EXTERNAL PEER REVIEW-HUMAN HEALTH RISK ASSESSMENT PROTOCOL FOR HAZARDOUS WASTE COMBUSTION FACILITIES

PEER REVIEW COMMENTS

U. S. EPA
RCRA DOCKET NO. F-1998-HHRA-FFFFF

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May 11, 2000

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I. INTRODUCTION

Background

The U.S. Environmental Protection Agency (EPA), Region 6 has developed the interim final draft document, *"Human Health Risk Assessment Protocols for Hazardous Waste Combustion Facilities"* (HHRAP) for the Center of Combustion Science and Engineering (CCSE), in coordination with the Office of Solid Waste. The HHRAP will provide guidance to risk assessors dealing with the direct and indirect exposure from potential contaminants emitted from stationary combustion sources. The HHRAP has undergone extensive internal peer review by EPA and State personnel. The HHRAP is intended to support the Office of Solid Waste in providing permitting authorities for hazardous waste combustion facilities with state-of-the -art methodology in predicting the risk associated with the exposure to compounds found in the air, soil and water. When published, the final document will serve as the national guidance for conducting human health risk assessments at hazardous waste combustion facilities. Because this guidance has been determined to be a "major scientific and technical work product", the Office of Solid Waste has requested that the HHRAP undergo an independent, external peer review.

Nine peer reviewers were selected according to the guidance set forth in EPA's "External Peer Review Guidance" document (EPA 100-B-98-001). The peer reviewers were chosen from the following disciplines: combustion engineering, atmospheric modeling, human exposure, chemical fate and transport, and human health toxicology. The selected peer reviewers are as follows:

Dr. William Schofield	Combustion Engineering
Dr. Water Dabberdt	Atmospheric modeling
Dr. James Butler	Human Exposure
Dr. Richard DeGrandchamp	Human Exposure
Mr. Steve Washburn	Human Exposure
Dr. Mary Davis	Human Health Toxicology
Dr. Thomas Gasiewicz	Human Health Toxicology
Dr. George Fries	Fate and Transport
Dr. Douglas Smith	Fate and Transport

Each peer reviewer was provided specific charge statements developed by the EPA Region 6, by which to evaluate the *HHRAP*. The peer reviewers were only responsible for answering their specific charge statements; however, they were given the option to review other sections of the *HHRAP*. Attached is a collation of the nine independent peer reviewers comments on the *HHRAP*. The

comments are organized according to topic areas (e.g, human exposure). Each peer reviewer name is associated with the specific topic area.

This report was prepared for the U.S. Environmental Protection Agency (EPA), Region 6, by TechLaw, Inc. in fulfillment of Contract No. 68-W-99-017, Work Assignment No. R06711. Any opinions, findings, and conclusions expressed herein are those of the TechLaw subcontractors and not necessarily those of the EPA or cooperating agencies. Mention of any company or product names is not to be considered an endorsement by the EPA.

II. COMBUSTION ENGINEERING

Dr. William Schofield

General Issues

In addition to providing review and comment on assigned specific technical issues, each reviewer should also address the following general issues, as applicable:

1. Comment on the organization of the section reviewed. Is the presentation of information clear and concise considering the technical complexity of the subject and intended audience?

   This draft is far superior to previous drafts in terms of logical consistency, clarity, and direction to permit writers, etc., about conducting risk assessments (RAs). However, inconsistencies continue as discussed on a specific basis, below,

   Comments on specific points:

   A. On Page 1-3 the guidance states: "Any decision to add permit conditions based on a site-specific RA under this authority must be justified . . . and the implementing Agency should explain the basis for this condition."

      However, on Page 2-5 the Guidance states: "If a facility desires to receive a permit with no limits other than those traditionally based . . . on a trial burn (TB), then risk testing should be conducted at trial burn or worst case conditions." Elsewhere on the page and elsewhere in the Guidance it is stated that TB risk data is expected to have higher emissions than would occur at normal conditions. Furthermore, no consideration is given in the statement about added permit conditions as to the level of risk shown in that facility's RA. For example, if a facility conducted a risk burn (not a trial burn) and shows a risk of 1.0 x 10-7, why would permit conditions be needed since a risk <<10-5 would seem to be protective without additional limits.

   B. On Page 1-6 the Guidance states: "The HHRAP recommends a process for evaluating reasonable - not worst case maximum potential risk . . . Conservative assumptions should be made only when needed to ensure that emissions do not pose unacceptable risks."

However, on Page 3-5 the Guidance states, "The lessor of the 95th percentile or the maximum stack gas concentration from the three trial burn runs should be used to develop the emission rate estimate used in the RA." As already discussed, the emissions during a TB are expected to exceed emission from long term average conditions.

If so, why the need for another level of conservatism? Why not apply this requirement only to RBs at more normal conditions. This argument is supported by the requirement that worst case waste be burned during the test and the resulting emission rates used to characterize long term risks. Level upon level of conservatism distorts the results of the RA process and jeopardizes its usefulness to reliably identify those HWC facilities that may pose a risk to the public. Other examples of worst possible assumptions include: (1) the CARB upset scale-up factor, and (2) use of RDL values for ND COPCs (both are discussed below).

C. Concerning the intended audience, I suggest that the stated purpose should explicitly include use by the regulated community since the document will be used by many facilities. Inclusion of the regulated community in your audience gives the Agency an opportunity to explain/justify conservative assumptions and reduce the perception of an overly conservative RA process which could well have severe repercussions to that group. Additionally, at several points in the guidance the phrase "Agency consensus" was used. This entire process would work more effectively if the consensus could be widened to include the regulated community.

2. Does the purpose of the HHRAP as stated in the Introduction (Chapter 1) accurately reflect the presented methodologies and scope?

In general, yes. However, it appears that as presented the RA process is overly conservative; a clear imbalance in emphasis on very fine detail on RB analytical data that is inconsistent with the level of accurate available in approximate/non-existent toxicity data, analytical methods, and fate and transport calculations.

3. Evaluate critically the scientific aspects (within your expertise) of the document, including methodologies, exposure factors and scenarios, parameters and defaults and risk characterization. Interpretations of scientific conclusions should be based on sound biological principles and accurate and legally defensible scientific support.

The current state of the art of RAs, including the parameter values used in the RA models, limitations in stack sampling/analytical, etc., could best be described as allowing an order of magnitude analysis of actual risks, i.e., with full site-specific model content and realistically conservative assumptions when needed. The models can suggest a risk that is 10-5, 10-6, 10-7, etc. Attempting a finer resolution of risk (1 x 10-6 *versus* 2 x 10-6) is unrealistic at this time. When overly conservative assumptions are made, higher than actual risks would be calculated. Several examples are discussed within these comments.

There appears to be an undue emphasis placed in the guidance on being able to say with legally defensible certainty that actual stack emissions from a given facility are less than those used in the facilities' RAs. To meet this objective, the guidance requires that RDL values be used for non-detect COPC stack compounds even when an unbiased estimate of actual concentration (in the form of J-values) is available. It appears that in an effort to have bullet-proof COPC stack concentration numbers, the best information available is ignored and replaced by frequently much higher RDL values. Why disregard this information, especially when it may jeopardize the usefulness of the overall process, i.e., facilities with few, if any, hits could fail the RA. The whole process loses credibility in this event. The RA process as suggested by the agency should produce the best possible estimate of actual risk and conservative assumptions should be used in the face of uncertainty. The agency should implement the RA process in a manner that is and appears to be outcome-independent.

The agencies have sufficient authority to add and defend permit limits where they are truly needed under the RCRA Omnibus Provision. This is especially true when the RA process is implemented as described above, i.e., a facility can reasonably protect its rights through technical discussions with the agencies to resolve issues rather than having to resort to the courts.

4. Give your opinion whether the risk assessment supports not only a credible interpretation of what is known, but also a credible interpretation of the hazard and risk that is predicted.

The RA Guidance provides a reasonable albeit very conservative interpretation of what is known but the process as described overstates actual risk. Some conservativism is necessary due to a wide range of issues where uncertainty exists. A conservative approach is appropriate and necessary to be protective of the public. However, the most conservative approach in each and every facet of the models distort the results.

Suggestion: Use the best information available, make conservative assumptions only where necessary, and avoid layers of conservative assumptions just to produce a very conservative result.

5. As with any risk assessment, there are always additional data and method development efforts that could be undertaken to reduce the level of uncertainty. However, are there any major data or methodological gaps within this guidance specific to the sections reviewed that would preclude using for regulatory decision making? If so, how should they be addressed?

The agency appears to be acutely aware of information gaps, the difficulty in filling those gaps, and is making reasonable efforts to fill in gaps as soon as possible.

One idea that would allow the agency and the regulated community to adopt a more focused approach to conducting risk assessments would involve the development of a comprehensive database of TB/RB/RA results that would be available to the public for conducting analyses of real-world situations. At this point the guidance basically requires a shotgun (unfocused) inclusion of almost all RA factors in all RAs regardless of site-specific circumstances. Analysis of a comprehensive database would permit more selectivity in including only those factors which would likely have an impact on a given facility's perceived risk. For example, on a national basis there are frequently two to six HWC units burning similar waste. Once the results from two or three of these units are included in the database, the agency, the public and the regulated community could search for the important factors applicable to the other units in that category.

At this point, there appears to be approximately $30 - 50 million worth of RB/TB data which is currently available to the agency (without requiring a long, drawn out research program) that is not being used to guide current and future RB/TB/RA efforts.

6. What long-term research would you recommend that could significantly improve risk assessments of this type in the future?

None in my area of interest.

Technical Issues

The reviewer is charged with considering and providing written comment recommendations on specific technical issues generally defined as being within the specific discipline of combustion engineering. These specific technical issues were identified through public comment as being significant and requiring additional external review. The reviewer should be familiar with the section of the HHRAP referenced within the technical issue.

1. Comments were received regarding guidance presented for inclusion of the "unknown" or unspeciated total organic emission (TOE) data when estimating stack emission rates (Section 2.2.1.3). Considering the technical complexity of this issue, is guidance on quantifying unspeciated TOE data for use in the risk assessment adequate and presented clearly?

   While the TOE method is imperfect and should be improved, it does provide information pertinent to a HWC RA. The use of TOE information in the Uncertainty Section of RA Reports is appropriate. By definition, if one does not know what compounds are present, one does not know what toxicity factor would apply. In the absence of information there is room for a substantial range of opinion concerning the relative toxicity of unidentified compounds to those compounds which were identified. (I have heard opinions ranging from $Risk_{UNK} = Risk_{Dioxin}$; $= Risk_{IDENT}$; to $<< Risk_{IDENT}$). As time passes and EPA improves EPA- approved analytical methods and as we learn more about the compounds present in the TOE_{GRAV} fraction by all available methods, this should be a less significant issue.

   For the reasons given above, scaling up emission rates or risk levels to reflect the unidentified fraction of TOE is inappropriate and has no scientific basis. This is especially true if such scale-ups lead to additional permit limits.

2. Comments were received regarding guidance presented for considering process upsets in estimating emission rates (Section 2.2.5). Is additional detail and clarification of guidance specific to this issue required?

   The default CARB upset factors are excessively conservative. No HWC facility would be allowed to operate in upset conditions for 20% (organics) and 5% (metals) of the time. Also, hazardous waste combustion is prohibited under RCRA during startup and shutdown periods by the automatic waste feed cut off (AWFCO) system. The only potential "upset" condition for a HWC facility is the

period of time when permit operating limits are exceeded while hazardous waste remains in the combustion chamber. This situation may occur at a facility burning significant quantities of solids and sludge which pass relatively slowly through the system to effect treatment (e.g. cement kilns, rotary kiln incinerators).

The technical information available to me also indicates that upset emissions are not close to 10-times normal emissions. Upsets occur fairly frequently during TB/RB sampling conditions with minor apparent impact on measured concentrations. L. Waterland reported results from the EPA research incinerator in Arkansas at the Incineration Conference (circa 1995). In this work, various types of upsets were intentionally caused while emissions data were collected. Little or no increase in emissions was observed.

Most facilities do not have concrete records that contain the durations of upsets and means of estimating emissions during the upsets. However, some do. As an example, the operation of the current GTX facility in LA (formerly Marine Shale Processors) was under heavy compliance scrutiny during much of the 1990s. This is a large commercial facility with substantial solid/sludge capabilities which was not perceived by the agency as a model facility. However, because of the compliance attention GTX had sufficient information to estimate upset factors, as summarized below.

PARAMETER	**CARB DEFAULT PARAMETERS**		**ACTUAL PLANT RESULTS**
Estimated Fraction of Operating Time Facility is Upset	For Organics:	0.2	0.0009
	For Metals:	0.05	0.0004
Estimated Emissions Increase During Upset	For Organics:	x 10	x 1.12
	For Metals:	x 10	x 1.46

These results were calculated from information which was reported contemporaneously to LDEQ and EPA Region VI. The work producing the values in the above table was performed in support of GTX' recent RA. This information was supplied to EPA Region VI in 1998 and the results accepted for use by EPA in completing GTX' RA.

Analysis of these reports, which were based largely on third party efforts (e.g., Geraighty & Miller, Focus Environmental) and available for review by the agency almost immediately, reveals a very large discrepancy between the CARB default values and actual field results regarding frequency/ duration of upsets. Most HWC facilities would be expected to have much lower upset durations (small, simple boilers from which combustion gases would exit the unit within 2 - 6 seconds, compared to approximately one hour for HW residues to clear a large rotary kiln such as GTX'). Also, most HWC facilities burn a relatively small number of clean liquid waste streams. Many of these streams are considered hazardous due to ignitability only, a characteristic that generally reflects its ability to burn well. However, most of these smaller, simpler facilities have the disadvantage of not having upset duration data or emissions estimates during upset conditions. Thus, per guidance, these less threatening facilities would be required to use the excessively conservative CARB default values. This is another example of inconsistency (see General Comments 1B, above). The CARB default values are worse than "worst case maximum potential risk". This approach would be very difficult to defend in a court.

3. Comments were received concerning definition and use of the 95th percentile emission rate in the risk assessment (Section 2.2). Is the guidance on quantifying emission rates of compounds for use in the risk assessment adequate and scientifically sound? Should the guidance specify use of the 95th percentile or 95th upper confidence limit (UCL) of the mean?

   From a statistical perspective, the best unbiased estimate of actual emissions of a particular species is the average of the emission values for that species during RB/ TBs. Use of 95% UCL or similar approach results in a known biased estimate. Since the reviewer believes that a RA should provide the best estimate of actual risk, then the use of the unbiased information available is recommended.

   Should the agency insist that a more conservative approach is necessary, then the methods described in the guidance are appropriate. I see little difference in the 95th percentile and 95% UCL, but of the two I generally prefer the 95% UCL.

4. Comments were received regarding guidance presented for selection of compounds of potential concern (COPCs) (Section 2.3). Review and comment on the combustion engineering aspects of the COPC selection process presented in the guidance. These aspects would include guidance provided regarding inclusion of (1) compounds initially present in the hazardous waste feed stream and not completely destroyed in the combustion process, (2) compounds that are

formed during the combustion process, and (4) compounds in the waste feed stream that should be evaluated as potential products of incomplete combustion (PIC) precursors.

My response to this question considers two methods of selecting COPCs: (1) selection of COPCs using analytical results from TB/RB on existing facilities, and (2) selection of COPCs for a pre-TB/RB emissions inventory for a new or currently non-operating facility.

Selection of COPCs from Analytical Results from TB/RB on Existing Facilities

Since the majority of facilities that will go through the TB/RB/RA process in the next five to ten years already exist and the TB/RB/RA process is being implemented as part of the permitting (or re-permitting) process, the vast majority of facilities will follow this procedure. This process/procedure is well defined in Section 2.3 of the guidance document, paraphrased by me as follows:

A. Review the analytical results of a TB/RB. Retain as COPC any compound that is detected in any sample fraction in one or more runs.

B. Review the wastestream analysis. Add as a COPC any compound present in the waste even if that compound is ND in the stack sample analytical results.

C. Do not include any other ND compounds as a COPC if toxicity data are unavailable.

D. Do not add to the COPC list any other compounds which does not have a high potential to be emitted as a PIC.

E. Review the list of the 30 largest TICs and consider inclusion of those compounds for which toxicity data are available or are believed to have toxicity values similar to those compounds which were detected.

F. Consider adding other compounds based on site-specific factors. Add compounds as COPCs that: (a) are of concern for site-specific reasons, and (b) may be emitted by the combustion unit.

Overall, this is a very reasonable approach. My primary suggestion would be to add a discriminator phase to the second step (Step 2, above, and elsewhere in the RA process). Throughout the process, and especially at this step, no distinction is made between compounds that may be emitted and those that may be emitted at a level which could impact risk. In Step 2 there should be a provision for excluding compounds that may be present in the waste in low concentrations. There is a significant difference in RA impact between a compound present in the waste at 10 ppm versus a compound present in percentage quantities. After completing the minimum 10-4 reduction (99.99% DRE) in the combustion process a compound present in the waste at 2% may represent a risk, but a compound present at 10 ppm is not likely to. The relative thermal refractivity of the COPC of interest should also be considered, e.g., Class 1 compounds from the University of Dayton System might use 4-9s DRE. Less refractive compounds such as nitrobenzene might use 5-9s DRE.

In summary, with the addition of a discriminator to exclude deminimus COPCs, it appears that each of the steps is conservative, but none appear to be excessively so.

Selection of COPCs for New or Currently Non-Operating Facility

I have personally prepared two pre-risk burn emissions inventories under the previous draft of this guidance. One was a private facility and one was commercial. These comments are guided by that experience.

The previous guidance was internally inconsistent and included numerous loose ends, putting a heavy burden on the facility to develop a defensible inventory. The natural outcome from that draft was that the resulting inventory had a much larger number of COPCs included than there was any reasonable reason to believe could ever be present in the stack. When combined with the RDL as the assumed concentration, the risk was highly overestimated.

The current draft guidance seems to move in the other direction (e.g., essentially nothing is said concerning how the pre-RB/TB emission inventory should be prepared). The agency has made it clear that its policy (hazardous waste combustion strategy) is to discourage the permitting of new units. As time passes, a number of facilities have reviewed their needs and have decided that their HWC needs do not justify the continued expenses of operating the HWC unit. Thus, a natural attrition has and will likely continue for the foreseeable future. However, there are cases where facility

upgrades are needed or additional capacity at a given facility is necessary. As an example, one facility which has operated a 1970 vintage liquid incinerator concluded that its entire HWC unit had to be replaced with more modern technology in order to meet the HWC MACT. This facility committed to the state agency to replace the old unit and was required to prepare a pre-RB emissions inventory upon which a RA would be required. Estimating the stack concentration of those organic constituents (fed with the waste) in the stack is relatively straight- forward through a DRE-type calculation (i.e., Mass Feed Rate of Constituent *i* x 0.0001 = Mass Emission Rate of constituent *i* in the stack). However, identifying and estimating COPCs which could be emitted as PICS is much less clear.

The guidance should provide a short procedure that lays out an approach that the agency would accept. This approach should not say, for example, that if organic nitrogen is present in the waste, then any nitrogen-containing compound for Table A-1 from the guidance should be included as a COPC. While the concept of these types of PICS could be present is probably true some means of selecting more likely candidates is needed, especially in view of the agency's use of high RDL values to estimate low concentration COPCs.

The following comments pertain to the guidance provided on the seven categories of potential COPCs:

A. PCDD/PCDF: I agree that dioxin/furan sampling/analysis is important and should be included in the first TB/RB/RA for every HWC facility. Reasonably sensitive analytical methods are available, these compounds can be present even with a low chlorine concentration, a small emission rate can result in risk impacts and dioxin/furan are widely known compounds to the general public. This comment is based on the assumption that the agency will not insist on detection limits for non-detects that would suggest significant risk even from non-detects.

As a note: The studies referred to on Page 2-40 appear to be lab scale/pilot scale efforts. ASME and others have reported a general lack of agreement between lab/pilot scale test results and full-scale industrial operations. This discussion appears to be one-sided and actually unnecessary. Test every facility at least once. Retest for dioxin/furan at appropriate re-permitting, etc., stages, but only when the first test reveals a credible risk. This comment also applies to the other constituent categories such as PAHs, PCBs, etc.

B. PAHs: Testing for PAHs should follow a path similar to the previously discussed path for dioxin/furan. PAHs have been detected in many stack tests of HWC units and have been risk drivers in RAs. One reservation to this (and comments A, above, and C, below) pertains to COPCs which have driven risk assessments only because they were included at very high RDL concentrations.

C. PCBs: With one or two exceptions, I agree with guidance concerning stack testing for PCB. Several reasonable discriminators are provided. However, the last sentence on Page 2-47 does not appear to agree with the previous discussion, i.e., consider requiring stack testing for PCBs even if the conditions favorable for emitting PCBs do not exist at a given site. Also, "highly chlorinated waste stream" needs some further definition, i.e., >60% Cl. Further, the presence or possible presence of PCBs in the waste should also have a threshold level. The presence or possible presence of PCBs at 100 ppm may not warrant stack testing for PCBs for RA purposes when DRE reductions are considered.. It should also be noted that the references to PCB stack gas emissions being equal to or greater than those for Dioxins and Furans, is based solely on mass and not on the toxicity of the PCBs. Further, a review of selected referenced documents indicates that some of the data may be in error. If PCB testing is conducted, it should examine the coplanar PCBs, which are reported to exhibit Dioxin-like toxicity. Review of available (limited) literature which actually examines the 2,3,7,8-TCDD TEQ contribution due to PCBs suggests that PCBs are insignificant PICs of non-PCB waste streams.

D. Nitroaromatics: The procedure for including these compounds appears to be reasonable, especially if a waste concentration threshold is provided. Again, 10 ppm nitrobenzene in the waste would be very unlikely to result in increased risk, especially after considering that these compounds must survive a >99.99% DRE combustion process.

E. Phthalate: I disagree that the absence of these compounds in the stack "should always be verified by stack testing". Some knowledgeable practitioners in the combustion engineering field believe that detected phthalates in a stack sample are usually the result of laboratory/sampling/sample transport extraction from plastics used or from cross contamination from the background. EPA has access to a significant database that could be mined to determine if phthalates appear in analytical results even where they are not present in the waste. These possible false positives can be risk drivers in the RA process.

The same concept of a threshold concentration in the waste should also apply here. Even if a facility burns plastics, phthalates are generally used in low concentrations and the phthalates present must survive a >>99.99% DRE process (for phthalates, the '>>' symbol is added due to their combustible nature) to reach the stack.

F. HCB and PCP: The same comments regarding nitroaromatics, above, applies here.

G. VOCs: This section is weak overall, but may be the best possible at this time. The substantial database available to EPA should be mined to provide a more focused view of VOCs as a risk driver in RAs. Results of current RAs have generally shown that VOCs are not risk drivers.

5. Comments were received regarding guidance presented for quantifying non-detect compounds when estimating stack emission rates (Section 2.4). Review and provide recommendations on the following detection limit related issues: (1) can the instrument detection limit (IDL) be substituted for the method detection limit (MDL) in determining the RDL for metals, (2) can sample condensates be combined to lower detection limits without effecting (sic) the quality assurance and control of the data generated from analysis, and (3) how should J-flagged or qualified data be used if it is below the calculated RDL; what about if it is also below the assigned MDL?

