because it is derived from the fraction organic carbon in soil, which was updated from 0.01 to 0.006 (U.S. EPA, 1996). The 0.045 value is derived from the soil organic carbon fraction using a fraction organic carbon (suspended sediment) to fraction organic carbon (soil) ratio of 7.5 (U.S. EPA, 1993a). The fraction of organic carbon in bottom sediment is used in modeling transport to the aquatic food chain. The fraction of organic carbon in bottom sediment also was updated from the value used in the proposed rule (0.04) to 0.014, the mean value reported in Suedel and Rodgers (1991). The value of 0.04 had been derived in a manner similar to that used to estimate the fraction of organic carbon in suspended sediment, using a ratio of the fraction organic carbon in bottom sediments to the fraction organic carbon in soil of 4 (U.S. EPA, 1993a). However, in the case of dioxins/furans, the Kd_{bs} was calculated based on the fraction of organic carbon in bottom sediment and the Koc. This calculation to derive Kd_{bs} was based on the fraction of organic carbon in bottom sediment; the value used was left as the value derived from the fraction organic carbon in soil. As a result, for the dioxins/furans the fraction of organic carbon in bottom sediment was 0.024 (4 x 0.006).

The soil enrichment ratio accounts for the fact that eroded soils are generally enriched by fine particles sizes and organic matter, as well as by chemical contaminants that bind to

organic matter, such as dioxins. It generally ranges from 1 to 5. EPA retained the value of 3 used in the proposed rule as a reasonable central tendency value for the final rule. This is the default value recommended in the 1993 Addendum (U.S. EPA, 1993a). EPA also retained the bed sediment porosity of 0.6 used as a central tendency value for consolidated sediments in the proposed rule. This value was calculated from the bed sediment concentration (assumed to be 10^6 mg/L for consolidated sediments) and the particle density (assumed to be 2.6 g/cm^3) and is consistent with the method used in the 1993 Addendum (U.S. EPA, 1993a).

Comment 224: The commenter feels that EPA's risk methodology does not properly predict the distribution of congeners found in beef data collected by the USDA and EPA, and that the EPA model overestimates the contribution from those congeners associated incineration while underestimating congeners associated with other sources. The commenter refers to previously submitted comments on the draft dioxin reassessment in which the commenter noted that USDA and EPA conducted a joint statistical analysis of dioxin/furan concentrations in beef fat (specifically cited by the SAB in its comments as indicating that background body burdens may be much less than previously estimated by EPA in the draft dioxin reassessment). The commenter indicates that these data provide a sound basis upon which to evaluate the accuracy of the MACT risk model as it is the only statistically designed study of dioxin concentrations in beef fat. The commenter notes that EPA has used similar data to evaluate the risk model used in the draft dioxin reassessment.

The commenter presents an analysis to calculate the dioxin/furan beef concentrations for the subsistence and average farmer for each of the case studies examined by EPA in the MACT risk assessment Background Document using an air matrix based on the background air matrix found in the dioxin reassessment. The commenter presents the resulting beef concentration congener distribution calculated from EPA's model was compared with the USDA data. As shown in the commenter's figures, the model overpredicts the percentage furans in the beef relative to the contribution from dioxins. This commenter indicates that this error is common to all pathways of the model and notes that, in particular, the model most overpredicts the contribution from the 2,3,4,7,8 PeCDF congener. The commenter notes that because furans are more likely to be associated with combustion processes, this error relates directly to an overestimation of the estimated dose from combustion sources, a discrepancy contributes to a 30 percent increase in the contribution of furans over dioxins when compared to the USDA beef data.

The commenter presents another figure with the results of a similar analysis conducted for dioxin/furan concentrations in fish. For the fish analysis, however, the fish data were derived from the North America data supplied in the dioxin reassessment. The commenter points out that the results for fish are similar to the beef discrepancy in that the model overestimates the 2,3,4,7,8-pentachlorofuran contribution by approximately 25 percent. The commenter feels that, given these results, it is clear that EPA's model is inaccurate and that because EPA's beyond-the-floor analysis relies upon the risks

predicted by its model, it is critical that EPA consider the overestimation of risk in its analysis of BTF control.

***Response 224**: EPA recognizes the importance of model validation in developing and applying indirect exposure models including the model used for the final rule to predict dioxin/furan concentrations in beef and fish.*

EPA's research program includes continued development of models for assessing indirect exposure to dioxins and other bioaccumulative compounds. While it has not been possible to validate the entire system of models that make up the indirect exposure methodology, components of the indirect exposure models have been validated using field data. In addition, many of the modeling components and supporting parameter values are based on empirical data (e.g., bio-uptake factors used to predict congener concentrations in plants, animals, and fish). Although EPA believes that the best models currently available and the most up-to-date parameter data have been used for the HWC final rule, EPA nevertheless recognizes that significant uncertainty is associated with media concentrations predicted by the models. Consequently, EPA has included an expanded discussion of uncertainty associated with the indirect exposure modeling in the Background Document (RTI, 1999) accompanying the final rule.

EPA recognizes the value of conducting model validation studies using empirical or field measurements. These studies compare output from the model, in the form of predicted media concentrations (e.g., soil, water, fish, beef), to measured data for those same media. By comparing the predicted concentrations to the measured concentrations, conclusions regarding the performance of the model can be drawn. However, the utility of these types of model validation studies is heavily dependent on two criteria related to the underlying measured data that form the basis of the analysis:

*First, there needs to be spatial and temporal correlation between model inputs and target model outputs. It is important that data used to characterize the input data be consistent with that used to characterize the observed concentrations that will be compared to model output data. This needs to be true from both a spatial and temporal standpoint (i.e., the two data sets should cover the same location and be drawn from similar time periods). If the input data and observed concentration data are obtained from different locations, then failure to match model output to observed data may be due to inconsistencies between the input data and the observed data. These inconsistencies may vary with regard to either dioxin/furan concentrations or congener mix. That is, the dioxin/furan congener mix in the model inputs (e.g., air and deposition data) may be different from the observed data (e.g., measured beef tissue concentrations) simply because they were taken from different locations and represent different time periods.*

Second, the measured data sets need to be representative. The data sets characterizing both the model input data and observed data to be compared to model output should be representative of the study conditions used for model validation.

If either of the above criteria is not satisfied, then a significant level of uncertainty will be associated with the validation results.

Although EPA commends the commenter for undertaking such an effort, the data that were used in the analysis do not meet these criteria. Limitations of the data include

Uncertainty in the data characterizing dioxin/furan background concentrations in rural ambient air: In the dioxin reassessment, EPA noted that there are insufficient data to characterize dioxin/furan ambient air concentrations in rural areas of the United States. Consequently, measured data for dioxin/furan concentrations in urban/suburban air were adjusted to reflect rural areas through the use of a simple scaling procedure that assumed that the mix of congeners in rural areas matches that in urban areas.

Background dioxin/furan concentrations for rural air and for soil, beef, and fish media are not well correlated: The commenter bases the model validation on background data sets provided in the dioxin reassessment. However, these data sets provide coverage for different areas of the U.S. that may be affected to a greater or lesser extent by dioxin/furan and may reflect different mixes of dioxin/furan congeners. The specific mix of congeners reflects the original anthropogenic sources; consequently, different locations can display different congener mixes.

Although data sets available for characterizing soil, beef, and fish tissue concentrations are more extensive than those available for characterizing ambient air, the issue of spatial correlation between the beef and fish tissue and soil data sets and the ambient air data set still exists.

EPA recognized in the dioxin reassessment the uncertainties implicit in conducting validation studies using such data. Although the results of the comparison performed by the commenter are of interest, EPA believes they are subject to considerable uncertainty and do not warrant the conclusions reached by the commenter.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the

CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA, or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Comment 225*:* The commenter states that EPA uses outdated TEF values to calculate dioxin TEQ emissions from the HWC industry although revised TEFs (based on 90-day feeding studies) are available and that use of revised TEFs (DBTEFs) developed by EPA results in a greater than 30 percent reduction in TEQs from fish and beef and a 25 percent reduction of total background emissions from hazardous waste combustion. The commenter feels that given the availability of DBTEFs, it is inappropriate for EPA to estimate the risks associated with chronic exposure to dioxins using the outdated TEFs, which are based on acute exposure. The commenter states that use of DBTEFs significantly affects EPA's conclusions regarding the contribution of dioxin/furans to the environment by cement kilns using waste-derived fuels as well as the absolute risk associated with individual kilns. While the DBTEFs do not include revised values for hexachloro- or heptachloro-substituted dioxins/furans (Birnbaum and DeVito, 1996), EPA acknowledges that, based upon the cancer bioassay of a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HxCDDs, the TEF for these congeners would be reduced by a factor of 2 (draft dioxin reassessment). The commenter calculates the following reductions in TEQ emissions (calculated as the percent reduction from TEQ emissions reported in the proposed rule) using actual emission data from on-site and commercial hazardous waste incinerators: DBTEF Correction Only—35 percent, and DBTEF and HxCDD Correction—45 percent. The commenter provides tabulated multipathway risk estimates for selected exposure scenarios, and these reductions in TEQ emissions are magnified when the DBTEFs are applied to EPA's MACT risk methodology. Because this methodology overestimates the contribution from furans (particularly 2,3,7,8-TCDF and 2,3,4,7,8-PCDF), the use of DBTEFs reduces the risks associated with ingestion of dioxin contaminated fish and beef by a factor of 2. These risks would be further reduced if the correction for HxCDD were applied. The commenter provides figures that compare the relative RTEQ contributions of dioxins and furans using empirical concentrations for beef and fish and to those predicted by the MACT risk model using DBTEFs. Of interest is that the use of DBTEFs corrects for some of the overestimation of furans in beef and fish. This is the only instance where dioxin TEQs are predicted to exceed furans in accordance with empirical results.

Response 225*: EPA recognizes that there is some uncertainty regarding the most appropriate values for the toxicity equivalence factors (TEFs) for the various chlorinated dioxin and furan congeners. TEFs were developed as a practical and scientifically defensible approach for addressing risks associated with complex environmental*

mixtures of dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs). The consensus TEF values presented in Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-dioxins and Dibenzofurans (CDDs and CDFs) and 1989 Update *(U.S. EPA, 1989) as well as recent updates to the TEFs by the World Health Organization (Van den Berg et al., 1998) were meant to be order of magnitude estimates. As such, small deviations are certainly within the range of uncertainty inherent in the TEF approach. The TEFs used in the final rule are those that were recently published in* Environmental Health Perspectives *(Van den Berg et al., 1998). These values, which were made available to EPA prior to their publication, reflect a consensus from the international community. The TEFs are not based solely on acute studies, as indicated by the commenter; rather, they were developed from all available mammalian, bird, and fish studies previously reviewed, as well as new studies published over the last several years. These studies were carefully evaluated and a database was developed using the following criteria:*

- *At least one CDD, CDF, or PCB congener and a reference compound must be included in the study.*
- *Either TCDD or PCB 126 must be included as a reference compound in the same experiment or studied with the same experimental design by the same authors in another experiment.*
- *The relevant end point should be affected by the congener studied as well as the reference compound.*

The revised TEFs reflect only limited changes to the 1989 interim TEFs. Most TEFs were not changed. Exceptions include OCDD and OCDF (reduced from 0.001 to 0.0001) and 1,2,3,7,8-PeCDD (raised from 0.5 to 1). EPA believes the revised TEFs from the World Health Organization are the most appropriate values for use in the HWC final rule.

***Comment 226*:** The commenter states that EPA's risk methodology overestimates exposure to dioxins and indicates that background soil and water related concentrations of dioxins are overstated by factors that range from 2.5 to 119. The commenter notes that the only complete set of calculations available from EPA is for the water pathway for Case H. Water-related concentrations were calculated using the background dioxin/furan matrix (2,3,7,8, substituted dioxin/furan congener distribution) provided in the draft dioxin reassessment rather than emissions matrix for Case H; EPA relied upon the background matrix as part of its validation of the beef pathway model. The commenter provides tabulated results for water column, sediment, fish, and soil background concentrations calculated by the MACT risk assessment model. and notes that the MACT risk model overestimates the dioxin contribution from water pathway when compared to actual background measurements found in the draft dioxin reassessment. Given the inaccuracies of the risk model used in the proposed rule and the implications for the basis of the rule, the commenter feels that EPA should revise its model.

***Response 226**: As indicated previously, EPA recognizes the importance of model validation in developing and applying indirect exposure models, including the model used to predict dioxin/furan concentrations in fish.*

EPA's research program includes continued development of models for assessing indirect exposure to dioxins and other bioaccumulative compounds. Although it has not been possible to perform a true model validation study on dioxins in surface water and uptake in fish, EPA notes that many of the modeling components and supporting parameter values are based on empirical data (e.g., bio-uptake factors used to predict congener concentrations in fish). Although EPA believes that the best models currently available and the most up-to-date parameter data have been used for the HWC final rule, EPA recognizes that significant uncertainty is associated with media concentrations predicted by the models. Consequently, EPA has included an expanded discussion of uncertainty associated with the indirect exposure modeling Background Document (RTI, 1999) accompanying the final rule.

EPA recognizes the value of conducting model validation studies using empirical or field measurements. These studies compare output from the model, in the form of predicted media concentrations (e.g., soil, water, fish), to measured data for those same media. By comparing the predicted concentrations to the measured concentrations, conclusions regarding the performance of the model can be drawn. However, the utility of these types of model validation studies is heavily dependent on two criteria related to the underlying measured data that form the basis of the analysis:

- *First, there needs to be spatial and temporal correlation between model inputs and target model outputs. It is important that data used to characterize the input data be consistent with that used to characterize the observed concentrations that will be compared to model output data. This needs to be true from both a spatial standpoint and a temporal standpoint (i.e., the two data sets should cover the same location and be drawn from similar time periods). If the input data and observed concentration data are obtained from different locations, then failure to match model output to observed data may be due to inconsistencies between the input data and the observed data. These inconsistencies may vary with regard to either dioxin/furan concentrations or congener mix. That is, the dioxin/furan congener mix in the model inputs (e.g., air and deposition data) may be different from the observed data (e.g., measured beef tissue concentrations) simply because they were taken from different locations and represent different time periods.*

- *Second, the measured data sets need to be representative. The data sets characterizing both the model input data and observed data to be compared to*

model output should be representative of the study conditions used for model validation.

If either of the above criteria is not satisfied, then a significant level of uncertainty will be associated with the validation results.

Although EPA commends the commenter for undertaking such an effort, the data that were employed in the analysis do not meet these criteria. Limitations of the data include:

- *Uncertainty in the data characterizing dioxin/furan background concentrations in rural ambient air: In the dioxin reassessment, EPA noted that there are insufficient data to characterize dioxin/furan ambient air concentrations in rural areas of the United States. Consequently, measured data for dioxin/furan concentrations in urban/suburban air were adjusted to reflect rural areas through the use of a simple scaling procedure that assumed that the mix of congeners in rural areas matches that in urban areas.*

- *Background dioxin/furan concentrations for rural air and for soil and fish are not well correlated: The commenter bases the model validation on background data sets provided in the dioxin reassessment. However, these data sets provide coverage for different areas of the U.S. that may be impacted to a greater or lesser extent by dioxin/furans.*

Although data sets available for characterizing both soil and fish tissue concentrations are more extensive than those available for characterizing ambient air, the issue of spatial correlation between the fish tissue and soil data sets and the ambient air data set still exists.

EPA recognized in the dioxin reassessment the uncertainties implicit in conducting validation studies using such data. Although the results of the comparison performed by the commenter are of interest, EPA believes they are subject to considerable uncertainty and do not warrant the conclusions reached by the commenter. In particular, the results of the commenter's comparison for fish suggests much closer agreement: the ratio of the fish concentrations calculated from background air concentrations to the background fish concentration ranges from 0.1 to 2.7, with a median of 0.5, suggesting a tendency toward underprediction, not overprediction.

Comment 228: The commenter states that high-end food consumption scenarios need to be re-evaluated in light of the recent USDA/EPA back fat study, which suggests average levels of dioxin are one-fourth lower than EPA presents in the draft dioxin reassessment and the proposed HWC MACT rule. Despite these data, the risk analysis uses high-end food consumption scenarios that are

based on an unverified assumption: that subsistence farmers and fishermen consume much of their diets from farms and streams that are contaminated. The commenter states that it already is known from recent studies of subsistence fishermen in Florida that the amount of fish in the diet of these fishermen is greatly overestimated, as confirmed by taking hair samples from the fishermen and analyzing for mercury. It was found that the mercury levels in the hair were far below those that would have been estimated by the mercury levels in fish in this area and the estimated diets of the subsistence fishermen.

Response 228: *The joint USDA/EPA data for dioxins/furans in beef fat are most representative of background exposure levels and are not comparable to exposures in areas affected by HWC emissions. Therefore, it is inappropriate to use the USDA/EPA data to draw conclusions about the results of the HWC risk analysis.*

For the final rule, EPA modified the exposure scenarios to be more representative of the exposed population at each facility. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing for recreation and for individuals engaged in farming or fishing for subsistence. EPA used central tendency exposure factors to estimate exposures, except for a few risk-driving pathways for which EPA performed an exposure factor variability analysis using Monte Carlo simulation. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative and, when taken together, are not likely to substantially overestimate (or underestimate) the range of possible exposures.

Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence farming and fishing does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals.

The same food intake rates were used for subsistence farmers as were used for farmers engaged in farming for commerce. Subsistence farmers were assumed to subsist entirely on food produced on the farm, while commercial farmers were assumed to consume only the primary commodity they produced for sale. EPA derived food intake rates from data collected on utilization of home-produced foods by households from the U.S. Department of Agriculture's Nationwide Food Consumption Survey as cited in the 1997 Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a). For subsistence fishing, EPA used fish intake rates representative of Native American tribes from the Columbia River basin, who obtain a significant portion of their dietary intake from fish. The use of exposure factors derived from such studies ensures the plausibility of the exposure scenarios EPA used to characterize risks to individuals who may be engaged in subsistence activities.*

Comment 1081, 1082: EPA erroneously concludes that the MACT rule's risk assessment is not conservative and does not cover the entire range of risks. The commenter cites statements in the

risk assessment Background Document that it is likely that the results of the risk assessment do not cover the range of possible individual risks across all of the facilities because the number of case studies for each category is relatively small compared to the total number of facilities in each category (4/162 incinerators, 5/26 cement kilns, and 2/7 lightweight aggregate kilns); although the facilities selected were representative with respect to the range in size and geographical location, their selection was influenced by availability of appropriate meteorological data; therefore 11 facilities cannot be considered statistically representative of all hazardous waste combustion units. The commenter says that, although each of the above statements are individually correct, taken together these statements could be interpreted to suggest that the risks calculated by Agency for the HWC industry are understated. However, the manner in which EPA has chosen its case studies and constructed exposure scenarios leads to overly conservative results.

EPA has, in fact, characterized the upper-tail of the risk distribution by selection of scenarios that go far beyond high-end exposures. By failing to re-evaluate the cancer slope factor and consider a threshold, EPA has over estimated risks. In addition, EPA chose to evaluate the HWC industry using 11 facilities (i.e., 4 of 20 commercial hazardous waste incinerators, 5 of 26 hazardous waste burning cement kilns, and 2 of 7 hazardous waste burning lightweight aggregate kilns). It is disingenuous of EPA to suggest that these facilities are not representative given that EPA chose these facilities to represent the HWC industry. EPA's admission that the risks associated with small on-site incinerators are overstated by the case studies chosen to represent hazardous waste incinerators provides further evidence of the overly conservative nature of the MACT risk assessment.

> ***Response 1081, 1082:*** *EPA received a number of public comments that the risk analysis for the proposed rule was unrepresentative of HWCs, including comments which noted the absence of smaller incineration facilities among the facilities analyzed. These comments are consistent with comments from the external peer review. Therefore, for the final rule, EPA increased the number of facilities and modified the way the facilities were chosen.*
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> *For the final rule, EPA evaluated 76 active HWC facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). This represents over 40 percent of the hazardous waste combustion facilities covered by the rule. To ensure that these facilities would be representative of all facilities covered by the rule, 66 of the 76 facilities were selected using stratified random sampling, while the remaining 10 were facilities that had been analyzed at proposal and were retained for the final rule (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure). Sample sizes were chosen such that the probability of selecting a "high risk" facility would be 90 percent or better. A high risk facility is one that lies above the 90th percentile of the risk distribution. Therefore, EPA believes the risk assessment for the final rule is representative of the*

range and types of hazardous waste combustion facilities. EPA also expects that the risk assessment adequately characterizes risks at the high end of the risk distribution.

For the final rule, EPA modified the exposure scenarios to be more representative of the exposed population at each facility. The risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and U.S. Census data to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming for an occupation or fishing for recreation and persons engaged in farming or fishing for subsistence. EPA used central tendency exposure factors for estimating exposures, except for a few risk driving pathways for which an exposure factor variability analysis was performed, using Monte Carlo simulation. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative and, when taken together, are not likely to either substantially overestimate or underestimate the range of possible exposures.

In addition, EPA has reevaluated the cancer slope factor for dioxins, specifically 2,3,7,8-TCDD as part of the dioxin reassessment. However, EPA fails to find convincing evidence of the existence of a threshold. Instead, EPA believes there are sufficient human and animal data suggesting a linear dose-response relationship to warrant continued use of a linear dose-response model. Until the dioxin reassessment is concluded, however, it is EPA's policy to continue to use the slope factor based on EPA's 1985 health assessment (U.S. EPA, 1985). EPA notes that its most recent analysis of the human and animal data lends considerable support to the 1985 slope factor estimate.

***Comment 229*:** EPA ignores the SAB comments regarding the conservative nature of the cancer slope factors used in the draft dioxin reassessment. By doing so, EPA implies that upper bound risk calculations are not conservative. The draft dioxin reassessment documents discuss (in considerable detail) a number of the uncertainties associated with both the cancer slope factor and the many parameters used in the exposure assessment.

The SAB specifically commented on the threshold nature of the dose-response curve for dioxin. In particular, the SAB explicitly charged EPA to re-evaluate the cancer slope factor and to incorporate a threshold. The importance of using a threshold for assessing risk is that risks for the low levels seen in the environment will be less than those calculated by the present model. Again, EPA has made a conservative decision without acknowledging either SAB's concerns or the conservative nature of its estimate. Had an appropriate cancer slope factor and threshold been used in EPA's analyses, the risks calculated (and the corresponding benefits resulting from risk reduction) for the proposed rule would be substantially less.

Response 229: *In its review of the 1994 draft dioxin reassessment, the EPA Science Advisory Board suggested that EPA consider alternatives to the linear nonthreshold*

model, allowing for minimal response at low environmental levels of exposure, and which would be consistent with the body of available health effects data. In response, EPA developed a mechanistic model for liver tumors in female rats using data from a 2-year feeding study in Sprague-Dawley rats (Kociba et al., 1978). EPA also summarized the results of simple empirical models that have been applied to other significant cancer findings in female Sprague-Dawley rats from the Kociba study and in Osborne-Mendel rats and B6C3F1-mice from the National Toxicology Program study (NTP, 1982). EPA's analysis indicates that most of the cancer findings exhibited response consistent with linearity in the observable range. Also, few of the mechanistic models that EPA identified in the scientific literature exhibited nonlinear dose-response in the observable region or predicted nonlinear dose-response in the low-dose (extrapolation) region. Furthermore, EPA's analysis of the data from the National Institute of Occupational Safety and Health (NIOSH) study (Fingerhut et al., 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin") found that the ratio of the average daily dose for the high- and low-dose groups was the same as the ratio of increased risk for respiratory cancer, indicating a linear dose-response curve (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d). Therefore, EPA believes there are sufficient human and animal data suggesting that response is proportional to dose to warrant continued use of the linearized multistage model.*

***Comment 389*:** The EPA administrative record is incomplete for evaluation of the risk assessment calculations: EPA has omitted the basic calculations from the administrative record and the administrative record contains mistakes. The administrative record for the rulemaking (i.e., the docket) does not include the spreadsheets (i.e., specific calculations and variables) upon which the risk calculations for each model scenario or pathway are based. The record contains mistakes and has omitted basic calculations. This is in contrast to the recently proposed HWIR rule where EPA made available a CD-ROM disk with some 50 megabytes of spreadsheets so all calculations could be reproduced.

The administrative record contains errors. Based on materials provided by EPA outside of the administrative record, a number of physical-chemical parameters identified in the documentation in the administrative record do not match those provided in EPA's calculations (that is, the values listed in the administrative record do not match those of the calculations provided by EPA). Examples of these errors include the Henry's Law constant of PeCDD, Fv for OCDF, and operating hours (7,324 hours vs. 7,560 hours listed in the documentation).

Response 389: *The background document for the proposed rule documents all equations and parameter values used in the risk analysis at proposal (see* Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February, 1996). Although EPA*

has not made the spreadsheets and other software that were used in HWC risk analysis part of the administrative record, EPA placed example spreadsheet calculations in the docket for the proposed rule. (This information was included as an attachment to a July 17, 1996, memo from D. Layland to the file documenting a meeting with cement industry consultants.) EPA also made all air dispersion model output files available to the public through the RCRA docket. (See "Availability of Electronic Files of Dispersion Modeling Results," memorandum from D. Layland to the RCRA Docket dated May 16, 1997.) In addition, much of the software that EPA used in the analysis is publicly available. This includes the Industrial Source Complex Short-Term (ISCST) model, which was used for modeling air dispersion and deposition in both the proposed and final rules, and the Integrated Exposure Uptake Biokinetic Model (IEUBK) model, which was used for modeling lead exposures in the final rule. EPA has carefully documented the calculations, variables, and data used in the risk assessment for the final rule and has placed these in the rulemaking docket. In addition, the IEM-2M model, which was used for modeling the fate and transport of mercury in surface water in the risk assessment for the final rule, is fully documented in the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c).*

The discrepancies noted by the commenter have been documented and placed in the administrative record. Errors found in the risk analysis for the proposed rule have been corrected for the final rule. EPA corrected the Henry's Law constant for PeCDD. EPA believes the value of the vapor fraction, Fv, for OCDF used in the proposed rule (0.002) is correct, and this is the value that was used for the final rule. For the final rule, each facility was assumed to operated 24 hours per day, 365 days per year, for a total of 8,760 hours.

***Comment 390**:* The commenter feels that EPA has grossly exaggerated the dangers of dioxin by selectively relying on the draft dioxin reassessment without considering the explicit comments of the Science Advisory Board (SAB), EPA's own research, and the comments received by EPA on the dioxin reassessment. The draft dioxin reassessment has been the subject of considerable controversy and unprecedented scientific review by the SAB and public. The commenter states that given the current unsettled nature of the draft dioxin reassessment, reliance upon this document to estimate health benefits of dioxin is inappropriate and premature. Anticipating a good faith effort by EPA to modify the draft dioxin reassessment in accordance with the comments and recommendations of the SAB and public, it is inappropriate at this time for the proposed MACT rule to rely on the key conclusions in the draft dioxin reassessment.

The commenter provides some specific examples of conclusions in the draft dioxin reassessment that the SAB took issue with, as well as some additional observations on health related issues. EPA's own research on TEFs derived from 90-day feeding studies establishes that the present TEQ system overestimates long-term risks by a factor of 4 to 10 depending on the emission matrix. The proposed MACT rule incorporates the use of the TEQ methodology endorsed in the dioxin

reassessment for assessing risks associated with exposures to dioxins (61 FR 17375). The proposed rule does not address the critical issue that the TEFs currently in use are likely overestimates of the relative toxicity of the 2,3,7,8-substituted congeners. Even as the dioxin reassessment was being prepared, data developed by EPA were available which demonstrated that the TEFs for several dioxin and furan congeners were less than the values currently in use. Birnbaum et al. (1993) concluded that TEFs may be overly conservative because they are based on acute rather than chronic studies. At Dioxin '93, Birnbaum and colleagues presented a paper on dose-response relationships for 7-Ethoxyresorufin-O-deethylase (EROD) induction in liver, lung, and skin for dioxin and dibenzofurans. In that paper, the authors concluded that the current TEFs may be overly conservative. These results have been published (Birnbaum and DeVito, 1996) and have significant implications for estimates of exposure and resulting body burdens of all dioxins, furans, and dioxin-like PCBs. Since dioxin-like PCBs make up the major portion of daily intake and resulting body burdens, the substantial reductions in the TEFs for this group of compounds means that dioxin-like PCB TEQ body burdens decline significantly. Also, typical PCDD/PCDF body burdens are reduced by approximately a factor of 5 (e.g., the TCDD and PCDD congeners with unchanged TEFs account for about 15 percent of body burden). The rest of the body burden is reduced by about a factor of 10. Consequently, the revised PCDD/PCDF body burden is about 20 percent to 25 percent of the present body burden. The result of these reductions in PCDD/PCDF body burdens may be significant. For many or most of the adverse effects hypothesized by EPA in the draft dioxin reassessment as occurring at or near background body burden levels (a position judged by the SAB as speculative and in need of reevaluation), the margin of exposure (MOE) will increase. This is because studies in animals are based almost exclusively on TCDD, while human body burdens are derived predominantly from other congeners (now with reduced TEFs). As the TEQ body burden declines, the MOE must increase.

The commenter feels that this finding, based on the results of the most recent research, demonstrates that the TEQ methodology should not be used for regulation until all TEFs for all dioxin and furan congeners are corrected.

> ***Response 390****: EPA has not relied on the conclusions of the 1994 dioxin health assessment (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin [TCDD] and Related Compounds, *U.S. EPA, 1994d,e) because this document remains a draft document. However, EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available, and EPA continues to rely on it for the final rule. This includes much of the information contained in the 1994 dioxin exposure assessment (*Estimating Exposure to Dioxin-like Compounds, *(external review draft), U.S. EPA, 1994a,b).*
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> *EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.*

EPA acknowledges that the SAB had a number of comments and concerns regarding the draft Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds *(draft dioxin assessment) and agrees that there are uncertainties associated with the TEQ approach. Although the SAB called for clarifications in the specification of TEFs for the various dioxin-like compounds for various health outcomes, the SAB concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds.*

While the draft dioxin assessment has been reviewed by the SAB and others, it has not yet been made final by EPA. Because the 1994 dioxin health assessment remains a draft document, EPA is not relying on the conclusions presented in the draft reassessment.

The TEFs used in the final rule are those recommended by the World Health Organization that were recently published in Environmental Health Perspectives *(Van den Berg et al., 1998). These values, which were made available to EPA prior to their publication, reflect a consensus from the international community. The TEFs are not based solely on acute studies, as implied by the commenter; rather, they were developed from all available mammalian, bird, and fish studies previously reviewed as well as new studies published over the last several years. These studies were carefully evaluated and a database was developed using the following criteria:*

- *At least one CDD, CDF, or PCB congener and a reference compound must be included in the study.*
- *Either TCDD or PCB 126 must be included as a reference compound in the same experiment or studied with the same experimental design by the same authors in another experiment.*
- *The relevant end point should be affected by the congener studied as well as the reference compound.*

The TEFs applicable to humans and mammals considered in vivo toxicity data, in vitro data, and quantitative structure-activity relationship (QSAR) data. In vivo data were given the highest priority; in fact, the TEFs applicable to humans were largely derived from in vivo data.

The revised international TEFs reflect only limited changes to the 1989 interim TEFs. Most TEFs were not changed. Exceptions include OCDD and OCDF (reduced from 0.001 to 0.0001) and 1,2,3,7,8-PeCDD (raised from 0.5 to 1). EPA believes the revised

international TEFs are the most appropriate values for use in the HWC final rule. Therefore, EPA disagrees that the dangers of dioxins have been grossly exaggerated.

***Comment 391*:** Given the uncertainty expressed by the SAB about EPA's assessment of potential cancer risks possibly associated with low environmental exposures to dioxin, it is premature for the proposed rule to rely on procedures or conclusions in the draft dioxin reassessment. The proposed rule relies on a total TEQ dose of 0.01 pg/kg/day as associated with an upper bound excess cancer risk of 10^{-6}. The commenter cites a statement in the preamble to the proposed rule that as many as 600 cancer cases may be attributable to dioxin exposures each year in the United States given the levels of background dioxin exposures (61 FR 17477). The commenter says it is premature for the proposed rule to incorporate either the dose of 0.01 pg/kg/day as associated with an upper bound excess cancer risk of 10^{-6} or the derived 600 annual cancer deaths attributed to dioxin exposures given the controversy about how low-dose dioxin cancer risks should be modeled. At this time, neither of these conclusions are scientifically supportable to the point where it is appropriate to include them in a discussion of human health benefits resulting from control of dioxin emissions.

***Response 391**: The annual number of cancer cases cited by the commenter was EPA's estimate in the preamble to the proposed rule of the potential cancer incidence from background exposures to dioxins, only a portion of which might be attributable to HWCs. The estimate was based on a risk-specific dose of 0.01 picograms on a toxicity equivalent (TEQ) basis per kilogram body weight per day, corresponding to an upper-bound lifetime excess cancer risk of 1 in 1 million and an average dietary intake of 120 picograms TEQ per day. Dietary intakes of dioxins have generally declined, and EPA now estimates an average dietary intake of approximately 40 pg TEQ per day for adults. This estimate is based on updated food intake rates and measurements of dioxin levels in food obtained from recent national surveys of beef, pork, poultry, and milk.*

*The risk-specific dose of 0.01 pg TEQ/kg-day was EPA's estimate in the 1994 draft dioxin reassessment. However, EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment should be used (*Health Assessment Document for Polychlorinated Dibenzo-p-dioxin, *U.S. EPA, 1985). That estimate is 156,000 [mg/kg-d]$^{-1}$. This corresponds to a risk-specific dose of 0.06 pg TEQ/kg-day for a 1 x 10^{-6} lifetime excess cancer risk. In response to comments received from the SAB, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d). This study lends considerable support to the earlier cancer potency estimates from the 1994 draft reassessment and the 1985 health assessment.*

For these and other reasons, EPA is no longer using the cancer incidence estimate mentioned in the preamble to the proposed rule. For the final rule, EPA estimated the

annual incidence of cancer associated with dietary intake of dioxins specifically from HWC sources. This was done by estimating the amount of dioxin contained in locally produced meat and milk and assuming that these products enter the national food commodity distribution system and are consumed by the general population. However, as explained in the risk assessment Background Document (RTI, 1999), the analysis for the final rule does not consider long-range transport of dioxins emitted from HWCs. Only exposures resulting from agricultural commodities produced within 20 kilometers of HWCs were considered. Therefore, EPA expects that its cancer incidence estimate understates the true cancer incidence in the general population and that the degree of underestimation could be significant.

***Comment 1056, 1057*:** For evaluating the possible carcinogenicity of dioxins and furans, the draft dioxin reassessment relies exclusively on the linear multistage model (LMS) which assumes no threshold. Based on this model, the draft dioxin reassessment derives a proposed revised cancer potency factor (CPF) for TCDD of 0.10 ng/kg/day, a 64 percent reduction from the previous CPF of 0.156 ng/kg/day. However, the SAB did not unequivocally endorse the use of the LMS model for assessing possible dioxin carcinogenicity: The commenter cites a statement by the SAB on the draft dioxin reassessment that a threshold model would provide an equivalent or nearly equivalent description of the data as EPA's preferred linear model and that this is the most important issue in the dose-response modeling.

The draft dioxin reassessment focuses exclusively on highly exposed cohorts from several occupational studies (primarily the NIOSH study by Fingerhut et al., 1991) as the underlying human data supporting the Kociba et al. (1978) study in rats for cancer risk extrapolations and estimates of possible excess cancer risks in the general population. However, consistent with the recommendations of the SAB that a threshold model for cancer be considered, the lack of excess cancer among less exposed subcohort in the NIOSH study must be considered. In this study, only in the cohort of workers with greater than one year of exposure to TCDD (and > 20 years of latency) were excess cancers observed and even this finding is controversial due to multiple confounding factors from smoking and exposure to asbestos and other potentially carcinogenic chemicals. However, in the cohort of workers with less than one year of exposure to TCDD (also with > 20 years of latency) no excess cancer was observed. These human data, in a cohort with initial estimated TCDD serum levels of 640 ppt, appear to highlight the rationale behind the SAB's recommendation that EPA consider a threshold approach for estimating possible carcinogenicity of dioxin and related compounds. In reviewing EPA's assessment of the epidemiology database, the SAB appears to concur with this interpretation. The commenter cites a statement in the SAB report that calls on EPA to comment on questions of external validity as sources of uncertainty, the most important of which are the high exposure to low exposure generalization, as well as on the relationships between agricultural and forestry and environmental exposure levels and the cancers observed at those exposure levels. The commenter says the basis for this statement appears to be the fact that the studies referred to (i.e., agricultural and forestry and

environmental exposure) do not demonstrate an association between exposure to dioxin and cancer. The low exposure cohort in the NIOSH study would be another example of this.

While not available during the time that the draft dioxin reassessment was being prepared, the most recent data from the extensive studies of the Ranch Hand are also relevant. Since the various exposed Ranch Hand cohorts cover the spectrum of exposure with initial TCDD serum levels ranging from approximately 25 to 300 ppt, there is hardly a more relevant database to determine whether exposures well above background levels are likely to result in excess risk of cancer. The results of the most recent evaluation of the Ranch Hand cohort for possible carcinogenicity associated with exposure to TCDD appear to support the SAB's concern and recommendation that EPA consider low dose exposure cohorts and a threshold-type model for cancer. In the 1992 Ranch Hand examination results, now with more than 20 years of latency since exposure to TCDD in Southeast Asia, there were no statistically significant group differences for any neoplasm. The conclusions of this recent evaluation regarding a possible association between exposure to dioxin and cancer are relevant: The commenter cites a statement in Wolfe et al., 1995, that at the end of a decade of surveillance and more than 20 years after the last exposure to Agent Orange in Vietnam, Ranch Hands and comparisons appear to be at equal risk for the development of all forms of neoplastic disease and there is no evidence to suggest a positive dose-response relationship between body burden of dioxin and neoplastic disease.

Given the clear recommendations by the SAB for EPA to consider a threshold-type cancer risk assessment for dioxin, it is premature to focus exclusively on non-threshold models as the sole basis for assessing possible carcinogenicity. Until this critical issue is resolved in the revised dioxin reassessment, it is premature to use any cancer potency factors (i.e., 0.156 ng/kg/day or 0.10 ng/kg/day) for dioxin in the risk assessment in the proposed rule. It must be assumed that EPA will take seriously the suggestion of the SAB to consider a threshold model for cancer risk assessment. Therefore, the proposed rule should not adopt the LMS approach for dioxin cancer risk assessment or any conclusions which derive from it until after EPA has responded to the SAB's suggestions.

> ***Response 1056, 1057:*** *In its review of the 1994 draft dioxin reassessment, the EPA Science Advisory Board suggested that EPA consider alternatives to the linear nonthreshold model, allowing for minimal response at low environmental levels of exposure, and which would be consistent with the body of available health effects data. In response, EPA developed a mechanistic model for liver tumors in female rats using data from a 2-year feeding study in Sprague-Dawley rats (Kociba et al., 1978). EPA also summarized the results of simple empirical models that have been applied to other significant cancer findings in female Sprague-Dawley rats from the Kociba study and in Osborne-Mendel rats and B6C3F1-mice from the National Toxicology Program study (NTP, 1982). In addition, EPA applied simple empirical models to evaluate the shape of the dose-response curve for significant noncancer findings from other animal studies. EPA compared the shapes of the dose-response curves across all the studies and grouped them into those that appeared to be linear across the experimental findings and those*

that appeared to be nonlinear (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d) EPA found that a majority of the dose-response curves are consistent with linearity but that some are highly nonlinear, appearing to have a clearly defined threshold. However, most of the cancer findings (9 of 13, or 70 percent) exhibited response consistent with linearity in the observable range.*

Also, few of the mechanistic models that EPA identified in the scientific literature exhibited nonlinear dose-response in the observable region or predicted nonlinear dose-response in the low-dose (extrapolation) region. Results of the two-stage modeling of the Kociba et al. female rat liver tumor data incorporating dioxin-altered hepatic foci data to estimate mutation and growth parameters provide nearly the same low-dose estimates as the linearized multistage (LMS) model using only the tumor data. Unless a protective effect of TCDD on mutation rates occurs at low doses, low-dose risk will remain proportionate to exposure and consistent with the linearized multistage model. If protective effects are allowed in the model, the low-dose risks may be substantially reduced; however, the focal lesion data and the biochemical markers generally agree and do not suggest a protective effect (U.S. EPA 1997d).

Therefore, there appears to be no strong support for general nonlinearity for TCDD's effects in the range of the data studied and little support for extrapolation into a lower dose range with a highly nonlinear model. To the contrary, EPA believes there are sufficient data suggesting that response is proportional to dose that, when considered together with the available human data, warrant concern that 2,3,7,8-TCDD will induce toxic effects in humans in the range of the experimental animal data. Furthermore, based on a lack of data indicating an immediate and steep change in slope of the dose-response curve, EPA believes the possibility of response 1 to 2 orders of magnitude below this range must be considered.

As the SAB pointed out in its report on the draft dioxin reassessment, understanding the operation of bias and chance is especially important when interpreting "negative" results from an epidemiological study, where no differences are apparent or where the differences are not statistically significant. Differences produced by real effects can easily be masked by poor exposure classifications (misclassification bias), a negative association may appear merely by chance by virtue of a small population available for study (poor statistical power), and potential risks can be rendered undetectable by observing the exposed population for too short a time (bias produced by failure to account for adequate latency). These are just a few of the factors complicating interpretation of negative outcomes. On the other hand, spurious positive associations are much less likely to occur in environmental epidemiological studies because more factors operate to lower the observed risks than to raise them. With respect to the

National Institute of Occupational Safety and Health (NIOSH) study (Fingerhut et al., 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"), no association was reported for the cohort exposed for a year or less. However, such an outcome is not unexpected given that the probability of observing cancer in this cohort is so much less than in the more highly exposed cohort. Therefore, EPA does not consider the NIOSH study to be evidence of a threshold of effect.

On the contrary, EPA's analysis of the NIOSH data done in response to the SAB review of the draft dioxin reassessment found that the ratio of the IADD (Intake Average Daily Dose) for the high- and low-dose groups was the same as the ratio of increased risk for respiratory cancer, indicating a linear dose-response curve. The results for all cancer mortality combined were less clear. However, a similar comparison for total cancer risk using data from the Manz et al. (1991) cohort was consistent with linearity ("Cancer mortality among workers in chemical plant contaminated with dioxin"). The IADD is the continuous dose which yields the same average area under the curve (AUC) for serum concentration versus time and was used as the dose metric in EPA's analysis (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d).*

In addition to the NIOSH study, there are epidemiological studies of a number of other cohorts that have found a positive association between dioxin exposure and cancer. These include cancers at many different sites, including malignant lymphomas, soft tissue sarcomas, hepatobiliary tumors, hematopoietic tumors, thyroid tumors, and respiratory tract tumors. These studies include: Hooiveld et al., 1998 ("Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants"); Kogevinas et al., 1997 ("Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: an expanded and updated international cohort study"); Becher et al., 1996 ("Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"); Kogevinas et al., 1995 ("Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: two nested case-control studies"); Kogevinas et al., 1993 ("Cancer incidence and mortality in women occupationally exposed to chlorophenoxy herbicides, chlorophenols and dioxins"); Bertazzi et al., 1993 ("Cancer incidence in a population accidentally exposed to 2,3,7,8-tetrachlorodibenzo-para-dioxin"); and Zober et al., 1990 ("Thirty-four year follow-up of BASF employees exposed to 2,3,7,8-TCDD after the 1953 accident"). Although the Ranch Hand study has yet to find evidence of an increased incidence of cancer, this cohort was not as highly exposed as the other cohorts studied. However, other health effects have been observed in the Ranch Hand veterans, including an increased incidence of diabetes, elevated cardiovascular disease, and a modest decrease in testosterone levels in the most highly exposed veterans.

Overall, EPA believes there are sufficient human and animal data suggesting that response is proportional to dose to warrant continued use of the linearized multistage model. EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment (U.S. EPA, 1985) should be used. That estimate, which is based on the Kociba et al. (1978) study, is 156,000 [mg/kg-d]$^{-1}$. EPA notes that the 1997 analysis of the human and animal data cited above lends considerable support to the 1985 estimate.

***Comment 392**:* The commenter states that the SAB was skeptical about a number of issues involving the use of the TEF/TEQ methodology. In relying on EPA's cancer risk estimate for dioxins on a TEQ basis, the commenter cites the proposed MACT rule, which indicates that "[t]oxicity equivalence is based on the premise that a series of common biological steps are necessary for most if not all of the observed effects, including cancer, from exposures to 2,3,7,8 chlorine-substituted dibenzo-p-dioxins and dibenzofuran compounds in vertebrates, including humans." The commenter notes that the relevance of this statement as the basis for relying on the TEQ methodology is questionable given the SAB's review of this same statement: "This pronouncement is too strong. Virtually all of the Committee believes that it is more accurate to state that binding of TCDD and related compounds to the Ah receptor is a marker of exposure, but has not yet been established to be necessary for the induction of several of the observed effects."

The commenter notes that while the TEQ methodology may be useful for estimating exposure, there is currently so much controversy and uncertainty in underlying assumptions, individual TEF values, and the role of the Ah receptor in certain postulated health outcomes that it should not be used for possible health effects assessment until some of the legitimate issues are resolved. The commenter points out that in reviewing the TEF/TEQ methodology, the SAB was clearly unwilling to accept its unqualified use without additional work on the part of EPA. The commenter states that the SAB also noted the presence of new data which have resulted in adjustments to several of the TEF values. The commenter states that some of the "new data" noted by the SAB were actually available during the time that the draft dioxin reassessment was in preparation (e.g., Birnbaum et al., 1993, "Dose Response Relationships for EROD Induction in Liver, Lung and Skin for Dioxin and Dibenzofurans," *DIOXIN93*) and that, given the implications of these data (which were developed in EPA's own laboratories), they should have been used for several critical conclusions in the draft dioxin reassessment despite not having been published. The commenter points out also that the SAB was skeptical that the TEQ methodology could be used to assess all toxic endpoints possibly associated with exposure to dioxins or furans.

The commenter asserts that in reviewing the draft dioxin reassessment, the SAB concluded that Chapters 8 and 9 needed "substantial revisions" while Chapters 1-7 were adequate. The commenter states that assuming that Chapters 1-7 remain essentially unchanged from their present draft versions, it must be noted that numerous statements in these chapters appear to refute the key underlying concept of the TEF/TEQ methodology (which was specifically rejected by the SAB) that the Ah receptor

participates in every biological response to TCDD. The commenter notes that the implications of this position in the draft dioxin reassessment are that whatever the degree of Ah receptor participation in the initiation of a biological response (e.g., enzyme induction), this same degree of participation carries through to all known endpoints. The commenter states that while future research may or may not validate such an inference, based on what is known today, it is scientifically inappropriate to assume that the Ah receptor participates, proportional to the assigned TEQ for each congener, in every biological response to TCDD, particularly at the low doses of regulatory concern.

The commenter points out that as acknowledged numerous times throughout the draft dioxin reassessment, few, if any, of the key adverse health endpoints highlighted as potentially occurring at or near background levels of exposure (i.e., altered levels of circulating reproductive hormones, immunotoxicity, reduced glucose tolerance) have been demonstrated to be Ah receptor-mediated responses. The commenter states that if they are not mediated by the Ah receptor, it is inappropriate to apply the TEQ concept to these effects as if they were Ah receptor-mediated. The commenter cites the draft dioxin reassessment as indicating that conclusive evidence that all known effects of TCDD are Ah receptor-mediated is still lacking. The commenter notes that Chapters 1-7 support this conclusion for a number of the specific endpoints highlighted in the risk characterization chapter of the draft dioxin reassessment, and that various statements in these chapters (in agreement with the view of the SAB) demonstrate that the Ah receptor either does not or is not known to participate in all effects. For example, in Chapter 4 (p. 4-19), in reviewing potential immunotoxic effects, "…host resistance is often accorded the 'bottom line' in terms of relevant immunotoxic end points." However, 3 pages later we discover that "...the role of the Ah receptor has not been addressed in terms of host resistance models...." The commenter states that since the host resistance studies form the central basis for the inference that animal data demonstrate potential adverse effects on the human immune system and lacking essentially any confirmation that the TEQ methodology for assessing this endpoint is relevant, it is unwarranted (or at least premature) to use the TEQ methodology to assess potential immunotoxic risks. Despite refinements in TEF values for individual dioxin and furan congeners, there continues to be uncertainty whether the Ah receptor is involved in many of the key noncancer health endpoints of purported concern. The commenter believes that given this uncertainty, and the unlikely event that it will be resolved even after the draft dioxin reassessment is finalized, caution needs to be exercised in assuming that the TEF/TEQ methodology can be uncritically applied to all endpoints of concern.

The commenter states that the unresolved uncertainty about the TEQ methodology argues strongly that the proposed MACT rule should not incorporate any use of the TEF/TEQ methodology until EPA has followed the advice of the SAB "...to include a peer reviewed appendix that will comprehensively review EPA's use of the TEF/TEQ approach in the exposure and health assessment documents." The commenter believes that the various conclusions from the SAB concerning the TEF/TEQ methodology and underlying assumptions underscores the need for additional research and reflection before this methodology can reliably be applied to either exposure or risk assessment issues in the proposed MACT rule. The commenter feels that use of either the current outmoded TEF values

for many dioxin/furan congeners of concern or reliance upon EPA's current version of the TEQ/TEF methodology prior to implementation of the SAB's recommendations is unwarranted.

Response 392: *EPA acknowledges that there are uncertainties associated with the use of the TEQ approach. In its review of the 1994 draft health assessment document (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds*, U.S. EPA, 1994d,e), the SAB called for clarifications in the specification of TEFs for the various dioxin-like compounds for various health outcomes. However, the SAB also concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds. The WHO review, which was recently published in* Environmental Health Perspectives *(Van den Berg et al., 1998), resulted in recommendations for changes to the values of several of the TEFs. The WHO TEFs reflect a consensus of the international community and, for this reason, were used by EPA in the risk assessment for the final rule.*

The TEF concept derives from mechanistic research indicating that binding to the aryl hydrocarbon (Ah) receptor is the initial event for many, if not all, of the toxic effects associated with dioxins. EPA acknowledges that the TEF approach is based on certain assumptions and limitations. Although the mechanism of action of dioxins is not completely understood, it has been extensively studied. To date, the evidence supports the premise that toxicity is related to Ah induction for a number of effects. These include lethality in guinea pigs (Eadon et al., 1986); body weight loss and thymic atrophy (Safe, 1987); epidermal responses (Knutson and Poland, 1982); suppression of splenic antibody response to sheep red blood cells (SRBC) (Kerkvilet et al., 1985); antiestrogenicity (Krishnan and Safe, 1993); and teratogenicity (Weber et al., 1985). Furthermore, the genetic data using inbred mouse strains differing only in the Ah locus strongly support the role of the Ah receptor as an initiating event for dioxin toxicity (ATSDR, 1998). While it is true, as pointed out by the SAB, that binding to the Ah receptor has not been established as necessary for the induction of all toxic effects, it is important to point out that the TEFs only apply to Ah receptor-mediated responses.

The WHO panel of experts also reviewed the uncertainties related to the TEQ approach. These included nonadditive interactions, differences in shapes of the dose-response curves, and differences in species' responsiveness. This panel concluded that the TEF model is both plausible and the most feasible approach for risk assessment of dioxin-like compounds. The panel also concluded that, in view of the available scientific evidence from studies of mixtures of dioxin-like compounds, use of the TEFs are not expected to

result in large errors in estimating concentrations of TEQs or responses at environmentally relevant levels.

EPA did not use the TEF/TEQ methodology to assess any specific noncancer endpoints in the risk assessment for the final rule. Additional research is needed to gain a better understanding of the underlying molecular mechanisms responsible for several key noncancer effects (e.g., immunotoxicity, neurobehavioral toxicity, and female reproductive toxicity) in order to develop biologically based dose-response models that can be used for noncancer risk assessment. Instead, EPA used a modified margin of exposure (MOE) approach to compare predicted exposure levels against background levels. The MOE approach, which incorporates the TEF/TEQ methodology to assess exposures from all 2,3,7,8 chlorine substituted dioxins and furans, was not used to assess risk for any specific noncancer endpoint but rather as a general indicator of potential noncancer hazards from this class of compounds.

***Comment 625*:** In both the draft dioxin reassessment and in the proposed rule, EPA has ignored the fact that no population of highly exposed individuals has demonstrated a broad spectrum of effects. The commenter cites a statement from the preamble to the proposed rule which relies on a conclusion from the draft dioxin reassessment that there is adequate evidence from both human populations and laboratory animals, as well as other experimental data, to support the inference that humans are likely to respond with a broad spectrum of noncancer effects from exposure to dioxins if exposures are high enough (61 FR 17477). The commenter questions the inference that humans are likely to respond with a broad spectrum of effects when numerous human studies have demonstrated the opposite. If only animal data were available, this inference might be necessary. However, this is not the case, and despite numerous studies, there are no studies of people highly exposed to TCDD (e.g., NIOSH, Seveso) or moderately exposed (e.g., Ranch Hand, Times Beach) which have demonstrated a spectrum of effects. If exposure is high enough, anything is toxic.

The conclusion (both in the draft dioxin reassessment and now in the proposed rule) that a spectrum of noncancer effects are likely to occur “if exposures are high enough” is a questionable approach with respect to evaluating the available data. Any risk characterization can include the statement that humans are likely to respond with a broad spectrum of effects from exposure to a chemical if the dose is large enough. Given the abundant database on humans exposed to varying amounts of dioxin (i.e., TCDD), it is simply incorrect to conclude that a spectrum of noncancer effects has occurred. While a few effects of questionable clinical relevance have been reported, such findings do not constitute a “spectrum of effects.” In particular, the extensive Ranch Hand studies and the series of studies on the NIOSH cohort underscore the fact that a “spectrum of effects” has not occurred, even in people with significant exposures to TCDD.

The commenter cites a statement made by the SAB in its review of the draft dioxin reassessment that there is no reason nor sufficient evidence to reject completely the EPA’s conclusion

but it should be revised to sharpen its message, better indicate areas of uncertainty, and reflect (with appropriate caveats) the total extant database. Since this has yet to be done, it is inappropriate to incorporate the conclusion concerning a spectrum of noncancer effects into the proposed rule. In particular, EPA's inference in the draft dioxin reassessment that the "spectrum of effects" was occurring at or near background exposure levels was specifically rejected by the SAB. The commenter cites a statement made by the SAB in its review of the draft dioxin reassessment that if EPA is inferring that adverse effects in humans may be occurring near current exposure levels, it has not presented findings that support this conclusion adequately. The commenter says this statement by the SAB is particularly relevant with respect to potential exposures from dioxin/furan emissions from combustion sources. Given the obvious disagreement between the EPA and the SAB on the issue of potential noncancer effects at or near background body burden levels and the likelihood that this position may be modified in order to meet the recommendations of the SAB, it is inappropriate for this conclusion to be incorporated into the proposed rule.

Response 625: *EPA agrees with the commenter that there is insufficient evidence to conclude that adverse noncancer effects are occurring in the general population at current background levels of exposure. As indicated in the draft dioxin reassessment, the available epidemiologic data are limited in this regard due to a number of possible factors, including the absence of a large body of human exposure data for the general population; the limited ability of epidemiologic studies to detect effects in relatively small populations when exposures are low and the outcomes are rare; and the difficulty of adjusting for all potentially confounding factors. EPA believes an evaluation of hazard and risk must rely on a weight-of-evidence approach in which all available data (animal and human) are examined together, a process that often requires extrapolation of effects across various animal species as well as to humans.*

Although evidence of adverse effects in humans at current background levels is limited, adaptive changes may be occurring at these levels. In commenting on this point in its review of the draft dioxin reassessment, the SAB said that the overall impact of certain biochemical changes seen at lower levels is not fully understood and that current knowledge of the mechanisms of TCDD toxicity has not identified the biological determinants of specificity that would allow one to extrapolate toxicities across species with confidence. The SAB agreed with EPA that dioxins produce a spectrum of effects in laboratory animals depending on the dose, context of exposure, and genetic background but concluded that adverse effects attributable to chronic low-level exposure in humans have not yet been adequately demonstrated.

A number of health effects associated with dioxin exposure have been observed in the Ranch Hand veterans, including an increased incidence of diabetes mellitus and adverse effects on glucose metabolism and insulin production. Elevated cardiovascular disease and a modest decrease in testosterone levels was observed in the most highly exposed

veterans, although no statistically significant association was seen between paternal serum dioxin levels and reproductive outcomes. An increased incidence in mortality associated with diseases of the digestive system has also been reported which appeared to be related to chronic liver cirrhosis. This study is continuing.

Evidence of noncancer health effects in humans at higher exposure levels has also been found in other studies. For example, an increased risk of ischemic heart disease was observed in an international study of phenoxyacid herbicide and chlorophenol workers (Vena et al., 1998, "Exposure to dioxin and nonneoplastic mortality in the expanded IARC international cohort study of phenoxy herbicide and chlorophenol production workers and sprayers"). Analysis of over 20,000 workers' mortality records showed an overall relative risk of 1.67 for ischemic heart disease and risks increased slightly with length of exposure.

There is also evidence from human data suggesting TCDD has effects on human reproduction, thyroid hormones, and neurotoxicity. Seveso area results suggest that there were sex ratio changes in children born to parents living near the plant (Mocarelli et al., 1996, "Change in sex ratio with exposure to dioxin"). Vietnam veterans (Operation Ranch Hand), Seveso females, and workers exposed to TCDD from industrial accidents show a consistent pattern of thyroid function changes, e.g., diabetes (Henriksen et al., 1997 "Serum dioxin and diabetes mellitus in veterans of Operation Ranch Hand;" Jennings et al., 1988, "Immunological abnormalities 17 years after accidental exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin;" Pesatori et al., 1998, "Dioxin exposure and non-malignant health effects: a mortality study;" and Zober et al., 1994,"Morbidity follow-up study of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin after a 1953 chemical reactor incident"). The overall evidence from case reports and epidemiological studies shows that exposure to CDDs is associated with central and peripheral nervous system changes shortly after exposure, although long-lasting abnormalities have not been found (Assennato et al., 1989, "Followup of subjects who developed chloracne following TCDD exposure at Seveso").

For the final rule, EPA used a modified margin of exposure approach to assess noncancer risks from dioxins. In this approach, the estimated average daily dose for HWCs is compared to background exposures in the general population. As a measure of risk, this "incremental margin of exposure" presupposes that if exposures are small relative to background, then risks from these exposures are likely to have limited significance for human health.

Comment 394: The SAB concluded that one of the EPA's key inferences in the draft dioxin reassessment concerning a smaller margin of exposure than previously estimated was "speculative and needs to be re-examined." Citing the preamble to the proposed rule (61 FR 17477) and given the

views of the SAB on this issue, the commenter says it is unfortunate that the proposed rule uncritically incorporates this conclusion from the draft dioxin reassessment. Only very few specific sentences in the dioxin reassessment were singled out by SAB, and this particular conclusion was specifically highlighted by the SAB as a problem area. Citing a statement in the draft dioxin reassessment that it is not currently possible to state exactly how or at what levels humans in the population will respond, but the margin of exposure (MOE) between background levels and levels where effects are detectable in humans in terms of TEQs is considerably smaller than previously estimated, the commenter asks what "previous estimates" are being referred to. If these are estimates made by EPA in another document, these should be provided or at least cited. However, since the SAB concluded that this conclusion is "speculative and needs to be reexamined," it is clearly unwarranted to include it in the proposed rule until EPA has reexamined and justified this conclusion.

> ***Response 394:*** *EPA concedes that sufficient supporting information may not have been provided for the statement originally made in the draft dioxin reassessment, as cited by the commenter. Therefore, EPA has removed the statement from the preamble to the final rule. EPA recognizes the difficulty of interpreting the available health effects information and drawing inferences regarding risks to human health. For this and other reasons, EPA has not developed a "reference dose" for 2,3,7,8-TCDD. Instead, EPA used a margin of exposure analysis for the final rule that compared the estimated average daily dose for HWCs to the average daily dose in the general population. As a measure of risk, this "incremental margin of exposure" presupposes that if exposures are small relative to background, then risks from these exposures have limited significance for human health.*

Comment 395, 1084: In the proposed rule, EPA inappropriately relies on the Seveso data to support a concern for potential carcinogenicity. The commenter cites a statement in the preamble to the proposed rule that the most well known incident of environmental contamination with dioxins occurred in Seveso, Italy, in an industrial accident and that, since then, significant increases in certain types of cancers have been observed (61 FR 17477). The commenter says it is inappropriate to cite results from the Seveso studies as though they provided support to the conclusions of the draft dioxin reassessment or the proposed rule concerning possible carcinogenicity following exposure to dioxin. Any reliance upon results of the latest two studies in the Seveso population is unwarranted. These two studies from Bertazzi's group on the cancer incidence in the Seveso population (i.e., children ages 0-19 and adults ages 19 and older) purport to demonstrate a cause and effect association between exposure to TCDD and subsequent development of cancer at a variety of sites as of 1986. However, taken together, these studies show no consistent pattern of elevated cancers between zones, sexes, or populations (young and adult).

The first study in adults (Bertazzi et al., 1993) is a cancer incidence study of people living in the vicinity of Seveso at the time of the accident. For purposes of cohort classification, all persons-years were attributed to the initial zone of residence (i.e., A, B, or R). The results reported were only for 10

years of latency and not 20 years as is typically believed to be necessary for chemically induced cancers. This study shows no logical pattern of dose-response and the results (increased incidence of hepatobiliary cancer and hematopoietic cancer) generally do not agree with the findings in the NIOSH cohort or any of the other occupational studies. While an increase in soft tissue sarcoma (STS) was reported in Zone R, this finding makes no sense on dose-response considerations alone. This finding is also inconsistent with the occupational cohort studies of Manz et al. (1991), Saracci et al. (1991), and Zober et al. (1990), none of which reported elevated mortality from STS. The increased STS reported in Fingerhut et al. (1991) was not only questioned by the authors, but additional analysis by Collins et al. (1993) suggests that simultaneous exposure to paraaminobiphenyl (PAB) was possibly a contributor to the observed increased.

The other recent Seveso study (Pesatori et al., 1994) is a review of cancer occurrence in approximately 20,000 people aged 0-19 years who lived in the various contaminated areas surrounding Seveso. Because of small sample sizes in zones A and B, all three contaminated zones (A, B, and R) were combined into a single group for purposes of this analysis which eliminates any possibility of determining a dose-response relationship between exposure to TCDD and outcome. The rationale for aggregating individuals aged 0-19 in this study and all others in the other study is not explained. It is possible that the results in both studies might be a function of where the age distribution was divided. The most important point about this study is the fact that none of the relative risks reported were statistically significant. Since no data are reported on average soil levels of TCDD in the "non-contaminated" reference zone, it is impossible to demonstrate that there are exposure differences between the majority of the exposed cohort and the reference cohort. Finally, none of the cancers discussed (i.e., ovary, brain, thyroid, Hodgkin's lymphoma, and leukemia) were elevated in the companion study in adults or in any of the occupational cohort studies.

The results of the various epidemiological studies on the Seveso population conducted to date should not be used in assessing possible cancer risks associated with exposure to TCDD. While these studies may prove useful in the future, without better data on possible exposures to assess dose-response relationships, and until the data following at least 20 years of latency are published, the results after only 10 years of latency are not a valid basis from which to infer possible effects. Currently, the available Seveso data fail to satisfy most of the recognized causation criteria (i.e., strength of association, dose-response, consistency of the association, specificity of the association, and coherence of the evidence).

> ***Response 395, 1084****: EPA recognizes the limitations of the Seveso studies and for this and other reasons EPA does not cite the Seveso incident in the preamble to the final rule. This 1976 accident is the most widely studied release of 2,3,7,8-TCDD primarily involving residential exposures. It was estimated that more than 1.3 kg of 2,3,7,8-TCDD was released into the atmosphere and that more than 17,000 people in a 2.8-km^2 area adjacent to the facility were exposed. To investigate this accident, the contaminated area was separated into regions A, B, and R based on soil levels of 2,3,7,8-TCDD. The regions*

had population sizes of 736, 4,737, and 31,800, respectively. These regional groupings do not take into consideration actual exposure levels and differences in within-zone 2,3,7,8-TCDD exposure. Zone B showed the most consistent pattern of cancers of the digestive and lymphohemopoietic systems. Cancer incidence results reported in 1976 and 1991 have been supported in the most recent studies of Seveso (Bertazzi et al., 1998,"The Seveso studies on early and long-term effects of dioxin exposure: a review").

EPA understands well that several inconsistencies and confounding factors exist for the results from the Bertazzi and Pesatori Seveso studies: the pattern of cancer cases does not support a dose response relationship (Bertazzi); childhood risks for Hodgkin's lymphoma, myeloid leukemia, and thyroid cancer for the exposed population were raised, but lacked statistical significance (Pesatori); latency periods, to date, may be too small to detect increases in cancer risks (Bertazzi and Pesatori); the population size in the highest exposed region may be too small to show possible cancer rate increases; and the exposure to dioxin was accompanied by exposure to other possible carcinogens. The weight of evidence for dioxin carcinogenicity from the Seveso studies, however, has shown a consistency of increased cancer risks over the past 23 years. Although the highest exposure group is small and has not shown significant increases in cancers, results for the second highest exposure group, Zone B, do support the cancer hypothesis. Both EPA and the international community have reviewed dioxin-cancer studies and have found strong causal evidence from animal studies and the cumulative evidence from other epidemiological studies that occupational exposure to dioxin-containing herbicides strongly points to dioxin carcinogenicity.

Despite the limitations and uncertainties surrounding the Seveso studies, there is ample epidemiologic evidence of a positive association between dioxin exposure and cancer that involve cancers at many different sites, including malignant lymphomas, soft tissue sarcomas, hepatobiliary tumors, hematopoietic tumors, thyroid tumors, and respiratory tract tumors. These include Fingerhut et al., 1991 ("Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"); Hooiveld et al., 1998 ("Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants,"); Kogevinas et al., 1997 ("Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: an expanded and updated international cohort study"); Becher et al., 1996 ("Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"); Kogevinas et al., 1995 ("Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: two nested case-control studies"); Kogevinas et al., 1993 ("Cancer incidence and mortality in women occupationally exposed to chlorophenoxy herbicides, chlorophenols and dioxins"); Bertazzi et al., 1993 ("Cancer incidence in a population accidentally exposed to 2,3,7,8-tetrachlorodibenzo-para-dioxin"); and Zober et al., 1990

("Thirty-four year follow-up of BASF employees exposed to 2,3,7,8-TCDD after the 1953 accident").

With respect to the concerns about soft tissue sarcomas (STS), there is a general indication that exposure to phenoxy acids with known dioxin contamination are associated with STS. While the New Zealand studies (e.g., Smith and Pearce, 1986, "Update on soft tissue sarcoma and phenoxy herbicides in New Zealand") did not find an increase in STS, other reports do suggest an increased risk. For example, Kramarova et al., 1998 ("Exposure to Agent Orange and occurrence of soft-tissue sarcomas or non-Hodgkin lymphomas: an ongoing study in Vietnam"), discusses an association between STS and phenoxy herbicide exposure in Sweden. The NIOSH study also provides evidence (Fingerhut et al., 1991) for significant increases in death from STS in the high-exposure cohort, although EPA acknowledges the concerns raised by the commenter regarding the findings of this study. Both Saracci et al. (1991) and Kogevinas et al. (1997) report increased STS rates for exposed workers in several countries, although EPA also recognizes the weaknesses that exist in these studies. In the case of Kogevinas et al. (1997), the increase in mortality for STS was not statistically significant and neither the latency periods nor the length of probable exposure to TCDD were associated with increased mortality or incidence of STS. Kramarova et al., 1998, also report an increased risk for STS from an analysis of the combined results of an analysis that combines the results from U.S. pesticide workers (Fingerhut et al., 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"), German workers (Becher et al., 1996,"Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"), and Dutch workers (Hooiveld et al., 1996, "preliminary results of the second follow-up of a Dutch cohort of workers occupationally exposed to phenoxy herbicides, chlorophenols and contaminants"). EPA also notes that other health effects have been observed in the Ranch Hand veterans, including an increased incidence of diabetes, elevated cardiovascular disease, and a modest decrease in testosterone levels in the most highly exposed veterans.

Despite the limitations of the various studies, EPA believes the weight of the evidence supports the conclusion that 2,3,7,8-TCDD and other dioxin-like compounds are probable human carcinogens. The SAB, in its review of the draft dioxin reassessment, agreed, concluding that dioxin is likely to increase human cancer incidence under some conditions of exposure. The SAB categorized dioxin and dioxin-like compounds as a B1 carcinogen under the 1986 EPA cancer guidelines (U.S. EPA, 1986) of "agents for which there is limited evidence of carcinogenicity from epidemiologic studies." In addition, the International Agency for Research on Cancer (IARC) recently reviewed the experimental data and epidemiological studies and also concluded that 2,3,7,8-TCDD is carcinogenic to humans (WHO, 1997; McGregor et al. 1998).

Comment 396, 397: The commenter states that EPA has misinterpreted studies based on inappropriate data to support unwarranted beyond-the-floor controls for mercury, and that EPA's *Mercury Study Report to Congress* (Mercury Report) fails to rely on the best available data. Section 4 (Summary of Interagency Reviewers Comments and Disposition) of the executive summary of the Mercury Report provides a synopsis of various comments from other federal agencies and EPA responses to these comments. The commenter cites an EPA response to an FDA comment concerning results of certain new studies (i.e., the Faroe Islands and Seychelles Islands studies), which show no or little neurological impairment in children exposed in utero to methylmercury similar to that observed in the Iraqi population. In its response, EPA indicated that only data which are available to the scientific community and have undergone a process of peer review can be used by EPA. However, the commenter feels that this position is clearly not consistent with previous EPA positions on this issue. For example, in the recent draft dioxin reassessment, EPA liberally relied upon non–peer-reviewed data to support several health-related conclusions

The commenter points out that with respect to potential adverse effects associated with exposure to methylmercury, the executive summary of the Mercury Report notes that the RfD developed for methylmercury was supported by studies on Cree Indians in Canada and New Zealanders' consuming large amounts of fish. However, in reviewing the supporting citations for the study on New Zealanders, there are two unpublished reports from the National Swedish Environmental Protection Board. The commenter believes that the use of these unpublished results, which are not readily available to the scientific community, seemingly refutes EPA's claim that only peer-reviewed data may be used. The commenter states that while peer review is clearly an important criterion for judging the quality of scientific data, the concept of using the best science available (in conjunction with sound scientific judgment) should be acknowledged.

The commenter states that the executive summary (p. 3-21) of EPA's Mercury Report incorrectly concludes that the RfD for methylmercury was supported by studies on Cree Indians in Canada and New Zealanders' consuming large amounts of fish. The commenter feels that the two studies do not necessarily support the Iraqi study (i.e., Marsh et al., 1987) from which the methylmercury RfD was derived

On reviewing EPA's summary of the New Zealand data, it is noted that the unpublished New Zealand study from the National Swedish Environmental Protection Board is based on only 31 matched pairs, and the results appear to be more heavily influenced by socioeconomic and cultural factors than by methylmercury. The fact that even EPA concludes that "[t]he Kjellstrom studies are limited for assessing methylmercury toxicity.... [G]reater significance was seen in differences of cultural origins of the children than the differences in maternal hair methylmercury concentrations" (p. 4-61) suggests that these studies do not support the Iraqi study. Also, it is important to note that the study on the New Zealand cohort involved exposure to methylmercury from chronic consumption of fish, while the study in Iraq (from which the RfD was derived) involved a subacute exposure to methylmercury in contaminated grain.

The commenter states that the data from the studies on Cree Indians in Canada are also a questionable basis of support for the findings of the Iraqi study. Overall, the description of this study (and quite possibly the study itself) is an insufficient basis for drawing any conclusions concerning a possible causal association between neurological findings and exposure to methylmercury. The EPA review of these data does not permit a determination of the basis for the indices of exposure (i.e., hair analyses or some other exposure index). While the numbers cited probably reflect hair concentrations, the failure to note this should be corrected (p. 4-60). The notation concerning a "possible influence of alcoholism and smoking among mothers," (p. 4-60) suggests that the tenuous findings might have been influenced by these factors, particularly excessive maternal alcohol consumption. Given the well-documented fetal effects of maternal alcohol consumption, any differences between groups must be considered, and merely mentioning that such differences exist does not constitute sufficient consideration of this potential confounding variable. It is also important to note that the study on Cree Indians involved exposure to methylmercury from chronic consumption of fish, while the study in Iraq (from which the RfD was derived) involved a subacute exposure to methylmercury in contaminated grain.

The commenter feels that, despite the conclusion in the executive summary of the Mercury Report, the studies on Cree Indians in Canada and New Zealanders do not provide compelling support for the findings of the Iraqi study. The fact that both of these studies involved chronic methylmercury exposure from consuming fish while the Iraqi study involved a subacute exposure from methylmercury-contaminated grain suggests that they are not comparable. The commenter believes that the likely differences in fetal exposure from subacute and chronic exposures needs to be addressed before these data can be assumed to be comparable. In particular, the commenter points out that the likelihood of neurological effects in the Iraqi infant population exposed to methylmercury in utero occurring as a consequence of maternal toxicity (or additively with fetal exposure) makes comparisons even more tenuous. The commenter states that the fact that there was evidence of maternal toxicity in the Iraqi study renders any confirmation by the Canadian and New Zealand studies even less likely.

> ***Response 396, 397****: For the final rule, EPA continues to use the same RfD for methylmercury that was used at proposal (i.e., 1 x 10^{-4} mg/kg-d). This is the same RfD that was developed for EPA's* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c). The 1997 MRTC was subject to extensive review, including review by EPA's Science Advisory Board (SAB). The SAB endorsed retention of EPA's RfD, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data. The SAB felt that the uncertainty factor used by EPA could even be increased, thus lowering the RfD, due to concerns about the acute nature of the exposure in the Iraqi study and at least some evidence that methylmercury has a longer half-life in the brain than in the blood.*

EPA believes the RfD for methylmercury is supported by the Cree Indian (northern Quebec) and New Zealand studies. The most frequently observed neurological abnormality among the offspring of Cree Indians was delayed deep tendon reflexes (McKeown-Eyssen et al., 1983). Developmental delays in fine motor and language function were reported in children of New Zealand mothers who had consumed three fish meals per week during pregnancy (Kjellstrom et al., 1986, 1989). Both studies had limitations but provide evidence of neurodevelopmental effects following in utero exposure to methylmercury that are concordant with the effects observed in the Iraqi study. Also, because the Cree Indians and New Zealanders are fish-consuming populations, the results are particularly relevant to the U.S. population where fish ingestion is the primary route of exposure. Despite the different durations of exposure between the Iraqi, Cree Indian, and New Zealand developmental studies, exposure occurred during gestation in all three studies with similar findings, lending additional support to the neurodevelopmental effects reported in the Iraqi study.

At the time of the finalization of the 1997 MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and marine mammal–consuming populations in the Seychelles and Faroe Islands. The SAB recognized the importance of these studies, commenting that "Because these data are so much more comprehensive and relevant to contemporary regulatory issues than the data heretofore available, once there has been adequate opportunity for peer review and debate within the scientific community, the RfD may need to be reassessed in terms of the most sensitive endpoints from these new studies." Because the majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to reevaluate the RfD for methylmercury.

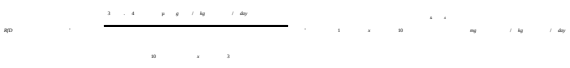
*EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data to undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy.*

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Comment 398: The commenter states that the Iraqi study (Marsh et al., 1987) of neurological effects in infants and children associated with a subacute poisoning episode is an insufficient basis for establishing an RfD for methylmercury. The proposed RfD for methylmercury in EPA's Mercury Report is based on data from a study on neurologic changes in 81 children exposed in utero after maternal poisoning from methylmercury-contaminated bread during pregnancy. The commenter believes that the Iraqi study is an inadequate basis for establishing an RfD because it presents a combination of short-duration, high exposure and exposure through a non-fish pathway. The commenter points out that despite acknowledging numerous shortcomings and uncertainties about this study, the report still concludes that the methylmercury RfD is a reasonable estimate based on currently available data. The fact that the doses of methylmercury were so high and that there was toxicity in many adults and children who consumed contaminated bread over a 3-month period further suggests that this study was not appropriate for derivation of an RfD.

The commenter notes that at the time the Mercury Report was released, the Iraqi data (Marsh et al., 1987) were not the best available data, as it appears that the massive body of data from the Seychelles Study of Fetal Methylmercury Exposure and Child Development were available (at least in a preliminary form) at the time the EPA Mercury Report was released. The commenter points out that the FDA deferred revisions to its action level for mercury concentrations in fish based on the knowledge that this study on almost 800 mother-infant pairs (compared to the 81 in the Iraqi study) was available. The commenter feels that given EPA's reliance upon unpublished data in the dioxin reassessment, any claim that the data from the Seychelles study could not be relied upon (even at the time the Mercury Report was released) because they had not been published is not a credible explanation.

The commenter feels that due to the importance of this issue, the best data should have been used in the derivation of the RfD of 1×10^{-4} mg/kg/day even if this meant a slight delay in the release of the report. The commenter notes also that, even based upon the questionable Iraqi data, there appears to be an error in the derivation of the RfD. The commenter cites the *Mercury Study Report to Congress* as indicating that a composite uncertainty factor of 10 and a modifying factor of 1 were used. The commenter notes, however, that the derivation of the RfD subsequently is shown as follows:



Based on the values given for the uncertainty factor and the modifying factor (i.e., UF = 10 and MF = 1), however, the denominator in the above equation should have been 10 and not 30. Using UF x MF = 10 in the RfD equation raises the derived RfD value to 3.4×10^{-4} mg/kg/day. It should be noted that EPA has published the incorrect RfD value of 1×10^{-4} mg/kg/day in its discussion of mercury benefits (61 FR 17478). In addition, the commenter cites the Mercury Report as indicating that confidence in the supporting database is medium in the derivation of the RfD. The commenter feels that given that data were available (i.e., the Faroe Islands and Seychelles Islands studies) to derive an RfD where both the supporting database and confidence in the RfD would be high, the failure to rely on such

data is unwarranted. The commenter believes that because appropriate studies are now available, it is no longer necessary to assume (as is necessary in accepting the proposed RfD) that a subacute poisoning exposure to high levels of methylmercury from contaminated grain is applicable to chronic consumption of fish containing low levels of methylmercury.

The commenter points out that in recent comments (April 1, 1996) from the Executive Office of the President to EPA concerning the Mercury Report, it is noted that the authors of the Iraqi study (i.e., Marsh et al.) have stated that this study is not suitable for estimating an RfD for methylmercury. The commenter feels that if correct, this admonition from the same authors involved with the Seychelles study (Marsh et al.), deserves serious consideration. The commenter cites a paper by many of the same authors (Cox et al., 1995, "Analysis of Data on Delayed Development from the 1971-72 Outbreak of Methylmercury Poisoning in Iraq: Assessment of Influential Points," *Neurotox.* 16:727-30) that further supports the concept that the data in Marsh et al. (1987) are not suitable for estimating an RfD for methylmercury is based on a rigorous reanalysis of the Iraqi data. The commenter notes that these authors point out that while the Iraqi study was important for establishing a dose-response relationship in humans, the uncertainty in the parameters of this relationship is large, the data set is limited, and the outcome measures used in this study were appropriate for the circumstances, but do not meet the same psychometric standards as modern developmental tests. The authors intended that the results of the Iraq study lead to more carefully controlled longitudinal studies. The commenter notes that the more carefully controlled longitudinal studies are available in the form of the Faroe Islands and Seychelles Islands studies. The commenter notes these studies are prospective, use the most up-to-date psychometric tests, are based on fish ingestion of low levels of methylmercury (instead of subacute exposure to methylmercury in contaminated grain), and involve chronic exposure (as opposed to subacute).