At this point, the RA methodology (with associated RB data, partitioning values, toxicity values, etc.) is capable of producing what can be described as an "order of magnitude analysis", i.e., is the risk 10-4, 10-5, 10-6, or 10-7. Where data is not available conservative estimates must be used. Where information is available, unbiased estimates should be used.

In an ideal situation sampling/analytical methods would be available to obtain very defensible concentrations even at the very low concentrations that many COPCs exist in the stack, i.e., RDL values would be sufficiently low that all or most of the COPC stack concentrations would be above it. This ideal situation does not exist today. Thus, the agency must adopt an approach which is less than ideal.

In the guidance (Section 2.4.1) descriptions of several different DLs are provided. For the purposes of a risk assessment as opposed to compliance enforcement and research purposes, I have provided somewhat different descriptions as follows:

IDL: Defines the level at which you can begin to answer the question "is the compound present - yes/no. As a concentration value increases above the IDL one can begin to quantify the concentration. By the time the concentration reaches the MDL you can say with 99% confidence that the compound is or is not present. For compliance enforcement purposes this (MDL) is the lowest level at which you can reasonably "prove" that the compound is present. However, for quantifying estimated emissions for the purpose of an "order of magnitude analysis" type RA, analytical values between the IDL and MDL contain useful information on the "true state of nature" regarding compound concentrations. As a default, if no signal for a given compound is present at the IDL, then that compounds concentration should be set at the IDL.

MDL: The lowest level at which you are reasonably certain that a given compound is or is not present and, if present, begins to provide legally defensible quantified values. As concentrations increase from the MDL to the RDL totally defensible quantitation (as would be needed in court for compliance enforcement) or for scientific purposes becomes progressively available.

RDL: Appropriate for enforcement and research purposes and not necessary or appropriate for obtaining information on low concentrations COPCs for RA purposes.

At least two primary options are available: (1) use MDLs or even RDLs as a substitute for estimating the actual compound concentrations. This obviously provides numbers that are more defensible to analytical purists but has the disadvantage of overstating (sometimes by orders of magnitude) the actual concentration, or (2) use J-values as best estimates available even though they could be attacked as not being hard or totally quantified numbers.

In response to the three issues presented in the statement of the question:

(1) As discussed herein, I do not believe that RDL values should be used for obtaining low level estimates of COPC concentrations for RA purposes. This approach obviously distorts the actual risks present and may result in an unworkable RA process - high reported risks from mainly ND values.

(2) Yes, analysis of combined sample train condensates is defensible and provides more accurate risk estimates. It appears that in an effort to have total confidence that RB emission values are conservative and bulletproof from criticism; the guidance jeopardizes the total RA effort by using high DL values. The RA process may show high risks even with stack concentrations which are mainly/totally non- detect. Compositing the factions from the sample train recovery would allow a substantially lower total sample DL regardless of which DL values the agency uses without materially jeopardizing the QA/QC of the data.

(3) From a statistical viewpoint, J-values contain information about what the concentration of a given species actually is in the stack sample. Use of a higher, or in this case of an RDL, a much higher DL value contains much less information. Since there is no reason to believe that J-value results will be consistently higher or lower than the "true" value, any error in one J-value would be offset by another. J-values are our best current estimates of the actual stack concentrations.

6. Comments were received regarding guidance on determination of stack-specific particle size distributions recommended for use in air dispersion modeling (Section 3.4). Is inclusion of stack specific particle size distributions warranted, or could general or default distribution be applied without inducing additional uncertainty in the risk assessment? Considering the possibility of particle agglomerations, would the collection of particle size distribution data, divided into a minimum of three size categories (i.e., <2 microns, 2-10 microns, >10 microns), via SEM utilizing a Colter Counter be technically valid for stacks with a wet scrubber?

To date the agencies have required that particle size distribution (PSD) data be obtained on every hazardous waste combustion facility regardless of their PM emission rate or stack condition which frequently make obtaining accurate PSD data difficult or nearly impossible.

In one case a facility had data which showed approximately 15 ppm ash in the waste (Solutia, Chocolate Bayou, Texas). In this instance, the outcome of the RA would not be affected by the PSD because there is essentially no PM to deposit after being emitted from the stack. A threshold PM value, say of 0.015 gr/DSCF should be used to select which facilities should be required to obtain PSD data.
It is also difficult/nearly impossible to obtain accurate and reliable PSD data from wet stacks regardless of methods used. It is not clear that PSD data from wet stacks, even with relatively high PM levels, is more accurate as default PSD values.

The agency has access to a substantial quantity of TB/RB/RA data now. What impact is PSD having on RA outcomes? Unless PSD has been a significant impact on a reasonable fraction of facilities, the agency should strongly consider reducing PSD data collection requirements. Instead, the guidance should allow the use of existing or improved default PDSs as an option to collecting site-specific PSD data (improve PSD defaults by considering the PSD data from the already completed TB/RBs).

III. ATMOSPHERIC MODELING

Dr. Walter Dabberdt

Specific Technical Issues

Overarching Comment:

Many if not all of the issues identified below can appropriately be addressed in the context of uncertainty and sensitivity as they pertain to the risk assessment process. Uncertainty of a given variable, parameter or process should be assessed relative to its impact on the overall uncertainty in the associated risk that results from all sources, and it should be further assessed relative to the uncertainty in each of the other components of the risk assessment process. This means that one needs to consider uncertainty of a variable in the context of the sensitivity of the estimated risk to that variable and its uncertainty. Accordingly, I recommend that the Agency incorporate both sensitivity studies and uncertainty analyses into its risk assessment protocol. The need for uncertainty analyses is discussed in Section 8 of the HHRAP, but neither in sufficient detail nor with sufficient emphasis to convey that this is an integral and necessary element of the HHRAP process. Further, the discussion does not address the issue of sensitivity.

1. Does conducting separate modeling runs for each emissions phase (vapor, particle, and particle-bound vapor) provide better resolution of air parameter inputs for use in the risk assessment?

   The issue is not whether separate model runs are required. Rather, the issue would appear to be whether it is important to distinguish among the atmospheric concentration of each of the three phases. In terms of indirect exposure through deposition and uptake, as well as direct exposure through inhalation, it is important to distinguish between the vapor and particle phases for COPCs. I defer to those expert in toxicology and environmental biology to comment on the degree of importance. And with reference to direct exposure, it is important to apportion airborne particle concentrations according to particle size and number. There is an in-house version of ISC called HWIR that provides outputs of airborne concentrations and surface deposition by particle size; the conventional version of the code available on the OAQPS bulletin board only provides as output a single value of particle concentration. However, the regulatory version of ISC can be used with some difficulty to apportion airborne concentrations and surface depositions according to phase, particle size, and source, although this requires each phase and particle size(s) to be treated as a separate source group in the ISC model. OAQPS has indicated a willingness to consider making more widely available the HWIR version of the code.

Comment on the scientific validity regarding use of the fraction in vapor (Fv) for partitioning emissions between particle and vapor phases.

If the fraction in vapor, Fv, can be reasonably well described or defined, then it is appropriate to use this parameter to partition emissions between particle and vapor phases. The challenge is to define a representative fraction through measurements in the hot plume environment where this fraction is likely to undergo significant change.

2. Is inclusion of stack-specific particle size distributions warranted, or could general or default distributions be applied without including additional uncertainty in the risk assessment? Is the guidance on determining particle size distributions for inclusion into the air model adequate? Should additional guidance be provided regarding how particle size distribution data can be collected?

Representative particle size distributions are important because of their importance to determining direct exposure through inhalation and also determining the rate of deposition (and hence also the in-plume concentrations, through depletion and settling effects). The default size distribution intended for use at facilities with ESPs or bag houses appears appropriate in a generic sense for the finer sizes. However, the larger (15 micron) particles may be too heavily represented since these are typically effectively captured by particle control systems. On a more general note, I urge EPA to proceed with caution in offering this distribution as a default file. It is important to provide clear guidance to the user as to where and when the default size distribution is allowable, and when and where a source-specific distribution is required.

I do not recommend specific guidance on collection of particle size data (presumable through measurement). It is more important to provide guidance on when site-specific measurement data are required. When they are, the risk assessors will need to call on recognized experts.

3. Is the guidance provided for estimating (dry) vapor phase deposition technically valid as applied?

The HHRAP should be updated to reflect the dry deposition methodology reflected in the 1999 version of the ISC documentation. There is also an article recently published (Wesely and Hicks, 2000: Atmospheric Environment, Vol. 34, Nos. 12-14) that reviews the state of the art in dry deposition modeling.

Comment on the scientific validity regarding the assumption that the wet deposition and precipitation rates are linear.

In theory, the below-cloud precipitation scavenging should be a function of hydrometeor size and number distribution, aerosol size, and fall velocity. The hydrometeor data are typically not available for risk assessment applications; they are obviously difficult to measure and to obtain representative space-time distributions. In-cloud scavenging processes are even more difficult to measure and model. For similar reasons, it is difficult to estimate robust relationships to particle size, although attempts have been made. For these reasons, it is this reviewer's opinion that it is appropriate for risk assessment applications to use empirical relationships that relate wet deposition to precipitation rate in a linear fashion.

4. Comment on the guidance presented for air modeling of fugitive emissions and emissions used to evaluate acute exposure.

The protocol does not focus on truly acute events, except to incorporate their emissions into "long-term average emission rates adjusted for upsets … or reasonable maximum emission rates measured during trial burn conditions." The ISCST3 dispersion model has the ability to deal with certain types of events which yield acute exposure, particularly those where the emissions can be appropriately characterized by hourly averages. However, for true acute risk assessment, a non-steady state dispersion model (e.g. CALPUFF) may provide a more representative estimate of concentration and deposition given this model's ability to treat time-varying emissions and time-and space-varying meteorological fields.

Will conducting air dispersion modeling of fugitive emissions add unwarranted complexity to the risk assessment …?

I do not view assessing risks from fugitive emissions as necessarily complex or unwarranted. Given the nature of the risk assessment process and the goal of providing the public with objective information, this analysis is reasonable and appropriate. The need to model fugitive emissions can best be addressed through consideration of the sensitivity of the risk to these emissions and their associated uncertainties.

5. Comment on default values provided in the guidance air modeling inputs.

I assume the question pertains to the appropriateness of the default values contained in Tables 3-1 through 3-4.

Table 3-1 provides generalized particle-size distribution data; please refer to comments provided in response to Question 2.

Table 3-2 provides representative values of the albedo for different surface types during each of the four seasons; as default values, they are appropriate.

Table 3-3 provides representative seasonal values of the daytime Bowen ratio. This is a difficult parameter to estimate without site-specific precipitation and wind data. I recommend using site-specific data wherever possible. [Note also that Bowen ratio is incorrectly defined on page 3-35; it is the ratio of the sensible heat flux to the evaporative or latent heat flux at the ground surface].

Table 3-4 estimates default values of the anthropogenic heat flux for different types of urban areas. These are appropriate for use as default values. There are more recent summary papers that might be reference here as well; for example, the work of Klysik et al. (Atmospheric Environment, 33(24-25), 3885-3895).

6. Should atmospheric degradation be incorporated for the purpose of assessing contaminant mass losses? What changes in the guidance would be required?

It is my understanding that "degradation" refers to depletion as a result of dry and wet deposition and chemical transformations. The regulatory version of ISC as described in the HHRAP documents has the ability to treat depletion due to deposition of particles, and a new algorithm has apparently been incorporated into ISC for treatment of dry deposition of gases. The latter presumable also has the ability to consider plume mass depletion from deposition. Not treating depletion will certainly result in more conservative, but less realistic, model estimates of atmospheric concentrations as well as surface depositions. I do not advocate ignoring depletion.

General Comments

1. General Inconsistent depth of treatment throughout the protocol document; some sections are at an appropriate level, while others are far too detailed implementation guidance.

2. General The document could be improved with careful editing; for example, there are many grammatical errors.

3. P 1-1 Meaning of "one oral dose?"

4. P 1-1 Sentence 5 of introduction makes an important distinction between relative focus of the protocol on direct inhalation risk vs. risk through indirect pathways. This sentence could be clarified, but it should also be expanded because this is an important consideration. For example, why is the primary focus on indirect pathways?

5. Section 3 This discussion is based on ISC model construct and documentation circa 1995. However, ISC documentation was updated in 1999 to reflect incorporation of the dry deposition algorithms for gases and also an optimized area-source algorithm. I offer two, more general comments as well. One, the physical assumptions embodied in ISC should be described. I believe it is important for the reader of the protocol document to have a high-level understanding of the algorithms (e.g. plume rise, diffusion) embodied in the dispersion model. Two, the document would benefit from a conceptual description of the various atmospheric processes that are embodied in the dispersion model. This would apply irrespective of which model was being used. Then, details and the implications of the various approved models would be described at an intermediate level of detail (discussion of low-level details would be referred to the users manuals). I offer this comment recognizing that ISCST3 is the model currently prescribed, but EPA may approve other models for regulatory use. In fact, the CALMET/CALPUFF dispersion modeling system is currently being formally proposed by EPA for regulatory applications.

6. S. 3.5.1.5 Is pressure adjusted to surface elevation? If so, it would appear to be important for high-altitude locations.

7. S. 3.5.1.6 ISC addendum 99155 incorporates a dry gas deposition algorithm.

8. S. 3.5.2 "Upper air data" and "mixing height data" are NOT equivalent terms.

9.	S. 3.6.1.1	This discussion is confusing. "L" in urban areas for nighttime stable conditions should have a (positive) maximum value that is smaller for more built-up cities than for less built-up cities (i.e. the former have less stable nighttime conditions).
10.	S. 3.6.1.3	The discussion of aerodynamic surface roughness and the sensitivity of atmospheric concentrations to this term is confusing and misleading.
11.	S. 3.6.1.6	Bowen ratio is not a "measure of surface moisture;" rather it is defined as the ratio of the sensible heat flux to the latent heat flux. The latter is sensitive to soil moisture.
12.	S. 3.6.1.8	This discussion is misleading. M-O length is sensitive to net radiation. The "fraction of net radiation absorbed" is a misnomer.
13.	S. 3.7	General comment: the level of detail in this subsection seems inconsistent with other sections. Should these details be provided by reference to the user manual?
14.	S. 3.7	First paragraph, 2nd sentence: a noun is missing (ambient atmospheric concentration and deposition?)
15.	S. 3.7.1	The introductory sentence is confusing.
16.	S. 3.7.3	I found this subsection to be very helpful.
17.	S. 3.7.5	The OSW recommendation that use of terrain grid files is not necessary is arbitrary; if ignored, I would think that the user would need to demonstrate that the particular application is not very sensitive to terrain. This would not require a full 5-year model run to demonstrate.
18.	S. 3.8	The discussion of run times for different computers is out of date. Also, Comment #17 applies to this section as well.
19.	S. 3.9.1	The reference to "theory" in the 3rd sentence might be better stated as "rationale" or some such term.
20.	S. 3.9.1.2	First line: Eq. 3-1 should read 3-2.

21.	S. 3.9.3.1	The reference to "five percent" is unclear.
22.	S. 3.10	The height of the fugitive source is defined as "one-half …"
23.	S. 5.1	Does this mean that all particles are inhaled, irrespective of size? If yes, then why? We know that the smaller particles are more effectively inhaled than the larger.
24.	S. 8	It is important to understand and quantify model "sensitivity" as well as uncertainty; see specific technical comments.
25.	S. 8.2	Discussion on dry vapor-phase deposition is dated; I believe the current regulatory version of ISC includes this process.
26.	S. 8.5	The sentence on "typical" accuracies of dispersion model estimates is arbitrary without further clarification of the conditions under which such accuracies are achieved. Many situations achieve much poorer accuracies.

IV. HUMAN HEALTH TOXICOLOGY

Thomas A. Gasiewicz, Ph.D.
Chapter 1. Introduction.

1. Overall, this chapter does a good job in outlining the scope of the problem, the factors considered in the need to perform a risk assessment, the purpose and goals of the document, and the risk assessment process that will be considered. Some relatively minor changes should be considered.

   p. 1-3, 2nd point of "several factors": Here would recommend changing wording to "whether the facility is known to exceed any final technical standards". Sometimes it may be difficult to determine "whether the facility is exceeding any final technical standards" unless the risk analysis is performed. The suggested wording makes the wording more precise to indicate the intended purpose of this statement.

   p. 1-4, 1st full paragraph under "1.1 OBJECTIVE AND PURPOSE": Would recommend adding a sentence to the end of this paragraph to indicate that "The organization of this document follows closely the process outlined in Figure 1-1."

   Figure 1-1: Sometimes the Exposure Scenario Selection may be based on the Estimation of Media Concentrations, especially considering site-specific characteristics. Would it be more appropriate to have the "Exposure Scenario Selection" box interweaved below that for "Estimation of Media Concentrations"? Or at least have data from "Estimation of Media Concentrations" feeding into "Exposure Scenario Selection". There might be some concern that the Scenario Selection would exclusively drive the Media selections. This should be a two-way process.

   p. 1-7, section 1.2 Related Trial Burn Issues: This is a very specific issue that seems to be out of place in the Introductory chapter. This discussion is better left for Facility Characterization.

II. Section 2.2.1.3. Estimates of the Total Organic Emission (TOE) Rate

This section describes a clear and very reasonable approach for dealing with unidentified organic compounds. Since these are unknowns, it is unreasonable to them to be treated equally as COPCs in the risk calculation. Nevertheless, these may represent a significant fraction of the emission. As such, and depending on the relative

fraction of these that are present, options are provided to the permitting authority to incorporate this information into the risk assessment process. These options appear to be clear and reasonable. In particular, the assumption that the unknown compounds are similar in toxicity and chemical properties to the known compounds taken as a whole is reasonable. For a particular facility with particular characteristics and given no other information, it seems a reasonable assumption that the characteristics of the unknowns might be similar to that of the known compounds. This should be assumed unless additional information is available. It would be unreasonable to assume the worst case, just as it would be unreasonable to assume no toxicity. However, clearly the decision to or not to incorporate these data should be a quantitative one based on the relative contribution of the unidentified compounds to the total organics (see below).

p. 2-11, option 2 at bottom: It might be useful to provide some guidance as to what should be considered a "significant portion of the emission profile". Is this number 2% or is it 10 or 25%? As this might be site specific, it might be useful for the authority to perform the calculations and determine precisely how much of an increased risk this represents to the particular facility. Clearly a direction should be indicated and not left up to some undefined authority with no specific guidance.

On p. 2-12 it would be useful to incorporate an addition section "Recommended Information for Risk Assessment Report" to provide guidance as to how this information is to be specifically handled. This specific guidance is necessary since for a specific site the fraction of TOE may be considerable. At present, there is no specific guidance. One should be clearly indicated.

Section 2.3.1.2. PCDD/PCDF Noncancer Hazards

Here the inherent assumption is that background exposure levels do not cause significant noncancer effects in humans. Although, there are suggestive data to indicate that this may not be correct, there is certainly no conclusive evidence to indicate that in fact background exposure levels have any noncancer effects in humans. Nevertheless, terminology which indicates this one way or the other should be avoided. Until such a time when a reference dose for noncancer effects of these chemicals becomes established, it is reasonable to use 1 pg TEQ/kg/day (adults) as a reasonable benchmark (Note that it should be specifically indicated as TEQ). However, guidance should be provided to clarify what would be considered "low compared to background exposure". Is an additional 10% of this background considered low? Without

additional qualifications and/or reference to sections which discuss this in more detail, this is very confusing issue. Nevertheless, this is a significant issue that should be clarified especially since 1) an assumption, that is not conservative, is made that background levels have no toxicity, 2) the "background" levels in the area of concern are not likely to be known and there is no specific directive to determine these. It should also be clearly indicated whether using 1 pg/kg/day as a benchmark inherently assumes that this levels of exposure assumes no level of risk or an acceptable level of risk. In either case, the basis of this assumption needs to be clearly stated. (Actually this appears to go against the previous risk assessment for the dioxins. If this is the case, then it is not defensible, based on the definition of a benchmark dose, to use this level as a reference dose.) Regardless, the use of 1 pg TEQ/kg/day as the reference dose should be consistent with definitions established and scientifically defensible (and I am not sure it is).

p. 2-43, line 4: Would recommend: ".....not expected to result in a significant increase in noncancer effects above that already observed in U.S. population." The reader should realize that there is a significant background in noncancer effects in the U.S. population that may be due to differing genetics, exposure to chemicals etc..

Actually, it seems inappropriate not to have an RfD for these chemicals. On the other hand, if and RfD is not available (from any source) and there is reasonable evidence to indicate that the analyses for risk using the cancer slope factor is conservative enough to protect against noncancer endpoints, then this should be clearly stated.

Section 2.3.3. Polychlorinated Biphenyls

p. 2-47, lines 1-3 from bottom: Recommend eliminating "that" in "..to confirm that the absence...." to make the sentence clearer. Also, it should be more definitive whether or not the permitting authority should confirm the absence of the PCBs. The way the sentence is written, the permitting authority has the option to confirm. Either indicate which should be done, or provide some guidance into the process by which the decision is made. It is this reviewer's opinion that given the potential toxicity of the PCBs the recommendation should be that the absence should be confirmed.

Based on the information available there is sufficient scientific information available to indicate that most if not all effects of the coplanar PCBs are mediated through the aryl hydrocarbon receptor. Thus, the guidance for evaluating coplanar PCB congeners in the risk assessment using dioxin TEFs is appropriate and reasonable.

The last paragraph on p. 2-49 and first two paragraph on p. 2-50: These paragraphs are a bit confusing. In particular, it is not clear if these paragraphs refer specifically to the non-coplanar PCBs. Part of this could be clarified if there were a separate section for the non-coplanar PCBs. In addition, in most cases "non-coplanar" PCBs should be specified. For example, in the "Criteria for Use" Table on p. 2-49, the slope factor 2 is indicated for early-life exposure by all routes to all PCB mixtures. Does this really mean all PCB mixtures or just the non-coplanar PCBs? It may be very confusing to the reader, especially since the risk assessment approach for the coplanar PCBs has been specified.

Likewise, on p. 2-50 in lines 4, 5 and 8, is it correct as is, or is non-coplanar PCBs meant? If planar and/or non-coplanar are not specified, again there is likely to be some confusion as to the process especially if it is required that analysis for coplanar PCB be performed (as suggested on p. 2-49). If the coplanar PCBs are separately assessed then it would seem that an additional assessment of the PCB mixtures would in fact include the coplanar congeners based on what is written in these paragraphs. Again, the wording needs to be more specific about how the non-coplanar PCBs should be assessed specifically.

Section 2.3.3.2. Here the guidance is somewhat clearer since only mixtures are discussed. However, since section 2.3.1.2.specifically designates guidance for PCDD/PCDF noncancer hazards, it would seem appropriate that the coplanar PCBs be assessed for noncancer hazards as described in 2.3.1.2. If this is done, then 2.3.3.2 should be changed to indicate how the noncancer hazards should be approached for non-coplanar PCBs.

p. 2-51: PCBs are a significant issue. There should be a "Recommended Information for Risk Assessment Report" section at the end of section 2.3.3.