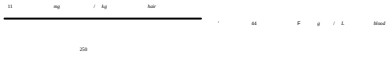
The commenter believes, therefore, that these studies, comprising 11 published articles (see *Neurotoxicology*, 1995, Vol. 16; *Special Issue: Methylmercury and Human Health*), and not the inappropriate Iraqi study (as acknowledged by the authors), should be the basis upon which an RfD for methylmercury is derived. The commenter acknowledges that while the results of all of these studies need to be carefully reviewed, the conclusions from the Seychelles Child Development Study are noteworthy. The commenter cites these results as indicative of a relationship between fetal mercury exposure and neurodevelopment in the pilot study at enrollment and at the 66-month evaluation; the results indicated that this association was highly dependent on how the data was analyzed and on a small number of outliers and influential scores. Further, the study indicated no association between mercury exposure and neurodevelopment was found at 62 months of age in the main study, one which had more extensive endpoints and covariates for statistical analysis. Although at the 19-month evaluations no associations between mercury exposure and the mental or psychomotor scales of the BSID was found, on the 29-month evaluations the activity level from the behavioral index of the Bayley Scales of Infant Development indicated an association. The results found that association only in boys, and not in the direction expected. The commenter believes that failure to use the best scientific data to establish a regulatory policy for methylmercury undermines confidence that the best science is being

used, and that even if the results of these studies conflict with the conclusions (e.g., RfD) already articulated in the draft Mercury Report, the conclusions should be altered to reflect the science.

***Response 398**: As indicated by the commenter, the RfD for methylmercury is based on the Iraqi study (Marsh et al., 1987) in which a number of people were exposed to methylmercury-treated seed grain that was mistakenly used for making bread. EPA defines the RfD as an estimate of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Latent toxicity was observed in many adults and children who had consumed contaminated bread over a 2- to 3-month period. Infants exposed in utero were the most sensitive group; infants often exhibited neurological effects, while their mothers showed no signs of toxicity. Marsh et al. (1987) examined neurological signs in 81 mother-infant pairs. The incidence of several neurological endpoints among infants, including late walking, late talking, delayed mental development or seizures, and low scores on clinical tests of nervous system function, was associated with mercury concentrations in maternal hair during pregnancy. Paresthesia was the most frequent maternal symptom during pregnancy.*

As explained in the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c), a benchmark dose approach was used to calculate the mercury level in maternal hair associated with no adverse effects in offspring. The benchmark dose is the intake of methylmercury associated with the lower bound on a 95 percent confidence interval of a dose producing a 10 percent prevalence of adverse effects. Hair concentrations were determined for periods during gestation when actual methylmercury exposure had occurred. Application of this approach resulted in the calculation of a 95 percent lower bound on the concentration of methylmercury in maternal hair of 11 ppm (11 mg/kg).*

The critical dose used for deriving the methylmercury RfD was based on the 95 percent lower bound on the maternal hair concentration of 11 mg/kg. A ratio of 250:1 (µg mercury/mg in hair:µg mercury/L of blood) was used to convert the maternal hair mercury level to a blood mercury level as follows:



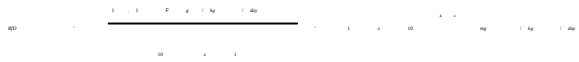
Assuming that steady-state conditions exist and first-order kinetics for mercury are being followed, the daily dietary intake, d, was calculated by:

where

d = daily dietary intake (µg of methylmercury per day)
C = concentration in blood (44 µg/L)
b = elimination constant (0.014 days^{-1})
V = volume of blood in the body (5 L)
A = absorption factor (unitless, 0.95)
f = fraction of daily intake taken up by blood (unitless, 0.05).

Solving for d results in a daily dietary intake of 64.8 µg/day. The daily dose was calculated by dividing the daily dietary intake by the average body weight of 60 kg for an adult female. The result is a dose of 1.1 µg/kg/day, which represents the total daily quantity that is ingested by a 60 kg individual to maintain a blood concentration of 44 µg/L, or a hair mercury concentration of 11 ppm. This is also called the benchmark dose.

The RfD of 1.0 x 10^{-4} mg/kg-d for methylmercury was derived by dividing the benchmark dose (1.1 µg/kg/day) by a composite uncertainty factor of 10. The uncertainty factor is applied to account for variability in the human population, in particular the variation in biological half-life of methylmercury and the variation that occurs in the hair-to-blood ratio, as well as for the lack of a two-generation reproductive study and lack of data for possible chronic manifestation of the adult paresthesia that was observed during gestation. The uncertainty factor and a default modifying factor of 1 are applied to the benchmark dose, as shown below:



Although fish consumption is the most likely route of exposure to methylmercury by the U.S. population, there are no data to indicate that methylmercury absorption is affected by food type. Despite the different durations of exposure between the Iraqi, Cree Indian, and New Zealand developmental studies, exposure occurred during gestation in all three studies with similar findings, lending additional support to the neurodevelopmental effects reported in the Iraqi study. The SAB, while recognizing the limitations of the Iraqi study, concluded in its review of the 1997 MRTC that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data, and that the current RfD should be retained.

At the time of the finalization of the 1997 MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and

marine mammal-consuming populations in the Seychelles and Faroe Islands. The SAB recognized the importance of these studies, commenting that: "Because these data are so much more comprehensive and relevant to contemporary regulatory issues than the data heretofore available, once there has been adequate opportunity for peer review and debate within the scientific community, the RfD may need to be reassessed in terms of the most sensitive endpoints from these new studies." Because the majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to re-evaluate the RfD for methylmercury.

*EPA and other federal agencies, including the FDA, participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing the EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Section 7
Chemical Manufacturers Association
RCSP-0128

***Comment 200*:** The commenter feels that, as a matter of substance, the proposal greatly overstates the quantity and even more so the risks posed by HWI emissions in an attempt to justify the arbitrary "beyond the floor" levels imposed for several pollutants. Particularly with respect to dioxins, the commenter feels that the proposal does not follow prior EPA risk assessment practice or use the most current EPA data. The commenter notes that, in fact, the risks posed by these pollutants are already at or below EPA's target levels for acceptable risks. The commenter states that Subpart O regulations and the RCRA permitting process have worked and are working.

***Response 200**: The establishment of NESHAP for sources that emit HAPs is required by Sections 112(c) and 112(d) of the CAA. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.*

The risk assessment for the final rule is based on methodology presented in the 1990 Indirect Exposure Document, Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IEM) *(U.S. EPA, 1990) and the 1993 Addendum to the 1990 Indirect Exposure Document (U.S. EPA, 1993a). These methods were further updated with respect to dioxins and mercury. For dioxins, the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist of the procedures published in the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. All parameter values used in assessing risks from dioxins were derived from the information in the draft dioxin reassessment or more recent information. For mercury, the assessment followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model developed especially for that report. For lead, EPA used the Integrated Exposure Uptake Biokinetic Model (IEUBK) (U.S. EPA, 1994c) to estimate blood lead concentrations in children. Human exposure factors were obtained from the 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a), including*

consumption rates for home-produced foods and recreationally caught fish. Therefore, EPA believes that the risk assessment for the final rule is consistent with current EPA practice and uses the best information currently available.

***Comment 205*:** The commenter states that EPA has not documented significant or widespread cancer risks from HWIs, given that a substantial number of people are not proven to be exposed to cancer risk levels greater than 1×10^{-6} and that the maximally exposed individual's (MEI) cancer risk from area sources is not proven to exceed 1×10^{-4}. The commenter notes that dioxin and metals are the only pollutants from HWIs for which EPA provides cancer risk-related data. The commenter states that although EPA claims that 600 cancer cases per year may be attributable to exposure to dioxin throughout the United States, and suggests that HWCs contribute to this increased risk, EPA does not specify the increase in cancer risk greater than 10-6 from HWCs (much less HWIs) except for "special subpopulations." However, the commenter notes that for these special subpopulations lifetime individual risk estimates from incinerators are generally below 1×10^{-6} with two exceptions: high-end risk range for the subsistence fisher scenario (ingestion) of 9×10^{-5} to 2×10^{-7}, and the farmer scenario (inhalation) of 1×10^{-6} to 1×10^{-7}.

The commenter then points out that even these risk estimates are extremely conservative, beyond EPA guidance for high-end risk. For example, the dioxin risk values are based on a "model plant" emitting at the 90th percentile for each congener, which is highly atypical (and also is biased, since EPA has differing amounts of data for the different congeners). Further, the fish ingestion pathway uses a lipid content for fish of 7 percent, when EPA data suggest an empirical range of 3-6.7 percent.

***Response 205**: In a departure from the approach used at proposal, the risk analysis completed for the final rule does not use model facilities and instead uses stratified random sampling to select a subset of facilities from the HWC facility universe for analysis using site-specific data. While the risk analysis completed at proposal evaluated 4 hazardous waste incinerators, for the final rule, the total number of incinerators evaluated was increased to 56 facilities (including 3 of the 4 incinerators evaluated at proposal). For the final rule, facility-specific emissions data were used rather than values obtained from the national emissions distribution, in the manner done at proposal. These data were obtained primarily from certificate of compliance tests and trial burn reports. When measured emissions data were not available for a specific facility, values were imputed from facilities with similar operational characteristics. Imputation was done such that emissions of individual congeners were all imputed from the same data set.*

EPA agrees with the commenter that the fish lipid content used in the proposed rule was too high to truly represent a central tendency value, as intended. For the final rule, EPA concluded that lipid content should represent a fraction of lipid in the fillet for a wide variety of species across the United States. For the human health analysis, EPA used a lipid content of 2.6 percent generated assuming 36 percent consumption of Trophic Level

3 fish (lipid content 2 percent) and 64 percent consumption of Trophic Level 4 fish (lipid concentration 3 percent).

EPA's risk analysis for the final rule indicates that cancer risks from hazardous waste incinerators are largely attributable to dioxins rather than metals. For the final rule, high-end risks from dioxins under baseline conditions are estimated to lie in a range from 7 x 10^{-7} to 8 x 10^{-6} (90th to 99th percentile) for children of dairy farmers and 5 x 10^{-6} to 4 x 10^{-5} for children of subsistence farmers. Risks are estimated to be higher for incinerators equipped with waste heat recovery boilers. High-end risks under baseline conditions are estimated to lie in a range from 3 x 10^{-6} to 2 x 10^{-5} for children of dairy farmers and 3 x 10^{-5} to 8 x 10^{-5} for children of subsistence farmers. Annual cancer incidence from all incinerators is estimated to be 0.3 cases per year under baseline conditions. However, this estimate does not account for any cancer that may result from transport of dioxin emissions beyond a distance of 20 kilometers of HWCs.

Comment 206: The commenter feels that the multipathway modeling used for subsistence fisher (ingestion) and the subsistence farmer (inhalation) scenarios employs multiple (not just one or two) very conservative assumptions, as illustrated by comparing that risk modeling to the range of values contained in the probabilistic models found in the draft *Mercury Report to Congress.* The commenter notes that the result is risk estimates far in excess of the 99th percentile.

Response 206: *EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. This is why, for the final rule, EPA characterized exposures using central tendency parameter values (rather than high-end values) in conjunction with site-specific data and used probabilistic modeling to consider the variability of these parameters. Monte Carlo simulation was used to evaluate the impact of variations in exposure parameters on individual risk for key risk-driving receptor population/constituent combinations (including beef and dairy farmers for dioxin). EPA believes that this approach ensures that the risk estimates for the HWC rule are not overly conservative.*

***Comment 207*:** The commenter feels that the risk reduction does not justify beyond-the-floor (BTF) standards based on the fact that EPA estimates the risk from dioxin/furan emissions at the MACT floor to be 5 x 10^{-5} to 3 x 10^{-9}. The commenter notes that only a few pages earlier, the preamble states that EPA's target risk level is 1 x 10^{-5}. The commenter fails to understand how EPA can view those risks as unprotective when the high-end risk from floor-level risks is in the same order of magnitude as the target risk level.

The commenter notes that the comments of the Chlorine Chemistry Council (CCC) explain at length the many ways in which the current proposal's risk assessment overstates risks from dioxin/furan

emissions, including (1) failing to use EPA's own more current research that shows that the TEFs used in this proposal are consistently overstated; (2) using cancer slope factors older than those used in the dioxin reassessment; (3) using assumptions and models from the dioxin reassessment and EPA's guidance for assessing indirect exposure risks from combustors, even though the Science Advisory Board criticized both; and (4) setting many parameters, not just one, to high-end values in determining high-end risks.

The commenter supports and endorses CCC's comments in their entirety and believes that even if none of these criticisms were valid, the difference between the floor risks and EPA's target risk level is so minor and uncertain that going beyond the floor is arbitrary and capricious.

Response 207: *The establishment of NESHAP for sources that emit HAPs is required by Sections 112(c) and 112(d) of the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6) of the CAA. EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in CAA Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing incinerators based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. However, Sections 3004(a) and (q) of RCRA mandate that standards governing the operation of HWC facilities be protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate whether the MACT standards satisfy this requirement in order to determine what, if any, RCRA standards for emissions from these sources may be needed. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.*

EPA modified the TEFs used in the final rule to be consistent with the most recent TEFs recommendations of an international panel of experts convened under the auspices of the World Health Organization (WHO) (Van den Berg el al., 1998). The WHO panel of experts reviewed all the available data on the relative toxicities of dioxin-like compounds and made recommendations for changes to the values of several of the TEFs from the 1989 interim values. The WHO TEFs reflect a consensus of the international community and, for this reason, were used by EPA in the risk assessment for the final rule.

With respect to the cancer slope factor, because the dioxin reassessment is not yet completed, EPA is not relying upon the conclusions of the 1994 assessment. However, in response to comments received from the Science Advisory Board, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response

Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d). This study lends considerable support to the earlier cancer potency estimates from the 1994 draft reassessment and the 1985 health assessment. However, EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment should be used (*Health Assessment Document for Polychlorinated Dibenzo-p-dioxin, *U.S. EPA, 1985). That estimate is 156,000 [mg/kg-d]$^{-1}$. This corresponds to a risk-specific dose of 0.06 pg toxicity equivalency quotient (TEQ)/kg-day for a 1 x 10^{-6} (or 1 in 1 million) lifetime excess cancer risk.*

*EPA is also not relying on other conclusions of 1994 draft dioxin reassessment because the assessment is not yet completed. However, EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available, and EPA continues to rely on it for the final rule. This includes the information contained in the 1994 dioxin exposure assessment (*Estimating Exposure to Dioxin-like Compounds *(external review draft), U.S. EPA, 1994a,b), including methods and data that were developed for assessing indirect exposures. EPA notes that the SAB commended EPA for its work on the dioxin reassessment, calling the estimating exposures document "a very credible and thorough job."*

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the Science Advisory Board. EPA intends to respond to the comments in an appropriate forum.

The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (*Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), as well as other sources of information.*

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board. EPA intends to respond to comments on the draft Addendum that it has received from the public, as well as from the Science Advisory Board, in an appropriate forum.

For the final rule, EPA modified the exposure scenarios to be more representative of the exposed population at each facility. The risk analysis for the final rule used site-specific

emission estimates to estimate media concentrations and U.S. Census and Census of Agriculture data to locate exposed individuals. For the final rule, EPA generated population-weighted cumulative distributions of risks to individuals, thereby eliminating the need to define central tendency and high-end exposure scenarios deterministically, in the manner done at proposal. EPA used central tendency exposure factors to estimate exposures, except for a few risk-driving pathways for which EPA performed an exposure factor variability analysis using Monte Carlo simulation. Separate exposure estimates were made for individuals engaged in farming as an occupation and persons engaged in farming for subsistence. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative.

Comment 0128-1: The commenter states that EPA has not put emissions from HWCs into proper perspective. Specifically, EPA failed to put PIC and PM_{10} emissions into proper perspective.

Response 0128-1*: For the final rule, EPA evaluated all chemical constituents for which adequate emissions data were available. Although EPA assessed the risks from chlorinated dioxins and furans for the final rule, risks from other organics that may be present as PICs could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans emissions measurement data of acceptable quality for nondioxin PICs are quite limited, and the data are highly variable and are therefore inadequate for making national emissions estimates. A number of PICs known to be emitted from HWCs are human carcinogens (e.g., hexachlorobenzene, benzo(a)pyrene, and others). Omission of quantitative risk estimates for these HAPs may have resulted in the potential risks being understated in the risk assessment for the final rule.*

As best as can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA anticipates that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

For the final rule, EPA also assessed the risks associated with particulate matter emissions ($PM_{2.5}$ and PM_{10}) apart from the chemical specific risks from individual chemical constituents, as the commenter suggested. This analysis evaluated the incidence of PM related health effects avoided by reducing PM emissions as a result of the MACT standards. Health endpoints considered included mortality, hospital admissions, and respiratory symptoms.

***Comment 456*:** The commenter states that EPA seems to have twisted science in interpreting mercury background fish data. The commenter cites the proposed rule in which EPA describes the collection and analyses of fish tissue for mercury. According to that description, chemical residues in fish data were collected from 388 locations nationwide; at 92 percent of the locations fish were found to contain detectable levels of mercury. The commenter cites EPA's comment regarding the location of the highest concentration detected (1.8 ppm) and the conclusion that the remote site could be considered to represent background conditions. The commenter also cites EPA's statement that similar results obtained in other studies strongly suggest that long-range atmospheric transport and deposition of anthropogenic emissions is occurring.

The commenter provides an attachment with a summary table for a study conducted by U.S. Geological Survey on toxic metals in U.S. surficial soil. In this study, samples of soils were taken, at a depth of approximately 20 cm from the surface, from locations about 80 km apart throughout the continental United States. Hence, the commenter notes that data represented soil characteristics without the impact of atmospheric deposition. The commenter refers to these tables to show that mercury as well as all other toxic metals naturally exist at low levels in the earth's topsoil. The commenter further states that their existence has nothing to do with "long-range atmospheric transport and deposition of anthropogenic emissions."

The commenter feels that EPA's risk characterization for this proposal, by crediting the highest concentration that was observed in the background fish samples as due to long-range atmospheric transport and depositions, has clearly violated Administrator's Browner instruction that "[r]isk assessments should be transparent, in that the conclusions drawn from the science are identified separately from policy judgments." The commenter states that if long-range atmospheric transport and deposition is the true reason for a given level of mercury, the observed concentration level would be uniformly distributed over a very wide area, rather than producing the highest background concentration at an isolated remote area, and concludes that EPA's statement seems to be trying to twist science to justify a policy decision

***Response 456**: EPA believes there is ample evidence that long-range transport of anthropogenic emissions of mercury are contributing to mercury levels in remote areas. As noted by Fitzgerald et al., 1998 ("The case for atmospheric mercury contamination in remote areas"), recent experimental results indicate that local-scale geochemical processes alone cannot explain the mounting number of lake sediment and peat profiles*

showing substantial increases of mercury flux during the past century. Atmospheric and aquatic cycling of mercury and the bioaccumulation of monomethylmercury in aquatic systems are driven by complex chemical and biological reactions involving trace amounts of mercury. Analytical advances in measuring environmental mercury at trace levels have greatly narrowed estimates of natural mercury fluxes and support the case for long-range atmospheric transport of mercury to remote areas. The similar timing and magnitude of recent increases and a concordance with spatial trends in measured mercury deposition strongly support that long-range transport of anthropogenic mercury is the cause of increasing mercury concentrations and fluxes in the sediments of lakes and sparsely populated regions that are not affected by localized human-related sources of mercury.

As explained in EPA's 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), mercury cycles in the environment as a result of natural and anthropogenic activities. Most of the mercury in the atmosphere is elemental mercury vapor, which circulates in the atmosphere for up to a year and can be dispersed and transported thousands of miles from the source of emission. A computer simulation of long-range transport of mercury suggests that about one-third of U.S. anthropogenic emissions are deposited through wet and dry deposition within the lower 48 states. The remaining two-thirds is transported outside of U.S. borders, where it diffuses into the global reservoir. The highest deposition rates from anthropogenic and global contributions are predicted to occur in the southern Great Lakes and Ohio River Valley, the Northeast, and scattered areas in the South. The location of sources, the chemical species of mercury emitted, and climate and meteorology are key factors in mercury transport and deposition. The flux of mercury from the atmosphere to land or water at any one location is composed of contributions from the natural global cycle including re-emissions from the oceans, regional sources, and local sources.*

For the final rule, EPA conducted a quantitative assessment of risks from mercury to both human and ecological receptors. The mercury analysis for the final rule evaluated impacts from mercury emissions on local bodies of water in the immediate vicinity of HWCs. However, EPA did not evaluate the specific impacts resulting from long-range transport of HWC emissions beyond local study areas for the final rule.

***Comment 211*:** The commenter points out that in its discussion of the health effects of mercury, EPA references a study of 81 maternal/infant pairs exposed to methylmercury in an accident in Iraq. The commenter notes that EPA recently announced that it is postponing the release of its mercury emissions report because one recent study on children in the Seychelles Islands directly contradicts the results of the Iraq study. The Seychelles Islands study involved 740 mother/infant pairs and found none of the neurological effects on children that were reported in the Iraq study. Also, the

commenter states that EPA is reevaluating these studies and plans to submit them, along with the draft mercury report, to the EPA Science Advisory Board for review.

> ***Response 211:*** *EPA released its* Mercury Study Report to Congress *(MRTC) in 1997 (U.S. EPA, 1997c) following a review of the report by the SAB. As explained in the mercury study, EPA's RfD for methylmercury is based on the Iraqi study. (EPA defines the RfD as an estimate of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime.) The SAB endorsed retention of the RfD, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data.*
>
> *Subsequently, EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*
>
> *The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.*

***Comment 212*:** The commenter notes that EPA concedes that "the current state of knowledge concerning the behavior of mercury in the environment does not allow for a meaningful quantitative risk assessment of emission sources which is precise enough to support regulatory decisions at the national level." The commenter states that, given this state of affairs, EPA should defer consideration of BTF controls for mercury until EPA develops adequate knowledge about mercury risks.

Response 212: *EPA is concerned about exposure to mercury from HWC emissions because mercury is a known neurological toxicant in humans. However, at the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA, 1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

The assessment of human health and ecological risks from mercury emissions followed the general modeling approach developed for the 1997 MRTC and used the IEM-2M surface water model developed especially for that report.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in CAA Section 112(d)(2) and (3). Mercury is singled out for regulation under MACT standards in Section 112(c)(6). For the final rule, EPA is setting the emission standard for mercury at the MACT floor.

Comment 209*:* The commenter feels that EPA has not demonstrated that risks associated with mercury emissions warrant beyond-the-floor (BTF) standards. The commenter notes that while EPA discusses its concern with exposures to methylmercury, primarily by ingestion of fish, in relating this concern to HWCs, EPA uses the terms “strongly suggesting,” “indicates,” and “it may be inferred” but fails to provide any data specific to HWCs to demonstrate that mercury emissions from these facilities actually pose a threat. The commenter states that it is unclear whether the small reduction in emissions achieved by BTF standards for hazardous waste incinerators (HWIs) is measurable, let alone a justification for the high cost of implementing the standard.

Response 209: *For the final rule, EPA conducted a quantitative risk assessment for mercury. The assessment followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model and methylmercury reference dose developed especially for that report. The results of EPA’s mercury analysis suggest that mercury emissions from HWCs in and of themselves are not likely to lead to exposures that exceed EPA's reference dose. However, exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing. EPA recognizes that its*

mercury analysis is subject to considerable uncertainty, including the fact that background exposures were not taken into account. Other sources of uncertainty include the speciation of mercury emissions, fate and transport of mercury in the atmosphere and in soils, methylation in surface water and uptake of mercury in fish, and the absence of site-specific information on the fishing activity of anglers.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). For the final rule, EPA is setting the emission standard for mercury at the MACT floor.

EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Comment 215: The commenter provides two papers that use different approaches to determine worst-case metals emissions from incinerators and notes that both conclude that under worst-case circumstances there is virtually no potential to create public health issues from short-term peak emissions from incinerators.

The commenter provides additional details on one paper. This paper, "Evaluation of the Potential for Health Effects Due to Short-term Emissions of Metals from Hazardous Waste Incinerators," evaluated the maximum half-hour ground-level concentrations (GLCs) of metals resulting from three worst-case hypothetical situations: (1) the extreme assumption that all the particulate emissions were a single metal, (2) the unlikely situation that 5 percent of the total feed to the incinerator was a single metal, and (3) a bypass of the air pollution control equipment. GLCs were compared with the Immediately Dangerous to Life and Health (IDLH) standards designed to protect workers from potentially dangerous short-term exposures. The IDLH was greater than the maximum GLC concentration by at least a factor of 26 in all cases and in most cases by a factor of thousands. GLCs were also determined using the maximum metals emissions concentration in a comprehensive U.S. waste combustion emissions database under worst-case dispersion conditions. The IDLH was greater than the maximum GLC for all hazardous waste incinerator emissions by a factor of 44,000. Maximum GLCs for two metals with Occupational Safety and Health Administration (OSHA) ceiling limits (cadmium and mercury) were compared to the to the OSHA ceilings; OSHA ceiling limits were at least 3,700 times larger than the maximum GLCs.

***Response 215**: EPA notes with interest the information submitted by the commenter. Although the risk assessment for the final rule focused on routine emissions and the risks associated with chronic exposures, EPA does not expect short-term emissions to be high enough to pose a significant risk from acute exposures except possibly under very unusual circumstances.*

EPA expects that under normal operating conditions, for most of the constituents evaluated in the HWC risk analysis, risks resulting from long-term exposure are likely to exceed risks resulting from short-term acute exposure to peak emissions levels. This is especially true for metals and dioxins/furans, which exhibit carcinogenic effects, although it is likely to also be true for noncancer effects. The risk analysis conducted for the final rule was designed to answer public policy questions concerning the MACT emission standards for HWCs, which are not specifically designed to limit emissions under extreme operational conditions of the sort that could pose a significant risk from acute exposures. However, under the omnibus provisions of section 3005(c)(3)of RCRA, permit writers may, on a case-by-case basis, establish additional permit conditions as may be necessary to protect human health and the environment.

Chemical Manufacturers Association
RCSP-0221

***Comment 238*:** The commenter feels that the risk assessment comments show that the asserted health benefits of beyond-the-floor (BTF) controls for dioxins/furans and mercury are significantly overstated and may be nonexistent in the case of mercury. Also, the commenter states that Section 3004(a) of RCRA, EPA's other basis for BTF controls, only authorizes EPA to promulgate emission standards for hazardous waste incinerators (HWIs) "as necessary to protect human health and the environment." The commenter states that the peer reviews regarding risk assessment likewise reveal that EPA cannot support BTF levels for dioxins/furans or mercury under this authority.

Response 238: *HWCs emit hazardous air pollutants listed under Section 112(b) of the CAA. In an effort to control emissions of HAPs from HWCs, EPA proposed NESHAP pursuant to Section 112(d) of the CAA that establish technology-based, not risk-based, emission standards based on application of maximum achievable control technology (MACT). However, Sections 3004(a) and 3004(q) of RCRA, as amended, require EPA to develop standards that are protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate human health and ecological risks from HWCs in order to determine what, if any, RCRA standards for emissions from these sources may be needed.*

The final rule sets emission standards for dioxins and mercury for existing incinerators based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule.

Regarding the peer review completed for the proposed rule, EPA gave much consideration to the comments of the peer-review panel. In response to these comments, EPA made a number of changes to the risk assessment in order to improve the overall representativeness of the risk assessment. EPA has prepared a separate response to comments document entitled Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for EPA's response to the peer review.*

***Comment 240*:** The commenter strongly opposes BTF regulation and cites the comments of peer reviewers intended to caution EPA on its risk approach and findings. Specifically, the commenter notes the conservative nature of the case study approach: "…in that the methodologies and assumptions used to characterize chemical emissions, fate and transport, exposure, and toxicity all lead to the overestimation of potential risk. Thus, it is misleading to characterize the case study approach as

providing accurate risk estimates." The commenter notes that while recognizing the usefulness of the case study approach, a peer reviewer had objected to its exposure bounding. The commenter cites the peer reviewers' comments on exposure bounding, indicating that it "…violates the case study objective of minimizing the number of generic default, worst-case assumptions incorporated into the risk estimates," and notes that the peer reviewer recommends deleting it. The commenter also notes that the peer reviewer cautioned that if "assumptions [of risk conditions] are made in a conservative manner, the risk outcome can potentially be vastly overstated."

Response 240: *For the final rule, EPA did not use a case study approach and instead used stratified random sampling to select a subset of facilities from the HWC universe for risk characterization. Each of these selected facilities was modeled using site-specific data when available. The use of stratified sampling in selecting facilities allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe they are designed to represent (e.g., confidence intervals). Such statements could not be made using the case study approach that was used at proposal.*

EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. That is why, for the final rule, EPA did not use high-end values. In addition, EPA did not make bounding estimates as referred to by the commenter. In all instances, EPA either used central tendency values in conjunction with site-specific data or conducted a full distributional analysis to account for the full range of exposure.

EPA used central tendency exposure factors (e.g., mean values) for estimating exposures, except for a few risk-driving pathways for which an exposure factor variability analysis was conducted using Monte Carlo simulation. Monte Carlo simulation allows a more complete characterization of the range of exposures for a population without use of overly conservative upper-bound parameters. In addition, the risk analysis for the final rule used site-specific emissions estimates to estimate media concentrations and concentrations in agricultural products. Also, U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing as a recreational sport and for persons engaged in farming or fishing for subsistence. EPA believes that these modifications to the risk methodology provide considerable assurance that the exposure estimates developed for the final rule are reasonable.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by

Sections 3004(a) and (q) of RCRA. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 241*:** The commenter cites a peer reviewer's comments regarding background exposure to caution EPA on its risk approach and findings. The peer reviewer states, "The finding that background dioxin exposure levels generally exceed the site-specific exposure levels the Agency has modeled for hazardous waste incinerators…is not surprising since these sources comprise a minority of the total sources contributing to background. It is likely that nearby non–hazardous waste-burning sources of these chemicals (e.g., coal-fired plants, etc.) are contributing to the observed levels."

***Response 241**: EPA gave much consideration to the comments of the peer-review panel and, in response to these comments, made a number of changes to the risk assessment to improve its overall representativeness. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for EPA's response to the issues in the above comment.*

***Comment 1101*:** The commenter notes that a peer reviewer summarized his findings by stating that when analyzing uncertainties, EPA should qualitatively state the over- or underestimation of risk attributable to assumptions made within the risk assessment, thereby enabling readers to "judge the level of confidence that should be placed on the deterministic risk estimates…."

***Response 1101**: EPA has prepared a separate response to comments document for the external peer review entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for a full response to the issues raised by the peer review panel.*

For the final rule, EPA gave considerable attention to the sources of uncertainty in characterizing the risks from HWCs. Furthermore, a number of modifications were made in the revised risk assessment which serve to reduce uncertainty. The risk assessment background document includes a detailed discussion of uncertainty associated with all major components of the HWC risk analysis including fate/transport modeling, exposure assessment, and risk characterization. When feasible, the uncertainty discussion includes a qualitative characterization of both the magnitude and direction of the impact from specific sources of uncertainty. However, with the exception of the uncertainty associated with statistical sampling error that was quantified, it was not possible to perform a quantitative uncertainty analysis for the HWC risk assessment.

***Comment 242*:** The commenter refers to a peer reviewer's comment on the appropriateness of using TEQ for estimating risk. The reviewer said, "Toxicity equivalent factors are expressly not to be used to estimate risks…but to be used only for screening purposes. This use as an estimator renders the estimate of risk invalid." While another reviewer believes that the TEQ approach for risk assessment rests on sound scientific principles, "its application in the [risk assessment] is an oversimplification of the exposure to chemical mixtures like dioxins with varying toxic potentials…."

***Response 242**: EPA has prepared a separate response to comments document on the external peer review entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for a full response to the issues raised by the peer-review panel.*

The concept of toxicity equivalence (TEQ) between compounds having similar chemical structures and a common mechanism of action is one that has long been used by EPA and the scientific community for assessing risks from chlorinated dioxins and furans. The approach uses toxicity equivalence factors (TEFs) that relate the toxicity of individual chlorinated dioxin and furan congeners to that of 2,3,7,8-TCDD, the most studied compound of the class. The TEFs are derived from experimental data across a variety of toxicological endpoints that are mediated by binding to the Ah receptor. While the TEQ approach is a simplification and has associated with it a number of uncertainties, EPA believes that the TEQ approach remains the most feasible approach for assessing risks from this class of compounds.

Comment 640: The commenter cites a peer reviewer comment that "'real world' exposure to chemical mixtures can be expected to alter considerably in exposures involving mixtures of TCDD and congeners" and that "the risk assessment does not provide a clear overview of the approach adopted in the exposure assessment and its associated limitation in the use of case-study specific exposure data."

***Response 640**: EPA has prepared a separate response to comments document on the external peer review entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for a full response to the issues raised by the peer-review panel. EPA is aware of the complexity of assessing risks from chemical mixtures and that is why EPA is committed to the TEQ approach for assessing risks from 2,3,7,8-TCDD and other chlorinated dioxins and furans. The commenter is referred to the risk assessment background document for an overview of the analytical framework used in the risk assessment for the final rule and a detailed discussion of the limitations and uncertainties in the HWC risk analysis.*

***Comment 243*:** The commenter notes that while the risk assessment panel was critical of the proposal's dioxin/furan risk assessment, the panelists were even more withering in their criticism of the proposal's mercury risk assessment. The commenter cites the review panel's conclusions that "the preamble makes no case that mercury emissions pose a threat to human health at any level"; that "EPA does not provide sufficient supporting evidence to reasonably conclude that adverse health effects associated with ingestion of fish are currently occurring"; and finally that "EPA does not provide a rationale to support the actual mercury limits being proposed for HWCs." The commenter feels that certain statements are particularly noteworthy:

Dr. Wilson
"The Agency has successfully shown that reductions beyond the floor do not lead to a significant reduction in mercury exposures: the reduction is almost certainly smaller than the uncertainty in the estimate of total mercury intake from all sources, so it is insignificant by definition."

Dr. Rao
"Section 4b states mercury's oral reference dose (RfD) as 1E-4 mg/kg-day. According to the Agency's…(IRIS),…there is no oral RfD for metallic mercury…. However, an oral RfD of 3E-4 mg/kg-day for inorganic mercury (HgC4) is…under review. So it is not clear as to from where the Agency obtained a new oral RfD of 1E-4 mg/kg-day for mercury?"

"The oral RfD of 1E-4 mg/kg-day is based on a human study (Iraqi study) with developmental neurological abnormalities as the end point…. It is not clear in the EPA RA as to how…a toxic end point that the Agency treats as a unique end point and outside of systemic effects, was actually used here to derive a RfD for general systemic effects?"

Dr. Anderson
"EPA has totally failed to reconcile this proposed reduction [in mercury emissions] with potential benefits to the aquatic environment…."

> ***Response 243:*** *EPA gave much consideration to the comments of the peer-review panel on the risk assessment for the proposed rule. In response to these comments, EPA made a number of changes to the risk assessment in order to improve the overall representativeness of the risk assessment, including a quantitative analysis of the risks from mercury emissions from HWCs. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments.*
>
> *With regard to the specific issues raised by the commenter, EPA recognizes the uncertainties associated with any assessment of risks from mercury emissions from HWCs, including those identified by the peer reviewers. However, EPA wishes to*

emphasize that the MACT standards are technology-based standards that are based solely on the factors made relevant under Section 112 of the CAA. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by sections 3004(a) and (q) of RCRA in order to determine what, if any, RCRA standards for emissions from these sources may be needed.

The oral RfD for mercury is derived from an epidemiological study of the effects of methylmercury on the neurological and behavioral development of infants exposed in utero, as explained in EPA's Mercury Study Report to Congress *(U.S. EPA, 1997c).*

Section 8
Chemical Waste Management, Inc.
RCSP-0153

***Comment 369*:** The commenter notes that in its description of the methodology for evaluating potential risks from dioxins to individuals living near HWCs, EPA presents a total of 11 "synthetic" HWC facilities that combined the use of site-specific characteristics with national emissions data were selected for evaluation. As expected, indirect exposure pathways, such as consumption of locally raised cattle, eggs, and dairy products predominated. The risk assessment is driven by the indirect risks from polychlorinated dibenzo-p-dioxin and polychlorinated dibenzofuran (PCDD and PCDF, respectively, hereafter referred to as dioxin/furan) toxic equivalents (TEQs).

The commenter states that an evaluation of the background support document and its appendices, however, reveals that EPA's summaries of the risk ranges are misleading and are based on receptor populations that are rarely encountered or do not exist in the immediate proximity of most HWCs. The commenter provides a brief summary that compares the findings of EPA's background support document used to quantify the level of risk in support of the proposed dioxin/furan emission standard with the risk ranges depicted in a table in the proposed rule. For the baseline, the low end of the dioxin risk range (2×10^{-9}) corresponds with "direct inhalation," low central tendency. The high end of the dioxin risk range (9×10^{-5}) corresponds to the high end for a "subsistence dairy farmer child." For the proposed floor standard, the low end of the dioxin risk range (3×10^{-9}) corresponds with the low central tendency value for a "home gardener from all pathways combined," although the low central tendency value for "direct inhalation" actually provides the lowest low-central tendency value (2×10^{-9}, the same as the baseline figure). The high end of the dioxin risk range (5×10^{-5}) again corresponds to the high end for a "subsistence dairy farmer child." For the proposed beyond-the-floor standard, the low end of the dioxin risk range (3×10^{-9}) corresponds with the low central tendency value for a "subsistence fisher" and "home gardener from all pathways combined," although the low central tendency values for "typical farmer," "recreational fisher," and "direct inhalation" actually provide the lowest low-central tendency value (2×10^{-9}, the same as the baseline figure). The high end of the dioxin risk range (2×10^{-6}) corresponds to the high end for a "subsistence beef farmer" and "subsistence dairy farmer."

The commenter states that EPA has provided no data or supporting information to document the existence of subsistence farmers or fishers located in proximity to HWCs and notes that in general, the majority of HWCs are associated with chemical manufacturing and processing industries and are typically located in highly industrial areas. Review of EPA's database, provided in the background support document, demonstrates that the upper bound of the acceptable risk range (that is, 1 in 10,000) is satisfied, at existing emission levels, for all exposed populations. In addition, the data demonstrate that EPA's "point of departure" (the lower bound of the acceptable risk range, 1×10^{-6}) is satisfied for most typical populations. In short, the commenter feels that EPA has not justified a risk-

based BTF standard for dioxin/furans when the existence of such affected individuals, from the most extreme tail of the exposure distribution curve, is not supported by EPA data.

Response 369: *In a departure from the approach used at proposal, the risk analysis completed for the final rule does not use model facilities and instead uses stratified random sampling to select a subset of facilities from the HWC facility universe for analysis using site-specific data. For the final rule, the number of facilities analyzed was expanded from 11 "synthetic" facilities to 76 active HWC facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). This group represents more than 40 percent of the HWC facilities covered by the rule. By increasing the overall number of facilities evaluated, EPA has increased the probability that the risk results will be representative of the overall universe of HWC facilities across the full range of exposures. To ensure that these facilities would be representative of all facilities covered by the rule, 66 of the 76 facilities were selected using stratified random sampling, while the remaining 10 were facilities that had been analyzed at proposal and were retained for the final rule (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure). Sample sizes for each combuster category were chosen such that the probability of selecting a high-risk facility (one that lies above the 90th percentile of the risk distribution) would be 90 percent or better. Therefore, EPA believes the risk assessment for the final rule is representative of the range and types of hazardous waste combustion facilities. EPA also expects that the risk assessment adequately characterizes risks at the high end of the risk distribution.*

The risk assessment for the final rule also was modified to account for the number of individuals exposed and their location. The analysis used U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. Individual risk results for the final rule include multiple risk percentiles (i.e., 50th, 90th, 95th, and 99th percentile risk estimates), which characterize the range of risk experienced by a specific receptor population/age group combination including both central tendency and high-end risk. However, it was not possible from census data to identify and locate individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios were retained in the risk analysis for the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to

those individuals. For this purpose, EPA assumed that subsistence farming takes place within certain prescribed distances of HWCs (that is, within 2, 5, 10, and 20 kilometers). This is a departure from the proposed rule, which located subsistence farmers at the point of maximum projected impact. EPA also assumed that subsistence fishing takes place at each body of water that was modeled in the risk analysis. EPA recognizes that these assumptions may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3). Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Section 9
Chlorine Chemistry Council
RCSP-0115

***Comment 478*:** The commenter states that EPA inappropriately relies upon its draft dioxin reassessment as the basis for many assumptions and conclusions about dioxin/furan emissions, risks, and other health-related issues. The commenter indicates that previous extensive comments were submitted on the document upon its release in 1995. At that point, the commenter argued that the draft reassessment did not accurately reflect the current scientific understanding of dioxin exposures and human health effects and therefore did not accurately characterize potential human health risks. The EPA Science Advisory Board also reviewed the draft dioxin reassessment and noted many of the same weaknesses in the document. In response to both public and Science Advisory Board comments, EPA is currently revising its draft dioxin reassessment but has not yet issued a final document. This revision is expected to significantly modify the risk characterization chapter, in particular. The current draft, with widely recognized flaws, is not an acceptable proxy for the final reassessment.

> ***Response 478**: EPA has not relied on the conclusions of 1994 dioxin reassessment, including specifically the risk findings of the draft health assessment document (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin [TCDD] and Related Compounds*, U.S. EPA, 1994d,e) because this document remains a draft document. However, EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available, and EPA continues to rely on it for the final rule. This includes the information contained in the 1994 dioxin exposure assessment (*Estimating Exposure to Dioxin-like Compounds *(external review draft), U.S. EPA, 1994a,b).*
>
> *EPA is continuing its work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to these comments in an appropriate forum.*

***Comment 479*:** The commenter believes that EPA's protocol for evaluating site-specific multipathway risk relies on unreasonable scenarios and invalidated models. The commenter feels that the risk assessments do not make use of the best available science and result in overly conservative risk estimates, and the commenter notes that the risk assessment methodology follows that outlined in the *Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (1990) and its 1993 Addendum, both of which were rejected by the Science Advisory Board as "not ready for release."

> ***Response 479:** It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect*

exposures to emissions from HWC facilities. As explained in the risk assessment for the proposed rule, EPA used the indirect exposure methodology as outlined in the 1990 document Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emission *(U.S. EPA, 1990). This document was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure with information gathered in conjunction with the dioxin reassessment (*Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress (*MRTC) (U.S. EPA, 1997c) as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, is the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."*

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board. EPA intends to respond to comments on the draft Addendum that it has received from the public, as well as from the Science Advisory Board, in an appropriate forum.

EPA disagrees with the commenter that the methodology is overly conservative. For the final rule, EPA used central tendency exposure factors in conjunction with site-specific data to estimate exposures, except for a few risk-driving pathways for which an exposure factor variability analysis was performed using Monte Carlo simulation. U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing as a recreational sport and for persons engaged in farming or fishing for subsistence. EPA believes that these steps provide considerable assurance that the exposure estimates developed for the final rule are reasonable.

Comment 480: The commenter notes that not only has EPA not yet formally addressed the SAB comments about uncertainties associated with the risk assessment methodology, but also EPA has derived overly conservative risk estimates by setting a number of key parameters to high-end risk values.

Response 480: *As indicated in response to the previous comment, EPA intends to respond to comments received from the Science Advisory Board on the 1993* Addendum *(U.S. EPA, 1993a) in an appropriate forum. EPA also acknowledges the uncertainty of the models used for assessing exposures from HWCs. However, EPA believes these models represent the best analysis tools available for assessing risks from HWCs. EPA*

has included a comprehensive discussion of the uncertainties in its characterization of risks in the risk assessment background document for the final rule.

EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. That is why, for the final rule, EPA did not use high-end values. In all instances, EPA either used central tendency values or conducted exposure parameter variability analysis to capture the range of exposure, in conjunction with site-specific data. The variability analysis was conducted for key exposure pathways and facilitates characterization of the full range of exposures without the use of high-end or upper-bound exposure assumptions.

***Comment 482*:** The commenter states that EPA does not use revised TEF values to calculate TEQ emissions, and that use of the new 90-day chronic TEFs developed by EPA results in 30 percent lower TEQs from fish and beef and a 25 percent reduction of total background emissions from incineration.

***Response 482**: For the final rule, EPA used the most recent TEFs recommended by an international panel of experts convened under the auspices of the World Health Organization (WHO). The WHO panel of experts reviewed all the available data on the relative toxicities of dioxin-like compounds and made recommendations for changes to the values of several of the TEFs from the 1989 interim values. The WHO TEFs reflect a consensus of the international community and for this reason were used by EPA in the risk assessment for the final rule. TEFs are meant to represent order of magnitude estimates relative to 2,3,7,8-TCDD. As such, a 25 to 30 percent reduction in TEQs relative to the 1989 values is within the range of uncertainty inherent in the TEFs.*

***Comment 483*:** The commenter states that EPA's administrative record for this proposed rule is incomplete, as EPA has not provided all risk assessment calculations as requested by the Chemical Manufacturer's Association (CMA) in a letter dated July 23, 1996. The commenter feels, therefore, that it is impossible to verify independently the accuracy of most of the risk scenarios relied upon by EPA.

***Response 483:** The background document for the proposed rule documents all equations and parameter values used in the risk analysis at proposal (see* Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). Although EPA has not made the spreadsheets and other software that were used in HWC risk analysis part of the administrative record, EPA placed example spreadsheet calculations in the docket for the proposed rule. (This information was included as an attachment to a July 17, 1996 memo from D. Layland to the file documenting a meeting with cement industry*

consultants.) EPA also made all air dispersion model output files available to the public through the RCRA docket (see "Availability of Electronic Files of Dispersion Modeling Results," memorandum from D. Layland to the RCRA Docket dated May 16, 1997). In addition, much of the software that EPA used in the analysis is publicly available. This includes the Industrial Source Complex Short-Term (ISCST) model, which was used for modeling air dispersion and deposition in both the proposed and final rules, and the Integrated Exposure Uptake Biokinetic Model (IEUBK) model (U.S. EPA, 1994c), which was used for modeling lead exposures in the final rule. EPA has carefully documented the calculations, variables, and data used in the risk assessment for the final rule and has placed these in the rulemaking docket. In addition, the IEM-2M model, which was used for modeling the fate and transport of mercury in surface water in the risk assessment for the final rule, is fully documented in the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c).*

***Comment 484*:** The commenter feels that EPA has overestimated background body burdens, as well as the toxicological risks of dioxin/furans, as EPA relies on a cancer risk estimate approach which overstates the health risks associated with background dioxin/furan exposure. The commenter believes that it is premature for the proposed rule to incorporate the dose of 0.01 pg/kg/day as associated with an upper bound excess cancer risk of 10^{-6} in light of SAB's specific recommendations to consider a threshold for cancer effects in humans. Further, the commenter states that neither the draft dioxin reassessment nor the proposed rule give proper weight to the extensive Ranch Hand studies which demonstrate that moderate dioxin/furan exposure is not a risk factor for cancer in humans. The commenter feels that EPA ignores the fact that no population of individuals exposed to dioxin/furan have demonstrated a "broad spectrum" of adverse health effects, and that EPA inappropriately relies on the Seveso studies to support the health benefits of dioxin/furan reduction. The commenter believes that EPA's use of TEF/TEQ methodology to assess risks from dioxin/furan is not appropriate, given that the SAB was quite explicit on the need for additional work on this methodology. The commenter notes that EPA's own research establishes that the present TEF/TEQ system overestimates long-term risks by a factor of 4 to 10.

***Response 484**: EPA agrees with the commenter that the background exposures referred to in the preamble to the proposed rule may have been overestimated. However, these estimates were not based on background body burden data, as indicated by the commenter, but rather were derived from dietary data. For the final rule, EPA used a background exposure estimate of 1.5 pg TEQ/kg-day. This estimate was made from background body burden data using a steady state pharmacokinetic model. EPA recognizes the uncertainty in this estimate of background exposures. Estimates derived from body burden data using a non-steady-state pharmacokinetic model suggest that background exposures could be lower than this. Estimates derived from current dietary data range from 0.6 to 2 pg/kg-day, depending on an person's age, with the higher*

values' being for children (these high values occur as a result of a child's greater dietary intake on a per-unit-body-weight basis).

In its review of the 1994 draft dioxin reassessment, the EPA Science Advisory Board suggested that EPA consider alternatives to the linear nonthreshold model, allowing for minimal response at low environmental levels of exposure; this method would be consistent with the body of available health effects data. In response, EPA developed a mechanistic model for liver tumors in female rats using data from a 2-year feeding study in Sprague-Dawley rats (Kociba et al., 1978). EPA also summarized the results of simple empirical models that have been applied to other significant cancer findings. EPA's analysis indicates that most of the cancer findings exhibited response consistent with linearity in the observable range. Also, few of the mechanistic models that EPA identified in the scientific literature exhibited nonlinear dose-response in the observable region or predicted nonlinear dose-response in the low-dose (extrapolation) region. Furthermore, EPA's analysis of the data from the National Institute of Occupational Safety and Health (NIOSH) study (Fingerhut et al., 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin") found that the ratio of the average daily dose for the high- and low-dose groups was the same as the ratio of increased risk for respiratory cancer, indicating a linear dose-response curve (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d).*

Therefore, EPA believes there are sufficient human and animal data suggesting that response is proportional to dose to warrant continued use of the linearized multistage model. EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment should be used. That estimate, which is based on the Kociba et al. (1978) study, is 156,000 $[mg/kg\text{-}d]^{-1}$. EPA notes that the 1997 analysis of the human and animal data cited above lends considerable support to the 1985 estimate.

EPA believes the weight of the evidence supports the conclusion that 2,3,7,8-TCDD and other dioxin-like compounds are probable human carcinogens. Although the Ranch Hand study (Henriksen et al., 1997, "Serum dioxin and diabetes mellitus in veterans of Operation Ranch Hand") has yet to find evidence of an increased incidence of cancer, epidemiological studies of a number of other cohorts exposed at higher levels than the Ranch Hand veterans have found a positive association between dioxin exposure and cancer, in addition to the NIOSH study. These include cancers at many different sites, including malignant lymphomas, soft tissue sarcomas, hepatobiliary tumors, hematopoietic tumors, thyroid tumors, and respiratory tract tumors. EPA also notes that other health effects have been observed in the Ranch Hand veterans, including an increased incidence of diabetes, elevated cardiovascular disease, and a modest decrease in testosterone levels in the most highly exposed veterans.

The SAB, in its review of the draft dioxin reassessment, agreed with EPA, concluding that dioxin is likely to increase human cancer incidence under some conditions of exposure. The SAB categorized dioxin and dioxin-like compounds as a B1 carcinogen under the 1986 EPA cancer guidelines (U.S. EPA, 1986) of "agents for which there is limited evidence of carcinogenicity from epidemiologic studies."

The SAB, in its review of the draft dioxin reassessment, agreed with EPA that dioxins produce a spectrum of effects in laboratory animals depending on the dose, context of exposure, and genetic background but concluded that adverse effects attributable to chronic low-level exposure in humans have not yet been adequately demonstrated.

Seveso area results suggest that there were sex ratio changes in children born to parents living close to the plant (Mocarelli et al., 1996, "Change in sex ratio with exposure to dioxin"). Seveso females, together with Ranch Hand veterans and workers exposed to TCDD from industrial accidents, show a consistent pattern of thyroid function changes, e.g., diabetes (Jennings et al., 1988, "Immunological abnormalities 17 years after accidental exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin;" Henriksen et al., 1997, "Serum dioxin and diabetes mellitus in veterans of Operation Ranch Hand;" Pesatori et al., 1998 "Dioxin exposure and nonmalignant health effects: a mortality study;" Zober et al., 1994, "Morbidity Follow up study of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin after a 1953 chemical reactor incident").

With regard to the TEQ methodology and the use of TEFs for assessing risks from dioxins, the SAB called for clarifications in the specification of TEFs for the various dioxin-like compounds for various health outcomes but concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds. The WHO review, which was recently published in Environmental Health Perspectives *(Van den Berg et al., 1998), resulted in recommendations for changes to the values of several of the TEFs. The WHO TEFs reflect a consensus of the international community and, for this reason, were used by EPA in the risk assessment for the final rule.*

***Comment 491, 575, 576, 577*:** The commenter states that EPA has established high-end risk estimates in a manner different from the procedures outlined in the preamble, ignoring its own long-standing guidance regarding exposure. The commenter also states that the cumulative risk estimates far exceed the 90th percentile. To evaluate individual risks, EPA projected both high-end and central tendency estimates of risks to the individuals of concern. The commenter cites the proposed rule's description of the method followed to derive high-end risk estimates and notes that with regard to the high-end risk estimates, EPA has created implicit regulatory controls at or above the 99.9th

percentile, which result from a focus on the high-end risk scenarios posed by the different subsistence individuals and by choosing predominately conservative modeling values at each step of the process. In other words, by setting virtually all key parameters (that is, those which have the greatest effect on the dose) to high-end values, the overall risk or dose modeled is far beyond the 90th percentile. Thus, the commenter feels that it is not valid to suggest that the analysis is reasonable based only on the fact that certain parameters were kept at central tendency values. The commenter attached two papers that discuss the problems with EPA's high-end risk modeling and noted that EPA's use of high-end risk at the 99.9th percentile also appears to be at odds with EPA's own guidance. To this end, the commenter attached a February 26, 1992, memorandum entitled "Guidance on Risk Characterization for Risk Managers and Risk Assessors," in which then–EPA Deputy Administrator F. Henry Habicht stated, "…Conceptually, high end risk means risk above about the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest risk."

The commenter feels that EPA incorrectly asserts that only one parameter was set to the high-end value. EPA identifies three parameters (exposure duration [ED], emission levels [Q], and [for two scenarios] the contaminated fraction [F]) as being varied in the risk analysis to construct high-end and central tendency scenarios. However, the commenter notes that EPA has chosen high-end default values for other parameters which are not explicitly identified; indeed, the values selected for these parameters in the proposed rule are different from values recently published in the HWIR rulemaking and *draft dioxin reassessment*. These parameters (which vary by scenario) affect total media concentrations and cause the model to calculate higher risks.

575, 576, and 577 are attachments A, B, and C referred to in the comment above

Response 491, 575, 576, 577: *EPA disagrees with the commenter's suggestion that virtually all key parameter were set to high-end values in the risk assessment at proposal. EPA stands by its depiction of the approach used for estimating high-end exposures in the preamble to the proposed rule and in the risk assessment background document (see* Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). This approach involved constructing high-end exposure scenarios by setting one or more parameters at high-end values and is fully consistent with EPA's risk characterization guidance. EPA concedes, however, that it is not possible to state precisely where on the distribution the high-end scenario might lie. For this and other reasons, EPA modified the approach for characterizing high-end exposures for the final rule.*

For the final rule, rather than defining high-end exposure scenarios deterministically, EPA characterized high-end risks by developing population-weighted cumulative distributions of risks to individuals explicitly. The commenter is referred to the risk assessment background document for a complete description of the analytical framework

and the methods and data used in the risk analysis for the final rule (RTI, 1999). Briefly, EPA selected a total sample of 76 facilities to represent various categories of HWCs. Each facility was assigned a sample weight based on its selection probability. A study area was defined for each facility as the area surrounding the facility out to a distance of 20 kilometers. Each study area was divided into 16 sectors. Media concentrations were estimated for each facility sector using facility-specific (for example, stack emissions) and site-specific (for example, land use) information combined with national-level defaults (for example, livestock feeding practices). Sector-specific exposures were then estimated from the media concentrations and age-specific exposure factors. Mean exposure factor values were used in order to estimate the mean, or arithmetic average, risk to individuals within a sector. These mean risk values were then aggregated across an HWC category, taking into account sector-specific population estimates and facility-specific sampling weights. Population estimates were derived from U.S. Census and Census of Agriculture data.

In addition, to further refine the estimates of risks from exposures to dioxins and furans via consumption of beef and milk (representing the risk-driving exposure pathways), EPA performed an exposure factor variability analysis. This involved generating a distribution of risks at the sector level that reflected the variability in exposure factors between individuals. A cumulative risk distribution was generated from the sector-level distributions using Monte Carlo analysis. A similar analysis was conducted to estimate exposures to methylmercury through ingestion of fish.

From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, and 99th percentiles. Using this approach, EPA could more precisely characterize the high end of the risk distribution while avoiding the need to set specific parameters to high-end values.

***Comment 0115-1*:** The commenter states that EPA has relied on a flawed risk assessment methodology and in so doing has overstated the incremental health benefits of BTF controls for HWIs. The commenter points out the following specifics.

The risk assessment relies on the *Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (1990) and its 1993 Addendum, a methodology rejected by the Science Advisory Board. The commenter notes that these documents both were evaluated by the SAB as late as December 1993 and rejected as “not ready for release.” In particular, the SAB was concerned about the uncertainties associated with the model. Yet in developing the proposed rule, EPA has relied upon the risk model as an accurate quantitative instrument.

EPA has only recently made the external peer review of the risk analysis available to the public. Because the results of this external review became public on August 5, 1996, EPA has not allowed sufficient time for adequate review of these critical materials.

EPA improperly relies on the draft dioxin reassessment to substantiate the health benefits of BTF controls. Much of the information used to derive the individual risk estimates for dioxins/furans is taken from EPA's 1994 review draft dioxin reassessment documents, which are documents that are to be neither cited nor quoted. Although the SAB reviewed the draft dioxin reassessment in 1995 and issued its report, *An SAB Report: A Second Look at Dioxin*, substantial changes remain to be made by EPA before final SAB approval can be rendered. Specifically, the SAB determined that the risk characterization chapter was not as thoroughly peer-reviewed as were earlier chapters and needs to be revised considerably to deal with a number of weaknesses. In particular, SAB noted that the presentation of scientific findings was unbalanced and that EPA tended to overstate the possibility of risks posed by dioxins/furans. In addition, important uncertainties associated with EPA's conclusions were not fully identified and subjected to feasible analyses. It is therefore inappropriate for EPA to utilize the draft dioxin reassessment in developing the proposed rule particularly when substantial changes to the risk characterization chapter, perhaps the most important aspect of the entire reassessment, are anticipated.

In addition, revisions to the draft dioxin reassessment and final SAB approval are not anticipated until the last quarter of 1996 and, consequently, will not be part of the public record for this proposed rule. Nonetheless, EPA has stated that revisions to the draft dioxin reassessment will be considered in the development of the final rule. Importantly, the public will be foreclosed from commenting on EPA's use of these revisions in developing the final rule, directly contrary to the Administrative Procedures Act.

> ***Response 0115-1****: It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from HWC facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment* (Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), as well as other sources of information.*
>
> *EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available, including specifically*

*information in the draft exposure document, for which the SAB commended EPA, calling it "a very credible and thorough job." However, EPA has not relied on the conclusions of 1994 dioxin reassessment, including specifically the risk findings of the draft health assessment document (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin [TCDD] and Related Compounds*, U.S. EPA, 1994d,e) because this document remains a draft document.*

As indicated by the commenter, both the 1993 draft Addendum and the 1994 draft dioxin reassessment were reviewed by EPA's Science Advisory Board. Also, public comments were received on both the Addendum and the dioxin reassessment. EPA intends to address the comments from the SAB on the 1993 Addendum to the indirect exposure methodology and the 1994 draft dioxin reassessment, as well as those from the public, in an appropriate forum.

As part of the rulemaking package, the risk assessment for the proposed rule was subject to full notice and comment procedures consistent with the Administrative Procedure Act. A notice of proposed rulemaking was published in the Federal Register *on April 19, 1996. The public comment period extended from April 19, 1996, to August 19, 1996, a period of more than 120 days. A public docket was established for the notice of proposed rulemaking, and all comments received on the proposed rule were placed in the public docket (U.S. EPA Docket Number F-96-RCSP-FFFFF). In addition, the risk assessment for the proposed rule was externally peer reviewed. A notice of data availability was published in the* Federal Register *on August 23, 1996, requesting comment on the report prepared by the peer reviewers. A 30-day public comment period was established. EPA believes that the 120-day comment period for the notice of proposed rulemaking, followed by the additional 30-day comment period on the peer review, provided ample opportunity for the public to comment on the risk assessment for the proposed rule. Indeed, EPA received numerous comments from this and other commenters.*

Comments received during the public comment periods were considered by EPA in the risk assessment for the final rule. EPA has prepared this response to comments document for the final rule that explains how EPA considered each of the comments received on the risk assessment at proposal. A response to comments document was also prepared on the report of the external peer review panel on the risk assessment for the proposed rule.

EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2)

and (3). For dioxins, the final rule sets an emission standard for existing sources based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule.

EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, which has not been finalized, either in the risk assessment or in setting the emission standards. Therefore, the commenter's notice and comment concerns about revisions to the risk methodology, which EPA regards as unfounded, do not relate to the emission standards themselves.

***Comment 492*:** The commenter notes that many parameter values are set to high-end values. The commenter provides as an example the grass diet of subsistence farmer cattle versus the diet of average farmer cattle. The daily diet of beef cattle changes from subsistence farmer to average farmer. The cattle of subsistence beef farmers eat a daily diet of forage, silage, grain, and soil totaling 12.27 kg/day, while the cattle of the typical beef farmer eat only 8.85 kg/day. The commenter provides a table that compares EPA beef cattle diets.

Response 492: *EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. This is why, for the final rule, EPA did not use high-end values. In all instances, EPA either used central tendency values or conducted exposure parameter variability analysis to capture range of exposure, in conjunction with site-specific data. The variability analysis was conducted for key exposure pathways and facilitates characterization of the full range of exposures without the use of high-end or upper-bound exposure assumptions.*

For the MACT rule, different consumption rates were used for livestock depending on whether the livestock were raised commercially or for subsistence purposes. As a result, commercial beef and dairy cattle were assumed to consume more grain and less forage than subsistence cattle. A diet high in grain intake for beef cattle raised commercially (i.e., cattle raised by the "typical" farmer at proposal) is consistent with national data on livestock production practices and was used for both the proposed and final rules.

***Comment 494*:** The commenter identifies soil mixing depth as an important parameter for estimating watershed soil concentrations and thus the impacts resulting from ingestion and dermal contact with contaminated surface water and ingestion of fish. Small soil mixing depths maximize dioxin/furan concentration (and therefore risk) by limiting the volume of soil into which it is mixed. The value chosen in the MACT risk analyses is 1 cm, while the value used in the draft dioxin reassessment is 10 cm.

Response 494: *For the final rule, EPA used a soil mixing depth of 1 centimeter for untilled soil (pasture land) and a mixing depth of 20 centimeter for tilled soil (tilled agricultural land), the same values that were used at proposal. In the SAB review of the draft Addendum (U.S. EPA, 1993a), the SAB agreed that these were reasonable values to use. These are also the same values used in the draft dioxin reassessment and the* Mercury Study Report to Congress *(U.S. EPA, 1997c).*

Comment 495: The commenter notes that the dose of dioxin/furan ingested from contaminated fish is proportional to the lipid content of edible portions of fish and that EPA has selected a lipid content of 7 percent for edible portions of fish based on limited data. The commenter points out that large compilations of the lipid content of fish found in waters of the United States (the Great Lakes and other water bodies) exist and that based on the Great Lakes compilation, the overall lipid content for edible portions of all game fish is approximately 5 percent. The commenter summarizes the lipid contents provided in the study as follows: edible portions of salmonids from the Great Lakes have a lipid content of approximately 6.7 percent, while nonsalmonids have a lipid content of approximately 3 percent. Many nonsalmonid species have lipid concentrations in edible portions that range from 1 to 2 percent; indeed, only one nonsalmonid species (channel catfish) has a lipid content greater than 5 percent. The commenter notes that because lipid content is proportional to dose, a reduction in the lipid content from 7 percent to 5 percent would result in a corresponding 30 percent reduction in risk.

Response 495: *EPA agrees with the commenter that the fish lipid content used in the proposed rule was too high to truly represent a central tendency value, as intended. Fish lipid content will vary greatly even within a given species depending on age, physiological state, availability of food, season, and other factors. For the final rule, EPA concluded that lipid content should represent a lipid fraction in the fillet for a wide variety of species across the United States. For the human health analysis, EPA used a lipid content of 2.6 percent, which was estimated assuming 36 percent consumption of trophic level 3 fish (lipid content 2 percent) and 64 percent consumption of trophic level 4 fish (lipid concentration 3 percent). This is consistent with a lipid content of 3.1 percent recommended in the final rule of the* Great Lakes Water Quality Initiative *for edible portions of trophic level 4 fish (60 FR 15373-15374).*

For the ecological risk assessment, EPA used higher lipid contents representative of the whole fish, including internal organs. A lipid content of 6.5 percent was used for trophic

level 3 fish and a lipid content of 10.3 percent for trophic level 4 fish. The intake of trophic level 3 and 4 fish was assumed to vary by ecological receptor.

***Comment 496, 493*:** The commenter highlights the effects of combining multiple high-end values for the subsistence farmer in a table. The commenter points out that EPA has combined several high-end variables (for example, the 90th percentile of emissions, high-end dietary practices, and farm location) to construct its high-end scenario and notes that this holds true even while maintaining the closer location (and therefore higher air concentrations) of the subsistence beef farmer scenario. Analysis of the sensitivity of the dioxin/furan concentration performed by the commenter shows that for the subsistence farmer, the concentration increases more than 300 percent from that calculated using the feed rate, cattle diet, and farm location of the typical farmer scenario. The commenter provides tabulated results in support of this statement.

The commenter notes that EPA changes the location of the farm and the river/stream for the subsistence farmer so as to maximize dioxin/furan concentrations. The commenter provides a table that shows the effects of changing receptor location with respect to maximizing dioxin/furan concentration and cattle diet. The commenter performs an analysis of the sensitivity of the dioxin/furan concentrations in beef to feed levels; this analysis shows that the central tendency dioxin/furan TEQ concentration for the subsistence farmer increases nearly 100 percent from that calculated using the farm location of the typical farmer scenario.

Response 496, 493: *In the risk assessment for the proposed rule, EPA did not change the location of farms to maximize dioxin/furan concentrations. As explained in the background document for the proposed rule (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, February 1996*), the locations of actual farms thought to be engaged in subsistence farming were identified by local officials. These locations were then used for assessing exposures to persons engaged in subsistence farming. In contrast, the typical farmer scenario used was intended to represent an entirely different exposure scenario, one that would provide a central tendency estimate for nonsubsistence farms located anywhere within 20 kilometers of HWCs. Both the typical farmer and subsistence farmer scenarios were assessed to illustrate the wide range of exposures that could result from emissions from HWCs.*

EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. For the final rule, EPA did not use high-end values. In all instances, EPA either used central tendency values or conducted exposure parameter variability analysis to capture a range of exposure, in conjunction with site-specific data. The variability analysis was conducted for key exposure pathways and facilitates

characterization of the full range of exposures without the use of high-end or upper-bound exposure assumptions.

For the final rule, EPA evaluated 76 active HWC facilities. Site-specific stack gas concentration data from trial burn and certificate of compliance tests were used in conjunction with stack gas flow rates to estimate emissions for each facility.

As indicated previously, different consumption rates were used for livestock, depending on whether the livestock were raised commercially or for subsistence purposes. Commercial beef and dairy cattle were assumed to consume more grain and less forage than subsistence cattle.

The risk assessment for the final rule was modified to account for both the number of individuals exposed and their location. The analysis used U.S. Census and Census of Agriculture data to locate and enumerate persons living in both farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. However, it was not possible to identify and locate from census data individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios were retained in the risk analysis for the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. To assess the potential risks, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA recognizes that this assumption may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

***Comment 497**:* The commenter provides a table of the effects of the use of high-end values rather than central tendency values for the subsistence fisherman scenario. (The parameters shown in this table were altered individually; all other values were set to the standard values found in the MACT risk analysis.) In particular, the commenter evaluated the effects of altering the values of lipid concentration, soil mixing depth, sediment organic carbon fraction, total suspended solids, enrichment ratio, and bed sediment porosity. The commenter notes that, not surprisingly, altering the input parameters for the aquatic food chain pathway can have a large impact on the final media concentration. The commenter notes that altering the fish lipid content or the enrichment ratio, for example, reduces the dioxin/furan TEQ concentration in fish to one-half to one-third of its standard

value. The high and low input values used by the commenter in this evaluation are found in the draft dioxin reassessment and the HWIR rule support documents.

***Response 497**: EPA recognizes the sensitivity of the estimated exposures for the subsistence fishing scenario to the parameter values selected for use in the surface water model. As explained in the response to the previous comment, EPA reduced the fish lipid content for human consumption from 7 percent to 2.6 percent for the final rule. The previously used fish lipid content of 7 percent represented the whole fish, as opposed to the fillet portion typically consumed by humans. This change results in a considerable reduction in dioxin levels in fish and in human exposures that occur through the consumption of fish.*

Also, as indicated in response to the previous comment, EPA retained the 1 centimeter soil mixing depth for untilled soils in the risk assessment for the final rule. This is the same mixing depth that was used for watershed soils and is consistent with the value used in the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c).*

EPA acknowledges that total suspended solids (TSS) concentrations can vary widely. For the proposed rule, a fixed default value was used for all waterbodies. However, due to the importance of this parameter in the partitioning of dioxins within the water column and between the water column and sediments, region-specific TSS values were used for the final rule depending on the type of waterbody (i.e., lakes vs. rivers and streams).

For the final rule risk analysis, EPA used a value of 0.045 for the fraction of organic carbon in suspended sediment, as compared to a value of 0.075 used for the proposed rule. This value was changed because it is derived from the fraction organic carbon in soil, which was updated from 0.01 to 0.006 (U.S. EPA, 1996). The 0.045 value is derived from the soil organic carbon fraction using a fraction organic carbon (suspended sediment) to fraction organic carbon (soil) ratio of 7.5 (U.S. EPA, 1993a). The fraction of organic carbon in bottom sediment is used in modeling transport to the aquatic food chain. The fraction of organic carbon in bottom sediment also was updated from the value used in the proposed rule (0.04) to 0.014, the mean value reported in Suedel and Rodgers (1991). The value of 0.04 had been derived in a manner similar to that used to estimate the fraction of organic carbon in suspended sediment, using a ratio of the fraction organic carbon in bottom sediments to the fraction organic carbon in soil of 4 (U.S. EPA, 1993a). However, in the case of dioxins/furans, the Kd_{bs} was calculated based on the fraction of organic carbon in bottom sediment and the Koc. This calculation to derive Kd_{bs} was based on the fraction of organic carbon in bottom sediment; the value used was left as the value derived from the fraction organic carbon in soil. As a result, for dioxins/furans, the fraction of organic carbon in bottom sediment was 0.024 (4 x 0.006).

The soil enrichment ratio accounts for the fact that eroded soils are generally enriched in fine particles sizes and organic matter, as well as chemical contaminants that bind to organic matter, such as dioxins. It generally ranges from 1 to 5. EPA retained the value of 3 used in the proposed rule as a reasonable central tendency value for the final rule. This is the default value recommended in the 1993 Addendum (U.S. EPA, 1993a). EPA also retained the bed sediment porosity of 0.6 used as a central tendency value for consolidated sediments in the proposed rule. This value was calculated from the bed sediment concentration (assumed to be 10^6 mg/L for consolidated sediments) and the particle density (assumed to be 2.6 g/cm^3) and is consistent with the method used in the 1993 Addendum (U.S. EPA, 1993a).

***Comment 498, 499, 1062**:* The commenter states that EPA's risk methodology does not properly predict the distribution of congeners found in beef data collected by the USDA and EPA. The commenter notes that, as described in the Chlorine Chemistry Council (CCC's) comments on the draft dioxin reassessment, USDA and EPA conducted a joint statistical analysis of dioxin/furan concentrations in beef fat (specifically cited by the SAB in its comments as indicating that background body burdens may be much less than previously estimated in the draft dioxin reassessment). Further, the commenter points out that this is the only statistically designed study of dioxin/furan concentrations in beef fat, and not only do its results provide a sound basis upon which to evaluate the accuracy of the MACT risk model, but EPA has used similar data to evaluate the risk model used in the draft dioxin reassessment.

The commenter states that the USDA/EPA analysis conducted by the commenter calculated the dioxin/furan beef concentrations for the subsistence and average farmer for each of the case studies examined by EPA in the MACT risk assessment Background Document using an air matrix based on the background air matrix found in the draft dioxin reassessment and the EPA model used at proposal. The resulting beef concentration congener distribution calculated from EPA's model was compared with the USDA data. The commenter provides figures that show that the model overpredicts the percentage of furans in the beef relative to the contribution from dioxins and notes that this error is common to all pathways of the model. Further, the commenter notes that the model's overestimation is particularly high for the 2,3,4,7,8 PECDF congener, and because furans are more likely to be associated with combustion processes, this error relates directly to an overestimation of the contribution of combustion sources. The commenter feels that this discrepancy contributes to a 30 percent increase in the contribution of furans relative to dioxins compared with the USDA beef data. The commenter feels that because the EPA model overestimates the contribution from those congeners associated with incineration and underestimates the contribution from those congeners associated with other sources, the model is inaccurate and should not serve as the basis for a beyond-the-floor dioxin/furan standard.

The commenter provides a figure with results of a similar analysis conducted for dioxin concentrations in fish using fish data derived from the North America data supplied in the draft dioxin reassessment. The commenter notes that similar results were found and that the model overestimates

the 2,3,4,7,8-pentachlorofuran contribution by approximately 25 percent. Given these results, the commenter feels that it is clear that EPA's model is inaccurate and should not serve as the basis for beyond-the-floor dioxin/furan standards.

***Response 498, 499, 1062**: EPA recognizes the importance of model validation in developing and applying indirect exposure models, including the model used for the final rule, to predict dioxin/furan concentrations in beef and fish.*

EPA's research program includes continued development of models for assessing indirect exposure to dioxins and other bioaccumulative compounds. While it has not been possible to validate the entire system of models that make up the indirect exposure methodology, components of the indirect exposure models have been validated using field data. In addition, many of the modeling components and supporting parameter values are based on empirical data (e.g., bio-uptake factors used to predict congener concentrations in plants, animals, and fish). EPA believes that the best models currently available and the most up-to-date parameter data have been used for the HWC final rule. Nevertheless, EPA recognizes that significant uncertainty is associated with media concentrations predicted by the models. Consequently, EPA has included an expanded discussion of uncertainty associated with the indirect exposure modeling in the Background Document (RTI, 1999) accompanying the final rule.

EPA recognizes the value of conducting model validation studies using empirical or field measurements. These studies compare output from the model, in the form of predicted media concentrations (e.g., soil, water, fish, beef), to measured data for those same media. By comparing the predicted concentrations to the measured concentrations, conclusions regarding the performance of the model can be drawn. However, the utility of these types of model validation studies is heavily dependent on two criteria related to the underlying measured data that form the basis of the analysis:

*First, there needs to be spatial and temporal correlation between model inputs and target model outputs. It is important that data used to characterize the input data be consistent with that used to characterize the observed concentrations that will be compared to model output data. This needs to be true from both a spatial and temporal standpoint (i.e., the two data sets should cover the same location and be drawn from similar time periods). If the input data and observed concentration data are obtained from different locations, then failure to match model output to observed data may be due to inconsistencies between the input data and the observed data. These inconsistencies may vary with regard to either dioxin/furan concentrations or congener mix. That is, the dioxin/furan congener mix in the model inputs (e.g. air and deposition data) may be different from the*

observed data (e.g. measured beef tissue concentrations) simply because they were taken from different locations and represent different time periods.

- *Second, the measured data sets need to be representative. The data sets characterizing both the model input data and observed data to be compared to model output should be representative of the study conditions used for model validation.*

If either of the above criteria is not satisfied, then a significant level of uncertainty will be associated with the validation results.

Although EPA commends the commenter for undertaking such an effort, the data that were employed in the analysis do not meet these criteria. Limitations of the data include:

- *Uncertainty in the data characterizing dioxin/furan background concentrations in rural ambient air: In the dioxin reassessment EPA noted that there are insufficient data to characterize dioxin/furan ambient air concentrations in rural areas of the United States. Consequently, measured data for dioxin/furan concentrations in urban/suburban air were adjusted to reflect rural areas through the use of a simple scaling procedure that assumed that the mix of congeners in rural areas matches that in urban areas.*

- *Background dioxin/furan concentrations for rural air and for soil, beef, and fish media are not well correlated: The commenter bases the model validation on background data sets provided in the dioxin reassessment. However, these data sets provide coverage for different areas of the U.S. that may be impacted to a greater or lesser extent by dioxin/furan and may reflect different mixes of dioxin/furan congeners. The specific mix of congeners reflects the original anthropogenic sources; consequently, different locations can display different congener mixes.*

Although data sets available for characterizing soil, beef, and fish tissue concentrations are more extensive than those available for characterizing ambient air, the issue of spatial correlation between the beef and fish tissue and soil data sets and the ambient air data set still exists.

EPA recognized in the dioxin reassessment the uncertainties implicit in conducting validation studies using such data. Although the results of the comparison performed by the commenter are of interest, EPA believes they are subject to considerable uncertainty and do not warrant the conclusions reached by the commenter.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA, and was done to determine if the technology-based standards are protective enough to satisfy RCRA, or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 501*:** The commenter notes that use of revised TEFs (RTEFs) developed by EPA would result in a greater than 30 percent reduction in TEQs from fish and beef and a 25 percent reduction of total background emissions from incineration. The commenter states that EPA does not use RTEFs (which are based on 90-day feeding studies) to calculate dioxin/furan TEQ emissions from the HWCs. The commenter feels that, given the availability of RTEFs, it is inappropriate for EPA to estimate the risks associated with chronic exposure to dioxin/furans using TEFs based on acute exposure. The commenter feels that the use of RTEQs would impact EPA's conclusions regarding the contribution of dioxin/furan emissions from HWIs as well as the absolute risk associated with particular incinerators. The commenter notes that while the RTEFs do not include revised values for hexachloro- or heptachloro-substituted dioxin/furans (Birnbaum and DeVito, 1996), EPA acknowledges in the draft dioxin reassessment that, based upon the cancer bioassay of a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HxCDDs, the TEF for these congeners would be reduced by a factor of 2.

Using actual emission data from on-site and commercial hazardous waste incinerators, the calculated reductions in TEQ emissions (as the percent reduction from TEQ emissions reported in the proposed rule) are shown below:

Category	**RTEF Correction Only**	**RTEF and HxCDD Correction**
On-site Incinerator	35 percent	49 percent
Commercial Incinerator	36 percent	37 percent

The commenter provides two tables that show multipathway risk estimates for selected exposure scenarios of incinerator case studies. These tables illustrate that reductions in TEQ emissions are magnified when the RTEFs are applied to EPA's MACT risk methodology. The commenter points out that because this methodology overestimates the contribution from furans (particularly 2,3,7,8-TCDF and 2,3,4,7,8-PCDF), the use of RTEFs reduces the risks associated with ingestion of dioxin/furan contaminated fish and beef by a factor of 2 and that these risks would be further reduced ff the correction for HXCDD were applied. The commenter provides two figures that compare the relative RTEQ contributions of dioxin/furans using empirical concentrations for beef and fish and those

predicted by the MACT risk model using RTEFs. Of interest is that the use of RTEFs corrects for some of the overestimation of furans in beef and fish. The commenter notes that this is the only instance where dioxin TEQs are predicted to exceed furans in accordance with empirical results.

Response 501*: EPA recognizes there is some uncertainty regarding the most appropriate values for the toxicity equivalence factors (TEFs) for the various chlorinated dioxin and furan congeners. TEFs were developed as a practical and scientifically defensible approach for addressing risks associated with complex environmental mixtures of dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs). The consensus TEF values presented in* Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-dioxins and Dibenzofurans (CDDs and CDFs) and 1989 Update *(U.S. EPA, 1989) as well as recent updates to the TEFs by the World Health Organization (Van den Berg et al., 1998) were meant to be order of magnitude estimates. As such, deviations of the magnitude cited by the commenter are certainly within the range of uncertainty inherent in the TEF approach. The TEFs used in the final rule are those that were recently published in* Environmental Health Perspectives *(Van den Berg et al., 1998). These values, which were made available to EPA prior to their publication, reflect a consensus from the international community. The TEFs were developed from all available mammalian, bird, and fish studies previously reviewed as well as new studies published over the last several years. These studies were carefully evaluated and a database was developed using the following criteria:*

- *At least one CDD, CDF, or PCB congener and a reference compound must be included in the study.*
- *Either TCDD or PCB 126 must be included as a reference compound in the same experiment or studied with the same experimental design by the same authors in another experiment.*
- *The relevant end point should be affected by the congener studied as well as the reference compound.*

The revised TEFs reflect only limited changes to the 1989 interim TEFs. Most TEFs were not changed. Exceptions include OCDD and OCDF (reduced from 0.001 to 0.0001) and 1,2,3,7,8-PeCDD (raised from 0.5 to 1). EPA believes the revised TEFs from the World Health Organization are the most appropriate values for use in the HWC final rule.