Chapter 2. Facility Characterization. (other comments for Chapter 2)

p. 2-1: Although the subsequent pages go into specific detail as to the types of emissions that should be considered, it might be worthwhile to (in one or two sentences) to define what is considered as "emissions".

p. 2-4, lines 7-8 from bottom, "Trial burn tests...": Something should be added here to indicate why this should be the case. If it is because of the conditions required for these tests, a simple phrase should be added to indicate this. Also somewhere in this section it should be indicated that waste feeds used during trial burns should be similar to used during normal operations.

p. 2-5, 2nd full paragraph, "High POHC...": The paragraph should state some conclusion. It begins by stating one aspect of the trial burn, then goes on to indicate something opposite by the PCDD and PCDF example. So what is the conclusion? There might be some confusion here.

p. 2-8, Recommended Information: In addition, there should be a statement, characterization, and justification for the "worst case" conditions.

p. 2-14, lines 3-5 from bottom, "U.S. EPA ...indicates...": It sounds like the EPA is not concerned about acute risks. Some clarification or addition should be made here.

p. 2-33, line 8 from bottom: Is wildlife also of concern here? Although this is clearly a "Human Health" risk assessment protocol, it might be indicated that either another document deals with this issue or there is too little data to approach such a protocol.
pp. 2-36 and 2-37: Many of the Table numbers as indicated in Appendix 1 are incorrect here.

p. 2-37, Step 3, "From compounds that are detected....": This is confusing. What is meant by a "similar" compound? Structurally or toxicologically similar? Additional specifics and guidance should be given here. Although a compound may look structurally similar it may have a very different potency and mechanism of action as a toxic chemical.

p. 2-37, Step 5: What is the basis for examining the "30 largest"? This statement fails to consider differences in potency. The largest peak may not be the chemical of greatest concern. Likewise, the smallest peak may be the chemical of greatest concern. There seems to be no scientific basis for this approach other than quantity.

p. 2-38, line 5: Table A-1.9-5 is missing from the Appendix (or it is not where it should be).

p. 2-38, 3rd full paragraph: The recommendation that one COPC list be developed which applies to both indirect and direct exposure analysis is a good one.

p. 2-46, line 13, "...emissions data indicate that ...: It has not been clarified here what a "significant" amount of noncarcinogenic PAH is. The concern, here and elsewhere, is that if the terminology is vague the chemicals will be largely ignored unless specific guidance is given.

p. 2-46: PAHs are a significant issue. At the end of 2.3.2 there should be a "Recommended Information for Risk Assessment Report" section.

p. 2-56: Based on the discussion of the VOCs from p. 2-55 it is not exactly clear what information, if any, is necessary for the risk assessment report. A clear statement should be made.

p. 2-9, Lead: Although this is a nice description of the recommended risk approaches for lead, no guidance has been provided as to the information on that is required for the risk assessment report. A specific guidance statement should be given.

pp. 2-69 to 2-70: A specific statement of guidance for Hydrogen chloride/Chlorine Gas should be made to conclude section 2.3.10.

Are there minimal site restrictions and requirements for the placement of such a hazardous waste combustion facility. If there are not, there probably should be. If there are not, a statement should indicate that EPA has not established minimal restrictions and requirements. If there are, these should be clearly presented and discussed.

Chapter 3. Air Dispersion and Deposition Modeling.

p. 3-22, Recommended Information. Since in some cases direct measurement of particle size distribution for a particular facility may not be available, some assumptions may be necessary to determine the likely particle size distribution. As part of the "Recommended Information...", the assumptions used should be indicated.

p. 3.40, end of section 3.6: Since additional manipulation of the meteorological data is necessary before use in the ISCST3 model, it would be useful to indicate in a

"Recommended Information for Risk Assessment Report" section a summary of the specific processes to be taken.

Chapter 4. Exposure Scenario Identification.

p. 4-3, 1st paragraph, last sentence: Here it would be useful to either be complete about identifying "instances" where worker exposure within the assessment area are considered within the risk assessment, or cross-reference to another section which does this.

p. 4-6, line 7 and p. 4-7, line 9: It is also important to consider populations outside the assessment area that may be using water contaminated in the assessment area, i.e. downstream. Would recommend avoiding terminology which limits the assessment to only those populations living in the assessment area and taking water from this same area. As indicated, local authorities would likely give some information to indicate whether any population living outside the assessment area consumes water within the assessment area. This seems also to be covered by the 2nd full paragraph on p. 4-7. Thus would recommend the following: On p. 4-6 - "...water is evaluated only if a population obtains...". On p. 4-7 - "...used for drinking water sources should generally be evaluated...".

Section 4.1.3. Special Subpopulation Characteristics: Might this also include populations significantly exposed, from other sources, to similar types of chemicals for which the risk might additive? <u>This is a significant issue that has been brought up at several incinerator facilities, and actually falls under the category of addressing "specific community concerns"</u>. The issue is very likely to be sights-specific. It should be discussed somewhere in this Protocol. Is it discussed elsewhere? If so, it should be cross-referenced in this particular section.

p. 4-10, top paragraph from previous page: Here it is specifically indicated that "..special subpopulations in such areas should be identified." Guidance should be given as to how these subpopulations are to be identified and who is responsible for this.

p. 4-10, last sentence of 1st full paragraph: It might also be mentioned that children with asthma might also be considered a particularly sensitive population.

p. 4-16, line 3 from bottom: Recommend "...ingestion rates of home-grown beef, poultry.....".

p. 4-22, sentence in lines 8-10: This sentence is extremely important. Would recommend putting in **bold**.

Chapter 5. Estimation of Media Concentrations

p. 5-9, lines 1-10: Here it is indicated that "..a first-order loss constant may be adequate to describe the loss of COPC from soil...". It is not clear here what "may be" means in terms of the error to the overall process. It should be indicated here how this assumption affects error on this parameter, and whether there could or could not be significant differences if kinetics other than first-order were considered.

p. 5-25: Here it is indicated that root uptake of COPCs is the primary mechanism through which aboveground protected produce becomes contaminated. A reference should be provided here to indicate the basis of this.

p. 5-29, line 2: A constant is missing here.

p. 5-18, Calculation of COPC Concentrations in Beef and Dairy Products: It should be indicated whether inhalation and water consumption are considered significant sources of exposure of contaminants to cattle. If no, then the basis of this should be stated. If yes, a cross-reference should be given to the specific section where this is discussed.

For Chapter 5 it would be useful to have a summary (perhaps in tabular form) at the end indicating whether data is available which indicate whether the predicted values using the models and assumptions presented in the chapter have been shown to agree or disagree with actual field measurements. Presenting this data, even though it is likely to be very limited, would present some confidence in the process.

Chapter 6. Quantifying Exposure

pp. 6-1 to 6-2: There should be more discussion of the proper dose-metric to be used. Dose to the receptor can be quantified in a variety of metrics. Some of these include daily intake, total body burden, or body burden averaged over a given period of time.

The metric used should clearly match the particular toxic endpoint. Developmental effects may utilize a metric which describes exposure at a particular time of development. On the other hand, responses such as cancer may be best described either by an exposure over time or a particular body burden. The appropriate metric used may also depend on the half-life of the particular chemical. There should be some guidance as to the particular dose metric to be used for quantifying exposure, and this guidance should consider characteristics of the particular chemical as well as the endpoint(s) of concern.

p. 6-4, 2nd full paragraph, "Intakes...": The first sentence presents a statement, and the second sentence presents a qualifier. An additional, sentence or two should be added here to draw some conclusion or guidance regarding the use of the toxicity factors for these calculations.

p. 6-6, assumptions for percentage of contaminated food: Here it is assumed that no other receptors, in addition to the subsistence farmer and subsistence farmer child, consume the contaminated animal tissues. Is there some basis for this assumption? If so, it should be referenced.

p. 6-6, lines 3-5 from bottom, "However, dermal...": Some specific guidance should indicate specifically what site-specific exposure setting characteristics would indicate a requirement to assess dermal exposure to soil and inhalation of resuspended dusts. If no guidelines are indicated, these potential sources of exposure might be largely ignored.

p. 6-7, Soil Ingestion: Based on what is presented here, it seems unreasonable to rule out pica behavior as part of the risk assessment, especially since 1) it is considered "a normal part of a child's development", and 2) children may be one of the most susceptible groups. The last paragraph goes on to indicate that this should be considered on a case-by-case and site-specific basis. This reviewer would recommend a statement indicating that this must be considered and the lack of this consideration be supported by documentation. Unless this recommendation is made this potential source of exposure may be largely ignored.

p. 6-8, Dermal Exposure to Soil: Here again, specific guidance should be provided to indicate what conditions or setting characteristics would require that this exposure pathway be evaluated. Without specific recommendations some potentially significant dermal exposures may not be considered.

p. 6-9, Soil Inhalation Resulting from Dust Resuspension: If there are any site-specific conditions that may require consideration of this pathway, they should be indicated here.

p. 6-13, Exposure Duration, last paragraph and data in Table on p. 6-14: Despite what is indicated here, there is a distinct possibility that many individuals growing up in a particular area may remain in that area all of their lives. This would seem to be a population that may be most susceptible by virtue of their life-long exposure. It would seem unreasonable not to consider this type of exposure duration, unless there is data from a particular area to indicate that this is not the case.

p. 6-14, Averaging Time: Despite the discussion, no specific recommendations are made, but they should be.

Chapter 6: Just as present in previous chapters, this chapter needs a summary of specific recommendations and processes to follow for quantifying exposure. Much of this may consider site-specific factors and the type of metric to be used for carcinogenic vs noncarcinogenic endpoints. Nevertheless, the specific guidelines should be made.

Chapter 7. Risk and Hazard Characterization

p. 7-2, last sentence in 1st full paragraph: It should be specified here what site-specific conditions would indicate calculation of population risks.

p. 7-6, lines 4-5: Background exposure levels and exposure from other man-made sources are issues that are not adequately addressed in this document. It is suggested here that consideration of other sources is largely left up to the discretion of the permitting authority. This reviewer would suggest that a requirement be made to at least consider other sources of exposure. This would require a statement that the HQ has or has not been adjusted based on the determined (or at least estimated) absence or presence of other significant sources of exposure. The importance of other sources should also be considered for cancer risk, but may be especially important for effects where thresholds are involved, especially if the exposure is near such a threshold.

p. 7-6, lines 4-7 from bottom, “This summation methodology assumes...”: It probably should be noted that this is a very conservative assumption especially for different

endpoints of concern. There is certainly only very limited evidence for any of the health effects of these chemicals to be additive. In fact, it is probably unlikely for many health effects. Nevertheless, the possibility exists. Thus, the assumption is made.

Chapter 8. Uncertainty Interpretation for Human Health Risk Assessment

p. 8-7, lines 5-6 from bottom: It might be useful to give some relative quantification of what is meant by high, medium and low effect. Thus, will a high effect give an uncertainty of 2 orders of magnitude vs 5-fold.

Chapter 9. Completion of Risk Assessment and Follow-On Activities

p. 9-1, Conclusion: Perhaps it would also be useful here to indicate the major sources of uncertainty and an estimate of how many fold the error might be.

p. 9-2, last paragraph: This section should specify a mimimal amount of time between reviews and the types of changes in process and procedures that would require a review of the facility. Here too much is left to the discretion of the facility and/or the permitting authority.

Appendix A-3. Compound Specific Parameter Values.

p. A-3-2, last sentence: Was any consideration given to variability. Depending on the variability and goodness of the data, using the geometric mean may not often be the best approach.

p. A-3-26, last line for discussion of PCDDs and PCDFs biotransfer factors: The last sentence "Therefore, the beef and milk..." relative to the discussion above this sentence is confusing. A more specific statement is needed to indicate what is applicable to the PCBs, PCDDs and PCDFs.

p. A-3-33, section A3.5.4.2: The exact difference between BAF as defined here an BCF as defined in A3.5.4.1 is not clear. This should be explained in simple terminology.

Section A3.6.3. Explanation of Calculated Toxicity Benchmark Values:

The extrapolation between Oral CSF and Inhalation CSF may be inappropriate for some agents. A particular example would be asbestos. On the other hand if Oral CSFs are missing, there is no data to believe that a particular deleterious effect of the chemical is dependent on route of exposure, and there is no data to indicate different levels of absorption, the inhalation CSF should be used. However, a qualifier should probably be added to indicate this. For the extrapolation of RfD to inhalation RfC, it probably should be indicated that this procedure should be used unless there is some basis to indicate thatabsorption by inhalation might be substantially less than 100%. Here the conservative assumption is made that absorption is 100%. This probably should be stated.

The assumption that Inhalation RfD = Oral RfD may not be valid for some chemicals. It should be indicated that 1) this assumes equal levels of absorption, and 2) this assumption should be made unless there is data to indicate that the levels of absorption of the particular chemical from these routes of exposure might be different or that toxicity is dependent on the route of exposure. For the top equation on p. A-3-40, the Inhal. URF in the numerator should be Oral CSF. Here again a statement should be made to indicate the assumptions as in the above paragraph. As above the assumption that Inhalation CSF = Oral CSF may not be valid. As above, the appropriate statements should be made to provide guidance and qualify this assumption.

Section 3.6.4: Although the assumption about the toxicity between routes is appropriate here when uncertainty is discussed, an additional statement should be made about the absorption. Although the same type of toxic endpoint might occur, differences in potency could be observed due to difference in absorption from the different routes. Very often judgment can be made that are often compound specific, and this aspect should be added as well. The degree of uncertainty will also be compound specific. Thus, the uncertainty for the overall risk assessment for a particular facility might be influenced predominantly by the relative abundance of a particular chemical. The consideration of an additional uncertainty factor should be made on a site specific basis, and this should be influenced by the relative abundance of chemicals for which greater uncertainty exists.

Table A-3: For “Health Benchmarks” the type of toxic effect on which the benchmark values are based on should be briefly indicated for each compound. Where values are

missing, it is assumed that values for these health benchmarks are not available? If so, it would be useful to clearly indicate this in a key at the beginning of the table. It is noted that for the halo-substituted dibenzodioxin and dibenzofurans no health benchmarks are listed. These values should be calculated on the basis of TEFs in comparison to the 2,3,7,8-tetrachlorodibenzo-p-dioxin (just as has been performed for the PAHs).

Appendix A-4, Acute Inhalation Exposure Criteria.

A more complete description of the effects of concern should be given for each compound and for each value listed in Table A-4. It is actually difficult to compare values from different sources without some indication of the effect.

This reviewer would make the following recommendation for the use of data presented in Table A-4:

1. Each chemical should be evaluated separately.

2. If only one value is available, that value should be used unless there is reason to believe that the criteria and/or background information on which that value is based are inappropriate.

3. If more than one value is available and the endpoints of concern on which the values are based are different, the value for the most sensitive value of concern should be used unless there is reason be believe that the criteria and/or background information on which that value is based are inappropriate.

4. If more than one value is available, the endpoints of concern are the same, and the values are greatly different, then the criteria upon which these values are based should be critically evaluated. A conservative approach would be to use the smaller value. However, for a variety of reasons, e.g. the use of unpublished or very limited data, and or scientifically unsound data, the use of the smaller value may be inappropriate. On the other hand, newer values may have incorporated additional information that may not have been available at the time the older data were obtained. If both values are listed, the differential criteria explaining the reasons for these differences should be clearly stated. A lower and or newer value should not automatically be accepted without a careful and critical evaluation. <u>The use of one value over another, regardless if it is a newer value, should be based on a sound scientific evaluation and the rationale should be clearly stated for each chemical.</u> This evaluation should include values provided in the California Office of Environmental Health Hazard Assessments (OEHHA) 1998 revised draft.

Dr. Mary Davis

General Issues

1. In general, the sections I reviewed were clear and concise. I found the organization of the document to be good. I could use the Table of Contents to find specific areas when I needed to refer back to them, and I am very grateful for that (with other documents I have been frustrated knowing I read something but unable to easily re-locate it). Parts were, I think, too brief. In particular, I had some trouble determining the purpose of the acute risk scenario and in understanding how the modeling of the fugitive emissions, acute risks and the routine emissions come together. I have reviewed the risk assessment for the WTI incinerator, so I am somewhat familiar with the process. Having this document prior to diving into that risk assessment would have been quite useful. I think it would be helpful for the uninitiated if there was a narrative description accompanying the flow diagram depicted in Figure 1-1 that would convey the type of information that flows between boxes and particularly the products of the Risk and Hazard Characterization step. The highlighted "Recommended information for risk assessment report" boxes are quite useful. My copy did not have the references, I was able to find the file on-line.

2. I think the HHRAP accurately reflects the presented methodologies and scope. It is a good overview and conveys what is involved in the process—what data are needed and how they are used. It communicates the complexity of the process. I think one could get an understanding of the process reading the HHRAP alone, however it does not equip one to do a risk assessment. I think it would equip one to understand a risk assessment, one of the objectives, and is a general guidance, pointing to where one can find all the details, if one wishes or needs to do a risk assessment.

3. See responses to Specific Technical Issues.

4. I'm not sure I understand this question; I think the HHRAP leads one to credible interpretations with reasonable discussion of uncertainty about the predictions.

5. Methodological gaps: The modeling of upset conditions discussed under item 2 of Specific Technical Issues.

6. Long term research recommendations: Assess how well the route-to-route extrapolations of slope factors and reference doses actually work and if there are additional parameters that could improve this. This is covered in more detail under item 3 of Specific Technical Issues.

Specific Technical Issues

1. Soundness of assigning toxicity values to the "unknown" or TOE portion of the emissions.

   The document suggests several strategies for using the TOE factor in the risk assessment, by addressing the uncertainty as a narrative, assigning toxicity values based on the toxicity of the identified emissions, requiring more testing or imposing more stringent permit conditions. Assigning toxicity values to the unknown component by assuming the toxicity is similar to the known has some logic in that the knowns are a subset of the universe of emitted chemicals, as are the unknowns. It requires the assumption that both are representative of the universe of emitted chemicals. This would be easier to accept if the chemicals that are measured were randomly selected from the universe of emitted chemicals. Both the measured and non-measured chemicals would be random samples of the same population and there is a good chance that the measured chemicals would be representative of the non-measured chemicals. One could argue that testing methods are biased towards chemicals that are perceived to have toxicity (the concern about them is what landed them in the list of chemicals to be measured) and so the measured chemicals are not representative of the universe of emitted chemicals. If that is the case, assigning the unknown fraction the same toxicity as the known fraction is defensible in that it is appropriately conservative and protective.

2. Issues pertaining to acute toxicity and recommended AIEC values:

   The purpose of acute exposure scenario was not clear to me, and the purpose is the important factor in determining which values are the most appropriate. In the risk assessment of the WTI plant, the external review panel recommended that risks of upset and accident conditions be considered, on the basis that the greatest exposure to the population would be during such episodes. Most of the various emergency exposure guidelines under consideration in Chapter 7 are clearly meant to deal with such episodes. Therefore, I was anticipating that acute exposure limit guidelines would be applied to the emissions that are estimated to result from process upsets. After re-reading of Chapter 2, I gather that this is not the case, rather upset conditions are handled by multiplying the stack emission rate by an upset factor that accounts for the proportion of time that the facility is operating in upset conditions. This has the effect of diminishing the impact of the upset event by lumping it into overall emissions. If the goal of the acute exposure scenario is to estimate the risks from upset conditions, I recommend that the sections that deal with this be revised after reconsidering how to best accomplish that goal. Regardless, I recommend that the sections dealing with the acute exposure scenario be revised to clearly specify the purpose of the scenario.

The AEGLs characterized as "*short-term threshold or ceiling exposure values intended for the protection of the general public, including susceptible or sensitive individuals, but not hypersusceptible or hypersensitive individuals.*" The AEGL-1 value is "*the airborne concentration (expressed as parts per millions (ppm) or milligrams (mg)/meters (m)^3) of a substance at or above which it is predicted that the general population, including ``susceptible'' but excluding ``hypersusceptible'' individuals, could experience notable discomfort. Airborne concentrations below AEGL-1 represent exposure levels that could produce mild odor, taste, or other sensory irritations.*" AEGL values are given for specific durations of exposure (0.5, 1, 4 and 8 hr). The HHRAP assumes a 1 hr exposure duration, so the 1 hr values would be appropriate.

The Relative Exposure Levels (REL) values established by California Office of Environmental Health Hazard Assessments 1998 draft. RELs are defined as "*The concentration level at or below which no adverse health effects are anticipated for a specified exposure duration is termed the reference exposure level (REL). RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact.*" RELs are based on 1 hr exposure.

Emergency Response Planning Guides (ERPGs) have been developed by the American Industrial Hygiene Association. Unlike the RELs and AEGLs, the ERPGs do not include safety factors and they are designed to predict response (and therefore plan appropriate emergency responses), not protect health. ERPG-1 is defined as "*The maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor.*" ERPGs are based on a 1 hr exposure. Temporary Emergency Exposure Limit (TEEL) values are estimated from existing data, such as toxicity and lethality data and occupational exposure limit values and are meant to serve as substitutes for ERPGs until ERPGs are developed. TEELs are developed by the Department of Energy.

The question of whether specific guideline values should be included in the document or not is particularly timely. Since the draft, the ERPG-1 and TEEL-1 values have been updated, and the CalEPA ATEL-1 values seem to have been replaced by REL values. As of February 4, 2000, there are ERPG-1 values on approximately 90 chemicals, and TEEL-1 values on another 1250 (or so) chemicals or specified mixtures. The AEGL-1 values are more limited than the

draft indicates, as AEGL-1 values were not determined for methylhydrazine, either of the dimethylhydrazines, ethylene oxide, arsine or phosphine. That is, AEGL-1 values were given for only 6 of the compounds (the remaining 6 do have AEGL-2 and AEGL-3 values). Additional AEGLs are expected to be developed. Similarly, REL values for some chemicals are for more severe effects, as it was not possible to estimate a mild response, or the most sensitive response is a severe effect. Generally speaking, the document clearly indicates that current values need to be sought. Many, if not all, of the values are available through the WWW. It would be useful if EPA created a page with links to pages with the current values and referred to that page in the document. EPA would need to maintain the page, in particular verifying that the links are still correct or updating them as needed. Links to support documents would also be good.

The question of the order in which the different guidelines should be consulted is a difficult question, particularly without knowing the objective of the acute risk scenario. As best I can determine, the acute exposure scenario is to consider the immediate effects of the emissions that occur on a routine basis. The ERPG-1 values do allow for "mild transient adverse effects" to occur. The ERPGs are geared towards accidental releases, not releases that occur from predictable operations, which for a hazardous waste incinerator are upset conditions such as start-up, shut-down and other upsets as described in the HHRAP. In contrast, the REL values consider the effects to the population from non-emergency routine releases. Thus, the REL values seem most suited to the purpose and would take precedence over ERPG-1 values.