Comment 643: The commenter feels that EPA's risk methodology overestimates exposure to dioxins/furans because background soil- and water-related concentrations of dioxins/furans are overstated by factors that range from 2.5 to 119. The commenter notes that the only complete set of calculations provided by EPA (outside of the Administrative Record) is for the water pathway for Case H in the Background Risk Assessment Document and that water-related concentrations were calculated using the background dioxin/furan matrix provided in the draft dioxin reassessment rather

than the emissions matrix for Case H. The commenter points out that EPA relied upon the background matrix as part of its validation of the beef pathway model.

The commenter includes a table to compare calculated to background dioxin TEQ concentrations for the water column, bed sediment, and soil. The commenter states that the table indicates that the MACT risk model overestimates the dioxin/furan contribution from the water pathway compared to actual background measurements found in the draft dioxin reassessment. The commenter also provides graphical representations of dioxin/furan congener distributions in beef to compare USDA study data and MACT model predictions, as well as dioxin/furan congener distributions in fish to compare the dioxin reassessment and MACT model predictions.

Response 643: *As indicated previously, EPA recognizes the importance of model validation in developing and applying indirect exposure models, including the model used to predict dioxin/furan concentrations in fish.*

EPA's research program includes continued development of models for assessing indirect exposure to dioxins and other bioaccumulative compounds. Although it has not been possible to perform a true model validation study on dioxins in surface water and uptake in fish, EPA notes that many of the modeling components and supporting parameter values are based on empirical data (e.g., bio-uptake factors used to predict congener concentrations in fish). Although EPA believes that the best models currently available and the most up-to-date parameter data have been used for the HWC final rule, EPA recognizes that significant uncertainty is associated with media concentrations predicted by the models. Consequently, EPA has included an expanded discussion of uncertainty associated with the indirect exposure modeling in the Background Document (RTI, 1999) accompanying the final rule.

EPA recognizes the value of conducting model validation studies using empirical or field measurements. These studies compare output from the model, in the form of predicted media concentrations (e.g., soil, water, fish), to measured data for those same media. By comparing the predicted concentrations to the measured concentrations, conclusions regarding the performance of the model can be drawn. However, the utility of these types of model validation studies is heavily dependent on two criteria related to the underlying measured data that form the basis of the analysis:

*First, there needs to be spatial and temporal correlation between model inputs and target model outputs. It is important that data used to characterize the input data be consistent with that used characterize the observed concentrations that will be compared to model output data. This needs to be true from both a spatial and temporal standpoint (i.e., the two data sets should cover the same location and be drawn from similar time periods). If the input data and observed*

concentration data are obtained from different locations, then failure to match model output to observed data may be due to inconsistencies between the input data and the observed data. These inconsistencies may with regard to either dioxin/furan concentrations or congener mix. That is, the dioxin/furan congener mix in the model inputs (e.g. air and deposition data) may be different from the observed data (e.g. measured beef tissue concentrations) simply because they were taken from different locations and represent different time periods.

- *Second, the measured data sets need to be representative. The data sets characterizing both the model input data and observed data to be compared to model output should be representative of the study conditions used for model validation.*

If either of the above criteria is not satisfied, a significant level of uncertainty will be associated with the validation results.

Although EPA commends the commenter for undertaking such an effort, the data that were used in the analysis do not meet these criteria. Limitations of the data include:

- *Uncertainty in the data characterizing dioxin/furan background concentrations in rural ambient air: In the dioxin reassessment EPA noted that there are insufficient data to characterize dioxin/furan ambient air concentrations in rural areas of the United States. Consequently, measured data for dioxin/furan concentrations in urban/suburban air were adjusted to reflect rural areas through the use of a simple scaling procedure that assumed that the mix of congeners in rural areas matches that in urban areas.*

- *Background dioxin/furan concentrations for rural air and for soil and fish media are not well correlated: The commenter bases the model validation on background data sets provided in the dioxin reassessment. However, these data sets provide coverage for different areas of the U.S. that may be impacted to a greater or lesser extent by dioxin/furan and may reflect different mixes of dioxin/furan congeners. The specific mix of congeners reflects the original anthropogenic sources; consequently, different locations can display different congener mixes.*

Although data sets available for characterizing both soil and fish tissue concentrations are more extensive than those available for characterizing ambient air, the issue of spatial correlation between the fish tissue and soil data sets and the ambient air data set still exists.

EPA recognized in the dioxin reassessment the uncertainties implicit in conducting validation studies using such data. Although the results of the comparison performed by the commenter are of interest, EPA believes they are subject to considerable uncertainty and do not warrant the conclusions reached by the commenter. In particular, the results of the commenter's comparison for fish suggests much closer agreement: the ratio of the fish concentrations calculated from background air concentrations to the background fish concentration ranges from 0.1 to 2.7, with a median of 0.5, suggesting a tendency toward underprediction, not overprediction.

Comment 503: The commenter states that, given the inaccuracies of the risk model used in the proposed rule and the implications for the basis of the rule, EPA should revise its model. The commenter states that EPA must validate the reasonableness of high-end food consumption scenarios based on new data because, by using old data, EPA overestimates exposure. The commenter believes that high-end food consumption scenarios need to be reevaluated in light of the recent USDA/EPA back fat study, which suggests that average levels are 25 percent lower than the levels EPA presents in the draft dioxin reassessment and the proposed rule. The commenter states that, despite these data, EPA relies on high-end food consumption scenarios, which are based on an unverified assumption: that subsistence farmers and fisherman actually consume much of their diets from farms and streams that are contaminated.

Response 503: *The joint USDA/EPA data for dioxins/furans in beef fat are most representative of background exposure levels and are not comparable to exposures in areas affected by HWC emissions. Therefore, it is inappropriate to use the USDA/EPA data to draw conclusions about the results of the HWC risk analysis.*

For the final rule, EPA modified the exposure scenarios to be more representative of the exposed population at each facility. Separate exposure estimates were made for individuals engaged in farming for an occupation or fishing for recreation and persons engaged in farming or fishing for subsistence. EPA used central tendency exposure factors to estimate exposures, except for a few risk-driving pathways for which an exposure factor variability analysis was performed, using Monte Carlo simulation. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative and, when taken together, are not likely to substantially overestimate (or underestimate) the range of possible exposures.

Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence farming and fishing does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals.

The same food intake rates were used for subsistence farmers as were used for farmers engaged in farming for commerce. Subsistence farmers were assumed to subsist entirely on food produced on the farm, while commercial farmers were assumed to consume only the primary commodity they produced for sale. EPA derived food intake rates from data collected on utilization of home-produced foods by households from the U.S. Department of Agriculture's Nationwide Food Consumption Survey (as cited in the 1997 Exposure Factors Handbook*, U.S. EPA, 1997a). For subsistence fishing, EPA used fish intake rates representative of Native American tribes from the Columbia River basin, who obtain a significant portion of their dietary intake from fish. The use of exposure factors derived from such studies ensures the plausibility of the exposure scenarios EPA used to characterize risks to individuals who may be engaged in subsistence activities.*

Comment 504: The commenter states that the EPA administrative record for the proposed rule is incomplete for evaluation of the risk assessment calculations because EPA has omitted the basic calculations for its risk assessment. Further, the commenter notes that the administrative record contains mistakes and does not include the spreadsheets (i.e., specific calculations and variables) upon which the risk calculations for each scenario or pathway are based, in contrast to the recently proposed HWIR rule (for which EPA made available a CD-ROM disk with some 50 megabytes of spreadsheets so all calculations could be reproduced). EPA did not grant or formally respond to CMA's request (letter from CMA dated July 23,1996) for inclusion of all calculations in the administrative record.

The commenter notes that the administrative record contains several errors in that a number of physical-chemical parameters identified therein did not match calculations EPA provided to CCC outside of the record (the commenter includes Attachment D, *Constants Used in Media Concentration Calculations for the Maximum of the High-end Exposures Through the Fish Ingestion Pathway for Cement Kilns* and *Walkthrough Calculations for the Maximum of the High-end Exposures Through the Fish Ingestion Pathway for Cement Kilns* in support of this point). Examples of these errors include the Henry's Law constant of PeCDD, Fv (vapor fraction) for OCDF, operating hours (7,324 hours vs. 7,560 hours listed in the documentation). The commenter believes that such errors make clear the need to provide complete model documentation so that all calculations may be reviewed.

Response 504: *The background document for the proposed rule documents all equations and parameter values used in the risk analysis at proposal (see* Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February, 1996). Although EPA has not made the spreadsheets and other software that were used in HWC risk analysis part of the administrative record, EPA placed example spreadsheet calculations in the docket for the proposed rule (this information was included as an attachment to a July 17, 1996, memorandum from D. Layland to the file documenting a meeting with cement industry consultants). EPA also made all air dispersion model output files available to the*

public through the RCRA docket (see "Availability of Electronic Files of Dispersion Modeling Results," memorandum from D. Layland to the RCRA Docket dated May 16, 1997). In addition, much of the software that EPA used in the analysis is publicly available. This includes the Industrial Source Complex Short-Term (ISCST)model, which was used for modeling air dispersion and deposition in both the proposed and final rules, and the Integrated Exposure Uptake Biokinetic Model (IEUBK) model, which was used for modeling lead exposures in the final rule. EPA has carefully documented the calculations, variables, and data used in the risk assessment for the final rule and has placed these in the rulemaking docket. In addition, the IEM-2M model, which was used for modeling the fate and transport of mercury in surface water in the risk assessment for the final rule, is fully documented in the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c).*

The discrepancies noted by the commenter have been documented and placed in the administrative record. Errors found in the risk analysis for the proposed rule were corrected for the final rule. EPA corrected the Henry's Law constant for PeCDD. EPA believes the value of the vapor fraction, Fv, for OCDF used in the proposed rule (0.002) is correct, and this is the value that was used for the final rule. For the final rule, each facility was assumed to operated 24 hours per day, 365 days per year, for a total of 8,760 hours.

Comment 1105: The commenter states that, based on materials provided by EPA (outside of the administrative record), a number of physical-chemical parameters identified in the documentation in the administrative record did not match those provided in EPA's calculations (that is, the values listed in the administrative record did not match those of the calculations provided by EPA). Examples of these errors include the Henry's Law constant of PeCDD, Fv for OCDF, and operating hours (7,324 hours vs. 7,560 hours listed in the documentation).

Response 1105: *The discrepancies noted by the commenter have been documented and placed in the administrative record. Errors found in the risk analysis for the proposed rule were corrected for the final rule. EPA corrected the Henry's Law constant for PeCDD. EPA believes the value of the vapor fraction, Fv, for OCDF used in the proposed rule (0.002) is correct, and this is the value that was used for the final rule. For the final rule, each facility was assumed to operate 24 hours per day, 365 days per year, for a total of 8,760 hours.*

***Comment 505*:** The commenter feels that EPA has overstated the risk of dioxin/furans, and therefore the incremental health benefits of BTF standards for HWIs, by selectively relying on the draft dioxin reassessment without considering the explicit comments of the Science Advisory Board (SAB), EPA's own research, or extensive public comments. The commenter states that in the proposed rule, EPA relies on a cancer risk estimate approach that has been criticized by the SAB and which

overstates the health risks associated with background dioxin/furan exposure. The commenter explains that the proposed rule relies on a total TEQ dose of 0.01 pg/kg/day as associated with an upper-bound excess cancer risk of 1 x 10^{-6} and in discussing the human health benefits cites the proposed rule as saying that dioxin risks associated with current background exposures are characterized as "600 annual cancer deaths attributed to dioxin exposures." The commenter believes that it is premature for the proposed rule to incorporate either the dose of 0.01 pg/kg/day as associated with an upper-bound excess cancer risk of 1 x 10^{-6} or the derived 600 annual cancer deaths attributed to dioxin/furan exposures from the draft dioxin reassessment since, at this time, neither of these conclusions is scientifically supportable to the point where it is appropriate to include them in a discussion of human health benefits resulting from of dioxin/furan emission reductions.

Response 505*: The annual number of cancer cases cited by the commenter was EPA's estimate in the preamble to the proposed rule of the potential cancer incidence from background exposures to dioxins, only a portion of which might be attributable to HWCs. The estimate was based on a risk-specific dose of 0.01 picograms on a toxicity equivalent (TEQ) basis per kilogram body weight per day, corresponding to an upper bound lifetime excess cancer risk of one in one million, and an average dietary intake of 120 picograms TEQ per day. Dietary intakes of dioxins have generally declined and EPA now estimates an average dietary intake of approximately 40 pg TEQ per day for adults. This estimate is based on updated food intake rates and measurements of dioxin levels in food obtained from recent national surveys of beef, pork, poultry, and milk.*

*The risk-specific dose of 0.01 pg TEQ/kg-day was EPA's estimate in the 1994 draft dioxin reassessment. However, EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment should be used (*Health Assessment Document for Polychlorinated Dibenzo-p-dioxin, *U.S. EPA, 1985*). *That estimate is 156,000 [mg/kg-d]$^{-1}$. This corresponds to a risk-specific dose of 0.06 pg TEQ/kg-day for a 1 x 10^{-6} lifetime excess cancer risk. In response to comments received from the SAB, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d). This study lends considerable support to the earlier cancer potency estimates from the 1994 draft reassessment and the 1985 health assessment.*

For these and other reasons, EPA is no longer using the cancer incidence estimate mentioned in the preamble to the proposed rule. For the final rule, EPA estimated the annual incidence of cancer associated with dietary intake of dioxins specifically from HWC sources. This was done by estimating the amount of dioxin contained in locally produced meat and milk and assuming that these products enter the national food commodity distribution system and are consumed by the general population. However, as explained in the risk assessment Background Document (RTI, 1999), the analysis for the

final rule does not consider long-range transport of dioxins emitted from HWCs. Only exposures resulting from agricultural commodities produced within 20 kilometers of HWCs were considered. Therefore, EPA expects that its cancer incidence estimate understates the true cancer incidence in the general population and that the degree of underestimation could be significant.

***Comment 506*:** The commenter states that with respect to evaluating the possible carcinogenicity of dioxin/furan, EPA's draft dioxin reassessment relies exclusively on the linear multistage (LMS) model which assumes no threshold. Using the LMS model, the draft dioxin reassessment derives a proposed revised cancer potency factor (CPF) for TCDD of 0.10 ng/kg/day, down from the previous CPF of 0.156 ng/kg/day. The commenter notes that both of these values are derived from the data in Kociba et al. (1978). However, the commenter points out that in reviewing EPA's conclusions regarding dioxin/furan carcinogenicity, the SAB report correctly noted that dioxin/furan was nongenotoxic and was not an initiator of cancer, and "thus is not a complete carcinogen." The commenter cites the resulting conclusions of the SAB regarding EPA's assessment of the possible carcinogenicity of dioxin/furans and underscores their significance. The commenter feels that, given the SAB's clear recommendations to consider a threshold-type cancer risk assessment for dioxin/furans, it is premature to focus exclusively on nonthreshold models as the sole basis for assessing possible carcinogenicity and it is unwarranted to use any cancer potency factors (i.e., 0.156 per ng/kg/day or 0.10 per ng/kg/day) for dioxin/furans in a risk assessment until this is resolved. Consequently, the commenter believes that the proposed rule should not adopt the LMS approach or any conclusions which derive from it until after EPA has responded to the SAB's recommendations.

Response 506: *In its review of the 1994 draft dioxin reassessment, the EPA Science Advisory Board suggested that EPA consider alternatives to the linear nonthreshold model, allowing for minimal response at low environmental levels of exposure, that would be consistent with the body of available health effects data. In response, EPA developed a mechanistic model for liver tumors in female rats using data from a 2-year feeding study in Sprague-Dawley rats (Kociba et al., 1978). EPA also summarized the results of simple empirical models that have been applied to other significant cancer findings in female Sprague-Dawley rats from the Kociba study and in Osborne-Mendel rats and B6C3F1-mice from the National Toxicology Program study (NTP, 1982). In addition, EPA applied simple empirical models to evaluate the shape of the dose-response curve for significant noncancer findings from other animal studies. EPA compared the shapes of the dose-response curves across all the studies and grouped them into those that appeared to be linear across the experimental findings and those that appeared to be nonlinear (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d) EPA found that a majority of the dose-response curves are consistent with linearity but that some are highly nonlinear,*

appearing to have a clearly defined threshold. However, most of the cancer findings (9 of 13, or 70 percent) exhibited response consistent with linearity in the observable range.

Also, few of the mechanistic models that EPA identified in the scientific literature exhibited nonlinear dose-response in the observable region or predicted nonlinear dose-response in the low-dose (extrapolation) region. Results of the two-stage modeling of the Kociba et al. female rat liver tumor data incorporating dioxin-altered hepatic foci data to estimate mutation and growth parameters provide nearly the same low-dose estimates as the linearized multistage (LMS) model using only the tumor data. Unless a protective effect of TCDD on mutation rates occurs at low doses, low-dose risk will remain proportionate to exposure and consistent with the linearized multistage model. If protective effects are allowed in the model, the low-dose risks may be substantially reduced; however, the focal lesion data and the biochemical markers generally agree and do not suggest a protective effect (U.S. EPA, 1997d).

Therefore, there appears to be no strong support for general nonlinearity for TCDD's effects in the range of the data studied and little support for extrapolation into a lower dose range with a highly nonlinear model. To the contrary, EPA believes there are sufficient data suggesting that response is proportional to dose that, when considered together with the available human data, warrant concern that 2,3,7,8-TCDD will induce toxic effects in humans in the range of the experimental animal data. Furthermore, based on a lack of data indicating an immediate and steep change in slope of the dose-response curve, EPA believes the possibility of response 1 to 2 orders of magnitude below this range must be considered.

***Comment 507*:** With respect to possible carcinogenicity associated with dioxin/furan exposure, the draft dioxin reassessment focuses exclusively on highly exposed cohorts from several occupational studies as the underlying human data for cancer risk extrapolations and estimates of possible excess cancer risks in the general population. EPA relies on this data in the proposed rule to support the conclusion that 600 annual cancer deaths can be attributed to dioxin/furan exposure. However, EPA must consider the lack of excess cancer among less-exposed subcohorts in these studies. For example, it is generally acknowledged that the most thorough of the occupational studies is the NIOSH study by Fingerhut et al. ("Cancer Mortality among U.S. Workers Employed in the Production of Chemicals Contaminated with 2,3,7,8- Tetrachlorodibenzo- P-dioxin (TCDD)," Centers for Disease Control, Cincinnati, Ohio, 1991). This study provides the principal human data relied upon by EPA in the draft dioxin reassessment for assessing potential carcinogenicity. This study is comprised of two exposed cohorts, a point generally overlooked in assessments of potential carcinogenicity. The highly exposed cohort of 1,520 men (with greater than 1 year of TCDD exposure and 20 years elapsed after first exposure) serves as the principal basis for EPA's risk assessment in the draft dioxin reassessment (i.e., statistically significant increases in all cancers combined, respiratory cancer, and soft tissue sarcoma). However, there is a legitimate controversy over whether these increases were due to

TCDD or confounders such as smoking, exposure to asbestos or to other chemicals. In the less exposed cohort of 1,516 men (with less than 1 year of exposure to TCDD and over 20 years elapsed since last exposure) with current mean TCDD serum levels of 78 ppt, no excess cancer of any kind was observed. The current mean TCDD serum levels in the less exposed cohort were extrapolated back by Fingerhut et al. to approximately 640 ppt at the time of initial exposure for workers in this group. These human data, showing no carcinogenic effects of TCDD exposure in a group of workers with initial TCDD serum levels of 640 ppt, appear to highlight the rationale behind the SAB's recommendation that EPA consider a threshold approach for estimating possible carcinogenicity of dioxin/furan. In reviewing EPA's assessment of the epidemiology database, the SAB appears to agree that consideration be given to results of studies in less exposed cohorts. The commenter cites a statement by the SAB on the draft dioxin reassessment that the conclusion that dioxin and related compounds are likely to present a cancer hazard to humans at exposure levels within one or two orders of magnitude above background is not well-supported by the existing human epidemiological database. The commenter concludes that EPA's assertion that 600 annual cancer deaths are attributed to dioxin/furan exposure is not supportable.

> ***Response 507*:** *As the SAB pointed out in its report on the draft dioxin reassessment, understanding the operation of bias and chance is especially important when interpreting "negative" results from an epidemiological study, where no differences are apparent or where the differences are not statistically significant. Differences produced by real effects can easily be masked by poor exposure classifications (misclassification bias), a negative association may appear merely by chance by virtue of a small population available for study (poor statistical power), and potential risks can be rendered undetectable by observing the exposed population for too short a time (bias produced by failure to account for adequate latency). These are just a few of the factors complicating interpretation of negative outcomes. On the other hand, spurious positive associations are much less likely to occur in environmental epidemiological studies because more factors operate to lower the observed risks than to raise them. With respect to the Fingerhut study (Fingerhut et al, 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"), no association was reported for the cohort exposed for a year or less. However, such an outcome is not unexpected given that the probability of observing cancer in this cohort is so much less than in the more highly exposed cohort.*
>
> *On the contrary, EPA's analysis of the NIOSH data performed in response to the SAB review of the draft dioxin reassessment found that the ratio of the IADD (Intake Average Daily Dose) for the high- and low-dose groups was the same as the ratio of increased risk for respiratory cancer, indicating a linear dose-response curve. The results for all cancer mortality combined were less clear. However, a similar comparison for total cancer risk using data from the Manz et al. (1991) cohort was consistent with linearity ("Cancer mortality among workers in chemical plant contaminated with dioxin"). The IADD is the continuous dose that yields the same average area under the curve (AUC) for serum*

concentration versus time and was used as the dose metric in EPA's analysis (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d).*

In addition to the NIOSH study, there are epidemiological studies of a number of other cohorts that have found a positive association between dioxin exposure and cancer. These include cancers at many different sites, including malignant lymphomas, soft tissue sarcomas, hepatobiliary tumors, hematopoietic tumors, thyroid tumors, and respiratory tract tumors. These studies include: Hooiveld et al., 1998 ("Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants"); Kogevinas et al., 1997 ("Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: an expanded and updated international cohort study;" Becher et al., 1996 ("Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"); Kogevinas et al., 1995 ("Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: two nested case-control studies"); Kogevinas et al., 1993 ("Cancer incidence and mortality in women occupationally exposed to chlorophenoxy herbicides, chlorophenols and dioxins"); Bertazzi et al., 1993 ("Cancer incidence in a population accidentally exposed to 2,3,7,8-tetrachlorodibenzo-para-dioxin"); and Zober et al., 1990 ("Thirty-four year follow-up of BASF employees exposed to 2,3,7,8-TCDD after the 1953 accident"). Although the Ranch Hand study has yet to find evidence of an increased incidence of cancer, this cohort was not as highly exposed as the other cohorts studied. However, other health effects have been observed in the Ranch Hand veterans, including an increased incidence of diabetes, elevated cardiovascular disease, and a modest decrease in testosterone levels in the most highly exposed veterans.

EPA believes there are sufficient data from animal studies suggesting that response is proportional to dose that, when considered together with the available human data, warrant concern that 2,3,7,8-TCDD will induce toxic effects in humans in the range of the experimental animal data. Furthermore, based on a lack of data indicating an immediate and steep change in slope of the dose-response curve, EPA believes the possibility of response 1 to 2 orders of magnitude below this range must be considered.

As indicated in response to a previous comment, EPA is no longer using the cancer incidence estimate from the preamble to the proposed rule cited by the commenter.

***Comment 508*:** The proposed rule does not give proper weight to the extensive Ranch Hand studies which demonstrate that dioxin/furan exposure is not a risk factor for cancer at moderate exposure levels. The extensive Ranch Hand studies do not support the statement in the proposed rule that humans are likely to respond with a "broad spectrum of effects" from exposure to dioxin/furans.

Although essentially dismissed in the draft dioxin reassessment, the Ranch Hand studies, including extensive clinical evaluations in 1982, 1985, 1987, and 1992 on a cohort of almost 1,000 dioxin/furan-exposed individuals, comprise the most comprehensive analysis ever conducted on a cohort of dioxin/furan-exposed individuals. The Ranch Hand data offer the best database for examining the likelihood of a broad range of effects from dioxin/furans at or near background body burdens of exposure. While it is true that the Ranch Hand cohort involves a significant number of individuals with present TCDD serum levels within the same range as the control group, it is also true that there are numerous individuals with TCDD serum levels substantially elevated from controls (see Table 7). It is critical that effects be considered in the context of the TCDD dose at or soon after exposure. Since the various exposed Ranch Hand cohorts cover the spectrum of exposure with initial TCDD serum levels ranging from approximately 25 to 300 ppt, there is hardly a more relevant database to determine whether dioxin/furan exposures are likely to cause adverse effects at or near background body burden levels of dioxin/furans. In particular, the results of the most recent evaluation of the Ranch Hand cohort appear to support the SAB's recommendation that EPA consider low dose exposure cohorts and a threshold-type model for cancer. In the 1992 Ranch Hand examination results, now with approximately 20 years of latency since exposure in Southeast Asia, there was no statistically significant group differences for any cancer. The conclusions in the most recent Ranch Hand evaluation regarding a possible association between exposure to dioxin/furans and cancer are relevant. The commenter cites a statement in Wolfe et al. (*Air Force Health Study: An Epidemiologic Investigation of Health Effects in Air Force Personnel Following Exposure to Herbicides, 1992 Followup Examination Results,* May 1992 to May 1995, Epidemiology Research Division, Armstrong Laboratory, Human Systems Division (AFSC), Brooks Air Force Base, Texas, 1995) that indicates that at the end of a decade of surveillance and more than 20 years after the last exposure to Agent Orange in Vietnam, Ranch Hands and comparisons appear to be at equal risk for the development of all forms of neoplastic disease and there is no evidence to suggest a positive dose-response relationship between body burden of dioxin and neoplastic disease. The commenter says these results are consistent with the finding of no excess cancer in the less exposed cohort of workers in the NIOSH study. Based on studies in which TCDD body burdens have been measured in at least some members of a cohort, at least three studies now suggest that TCDD serum levels of 143-640 ppt are not associated with increased cancer risk (Fingerhut et al., 1992; Smith et al., 1992; and the 1992 Ranch Hand results). The commenter provides a table that illustrates the relevant data. These data were summarized in comments submitted to EPA (as well as to the SAB) by CDC (Fingerhut, et al., 1991, *Cancer Mortality among U.S. Workers Employed in the Production of Chemicals Contaminated with 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD),* Centers for Disease Control, Cincinnati, Ohio; Smith, A.H. and Pearce, N.E. 1986, "Update on Soft Tissue Sarcoma and Phenoxyherbicides in New Zealand," *Chemosphere* 15:1795-1798; Smith et al., 1992, "Serum 2,3,7,8-tetrachlorodibenzo-p-dioxin Levels of New Zealand Pesticide Applicators and Their Implication for Cancer Hypotheses," *J. Natl. Cancer Inst.* 84(2):104-108.)

> ***Response 508****: EPA believes an evaluation of hazard and risk must rely on a weight-of-evidence approach, in which all available data (animal and human) are examined*

together, a process that often requires extrapolation of effects across various animal species as well as to humans. EPA believes the weight of the evidence supports the conclusion that 2,3,7,8-TCDD and other dioxin-like compounds are probable human carcinogens. Although the Ranch Hand study has yet to find evidence of an increased incidence of cancer, epidemiological studies of a number of other cohorts exposed at higher levels than the Ranch Hand veterans have found a positive association between dioxin exposure and cancer, in addition to the NIOSH study, as indicated in response to a previous comment. These include cancers at many different sites, including malignant lymphomas, soft tissue sarcomas, hepatobiliary tumors, hematopoietic tumors, thyroid tumors, and respiratory tract tumors. With respect to the concerns about soft tissue sarcomas (STS), there is a general indication that exposure to phenoxy acids with known dioxin contamination are associated with STS. While the New Zealand studies (e.g., Smith and Pearce, 1986, "Update on soft tissue sarcoma and phenoxy herbicides in New Zealand") did not find an increase in STS, other reports do suggest an increased risk. For example, Kramarova et al., 1998 ("Exposure to Agent Orange and occurrence of soft-tissue sarcomas or non-Hodgkin lymphomas: an ongoing study in Vietnam"), discusses an association between STS and phenoxy herbicide exposure in Sweden. The NIOSH study also provides evidence (Fingerhut et al., 1991) for significant increases in death from STS in the high-exposure cohort, although EPA acknowledges the concerns raised by the commenter regarding the findings of this study. Both Saracci et al. (1991) and Kogevinas et al. (1997) report increased STS rates for exposed workers in several countries, although EPA also recognizes the weaknesses that exist in these studies. In the case of Kogevinas et al. (1997), the increase in mortality for STS was not statistically significant and neither the latency periods nor the length of probable exposure to TCDD were associated with increased mortality or incidence of STS. Kramarova et al., 1998, also report an increased risk for STS from an analysis of the combined results of an analysis that combines the results from U.S. pesticide workers (Fingerhut et al., 1991, "Cancer Mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"), German workers (Becher et al., 1996,"Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"), and Dutch workers (Hooiveld et al., 1996, "Preliminary results of the second follow-up of a Dutch cohort of workers occupationally exposed to phenoxy herbicides, chlorophenols and contaminants"). EPA also notes that other health effects have been observed in the Ranch Hand veterans, including an increased incidence of diabetes, elevated cardiovascular disease, and a modest decrease in testosterone levels in the most highly exposed veterans.

The SAB, in its review of the draft dioxin reassessment, agreed with EPA, concluding that dioxin is likely to increase human cancer incidence under some conditions of exposure. The SAB categorized dioxin and dioxin-like compounds as a B1 carcinogen under the 1986 EPA cancer guidelines (U.S. EPA, 1986) of "agents for which there is limited evidence of carcinogenicity from epidemiologic studies." In addition, the

International Agency for Research on Cancer (IARC) recently reviewed the experimental data and epidemiological studies and also concluded that 2,3,7,8-TCDD is carcinogenic to humans (WHO, 1997; McGregor et al., 1998).

As the SAB pointed out in its report on the draft dioxin reassessment, understanding the operation of bias and chance is especially important when interpreting "negative" results from an epidemiological study, where no differences are apparent or where the differences are not statistically significant. Differences produced by real effects can easily be masked by poor exposure classifications (misclassification bias), a negative association may appear merely by chance by virtue of a small population available for study (poor statistical power), and potential risks can be rendered undetectable by observing the exposed population for too short a time (bias produced by failure to account for adequate latency). The effect of these factors is magnified at lower exposures. These are just a few of the factors complicating interpretation of negative outcomes. On the other hand, spurious positive associations are much less likely to occur in environmental epidemiological studies because more factors operate to lower the observed risks than to raise them.

***Comment 509*:** The commenter cites the proposed rule and states that EPA inappropriately relies on studies suggesting cancer increases in the Seveso population to support the proposed rule. The commenter asserts that it is inappropriate for EPA to cite results from the Seveso studies to support to the conclusions of the draft dioxin reassessment or the proposed rule concerning possible carcinogenicity following exposure to dioxins/furans given that cancer incidence data recently have been published from the first 10-year follow-up studies (1976-1986) on the exposed cohorts (Bertazzi et al., 1993; Pesatori et al., 1994). The commenter summarizes the findings and notes that in the zones with the highest presumed exposure to TCDD, there have been no higher incidence of soft tissue sarcoma (STS) than would be expected in a population this size. According to the commenter, the two recent studies of the cancer incidence in the Seveso population purport to demonstrate an association between exposure to TCDD and subsequent development of cancer at a variety of sites. These two studies report separately on cancer incidence in the children (ages 0-19) and adult (ages 19 and older) populations as of 1986, representing a latency period of only 10 years and not 20 years, as is typically believed to be necessary for chemically induced cancers. These studies show no consistent pattern of elevated cancers between zones, sexes, or populations (young and adult). The commenter states that the results from Seveso on people aged 0-19 fail to support the conclusion of EPA in the proposed rule that significant increases in certain cancers have occurred and that none of the results in the Seveso study on people age 0-19 (Pesatoti et al., 1994) are statistically significant. Further, the commenter notes that the results of this study are inconsistent with the Seveso study on adults (Bertazzi et al., 1993) as well as with all of the occupational cohort studies. This study is a review of cancer occurrence in approximately 20,000 people age 0-19 who lived in the various contaminated areas surrounding Seveso. Because of small sample sizes in zones A and B, all three contaminated zones (A, B, and R) were combined into a single group for purposes of this analysis. This eliminated any possibility of

determining a dose-response relationship between exposure to TCDD and outcome. The rationale for aggregating individuals age 0-19 in this study and all adults in the other study is not explained. It is possible that the results might be a function of where the age distribution was divided. One might also speculate whether any results would still be positive if both populations were combined. The commenter emphasizes that the most important point about this study is the fact that none of the relative risks reported were statistically significant. While the absolute number of subjects living in each zone was not reported, it is clear that people from zone R (the reference zone) dominates the exposed cohort. Since there are no data on average soil levels of TCDD in the "noncontaminated" reference zone, it is impossible to demonstrate that there are exposure differences between the majority of the exposed cohort and the reference cohort. Finally, none of the cancers discussed (i.e., ovary, brain, thyroid, Hodgkin's lymphoma, and leukemia) were elevated in the companion study in adults nor in any of the occupational cohort studies.

In summary, the commenter believes that the results of the various epidemiological studies on the Seveso population conducted to date should not be used as the basis for inferring or assessing possible cancer risks associated with exposure to TCDD. Currently, the available Seveso data fail to satisfy most of the recognized causation criteria (i.e., strength of association, dose-response, consistency of the association, specificity of the association, and coherence of the evidence.) Given these shortcomings, the commenter feels EPA should not rely on these studies to support inferences of possible carcinogenicity of dioxins/furans and that adverse effects attributable to chronic low-level dioxin exposure have not been adequately demonstrated.

> ***Response 509****: EPA recognizes the limitations of the Seveso studies and for this and other reasons EPA does not cite the Seveso incident in the preamble to the final rule. This 1976 accident is the most widely studied release of 2,3,7,8-TCDD primarily involving residential exposures. It was estimated that more than 1.3 kg of 2,3,7,8-TCDD was released into the atmosphere and that more than 17,000 people in a 2.8 km^2 area adjacent to the facility were exposed. To investigate this accident, the contaminated area was separated into regions A, B, and R based on soil levels of 2,3,7,8-TCDD. The regions had population sizes of 736, 4,737, and 31,800, respectively. These regional groupings do not take into consideration actual exposure levels and differences in within-zone 2,3,7,8-TCDD exposure. Zone B showed the most consistent pattern of cancers of the digestive and lymphohemopoietic systems. Cancer incidence results reported in 1976 and 1991 have been supported in the most recent studies of Seveso (Bertazzi et al., 1998, "The Seveso studies on early and long-term effects of dioxin exposure: a review").*
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> *EPA understands well that several inconsistencies and confounding factors exist for the results from the Bertazzi and Pesatori Seveso studies: the pattern of cancer cases does not support a dose response relationship (Bertazzi); childhood risks for Hodgkin's lymphoma, myeloid leukemia, and thyroid cancer for the exposed population were raised but lacked statistical significance (Pesatori); latency periods, to date, may be too small to*

detect increases in cancer risks (Bertazzi and Pesatori); the population size in the highest exposed region may be too small to show possible cancer rate increases; and the exposure to dioxin was accompanied by exposure to other possible carcinogens. The weight-of-evidence for dioxin carcinogenicity from the Seveso studies, however, has shown a consistency of increased cancer risks over the past 23 years. Though the highest exposure group is small and has not shown significant increases in cancers, results for the second highest exposure group, Zone B, does support the cancer hypothesis. Both EPA and the international community have reviewed dioxin cancer studies and have found strong causal evidence from animals studies and the cumulative evidence from other epidemiological studies of occupational exposure to dioxin containing herbicides that strongly points to dioxin carcinogenicity.

Despite the limitations and uncertainties surrounding the Seveso studies, there is ample epidemiologic evidence of a positive association between dioxin exposure and cancer that involve cancers at many different sites, including malignant lymphomas, soft tissue sarcomas, hepatobiliary tumors, hematopoietic tumors, thyroid tumors, and respiratory tract tumors. These include Fingerhut et al., 1991 ("Cancer Mortality in Workers Exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"); Hooiveld et al., 1998 ("Second follow-up of a Dutch cohort occupationally exposed to phenoxy herbicides, chlorophenols, and contaminants,"); Kogevinas et al., 1997 ("Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: an expanded and updated international cohort study"); Becher et al., 1996 ("Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"); Kogevinas et al., 1995 ("Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: two nested case-control studies"); Kogevinas et al., 1993 ("Cancer incidence and mortality in women occupationally exposed to chlorophenoxy herbicides, chlorophenols and dioxins"); Bertazzi et al., 1993 ("Cancer incidence in a population accidentally exposed to 2,3,7,8-tetrachlorodibenzo-para-dioxin"); and Zober et al., 1990 ("Thirty-four year follow-up of BASF employees exposed to 2,3,7,8-TCDD after the 1953 accident").

With respect to the concerns about soft tissue sarcomas (STS), there is a general indication that exposure to phenoxy acids with known dioxin contamination are associated with STS. While the New Zealand studies (e.g., Smith and Pearce, 1986, "Update on soft tissue sarcoma and phenoxy herbicides in New Zealand") did not find an increase in STS, other reports do suggest an increased risk. For example, Kramarova et al., 1998 ("Exposure to Agent Orange and occurrence of soft-tissue sarcomas or non-Hodgkin Lymphomas: An ongoing study in Vietnam"), discusses an association between STS and phenoxy herbicide exposure in Sweden. The NIOSH study also provides evidence (Fingerhut et al., 1991) for significant increases in death from STS in the high-exposure cohort, although EPA acknowledges the concerns raised by the commenter

regarding the findings of this study. Both Saracci et al. (1991) and Kogevinas et al. (1997) report increased STS rates for exposed workers in several countries, although EPA also recognizes the weaknesses that exist in these studies. In the case of Kogevinas et al. (1997), the increase in mortality for STS was not statistically significant and neither the latency periods nor the length of probable exposure to TCDD were associated with increased mortality or incidence of STS. Kramarova et al., 1998, also report an increased risk for STS from an analysis of the combined results of an analysis that combines the results from U.S. pesticide workers (Fingerhut et al., 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"), German workers (Becher et al., 1996,"Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"), and Dutch workers (Hooiveld et al., 1996, "Preliminary results of the second follow-up of a Dutch cohort of workers occupationally exposed to phenoxy herbicides, chlorophenols and contaminants"). EPA also notes that other health effects have been observed in the Ranch Hand veterans, including an increased incidence of diabetes, elevated cardiovascular disease, and a modest decrease in testosterone levels in the most highly exposed veterans.

Despite the limitations of the various studies, EPA believes the weight of the evidence supports the conclusion that 2,3,7,8-TCDD and other dioxin-like compounds are probable human carcinogens. The SAB, in its review of the draft dioxin reassessment, agreed, concluding that dioxin is likely to increase human cancer incidence under some conditions of exposure. The SAB categorized dioxin and dioxin-like compounds as a B1 carcinogen under the 1986 EPA cancer guidelines (U.S. EPA, 1986) of "agents for which there is limited evidence of carcinogenicity from epidemiologic studies." In addition, the International Agency for Research on Cancer (IARC) recently reviewed the experimental data and epidemiological studies and also concluded that 2,3,7,8-TCDD is carcinogenic to humans (WHO,1997; McGregor et al. 1998).

***Comment 510*:** The commenter cites the proposed rule regarding the assessment of dioxin/furan-related noncancer health effects and suggests that if occupational studies have not provided a sufficiently high dose of TCDD to produce a spectrum of effects, it seems questionable whether environmental exposures would be capable of providing a sufficiently high dose. The commenter notes that the SAB, in reviewing the draft dioxin reassessment conclusion that "...a picture emerges of TCDD and related compounds as potent toxicants in animals with the potential to produce a spectrum of effects. Some of these effects may be occurring in humans at very low levels, and some may be resulting in adverse impacts on human health," observed: It is difficult to determine what EPA is inferring in the last sentence in the above cited conclusion. If it is intended to state that adverse effects in humans may be occurring near current exposure levels, it is the Committee's judgment that EPA has not presented findings that support this conclusion adequately.

The commenter feels that given the contentious nature of EPA's conclusion in the draft dioxin reassessment concerning potential effects at or near background body burden levels, and the likelihood that this conclusion may be modified in order to meet the recommendations of the SAB, it is inappropriate for EPA to use this conclusion as a basis for calculating benefits of reducing dioxin/furan emissions in the proposed rule.

***Response 510**: EPA has not relied on the conclusions of 1994 dioxin reassessment in the final rule, including specifically the risk findings of the draft health assessment document (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin [TCDD] and Related Compounds*, U.S. EPA, 1994d,e) because this document remains a draft document. However, EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available, and EPA continues to rely on it for the final rule.*

EPA agrees with the commenter that there is insufficient evidence to conclude that adverse noncancer effects are occurring in the general population at current background levels of exposure. As indicated in the draft dioxin reassessment, the available epidemiologic data are limited in this regard due to a number of possible factors, including the absence of a large body of human exposure data for the general population; the limited ability of epidemiologic studies to detect effects in relatively small populations when exposures are low and the outcomes are rare; and the difficulty of adjusting for all potentially confounding factors. EPA believes an evaluation of hazard and risk must rely on a weight-of-evidence approach in which all available data (animal and human) are examined together, a process that often requires extrapolation of effects across various animal species as well as to humans.

The nature of responses across mammalian species to dioxins/furans supports the view that low-level responses found in animal studies could be expected for humans as well. There is a strong indication that the mechanism of action for TCDD in animals and humans is similar. The Ah receptor response is common to animals and humans (McGregor et al., 1998, "An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis") and is involved in many of the toxic responses, including both carcinogenic and noncarcinogenic responses. Although evidence of adverse effects in humans at current background levels is limited, adaptive changes may be occurring at these levels. In commenting on this point in its review of the draft dioxin reassessment, the SAB said that the overall impact of certain biochemical changes seen at lower levels is not fully understood and that current knowledge of the mechanisms of TCDD toxicity has not identified the biological determinants of specificity that would allow one to extrapolate toxicities across species with confidence. The SAB agreed with EPA that dioxins produce a spectrum of effects in laboratory animals depending on the dose, context of exposure, and genetic background

but concluded that adverse effects attributable to chronic low-level exposure in humans have not yet been adequately demonstrated.

A number of health effects associated with dioxin exposure have been observed in the Ranch Hand veterans, including an increased incidence of diabetes mellitus and adverse effects on glucose metabolism and insulin production. Elevated cardiovascular disease and a modest decrease in testosterone levels was observed in the most highly exposed veterans, although no statistically significant association was seen between paternal serum dioxin levels and reproductive outcomes. An increased incidence in mortality associated with diseases of the digestive system has also been reported which appeared to be related to chronic liver cirrhosis. This study is continuing.

There is also evidence from human data suggesting TCDD has effects on human reproduction, thyroid hormones, and neurotoxicity. Seveso area results suggest that there were sex ratio changes in children born to parents living near the plant (Mocarelli et al., 1996, "Change in sex ratio with exposure to dioxin"). Vietnam veterans (Operation Ranch Hand), Seveso females, and workers exposed to TCDD from industrial accidents show a consistent pattern of thyroid function changes, e.g., diabetes (Henriksen et al., 1997, "Serum dioxin and diabetes mellitus in veterans of Operation Ranch Hand;" Jennings et al., 1988, "Immunological abnormalities 17 years after accidental exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin;" Pesatori et al., 1998 "Dioxin exposure and nonmalignant health effects: a mortality study;" Zober et al., 1994, "Morbidity follow-up study of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin after a 1953 chemical reactor incident"). The overall evidence from case reports and epidemiological studies shows that exposure to CDDs is associated with central and peripheral nervous system changes shortly after exposure, although long-lasting abnormalities have not been found (Assennato et al., 1989, "Follow up of subjects who developed chloracne following TCDD exposure at Seveso").

For the final rule, EPA used a modified margin of exposure approach to assess noncancer risks from dioxins. In this approach, the estimated average daily dose for HWCs is compared to background exposures in the general population. As a measure of risk, this incremental margin of exposure (MOE) presupposes that if exposures are small relative to background, then risks from these exposures are likely to have limited significance for human health.

***Comment 511*:** The commenter believes that EPA inappropriately relies on TEQ methodology in assessing dioxin/furan risks. The commenter cites the proposed rule regarding relying on EPA's cancer risk estimate for dioxin/furans on a TEQ basis. The commenter feels that this is particularly inappropriate given the SAB comment on this reliance: "This pronouncement is too strong. Virtually all of the Committee believes that it is more accurate to state that binding of TCDD and related

compounds to the Ah receptor is a marker of exposure, but has not yet been established to be 'necessary' for the induction of several of the observed effects." While the TEQ methodology may be a useful tool for regulatory purposes, its use in assessing potential health effects is problematic. Currently there is such controversy and uncertainty in the underlying assumptions, the specific endpoints to which TEFs can be applied, individual TEF values, and the role of the Ah receptor in certain postulated health outcomes that methodology should not be used until some of these legitimate issues are resolved. The commenter notes that the SAB was quite explicit on the need for more work on the part of EPA before the TEF/TEQ methodology could be used to assess risks: "Since the TEQ approach has been used throughout the assessment document, and many of its conclusions (e.g., the proposition that levels 10-100 times over background pose a possible human health hazard) hinge on the validity of the TEF values and assumptions used, the Committee advises EPA to include a peer-reviewed appendix that will comprehensively review EPA's use of the TEF/TEQ approach in the exposure and health assessment documents." In addition, the commenter points out that the SAB noted: "New data, which became available since the release of the document have resulted in adjustments to several of the TEF values. The Committee suggests that a comprehensive review of all TEF values be summarized within the appendix for each congener that has previously been assigned a value…. Finally, since TEF values can vary dramatically based upon the species and response examined, EPA should justify the TEF value that has been selected for evaluating human risk."

The commenter believes that endorsement of either the current outmoded TEF values for many dioxin/furan congeners of concern or EPA's current version of the TEQ/TEF methodology prior to implementation of the SAB's recommendations is unwarranted and notes that the scientific community generally supports a cautious approach with respect to relying on the TEQ methodology. The commenter emphasizes that even prior to the release of the draft dioxin reassessment, there was considerable skepticism concerning the issue of using the TEQ methodology for risk assessment purposes. Many of these concerns center around acute vs. chronic dosing to derive TEFs and species differences. The commenter provides an example and notes that in reviewing this issue, Schlatter (1994) stated: "Because all toxicological data [for derivation of TEFs] were obtained in vitro or from studies with rats, they cannot *a priori* be used to evaluate the situation in humans. marked differences in kinetics and organ distribution according to dosage and species must be taken into account." The commenter feels that regarding reliance upon the TEQ methodology for risk characterization, the following conclusion of Schlatter (1994) is noteworthy: "Consideration of such differences leads to substantially different values for TEFs. In most cases, the resulting human TEF is lower than the original rat TEF. Thus the human burden in terms of TCDD-equivalence has been overestimated. Unfortunately, almost all studies and evaluations have been based on values of rat TEF[s]." The commenter states that additional specific examples of statements by knowledgeable experts cautioning restraint with respect to relying too heavily on TEFs can be found in the extensive comments submitted by CCC to EPA concerning the draft dioxin reassessment. The commenter states that EPA's own research establishes that the present TEF/TEQ system overestimates long-term risks by a factor of 4 to 10 depending on the emission matrix and that the proposed rule does not address the issue that most

current TEFs (as used in the draft dioxin reassessment) are almost certainly overestimates of the relative toxicity of the 2,3,7,8-substituted congeners.

The commenter cites the SAB's awareness of "new data" following the release of the draft dioxin reassessment that would have impacted some of the TEF values; the commenter points out that some of the "new data" noted by the SAB were actually available during the time that the draft dioxin reassessment was in preparation (e.g., Birnbaum et al., 1993, "Dose Response Relationships for EROD Induction in Liver, Lung and Skin for Dioxin and Dibenzofurans," *DIOXIN93*). In that paper, the authors conclude that current TEFs may be overly conservative. The commenter notes that to illustrate how "new data" can change TEF values as a result of more appropriate testing. Birnbaum and DeVito (1996) recently published their Dioxin '93 data on revised TEF values for several dioxins, furans, and PCBs. These were based on 90-day feeding studies instead of the acute studies, which are the basis for all of the TEFs currently in use. This kind of testing more closely approximates the way people are exposed to these compounds and is therefore more appropriate for generating TEFs. The commenter summarizes the results of the study as follows: For one dioxin congener, the TEF value was unchanged, and for three furans the TEFs decreased by a factor of 5-10. The commenter believes that these data demonstrate why, as noted by the SAB, it is premature to use the TEF/TEQ methodology to estimate either body burdens or health effects of dioxins/furans. The commenter emphasizes that the results of the study by Birnbaum and DeVito (1996) have significant implications for estimates of exposure and resulting body burdens of all dioxins/furans. Also, the commenter notes that typical PCDD/PCDF body burdens are also reduced by approximately a factor of 5 and that the result of reductions in PCDD/PCDF body burdens may be significant.

The commenter points out that for many or most of the adverse effects hypothesized by EPA as occurring at or near background body burden levels, the margin of exposure (MOE), i.e., the "safety factor" between an effect level in animals and typical amounts in humans, will increase. This is because studies in animals are based almost exclusively on TCDD, and human body burdens are derived predominantly from other congeners (now with reduced TEFs). As the TEQ body burden declines, the MOE must increase. The above noted research results also suggest that if TEFs were derived from longer term feeding studies (i.e., 180 days) the TEF values would be even lower, thereby further reducing body burdens and increasing the MOE. [Footnote: The TCDD and PCDD congeners with unchanged TEFs account for about 15 percent of body burden. The remainder of the PCDD/PCDF body burden is reduced by about a factor of 10. Consequently, the revised PCDD/PCDF body burden is about 20 percent to 25 percent of the present body burden.] The commenter also states that it should be noted that the current TEQ methodology is always described as an "interim" procedure, and that it is clear that additional research is required before the TEQ methodology can be relied upon for risk assessment purposes.

> ***Response 511****: EPA acknowledges that there are uncertainties associated with the use of the TEQ approach. In its review of the 1994 draft health assessment document (*Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related

Compounds*, U.S. EPA, 1994d,e), the SAB called for clarifications in the specification of TEFs for the various dioxin-like compounds for various health outcomes. However, the SAB also concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds. The WHO review, which was recently published in* Environmental Health Perspectives *(Van den Berg et al., 1998), resulted in recommendations for changes to the values of several of the TEFs. The WHO TEFs reflect a consensus of the international community and, for this reason, were used by EPA in the risk assessment for the final rule.*

The TEF concept derives from mechanistic research indicating that binding to the aryl hydrocarbon (Ah) receptor is the initial event for many, if not all, of the toxic effects associated with dioxins. EPA acknowledges that the TEF approach is based on certain assumptions and limitations. Although the mechanism of action of dioxins is not completely understood, it has been extensively studied. To date, the evidence supports the premise that toxicity is related to Ah induction for a number of effects. These include lethality in guinea pigs (Eadon et al., 1986); body weight loss and thymic atrophy (Safe, 1987); epidermal responses (Knutson and Poland, 1982); suppression of splenic antibody response to SRBC (Kerkvilet et al., 1985); antiestrogenicity (Krishnan and Safe, 1993); and teratogenicity (Weber et al, 1985). Furthermore, the genetic data using inbred mouse strains differing only in the Ah locus strongly support the role of the Ah receptor as an initiating event for dioxin toxicity (ATSDR, 1998). While it is true, as pointed out by the SAB, that binding to the Ah receptor has not been established a necessary for the induction of all toxic effects, it is important to point out that the TEFs only apply to Ah receptor-mediated responses.

EPA recognizes that reliance on animal data for the specification of TEFs has some uncertainty but disagrees with the commenter's implication that this represents a major failing of the TEQ approach. EPA believes that the use of animal data is well supported given the biochemical and biological similarities between laboratory animals and humans, scientific understanding of some of the fundamental impacts of this class of compounds on biological systems, and comparable responses from animal and human studies both in vitro and in vivo.

The issue of differences between human and rodent TEFs was considered by the World Health Organization (Van den Berg et al., 1998). Data on ligand binding specificity of the Ah receptor in humans and rodents are available. The Ah receptor is detected in a wide variety of human tissues and can be activated both in vivo and in vitro into a DNA binding state by a variety of halogenated aromatic hydrocarbons (Van den Berg et al.,

1998). As in mice, a similar situation with several different allelic variants of the receptor might be present in humans. Although the mean 2,3,7,8-TCDD binding affinity for the human Ah receptor may be lower than that observed in responsive mouse strains, there exists a range of binding affinity values similar in magnitude to the range observed between responsive and nonresponsive mouse strains (Van den Berg et al., 1998).

The TEFs used in the final rule are those recommended by the World Health Organization that were recently published in Environmental Health Perspectives *(Van den Berg et al., 1998). These values, which were made available to EPA prior to their publication, reflect a consensus from the international community. The TEFs are not based solely on acute studies as implied by the commenter; rather, they were developed from all available mammalian, bird, and fish studies previously reviewed as well as new studies published over the last several years. These studies were carefully evaluated, and a database was developed using the following criteria:*

- *At least one CDD, CDF, or PCB congener and a reference compound must be included in the study.*
- *Either TCDD or PCB 126 must be included as a reference compound in the same experiment or studied with the same experimental design by the same authors in another experiment.*
- *The relevant end point should be affected by the congener studied as well as the reference compound.*

The TEFs applicable to humans and mammals considered in vivo toxicity data, in vitro data, and quantitative structure-activity relationship (QSAR) data. In vivo data were given the highest priority, and, in fact, the TEFs applicable to humans largely were derived from in vivo data.

The revised international TEFs reflect only limited changes to the 1989 interim TEFs. Most TEFs were not changed. Exceptions include OCDD and OCDF (reduced from 0.001 to 0.0001) and 1,2,3,7,8-PeCDD (raised from 0.5 to 1). EPA believes the revised international TEFs are the most appropriate values for use in the HWC final rule. EPA notes that the impact of these revisions on the margin of exposure is relatively minor, so there is relatively little effect on the estimated risks from HWCs.

The WHO panel of experts also reviewed the uncertainties related to the TEQ approach. These included nonadditive interactions, differences in shapes of the dose-response curves, and differences in species' responsiveness. This panel concluded that the TEF model is both plausible and the most feasible approach for risk assessment of dioxin-like compounds. The panel also concluded that, in view of the available scientific evidence from studies of mixtures of dioxin-like compounds, use of the TEFs are not expected to

result in large errors in estimating concentrations of TEQs or responses at environmentally relevant levels.

***Comment 512**:* The commenter states that in the proposed rule, EPA relies on an assessment of the adequacy of the margin of exposure for adverse human health effects that the SAB has concluded are "speculative and needs to be reexamined." The commenter notes in particular that the proposed rule draws the following from the draft dioxin reassessment: "Although it is not possible given existing information to state exactly how or at what levels exposed humans will respond, the margin of exposure (MOE) between background TEQ levels and levels where effects are detectable in humans is smaller than previously thought." The commenter feels that it is unfortunate that the proposed rule incorporates this particular conclusion from the draft dioxin reassessment given that although very few specific sentences in the draft dioxin reassessment were singled out by SAB, this particular conclusion was specifically highlighted by the SAB as "...thought to be speculative and needs to be reexamined." The commenter further cites the SAB review comments: "In effect, it states that we don't know what will occur, or at what level this unknown [response] will occur, but we know that it will occur (in terms of TEQ) closer to background levels than previously estimated." The commenter feels that, given the SAB's concern on this issue, it is clearly unwarranted to include EPA's speculative conclusion in the proposed rule until EPA has reexamined and justified this conclusion. Further, the commenter believes that EPA relies on questionable assumptions when concluding that the margin of exposure between background levels and levels where effects are detectable in humans is small, a conclusion that hinges to a large extent on the metric used to estimate the MOE. The commenter notes that the draft dioxin reassessment (and now presumably the proposed rule) relies on body burden as the dose metric on which to base these estimates of MOE although it is by no means clear that this is the most appropriate dose metric for this purpose. The commenter states that serum concentrations (i.e., area under the curve) may be more appropriate than body burden and that rather than arbitrarily concluding that body burden is the only dose metric worthy of consideration, EPA should initiate a dialog with scientists, including the SAB, to determine which approach has the broadest support in the scientific community. The commenter included four attachments as follows:

Attachment A: The Benefits of Probabilistic Exposure Assessment: Three Case Studies Involving Contaminated Air, Water, and Soil
Attachment B: Uncertainty and Variation in Indirect Exposure Assessments: An Analysis of Exposure to Tetrachlorodibenzo-p-Dioxin from a Beef Consumption Pathway
Attachment C: Memo from Henry Habicht on Guidance on Risk Characterization for Risk Managers and Risk Assessors
Attachment D: Risk Assessment data provided by commenter.

***Response 512**: EPA concedes that sufficient supporting information may not have been provided for the statement originally made in the draft dioxin reassessment, as cited by the commenter. Therefore, EPA has removed the statement from the preamble to the final rule. EPA recognizes the difficulty of interpreting the available health effects*

information and drawing inferences regarding risks to human health. For this and other reasons, EPA has not developed a "reference dose" for 2,3,7,8-TCDD.

Regarding the use of serum concentrations as an alternative dose metric, EPA feels that the dose metric used should be most appropriate to the circumstances. There is good evidence that serum TCDD, coupled with measurement of serum lipid content, provides a valid estimate of the TCDD concentration in adipose tissue under steady-state, low-dose conditions. However, for risk assessment purposes, EPA generally prefers to use the administered dose (i.e., daily intake per unit body weight).

For the final rule, EPA used a margin of exposure (MOE) analysis to assess the potential for noncancer effects. In this analysis, EPA compared the estimated average daily dose for HWCs to the average daily dose in the general population. As a measure of risk, this incremental MOE presupposes that if exposures are small relative to background, then risks from these exposures have limited significance for human health.

***Comment 49, 50*:** The commenter states that EPA has overestimated the relative contribution of HWCs and HWIs to total dioxin/furan emissions. The commenter points out that the peer-review panel concluded that the risk assessment should "consider total exposure from all recognized dioxin sources" since background dioxin/furan levels generally exceed site-specific exposure levels attributed to HWC emissions and their sources constitute a minority of the total sources contributing to background levels.

Response 49, 50: *EPA does not believe it is necessary to perform a comprehensive assessment of exposures to all anthropogenic emission sources within the context of the risk assessment for the hazardous waste combustion rule. EPA points to the ongoing work on the dioxin reassessment to inventory all sources of dioxin emissions in the U.S. (see* The Inventory of Sources of Dioxin in the United States, External Review Draft, *April 1998, U.S. EPA, 1998c).*

In assessing risks from dioxins for the final rule, EPA used a margin of exposure (MOE) analysis for assessing noncancer risks. In this analysis, EPA compared the estimated average daily dose for HWCs to the average daily dose in the general population. The estimates of background exposure that were used for the general population represent total exposure to all sources of dioxins. Therefore, this analysis provides a direct measure of the relative exposure attributable to HWCs vis-a-vis total exposure from all sources.

***Comment 247*:** The commenter states that EPA's case study approach relies on many worst-case assumptions, and the commenter agrees with the peer-review panel's conclusion that "it is misleading to characterize the case study approach as providing accurate risk estimates." As noted in the peer-review panel's review and the commenter's comments on the proposed rule, EPA bases its "average case" calculations on several worst-case assumptions, thereby overestimating potential risks. The commenter believes that using conservative parameter values and methodologies to characterize dioxin/furan emissions' fate and transport, exposure, and toxicity violates the stated intent of using the case study approach to steer away from reliance on these assumptions. The commenter agrees with the peer-review panel's conclusion that the bounding-level exposure estimates should be deleted from the risk assessment because these hypothetical estimates rely on so many worst-case assumptions about exposure that the calculated level is far beyond any exposure reasonably expected to occur. According to the commenter, the peer-review panel recommended that if EPA believes that the case study approach does not represent the risk of the "worst offender" facility in a source category, EPA should evaluate additional facilities instead of relying on a hypothetical bounding estimate. The commenter agrees with the peer-review panel that EPA inappropriately relies on these models without adequately addressing their severe limitations.

Response 247: *For the final rule, EPA did not use a case study approach and instead used stratified random sampling to select a subset of facilities from the HWC universe for risk characterization. Each of these selected facilities was modeled using site-specific data when available. The use of stratified sampling in selecting facilities allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe they are intended to represent (e.g., confidence intervals). Such statements could not be made using the case study approach that was used at proposal.*

EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. That is why, for the final rule, EPA did not use high-end values. In addition, EPA did not make bounding estimates, as referred to by the commenter. In all instances, EPA either used central tendency values in conjunction with site-specific data or conducted a full distributional analysis to account for the full range of exposure.

EPA used central tendency exposure factors (e.g., mean values) for estimating exposures, except for a few risk-driving pathways for which an exposure factor variability analysis was conducted using Monte Carlo simulation. In addition, the risk analysis for the final rule used site-specific emissions estimates to estimate media concentrations and concentrations in agricultural products. Also, U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing as a recreational sport and for persons engaged in farming or fishing for subsistence. EPA believes that these modifications to the risk methodology provide considerable assurance that the exposure estimates developed for the final rule are reasonable.

Comment 1093: The commenter states that the risk assessment does not adequately address uncertainties. The commenter elaborates that the steady-state surface water model and terrestrial food chain models introduce high levels of uncertainty in the risk assessment, particularly because they are derived from "highly limited" data sets. The commenter points out that the Panel noted that "long-term observation of soil and sediment levels in the vicinity of a source would be required to assess the degree to which accumulation of environmentally persistent compounds affects the suitability of the steady-state assumption."

Response 1093: *EPA acknowledges the uncertainty of the data and models used in the risk assessment in support of the final rule. However, EPA believes they represent the best analysis tools currently available. The uncertainty associated with data and models is discussed in the risk assessment background document (RTI, 1999).*

EPA has emphasized the uncertainties in its characterization of risks for the final rule. The risk assessment Background Document (RTI, 1999) includes a detailed discussion of uncertainty associated with all major components of the HWC risk analysis including fate/transport modeling, exposure assessment, and risk characterization. When feasible, the uncertainty discussion includes a qualitative characterization of both the magnitude and direction of the impact from specific sources of uncertainty. However, with the exception of the uncertainty associated with statistical sampling error that was quantified, it was not possible to perform a quantitative uncertainty analysis for the HWC risk assessment.

In the development of emerging methodologies, EPA must often accept the limitations of the data available for model validation. Although additional studies would help further refine the models, EPA believes there are currently sufficient evidence and data available to evaluate exposures from the surface water and terrestrial food chain and that it is important to assess the risks attributable to such exposures.

EPA agrees that the steady-state assumption may in some cases overestimate media concentrations. This is mitigated to a degree by the use of simple empirical relationships derived from field data to model bioaccumulation in the terrestrial and aquatic food chain. For aquatic systems, the steady state assumption is a greater limitation in larger bodies of water that have a long turnover time, such as drainage lakes. In the risk assessment for the final rule, a surface water model (IEM-2M) was used for modeling mercury water concentrations that does not assume steady state, although a simple empirical relationship was used for modeling bioaccumulation (of methylmercury) in fish. This is consistent with the modeling approach used in EPA's Mercury Study Report to Congress *(U.S. EPA, 1997c).*

***Comment 1094*:** The commenter states, furthermore, the Panel noted that the food chain models "cannot reliably estimate bioconcentration and biotransfer factors, particularly in environments significantly different from those in which the available data were collected."

Response 1094: *Extrapolation of data from one set of conditions to another is common practice in risk assessment and many other fields of endeavor and may contribute significantly to the uncertainty of the analysis. However, gathering information to establish site-specific food chain models and selecting intermedia transfer coefficients reflective of site conditions was not possible within the context of the hazardous waste combustion rule.*

Comment 1095: The commenter feels that the risk assessment's exposure scenarios tend to give a false picture of the amount of exposure expected because they do not provide probabilities of a particular scenario being realized. The commenter agrees with the peer-review panel's conclusion that

not providing these probabilities or the number of people likely to be exposed through each scenario is a "gross failing" on EPA's part.

Response 1095: *The risk assessment for the final rule was modified to account for the number of individuals exposed and their location. The analysis employed U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, and 99th percentiles.*

Comment 1096**:** The commenter points out that the Panel concluded that EPA must do more than expand its qualitative discussion of uncertainties. The Panel indicated that an "add on" section describing uncertainties is "of very little value" if those uncertainties are not addressed in the risk assessment. The commenter notes that a Panel member noted several ways the risk assessment could quantitatively address the uncertainties of parameter estimates and exposure models using sensitivity analysis. The commenter agrees that this quantitative analysis is necessary to provide useful information on the degree to which key assumptions influence the risk assessment's conclusions.

Response 1096: *EPA gave much consideration to the comments of the peer-review panel and made a number of changes to the risk assessment in order to improve the risk assessment. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for EPA's response to the issues raised by the peer-review panel.*

EPA modified its risk analysis for the proposed rule in a number of important areas as a result of public comments and comments submitted by the peer-review panel. The risk assessment for the final rule was based on exposure characterization that included components that directly address the issue of providing representative coverage for exposed individuals. The goal of these modifications was to increase representativeness and consequently reduce uncertainty.

EPA acknowledges the uncertainty of the data and models used in the risk assessment in support of the final rule. However, EPA believes they represent the best analysis tools currently available. The uncertainty associated with data and models is discussed in the risk assessment background document (RTI, 1999).

EPA has emphasized the uncertainties in its characterization of risks for the final rule. The risk assessment background document includes a detailed discussion of uncertainty

associated with all major components of the HWC risk analysis including fate/transport modeling, exposure assessment, and risk characterization. When feasible, the uncertainty discussion includes a qualitative characterization of both the magnitude and direction of the impact from specific sources of uncertainty. However, with the exception of the uncertainty associated with statistical sampling error that was quantified, it was not possible to perform a quantitative uncertainty analysis for the HWC risk assessment.

***Comment 248*:** The commenter states that EPA inappropriately relies on TEQs for assessing dioxin/furan risks and that although the Panel's review of EPA's use of TEQs to estimate dioxin/furan risks was mixed, the Panel unanimously agreed that the TEQ approach for dioxin/furan congeners requires additional consideration. The commenter cites Dr. Rao and the SAB report *A Second Look at Dioxin* as consistently supporting the fact that the application of TEQ methodology in the risk assessment is an oversimplification. Further, the commenter cites and strongly agrees with Dr. Wilson's comment regarding EPA's policy statement on the appropriate use of TEQs as a "worst case" or "upper bound" screen only and not a tool used to estimate risks. Dr. Wilson indicated that such use of TEQs renders the estimate of risk invalid.

The commenter agrees with the Panel's conclusion that TEFs are not predictive of responses at low exposures levels. By definition, the TEF is the factor necessary to convert the dose of one dioxin-like chemical to the dose of 2,3,7,8-TCDD that would produce the same response. Since not all dose response curves are strictly parallel, accurate TEF values should vary with the level at which the relative potency of the doses is to be determined. Because TEFs for dioxins/furans are derived from median-response ratios, predicted toxicity at very low exposures may be highly inaccurate. The commenter notes that although TEQ methodology may be a useful tool for regulatory purposes, its use in assessing potential health effects is problematic.

Response 248: *EPA acknowledges that there are uncertainties associated with the use of the TEQ approach. However, despite the uncertainties, the SAB concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds. The WHO review, which was recently published in* Environmental Health Perspectives *(Van den Berg et al., 1998), resulted in recommendations for changes to the values of several of the TEFs. The WHO TEFs reflect a consensus of the international community and, for this reason, were used by EPA in the risk assessment for the final rule.*

The WHO panel of experts also reviewed the uncertainties related to the TEQ approach. These included nonadditive interactions, differences in shapes of the dose-response curves, and differences in species' responsiveness. This panel concluded that the TEF model is both plausible and the most feasible approach for risk assessment of dioxin-like

compounds. The panel also concluded that, in view of the available scientific evidence from studies of mixtures of dioxin-like compounds, use of the TEFs are not expected to result in large errors in estimating concentrations of TEQs or responses at environmentally relevant levels.

Section 10
Citizens Aware and United for a Safe Environment
RCSP-0169

Comment 550: The commenter states that EPA fails to characterize, discuss, or assess the health effects associated with the proposed massive increases in acid gas emissions (HCl) and their endorsement of usage of high sulfur coal resulting in SO_2 and reduced sulfur.

Response 550: *EPA assessed the potential health risks associated with emissions of hydrogen chloride (HCl) from HWCs in the risk assessment for the final rule. EPA's analysis indicates that emissions of hydrogen chloride are not likely to pose a significant risk to human health. Inhalation exposures are well below EPA's reference concentration (RfC) for HCl. As a result, hazard quotients for HCl are generally quite low. However, for certain HWCs with relatively high HCl emissions, a potential may exist for ecological effects in close proximity to the facility as a result of wet deposition of hydrochloric acid. EPA is confident that the MACT standards for total chlorine will significantly decrease HCl emissions from such sources. EPA believes this will minimize any ecological threat posed by hydrogen chloride emissions.*

EPA does not expect that sulfur gases from HWCs will pose a significant threat to human health and the environment due to the relatively low sulfur emissions from HWCs in comparison with other sources. Sulfur dioxide is not a hazardous air pollutant (HAP) under the Clean Air Act and is not subject to MACT controls. Therefore, EPA is not setting an emission standard for sulfur emissions in the MACT rule.

***Comment 1087*:** The commenter notes that the synergistic interactions of acid gases which irritate and damage lung tissue and other mucus membranes is not addressed by EPA.

Response 1087: *Although interactions in response to exposures to chemical mixtures can be important, there is little reason to expect synergism in responses to mixtures of acid gases. Rather than synergism, EPA expects an additive response due to similarities in the mode of action. Chlorine gas can be converted to hydrogen chloride in the atmosphere, thereby further limiting the potential for nonadditive interactions.*

For the risk analysis for the final rule, EPA characterized the potential risk from chemical mixtures using a hazard index as the risk descriptor. An inhalation hazard index was calculated by summing the individual inhalation hazard quotients for each chemical constituent for which inhalation exposures were assessed, including hydrogen chloride and chlorine. EPA's analysis indicates that there is little potential for risk from exposures to HCl or chlorine, either individually or as a mixture.

Comment 1014: The commenter states that organics in the raw materials of cement production can react with chlorine in the hazardous waste to form toxic PICs. The commenter feels that these PICs and dioxins are of great concern with regard to public health. The commenter states that the risk involved from exposure to harmful products of incomplete combustion is the same regardless of whether it comes from the hazardous waste itself or whether from indirect chemical reactions of the raw material.

Response 1014: *Although EPA assessed the risks from chlorinated dioxins and furans for the final rule, risks from other organics that may be present as products of incomplete combustion (PICs) could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited, highly variable and, therefore, inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.*

Comment 399*: (This summary is not a part of original comment RCSP-0169.)* The commenter believes that EPA and FDA should reach consensus on the use of the Faroe Islands and Seychelles Islands studies to establish consistent exposure guidelines. The commenter feels that too often, EPA and FDA reach inconsistent conclusions based on analysis and interpretation of the same data. While recognizing that regulatory mandates differ, the commenter believes that it would be desirable if RfDs and action levels were consistent with respect to acceptable exposure levels. To this end, the commenter feels that prior to the release of EPA's revised Mercury Report, EPA and FDA scientists should meet to ensure that data from the Faroe Islands and Seychelles Islands studies are interpreted and used in a consistent manner.

Response 399*: For the final rule, EPA is using a reference dose(RfD) for methylmercury of 1 x 10^{-4} mg/kg-d developed for EPA's 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c).EPA defines the RfD as an estimate of a daily exposure to the human*

population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD for methylmercury is based on the data from a study of an incident in Iraq in which methylmercury treated seed grain was used for making bread (Marsh et al., 1987). EPA's Science Advisory Board, in its review of the Mercury Study Report to Congress*, concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including studies on the Cree Indians and in New Zealand) as well as experimental animal data, and that the above RfD should be retained.*

At the time of the finalization of the MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and marine mammal–consuming populations in the Seychelles and Faroe Islands. Because the majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to re-evaluate the RfD for methylmercury.

*EPA and other federal agencies, including the FDA, participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury*, November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing the EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Section 11
Coalition for Responsible Waste Incineration
RCSP-0013

***Comment 476**: (The original comment for RCSP-0013 was directed to EPA's* Draft Combustion Emissions Technical Resource Document; *therefore, there is no reference to the summary text cited below in this Docket I.D. RCSP-0013).* The commenter feels that EPA relied upon flawed indirect risk assessment protocols. The commenter states further that one of the other reasons EPA may have overestimated risks in developing this proposed rule is EPA's use of a flawed indirect risk assessment methodology. The commenter notes that the risk assessment protocols EPA used also have been criticized by the SAB, which pointed out that the protocols included poor information on upsets, lacked measured data, ignored key additional data points, and failed to assess unique differences among pollution sources. The commenter states that the SAB also noted that EPA needs to develop and implement a plan to collect data for models, to validate the methodology, and to reduce the reliance on generic default values. In short, the SAB believed that EPA should not rely on highly conservative generic assumptions when more accurate data are available. The commenter cites the SAB conclusion that the new EPA guidance is not ready for release as an "EPA methodology" for routine use due to the substantial scientific uncertainties in the models and the absence of information on them in the EPA guidance.

> ***Response 476**: It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from hazardous waste combustion facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the* Mercury Study Report to Congress *(December 1997) U.S. EPA, 1997c), as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."*

EPA acknowledges the uncertainty implicit in the use of models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board. EPA intends to respond to comments on draft Addendum that have been received from the public, as well as from the SAB, in an appropriate forum.

Coalition for Responsible Waste Incineration
RCSP-0222

***Comment 244, 47*:** The commenter believes that HWCs are not a major contributor to the mass loading of dioxins to the environment relative to other significant sources. Furthermore, the commenter expresses concern about the nature of question 10 for the reviewers. This question states, "Dioxin exposure from background sources normally exceed those estimated as due to exposure to incinerator emissions." If this statement is correct, it supports the commenter's August 19 comments that the HWC industry is not a major source of dioxin/furan emissions.

***Response 244, 47**: It is possible for a given industrial source category to have significant risk impacts to local receptor populations even when emissions from that source category represent a relatively minor contribution to overall anthropogenic emissions nationwide. This is especially true for those constituents that have the potential to bioconcentrate up the food chain and display elevated toxicity, such as dioxins and furans. The magnitude of indirect exposures through the food chain for these constituents depends on a number of facility-specific and site-specific factors including (a) plume dispersion characteristics for the emissions source; (b) location of human receptors, including farms, relative to the emissions source; (c) physical properties related to fate/transport modeling (e.g., waterbody TSS, and USLE parameters related to erosion modeling); and (d) behavioral characteristics linked to exposure for the human receptor populations under evaluation, including exposure duration and dietary ingestion rates. Because the magnitude of risk impacts to specific receptor populations depends on facility-specific and site-specific factors and because it is possible for relatively low mass emissions to pose significant risks to local populations, it is necessary to conduct a quantitative risk analysis to determine whether significant risks exist for a specific study area.*

EPA is continuing to inventory all sources of dioxin emissions in the U.S. as part of the ongoing work on the dioxin reassessment (see The Inventory of Sources of Dioxin in the United States, External Review Draft, *April 1998, U.S. EPA, 1998c).*

***Comment 1049*:** The commenter feels that the current methodology of indirect risk assessment overstates the risk from dioxin-like compounds.

***Response 1049:** The risk assessment for the final rule is based on methodology presented in the 1990 indirect exposure document,* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(IEM) (U.S. EPA, 1990) and the 1993 Addendum (U.S. EPA, 1993a) to the 1990 indirect exposure document. These methods were further updated with respect to dioxins, as the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist*

of the procedures published in the Estimating Exposure to Dioxin-Like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. Human exposure factors were obtained from the 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a), including consumption rates for home-produced foods and recreationally caught fish. Risks from both cancer and noncancer effects were assessed.*

The risk assessment for the final rule was modified in a number of important areas, including the use of stratified random sampling to select facilities for analysis, use of facility- and site-specific data, use of central tendency exposure parameters, and use of population-based risk characterization. The risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and U.S. Census data to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming for an occupation or fishing for recreation and persons engaged in farming or fishing for subsistence. These modifications are designed to increase the representativeness of risk results generated for the analysis including those specific to dioxin/furan exposure. The commenter is referred to the risk assessment background document for a full discussion of the data and methods used in the risk assessment for the final rule (RTI, 1999).

EPA believes the modifications that have been made to the risk assessment framework for the final rule provide considerable assurance that the exposure estimates are reasonable and, when taken together, are not likely to either substantially overestimate or underestimate the range of possible exposures.

Comment 245: The commenter has concerns similar to those of the peer reviewers on the risk assessment section. The individual concerns with certain expansions are listed below.

The commenter agrees with the peer reviewers that the preamble does not make a sufficient case that mercury is a threat to human health. The reviewers go on to state that EPA has provided no rationale to support the actual mercury limit proposed.

The commenter supports EPA in controlling mercury emissions from HWCs. However, as the commenter suggested in previously submitted comments, the commenter believes that EPA should incorporate the new data expected to be available in 1997 on the Seychelles Islands and Faeroe Island studies and reexamine the total database to determine if currently proposed mercury standards are justified.

Response 245: *EPA gave much consideration to the comments of the peer-review panel and, in response to these comments, made a number of changes to the risk assessment to improve its overall representativeness. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for

the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for EPA's responses to the issues raised by the peer reviewers.*

The health effects of mercury are well documented. Clinically observed neurotoxicity has been observed following exposure to mercury. Generally the most subtle indicators of mercury toxicity are neurological changes that include losses of motor skills and sensory ability. Humans, plants, and animals are routinely exposed to mercury, potentially resulting in a variety of ecological and human health impacts. EPA's 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c) concluded that exposure to methylmercury through consumption of fish is most important to human health.*

For the final rule, EPA conducted a quantitative risk assessment for mercury. The assessment followed the general modeling approach developed for the 1997 Mercury Study Report to Congress *and used the IEM-2M surface water model and methylmercury reference dose developed especially for that report. The results of EPA's mercury analysis suggest that mercury emissions from HWCs in and of themselves are not likely to lead to exposures that exceed EPA's reference dose. However, exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing. EPA recognizes that its mercury analysis is subject to considerable uncertainty, including the fact that background exposures were not taken into account. Other sources of uncertainty include the speciation of mercury emissions, fate and transport of mercury in the atmosphere and in soils, methylation in surface water and uptake of mercury in fish, and the absence of site-specific information on the fishing activity of anglers.*

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

*EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faeroe Islands (*Report of the Workshop on Scientific Issues

Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

***Comment 46*:** The commenter agrees with the reviewers that the lack of field data necessary to validate the dioxin/furan models creates major uncertainties in the model predictions. The commenter also realizes that the development of such data is difficult and costly.

***Response 46:** In the development of emerging methodologies, EPA must often accept the limitations of the data available for model validation. Although additional studies would help further refine the models, EPA believes there are currently sufficient evidence and data available to evaluate exposures from the surface water and terrestrial food chain and that it is important to assess the risks attributable to such exposures. EPA acknowledges the uncertainty of the data and models used in the risk assessment in support of the final rule. However, EPA believes they represent the best analysis tools currently available. The uncertainty associated with data and models is discussed in the risk assessment background document (RTI, 1999).*

Comment 1017: The commenter believes that EPA should not be developing regulations based on uncertain predictions from unvalidated models.