To address this question and the question of comparability of the RELs to other AIECs, I compared the values from the various guidelines. Table 1 includes the compounds for which there are AEGL values but shows only the AEGL-1 values; ERPG-1/TEEL-1 and REL values are also shown. The characterization of the AEGL-1 and REL values would suggest that they would result in similar values. There are both AEGL-1 and REL values for just two compounds and for both the REL is about 7% of the AEGL-1 value. For chlorine this stems from a difference in evaluation of human data in the same study. For nitric oxide, the levels are based on different human studies. The AEGL-1 is based on a small uncertainty factor for sensitive populations because irritant effects are considered to not vary much among individuals. The REL is based on a study in asthmatic adolescents. For nitric oxide, the difference between the two guidelines highlights that the two are different in how they address asthmatics, a sensitive population. While the general purpose is similar, the outcome is very different, however the comparison is based on only two compounds.

Table 2 is a comparison of ERPG-1/TEEL-1 and REL values. The last column of the table the REL is shown as a percent of the ERPG-1/TEEL-1. With the exceptions of vinyl chloride and

acrylic acid, the REL values are more conservative than are the ERPG-1/TEEL-1 values, and often considerably so.

The California OEHHA comments that the REL values are designed for preparation of risk assessments for non-emergency routine releases and that the AEGL values are for emergency planning purposes. The approach taken in determining the REL does focus on the most sensitive effect. Both use similar techniques to adjust for time of exposure. The ERPG-1 value does allow an individual to experience transient, mild adverse effects. Thus, it is not intended to be as protective as the REL, and it is not. The TEEL-1 values are extrapolated from other toxicity values and incorporate simplifying assumptions. Nonetheless, excluding an outlier in each sub-group (vinyl chloride in the TEEL-1 group and chloroform in the ERPG-1 group), TEEL-1 and ERPG-1 each predicted the REL similarly (the ratio of REL to TEEL-1 or ERPG-1 was 15% for TEEL-1 vs 11% for ERPG-1; 12% for the two combined).

The acute toxicity risk assessment is done using a hazard quotient. For that, effects are summed for a particular response. To do that, the risk assessor must know to what response each guidance value applies. This is clearly specified for the AEGL-1 and the REL values, but not for the TEEL-1 values. To be able to use the TEEL-1 values, the effects of concern would need to be provided.

3. Is route-to-route extrapolation appropriate and conservative to determine benchmark values for use in an initial screen for which peer reviewed toxicity benchmarks are not available?

I considered three specific extrapolations, between (1) the RfD and RfC, (2) oral vs inhalation CSF and (3) inhalation CSF vs one calculated from the inhalation URF. For some of the chemicals listed in Appendix A there are values for several of these parameters and these can be used to compare extrapolated values to actual values. I did this using the equations in Appendix A3, pages A-3-39 to A-3-40 (correction: the equation for calculating inhalation URF from oral CSF needs to have oral CSF on the right side of the equation at the top of page A-3-40).

Table 3 shows the comparison of RfCs calculated from RfDs to actual RfCs, expressed as a number and, in the last column of the table, as percent of RfC. Of the 38 compounds, the range was 5.3% to 250000%. For some compounds, this approach is not conservative; the extrapolated value is about three orders of magnitude higher than the actual value. For some compounds, the extrapolation is overly conservative, and for others the extrapolation is reasonable. So the question becomes, when is route-to-route

extrapolation a reasonable thing to do. For a compound for which this extrapolation is not conservative, toxicity benchmarks that have not been peer-reviewed may be more conservative.

Table 4 shows calculations and comparisons involving CSFs. Frequently, the oral and inhalation CSFs are the same value, however for some compounds they are quite divergent, ranging from 8% to 58333%. The CSF calculated from inhalation URFs agreed well, with only two compounds being off by more than 5%. The inhalation URFs are potentially derived from the same data as the CSFs, so this is not surprising.

With these extrapolations, the divergences from the "real" value are substantial and in both directions, that is both over- and underestimating the benchmark. On average, for this subset of compounds, the extrapolated RfCs are higher than the real RfCs and would not conservative. Whether this would apply to a larger subset is unknown nor, more importantly, is it known if chemicals that have not been studied would have the proportion of over-estimators and under-estimators.

I believe this preliminary analysis is reasonable and that I have entered the numbers correctly. I believe this highlights some issues to be considered more fully, particularly if there are properties of chemicals that can be used to predict which ones would not be amenable to route-to-route extrapolation that would be useful for the risk assessor to consider in making an informed decision on the validity. These might be toxicokinetic parameters including differences in the permeability of the different barriers involved and effectiveness of pre-systemic clearance mechanisms, and target site for the effect of concern.

Appendix A3.6.1 gives the order of preference for sources of RfD and RfC values. HEAST values from 1995 are preferred to those from 1997. I would expect that the most current values would be preferred. Why is 1995 preferred?

4. Is it appropriate to use as the recommended benchmark for noncarcinogenic effects of dioxin the national average background exposure (1 pg/kg/d for adults)?

In the absence of a reference dose for PCDD and PCDF congeners, it is useful to compare exposure due to incinerator emissions to the background dioxin exposure. Interpretation will be difficult if the plant emissions approach background exposure. The non-cancer dioxin effects correlate with body burden and dioxin has a half life on the order of 7-10 years and so accumulates over a long time. The exposure from the

incinerator emissions would need to be well below the background exposure for this analysis to be re-assuring. In the WTI risk assessment, the exposure to the most highly exposed scenario (subsistence farmer in high exposure area) had an intake of approximately 7E-03 pg TEQ/kg/day (based on PCDD and PCDF congeners), suggesting it is unlikely that exposure from plant emissions would approach background exposures. Convincing the local population of that may be a challenge. Local populations, particularly in rural areas, may think their environment is more pristine than average and that their background exposure is correspondingly less so that the impact of the incinerator emissions is correspondingly more.

5. Scientific validity of evaluating coplanar PCB congeners using dioxin TEFs. Technical validity of using Aroclor 1254 and 1060 for other PCB congeners.

The HHRAP recommends the risk of exposure to certain PCB congeners be assessed as dioxin-like compounds by applying a dioxin TEF. The use of dioxin TEFs for those coplanar PCBs for which they have been established through a rigorous review and consensus process is scientifically valid; indeed one could argue persuasively it would not be scientifically valid not to use such an approach. The TEF approach incorporates more science into the risk assessment and thereby diminishes the uncertainty inherent in default assumptions. The risk assessment of the remaining PCBs is based on using the characteristics of specific PCB mixtures (the common commercial mixtures, for which there is much toxicity information) as an estimate of the toxicity of specific PCB congeners. The approach was developed for assessments of environmental exposure to residual PCB left from contamination by the common commercial mixtures, taking into account that various environmental processes alter the various component PCB congeners differently. Because of those alterations, PCB contamination in the environment no longer has the same composition as the original mixtures. The exposure from hazardous waste incinerators is similar in that the composition of PCB congeners in the stack emissions would not be expected to be similar to the original commercial mixtures. The use of Aroclor 1254 or 1016, depending on congener composition of the actual emissions, is a reasonable approximation. The table at the bottom of page 2-49 has been condensed from the original document but does not include the notation that the 2 slope factor should be used for dermal exposure only if an absorption factor has been applied and the 0.4 slope should be used for dermal exposure if no absorption factor has been applied. The HHRAP notes that dermal absorption will rarely be modeled so this is a minor issue.

Table 1: Comparison of ERPG-1 or TEEL-1, REL and AEGL-1 Values

Chemical	CAS Number	**ERPG-1**[a] or TEEL-1	REL	AEGL-1[b]
Arsine	007784-42-1	**0.16**	0.16[c]	ND
Aniline	000062-53-3	23		30
Ethylene oxide	000075-21-8	**5**		ND
Fluorine	007782-41-4	**1**		3.1
Chlorine	007782-50-5	**3**	0.21	2.9
Dichloroethylene, 1,2-[d]	000540-59-0	2377		53
Dichloroethene, trans-1,2[d]	000156-60-5	50		
Dichloroethene, cis-1,2[d]	000156-59-2	792		
Dimethylhydrazine, 1,1-	000057-14-7	0.1		ND
Dimethylhydrazine, 1,2-	000540-73-8	4		ND
Monomethylhydrazine; (Methyl hydrazine)	000060-34-4	0.4		ND
Nitric acid	007697-37-2	3	0.09	1.3
Phosphine	007803-51-2	**1**		ND

[a] values in bold are ERPG-1, others are TEEL-1

[b] ND means not determined, generally because data did not support a mild effect

[c] the arsine REL is for a severe effect, not a mild effect

[d] There are three different TEEL-1 values for dichloroethylene. It is not clear to me why the mixture has a higher TEEL-1 than either of its components. The AEGL-1 considers the greater toxicity of the trans isomer, and the TEEL-1 and AEGL-1 are in excellent agreement. The AEGL-1 is listed based on the CAS number appearing in the Federal Register.

Table 2: Comparison of ERPG-1 or TEEL-1 values to REL Values

Chemical	CAS Number	**ERPG-1**[a] or TEEL-1	REL[b]	REL/TEEL-1 or REL/ERPG-1
Acrolein	000107-02-8	**0.2**	0.0002	**0.09%**
Acrylic acid	000079-10-7	6	6	101.85%
Ammonia	007664-41-7	**17**	3	**18.38%**
Arsenic (inorganic cmpds as As2O3)	001327-53-3	0.24	*0.00002*[c]	0.00%
Arsine	007784-42-1	**0.16**	*0.16*	**3.35%**
Benzene	000071-43-2	**160**	1.3	**0.81%**
Benzyl chloride	000100-44-7	**5**	0.2	**4.64%**
Butoxyethanol, 2-; (Glycol ether EB)	000111-76-2	242	14	5.80%
Carbon disulfide	000075-15-0	**31**	*6*	**0.40%**
Carbon monoxide	000630-08-0	**229**	23	**10.05%**
Carbon tetrachloride	000056-23-5	**126**	*2*	**0.04%**
Chlorine	007782-50-5	**3**	0.2	**7.25%**
Chloroform	000067-66-3	**10**	*0.2*	**0.00%**
Chloropicrin; (Trichloronitromethane)	000076-06-2	**1**	0.03[d]	**4.32%**
Copper	007440-50-8	8	0.1	1.28%
Diethyleneoxide, 1,4-; (1,4-dioxane)	000123-91-1	90	3.0	3.33%
Epichlorohydrin	000106-89-8	**8**	1.3	**17.19%**
Ethoxyethanol, 2-	000110-80-5	55	*0.4*	0.02%
Ethoxyethylacetate, 2-	000111-15-9	81	*0.1*	0.01%
Ethylene Glycol Monomethyl Ether	000109-86-4	78	*0.1*	0.00%
Formaldehyde	000050-00-0	**1**	0.1	**7.66%**
Hydrogen chloride; (Hydrochloric acid)	007647-01-0	**4**	2.1	**46.97%**
Hydrogen cyanide; (Hydrocyanic acid)	000074-90-8	**5**	*0.3*	**1.23%**
Hydrogen fluoride; (Hydrofluoric acid)	007664-39-3	**2**	0.2	**14.67%**
Hydrogen selenide	007783-07-5	0	0.005	3.02%
Hydrogen sulfide	007783-06-4	**21**	0.04	**0.20%**
Isopropyl alcohol	000067-63-0	983	3	0.33%
Mercury (elemental & inorganic as Hg)	007439-97-6	1	*0.002*	0.00%
Methyl alcohol; (methanol)	000067-56-1	**262**	28	**10.69%**
Methyl bromide	000074-83-9	**12**	4	**33.50%**
Methylene chloride	000075-09-2	**694**	14	**2.02%**
Nickel	007440-02-0	7	0.006[e]	0.08%
Nitric acid	007697-37-2	3	0.09	3.34%
Nitrogen dioxide	010102-44-0	4	0.47	12.50%
Ozone	010028-15-6	0	0.18	91.75%
Perchloroethylene; (Tetrachloroethylene)	000127-18-4	**678**	20	**2.95%**
Phenol	000108-95-2	**38**	6	**15.08%**
Phosgene	000075-44-5	**0**	0.004	**0.99%**
Propylene oxide; (Methyl ethylene oxide)	000075-56-9	**119**	3	**2.61%**
Sodium hydroxide	001310-73-2	**1**	0.01	**0.98%**
Styrene	000100-42-5	**213**	21	**9.87%**
Sulfur dioxide	007446-09-5	**1**	0.7	**84.03%**
Sulfuric acid, Sulfur trioxide (7446-11-9)	007664-93-9	**8**	0.1	**1.50%**
Toluene	000108-88-3	**188**	37	**19.65%**
Trichloroethane, 1,1,1-; (Methyl chloroform)	000071-55-6	**1908**	68	**3.56%**
Triethylamine	000121-44-8	12	3	22.56%
Vanadium pentoxide; (Vanadium(V) oxide)	001314-62-1	1	0.03	5.38%
Vinyl chloride	000075-01-4	13	180	1409.30%
Xylene	001330-20-7	651	22	3.38%

[a] values in bold are ERPG-1, others are TEEL-1
[b] a REL given in italics indicates the REL is based on a severe (non-transitory) effect
[c] arsenic and inorganic arsenic compounds
[d] copper and copper compounds
[e] nickel and nickel compounds

Table 3: Extrapolation of RfDs to RfCs

Chemical name		Values		Calculated value	
	CAS number	RfD	RfC	RfC[a]	% EPA RfC[b]
	average				12663.9%
	minimum				5.3%
	maximum				250000.0%
	n~100%				11
acetaldehyde	75-07-0	2.60E-03	9.00E-03	9.10E-03	101.1%
acrylonitrile	107-13-1	1.00E-03	2.00E-03	3.50E-03	175.0%
aniline	62-53-3	2.90E-04	1.00E-03	1.02E-03	101.5%
beryllium	7440-41-7	2.00E-03	2.00E-02	7.00E-03	35.0%
carbon disulfide	75-15-0	1.00E-01	7.00E-01	3.50E-01	50.0%
chlordane	**57-74-9**	**5.00E-01**	**7.00E-04**	**1.75E+00**	**250000.0%**
chlorine	**7782-50-5**	**1.00E-01**	**2.00E-04**	**3.50E-01**	**175000.0%**
chlorobenzene	108-90-7	2.00E-02	6.00E-02	7.00E-02	116.7%
chloroethane	**75-00-3**	**4.00E-01**	**1.00E+01**	**1.40E+00**	**14.0%**
chloroform	67-66-3	1.00E-02	3.50E-02	3.50E-02	100.0%
chromium hexavalent	**18540-29-9**	**3.00E-03**	**1.40E-04**	**1.05E-02**	**7500.0%**
cumene	98-82-8	1.00E-01	4.00E-01	3.50E-01	87.5%
1,2-dibromo-3-chloropropane	96-12-8	5.70E-05	2.00E-04	2.00E-04	99.8%
1,4-dichlorobenzene	**106-46-7**	**3.00E-02**	**8.00E-01**	**1.05E-01**	**13.1%**
1,1-dichloroehane	75-34-3	1.00E-01	5.00E-01	3.50E-01	70.0%
1,2-dichloropropane	78-87-5	1.10E-03	4.00E-03	3.85E-03	96.3%
(cis)-1,3-dichloropropene	**542-75-6**	**3.00E-04**	**2.00E-02**	**1.05E-03**	**5.3%**
dichlorvos	**62-73-7**	**5.00E-04**	**5.00E-04**	**1.75E-03**	**350.0%**
epichlorohydrin	**106-89-8**	**2.00E-03**	**1.00E-03**	**7.00E-03**	**700.0%**
ethylbenzene	100-41-4	1.00E-01	1.00E+00	3.50E-01	35.0%
ethylene dibromide	106-93-4	5.70E-05	2.00E-04	2.00E-04	99.8%
hexachlorocyclopentadiene	**77-47-4**	**7.00E-03**	**7.00E-05**	**2.45E-02**	**35000.0%**
methacrylonitrile	126-98-7	1.00E-04	7.00E-04	3.50E-04	50.0%
methyl bromide	74-83-9	1.40E-03	5.00E-03	4.90E-03	98.0%
methyl ethyl ketone	78-93-3	6.00E-01	1.00E+00	2.10E+00	210.0%
methyl isobutyl ketone	**108-10-1**	**8.00E-02**	**8.00E-02**	**2.80E-01**	**350.0%**
methylene chloride	**75-09-2**	**6.00E-02**	**3.00E+00**	**2.10E-01**	**7.0%**
naphthalene	**91-20-3**	**2.00E-02**	**3.00E-03**	**7.00E-02**	**2333.3%**
2-nitroaniline	88-74-4	6.00E-05	2.00E-04	2.10E-04	105.0%
nitrobenzene	98-95-3	5.00E-04	2.00E-03	1.75E-03	87.5%
phthalic anhydride	**85-44-9**	**2.00E+00**	**1.20E-01**	**7.00E+00**	**5833.3%**
styrene	100-42-5	2.00E-01	1.00E+00	7.00E-01	70.0%
tetrachloroethylene	**127-18-4**	**1.00E-02**	**4.00E-01**	**3.50E-02**	**8.8%**
tetrahydrofuran	109-99-9	2.00E-01	3.00E-01	7.00E-01	233.3%
toluene	108-88-3	2.00E-01	4.00E-01	7.00E-01	175.0%
1,2,4-trichlorobenzene	**120-82-1**	**1.00E-02**	**2.00E-01**	**3.50E-02**	**17.5%**
trichlorofluoromethane	75-69-4	3.00E-01	7.00E-01	1.05E+00	150.0%
vinyl acetate	**108-05-4**	**1.00E+00**	**2.00E-01**	**3.50E+00**	**1750.0%**

[a] RfC calculated from the RfD

[b] calculated RfC expressed as percent of actual RfD

Entries with pronounced differences between calculated and actual value are shown in bold.

Table 4:Route-to-Route Extrapolations of CSFs

Chemical name	CAS	Values Oral CSF	Inhal URF	Inhal CSF	oral/inhal[a]	Calculated value Inhal CSF[b]	cal % val[c]
Average					2076%		98%
minimum					8%		54%
maximum					58333%		105%
n~100%					22		35
acetaldehyde	75-07-0	7.70E-03	2.20E-06	7.70E-03		7.70E-03	100.0%
acrylonitrile	**107-13-1**	**5.40E-01**	**6.80E-05**	**2.40E-01**	**225%**	**2.38E-01**	**99.2%**
aldrin	309-00-2	1.70E+01	4.90E-03	1.70E+01	100%	1.72E+01	100.9%
aniline	62-53-3	5.70E-03	1.60E-06	5.70E-03	100%	5.60E-03	98.2%
arsenic	**7440-38-2**	**1.50E+00**	**4.30E-03**	**1.50E+01**	**10%**	**1.51E+01**	**100.3%**
benzene	71-43-2	2.90E-02	8.30E-06	2.90E-02	100%	2.91E-02	100.2%
alpha-BHC	319-84-6	6.30E+00	1.80E-03	6.30E+00	100%	6.30E+00	100.0%
bis(2-chloroethyl)ether	111-44-4	1.10E+00	3.30E-04	1.10E+00	100%	1.16E+00	105.0%
bromoform	**75-25-2**	**7.90E-03**	**1.10E-06**	**3.90E-03**	**203%**	**3.85E-03**	**98.7%**
carbon tetrachloride	**56-23-5**	**1.30E-01**	**1.50E-05**	**5.30E-02**	**245%**	**5.25E-02**	**99.1%**
chlordane	57-74-9	3.50E-01	1.00E-04	3.50E-01	100%	3.50E-01	100.0%
chlorobenzilate	510-15-6	2.70E-01	7.80E-05	2.70E-01	100%	2.73E-01	101.1%
chloroform	**67-66-3**	**6.10E-03**	**2.30E-05**	**8.10E-02**	**8%**	**8.05E-02**	**99.4%**
chromium hexavalent	18540-29-9		1.20E-02	4.10E+01		4.20E+01	102.4%
4,4'-DDT	50-29-3	3.40E-01	9.70E-05	3.40E-01	100%	3.40E-01	99.9%
1,2-dibromo-3-chloropropane	**96-12-8**	**1.40E+00**		**2.40E-03**	**58333%**		
1,2-dichloroethane	107-06-2	9.10E-02	2.60E-05	9.10E-02	100%	9.10E-02	100.0%
1,1-dichloroethylene	**75-35-4**	**6.00E-01**	**5.00E-05**	**1.80E-01**	**333%**	**1.75E-01**	**97.2%**
(cis)-1,3-dichloropropene	542-75-6	1.80E-01	3.70E-05	1.30E-01	138%	1.30E-01	99.6%
dieldrin	60-57-1	1.60E+01	4.60E-03	1.60E+01	100%	1.61E+01	100.6%
1,2-diphenylhydrazine	122-66-7	8.00E-01	2.20E-04	8.00E-01	100%	7.70E-01	96.3%
epichlorohydrin	**106-89-8**	**9.90E-03**	**1.20E-06**	**4.20E-03**	**236%**	**4.20E-03**	**100.0%**
ethylene dibromide	**106-93-4**	**8.50E+01**	**2.20E-04**	**7.70E-01**	**11039%**	**7.70E-01**	**100.0%**
ethylene oxide	**75-21-8**	**1.02E+00**	**1.00E-04**	**3.50E-01**	**291%**	**3.50E-01**	**100.0%**
heptachlor	76-44-8	4.50E+00	1.30E-03	4.50E+00	100%	4.55E+00	101.1%
heptachlor epoxide	1024-57-3	9.10E+00	2.60E-03	9.10E+00	100%	9.10E+00	100.0%
hexachlorobutadiene	87-68-3	7.80E-02	2.20E-05	7.80E-02	100%	7.70E-02	98.7%
hexachlorobenzene	118-74-1	1.60E+00	4.60E-04	1.60E+00	100%	1.61E+00	100.6%
hexachloroethane	67-72-1	1.40E-02	4.00E-06	1.40E-02	100%	1.40E-02	100.0%
methyl chloride	**74-87-3**	**1.30E-02**	**1.80E-06**	**6.30E-03**	**206%**	**6.30E-03**	**100.0%**
N-nitroso-di-N-butylamine	924-16-3	5.40E+00	1.60E-03	5.40E+00	100%	5.60E+00	103.7%
N-nitrosodipropylamine	621-64-7	7.00E+00	2.00E-03	7.00E+00	100%	7.00E+00	100.0%
TCDD	1746-01-6	1.50E+05	3.30E+01	1.50E+05	100%	1.16E+05	77.0%
1,1,1,2-tetrachloroethane	630-20-6	2.60E-02	7.40E-06	2.60E-02	100%	2.59E-02	99.6%
1,1,2,2-tetrachloroethane	79-34-5	2.00E-01	5.80E-05	2.00E-01	100%	2.03E-01	101.5%
tetrachloroethylene	**127-18-4**	**5.20E-02**	**5.80E-07**	**2.00E-03**	**2600%**	**2.03E-03**	**101.5%**
tetrahydrofuran	109-99-9	7.60E-03	1.90E-06	6.80E-03	112%	6.65E-03	97.8%
1,1,2-trichloroethane	79-00-5	5.70E-02	1.60E-05	5.70E-02	100%	5.60E-02	98.2%
trichloroethylene	**79-01-6**	**1.10E-02**	**1.70E-06**	**1.10E-02**		**5.95E-03**	**54.1%**
2,4,6-trichlorophenol	88-06-2	1.10E-02	3.10E-06			1.09E-02	
vinyl chloride	**75-01-4**	**1.90E+00**	**8.40E-05**	**3.00E-01**	**633%**	**2.94E-01**	**98.0%**

[a] oral CSF as percent of inhalation CSF

[b] inhalation CSF calculated from inhalation URF

[c] inhalation CSF calculated from inhalation URF, expressed as percent of inhalation CSF value

Entries with pronounced differences between calculated and actual value are shown in bold.

V. HUMAN HEALTH EXPOSURE

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General Issues

1. Organization of the Document

Considering the technical complexity of the material, the HHRAP is presented in a clear and relatively concise manner. The level and presentation of the material are appropriate for an intended audience of risk practitioners. It should also be a useful resource for permit writers, risk managers, and community relations personnel, to varying degrees. While it appears that care was taken to write the document in "plain English", it is unlikely that many members of the general public will understand all the procedures and, therefore, cannot be considered an intended audience. However, including an executive summary would greatly assist some of the readers of the document.

Generally, the organization of the report follows a logical format. One exception is the discussion of infant exposure to PCDDs and PCDFs via the ingestion of their mother's breast milk. This exposure pathway is briefly discussed in Chapter 2 and Appendix C, but not included as a separate section in the chapters on exposure scenarios, media concentrations, and quantifying exposure. Although it is stated in Chapter 4 that the ingestion of breast milk exposure pathway is evaluated separately in Chapter 2, this does not appear to be the case (it is just briefly mentioned in Chapter 2). In any event, a full evaluation of breast milk exposures would be out of place in the facility characterization chapter. I recommend including a discussion of the relevant aspects concerning the ingestion of breast milk exposure pathway in each of the exposure-related chapters.

There is a reasonable balance between the explanation of the procedures in the text and the extensive amount of chemical and media-specific data and model equations in the appendices. Finally, the authors have done a good job of stressing the importance of addressing data limitations, model uncertainties, and scenario assumptions in a discussion of risk assessment uncertainty.

2. Does Purpose Accurately Reflect Methodologies and Scope?

The stated purpose of the document is to explain how risk assessments should be performed at hazardous waste combustion facilities and provide a comprehensive source of data needed to complete the assessment. While this a fairly accurate capsule summary, it is too understated in several ways. It inadvertently misleads the reader into thinking that performing a risk assessment at a hazardous waste combustion facility is a straightforward task that simply involves plugging data into the equations, all of

which are provided in the document. While the HHRAP does pull together existing methodologies, there are many cases where site-specific conditions will warrant additional analyses, for which little or no guidance is provided in the document. The use of expert judgement on the part of the risk assessor is extremely important at the more complex sites and facilities, and that concept is lost in stating that "EPA OSW's objective is to present a user-friendly set of procedures for performing risk assessments..." Throughout the document, specific instances are cited where exceptional conditions may require the risk assessor to extend the analysis beyond the scope of the HHRAP; this should be reflected in the purpose statement in Chapter 1.

The scope of the document is also somewhat misrepresented in the final chapter, where it is stated that "the main purpose of developing the HHRAP was to provide risk assessors with a tool for completing quality, consistent, and defensible risk assessments in a short amount of time, rather than spending years to determine which COPCs, exposure pathways, and receptors the risk assessment report should include and evaluate." While it is clear that the procedures presented in the HHRAP will help to streamline the process somewhat, I do not believe that risk assessments will now be able to be completed in a short amount of time (especially at complex sites where at least some site-specific data and analysis may be required). However, it is also stated in Chapter 9 that another purpose was to provide "...the tools needed to clearly communicate the procedures, results, and limitations of the risk assessment process." I feel that this is an accurate statement of one purpose of the document.

3. Critical Evaluation of Scientific Aspects of Document

Scientific aspects of the exposure-related sections appear to conform to accepted EPA approaches for conducting risk assessments, much of which has also been reviewed and supported by the Science Advisory Board. I do, however, have specific concerns about several technical aspects of the methodology and interpretation of the results (e.g., uncertainties, cumulative risks, and accidents); these issues are addressed below in General Issue #5 and Additional Comments.

4. Are Risk Assessment Results Credible?

Whether or not the results of HHRAP-based risk assessments can be considered credible depends on how these risk estimates will be applied. Given the considerable uncertainties and limitations in using this methodology, the risk results cannot serve as the sole basis for making permitting decisions. The HHRAP recommends a process for evaluating "reasonable" potential risks, not "theoretical worst-case maximum" risks. Although I agree that worst-case maximum risk estimates would not be appropriate, assumptions should err somewhat on the conservative side to compensate for the many sources of uncertainty in these risk assessments. For example, there is a paucity of data on interactions (e.g., synergism, antagonism) between specific chemicals in complex environmental mixtures. The necessary

simplifying assumptions regarding chemical interactions and risk additivity can be discussed in the uncertainty section of a risk assessment. Nevertheless, the quantitative estimates of risk do not account for these types of analytical uncertainties and current data gaps and, therefore, should be considered in only a relative sense for environmental decision making. In other words, the risk results are probably credible enough to be one of several technical and non-technical inputs to an integrated risk management process, but not as a definitive statement of absolute risks or hazards.

5. Major Data and Methodological Gaps

It appears that most of EPA's current risk assessment guidance has been incorporated or adapted for use in the HHRAP. The limitations of the methodology and site-specific conditions that may warrant additional analysis (beyond the scope of this protocol) have been identified. To use the results of this analysis for risk-based decision making, however, several additions to the document are recommended.

a) The risk assessment methodology should address the issue of background exposures more consistently. The general approach to estimating exposures and doses in the HHRAP involves assessing incremental intakes of chemicals emitted from a facility. For some contaminants, it is necessary to also account for existing body burdens and intakes from other sources. This is especially important for compounds that are retained in the body, have relatively low thresholds, or have other significant sources in the environment. For example, the HHRAP recommends the use of the IEUBK model for predicting blood lead levels in children (this should be required for any site with lead as a COPC). Therefore, estimated existing body burdens and intakes of lead from other sources for children living in close proximity to hazardous waste combustion facilities are factored into the analysis. Noncancer health effects of dioxins are evaluated simply by comparing exposures from the facility's emissions to national average background exposure levels for dioxins. Background intake of methylmercury through the consumption of non-local fish and seafood is not considered in the exposure modeling for the residential and farmer scenarios, for whom consumption of commercial seafood and fish is the primary source of methylmercury intake. If local conditions suggest that nearby surface water bodies may be used as a potential source of fish for consumption by residents, then it is important to evaluate if that additional exposure to methylmercury from facility emissions could present a health risk when combined with current intake levels. It does not appear that the HHRAP recommendation of reducing the risk level and hazard index benchmarks (on a site-specific basis) is sufficient to account for the whole spectrum of background exposures in different subpopulations with varying susceptibilities.

b) The data or methodological gaps may or may not preclude the use of this risk assessment protocol for decision making. The reason it is unclear is because the document does not include a discussion of possible risk management or policy actions that could be taken based on the results. How will the results specifically be used in the permitting process? For example, will the risk assessment results be used to define exposure/engineering control options or as the only basis for setting emission limits? The HHRAP could also be a useful tool for risk reduction during the permitting process by providing input to other risk management options, e.g., pollution prevention, regulatory changes, prioritization, and education/outreach. The analytical limitations have different implications for regulatory decision making, so this document needs to identify the most likely use of the findings.

c) It is stated in the HHRAP that accidental releases are not considered within the scope of this guidance and that a decision to consider accidental releases should be made on a site-specific basis. Given that the peer review panel for the WTI Incinerator risk assessment felt that accidents were potentially among the most significant risks at that facility, an accident analysis should be required at sites in similarly populated areas. In addition to referring the reader to current EPA guidance on this topic, additional guidance should be provided that specifies under what conditions this analysis would be necessary and the types of accidents that would need to be evaluated.

6. Research Needs

The following are my suggestions for long-term research that could improve the risk assessment methodology:

It is important to develop methods to integrate cumulative risk estimates from ambient air and other pathways to provide a basis for comparing the contributions from various sources and for contrasting the risk levels among different locations with different types of sources. The next important step would be the development of approaches to conduct integrated assessments that consider different types of possible effects: not only human and ecological health risks, but also social, cultural, and economic impacts.

The importance of considering environmental health risks to children is obvious. Specific issues include: proportionally greater intake of food, water, and air than adults; nursing infants; more hand-to-mouth behavior; immature metabolic pathways; dependence on adults for "risk management" decisions; and more future years to develop chronic diseases from environmental exposures. In general, the HHRAP attempts to evaluate risks to children by making some adjustments to exposure factors, which is a logical first step. Methods for assessing childhood risks need to be further developed, however, to

more directly account for childrens' unique susceptibilities and sensitivities to specific contaminants and chemical mixtures. In particular, there is a need to improve knowledge regarding the relative importance of exterior dust and soil as lead exposure sources for children in various residential environments.

The idea of simply reducing the acceptable risk range and hazard benchmark level as a means to realistically account for background contributions from exposures to a wide range of contaminants should be further evaluated.

There is a need for chemical mixture-related research, especially with respect to predicting the health effects of the unidentified organic compounds in the total organic emissions fraction. The most relevant research would be toxicity studies of the whole mixture of emitted chemicals from representative facilities.

Because of the uncertainty associated with the inclusion of unspeciated total organic emission data when estimating stake emission rates, research is also needed to evaluate this component of the facility characterization, e.g., what is the composition of the gravimetric fraction typically and how appropriate is the approach for attributing a risk to this unknown portion of emissions?

Because of the increasing involvement of stakeholders in the risk assessment and management process, improved approaches are needed for managing environmental risk data and communicating risk-related information. A variety of tools should be utilized and further developed (e.g., World-Wide Web, geographic information systems) to not only disseminate risk results, but also facilitate an exchange of information with various stakeholder groups.

There is a need to collect environmental monitoring data for model input parameters that drive the exposure assessment in order to validate the modeling approach. In particular, fate and transport data are needed to track long-term exposures and health effects from combustion emissions. Large-scale epidemiological studies should be carried out to verify that the health effects results predicted by these risk assessments are valid.

To address the substantial uncertainties inherent in mercury risk assessments, prioritized research needs have recently been identified in EPA's *Mercury Research Strategy* (NCEA-I-0710, Nov. 1999). This report describes a research program that provides information, methods, models, and data for addressing key scientific questions, which are clearly needed to reduce the uncertainties in the HHRAP approach to estimating mercury exposures and risks.

SPECIFIC TECHNICAL ISSUES

1. 95th Percentile Emission Rate

The guidance for quantifying emissions rates of compounds is generally adequate, although, for clarity, it should specify the 95 percent upper confidence limit of the mean emission rate. There should also be additional guidance that discusses under what circumstances the risk assessor is required to use the emission rate data developed from maximum operating conditions.

2. Treatment of Non-Detected Compounds in Estimating Stack Emission Rates

The recommendation in the HHRAP is to assume that COPCs are present at a concentration equivalent to the MDL-derived RDL for non-isotope dilution methods, or the method-defined EDL for isotope dilution methods. For compounds that have already been retained as COPCs (i.e., are in the waste stream and/or detected during trial burns), it would be inappropriate to assume a zero for every non-detect. On the other hand, it seems overly conservative to assume that for every non-detect, a compound is present at its detection limit (which is already several times higher than the method detection limit). For estimating stack emission rates, I recommend using one-half of the detection limit (as defined above), which is typically the intermediate approach that EPA and others have used for handling non-detects for COPCs.

3. Treatment of TOE Data in Estimating Stack Emission Rates

As noted in the document, the inclusion of the "unknown" or unspecified total organic emission (TOE) data when estimating stack emission rates has many limitations. The assumption that the mass of unidentified organic compounds has the same toxicity as an equal mass of identified compounds cannot be considered scientifically accurate. However, in the absence of additional data on mixtures toxicity (see General Issue question #6), this is simply a risk assessment technique to account for, and possibly bound, the toxicity of the whole organic fraction of emissions. A common approach is to select representative (surrogate) organic compounds in stack emissions with the highest quantity, toxicity, and persistence. The scaling of toxicity-weighted emission rates in the HHRAP is an attempt to be more comprehensive in the treatment of unknown organics, which is particularly important whenever the unknown fraction of emissions is large. The considerable limitations of the technique should be discussed in the uncertainty section of the risk assessment, and additional toxicity research should be conducted to investigate the significance of the unknown organics fraction in complex environmental mixtures such as hazardous waste combustion emissions.

4. Mercury Speciation and Modeling

The approach presented in the HHRAP for speciating and modeling of mercury is generally consistent with recently published EPA guidance, in particular the *Mercury Study Report to Congress* (EPA-425/R-97-003, Dec. 1997). The potential risk from mercury emissions is estimated based on a number of simplifying assumptions concerning forms and speciation of mercury, as well as its transport, fate, and uptake. The document points out limitations of the models, and includes the caveat that some risk from mercury not depositing locally will be missed in a site-specific risk assessment. In addition, there is appropriate flexibility for a facility to use more complex models and detailed site-specific data to predict the fate and transport of mercury at exposure points. Given the uncertainties, it is also reasonable to be conservative and assume that the sum of the divalent and methylmercury fish concentrations is 100 percent methyl mercury in estimating potential risk.

5. Determination and Application of Biotransfer Values

I do not have any additional information on specific biotransfer values.

6. Ingestion of Contaminated Water by Cows

There are limited data available on water ingestion by cows as a potential COPC uptake mechanism. Assuming the same source of water, direct exposures of human receptors to contaminants in drinking water is likely to be a more significant pathway than secondary exposures from meat and milk. However, a recent U.S. DOE environmental impact statement included ingestion rates of water for meat and dairy cows of 50 l/day and 160 l/day, respectively, assuming they ingest groundwater and fodder that are irrigated with contaminated groundwater (DOE/EIS-0269, April 1999). Exposure pathways involving groundwater are not generally evaluated for combustion units, unless site-specific conditions dictate otherwise (e.g., infiltration of COPCs into very shallow aquifers). Because the protocol focuses on the most significant exposure pathways, it is probably not necessary to account for the ingestion of contaminated water by cows unless pasture grasses are also irrigated with contaminated groundwater from shallow wells or surface water.

7. Selection of Exposure Scenario Locations

The guidance for identifying the most representative exposure scenarios at actual receptor grid nodes is adequately explained. There appears to be sufficient flexibility in the methodology that additional exposure scenario locations within a particular land use area can be added based on site-specific conditions. One situation that needs to be clarified is for identifying exposure scenario locations at large

government installations, where the recommended receptor grid node array would need to be extended out much more than 10 km from the facility emission sources. Additional guidance is needed on both the method for providing additional coverage of the area of concern and practical considerations for when all of the HHRAP-recommended receptors would be located offsite at a much greater distance than 10 km.

8. Non-Cancer Effects from Dioxins and Furans

The approach for evaluating noncancer health effects of dioxins involves a comparison of exposures resulting from facility emissions to national average background exposure levels for dioxins. The need for consistency in addressing background exposures is discussed above (see General Comment #5a). In addition, guidance is needed on how low facility-related exposures must be compared to background exposures in order to be deemed acceptable. The HHRAP assumption of 4% as the percentage of fat in breast milk is near the upper end of the range and is the value often used as the default value in risk assessments (e.g., *Mercury Study Report to Congress*). It does appear that depletion of dioxin in breast milk occurs over the first year, approaching steady state at around 30-50% of initial levels. The theoretical models and limited data discussed in the *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustion Emissions* (EPA 600-R-98/137, Dec. 1998) suggest that overprediction of breast milk concentrations will occur and, therefore, there is a need to account for the decreasing dose to nursing infants.

ADDITIONAL COMMENTS

Cumulative Risks

The risk assessment methodology does not address cumulative risks, defined as the total health risk associated with multiple stressors from multiple sources. The methodology focuses on the incremental risks of contaminants emitted from a single hazardous waste combustion facility or site, which is the approach that EPA risk assessments have historically followed. Although a total measure of cumulative carcinogenic risks or noncancer hazards for all possible exposures is not currently feasible, the risk assessments should attempt to evaluate additional, major sources of potential exposure for significant contaminants of concern from a facility. For example, exposure and risk from multiple point sources within a given geographic area should be quantitatively evaluated. The analysis of air emissions from multiple point sources (and eventually mobile and area sources) would provide stakeholders with a more complete picture of a facility's emissions in relation to environmental loadings from currently operating facilities in the same community. While the methodology for evaluating multiple and

cumulative exposures and risks has not been fully developed, the document should at least follow the Administrator's Cumulative Risk Assessment Guidance on Planning and Scoping (U.S. EPA, "Guidance on Cumulative Risk Assessment, Part 1. Planning and Scoping," Science Policy Council, Washington, D.C., July 3, 1997).

Policy Decisions

The HHRAP reflects numerous policy decisions that were made in developing the risk assessment methodologies. For example, it is stated on page 8-3 that "The models specified for use in this document were selected on the basis of scientific policy." It is difficult, if not impossible, to conduct a full technical review of the document without a consideration of the science policy decisions that have guided its development. Therefore, policy considerations and other risk management factors that affect the HHRAP methodologies should be explicitly identified and their relevance discussed. It appears that EPA Region 6 recognized this need in publishing a "Risk Management Addendum to the HHRAP" <http://www.epa.gov/earth1r6/6pd/ rcra_c/protocol/r6add.pdf>. This analysis should be expanded and incorporated into the main document.

Uncertainties and Limitations

Chapter 8 provides a good overview of uncertainties and resulting limitations in the risk assessment process. I strongly support the suggestion to include a table that lists the key assumptions in the risk assessment, the rationale for those assumptions, their effects on risk estimates, and the magnitude of the effects. However, more information is needed on how to conduct a quantitative uncertainty analysis (unless the reader is referred to another source, e.g., EPA's Draft *Risk Assessment Guidance for Superfund Volume 3 - Part A: Process for Conducting Probabilistic Risk Assessment*, Dec. 1999). But even more important, the reader is not given any guidance in the HHRAP for deciding when a detailed quantitative treatment of uncertainty is required. In addition, while there is enough guidance in the chapter about the qualitative description of uncertainties, there is no discussion about how this type of information should be incorporated into the risk-based decision-making process.

Recommended Exposure Scenarios

There are some inconsistencies and omissions in the recommended exposure pathways and receptors. As noted in the document, the subsistence farmer (and child) scenarios are assumed to have "reasonable" intakes of food items. The rationale for including subsistence receptors is to provide an upper bound on exposures for estimating high-end risks. The use of reasonable dietary intakes defeats this purpose. Similarly, omitting the exposure pathway of ingestion of locally caught fish for subsistence

farmers could potentially result in an underestimate of high-end risks. Because the final risk and hazard estimates are sums of cancer risks or hazard indices (for similar health effects) from multiple exposure pathways, it is important to avoid this kind of artificial division between receptor populations. Therefore, it may be appropriate to introduce intermediate categories to account for partial homegrown food and fish consumption, instead of attempting to represent subsistence conditions with more typical food intakes. It would also be a good idea to provide examples of exposure scenarios that may be necessary to consider as a result of site-specific conditions, e.g., hunters, trespassers, and noninvolved workers (persons working at a site but not directly involved with the handling of hazardous materials).

Modifying IRIS Risk Values

Appendix 1A of the updated *Exposure Factors Handbook* (EPA/600/P-95/002Fa, Aug. 1997) discusses procedures for ensuring that assumptions about population parameters in the dose-response analysis are consistent with the population parameters used in the exposure analysis. These procedures include correction factors for dose-response parameters and intake data. For example, in the HHRAP the mean drinking water intake of 1.4 L/day is used instead of 2 L/day. Therefore, it may be necessary to adjust the IRIS values for certain contaminants.

Miscellaneous

Chapter 1: The introduction should discuss the applicability of this guidance to other type of combustion facilities. It is clearly stated that the HHRAP is intended for use in evaluating hazardous waste combustors. However, it is obvious that the methodology can, and probably will, be used at other facilities, such as medical waste incinerators, waste-to-energy plants, etc. Caveats about the relevance of using the HHRAP guidance for risk assessments of other combustion facilities need to be spelled out.

Section 1.1: I am generally in favor of retaining flexibility in the HHRAP for site-specific modifications, but the flip side is that for a guidance document ("a set of user-friendly procedures for performing risk assessments"), much is left for the risk assessor to figure out. More guidance would be helpful for deciding if and how modifications to the protocol should be made.

Section 1.3: Should include the *Risk Assessment for the Waste Technologies Industries (WTI) Hazardous Waste Incineration Facility (East Liverpool, Ohio)* (EPA-905-R97-002, May 1997) as one of the reference documents used to prepare the HHRAP, because it appears that much of the methodology was developed and/or applied in the WTI study.

Section 1.3: The EPA/NCEA guidance, *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustion Emissions* is mentioned as in press, but it has since been released. More important, there should be some discussion about what connection there is between the two documents. Should the NCEA methodologies and data be integrated into or coordinated with the approaches in the HHRAP?

p. R-1: The References section is missing from the bound copy of the peer review draft of the document, although it is available on the OSW and Region 6 HHRAP Web sites.

Table A-1: It would be more helpful to have the compound names listed alphabetically. Without access to the electronic file, it is doubtful that readers will be able to look up many compounds by CAS numbers. It may be best to simply include two versions of Table A-1 with compounds sorted both ways.

Dr. Richard L. DeGrandchamp

General Comments

USEPA and EPA Region 6 should be commended for developing an outstanding risk assessment guidance document that is very detailed and comprehensive. This document presents a correct risk assessment methodology and a compendium of information necessary to quantify risks associated with hazardous waste combustion facilities. The breadth and depth of information presented in this guidance document is unprecedented as a stand-alone USEPA risk assessment guidance document.

One general improvement, however, is to clearly state early in the document whether the guidance is based on risk assessment *policy* or *science*. If the guidance is based on risk assessment policy, rather than science, for the purpose of achieving consistency and uniformity, with the ultimate goal of protecting the general public, few changes are needed. The only necessary change would be a clear statement of that purpose and a stated recognition that, for many facilities, risks are intentionally being overestimated. If the purpose of the HHRAP is to provide guidance for conducting a scientifically defensible risk, some changes are required.

The seminal work for all risk assessment paradigms is the National Academy of Sciences' (1983) *Risk Assessment in the Federal Government: Managing the Process* (also known as the Red Book). This document forms the framework for all USEPA risk assessment guidance and, most notably, *Risk Assessment Guidance for Superfund, Parts A, B, C, and D* (and all supplemental guidance). The approach suggested in HHRAP was compared with the Red Book and all subsequent USEPA Superfund risk assessment guidance. Although for the most part, the HHRAP appears to follow generally accepted risk assessment practices, the HHRAP methodology is much more prescriptive and based primarily on default assumptions. There is little latitude to develop the best scientific approach for the site using site-specific information.

The single most important goal of any risk assessment is to accurately predict risks associated with actual or likely exposures based on the best scientific information. Risk assessments should not intentionally overestimate risks because it is "health protective." That is the function of risk management personnel (permit writers), who can introduce safety factors into the final decision based on the confidence of the final risk estimate.

If the intent of the HHRAP is to produce accurate, precise, and scientifically tenable risk estimates, greater emphasis should be placed on conducting risk assessments with site-specific information (adequately supported with documentation), rather than default parameters. It should be noted that

there is a great deal of conservatism already built into the overall risk assessment methodology. Many assumptions and exposure scenarios are recognized by most risk assessors as highly unrealistic, but are routinely used to protect the general public. The HHRAP continues to follow the *status quo*.