***Response 1017:** EPA must often accept limitations and uncertainties in the information available for making decisions. EPA acknowledges the uncertainty of the data and models used in the risk assessment in support of the final rule. However, EPA believes they represent the best analysis tools currently available. EPA has included a comprehensive discussion of the uncertainties in its characterization of risks in the risk assessment Background Document (RTI, 1999) for the final rule.*

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA, and was done to determine if the technology-based standards are protective enough to satisfy RCRA, or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Section 12
Continental Cement Company
RCSP-0147

Comment 0147-1: The commenter indicates that the proposed rule incorporates the use of inchoate documents that the EPA has specifically stated are not ready to be the basis of regulatory standards. The commenter points out three such examples. First, EPA uses the results of its draft dioxin reassessment to characterize the risks associated from dioxin/furan emissions from cement kilns and other HWCs. Given the recent review of the reassessment by the EPA Science Advisory Board (SAB), the commenter believes that reliance upon the reassessment in the current context is legal error. In support of this argument, the commenter cites 42 U.S.C. Section 7607(d)(8), which indicates that "there is a substantial likelihood that the rule would have been significantly changed if such errors had not been made." The commenter views the dioxin/furan risk levels as being of central importance to the case made for going "beyond-the-floor."

Response 0147-1*: EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards.*

EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment are the best information currently available.

Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job."

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

***Comment 232*:** The commenter points out that the draft Mercury Report to Congress (due to have been released on April 15, 1996) was unreleased at the time of the April 19 proposal. The commenter sees this as significant because the Mercury Report provides the only health basis for the unduly harsh mercury standards that also go beyond the floor of the MACT facilities. Therefore, the commenter believes that without this mercury risk data, EPA would not have been able to set such inflexible and improbable mercury standards.

The commenter states that the Mercury Report is extraordinarily flawed, as it essentially bases its conclusion on data gathered in rural Iraq and predicated upon recollections a decade past the event. The commenter points out that more recent and controlled data (gathered in the Seychelles Islands) appears to strongly dispute the Iraqi conclusions. The commenter cites an April 1996 letter from Martha Keating, U.S. EPA, to other federal agencies, as discussed in *Air/Water Pollution Report's Environment Week*, April 19, 1996, indicating that EPA itself has recognized these flaws and purportedly promised not to utilize the Mercury Report until such flaws were addressed. Further, the commenter notes that it is a legal error to utilize the Mercury Report to Congress to establish mercury risk levels.

Response 232: *EPA is concerned about exposure to mercury from HWC emissions because mercury is a known neurological toxicant in humans. However, at the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA,1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA,1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from hazardous waste combustors. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

At the time of the finalization of the MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and marine mammal–consuming populations in the Seychelles and Faeroe Islands. Because the

majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to reevaluate the RfD for methylmercury. For the final rule, EPA continues to use the same RfD for methylmercury that was used at proposal (i.e., 1 x 10^{-4} mg/kg-d). This is the same RfD that was developed for EPA's MRTC and was subject to extensive review, including review by EPA's Science Advisory Board (SAB). The SAB endorsed retention of EPA's RfD, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data. The SAB felt that the uncertainty factor used by EPA could even be increased, thus lowering the RfD, due to concerns about the acute nature of the exposure in the Iraqi study and at least some evidence that methylmercury has a longer half-life in the brain than in the blood.

*EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faeroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data to undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury, but that significant uncertainties remain because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

***Comment 233*:** The commenter states that the indirect, multi-pathway risk methodology is an incomplete and inaccurate document used to substantiate the risk case (used to make the case for biomagnification in the food chain) made within the April 19 proposal. The commenter points out that in recent comments, EPA's SAB was highly critical of the multipathway risk model that EPA employs, noting that it lacked sufficient scientific validity for regulatory use. The commenter feels that it is legal error to use the multipathway risk assessment unless and until EPA fully corrects the technical errors in the methodology indicated by EPA's own SAB.

Response 233: *It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess risks associated with indirect exposures to emissions from hazardous waste combustion facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress *(U.S. EPA, 1997c), as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."*

EPA acknowledges the uncertainty implicit in the use of models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board. EPA intends to respond to comments on draft Addendum that have been received from the public, as well as from the SAB, in an appropriate forum.

***Comment 260*:** The commenter generally supports the conclusions ("Summary of Key Points") of the Risk Analysis Peer Review Panel.

Response 260: *EPA is committed to the use of the peer review process for major scientific studies and technical analyses used in decision-making. EPA has incorporated comments from peer reviewers in the risk analysis for the final rule and has prepared a response to comments document on the peer review.*

***Comment 261*:** The commenter emphasizes an observation concerning exposure scenarios that has been general criticism of EPA for many years. The selected exposure scenarios tend to give a false picture of the amount of exposure expected because they include no information by which one can judge the likelihood that any particular scenario will be realized.

Response 261: *The risk assessment for the final rule was modified to account for the number of individuals exposed and their location. The analysis used U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions which explicitly account for the numbers of persons exposed at differing levels of exposure. From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, and 99th percentiles.*

***Comment 1091*:** The commenter cites a peer-review comment that indicates that EPA's theoretical assumptions may yield estimates greater than actual values in most cases. The commenter feels that this statement sums up the weaknesses of the approaches for airborne emissions and that this is clearly another exaggeration activity to bolster EPA's tenuous position.

Response 1091*: Regarding the peer review completed for the proposed rule, EPA gave much consideration to the comments of the panel. In response to these comments, EPA made a number of changes to the risk assessment in order to improve the overall representativeness of the assessment. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer review comments. The commenter is referred to this document for EPA's response to the issues raised by the panel.*

***Comment 1069*:** The commenter emphasizes a peer-review panel statement that the "...relative contributions of background and hazardous waste combustor facilities (HWCs) sources on cumulative exposure was considered an intractable problem." The commenter notes, more specifically, that the reviewer stated that "[b]ackground exposures to dioxins generally exceed site-specific exposure levels attributed in the report as due to dioxin emissions from HWCs. Moreover, there is no evidence to support the claim that long-range transport of emissions from HWCs are solely responsible for elevated background levels."

The commenter notes that a reviewer stated that "[t]he finding that background dioxin exposure levels generally exceed the site-specific exposure levels the Agency has modeled for hazardous waste incinerators and hazardous waste burning cement kilns is not surprising since these sources comprise a minority of the total sources contributing to background." The commenter feels that this statement is in direct refutation to EPA's statements in the preamble to the HWC, FR 17366, which ascribes approximately 46 percent of the total PCDD/PCDF TEQ emitted each year to cement kilns.

***Response 1069**: EPA does not believe it is necessary to perform a comprehensive assessment of exposures to all anthropogenic emission sources within the context of the risk assessment for the hazardous waste combustion rule. EPA points to the ongoing work on the dioxin reassessment to inventory all sources of dioxin emissions in the U.S. (see* The Inventory of Sources of Dioxin in the United States, External Review Draft, *April 1998, U.S. EPA, 1998c).*

In assessing risks from dioxins for the final rule, EPA used a margin of exposure analysis for assessing noncancer risks. In this analysis, EPA compared the estimated average daily dose for HWCs to the average daily dose in the general population. The estimates of background exposure that were used for the general population represent total exposure to all sources of dioxins. Therefore, this analysis provides a direct measure of the relative exposure attributable to HWCs vis-a-vis total exposure from all sources.

The wording in the preamble has been modified to clarify the statement regarding the relative contribution of individual source categories.

***Comment 1067*:** The commenter points out a comment/observation that has been made over and over, and states that one of EPA's selected reviewers finally has implicated EPA risk assessments as gross overestimates. The commenter cites the following peer-review comment: "The guidelines in the HWC Emission Database (Volume II) to adopt detection limits for various congeners, when in most cases none will be emitted, may have an adverse impact on the final risk outcome for dioxin, particularly for the indirect exposure pathways."

The commenter cites a peer reviewer's recommendation regarding the treatment of nondetect dioxin congeners. The reviewer recommended that "...if there are no hits for any of the congeners, the

congeners should be dropped from the analysis. Where there is some hit, the analysis should be carried through at half the detection limit or at a level defined by a statistical approach that relies on the number of total hits out of the number of samples taken. A similar method should be adopted for all other chemicals that are included in the analysis." The commenter feels that this suggested method of adjusting the way in which PCDD/PCDF emissions are reported is decidedly different than current practice, and notes that currently, PCDDs/PCDFs at levels below the detection limit are discounted in computing the toxic equivalent quantity. The commenter notes that if the peer reviewer's method of calculating PCDD/PCDF emissions is adopted, the proposed HWC limit for PCDD/PCDF emissions must be reexamined because it is the current TEQ method that was used to establish this limit. Further, the commenter feels that applying the peer reviewer's proposed method to "other chemicals" which, unlike PCDDs/PCDFs, do not have an interrelationship, is an instance of "guilt by association" where individual chemicals are to be deemed present at certain levels (say, ½ of the detection limit) simply because other chemicals are detected at the lower detection limit.

> ***Response 1067:*** *For the final rule, EPA assumed that dioxin and furan congeners that were below the limit of detection were present at one-half the detection limit. This represents a middle course that avoids the conservatism that could be introduced by assuming congeners are present at their full detection limit yet accounts for the likelihood that the congeners are, in fact, present but at levels below the detection limit of the measurements. Although the issue of compounds being present at levels below the limit of detection contributes to uncertainty, the uncertainty is sufficiently small so as not to have a material effect on the findings and conclusions of the risk assessment. In particular, individual risks associated with high-end exposures are attributable to emissions of congeners that are present at levels well above the limit of detection rather than congeners that are below the limit.*

Comment 1072: The commenter cites a peer reviewer who states that "EPA's argument does not provide a logical rationale to support the actual mercury limits being proposed…. This statement culminates in the total trashing by EPA's arguments in the preamble to the proposed HWC regulations on the basis for the proposed mercury emission limit. The commenter feels that, as stated by the peer reviewer, "EPA has totally failed to reconcile this proposed reduction [due to the limits in the HWC] with potential benefits to the aquatic environment." The commenter notes that in response to EPA's general statement regarding potential health benefits from reduced mercury levels in fish over time and thus reduced health effects occurring in fish consuming populations, a peer reviewer stated "...the Agency does not provide sufficient supporting evidence to reasonably conclude the adverse health effects related to fish ingestion are currently occurring." The commenter notes that the peer reviewer supported this statement by referencing recent findings by the National Institute of Environmental Health Sciences. The commenter feels that the peer reviewer clearly has cause to make the concluding statement noted above.

> ***Response 1072****: The health effects of mercury are well documented. Clinically observed*

neurotoxicity has been observed following exposure to mercury. Generally, the most subtle indicators of mercury toxicity are neurological changes that include losses of motor skills and sensory ability. Humans, plants, and animals are routinely exposed to mercury, potentially resulting in a variety of ecological and human health impacts. EPA's 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) concluded that exposure to methylmercury through consumption of fish is most important to human health.*

For the final rule, EPA evaluated human health and ecological risks associated with exposure to elemental mercury, divalent mercury, and methylmercury. With respect to methylmercury, the human health evaluation completed for the final rule shows that exposures are projected to be below EPA's reference dose. However, the analysis is subject to considerable uncertainty, including the fact that background exposures are not taken into account. Exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing.

EPA conducted a screening-level ecological assessment in the risk assessment for the final rule to evaluate the impacts of ecological exposure to mercury resulting from emissions from HWCs. In this approach, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water and compared to model-predicted media concentrations to assess the potential for ecological risk. With respect to methylmercury, the ecological analysis shows that surface water concentrations are projected to be below EPA's wildlife criteria for the protection of aquatic wildlife, suggesting a low potential for ecological risk. However, as with the human health evaluation, the analysis is subject to considerable uncertainty, including the fact that background levels were not considered. Therefore, the potential for ecological risk may be higher than the quantitative analysis would suggest.

Comment 1099: The commenter strongly agrees with and emphasizes the peer reviewer's comment that "...most of EPA commonly forgets that 'Reference Dose (RFD),' 'Risk specific Dose (RsD),' and related administrative values represent exposure that can be considered safe: such exposure, for a lifetime, poses no or a negligible risk to those so exposed. The product of an RFD and an estimate of exposure does not produce a 'risk' or predicted incidence of injury. EPA has defined the risk at the RFD as zero, so this product is identically zero. One can conclude, based on the product of an exposure times an RfD or RsD, that the exposure poses no risk and thus conclude that no further action is required. One cannot conclude that further reduction in exposure reduces risk."

Response 1099: *EPA has prepared a separate response to comments document entitled*

Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer review comments. The commenter is referred to this document for EPA's response to the issues raised by the peer reviewers. The risk-specific dose is the dose or intake level corresponding to a specific level of risk (usually a HQ of 1 or an excess lifetime cancer risk of 1 in 1 million).*

EPA considers that the ingestion reference dose (RfD) represents an average daily ingestion exposure rate that is considered to pose no risk to the population. RfD values are published in EPA's Integrated Risk Information System (IRIS, U.S. EPA, 1998a) database or in EPA's Health Effects Assessment Summary Tables (HEAST, U.S. EPA, 1997b). For the final rule, EPA relied on RfDs to quantitatively express risk as hazard quotients (HQs) for the ingestion of noncarcinogenic constituents. For this analysis the ratio of a single substance exposure level over a specified time period to a reference dose for that substance derived from a similar exposure period is the HQ. In the HWC risk assessment modeled, intake divided by the RfD is represented by the hazard quotient. If hazard quotients exceed 1 (e.g., the exposure level [intake] exceeds the RfD), there may be concern for potential noncancer effects. As a rule, EPA considers that the greater the value of HQ above unity, the greater the level of concern.

Comment 1073: The commenter emphasizes the peer reviewer's comment regarding the toxicology of hydrogen chloride that "…[t]he toxicology of hydrogen chloride is well known: for instance, the mean lethal concentration in air is almost the same as hydrogen cyanide, and the toxic effects are precisely those to be expected from a strong acid condensing on tissue. Thus, I find it exceedingly surprising that EPA reports 'low confidence' in the very protective RfC that is quoted (page 120). It is stupidities like this that give EPA science its poor reputation."

Response 1073: *EPA acknowledges that acute effects on humans exposed to hydrogen chloride include coughing, choking, inflammation and ulceration of the respiratory tract, chest pain, and pulmonary edema. EPA established a reference concentration (RfC) for hydrogen chloride of 2.0×10^{2} mg/m^{3} based on a chronic rat inhalation study that reported an increased incidence of hyperplasia of nasal mucosa as well as laryngeal-tracheal segments in the group exposed to hydrochloric acid (Sellakumar et al., 1985). EPA has low confidence in the chronic inhalation study on which the RfC was based because the study used only one dose and had limited toxicological measurements. Confidence in the database is also low because the supporting data consisted of only subchronic bioassays and the database does not provide any additional chronic or reproductive studies. Therefore, EPA has low confidence in the RfC.*

Section 13
Department of Energy
RCSP-0124

***Comment 1048*:** The commenter notes that the BID uses the methodology described in the *Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions* (EPA/600/AP-93/003, November 1993). The commenter points out that this document (which is a review draft) contains the statement that the methodology should not be construed to represent EPA policy, nor should it be cited or quoted and, consequently, this situation presents a problem for RCRA permit applicants as there is no EPA-approved methodology for the performance of indirect exposure assessment. To address the situation, the commenter urges EPA to provide guidance for the performance of indirect risk assessments if a site-specific risk assessment will continue to be required for RCRA permitting.

Response 1048: *The risk analysis for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level. Therefore, the risk methodology may differ from what is typically used in a permitting context. Specifically, EPA did not follow the 1994 draft guidance for performing screening level analyses at hazardous waste combustion facilities. The risk analysis provided in the final rule is not intended to provide guidance for the kind of site-specific analyses required for permitting. EPA considers that the risk assessment methodology provided in this document does not comprise a set of guidelines or recommended approaches that should be applied in all circumstances. Rather, it provides a set of procedures that the risk assessor can draw upon where applicable for site-specific exposure scenarios.*

It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from hazardous waste combustion facilities. As explained in the risk assessment for the proposed rule, EPA used the indirect exposure methodology as outlined in the 1990 document Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emission *(Interim Final, January 1990) (U.S. EPA, 1990). This document was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure with information gathered in conjunction with the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b), the 1997* Mercury Report to Congress *(MRTC) (U.S. EPA, 1997c), and other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best*

information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."

***Comment 0124-1*:** The commenter states that EPA's presentation of risk from existing incinerators is misleading as it assumes the presence of all receptor pathways. Only the appropriate receptor pathways should be used. This is especially relevant to mercury, which is a target of the proposed rulemaking. EPA states that the most significant receptor pathway is through the aquatic food chain; if this pathway does not exist due to a facility's location, then mercury risk would be much less.

***Response 0124-1**: To meet the current MACT requirements under the CAA and satisfy RCRA's requirement, this multimedia, multipathway risk analysis was conducted to support the MACT standard rulemaking for HWCs by evaluating all pathways and constituents of concern for which adequate data were available. To ensure consideration of all receptor populations potentially exposed to emissions from HWC facilities, the analysis completed for the final rule evaluated the following receptor populations: subsistence farmer, subsistence fisher, commercial beef farmer, commercial dairy farmer, commercial pork farmer, commercial produce farmer, resident, home gardener, and recreational fisher. All individuals were assumed to be exposed through inhalation and incidental soil ingestion. If a modeled waterbody was designated as a drinking water source, then all receptors were assumed to be exposed through drinking water consumption, as well. Additionally, receptors were assumed to be exposed through population-specific pathways (i.e., the beef farmer through beef ingestion).*

Assessing human exposures requires that certain assumptions be made about the receptors and pathways of exposure. The analysis used U.S. Census and Census of Agriculture data to locate and enumerate persons living in farm and nonfarm households. This improved characterization of both population and individual risk estimates. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. However, it was not possible from census data to identify and locate individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios were retained in the risk analysis for the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. To assess the potential risks, EPA assumed that subsistence farming could take place in any

of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA also assumed that subsistence fishing takes place at each body of water that was modeled in the risk analysis. EPA recognizes that these assumptions may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

***Comment 518*:** The commenter notes that health risk assessments conducted by the U.S. Army Center for Health Promotion and Preventive Medicine for the U.S. Army Chemical Stockpile Disposal Program have shown that the risks posed to individuals by mercury emissions from the chemical demilitarization incinerators are insignificant. The commenter offers that the worst-case mercury exposure scenario (mercury uptake resulting from subsistence fishing) indicates individual exposures that are approximately 150 times lower than the allowable exposure. The commenter notes that this conclusion was determined using an emission rate of 10 µg/dscm and offers to provide EPA these data if requested. The commenter recommends that the final rule should recognize that not every HWC emits mercury in concentrations that pose a risk to individual health.

Response 518: *Sections 112 (a) and (d) of the CAA direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). For the final rule, EPA is setting the emission standard for mercury at the MACT floor. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA, and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.*

EPA acknowledges that risk reduction associated with implementation of the regulatory options may vary by source category, exposure parameters, exposure scenario, and constituent. However, the risk analysis for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level.

In addition, the level of risk associated with a specific combustion facility, such as the chemical demilitarization incinerators mentioned in the comment, is dependent on a variety of factors linked to that facility's operation (e.g., stack parameters, emissions rates) and site conditions surrounding the facility (e.g., meteorological conditions, surrounding terrain, demographic/agricultural patterns). Therefore, care must be taken in comparing risk results generated for a specific facility to those generated for a specific

combustor category since key factors linked to risk can vary between the individual facility and facilities making up the combustor category.

***Comment 0117-1*:** The commenter states that EPA has indicated that the draft dioxin reassessment has received "a large number of public comments" and that the risk analysis supporting this proposed rule is undergoing an external peer review. The commenter cites the proposed rule as indicating that comments received on these two items will be considered in the development of the final rule. EPA will us these items to assist in determining if the proposed MACT standards are consistent with the RCRA mandate that EPA establish standards for hazardous waste management facilities that are protective of human health and the environment. In light of the importance of the dioxin reassessment and the risk analysis used for the proposed rule, if EPA anticipates revisions to this proposed rule due to comments received on either one of these two items, the commenter believes an additional comment period should be provided. This additional comment period would allow both the regulated community and the public the opportunity to comment on the revisions prior to development of the final rule.

The commenter recommends that if EPA plans to revise the proposed rule based on comments received on either the dioxin risk reassessment or the external peer review of the risk analysis, another comment period should be provided prior to promulgation of the final rule.

Response 0117-1: *EPA believes there has been ample opportunity for commenters to comment on the HWC risk assessment. As part of the rulemaking package, the risk assessment was subject to full notice and comment procedures consistent with the Administrative Procedure Act. A notice of proposed rulemaking was published in the* Federal Register *on April 19, 1996. The public comment period extended from April 19, 1996, to August 19, 1996, a period of more than 120 days. A public docket was established for the notice of proposed rulemaking, and all comments received on the proposed rule were placed in the public docket (U.S. EPA Docket Number F-96-RCSP-FFFFF). In addition, the risk assessment for the proposed rule was externally peer reviewed. A notice of data availability was published in the* Federal Register *on August 23, 1996, requesting comment on the report prepared by the peer reviewers. A 30-day public comment period was established. All comments received during the public comment periods were considered by EPA, and revisions were made to the risk assessment for the final rule to reflect the comments received. EPA believes that the 120-day comment period for the notice of proposed rulemaking, followed by the additional 30-day comment period on the peer review, provided ample opportunity for the public to comment on the risk assessment for the proposed rule. Indeed, EPA received numerous comments from this and other commenters.*

Comments received during the public comment periods were considered by EPA in the risk assessment for the final rule. EPA also has prepared a response to comments

document for the final rule that explains how EPA considered each of the comments received on the risk assessment at proposal. A response to comments document was also prepared on the report of the external peer review panel on the risk assessment for the proposed rule.

The risk assessment for the final rule continues to rely on technical information developed for EPA's draft dioxin reassessment, which has yet to be concluded. Although EPA has updated some of the technical information from the 1994 draft reassessment document, no changes have been made in the HWC risk assessment as a result of any new conclusions having been reached in the dioxin reassessment effort.

The risk assessment background document for the final rule highlights the changes made to the risk assessment since proposal (RTI, 1999).

Section 15
The Dow Chemical Company
RCSP-0245

***Comment 56*:** The commenter agrees with CRWI, CMA, and the peer reviewers that EPA's risk methodology will overstate risks from dioxins and needs to be significantly refined before it is of practical value in assessing risks from HWCs.

> ***Response 56****: EPA gave much consideration to the comments of the peer-review panel on the risk assessment for the proposed rule. In response to these comments, EPA made a number of changes to the risk assessment in order to improve the overall representativeness of the risk assessment, including addition of combustor categories, use of facility-specific and site-specific data, use of central tendency parameters, and use of population-based risk characterization. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments.*
>
> *EPA used the indirect exposure methodology, as outlined in the 1990 document* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emission *(U.S. EPA, 1990), updated in 1993 with the draft Addendum (U.S. EPA, 1993a). For the risk assessment for the final rule, EPA updated the indirect exposure document with information gathered in conjunction with the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b), as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment is the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." EPA acknowledges the uncertainty implicit in the use of the models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.*

***Comment 57*:** The commenter strongly encourages EPA to incorporate the comments of the risk assessment peer reviewers into the methodology before using these to calculate risks from specific operating facilities. The commenter feels that, left unchanged, use of the current methodology will raise public concern needlessly.

> ***Response 57****: As noted in the response to the previous comment, EPA incorporated comments from peer reviewers in the risk analysis for the final rule by implementing modifications to the risk analysis framework designed to improve overall analysis representativeness. The commenter is referred to the separate response to comments document (U.S. EPA, 1999b) that addresses the peer-review comments explicitly.*

Section 16
DuPont Engineering
RCSP-0180

***Comment 405*:** The commenter performed a direct comparison of realistic dioxin risk ranges for at the floor levels and beyond the floor levels to show that at the floor controls are protective and no risk justification exists for going beyond the floor. The commenter explains that EPA tabulates ranges of potential HWI individual cancer risks associated with dioxin exposures for at the floor and beyond the floor scenarios. In the Risk Assessment Background Document, EPA calculates risks for four cases: Central Tendency/Low End, Central Tendency/High End, High End/Low End, and High End/High End. However, the commenter's review of the Background Document indicates that the risk range reported for at the floor includes a high end/high end risk estimate and that the risk range reported for beyond the floor only considers a low end/high end estimate of risk. The commenter supports EPA's decision to reject the High End/High End case for the beyond the floor risk range as a more appropriate and a more realistic way to convey the potential risks associated with beyond the floor controls. However, the commenter does not consider it appropriate to compare risk ranges as the justification for additional controls in this proposal when the two risk ranges have different bases. A direct comparison of the HWI dioxin risk ranges (without the High End/High End risk case) would be 3×10^{-9} to 2×10^{-6} for at the floor and 3×10^{-9} to 2×10^{-6} for the beyond the floor. Therefore, the commenter notes that this more realistic risk range for at the floor indicates that at the floor controls for dioxin would satisfy RCRA's protectiveness standard. The commenter states that because the direct comparison does not indicate a reduction in dioxin risk attributable to beyond the floor controls and because the corrected estimate of dioxin risk at the floor is protective, EPA should set the HWI MACT emission standard for dioxin at the MACT floor.

> ***Response 405:*** *Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made*

relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

For the final rule, rather than defining high-end exposure scenarios deterministically, EPA characterized high-end risks by developing population-weighted cumulative distributions of risks to individuals explicitly. The commenter is referred to the risk assessment Background Document (RTI, 1999) for a complete description of the analytical framework and the methods and data used in the risk analysis for the final rule.

***Comment 406*:** The commenter states that EPA should document calculations and assumptions in the Risk Assessment Background Document and should re-open comment on the revised Background Document. The commenter suggests that EPA should properly document all calculations for all scenarios in the Background Document. The commenter believes that the *Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document* and supporting appendices do not provide sufficient detail to allow verification of the calculations and therefore conclusions.

The commenter also notes that EPA presents environmental media concentrations of dioxin and furan congeners for only two scenarios; from the commenter's perspective, this is not sufficient and, at a minimum, chemical concentrations in all media (soil, water, air, animal tissue, etc.) should be provided for each facility modeled. The commenter feels that this would enable review of the risk assessments in the document and is consistent with the level of detail that EPA would require a owner/operator to submit for a site-specific risk assessment.

In addition, the commenter notes that although the Background Document calculates the potential risk at 11 facilities, the document only summarizes the risks from the best-case and worst-case facilities. Because all of the calculated cancer risk estimates and hazard indices are not presented, the commenter feels that it is not possible to fully evaluate and comment on the potential spectrum of risks associated with different facilities.

Response 406: *The risk assessment for the proposed rule was fully documented in the risk assessment background document (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). As part of the rulemaking package, the risk assessment was subject to full notice and comment procedures consistent with the Administrative Procedure Act. A notice of proposed rulemaking was published in the* Federal Register *on April 19, 1996. The public comment period extended from April 19, 1996, to August 19, 1996, a period of more than 120 days. A public docket was established for the notice of proposed rulemaking and all comments received on the proposed rule were placed in the public docket (U.S. EPA Docket Number*

F-96-RCSP-FFFFF). In addition, the risk assessment for the proposed rule was externally peer reviewed. A notice of data availability was published in the Federal Register *on August 23, 1996, requesting comment on the report prepared by the peer reviewers. A 30-day public comment period was established. EPA believes that the 120-day comment period for the notice of proposed rulemaking, followed by the additional 30-day comment period on the peer review, provided ample opportunity for the public to comment on the risk assessment for the proposed rule. Indeed, EPA received numerous comments from this and other commenters.*

Comments received during the public comment periods were considered by EPA in the risk assessment for the final rule. EPA has prepared this to comments document for the final rule that explains how EPA considered each of the comments received on the risk assessment at proposal. A response to comments document was also prepared on the report of the external peer review panel on the risk assessment for the proposed rule.

Although EPA has not made the spreadsheets and other software that were used in HWC risk analysis part of the administrative record, EPA placed example spreadsheet calculations in the docket for the proposed rule. This information was included as an attachment to a July 17, 1996, memo from D. Layland to the file documenting a meeting with cement industry consultants. EPA also made all air dispersion model output files available to the public through the RCRA docket (see "Availability of Electronic Files of Dispersion Modeling Results," memorandum from D. Layland to the RCRA Docket dated May 16, 1997). In addition, much of the software that EPA used in the analysis is publicly available. This includes the Industrial Source Complex Short-Term (ISCST) model, which was used for modeling air dispersion and deposition in both the proposed and final rules, and the Integrated Exposure Uptake Biokinetic Model (IEUBK) model, which was used for modeling lead exposures in the final rule. EPA has carefully documented the calculations, variables, and data used in the risk assessment for the final rule and has placed these in the rulemaking docket. In addition, the IEM-2M model, which was used for modeling the fate and transport of mercury in surface water in the risk assessment for the final rule, is fully documented in the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c).*

EPA has provided all results (by facility type, exposure scenario, and age group) of the risk assessment in a multivolume document that accompanies the technical support document. This additional documentation prepared for the risk assessment for the final rule—including example calculations, expanded information on media concentrations, and facility-specific risk results for certain receptors (i.e., subsistence exposure scenarios)—provides the information the commenter is seeking.

Comment 1103: The commenter suggests that EPA should provide data to support

overconservative exposure assumptions used to support the proposed dioxin emission standard. The commenter points out that EPA supports the need for the beyond-the-floor HWI dioxin emission standard through the worst-case subsistence risk scenarios and for these scenarios (as is the case for most of the scenarios) the exposure assumptions are extremely conservative for estimating the total doses from produce and animal tissue ingestion. As an example, the commenter points out that subsistence farmers are assumed to ingest 100 percent of the animal product (i.e., beef, dairy, pork) produced at the farm, along with 100 percent of their produce for the entire year. The commenter suggests that EPA provide the data that support these assumptions.

***Response 1103**: The final rule uses an exposure assessment methodology that includes many components designed to evaluate central tendency exposure conditions at modeled facilities and avoid the generation of overly conservative risk estimates. EPA modified the exposure methodology used at proposal to improve the representativeness of the analysis. For the final rule, EPA used central tendency exposure factors in conjunction with site-specific data for estimating exposures, except for a few risk-driving pathways for which an exposure factor variability analysis was performed using Monte Carlo simulation. In addition, the risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and concentrations in agricultural products, and U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing as a recreational sport and persons engaged in farming or fishing for subsistence. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative.*

Subsistence populations, including those engaged in both farming and fishing, can experience elevated exposure to dioxins/furans through consumption of locally raised milk and beef, and locally caught fish. Consequently, the subsistence scenarios present at proposal were retained in the risk analysis for the final rule. Subsistence exposure estimates were generated using dietary intake rates and exposure durations reflective of subsistence activity (i.e., subsistence farmers were assumed to receive nearly all of their dietary intake from home-produced foods, while subsistence fishers were assumed to obtain a significant portion of their dietary intake from self-caught fish). Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Therefore, it was not possible from census data to identify and locate individual farms that may be engaged in subsistence farming. Because it was not feasible to characterize the specific location of subsistence receptors around modeled HWC facilities, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA also assumed that subsistence fishing takes place at each body of water modeled in the risk analysis. EPA recognizes that these

assumptions may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

In modeling chemical exposure for subsistence receptor populations, the same food intake rates were used for subsistence farmers as for farmers engaged in farming for commerce, although the subsistence farmers were assumed to ingest all modeled agricultural commodities as home-produced items. These rates were derived from data collected on consumption of home-produced foods from the U.S. Department of Agriculture's Nationwide Food Consumption Survey (as cited in the 1997 Exposure Factors Handbook*, U.S. EPA, 1997a). For subsistence fishing, EPA used fish intake rates representative of Native American tribes from the Columbia River basin, who obtain a significant portion of their dietary intake from fish. Subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. The use of exposure factors derived from such studies ensures the plausibility of the exposure scenarios EPA used to characterize risks to individuals who may be engaged in subsistence activities.*

***Comment 1104*:** The commenter notes that there are errors in the Background Document should be corrected. Specifically, Table IV.2 of the Risk Assessment Background Document presents an incorrect TEQ emission concentration for 1,2,3,7,8,9-HxCDF. The correct value should be 0.063 ng/dscm rather than 6.03 ng/dscm.

Response 1104: *For the final rule, EPA corrected all errors known to have occurred in the risk analysis for the proposed rule. Emissions data, presented in EPA's document* Final Technical Support Document for HWC MACT Standards, Volume V: Emission Estimates and Engineering Costs *(U.S. EPA, 1999a), are now presented as annualized values.*

***Comment 407*:** The commenter feels that EPA should improve its knowledge of HWI mercury emissions and their potential impacts prior to proposing beyond the floor mercury standards not supported by science. The commenter cites the proposed rule, in which EPA acknowledges that meaningful quantitative risk assessment of emission sources precise enough to support national-level regulatory decisions is not possible. Instead, the commenter notes that EPA is proposing beyond the floor emission standards for mercury as the result of suspicion of significant individual risks near hazardous waste combustion facilities. The commenter supports the regulation of emissions at levels that limit risks based on technically defensible science, not on suspicion. Therefore, the commenter believes that EPA should document a cause and effect relationship between specific point sources and increased mercury concentrations in fish prior to promulgating emission limits based on suspicion.

The commenter cites the emissions rates of hazardous waste incinerators as given in the proposed rule, noting that the rate of 4.2 metric tons per year of mercury represents less than 2 percent

of total anthropogenic emissions of mercury in the U.S. The commenter cites EPA's (January 1995) *Mercury Study Report to Congress* as indicating that there is not sufficient data to estimate mercury emissions from hazardous waste incinerators. Further, the commenter notes that the draft report to Congress does not include HWIs as a significant source of mercury emissions in the U.S.

The commenter states that in light of the protection offered by existing inhalation risk-based mercury emission limits on HWIs and stringent RCRA Land Disposal Restrictions, which largely preclude feeding mercury to HWIs, EPA should improve its knowledge on mercury emissions and their potential impacts prior to proposing a beyond the floor national emission standard on mercury emissions from HWIs.

***Response 407**: EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.*

EPA is concerned about exposure to mercury from HWC emissions because mercury is a known neurological toxicant in humans. However, at the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its Mercury Study Report to Congress *(MRTC) (U.S. EPA,1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA,1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

For the final rule, the concentrations of mercury species in HWC emissions were determined from emissions tests at HWC facilities. Specifically, trial burn and certificate of compliance emissions measurements from the standard stack gas sampling train, EPA Method 29, were used for estimating emissions of mercury species. The EPA Method 29 sampling train was originally developed for quantifying total metals in the stack gas. However, due to the manner in which the mercury is captured (and subsequently analyzed) in the sampling train, it has been proposed that Method 29 measurements can

be used to estimate three general forms for mercury: condensed solid phase mercury (primarily HgO); soluble vapor phase ionic mercury forms (such as $HgCl_2$); and elemental mercury (Hg0). The condensed mercury and water-soluble mercury vapor represent the divalent forms of mercury in the particulate and vapor phases, respectively. This approach was taken in the 1997 MRTC and was also used for the HWC risk assessment.

The risk assessment for the final rule also followed the same general modeling approach developed for the 1997 MRTC and used the IEM-2M surface water model and the methylmercury reference dose developed especially for that report. The 1997 MRTC was critically reviewed by EPA's Science Advisory Board. Although the SAB had some concerns with the surface water modeling in the draft report (which EPA believes were addressed in the final report), the subcommittee felt that EPA's reference dose for methylmercury was well supported by the available data, while recognizing that important human studies were still ongoing.

***Comment 408*:** The commenter states that EPA concludes that baseline emissions of semivolatile metals and low volatility metals do not pose a significant risk through indirect exposure routes. The commenter cites the proposed rule as indicating that for all metals save mercury, significant accumulation is not expected in the food chain and the risks from other indirect exposure routes (i.e., deposition on soil and subsequent incidental ingestion) are not projected to be significant. The commenter feels that this statement confirms that the current use of setting RCRA permit emission limits on HWIs based on inhalation risks and air dispersion on a site-specific basis via guidance is sufficiently protective.

Response 408: *EPA is revising standards for hazardous waste combustion facilities under the CAA to limit emissions of HAPs that could adversely affect the general public. The revised standards are technology-based standards being promulgated under the authority of section 112 of the CAA.*

The risk analysis for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level. Therefore, the risk methodology may differ from what is typically used in a permitting context. In particular, the risk analysis for the final rule is not intended to serve as guidance for performing site-specific risk assessments that may be required for permitting. EPA has issued other guidance for that purpose.

Section 17
Eastman Chemical Company
RCSP-0181

***Comment 553*:** The commenter feels that EPA has not demonstrated that risks with mercury emissions warrant BTF standards. The commenter cites 61 FR 17478, in which EPA discusses its concern with exposures to methylmercury, primarily the ingestion of fish. In relating this concern to HWCs, EPA uses the terms "strongly suggesting," "indicates," and "it may be inferred" but fails to provide any data specific to HWCs to demonstrate that mercury emissions from these facilities actually pose a threat. The commenter feels that it is unclear whether the small reduction in mercury emissions achieved by BTF standards for hazardous waste incinerators is even measurable, let alone justification for the high cost of implementing that standard.

***Response 553**: For the final rule, EPA conducted a quantitative risk assessment for mercury. The assessment followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model and methylmercury reference dose developed especially for that report. The results of EPA's mercury analysis suggest that mercury emissions from HWCs in and of themselves are not likely to lead to exposures that exceed EPA's reference dose. However, exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing. EPA recognizes that its mercury analysis is subject to considerable uncertainty, including the fact that background exposures were not taken into account. Other sources of uncertainty include the speciation of mercury emissions, fate and transport of mercury in the atmosphere and in soils, methylation in surface water and uptake of mercury in fish, and the absence of site-specific information on the fishing activity of anglers.*

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). For the final rule, EPA is setting the emission standard for mercury at the MACT floor.

EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT

standards implement those provisions alone.

***Comment 554*:** The commenter states that, in its discussion of the health effects of mercury, EPA references a study of 81 maternal/infant pairs exposed to methylmercury in an accident in Iraq (61 FR 17478). However, the commenter notes that EPA recently has announced that it is postponing the release of its mercury emissions report because one recent study, on children in the Seychelles Islands, directly contradicts the results of the Iraq study. The Seychelles Islands study involved 740 mother/infant pairs and found none of the neurological effects on children that were reported in the Iraq study. EPA is reevaluating these studies and plans to submit them, along with EPA's draft mercury report, to the EPA Science Advisory Board for review.

***Response 554**: EPA released its* Mercury Study Report to Congress *(MRTC) in 1997 (U.S. EPA, 1997c) following a review of the report by the SAB. As explained in the mercury study, EPA's RfD for methylmercury is based on the Iraqi study. EPA defines the RfD as an estimate of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime. The SAB endorsed retention of the RfD, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data.*

*Subsequently, EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faeroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data to undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

***Comment 556*:** The commenter feels that a BTF level for dioxin/furan is not justified on a risk basis despite the fact that EPA uses an extensive RCRA risk analysis of emissions from incinerators to conclude that BTF dioxin/furan controls should be required for this source category. The commenter believes that the EPA risk evaluation fails to support a BTF requirement for incinerators, and, furthermore, the risk evaluation demonstrates that MACT floor levels are sufficient to satisfy the risk mandate of RCRA. The commenter states that, consistent with other dioxin/furan studies, EPA's analysis shows that direct inhalation is a minor exposure pathway. The maximum predicted individual risk for the maximum exposed individual (MEI) from incinerator dioxin/furan emissions is 8×10^{-7}, which is well below the 1×10^{-5} RCRA risk criterion. The commenter notes that the baseline, floor, and BTF emission scenarios are well below the criterion and that there certainly is no justification for BTF controls based upon the MEI calculations. The commenter states that most of EPA's risk Background Document (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes,* February 20, 1996) focuses on indirect risk pathways. The commenter summarizes this risk analysis as follows. Risks were quantified using case studies from four hazardous waste incinerators. Scenarios representing both general population (e.g., typical adult and child resident and typical farmer), and special subpopulations (e.g., subsistence farmers, subsistence fishers, and home gardeners) were modeled. Scenarios included both adults and children. EPA estimated both central tendency (transport and exposure variables set at 50th percentile levels) and high-end (selected exposure variables set at 90th percentile) risks. Of all the exposure pathways and populations evaluated at the floor level, only the extreme high-end risk scenarios exceed the 1×10^{-5} criterion. Central tendency risk estimates for HWIs at the floor level ranged from 3×10^{-9} to 2×10^{-6}, well below the 1×10^{-5} criterion risk level. Highest estimated risk to the typical resident was 8×10^{-7}. Even for the high-end (90th percentile) estimates, the estimated risks ranged from 1×10^{-7} to 5×10^{-5}, the highest risk estimate being within an order of magnitude of the acceptable risk level. Only for three exposure scenarios (subsistence dairy farmer (adult and child), subsistence beef farmer, and subsistence poultry farmer), did the high-end estimated risk exceed the 1×10^{-5} level. These levels were:

- Subsistence Beef Farmer—4×10^{-5}
- Subsistence Poultry Farmer—3×10^{-5}
- Subsistence Dairy Farmer—4×10^{-5}
- Subsistence Dairy Farmers (child)—5×10^{-5}

Therefore, the risk data upon which EPA concludes that BTF dioxin/furan standards are justified are based upon the simultaneous occurrence of subsistence farming scenarios and high-end dioxin/furan emission rates.

The commenter notes that, as shown above, only subpopulations that potentially rely on beef, poultry and dairy products for their subsistence were shown, by EPA's high-end risk assessment, to be exposed to a risk slightly greater than 1×10^{-5}. However, the commenter cites EPA's *Risk Assessment*

Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes, February 20, 1996, as indicating that even for the example facilities modeled in EPA's risk study, location of actual farms where subsistence type activities might be occurring were not identified. Therefore, the commenter feels that it is not clear that any of the estimated risk levels that exceeded the 1 x 10^{-5} level (i.e., subsistence farmers) represent any actual incinerator scenarios. The commenter points out that most on-site hazardous waste incinerators are located within an industrial facility where the likely impact on a subsistence population is very improbable. Furthermore, the commenter believes that the likelihood of subsistence population impacts analogous to those modeled for hazardous waste incinerators is very low, if at all existent.

The commenter also notes that according to EPA's risk assessment support document, February 20, 1996, all of the floor level exposure scenarios that had modeled risk levels exceeding 1 x 10^{-5} were assumed to be emitting dioxin/furan at a rate of 4 ng/dscm. However, the commenter points out that the highest dioxin/furan emitting source in EPA's MACT database that indicated that its APCD inlet temperature was below 400EF, was facility 222C3. Facility 222C3 indicated an average emission level of 2.22 ng/dscm.

The commenter notes that if the MACT floor level is redefined as suggested in subsequent comments (rapid quench and no dioxin precursor feeds), the highest emitting facility meeting the MACT floor standard would be facility 221C5, with an average emission rate of 0.78 ng/dscm. The commenter states that risk estimates based upon this facility's emission level would be less than 1 x 10^{-5}, even in EPA's worst-case high-end analysis, and, furthermore, that it is highly unlikely that many, if any facilities meeting EPA proposed MACT floor (especially if waste heat recovery boilers and feeding of dioxin/furan precursors are prohibited) will have dioxin emissions as high as the 4.0 ng/dscm that was modeled.

> ***Response 556:*** *Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made*

relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

In response to comments received from both peer reviewers and the public, EPA implemented a number of modifications to the risk analysis framework for the final rule to improve the representativeness of the assessment. A number of these modifications address issues raised in this comment.

The risk assessment for the final rule was modified to account for the number of individuals exposed and their location, using U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. However, it was not possible from census data to identify and locate individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios present at proposal were retained in the risk analysis for the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, EPA acknowledges that subsistence behavior does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. Separate exposure estimates were made for individuals engaged in farming for an occupation or fishing for recreation and persons engaged in farming or fishing for subsistence.

EPA used central tendency exposure factors for estimating exposures, except for a few risk-driving pathways for which an exposure factor variability analysis was performed, using Monte Carlo simulation. The Monte Carlo simulation allows a more complete characterization of the range of exposures for a population without the use of conservative upper-bound parameters which can serve to compound conservatism. For the final rule, EPA relied on data reported in the 1997 Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a) on total time in the same residence to determine the duration of exposure.*

EPA increased the representativeness of the facilities evaluated. For the final rule, EPA used stratified random sampling to select a subset of facilities for risk characterization. The use of stratified random sampling allows risk results generated for a subset of modeled facilities to be adjusted to reflect the entire universe of facilities in a statistically meaningful manner. EPA agrees that site-specific emissions data are preferable to national-level data and, in the risk assessment for the final rule, EPA used site-specific

information whenever possible. Facility data were used to characterize emissions and the conditions of stack release. Site-specific stack gas concentration data from trial burn and certificate of compliance tests were used in conjunction with stack gas flow rates to estimate emissions. In those instances where emissions measurements were not available, stack gas concentrations were imputed from a pool of emissions measurements for other similar facilities.

EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative.

***Comment 1106*:** The commenter questions a number of parameters, including the exposure duration, contaminated fraction, deposition characteristics, and exposure frequency. In particular, the commenter believes that in today's mobile society the number of subsistence farmers who would have a 40-year exposure duration is likely extremely small. The commenter also believes that assuming the fraction of the product (beef, dairy, poultry, etc.) consumed by subsistence farmers that is contaminated by incinerator emissions is 100 percent (or that every bite of beef, dairy products, poultry, etc. that a farmer eats for 40 years is contaminated to the maximum extent) is highly improbable. The commenter notes that the pollutant deposition rates of the highest facility was used for all high-end modeling. Finally, the commenter notes that the exposure frequency of 350 days per year assumed for all exposure scenarios assumes that an individual is at a different, uncontaminated environment for only 15 days per year. The commenter feels that, taken individually, any of these modeling assumptions may cast doubt on whether any actual incinerator exceeds the 1×10^{-5} risk criterion. Taken collectively (assuming they are all true simultaneously), the prospect of a hazardous waste incinerator (complying with the MACT floor level standards) exceeding the 1×10^{-5} risk criterion is extremely small, certainly limited to a very small subset of facilities. Additionally, the commenter finds it difficult to understand how EPA can conclude based on its risk assessment that BTF dioxin/furan controls are justified, given the extreme uncertainty surrounding the science of indirect risk assessment.

The commenter cites EPA's Risk Assessment Support Document indicating that the risk analysis is based on the *Methodology for Assessing Health Risks with Indirect Exposure to Combustor Emissions* (U.S. EPA, 1990b) and its Addendum (U.S. EPA, 1993a). The commenter notes that both of these documents were reviewed by the Agency's Science Advisory Board (SAB). The commenter cites a July 29, 1994, letter to Administrator Browner, in which Drs. Daisey and Matanoski (SAB Chair) stated that inherent uncertainties in the model prohibited the release of the Addendum as an EPA methodology. The commenter notes that EPA is still evaluating these comments, and that EPA's reliance on methodologies that are still highly debatable and subject to revision brings into question the accuracy and precision of the risk results presented in the MACT proposal. The commenter believes that EPA is ill-advised to infer that a BTF dioxin/furan standard is justified on such imprecise risk estimates, especially considering the extremely high incremental costs of implementing the BTF standard and that, even under the most worst-case assumptions, estimated risk levels for HWIs were within a half-an-order of magnitude of the 1×10^{-5} risk criterion.

***Response 1106**: EPA has taken a number of steps to address comments and concerns raised by the public and the peer reviewers, including those identified by the commenter. EPA believes that these steps provide considerable assurance that the exposure estimates developed for the final rule are reasonable.*

For the final rule, rather than defining high-end exposure scenarios deterministically, EPA characterized high-end risks by developing population-weighted cumulative distributions of risks to individuals explicitly. The commenter is referred to the risk assessment Background Document (RTI, 1999) for a complete description of the analytical framework and the methods and data used in the risk analysis for the final rule. Briefly, EPA selected a total sample of 76 facilities to represent various categories of HWCs. Each facility was assigned a sample weight based on its selection probability. A study area was defined for each facility as the area surrounding the facility out to a distance of 20 kilometers. Each study area was divided into 16 sectors. Media concentrations were estimated for each facility sector using facility-specific (for example, stack emissions) and site-specific (for example, land use) information combined with national-level defaults (for example, livestock feeding practices). Sector-specific exposures were then estimated from the media concentrations and age-specific exposure factors. Mean exposure factor values were used in order to estimate the mean, or arithmetic average, risk to individuals within a sector. These mean risk values were then aggregated across an HWC category, taking into account sector-specific population estimates and facility-specific sampling weights. Population estimates were derived from U.S. Census and Census of Agriculture data.

For the final rule, EPA relied on data reported in the 1997 EFH on total time in the same residence to determine the duration of exposure. The data indicate that farm households have, on average, a longer residence time than nonfarm households. This is not surprising given that farmers are typically a less mobile population group than the general population. For farm households, EPA used a mean residence time of 6.5 to 17.3 years, depending on an individual's age.

For the final rule, EPA assumed that beef farmers eat beef produced on the farm, similar to the approach taken at proposal. However, the intake rate of home-produced beef for the risk assessment for the final rule was based on consumption estimates specific to individuals who report consuming home-produced beef, as presented in the 1997 EFH. Previously, EPA had used beef consumption rates for the general population. EPA believes it is reasonable to assume that beef that is home-produced is produced by the farmer and therefore is subject to dioxin contamination from local sources. As such, the contaminated fraction is 100 percent because all locally produced beef will contain some level of contamination from local sources depending on the location of the farm.

An exposure frequency of 350 days per year is a standard default parameter value (see Fields and Diamond, 1991). EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. In recognition of this fact, EPA improved the representativeness of the risk analysis for the final rule through the use of central tendency parameters.

For dioxins/furans, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA used the MACT process to set technology-based emission standards. However, Sections 3004(a) and (q) of RCRA mandate that standards governing the operation of hazardous waste combustion facilities be protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate whether the MACT standards satisfy this requirement in order to determine what, if any, RCRA standards for emissions from these sources may be needed.

It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess risks associated with indirect exposure to emissions from HWC facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (*Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job."*

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board (SAB). EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to comments on these documents that have been received from the public, as well as from the SAB, in an appropriate forum.

***Comment 1107*:** The commenter cites EPA's Risk Assessment Support Document as

indicating that the recent dioxin reassessment (U.S. EPA, 1994b and c) was a source of both physical and chemical properties and additional methodology equations used in the risk analysis. The commenter notes that EPA received extensive comments and criticism on the dioxin reassessment, from EPA's Science Advisory Board and a host of other commenters

Response 1107: *EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards. However, EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document, for which the SAB commended EPA, calling it "a very credible and thorough job." In response to comments received from the SAB, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d). This study lends considerable support to the earlier cancer potency estimates from the 1994 draft reassessment and the 1985 health assessment. However, EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment (U.S. EPA, 1985) should be used.*

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

Comment 250: The commenter cited the peer-review comments that the estimated risk levels may be overstated and that the risk analysis failed to fully consider the degree of uncertainty associated with those risk estimates in drawing conclusions from the risk study.

Response 250: *EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *that addresses the peer review comments (U.S. EPA, 1999b). The commenter is referred to this document for EPA's response to the issues raised by the peer reviewers.*

EPA modified its risk analysis for the proposed rule in a number of important areas as a result of public comments and comments submitted by the peer-review panel. The risk assessment for the final rule was based on exposure characterization that included components that directly address the issue of providing representative coverage for exposed individuals. The goal of these modifications was to increase representativeness, and, in association, reduce uncertainty.

EPA acknowledges the uncertainty implicit in the use of the models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.

***Comment 1097*:** The commenter cites and concurs with the peer-review comment by Wilson that the selected exposure scenarios tend to give a false picture of the amount of exposure expected because they include no information by which one can judge the likelihood that any particular scenario will be realized. The uncertainty introduced by failing to indicate the likelihood of each scenario or the number of people likely to be exposed through each scenario also was not addressed explicitly.

Response 1097: *The risk assessment for the final rule was modified to account for the number of individuals exposed and their location. The analysis employed U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions which explicitly account for the numbers of persons exposed at differing levels of exposure. From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, and 99th percentiles.*

Comment 251: The commenter cites the peer-review comment relative to detection limits.

The commenter believes that the guidelines in the HWC Emissions Database (Volume II) to adopt detection limits for various congeners, when in most cases none will be emitted, may have an adverse impact on the final risk outcome for dioxin, particularly for the indirect exposure pathways.

Response 251: *For the final rule, EPA assumed that dioxin and furan congeners that were below the limit of detection were present at one-half the detection limit. This represents a middle course that avoids the conservatism that could be introduced by assuming congeners are present at their full detection limit yet accounts for the likelihood that the congeners are, in fact, present but at levels below the detection limit of the measurements. Although the issue of compounds being present at levels below the limit of detection contributes to uncertainty, the uncertainty is sufficiently small so as not to have a material effect on the findings and conclusions of the risk assessment. In particular, individual risks associated with high-end exposures are attributable to emissions of congeners that are present at levels well above the limit of detection rather than congeners that are below the limit.*

Comment 252: The commenter cited the peer-review comment that the use of the TEQ approach generally yields higher risk estimates for the more abundant and less toxic congeners of dioxins. However, a much more important problem is the gross error introduced by adopting TEQ as a true indicator of toxic hazard for exposures to dioxin mixtures. The risk analysis must address uncertainties associated with the use of TEQ approach.

Response 252: *EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *that addresses the peer review comments (U.S. EPA, 1999b). The commenter is referred to this document for EPA's response to the issues raised by the peer reviewers.*

EPA acknowledges that the SAB had a number of comments and concerns regarding the draft Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds *(draft dioxin assessment) and agrees that there are uncertainties associated with the TEQ approach. Although the SAB called for clarifications in the specification of TEFs for the various dioxin-like compounds for various health outcomes, the SAB concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds.*

Comment 253: The commenter cites the peer-review comment that the theoretical assumptions of the risk analysis may yield estimates greater than actual values in most cases. For

example, the deposition rate may be in error because of the assumption that little or no TCDD deposits on the relatively large mineral particles (fly ash) in the gas stream, when it is known that such condensation occurs.

***Response 253**: As noted in previous comments, EPA gave much consideration to the comments of the peer-review panel and, in response to these comments, made a number of changes to the risk assessment to improve its overall representativeness. EPA has prepared a separate response to comments document that addresses the peer-review comments. The commenter is referred to this document for EPA's detailed responses to the issues raised by the peer-review panel.*

The low amount of TCDD deposited on relatively large mineral particles may result partially from the use of a default particle size distribution that includes a relatively low fraction of large mineral particles. This should not have a strong impact on the analysis because, as discussed in ORD's dioxin exposure assessment and the 1993 Addendum (U.S. EPA, 1993a), evidence points to the fact that semivolatiles condense preferentially on smaller particles due to the larger surface area–to–volume ratio. Thus not only would these large particles be more efficiently collected in the air pollution control devices, resulting in little or no release of large particles to the atmosphere, but the smaller particles would also contain more deposited dioxin than the larger particles due to their relatively larger surface area.

Comment 254: The commenter cited the peer-review panel's comment that steady-state models tend to overestimate the nature and extent of risk. Depending upon the type of steady-state aquatic model used in the study, the results could vary by several orders of magnitude.

***Response 254**: As noted in previous comments, EPA made a number of changes to the risk assessment to improve its overall representativeness. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for EPA's response to the issues in the above comment.*

EPA agrees that the steady-state assumption may in some cases overestimate media concentrations. This is mitigated to a degree by the use of simple empirical relationships derived from field data to model bioaccumulation in the terrestrial and aquatic food chain. For aquatic systems, the steady state assumption is a greater limitation in larger bodies of water that have a long turnover time, such as drainage lakes. In the risk assessment for the final rule, a surface water model (IEM-2M) was used for modeling mercury water concentrations that does not assume steady state, although a simple empirical relationship was used for modeling bioaccumulation (of methylmercury) in fish.

This is consistent with the modeling approach used in EPA's Mercury Study Report to Congress *(U.S. EPA, 1997c).*

Comment 255: The commenter concurred with the peer-review panel with respect to the use of terrestrial food chain models for assessing risks from consumption of meat, eggs, milk, and other food products, stating that data gaps on media-and contaminant-specific transfer coefficients introduce substantial ambiguity in the risk analysis.

Response 255: *In the development of emerging methodologies, EPA must often accept limitations of data available. However, EPA believes there are currently sufficient evidence and data available to evaluate exposures from the aquatic and terrestrial food chain and that it is important to assess the risks attributable to such exposures. EPA has emphasized the nature and extent of the uncertainties involved in its characterization of risks for the final rule.*

***Comment 53*:** The commenter cites a peer-review commenter who states that background exposures to dioxins generally exceed site-specific exposure levels attributed in the report as due to dioxin emissions from the HWCs. The commenter further notes that the peer reviewer stated that there is no evidence to support the claim that long-range transport of emissions from HWCs are solely responsible for elevated background levels.

***Response 53*:** *EPA does not believe it is necessary to perform a comprehensive assessment of exposures to all anthropogenic emission sources within the context of the risk assessment for the hazardous waste combustion rule. EPA points to the ongoing work on the dioxin reassessment to inventory all sources of dioxin emissions in the U.S. (see* The Inventory of Sources of Dioxin in the United States, External Review Draft, *April 1998, U.S. EPA, 1998c).*

In assessing risks from dioxins for the final rule, EPA used a margin of exposure analysis for assessing noncancer risks. In this analysis, EPA compared the estimated average daily dose for HWCs to the average daily dose in the general population. The estimates of background exposure that were used for the general population represent total exposure to all sources of dioxins. Therefore, this analysis provides a direct measure of the relative exposure attributable to HWCs vis-a-vis total exposure from all sources.

***Comment 627*:** The commenter, in agreement with peer reviewers, states that there is no evidence to support the claim that long-range transport of emissions from HWCs are solely responsible for elevated background levels.

Response 627: *As explained in EPA's 1997* Mercury Study Report to Congress *(U.S. EPA, 1997c) EPA identifies that mercury cycles in the environment as a result of natural and*

anthropogenic activities. Most of the mercury in the atmosphere is elemental mercury vapor, which circulates in the atmosphere for up to a year and can be dispersed and transported thousands of miles from the source of emission. A computer simulation of long-range transport of mercury suggests that about one-third of U.S. anthropogenic emissions are deposited within the lower 48 states through wet and dry deposition, and that the remaining two-thirds is transported outside of U.S. borders where it diffuses into the global reservoir. The location of sources, the chemical species of mercury emitted, and climate and meteorology are key factors in mercury transport and deposition. The highest deposition rates from anthropogenic and global contributions are predicted to occur in the southern Great Lakes and Ohio River Valley, the Northeast, and scattered areas in the South. The flux of mercury from the atmosphere to land or water at any one location is comprised of contributions from the natural global cycle including re-emissions from the oceans, regional sources, and local sources.

Comment 257: The commenter concurs with the peer-review panel that the risk analysis had failed to reconcile the proposed reduction in mercury emissions with potential benefits to the aquatic environment.

Response 257: *EPA conducted a screening-level ecological assessment in the risk assessment for the final rule to evaluate the impacts of ecological exposure to mercury resulting from emissions from HWCs. In this approach, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water and compared to model-predicted media concentrations to assess the potential for ecological risk.*

The ecological assessment evaluated all chemical contaminants that were assessed in the human health assessment completed in support of the final rule. This includes dioxins and furans, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, lead, manganese, mercury, nickel, selenium, silver, and thallium. In addition, an ecological assessment was performed for copper. A somewhat more detailed analysis was done for aquatic mammals and birds for evaluating ecological risks from dioxins and furans. In this analysis, fish intake rates and congener-specific concentrations in fish were used together with mammalian and avian toxicity equivalence factors (TEFs) to estimate a TCDD-TEQ dose to these ecological receptors for comparison with ecotoxicological benchmarks for 2,3,7,8-TCDD.

Comment 258: The commenter cites the peer-review panel's comments that the preamble makes no case that mercury emissions pose a threat to human health at any level; there is a lack of supporting evidence to reasonably conclude that adverse health effects associated with ingestion of fish are currently occurring. The risk analysis does not provide a rationale to support the actual mercury

limits being proposed for HWCs.

Response 258: *The health effects of mercury are well documented. Clinically observed neurotoxicity has been observed following exposure to mercury. Generally, the most subtle indicators of mercury toxicity are neurological changes that include losses of motor skills and sensory ability. Humans, plants, and animals are routinely exposed to mercury, potentially resulting in a variety of ecological and human health impacts. EPA's 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) concluded that exposure to methylmercury through consumption of fish is most important to human health.*

For the final rule, EPA evaluated human health and ecological risks associated with exposure to elemental mercury, divalent mercury, and methylmercury. With respect to methylmercury,the human health evaluation completed for the final rule shows that exposures are projected to be below EPA's reference dose. However, the analysis is subject to considerable uncertainty, including the fact that background exposures are not taken into account. Exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing.

The final rule sets emission standards for mercury for existing HWCs based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule.

Comment 259**:** The commenter noted that only in three worst-case subsistence farmer scenarios did the floor-level risk estimates exceed the 1×10^{-5} RCRA criterion. Even for these three scenarios, risk estimates ranged from 2×10^{-5} to 5×10^{-5}, only slightly higher than the risk criterion. Given that these estimates are worst case, likely overstated, surrounded by a great deal of uncertainty and not necessarily representative of any actual combustion facility, the commenter believes that the risk analysis has failed to demonstrate that BTF dioxin/furan or mercury standards are justified. In fact, the commenter considers that the risk analysis shows that the proposed floor-level standards are protective.

Response 259: *Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed*

and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

The risk assessment for the final rule was modified to improve components of the exposure characterization that directly address the issue of providing representative coverage for exposed individuals. EPA received a number of public comments that the risk analysis for the proposed rule was not sufficiently representative of HWC facilities, including comments that noted the absence of smaller incineration facilities among the facilities analyzed. These comments were affirmed by the external peer review. Therefore, for the final rule, EPA increased the number of facilities and modified the way these facilities were selected for risk characterization from the HWC facility universe.

EPA did not rely on the case study approach in the final rule due to the number of comments received questioning the representativeness of the case study facilities. Instead, EPA used stratified random sampling to select an additional subset of facilities from the HWC universe for further analysis. The use of stratified sampling in selecting facilities allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe they are intended to represent (e.g., confidence intervals). Such statements could not be made using the case study approach. The use of stratified random sampling also allows risk results for a subset of facilities to be adjusted to represent an entire combustor category in a statistically meaningful manner based on their selection probabilities. However, other attributes of the case study approach were retained (or expanded) for the final rule, such as the use of facility-specific stack parameters (and emission rates) and site-specific meteorological, topographic, hydrologic, and land use data (as well as population and soils data). EPA believes that the use of stratified random sampling in conjunction with facility-specific and site-specific data ensures that the results of the risk analysis are representative of the larger universe of HWCs.

In the risk assessment for the final rule, EPA used site-specific parameter values whenever possible. These included facility-specific parameters such as measured emissions data for specific constituents including divalent and elemental mercury.

EPA acknowledges the uncertainty implicit in the use of the models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.

Section 18
Eli Lilly and Company
RCSP-0141

Comment 0141-1: The commenter states that EPA has not adequately characterized the risks posed by hazardous waste incinerators and therefore has failed to properly determine whether the rule is consistent with RCRA. In particular, wide criticism of the Dioxin Reassessment undermines the basis for the combustor MACT and requires reconsideration of the rule.

The commenter notes that a driving force behind the development of the MACT standards is the presumed exposure to dioxins from incinerator emissions, particularly as described in the three-volume report entitled *Health Assessment Document for 2,3,7,8-TCDD and Related Compounds* and its three-volume companion set on exposure (collectively termed "Dioxin Reassessment") published in 1994.

The Dioxin Reassessment has come under strong criticism, primarily from the international and U.S. scientific community as well as from within EPA itself. Much of this criticism was reported in written comments submitted on the Dioxin Reassessment, in a subsequent meeting of a special 39-member dioxin panel of the EPA's Science Advisory Board (SAB), and in published newsletters and magazine articles. For example, HWCs, cited in the Dioxin Reassessment as a "major" source of dioxin in the U.S., were found in testimony before the SAB to be well less than 1 percent, These figures have been replicated and published in many other reports in the U.S. and internationally. Health effects aside, one questions whether reducing a source which is already below 1 percent of emissions to levels far lower could have a significant impact on presumed health effects. The costs required to achieve these questionable results makes the issue more pressing still.

The errors inherent in the Dioxin Reassessment's summary chapter on the health effects of dioxin have caused even greater concern. In December 1994, several external authors of the Dioxin Reassessment took the unusual step of writing a letter to the editor of the well-known journal of the American Academy for the Advancement of Science (AAAS), which disputed the report's conclusions. This very publically expressed level of concern among scientists and participants in the reassessment process is an alarming commentary on the inherent conflict between policy and science within the EPA, one which has apparently manifested itself in the proposed MACT regulations. EPA's policy positions on MACT and dioxin clearly are not supported by the extensive data generated by billions of dollars worth of research over the past two decades.

It is the commenter's understanding that EPA has been asked to rewrite the conclusions of the Dioxin Reassessment to reflect data contained in earlier chapters. If the document forming the acknowledged basis of the combustor MACT rule has come under such vehement scientific criticism, it stands to reason that the need for MACT rule itself must be reconsidered. Given the inherent flaws in

the Dioxin Reassessment, the commenter believes that the scientific and technical basis for the proposed combustor MACT rule clearly has not been adequately established. The commenter recommends that the EPA's policy position on MACT and dioxin be reconciled with the available exposure and health effects before the MACT rule is promulgated. This is particularly appropriate given that EPA's own published schedule for issuing a MACT standard for hazardous waste incinerators is the year 2000.

***Response 0141-1**: EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards.*

However, EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job." In response to comments received from the SAB, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD), *U.S. EPA, 1997d). This study lends considerable support to the earlier cancer potency estimates from the 1994 draft reassessment and the 1985 health assessment. However, EPA's policy is that until the dioxin reassessment is concluded, the cancer slope factor from EPA's 1985 assessment should be used (*Health Assessment Document for Polychlorinated Dibenzo-p-dioxin, *U.S. EPA, 1985). That estimate is*

156,000 [mg/kg-d]$^{-1}$.

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

Section 19
The Environmental Technology Council
RCSP-0130

***Comment 527*:** The commenter attached a report that provides an evaluation of the risk analysis methodology used by EPA in justifying the BTF standards for the MACT emission standards. Although the commenter does not agree with some of the high end assumptions in the risk analysis applied to high end tendency data, the commenter does concur that EPA's risk analysis does show significant risk reduction for the BTF emission standards. The commenter notes also that no BTF analysis is needed for the particulate standard if EPA uses a valid statistical outlier test on the expanded MACT pool data. Given the toxicity of dioxins/furans and the concerns raised in the 1994 draft dioxin reassessment report, and considering the uncertainties in risk assessment, the commenter feels that it is sufficient for EPA to demonstrate a risk reduction trend in setting BTF standards for dioxins/furans. The commenter states that the risk analysis by EPA certainly does this, as shown in Figure 1 and Table 1 of the attached report.

Response 527: *EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. For the final rule, EPA did not use high-end values. In all instances, EPA either used central tendency values or conducted exposure parameter variability analysis to capture a range of exposure, in conjunction with site-specific data. The variability analysis was conducted for key exposure pathways and facilitates characterization of the full range of exposures without the use of high-end or upper-bound exposure assumptions.*

EPA evaluated all constituents for which data were available, including $PM_{2.5}$ and PM_{10}. The PM analysis evaluated the incidence of PM-related health effects avoided by reducing PM emissions as a result of the MACT standards. Health endpoints considered included mortality, hospital admissions, and respiratory symptoms.

Although EPA's analysis demonstrates the risk reductions that are achieved by the MACT standards, EPA set the level of the standards as prescribed in Section 112(d)(2) and (3) of the CAA. Emission standards for existing sources are based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as required by the CAA, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective

enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 1031*:** The commenter notes that statistics in the risk assessment document only give a summary of the results, so that it is difficult to do a detailed analysis in terms of which receptors and which individual facilities (if any) drive the risk estimates. The commenter strongly recommends that the EPA make their data more transparent so that these evaluations can be done.

***Response 1031**: The risk analysis completed for the final rule provides an expanded set of individual-level and population-level risk results that are differentiated as to receptor population, age group, constituent type and combustor category. Individual risk results for the final rule include multiple risk percentiles (i.e., 50th, 90th, 95th, and 99th percentile risk estimates) which characterize the range of risk experienced by a specific receptor population/age group combination including both central tendency and high-end. Similarly, population-level risk results generated for the final rule are differentiated (depending on the specific category of risk results) as to receptor population, chemical constituent, and combustor category. EPA believes the national-level risk information generated by the risk assessment for the final rule is the most appropriate information within a national standards setting context.*

While the risk analysis completed for the final rule does not provide a full set of risk results for each modeled facility, which would be prohibitive in any case due to the large volume of data generated in the analysis, EPA has fully documented the site-specific information used in the analysis. Other documentation includes all algorithms and parameter data used in the analysis, example calculations, expanded information on media concentrations, and facility-specific risk results for certain receptors and modeled sectors (i.e., maximum risk sectors for subsistence exposure scenarios). EPA believes that this collection of data and information, all of which is available in the docket for the final rule, satisfies EPA's goal of transparency in its risk assessments.

***Comment 1032*:** The commenter notes that summaries of the ranges of risks (low, median and high values for the so-called high end receptors) for incinerators and cement kilns show an overall trend toward risk reduction with increasing emissions controls. The commenter feels that it is important that this trend is observed at the low and median portions of the risk range, because the commenter is skeptical that the high values for the high end receptors can be used as a reasonable test (these receptors may be too extreme to be considered reasonable). The commenter notes that no risks, with the exception of the highest values of the high-end receptor, are particularly high, but the trend is toward lower risk with increasing controls.

Response 1032*: For the final rule, EPA modified the exposure scenarios to be more representative of the exposed population at each facility. The risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and U.S. Census and Census of Agriculture data to locate exposed individuals.*

For the final rule, EPA generated population-weighted cumulative distributions of risks to individuals, thereby eliminating the need to define central tendency and high-end exposure scenarios deterministically, in the manner done at proposal. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, 95th, and 99th.

EPA used central tendency exposure factors to estimate exposures, except for a few risk-driving pathways for which EPA performed an exposure factor variability analysis using Monte Carlo simulation. Monte Carlo simulation allows for a more complete characterization of the range of exposures for a population without use of overly conservative upper-bound parameters. Using this approach, EPA could more precisely characterize the high-end of the risk distribution while avoiding the need to set specific parameters to high-end values. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative.

Comment 1018: The commenter believes that because EPA used actual sites to develop many of the parameter values used in the risk assessment, the validity of these results for national standard-setting is dependent on the sites being representative of the universe of hazardous waste combustion facilities. In view of the importance of this factor, the commenter suggests that EPA develop a comprehensive program to determine the representativeness of the facilities chosen for study.

Response 1018: *EPA recognizes the importance of developing a representative facility set for the HWC risk analysis. For the final rule, EPA evaluated 76 active HWC facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). This set represents more than 40 percent of the hazardous waste combustion facilities covered by the rule. To ensure that these facilities would be representative of all facilities covered by the rule, 66 of the 76 facilities were selected using stratified random sampling, while the remaining 10 were facilities that had been analyzed at proposal and were retained for the final rule (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure).*

The use of stratified random sampling allows EPA to make clear statistical statements regarding the representativeness of risk results for the HWC facility universe. Specifically, for the final rule, confidence intervals reflecting sampling error (i.e., error

introduced into the analysis by not having modeled all HWC facilities) were generated for the majority of risk results. Sampling error is a key source of uncertainty that impacts the representativeness of risk results generated for the HWC risk analysis. Use of a nonstatistical selection strategy, such as the case study approach used at proposal, does not allow the representativeness of risk results to be assessed (e.g., confidence intervals reflecting sampling error cannot be generated). Sample sizes for each combustor category were based on the goal of having a 90 percent probability of selecting a facility from the top 10 percent of facilities within a given combustor category with regard to risk (i.e., a 90 percent probability of having included a high-risk facility in the sample). EPA's analysis for the final rule met or exceeded this goal. Stratified random sampling also reduces the level of bias in the overall analysis by avoiding the use of purposive or biased selection of modeled facilities. For these reasons, EPA believes the risk assessment for the final rule is representative of the range and types of HWC facilities. Furthermore, because of the sample sizes used in the analysis, EPA also expects that the risk assessment adequately characterizes risks at the high end of the risk distribution.

In addition to using stratified random sampling to select facilities for risk characterization, the risk analysis completed for the final rule also utilized facility-specific and site-specific data when available to improve the representativeness of both fate/transport modeling and exposure assessment. For example, U.S. Census and Census of Agriculture data were used to allow the distribution of human receptor populations and livestock around modeled HWC facilities to be reflected in the cumulative risk distributions used to characterize human health risk. Facility-specific emission measurement data were used when available (when these data were not available, values were imputed from facilities with similar operational characteristics). Site-specific data including waterbody-specific parameters and land-use classification data (i.e., GIRAS) were used to support fate/transport modeling in multiple media compartments including watershed soils and waterbodies.

***Comment 1033*:** The commenter briefly reviewed certain site-specific factors (e.g., deposition rates, distance to special receptors) and found nothing overtly different from facilities we have assessed in the past. Thus, the commenter feels that there is no obvious indication that the selected sites would over- or underestimate risk for hazardous waste combustion facilities in general. The commenter notes that this conclusion does not mean that risk assessment of individual incinerators will not result in both higher and lower risk estimates than presented for the study incinerators.

***Response 1033**: EPA has made a number of modifications to the risk analysis framework used for the final rule in order to increase the representativeness of the analysis. One of the modifications is the use of stratified random sampling to select HWC facilities for risk characterization. The use of stratified random sampling provides a statistically rigorous means for selecting facilities. With this approach, risk estimates*

generated for the subset of HWC facilities that are selected can be adjusted to reflect the universe of HWC facilities in a statistically meaningful manner on the basis of their selection probabilities. Stratified random sampling also allows clear statements to be made regarding the representativeness of the sampled facilities for the universe of HWC facilities (e.g., confidence intervals). In addition to implementing the use of stratified random sampling, EPA also selected a larger number of facilities for risk characterization for the final rule than were evaluated at proposal—11 case study facilities were evaluated at proposal, while 76 facilities were evaluated for the final rule. By increasing the overall number of facilities evaluated, EPA has increased the probability that the risk results will be representative of the overall universe of HWC facilities across the full range of exposures.

Comment 1019: Neither mercury nor particulate matter were included.

Response 1019*: EPA performed a quantitative analysis of risks from mercury and PM for the final rule, as the commenter suggests.*

EPA's PM analysis estimated the reductions in risk associated with a number of health endpoints that are projected to occur with the MACT standards, including:

- *Mortality from long-term exposure and from short-term exposure*
- *Hospital admissions for all respiratory infections, congestive heart failure, and ischemic heart disease*
- *Chronic bronchitis*
- *Acute bronchitis, lower respiratory symptoms, and upper respiratory symptoms*
- *Work loss days, minor restricted activity days, and restricted activity days.*

The results of the analysis indicate that risk reductions are expected for a number of health endpoints, including reductions in mortality, hospital admissions, chronic bronchitis, and respiratory symptoms, although the reductions are modest relative to those achieved by controls on other sources of PM.

The risk assessment for the final rule included an analysis of risks from mercury that followed the general modeling approach developed for the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c) and used the IEM-2M surface water model and reference dose for methylmercury developed especially for that report. For the final rule EPA evaluated human health and ecological risks associated with exposure to elemental mercury, divalent mercury and methylmercury. With respect to methylmercury, the human health evaluation shows that exposures are projected to be below EPA's reference dose. However, the analysis is subject to considerable uncertainty, including*

the fact that background exposures were not taken into account. Exposures from HWC emissions, when taken together with background exposures, could pose a cumulative risk to human health in special populations, such as persons engaged in subsistence fishing.

***Comment 1035*:** The commenter states that risks for receptors were calculated separately for the 50th and 90th percentile emissions estimates and that risk estimates were reported by facility type (incinerator, cement kill aggregate kiln) in summary form only. Thus, while ranges of risks for various receptors were reported, the commenter feels that it is not possible to tell the overall distribution of risk estimates among particular facilities or even between the emission estimates. Total risks for all receptors were summarized in a series of tables in Appendix B of the report and range over several orders of magnitude. For "central tendency" receptors, the risks are low and showed no apparent trend toward risk reduction with increasing levels of technological emissions control. The commenter notes that "low" is, of course, a relative term. The commenter notes that in the *Federal Register* citation, EPA expresses an interest in protecting high-end receptors at the 1 in 100,000 (1×10^{-5}) excess cancer risk level. To the commenter, this leaves in question what level of protection is required for central tendency receptors. For the purposes of its review, the commenter describes any risk below 10^{-5} (regardless of receptor type) as low.

For its review, the commenter concentrated on high-end receptors because this was the group upon which EPA focused. Further, the commenter concentrated on reports of overall dioxin cancer risks (i.e., total risks from all pathways reported for all receptors) for this group because this is the only summary information in the risk report providing median values as well as the upper and lower bounds of the results. The commenter provides a table to report overall dioxin risks in high-end receptors and a figure to provide a graphical representation. The commenter states that risks among high-end receptors are, for the most part, low as well—the only risks reported to exceed the 1 in 100,000 (1×10^{-5}) excess risk level were the high bound of the high-end receptors. However, the commenter states that as a result of the summary nature of the reporting, it is not possible to determine how "deeply" into the distribution of risks among different receptor scenarios risks in excess of 1×10^{-5} persist. The commenter notes that there is a clear downward trend in risk estimates, with increasing emission controls for incinerators and cement kilns that is not apparent for aggregate kilns. The risk reduction trend for incinerators and cement kilns exists over the entire range of the risk estimates. That is, it is apparent in the lower bound and at the median of the risk estimates as well as the high bound. The commenter states that this indicates that the risk estimate is being controlled by the emission estimate in high-end receptors and is not being somehow suppressed by the differing exposure assumptions applied to various receptors in the range.

***Response 1035**: EPA made a number of modifications to the HWC risk analysis for the final rule in order to improve the representativeness of risk results. Some of these modifications address explicitly the need to more completely characterize the range of risk (including both central tendency and high-end risk) experienced by modeled receptor populations located in the vicinity of HWC facilities. These include the use of (a) a study*

area out to 20 kilometers divided into 16 sectors to gain increased spatial resolution in evaluating both individual and population-level exposure around modeled facilities; (b) population-weighted cumulative risk distributions as the basis for identifying both central tendency and high-end risk levels for modeled receptor populations; (c) four different age cohorts (ages 0-5, 6-11, 12-19, and over 19 years) to reflect age-related differences in behavior in risk characterization; and (d) an exposure parameter variability analysis for key exposure pathways in order to refine estimates of the range of exposures occurring within certain receptor populations. Together these modifications result in an analysis that more completely characterizes the range of individual risk experienced by modeled receptor populations.

The use of population-weighted cumulative risk distributions as the basis for identifying high-end risk levels (i.e., 90th, 95th, and 99th percentiles obtained from those distributions) eliminates the need to define high-end exposure scenarios deterministically using conservative exposure assumptions, in the manner done at proposal. In addition, because the cumulative risk distributions are population-weighted, they reflect the distribution of individuals relative to the modeled HWC facilities, which significantly increases the representativeness of the analysis.