Regardless of the scientific approach, it is necessary to carefully examine the underlying assumptions in the risk assessment so that correct risk management decisions can be made. This is the central theme discussed in *EPA Risk Characterization Guidance* (EPA 1995):

> "*...we must adopt as values transparency in our decision making process and clarity in communication with each other and the public regarding environmental risk and the uncertanties associated with our assessments of environmental risk. This means that we must fully, openly, and clearly characterize risks. In doing so, we will disclose the scientific analyses, uncertainties, assumptions, and science policies which underlie our decisions as they are made throughout the risk assessment and risk management process.*"

To provide a high margin of safety, conservative health-protective default (or generic) assumptions are intentionally incorporated into risk-based screening. These assumptions are not intended to be "most likely or best estimates" and do not apply to the most of the population, but represent upper-bound estimates, ensuring *no* hazardous waste will pose unacceptable human health risk to any person. Like Superfund risk assessment guidance, there are numerous aspects of the HHRAP risk assessment approach that do not represent realistic conditions and will result in overestimating risk. The exposure assumptions (which are implicit and not often articulated) that are made to estimate the Reasonable Maximum Exposure (RME) risk for the residential receptor is a good example. The RME is defined as the highest exposure that is reasonably expected to occur at a facility. However, it is important to stress that the calculated risk is for a *single individual*. That is, it is assumed that the *same* individual is simultaneously exposed to the contaminants in all environmental media within an *a priori* defined exposure area. The carcinogenic risk is estimated as the incremental probability of this hypothetical person of developing cancer over a lifetime (prorated for the time spent at the location) as a result of exposure to the potential carcinogen. The assumptions underlying this risk estimate are not realistic. For example, it is assumed that this resident is essentially born on the lot at the facility and (except for 2 weeks per year) does not leave the yard. This person remains in the yard 24-hours per day, 7 days a week. What is most important to note is that this hypothetical person does not attend grade school, high school/prep school, or college, or even hold a job outside the home, until he/she reaches 30 years of age (even though the median time at the same residence is 9 years). Additionally, this person is (most often) assumed to be exposed to the maximum detected concentration in each environmental medium each day. Clearly, few U.S. citizens will ever be exposed in this manner because these

exposure conditions are almost impossible to imagine and would only apply to a situation where a child would essentially be infirm and remain infirm and restricted to bed through adulthood because of illness. However, for the infirm individual confined to their bed for the entire 30 years, the risk assessment is conservative because the risk assessment assumes that the person remains outside for the entire 30 years and is in direct contact with contaminated soil and other environmental media. In fact, for the truly infirm, the risks would be much lower because the only contact would be with contaminated *dust*, which is only a fraction of the soil concentration. The dust concentration is typically only between 20 and 60 percent of that found outdoors (recent USEPA Soil Screening recognizes this reality and provides for the adjustment for office workers). It is necessary to recognize that these conservative assumptions and features are already built into the risk assessment paradigm so that additional conservatism is not added capriciously.

It should be noted that, in addition to the exposure assessment, conservatism or bias is intentionally introduced into all other parts of the HHRAP (similar to other USEPA guidance documents), including data analysis, toxicity assessment, and risk characterization. There is nothing inherently wrong with this approach as long as the objectives and aims of the risk assessment are discussed and the results communicated to all stakeholders.

Technical Accuracy

The overall technical accuracy of the HHRAP is very high. Many technical errors have apparently been identified and corrected in the errata. However, there are several (important) instances where the HHRAP deviates from USEPA Superfund risk assessment guidance (specific issues are addressed in subsequent sections). This does not necessarily make the HHRAP incorrect, but it is important to justify deviations from existing risk assessment approaches and provide rationale for making the changes.

Completeness

The HHRAP is very complete. It is readily apparent that considerable attention has been made to provide all pertinent information for conducting a very complex risk assessment for every possible exposure pathway. However, for most risk assessments, only a few chemicals for one or two routes of exposure account for nearly all (90 to 95 percent) of the risk for the RME individual. Accordingly, it would streamline the HHRAP and make it more cost effective to provide a screening step to identify significant exposure pathways and chemicals prior to initiating the risk assessment. Although USEPA is to be lauded for the attention to detail in estimating risk for all potential points of exposure, it may be unnecessary to evaluate all pathways equally.

Minor pathways posing insignificant risk should not be evaluated in the detail that the predominant pathways are.

The risk assessment approach of winnowing insignificant pathways has recently become the *de facto* scientific paradigm in recent USEPA risk assessment guidance. For example, before initiating a risk assessment, most facilities first screen the site to identify high-risk chemicals of concern (COCs) and exposure pathways. The Presidential/Congressional Commission on Risk Assessment and Risk Management's reports, *Framework for Environmental Health Risk Management* and *Risk Assessment and Risk Management in Regulatory Decision-Making*, strongly recommend this approach to wisely use valuable resources, time and effort to conduct a risk assessment to determine if significant and meaningful risks are posed by hazardous waste. To conduct a risk assessment for every conceivable permutation of exposure just because computer spreadsheet software is available may be a waste of valuable resources and divert attention from truly protecting the public health.

When a chemical is present in emissions at a concentration below some acceptable level (based on conservative exposure assumptions), based on a back-calculation similar to the approach used to develop chemical-specific *Preliminary Remediation Goals* ([PRG] EPA Region 9), *Media Specific Screening Levels* ([MSSLs] EPA Region 6), *Soil Screening Levels* ([SSLs] USEPA), as well as many state-derived screening levels, it would provide needed focus. Little effort would be wasted on quantifying risk for low risk chemicals and pathways in the HHRAP. Employing a screening tool can also resolve the dilemma addressed in the HHRAP pertaining to non-detect chemicals.

When the analytical detection limit is below the screening level (based on an *a priori* acceptable level), it can be concluded that the non-detect chemical can be confidently eliminated, even if it assumed to be present just below the detection limit. This approach circumvents the scientifically questionable approach of using the detection limit concentration for non-detects in the risk assessment.

Typically, a scientifically-based risk assessment attempts to first identify the most important exposure routes (through a sensitivity analysis), then considerable effort is made to collect site-specific information pertinent to those pathways to precisely estimate risk. By giving equal weight and consideration to primary, secondary, and tertiary pathways, and exerting equal effort for all pathways, the risk assessment effort is diluted. If it is more important to USEPA to be able to compare different facilities burning hazardous waste, there may be no alternative to ensuring each facility conduct a policy-based default-type risk assessments with the comprehensive approach suggested in the HHRAP. This will, of course, achieve uniformity. In that case, few changes to the HHRAP are necessary.

Scientific Soundness

With a few exceptions, the overall scientific approach parallels the approach described in other USEPA risk assessment guidance documents. As previously mentioned, using the default approach with individual assumptions will result in overestimating risks, as is the case with other risk assessment protocols. However, one major difference between other risk assessment methodologies (e.g., Superfund risk assessment guidance) is that risks are based on actual samples collected from all different environmental media. With the HHRAP, primary (direct), as well as secondary and tertiary (indirect) risks, are (primarily) calculated from stack emissions. Concentrations in all environmental media, as well as risks, are estimated solely through mathematical modeling. Any conservatism associated with the original input emission concentrations would be tremendously amplified through the secondary and tertiary pathways.

As with any mathematical model, confirming the scientific veracity of the risk assessment approach and confirming the results requires comparing the predicted risk estimates with the actual incidence of carcinogenic and noncarcinogenic effects. This is similar to confirming air modeling results with samples collected from actual air monitoring. The problem with risk assessments is that (as discussed previously) we will never have a large population continuously exposed to hazardous waste for 30 years. Furthermore, it would take an exposed population of 5 trillion people (about 1,000 times the Earth's population) at the facility to detect tumors at a 1E-6 risk level (due to the background incidence of cancer ~ 3E-1 in the United States). Therefore, the veracity of the risk assessment method (scientific soundness) can only be evaluated on the basis of the mathematical model itself.

As previously mentioned, all environmental media concentrations and risks are estimated from a relatively few point source stack emission samples. These concentrations are modeled to estimate the total RME risk for the hypothetical individual at the facility. According to USEPA (1991), the RME for an individual pathway should represent the 90 to 99 percentile individual:

> *"Readers are reminded that the goal of the RME is to combine upper-bound and mid-range exposure factors in the following equation so that the result represents an exposure scenario that is both health protective and reasonable; not the worst case."*

USEPA (1989) cautions that combining pathways for estimating the RME risk should not be a matter of simply summing the RME risk for each pathway:

> *"After estimating the RME for individual pathways, there are two steps required to determine whether risks or hazard indices for two or more pathways should be*

combined for a single exposed individual or group of individuals . The first is to identify reasonable exposure pathway combinations. The second is to examine whether it is likely that the same individuals would consistently face the "reasonable maximum exposure" (RME) by more than one pathway…

Identify exposure pathways that have the potential to expose the same individual or subpopulation at the key exposure areas evaluated in the exposure assessment, making sure to consider areas of highest exposure for each pathway for both current and future land-uses (e.g., nearest downgradient well, nearest downwind receptor). For each pathway, the risk estimates and hazard indices have been developed for a particular exposure area and time period; they do not necessarily apply to other locations or time periods. Hence, if two pathways do not affect the same individual or subpopulation, neither pathway's individual risk estimate or hazard index affects the other, and risks should not be combined. Once reasonable exposure pathway combinations have been identified, it is necessary to examine whether it is likely that the same individuals would consistently face the RME as estimated by the methods described in Chapter 6. Remember that the RME estimate for each exposure pathway includes many conservative and upper-bound parameter values and assumptions (e.g., upper 95th confidence limit on amount of water ingested, upper-bound duration of occupancy of a single residence)."

The more pathways are summed, the greater the probability that risks are overestimated. There doesn't seem to be any conscious effort in the HHRAP to ensure that the final RME risk estimate will represent ~ 95 percentile individual. When all the pathways in the HHRAP are aggregated, the final risk estimate will far exceed the target percentile risk. That is because conservatism is multiplicative, not additive, in the risk assessment. Aggregating the maximum RME for all pathways to calculate the cumulative risk for the RME individual will be unrealistic and likely to far exceed the 99 percentile individual. In other words, the risk estimates will be meaningless because the likelihood of exposure actually occurring as modeled is nil. It will, however, be health protective.

USEPA has recently developed an approach that circumvents the compounding conservatism inherent in the deterministic approach in the HHRAP. It involves applying probabilistic methods. The *Draft Risk Assessment Guidance for Superfund, Volume 3 - Part A, Process for Conducting Probabilistic Risk Assessment* presents a probabilistic approach that will yield a more precise, legally and scientifically tenable risk estimate, even when the myriad pathways suggested in the HHRAP are evaluated. Conducting a probabilistic risk assessment should be an option available to risk assessors using the HHRAP. The advantages would be as follows:

Avoiding "compounding conservatism," which is inherent in single-point, deterministic approaches;

Complete utilization of the entire database for each exposure pathway;

The ability to conduct detailed sensitivity analyses to identify and rank exposure routes exposure parameters that dominate the risk assessment to guide additional data collection;

Precluding disagreements regarding selecting the single "most appropriate" input value for a particular parameter (since the entire data population is used);

Estimating the entire risk range instead of the RME and;

Quantifying the uncertainty surrounding the risk estimate.

Probabilistic risk assessments model exposure and risks to a population of human receptors by interactively calculating risk for each person in an exposed population. Each iteration represents a statistical model of one person drawn from the hypothetical facility population. When these iteration results are combined, individual risk to any receptor (i.e., 95th percentile RME individual) can be estimated. Probabilistic techniques have been used for decades in many scientific disciplines and the recent draft USEPA guidance (1999) provides all the tools necessary. The recent USEPA guidance is very well written and could be simply be cited as an option for those facilities having the resources and inclination to calculate precise estimates of risk.

Specific General Questions:

1. Is the presentation of information clear and concise, considering the technical complexity of the subject and intended audience?

   The organization is well thought out and presented. Although the HHRAP presents and uses slightly different nomenclature and terms, the organization generally follows other USEPA risk assessment guidance.

2. Does the purpose of the HHRAP as stated in the Introduction (Chapter1) accurately reflect the presented methodologies and scope?

   Yes, the document follows the purpose stated in Chapter 1. However, the HHRAP suggests conducting a default-type "black box" risk assessment and, if the estimated

risks are unacceptable, then a site-specific risk assessment "can" be conducted. Experience has shown that this approach could be doomed for failure because, unfortunately, conducting two risk assessments appears to the general public *as "investigating until you get the right answer you are looking for."* It is recommended that, instead of conducting a default and site-specific risk assessment, the data sets be compared with screening values (an exercise that should take no longer than one-half hour) and only one site-specific risk assessment be conducted. Although there are many intentional and unintentional sources of conservatism introduced into the risk assessment that risk assessors are keenly aware of, it is difficult to communicate to the general public *"why the risk wasn't calculated correctly in the first place."*

It would streamline the process considerably and allow PRPs to identify important exposure pathways and spend the finite resources on estimating risks for significant and meaningful sources of risk resulting in *one* final scientifically tenable risk assessment.

3. Are the interpretations based on sound biological principles, accurate, and legally defensible scientific support?

Overall, the HHRAP interpretation of the existing risk methodology is good. However, as in previous USEPA risk assessment guidance documents, there is little information or biological support for many of the assumptions. Without going into great detail, there are many areas of hidden conservatism, such as the USEPA toxicity values, which are presented in the HHRAP as a given. There is general agreement among toxicologists that thresholds do exist for carcinogens, but the USEPA policy is that low-dose extrapolation models must go through zero. This may sound minor, but the difference between low-dose extrapolation models can result in a difference in risk estimates of around 20 orders of magnitude for some chemicals. Although this is well known amongst toxicologists, this area of conservatism is not generally discussed in the HHRAP. The HHRAP is scientifically defensible in that it follows the *status quo*.

4. Does the risk assessment support a credible interpretation of what is known and risk predicted?

Yes, the approach parallels previous risk assessment guidance and is health protective. However, it does not present a methodology that will result in precise, scientifically based risk estimates.

5. Are there any methodology gaps that would preclude using the HHRAP for regulatory decision making?

No, except that it presents a slightly different methodology from Superfund risk assessment guidance. The private sector could conceivably accuse USEPA of being inconsistent and applying different scientific guidance and regulating RCRA and CERCLA sites differently.

The differences lie primarily in identifying COCs and quantifying exposure point concentrations, which, in the case of HHRAP, is one of the most important aspects because the risk assessment is completely dependent on the initial input emission concentrations for all pathways.

6. What long-term research would you recommend that could significantly improve risk assessments of this type in the future?

As mentioned earlier, the litmus test for any risk assessment is to compare the predicted risks with the actual incidence of health effects. For example, when applying the conventional USEPA risk assessment methodology (similar to the HHRAP) to estimate risks for exposure to ***naturally occurring*** levels of arsenic in U.S. soils (using the 95UCL of the mean background concentration), it is estimated that there would be slightly more than 1 million cases of arsenic-induced tumors (lung cancer, hyperplastic keratosis, etc.) every year (since we know that all US citizens are exposed to background arsenic in soil regardless of where they go their entire lives, we can be certain we have a real exposure population). Obviously, we don't observe 1 million cases of arsenic-induced cancer from soil in the United States each year; if we did, it would be a national emergency. Since the predicted tumor rate for arsenic exposure to *background* levels is insignificant, it is apparent the risk assessment model is incorrect.

Likewise, a long-term study needs to be initiated at hazardous waste facilities to confirm that the HHRAP model is correctly predicting risk estimates. If would be difficult to conduct epidemiological studies for cancer, but biomarker studies could be used to at least determine whether exposures are actually occurring.

It will also be necessary to confirm that the modeled concentrations actually exist in all the environmental media for all the intake sources in the risk assessment. A sensitivity analysis also needs to be conducted to confirm that the indirect pathways are truly significant and

warrant inclusion in the risk assessment. The sensitivity analysis should not be mathematical, but should be based on actual biological measurements from foodstuffs (e.g., eggs, chickens, meat, milk, etc.)

Specific Technical Questions:

1. What is the scientifically-defensible estimate of the emission rate (i.e. 95th percentile emission, 95 UCL of the mean)?

Since the emission rate is being used to model not only direct but indirect exposures, it is important that the concentration represent a relatively steady-state concentration to which a receptor could be exposed over the entire 30-year period. The conditions of the trial burn are such that it is highly likely that the emissions represent the worst case, which may be unreasonable. Therefore, the first issue that must be addressed is how well the combustion conditions represent the long-term exposure conditions for residents and farmers. In other words, do the trial burns themselves represent long-term exposure conditions or should they be considered a worst case situation that would be infrequent?

Selecting the most appropriate concentration to use in the risk assessment depends on the number of samples collected, the variance within the data set for a particular chemical, and the confidence in the data set that the investigator needs in estimating risk. The 95th percentile concentration is calculated by the following equation:

$$\mathbf{k = p(n + 1)}$$

where:

k = 95th percentile concentration (concentration to be calculated)
p = 0.95 (or the percentile that is to be calculated)
n = number of samples

As shown by the equation, the 95th percentile is a relatively simple statistic to calculate and does not depend on the number of samples collected or the variance in the data set. That is, collecting more data (from the same population) will not reduce the 95th percentile concentration. For practical purposes, it is important to note that the 95th percentile concentration ***cannot*** be larger than the maximum detected concentration (as suggested on page 2-5).

In contrast, the 95 UCL (95th percent upper confidence limit of the mean [geometric or arithmetic]) ***is*** dependent on the number of samples and the variance (USEPA 1992). For a lognormally distributed data, the UCL is calculated with the following equation:

$$UCL = e^{(mean + 0.5s^2 + sH/(n-1)^{1/2})}$$

where:

UCL = upper confidence limit
e = constant
mean = mean concentration of transformed data
s = standard deviation of the transformed data
H = H-static (from statistical table)
n = number of samples

For a normally distributed data set, the UCL is calculated with the following equation:

$$UCL = mean + t\,(s/(n)^{1/2})$$

where:

UCL = upper confidence limit
mean = mean concentration of data set
s = standard deviation of the data set
t = Student-t statistic (from statistical table)
n = number of samples

As shown, the UCL is dependent on the number samples and the variability of the data set for both normal and lognormal data sets. That is, as the number of samples increases and variability decreases, the 95UCL approaches the mean (this was a way to "reward" PRPs for collecting more samples—the exposure point concentration would be lower). With a large number (thousands) of samples, the 95UCL is identical to the mean concentration (which is intuitive because, with an infinite number of samples, we can be quite confident of the mean concentration). However, with high variability in the data set or a few number of samples in the data set, the 95UCL can exceed the maximum detected concentration. USEPA guidance suggests that, when this happens, the maximum detected concentration should be used. For this reason, there appears to be little advantage in using the UCL based on the H-statistic method, since the data sets for trial burns are typically small. Incidentally, USEPA (1997) has shown that both UCL methods using the H-statistic are flawed and can lead to significantly overestimating the "true" 95UCL by one-to-two orders of magnitude when the data set distribution is not lognormal. New supplemental guidance for calculating the 95UCL is now being developed (personal communication with Dr. Susan Griffin, EPA Region 8).

The initial impetus for using the 95UCL in Superfund risk assessments was based on the presumption of random exposure to *soil* contamination by a receptor. The average concentration across the exposure area is thought to mirror or represent random contact with all areas of the site. However, since soil samples are not collected in the HHRAP, the concept of contact area is irrelevant and the 95UCL does not represent random contact. What appears to be important for the HHRAP instead is the *temporal* representativeness of air emissions. With a 30-year exposure duration for residential receptors, the emission rate must represent the average concentration over the entire 30-year period and lead to uniform contamination in the exposure area.

Another reason against using the 95UCL (or *de facto* maximum) concentration is that USEPA Superfund risk assessment guidance (USEPA 1989) suggests that the maximum concentration not be used in the risk assessment:

> *"The concentration term in the intake equation is the arithmetic average of the concentration that is contacted over the exposure period. Although this concentration does not reflect the maximum concentration that could be contacted at any one time, it is regarded as a reasonable estimate of the concentration likely to be contacted over time. This is because in most situations, assuming long-term contact with the maximum concentration is not reasonable."*

The problem with estimating the 95UCL emission concentration is that, from so few samples (less than 20), it is likely that it will be difficult to determine whether the data set is normal or lognormally distributed in the preliminary analysis, which is necessary to choose the correct equation (presented above).

In summary, from a practical standpoint, with just a few number of samples, the 95UCL (using the H-statistic approach) will likely exceed the maximum detected concentration for many chemicals. If the HHRAP recommends the 95UCL be used, risks will actually be calculated based on maximum concentration (when the 95UCL exceeds the maximum concentration, the maximum value is used). If this maximum concentration is derived from a trial burn where the emission source is maximized, this approach would be ultraconservative and overestimate risks (over the 30-year period). Using the 95th percentile value would at least eliminate the possibility that the risk would not be based on a resident's being exposed to the maximum concentration constantly for 24 hours per day, seven days per week, for 30 years. It may be most appropriate; however, to take into account the conditions of the trial burn to determine whether the 95th percentile concentration would also overestimate risks. For example, if

the trial burn is expected to produce maximum emissions far in excess of the long-term conditions, it may be most appropriate to use the average or 50th percentile emission concentration.

2. Is the guidance on quantifying non-detect compounds for use in the risk assessment adequate and scientifically sound. Should additional guidance be provided regarding what risk management factors are to be considered if the risk for a non-detected compound exceeds a regulatory trigger level?

No, it appears the non-detect guidance is not adequate and scientifically sound. It also differs from existing Superfund risk assessment guidance. It is unreasonable to simply assume a non-detect chemical is present in emissions. It is recommended that the section on non-detect data be expanded to suggest a careful evaluation not only of the non-detect data, but of the entire data set. The approach should be based on professional judgment only after the analytical limits are evaluated with regard to potential risk and it is determined that the chemical is present in other samples. According to USEPA (1989):

"Most analytes at a site are not positively detected in each sample collected and analyzed. Instead, for a particular chemical the data set generally will contain some samples with positive results and others with non-detected results. The non-detected results usually are reported as SQLs. These limits indicate that the chemical was not measured above certain levels, which may vary from sample to sample. The chemical may be present at a concentration just below the reported quantitation limit, or it may not be present in the sample at all (i.e., the concentration in the sample is zero). In determining the concentrations most representative of potential exposures at the site (see Chapter 6), consider the positively detected results together with the non-detected results (i.e., the SQLs). If there is reason to believe that the chemical is present in a sample at a concentration below the SQL, use one-half of the SQL as a proxy concentration. The SQL value itself can be used if there is reason to believe the concentration is closer to it than to one-half the SQL."

In other words, if the chemical is detected in at least one sample, the conventional approach (used almost without exception) is to use *one-half* the SQL. This is the compromise between positions from those who believe the chemical is not present and those who believe that the chemical is present in the sample just below the analytical limit.