For the final rule, EPA characterized both central tendency risk and high-end risk using specific percentile values obtained from the population-weighted cumulative risk distributions. Central tendency risk was defined as the 50th percentile of the cumulative risk distributions, while high-end risk was defined as a range of values obtained from the cumulative risk distributions including 90th, 95th, and 99th percentiles). To provide a comprehensive set of individual risk results, these central tendency and high-end risk percentiles are provided for all modeled constituents, receptor populations, age groups, and combustor categories by route of exposure (e.g., 95th percentile ingestion cancer risk for dioxins for the 0- to 5-year-old children of commercial beef farmers residing in the vicinity of commercial incinerators). In order facilitate analysis of overall impacts to receptor populations, several different aggregated individual risk levels are also provided for the final rule (these are documented in the Background Document completed for the final rule). EPA also characterized population-level risk for the final rule in order to provide a more comprehensive set of risk results for assessing benefits. The final rule includes population-level risk characterization for dioxins and furans, carcinogenic metals, lead, and PM.

***Comment 143*:** The commenter provides the following general comments on the risk assessment. EPA chose to conduct this assessment using the Combustor Guidance and the Addendum; the commenter does not consider this guidance flawless in any way. Instead, the commenter finds it to be cumbersome, prone to overestimate risk, and not entirely supported by empirical findings. Nonetheless, the commenter acknowledges that it is the standard guidance for evaluating the risks of

hazardous waste combustion facilities seeking permits under RCRA. The commenter has used the guidance for risk assessment of hazardous waste (and other) combustion facilities and, indeed, has relied heavily on comparisons of our experiences using this approach to evaluate the risk report. The commenter notes that, as such, it is true that EPA is using a consistent and standard approach to risk evaluation in support of the proposed rule. The other standard approach would be to employ methods outlined in *Guidance for Performing Screening Level Risk Analyses at Combustion Facilities Burning Hazardous Wastes* ("The Screening Guidance," 1994). The commenter believes that this approach is so conservative as to yield meaningless results. Thus, the commenter feels EPA is to be commended for following an approach that yields at least plausible risk estimates. However, the commenter notes that even if an appropriate methodology is employed, it is still possible to generate misleading answers if the risk model is parameterized inappropriately.

In addition, the commenter is aware that much scientific debate has ensued since the Combustor Guidance and the Addendum documents were released concerning appropriate risk assessment approaches and assumptions. However, the commenter notes that the above EPA guidance has been required for performing risk assessments supporting the regulation of various combustion devices. It should be noted that the commenter did not review and evaluate every one of the many equations and input parameters. The commenter's evaluation was an overview evaluation designed to determine if there were major, obvious technical flaws or deviations from standard risk assessment practice. The commenter concludes that EPA's approach in the current document is generally consistent with the above guidance documents and historical precedents. In addition, the commenter notes that EPA appears, in most cases, to have moved in the direction of more realism when deviations from recent EPA assessments and approaches were found. The commenter feels that EPA should be applauded for adopting these realistic approaches.

> ***Response 143****: As noted by the commenter, EPA did not follow the risk methodologies that typically have been used for permitting HWCs due to differences in the objectives of the risk assessment for the final rule and those of the site-specific risk assessments conducted in a permitting context. Specifically, EPA did not follow the 1994 draft guidance for performing screening level analyses at hazardous waste combustion facilities.*
>
> *The risk assessment for the final rule is based on methodology presented in the 1990 Indirect Exposure Document,* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IEM) *(U.S. EPA, 1990) and the 1993 Addendum (U.S. EPA, 1993a). These methods were further updated with respect to dioxins, mercury, and lead. For dioxins, the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist of the procedures published in the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. For mercury, the assessment followed the general modeling approach developed for the 1997*

Mercury Study Report to Congress *(U.S. EPA, 1997c). Human exposure factors were obtained from the 1997* Exposure Factors Handbook *(U.S. EPA, 1997a), including consumption rates for home-produced foods and recreationally caught fish. For lead, EPA used the Integrated Exposure Uptake Biokinetic Model (IEUBK) to estimate blood lead concentrations in children.*

EPA modified the exposure methodology used at proposal to improve the representativeness of the analysis. For the final rule, EPA used central tendency exposure factors in conjunction with site-specific data for estimating exposures, except for a few risk driving pathways, for which an exposure factor variability analysis was performed using Monte Carlo simulation. In addition, the risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and concentrations in agricultural products. Also, U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in farming as an occupation or fishing as a recreational sport and persons engaged in farming or fishing for subsistence. EPA believes that these steps provide considerable assurance that the exposure estimates are reasonable and are not overly conservative.

***Comment 1036*:** The commenter states that the Combustor Guidance and Addendum contain certain approaches and parameter values that the commenter considers scientifically questionable. The commenter also raises the possibility that certain parameter values could have been set at indefensible levels in the risk report. Therefore, the commenter evaluated EPA's implementation of the risk model. The commenter feels that EPA is "keeping up" with new science since the issuance of the above-referenced guidance. The commenter notes that the review did find several parameter values to be more conservative than values the commenter had used in past assessments, but, on the other hand, the commenter found others at values equal to or less stringent than assumptions the commenter had made. The commenter feels that the report is a credible attempt to maintain plausibility of the risk estimates within the constraints of the Combustor Guidance. The commenter notes that this presents some difficulties with the reporting of data as it has been done in the risk report. The commenter also points out that compounding the difficulty is the fact that, since only the bounds and the median values are reported, the influence of the extreme receptors on the overall reporting of risk cannot be determined.

Response 1036: *As explained in response to the previous comment, EPA used the indirect exposure methodology for assessing risks from HWCs. Algorithms and parameter values from the 1993 Addendum (U.S. EPA, 1993a) were updated based on methods developed for the mercury study and the ongoing dioxin reassessment. EPA acknowledges the uncertainty implicit in the use of models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. In the development of emerging methodologies, EPA must often*

accept the limitations of the data available. However, EPA believes there are currently sufficient evidence and data available to evaluate exposures from the aquatic and terrestrial food chain and that it is important to assess the risks attributable to such exposures. EPA has emphasized the nature and extent of the uncertainties involved in its characterization of risks for the final rule.

For the final rule, individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, 95th, and 99th. Using this approach, it was possible for EPA to characterize the high end of the risk distribution more precisely, while avoiding the need to set specific parameters to high-end values.

Comment 1020: The commenter notes that EPA continues to evaluate risks in "subsistence" receptors (individuals who consume a substantially greater than average amount of one or another foodstuff, with a greater than average portion of the foodstuff coming from areas affected by combustion facility emissions) with absolutely no indication of the prevalence (or even the existence) of such subsistence behavior in the study areas. The commenter notes that when these exposure assumptions are combined with high-end (e.g., 90th percentile) emissions estimates and fate and transport model results for the most poorly situated facility, it is distinctly possible that an exposure estimate so extreme as to be implausible results. The commenter therefore rejects (as in past comments to EPA) the "high end of the high end" as a receptor that is useful for decision making.

Response 1020: *EPA has retained the use of subsistence scenarios for assessing risks in the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals.*

To assess the potential risks, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA also assumed that subsistence fishing could take place at any of the bodies of water that were modeled in the risk analysis. It was not possible to identify and locate individual farms that may be engaged in subsistence farming from census data or to characterize the location and prevalence of subsistence fishing activity. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. EPA recognizes that the assumptions made in order to assess risks associated with subsistence activities may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

***Comment 1021*:** The commenter states that it appears that the summary risks for high-end receptors include both the 50th and 90th percentile emissions estimates. Thus, the high bound on reported estimates are not just the high-end receptor as described in the risk report (central tendency except for the 90th percentile on exposure duration) but are probably the extreme receptors. In this way, the commenter believes EPA is misrepresenting their data to some extent

Response 1021: *In the risk assessment for the proposed rule, EPA conducted a site-specific modeling analysis for a small number of HWC facilities, specifically 5 cement kilns, 4 incinerators, and 2 lightweight aggregate kilns. For each of these example facilities, EPA constructed central tendency and high-end exposure scenarios for various receptor populations. High-end scenarios were constructed by setting one or two exposure parameters to high-end values, such as emissions and exposure duration. For certain receptor populations (i.e., home gardeners and the "typical farmer"), a third parameter, the contaminated fraction, also was set to high-end values. The contaminated fraction was the fraction of the intake of a food commodity that was assumed to be home-grown and contaminated at a higher level than the same food commodity obtained from local markets. A more detailed description of the contaminated fraction analysis, as well as the overall approach used to characterize central tendency and high-end exposures at proposal, is found in the background information document for the proposed rule (see* Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document*, February 1996). Results from the central tendency and high-end exposure scenarios were reported as a range. The range was the range of estimated risks for a given receptor across the example facilities in a source category.*

For the final rule, EPA modified the approach for characterizing the range of exposures from HWCs in a number of ways. First, EPA used stratified random sampling to increase the number of facilities analyzed in each combustor category. Second, EPA used U.S. Census and Census of Agriculture data to locate and enumerate exposed populations within 20 kilometers of HWCs. Third, EPA used central tendency exposure factors (e.g., mean values) in conjunction with facility-specific emissions estimates and other site-specific data to estimate exposures for these populations. Fourth, EPA performed an exposure factor variability analysis using Monte Carlo simulation for certain risk-driving pathways. These modifications allowed EPA to generate cumulative frequency distributions of risks to individuals living in the vicinity of HWCs and to more precisely characterize central tendency risks (i.e., at the 50th percentile) and risks at the high end of the distribution (i.e., the 90th, 95th, and 99th percentiles).

These modifications to the risk analysis for the final rule work in concert to ensure that characterization of both central tendency and high-end risk more accurately reflects site

conditions in the vicinity of HWC facilities as well as facility-specific operational parameters. EPA believes that these modifications to the risk methodology provide considerable assurance that the exposure estimates developed for the final rule are reasonable and are not overly conservative.

***Comment 1037*:** The commenter anticipates significant debate concerning the level to which the Risk Report supports EPA's proposed rule and believe EPA could greatly improve the evaluation necessary to resolve the debate by providing a more transparent report of its results. The commenter feels that this should include full reports of risk levels for separate facilities, as well as risk results separated by emissions assumptions (50th versus 90th percentile).

Recognizing the mass of data this represents, the commenter believes that EPA should consider some means of graphic representation of the range of results, such as the use of scattergrams. In view of the commenter's skepticism relative to the usefulness of the high bound estimates, it is extremely important to note that the observed risk reduction trend persists throughout the risk range reported by EPA. Therefore, the commenter notes that risk reduction is apparent even for more plausible exposure assumptions included in the data.

***Response 1037**: The risk analysis completed for the final rule provides an expanded set of individual-level and population-level risk results that are differentiated as to receptor population, age group, constituent type, and combustor category. With regard to individual risk, results for the final rule include multiple risk percentiles (i.e., 50th, 90th, 95th, and 99th percentile risk estimates), which characterize the range of risk experienced by a specific receptor population/age group combination, including both central tendency and high-end risk. Similarly, population-level risk results generated for the final rule are differentiated (depending on the specific category of risk results) as to receptor population, chemical constituent, and combustor category. Due to the large volume of data generated by the risk analysis, EPA has not produced a complete set of risk results for each of the facilities modeled. However, EPA has produced facility-specific risk estimates for the subsistence scenarios which includes risk results by exposure pathway. This information may be found in the docket for the final rule.*

EPA believes that the compilation of risk results generated for the final rule presents a thorough and comprehensive set of the results that, together with the detailed documentation provided of methods and data used in the analysis, fully satisfies EPA's goal of transparency in all its risk assessments.

***Comment 1022**: The commenter notes that in applying a partially site-specific approach, the usefulness of the results for decision-making on a national scale is extremely dependent on how the results may be extrapolated to hazardous waste combustion facilities as a whole. The commenter feels that this is of particular importance due to the fact that the number of study cases is small relative to the

total number of facilities for incinerators and cement kilns in the U.S. (there are only 7 aggregate kilns, total, reported to be burning hazardous waste, so the issue is perhaps less important for these facilities). The commenter conducted a brief review of certain site-specific factors and found nothing overtly different from real or proposed facilities they have assessed in the past. The commenter acknowledges that while this is a crude test, it suggests no obvious indication that the selected sites would over- or underestimate risk for hazardous waste combustion facilities in general. The commenter caveats that this does not mean that risk assessment of individual incinerators will not result in both higher and lower risk estimates than presented for the study incinerators.

***Response 1022**: In response to concerns raised by this and other commenters regarding the representativeness of risk results generated at proposal, EPA made a number of modifications to the risk characterization framework implemented for the final rule designed to improve the representativeness of risk results. Key among these modifications was the use of stratified random sampling to select a larger subset of HWC facilities from the HWC facility universe for risk characterization. The use of stratified random sampling allows risk results generated for the subset of modeled facilities to be extrapolated to represent the universe of HWC facilities in a statistically rigorous manner. Specifically, the use of stratified random sampling allows facility sampling weights to be derived and applied to adjust risk estimates generated for a subset of facilities to represent an entire combustor category. In addition, stratified random sampling allows confidence intervals reflecting sampling error to be constructed for the majority of risk results generated for the final rule. Sampling error refers to error introduced into an analysis by not having sampled and evaluated all facilities in a combustor category. By providing confidence intervals reflecting sampling error, a potentially significant source of uncertainty in the overall analysis has been quantified.*

In addition, EPA used a variety of facility and site-specific data in the risk analysis for the final rule. These include facility-specific emissions and stack data and site-specific population, meteorological, topographic, hydrologic, land use, and soils data. EPA believes that the use of stratified random sampling in conjunction with site-specific data ensures that the risk analysis is representative of the actual universe of HWCs.

***Comment 1023*:** The commenter believes the EPA risk report would benefit from a far more comprehensive evaluation of the representativeness of the selected facilities and may be able to use methods similar to the commenter's evaluation. The commenter suggests that as EPA is undoubtedly in possession of a number of risk assessments of hazardous waste combustion facilities, EPA may wish to compare the present study to the site characteristics, intermediate exposure estimates, and risk estimates presented in those reports.

***Response 1023**: As noted in response to a previous comment, EPA did not follow the risk methodologies that typically have been used for permitting HWCs due to differences*

in the objectives of the risk assessment for the final rule and those of the site-specific risk assessments conducted in a permitting context. Because of these methodological differences, comparisons between the risk analysis for the final rule and site-specific risk assessments that have been completed for permitting actions can be difficult to interpret. For instance, site-specific risk assessments are typically designed to assess risks to specific types of receptors (e.g., subsistence farmers) at allowable emission levels established in the permit. However, the risk assessment for the final rule is intended to assess the risks to the entire population based on current actual emissions and to project how those risks would change under the MACT standards. Due to these and other differences, EPA expects that risks obtained from the risk analysis for the MACT rule could be significantly different than the risks obtained from a risk analysis done for an individual permit.

***Comment 1038 and 144*:** The commenter states that it evaluated the risk report to determine if the approaches and specific input assumptions were consistent with or at variance with the Combustor Guidance or the Addendum. The commenter does not endorse these EPA guidance documents and the specific input parameters defined therein as the appropriate way to estimate the indirect risks posed by emissions from a combustion device. In addition, the commenter is aware that much scientific debate has ensued since both documents were released concerning appropriate risk assessment approaches and assumptions. However, the commenter notes that the above EPA guidance has been required for the performance of risk assessments supporting the regulation of various combustion devices. It should be noted that the commenter did not review and evaluate every one of the many equations and input parameters. The commenter's evaluation was an overview evaluation designed to determine if there were major, obvious technical flaws or deviations from standard risk assessment practice. The commenter concludes that EPA's approach in the current document is generally consistent with the above guidance documents and historical precedents. In addition, the commenter notes that EPA appears, in most cases, to have moved in the direction of more realism when deviations from recent EPA assessments and approaches were found.

***Response 1038 and 144**: It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from HWC facilities as part of the RCRA permitting process. The risk assessment for the final rule is based on methodology presented in the 1990 Indirect Exposure Document,* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IEM) *(U.S. EPA, 1990) and the 1993 Addendum to the 1990 Indirect Exposure Document (U.S. EPA, 1993a). These methods were further updated with respect to dioxins and mercury. For dioxins, the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist of the procedures published in the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. All parameter values used for assessing risks from dioxins were*

derived from the information in the draft dioxin reassessment or more recent information. For mercury, the assessment followed the general modeling approach developed for the 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c) and used the IEM-2M surface water model developed especially for that report. For lead, EPA used the Integrated Exposure Uptake Biokinetic Model (IEUBK) to estimate blood lead concentrations in children. Human exposure factors were obtained from the 1997* Exposure Factors Handbook*, including consumption rates for home-produced foods and recreationally caught fish. Therefore, EPA believes that the risk assessment for the final rule is consistent with current EPA practice and uses the best information currently available.*

***Comment 1024*:** The commenter feels that there are still some approaches and parameters that are unrealistic and inappropriate for a site-specific or generic combustor risk assessment. For instance, the commenter notes that EPA assumes in the current document that the lipid content of all fish in all locations is 7 percent and points out that such a value is certainly an overestimate for fish in warm waters, such as rivers and lakes in the southern United States.

***Response 1024**: EPA has made every effort to ensure that the data and methods used in the risk analysis for the final rule are realistic and appropriate.*

EPA agrees with the commenter that the fish lipid content used in the proposed rule was too high to truly represent a central tendency value, as intended. Fish lipid content will vary greatly even within a given species depending on age, physiological state, availability of food, season, and other factors. For the final rule, EPA concluded that lipid content should represent a lipid fraction in the fillet for a wide variety of species across the United States. For the human health analysis, EPA used a lipid content of 2.6 percent, which was estimated assuming 36 percent consumption of trophic level 3 fish (lipid content 2 percent) and 64 percent consumption of trophic level 4 fish (lipid concentration 3 percent). This is consistent with a lipid content of 3.1 percent recommended in the final rule of the Great Lakes Water Quality Initiative *for edible portions of trophic level 4 fish (60 FR 15373-15374).*

For the ecological risk assessment, EPA used higher lipid contents representative of the whole fish, including internal organs. A lipid content of 6.5 percent was used for trophic level 3 fish and a lipid content of 10.3 percent for trophic level 4 fish. The intake of trophic level 3 and 4 fish was assumed to vary by ecological receptor.

***Comment 1086*:** The commenter states that EPA in the current document deviates from the draft dioxin reassessment and assumes that 60 percent of the dioxin present in wet deposition adheres to plant surfaces. The commenter feels that the value used in the draft dioxin reassessment (30 percent) is more realistic, but itself may be an overestimate.

Response 1086: *The 1993 draft Addendum (U.S. EPA, 1993a) recommended a value of the fraction of wet deposition that adheres to plant surfaces (Fw) of 100 percent. The Science Advisory Board (SAB) commented on this parameter in its review of the draft Addendum (U.S. EPA, 1993a). The SAB addressed the question of whether a wet retention fraction of 30 percent might be more appropriate than assuming 100 percent retention for contaminants that have high sorptive tendencies. The SAB agreed that 100 percent was not appropriate for such contaminants, arguing that it is the particle as a whole and not the sorption of the individual contaminants that determines the degree of retention. Subsequently, EPA derived a wet deposition fraction based on Hoffman's interception fractions for particulates (Hoffman et al., 1992). To obtain a wet deposition fraction for particles, Hoffman's interception fractions for three different particle sizes were adjusted by an assumed interception fraction of 0.47 for forage. Based on the results for the three particle sizes, a value of 0.6 was chosen as a representative central tendency value. This is the same value that was used in the* Mercury Study Report to Congress *(U.S. EPA, 1997c). EPA acknowledges that there is some uncertainty in these estimates. The sources of uncertainty include experimental errors, uncertainty of applying experimental results to other pollutants, variations in rainfall intensity, and uncertainty of long-term retention.*

Comment 1039**:** The commenter describes various changes from recent EPA risk assessment approaches used in the current document that are more realistic and should be adopted for site-specific combustor risk assessments. In particular, the commenter discusses actual versus hypothetical receptor locations. The commenter notes that regulatory agencies have historically required risk assessors to assume that large quantities of fish were ingested from nearby ponds and lakes regardless of the carrying capacity of the waterbody or actual knowledge of fishing activities. In the current document, the commenter notes that EPA appears to be focusing on waterbodies where fish consumption can and does occur. Similarly, the locations evaluated for produce, beef, dairy, pork, and chicken consumption scenarios appear to have been defined as locations where such activities are actually known to occur, instead of locations were someone could in theory raise produce or livestock. The commenter feels that this use of actual site-specific information should be encouraged.

Response 1039: *As indicated by the commenter, EPA attempted to determine the actual locations of farms and bodies of water used for fishing. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Instead, EPA used U.S. Census and Census of Agriculture data to locate farm households. Within a study area, up to four bodies of water were chosen for analysis based on their proximity to the sample facility and the likelihood of their being used for recreational purposes, as indicated by factors such as size and accessibility. Waterbodies were also chosen if they were used to supply drinking water to the surrounding community. However, it was not possible to identify*

and locate individual farms that may be engaged in subsistence farming from census data or to characterize the location and prevalence of subsistence fishing activity. Therefore, to assess the potential risks from subsistence farming, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA also assumed that subsistence fishing could take place at any of the bodies of water that were modeled in the risk analysis. EPA acknowledges that the assumptions made in order to assess risks associated with subsistence activities may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

The risk analysis for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level. Therefore, the risk methodology may differ from what is typically used in a permitting context. In particular, the risk analysis for the final rule is not intended to serve as guidance for performing site-specific risk assessments that may be required for permitting. EPA has issued other guidance for that purpose.

***Comment 1040*:** The commenter states that EPA did not perform risk assessment in numerous dispersion and deposition-defined zones around the facilities, as proposed in the Addendum. The commenter notes that numerous commenters over the years have noted that the proposal was complex and without clear decision-making benefit compared to the resources required to execute it. The commenter applauds EPA for recognizing that the zonal analyses are not necessary for assessing the risks posed by combustor emissions.

***Response 1040**: For the HWC risk analysis, EPA defined a study area as the area surrounding HWC facilities out to a distance of 20 kilometers. Within a study area, a 16-sector template was used within a GIS to provide the spatial resolution required to meet the objectives of the assessment. The 16-sector template (made up of concentric rings at 2, 5, 10, and 20 km transected by north-south and east-west axes) provided increased resolution close to the facility, where deposition and air concentrations are likely to be elevated. By applying the 16-sector template within a GIS, digital spatial data (including U.S. Census data and Census of Agriculture data) could be used to establish residential and commercial farming populations for each of the sectors. This population density data allowed both individual-level and population-level risk estimates to be generated, reflecting the actual locations of residential and farming populations. The use of the 16-sector template also for spatial enhanced resolution in characterizing exposure because separate air concentration/deposition values were generated for each of the sectors and fate/transport modeling through the food chain was performed for each sector.*

***Comment 1041*:** The commenter feels that the beef, dairy, and fish ingestion rates appear not to be unreasonable values. In particular, the commenter notes that EPA has used fish ingestion rates of

1.64 g/day for the adult resident and farmer, 30 g/day for the recreational fisher, and 60 g/day for the subsistence fisher. The commenter acknowledges that while there is considerable debate about the appropriate values to use depending on the nature of the water body (fresh water versus salt water), these values are more reasonable and realistic than some values recently proposed by EPA. In particular, the commenter feels that the 140 g/day ingestion rate proposed in the 1994 screening guidance was not justified by the scientific literature.

Regarding plants and produce, the commenter feels that EPA's approach to assume that grain is a "protected crop" that does not receive vapor uptake or particle deposition appears reasonable.

***Response 1041**: In the HWC risk analysis, the only consumers of fish are the recreational fisher, the subsistence fisher, and the subsistence farmer. However, unlike the recreational and subsistence fishers, who are assumed to fish from local bodies of water, the subsistence farmer is assumed to raise fish in farm ponds. For the adult subsistence farmer and the adult recreational fisher, a fish ingestion rate of 0.16 g/kg-day was used for the final rule. Assuming an adult body weight of 71.8 kg, this corresponds to a daily ingestion rate of 11.5 g/day. The ingestion rate for the subsistence farmer was assumed to be equivalent to that of the recreational fisher because data were not available on fish consumption by farmers. For the adult subsistence fisher, an ingestion rate of 0.97 g/kg-day (equivalent to 69.6 g/day) was used. Ingestion rates for the subsistence fisher and recreational fisher/subsistence farmer all were derived from EPA's 1997* Exposure Factors Handbook *(EFH) (U.S. EPA 1997a).*

For the final rule, EPA maintains the distinction between"protected" and "unprotected" portions of above-ground plants. In particular, grains used for animal feed are assumed to be protected. Because the outer covering on the protected parts of the plant acts as a barrier, contamination of this part of the plant through deposition of particles and vapor transfer is assumed to be negligible, and contamination is assumed to occur only through root uptake.

***Comment 1042*:** The commenter states that there was insufficient time allotted to allow the commenter to evaluate the new air-to-plant biotransfer factors used in the current document, noting that they vary from those used in EPA's Addendum. The commenter believes that the biotransfer factor approach overestimates the uptake of vapor phase dioxin congeners into plants (see Magee and Smith, 1995). However, the commenter acknowledges that the values used in this document are more realistic than the values proposed in the Addendum.

***Response 1042**: The air-to-plant biotransfer factors used in the final rule were derived from the ongoing dioxin reassessment and were published in a 1995 paper, "Development of Air-to-Leaf Vapor Phase Transfer Factor for Dioxins and Furans" (Lorber, 1995). The biotransfer factors were estimated from experimental data on the*

uptake of dioxins by welsh ray grass and are lower than the values in the 1994 draft dioxin reassessment derived using the Bacci algorithm and an empirical adjustment factor. For example, the value for 2,3,7,8 -TCDD was 6.1 x 10^4 (µg TCDD/g plant tissue DW(/(µg TCDD/g air) for Welsh ray grass vs. 1.0 x 10^5 from the 1994 dioxin reassessment. Values for the other dioxin congeners ranged from 8.1 x 10^4 to 8.6 x 10^6 for Welsh ray grass. The values used by EPA for the final rule are presented in Appendix D of the risk assessment Background Document (RTI, 1999).

***Comment 1043*:** The commenter applauds EPA for performing congener-specific fate and transport modeling into fish, beef, and dairy products given that it is well recognized that the uptake of dioxin congeners into meat and milk varies and that higher molecular weight congeners are biotransferred to a lesser extent than is 2,3,7,8-TCDD and other lower molecular weight congeners. The commenter feels that the milk and beef biotransfer factors are reasonable. The commenter encourages EPA to continue using congener-specific procedures but to reevaluate the specific values used in the current document.

***Response 1043**: For the HWC risk analysis, EPA followed the site-specific modeling procedures developed for the 1994 dioxin reassessment, including congener-specific modeling of fate and transport. As indicated by the commenter, bioaccumulation varies for the different dioxin and furan congeners and EPA believes it is important that these differences be considered in the risk analysis.*

Regarding biotransfer factors in beef and milk, EPA is using values derived from an experimental data set for a lactating cow published by McLachlan et al. (1990). This is the same data set that was used in the 1994 dioxin reassessment to derive bioconcentration factors for beef and milk. The biotransfer values used in the final rule are found in Appendix D of the risk assessment Background Document (RTI, 1999). Although there are uncertainties in the use of these data for estimating biotransfer factors, including the uncertainties implicit with the use of the same data set to derive biotransfer factors for both lactating and nonlactating animals, EPA believes these are the best data currently available on the full range of congeners that are needed for assessing risks from dioxins and furans.

***Comment 1044*:** The commenter notes that the fish biota to sediment accumulation factors (BSAFs) for dioxin congeners were taken from the dioxin reassessment. While the commenter encourages the use of congener-specific approaches, the commenter does not agree that the BSAFs proposed by EPA in 1994 are appropriate for risk assessment of dioxin and furan congeners. The commenter provides a table that shows BSAFs derived for the various congeners included in the two studies from the dioxin reassessment (Kuehl et al., 1987, and Kjeller et al., 1990) that investigated accumulation of multiple congeners in one species of fish. The commenter notes that these BSAFs clearly demonstrate the expected trend of decreasing BSAF with increasing chlorination. The table also

shows the BSAFs proposed by EPA in the *Federal Register* on August 30, 1994. The commenter encourages EPA to continue using congener-specific procedures but to reevaluate the specific values used in the current document.

> ***Response 1044****: As indicated by the commenter, the biota-sediment accumulation factors (BSAF) used in the risk assessment for the proposed rule were taken from the 1994 dioxin reassessment (U.S. EPA, 1994a,b). However, the appropriateness of these values, which were derived from studies in the Great Lakes, has been questioned. For the final rule, EPA revisited the BSAF values used to calculate the concentration of dioxins/furans in fish and derived BSAF values from fish tissue and sediment data presented in a 1992 report from the State of Connecticut,* Multivariate Statistical Analyses of Dioxin and Furan Levels in Fish, Sediment, and Soil Samples Collected Near Resource Recovery Facilities *(Bauer, 1992). BSAF values for 2,3 7,8- TCDD, corrected for lipid content and fraction organic carbon, ranged from 0.16 to 0.97 across five sites in Connecticut. Values from the Connecticut study were higher than BSAFs in the draft dioxin for all congeners. This trend is consistent with the view that biota-sediment accumulation factors are higher in aquatic environments that are experiencing ongoing contamination, such as in the Connecticut study, than in the Great Lakes where impacts are largely due to historical contamination. Because the HWC risk analysis is intended to assess the risks from operating HWCs, EPA concluded that the BSAF values used in the final rule should be based on the Connecticut study and not the Great Lakes. The BSAF values for the final rule are found in Appendix D of the risk assessment background document (RTI, 1999).*

Comment 1045**:** The commenter feels that EPA excluded from its risk assessment several exposure pathways that typically result in insignificant risks in a multipathway indirect risk assessment compared to other exposure pathways. The commenter states that these pathways include dermal absorption of chemicals from soil, dermal contact with surface water (during swimming or wading), and incidental ingestion of chemicals in surface water (during swimming or wading). In the commenter's opinion, it is reasonable to exclude these pathways from such risk assessments. The commenter encourages EPA to recommend their exclusion from all combustor risk assessments.

> ***Response 1045****: EPA did not conduct a quantitative assessment of risks from dermal exposures for the HWC risk analysis. EPA expects that risks associated with direct inhalation exposures and indirect exposures through the food chain are considerably greater than any risks associated with dermal exposures. This has consistently been shown to be the case in studies that have examined dermal exposures from combustion sources. Therefore, for the final rule EPA did not evaluate dermal exposure pathways. However, this does not mean EPA has reached the same conclusion regarding other releases from industrial sources. For the final rule, EPA assessed risks associated with a variety of other exposure pathways, including direct inhalation, incidental soil ingestion,*

drinking water ingestion, and consumption of home-produced meat, milk, eggs, poultry, fish, and produce.

The risk analysis for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level. Therefore, the risk methodology may differ from what is typically used in a permitting context. In particular, the risk analysis for the final rule is not intended to serve as guidance for performing site-specific risk assessments that may be required for permitting. EPA has issued other guidance for that purpose.

***Comment 1046*:** The commenter notes that EPA did not include the breast milk ingestion pathway in the quantitative risk assessment as suggested in the Addendum by adding lifetime excess cancer risk from breast milk ingestion to risks posed by other exposure pathways. Instead, the level of incremental TCDD toxic equivalents was calculated and compared to baseline concentrations in breast milk. The commenter states that EPA has recognized the inherent difficulties in adding numerical risk values as has been done in the past. Historically, risk assessors have assumed that the receptor living near a combustor did so from birth and received chemical doses from breast milk, produce, beef, fish, etc. for varying periods of time up to 70 years, in some cases. The commenter notes that to have a breast milk dose over the first year of life required that the risk assessor assume that the mother also lived near the facility for varying periods of time, usually up to 30 years. The commenter feels that such an approach was unrealistic because it assumed that people were exposed to facility-associated chemicals for a period longer than the typical facility lifetime. The commenter points out that in the current document, EPA has avoided such an unrealistic approach by assessing the impacts of the facility on breast milk separately from the incremental cancer.

***Response 1046**: EPA agrees with the commenter that it may not be realistic to combine breast milk exposures with exposures that occur later in life. The probability that an individual would spend his or her entire life in the same geographic location is very small. Of greater likelihood is that an infant that breastfeeds at birth may continue to be exposed well into childhood and that the exposures that occur during the first year of life could make a significant contribution to a child's average exposure.*

For the final rule, EPA did not attempt to characterize the risk of cancer for exposures that occur in the first year of life from breastfeeding. There is considerable uncertainty in estimating cancer risks in children on the basis of lifetime average daily dose, in the manner normally done for adults, and this uncertainty is further exacerbated when the exposures occur in the first year of life. However, because exposures during nursing may substantially exceed exposures later in childhood, EPA believes that it is important to assess the potential <u>noncancer</u> risks associated with such exposures. Recent research suggests that the primary toxicity endpoint of concern from exposures to dioxins/furans in infants may be developmental effects.

For the final rule, EPA assessed the potential for noncancer health effects using a modified margin of exposure (MOE) approach. In this approach, the estimated average daily dose to the infant from HWCs was compared to background exposures to infants in the general population that breastfeed. Because exposures through breast milk are expected to be among the highest exposures that occur during early childhood, and given the concern for developmental effects, EPA believes that it is important to assess the potential risks during this critical exposure period; furthermore, if it can be concluded that the potential for risks during that time period is small, then exposures at other times and through other pathways are not likely to be cause for concern. This is the approach EPA took in the risk assessment for the final rule.

***Comment 145*:** The commenter evaluates the representativeness of the study facilities that EPA used to perform a generic site-specific risk assessment of MACT emission standards and proposed BTF-MACT standards. The commenter states that EPA did this by performing the risk assessment on a site-specific basis for a limited number of actual facilities including four incinerators, five cement kilns, and two lightweight aggregate kilns. The commenter observes that EPA's risk assessment results are meaningful only if the selected facilities are representative of the approximately 200 combustors to which the standards would apply and notes that EPA is aware of the representativeness issue. To illustrate that EPA is aware of the representativeness issue, the commenter cites the proposed rule as indicating that it is likely that the results do not cover the range of possible individual risks across all of the facilities as a result of the small number of facilities in each category. Also, the commenter cites EPA as stating that 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, so the facilities cannot be considered statistically representative of all hazardous waste combustion units. Accordingly, the commenter evaluated the facilities to determine if they were at least reasonably representative of the entire class of facilities or if they were clearly unusual compared to other members of the class. This evaluation was by necessity a limited, nonrigorous exercise. The commenter compared the proximity of the facilities to potential receptors and selected modeling results for each facility and for facility type categories (incinerator, cement kiln, lightweight aggregate kiln). The commenter prepared a table that shows the proximity of each facility to the designated beef farm, dairy farm, and the closest of several fishing locations, a table that shows for each facility type the shortest distance to these three receptor locations, and a table that shows that average distance to each of the three receptor locations. The commenter notes that, as shown in these tables, EPA's facilities are for the most part facilities that are close to farming and fishing locations and that there are no obvious, major differences between facility types regarding proximity to such potential receptor locations. In the commenter's experience doing site-specific assessments, such receptor locations are often within several miles of any given combustion facility. In other cases, actual farms may not be located so close (1-5 km) to a combustion facility, but agricultural land that could be used for beef or dairy farming is closely located. The commenter does note, however, that there may exist some combustion facilities in highly urbanized areas that are many miles from any agricultural lands, in which case the 11 facilities used in EPA's risk assessment would not be representative.

The commenter provides another table that shows selected air dispersion modeling results for each facility and notes that there is a considerable range of values. For instance, the commenter points out that the particle concentration at the nearest residential location varies from 0 to 0.3 $\mu g/m^3/g/sec$. Also, the highest total particle deposition rate for the beef or dairy farm varies from 0.0006 to 0.11 g/m^2-yr/g/sec. These modeling results are all within the range of values observed by the commenter for other combustor facilities. The commenter provides a table that shows the highest of the air and deposition modeling results for each facility type as well as a table that shows the average value for these results for each facility type. The commenter also provides a table that shows the average value for total particle deposition rate at the beef farm, the dairy farm, and all fishing locations for each facility type category. As noted by the commenter, from these three tables, the facilities appear to be similar between facility types with the exception that the particle air concentration at the closest residential location is clearly lower for the cement kiln category than for the other two categories. Also, the commenter notes that the particle deposition rate for the fishing locations is higher for the aggregate kiln category compared to the other two categories.

The commenter feels that because only a selected number of comparisons was made in this preliminary evaluation, it is not possible to draw a firm conclusion about the representativeness of the selected facilities compared to all regulated facilities. In general, however, the commenter concludes from this evaluation that there are no obvious major differences between the facility setting and meteorology between facility type categories. Also, the commenter notes that facilities modeled are close to potential receptor locations (1-5 km), which is not unreasonable. The commenter feels that the risk assessment results presented by EPA are probably not underestimated for all facilities due to reasons of receptor proximity and meteorologic conditions. However, the commenter recognizes that it is possible that actual risk assessment results for some facilities subject to the proposed rule would be overestimated by EPA's generic analysis.

Despite encountering some difficulties in evaluating the data relating to EPA's reporting format, the commenter's overall impression is that EPA has done a credible job of evaluating health risks of dioxin emissions from hazardous waste combustion facilities, using a standard methodology. The commenter agrees that results indicate a risk reduction trend as one proceeds from baseline to MACT to BTF-MACT.

> ***Response 145****: For the final rule, EPA made a number of changes to the risk assessment to improve the representativeness of the analysis. These include the use of stratified random sampling to select facilities for analysis, facility- and site-specific data, central tendency exposure parameters, and population-based risk characterization. The commenter is referred to the risk assessment background document for the final rule for a complete discussion of the modifications made to the risk assessment framework.*
>
> *With regard to facility selection, the use of stratified random sampling provides a statistically rigorous means for selecting facilities from the HWC facility universe for risk*

characterization. With this approach, risk estimates generated for the subset of HWC facilities that are selected can be adjusted to reflect the universe of HWC facilities in a statistically meaningful manner based on their selection probabilities. The use of stratified random sampling also allows clear statements to be made regarding the representativeness of the sampled facilities for the universe of HWC facilities. EPA also selected a larger number of facilities for analysis for the final rule than were evaluated at proposal, increasing the number of facilities evaluated to 76 (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators), including 10 of the 11 facilities analyzed at proposal (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure). By increasing the overall number of facilities that are modeled, EPA has increased the probability that the facilities selected will be representative of the universe of HWC facilities with regard to key locational characteristics (e.g., presence of commercial dairy/beef farms, meteorological conditions, population density) and facility characteristics (e.g., stack parameters, emissions rates).

For the final rule, EPA modified the exposure scenarios to be more representative of the exposed population at each facility. The risk analysis for the final rule also used site-specific emission estimates to estimate media concentrations and U.S. Census and Census of agriculture data to locate exposed individuals and livestock within modeled study areas. Site characterization was supported by the use of a GIS, which facilitated incorporation of digital spatial data including census data and GIRAS land-use data. An exposure parameter variability analysis was also included for key exposure pathways to more fully reflect the range of exposure experienced by individuals residing in the vicinity of HWC facilities.

The Environmental Technology Council
RCSP-0243

***Comment 279*:** The commenter shares the concerns raised by the peer reviewers regarding the use of extreme generic worst case assumptions in producing a hypothetical upper bound risk in any risk assessment. Nevertheless, the commenter recognizes a clear trend of significant risk reduction is demonstrated in the BTF standards for dioxins/furans by EPA's risk analysis, even for the central and high-end portions of the risk range. Therefore, the commenter feels EPA's risk assessment provides a sound basis for concluding that the BTF emission limits for dioxins/furans will result in significant risk reduction compared to the pollution control costs, without reliance on the hypothetical upper bound risk data. The commenter states that EPA has succeeded in demonstrating risk reduction at the proposed BTF standard for dioxins/furans. The commenter notes that it is also important to recognize that the risk evaluation is just one portion of the decision-making process to set a MACT emission standard beyond the floor (BTF) and that other considerations include technical feasibility and cost.

The commenter notes that in 1994, EPA released its draft dioxin reassessment report and that this report presented additional toxicological data and information on the noncancer health impacts of dioxin and the high background of dioxin compounds in the environment relative to these toxicological benchmarks. The commenter notes that this report also justifies EPA's taking actions in this MACT rule for HWCs to reduce dioxins/furans emissions to the maximum extent feasible. Therefore, apart from EPA's risk assessment and the peer-review panel's concerns, the commenter believes the 0.2 ng/dscm TEQ dioxin/furan standard is reasonable and achievable.

***Response 279**: EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to the particular parameters. That is why, for the final rule, EPA did not use high-end values. In addition, EPA did not make bounding estimates as referred to by the commenter. In all instances, EPA used central tendency values, and in some cases, EPA conducted an additional exposure parameter variability analysis. The variability analysis was conducted for key exposure pathways and facilitates characterization of the full range of exposures without the use of high-end or upper-bound exposure assumptions.*

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains

necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 280*:** The commenter notes that for mercury, the peer reviewers commented that EPA has not made the case for MACT controls since a quantitative risk assessment was not done to support the BTF standard. Although the risk analysis report does not include risk calculations on mercury, the commenter does feel that EPA has adequately justified setting BTF limits for mercury in this rulemaking record. First, the extraordinarily high prevalence of fish advisories for over 1,700 water bodies in 47 states due to mercury levels is alarming. The commenter feels that this provides justification to control mercury emissions to the extent practical and feasible. Second, although the Report to Congress on mercury is being delayed, the reason for the delay is not disagreement over whether mercury is toxic, but over what the reference dose should be. The commenter believes that the draft report does demonstrate that mercury toxicity is a serious issue in the environment and that its impacts are potentially severe. The commenter believes that it would be unfortunate if EPA allowed the debate among toxicologist over the appropriate reference dose to delay an opportunity to control mercury emissions from HWCs. The commenter feels that enough is known now, and justification exists, to take actions to control mercury emissions in this MACT rule, without the need for a quantitative risk analysis.

Response 280: *For the final rule, EPA conducted a quantitative analysis of both the human health risks resulting from exposure to mercury released from HWC facilities as well as an analysis of the potential for adverse impacts to ecological receptors following mercury exposure. The mercury analysis followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(U.S. EPA, 1997c) and used the IEM-2M surface water model and methylmercury reference dose developed especially for that report.*

EPA's Science Advisory Board, in its review of the mercury study, endorsed retention of the RfD based on the Iraqi study of methylmercury contaminated seed grain, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). For the final rule, EPA is setting the emission standard for mercury at the MACT floor. EPA performed a risk assessment in order to evaluate whether the MACT standards are

generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 281*:** The commenter feels that the case study approach used by EPA was adequate to demonstrate on a national basis the justification to set BTF limits for dioxins/furans. The commenter notes that the peer-review panel seems to support this approach also, and notes that this departure from "conventional" approaches has minimal impact on the risk numbers. The commenter feels that the 11 sites used in the case studies are representative.

***Response 281**: EPA did not rely on the case study approach in the final rule due to the number of comments received questioning the representativeness of the case study facilities. Instead, EPA used stratified random sampling to select an additional subset of facilities from the HWC universe for further analysis. The use of stratified sampling in selecting facilities allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe they are intended to represent (e.g., confidence intervals). Such statements could not be made using the case study approach,. The use of stratified random sampling also allows risk results for a subset of facilities to be adjusted to represent an entire combustor category in a statistically meaningful manner based on their selection probabilities. However, other attributes of the case study approach were retained (or expanded) for the final rule, such as the use of facility-specific stack parameters (and emission rates) and site-specific meteorological, topographic, hydrologic, and land use data (as well as population and soils data). EPA believes that the use of stratified random sampling, in conjunction with facility-specific and site-specific data, ensures that the results of the risk analysis are representative of the larger universe of HWCs.*

Section 20
Gossman Consulting, Inc.
RCSP-0217

***Comment 464*:** The commenter presents two tables that compare the carcinogenic products of incomplete combustion (PIC) emissions from the Clarksville cement kiln with the Aptus incinerator in Utah. The commenter makes the following points:

- # A comparison of carcinogens was used because the relative risks from noncarcinogens is so low that carcinogens become the dominant factor. Likewise dioxins were not included because they become a much greater factor than the other PICs.
- # The Aptus, Utah, incinerator was chosen because it was the only commercial incinerator trial burn that reported PICs, dioxins, hydrocarbons (HC), and carbon monoxide (CO).
- # Dioxin emissions on a toxic equivalents (TEQ) basis ranged from 0.5 to 1.4 ng/dscm under baseline conditions for Clarksville. While burning hazardous waste feed (HWF), levels ranged from 0.04 to 0.7 ng/dscm. The Aptus incinerator dioxin emissions ranged from 11 to 28 ng/dscm at 7 percent O_2, TEQ.
- # The Aptus incinerator operated at 5-10 ppm HC and 5-10 ppm CO, while the Clarksville cement kiln operated at 150-220 ppm HC and 400-550 ppm CO.
- # Neither the Clarksville nor the Lone Star, Greencastle, cement kiln detected any chlorobenzene in its emissions despite being specifically looked for. This previously observed phenomena may be dependent on preheater kilns or specific precursors in the raw materials.
- # Each set of tables presents the raw data from the respective reports at the top, the data converted to a mg/ml at 7 percent O_2 basis in the middle, and then those concentration values multiplied by the unit risks taken from Appendix V of the boilers and industrial furnaces (BIF) regulations at the bottom.
- # The dominant carcinogenic PIC factors in the cement kiln are polyaromatic hydrocarbons, and there is little difference when comparing baseline with HWF. The dominant factors in the incinerator PIC emissions are volatile chlorinated hydrocarbons, most of which were not found in cement kiln emissions.
- # If the concentration times unit risk factor is totaled, a comparison can be made of the relative carcinogenicity of a cubic meter of emissions from each device. On this basis, the Clarksville cement kiln had carcinogenic PIC emissions 50-100 times lower than the “well operated” incinerator. Even on a mass emission basis, the comparison is quite favorable.

The commenter believes that this data is very convincing in demonstrating that PIC emissions from cement kilns with high HC levels are not a significant health threat when compared with baseline

(no HWF) or a "well operated" incinerator. The commenter also included a copy of the original direct exposure risk analysis (performed in compliance with existing BIF regulations for trial bums as part of the BIF compliance test at Clarksville) for Clarksville.

Response 464: *It is important to note that in order to draw clear conclusions regarding the relative potential for PIC formation and release for different combustor categories, it is necessary to obtain emissions data from a representative sample of facilities from each combustor category (that is, having data from a single facility is not sufficient to draw conclusions regarding the performance of an entire combustor category). In addition, risks from PICs could not be assessed quantitatively in the HWC analysis due to limitations of the data available, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited, are highly variable, and are therefore inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.*

***Comment 618*:** The commenter points out that exposure scenarios tend to give a false picture of the amount of exposure expected because they include no information by which one can judge the likelihood that any particular scenario will be realized.

***Response 618**: The risk assessment for the final rule was modified to account for the number of individuals exposed and their location. The analysis employed U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions which explicitly account for the numbers of persons exposed at differing levels of exposure. From the cumulative risk distributions, individual risks were estimated at various percentiles, such as the 50th, 90th, and 99th percentiles.*

Comment 1068: It has appeared over the years that every time EPA comes out with a set of regulations or additional guidelines for hazardous waste burning cement kilns that cement kilns successfully comply with, a new and more stringent attempt is made. At one point there was the boiler and industrial furnace (BIF) regulations. When cement kilns complied successfully with the BIF regulations, then there came the health risk issue. When it appeared that most cement kilns were going to successfully pass certain basic screening levels of a risk assessment, then indirect risk assessment became an issue. Cement kilns were looking pretty good in this department as well, and now we have the proposed HWC rule. It was refreshing to read the following reviewer's comment: "Lack of field data that corroborate models' predictions is a major uncertainty in terrestrial food-chain models." It would appear that EPA is relying more and more on making things up in their attempts to make hazardous waste burning cement kilns out as the bad guys.

***Response 1068:** In the development of emerging methodologies, EPA must often accept the limitations of the data available for model validation. Although additional studies would help further refine the models, EPA believes there are currently sufficient evidence and data available to evaluate exposures from the surface water and terrestrial food chain and that it is important to assess the risks attributable to such exposures.*

Section 21
Holnam, Inc.
RCSP-0113

***Comment 343*:** The commenter states that the baseline air emission risks from cement kilns may even be less than what EPA estimates because of EPA's reliance on flawed risk information, including the use of flawed indirect risk assessments, and the much-criticized *Mercury Study Report to Congress* and dioxin reassessment. EPA has also not evaluated information from the State of Texas and Region VI that show no real risk from cement kilns operating under the BIF standards.

Response 343: *It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess risks associated with indirect exposure to emissions from HWC facilities as part of the RCRA permitting process. The indirect exposure methodology, which is used in the risk assessment for the HWC final rule, was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (*Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."*

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688). The draft Addendum was also reviewed by EPA's Science Advisory Board (SAB). EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to comments on these documents that have been received from the public, as well as from the SAB, in an appropriate forum. The 1997 MRTC was critically reviewed and strongly supported by SAB. EPA performed a risk assessment for the HWC rule to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control

remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 468*:** The commenter feels that EPA has failed to articulate why indirect risk assessment is used to support this rule but not other RCRA rules currently under development. Furthermore, the commenter believes that the use of indirect risk assessment here is particularly problematic in light of the harsh criticism EPA's indirect risk assessment methodology has received.

***Response 468**: EPA performs risk assessments for hazardous waste combustion that include indirect exposures because this has been EPA's policy since 1993, when EPA released its draft hazardous waste minimization and combustion strategy.*

On May 18, 1993, the EPA Administrator announced a series of steps that EPA was undertaking to ensure the safety and reliability of hazardous waste combustion. EPA's hazardous waste minimization and combustion strategy, announced in final form in November 1994, established the general policy that risk assessments, which include indirect exposure pathways, should be performed prior to final permit determinations for all hazardous waste combustion facilities. In order to evaluate whether the technology-based MACT standards for HWCs are protective of human health and the environment under RCRA EPA performed a risk assessment that assessed the risks from a variety of exposure pathways, including both direct and indirect exposures.

***Comment 471*:** The commenter states that current air emission risks from cement kilns may even be less than what EPA estimates because of EPA's reliance on flawed risk information. The commenter states that in evaluating risks from the two pollutants for which EPA is proposing BTF standards, EPA relied upon two documents that have received considerable criticism: the draft dioxin reassessment and the draft *Mercury Report to Congress*. The commenter indicates that both documents have been thoroughly criticized in peer reviews and returned to EPA for further work. The commenter notes that EPA relies on the draft dioxin reassessment throughout the rule to support EPA's decision to establish beyond the floor (BTF) standards and in evaluating the health benefits of dioxin reduction. The commenter states that this document is a draft document that has never been finalized and still is stamped "Review Draft: Do Not Cite or Quote." The commenter states that EPA's assessment has received considerable public attention and has generated significant controversy and, to address issues raised about the reassessment, the Science Advisory Board (SAB) conducted a thorough review of EPA's work and published a report in September 1995 in which the SAB recommended that EPA make several modifications to its report before publishing a final version.

***Response 471**: EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in*

Section 112(d)(2) and (3). Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control achieved in practice by sources using the same control technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). For the final rule, EPA is setting the emission standard for mercury at the MACT floor. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards.

However, EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job." EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

The risk assessment for the final rule also followed the same general modeling approach developed for the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model and the methylmercury reference dose developed especially for that report. The MRTC was critically reviewed by EPA's Science Advisory Board. Although the SAB had some concerns with the surface water modeling in the draft report (which EPA believes were addressed in the final report), the subcommittee felt that EPA's reference dose for methylmercury was well supported by the available data while recognizing that important human studies were still ongoing.*

***Comment 472*:** The commenter states that the draft dioxin reassessment relies on a linear model that does not take into account the fact that dioxin is nongenotoxic and is not an initiator of cancer. Because it is not a "complete carcinogen," a model that does not include a threshold

assessment is incomplete. SAB noted this problem and recommended that a possible threshold dose-response relationship not be ignored in assessing the possible carcinogenic nature of dioxin.

***Response 472**: In its review of the 1994 draft dioxin reassessment, the EPA Science Advisory Board (SAB) suggested that EPA consider alternatives to the linear nonthreshold model, allowing for minimal response at low environmental levels of exposure, and that would be consistent with the body of available health effects data. In response, EPA developed a mechanistic model for liver tumors in female rats using data from a 2-year feeding study in Sprague-Dawley rats (Kociba et al., 1978). EPA also summarized the results of simple empirical models that have been applied to other significant cancer findings in female Sprague-Dawley rats from the Kociba study and in Osborne-Mendel rats and B6C3F1-mice from the National Toxicology Program study (NTP, 1982). In addition, EPA applied simple empirical models to evaluate the shape of the dose-response curve for significant noncancer findings from other animal studies. EPA compared the shapes of the dose-response curves across all the studies and grouped them into those that appeared to be linear across the experimental findings and those that appeared to be nonlinear (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d). EPA found that a majority of the dose-response curves are consistent with linearity but that some are highly nonlinear, appearing to have a clearly defined threshold. However, most of the cancer findings (9 of 13, or 70 percent) exhibited response consistent with linearity in the observable range.*

Also, few of the mechanistic models that EPA identified in the scientific literature exhibited nonlinear dose-response in the observable region or predicted nonlinear dose-response in the low-dose (extrapolation) region. Results of the two-stage modeling of the Kociba et al. female rat liver tumor data incorporating dioxin-altered hepatic foci data to estimate mutation and growth parameters provide nearly the same low-dose estimates as the linearized multistage (LMS) model using only the tumor data. Unless a protective effect of TCDD on mutation rates occurs at low doses, low-dose risk will remain proportionate to exposure and consistent with the LMS model. If protective effects are allowed in the model, the low-dose risks may be substantially reduced; however, the focal lesion data and the biochemical markers generally agree and do not suggest a protective effect (U.S. EPA, 1997d).

Therefore, there appears to be no strong support for general nonlinearity for TCDD's effects in the range of the data studied and little support for extrapolation into a lower-dose range with a highly nonlinear model. To the contrary, EPA believes there are sufficient data suggesting that response is proportional to dose that, when considered together with the available human data, warrant concern that 2,3,7,8-TCDD will induce toxic effects in humans in the range of the experimental animal data. Furthermore, based

on a lack of data indicating an immediate and steep change in slope of the dose-response curve, EPA believes the possibility of response 1 to 2 orders of magnitude below this range must be considered.

***Comment 473*:** The commenter cites the SAB report as indicating that the use of a TEQ methodology, which EPA used in the reassessment and in this rule, has not been adequately confirmed as being accurate and as advising EPA to include a peer-reviewed appendix that will comprehensively review EPA's use of the TEF/TEQ approach in the exposure and health assessment documents. The commenter also states that SAB noted that new data had since been made available which would adjust several of the TEF values. The commenter indicates that much of this data was actually available at the time the reassessment was being conducted and would demonstrate a lower TEF values for several dioxins and furans. The commenter notes that this information and the additional criticisms by the SAB have not yet been addressed by EPA. The commenter feels, hence, that until the reassessment is made final, its risk information should not be used to justify more stringent standards for combustion devices.

***Response 473**: EPA used technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job." EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.*

EPA acknowledges that the SAB had a number of comments and concerns regarding the draft Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds *(U.S. EPA, 1994d,e) and agrees that there are uncertainties associated with the TEQ approach. Although the SAB called for clarifications in the specification of TEFs for the various dioxin-like compounds for various health outcomes, the SAB concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds.*

The TEFs used in the final rule are those recommended by the World Health Organization that were recently published in Environmental Health Perspectives *(Van den Berg et al., 1998). These values, which were made available to EPA prior to their publication, reflect a consensus from the international community. The TEFs are not based solely on acute studies, as implied by the commenter; rather, they were developed*

from all available mammalian, bird, and fish studies previously reviewed, as well as new studies published over the last several years. These studies were carefully evaluated and a database was developed using the following criteria:

- *At least one CDD, CDF, or PCB congener and a reference compound must be included in the study.*
- *Either TCDD or PCB 126 must be included as a reference compound in the same experiment or studied with the same experimental design by the same authors in another experiment.*
- *The relevant end point should be affected by the congener studied as well as the reference compound.*

The TEFs applicable to humans and mammals considered in vivo toxicity data, in vitro data, and quantitative structure-activity relationship (QSAR) data. In vivo data were given the highest priority; in fact, the TEFs applicable to humans were largely derived from in vivo data. The revised international TEFs reflect only limited changes to the 1989 interim TEFs. Most TEFs were not changed. Exceptions include OCDD and OCDF (reduced from 0.001 to 0.0001) and 1,2,3,7,8-PeCDD (raised from 0.5 to 1). EPA believes the revised international TEFs are the most appropriate values for use in the HWC final rule.

The WHO panel of experts also reviewed the uncertainties related to the TEQ approach. These included nonadditive interactions, differences in shapes of the dose-response curves, and differences in species' responsiveness. This panel concluded that the TEF model is both plausible and the most feasible approach for risk assessment of dioxin-like compounds. The panel also concluded that, in view of the available scientific evidence from studies of mixtures of dioxin-like compounds, use of the TEFs is not expected to result in large errors in estimating concentrations of TEQs or responses at environmentally relevant levels.

EPA did not use the TEF/TEQ methodology to assess any specific noncancer endpoints in the risk assessment for the final rule. Additional research is needed to gain a better understanding of the underlying molecular mechanisms responsible for several key noncancer effects (e.g., immunotoxicity, neurobehavioral toxicity, and female reproductive toxicity) in order to develop biologically based dose-response models that can be used for noncancer risk assessment. Instead, EPA used a modified margin of exposure (MOE) approach to compare predicted exposure levels against background levels. The MOE approach, which incorporates the TEF/TEQ methodology to assess exposures from all 2,3,7,8 chlorine substituted dioxins and furans, was not used to assess risk for any specific noncancer endpoint but rather as a general indicator of potential noncancer hazards from this class of compounds.

***Comment 474*:** The commenter questions EPA's use of the *Mercury Study Report to Congress*, given that the report has received significant criticism from the SAB. The commenter notes that, in particular, the SAB report identified problems with EPA's of the use of the Iraqi study on neurological impacts, since this study relied on data from children exposed in utero to bread contaminated with methylmercury. The commenter cites the SAB criticisms of a type of exposure not expected to be prevalent in the U.S., a short duration, and high exposure levels, all of which differ greatly from more common mercury exposures. The commenter indicates that the SAB report noted several other shortcomings and uncertainties in this study, including the reliability of the estimates of ingested mercury doses, the small population size, and the effects of reporting 30 months after birth. As has been pointed out by other commenters, a much more comprehensive study, the Seychelles Study of Fetal Methylmercury Exposure and Child Development, was available at the time the EPA study was released. This study included 800 mother-infant pairs and is likely to be a much more accurate study from which to derive an RfD.

***Response 474**: EPA is concerned about exposure to mercury from HWC emissions because mercury is a known neurological toxicant in humans. However, at the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), a study that includes quantitative modeling analyses of mercury exposures and that has been subject to extensive peer review. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

For the final rule, EPA continues to use the same RfD for methylmercury that was used at proposal (i.e., 1 x 10^{-4} mg/kg-d). This is the same RfD that was developed for EPA's MRTC. The 1997 MRTC was subject to extensive review, including review by EPA's Science Advisory Board (SAB). The SAB endorsed retention of EPA's RfD, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data. The SAB felt that the uncertainty factor used by EPA could even be increased, thus lowering the RfD, due to concerns about the acute nature of the exposure in the Iraqi study and at least some evidence that methylmercury has a longer half-life in the brain than in the blood.

At the time of the finalization of the MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and marine mammal–consuming populations in the Seychelles and Faeroe Islands. Because the majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to reevaluate the RfD for methylmercury.

*EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faeroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Holnam, Inc.
RCSP-0238

***Comment 621*:** In general, the commenter believes the risk assessment panel identified several key issues with EPA's analysis of risks in the proposed rule.

- **#** EPA's use of a case study approach does not provide an accurate assessment of risk. The panel concluded that EPA's case study approach does not deviate from the traditional reliance on worst-case default estimates and overestimates risk. Panelists viewed the case study approach as only being useful for a "screening analysis" and not as a reliable approach to make beyond-the-floor determinations.

- **#** The assumptions used to estimate the pathways for airborne emissions "may yield estimates greater than actual values in most cases."

- **#** The use of steady-state models for estimating risks from consumption of drinking water and risk also tend to overestimate the nature and extent of risk.

- **#** The panel also noted significant uncertainty in EPA's use of a terrestrial food chain model for assessing risks from consumption of meat, eggs, milk, and other food products.

Thus, the commenter feels that the summary report makes it clear that the panel members found several flaws in EPA's risk analysis, all of which lead to an overestimation of risk from hazardous waste combustion.

***Response 621**: EPA gave much consideration to the comments of the peer-review panel on the risk assessment for the proposed rule and made a number of changes to the risk assessment for the final rule as a result of the comments received from the peer reviewers. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for further details on EPA's response to specific issues raised by the peer reviewers.*

Section 22
Hunton & Williams for the Utility Air Regulatory Group
RCSP-0125

Comment 424: The commenter is concerned with certain statements made by EPA in the proposed rule regarding mercury in the environment and resulting health effects. The commenter notes that EPA is currently working on a study pursuant to Section 112(n)(2) of the CAA on mercury emissions from all sources and the health and environmental effects from such emissions, the results of which will be used in another study under Section 112(n)(1) of the health effects resulting from HAP emissions by electric utilities. The commenter notes that in the utility study, EPA is to determine whether regulation of HAP emissions from electric utilities is "appropriate and necessary." The commenter points out that to the extent EPA sets mercury standards in this rule on inaccurate or unsubstantiated assumptions about mercury, the electric utility industry could be affected if EPA were to use those same assumptions in its mercury study and utility study.

> ***Response 424****: EPA is concerned about exposure to mercury from HWC emissions because mercury is a known neurological toxicant in humans. However, at the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA, 1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

***Comment 0125-1*:** The commenter cites the preamble to the proposed rule and indicates that EPA makes several statements therein that are not supported by current scientific knowledge. The commenter states that EPA should delete or qualify these statements in its final rule as described in the following text.

The commenter notes that, in discussing mercury contamination in fish, EPA concludes that low levels of mercury in surface water can lead to high levels of mercury in fish, and that a reduction in mercury emissions therefore would be correlated with a subsequent reduction in mercury levels in fish over time. The commenter notes that these conclusions depend on simplistic assumptions that are not supported by current scientific knowledge of mercury cycling in the environment or bioaccumulation of mercury in fish. Specifically, EPA presumes a direct causal link between current mercury emissions and

fish contamination. EPA concludes that if emissions are reduced, levels of fish concentration will decrease. Yet, contrary to EPA's assumption, mercury emissions in this country and elsewhere have declined significantly since the 1950s, but there has been little or no corresponding decline in mercury levels in fish. These findings indicate the significance of natural sources of mercury and historical sediment concentrations of mercury. Current mercury emissions appear to contribute little to the overall mercury pool and, therefore, reductions in these emissions would have little effect on fish contamination.

The commenter notes also that EPA's use of a bioaccumulation factor (BAF) to draw conclusions about mercury concentrations in fish from mercury concentrations in surface water oversimplifies the way mercury reaches fish. Understanding mercury concentrations in fish requires understanding of a variety of chemical and biological factors that relate to the form of mercury in the water column, its bioavailability, and the portion of mercury that ends up in sediments. Research is ongoing to understand more about the cycling of mercury in the environmental and the relative contributions from natural, historic, and current anthropogenic sources of mercury levels in fish. EPA should delete the statements cited by the commenter given that they are not supported by, and are, in fact, contrary to, existing scientific knowledge about mercury.

The commenter points out that in its discussion of state fish advisories, EPA concludes that these advisories indicate the "severity" of mercury contamination in fish. A closer look at the bases for state advisories reveal that EPA's conclusion does not follow. Many state fish advisories are based on levels of contamination below the level the Food and Drug Administration (FDA) considers safe for the sale of fish in commerce. For example, Minnesota uses an action level for some of its fish advisories that is more than five times lower than the FDA level, while Michigan and Wisconsin use an action level one-half the federal level. Thus, state fish advisories, in themselves, do not indicate "severe" mercury contamination because many fish subject to state advisories would be considered safe for consumption by the FDA.

The commenter also notes that the proposed rule uses a mercury reference dose (RfD) of 0.01 µg/kg body weight/day to estimate potential adverse health effects from consuming fish. The commenter has previously criticized this RfD because it is based on 20-year-old data from an acute exposure incident. The acute nature of the exposures make them inappropriate for setting acceptable chronic exposure limits for low levels of methylmercury. Studies of low-level chronic exposure to mercury through fish consumption are being conducted in the Seychelles Islands and Faroe Islands. The data and results from these studies should be available within the next year. The commenter states that EPA notes in the preamble to the rule that it intends to reevaluate this RfD when the results from the Seychelles and Faroe studies are available. Because the current RfD is scientifically questionable, it is inappropriate for EPA to use it to estimate health effects from fish consumption.

Response 0125-1: *EPA is concerned about exposure to mercury from HWC emissions because mercury is a known human toxicant. In the HWC risk analysis, the concentrations of mercury species in the atmosphere were determined from emissions*

tests at the individual sites. Mercury was measured at the stack (point of emissions) as a fraction of divalent mercury (particle-bound) and elemental mercury (vapor phase only).

For mercury, the assessment followed the general modeling approach developed for the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model developed especially for that report. The IEM-2M model, which was used for modeling the fate and transport of mercury in surface water, is fully documented in the 1997 MRTC, which was critically reviewed and strongly supported by EPA's Science Advisory Board. IEM-2M is composed of two integrated modules that simulate mercury fate using mass balance equations describing watershed soils and a waterbody. The mass balances are performed for each mercury component, with internal transformation rates linking elemental, divalent, and methylmercury.*

Contemporary anthropogenic emissions of mercury are only one component of the global mercury cycle. Releases from human activities today are adding to the mercury reservoirs that already exist in land, water, and air, both naturally and as a result of previous human activities. Given the present understanding of the global mercury cycle, the flux of mercury from the atmosphere to land or water at any one location is made up of contributions from the natural global cycle, the global cycle perturbed by human activities, regional sources, and local sources. EPA acknowledges in the 1997 MRTC that although considerable uncertainty still exists, it has become increasingly evident that anthropogenic emissions of mercury to the air rival or exceed natural inputs. Recent estimates place the annual amounts of mercury released into the air by human activities at between 50 and 75 percent of the total yearly input to the atmosphere from all sources. Recycling of mercury at the earth's surface, especially from the oceans, extends the influence and active lifetime of anthropogenic mercury releases. EPA believes that this factor indicates a causal link between combustor emissions and fish tissue concentrations, even if the effects of limiting emissions are not immediately apparent.

Methylmercury concentrations in fish are derived from dissolved methylmercury water concentrations using bioaccumulation factors (BAF). EPA recognizes that the BAF contains a substantial level of uncertainty. The technical basis for the derivation of the BAF values, and a discussion of the associated uncertainty, is presented in Appendix D (Aquatic Bioaccumulation Factor Development and Analysis) of Volume III of the 1997 MRTC.

The 1997 MRTC indicates that, based on 1996 data compiled by U.S. EPA's Office of Water, advisories have been issued in 39 states that warn against the consumption of certain amounts and species of fish that are contaminated with mercury. Ten states have statewide advisories (i.e., advisories posted on every fresh waterbody in that state). These advisories are based on the results of sampling surveys that measure mercury

levels in representative fish species collected from waterbodies. The advisories are intended for people who catch and eat fish from those waterbodies. EPA feels confident in interpreting these advisories as indicators of a severe problem.

For the final rule, the RfD for methylmercury of 1 x 10^{-4} mg/kg-d cited in IRIS (U.S. EPA, 1998a) and EPA's 1997 MRTC was used. The final MRTC has been critically reviewed and approved by EPA's Science Advisory Board (SAB). Therefore, EPA has used the conclusions from this report in support of the final rule.

The SAB commented that "[b]asing the RfD on the Iraqi study is not ideal. The study involved an acute high level exposure from contaminated seed grain, rather than the chronic low-level exposure from fish consumption that is the principal risk the RfD is designed to protect against." However, the SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including the Cree Indian and New Zealand studies) as well as experimental animal data, and that the current RfD should be retained.

At the time of the finalization of the MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and marine mammal–consuming populations in the Seychelles and Faeroe Islands. Currently smaller-scale studies are being carried out around the U.S. Great Lakes. The SAB stated, "Because these data are so much more comprehensive and relevant to contemporary regulatory issues than the data heretofore available, once there has been adequate opportunity for peer review and debate within the scientific community, the RfD may need to be reassessed in terms of the most sensitive endpoints from these new studies." Because the majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to change the RfD for methylmercury.

*EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faeroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary*

for their full potential to be realized.

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Section 23
Indiana Department of Environmental Management
RCSP-0160

***Comment 374*:** The commenter asks if the Monte Carlo method was considered for use in determining the "high-end" and "central tendency" estimates of risk. Further, the commenter inquires if so, what was the reason for using the Monte Carlo method, and, if not, and considering the past EPA preference and use of the Monte Carlo method noted below, can EPA use the Monte Carlo method in determining these risks?

The commenter cites the following risk assessment guidance as using the Monte Carlo method:

- Habicht Memo (2-26-92)
- Guidelines for Exposure Assessment (57 FR 22,888)
- Science Advisory Board (Feb, 1992)

Also, it is used in the following rulemaking and implementation:

- Delisting Petitions Using Composite Model for Landfills (56 FR 67,197)
- RCRA TCLP Rulemaking (55 FR 11,798)
- Water Quality-Based Toxics Control (March 1991)

Finally, the Monte Carlo method is used in the Superfund Risk Assessment:

- Rohm & Haas Bristol Landfill (U.S. EPA CONTRACT 68-01-7250)

***Response 374**: For the final rule, EPA made a number of changes to the risk assessment to address commenters' concerns and to improve the representativeness of the analysis. In particular, EPA used central tendency exposure factors for estimating exposures, except for a few risk-driving pathways for which an exposure factor variability analysis using Monte Carlo simulation was performed. The Monte Carlo simulation allows a more complete characterization of the range of exposures for a population without the use of high end assumptions. The risk analysis completed for the final rule includes an exposure parameter variability analysis for key receptor population/exposure pathway combinations designed to reflect the impact of interindividual variability in exposure parameters on risk results. Specifically, an exposure parameter variability analysis was conducted for the commercial beef and dairy farmer receptors and for the recreational fisher (for the beef, milk, and fish ingestion pathways, respectively). The analysis used Monte Carlo simulation along with variability distributions for dietary intake, exposure duration, and the age-crossing correction factor (the last parameter corrects for*

differences in dietary intake rates as a modeled individuals age from one cohort to the next). The use of the exposure parameter variability analysis allows those individuals with elevated exposures due to higher than average ingestion rates or residence times to be reflected in the cumulative risk distributions used to characterize individual risk, without the use of conservative upper-bound exposure assumptions.

It is important to note that, for the commercial beef and dairy farmer receptors, which are both enumerated using U.S. Census and Census of Agriculture data, the exposure parameter variability analysis was integrated with sector-level population data in generating cumulative risk distributions. Consequently, specific risk percentiles established in the analysis for these receptors not only reflected the range of variability in exposure parameters across the population, they also reflected the distribution of individuals across the modeled sectors (i.e., their position and density relative to the modeled facilities).

Section 24
International Brotherhood of Boilermakers, Iron Ship Builders, Blacksmiths, Forgers, and Helpers
RCSP-0120

***Comment 525*:** The commenter states that EPA has inappropriately relied upon documents in formulating the HWC MACT proposal. The commenter notes that these documents—the Dioxin Exposure and Health Effects Reassessment; the Mercury Study Report to Congress; and the Indirect Multi Pathway Risk Assessment Methodology—have been the subject of sharp criticism from EPA's own Science Advisory Board and that EPA has withdrawn portions or all of these reports and has even insisted that they not be cited by commentators. The commenters think EPA's use of these documents is reversible legal error.

Response 525: *It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from hazardous waste combustion facilities as part of the RCRA permitting process. The indirect exposure methodology is the underlying methodology used in the risk assessment for the HWC final rule. The methodology was issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) and was updated in 1993 with the draft Addendum (U.S. EPA, 1993a). Since the draft Addendum was completed, scientific knowledge and understanding have continued to improve. For the risk assessment for the final rule, EPA updated the indirect exposure methodology based on information from the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), as well as other sources of information. EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."*

EPA solicited public comment on the indirect exposure methodology in 1993 (see 58 FR 61688) and the dioxin reassessment in 1994 (see 59 FR 46980-46982). The draft Addendum and the dioxin reassessment were also reviewed by EPA's Science Advisory Board. EPA intends to address the comments from the SAB on the 1993 Addendum to the indirect exposure methodology and the 1994 draft dioxin reassessment in the appropriate forum. EPA has responded to the SAB's concerns regarding the cancer slope factor for dioxin with Chapter 8 of the 1997 report Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds *(U.S. EPA, 1997d). In*

addition, EPA responded to the comments from the SAB review of the MRTC and finalized the report in 1997. Although the Addendum and dioxin reassessment are draft documents that have not been finalized, EPA believes that the technical information contained in them is appropriate for use in the HWC risk assessment. EPA acknowledges the uncertainty implicit in the use of the models to analyze complex physical and chemical processes. However, EPA believes the models represent the best analysis tools currently available. EPA has emphasized the nature and extent of the uncertainties in its characterization of risks for the final rule.

Section 25
Lehigh Portland Cement Company
RCSP-0033

Comment 597: *(Docket ID RCSP-0033 is a request for an extension of comment period. This is the only docket from Lehigh Portland Cement Company in RTI's database.)* The commenter notes that EPA continues to rely upon the risk characterization analysis present in the draft dioxin reassessment. The commenter states that the use of the reassessment may constitute legal error because of the severe and unaddressed criticisms the document received by the EPA Science Advisory Board and other competent sources.

Response 597: *EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. Sections 112 (a) and (d) of the Clean Air Act direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards.*

However, EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job."

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

Section 26
Lone Star Industries
RCSP-0232

***Comment 262*:** The commenter cites a peer-review comment that implicates EPA risk assessments as gross overestimates: "The guidelines in the HWC Emission Database (Volume II) to adopt detection limits for various congeners, when in most cases none will be emitted, may have an adverse impact on the final risk outcome for dioxin, particularly for the indirect exposure pathways."

***Response 262**: For the final rule, EPA assumed that dioxin and furan congeners that were below the limit of detection were present at one-half the detection limit. This represents a middle course that avoids the conservatism that could be introduced by assuming congeners are present at their full detection limit yet accounts for the likelihood that the congeners are, in fact, present but at levels below the detection limit of the measurements. Although the issue of compounds being present at levels below the limit of detection contributes to uncertainty, the uncertainty is sufficiently small so as not to have a material effect on the findings and conclusions of the risk assessment. In particular, individual risks associated with high end exposures are attributable to emissions of congeners that are present at levels well above the limit of detection rather than congeners that are below the limit.*

***Comment 263*:** The commenter strongly agrees with and emphasizes the peer reviewer's comment that "...most of EPA commonly forgets that 'Reference Dose (RFD),' 'Risk-specific Dose (RsD)', and related administrative values represent exposure that can be considered safe: such exposure, for a lifetime, poses no or a negligible risk to those so exposed. The product of an RFD and an estimate of exposure does not produce a 'risk' or predicted incidence of injury. EPA has defined the risk at the RFD as zero, so this product is identically zero. One can conclude, based on the product of an exposure times an RfD or RsD, that the exposure poses no risk and thus conclude that no further action is required. One cannot conclude that further reduction in exposure reduces risk."

***Response 263**: EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *that addresses the peer review comments (U.S. EPA, 1999b). The commenter is referred to this document for EPA's response to the issues raised by the peer reviewers. Risk-specific dose is the dose or intake level corresponding to a given level of risk (e.g., usually a hazard quotient [HQ] of 1 or an excess lifetime cancer risk of 1 in 1 million).*

EPA considers that the ingestion reference dose (RfD) represents an average daily ingestion exposure rate that is considered to pose no risk to the population. RfD values

are published in EPA's Integrated Risk Information System (IRIS, U.S. EPA, 1998a) database or in EPA's Health Effects Assessment Summary Tables (HEAST, U.S. EPA, 1997b). For the final rule, EPA relied on RfDs to quantitatively express risk as hazard quotients for the ingestion of noncarcinogenic constituents. For this analysis, the ratio of a single substance exposure level over a specified time period to a reference dose for that substance derived from a similar exposure period is the HQ. In the HWC risk assessment, modeled intake divided by the RfD is represented by the HQ. If HQs exceed 1 (e.g., the exposure level [intake] exceeds the RfD), there may be concern for potential noncancer effects. As a rule, the greater the value of HQ above unity, the greater the level of concern.

***Comment 620*:** The commenter emphasizes the peer reviewer's comment regarding the toxicology of hydrogen chloride that "[t]he toxicology of hydrogen chloride is well known: for instance, the mean lethal concentration in air is almost the same as hydrogen cyanide, and the toxic effects are precisely those to be expected from a strong acid condensing on tissue. Thus, I find it exceedingly surprising that EPA reports 'low confidence' in the very protective RfC that is quoted (page 120). It is stupidities like this that give EPA science its poor reputation."

***Response 620**: As noted in the previous response, EPA has prepared a separate response to comments document. The commenter is referred to this document for EPA's response to the issues raised by the peer reviewers.*

EPA is aware that humans exposed to hydrogen chloride experience acute effects that include coughing, choking, inflammation and ulceration of the respiratory tract, chest pain, and pulmonary edema. However, chronic toxicity data are limited. EPA established a reference concentration (RfC) for hydrogen chloride of 2.0×10^{-02} mg/m^3 based on a chronic rat inhalation study, which reported an increased incidence of hyperplasia of nasal mucosa as well as laryngeal-tracheal segments in the group exposed to hydrochloric acid (Sellakumar et al., 1985). EPA has low confidence in the chronic inhalation study on which the RfC was based because the study used only one dose and had limited toxicological 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 has low confidence in the RfC, although it was used to derive the noncancer risk results presented for the final rule.

Section 27
Molten Metal Technology, Inc.
RCSP-0136

***Comment 349*:** The commenter notes, for reasons not apparent in the risk assessment, high-end health and environmental risk evaluations were not conducted for certain pollutants and exposure scenarios, including inhalation of hydrogen chloride (HCl).

Response 349: *In the risk analysis for the proposed rule, EPA set one or more parameters at high-end values deterministically in order to characterize risks associated with high-end exposures. For HCl, emission rates were set to high-end values based on the 90th percentile of the distribution of stack gas concentrations. However, as a result of the comments received, EPA is no longer using that approach for the final rule.*

For the final rule, EPA modified the approach for characterizing the range of exposures from HWCs in a number of ways. First, EPA used stratified random sampling to increase the number of facilities analyzed in each combustor category. Second, EPA used U.S. Census and Census of Agriculture data to locate and enumerate exposed populations within 20 kilometers of HWCs. Third, EPA used central tendency exposure factors (e.g., mean values) in conjunction with facility-specific emissions estimates and other site-specific data to estimate exposures for these populations. These modifications allowed EPA to generate cumulative frequency distributions of risks to individuals living in the vicinity of HWCs and to more precisely characterize central tendency risks (i.e., at the 50th percentile) and risks at the high end of the distribution (i.e., the 90th, 95th, and 99th percentiles).

***Comment 350*:** The commenter feels that deficiencies in the risk assessment potentially result in significant underestimations of risk to human health and the environment. Consequently, the commenter feels that in its current form, EPA cannot rely upon the risk assessment to either discount the need to conduct a BTF analysis for particular pollutants, or select a BTF standard not reflective of available and aggressive source reduction/waste minimization opportunities and/or pollution control technologies.

The commenter feels that no compelling evidence is presented that the 11 facilities evaluated are representative of either the regulated universe itself or reasonable worst case conditions at facilities within the universe. The commenter notes that none of the incinerators evaluated are on-site facilities, which tend to be located in more heavily developed areas where other sources of emissions can contribute substantially to risks along the various exposure pathways. The commenter also notes that the location of the potential receptors and the nature of the terrain surrounding the 11 combustors are of particular concern. The commenter states that EPA attempted to use general geographic location as an

imprecise surrogate for land use, agricultural practices, and location of surface water bodies; consequently, most of the facilities evaluated are not located near people and/or subsistence farmers and fishers. The commenter points out that not one of the facilities evaluated is closer than 1 km to a water body potentially used by subsistence fishers, that although the closest subsistence farmer to any of the incinerators assessed is 1 km, and the closest subsistence farmer to half of the incinerators evaluated is 5 km. The commenter notes that the two LWAKs are 3 and 5 km from the closest subsistence farmers; at one of the five cement kilns evaluated, the closest subsistence farmer is 16 km from the kiln; and only one of four incinerators is located in an area considered even partly residential. Furthermore, the commenter notes that none of the five cement kilns are located in even partly residential areas; indeed, two of the five are in "forests."

The commenter notes that, in addition, EPA did not evaluate the proximity of the 11 combustion devices to schools, day care centers, recreational areas, or other locations where people regularly congregate, particularly children and other vulnerable populations. Furthermore, the commenter points out that despite EPA acknowledgment that the presence of complex terrain can have a substantial impact on risk results, the majority of the facilities evaluated are in "flat" terrain, and only 2 of 11 facilities are in complex terrain. The commenter notes that EPA eliminated facilities in complex terrain from the risk evaluation because of a lack of site-specific meteorological information reflecting those complex conditions, resulting in an underrepresentation of complex sites.

The commenter states that it is also unclear whether the two "complex" terrain facilities represent reasonable worst case terrain/exposure conditions in the regulated universe. As an example, the commenter notes that it is not certain how these two terrains compare to the topography surrounding the Waste Technologies Industries (WTI) incinerator in Ohio or other locations with complex terrain. In summary, the commenter feels that the 11 case studies around which the entire risk assessment is based may not be indicative of the potential risks posed by HWC emissions, because those case studies do not reflect high risk exposure/terrain scenarios.

> ***Response 350:*** *EPA acknowledges the concerns raised by the commenter about the representativeness of the example facilities used in the risk assessment for the proposed rule. Although EPA has not concluded that the facilities analyzed at proposal were not representative (with the exception of small on-site incinerators, which were not represented), EPA recognizes the difficulty of demonstrating the extent to which the facilities may or may not be representative. For this and other reasons, EPA decided to increase the number of facilities analyzed and to use stratified random sampling to select the facilities for analysis. Stratified random sampling allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe they are designed to represent (e.g., confidence intervals). Such statements could not be made using the case study approach. The use of stratified random sampling also allows risk results for a subset of facilities to be adjusted to represent an entire combustor category in a statistically meaningful manner based on their selection probabilities. For*

the final rule, sample sizes were established based on a criterion that there be a 90 percent probability of sampling at least one facility in the upper 10 percent of the risk distribution. This, combined with the facilities analyzed at proposal, resulted in a total sample size of 76 facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators), which represents over 40 percent of the HWC facilities covered by the rule. By evaluating a larger number of facilities, the risk analysis for the final rule has greatly increased the probability of capturing the range of site conditions (e.g., meteorology, terrain, land use, population density, waterbody types/conditions) occurring at HWC facilities located across the nation.

EPA recognizes the potential for those engaged in subsistence fishing and farming activities to experience heightened exposures through dietary pathways. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population and EPA believes it is important to evaluate the risks to those individuals. Separate exposure estimates were made for individuals engaged in farming or fishing for subsistence from individuals engaged in farming for commerce or recreational fishing. Exposures were estimated using dietary intakes reflective of subsistence activity (i.e., subsistence farmers were assumed to receive nearly all of their dietary intake from home-produced foods, and subsistence fishers were assumed to obtain a significant portion of their dietary intake from self-caught fish). Because site-specific information was not available, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA also assumed that subsistence fishing could take place at any of the bodies of water that were modeled in the risk analysis. It was not possible to identify and locate individual farms that may be engaged in subsistence farming from census data or to characterize the location and prevalence of subsistence fishing activity. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. EPA recognizes that the assumptions made in order to assess risks associated with subsistence activities may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

For characterizing risk associated with the ingestion of fish caught by both recreational fishers and subsistence fishers, the number and location of waterbodies selected for analysis reflected both the prevalence of waterbodies in a given study area as well as a goal of selecting those waterbodies that (a) are located in more heavily affected areas of the study area; (b) serve as sources of drinking water (the drinking water ingestion pathway was evaluated using concentrations modeled for these specific waterbodies);

and (c) are likely to experience recreational fishing activity (based on waterbody size and accessibility). While consideration of drinking water status and recreational activity status could result in the selection of waterbodies that are not in heavily affected portions of a study area, in general, waterbody selection favored areas likely to be more heavily affected by facility emissions (i.e., those areas in close proximity to the HWC facility). Consequently, the HWC risk analysis focused risk characterization on waterbodies that are relatively close to HWC facilities and likely to be more heavily affected by deposition.

For the final rule, EPA used a 16-sector template implemented within a GIS as the basis for site characterization. The 16-sector template provides enhanced spatial resolution in evaluating exposure for both human and ecological receptors. The GIS component allows digital spatial data to be used in support of both fate/transport modeling and exposure assessment. For example, GIRAS land-use data is used in support of USLE modeling of erosion loads to waterbodies, while U.S. Census and Census of Agriculture data is used to derive sector-level receptor population and livestock populations. Enumeration of receptor populations included establishing sector-level population counts for four age-groups for each receptor: ages 0-5, 6-11, 12-19, and over 19 years. While the risk analysis for the final rule did not identify the locations of schools and daycare centers, the generation of sector-level population counts for the age groups listed above should ensure that the distribution of younger individuals relative to the HWC facilities is reflected in the risk estimates for the final rule. EPA did not attempt to characterize the daily activity patterns of individuals, which would have been required to assess exposures that occur apart from an individual's place of residence. Instead, EPA assumed that exposures (e.g., inhalation, incidental soil ingestion) occur at the location of residence, as indicated by the census data.

For the final rule, site-specific terrain was evaluated as part of the modeling analysis whenever there was significant elevation of terrain (relative to stack height) close to the facility. In these cases, terrain elevations were explicitly considered for dispersion modeling. In all, 13 facilities were judged to be located in areas of complex terrain and modeled using site-specific terrain elevations. Therefore, EPA believes that the risk estimates for the final rule do reflect the influence of complex terrain on ground-level concentration.

***Comment 626*:** The commenter states that the list of contaminants assessed is substantially incomplete. For example, no products of incomplete combustion (PICs) other than dioxin were assessed, even though many PICs are both highly toxic and bioaccumulative (such as polycyclic aromatic hydrocarbons, or PAHs). The commenter notes that Table 1 of EPA's *Draft Technical Support Document for the HWC MACT Standards, Volume 2*, contains a long list of PICs and other organic contaminants that are emitted from HWCs. The commenter notes that these contaminants may

be released in significant concentrations, notwithstanding the operating standards in effect at some facilities that attempt to control PIC formation. For example, the commenter points out that two of the pollutants posing the greatest risks at Waste Technology Industries (WTI), according to EPA's recent risk assessment, are benzo(a)pyrene and hexachlorobenzene; however, neither of those contaminants were included in the risk assessment for the instant rulemaking. Moreover, the commenter points out that 143 nondioxin PICs were evaluated in the WTI risk assessment. Yet the peer-review panel for the risk assessment remained concerned about the 60 percent of organic emissions that still remained uncharacterized. According to the panel, "the large number of uncharacterized emissions increases the likelihood that one of the organics is a supercarcinogen"; therefore carcinogenicity testing of the actual emissions was suggested. The commenter notes that in the instant rulemaking, all the nondioxin PICs remain uncharacterized and unevaluated.

> ***Response 626:*** *The risk assessment for the final rule assessed risks from chlorinated dioxins and furans. However, risks from other organics that may be present as PICs could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. For example, while it is known that a variety of PICs are emitted from HWCs (including hexachlorobenzene, benzo(a)pyrene, and other polyaromatic hydrocarbons, as indicated by the commenter), unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited and the data are highly variable, so they are inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.*

***Comment 352*:** The commenter notes that emissions of several important metals were not evaluated as part of the risk assessment. As an example, the commenter notes that while EPA recently ranked copper as the fourth most toxic metal when destined for combustion, based on the metal's persistence, bioaccumulation potential, and ecologic toxicity, copper emissions were not evaluated in the risk assessment. The commenter notes that other metals not evaluated (and for which the European Union sets emissions standards in its hazardous waste incinerator directive) include cobalt, tin, vanadium, and manganese.

The commenter notes that for each metal dust or fume there are specific health effects documented for employee exposure, and the commenter summarizes these results as follows. Cobalt can cause hard metal disease which is characterized by pulmonary problems, asthma, and dermatitis. Cobalt has been found to be carcinogenic in animal testing, and is a suspected human carcinogen. Manganese exposures affect central nervous system function. Employees exposed to manganese show sign of sleepiness, weakness in the legs, uncontrollable laughter, and difficulty walking. Manganese exposures can cause degenerative brain changes, loss of motor activity, and muscle weakness. The dust is a skin and eye irritant. There have been cases showing a high incidence of pneumonia in workers exposed to manganese dust and fume. Elemental tin and inorganic tin compounds have low toxicity. However, organic tin compounds are suspected carcinogens. Vanadium metal is non-toxic. However, vanadium is usually found in dust form as vanadium pentoxide (V205). Vanadium pentoxide dust and fumes are highly toxic and suspected as being carcinogenic. V205 is a poison by inhalation and ingestion as the routes of entry. It causes systemic effects to the respiratory system via inhalation and gastrointestinal disorders if ingested.

The commenter also noted that no products of incomplete combustion (PICs) other than dioxin were assessed, even though many PICs are both highly toxic and bioaccumulative (such as PAHs). In summary, the commenter states that many contaminants potentially posing significant risks to human health and the environment from the combustion of hazardous waste were not evaluated in EPA's risk assessment. The commenter feels that given the nature and extent of the contaminants omitted from the risk assessment, EPA cannot simply conclude an absence of data means an absence of risk.

Response 352: *In the risk analysis for the final rule, EPA evaluated risks associated with all constituents for which adequate emissions data were available. These included the following: antimony, arsenic, barium, beryllium, cadmium, chromium (III), chromium (VI), cobalt, copper, lead, manganese, elemental mercury, divalent mercury, methylmercury, nickel, selenium, silver, and thallium. Adequate emissions data were not available for zinc, tin, and vanadium, so risks associated with exposure to these metals were not evaluated. EPA also assessed the risks from hydrogen chloride and chlorine. In addition, risks from exposure to $PM_{2.5}$ and PM_{10} emissions were evaluated.*

Although EPA assessed the risks from chlorinated dioxins and furans, risks from other organics that may be present as products of incomplete combustion (PICs) could not be assessed due to limitations of the data available for analysis, as explained in response to the previous comment. However, EPA expects that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

For the final rule, EPA evaluated human health risks resulting from HWC emissions within a 20 km radius surrounding each facility. This risk analysis targeted risks to individuals residing within the study areas, rather than risks to facility workers from occupational exposures.

Comment 354: The commenter notes that the risk assessment did not evaluate the risks posed by emissions arising from upset or abnormal conditions. The commenter points out that such conditions may cause substantially higher PIC and other emissions than observed during routine operations and that in EPA's draft Waste Technology Industries (WTI) risk assessment, EPA evaluated a variety of onsite and offsite accident scenarios. The commenter notes, however, that the peer-review panel for the risk assessment recommended that EPA "adjust the estimate of normal PIC emissions to reflect emissions during abnormal operations (i.e., based upon the percentage of operation time during which emission violations or automatic waste feed shutoffs occur)." The commenter states that the panel also recommended expanding the accident analysis to include additional scenarios, such as pressurized jet releases resulting in aerosol formation due to mixing of chemicals or a fire. The lack of an accident analysis in the ecological risk assessment was also noted. Accordingly, since EPA's risk assessment for the instant rulemaking does not take into account abnormal operating conditions, the commenter believes that the results do not reflect the potential risks posed by real-world operating circumstances. Significantly, EPA's proposed rule would not require reporting of "excessive" automatic waste feed cutoffs associated with violations of operating limits or monitored emission standards until the number of events reached 10 during a 60-day period. See 61 FR 17440 (April 19, 1996). Clearly, abnormal operating conditions are not necessarily very infrequent.

Response 354: *The risk analysis for the HWC rule was designed to answer public policy questions concerning the MACT emission standards for HWCs. Such standards are established pursuant to the provisions of Section 112(d)(2) and (3) of the CAA and are not specifically designed to limit emissions under extreme operational conditions of the sort that could pose a significant risk from acute exposures. Under the general provisions of the MACT program, facilities are required to address startup, shutdown, and malfunction or upset conditions. Additionally, HWCs are already required to stop feeding hazardous wastes during process upsets (referred to as automatic waste feed cutoffs). While the MACT standards may lead to improvements in the air pollution control equipment required to meet the standards during hazardous waste operations, the rule would not specifically control emissions during periods when no hazardous waste is in the combustion chamber. Any air emissions during such times should be of relatively short duration and should be no greater than from any other combustion device burning a nonhazardous waste fuel. However, if process upsets are of such a magnitude or frequency to generate health and safety concerns, permit writers may, on a case-by-case basis, establish additional permit conditions as may be necessary to protect human health and the environment under the omnibus provisions of section 3005(c)(3) of RCRA.*

With regard to accidents, although an accident analysis was done at WTI to address community concerns, the MACT rule will not impact the frequency or magnitude of industrial accidents that might occur either during hazardous waste operations or other times. It would not be proper for EPA to consider the risk of such accidents occurring under the provisions of Section 112 of the CAA, nor did EPA consider such risks as a factor in setting the level of the MACT standards for HWCs.

***Comment 357*:** The commenter states that the risk assessment fails to consider cumulative risks posed by combustion emissions in several significant ways. The commenter notes that while risk estimates are summed across pathways for each chemical, the risks are not summed across chemicals for carcinogens generally, or for non-carcinogens where the chemicals target the same organ. The commenter feels that this failure to sum risks across constituents is inconsistent with other RCRA risk assessment policies, including policies governing hazardous waste listing determinations.

Response 357: *The commenter is raising the issue of risks from chemical mixtures. EPA considered the risks from chemical mixtures by assuming additivity of response by route of exposure. Specifically, chemical-specific cancer risks were summed for oral (ingestion) exposures and for inhalation exposures. Similarly, noncancer risks (hazard quotients) were summed for oral exposures and for inhalation exposures. EPA views this as a conservative yet reasonable approach to assessing mixture effects because it considers possible portal of entry effects but not effects on specific organs or mechanisms of action. EPA notes, however, that for the purpose of assessing population level cancer risks (e.g., excess cancer incidence), EPA conservatively assumed that effects were additive across exposure routes (i.e., cancer risks from inhalation and ingestion exposures were summed).*

Other categories of individual-level and population-level risks are constituent-specific (e.g., individual PbB analysis for lead, margin of exposure analysis relative to background exposures for dioxins/furans and the PM analysis), so the issue of additive risks generally does not apply. EPA notes, however, that in assessing risks from dioxins and furans, EPA used the toxicity equivalence (TEQ) concept, which assumes additivity of response among the various 2,3,7,8-substituted congeners.

The final rule does not include a comprehensive analysis of target organ effects for individual constituents. Instead, except as noted above, it was assumed that risks were additive separately across each route of exposure, reflecting concerns for portal-of-entry effects. It is also important to note that synergistic and antagonistic interactions between constituents were not specifically evaluated. EPA acknowledges the uncertainties related to the possibility of nonadditive, synergistic, or antagonistic effects between chemicals. However, at the present time, synergistic and antagonistic interactions have not been sufficiently characterized to allow consideration of these effects quantitatively for risk

characterization purposes. EPA believes that the additive risks generated for the final rule provide an adequate level of analysis for assessing the potential cumulative effect of multiple chemical exposures. EPA notes that cancer risks from HWCs generally appear to be dominated by risks from dioxins and furans. Therefore, the issue of multiple chemical exposures and chemical interactions with other classes of chemical compounds may be of lesser significance than it might otherwise be.

***Comment 365*:** The commenter notes that exposure to workers was not assessed, despite the fact that much of the combusted hazardous waste is managed onsite, and many onsite combustion units are part of large manufacturing operations, thus emissions from combustion units may affect many thousands of people. The commenter cites the recent HWIR rulemaking (60 FR 66365, December 21, 1995), in which onsite workers were evaluated in the supporting risk assessment for the inhalation and dermal contact with contaminated soil pathways. The commenter states that the existing risk assessment does not account for these onsite exposures, particularly given the surrounding land use patterns in the 11 case studies.

Response 365: *EPA is revising standards for hazardous waste combustion facilities under the Clean Air Act to limit emissions of HAPs that could adversely affect the general public. The revised standards are technology-based standards being promulgated under the authority of Section 112 of the CAA. EPA's express intent is to minimize duplication in regulations and regulatory actions. Accordingly, the MACT standards for incinerators, cement kilns, and lightweight aggregate kilns that were developed under CAA authority replace the existing RCRA emission standards for these sources. EPA believes it would be inappropriate to promulgate standards that would be duplicative with respect to existing standards for the health and safety of industrial workers promulgated under the authority of the Occupational Safety and Health Act (OSHA).*

EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by sections 3004(a) and (q) of RCRA, in order to determine what, if any, RCRA standards for emissions from these sources may be needed. For this reason, EPA evaluated human health risks resulting from HWC emissions within a 20 km radius surrounding each facility. This risk analysis targeted risks to individuals residing within the study areas, rather than facility workers exposed occupationally, for the reasons stated.

***Comment 367*:** The commenter believes that there is a lack of justification for selection of high-end values. The commenter notes that EPA's high-end risk estimates for the proposed emission standards were derived by adjusting one or two exposure parameters from central tendency to so-called 90 percent high-end values. The commenter feels, however, that since EPA did not conduct a sensitivity analysis to determine which exposure parameters were of principal importance, it is far from clear EPA adjusted the appropriate parameters to produce a real high-end analysis. The commenter

illustrates this point with the following example. EPA's adult subsistence fisher consumption rate in the risk assessment is only 60 g/d, while the high-end value associated with recreational fishers is 140 g/day, and the subsistence rate is substantially higher. However, EPA did not run a sensitivity analysis to determine whether adjusting this exposure parameter would produce significant high-end risks to human health.

Response 367: *For the final rule, EPA used central tendency values, rather than high-end values, for estimating exposures. As explained in the risk assessment Background Document (RTI, 1999), EPA generated population-weighted cumulative distributions of risks to individuals, thereby eliminating the need to define central tendency and high-end exposure scenarios deterministically, in the manner done at proposal. EPA used central tendency exposure factors to estimate exposures, including fish and other food intake rates, except for a few risk-driving pathways, for which EPA performed an exposure factor variability analysis using Monte Carlo simulation. Monte Carlo simulation allows for a more complete characterization of the range of exposures for a population without use of overly conservative upper-bound parameters. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative.*

For the final rule, an exposure parameter variability analysis was conducted for certain key exposure pathways in order to refine the estimates of exposures to account for interindividual variability in behavior linked to exposure. The exposure parameter variability analysis was conducted for the consumption of commercially raised beef, milk, and recreationally caught fish. Risk from these exposure pathways were identified at proposal as potentially experiencing the highest risk levels as a result of HWC emissions. The exposure parameter variability analysis included consideration of interindividual variability in exposure duration, ingestion rates, and age-crossing adjustment factors (the latter factor was used to adjust intake rates to reflect the aging of cohorts from one modeled age group to the next because intake rates varied between different age cohorts).

Although subsistence receptors including subsistence farmers and fishers could experience risks higher than commercial beef and dairy farmers and recreational fishers, it was not possible to characterize the distribution of subsistence activity across modeled HWC study areas, so risk estimates for these receptor populations are especially uncertain. For this reason, an exposure parameter variability analysis was not performed for subsistence receptors. Instead, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA also assumed that subsistence fishing could take place at any of the bodies of water that were modeled in the risk analysis.

For the final rule, EPA also used U.S. Census and Census of Agriculture data to characterize the location and density of receptor populations relative to each modeled HWC facility. These population data were then used to population-weight the sector-level risk estimates that form the basis for the cumulative risk distributions used to characterize risk in the analysis. In this way, the distribution of receptor populations across modeled study areas could be reflected in both central tendency and upper-end risk estimates.

Regarding the specific fish ingestion rates mentioned by the commenter, for the final rule exposures through the fish ingestion pathway were considered for the following receptors: the recreational fisher, the subsistence fisher, and the subsistence farmer. For the adult subsistence farmer and the adult recreational fisher, the final rule assumes a fish ingestion rate of 0.16 g/kg-day based on data on the consumption of freshwater by recreational anglers. Data on consumption of fish by subsistence farmers who raise fish in farm ponds are not available. Assuming an adult body weight of 71.8 kg, this corresponds to a daily ingestion rate of 11.5 g/day. For the adult subsistence fisher, an ingestion rate of 0.97 g/kg-day (equivalent to 69.6 g/day) was used based on data for Native Americans, who have a diet high in fish. Ingestion rates for the subsistence fisher and recreational fisher/subsistence farmer, both of which represent central tendency (mean) ingestion rates, were derived from EPA's 1997 Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a).*

***Comment 370*:** The commenter notes that EPA assumed in the risk assessment that no hazardous waste combustion facility would operate for longer than 30 years, thus EPA used 30 years as the maximum exposure duration for inhaling contaminated air. The commenter states, however, that no evidence is presented to support this assumption, and that the assumption is particularly suspect in the case of cement kilns and LWAKs, where the units are principally functioning to perform an economic activity other than waste management, and many of the existing facilities exceed 30 years of age. The commenter cites the Portland Cement Association as indicating that the majority of the cement plants covered by this rulemaking include one or more kilns over 30 years of age. Specifically, at least 15 of the 26 plants in the EPA database began operations or last modernized in 1966 or before. One plant, Lafarge at Fredonia, Kansas, began operations or last modernized in l921. Clearly, since these plants remain in operation today, cement plants routinely operate for much longer than 30 years and may elect to burn hazardous waste as part of such operations. The commenter points out that these data likely understate the age of the plants, since they indicate the later of the startup or modernization dates. For risk assessment purposes, the relevant date is initial startup, since this date better reflects the duration of the active life of the facility.

Response 370: *For the final rule, EPA assumed a 30-year facility lifetime, the same value as used at proposal. EPA recognizes that specific facilities may well have operational lifetimes in excess of the 30 years or may have lifetimes less than 30 years*

and that this is a source of uncertainty in the risk analysis. However, several factors mitigate against an over- or underestimation of exposures and risk stemming from the uncertainty surrounding a given facility's operating life. These include the residence times of exposed populations, the sensitivity of media concentrations to the operational period, and the risk descriptors used to characterize the risks. The role of these factors is discussed below.

The duration of exposure to a contaminant is directly related to the period of time an individual resides at a given site and the individual cancer risk associated with exposures that occur over that time period. This period of time is often considerably shorter than the operating life of a facility. Information on population mobility and residence time indicate that for most sectors of the U.S. population, the likelihood of anyone residing at the same location for an average life expectancy of 70 years is relatively small. For example, Census Bureau data show that over half of all households have lived in their current residence for 9 years or less, and only 1 percent have lived in their current residence for more than 50 years. Approximately 90 percent have lived in the same residence for 30 years or less. Therefore, the great majority of individuals' exposures will be less than 30 years in duration.

For the final rule, EPA relied on data reported in the 1997 Exposure Factors Handbook *(U.S. EPA, 1997a) on total time in the same residence to determine the duration of exposure. EPA used residence times of 6.5 to 13.5 years for nonfarm households and 6.5 to 17.3 years for farm households, depending on an individual's age. These estimates were taken from a study that used a mathematical model to simulate the probability over time that an individual would either move away or die. The model was based on survey data obtained from the Census Bureau and vital statistics data from the National Center for Health Statistics. EPA selected mean values from the distribution of total residence time for each age group. In addition, EPA fitted a distribution to data on total residence time specific to farm households for the purpose of assessing risks to persons living in farm households that are exposed to dioxins through consumption of home-produced beef and milk, as explained in the risk assessment background document. From the fitted distribution, it was estimated that 50 percent of adult farmers have a total residence time of 9 years or less, 75 percent a residence time of 19 years or less, 90 percent a residence time of 40 years or less, and 95 percent a residence time of 62 years or less. The same percentiles (i.e., 50th, 75th, 90th, and 95th) for farm children ages 0-5 were estimated to be 3, 7, 15, and 23 years, respectively.*

With respect to media concentrations, chemical constituents can accumulate in specific media (e.g., surface soil and sediment) over the operational lifetime of a facility, resulting in more highly elevated levels in later years of operation. EPA recognizes this fact and consequently has based media concentrations used for estimating exposure for assessing

both cancer and noncancer risk that are drawn from the tail end of the 30-year facility lifetime. Specifically, for estimating the average daily dose (ADD) used to assess noncancer risks, EPA estimated concentrations in soils and surface water that coincide with year 30 of the facility operating period. For estimating the lifetime average daily dose (LADD) used to assess cancer risks, EPA averaged the concentrations over the final years of a facility's operations for a length of time that corresponded to the assumed duration of exposure. For chemical constituents that quickly reach steady state concentrations, media concentrations are insensitive to the assumed 30-year operating lifetime. However, for chemical constituents that accumulate slowly in surface soils or large bodies of water over relatively long periods without reaching steady state (such as dioxins and mercury), this approach ensures that relatively higher concentrations for these media will be used for estimating exposures. However, it is important to recognize that several of the more important exposure pathways evaluated for the HWC risk analysis are primarily dependent on air concentrations that are not linked to the operational lifetime of a facility. These key exposure pathways include inhalation for all chemical constituents as well as indirect exposure pathways for dioxins/furans that have bioaccumulated in beef and milk. The bioconcentration of dioxins/furans in beef and milk results from their uptake into feed crops, which is primarily the result of direct vapor uptake by the plants (soil-to-root uptake is not a significant source of contamination). Mercury uptake into fish, another important exposure pathway in the analysis, is sensitive to the build up and accumulation of mercury in surface water over time. However, EPA believes that the use of fish concentrations drawn from year 30 avoids significant underestimation of mercury exposures in most cases and may overestimate exposures for some facilities that operate for a shorter period of time.

With regard to risk descriptors, EPA developed annual incidence risk estimates, rather than risk estimates over the entire life of the facility (e.g., annual excess cancer incidence, rather than the total number of statistical cancer cases over the life of the facility), at the national level for use in assessing population level risks and quantifying the associated economic benefits. Annual incidence estimates were made for cancer risks from dioxins and furans and all carcinogenic metals, as well as for risks from lead and PM. These estimates are not directly dependent on the operating life of HWC facilities and can be projected out into the future indefinitely as long as hazardous wastes are burned at current levels. Consequently, with regard to population-level risk, the question of facility operational life is not a significant issue, except as it may impact media concentrations as already discussed.

***Comment 1090*:** The commenter feels that the ecological risk portion of the assessment is extremely incomplete, since it consisted entirely of comparing estimated surface water concentrations to chronic ambient water quality criteria. The commenter points out that no assessment of impacts to terrestrial organisms were conducted, animal or plant, endangered or otherwise and that, in contrast,

the risk assessment supporting EPA's HWIR rulemaking evaluated the potential effects of waste releases on sediment-dwelling organisms, mammals, birds, soil fauna, and terrestrial plants. The commenter points out that, similarly, the draft Waste Technology Industries (WTI) risk assessment evaluated impacts to a variety of terrestrial plants and animals, and birds. The commenter indicates that no rationale is offered for the limited scope of the instant rulemaking. The commenter points out that, interestingly, the HWIR effort was criticized by the Science Advisory Board for the arbitrary exclusion of chemicals evaluated, not the breadth of receptors considered.

Response 1090: *EPA conducted a screening-level ecological assessment for the final rule that goes beyond the use of ambient water quality criteria. In this approach, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water. These criteria were then compared to model-predicted media concentrations in order to assess the potential for ecological risk. The ecological assessment evaluated all chemical contaminants that were assessed in the human health assessment. This includes dioxins and furans, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, lead, manganese, mercury, nickel, selenium, silver, and thallium. In addition, an ecological assessment was performed for copper. A somewhat more detailed analysis was done for aquatic mammals and birds for evaluating ecological risks from dioxins and furans. In this analysis, fish intake rates and congener-specific concentrations in fish were used, together with mammalian and avian toxicity equivalence factors (TEFs) to estimate a TCDD-TEQ dose to these ecological receptors for comparison with ecotoxicological benchmarks for 2,3,7,8-TCDD.*

EPA agrees with the commenter that an ecological risk assessment should consider a broad suite of ecological receptors because ecological receptors have varying levels of intrinsic value to ecosystems. Because the risk assessment must be appropriate for a national rulemaking, EPA selected a suite of ecological receptors that represented major trophic elements of ecosystems into which constituents may be released based upon (1) spatial distribution of chemical stressors in relation to the receptor and (2) the availability of data with which to assess the risks to that receptor.

EPA evaluated ecological impacts on freshwater mammals (e.g., mink, river otter) and birds (e.g., bald eagle, osprey, great blue heron), terrestrial mammals (e.g., short-tailed shrew and white tailed deer) and birds (e.g., red-tailed hawk, and American woodcock), the benthic community, the soil community (nematodes, soil mites, springtails, annelids, arthropods, molluscs), the plant community, the freshwater community (fish, aquatic invertebrates), and algae and aquatic plants. The ecological risk analysis considered impacts to selected representative ecological receptors that were likely to inhabit ecosystems surrounding HWCs.

Comment 372: The commenter notes that the risk analysis failed to consider that direct deposition of hydrophobic compounds is not immediately mixed through the water column, but remains concentrated in surface microlayers that would affect the exposure concentrations experienced by biota.

Response 372: *In terms of surface deposition, EPA recognizes that hydrophobic compounds will, depending on environmental conditions, tend to concentrate in surface microlayers. As a result, the actual concentrations to which various aquatic biota are exposed (either directly or indirectly) may differ from or be more variable than the average concentration in the water column. However, the extent of contaminant mixing in surface water is a function of meteorological conditions as well as the site-specific characteristics of the waterbody itself (e.g., flow rate).*

In the case of dioxins, the issue of aquatic microlayers is somewhat minimized because the primary mechanism of transport to waterbodies following stack release is through particulate deposition and not vapor deposition. Once particulates containing sorbed dioxin are deposited to waterbodies, they can mix within the water column as suspended sediment and ultimately settle out, contributing to dioxin loads to benthic sediment. Because a significant portion of the dioxin transported to waterbodies following HWC stack release may remain sorbed to particulates within the waterbody (either as suspended or benthic sediment), the amount of dioxin that is available for mixing within the surface microlayer may be significantly reduced. It is also important to note that the BSAFs used in projecting fish tissue concentrations which relate the concentration of dioxin in sediment to the concentration of dioxin in fish tissue, are empirically derived. Because they are empirically derived, BSAFs implicitly account to a certain extent for the complexities of dioxin fate/transport within the waterbody including such phenomenon as microlayer mixing. Consequently, while some uncertainty is introduced into the analysis by not explicitly accounting for the concentration of lipophilic compounds such as dioxin in the surface microlayer of waterbodies, EPA does not believe that this represents a significant source of uncertainty for the analysis.

***Comment 375*:** The commenter points out that EPA's risk assessment uses 55 percent vapor for atmospheric dioxins/furans based upon an equilibrium calculation for the partitioning between the vapor phase and the absorbed phase of TCDDs. However, measured data discussed in EPA's dioxin reassessment yield vapor/particle (V/P) ratios from 2 to 10 times higher than those calculated assuming equilibrium partitioning. The commenter feels that this discrepancy indicates the assumption of equilibrium behavior may not be valid. The commenter believes that since nonequilibrium conditions appear to dominate in dioxin/furan emissions, a higher percentage of vapor phase dioxins/furans should be used in the risk assessment for the instant rulemaking. The commenter points out that increasing the vapor fraction would ensure protection of human health and the environment because dioxins/furans in

the vapor form have been demonstrated to enter the food chain more rapidly than emissions in particulate form due to higher depositional velocities and more efficient uptake by vegetative matter. The commenter also notes that EPA's risk assessment uses a deposition rate that may be too small by up to a factor of five, given that previous research (Sehmel, "Particle and Dry Deposition: A Review," *Atmospheric Environment* 14: 983-1011) indicates a vapor deposition rate of 1 cm/s is appropriate.

Response 375: *For the final rule, EPA used the same vapor-particle partitioning factors that were used in the risk assessment for the proposed rule. Vapor-particle partitioning was varied among congeners, ranging from as high as 0.7 for 2,3,7,8-TCDF to as low as 0.0002 for OCDD. The value of 0.55 percent is the value used for 2,3,7,8-TCDD. These values were calculated according to the methods outlined in the site-specific assessment procedures developed for the dioxin reassessment (Volume III of* Estimating Exposure to Dioxin-Like Compounds*, External Review Draft, June 1994, U.S. EPA, 1994b). The method accounts for both the effect of ambient temperature and the amount of particulate matter surface area available for sorption, as well as the physical-chemical properties of the various congeners.*

With regard to the ambient measurements referred to by the commenter, Volume II of the dioxin exposure document clearly states that the ambient monitoring methods have certain limitations that may prevent obtaining a true measurement of vapor/particle partitioning in the ambient air. First, the glass fiber filter is designed to capture and retain particulate matter greater than or equal to 0.1 µg. Particles with diameters smaller than this may pass through the filter and be retained in the polyurethane foam vapor trap downstream. If this is the case, the amount of particle-bound constituent would be underestimated and the amount of vapor-bound constituent would be overestimated. Second, the relatively high volume of sampled air passing through the system may redistribute the more volatile congeners from the filter to the adsorbent trap by a process known as "blow-off." Again, this would lead to an overestimate of the fraction in the vapor phase.

Volume III of the dioxin exposure document recommends that until sampling methods are improved and modified such that they give results that indicate the true vapor/particle ratio of dioxins/furans in ambient air, the theoretical vapor-particle partitioning model described by Bidleman (1988) be used to calculate the V/P ratio for purposes of air dispersion and deposition modeling. This is the approach taken in the risk assessment for the final rule.

For estimating dry vapor deposition to soils, EPA used a deposition velocity of 0.2 cm/sec for dioxins/furans in the risk assessment for the final rule. This is the same value that was used at proposal and is the value reported by Koester and Hites (1992) from field measurements. EPA acknowledges that there is considerable uncertainty

regarding the use of this value for specific application to vapors but chose to use it because it is based on direct measurements of deposition of dioxins and furans. The Sehmel paper referred to by the commenter (1980, "Particle and Dry Deposition: A Review*,") summarized the values of particle and gas dry deposition velocities based on the published literature prior to 1980. The Sehmel paper offers no data on the specific gaseous dry deposition velocity for dioxins/furans. Instead it notes that a deposition velocity of 1 cm/s is sometimes assumed in lieu of data. However, as pointed out by the author, dry deposition velocities for gases range over four orders of magnitude. Therefore, EPA believes that it would be inappropriate to this value in the HWC risk analysis.*

Comment 377: The commenter states that even though EPA acknowledges breast-fed infants are "among the most highly exposed and most susceptible human populations," EPA used an extremely low breast milk ingestion rate when evaluating whether dioxin measures through the breast milk would exceed background concentrations. The commenter notes that in the instant risk assessment, EPA used 0.8 g/d as the infant breast milk ingestion rate, while the Waste Technology Industries (WTI) risk assessment used central and high-end values of 700 and 850 g/d. Moreover, EPA's *Exposure Factors Handbook* recommends ingestion rates over three orders of magnitude higher than the 0.8 g/d value. The commenter feels that the low ingestion rate used may explain why the risk assessment indicated even baseline breast milk exposures do not exceed background levels. The commenter feels that the same deficiency applies to the 0.35 g/d child fish consumption rate used in the risk assessment.

Response 377: *The value cited by the commenter of 0.8 g/d for breast milk ingestion is a typographical error. As indicated in Appendix C of the risk assessment background information document for the proposed rule (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document*, February 1996) the value EPA used was 0.8 kg/d (800 g/d). For the final rule, EPA based the ingestion rate upon data provided in its 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a). EPA assumed that 0.742 L of breast milk were ingested per day. Assuming a density of 1.03 mg/mL, the ingestion rate is approximately equal to 0.76 kg/day (760 g/day), a value that is consistent with the values of 700 and 850 g/d cited by the commenter from the WTI risk assessment.*

The value cited by the commenter for the child fish ingestion rate (0.35 g/day) is a relatively low value because it represents the amount of freshwater fish consumed in the general population. For the final rule, EPA assessed risks to the subpopulation of children that live in households that consume recreationally caught fish. Fish consumption for these individuals are much higher than for the general population. EPA's fish consumption rates for the final rule were derived from data for specific populations that consume recreationally caught freshwater fish, as reported in the 1997

EFH. The specific values EPA used were 5.3, 8.6, and 8.8 g/day for children ages 0-5, 6-11, 12-19, respectively.

For children of subsistence fishers, EPA used fish ingestion rates of 20, 42, and 57 g/day for age groups 0-5, 6-11, and 12-19, respectively. These estimates were derived from data on fish consumption by Native Americans in the Columbia River Basin that have a diet high in fish.

***Comment 380*:** The commenter notes that the risk assessment did not evaluate or even discuss the noncarcinogenic effects of dioxin compounds, despite the accumulative evidence of these effects. The commenter notes that noncancer effects include damage to reproductive, hormone, and immune systems and that these effects of dioxin, in animals and humans, are much worse than was previously thought. Further, the commenter states that some of these effects are already occurring in the U.S. population. The commenter points out that, according to EPA, on average we are likely to have body burdens of 9 ppt of dioxin TEQs. The commenter summarizes some of the effects of dioxin body burdens as follows: EPA indicates that monkeys get endometriosis at 27 ppt. Decreased testosterone occurs in men at 13 ppt. Altered glucose tolerance (suggesting the threat of diabetes) occurs at 14 ppt in one study. Enhanced viral susceptibility is found in mice at 7 ppt, implying that we could be less resistant to viral illness at our current body level of dioxins. Dioxins stimulate the production of certain enzymes, which could have effects on cell processes, and this effect is found in rats at 1 ppt. The commenter feels that, given the ability of dioxins to bioaccumulate, the noncarcinogenic effects of the combustor emissions are likely significantly underestimated.

Response 380: *EPA agrees with the commenter that dioxins have been shown to exhibit a wide variety of toxic effects other than cancer. There is evidence that occupational and environmental exposures to TCDD have been associated with a number of noncancer health effects, but at levels above background concentrations. Many of dioxins' toxic effects have mechanisms of action that involve the Ah receptor and are common across mammalian species. Animal test results show responses at low levels of exposure, supporting the belief that humans could be affected, as well, at low levels.*

In the absence of a verified reference dose (RfD) for exposure to 2,3,7,8-TCDD, EPA evaluated the potential for noncarcinogenic effects with an incremental margin of exposure (MOE) analysis for all modeled receptor populations/age groups. The incremental MOE analysis compared the estimated incremental average daily dose from dioxins/furans associated with HWC emissions to background exposures in the general population. EPA recognizes the difficulty in interpreting the results of this analysis because the incremental MOE approach compares modeled intake rates to background-based levels that are presumed to be without adverse effects but that are higher than any RfD that could be calculated based on a no observed adverse effect level (NOAEL).

For estimating background exposures, EPA used pharmacokinetic modeling to derive a daily intake rate based on background body burden levels of TCDD-TEQ in human adipose tissue. Specifically, a background body burden level of 30 ppt in human adipose tissue and an assumed half-life of 7 years were used to estimate an intake rate of 110 pg/day. For a 70 kg adult, the 110 pg/day intake corresponds to an average daily dose of 1.5 pg/kg/day. This value was used in the risk analysis for the final rule and compares well with other estimates of background exposures that lie in the range of 1 to 2 pg/kg/day. However, other estimates of background exposure derived from recent dietary surveys suggest that while childhood exposures may lie in this range, adult exposures may be below 1 pg/kg/day. For the final rule, EPA assumed a background exposure of 1.5 pg/kg/day for all age groups.

***Comment 382*:** The commenter feels that EPA should take steps in the instant rulemaking to improve the risk characterization that include modifying the risk assessment to address some of the technical deficiencies noted in these comments (i.e., summing risks across chemicals, duration of exposure, ecologic risk assessment, case study representation).

Response 382: *For the final rule, EPA summed risk estimates for individuals across exposure pathways and across chemicals by route of exposure (inhalation and ingestion). This was done for both cancer risks and noncancer risks (i.e., hazard quotients). This is a conservative approach that assumes additivity of response but accounts for possible portal of entry effects. However, for the purpose of estimating population-level cancer risks, cancer incidence estimates were also summed across route of exposure.*

For the final rule, EPA derived values for the duration of exposure based on data on residential occupancy periods presented in EPA's 1997 Exposure Factors Handbook *(U.S. EPA, 1997a). EPA used age-specific estimates of the mean duration of exposure for farm and nonfarm households to estimate the lifetime average daily dose (LADD) to these receptors. In addition, for key risk-driving exposure pathways, EPA conducted an exposure parameter variability analysis using Monte Carlo simulation that included variability in the duration of exposure for farm receptors. The commenter is referred to the risk assessment background document for the final rule for a complete discussion of exposure duration and other exposure factors that were used in the risk analysis.*
For the final rule, EPA conducted a screening-level ecological assessment. In this analysis, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water and compared to model-predicted media concentrations in order to assess the potential for ecological risk. The ecological risk assessment served as a screening-level analysis designed to identify the potential for adverse effects. The process was based on current EPA guidelines for ecological risk assessment and began with the

selection of assessment endpoints (e.g., the actual environmental values to be protected) that reflect the management goals for the analysis and relate to (1) a valued ecological entity such as a wildlife species, and (2) an attribute of that entity that is important to protect (for example, reproductive fitness).

For the final rule, EPA evaluated 76 active HWC facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). This represents over 40 percent of the hazardous waste combustion facilities covered by the rule. To ensure that these facilities would be representative of all facilities covered by the rule, 66 of the 76 facilities were selected using stratified random sampling, while the remaining 10 were facilities that had been analyzed at proposal and were retained for the final rule (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure). The use of stratified random sampling also allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe. Specifically, for the final rule, confidence intervals reflecting sampling error (i.e., error introduced into the analysis by not having modeled all HWC facilities) were generated for the majority of risk results. Sampling error is a key source of uncertainty that impacts the representativeness of risk results generated for the HWC risk analysis. Sample sizes for each combustor category were based on the goal of having a 90 percent probability of selecting a facility from the top 10 percent of facilities within a given combustor category with regard to risk (i.e., a 90 percent probability of having included a "high risk" facility in the sample). By taking this approach to facility selection, EPA believes the risk assessment for the final rule is representative of the range and types of HWC facilities and adequately characterizes risks at the high end of the risk distribution.

***Comment 1100*:** The commenter feels EPA should carefully and comprehensively identify all the limitations in the risk assessment which affect whether and under what circumstances definitive conclusions that the proposed standards are fully protective can be made at this time. The commenter feels that uncertainties should foreclose the use of the risk assessment as justification for not considering BTF emission standards. The commenter feels that EPA should take aggressive steps in this rulemaking to minimize the nature and extent of hazardous waste combustor emissions. While Section 112(f) of the CAA suggests EPA may address residual risks from HWCs in a subsequent rulemaking, this rulemaking is based upon both RCRA and CAA authorities, and pursuant to RCRA EPA is required to address potential risks now to fulfill the RCRA mandate to protect human health and the environment. The commenter believes that the suggested modifications to the risk assessment will further document these potential risks.

Response 1100: *EPA discusses the limitations and uncertainty associated with the data and models used in the risk analysis in the risk assessment background document for the final rule* (*RTI, 1999). EPA has emphasized these uncertainties in its characterization of*

risks from HWCs. The Background Document includes a detailed discussion of uncertainty associated with all major components of the HWC risk analysis including fate/transport modeling, exposure assessment, and risk characterization.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology. For existing sources, EPA is required to set the emission standard based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources. However, EPA may establish emission standards that are more stringent than this.

EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Section 28
National Cement Company of California, Inc.
RCSP-0094

***Comment 307*:** The commenter notes that in the proposed MCT rule EPA has required a decrease in APCD inlet gas temperature, which will reduce stack emission temperature. The commenter notes that because dispersion of pollutants from a stack is directly related to exit gas temperature, individuals residing in close proximity to cement plants may experience increased exposure to pollutants and suffer increased health risk.

Response 307: *Temperature is one of many parameters that affect exhaust plume dispersion from cement kilns. Temperature can influence ultimate plume rise, thus effectively causing the dispersion of constituents to be at elevations that are higher that the physical height of the stack. Although the MACT standards may require some HWCs to lower their exhaust gas temperatures, the thermal buoyancy of the exhaust gases from cement kilns, which is quite substantial, combined with the relatively tall stacks found at cement kilns should be more than sufficient to promote good atmospheric mixing and dispersion.*

Indirect pathway exposures tend to be more important for constituents that contribute most to human risk (e.g. lead, dioxins/furans, mercury). Indirect pathway risks are a result of constituent deposition over broad areas. Lower exhaust temperatures may alter the pattern of deposition, but the total mass of material deposited within the 20 km study area is not likely to be significantly affected. Therefore, aquatic pathways will be little affected by lower exhaust temperature because the total deposition over a watershed area will not be significantly different and total loads to waterbodies will be little changed. More important is the reduction in deposition that results from lowered emissions.

EPA's risk analysis indicates that direct inhalation pathways are of relatively little importance for cement kilns. The effect of lower exhaust temperature on average ground-level concentrations is uncertain but is not expected to be large. However, reductions in emissions associated with pollution controls that lower exhaust temperature will likely overshadow the temperature effect, resulting in an overall lowering of ground-level concentrations.

EPA believes it is unlikely that individuals residing near cement kilns will be at increased risk as a result of the MACT standards. On the contrary, EPA believes that the emission reductions achieved with the MACT standards will decrease risks to nearly all individuals living in the vicinity of hazardous waste–burning cement kilns.

Section 29
New Jersey Department of Environmental Protection
RCSP-0161

***Comment 378*:** The commenter commends EPA for conducting an extensive risk assessment that evaluated direct and indirect pathways from model incinerators and expects that this will serve as a good foundation for the residual risk assessment which will be required under Section 112(f). However, the commenter disagrees with the use of a 30-year exposure period and recommends a 70-year lifetime exposure for a source of this type. The commenter feels that risk assessment procedures should be applied to each incinerator within 1 year of promulgation of these standards to show the existing risk and the reduced risk after compliance is achieved.

Response 378: *The duration of exposure to a contaminant is directly related to the period of time an individual resides at a given site. This period of time is often considerably shorter than the operating life of a facility. Information on population mobility and residence time indicate that for most sectors of the U.S. population, the likelihood of anyone residing at the same location for 70 years is relatively small. For example, Bureau of Census data show that over half of all households have lived in their current residence for 9 years or less and only 1 percent have lived in their current residence for more than 50 years.*

For the final rule, EPA derived values for the duration of exposure based on data on residential occupancy periods presented in EPA's 1997 Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a). EPA used age-specific estimates of the mean duration of exposure for farm and nonfarm households. In addition, for key risk-driving exposure pathways, EPA conducted an exposure parameter variability analysis using Monte Carlo simulation that included variability in the duration of exposure for farm receptors. The commenter is referred to the risk assessment background document for the final rule for a complete discussion of exposure duration and other exposure factors that were used in the risk analysis.*

Section 30
New York State Department of Environmental Conservation
RCSP-0207

***Comment 568*:** The commenter notes that in the evaluation of protection provided by the proposed MACT standards, EPA has considered health impacts of dioxin, arsenic, beryllium, cadmium, and hexavalent chromium only; however, impacts due to emissions of nickel (a carcinogenic metal), mercury, and nondioxin PICs have not been considered. In addition, the commenter points out that the proposed rules have no standards for other metals such as selenium, cobalt, manganese, etc. The commenter feels that, unless a site-specific risk assessment is done, the proposed standards will not address the cancer risk due to nickel emissions and noncancer impacts due to emissions of other metals at specific sites.

Response 568: *In the final rule, EPA evaluated human health and ecological risks for all metals for which adequate emissions data were available. These included selenium, nickel, mercury (elemental, divalent, and methylmercury), cobalt, and manganese, as well as antimony, arsenic, barium, beryllium, cadmium, chromium (III), chromium (VI), copper, lead, silver, and thallium.*

The risk assessment for the final rule included a quantitative analysis of risks from mercury based on information contained in the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c). This report was subject to extensive peer review, including review by EPA's Science Advisory Board, and was the basis for the modeling approach used in the analysis, as well as the toxicological benchmarks used to evaluate risks associated with exposures to mercury.*

The risk assessment for the final rule assessed risks from chlorinated dioxins and furans. However, risks from other organics that may be present as PICs could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited and the data are highly variable, so they are inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of

the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

***Comment 41*:** The commenter notes that information in the "Evaluation of Protectiveness" sections of the *Federal Register* notice indicates that for existing facilities, the range of estimated cancer risks is lower when baseline (i.e., current) emission levels are used than when MACT emission levels are used. The commenter feels that this does not make sense and is inconsistent with EPA's goal of providing increased protection to human health and the environment over existing RCRA standards. The commenter notes that in the case of metals, this occurs for every facility type with floor emission levels; in the case of dioxins and furans, this occurs only for lightweight aggregate kilns with beyond-the-floor emission levels.

Response 41: *EPA agrees with the commenter that risks should not be lower at baseline than at the MACT standards. This outcome was a consequence of the way in which emission rates were determined. At proposal, baseline emissions (reflecting current conditions) were estimated from the national distribution of stack gas concentrations for a given category of sources (e.g., cement kilns, incinerators, and lightweight aggregate kilns). Central tendency and high-end emissions estimates were made by taking the 50th and 90th percentiles of the stack gas concentration distributions and calculating the emission rate from an individual facility's stack gas flow rates. Then, for the purpose of evaluating risks associated with the emission standards, it was assumed that facilities would emit at the design level (the level that a facility would likely have to design to in order to meet the standard), even if this meant an increase in emissions over baseline (which in many instances it did). Furthermore, it was also assumed that a facility could emit each of the metals that were subject to the same emission standard up to the design level (e.g., for the SVM standard, a facility could emit both cadmium and lead up to the design level). As a result, the risk estimates were often higher for the proposed standards than at baseline, as noted by the commenter.*

As explained in the preamble to the final rule, EPA modified the approach for estimating emissions. For the final rule, EPA estimated emissions for a given facility using the available stack gas emissions concentration measurements for that facility. In instances where emissions measurements were not available, stack gas concentrations were imputed from emission measurements at similar facilities (U.S. EPA, 1999a), for a detailed discussion of emissions estimation and data imputation methods). Then, with this as the baseline level of emissions, EPA projected what the emissions would likely be under the MACT standards. EPA assumed that facilities emitting above the design level would reduce their emissions to the design level (which was taken as 70 percent of the MACT standard). EPA determined the percentage reduction needed to meet the design

level and applied that percentage reduction to each chemical constituent to which the standard applies. If a facility's baseline emissions were already estimated to be below the design level, EPA assumed no change and the affected emissions would remain the same. This is the same approach EPA used for estimating national emissions and for determining the costs of the rule. EPA believes this approach gives a more realistic portrayal of emissions under the MACT standards and the associated risks.

***Comment 449*:** The commenter notes that emission levels that EPA used in the risk assessment do not always match the emission levels given in the *Federal Register* notice. As an example, the commenter cites footnote 4 of Table III.C.1 (page 17389) of the *Federal Register* notice that indicates that dioxin cancer risk estimates for floor level emissions from existing incinerators were based on a dioxin concentration of 20 nanograms per dry standard cubic meter (ng/dscm) measured as 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) toxic equivalents (TEQ). The commenter notes that although the *Federal Register* notice indicates that this represents the highest level known to be emitted at the floor, the risk assessment document indicates that the estimated "high-end" floor-level dioxin risk estimate for existing incinerators was estimated based on an emission level of 4 ng/dscm TEQ. The commenter notes that the risk assessment similarly used an emission level of 120 µg/dscm to estimate cancer risks for MACT floor-level emissions of semivolatile metals from existing incinerators, while the *Federal Register* notice indicated that the cancer risk estimates were based on the air pollution control equipment (APCE) design value corresponding to the proposed MACT floor of 270 µg/dscm. The commenter feels that these apparent inconsistencies between the risk assessment and the *Federal Register* notice are confusing and make it difficult to determine if EPA has complied with the RCRA requirement of demonstrating that the proposed standards are protective of human health. The commenter states that the *Federal Register* notice and the risk assessment should be consistent with respect to the baseline, floor, beyond-the-floor, and continuous emission monitor option emission levels upon which risk estimates are based.

Response 449: *The emissions levels EPA used in the risk assessment at proposal are the levels given in the risk assessment background information document (see* Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). For dioxins and furans, the MACT floor was defined as a level of 0.2 ng/dscm, or 400EF. The use of temperature control to limit emissions results in variable emissions levels depending on the particular combustion unit. EPA projected the effect of temperature control on emissions for incinerators with dioxin emissions data and selected the 90th percentile of the distribution as a high-end value. This value was 4 ng/dscm and was used in the risk assessment at proposal for characterizing high-end risks. The value given in the footnote to table III.C.1. on page 17389 of the April 19, 1996, notice of proposed rulemaking (i.e.,20 ng/dscm) is a typographical error. For semivolatile metals (cadmium and lead), the risk analysis was apparently based on a level of the standard for incinerators of 120 µg/dscm, as indicated in the background information document.*

However, the correct level was 270 µg/dscm, as indicated in the April 19, 1996, Federal Register *notice. Therefore, it appears that the risk results for semivolatile metals for incinerators as presented at proposal were in error.*

***Comment 453*:** The commenter believes that the risk assessment does not contain enough information to allow a reader to independently verify EPA's risk estimates for each of the receptors in each of the 11 cases evaluated. For example, the risk assessment does not identify the risk estimates for individual receptors at each facility. The commenter suggests that EPA should ensure that all pertinent information is provided in the risk assessment in a manner that allows readers to readily determine how exposures and risks were estimated and to permit verification of those estimates.

Response 453: *As indicated by the commenter, the background information document for the proposed rule did not present facility-specific risk results for any receptor. While all site-specific information that was used to characterize the environmental settings of the example facilities was fully documented in the appendices to the background information document, EPA's intent was to characterize the range of risks across the facilities within a source category (i.e., cement kilns, incinerators, and lightweight aggregate kilns) taken together as a class. EPA thought it would be inappropriate to present risk results for individual facilities because the analysis did not use site-specific emissions data. As EPA stated in the preamble to the proposed rule, the example facility analyses were considered useful for providing information to evaluate national standards on a generic basis but they are not site-specific assessments of any given individual facility and cannot be regarded as such.*

The risk analysis completed for the final rule provides an expanded set of individual-level and population-level risk results that are differentiated as to receptor population, age group, constituent type, and combustor category. With regard to individual risk, results for the final rule include multiple risk percentiles (i.e., 50th, 90th, 95th, and 99th percentile risk estimates), which characterize the range of risk experienced by a specific receptor population/age group combination, including both central tendency and high-end risk. Similarly, population-level risk results generated for the final rule are differentiated (depending on the specific category of risk results) as to receptor population, chemical constituent, and combustor category. Although the risk results are based on site-specific emissions estimates, due to the large volume of data generated by the risk analysis, EPA has not produced a complete set of risk results for each of the facilities modeled. However, EPA has produced facility-specific risk estimates for the subsistence scenarios, which include risk results by exposure pathway. This information may be found in the docket for the final rule. In addition, full documentation has been prepared of the methods and data used in the analysis, including example calculations and expanded information on media concentrations. EPA believes that this body of

information should be sufficient to facilitate an in-depth review of the risk assessment for the final rule and that it satisfies EPA's goal of transparency in all its risk assessments.

Comment 454: Since the risk assessment only evaluated 11 of 195 existing HWCs, potential worst-case risks may not be reflected in the risk assessment results. A subsistence farmer at the location of maximum facility impact would probably be a worst-case receptor. EPA could add this scenario to the risk assessment as an evaluation of potential worst-case risks for facilities not addressed in the risk assessment.

Response 454: *In response to comments received from both peer reviewers and the public, EPA implemented a number of modifications to the risk analysis framework for the final rule to improve the representativeness of the assessment. Specifically, rather than using a case study approach to evaluate 11 existing facilities, EPA used stratified random sampling to select a subset of facilities from the HWC facility universe for risk characterization for the final rule.*

For the final rule, EPA evaluated 76 active HWC facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). This group represents more than 40 percent of the HWC facilities covered by the rule. To ensure that these facilities would be representative of all facilities covered by the rule, 66 of the 76 facilities were selected using stratified random sampling, while the remaining 10 were facilities that had been analyzed at proposal and were retained for the final rule (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure). The use of stratified random sampling allows EPA to make clear statistical statements regarding the representativeness of risk results for the HWC facility universe. Specifically, for the final rule, confidence intervals reflecting sampling error (i.e., error introduced into the analysis by not having modeled all HWC facilities) were generated for the majority of risk results. Sampling error is a key source of uncertainty that impacts the representativeness of risk results generated for the HWC risk analysis. Use of a nonstatistical selection strategy, such as the case study approach used at proposal, does not allow the representativeness of risk results to be assessed (e.g., confidence intervals reflecting sampling error cannot be generated). Sample sizes for each combustor category were based on the goal of having a 90 percent probability of selecting a facility from the top 10 percent of facilities within a given combustor category with regard to risk (i.e., a 90 percent probability of having included a high-risk facility in the sample). EPA's analysis for the final rule met or exceeded this goal. Stratified random sampling also reduces the level of bias in the overall analysis by avoiding the use of purposive or biased selection of modeled facilities. For these reasons, EPA believes the risk assessment for the final rule is representative of the range and types of HWC facilities.

Furthermore, because of the sample sizes used in the analysis, EPA also expects that the risk assessment adequately characterizes risks at the high end of the risk distribution.

The risk assessment for the final rule was modified to account for the number of individuals exposed and their location using U.S. Census and Census of Agriculture data to both locate and enumerate persons living in farm and nonfarm households. Individual risks were characterized by generating cumulative frequency distributions that explicitly account for the numbers of persons exposed at differing levels of exposure. However, it was not possible from census data to identify and locate individual farms that may be engaged in subsistence farming. Although local officials were contacted to identify the location of subsistence farms at proposal, this was not possible for the final rule due to the large number of facilities evaluated and restrictions on collecting information from nonfederal sources. Despite this limitation, subsistence scenarios were retained in the risk analysis for the final rule. Although it is not known precisely how many individuals are engaged in subsistence activities or exactly where those activities take place, subsistence does occur in some segments of the U.S. population, and EPA believes it is important to evaluate the risks to those individuals. For the purpose of assessing the potential risks, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations in the final rule. EPA recognizes that this assumption may lead to risk estimates that have a relatively low probability of actually occurring in the population of interest.

***Comment 455*:** The commenter feels that EPA's approach for identifying receptor locations should have included contacts with appropriate regulatory agencies. The commenter notes that EPA did contact local officials, use maps, and conduct telephone surveys to identify receptors and this is reasonable. However, appropriate regulatory agencies could provide EPA with existing risk assessments for facilities of interest, and EPA could use these assessments to verify the accuracy of receptor locations.

Response 455: *For the final rule, EPA visited a limited number of state offices to collect facility-specific information to improve the characterization of both facility operations and land use surrounding HWC facilities. These visits focused on obtaining permitting information, including site-specific risk assessments, as well as information obtained directly from regulatory staff who were familiar with specific facilities. The kinds of information collected included facility status (e.g., operational or undergoing RCRA closure), location, key facility operational parameters (i.e., stack height, exit velocity), and facility-specific emissions data.*

For the final rule, EPA used U.S. Census and Census of Agriculture data to characterize the location/density of receptor populations relative to the modeled facilities. These locational data were used to generate population-weighted individual risk distributions

from which specific percentiles (e.g., 50th, 90th, 95th, and 99th) could be estimated. The U.S. Census provides detailed population density data, which is broken down into the number of total persons and the number of persons in rural areas on farms. The type of farm was determined from Census of Agriculture data (e.g., beef cattle farms vs. dairy farms). The commenter is referred to the risk assessment background document for a complete discussion of the use of census data to enumerate and locate receptor populations for the final rule (RTI, 1999). EPA believes that the use of census data allows a more complete picture of risks to individuals and population level risk than could be achieved by locating a small number of individual farms in close proximity to HWCs.

***Comment 457*:** The commenter notes that, for each facility in the risk assessment, EPA evaluated a number of receptors including a "maximally exposed individual" (MEI), who resides at the point of maximum facility impact. The commenter points out that EPA only evaluated inhalation exposure for the MEI, yet indirect exposure pathways as well as inhalation were evaluated at other locations. The commenter notes another approach has been to evaluate the MEI on a case-by-case basis to determine if indirect routes of exposure are important for that individual. The commenter feels that EPA should also have evaluated home gardening, soil ingestion, and breast milk consumption for facilities for which the point of maximum impact was in a residential or rural area, and that children should have also been evaluated at the MEI location. The commenter notes that adding indirect exposure pathways at the MEI location would not affect EPA's risk estimates for the MEI for metals but would increase dioxin risk estimates for the MEI by a factor of about 10.

***Response 457*:** *EPA received a number of comments on the use of bounding estimates in the risk assessment, such as the "maximally exposed individual," or MEI. While EPA believes that bounding estimate have a role to play in risk analysis, EPA decided that bounding estimates were not needed as part of the risk assessment for the final rule. Instead, EPA concluded that it was most important to characterize the distribution of risks in the exposed population based on actual data on the number and locations of exposed individuals. As indicated in response to the previous comment, EPA used U.S. Census and Census of Agriculture data to enumerate and locate receptor populations that could be identified from these data.*

The population-based risk analysis for the final rule had two components:

- *Assessing direct and indirect exposures to a representative individual for each receptor population within each of 16 sectors of a 20 km study area around a HWC facility*
- *Projecting the number of individuals experiencing that level of exposure.*

Receptor populations evaluated in the final rule include commercial beef, dairy, hog, and produce farmers; residents; home gardeners; recreational fishers; subsistence farmers;

and subsistence fishers. In addition to modeling adult exposures (i.e., those over 19 years), EPA also modeled three younger age groups (ages 0-5, 6-11, and 12 -19) for each receptor population.

The analysis also included an assessment of risks to sensitive subpopulations that included the following:

- *Incremental margin of exposure (MOE) analysis for exposure to dioxin TEQ and for infant exposure through breast milk*
- *Blood lead level (PbB) analysis for 0- to 5-year-olds in each receptor population*
- *Assessment of the risks (HQ) of developmental effects from prenatal and childhood exposures to methylmercury*
- *Characterization of premature mortality and respiratory and cardiovascular disease (annual incidence avoided) for individuals over 65 years old as a result of reductions in exposure to PM_{10} and $PM_{2.5}$*
- *Characterization of risks (cancer and noncancer) to persons engaged in subsistence farming and subsistence fishing.*

All receptor populations were evaluated for inhalation and soil ingestion (excluding pica behavior). Only those individuals residing within study areas identified as having surface waterbodies that are sources of drinking water were evaluated for risks associated with tap water ingestion.

EPA notes that in characterizing risks associated with subsistence activities, EPA assumed that subsistence farming could take place in any of the 16 sectors used to differentiate the locations of exposed populations within a study area. EPA also assumed that subsistence fishing could take place at any of the bodies of water that were modeled in the risk analysis. Therefore, EPA believes that it is unlikely that the risk analysis for the final rule underestimated risks to these highly exposed subpopulations or the other sensitive subpopulations indicated above.

***Comment 458*:** The commenter notes that although EPA evaluated risks associated with baseline emissions of nickel, EPA did not propose an emission standard for nickel because EPA determined that existing emissions data were insufficient. The commenter points out that risk assessments for HWCs in New York State (e.g., Kodak, Nepera, Norlite, Schenectady International) have evaluated nickel based on measured emission data and EPA could use these data to develop proposed emission standards.

Response 458: *In the risk assessment for the final rule, EPA evaluated human health and ecological risks for all metals (including nickel) for which adequate emissions data*

were available. The risk analysis for the final rule specifically evaluated risks from inhalation exposures to nickel in HWC emissions.

Comment 459: The commenter notes that the *Federal Register* notice includes proposed emission standards for mercury. However, the notice indicates that the health risk assessment did not include an evaluation of mercury because of insufficient information on the environmental fate of mercury. The commenter points out that an assessment of mercury could be performed using EPA's own *Implementation Guidance for Conducting Indirect Exposure Analysis at RCRA Combustion Units*, which is frequently used in the RCRA program, a document that provides guidance on estimating risks for mercury. The commenter states that if EPA believes that the uncertainty in the environmental fate of mercury is significantly greater than for other chemicals assessed in the risk assessment (such as dioxins/furans), they could incorporate a quantitative uncertainty analysis into the assessment. The commenter notes that EPA incorporated variability and uncertainty in the exposure parameters for the other chemicals evaluated in the risk assessment and they could do the same with the mercury environmental fate parameters. The commenter suggests the resulting risk estimates for mercury could be presented as a range that includes the uncertainty in environmental fate parameters and the variability in exposure parameters.

Response 459: *At the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA, 1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

The mercury assessment followed the general modeling approach developed for the 1997 MRTC and used the IEM-2M surface water model developed especially for that report.

For the final rule, EPA assessed the human health and ecological risks associated with elemental, divalent, and methylmercury. EPA's analysis shows that the most significant exposures come from methylmercury, due primarily to deposition and runoff of divalent mercury to surface water and subsequent methylation and bioaccumulation in fish.

The uncertainty associated with modeling mercury exposure and assessing the potential for risk stem from (a) complexities associated with modeling the fate/transport of different mercury species through different environmental media following release from

HWC facilities, (b) difficulties in accurately modeling individual behavior related to mercury exposure (e.g., recreational fishing activities at specific locations and amounts of fish consumed), and (c) challenges in evaluating differing health effects studies and accurately characterizing the dose-response relationship (i.e., RfD) for methylmercury. The risk assessment background document for the final rule includes a detailed discussion of uncertainty associated with all major components of the HWC risk analysis, including fate/transport modeling, exposure assessment and risk characterization for mercury.

***Comment 460*:** The commenter notes that while toxicity values to assess noncancer risks for oral and inhalation exposure to 2,3,7,8-TCDD equivalents were not included in the risk assessment, data are available in recent scientific literature to derive a noncancer oral toxicity value for 2,3,7,8-TCDD. The commenter suggests several studies on the reproductive effects of 2,3,7,8-TCDD in rhesus monkeys have recently been published (Bowman et al., 1989a, b; 1990a,b; Rier et al., 1993; Schantz and Bowman, 1989; Schantz et al., 1992) and summarizes the results as follows. These studies report an increased incidence of endometriosis in the adult females exposed to 0.13 nanograms per kilogram body weight per day (ng/kg/day) and developmental effects (reduced growth during nursing and post-weaning behavioral/learning effects) in their offspring. Thus, the dose of 0.13 ng/kg/day may be considered a lowest-observed-effect level for maternal and developmental toxicity. The commenter feels that this dose level could be used to derive a reference dose for 2,3,7,8-TCDD toxicity equivalents. Although the exact magnitude of the uncertainty factor is debatable, the commenter suggests that if an uncertainty factor of 1,000 were used, the resulting reference dose (1×10^{-10} milligrams per kilogram body weight per day [mg/kg/day]) could be applied to the oral and inhalation exposure estimates for 2,3,7,8-TCDD equivalents to evaluate noncancer health risks.

Response 460: *EPA assessed the body of literature on the health effects of 2,3,7,8-TCDD as part of the dioxin reassessment (see* Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin [TCDD] and Related Compounds, *U.S. EPA, 1994d,e). As EPA explained then, calculation of an RfD based on human and animal data and including standard uncertainty factors to account for species differences and sensitive subpopulations would likely result in a reference intake level on the order of 10 to 100 times below the current estimates of daily intake in the general population. For most compounds where RfDs are applied, the compounds are not persistent and background exposures that are generally low are not taken into account. Dioxin and related compounds present an excellent example of a case where background levels in the general population are likely to have significance for evaluation of the relative impact of incremental exposures associated with a specific source. Since RfDs refer to the total chronic dose level, the use of the RfD in evaluating incremental exposures in the face of a background intake exceeding the RfD would be inappropriate and make the calculation of an RfD for dioxin-like compounds of doubtful significance. Although the health assessment document is a draft document and the dioxin reassessment has yet to be*

finalized, the considerations outlined above remain true today. EPA notes that use of the lowest observed adverse effects level and uncertainty factor suggested by the commenter results in an intake exposure level that is an order of magnitude <u>below</u> current background exposures, which EPA estimates lie in the range of 1 to 2 pg/kg/day.

With respect to the possibility of health effects at current background levels, EPA believes there is insufficient evidence thus far to conclude that adverse noncancer effects are occurring in the general population at current background levels of exposure. As indicated in the draft dioxin reassessment, the available epidemiologic data are limited in this regard due to a number of possible factors, including the absence of a large body of human exposure data for the general population; the limited ability of epidemiologic studies to detect effects in relatively small populations when exposures are low and the outcomes are rare; and the difficulty of adjusting for all potentially confounding factors. EPA believes an evaluation of hazard and risk must rely on a weight-of-evidence approach in which all available data (animal and human) are examined together, a process that often requires extrapolation of effects across various animal species as well as to humans.

Although evidence of adverse effects in humans at current background levels is limited, adaptive changes may be occurring at these levels. In commenting on this point in its review of the draft dioxin reassessment, the SAB said that the overall impact of certain biochemical changes seen at lower levels is not fully understood and that current knowledge of the mechanisms of TCDD toxicity has not identified the biological determinants of specificity that would allow one to extrapolate toxicities across species with confidence. The SAB agreed with EPA that dioxins produce a spectrum of effects in laboratory animals depending on the dose, context of exposure, and the genetic background but concluded that adverse effects attributable to chronic low-level exposure in humans have not yet been adequately demonstrated.

For the final rule, EPA used a modified margin of exposure (MOE) approach to assess possible noncancer risks from dioxins. In this approach, the estimated average daily dose for HWCs is compared to background exposures in the general population. As a measure of risk, this incremental MOE presupposes that if exposures are small relative to background, then risks from these exposures are likely to have limited significance for human health.

Comment 1058: The commenter notes that the risk assessment did not provide noncancer inhalation toxicity values for most of the contaminants. The commenter suggests that the New York State Department of Health (NYS DOH) has developed noncancer ambient air criteria for three of the contaminants which may be used to assess the noncancer inhalation risks. The ambient air criteria values are 0.02 micrograms per cubic meter of air ($\mu g/m^3$) for both cadmium and nickel and 0.1 pg/ml

for total chromium. The commenter provides the NYS DOH ambient air criteria documents for these contaminants.

The commenter also points out that the inhalation reference concentration for barium was incorrectly listed as 0.0005 pg/mL. The *Health Effects Assessment Summary Tables* (U.S. EPA, 1995) list a barium reference concentration of 0.0005 µg/m^3. The commenter suggests that exposure estimates for beryllium could be compared to the NESHAP standard of 0.01 µg/m^3 (U.S. EPA, 1973, "National Emissions Standards for Hazardous Air Pollutants," 38 FR 8820-8850). For the remaining contaminants having oral reference doses based on systemic toxicity (antimony, arsenic, selenium, silver, and thallium), the commenter suggests that oral reference doses may be used as surrogates to evaluate noncarcinogenic inhalation health risks.

Response 1058: *EPA estimated noncancer inhalation risks (i.e., hazard quotients) for barium, chlorine, hydrogen chloride, manganese, and elemental mercury. Although EPA has derived a reference air concentration (RfC) for beryllium of 0.02 µg/m^3, EPA did not assess noncancer inhalation risks from beryllium in the final rule. However, EPA did assess inhalation cancer risks for beryllium. Because the air concentration associated with a 1 x 10^{-5} lifetime excess cancer risk is 4 x 10^{-3} µg/m^3, and the highest cancer risks for beryllium were estimated to be 4 x 10^{-9}, EPA believes it is highly unlikely that the RfC for beryllium could have been exceeded for any of the receptor populations evaluated in the risk assessment for the final rule. The NESHAP standard for beryllium suggested by the commenter is not a health-based number, so EPA does not believe it is appropriate to use such a number for assessing risks.*

Although EPA estimated noncancer inhalation risks for barium using a provisional reference air concentrations from HEAST (Health Effects Assessment Summary Tables, U.S. EPA, 1997b), EPA's National Center for Environmental Assessment does not currently recommend an RfC for barium. As indicated in the IRIS (Integrated Risk Information System, U.S. EPA, 1998a) summary for barium, the human and animal inhalation and intratracheal studies suggest that the respiratory system is a target of barium toxicity. The data also suggest that systemic effects, such as hypertension, may occur following inhalation exposure. However, the human studies cannot be used to derive an RfC for barium because exposure concentrations were not reported. Specifically, barium exposure levels were not measured in the group of workers with increased incidence of hypertension in the NIOSH (1982) study ("Health Hazard Evaluation Report: Sherwin Williams Company, Coffeyville, Kansas," National Institute for Occupational Safety and Health, Centers for Disease Control, Cincinnati, OH. NIOSH Report No. HETA/81-356-1183) as cited in IRIS (U.S. EPA, 1998a). Furthermore, deficient reporting of methods and results from the only animal subchronic/chronic inhalation study (Tarasenko et al., 1977) precludes deriving an RfC for barium from animal data.

EPA did not estimate noncancer risks for cadmium or nickel because RfCs were not available in IRIS (U.S. EPA, 1998a) or HEAST. EPA follows a general policy of using toxicological benchmarks from IRIS or HEAST, although EPA may adopt other values on an interim or provisional basis if values are not available from either of these sources. The values in IRIS have undergone extensive peer review and represent agency consensus on the toxicity of a chemical. The values in HEAST have not undergone the same level of review, but they represent provisional health values from EPA's National Center for Environmental Assessment, which is a part of EPA's Office of Research and Development. EPA notes, however, that inhalation cancer risks were assessed for both cadmium and chromium and that the air concentration associated with a 1 x 10^{-5} lifetime excess cancer risk is 6 x 10^{-3} µg/m^3 for cadmium and 4 x 10^{-2} µg/m for nickel. These compare with an ambient air criteria value of 0.02 µg/m^3, suggested by the commenter. Because the highest cancer risks for cadmium and nickel were estimated to be 5 x 10^{-7} for cadmium and 1 x 10^{-8} for nickel, EPA believes it is highly unlikely that the 0.02 µg/m^3 value suggested by the commenter would have been exceeded for any of the receptor populations evaluated in the risk assessment for the final rule.

EPA recently derived two RfCs for chromium (VI). The RfC for chromium particulates is 1 x 10^{-4} mg/m^3. The RfC for chromic acid mists and dissolved chromium aerosols is 8 x 10^{-6} mg/m^3. However, because these values were not available at the time EPA performed the risk analysis, EPA did not assess noncancer inhalation risks for chromium (VI) in the final rule. However, EPA did assess inhalation cancer risks for chromium (VI). Because the air concentration associated with a 1 x 10^{-5} lifetime excess cancer risk is 8 x 10^{-4} µg/m^3 (8 x 10^{-7} mg/m^3), and the highest cancer risks for chromium (VI) were estimated to be 4 x 10^{-7}, EPA believes it is very unlikely that even the lower of the two RfC's for chromium (VI) could have been exceeded for any of the receptor populations evaluated in the risk assessment for the final rule. The ambient air criteria value for total chromium suggested by the commenter (0.1 pg/mL, assumed to be equal to 0.1 µg/m^3) is equivalent to the RfC for particulate chromium (VI) and well above the RfC for chromic acid mists.

Regarding the commenter's suggestion that oral RfDs be used as surrogates to evaluate noncancer inhalation health risks, EPA disagrees as a general matter that route to route extrapolation is appropriate. In certain circumstances route to route extrapolations may be justified, such as if inhalation studies show a systemic effect and provide sufficient pharmacokinetic information. In general, route-to-route extrapolation is not recommended because of the potential for portal-of-entry effects (e.g., respiratory tract effects following inhalation exposure) and/or first-pass effects (i.e., the metabolism that takes place in the portal-of-entry tissue prior to entry into the systemic circulation, thereby resulting in differences in toxicity between oral and inhalation exposures). Unless

the first-pass effect and dosimetry are adequately understood, substantial error can be introduced performing route-to-route extrapolation. In the absence of data to determine dosimetry via inhalation, when a chemical is thought to be susceptible to first-pass effects, or where a potential for portal-of-entry effects is indicated, then route-to-route extrapolation for the derivation of an RfC is not appropriate (U.S. EPA, 1994f, Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry*).*

Oral toxicity data may be useful to derive an RfC only when the respiratory tract effects, or "first-pass" effects (metabolism that can take place in the portal-of-entry tissue, such as the respiratory tract, or in the liver prior to entry into the systemic circulation), can be ruled out. However, unless first-pass effect and dosimetry are adequately understood, substantial error can be introduced in route-to-route extrapolation.

***Comment 1059*:** The commenter points out that EPA's inhalation cancer potency factor for chromium (VI) is based on an epidemiological study of lung cancer in chromium workers, and was derived using total chromium as a surrogate for chromium (VI) exposure (U.S. EPA, 1984, *Heath Assessment Document for Chromium. Final Report,* EPA-600/8-83-014F, Environmental Criteria and Assessment Office, Research Triangle Park, NC). Based on the same study, the commenter points out that NYS DOH derived an inhalation cancer potency factor of 175 $(mg/kg/day)^{-1}$ (NYS DOH, 1990, *Ambient Air Criteria Document for Chromium,* Bureau of Toxic Substance Assessment, Albany, NY). The NYS DOH number is based solely on chromium (VI) exposure, which was estimated using analytical speciation data for the chromium facility under study. The commenter provides the NYS DOH Air Criteria Document for Chromium and points out that application of the NYS DOH cancer potency factor to chromium (VI) exposure estimates increases the estimated inhalation cancer risk by a factor of 4.3.

Response 1059: *EPA recently completed a toxicological review for hexavalent chromium (see* Toxicological Review of Hexavalent Chromium*, August 1998, U.S. EPA 1998d, as cited in IRIS, U.S. EPA 1998a). EPA used the data of Mancuso (1975), an occupational study of chromate production workers, to estimate an inhalation unit risk factor of 1.2 x 10^{-2} per µg/m^3. This is equivalent to a slope factor of 42 per mg/kg/day. The same study was used to derive the slope factor of 175 per mg/kg/day cited by the commenter.*

EPA estimated the risk of hexavalent chromium on the basis of the total chromium, both soluble and insoluble, to which the chromium workers were exposed. Since there are likely differences in the potency of the chromium compounds that the workers were exposed to, the potency of hexavalent chromium compounds may have been underestimated. However, other factors may have lead to some overestimation of the potency, including possible underestimation of worker exposures (the industrial hygiene data used for estimating exposures were collected more than a decade after the

exposures occurred) and failure to account for the higher prevalence of smoking generally found in industrial workers.

One difference between the New York Department of Health potency estimate and EPA's is that NYDOH used data from another study to estimate the ratio of trivalent to hexavalent chromium to adjust the slope factor. Bourne and Yee (1950) reported that the ratios of chromium (III) to chromium (VI) concentrations in the airborne dust in nine major departments in the plant in which the Mancuso cohort worked ranged from 1 to 3, except for two departments where the ratios were 6 for the lime and ash operation and 52 for the ore operation. Excluding the ore operation, the maximum ratio of trivalent chromium to hexavalent chromium is 6. Based on this, EPA believes any underestimation of the risk for hexavalent chromium is unlikely to be greater than sevenfold and is most probably less, especially considering that other factors work to overestimate the risk, as discussed above.

***Comment 1060*:** The commenter states that the risk assessment does not clearly identify the unit risk value used to estimate cancer inhalation risks for 2,3,7,8- TCDD. The commenter notes that the toxicity profile cites ranges of unit risks for lung cancer and all cancers based on human exposure, which were derived by EPA in the recent *Health Assessment Document for 2,3,7,8-TCDD* (U.S. EPA, 1994). The commenter points out that both of these values are draft and are under review and that, typically, an inhalation unit risk of 3.3×10^{-5} $(pg/mL)^{-1}$ listed in the *Health Effects Assessment Summary Tables* (U.S. EPA, 1995) is used to estimate cancer inhalation risks for 2,3,7,8-TCDD exposure. The commenter suggests the document should clearly identify the unit risk value that was used to calculate the inhalation cancer risk for 2,3,7,8-TCDD.