For those chemicals that have not been detected in any sample, it should be assumed that the chemical is not present in the emissions. According to USEPA (1989):

> *"After considering the discussion provided in the above subsections, generally eliminate those chemicals that have not been detected in any samples of a particular medium."*

However, the risk assessor should always check to determine whether the detection limits were reasonable or low enough to detect a concentration that may pose risk (this is another useful application of the screening step recommended earlier). If the detection limit significantly exceeds a screening level, then assuming the chemical is not present would be suspect and the samples should be reanalyzed. It would not automatically be eliminated.

To summarize, when the chemical is detected in at least one sample, one-half the SQL should be used as a proxy value to calculate the 50^{th} or 95^{th} percentile emission concentration (or the concentration representing the long term exposure). When the chemical has not been detected in *any* sample, and the detection limit is at health protective levels, it should be concluded that the chemical is *not* present in the emissions and eliminated from the risk assessment.

3. Given the objectives of the HHRA and limitations associated with analyses of stack gas, is the guidance on inclusion of TOE data in the risk assessment adequate and scientifically sound?

The TOE approach appears to be scientifically untenable. It is recommended that the TOE not be used to *quantify* risks. However, the information could be useful for the uncertainty analysis and for the permitting process.

It should be noted that USEPA has taken considerable effort over many years to develop toxicity values for the chemicals of concern at most hazardous waste facilities (these are presented in the IRIS database). Analytical methods have been developed to detect these chemicals. When these methods are applied to determine the presence of these individual chemicals in emissions, the results can be used to quantify the amount of each individual chemical. To simply assume that a fraction of the TOE also contains the chemical previously analyzed and quantified seems unreasonable. Either an individual chemical has been detected or it hasn't. Once detected, it should not be

assumed to be present in another fraction. The TOE method seems to imply a double counting approach. The individual chemical is accounted for by individual analysis, but then the chemical is also assumed to be present in the TOE fraction that was somehow ignored in the analysis. The rationale is not clear. If toxic chemicals are present in the emissions, they will be individually detected through sampling for each compound.

It is unreasonable to assume that a toxic "rogue" chemical, not yet identified by USEPA, is present in the TOE fraction. It is more likely that the TOE fraction contains fairly non-toxic straight chain organic compounds.

The only case where it may be appropriate to implement the TOE approach is (1) when individual chemicals are not sampled or (2) when detection limits are too high. In this case, an individual chemical may be present in the TOE fraction, but go undetected. In this case, there may be no alternative but to assume a certain fraction of the TOE contains a toxic individual chemical presumed to be present in the emissions. This situation can be avoided by carefully implementing the sampling and analysis plan to sample for individual chemicals using appropriate detection limits.

4. Is the methodology for speciating and modeling of mercury in the risk assessment scientifically defensible?

There was insufficient time to carefully evaluate fate and transport of mercury.

5. Are the biotransfer factors scientifically defensible?

No. There appear to be two problems with the biotransfer factor assumptions. First, it is automatically assumed that the chemical taken up in plants or ingested in animals is in a "free and unaltered state" when the plant or animal is subsequently ingested by humans. When a chemical is ingested by an animal (e.g., chicken or cow), the chemical undergoes toxicokinetic changes similar to those in humans. The chemical can be absorbed and covalently bound to body tissues and organs, it can be detoxified, or it can be rapidly eliminated. It should not be automatically assumed that the chemical is bioconcentrated in those tissues in an unaltered state in those tissues later consumed by humans. Food preparation of animal products can also change the chemical structure, rendering it nontoxic.

To simply assume a chemical is in a free state ready to be totally absorbed through the gastrointestinal tract of a human is not scientifically tenable. As occurs in humans, ingested chemicals are detoxified in the liver through a "first pass" effect in animals. After detoxification in which some chemicals are made water soluble, the chemical is eliminated in the urine. The biological half-life depends on the physicochemical characteristics of the chemical.

Some lipophilic chemicals can be sequestered and bioaccumulated in fat stores. However, unless the fat is preferentially eaten in the human diet, only a small amount of lipophilic chemicals will be ingested and absorbed through the gastrointestinal tract. In other words, the chemical may accumulate in the animal, but unless the accumulating tissue or organ is ingested by the receptor, the biotransfer factor will significantly overestimate chemical intake. It is probably the oversimplification (overestimation) of biotransfer that has resulted in the conclusion stated in the HHRAP that indirect pathways pose significant risks. (If this pathway was further investigated with biological data and based on toxicokinetic principles of absorption, distribution, biotransformation, and excretion (in animals) instead of a simple "black box," where it is simply assumed that whatever chemical ingested by the animal is available, in its unaltered form, for absorption in humans, the pathway would likely be shown to be much less significant than currently thought.) As an example, when an animal eats lead-contaminated soil and then a human eats the animal, the amount of lead eaten by the animal cannot be used to estimate intake in the human. First, not all the lead eaten by the animal is absorbed by the GI tract and gets into the body. It will depend on the bioavailability of the lead. Secondly, only a small fraction of the lead absorbed by the animal will be available to the human because most of the lead will be sequestered into the bones, which are not conventionally eaten.

The other reason the biotransformation methodology may be unscientific is that it appears to violate mass balance. For example, the concentration in eggs is significantly more than the chicken is originally ingesting from soils.

6. Should ingestion of contaminated water by cows be included in the calculation of exposure concentrations in beef and milk?

There was insufficient time to carefully evaluate fate and transport through this pathway.

7. Is the selection of exposure scenario locations correct?

There is some ambiguity in the text, and the exposure areas are vaguely described. It is reasonable to assume that current exposures can be defined by current land use and zoning laws or restrictions. Chemical concentrations should be modeled for the nearest residential or agricultural property or property zoned for that use. However, this zoning would be outside the facility boundary. Modeling exposures should, therefore, be conducted at the nearest point that is currently zoned for residential or agricultural, whether or not the property is currently being used for those purposes. Current residential or agricultural exposures should not be modeled inside the operating facility.

For future land use, it may be appropriate to model chemical concentrations and risks near the source on the facility for both residential or agricultural use. Residential and agricultural development could potentially occur through rezoning of the facility after closing. However, rezoning and developing the property for residential or agricultural used would require combustion activities to cease. For future residential and agricultural exposures, it would be unreasonable to evaluate direct exposure via inhalation of emissions. The risk assessment on site could evaluate residual contamination in soils, sediments, and surface water historically deposited from the emission source.

It does not seem reasonable to simply assume a residential or agricultural receptor will be directly exposed to ongoing emissions within the facility boundary either under current or future exposure conditions.

8. Are the noncarcinogenic effects from dioxins and furans correctly presented? Is the breast feeding pathway correctly modeled?

There is no general consensus among toxicologists yet regarding carcinogenic or non-carcinogenic effects resulting from dioxins and furans. Although in the past, dioxins and furans were thought to be extremely toxic even in minute amounts, recent evidence has shown those fears to be unfounded. For example, studies conducted on Air Force personnel exposed to high concentrations of dioxin (from Agent Orange) actually exhibited an incidence of cancer (with a 20-year latency period) that was 22 percent lower than that of the general population, suggesting dioxin may actually be protective against cancer (Ketchum et al. 1999). Indeed, evidence that dioxin may actually have cancer-suppressing effects at low doses has recently been published (Calabrese *et al.*

1999). As evidenced by the withdrawal of the dioxin toxicity profile from IRIS (USEPA's only verified toxicity database), no conclusions have yet been reached on the carcinogenic potential of dioxin. Cause and effect has not yet been established for the non-carcinogenic effects. Therefore, it is premature to develop a toxicity value.

The approach in the HHRAP in which dioxin emissions are compared with background is unsupportable and does not appear to belong in the risk assessment. This information provides no information about potential health risks. This approach would be better included in the permit as a not to exceed value. Unless USEPA has supporting information to state:

> "If exposures due to the facility's emissions during the exposure duration of concern are low compared to background exposures, then the emissions are not expected to cause noncancer effects."

It would be advisable to eliminate it from the HHRAP.

Specific Recommendations:

Page 2-5, Second Paragraph

> *"The lessor of the 95th percentile or maximum stack gas concentration from the three trial burn runs should be used to develop the emission rate estimate used in the risk assessment"*

Recommendation: Delete or modify the sentence. As discussed earlier, the 95th percentile ***cannot*** be higher than the maximum concentration.

Figure 2-3, COPC Identification/Page 2-37, Step 3.

Recommendation: This figure (and accompanying text) suggests identifying non-detect chemicals, that have toxicity values, as COCs. This is unreasonable for facilities that have implemented a robust sampling and analysis program with appropriate detection limits. If the chemical is not detected, it should not be identified as a COC. A "what if" scenario can be discussed in the uncertainty section.

Page 4-4, Section 4.1.1

This section provides a brief overview of information used to evaluate future land use. It is recommended that recent USEPA guidance be included.

The development of assumptions regarding the reasonably anticipated future land use should not become an extensive, independent research project. Site managers should use existing information to the extent possible, much of which will be available from local land use planning authorities. Sources and types of information that may aid EPA in determining the reasonably anticipated future land use include, but are not limited to, the following:

5. Current land use;
6. Zoning laws;
7. Zoning maps;
8. Comprehensive community master plans;
9. Population growth patterns and projections (e.g., Bureau of Census projections)
10. Accessibility of site to existing infrastructure (e.g., transportation and public utilities);
11. Institutional controls currently in place;
12. Site location in relation to urban, residential, commercial, industrial, agricultural, and recreational areas;
13. Federal/State land use designation (Federal/State control over designated lands range from established uses for the general public, such as national parks or State recreational areas, to governmental facilities providing extensive site access restrictions, such as Department of Defense facilities);
14. Historical or recent development patterns;
15. Cultural factors (e.g., historical sites, Native American religious sites);
16. Natural resources information;
17. Potential vulnerability of groundwater to contaminants that might migrate from soil;
18. Environmental justice issues;
19. Location of on-site or nearby wetlands;
20. Proximity of site to a floodplain;
21. Proximity of site to critical habitats of endangered or threatened species;
22. Geographic and geologic information; and
23. Location of Wellhead Protection areas, recharge areas, and other areas identified in a state's Comprehensive Groundwater Protection Program.

These types of information should be considered when developing the assumptions about future land use. Interaction with the public, which includes all stakeholders affected by the site, should serve to increase the certainty in the assumptions made regarding future land use at an NPL site and increase the confidence that expectations about anticipated future land use are, in fact, reasonable.

Stephen T. Washburn

Preliminary Comments

The following comments are based on a preliminary review of EPA's July 1998 Peer Review Draft of the *Human Health Risk Assessment Protocols for Hazardous Waste Combustion Facilities* (HHRAP). This review has focused on the specific technical issues identified in a January 6, 2000 *Charge to Human Health Exposure Reviewers*, in preparation for a Peer Review Workshop scheduled for May 2000. Addenda or amendments to these comments may be prepared prior to the Workshop.

Comments were received concerning definition and use of the 95th percentile emission rate in the risk assessment (Section 2.2). Is the guidance on quantifying emission rates of compounds for use in the risk assessment adequate and scientifically sound? Should the guidance specify the use of the 95th percentile or 95th upper confidence limit (UCL) of the mean?

> In evaluating chronic health risks, it is appropriate to rely on average, long-term emission rate estimates (i.e., generally averaged over a period of at least a year). It is recognized, however, that there will be uncertainty in estimating long-term average emission rates for individual Chemicals of Potential Concern (COPCs), due to variability in emissions over time. Use of the 95 percent upper confidence limit (UCL) on the mean derived from data that are representative of long-term facility operations is a conservative way of addressing such uncertainty in the risk assessment. Thus, the 95 percent UCL on the mean is appropriate for estimating "reasonable maximum" long-term emissions, while the mean itself would be more appropriate for estimating "central tendency" long-term emissions.
>
> In estimating conservative emission rates to assess chronic health risks, the 95 percent UCL on the mean is more appropriate than the 95th percentile, because the 95th percentile is not a measure of the long-term average. Further, the proper calculation of an upper confidence limit is dependent upon the distribution of the data, and thus the HHRAP should allow for flexibility in the method to be applied in estimating the 95 percent UCL. There may also be uncertainty in calculating the 95 percent UCL when only a limited number of data points are available. As proposed in the HHRAP, use of the maximum measured emission rate for an individual COPC is appropriate if the calculated 95 percent UCL exceeds the maximum.
>
> In the HHRAP, U.S. EPA proposes to allow the use of data collected during either a trial burn or a risk burn in conducting a risk assessment. The risk burn is intended to provide data

reflecting long-term normal operating conditions, and thus is generally more appropriate than the trial burn as a foundation for the risk assessment. However, the HHRAP specifies that the risk burn should be conducted with the "worst case" waste handled by the combustion unit (Section 2.2.1.2, p.2-7). This requirement could result in a significant overestimate of long-term average emissions if the "worst case" waste represents only a relatively small fraction of the total annual throughput for the combustion unit. (This conservative approach is compounded when the 95 percent UCL of emission data for the "worst case" waste is calculated and used in the risk assessment). U.S EPA should allow for a consideration of both the composition and the annual throughput of various waste streams in determining which waste stream(s) should be tested in the risk burn.

Comments were received regarding guidance presented for quantifying non-detect compounds when estimating stack emission rates (Section 2.4). Is the guidance on quantifying non-detect compounds for use in the risk assessment adequate and scientifically sound? Should additional guidance be provided regarding what risk management factors are to be considered if the risk for a non-detected compound exceeds a regulatory trigger level?

Section 2.4 of the HHRAP provides a good overview of the different types detection limits, and their derivation.

According to Figure 2-3 of the HHRAP, compounds that are not detected in any sampling event for a combustion unit (i.e., "non-detect compounds") must still be included in the risk assessment if the compound: 1) is present in the waste being burned; 2) has a high potential to be emitted as a PIC; or 3) is of concern due to site-specific factors. If this approach is ultimately adopted, it is recommended that non-detect compounds included in the risk assessment be addressed only in the uncertainty section of the risk assessment report, rather than included in the base case risk estimates. Furthermore, additional guidance should be provided on how to interpret a result where the risk for a non-detect compound exceeds a regulatory trigger level, or causes such a trigger to be exceeded.

If non-detect compounds are included in the risk assessment, the concentrations considered in the uncertainty section should be based on the lowest detection limit not expected to result in a significant possibility of "false negatives" (i.e., not expected to indicate a compound is not present above the detection limit, when it actually is present above the detection limit). According to Section 2.4.2 of the HHRAP, the method detection limit (MDL) "has only a 1 percent chance the detects will be misidentified as negative, when the compound of concern was present" (p. 2-80). If a compound is not detected above the MDL, then the MDL (or

possibly half of the MDL) should be used in the risk assessment. There does not appear to be a good rationale for using the higher reliable detection limit (RDL) or estimated detection limit (EDL), as proposed in Section 2.4.4 of the HHRAP, if the compound has not been detected above the MDL.

There may be instances when use of the MDL is not possible or inappropriate. For example, when interference prevents a laboratory from achieving the MDL in analysis of a specific sample, the elevated sample-specific detection limit reported by the laboratory could be used.

Comments were received regarding guidance presented for inclusion of the "unknown" or unspeciated total organic emission (TOE) data when estimating stack emission rates (Section 2.2.1.3). Given the objectives of the HHRAP and limitations associated with analyses of stack gas, is the guidance on inclusion of TOE data in the risk assessment adequate and scientifically sound?

The HHRAP approach of considering TOE data in the uncertainty section of the risk assessment report, rather than including it in calculating the base case risk estimates, is appropriate. While the TOE data may be helpful in better understanding, and communicating, uncertainties in the available emission data for a facility, there is little scientific basis for calculating risks based on unspeciated TOE data.

Prorating the emission rates of identified compounds to account for unknown compounds (one of the options presented in Section 2.2.1.3 for evaluating the TOE data in the uncertainty section) would be expected to be conservative in instances where stack emissions have been characterized according to standard U.S. EPA procedures. Thus, exceeding a regulatory trigger when a TOE factor is applied should not be taken as an indication that the risks of the facility are unacceptable. Instead, closer scrutiny of the likely composition of the uncharacterized fraction of the emissions may be warranted. For example, to the extent that the TOE data include simple aliphatic hydrocarbons, such as methane and ethane, the risks of the TOE would be lower than the risks of the more toxic compounds that are targeted in stack testing.

Comments were received regarding guidance for speciating and modeling of mercury in the risk assessment (Section 2.3.8.3; Appendix B; and Appendix C). Review and comment on the technical validity of key elements of the mercury modeling…

The HHRAP presents a considerable amount of information on mercury speciation, and the fate and transport of the different species of mercury in the environment. The key concern with

respect to mercury, and the reason that it is a focus of the HHRAP, is believed to be the potential human health and ecological risk of mercury entering surface water bodies.

A key element of the modeling in the HHRAP is the use of the Universal Soil Loss Equation (USLE) and associated equations for estimating the contribution of mercury in surface water from soil carried by storm runoff. However, the USLE provides only an approximation of long-term average soil loss rates under fairly specific conditions (e.g., sheet erosion of agricultural fields), and was not developed for use in predicting soil loads to surface water bodies. For example, the USLE does not really address where the eroded soil goes (i.e., does it reach a perennial surface water body), or how quickly it gets there. In reality, most soil entering a surface water body would likely occur during large storm events, when the flow in a river or stream is very different than under normal circumstances, rather than evenly over the period of the year as calculated using the equations in the HHRAP. The potentially conservative nature of the USLE can produce highly unlikely results. For example, in EPA Region III's risk assessment of a proposed soil incinerator at the Drake Chemical Site, mercury runoff from soils into surface water was calculated to result in an ecological risk hazard quotient (HQ) of approximately 15. However, the estimated mercury concentration in soil causing this elevated HQ was only about 0.001 mg/kg, which is well below the range of "background" mercury concentrations reported for the eastern United States (i.e., 0.01 to 3.5 mg/kg).

The time available for this review does not allow for a more complete evaluation of the mercury modeling in the HHRAP. However, at this point it is recommended that U.S. EPA focus on refining the portions of the surface water modeling that address soil carried by soil runoff, rather than on further evaluating mercury speciation issues.

Comments were received regarding the recommended determination and application of biotransfer (Ba) values (Chapter 5; Appendix A-3; and Appendix B). Considering available scientific literature, review and comment on the technical validity of guidance presented for determination and application of Ba values…

The bioconcentration factors (BCF) presented for eggs and chicken in Table 3 of Stephens et al. (1995) should be applied to the transfer of dioxins/furans from *feed*, rather than *soil*, since the fraction of feed that is soil is already incorporated in deriving the BCF values. In other words, in calculating Ba values for egg and chicken, the BCF values in Table 3 should be divided by the daily feed intake (0.2 kg DW/day), rather than the daily soil intake (0.02 kg DW/day).

Whenever possible, biotransfer factors should be derived from available chemical-specific data on uptake in livestock and vegetation, rather than from regression equations based on physical/chemical properties. The regression equation presented in Travis and Arms (1988) for estimating concentrations in vegetation based on octanol-water partition coefficient (Kow) is particularly suspect. As indicated by the data shown in Figure 3 of Travis and Arms (1988), the slope of the assumed linear relationship between the log(BCF) for vegetation and log(Kow) is very dependent upon only a few of the 29 datapoints used in developing the correlation (particularly the chemicals with the lowest and highest log(Kow) values). Furthermore, the regression equations for meat, milk and vegetation in Travis and Arms (1988) do not account for certain potentially important differences between chemicals, such as the tendency to metabolize. There is also no indication that the measured concentrations used in the regression equations represent comparable "steady-state" conditions for all chemicals. For these and other reasons, there is significant uncertainty in using the regression equations to estimate biotransfer factors for the risk assessment.

U.S. EPA should ensure that available data for directly estimating biotransfer factors for common, particularly toxic COPCs have been reviewed for use in the HHRAP. It is recognized, however, that there are gaps in the data available to estimate biotransfer factors for many chemicals in a variety of types of produce, livestock, dairy products, and other sources of food. Simply eliminating chemicals from the risk assessment because biotransfer data are not available may lead to an underestimate of risk. Thus, continued reliance on theoretical or indirect methods of estimating biotransfer factors for at least some chemicals in at least some potential sources of food would appear necessary. However, the regression equations presented in Baes et al. (1984) and Travis and Arms (1988) do not include any data that might have been published over the past decade or more, and that might lead to improved correlations. Efforts should be made to improve the regression equations using recent data. Chemicals with characteristics that are not reflected in the regression equations (such as potential for significant metabolism) should also be identified. U.S. EPA may also wish to consider presenting the results of foodchain modeling performed using biotransfer factors based on regression equations in the uncertainty section of the risk assessment only, rather than including them in the base case risk estimates.

Given their derivation, it is possible that application of the biotransfer factors as outlined in the HHRAP could result in a situation where mass is not conserved. If risks estimated using the biotransfer factors are judged to be significant, and might affect risk management decisions at a facility, then the calculations should be checked to ensure that mass is conserved.

Comments were received regarding not including the water ingestion by cows as a potential COPC uptake mechanism (Sections 4.2 and 5.4). Should ingestion of contaminated water by cows be included in the calculation of exposure concentrations in beef and milk? If ingestion of contaminated water by cows is included as a pathway, will adjustments to the recommended Ba beef and Ba milk values be required?

> The HHRAP does not include water ingestion by animals, because the contribution of this pathway to total risk "is anticipated to be negligible in comparison with that of the other exposure pathways being evaluated" (Section 4.2, p.4-13). This approach appears to be reasonable in most instances, given the likely sources of water and expected concentrations of compounds in the water. (It is noted that the HHRAP does allow for the inclusion of the water ingestion pathway on a case-by-case basis, as warranted by site-specific exposure setting characteristics). However, the HHRAP should provide additional support for the exclusion of the water ingestion pathway if possible. Furthermore, the soil runoff into surface water pathway, which could conceivably affect the evaluation of water ingestion by livestock, should be refined as discussed in Comment 4 above.

Review and comment on guidance provided for selection of exposure scenario locations.

> The guidance provided for selection on exposure scenario locations appears to be reasonable.

Comment on the guidance for addressing the non-cancer effects from dioxins and furans (Section2.3.1.2). Is the default value for the breast milk fat intake for the breast milk pathway appropriately set, or is there new data available to suggest that different values may be more appropriate? Is there enough scientific evidence to show that depletion in the concentration of dioxin in breast milk over the first year of nursing should be accounted for in this pathway?

> The approach outlined in the HHRAP for addressing the non-cancer effects from dioxins and furans appears reasonable, given the lack of U.S. EPA Reference Dose (RfD) values for these compounds. It should be noted, however, that the reviewer preparing these comments is not a toxicologist.
>
> The approaches and default values for the breast milk pathway generally appear to be reasonable, given available data. However, this reviewer is not necessarily familiar with the most recent research in this area; this comment may be amended prior to the Workshop.

VI. CHEMICAL FATE AND TRANSPORT

Dr. George F. Fries

General Issues

1. Organization.

The focus of this review is on chapters 4 and 5 with emphasis on the exposure scenarios involving animal products. Generally, the descriptions of the exposure scenarios are clear and are consistent with past practice. Some terminology could be defined more precisely and specific suggestions will be made later in this review. The terms used for animal feeds should be defined and the terms should be consistent with those normally used in production agricultural. A clear description of the subsistence farmer should be provided because this class, or their children, will probably drive the assessment for many compounds. This class could be construed as individuals whose primary source of income was from off the farm and any agricultural activity was marginal. An alternative would be a large scale commercial producers who just incidentally use some of their product for personal consumption. Such factors as which feeds are used, and the likelihood of the farm resident consuming products from a combination of species would be influenced by the choice. A hypothetical construct for the subsistence farmer might be worthwhile in order to reduce confusion and misunderstanding, and to provide a degree of uniformity among assessments.