Response 1060: *In the human health risk assessment for the HWC rule, EPA used the same cancer slope factor for 2,3,7,8-TCDD for inhalation and ingestion ($1.56 \times 10^{+5}$ per mg/kg-d). This is the cancer slope factor from EPA's 1985 health assessment* (Health Assessment Document for Polychlorinated Dibenzo-p-dioxins*, U.S. EPA, 1985*). *The cancer slope factor was derived from liver tumor data from an oral study in rats that were exposed to 2,3,7,8-TCDD in the diet (Kociba et al., 1978). For the dioxin reassessment, EPA conducted additional dose-response modeling for 2,3,7,8-TCDD (see* Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d). This study lends considerable support to the cancer potency estimate in the 1985 health assessment, as well as the estimate in the 1994 draft reassessment (see* Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds*, U.S. EPA, 1994d,e).*

EPA did not use a unit risk factor for estimating inhalation cancer risks. Instead, EPA use an inhalation slope factor, as explained in the risk assessment background document

for the final rule (RTI, 1999). The unit risk factor is derived by making certain exposure assumptions, such as the inhalation rate and the duration of exposure, which EPA varied among different receptor populations in the risk analysis for the final rule.

Section 31
PermaFix Environmental Services, Inc.
RCSP-0193

***Comment 230*:** The commenter notes recent attendance at the Dioxin96 International Symposium held in Amsterdam, Holland, at which many of the discussions were pertinent to the dioxin standards being proposed for hazardous waste combustors. The commenter notes that the studies presented at the symposium indicated that the source of dioxins and furans is not predominantly from HWCs. Rather, 95 percent of all human health exposure to dioxins is from food. Recent studies completed by the U.S. Department of Agriculture point to the abilities of cattle to convert pentachlorophenol into dioxins and furans. A significant portion of beef TCDD contaminants are from pentachlorophenol wood treatment materials. Also, cattle generate more TCDD than they consume. The commenter indicated that copies of additional relevant papers from the symposium would be forwarded with the hard copy of the comments. A foodchain pathway study performed in an area surrounding a cement kiln burning hazardous waste also confirmed that the impact of constituents from cement kiln operations on beef, fish and vegetables is negligible. The dioxin and furan congeners in vegetables, soil, and beef were demonstrated to be nonexistent or at background levels. Prior to making further determinations as to the source of dioxins and furans, the commenter suggests EPA consult with the Department of Agriculture and the USDA.

Overall, the commenter thinks that the effort put into this proposed rulemaking is an extremely poor use of taxpayer's money. The commenter believes that there are many important environmental issues that should be the focus of EPA, rather than politically motivated issues.

> ***Response 230:*** *HWCs emit hazardous air pollutants (HAPs), listed under Section 112(b) of the CAA. In an effort to control emissions of HAPs from HWCs, EPA proposed NESHAP pursuant to Section 112(d) of the CAA that establish technology-based, not risk-based, emission standards based on application of maximum achievable control technology (MACT). However, Sections 3004(a) and 3004(q) of RCRA, as amended, require EPA to develop standards that are protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate human health and ecological risks from HWCs in order to determine what, if any, RCRA standards for emissions from these sources may be needed. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.*
>
> *For the proposed and final rules, EPA examined risks associated with the ingestion of fish, beef, pork, dairy, poultry, and egg products contaminated with TCDD from HWC emissions. This analysis assessed the incremental risks resulting from emissions emitted*

directly by an HWC unit. The ability of cattle to covert pentachlorophenol to dioxins is not relevant for this risk analysis.

Section 32
Rollins Environmental Inc.
RCSP-0246

***Comment 59*:** The commenter notes the Peer Panel agreed that the use of the toxicity equivalence (TEQ) approach "requires additional consideration" and that use of TEQ "is an oversimplification of the exposure to chemical mixtures like dioxins." The TEQ is a simplification that relates the toxicity of the 209 Poly-Chlorinated Dibenzo Dioxins (PCDD) and Poly-Chlorinated Dibenzo Furans (PCDF) to the highly studied 2,3,7,8-Tetrachlorodibenzodioxin (TCDD) to assess risks of exposure to mixtures of PCDDs and PCDFs.

Response 59: *The TEQ approach and associated TEFs were developed as a practical and scientifically defensible approach for addressing risks associated with complex environmental mixtures of dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs). EPA acknowledges that there are uncertainties associated with the use of the TEQ approach. However, despite the uncertainties, the SAB concluded that the use of TEFs is "clearly justifiable" from a public health standpoint. EPA believes that the comprehensive review of the values of the TEFs called for by the SAB was accomplished by a panel of experts organized by the World Health Organization (WHO), who reviewed all the available data on the relative toxicities of dioxin-like compounds. The WHO review, which was recently published in* Environmental Health Perspectives *(Van den Berg et al., 1998), resulted in recommendations for changes to the values of several of the TEFs. The WHO TEFs reflect a consensus of the international community and, for this reason, were used by EPA in the risk assessment for the final rule.*

***Comment 60*:** The commenter maintains that risk assessments should address the toxic effects of individual congeners on human health, particularly the noncarcinogenic effects referred to in the EPA study *Estimating Exposure to Dioxin-Like Compounds*. The commenter states that it is probable that the relative toxicities of individual congeners will be different from the Toxic Equivalence Factors (TEF) when more is learned about the noncarcinogenic health effects. For this reason, the commenter supports a total dioxin MACT emission standard for HWCs in addition to a TEQ MACT standard.

Response 60: *EPA considers that risk values for each dioxin congener based on appropriate toxicity studies would be preferable to the TEF approach; however, sufficient information is not available to conduct separate quantitative risk assessments. As indicated in the response to the previous comment, the TEQ approach and the use of TEFs were developed as a practical and scientifically defensible approach for addressing risks associated with complex environmental mixtures of dioxins/furans. As also indicated previously, EPA acknowledges that there are uncertainties associated with the*

use of the TEQ approach. However, despite the uncertainties, the SAB concluded that the use of TEFs is "clearly justifiable" from a public health standpoint.

Section 33
RSR Corporation
RCSP-0096

Comment 333: *(This Comment is not a part of the original comment for Docket RCSP-0096. There is not another docket for RSR Corporation. An identical comment is given in Docket RCSP-0128, Chemical Manufacturers Association.)*

The commenter states that EPA seems to have twisted science in interpreting mercury background fish data. On 17478, EPA states that "EPA collected data on chemical residues in fish taken from 388 locations nationwide and found that at 92 percent of the locations, fish contained detectable levels of mercury." EPA further stated that "[t]he highest concentration, 1.8 ppm, was measured at a remote site considered to represent background conditions. Similar results have been obtained in other studies, strongly suggesting that long-range atmospheric transport and deposition of anthropogenic emissions is occurring."

The commenter provides an attachment with a summary table for a study conducted by U.S. Geological Survey on toxic metals in U.S. surficial soil. In this study, samples of soils were taken, at a depth of approximately 20 cm from the surface, from locations about 80 km apart throughout the continental United States. Hence, the commenter notes that data represented soil characteristics without the impact of atmospheric deposition. The commenter refers to this tables to show that mercury as well as all other toxic metals naturally exist at low levels in the earth's top soil. The commenter further states that their existence has nothing to do with " long-range atmospheric transport and deposition of anthropogenic emissions."

The commenter feels that EPA's risk characterization for this proposal, by crediting the highest concentration that was observed in the background fish samples as due to long-range atmospheric transport and depositions, has clearly violated Administrator's Browner instruction that "[r]isk assessments should be transparent, in that the conclusions drawn from the science are identified separately from policy judgments." The commenter states that if long-range atmospheric transport and deposition is the true reason for a given level of mercury, the observed concentration level would be uniformly distributed over a very wide area, rather than producing the highest background concentration at an isolated remote area, and concludes that EPA's statement seems to be trying to twist science to justify a policy decision.

> ***Response 333:*** *EPA believes there is ample evidence that long-range transport of anthropogenic emissions of mercury are contributing to mercury levels in remote areas. As noted by Fitzgerald et al., 1998 ("The Case for Atmospheric Mercury Contamination in Remote Areas"), recent experimental results indicate that local scale geochemical processes alone cannot explain the mounting number of lake sediment and peat profiles*

showing substantial increases of mercury flux during the past century. Atmospheric and aquatic cycling of mercury and the bioaccumulation of monomethylmercury in aquatic systems are driven by complex chemical and biological reactions involving trace amounts of mercury. Analytical advances in measuring environmental mercury at trace levels have greatly narrowed estimates of natural mercury fluxes and support the case for long-range atmospheric transport of mercury to remote areas. The similar timing and magnitude of recent increases and a concordance with spatial trends in measured mercury deposition strongly support that long-range transport of anthropogenic mercury is the cause of increasing mercury concentrations and fluxes in the sediments of lakes and sparsely populated regions that are not affected by localized human-related sources of mercury.

As explained in EPA's 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), mercury cycles in the environment as a result of natural and anthropogenic activities. Most of the mercury in the atmosphere is elemental mercury vapor, which circulates in the atmosphere for up to a year and can be dispersed and transported thousands of miles from the source of emission. A computer simulation of long-range transport of mercury suggests that about one-third of U.S. anthropogenic emissions are deposited through wet and dry deposition within the lower 48 states. The remaining two-thirds is transported outside of U.S. borders, where it diffuses into the global reservoir. The highest deposition rates from anthropogenic and global contributions are predicted to occur in the southern Great Lakes and Ohio River Valley, the Northeast, and scattered areas in the South. The location of sources, the chemical species of mercury emitted, and climate and meteorology are key factors in mercury transport and deposition. The flux of mercury from the atmosphere to land or water at any one location is made up of contributions from the natural global cycle, including reemissions from the oceans, regional sources, and local sources.*

For the final rule, EPA conducted a quantitative assessment of risks from mercury to both human and ecological receptors. The mercury analysis for the final rule evaluated impacts from mercury emissions on local bodies of water in the immediate vicinity of HWCs. However, EPA did not evaluate the specific impacts resulting from long-range transport of HWC emissions beyond local study areas for the final rule.

Section 34
Safety-Kleen Corp.
RCSP-0108

***Comment 33*:** The commenter believes that one of the reasons EPA overstated risks in developing this proposed rule is the Agency's use of a flawed indirect risk assessments methodology. The commenter notes that the risk assessment protocols used by EPA have been criticized by EPA's own Science Advisory Board, which pointed out that the protocols included poor information on upsets, lacked measured data, ignored key additional data points, and failed to assess unique differences pollution sources.

Response 33: *The risk assessment for the final rule is based on methodology presented in the 1990 indirect exposure document,* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IEM) *(U.S. EPA, 1990) and the 1993 Addendum to the 1990 indirect exposure document (U.S. EPA, 1993a). These methods were further updated with respect to dioxins and mercury. For dioxins, the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist of the procedures published in the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. For mercury, the assessment followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model developed especially for that report. Human exposure factors were obtained from the 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a), including consumption rates for home-produced foods and recreationally caught fish. For lead, EPA used the Integrated Exposure Uptake Biokinetic Model (IEUBK) (U.S. EPA, 1994c) to estimate blood lead concentrations in children. Risks from both cancer and noncancer effects were assessed.*

The MACT rule making effort covers HWC stack emissions. Emission rates used in the risk analysis are based on facility-specific compliance or trial burn source tests. For those facilities for which data were missing, parameter values were imputed based on data available for similar sources. Startup, shutdown, or malfunction (upset) conditions were not reflected in the data.

Although EPA acknowledges the uncertainty of the models, EPA believes the models represent the best analysis tools available at the present time. EPA has included a comprehensive discussion of the uncertainties in its characterization of risks for the final rule. The draft Addendum was reviewed by EPA's Science Advisory Board, as indicated by the commenter. EPA intends to respond to the comments of the SAB in an appropriate

forum. EPA notes that the SAB also reviewed the MRTC, and EPA finalized that report in December 1997, taking into account the SAB's comments.

***Comment 34*:** The commenter notes that for the two HAPs EPA has decided to regulate BTF (dioxin/furan and mercury), EPA relied on two studies that have received considerable criticisms: the dioxin reassessment and draft *Mercury Study Report to Congress*. The commenter states that numerous commenters have identified flaws in both data and methodology in these reports and neither appear sufficient to rely on for the purposes of making accurate assessments of risk.

Response 34: *The establishment of NESHAP for sources that emit HAPs is required by Sections 112(c) and 112(d) of the Clean Air Act. The CAA directs EPA to set standards for stationary sources that are major sources of HAPS as defined in the CAA. Dioxins and mercury are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For existing sources, EPA is required to set the emission standard based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources. However, EPA may establish emission standards that are more stringent than this.*

Sections 3004(a) and (q) of RCRA mandate that standards governing the operation of HWC facilities be protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate whether the MACT standards satisfy this requirement in order to determine what, if any, RCRA standards for emissions from these sources may be needed. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

For the final rule, EPA is setting the emission standards for mercury at the MACT floor for all source categories. For dioxins, the final rule sets the emission standard at the MACT floor except for incinerators equipped with waste heat recovery boilers and lightweight aggregate kilns, for which a beyond-the-floor standard is established, as explained in the preamble to the rule.

The risk assessment for the final rule is based on methodology presented in the 1990 indirect exposure document, Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions (IEM) *(U.S. EPA, 1990), and the 1993 Addendum (U.S. EPA, 1993a). These methods were further updated with respect to dioxins and mercury. For dioxins, the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment were used. These consist of the procedures published*

in the 1994 Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. For mercury, the assessment followed the general modeling approach developed for the 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model developed especially for that report.*

EPA believes that the technical information gathered as a part of the ongoing dioxin reassessment, as well as that from the mercury study, are the best information currently available. The SAB commended EPA for its work on the dioxin exposure document, calling it "a very credible and thorough job." Regarding the mercury study, the SAB said the major findings of the report "are well supported by the scientific evidence."

***Comment 36*:** The commenter feels that EPA's estimates of risk lack important upcoming data, and that EPA has proceeded to propose costly BTF standards while admitting that the risk information it is relying on is incomplete and may be improved in the future. Specifically, the commenter points out that there are several ongoing activities that are likely to provide EPA with significantly better estimates of the health and environmental risks associated with hazardous waste combustion emissions. The commenter mentions the National Research Council (NRC) Waste Combustion Study and notes that EPA is aware that the NRC is in the midst of a comprehensive evaluation of hazardous waste combustion due to be released in September 1996. The commenter points out that the NRC's work is being funded by EPA, the Department of Energy, and the Agency for Toxic Substances and Disease Registry (ATSDR) and that the study will provide additional and needed expert assessment on hazardous waste combustion health effects. The commenter points out that the NRC study is of particular importance given the criticism over EPA's indirect risk assessment protocols, and that the panel of experts convened by NRC for its study offered EPA the opportunity to examine its health effects assumptions before issuing the proposal, thereby ensuring that the final MACT regulation is immune from such criticisms.

Response 36: *As of this time, the NRC report,* Waste Incineration and Public Health *(Committee on Health Effects of Waste Incineration, National Research Council) is a forthcoming title from the National Academy Press. EPA looks forward to the publication of this report.*

***Comment 38*:** The commenter notes that EPA acknowledges in the proposal that much of the information used to derive the risk estimates for dioxins was taken from the Agency's draft dioxin reassessment documents (61 FR 17371). The commenter states that EPA also acknowledges that there are a considerable number of uncertainties associated with the dioxin reassessment, that EPA has received numerous comments on the draft, and that EPA may revise its assessment of either the toxicity or exposure associated with dioxins. Given that the high costs associated with the MACT proposal are due in great part to meeting the proposed dioxin floor and BTF standards, the commenter suggests that EPA coordinate the MACT rulemaking with its review of the public input on the dioxin reassessment.

The commenter points out that if that public input requires EPA to revise the reassessment, EPA should not only incorporate those findings into the final rule, but should reissue the revised MACT standards for public comment. Given that the baseline dioxin risks in the proposed rule are low even using the current dioxin reassessment, the commenter believes the drawbacks of delaying the final rule by reproposing based on a revised dioxin reassessment are minimal. The commenter also notes that EPA has relied on the draft Mercury Report to Congress, which is undergoing significant revisions, and any revised health data from the revision should be reconsidered in reproposing these standards.

Response 38: *For dioxins, EPA used the site-specific modeling approaches developed for EPA's ongoing dioxin reassessment. These consist of the procedures published in the 1994* Estimating Exposure to Dioxin-like Compounds *(external review draft) (U.S. EPA, 1994a,b) and subsequently updated for the final HWC rule. EPA considers that technical information gathered as a part of the reassessment is the best data currently available and relied on this information in the proposed and final rule. EPA acknowledges that the Science Advisory Board had many comments and concerns regarding the draft* Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds *(U.S. EPA, 1994d,e). EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the Science Advisory Board. EPA intends to respond to the comments in an appropriate forum.*

For mercury, the assessment followed the general modeling approach developed for the 1997 Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c) and used the IEM-2M surface water model and methylmercury reference dose developed especially for that report. The MRTC has been extensively reviewed. EPA's Science Advisory Board, in its review of the mercury study, endorsed retention of the RfD based on the Iraqi study of methylmercury contaminated seed grain, calling the data "overwhelmingly supportive," at least until the ongoing Faeroe and Seychelles Islands studies have progressed much further and been subjected to the same scrutiny as the Iraqi data. The SAB concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish as well as by experimental animal data.*

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Dioxins and mercury are singled out for regulation under MACT standards in Section 112(c)(6). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA, and was done to determine if the technology-based standards are protective enough to satisfy RCRA, or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the

MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Section 35
Shell Oil Company
RCSP-0152

***Comment 368*:** The commenter cites the preamble and states that EPA does not currently possess knowledge of the health effects due to exposure to combustion products from hazardous waste incinerators. The commenter believes that since EPA has no evidence that the current standards are unprotective, it is very premature to set further, more restrictive emission standards. The commenter suggest that EPA wait until a risk assessment is performed to determine the safe exposure levels. Until then, the commenter feels EPA is setting standards for substances based on very conservative estimates that may or may not be valid.

Response 368: *The establishment of NESHAP for sources that emit HAPs is required by Sections 112(c) and 112(d) of the CAA. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards. However, Sections 3004(a) and (q) of RCRA mandate that standards governing the operation of HWC facilities be protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate whether the MACT standards satisfy this requirement in order to determine what, if any, RCRA standards for emissions from these sources may be needed.*

EPA performed a risk assessment for the HWC rule is designed to answer public policy questions about the general protectiveness of the MACT standards and to quantify the benefits of the rule at the national level, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA, and was done to determine if the technology-based standards are protective enough to satisfy RCRA, or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Section 36
Sierra Club
RCSP-0085

***Comment 82*:** The commenter states that EPA failed to discuss the potential for synergistic interactions between acid gases, which irritate and damage lung tissue and other mucus membranes, and the enhancement of transport of toxic chemicals via particulate inhalation, which may result when lung tissues are damaged.

Response 82: *Although interactions in response to exposures to chemical mixtures can be important, there is little reason to expect synergism in responses to mixtures of acid gases. Rather than synergism, EPA expects an additive response due to similarities in the mode of action. Chlorine gas can be converted to hydrogen chloride in the atmosphere, thereby further limiting the potential for nonadditive interactions.*

For the risk analysis for the final rule, EPA characterized the potential risk from chemical mixtures using a hazard index as the risk descriptor. An inhalation hazard index was calculated by summing the individual inhalation hazard quotients for each chemical constituent for which inhalation exposures were assessed, including hydrogen chloride and chlorine. EPA's analysis indicates that there is little potential for risk from exposures to HCl or chlorine, either individually or as a mixture.

For the final rule, EPA evaluated risks associated with particulate matter based on epidemiological studies of sensitive subpopulations (i.e., asthmatics and the elderly). While EPA did not specifically examine the issue of interactions between acid gases and inhalation of particulates, acidic gases such as sulfates and nitrates are normally present with particulate matter in ambient air. To the extent that interactions are occurring and contributing to adverse health effects, these effects may exert an influence on epidemiological studies that examine the association between PM levels and the incidence of respiratory disease.

***Comment 647*:** The commenter recommends that, given the number of HWCs located in communities with significant black populations and communities with below-average per capita income, EPA specifically evaluate the increased risk of lead poisoning posed to black and poor communities. Black and poor children are known to be at higher risk than white children because they live in older homes with deteriorating lead paint. Aging lead paint can expose children to lead in both house dust and in soil surrounding their homes, and maternal blood lead levels may also vary with race and income. A set of ATSDR data is included by the commenter (ATSDR, 1988, *Nature and Extent of Lead Poisoning: A Report to Congress)* that lists various data that may be used in calculating children's blood levels as it relates to race and income. The IEUBK model, version 0.99d, is used by EPA to

determine the concentration of lead in blood integrates children's exposures to lead from air, diet, drinking water, soil, indoor dust, paint, and maternal blood to predict a blood-lead level for a hypothetical child or population of children. The commenter states that it is well within EPA's ability to assess excess risk posed to black and poor communities by lead emissions.

The commenter notes that the town of Alpena, Michigan, has substantial background lead contamination, apparently from industrial sources. The commenter recommends this town as one that should be evaluated.

Response 647: *EPA has long recognized lead as an environmental toxicant of great concern. As a neurodevelopmental toxin in children, lead is a particular concern to minority and low income persons who may be among the most highly exposed. CDC's National Health and Nutrition Examination Surveys (NHANES), an ongoing series of national examinations of the health and nutritional status of Americans, have been the primary source for monitoring blood lead levels in the U.S. Data from NHANES indicate that blood lead levels in the U.S. population ages 1 to 5 were more likely to be elevated among those who were poor, non-Hispanic black, living in large metropolitan areas, or living in older housing. The NHANES data indicate that although the blood lead levels of 4.4 percent of all children ages 1 to 5 are above 10 micrograms per deciliter (µg/dL), the percentage increases to 8.0 percent for low income households and 11.2 percent for blacks (CDC, 1997).*

EPA acknowledges the limitations its risk assessment for lead, which focused on risks to the general population. Given this focus, it is difficult to draw conclusions about the risks to minorities and low income families. EPA recognizes this as an important uncertainty in the risk assessment and has identified it as such in both the preamble to the final rule and the risk assessment background document. At the same time, EPA's analysis shows that lead exposures from HWCs are relatively small in comparison with other sources of lead and that the final rule will further reduce lead exposures from HWCs.

***Comment 582*:** The commenter notes that it has been the contention of the environmental justice community that certain poor and/or minority communities have been disproportionately burdened with sources of pollution. The commenter states that, in an effort to address this concern, EPA has in some cases considered multiple pollution sources in evaluating the impact of facilities. The commenter cites a case study of the East Baton Rouge Parish, Louisiana, as presented to EPA at the Dioxin Reassessment Hearings, December 13, 1994, Arlington, Texas, by Mrs. Florence T. Robinson. She described a community with "six major petrochemical plants, a lead-smelter, a coke plant, a Superfund Site…a hazardous waste incinerator, and numerous hazardous waste landfills"; her comments are included for the record.

The commenter recommends that EPA evaluate the cumulative and synergistic effects of multiple-source pollution on communities that have been selected to host HWC facilities.

Response 582: *The risk analysis for the HWC rule was designed to answer public policy questions concerning the MACT emission standards for HWCs. Such standards are established pursuant to the technology provisions of Section 112(d)(2) and (3) of the CAA. EPA performed a risk assessment in order to evaluate whether the MACT standards for HWCs are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary.*

Other provisions of the CAA address air toxics concerns more generally. EPA recently issued its Integrated Urban Air Toxics Strategy under Section 112(k) of the CAA. The strategy includes national, regional, and community-based initiatives to focus on multimedia and cumulative risks. The strategy also includes national air toxics assessments that will help identify areas of concern by inventorying emissions sources, monitoring air toxics in ambient air, and developing multimedia models and other exposure assessment tools.

The risk analysis completed for the final rule evaluated national risks resulting from the impact of individual HWC facilities within specific combustor categories on receptor populations—the analysis did not assess simultaneous impacts from multiple facilities. A multifacility assessment was not carried out because this kind of analysis requires the development, parameterization, and use of site-specific models that require the inclusion of a host of factors that account for the collocation of facilities. Uncertainty is introduced into the HWC risk analysis by not reflecting the simultaneous impact of emissions from multiple facilities on the same receptor populations, especially for those chemical constituents exhibiting a threshold for adverse effects, including noncancer human heath effects and ecological effects.

EPA believes that consideration of location-specific multiple facility impacts is more appropriately a part of facility-specific permitting actions. Where the circumstances warrant it, permit writers may, on a case-by-case basis, establish additional permit conditions for HWCs as may be necessary to protect human health and the environment under the omnibus provisions of Section 3005(c)(3) of RCRA.

***Comment 84*:** The commenter feels that the full impact of dioxin emissions from HWCs on public health has yet to be assessed because EPA has not yet compared the locations of hazardous waste-burning sources to the most significant receptors, farm animals, on a national basis. The

commenter cites EPA estimates that hazardous waste-burning sources represent about 9 percent of total anthropogenic emissions of dioxins in the U.S. and notes that in preliminary analyses, EPA attributes about 3/4 of the background levels of dioxin in Americans to beef and dairy ingestion. The commenter points out that it does not follow that because HWCs contribute 9 percent of emissions that they also account for 9 percent of the risk posed by beef and dairy ingestion. The commenter notes that EPA states that the "impact of emissions on exposure and risk depends on the relative geographic locations of the emission sources and receptors which contribute to exposure and risk, primarily farm animals." The commenter points out that only by comparing the locations of cattle country relative to the locations of hazardous waste combustion can one get a realistic picture of the potential impact of hazardous waste combustion on our nation's beef supply. The commenter provides a table that compares the location of HWCs to a
list of the nation's top cattle-producing states.

The commenter remarks that cattle country, defined here as the top 6 cattle-producing states (Texas, Nebraska, Kansas, Oklahoma, Missouri, and California), produces 40 percent of the nation's cattle, and that the table provided indicates that 1/4 of the nation's commercial HWC facilities (23/93) are located in the six states comprising cattle country. Further, the commenter notes that both of the nation's commercial dioxin incinerators and a Superfund dioxin incinerator (Times Beach, MO) are located in the cattle states. Of particular concern to the commenter is the fact that nearly half of the HWCKs (11/25) are located in cattle states; the commenter notes that HWCKs burn enormous amounts of waste commercially and are considered significant dioxin sources by EPA. The commenter also points out that although the number 4 cattle state, Oklahoma, does not host a commercial HWC facility, combustors are located along the Arkansas and Kansas borders, and Tulsa maintains a large municipal waste incinerator as well.

The commenter performed a similar analysis on the location of combustion facilities with regard to the top 8 dairy states (WI, CA, NY, MN, PA, TX, MI, OH) and found that 1/4 of waste combustion facilities were located in these top dairy states as well. However, unlike the case in the cattle states, one type of facility did not predominate in dairy country. The commenter feels that due to the disproportionate location of commercial hazardous waste combustion facilities including dioxin incinerators in cattle and dairy states, the impacts of HWCs on the nation's beef and dairy supply must be much greater than might be estimated from their relative contribution to the nation's total dioxin emissions.

The commenter requests that EPA reevaluate the risk based limits set for dioxin emissions by combustors especially cement kilns vis-a-vis their disproportionate location in cattle country.

> ***Response 84:*** *For the final rule, EPA increased the number of HWCs analyzed in order to ensure that the results of the risk assessment would be representative of all HWCs. EPA used stratified random sampling to select 66 additional facilities for analysis, in addition to 10 of the 11 facilities analyzed at proposal (the 11th facility analyzed at*

proposal has ceased to burn hazardous waste and is undergoing RCRA closure). Therefore, a total of 76 active HWC facilities were analyzed for the final rule (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). This group represents more than 40 percent of the HWC facilities covered by the rule. Of these, 20 are located in the 6 states identified by the commenter as "cattle country" and 29 are in the states the commenter identified as the top 8 dairy states.

EPA determined livestock populations for each of these HWCs using U.S. Census block-group-level data and Census of Agriculture county-level data. The U.S. Census data provided detailed estimates of the number of farm households located within each of 16 sectors within a 20-km study area radius used for site characterization. As explained in the risk assessment background document for the final rule, these sector-level estimates were combined with Census of Agriculture data to estimate the total number of livestock located within each sector, including beef cattle, dairy cattle, and hogs,. This information was used to estimate cancer incidence in the general population associated with dioxin exposures resulting from consumption of agriculture commodities raised within the study areas. In addition, EPA assessed the risk to individuals in the local population who reside within the study areas and consume home-produced meat and milk. EPA believes that these analyses address the commenter's concerns regarding HWCs located in agricultural areas.

Comment 584, 85: The commenter states that EPA has failed to assess the impact of metal emissions on crop yield, despite the fact that it has been known for some time that metals will decrease crop yield. The commenter includes an old table of data on the effect of various levels of metal pollutants on various crops to illustrate the type of information that EPA should have gathered. The commenter notes also that contaminated fodder may adversely affect various grazing animals or confined herds and provides as an example a case of Midlothian, Texas, in which a leased agricultural field adjacent to a Chaparral Steel plant was found by the Texas Natural Resource Conservation Commission (TNRCC) to produce hay that could potentially cause adverse health effects in grazing cattle and in humans consuming the grazing cattle. The commenter notes that the origin of the contamination was not identified by TNRCC; however, both the steel plant and the adjacent HWCK (TX Midlothian, TX) emit metal pollutants. The commenter included the TNRCC report so that EPA can evaluate the potential risk to farmland posed by combustion facilities.

The commenter requests that EPA assess the risk to the productivity of farmland posed by the emissions that would be released under this proposed rule. The commenter requests that EPA quantify costs associated with any decreased productivity of nearby farmlands and establish how these costs will be recovered by the agricultural sector.

Response 584, 85: *EPA performed a screening-level ecological assessment to assess the potential ecological risks from HWC emissions. In this approach, ecotoxicological criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water. These criteria were then compared to model-predicted media concentrations in order to assess the potential for ecological risk. The ecological assessment evaluated all chemical contaminants that were assessed in the human health assessment. This includes dioxins and furans, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, lead, manganese, mercury, nickel, selenium, silver, and thallium. In addition, an ecological assessment was performed for copper.*

The ecological risk assessment served as a screening-level analysis designed to identify the potential for adverse effects. The process was based on current EPA guidelines for ecological risk assessment and began with the selection of assessment endpoints (e.g., the actual environmental values to be protected) that reflect the management goals for the analysis and relate to (1) a valued ecological entity such as a wildlife species, and (2) an attribute of that entity that is important to protect (for example, reproductive fitness). EPA selected a suite of ecological receptors that represented major trophic elements of ecosystems into which constituents may be released based upon (1) spatial distribution of chemical stressors in relation to the receptor and (2) the availability of data with which to assess the risks to that receptor.

The terrestrial plant community was among the assemblages of ecological receptors evaluated for the final rule. For the terrestrial plant community, toxicological benchmarks were identified from a summary document prepared at the Oak Ridge National Laboratory (ORNL): Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Terrestrial Plants: 1997 Revision *(Efroymson et al., 1997). The measurement endpoints generally consisted of growth and yield parameters. Such endpoints are the most common class of response reported in phytotoxicity studies and, therefore, allow for criterion calculations for a large number of constituents. Effects on growth and yield represent ecologically significant responses both in terms of plant populations and, by extension, the ability of producers to support ecological receptors at higher trophic levels.*

Comment 89: The commenter believes that EPA's failure to include mercury and copper in the ecological risk assessment constitutes a significant failure by EPA to perform a reasonable evaluation.

Response 89: *As described in the response to the previous comment, EPA conducted a screening-level ecological assessment in which ecotoxicologic criteria protective of*

various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, and the soil community, were developed. Criteria were developed for soils, sediments, and surface water and compared to model-predicted media concentrations to assess the potential for ecological risk. The chemical constituents evaluated included mercury and copper, as suggested by the commenter, as well as 2,3,7,8-TCDD and other chlorinated dioxins and furans, antimony, arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, selenium, silver, and thallium. EPA assessed the ecological risks from mercury in aquatic ecosystems using wildlife criteria for methylmercury developed for EPA's 1997 Mercury Study Report to Congress *(U.S. EPA, 1997c).*

***Comment 152*:** The commenter notes that dioxin, in particular, is known to affect fish and wildlife populations and that current scientific evidence indicates that presence of the Ah receptor indicates the susceptibility of an organism to dioxin and dioxin-like compounds. The commenter points out that the Ah receptor has been found in all vertebrates tested except agnathans, primitive jawless aquatic creatures with cartilaginous skeletons which include hagfish and lampreys. The commenter states that fish are among the most susceptible vertebrates, and that a lethal dose in fish fry or eggs may be an order of magnitude lower than a lethal dose in juvenile fish. The commenter states that, given the extremely low survival rates of eggs to adult in fish, even a slight reduction in egg survival rate can produce precipitous drops in population levels in sensitive species. Further, the commenter cites a source indicating that due to the steep dose/response curves for TCDD and fish, there is likely little difference in exposures between no effects on populations and very severe effects. Also, the most sensitive and most heavily exposed fish and wildlife species appear to be at the top of aquatic food webs.

The commenter notes, thus, while risk assessment depends upon health effects demonstrated by individuals, it must be understood that adverse effects for fish and wildlife may be even more profound at the population level. Further, the commenter notes that populational changes within a given species may change an entire ecosystem through alterations in the food web; this could be translated into the loss of a sport fish population and the correlated reduced value of the aquatic system to society. Put more bluntly by the commenter, both the recreational value and the community economic benefit associated with recreational fishing are lost when a day on the lake yields a bucket of dioxin-resistant lamprey instead of a string of dioxin-sensitive trout.

The commenter cites a recent review article as containing many useful references for determining toxicity in various fish species. Unfortunately, for many species, low-level effects such as behavioral effects, immunological effects, or fertility effects are unknown: the most frequent measure of adverse effect is death. The commenter notes that, since fish populations may be damaged by sublethal effects, the available information does not allow full protection of fish populations or ecosystem integrity. The commenter cites the U.S. Fish and Wildlife Service (1994) recommendation of 10-12 ppt 2,3.7,8-TCDD in food items (prey) for birds and mammals to "protect" wildlife and states that this level

is considered protective of terrestrial wildlife. Similarly, the commenter notes that the U.S. Fish and Wildlife Service has recommended that the 2,3,7,8-TCDD level in water that is still protective of aquatic wildlife (fish) should be 0.01 ppt = 10 parts per quadrillion.

The commenter cites the proposed rule as indicating that the national median concentration of 2,3,7,8-TCDD in fish was 3 ppt and that the 90th percentile was 30 ppt TEQ. The commenter believes that these numbers indicate terrestrial wildlife is at significant risk when ingesting aquatic prey. The commenter cites the MACT risk assessment as indicating that long-term ecological impacts may results from the exceedence of water quality criteria for TCDD and congeners.

The commenter requests that EPA describe how it will modify the proposed rule to protect fish and wildlife or how the loss of fish, wildlife, critical habitat, and natural resources will be mitigated under this proposed rule. The commenter recommends that EPA comply with NEPA (the National Environmental Policy Act) by fully discussing alternatives to the proposed rule which could prevent this serious degradation of natural resources.

Response 152: *EPA recognizes that vertebrates (e.g., mammals, birds, fish, amphibians) are particularly sensitive to the effects of dioxins when compared to other nonvertebrate ecological receptors. The HWC screening ecological risk assessment (SERA) evaluated the risk to piscivorous mammals and birds because of their higher dioxin sensitivity compared to fish. These species are at the top of the aquatic food chain, and for this reason, EPA has a high level of confidence that other vertebrate species, including fish, will be protected as well. Fish were not specifically evaluated in the SERA because EPA has not yet finalized a national ambient water quality criteria (NAWQC) protective of fish, amphibians, and other freshwater community biota.*

Effects and exposure conditions can be used to specify water and sediment concentrations that are associated with varying levels of risk to both aquatic life and aquatic-associated wildlife. For wildlife, significant risk is potentially more widespread than for aquatic life. EPA has proposed a range of 0.6 to 3.1 pg/L 2,3,7,8-TCDD that indicates "low" risk to fish species. However, concentrations ranging from 0.008 to 0.04 pg/L represent "low" risk for mammalian receptors. These ranges suggest that mammalian species show higher sensitivity to 2,3,7,8-TCDD in water than do fish in freshwater ecosystems. Studies have shown that completely piscivorus wildlife, especially mammals, are at the highest risk from exposure to 2,3,7,8-TCDD (U.S. EPA, 1993b).

Ecotoxicity studies suggest that rainbow trout exposed to 2,3,7,8-TCDD showed decreased survival and growth at concentrations of 38 pg/L (Mehrle et al., 1988,"Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout"). Significant impacts to growth and reproductive endpoints were also indicated in eggs, fry, and juvenile stages of rainbow

*trout at surface water concentrations of 0.1 ppt (100 pg/L) (Helder, 1981,"Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on Early life stages of rainbow trout (*Salmo gairdneri, *Richardson)"). Using this information, the Fish and Wildlife Service (FWS) has proposed a protective concentration of 0.01 ppt (10 pg/L), an order of magnitude below the low-effects surface water concentrations indicated in these studies (Eisler, 1986, "Dioxin hazards to fish, wildlife, and invertebrates: synoptic review").*

Given these findings in rainbow trout, one of the more dioxin-sensitive fish species, "protective" surface water concentrations for fish have a high probability to impact mammalian wildlife receptors. This potential impact is due to the fact that high-risk surface water concentrations for mammals range from 0.08 to 0.4 pg/L (U.S. EPA, 1993b). Low-risk concentrations presented here are derived from no-effects thresholds for reproductive effects (mortality and young) in sensitive species. High-risk concentrations are derived from TCDD doses expected to cause 50 to 100 percent mortality in embryos and young of sensitive species.

***Comment 155*:** The commenter cites a recent EPA publication that indicates that there are currently 1,740 waterbodies under fish advisories in 47 states, the District of Columbia, and Samoa. The commenter includes the document *EPA Fact Sheet: Update: National Listing of Fish and Wildlife Consumption Advisories,* EPA-823-F-96-006, June 1996, as an attachment. Fish advisories for mercury have increased from 899 to 1,308 nationally from 1993 to 1995. The number of fish advisories issued for mercury contamination far outweighs the number of advisories issued for any other contaminant. Yet, despite the tremendous impact of the mercury contaminant on our nation's natural resources and the risk posed to human health by the contaminated fisheries, EPA failed to produce an ecological risk assessment for mercury emissions.

The commenter notes that in addition to omitting mercury from the ecological risk assessment, EPA omitted copper. The commenter points out that the National Contaminant Biomonitoring Program followed the concentrations of arsenic, cadmium, copper, lead, mercury, selenium, and zinc in U.S. freshwater fish from 1976 through 1984 and has published these results. Lead concentrations in fish tissue have shown a decline, and, to a lesser extent, so have arsenic, cadmium, and selenium. However, zinc, copper, and mercury are not declining. The commenter notes that with respect to health risk considering persistence, bioaccumulation, and toxicity, mercury and copper are among the top-five ranking metals (others being lead, cadmium, and selenium). Thus, the commenter feels that it appears that both mercury and copper present a particularly important regulatory challenge in that they are not declining in the environment and they pose significant ecological risk. The commenter includes mercury data for Oklahoma fish and requests that EPA produce an ecological risk assessment for copper and mercury.

***Response 155:** For the final rule, EPA assessing the ecological risks for both copper and mercury. As indicated in response to previous comments, EPA conducted a screening-*

level ecological assessment in which ecotoxicologic criteria protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community, were developed. Criteria were developed for soils, sediments, and surface water and compared to model-predicted media concentrations to assess the potential for ecological risk. For mercury, EPA assessed the potential for ecological risks in aquatic ecosystems using wildlife criteria for methylmercury developed for EPA's 1997 Mercury Study Report to Congress*. For copper, EPA used the final chronic value (FCV) developed for the National Ambient Water Quality Criteria (NAWQC) for the protection of freshwater aquatic life. The commenter is referred to the risk assessment background document for the final rule for a complete discussion of the ecological assessment (RTI, 1999).*

Comment 157: The commenter requests that EPA reevaluate the risk-based emissions limits for mercury vis-a-vis the environmental and human health risk posed by this toxic, persistent, and bioaccumulative pollutant.

Response 157*: The MACT standards are not risk-based. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Mercury is singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology. For existing sources, EPA is required to set the emission standard based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources. However, EPA may establish emission standards that are more stringent than this.*

EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 162*:** The commenter points out that the ecological and human health impact of multiple facilities is of particular concern. As an example, the commenter points out that along the Oklahoma-Kansas border, within 50 mile radius, there are HWCKs at Chanute, Fredonia, and Independence, Kansas. In addition, the commenter notes that a dioxin incinerator operates along the border at Coffeyville, Kansas. All of these facilities appear to impact the watershed area for the

Verdigris River, which drains part of the Tri-State Mining District where lead, zinc, and cadmium were mined for many years and which contains abandoned mines and ore-processing facilities. Also, a zinc smelter is located in Bartlesville, OK. The commenter notes that while cadmium appears to be declining, it has been relatively high in the Verdigris River. The commenter asks both what is the collective ecological impact of the four hazardous waste facilities on the recovering Verdigris River system and what is the collective human health impact of these four facilities on the people of Nowata County, Oklahoma, who fish the Verdigris River?

Response 162: *The risk analysis completed for the final rule evaluated national risks resulting from the impact of individual HWC facilities within specific combustor categories on receptor populations—the analysis did not assess simultaneous impacts from multiple facilities. A multifacility assessment was not carried out because this kind of analysis requires the development, parameterization, and use of site-specific models that require the inclusion of a host of factors that account for the collocation of facilities. Uncertainty is introduced into the HWC risk analysis by not reflecting the simultaneous impact of emissions from multiple facilities on the same receptor populations, especially for those chemical constituents exhibiting a threshold for adverse effects, including noncancer human heath effects and ecological effects.*

As a general matter, EPA believes that consideration of location-specific multiple facility impacts is more appropriately a part of facility-specific permitting actions. Where the circumstances warrant it, permit writers may, on a case-by-case basis, establish additional permit conditions for HWCs as may be necessary to protect human health and the environment under the omnibus provisions of Section 3005(c)(3) of RCRA.

EPA's risk analysis for the final rule included an assessment of risks for the three cement kilns and the incinerator identified by the commenter. For the HWC facilities in Fredonia, Independence, and Coffeyville, the analysis included an assessment of risks associated with exposures from the Verdigris River. For the HWC facility in Chanute, EPA assessed risks from the Neosho River which is closer to the facility. For all four of the facilities evaluated, the estimated risks associated with fish consumption were relatively low. Assuming a high rate of fish consumption characteristic of subsistence fishing, the highest cancer risks were not greater than 1×10^{-7} (dioxins) and the highest noncancer risks (hazard quotients) were not greater than 0.02 (methylmercury) for any of the four facilities. Noncancer ingestion risks from cadmium were not greater than 2×10^{-3} (hazard quotient). Even if the highest exposures from each of the four facilities were assumed to occur concurrently (a physical impossibility), the risks would be below any level of human health significance. Furthermore, EPA's ecological assessment found no evidence of a significant potential for ecological risks to aquatic ecosystems under the final MACT standards. Although the risk analysis for the final rule did not evaluate zinc due to a lack of adequate emissions information, EPA evaluated 15 other metals. On the

basis of the results of the risk analysis, EPA concludes that the combined impact of the HWC facilities in Chanute, Fredonia, Independence, and Coffeyville is not likely to warrant special concern.

***Comment 163*:** The commenter notes that while EPA has apparently assumed that impacts on endangered species would be localized and would be addressed through risk assessment, cement plants are frequently sited next to raw materials which includes limestone. The commenter states that limestone formations include caves and other natural features conducive to endemic species and which include sensitive and unique habitats. The commenter concludes that since cement kilns impact sensitive species and habitats nationally, EPA should assess the impact of this rule on sensitive species and their habitats.

The commenter cites a previous EPA acknowledgment that "for those cement plants for which the Natural Heritage Programs were able to provide data, almost half had observed occurrences of species of concern within a one-mile radius or ten miles downstream and a majority had observed occurrences within a five mile radius." In this case, "species of concern" included species listed as threatened or endangered at the federal or state level as well as federal or state highly ranked species. The commenter states that EPA concluded that the colocation of cement plants with karst formations that are mined for raw materials in the cement manufacturing process creates the potential for an increased frequency of occurrences of certain species and habitats. The commenter notes that many of the 115 cement plants are located in karst areas, which are most commonly underlain by limestone or dolomite rock and are characterized by features such as sinkholes, springs, caves, and sloping rocky terrain. These areas are important because:

- **#** These areas often support unique plant and animal species. Certain unique species of blind catfish and shrimp are also known to exist in underground solution cavities in the vicinity of some cement plants, as are endemic species of certain insects and crustaceans in the springs that emanate from surrounding karst features.

- **#** Karst areas often are conducive to certain types of habitats. For example, the limestone soils of the Edwards Plateau in Texas support the habitat, including oak-juniper woodlands, of the Black-capped Vireo, a bird listed as endangered both federally and in Texas.

The commenter believes that under the Endangered Species Act dioxin impacts on endangered species may result in unauthorized "take" or "adverse modification to critical habitat." The commenter points out that HWCKs, which are known to impact endangered species, are unpermitted facilities. This rulemaking would be the first federal equivalent of permitting the emissions which these facilities have been generating for many years under the "interim status" provisions. Therefore, the commenter believes that it falls upon this rulemaking to meet the requirements of the Endangered Species Act especially as it impacts previously unpermitted facilities.

Response 163: *The HWC screening ecological risk assessment (SERA) completed for the final rule did not include an explicit assessment of threatened and endangered species because this kind of evaluation requires examination of a host of factors (e.g., habitat loss), many of which are unrelated to the HWC rule. EPA believes that, as a general matter, an evaluation of potential impacts to threatened and endangered species is more appropriately assessed on a site-specific basis. For example, if during the course of permitting an HWC facility, a site-specific ecological assessment identifies a significant potential for ecological risk and the circumstances warrant it, permit writers may, on a case-by-case basis, establish additional permit conditions as may be necessary to protect human health and the environment under the omnibus provisions of Section 3005(c)(3) of RCRA. EPA notes, however, that any hypothesized impacts to threatened or endangered species endemic to karst terrain are more likely to be associated with mining activities than with the burning of hazardous wastes.*

The SERA for the final rule is a screening-level analysis designed to assess the potential for adverse impacts to ecological receptors. Among the management goals EPA selected for the analysis was the protection of reproductive fitness. Reproductive effects are directly related to population impacts and, as an assessment endpoint, are directly relevant to the question of impacts on threatened or endangered species. The ecological receptors chosen for analysis represent a broad diversity of commonly occurring species and EPA has little reason to believe these ecological receptors are more or less sensitive to chemical exposures than are threatened or endangered species. As a screening-level analysis, the SERA utilizes a number of conservative assumptions in evaluating wildlife exposures (e.g., use of no-effects levels for developing protective wildlife criteria, use of high-end rates of bioaccumulation of chemical constituents in prey, and assuming all of a predator's diet is made up of prey from contaminated areas). By making these conservative assumptions, the threshold level is set to protect the more sensitive species in the habitats surrounding HWC facilities. EPA believes that all of these factors work together so as to confer a considerable degree of protection to all ecological receptors, including perhaps threatened and endangered species. While the SERA provides insight into the potential for adverse ecological impacts, EPA acknowledges that the results of the analysis cannot be extrapolated to all potentially exposed receptors with certainty, and may not apply to those that are threatened or endangered. A complete discussion of the ecological assessment and its uncertainties is found in the risk assessment background document for the final rule (RTI, 1999).

The results of the SERA analysis indicate that the potential for ecological risks from HWCs is relatively low and that the MACT standards will further reduce the potential for risk. For this and other reasons, EPA believes that the issue of takings under the Endangered Species Act, as identified by the commenter, does not arise.

***Comment 164*:** The commenter states that the Missouri River System is an area of particular concern as it contains numerous sensitive habitats and endangered species including aquatic and cave species. HWCKs operate along the Missouri at Festus, Cape Girardeau, Clarksville, and Hannibal, Missouri. The commenter notes that the River Cement facility in Festus was found to be emitting, and probably generating, tremendous amounts of dioxin in its cement kiln dust in the studies reported in the *Report to Congress on Cement Kiln Dust*. The commenter notes that, in addition, EPA has sited a dioxin incinerator at Times Beach, Missouri, as a Superfund treatment facility. The commenter points out that, although not covered under this rule, Superfund facilities may emit significant pollutants and should be considered as part of any assessment of cumulative emissions. The commenter also states that EPA should assess the potential ecological impact of the HWCKs and the dioxin incinerator on endangered, threatened, or rare birds which feed on aquatic organisms (great egret, great blue heron, interior least terns) and the endangered mollusks of the Missouri. Pallid sturgeons and paddle fish, a candidate for federal listing, are big river fish that range widely in the Mississippi and Missouri Rivers. The commenter cites the Missouri Department of Conservation as supporting evaluation of possible impact to pallid sturgeon populations for any project that modifies big river habitat or can impact water quality. The commenter notes that EPA, however, has provided no assessment of probable damage already done to these sensitive populations and their habitats by cement plants along the Missouri or of future impacts from emissions under this proposed rule.

Response 164: *In the risk assessment for the final rule, EPA analyzed 15 of the 18 cement kiln facilities that are currently known to be burning hazardous wastes. Since proposal, a number of cement kilns have stopped burning hazardous wastes, including the facility at Festus, Missouri, identified by the commenter. Of the other facilities the commenter identified, only the Clarksville facility was analyzed by EPA for the final rule. The estimated risks from dioxin exposures associated with fish consumption for the Clarksville facility were relatively low and were projected to be further reduced under the final rule. Assuming a high rate of fish consumption characteristic of subsistence fishing, the highest cancer risk for this facility under the final rule was estimated to be* 1×10^{-6} *for a nearby tributary of the Mississippi River. Furthermore, EPA's ecological assessment found no evidence of a potential for ecological risks to aquatic ecosystems from dioxins.*

As explained int the risk assessment background document for the final rule (RTI, 1999), EPA conducted a screening-level ecological assessment (SERA) for HWCs. In this analysis, ecotoxicologic criteria were developed that are protective of various assemblages of ecological receptors, such as terrestrial mammals, the aquatic community, or the soil community. Criteria were developed for soils, sediments, and surface water and compared to model-predicted media concentrations in order to assess the potential for ecological risk. The ecological risk assessment served as a screening-level analysis designed to identify the potential for adverse effects. The process was

based on current EPA guidelines for ecological risk assessment and began with the selection of assessment endpoints (e.g., the actual environmental values to be protected) that reflect the management goals for the analysis and relate to (1) a valued ecological entity such as a wildlife species, and (2) an attribute of that entity that is important to protect.

Among the management goals EPA selected for the analysis was the protection of reproductive fitness. Reproductive effects are directly related to population impacts and, as an assessment endpoint, are directly relevant to the question of impacts on threatened or endangered species. While the protection of threatened or endangered species was not specifically one of the management goals of the ecological assessment, the ecological receptors chosen for analysis represent a broad diversity of commonly occurring species and EPA has little reason to believe these ecological receptors are more or less sensitive to chemical exposures than are threatened or endangered species. As a screening-level analysis, the SERA utilizes a number of conservative assumptions in evaluating wildlife exposures (e.g., use of no-effects levels for developing protective wildlife criteria, use of high-end rates of bioaccumulation of chemical constituents in prey, and assuming all of a predator's diet is made up of prey from contaminated areas). By making these conservative assumptions, the threshold level is set to protect the more sensitive species in the habitats surrounding HWC facilities. EPA believes that all of these factors work together so as to confer a considerable degree of protection to all ecological receptors, including perhaps threatened and endangered species. While the SERA provides insight into the potential for adverse ecological impacts, EPA acknowledges that the results of the analysis cannot be extrapolated to all potentially exposed receptors with certainty, and may not apply to those that are threatened or endangered. Additional uncertainty is introduced into the SERA by not reflecting the simultaneous impact of emissions from multiple facilities on ecological receptors. A complete discussion of the ecological assessment and its uncertainties is found in the risk assessment background document for the final rule.

***Comment 165*:** The commenter believes that EPA should assess the impact of multiple facilities, in particular, the four facilities on the Kansas-Oklahoma border (the incinerator at Coffeyville, KS, and the HWCKs at Fredonia, Independence, and Chanute, KS) on the Neosho River and its endangered catfish, the Neosho madtom. The range for this fish includes Craig and Ottawa Counties in Oklahoma; Cherokee, Labette, Neosho, Allen, Woodson, Coffey, Lyon, and Chase Counties in Kansas; and Jasper County, Missouri. The commenter notes that the Neosho River watershed and the deposition from the four HWC facilities overlap and states that while habitat destruction and alteration account for the decline of populations of the madtom over much of its former range, water pollution also has contributed to its decline.

Response 165: *The HWC screening ecological risk assessment (SERA) completed for the final rule did not include an explicit assessment of threatened and endangered species because this kind of evaluation requires examination of a host of factors (e.g., habitat loss), many of which are unrelated to the HWC rule. EPA believes that, as a general matter, an evaluation of potential impacts to threatened and endangered species and consideration of multiple facility impacts is more appropriately assessed on a site-specific basis in relation to facility-specific permitting actions. For example, if during the course of permitting an HWC facility, a site-specific ecological assessment identifies a significant potential for ecological risk and the circumstances warrant it, permit writers may, on a case-by-case basis, establish additional permit conditions as may be necessary to protect human health and the environment under the omnibus provisions of Section 3005(c)(3) of RCRA.*

For the final rule, EPA analyzed the potential impact on human health and the environment of the HWCs at Coffeyville, Fredonia, Independence, and Chanute. Human health risks were discussed in response to a previous comment. In addition, EPA assessed the potential for ecological risk of the four facilities. EPA analyzed the impact of the Chanute facility on the Neosho River, which is located in close proximity to the Neosho. EPA analyzed the impact of the HWCs in Coffeyville, Fredonia, and Independence on the Verdigris River, since these facilities are located in the watershed of the Verdigris River. The results of the ecological assessment indicate that, under the final rule, none of the HWCs EPA analyzed are expected to have any potential for risk to aquatic ecosystems for any chemical contaminant, including dioxins and mercury. The only exception was an exceedance of the ecotoxicological criteria for selenium that is of questionable ecological significance. The exceedance occurred for a single HWC, involved only one ecological receptor (the river otter), and affected only a small area. No exceedances were found for fish.

As indicated in response to a previous comment, one of the management goals EPA selected for the ecological assessment was the protection of reproductive fitness. Reproductive effects are directly related to population impacts and, as an assessment endpoint, are directly relevant to the question of impacts on threatened or endangered species. While the protection of threatened or endangered species was not specifically one of the management goals of the ecological assessment, the ecological receptors chosen for analysis represent a broad diversity of commonly occurring species and EPA has little reason to believe these ecological receptors are more or less sensitive to chemical exposures than are threatened or endangered species. As a screening-level analysis, the SERA utilizes a number of conservative assumptions in evaluating wildlife exposures (e.g., use of no-effects levels for developing protective wildlife criteria, use of high-end rates of bioaccumulation of chemical constituents in prey, and assuming all of a predator's diet is made up of prey from contaminated areas). By making these

conservative assumptions, the threshold level is set to protect the more sensitive species in the habitats surrounding HWC facilities. EPA believes that all of these factors work together so as to confer a considerable degree of protection to all ecological receptors, including perhaps threatened and endangered species. While the SERA provides insight into the potential for adverse ecological impacts, EPA acknowledges that the results of the analysis cannot be extrapolated to all potentially exposed receptors with certainty, and may not apply to those that are threatened or endangered. Additional uncertainty is introduced into the SERA by not reflecting the simultaneous impact of emissions from multiple facilities on ecological receptors.

Comment 614: The commenter notes that HWCs have a variety of fugitive emissions from pipes, vales, seals, and transfer equipment which may contribute significantly to the total emissions. In addition, HWCKs routinely experience "upsets" where control over emissions is lost and emissions belch out of the stack or backfire out of the back end of the kiln. These upset emissions are generally believes to be laden with metals. Risk assessments frequently skip fugitive and upset emissions; however, stakeholders who smell the escaping gases and have to clean the thick blanket of upset emissions off their windshields do not take these emissions lightly.

The commenter notes that other emissions are also associated with HWCs. Combustors are frequently part of "hazardous waste management facilities." Theses adjacent facilities include blending, storage, transfer, fuel transfer and storage, and cement kiln dust management units. Each of these adjacent facilities may have significant impacts on the environment and the local community. The EPA has already done assessment on cement kiln dust management units so these figures are available.

The commenter states that documented air damage cases have resulted from excess emissions resulting from upsets, fugitive emissions, and mechanical breakdown of air pollution control equipment. The commenter includes an attachment, Exhibit 5-17, Summary of Air Damage Case Findings, in support of this argument.

The commenter requests that EPA include emissions from associated facilities such as transfer, storage, and blending facilities, and cement kiln dust management units as well as fugitive and upset emissions in all risk assessments, both for human health and ecological impact.

The commenter also requests that EPA utilize the information in the above-referenced attachment, which details the air damage cases to complete the MACT risk assessment by including such emissions in a reassessment.

Response 614: *The risk analysis for the HWC rule was designed to answer public policy questions concerning the MACT emission standards for HWCs. Such standards are established pursuant to the provisions of Section 112(d)(2) and (3) of the CAA. These standards are intended to control air emissions during routine operations and are not*

specifically designed to limit emissions under extreme operational conditions. Under the general provisions of the MACT program, facilities are required to address startup, shutdown, and malfunction or upset conditions. Additionally, HWCs are already required to stop feeding hazardous wastes during process upsets (referred to as automatic waste feed cutoffs), startup, and shutdowns. While the MACT standards may lead to improvements in the air pollution control equipment required to meet the standards during hazardous waste operations, the rule would not specifically control emissions during periods when no hazardous waste is in the combustion chamber. Any air emissions during such times should be of relatively short duration and should be no greater than from any other combustion device burning a nonhazardous waste fuel. However, if process upsets are of such a magnitude or frequency to generate health and safety concerns, permit writers may, on a case-by-case basis, establish additional permit conditions as may be necessary to protect human health and the environment under the omnibus provisions of Section 3005(c)(3) of RCRA.

Emissions from waste management operations such as blending and storage are already subject to RCRA standards at 40 CFR 264 Subparts AA, BB, and CC, which cover process vents, equipment leaks, tanks, and containers. EPA is developing a regulatory program for the safe management of cement kiln dust (CKD) (see 60 FR 7366).

***Comment 168*:** The commenter notes that 1/3 of waste combustors are located in the six cattle states. Thus, the commenter views exposure of farm families through subsistence ingestion of home-grown beef as a likely scenario. The commenter points out that in addition, in this part of the country, a great deal of fishing occurs in farm ponds that are maintained to provide water for cattle and to control erosion. The commenter points out that these ponds provide a supplemental food source to farm families, and that anyone who has flown into Dallas has seen the sparkling vista of farm ponds across the rich cattle pasture around Dallas-Fort Worth. As an example, the commenter notes that in Pontotoc County, Oklahoma (location of Holnam Cement, Ada, OK), there are about 4,000 farm ponds distributed over 1,000 local ranches. The commenter indicates that native fish species in Oklahoma's farm ponds include bass, catfish, perch, and crappie, and that state farm pond stocking programs improve pond productivity by balancing the fish population. The commenter cites the Oklahoma Department of Wildlife Conservation as indicating that a sustainable annual yield per acre of farm pond for this region of the country is 7-10 lbs of bass and 100-125 lbs of bluegill, with an annually replenished yield of 200 lbs of catfish. The commenter also notes that, for ranchers, farm ponds can provide both recreation and sustenance.

As EPA guidance specifies that "parameters that are known to be highly correlated should be varied together" in risk assessment, the commenter feels that it is clear that farm families have both cattle and cattle watering holes, which double as fish ponds. The commenter suggests that exposure pathways for home-grown beef consumption and recreational fishing be combined.

Response 168: *For the final rule, EPA assessed the risks from exposures to both home-produced beef and fish raised in farm ponds. Risks from the consumption of home-produced beef were assessed for farmers that raise beef cattle commercially and farmers engaged in subsistence farming. Unlike commercial beef farmers, whose only dietary exposure was through consumption of home-produced beef; subsistence farmers were assumed to derive substantially all of their dietary intake from home-produced foods. These included consumption of home-produced beef, pork, poultry, fish milk, and eggs, as well as fruits and vegetables. As suggested by the commenter, the fish consumed by the subsistence farmer were assumed to be raised in farm ponds. The intake of farm-raised fish was assumed to be equivalent to the recreational fish intake of freshwater anglers.*

Comment 613**:** It has been assumed that chemicals interact additively. However, according to a recent publication in *Science*, "Combinations of two weak environmental estrogens, such as dieldrin, endosulfan, or toxaphene, were 1,000 times as potent in hER-mediated transactivation as any chemical alone." Thus, the commenter feels that EPA may have underestimated human health effects by several orders of magnitude in failing to account for greater-than-additive synergistic effects of environmental hormones. In addition, ecological models also fail to account for synergistic effects. Thus, damage from chemical pollutants to sensitive populations, sensitive habitats, and ecosystem function may be more severe than currently estimated.

Response 613: *The commenter is raising the issue of risks from chemical mixtures. EPA considered the risks from chemical mixtures by assuming additivity of response by route of exposure. Specifically, chemical-specific cancer risks were summed for oral (ingestion) exposures and for inhalation exposures. Similarly, noncancer risks (hazard quotients) were summed for oral exposures and for inhalation exposures. EPA views this as a conservative yet reasonable approach to assessing mixture effects since it considers possible portal of entry effects but not effects on specific organs or mechanisms of action. EPA notes, however, that for the purpose of assessing population level cancer risks (e.g., excess cancer incidence), EPA conservatively assumed that effects were additive across exposure routes (i.e., cancer risks from inhalation and ingestion exposures were summed).*

Other categories of individual-level and population-level risks are constituent-specific (e.g., individual PbB analysis for lead, margin of exposure analysis relative to background exposures for dioxins/furans and the PM analysis), so the issue of additive risks generally does not apply. EPA notes, however, that in assessing risks from dioxins and furans, EPA used the toxicity equivalence (TEQ) concept, which assumes additivity of response among the various 2,3,7,8-substituted congeners.

Except as noted above, synergistic and antagonistic interactions between chemical constituents were not specifically evaluated. EPA acknowledges the uncertainties related

to the possibility of nonadditive, synergistic or antagonistic effects between chemicals. However, at the present time, synergistic and antagonistic interactions have not been sufficiently characterized to allow consideration of these effects quantitatively for risk characterization purposes.

EPA believes that the additive risks generated for the final rule provide an adequate level of analysis for assessing the potential cumulative effect of multiple chemical exposures. EPA notes that cancer risks from HWCs appear generally to be dominated by risks from dioxins and furans. Therefore, the issue of multiple chemical exposures and chemical interactions with other classes of chemical compounds may be of lesser significance than it might otherwise be.

***Comment 170*:** The commenter requests that EPA reassess human health risks posed by inhalation of particulate matter and refers EPA to data and comments included in the following publication, which has been submitted by the authors for the record at the commenter's request: Natural Resources Defense Council, 1996, *Breath Taking: Premature Mortality Due to Particulate Air Pollution in 239 American Cities*, New York: NRDC Publications, 154 pages.

Response 170: *EPA recently revised its ambient air quality standards for PM and has put in place a national strategy for implementing the new standards. For HWCs, EPA has set technology-based emission standards using the MACT process, as explained in the preamble to the final rule. These include standards for PM. In developing the final rule, EPA examined the potential for reducing risks from ambient PM by controlling HWC stack emissions. In particular, EPA estimated the reductions in risk associated with a number of health endpoints, including:*

- *Mortality from long-term exposure and from short-term exposure*
- *Hospital admissions for all respiratory infections, congestive heart failure, and ischemic heart disease*
- *Chronic bronchitis*
- *Acute bronchitis, lower respiratory symptoms, and upper respiratory symptoms*
- *Work loss days, minor restricted activity days, and restricted activity days.*

The results of the analysis indicate that risk reductions are expected for a number of health endpoints, including reductions in mortality, hospital admissions, chronic bronchitis, and respiratory symptoms, although the reductions are modest relative to those achieved by controls on other sources of PM.

Comment 172: The commenter notes that EPA selected a figure from a Columbia River study to represent fish ingestion rates by subsistence fishers. However, the commenter notes that while the

Columbia River study includes subsistence fishers, it also includes recreational fishers and both fish consumers and individuals who never eat fish (46/500 adult respondents in the study). The commenter feels that the selection by EPA of the mean ingestion rate of 60 g/p/d from the Columbia River study is not protective of the subsistence fishers in the Population. The 90th percentile for the population is between 97.2 and 130 g/p/d. The maximum value given for the population is 389 g/p/d, a figure very close to the 95th percentile value of 338.8 g/p/d provided by Puffer (1981) and the 100th percentile value of 381 g/p/d figure provided by Pierce et al. (1981). The commenter feels that while the 90th percentile figure would not protect all consumers, it is a more conservative and realistic standard value for protecting subsistence fishers than the one selected by EPA. The commenter recommends that EPA revise the fish ingestion rate for subsistence fishers to the 90th percentile for the Columbia River Study, a figure more consistent with the empirical data and more conservative.

Response 172: *For the final rule, EPA used a fish ingestion rate of 70 g/day to represent fish consumption by individuals engaged in subsistence fishing. This is the value for the mean fish intake recommended by EPA in the 1997* Exposure Factors Handbook *(U.S. EPA, 1997a). EPA believes that this ingestion rate is representative of fish consumption by subsistence populations. This value is supported by a study by Wolfe and Walker (1987) (as cited in U.S. EPA, 1997a) of fish harvests by Alaska Natives in small communities in Alaska, as well as the Columbia River Basin study of Native American tribes (CRITFC, 1994). Also, because EPA conservatively assumed that subsistence fishing could take place at any of the more than 260 bodies of water EPA analyzed for the final rule and that each of these bodies of water could provide the entire dietary intake of fish for individual subsistence fishers, EPA does not believe it would be appropriate to also use an upper percentile on the fish ingestion rate for characterizing risks from subsistence fishing, as suggested by the commenter. EPA believes there is only a low probability that subsistence fishing actual occurs at many of the bodies of water it analyzed.*

For the final rule, EPA did not use high-end values for exposure factors such as fish ingestion. Instead, EPA generated cumulative distributions of risks to individuals, thereby eliminating the need to define exposure scenarios deterministically, in the manner done at proposal. In all instances, EPA either used central tendency values or conducted a full distributional analysis to account for the full range of exposure. EPA notes that an exposure parameter variability analysis was performed using Monte Carlo simulation for freshwater anglers exposed to mercury through consumption of recreationally caught fish This analysis considered the variability of fish intake rates in this population. The commenter is referred to the risk assessment background document for a full discussion of EPA's analysis (RTI, 1999).

Comment 173: The commenter cites an EPA announcement on October 23, 1995, that risks to infants and children are to be considered "consistently and explicitly" as a part of all risk assessments

developed for EPA decisions and policies. The commenter then cites the risk assessment as indicating that only consumption of soil, fruits and vegetables, and milk were modeled to highlight the child's increased consumption per body weight; therefore, the commenter concludes that EPA disregards the child's increased consumption nor body weight in the fish consumption scenarios.

The commenter notes that an amount of 0.35 g/d for a child age 0-6 is presented in the risk assessment and described as a "scaled adult value based on body weight." The commenter states, however, that if one reverses the EPA's arithmetic it is apparent that an error in calculation has been made. The commenter queries that if adults and children consume fish in direct proportion to their body weight as EPA assumes, then how much would a child (ages 0-6 in this EPA scenario) consuming 0.35 g/d of fish weigh, himself, if the 70 kg adult eats 30g/d? In answer, the commenter states that the hypothetical child age 0-6 modeled in this EPA scenario would weigh 0.8 kg less than 2 pounds. The commenter concludes that the EPA value for child fish ingestion rate clearly is erroneous and has resulted in the underestimation of exposure of children to pollutants via the fish ingestion pathway by several orders of magnitude.

Further, the commenter notes that the results of the MACT risk assessment indicate that the highest risk posed by dioxin in adult scenarios is posed by the subsistence fisher scenario. The commenter concludes that the risk posed to children by recreational and subsistence fish pathways is a critical piece of information which is missing from the risk assessment.

The commenter cites EPA guidance (EPA Office of Water, 1994, *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume 11, Risk Assessment and Fish Consumption Limits,* EPA 823-B-94-004, June 1994) as providing a child recreational fisher standard of 85 grams/person/day for ages 0-4. Although the commenter recognizes that this value is extremely conservative, the commenter proposes that EPA adopt a child fish-ingestion rate based empirical data found in a number of peer-reviewed studies discussed by the commenter.

The commenter acknowledges that because different studies on fish ingestion use such different methodologies, one cannot compare adult and child fish ingestion rates between different studies. However, the commenter cites three solid, peer-reviewed studies which contain both adult and child fish ingestion rates and states that utilizing the fish ingestion rates provided in these studies and converting the rates (grams/person/day, or gpd) to doses (grams fish/kg human body weight/day, or g/Kg/d) allows determination of the child's increased consumption per body weight of fish. The commenter's discussion of these studies follows.

In performing these calculations, it was assumed that adults weigh 70 kg. The weight for children of various age ranges was calculated by averaging the 50th percentile for the weight over the range of ages per EPA methodology and using EPA standard weight tables. The three studies are a study of Native Americans ingesting self-caught fish harvested from the Columbia River in Washington; a West et al. (1992) study of ingestion of fish from all sources (self- caught. store-bought, restaurant)

by Michigan anglers; and a Pao et al. (1982) survey of the general U.S. public (anglers and nonanglers) of fish ingestion from all sources.

The commenter presents the results for the central tendency values presented in the studies, either the average (West et al. and Pao) or the mean (Columbia R.). The commenter performed identical calculations for the maximum ingestion value (the 99th percentile value) given in each study and reports those results as well. The commenter presents a table of fish ingestion rate data with age range and calculated child body weight (Kg), child fish ingestion dose, and adult fish ingestion dose in grams fish ingested/kg human body weight/day (g/Kg/d) for three peer-reviewed studies.

The commenter points out that, as can be seen from the ratio of child/adult fish consumption on a per-body-weight basis, children receive about twice the "dose" of fish in each of the three studies. That is, children ingest about twice as much fish per unit body weight as adults. The commenter performed this same analysis on the maximum ingestion rates provided in the three studies, the 99th percentile, and obtained approximately the same results. Assuming that the 99th percentile represents fish ingestion rates among high-end subpopulations detected in each study, the commenter notes that one can conclude that both recreational and subsistence fisher children consume double the amount of fish as adults on a per-body-weight basis.

The commenter recommends the following:

- **#** The Columbia River study (which includes 194 children and includes subsistence fishers) be utilized to represent children exposed through the fishing pathways.
- **#** The 91.2 percentile value of 48.6 g/d be used as the child subsistence fish ingestion rate.
- **#** The 51 percentile value of 12.2 g/p be utilized for recreational fish ingestion rate. The study considered children ages 1-5 so some adjustments may be necessary. Alternatively, the child ingestion rate for recreational and subsistence fisher pathways could be set at twice the per-body-weight ingestion of adults.
- **#** EPA tighten the proposed risk-based standards for dioxin emissions if the reassessment of risks to children show unacceptable levels.

Response 173: *EPA agrees with the commenter that children consume more fish per unit body weight than do most adults. For the final rule, EPA assessed the risks to children from the consumption of fish related to both subsistence and recreational fishing activity. The value used in the risk analysis for the proposed rule (0.35 kg/dy) was intended to represent consumption of freshwater fish in the general population.*

For the final rule, EPA assessed risks to children in three age groups (0-5, 6-11, 12-19 years of age). EPA's fish ingestion rates for subsistence fishing in each of the three age groups were based on data provided in Chapter 10 of EPA's 1997 Exposure Factors

Handbook *(EFH) (U.S. EPA, 1997a). EPA used mean values for exposure parameters, including fish ingestion rates, consistently throughout the risk analysis.*

For subsistence fishing, data from the Columbia River Study give a mean fish intake rate of 19.6 g/day for children under 5 years of age. Using a body weight of 14.3 kg for children age 0-5, EPA calculated an ingestion rate of 1.4 g/kg/day. Because of the lack of data for 6- to 11-year-olds, EPA used the age 0-5 ingestion rate of 1.4 g/kg/day, along with a body weight of 30.7 kg, to calculate a total fish intake rate of 42 g/day for 6- to 11-year-olds. For children age 11-19, EPA estimated the fish ingestion rate from data on adults. Using the mean fish intake of 70 g/day recommended in the EFH, EPA derived an ingestion rate of 0.97 g/kg/day for adults The adult ingestion rate of 0.97 g/kg/day was used, along with a body weight of 58.3 kg to calculate a total fish intake rate of 57 g/day for children age 11-19.

For recreational fishing, EPA used data from Table 10-61 of the EFH, "Mean Fish Intake Among Individuals Who Eat Fish and Reside in Households with Recreational Fish Consumption." Data in this table were taken from a 1989 report by West et al., Michigan Sport Anglers Fish Consumption Survey. *Using the West et al. fish ingestion rate of 0.37 g/kg-day for the 0- to 5-year-old group and a body weight of 14.3 kg for this group, EPA calculated a total fish intake rate of 5.3 g/day for children age 0-5. The ingestion rate for the 6- to 11-year-old group was assumed to be the same as the value provided by West and coworkers for the 6- to 10-year-old group (0.28 g/kg/day). Using this ingestion rate and a body weight of 30.7 kg, EPA calculated a total fish intake rate of 8.6 g/day. The 12- to 19-year-old group was extrapolated using data from West et al. on the sample sizes and ingestion rates for the 1-5, 6-10, and 1- to 20-year-old age groups. This resulted in a fish ingestion rate of 0.15 g/kg/day and, using a body weight of 58.3 kg, EPA calculated a total fish intake of 8.7 g/day.*

For the final rule, EPA assumed that subsistence farmers raise fish in farm ponds. Because no other data are available on fish intake related to home consumption of farm-raised fish, EPA used the fish ingestion rates for recreational fishing, as described above.

EPA wishes to emphasize that the MACT standards are technology-based standards that are based solely on the factors made relevant under Section 112 of the CAA. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA in order to determine what, if any, RCRA standards for emissions from these sources may be needed.

***Comment 175*:** The commenter notes that risk assessment for exposure of breast-feeding infants is usually performed with the assumption that infants are weaned from the breast at 1 year. The commenter enclosed a data set in support of the argument that this assumption is unwarranted; the statistics collected by Ross Laboratories found that in 1992, 5.6 percent of infants were still nursing at 12 months. Thus, the commenter believes that EPA has failed to assess the risk posed to about 6 percent of American infants by dioxin and dioxin-like compounds. In addition, the commenter notes that the data show that at 5 to 6 months, infants of older mothers are more likely to be nursed. The commenter points out that if older mothers have accumulated additional dioxin in their breast milk, it may be these infants that are at greatest risk. The commenter also points out that infants at relatively high risk of exposure to dioxin would be nursing infants of women who consume fish. The commenter cites a survey of the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin in which it was found that nursing mothers or mothers who have nursed have between 0 and 10 fish meals per week with the 50th percentile at about 1 fish meal per week and the 90th percentile at about 3 fish meals per week. Within this population, 15 percent of infants are still nursing at 1 year of age (n = 99). At 24 months, 99.3 percent of infants are weaned. The commenter feels that by utilizing the 1-year limitation on assessing risks attended to nursing, EPA has failed to assess the risks posed to 15 percent of the infants in this Northwest Native American population as well. The commenter believes that all women should be able to safely nurse their infants for a full 2 years without having to "assess" for themselves whether this natural reproductive function poses a risk to the infant. The commenter further believes that it is EPA's responsibility to assess the risk to infants posed by dioxin and to ensure that infants are fully protected from adverse affects of contamination regardless of when weaning occurs.

The commenter cites the risk assessment as presenting a breast milk consumption rate of 0.80 g/d, although the reference cited in the risk assessment (*Estimating Exposure to Dioxin-Like Compounds, Vol. III*, 1994) gives a value of 0.80 kg/d. The commenter indicates that if EPA did indeed use this erroneous figure in determining the average daily dose to infants of dioxin, then the risk assessment has underestimated the ADD (average daily dose) by several orders of magnitude.

The commenter makes the following requests:

- That EPA correct the risk assessment if the erroneous figure was indeed used in the calculations
- That EPA assess the impact on infants of 2 years of nursing, especially by those subpopulations known to nurse for extended periods (i.e., older mothers who are at risk for elevated dioxin body burdens by virtue of their age, and Northwest Native American mothers who are at risk for elevated dioxin body burdens by virtue of their reliance on fish for sustenance or subsistence)
- That EPA tighten the proposed risk-based dioxin emission standard if the correction of this portion of the risk assessment demonstrates that nursing infants are placed at unacceptable risk levels.

The commenter states that EPA apparently has confused grams with kilograms in both the child fisher scenario and in the breastfeeding child scenarios, and in both cases this has resulted in a underestimation of exposures to children by several orders of magnitude. The preparers and reviewers appear to be unfamiliar with the units of measure and what they signify. The commenter strongly recommends that EPA utilize a competent reviewer to exert quality control over this document and states that in its current condition it is unacceptable.

Response 175: *For the final rule, EPA assessed the risk to infants from exposures to dioxins through ingestion of breast milk for all receptor populations, including subsistence receptors, using a modified margin of exposure (MOE) approach. In this approach, the estimated average daily dose (ADD) to the infant from HWCs was compared to background exposure levels to infants in the general population that breastfeed. Since exposures through breast milk are expected to be among the highest exposures that occur during early childhood and given the concern for developmental effects, EPA believes that it is important to assess the potential risks during this critical exposure period. If it can be concluded that the potential for risks during this time period is small, then exposures at other times and through other pathways are not likely to be cause for concern. This is the approach EPA took in the risk assessment for the final rule.*

The approach for assessing risks from breast milk begins with estimating the level of dioxins in maternal milk. This was done according to the methods outlined in Volume II of the dioxin reassessment exposure document (Estimating Exposure to Dioxin-like Compounds*, U.S. EPA, 1994a,b). In this approach, the average daily dose to the mother is used to estimate a steady state concentration in breast milk assuming a maternal half-life of 7 years. The ADD to the infant is derived from the concentration level in breast milk and corresponds only to the period of time during which breastfeeding actually occurs. The estimated ADD using this approach is constant because the concentration level in breast milk is assumed to be at steady-state and the ADD is calculated for the period of time the infant is nursing. The length of time the infant nurses does not effect the ADD calculation, so the dose to the infant and the risk remain the same.*

*The value cited by the commenter of 0.8 g/day for breast milk ingestion is a typographical error. As indicated in Appendix C of the risk assessment background information document for the proposed rule (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document*, February 1996) the value EPA used was 0.8 kilograms/day (800 g/day). For the final rule, EPA based the ingestion rate upon data provided in its 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a) EPA assumed*

that 0.742 L of breast milk were ingested per day. Assuming a density of 1.03 mg/mL, the ingestion rate is approximately equal to 0.76 kg/day (760 g/day).
As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

***Comment 176*:** The commenter requests that EPA evaluate the human health impact of the emissions proposed to be released under this rule on disabled Americans as required by the Americans with Disabilities Act. For dioxin, the commenter notes that it appears that suppression of the immune system may be one of the most sensitive toxicological endpoints and points out that for individuals suffering various immune suppression resulting from infection or chemotherapy, the suppression of the immune system may prove life-threatening. The commenter notes, however, that EPA simply states that no reference dose has been established and dismisses the matter. The commenter requests that EPA assess the risk posed by dioxin's suppression of the immune system especially to individuals disabled by immunological disease or suppression.

Response 176: *While 2,3,7,8-TCDD has been shown to exhibit immunological effects in laboratory animals, at the present time, there is insufficient toxicological or epidemiological data to indicate that such effects are occurring in humans as a consequence of environmental exposures. Therefore, EPA has little reason to believe that individuals with suppressed immune systems are at greater risk from dioxin exposures.*

For the final rule, EPA used a modified margin of exposure (MOE) approach to assess possible noncancer risks from dioxins. In this approach, the estimated average daily dose for HWCs is compared to background exposures in the general population. As a measure of risk, this incremental MOE presupposes that if exposures are small relative to background, then risks from these exposures are likely to have limited significance for human health. While the incremental MOE analysis is not specific to any particular health endpoint, such as immune suppression, it does allow direct comparison of exposures related to HWCs to background dioxin/furan exposure experienced by the general population. EPA's analysis indicates that with the exception of persons engaged in subsistence farming, the incremental MOE is projected to be below 0.1 for all receptor population evaluated under the final rule. This means that exposures from HWCs are

expected to be less than 10 percent of current background exposures, even for the most exposed individuals. For subsistence farming, the incremental MOE may exceed 0.1, at a frequency of 10 percent or less. The commenter is referred to the risk assessment background document for the final rule for a complete discussion of the MOE analysis (RTI, 1999).

Comment 178: The commenter states that pregnant women are considered to be covered under the Americans with Disabilities Act for certain health coverage rights. However, the commenter points out that in regard to the protection of pregnant women from risks associated with contaminated fish ingestion, the only protection afforded are advisories that pregnant women not eat fish. The commenter notes that approximately one quarter (1,042/3,710) of fish advisories issued in 1995 were issued to "sensitive subpopulations" meaning that pregnant women, women of childbearing age, and children were advised to manage the excess risk posed to them by contaminated fish by not eating. The commenter requests that EPA assess the risk posed by the mercury and dioxin to pregnant women, with the understanding that because that the risk posed is to the developing fetuses rather than the lives of women, the risk may have to be presented as a hazard quotient.

Response 178: *For the final rule, EPA assessed the risks to women of child-bearing age, the unborn fetus, and young children exposed to mercury in fish. The risk analysis utilized a reference dose (RfD) for methylmercury, the most toxic form of mercury, that is based on neurological effects in children following in utero exposure. Therefore, the methylmercury RfD is protective of pregnant women and their offspring. The risk analysis for the final rule also used the same RfD for assessing the risk of developmental effects in children. The risk of developmental effects was expressed as a hazard quotient, the ratio of the estimated average daily dose (ADD) for HWCs from fish consumption to an individual to the reference dose.*

For dioxins, EPA assessed the risks to both adolescent and adult women, including women of child-bearing age, and to nursing infants and young children from consumption of fish using a modified margin of exposure analysis. In this approach, the estimated average daily dose for HWCs is compared to background exposures in the general population. As a measure of risk, this incremental MOE presupposes that if exposures are small relative to background, then risks from these exposures are likely to have limited significance for human health. For assessing risks in adolescent and adult women, EPA compared the estimated ADD for HWCs from fish consumption to background exposures in the general population. A similar approach was used for assessing risks in young children. For nursing infants, EPA estimated the infant's ADD from ingestion of breast milk based on the mother's fish intake and compared this to background exposures for infants that breastfeed in the general population. While the incremental MOE analysis is not specific to any particular health endpoint, such as developmental effects, it does allow direct comparison of exposures related to HWCs to

background dioxin/furan exposure experienced by the general population. Since exposures through breast milk are expected to be among the highest exposures that occur during early childhood and given the concern for developmental effects, EPA believes that it is important to assess the potential risks during this critical exposure period. If it can be concluded that the potential for risks during this time period is small, then exposures at other times and through other pathways are not likely to be cause for concern.

***Comment 179*:** The commenter notes that persons with multiple chemical sensitivity are sometimes disabled by this syndrome. The commenter points out that EPA has not assessed the risk posed to chemically sensitized individuals, and requests that EPA evaluate those specific emissions which may induce adverse reactions in chemically sensitive individuals. In particular, the commenter feels that persons suffering chronic obstructive lung disease including bronchitis, emphysema, and asthma are likely to be adversely affected by inhaling acidic emissions such as HCl and particulate emissions, and therefore requests that EPA evaluate the impact of HCl and particulate emissions on chronic lung disease.

Response 179: *For the final rule, EPA assessed the risks to sensitive subpopulations from a variety of exposures. These include an incremental margin of exposure (MOE) analysis for infants exposed to dioxin through breast milk, a hazard quotient analysis for methylmercury exposures in utero, an analysis of blood lead for 0- to 5-yr-old children, a hazard quotient analysis for inhalation of hydrogen chloride and chlorine for children ages 0 to 5 and 6 to 11, an analysis of risks to highly exposed persons engaged in subsistence farming and subsistence fishing, and an analysis of risks from exposure to fine particulate matter ($PM_{2.5}$ and PM_{10}) for children and the elderly, including bronchitis and chronic obstructive pulmonary disease. Furthermore, the toxicological benchmarks that EPA uses to assess noncancer effects (i.e., the RfD for oral exposures and the RfC for inhalation exposures) reflect an added margin of safety to account for the possibility of increased susceptibility in sensitive individuals.*

While the risk assessment for the final rule did consider risk for a number of sensitive subpopulations (as described above), the analysis did not explicitly evaluate risk for those individuals with multiple chemical sensitivity syndrome. EPA recognizes that certain individuals may be highly sensitive and exhibit idiosyncratic effects from concurrent exposure to multiple chemicals. Multiple chemical sensitivity can be considered a special category of synergistic chemical effect. The risk analysis completed for the final rule assumed additivity of noncancer risks (hazard quotients) for chemicals evaluated for the same route of exposure. However, the analysis did not explicitly consider either synergistic or antagonistic chemical effects. EPA acknowledges the uncertainties related to the possibility of nonadditive, synergistic effects between chemicals in persons with multiple chemical sensitivity syndrome. However, at the present time, synergistic and

antagonistic interactions have not been sufficiently characterized to allow consideration of these effects quantitatively for risk characterization purposes.

Section 37
Small Business Administration
RCSP-0237

***Comment 0237-1*:** The commenter notes that the risk panel had raised several concerns about the risk assessment methodology including that fact that "it was unclear how the more important region-to-region differences in human activity patterns is reflected in these selected studies" and that "the document is unclear about the basis for the selection of exposure pathways and exposed groups and the selection of land-use considerations." The commenter notes further that, with respect to air concentrations, the panel stated that "the theoretical assumptions of the EPA may yield estimates greater than actual values in most cases. For example, deposition rates may be in error because of the assumption that little or no TCDD, etc., deposits on relatively large mineral particles (fly ash) in the gas stream, when it is known that such condensation occurs." The commenter states that it appears the risk analysis conducted by EPA may have overestimated the risk and contains some fundamental shortcomings.

> ***Response 0237-1:*** *EPA gave much consideration to the comments of the peer-review panel and made a number of changes to the risk assessment in order to improve the risk assessment. EPA has prepared a separate response to comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *(U.S. EPA, 1999b) that addresses the peer-review comments. The commenter is referred to this document for EPA's response to the issues raised by the peer-review panel to the peer review.*

***Comment 266*:** The commenter notes that EPA reassessed the impact of TCDD (dioxin) on human health and the environment in 1994 and that the two draft documents, entitled *Health Assessment Document for 2,3,7,8- Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds* and *Estimating Exposure to Dioxin-like Compounds*, form the scientific foundation for regulation of dioxin emissions in the proposed Hazardous Waste Combustor Rule. The commenter points out that EPA has based the standards on MACT and is estimating that once promulgated, will reduce emissions from combustion units (incinerators, cement kilns, and light-weight aggregate kilns) by 97 percent. However, the commenter notes that regulated industry responded to the documents by saying that the EPA had over-assessed dioxins impact to health and the environment and that EPA has estimated that to achieve the proposed MACT standards, hydrocarbons will incur high removal costs per gram of dioxin removed. The commenter believes, therefore, additional research concerning the effects of dioxin are warranted.

> ***Response 266:*** *EPA did not use the draft dioxin reassessment as the basis for setting the emission standards for the final rule. Sections 112 (a) and (d) of the CAA direct EPA to*

set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns, based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone. Thus, EPA did not rely on the conclusions of the draft dioxin reassessment, either in the risk assessment or in setting the emission standards.

However, EPA did use technical information from the dioxin reassessment in assessing risks from HWCs, including methods and data that were developed for the dioxin reassessment for assessing indirect exposures. Much of this information was derived from the draft exposure document for which the SAB commended EPA, calling it "a very credible and thorough job."

EPA is continuing work on the dioxin reassessment and is considering all comments received on the 1994 draft assessment, including comments from the public and the SAB. EPA intends to respond to the comments in an appropriate forum.

EPA believes that the use of the aforementioned documents and modifications made to the risk assessment in support of the final rule do not overassess the impact of dioxins to health and the environment. EPA believes that these steps provide considerable assurance that the exposure estimates are not overly conservative and, when taken together, are not likely to either substantially overestimate (or underestimate) the range of possible exposures.

***Comment 1050*:** Commenters, representing the regulated industry, assessed issues relating to the toxicity, carcinogenicity, noncarcinogenic health effects, dose-response modeling, and risk characterization of dioxin and its related compounds. A primary concern of affected industry was the extrapolation of animal-related test results to humans. Emphasizing pharmacokinetic and metabolic response differences among species, it was stated that dioxin's toxic, immunotoxic, and carcinogenic

impacts on rats, mice, and sheep should not be applied to humans. The commenters cited tests on human immunotoxic response to dioxin (Neubert et al., 1993, and Roegner et al., 1991), which demonstrated no immune alterations in humans exposed to dioxin levels far in excess of levels which alter animal immune functions. Other epidemiological studies, which disprove EPA's findings that claim to justify carcinogenicity at low levels of dioxin, were also examined (Rockett and Arena, 1983). Concern was expressed regarding the low-dose linearity and nonthreshold assumptions of the dose-response models selected in the draft document. These assumptions imply that dioxin would produce harmful effects on body cells at a single dose. Risk models were criticized for their reliance on inconclusive epidemiological evidence on carcinogenicity. EPA's analysis of data was also scrutinized, and it was pointed out that a female rat study presented in the draft document actually demonstrated nonlinear tumor response to varying dioxin doses and low-dose reductions in total tumors.

Response 1050: *Dioxin has received a great deal of health and biological research attention over the past 25 years. The body of knowledge ranges from numerous epidemiological studies, to studies at the biochemical level and includes in vitro, ex vivo, and in vivo evidence. EPA agrees with the commenters that there are insufficiencies in the pharmacokinetic and disposition evidence in humans to model tissue doses, which limits extrapolation from animals to humans. However, the understanding of mechanisms of action and toxicity, in general, for dioxin is well developed. Association of the Ah receptor mediated response to dioxin exposure in many areas of toxicity, e.g. cancer, immunotoxicity, and developmental toxicity, is broadly accepted. Dioxin's induction of cytochrome P450 has been rigorously established. The biological consequences of this cytochrome induction have not been fully and mechanistically described, yet the common features of animal and human Ah receptor response are adequately proven. EPA does not reject the extrapolation of animal results to humans based on weaknesses in some evidence of dioxin's mechaisms of action in light of the Ah receptor insights.*

Regarding carcinogenicity, 2,3,7,8-TCDD has been shown to be carcinogenic in many independent rodent bioassays, and oral exposure results in increased incidences of a variety of tumor types. The 1982 bioassay by the National Toxicology Program demonstrating 2,3,7,8-TCDD to be an animal carcinogen in both rats and mice is one important study. As to the appropriateness of extrapolating animal results to human risk assessment, there is a strong indication that the mechanism of action for TCDD in animals and humans is similar. The Ah receptor response is common to animals and humans and TCDD is a multisite carcinogen (McGregor et al., 1998, "An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis"). Tissue concentrations in heavily exposed individuals is similar to those in some experimental animals that have developed tumors.

Cancer epidemiological evidence is most strongly found in the Swedish studies that associate soft tissue sarcoma with dioxin/phenoxyherbicide exposure. EPA and SAB

support the view that these studies have stood up over the intervening years. Supporting, although weaker, epidemiological evidence for cancer is provided by the NIOSH study of lung cancer in workers (Fingerhut et al., 1991). Kramarova et al., 1998, also report an increased risk for STS from an analysis of the combined results of an analysis that combines the results from U.S. pesticide workers (Fingerhut et al., 1991, "Cancer mortality in workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin"), German workers (Becher et al., 1996, "Cancer mortality in German male workers exposed to phenoxy herbicides and dioxins"), and Dutch workers (Hooiveld et al., 1996, "Preliminary results of the second follow-up of a Dutch cohort of workers occupationally exposed to phenoxy herbicides, chlorophenols and contaminants").

EPA's analysis indicates that most of the cancer findings exhibited response consistent with linearity in the observable range. Also, few of the mechanistic models that EPA identified in the scientific literature exhibited nonlinear dose-response in the observable region or predicted nonlinear dose-response in the low-dose (extrapolation) region. Furthermore, EPA's analysis of the data from the National Institute of Occupational Safety and Health (NIOSH) study (Fingerhut et al., 1991) found that the ratio of the average daily dose for the high- and low-dose groups was the same as the ratio of increased risk for respiratory cancer, indicating a linear dose-response curve (see Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds. Chapter 8, Dose-Response Modeling for 2,3,7,8 Tetrachlorodibenzo-p-Dioxin (TCDD)*, U.S. EPA, 1997d). Therefore, EPA believes there are sufficient human and animal data suggesting that response is proportional to dose to warrant continued use of the linearized multistage model.*

Results of the two-stage modeling of the Kociba et al. female rat liver tumor data incorporating dioxin-altered hepatic foci data to estimate mutation and growth parameters provide nearly the same low-dose estimates as the linearized multistage model using only the tumor data. Unless a protective effect of TCDD on mutation rates occurs at low doses, low-dose risk will remain proportionate to exposure and consistent with the linearized multistage model. If protective effects are allowed in the model, the low-dose risks may be substantially reduced; however, the focal lesion data and the biochemical markers generally agree and do not suggest a protective effect (U.S. EPA, 1997d). Therefore, there appears to be no strong support for general nonlinearity for TCDD's effects in the range of the data studied and little support for extrapolation into a lower dose range with a highly nonlinear model. To the contrary, EPA believes there are sufficient data suggesting that response is proportional to dose that, when considered together with the available human data, warrant concern that 2,3,7,8-TCDD will induce toxic effects in humans in the range of the experimental animal data. Furthermore, based on a lack of data indicating an immediate and steep change in slope of the dose-response

curve, EPA believes the possibility of response 1 to 2 orders of magnitude below this range must be considered.

The evidence for immunotoxicity is extensive in animals and very limited in humans. In the Scientific Advisory Board's review of the EPA dioxin reassessment document, the majority of reviewers agreed that sufficient data exist to indicate immune effects could occur in the human population from exposure to dioxin at some dose levels. Contaminated rice oil exposure in Taiwan provides evidence of immunosuppression and increased infection rates and some studies have reported immunoglobulin (Ig) level and NK cell activity changes in humans. Other human studies reported no effects, e.g. Ranch Hand/USA and Seveso, Italy. EPA accepts that there is large variability in the immune response in humans, limited numbers of tests conducted, and poor exposure characterization. Characterization of human dioxin sensitivity with regard to other species cannot be supported, because of insufficient clinical data. EPA does not conclude that humans are less sensitive, as suggested by the commenters. Extensive animal evidence over the past 25 years demonstrate the immune system toxicity of dioxins and related compounds. In vivo studies provided provide understanding of the cellular and molecular mechanisms of TCDD toxicity.

As the SAB pointed out in its report on the draft dioxin reassessment, understanding the operation of bias and chance is especially important when interpreting "negative" results from an epidemiological study, where no differences are apparent or where the differences are not statistically significant. Differences produced by real effects can easily be masked by poor exposure classifications (misclassification bias), a negative association may appear merely by chance by virtue of a small population available for study (poor statistical power), and potential risks can be rendered undetectable by observing the exposed population for too short a time (bias produced by failure to account for adequate latency). These are just a few of the factors complicating interpretation of negative outcomes. On the other hand, spurious positive associations are much less likely to occur in environmental epidemiological studies because more factors operate to lower the observed risks than to raise them.

***Comment 1051*:** The commenter notes that the commenters representing the regulated industry pointed out a lack of updated emissions data in the EPA's dioxin emissions inventory. The commenter cites comments from the industry representatives indicating that EPA overestimated emissions of dioxin based on this incomplete database. In the instance of cement kilns, it was stated that the EPA had relied on test data which was unrepresentative of the industry, thereby leading to an incorrect estimation of dioxin emissions.

Response 1051: *EPA used facility-specific annual emissions estimates for baseline and the three MACT options evaluated in the final rule. Baseline emission rates were*

generated from compliance and trial burn data that combined facility-specific emission concentration data (for each constituent) with facility-specific dry standard stack flow rate data using the assumption that facilities operate 24 hours per day for 365 days per year. These data have been revised since the proposed rule to incorporate additional facility-specific information as it became available and to address data issues raised in public comments. Specifically, the database has been augmented with facility-specific information obtained during an initial comment period (at proposal), a subsequent Notice of Data Availability (NODA) comment period, and further data-gathering efforts involving visits to Regional EPA and state environmental offices, which were conducted in the fall of 1997.

During the model facility sampling for the final rule, visits to state/Regional EPA offices were conducted to obtain facility-specific information on 60 of the 76 modeled facilities for purposes of improving characterization of both facility operations and land use surrounding the facilities. These visits focused on obtaining permitting information, including site-specific risk assessment, as well as information obtained directing from state and EPA staff familiar with specific facilities. Types of information obtained through these visits included facility-specific emissions data, facility status (i.e., operational or undergoing RCRA closure), specific facility location, and key facility operational parameters (stack height, velocity, etc.).

A complicated process of matching facilities with available emissions data to similar facilities and imputing emissions values was developed and implemented for those 16 modeled facilities that did not have measured emission data.

EPA published in the Federal Register *a notice covering the database that was used to set the floor levels via a NODA last year (January 7, 1997). The database noticed in January 1997 contained all the information available from trial burn and certificate of compliance reports that was used in the rulemaking, including emissions data and engineering information on control equipment and operating parameters as well as stack information. This information was used to characterize stack emissions where measurements were available and to impute exhaust gas concentrations in cases where they were not.*

This additional information, obtained chiefly from newly identified trial burn and compliance test reports, resulted in adjustments to facility-specific engineering parameters and emissions estimates. In some cases, the new information resulted in EPA's removing a facility from the database (e.g., because of closure) or changing its classification from one source category to another (e.g.,from a large on-site incinerator with waste heat boiler to a large on-site incinerator without waste heat boiler).

It was assumed that, to satisfy these MACT emission limits, facilities would have to operate at 70 percent of the MACT limit. These emission rates were used in the risk analysis. Moreover, it was further assumed that facilities operating below new regulatory limits, for whatever reason, would not increase their actual emission rates after new MACT standards have been promulgated.

Comment 1052: The commenter states that the cement kiln industry found that EPA's estimates of particle size distribution, incorporated in the site-specific assessment of cement kilns, overestimated dioxin concentrations in kiln dust due to insufficient size and surface considerations.

Response 1052: *The low amount of TCDD deposited on relatively large mineral particles may partially result from the use of a default particle size distribution that includes a relatively low fraction of large mineral particles. This should not have a strong impact on the analysis because, as discussed in ORD's dioxin exposure assessment and the 1993 Addendum (U.S. EPA, 1993a) to* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA,1990), evidence points to the fact that semivolatiles condense preferentially on smaller particles due to the larger surface area–to–volume ratio. Thus not only would these large particles be more efficiently collected in the air pollution control devices, resulting in little or no release of large particles to the atmosphere, but the smaller particles would also contain more deposited dioxin than the larger particles due to their relatively larger surface area.*

***Comment 1053*:** An analysis of EPA's findings on background exposure presented in the draft document was also addressed. The EPA had assumed a "steady state," or constant rate of dioxin intake, in calculating daily dose rates. In response, commenters representing the regulated industry emphasized temporal fluctuations in human exposure to dioxin.

Response 1053: *The background exposure level for dioxin toxicity equivalency concentration (TEQ) that is used for the final rule is subject to uncertainty resulting from the approach used in generating the value. This background value was generated using pharmacokinetic modeling and steady state assumptions to back-calculate the dose estimate from an adult background body burden value. EPA recognizes the uncertainty in this estimate of background exposures. The steady state assumption used in this calculation implies that past exposure to dioxin TEQ has been constant. However, evidence suggests that current body burden levels are the result of nonconstant (i.e., variable) exposure levels over the past few decades. When non-steady state conditions are used in conducting pharmacokinetic modeling for purposes of back-calculating dose estimates from these background body burden values, the resulting dose estimates can be significantly lower than values generated assuming steady state conditions (i.e., continuous exposure). Therefore, the generation of background dose estimates from*

background body burden values assuming steady state conditions, as was conducted for the HWC risk analysis, could result in an overestimation of background dose levels, which in turn would result in an underprediction of incremental MOE.

***Comment 1054*:** The commenter notes that, considering the amount of dioxin/furan emissions from combustion units and its minority contribution to total emissions of dioxins/furans, it is important for EPA to relate the stringent MACT standard with significant improvements in human health. The commenter notes that while EPA estimates a dose of 0.01 picograms per kilogram (pg/kg) of body weight per day leads to a 1 in 1 million risk of cancer, the World Health Organization claims that 10 pg/kg per day of dioxin constitutes a "tolerable daily intake."

Response 1054: *Sections 112 (a) and (d) of the CAA direct EPA to set standards for stationary sources that are major sources of HAPs, as defined in the CAA. Dioxins are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology, as prescribed in Section 112(d)(2) and (3). For dioxins, the final rule sets an emission standard for existing cement kilns based on the level of control in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources, as explained in the preamble to the rule. EPA performed a risk assessment in order to evaluate whether the MACT standards, as outlined above, are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.*

***Comment 1055*:** The commenter states that the comments submitted by industry representatives on the two draft dioxin documents question EPA's assessment of dioxin's impact on human health as well as the levels of combustion emissions and subsequent human exposure to harmful levels of dioxin. The commenter stresses that EPA's tests of health risk and exposure involve biases in the selection of models, data, and data interpretation. The commenter feels that these biases favor conservative estimates of dioxins health effects and presence and may overstate dioxin's health impacts. The commenter notes that, even with these biases, the EPA concludes that dioxin is a "likely" human carcinogen, rather than a "known"carcinogen. The commenter thus feels the justification of the stringent standards and associated costs through health risk is uncertain, given the possible uncertainty of EPA's health and exposure assessments.

Response 1055: *As indicated in response to the previous comment, EPA performed a risk assessment to evaluate whether the technology-based MACT standards are generally*

protective of human health and the environment, as required by RCRA Sections 3004(a) and (q). The level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

Combustion emissions were measured or derived for each facility in the analysis for the final rule. For each facility for which emission measurement data were available, site-specific stack gas concentration data from trial burn and certificate of compliance tests were used in conjunction with stack gas flow rates to estimate emissions. However, in those instances where emissions measurements were not available, stack gas concentrations were imputed from a pool of emissions measurements for other, similar facilities. Imputation was done such that emissions of individual congeners were all imputed from the same data set. EPA believes that this approach makes the best use of the available data and improves the representativeness of the analysis.

EPA develops its regulations within a well-defined set of requirements and policies that include rigorous peer-review (Science Advisory Board [SAB]) and EPA internal reviews. This process is designed to bring the best possible science to the rules and regulations that EPA develops. As part of the regulatory process, risk assessment is performed with the understanding that there is uncertainty in the collective understanding of exposure, health effects, and risks to the American public. It is appropriate to adjust for this uncertainty in conservative ways.

Despite the wealth of studies for chemicals, such as dioxin, there remain unknowns that EPA has carefully considered in its health and exposure risk assessments. The 1988 and 1994 documents developed for assessing dioxin's risks received very favorable reviews by the SAB, which concluded that the 1988 documents "were carefully and well written," the 1994 Exposure Assessment draft document was "very credible and thorough," and the 1994 Health Assessment draft document "provides a comprehensive review of the literature…and [the SAB] commends the EPA staff for this considerable accomplishment."

EPA believes the weight of the evidence supports the conclusion that 2,3,7,8-TCDD and other dioxin-like compounds are probable human carcinogens. The SAB, in its review of the draft dioxin reassessment, agreed, concluding that dioxin is likely to increase human cancer incidence under some conditions of exposure. The SAB categorized dioxin and dioxin-like compounds as a B1 carcinogen under the 1986 EPA cancer guidelines of "agents for which there is limited evidence of carcinogenicity from epidemiologic studies." In addition, the International Agency for Research on Cancer (IARC) recently reviewed the experimental data and epidemiological studies and also concluded that 2,3,7,8-TCDD is carcinogenic to humans (WHO, 1997; McGregor et al., 1998). Health

assessment for dioxins involves both cancer and noncancer effects. For the final rule, EPA used a margin of exposure (MOE) approach to assess noncancer risks. In this approach, the estimated average daily dose for HWCs is compared to background exposures in the general population. As a measure of risk, this incremental MOE presupposes that if exposures are small relative to background, then risks from these exposures are likely to have limited significance for human health.

Section 38
Solite Corporation
RCSP-0244

***Comment 283*:** The commenter generally agrees with the comments of the Risk Assessment Peer Review Panel. In particular, the commenter notes that the Panel's comments highlight two deficiencies in the risk assessment and EPA's use of the risk assessment.

- **#** The commenter feels that the panel has persuasively shown that the cumulative impact of the conservative assumption in the risk assessments results in a large exaggeration of the actual risks associated with the emissions from combustion units.

- **#** The commenter believes that the risk assessment fails to support any BTF standards because MACT floor standards have been shown to be adequately protective. The commenter feels any health or environmental benefits of BTF standards would be negligible, at best, and that this is especially the case for dioxins/furans, mercury, and HCl.

Response 283: *EPA gave much consideration to the comments of the peer-review panel on the risk assessment for the proposed rule. EPA made a number of changes to the risk assessment for the final rule as a result of the comments received from the peer reviewers. EPA has prepared a separate response-to-comments document entitled* Report of the Peer Review Panel on the Risk Assessment for the Hazardous Waste Combustion Proposed Rule: Response to Comments *that addresses the peer-review comments. The commenter is referred to this document for further details on EPA's response to specific issues raised by the peer reviewers. As a result of the comments received from the peer reviewers and the public, EPA took a number of steps to improve the representativeness of the risk analysis for the final rule. In particular, for the final rule, EPA generated population-weighted cumulative distributions of risks to individuals, thereby eliminating the need to define central tendency and high-end exposure scenarios deterministically, in the manner done at proposal. To accomplish this, EPA selected a total sample of 76 facilities to represent various categories of HWCs. Of these, 66 were selected at random and 10 had previously been selected at proposal. All 5 lightweight aggregate kiln facilities that burn hazardous waste were selected for analysis in the final rule. A study area was defined for each facility as the area surrounding the facility out to a distance of 20 kilometers. Each study area was divided into 16 sectors. Media concentrations were estimated for each facility sector using facility-specific (e.g., stack emissions) and site-specific (e.g., land use) information. Sector-specific exposures were then estimated from the media concentrations and age-specific exposure factors derived from the 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a). Mean exposure factor values were*

used in order to estimate the mean, or arithmetic average, risk to individuals within a sector. These mean risk values were then aggregated across an HWC category taking into account sector-specific population estimates and facility-specific sampling weights. For certain risk-driving pathways, EPA conducted an exposure parameter variability analysis using Monte Carlo simulation. Population estimates were derived from U.S. Census and Census of Agriculture data. EPA believes that these steps provide considerable assurance that the exposure estimates for the HWC rule are not overly conservative.

As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2) and (3) of the CAA. Dioxins and mercury are singled out for regulation under MACT standards in Section 112(c)(6). EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology. For existing sources, EPA is required to set the emission standard based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources. However, EPA may establish emission standards that are more stringent than this.

EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under Section 112 of the CAA, and the MACT standards implement those provisions alone.

For the final rule, EPA is setting the emission standards for mercury at the MACT floor for all source categories. For dioxins, the final rule sets the emission standard at the MACT floor except for incinerators equipped with waste heat recovery boilers and lightweight aggregate kilns, for which a beyond the floor standard is being established, as explained in the preamble to the rule. For total chlorine, the final rule sets the emission standards at the MACT floor except for lightweight aggregated kilns, for which a beyond the floor standard is being established, as explained in the preamble to the rule.

Section 39
Texas Natural Resource Conservation Commission
RCSP-0089

***Comment 287*:** The commenter cites a statement from the proposed rule ("The analysis focused primarily on dioxins and related compounds…. The individual risk analysis did also include risks from inhalation of metals, hydrogen chloride, and chlorine (Cl_2)…. The Agency conducted an evaluation of risks from metals through indirect exposure routes.") and notes that it is incomplete and somewhat misleading in terms of the specific HAPs evaluated by the EPA in the generic risk assessment. The commenter notes that according to *Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document Final Report* (February 20, 1996), EPA did evaluate dioxins and furans (as TEQs) but did not address any other dioxin-related compounds.

Response 287: *For the final rule, EPA evaluated the human health and ecological risks for all constituents released from HWC facilities for which adequate emissions data were available. These included the following: antimony, arsenic, barium, beryllium, cadmium, chromium (III), chromium (VI), cobalt, copper, lead, manganese, elemental mercury, divalent mercury, methylmercury, nickel, selenium, silver, and thallium. EPA also assessed the risks from hydrogen chloride and chlorine. EPA evaluated risk associated with 2,3,7,8-TCDD and the 16 other 2,3,7,8-substituted chlorinated dioxins and furans based on the concept of toxicity equivalence (TEQ). However, EPA has little or no data on emissions of other dioxin-like compounds from HWCs, such as coplanar PCBs.*

The risk assessment for the final rule assessed risks from chlorinated dioxins and furans. However, risks from other organics that may be present as PICs could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited and the data are highly variable and are therefore inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that in implementing the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3),

including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

***Comment 1092, 288*:** The commenter notes that risk results are presented for both hydrogen chloride and chlorine based on assuming 100 percent HCL and 90 percent HCL + 10 percent Cl2, respectively. The commenter notes that risk estimates for hydrogen chloride (only) are provided in the *Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document Final Report* (February 20, 1996). The commenter feels that the basis of the risk estimates for Cl2 should be clearly discussed in the aforementioned technical support document.

Response 1092, 288: *The omission of risk results for chlorine in the background information document for the proposed rule was an oversight. Chlorine risk results were documented separately and included in the docket for the proposed rule (see memorandum from D. Layland to the file dated April 15, 1996, on Hazard Quotients for Chlorine). For the final rule, hazard quotients for both chlorine and hydrogen chloride are presented in the risk assessment background (RTI, 1999).*

Chlorine is a potent irritant in humans to the eyes, upper respiratory tract, and lungs. It is also extremely irritating to the skin and can cause severe burns at high concentrations. In the final rule, EPA assessed the risks associated with chlorine and hydrogen chloride using the inhalation hazard quotient as the risk descriptor for these two chemical constituents.

Chlorine risks were based on an interim RfC of 1 x 10^{-3} mg/m^3, derived from a lifetime inhalation study of rats and mice. In this study, the lowest exposure concentration of 0.4 ppm was selected as the NOAEL. The NOAEL was adjusted for intermittent exposure and a human equivalent concentration NOAEL was calculated. An uncertainty factor of 30 was used (3 for interspecies extrapolation and 10 to account for sensitive individuals). The commenter is referred to the risk assessment background document for the final rule for a complete discussion of the derivation of the RfC for chlorine (RTI, 1999).

***Comment 289*:** The commenter agrees that due to the complexity of mercury chemistry, a quantitative risk assessment predicated on a combination of "generic" emission estimates for mercury and "site-specific" exposure parameters may not be precise enough to support regulatory decisions at a national level. However, the commenter's opinion is based more on concerns about the "generic" risk assessment approach as a whole rather than on the uncertainties associated with mercury. The commenter supports the continued use of quantitative risk assessment to evaluate the potential health effects associated with mercury, at least on a site-specific basis. The commenter has noted that some Regional EPA offices are now discounting noncancer exposures to mercury because of the concern that the exposure methodology contains too much uncertainty. To the contrary, the commenter believes that

it is precisely because there is an abundance of data for mercury that the merits of the fate and transport and exposure assumptions for mercury can be debated. The commenter feels that many of the complexities associated with mercury chemistry can be dealt with in a site-specific risk assessment by collecting site-specific information.

Response 289: *At the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. EPA has since issued its* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA, 1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule. EPA recognizes, however, that significant uncertainties remain and the results of the mercury analysis should be interpreted with caution and be used only qualitatively. EPA agrees with the commenter that performing a site-specific analysis based on the collection of site-specific information (e.g., speciation of mercury emissions, methylation rates in the water column, background levels of mercury, etc.) could reduce the uncertainty involved in assessing risks from mercury exposures for HWCs.*

The mercury assessment followed the general modeling approach developed for the 1997 MRTC and used the IEM-2M surface water model developed especially for that report. For the final rule, EPA assessed the human health and ecological risks associated with elemental, divalent, and methylmercury. EPA's analysis shows that the most significant exposures come from methylmercury, due primarily to deposition and runoff of divalent mercury to surface water and subsequent methylation and bioaccumulation in fish.

The uncertainty associated with modeling mercury exposure and assessing the potential for risk stems from (a) complexities associated with modeling the fate/transport of different mercury species through different environmental media following release from HWC facilities, (b) difficulties in accurately modeling individual behavior related to mercury exposure (e.g., recreational fishing activity at specific locations and amounts of fish consumed), and (c) challenges in evaluating differing health effects studies and accurately characterizing the dose-response relationship (i.e., RfD) for methylmercury. The risk assessment background document for the final rule includes a detailed discussion of uncertainty associated with all major components of the HWC risk analysis including fate/transport modeling, exposure assessment, and risk characterization for mercury.

***Comment 290, 291*:** The commenter cites a statement from the proposed rule ("The example facilities represent a variety of environmental settings and facility characteristics. The purpose of using example facilities was to incorporate as much realism as possible into the Agency's risk assessment and to reduce the reliance on hypothetical, conservative assumptions about either location or source type characteristics.") and notes that it is important to recognize that risk can only be accurately calculated on a site-specific basis. The commenter states that assessment of potential adverse effects on human health from chemicals emitted from incinerator stacks depends on the design of the facility, operating conditions, feedstock of the incinerator, specific characteristics of the site, environmental conditions, and types of receptors in the vicinity of the incinerator. The commenter feels, therefore, that it is unclear whether the MACT standards are truly protective in all cases. Further, the commenter questions whether the "example" facilities are in fact representative, based on issues of size, type, and location.

The commenter states that the example facilities chosen do not appear to be truly representative in terms of size. The commenter cites the *Background Information Document Final Report* (February 20, 1996; p. 50), as indicating that small on-site incinerators are not represented by the case studies. Therefore, the commenter feels that the emissions that form the basis of the risk assessment are not truly representative of all the facilities that burn hazardous waste in terms of size. In addition, the commenter states the 11 example facilities used in the risk assessment only represent 6 percent of the universe of facilities burning hazardous waste. Therefore, the commenter believes it is difficult to ascertain whether small facilities were adequately represented in the "generic" risk assessment.

Also, the commenter states that the example facilities chosen do not appear to be truly representative in terms of type and notes that for the risk assessment, four hazardous waste incinerators (HWIs), five cement kilns, and two lightweight aggregate kilns (LWAKs) were evaluated. Therefore, of the example facilities evaluated (11) in the risk assessment, HWIs made up 36 percent, cement kilns made up 45 percent, and LWAKs made up 18 percent. However, based on a survey conducted by the commenter of the actual universe of facilities burning hazardous waste in the U.S., HWIs make up approximately 83 percent (162 facilities), cement kilns comprise approximately 13 percent (26 facilities), and LWAKs make up approximately 4 percent (7 facilities). The commenter feels, therefore, it appears that HWIs are underrepresented and hazardous waste burning cement kilns and LWAKs are overrepresented.

The commenter further states that the example facilities chosen do not appear to be truly representative in terms of geographic location, given that most of the facilities chosen for this exercise are located in the eastern U.S. The commenter states that a large number of the facilities that will be affected by the proposed MACT standards (approximately 50 percent) are located in the South and Midwest and notes that it would have been more appropriate to select model facilities from a wider range of geographic regions. The commenter states that regional differences in weather patterns and terrain could significantly affect the fate and transport modeling and activity patterns and, therefore, the estimated risks associated with each model facility. The commenter notes specifically that the State of Texas has approximately 20 percent, 4 percent (previously 8 percent), and 14 percent, respectively, of

the universe of HWIs, cement kilns, and LWAKs that burn hazardous waste in the U.S. The commenter points out that only one Texas facility (HWI) was used as an example facility for the risk assessment. The commenter believes that since geographic location served as a surrogate for a number of variables such as precipitation, land use, agricultural practices, and surface water bodies, all of which significantly impact exposure estimates, terrain features, and meteorology specific to the State of Texas were adequately represented in the "generic" risk assessment that was conducted to evaluate the protectiveness of the standards on a national level. Therefore, the commenter feels MACT standards have not necessarily been shown to be adequately protective for the State of Texas.

Response 290, 291: *EPA received many comments on the risk assessment completed for the proposed rule regarding the representativeness of the example facilities and associated risks in relation to the overall HWC universe. In response to theses comments, several modifications were made to the risk analysis framework for the final rule in order to improve the representativeness of the assessment. These modifications included (a) the use of stratified random sampling to select a subset of HWC facilities for analysis; (b) the use of a study area out to 20 kilometers divided into 16-sectors implemented within a GIS for site characterization; and (c) the use of site-specific data, including U.S. Census and Census of Agriculture data, for exposure assessment.*

Both peer review and public comments regarding the representativeness of the risk analysis completed at proposal made observations that certain combustor categories (e.g., small on-site incinerators) were not represented in the facilities modeled at proposal and that the model facilities may not have accurately reflected the geographic distribution of HWC facilities nationwide. Therefore, for the final rule, EPA increased the number of facilities and modified the way the facilities were chosen.

For the final rule, EPA evaluated 76 active HWC facilities (15 cement kilns, 5 lightweight aggregate kilns, 13 commercial incinerators, 25 small on-site incinerators, and 18 large on-site incinerators). Of these, 11 are located in Texas. To ensure that these facilities would be representative of all facilities covered by the rule, 66 of the 76 facilities were selected using stratified random sampling, while the remaining 10 were facilities that had been analyzed at proposal and were retained for the final rule (the 11th facility has ceased to burn hazardous waste and is undergoing RCRA closure). The use of stratified random sampling also allows clear statistical statements to be made regarding the representativeness of risk results for the HWC facility universe. Specifically, for the final rule, confidence intervals reflecting sampling error (i.e., error introduced into the analysis by not having modeled all HWC facilities) were generated for the majority of risk results. Sampling error is a key source of uncertainty that impacts the representativeness of risk results generated for the HWC risk analysis. Use of a nonstatistical selection strategy, such as the case study approach used at proposal, does not allow the representativeness of risk results to be assessed (e.g., confidence intervals

reflecting sampling error can not be generated). Sample sizes for each combustor category were based on the goal of having a 90 percent probability of selecting a facility from the top 10 percent of facilities within a given combustor category with regard to risk (i.e., a 90 percent probability of having included a high-risk facility in the sample). EPA's analysis for the final rule met or exceeded this goal. For these reasons, EPA believes the risk assessment for the final rule is representative of the range and types of HWC facilities. Furthermore, because of the sample sizes used in the analysis, EPA also expects that the risk assessment adequately characterizes risks at the high end of the risk distribution.

With the increased number of facilities selected to represent the HWC universe, there is a more representative geographic distribution of facility locations. Model facilities for the final rule represent a wider range of geographic regions than did the case studies selected for the proposed rule.

For the final rule, EPA incorporated the use of GIS in order to (a) allow georegistered spatial data, including U.S. Census and Census of Agriculture data, to be incorporated into the analysis and (b) increase the accuracy of spatial analytical tasks associated with fate/transport modeling and exposure assessment. For the final rule, a GIS provided the platform for projecting the impact of HWC emissions on individual study areas and watersheds/waterbodies and for characterizing land use within study areas (i.e., location, shape, and size of watersheds and waterbodies and densities of human populations and livestock). To provide sufficient spatial resolution for the analysis, a 20-km radius polar grid, centered on the facility, was divided into 16 sectors at site radii of 2, 5, 10, and 20 km and along the north-south and east-west axes.

EPA agrees with the commenter that the potential for adverse effects on human health and the environment depend on a variety of site-specific factors. In the risk assessment for the final rule, EPA used site-specific information whenever possible. Facility data were used for characterizing emissions and the conditions of stack release. Facility-specific emission measurement data were used when available (when these data were not available, values were imputed from facilities with similar operational characteristics). Air modeling employed meteorological data from the nearest weather station and site-specific terrain and land use data. Exposed populations were located using a combination of block-level U.S. Census data and county-level Census of Agriculture data. Bodies of water selected for analysis were characterized using stream gauging and other site-specific data. Watersheds were delineated using site-specific topographic data and were further characterized using site-specific soils and land use data.

Five years of meteorological data were assembled from surface and upper air data for each of the HWC facilities modeled for the final rule. Surface and upper air stations were

assigned to each facility based upon location relative to one another, terrain effects on climate and wind patterns, and proximity to major bodies of water. In some locations, particularly, data-sparse areas such as the Rocky Mountains, efforts were made to select the most representative stations. Land use was characterized for each site using Anderson land use codes created from land use data within a GIS (Anderson et al., 1976). Roughness length, Bowen ratio, noontime albedo, and minimum Monin-Obukhov length were estimated based on the land use codes. EPA interpolated missing data using preceding and subsequent records.

Site-specific terrain was evaluated as part of the modeling analysis whenever there was significant elevation of terrain (relative to stack height) in close proximity to the facility. In these cases, terrain elevations were explicitly considered for dispersion modeling.

Based on site-specific modeling results, EPA generated cumulative frequency distributions for risk characterization for each category of HWCs. U.S. Census and Census of Agriculture data were used to generate sector-level population totals for enumerated receptor populations. This enabled the cumulative frequency distributions to be population-weighted to reflect the spatial distribution of individuals around HWC facilities. Central tendency risks were characterized based on the 50th percentile risk estimates from the population-weighted cumulative risk distributions, while upper end risks were characterized based on the 90th to 99th percentile risk estimates.

***Comment 292*:** The commenter notes that the maximally impacted receptor was identified by default as the closest receptor, rather than through modeling. The most impacted receptor was located by estimating the location of maximum air concentrations and deposition based upon information on the prevailing winds. It is not clear to the commenter how the maximum impacted locations can be determined by this method. Other factors, such as precipitation, impact the locations of the maximums. In risk assessments conducted in-house by the commenter using similar methodology to that employed in the "generic" MACT risk assessment, the commenter has noted several instances in which the model has shown that maximum deposition is in the opposite direction of the maximum air concentration and prevailing wind. The commenter states that the nearest receptor may not always be the maximum impacted because the design of the stack may cause the plume centerline to overshoot the nearest receptor. This could cause a greater impact at a further distance. In addition, the impact at a residence depends on the combined impacts of deposition and concentration. Therefore, the commenter emphasizes that the greatest combined impact may not always be at the nearest receptor.

Response 292: *EPA agrees with the commenter that a number of site-specific factors determine the location of the most impacted receptor. For the final rule, a 16-sector template implemented within a GIS framework was used to provide the necessary spatial resolution for fate/transport modeling and estimating exposure for all receptor populations that were evaluated. The 16-sector template also formed the basis of the*

screening-level ecological risk analysis completed for the final rule. The 16-sector template was made up of concentric rings at 2, 5, 10, and 20 km subdivided by north-south and east-west transects and provided enhanced spatial resolution nearer to the facility (within 10 km) where areas of higher deposition/air concentration are likely to occur. In the final rule risk analysis, sector-level air concentration/deposition values were generated for each chemical constituent. These sector-level concentration/deposition values were then used to derive sector-level concentrations for other media and ultimately sector-level exposure and risk estimates for modeled receptor populations. Up to four waterbodies were also selected for analysis within the GIS for each study area in order to assess surface water impacts to human and ecological receptors.

As indicated in response to the previous comment, EPA used a population-based approach to characterize the distribution of risk associated with HWC facilities in the final rule. U.S. Census and Census of Agriculture data were used to characterize the location/density of enumerated receptor populations at the sector-level (i.e., sector-level population counts were generated for those receptor populations that are referenced within the census data). These sector-level population counts were then used to weight the sector-level risk estimates by population to form the basis for cumulative risk distributions generated in the analysis. These population-weighted cumulative risk distributions were used to characterize central tendency and high-end risks based on various percentiles of the risk distribution (e.g., 50th, 90th, 95th, and 99th percentiles) for. Using the population data ensures that the exposure assessment is based on the actual location of receptor populations and that these form the basis of the high-end risk characterization, rather than assumptions about the location of the most impacted receptor. Methodologies developed and implemented for the final rule also allowed the aggregated impact of exposure parameter variability (interindividual variability), intersector variability, and interfacility variability to be assessed. EPA believes the cumulative risk distributions that reflect all of these sources ofvariability, in addition to population density, represent a significant improvement in characterizing the distribution of risk within a given receptor population.

***Comment 103, 104*:** The commenter cites the conclusion that "EPA's risk analysis developed for purposes of RCRA in fact shows that dioxin/furan emissions from hazardous waste incinerators could pose significant risks by indirect exposure pathways and that these risks would be reduced by BTF controls." The commenter feels that the results of this analysis are not so clear cut, and notes that depending on the source category, exposure scenario (i.e., whether subsistence activities are assumed), and whether high-end or central tendency emission estimates were used, going beyond the floor resulted in marginally lesser risks, identical risk, or higher risks.

Response 103, 104: *As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards, as prescribed in Section 112(d)(2)*

and (3) of the CAA. EPA is required to develop emission standards that are no less stringent than the performance achieved by the best control technology. For existing sources, EPA is required to set the emission standard based on the level of control achieved in practice by sources using the same technology, when properly designed and operated, as used by the average of the best performing 12 percent of sources. However, EPA may establish emission standards that are more stringent than this. EPA performed a risk assessment in order to evaluate whether the MACT standards are generally protective of human health and the environment, as required by Sections 3004(a) and (q) of RCRA. This analysis was necessary to satisfy EPA's concurrent obligations under RCRA and was done to determine if the technology-based standards are protective enough to satisfy RCRA or whether additional RCRA control remains necessary. EPA wishes to emphasize, however, that the level of the MACT standards is based solely on the factors made relevant under section 112 of the CAA, and implements those provisions alone.

EPA modified the risk assessment for the final rule in a number of important areas to increase the representativeness of the assessment and to make interpretation of risk results more transparent. EPA agrees with the commenter that the degree of risk reduction achieved by any given standard depends on the source category, exposure parameters, exposure scenario, and chemical constituent. Modifications made to the risk assessment conducted in support of the final rule include the addition of combustor categories and population-based risk characterization and the use of facility-specific emissions data and other site-specific data. In addition, for the final rule EPA projected emission reductions for each of the HWC facilities modeled on a site-specific basis considering baseline (i.e., current) emissions and the level of the standard. EPA believes that these modifications serve to better characterize the risk reductions that are likely to occur as a result the MACT standards.

Comment 20: The commenter cites the proposed rule's discussion of the cancer and noncancer risk results for hazardous waste incinerators as indicating that the results are expressed as a range that represents the variation in exposures across the example facilities (and example water bodies for surface water pathways) for the high-end and central tendency exposure characterizations across the exposure scenarios of concern. The commenter indicates that through experience in conducting risk assessments in-house using similar methodologies to those used for the risk assessment conducted to support the MACT standards, there is a linear relationship between the emission estimate and the risk results based on those estimates. The commenter notes, however, that data in some tables appear to indicate that the relationship is nonlinear. For example, if the carcinogenic risk result corresponding to the floor was based on an emission estimate derived for a particular example facility and 20 ng/dscm (the highest level known to be emitted at the floor) and the risk result corresponding to the BTF level was based on an emission estimate derived for the same example facility and 0.2 ng/dscm (proposed MACT standard), then the risk results for the two regulatory options should differ by two orders of

magnitude. The commenter states, however, the risks for the two regulatory options vary by considerably less than that, depending on whether the high or low end is considered. The commenter was unable to trace the source of the risk values presented in the summary tables because of the way that the *Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document Final Report*, (February 20, 1996) is organized.

Response 20: *EPA's risk analysis for HWCs uses the indirect exposure methodology issued in 1990 as the* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions. *The methodology was updated in 1993 with the draft Addendum (*Addendum to Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions, External Review Draft, *November 1993). For the HWC risk analysis, EPA further updated the indirect exposure methodology based on methods and data developed for the dioxin reassessment (e.g.,* Estimating Exposure to Dioxin-Like Compounds *(external review draft), U.S. EPA, 1994a,b) and the* Mercury Study Report to Congress *(U.S. EPA, 1997c), as well as other sources of information. EPA agrees with the commenter that, on a site-specific basis, the methodology exhibits a linear relationship between emissions, exposure, and risk (assuming all other modeling parameters remain the same).*

The emissions levels EPA used in the risk assessment at proposal are the levels given in the risk assessment background information document (see Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document, *February 1996). For dioxins and furans, the MACT floor was defined as a level of 0.2 ng/dscm, or 400EF. The use of temperature control to limit emissions results in variable emissions levels depending on the particular combustion unit. EPA projected the effect of temperature control on emissions for incinerators with dioxin emissions data and selected the 90th percentile of the distribution as a high-end value. This value was 4 ng/dscm and was used in the risk assessment at proposal for characterizing high-end risks. The value given in the footnote to table III.C.1. on page 17389 of the April 19, 1996, notice of proposed rulemaking (i.e.,20 ng/dscm) is a typographical error. Therefore, emissions at the MACT floor were substantially less the notice of proposed rulemaking would suggest.*

As indicated in the preamble to the proposed rule, risk results in the preamble were presented as a range where the range represented the variation in exposures across the example facilities and exposure scenarios for the high-end and central tendency exposure characterizations. Because the high-end and central tendency characterizations included emissions as one of the parameters that was varied, the risks at the low end of the range were based on emissions that were different than the risks at the high end of the range,

giving the appearance that the risks results were nonlinear when, in fact, the results were linear with respect to emissions.

For the final rule, EPA used facility-specific emissions estimates and projected emission reductions for each of the HWC facilities modeled on a site-specific basis considering baseline (i.e., current) emissions and the level of the standard. Facilities that were currently projected to be emitting below the level of the standard were assumed to continue to emit at baseline levels. Therefore, the relative reduction in emissions varied from facility to facility and between alternative MACT options levels (e.g., between the MACT floor and the standards) depending on a facility's baseline emissions.

Individual risk results for the final rule are presented as population-weighted cumulative risk distributions that are aggregated across all facilities modeled within a a given HWC category). Consequently, the aggregate risk reductions do not correspond in a 1:1 relationship with the relative reductions between alternative MACT options. Furthermore, the relative risk reductions for different receptor populations will differ because the cumulative risk distributions are weighted by population size. Therefore, although a reduction in a particular facility's emissions will result in a corresponding risk reduction for a given receptor, in the aggregate, the projected risk reductions will not be linear relative to the standards and will not be the same for different receptor populations.

Comment 21: The commenter feels that it is unclear why, in some cases, the baseline cancer and noncancer risks are less than those associated with the various regulatory options. This clearly needs to be explained given that promulgation of the MACT standards should result in some environmental benefit. Again, it has been the commenter's experience that the reductions in the risk values should be proportional to reductions in emissions.

Response 21: *EPA agrees with the commenter that the MACT standards will result in a reduction in risks from HWCs. EPA recognizes that, in the risk assessment for the proposed rule, the relationship between the estimated risks under the proposed standards and those at baseline is counterintuitive because the results appear to suggest an increase in risks. However, the baseline risks and the risks under the proposed standards are not directly comparable. In the risk assessment for the proposed rule, baseline emissions were set at either the 50th or 90th percentiles of the national distribution of stack gas emission concentration measurements for each source category depending on whether central tendency or high-end exposure scenarios were being characterized. Emissions under the MACT standards, on the other hand, were set at a (fixed) level that a facility would have to design to in order to meet the standards on a continuous basis (i.e., the design level, taken as 70 percent of the level of the standard). Depending on the level of the standard in relationship to the national distribution, the effect could be to either reduce or increase*

emissions under the proposed standards using this approach. As explained in the preamble to the proposed rule, the level of the standard was based on the highest emitter in the expanded MACT pool. Therefore, the design level correponding to the standard could be above the 50th percentile, or even the 90th percentile, of the stack gas emissions concentration measurements distribution for a given source category. In these instances, the effect was to increase emissions relative to baseline and, consequently, risks were also increased, as noted by the commenter.

As explained in response to a previous comment, for the final rule EPA used facility-specific estimates of emissions at baseline and projected emission reductions under the MACT standards for each of the HWC facilities modeled in the risk analysis on a site-specific basis considering baseline emissions and the level of the standard. In particular, facilities which were currently projected to be emitting below the design level corresponding to the standard were assumed to continue to emit at baseline levels while facilities that were currently projected to be emitting above the design level were assumed to lower their emissions to the design level. EPA believes that this approach provides a more realistic portrayal of how HWCs will behave as an industry in response to the revised emissions standards promulgated under the final rule. EPA recognizes, however, that any particular HWC facility could behave differently than assumed and that any set of assumptions regarding future emissions levels are inherently uncertain.

***Comment 294*:** The commenter received from EPA a set of hand-calculations (unfortunately they were for mercury, which has since been removed from the quantitative risk assessment) that allowed verification of portions of the basic methodology used in the risk assessment conducted in support of the proposed MACT standards. However, the commenter still found it difficult to navigate the risk assessment support document and had difficulty locating information necessary for verification of the results of EPA's risk assessment for the various regulatory alternatives. Specifically, the commenter was unable to locate the facility-specific emissions estimates calculated using the emissions data from stack gas concentrations in EPA's database. As a result, the commenter was unable to determine, for example, whether design levels were actually used in the risk assessment. If the design value is in fact always a lower value, and this was the value that was evaluated in the generic risk assessment, then the risks may in fact be underestimated. The commenter feels that, until this issue is clarified, it cannot be determined whether the MACT standards are in fact protective of human health.

Response 294: *As indicated in the preamble to the proposed rule, EPA used the design value for evaluating the human health impacts of the proposed standards. The design value is the level EPA expects a source would have to design to in order to be assured of meeting the standard on a continuous basis and hence is a lower value than the actual standard. While EPA acknowledges the uncertainty in determining a reasonable design value given the variability in the performance of air pollution control technology, EPA*

believes that most HWCs will operate at levels more typical of the design value rather than the actual level of the standard and, therefore, the design value is an appropriate basis for evaluating whether the MACT standards are generally protective of human health and the environment.

For the final rule, EPA prepared thorough documentation of all aspects of the risk analysis, including emissions and all other facility and site-specific data for modeled HWCs, model input values, algorithms, parameter values, and example calculations, as well as information on media concentrations and facility-specific risk results for certain receptors and sectors (i.e., maximum risk sectors for subsistence exposure scenarios). EPA believes that this information, together with the detailed description and discussion of the risk analysis framework, methods, and data provided in the risk assessment background document, is sufficiently detailed and complete to allow verification of key results and satisfies EPA's goal of transparency in all of its risk assessments.

At the time of proposal, a number of issues related to assessing risks from mercury had not been adequately resolved that would have allowed EPA to proceed with a quantitative analysis of mercury exposures and risks. As a consequence, the mercury calculations referred to by the commenter, which were provided to the Texas Natural Resource Conservation Commission in its capacity as co-regulator with EPA and participant on EPA's internal Agency work group for the HWC rule, were not included in the public docket as part of the record for the proposed rule. EPA has since issued its Mercury Study Report to Congress *(U.S. EPA, 1997c), a study that has been subject to extensive peer review, and the* Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units -- Final Report to Congress *(U.S. EPA, 1998b), both of which include quantitative modeling analyses of mercury exposures. Therefore, EPA now believes that sufficient technical basis exists for conducting a quantitative assessment of mercury exposures from HWCs. Such an analysis was performed for the final rule and followed the general modeling approach developed for the 1997 MRTC and used the IEM-2M surface water model developed especially for that report. EPA recognizes, however, that significant uncertainties remain and believes that the results of the mercury analysis should be interpreted with caution and be used only qualitatively.*

Comment 0089-2: The commenter cites EPA's contention in the proposed rule that exposures to mercury are significant when compared to EPA's estimate of the threshold at which effects may occur, given that the true threshold may be higher than EPA's estimate. The commenter requests that EPA elaborate on this discussion further given the extreme controversy concerning the current reference dose (RfD) for methylmercury (1×10^{-4} mg/kg/day). The commenter points out that EPA recently delayed release of the *Mercury Study Report to Congress* to await release of results from several studies designed to evaluate the effects of methylmercury in fish-eating populations (Seychelles Islands study, Faroe Islands study, Mancora study). To date, preliminary findings from

these studies have not shown a correlation between milestone attainment in infants or toddlers and prenatal methylmercury exposure. These findings may significantly alter EPA's threshold estimate for methylmercury.

Response 0089-2: *For the final rule, EPA is using a reference dose (RfD) for methylmercury of 1 x 10^{-4} mg/kg-day developed for EPA's 1997* Mercury Study Report to Congress *(U.S. EPA, 1997c). EPA defines the RfD as an estimate of a daily exposure to the human population, including sensitive subgroups, that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD for methylmercury is based on the data from a study of an incident in Iraq in which methylmercury treated seed grain was used for making bread (Marsh et al., 1987). EPA's Science Advisory Board, in its review of the MRTC, concluded that the RfD is supported by several epidemiological studies involving chronic exposure from fish (including studies on the Cree Indians and in New Zealand) as well as experimental animal data, and that the above RfD should be retained.*

At the time of the finalization of the MRTC, considerable new data on the health effects of methylmercury were emerging. These data included large studies of fish and marine mammal–consuming populations in the Seychelles and Faeroe Islands. Because the majority of the Seychellois and Faeroese data have not been subject to rigorous review, EPA considered it premature to re-evaluate the RfD for methylmercury.

*EPA and other federal agencies participated in an interagency review of available human neurodevelopmental data on methylmercury, including the most recent studies from the Seychelles and Faeroe Islands (*Report of the Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury, *November, NIEHS, 1998). The purpose of this review was to evaluate the major epidemiologic studies associating methylmercury exposure with an array of neurodevelopmental measures in children and to facilitate agreement on risk assessment issues. The workshop was a response to the need for the Seychellois and Faeroese data undergo a level of scrutiny beyond journal peer review if they are to be used in setting policy. The panel concluded that the results from the Faeroes and Seychelles studies provide valuable insights in the potential health effects of methylmercury but that significant uncertainties remain, because of issues related to exposure, neurobehavioral endpoints, confounders, statistics, and study design. The panel felt that continuation of these studies is necessary for their full potential to be realized.*

The National Academy of Sciences (NAS) is currently independently assessing EPA's RfD for methylmercury. Pending the completion of the NAS study, EPA will reevaluate the RfD for methylmercury following careful review of the results of the NAS study.

Comment 295: The commenter cites the proposed rule as indicating that air concentrations were the sum of the vapor and particulate concentration. The commenter notes that it would appear that air intakes were calculated as opposed to directly comparing predicted air concentrations to inhalation reference concentrations and unit risk factors. It is the commenter's understanding that EPA's current position is that, due primarily to portal-of-entry effects, it is more accurate to evaluate risk from exposure to air by doing a direct comparison between the predicted air concentration and the toxicity value presented in the Integrated Risk Information System (IRIS) (which are in terms of an air concentration) rather than calculating an internal dose.

Response 295: *For both the proposed and final rules, EPA calculated air concentrations of chemical contaminants as the summation of vapor air concentration and particle-bound air concentration. For the purpose of assessing the potential for noncancer effects, EPA compared the estimated air concentration (vapors and particles) to EPA's reference concentration (RfC). The RfC is an estimate of a concentration in air that is likely to be without an appreciable risk of deleterious effects in the human population, including sensitive subgroups, from continuous exposures over a lifetime. For assessing cancer risks via inhalation, EPA derived an inhalation slope factor (inh CSF) from the inhalation unit risk factor (inh URF). The inhalation URF assumes lifetime exposures and a default inhalation rate and for this reason, as well as for reasons of consistency, EPA wanted to use the same exposure assumptions that were made for noninhalation exposures. Therefore, instead of using the air concentration and inhalation URF, EPA evaluated cancer risks using the inhalation CSF and inhalation rates provided in EPA's 1997* Exposure Factors Handbook *(EFH) (U.S. EPA, 1997a) to calculate an inhaled dose. As indicated by the commenter, EPA is concerned about portal of entry effects and, for this reason, EPA did not as a general matter use oral slope factors to assess cancer risks from inhalation exposures but instead used inhalation slope factors that were derived from the inhalation unit risk factor.*

For the final rule, EPA used inhalation rates that are the same across different receptor populations but differ between age groups. Inhalation rates of 6.5 m^3/day, 11.8 m^3/day, 14.0 m^3/day, and 13.3 m^3/day were used for those persons age 0-5 years, 6-11 years, 12-19 years, and over 20 years, respectively. Using age-group-specific inhalation rates, EPA calculated an inhalation lifetime average daily dose. The cancer risk was calculated as the product of the inhalation lifetime average daily dose and the inhalation cancer slope factor (inh CSF). Inhalation CSFs were used to characterize cancer risks associated with inhalation exposures by adults and children.

***Comment 296*:** Whole weight consumption rates were used for dioxins and all of the metals except cadmium and selenium. It is unclear why a dry weight consumption rate was used for cadmium and selenium.

Response 296: *In the risk analysis for the final rule, biotransfer factors for cadmium and selenium in beef and milk were taken from EPA's 1992* Technical Support Document for Land Application of Sewage Sludge *(U.S. EPA, 1992). In this document, uptake slopes based on dry weight animal tissue concentrations were developed for metals commonly detected in sewage sludge. EPA converted these uptake slopes to biotransfer factors based on animal intake rates. The resulting biotransfer factors represent the dry weight animal tissue concentration per unit daily metal intake. However, human consumption rates for animal food products are expressed in whole weight terms, rather than dry weight. Therefore, the dry weight animal tissue concentrations calculated using the biotransfer factors from the sewage study were converted to a whole weight basis by multiplying by the dry weight fraction. The 1990* Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions *(U.S. EPA, 1990) suggests that the water content for beef is 0.6, which translates to a dry weight fraction of 0.4, and the water content for cow's milk is 0.9, which translates to a dry weight fraction of 0.1. The biotransfer factors for the remaining metals evaluated were taken from a 1984 study performed by Oak Ridge National Laboratory entitled* A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture *(Baes et al., 1984). These factors were based on a whole weight basis, so conversion from a dry weight was not necessary.*

Comment 297: The commenter notes the utilization of health benchmarks that are verified through the EPA work groups and available on EPA's Integrated Risk Information System, with the exception of the dioxin toxicity equivalency methodology. However, the commenter points out that neither the slope factor nor the inhalation unit risk factor for 2,3,7,8-TCDD are currently available on IRIS. Likewise, neither the health-based level in soil for lead nor the benchmark value used for comparison with an average daily dose to an infant via exposure to dioxin-contaminated breast milk are available on IRIS. The commenter feels that it is also debatable whether it is appropriate to state that any of the benchmarks mentioned here have been verified through the EPA work groups.

Response 297: *For the HWC risk analysis, EPA used the cancer slope factor (CSF) for 2,3,7,8-TCDD from EPA's 1985 health assessment document,* Health Assessment Document for Polychlorinated Dibenzo-p-dioxin *(U.S. EPA, 1985). Because dioxins are associated with cancers in a variety of sites in the body and portal of entry effects are not expected, EPA used the slope factor from the 1985 assessment for both oral and inhalation exposures. Although EPA is reevaluating the cancer slope factor for the dioxin reassessment, until the assessment is concluded EPA is using the slope factor from the 1985 assessment.*

For the final rule, EPA assessed the risk to infants from exposures to dioxins through ingestion of breast milk using a modified margin of exposure (MOE) approach. In this approach, the estimated average daily dose (ADD) to the infant from HWCs was

compared to background exposure levels to infants in the general population that breastfeed. Since exposures through breast milk are expected to be among the highest exposures that occur during early childhood and given the concern for developmental effects, EPA believes that it is important to assess the potential risks during this critical exposure period. If it can be concluded that the potential for risks during this time period is small, then exposures at other times and through other pathways are not likely to be cause for concern. EPA estimated background exposures from breast milk ingestion to be 50 pg/kg/day as TCDD-TEQ, based upon a measured U.S. background level of 16 ppt in the lipid portion of breast milk, a maternal milkfat fraction of 0.04, a breast milk ingestion rate of 0.8 kg/day, and an infant body weight of 10 kg from data provided in the Volume III of the draft dioxin health assessment document.

For the final rule, EPA assessed the risks from lead by evaluating the incremental blood lead level (PbB) in children from HWCs. PbB levels were modeled using EPA's Integrated Exposure Uptake Biokinetic Model (IEUBK). EPA compared the modeled PbB levels to a community action level of) of 10 µg/dL established by the Centers for Disease Control and Prevention in 1991 (CDC, 1991). *EPA's goal is to reduce children's blood lead to below this level. Although EPA did use a presumptively protective soil level for lead in the proposed rule, for the final rule EPA used the IEUBK blood lead model, which considers dietary, inhalation, and dust exposures in addition to soil exposures. EPA believes it is important to consider the combined exposures from all these exposure pathways in assessing risks from lead.*

***Comment 299, 300*:** The commenter notes that the source for the dry deposition velocity (Vdv) of 0.2 cm/s for dioxins should be cited. Further, the commenter notes that this value for Vdv of 0.2 cm/s for dioxins is reiterated in Table C-3.1, Soil Concentration Due to Deposition for Noncarcinogenic Compounds. The commenter notes that this appears to be in error since the noncarcinogenic effects of dioxins were not evaluated in the risk assessment conducted in support of the proposed MACT standards. According to the letter sent to the commenter on September 28, 1995, containing equations and hand calculations for mercury, the Vdv value used for noncarcinogenic constituents should be 3 cm/s. The commenter notes that the source of the Vdv value of 3 cm/s should also be added.

Response 299, 300: *For the final rule, EPA evaluated the noncarcinogenic effects of dioxins using an incremental margin of exposure analysis. For estimating vapor deposition to soils, EPA used a dry deposition velocity (Vdv) of 0.2 cm/s), from a 1992 publication "Wet and dry deposition of chlorinated dioxins and furans" (Koester and Hites, 1992). Use of this value for characterizing vapor deposition is uncertain and may overestimate vapor deposition because it presumes that dioxin vapors and particles have the same dry deposition velocities. EPA believes that a value of 3 cm/sec as indicated by the commenter is inappropriate for dioxins. With regard to mercury, for the final rule,*

EPA used a site-specific dry deposition velocity for divalent mercury vapor depending on land use. The mercury calculations referred to by the commenter, which were provided to the Texas Natural Resource Conservation Commission in its capacity as co-regulator with EPA and participant on EPA's internal Agency work group for the HWC rule, were not used and were not included in the public docket as part of the record for the proposed rule.

***Comment 301*:** The commenter notes that there appears to be an error in Table C-5.2 Contaminant Intake from Aboveground Produce and Root Vegetable Intake, in the units listed for Iag. The units for Iag are listed as mg/kg. However, the commenter notes that as it is written, the equation produces an intake in mg/day. Similarly, the commenter notes that there appears to be an error in this same table in the units listed for Ibg. The units for Ibg are listed as mg/kg. The commenter notes that as it is written, the equation produces an intake in mg/day.

Response 301: *Table C-5.2 of the risk assessment background information document for the proposed rule (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document*, February 1996) contains a typographical error in the units of the equations for daily contaminant intake from aboveground vegetables (Iag) and belowground vegetables (Ibg). As pointed out by the commenter, the correct units are mg/day. These are the intake units EPA used for the final rule.*

Comment 302: The commenter notes that there appears to be an error in Table C-6.1, Concentration in Maternal Milk, in the units listed for C (milkfat). The units for C (milkfat) are listed as pg/kg. The commenter notes that as it is written, the equation does not produce a C (milkfat) on a per-kg basis. The commenter notes that to produce C (milkfat) on a per-kg basis, the adult body weight (i.e., 70 kg) should be added to the denominator of the equation.

Response 302: *Table C-6.1 in the risk assessment background information document for the proposed rule (*Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Information Document*, February 1996) omits the maternal body weight from the calculation, as indicated by the commenter. The maternal body weight is required to calculate the maternal dose in mg/kg/day. For the final rule, EPA calculated the maternal dose using the body weight of nursing mothers. The body weight for women of child-bearing age was taken as 65 kg (mean female body weight [18-75 yr]).*

Section 40
Vulcan Chemicals
RCSP-0097

***Comment 121, 122*:** The commenter states that EPA cannot currently assess the magnitude of risks that can routinely be expected while burning of hazardous waste in HWCs. According to the commenter, EPA states, "With respect to mercury and non-dioxin PICs, the Agency does not at this time have sufficient reliable data to be able to assess, on a national basis, the magnitude of the risks that can routinely be expected from burning hazardous waste in HWCs." The commenter notes that EPA does not currently posses the knowledge of the health effects due to exposure to combustion products from hazardous waste incinerators. The commenter states that since EPA has no evidence that the current standards are unprotective, it is premature to set further more restrictive emission standards. The commenter suggests that EPA wait until the a risk assessment is done on these emissions to determine the safe exposure levels. The commenter feels that until EPA is able to do this, it is setting standards for substances based on very conservative estimates that may or may not be true.

Response 121, 122: *The establishment of NESHAP for sources that emit HAPs is required by Sections 112(c) and 112(d) of the Clean Air Act. As explained in the preamble to the final rule, EPA used the MACT process to set technology-based emission standards. However, Sections 3004(a) and (q) of RCRA mandate that standards governing the operation of HWC facilities be protective of human health and the environment. Therefore, EPA conducted a risk assessment to evaluate whether the MACT standards satisfy this requirement in order to determine what, if any, RCRA standards for emissions from these sources may be needed.*

EPA considers the environmental impact of pollutants in combustor emissions to be a fundamental issue associated with the increasing use of incineration. Recent studies have linked elevated levels of pollutants in soils, lake sediments, and cow's milk to atmospheric transport and deposition of pollutants from combustion sources. Typical pollutants emitted from HWCs include acid gases (HCl, SO_2, NO_x, and Cl_2), dioxins/furans, metals (such as mercury, lead, nickel, and cadmium), particulates, and products of incomplete combustion. These pollutants, through wet and dry deposition, become deposited on soil and waterbodies, resulting in accumulations in plants and animals. Humans and wildlife may become exposed through ingestion of soil, ingestion of food products, ingestion of drinking water, and inhalation. The final rule evaluates risks associated all constituents for which adequate emissions data were available.

The risk assessment for the final rule included an analysis of risks from mercury. Although EPA assessed the risks from chlorinated dioxins and furans, risks from other organics that may be present as PICs could not be assessed quantitatively due to

limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited, are highly variable, and are therefore inadequate for making national emissions estimates. As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit, circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

***Comment 126, 131*:** The commenter points out that PIC emissions need to be put in the proper perspective, and that EPA failed to do so. The commenter notes that, in preparing the emission standards for hazardous waste burning boilers and industrial furnaces (BIF), EPA compiled and assessed data on typical PIC emissions from eight full-scale HWC sources (54 FR 43737). The commenter cites EPA's conclusion from this assessment, which says PIC emissions do not pose significant risks when incinerators are operated under optimum conditions. The commenter further cites EPA's definition of high combustion efficiency and the correlated minimum emissions of unburned (or incompletely burned) organics.

Response 126, 331: *EPA evaluated all chemical constituents for which adequate emissions data were available. Although EPA assessed the risks from chlorinated dioxins and furans, risks from other organics that may be present as products of incomplete combustion (PICs) could not be assessed quantitatively due to limitations of the data available for analysis, including a lack of adequate emissions data on nondioxin PICs. While it is known that a variety of PICs are emitted from HWCs, unlike dioxins and furans, emissions measurement data of acceptable quality for nondioxin PICs are quite limited, are highly variable, and are therefore inadequate for making national emissions estimates. A number of PICs known to be emitted from HWCs are human carcinogens (e.g., hexachlorobenzene, benzo(a)pyrene, and others). Omission of quantitative risk estimates for these HAPs may have resulted in the potential risks being understated in the risk assessment for the final rule.*

As best as it can be determined now, formation of nondioxin PICs is a site-specific phenomenon and depends, among other things, on the type of combustion unit,

circumstances of combustion, and types of hazardous wastes burned. Under these circumstances, EPA believes the uncertainty is too great to attempt to quantify risks from nondioxin PICs at the national level. Although it is unclear whether nondioxin PICs pose a significant risk, given the certainty that nondioxin PICs are formed and will be emitted, EPA continues to be concerned about such emissions. Therefore, EPA expects that during implementation of the rule, permitting authorities will evaluate the need for risk assessments for individual HWCs on a case-by-case basis under the omnibus provision of RCRA Section 3005(c)(3), including the need to assess any risks from nondioxin PICs. Additional permit conditions may be established if necessary to reduce risks from such emissions.

***Comment 328*:** The commenter believes that Administrator Carol Browner's new Risk Characterization Policy is not being followed. The commenter cites Administrator Browner's March 21, 1995, memorandum"EPA Policy for Risk Characterization" to support this point. The commenter notes that, unfortunately, risk characterization discussion in the preamble of this rule mixed science with policy decision. The commenter feels that science was often twisted to support policy decision. The commenter states that in the proposed rule, EPA is still continuing the past practice of using the most conservative approach in order to cover all potential threat no matter how trivial or how remote, and common sense is rarely considered.

Response 328: *EPA recognizes that the risk analysis performed at proposal had inherent compounded conservatism, and EPA made several major modifications to the risk analysis completed in support of the final rule to address this issue. These modifications included updates to both the methodology and parameter values used.*

It has been EPA's policy since release of its draft hazardous waste minimization and combustion strategy in 1993 to assess the risks associated with indirect exposures to emissions from HWC facilities as part of the RCRA permitting process. EPA has chosen to use the indirect exposure methodology issued in 1990 as the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions, *(U.S. EPA, 1990), which was updated in 1993 with the draft Addendum (U.S. EPA, 1993a) in the risk assessment for the HWC final rule. Scientific knowledge and understanding have continued to improve since the draft Addendum was completed. Consequently, EPA updated the indirect exposure methodology based on information from the dioxin reassessment* (Estimating Exposure to Dioxin-Like Compounds, External Review Draft, *June 1994, U.S. EPA, 1994a,b) and the December 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), as well as other sources of information for the risk assessment for the final rule.*

EPA agrees that combining several high-end parameter values can result in an estimate that lies well out on the tail of the distribution, depending on the model's sensitivity to

the particular parameters. This is why, for the final rule, EPA characterized exposures using central tendency parameter values(rather than high-end values) in conjunction with site-specific data and used probabilistic modeling to consider the variability of these parameters. Monte Carlo simulation was used to evaluate the impact of variations in exposure parameters on individual risk for key risk-driving receptor population/constituent combinations (including beef and dairy farmers for dioxin and recreational fishers for methylmercury).

In addition, the risk analysis for the final rule used site-specific emission estimates to estimate media concentrations and concentrations in agricultural products. Also, U.S. Census and Census of Agriculture data were used to locate exposed individuals. Separate exposure estimates were made for individuals engaged in several different types of commercial farming as an occupation or fishing as a recreational sport and persons engaged in farming or fishing for subsistence. All of these modifications were designed to produce exposure estimates that are more representative of the different receptor populations being modeled with respect to both behavior and location.

EPA believes that these steps provide considerable assurance that the exposure estimates for the HWC rule are not overly conservative.

***Comment 332*:** The commenter notes that the risk analysis did not put PM emissions from HWC sources into proper perspective.

Response 332: *EPA agrees with the commenter concerning the health effects of fine particulate matter (PM). For the final rule, EPA assessed the risks associated with particulate matter emissions ($PM_{2.5}$ and PM_{10}) apart from the chemical specific risks from individual chemical constituents. This analysis evaluated the incidence of PM related health effects avoided by reducing PM emissions as a result of the MACT standards. Health endpoints considered included mortality, hospital admissions, and respiratory symptoms. The results of the analysis indicate that risk reductions are expected for a number of health endpoints, including reductions in mortality, hospital admissions, chronic bronchitis, and respiratory symptoms, although the reductions are modest relative to those achieved by controls on other sources of PM.*

***Comment 0097-1*:** The commenter states that EPA seems to have twisted science in interpreting mercury background fish data. The commenter cites the proposed rule in which EPA describes the collection and analyses of fish tissue for mercury. According to that description, chemical residues in fish data were collected from 388 locations nationwide; at 92 percent of the locations, fish were found to contain detectable levels of mercury. The commenter cites EPA's comment regarding the location of the highest concentration detected (1.8 ppm) and the conclusion that the remote site could be considered to represent background conditions. The commenter also cites EPA's statement that

similar results obtained in other studies strongly suggest that long-range atmospheric transport and deposition of anthropogenic emissions is occurring.

The commenter feels that EPA's risk characterization for this proposal, by crediting the highest concentration that was observed in the background fish samples as due to long-range atmospheric transport and depositions, has clearly violated Administrator's Browner instruction that "[r]isk assessments should be transparent, in that the conclusions drawn from the science are identified separately from policy judgments." The commenter states that if long-range atmospheric transport and deposition is the true reason for a given level of mercury, the observed concentration level would be uniformly distributed over a very wide area, rather than producing the highest background concentration at an isolated remote area, and concludes that EPA's statement seems to be trying to twist science to justify a policy decision.

> ***Response 0097-1:*** *EPA believes there is ample evidence that long-range transport of anthropogenic emissions of mercury are contributing to mercury levels in remote areas. As noted by Fitzgerald et al., 1998 ("The case for atmospheric mercury contamination in remote areas"), recent experimental results indicate that local-scale geochemical processes alone cannot explain the mounting number of lake sediment and peat profiles showing substantial increases of mercury flux during the past century. Atmospheric and aquatic cycling of mercury and the bioaccumulation of monomethylmercury in aquatic systems are driven by complex chemical and biological reactions involving trace amounts of mercury. Analytical advances in measuring environmental mercury at trace levels have greatly narrowed estimates of natural mercury fluxes and support the case for long-range atmospheric transport of mercury to remote areas. The similar timing and magnitude of recent increases and a concordance with spatial trends in measured mercury deposition strongly support that long-range transport of anthropogenic mercury is the cause of increasing mercury concentrations and fluxes in the sediments of lakes and sparsely populated regions that are not affected by localized human-related sources of mercury.*
>
> *As explained in EPA's 1997* Mercury Study Report to Congress *(MRTC) (U.S. EPA, 1997c), mercury cycles in the environment as a result of natural and anthropogenic activities. Most of the mercury in the atmosphere is elemental mercury vapor, which circulates in the atmosphere for up to a year and can be dispersed and transported thousands of miles from the source of emission. A computer simulation of long-range transport of mercury suggests that about one-third of U.S. anthropogenic emissions are deposited through wet and dry deposition within the lower 48 states. The remaining two-thirds is transported outside of U.S. borders, where it diffuses into the global reservoir. The highest deposition rates from anthropogenic and global contributions are predicted to occur in the southern Great Lakes and Ohio River Valley, the Northeast, and scattered areas in the South. The location of sources, the chemical species of mercury*

emitted, and climate and meteorology are key factors in mercury transport and deposition. The flux of mercury from the atmosphere to land or water at any one location is comprised of contributions from the natural global cycle including re-emissions from the oceans, regional sources, and local sources.

For the final rule, EPA conducted a quantitative assessment of risks from mercury to both human and ecological receptors. The mercury analysis for the final rule evaluated impacts from mercury emissions on local bodies of water in the immediate vicinity of HWCs. However, EPA did not evaluate the specific impacts resulting from long-range transport of HWC emissions beyond local study areas for the final rule.

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