2. Accuracy of Purpose.

Generally, the purpose as stated in the Introduction reflects the scope of the methodologies of the HHRAP. It is commendable that the protocols recommend use of a reasonable - not a theoretical worst-case maximum – potential risk. In practice, the large number uncertainties and data gaps have led to use of very conservative assumptions and default values. It is probable that assessments will substantially overstate the risk, possibly by orders of magnitude. Thus, the results are more appropriately used as screening assessment than as a decision document.

3. Scientific Aspects.

The design of the protocols are generally consistent with accepted practice. There are, however, many data gaps and it is not practical to include all of possible variations in the exposure pathways. Thus, the final result of any risk assessment can cover fairly wide range of uncertainties. Provided that the results of the assessment are used as a general guide in decision-making, these gaps will not preclude making reasonable decisions.

4. Credibility of Interpretations.

The credibility of the interpretations will depend on how the results of the risk assessment are applied. As noted, the conservatism of the assumptions, and the uncertainties and gaps in the underlying data will generally overstate the risk. Thus, it is important that the results and uncertainties are properly described and characterized. The results of the assessments can only be used a general guides for decision making should not be viewed as definitive result to be used as the sole basis for making decisions.

5. Data and Methodology Gaps.

Within the exposure section of the document, the largest methodology and data gaps involve the necessity to use derived values instead of actual measurements for many of the physical parameters and transport coefficients listed in Appendix A. The seriousness of this lack of measured values depends upon several factors. Many compounds in Appendix A can be eliminated for evaluation in indirect pathways based on low toxicity, lack of occurrence, or lack of persistence. Most of the default values listed in Appendix A are maximums and it is not necessary characterize these values if risk assessment results consistently show that a compound or class of compounds do not pose a significant risk. The use of maximum values as defaults for a number of steps is a conservative procedure that poses no problems if the risk is negligible, but it could be a problem if unacceptable risks are suggested when using a series of maximum default values. Greater use could be made of research on laboratory animals to draw inferences concerning metabolism. Examples will be provided in a subsequent section.

Fugitive and upset emissions were discussed in the earlier chapters, but there appears to be no mention of these topics in the sections on indirect exposure. Is it to be inferred that indirect exposure is not considered important for these transitory releases? Or, are the transitory releases averaged over some time period like a year? A brief clarification would be useful.

The seriousness of these methodology and data gaps are functions of the degree of uncertainty that can be tolerated in the final assessment. If the risk assessment is only used as one factor of many in making a decision, the data gaps become less serious.

6. Long-term Research.

In the long-term, an effort should be made to resolve some of the uncertainties and data gaps suggested in preceding section. Priorities for long-term research should be based on the toxicity and volume of a

compound released from an incinerator. Also, as a long-term goal, there should be continuing reviews of the literature so that new information on persistence, metabolism and transport can be incorporated in a timely manner. Efforts should also be made to draw inferences concerning metabolism and bioaccumulation in farm animals from the results of research with laboratory animals.

There has been a effort in recent years to evaluate polychlorinated biphenyls (PCBs) as individual compounds rather than as Aroclor mixtures. This has been especially true for those congeners with dioxin-like activity. Serious consideration should be given to providing individual PCB congener data in Appendix A for those PCBs of greatest toxicological concern.

SPECIFIC TECHNICAL ISSUES

1. Mercury Speciation and Modeling.

Methyl mercury from aquatic sources appears to being the only important route for human exposure to mercury through indirect pathways. The speciation of mercury is based upon a reasonable interpretation of the literature. The rates of conversion of divalent mercury to methyl mercury is quite variable in many situations. The literature provided for review suggests that 20% is a conservative upper bound value for the conversion of divalent mercury to methyl mercury in a variety of natural waters. Use this 20% value will tend to maximize the predicted exposure. The modeling of mercury to the appropriate water compartments is reasonable. No evidence has been presented to indicate that divalent mercury is taken up significantly by fish. The guidance does provide the option for modifying these assumptions depending on site-specific circumstances. Overall, the conservatism in evaluating mercury exposure appears to be consistent with or less than the conservatism many of the other processes in the protocols.

2. Biotransfer Values.

A great deal of confusion has been generated by reference to soil in listing Ba_{egg} and $Ba_{chicken}$ values for dioxins and furans. The soil term should not be used in converting the BCFs of Stephens et al. (1995) to the transfer coefficients in the protocol. Technically, the normal definition of a bioconcentration factor (BCF) is the ratio of the concentration in a product or tissue divided by concentration in the diet. In Stephens et al., the intake of dioxins and furans was assumed to be solely from the soil portion of the diet. Since soil could be analyzed with fewer non-detects than the total diet, the soil analysis multiplied by 0.1 was used as the concentration in the diet rather than using an analysis of the complete mixture. Thus, the BCF in Stephens et al. is consistent with the term as it is normally used. Conversion of the Stephens et al. BCF to the biotransfer factor (BTF) as used by Travis and

Arms (1988) would be a simple matter of multiplying by the daily feed intake (0.22 kg). Soil intake is irrelevant because the BCFs and BTFs would apply to dioxins and furans in any dietary matrix. Soil intake for these compounds should be treated in the same manner as soil intake is treated for other compounds and for other species.

The prediction equations of Travis and Arms (1988) presents a number of difficulties in theory and practice. However, the same difficulties would arise in any other prediction system based on the physical properties of compounds. Some of the more serious limitations and uncertainties are discussed here and these should be addressed in the protocols or in risk assessments prepared under the protocols.

Non-applicability at high log K_{ow}. There is ample evidence to demonstrate inverse relationships between log K_{ow} and biotransfer rates when log K_{ow} is > 6 . Comprehensive data sets illustrating the inverse relationships are available for dioxins, furans and PCBs (McLachlan, 1993; Thomas et al., 1998; Fries et al., 1999). It is not known if the mechanism for the inverse relationship is log K_{ow} or some other physical property that is coincidently related to log K_{ow}. In addition to the compound classes listed here, it can be expected that the inverse relationship would also apply to other classes of compounds with log K_{ow} > 6.

Failure to account for metabolism. The Travis and Arms equations fail to predict which compounds are susceptible to metabolism. This failure has been recognized in the alteration of the metabolism factor for phthalates in the case of mammals. The PAHs are another example of the failure of the equations. Although there has been no work with farm animals, these compounds are metabolized by epoxidation, hydrolyzation and conjugation in laboratory animals (Fries, 1995). The metabolites are excreted as water soluble products and there is little storage of the parent compounds as would be predicted by the log K_{ow}'s. The data from laboratory animals should allow development of metabolism factors for PAHs and other compounds. The failure to predict metabolism is also illustrated in the case of homologous series of halogenated compounds like PCBs, dioxins, and furans. Typically, congeners with the same number of chlorines have comparable K_{ow}'s. The distribution of chlorines on the ring will then determine if the compound is metabolized. One congener may be totally metabolized, whereas another congener with the same number chlorines in a different arrangement, will not be metabolized (McLachlan, 1993; Thomas et al., 1998; Fries et al., 1999).

Equations may not apply compounds that are ionized at some pHs or have low Kow's. For example, pentachlorophenol (PCP) from its log Kow is predicted to be bioconcentrated in fat. Experiments involving cattle dosed with ^{14}C-pentachlorophenol showed little tissue or milk accumulation. Most of the administered dose was eliminated in urine as either the parent compound or

as a conjugated metabolite(Kinzell et al., 1985; Hughs et al., 1985). The concentrations in tissues were highest in liver and kidney, and were not related to fat content in other tissues. Residues in milk were both free and bound, and were predominantly contained in the water fraction. Because of these findings, one cannot predict concentrations in tissues or products of other species based on the fat content. As in the case metabolism, when compound specific information is available, it should be incorporated into Appendix A, or users should be encouraged search for this information when a compound is predicted to be unusually important in the risk assessment. Reasonable extrapolations from laboratory species should be encouraged.

The data set for derivation of tissue equations is not adequate for the intended purpose. The Travis and Arms paper indicates beef studies were at least 60 days in length. It is not possible to reach a steady state concentration in tissue in that short time period with many persistent lipophilic like many halogenated hydrocarbons. Also, some of the beef tissue values were obtained in studies with lactating cattle. If it is assumed that the fractional absorption of a compound from the gastrointestinal tract is similar in both lactating and non-lactating cattle, it follow that a smaller fraction of the intake would be deposited in tissue of the lactating cattle than in non-lactating animals. On the other hand, lactating cattle would have lower percentage of fat in their bodies than non-lactating cattle. In summary, there is a great deal of uncertainty concerning the interchangeability of tissue results between lactating and non-lactating cattle. It is also not clear from the Travis and Arms paper what value the they used for feed intake when using tissue values obtained in lactating cows. Lactating animals will consume more feed than non-lactating animals.

The approach may inappropriately assume an equilibrium or steady state condition. The approach assumes a constant level of contaminant intake, which requires a constant level of feed intake. This is reasonable in the case of milk because most studies were carried out in mid-lactation where feed consumption and productivity would be representative of the average values for the complete lactation. However, the assumption of constant feed intake causes problems in the cases of beef and pork. Almost all beef and pork is obtained from growing the animals. Feed intake is a function of the body weight. Thus, in the growing animal, feed intake increases in amounts per day as the animal grows. Similarly, the body pool size is expanding. It has been shown by modeling feed intake and body fat content in pigs from weaning to slaughter that, with a constant concentration of contaminant in the diet, the concentration of contaminant in body fat will be at a stable level for a considerable period of time before slaughter (Fries, 1996a). After the animal reaches maturity, however, concentrations would tend to increase. There would be similar expectations for beef cattle. The issue that requires some examination is whether or not the biotransfer factor as defined by Travis and Arms is the appropriate term to evaluate transfer of compounds from feed to tissue in the growing animals, which are the primary sources of beef and pork.

3. Vapor Phase Deposition.

The recommendations concerning dry vapor phase deposition to water bodies appears to be deficient in theory, but this deficiency may not be important in practice. Vapor phase transport to a water body should properly be a two-way process with volatilization from the water surfaces occurring simultaneously with deposition. Over time, the concentrations in air and water would reach an equilibrium in which contaminant flux in either direction is equal, and there would be no net flux. Rather than being expressed as a transfer rate, a partition coefficient relating the concentration in air and water would be more sound in a theory. As expressed in the equation B-4-12, deposition appears to viewed as a one-way process with the water body as a sink. Expression of transfer as a one-way process suggests the possibility that the amount of vapor transferred to water could exceed the amount vapor emitted as was discussed in the comments on transfer of vapor to plants. While not pleasing aesthetically, the result of the theoretical deficiency may not be great in practice. The issue should be examined in the same sense as it was in the comments on vapor uptake by plants. The uncertainties associated with this process may not be great compared to the uncertainties with other parts of the risk assessment process.

4. Conservation of Mass.

The failure to account for a loss of contaminants by removal of vegetation is not a serious issue. Given the shortness of the growing season and application of the interception fractions, it has been shown that more than 90% of the particulate deposition in area will ultimately be deposited in soil (Fries and Paustenbach, 1990). In addition, if plant material is removed as animal feed, a portion of the contaminant will be unabsorbed and returned to the soil with the manure applications. Comments were received concerning the apparent deposition of more material from the vapor phase on plants than was emitted from the stacks. Is not clear whether this result arose from errors in the dispersion model, or inappropriate estimation of the distribution coefficient between air and plants. As noted above for dry deposition into water, there should be an equilibrium between concentrations on plants and in air. It is not realistic that all volatile material in air can be transported to plants as the model suggests. This apparent abnormal result should be examined to determine if the error lies with the dispersion model, or with the air to plant transfer coefficients.

5. Soil Erosion.

The assumption that no contaminant is lost from soil by erosion is appropriate. The soil loss equation that is used to estimate erosion does not specify the distance that the eroded soil may have moved. Thus, the eroded soil may still be within the agricultural system that is being evaluated. In addition, soil

from outside may be transported into the area of concern. Therefore, it prudent to assume that there is no contaminant loss due to erosion.

6. Atmospheric Degradation.

A number studies have demonstrated the photodegradation of PCBs, dioxins, and other halogenated compounds. This process would be expected to occur only with compounds in the vapor phase, and it would not apply to those compounds adsorbed or incorporated in particles. Laboratory studies indicate that photodegradation processes occur in the vapor phase and that the reactions follow first order rates. The process appears to be mainly dehalogenation. Thus, photodegradation may involve a reduction of the toxicity, or in some cases, the formation of more toxic congeners. The differential degradation rates of various compounds during aerial transport may be inferred from such finding as the reduction in concentrations of furans relative to dioxins as one moves from stack emissions to environmental samples obtained at a distance from the presumed sources. Photodegradation of non-halogenated and compounds is less well characterized than halogenated compounds. It appears prudent not to consider destruction during aerial transport because of lack of experimental knowledge on the rates of photodegradation of most compounds.

OTHER ISSUES

The items discussed in this category involve issues that were not raised in the charge to reviewers. *Contributions of Air and Water to Animal Exposure.* In addition to feeds, inhalation and water consumption also might be considered routes of animal exposure to emission contaminants. However, these pathways are relatively unimportant. Inhalation contributed less than 1% of the intake of dioxins (PCDDs), furans (PCDFs), and PCBs to dairy cattle exposed to normal background levels of these compounds (McLachlan, 1993; McLachlan et al., 1990; Thomas et al., 1999). The conclusions were based on measured concentrations in air and the assumption that all of the inhaled chemicals were absorbed. It is quite reasonable that the same conclusion would apply to other species and other air-borne contaminants. The water component of oral ingestion is not an important consideration for hydrophobic compounds like PCDD/Fs and PCBs because the low solubility of these compounds limits the potential intake. Water samples collected in a mass balance studies with dairy cows had concentrations of PCDD/Fs and PCBs below the limit of detection (McLachlan, 1993; McLachlan et al., 1990). Others have concluded that water analysis was unnecessary because the low solubilities of the compounds would preclude significant intake at normal rates of water intake (Thomas et al., 1999). Compounds with lower log K_{ow}'s might be present in surface waters consumed by animals, but as noted previously, it is likely that these compounds would not be accumulated because of rapid rates of metabolism and/or urinary excretion.

Animal Feed Usage and Terminology. The terms used for cattle feed should be defined. The use of the terms "forage" and "silage" in the protocols do not appear to be consistent with the definitions of these terms as used in the animal production industry and in academic animal science. "Grain" is satisfactory, but the more usual term is "concentrate". Concentrate, in addition to a base of feed grains, will usually contain protein, vitamin, and mineral supplements that are produced off-site. A small subsistence farmer is unlikely to produce grains, or to have the grinding and mixing facilities required to produce the concentrate mix. Thus, this portion of the dairy cattle diet usually would be from off-site.

"Forage" in normal usage is applied to fibrous feeds composed primarily of plant leaves and stems. Thus, silage would be considered forage in most animal production circumstances.

"Silage" in the context of the protocols and Baes *et al*. (1984) appears to be limited to corn silage. In practice, various grasses and grass-legume mixtures are also ensiled. "Forages" as used in the protocols appears to include pasture, hay, and grass-legume silage. The distinction between forage and silage is thus based on plant type and interception factors rather than on the harvesting and storage methods, which are the appropriate basis for classifying feeds used in animal production. At a minimum the terms should be defined to eliminate confusion. The so-called forage interception factor is applicable to pasture, hay and grass-legume silage. The silage interception factor would apply only to corn silage.

Silage like the concentrate mix requires special production equipment and storage facilities. Silage, while very important commercial-scale animal production, would probably be of little importance for the subsistence producer if the subsistence producer is defined as an individual with a small number of animals whose primary source of income is off-farm. Site-specific investigation may reveal that silage does not require consideration.

Swine are not fed silage and this plant type should be removed from Section 5.5.1.2. Realistically, ground grains with the appropriate protein, mineral, and vitamin supplements are the only feeds used for swine and poultry. As noted for concentrates in cattle, it is likely that these feeds would be obtained from a source off-site for most small operators.

Feed Intake.

Questions have been raised about the discrepancy in values used for feed intake of various animal species. These discrepancies have arisen because values that were reported in individual research studies were selected for application on a more general basis. Feed intake is a complex function that

involves animal size, growth rate, productivity (milk or eggs), energy density of the feed, and ambient temperature (National Research Council, 1987). It might be useful to establish "standard animals" for each species. This would insure uniformity in the factors used for converting data in the literature to the form used in the assessment. The National Research Council publication provides a comprehensive review the feed intake literature for all species of interest, and it should be considered the most authoritative document on this subject.

Soil Ingestion.

Several points concerning soil ingestion are in order. The soil ingestion values presented in the protocols should be considered maximum values. In practice the values may be considerably less with changes in the assumptions concerning site specific conditions, and the normal range of animal management practices. Some of the limitations and qualifiers of the values for individual species values are listed.

Beef cattle: The 8% intake is a yearly average value that was derived in from cattle on arid western ranges and from dairy cattle in New Zealand (Fries and Paustenbach, 1990). The animals were offered no feed other than pasture. The amount of soil ingested was strongly related to the amount of standing forage available. If animals are offered supplementary feed (grain, hay, or silage), soil as a fraction to dry matter intake will be greatly reduced. Year-round grazing with no supplementary feed would not be realistic in many areas of the United States..

Dairy cattle: The 2% soil intake value for dairy cattle was derived from the New Zealand studies in which supplementary feed was offered to the cows (Fries and Paustenbach, 1990). It must be emphasized, however, that this value only applies to the time that the animals are on pasture. If climatic conditions prevent year-round grazing, a lower value applies. It should be noted that few commercial dairy cows have access to pasture in the United States.

Swine: The 8 % value for soil intake was derived from single study (Fries *et al.*, 1982). This was the maximum value in the study in which the range of range of values was from 3 to 8%. Unlike cattle, soil ingestion by swine is not an adjunct of grazing. It is the biological nature of swine to dig (root) in soil in search edible roots and tubers. Soil ingestion is incidental to this activity. Swine confined indoors, or on a concrete slab without access to soil, would have the no soil intake.

Poultry: There is no direct experimental work concerning soil intake of poultry. The 10% value used by Stevens *et al.* (1995) was an arbitrary selection. Interestingly, however, Beyer et al. (1994) in studies of soil ingestion by wildlife found that wild turkeys ingest approximately 9% soil. Given the

behavioral similarity of turkeys and chickens, this finding supports the 10% value suggested for chickens. It should be noted that soil ingestion only occurs with the so-called “free range” chickens. The value should be reduced for periods of time that the birds would not have access to soil.

Dr. Douglas Smith

The ENSR team of external peer reviewers is pleased to submit these comments to TechLaw on the U.S. EPA's Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP) and Errata. In accordance with the Charge Statement, ENSR's review team focused on the fate and transport components of the guidance. The ENSR Peer Review team consisted of the following four individuals:

- Mark Gerath
- Dr. Michael Mills
- Marcus Garcia
- Betsy Ruffle

We would also like to acknowledge a number of additional individuals who provided assistance and insight on this review, including: Dr. Ishrat Chaudhuri, Dr. Andrew Friedmann, and Dr. Ken Heim.

ENSR's approach to preparation of comments on the current draft of the HHRAP documents has necessarily focused upon those topic areas in which our group has the most experience. Over the last several years, our team members have presented several papers on the various transport processes and the technical issues that are involved in the current selection of methods (and specific default parameters) by the U.S. EPA Region 6 guidance. As early as 1994, some of our scientists participated in raising some of the specific comments that are presented in the Charge Statement as points of continuing concern.

ENSR has always supported the use of current scientific information, particularly when it has the benefit of independent confirmation or field validation. When available evidence is fragmentary, it is important to limit the consequences for the regulatory decision process, so that every decision does not have to await the next round of scientific progress. However, the decision process should also assure that decisions that are to be made include an adequate margin of safety for the affected public. It can best accomplish this latter goal by establishing a realistic risk sorting process that builds effectively upon previous knowledge, rather than treating every case as a new research project. This will ultimately allow resource allocation to resolve major sources of risk, and minimize time and resources spent resolving truly inconsequential levels of risk.

We hope that the comments provided below will be applied to improving the science behind the particular models chosen for inclusion in the present HHRAP Guidance. We also hope that this process helps to clarify which modeling areas are "not ready for prime time" in the regulatory decision arena.

General Comments

Each of the six general comments presented in the Charge Statement are addressed below.

1. Organization and Documentation:

The HHRAP procedures are generally very well and completely described. In addition to the overview of each set of equations, the accompanying appendices provide a detailed justification for the parameters selected including a review of other options.

The discussion titled "Water Bodies and Their Associated Watersheds" (Section 4.1.2), should be expanded to provide important guidance on selecting the water bodies for evaluation under the HHRAP protocols. In particular, criteria for selection of target water bodies should be specified. Priority should be given to those water bodies in which relevant risk pathways are complete (e.g., trophic level 4 fish occur and are likely to be sought by anglers) while the average rate of COPC deposition to watershed is high relative to the likely dilution available. This can present a challenge as these criteria tend to be mutually exclusive. An extensive fishery may depend on a large watershed while the highest COPC concentrations are likely to occur in small watersheds located close to the source.

Another important topic for fuller discussion is the "discretization" of the watershed into both its component land types (various types of pervious surfaces and impervious surfaces) as well as the different deposition rates to arrive at the area-weighted land surface types.

2. Alignment of Purpose and Methods:

In general, the U.S. EPA has increasingly shown its interest in improving the models that are used to support regulatory decision-making, while remaining aware of the practical limits that affect our ability to predict risk results. In 1994, an attempt was made to create a template for a "screening-level" version of a combustion facility risk assessment (U.S. EPA, Dec. 1994) that could be used to "screen out" those cases in which conservative estimates of total risks showed that a particular project was not even marginally of concern, and that further risk assessment was unnecessary. Unfortunately, that effort rushed out a somewhat flawed document that did not adequately reference its technical content, limiting its ability to be used knowledgeably by the intended audience. It did make a valiant attempt, however, to effectively address the complaints that came from both industry and state and federal permit writers that the earlier 1990 and 1993 guidance documents were too complex to apply to every facility unless a need was clearly established. The State of North Carolina recognized many of the limitations to the

1994 Screening Guidance, and volunteered a significant improvement with its own version (NC 1997). Meanwhile, the U.S. EPA, in what appears to be a conscientious effort to address the documentation limitations of the 1994 document, has also simultaneously expanded the scope to include virtually every potentially toxic chemical that had ever been measured or mentioned in connection with hazardous waste incineration.

While expanding the scope to include a longer list of chemicals, the U.S. EPA seized the opportunity to include more source types and routes of exposure. This effort was presumably to address other comments that claimed that the Screening Guidance was not sufficiently complete to characterize potential risks from major sources.

The attempt to make the HHRAP both sufficiently simple to apply to a wide range of sources with a wide range of chemicals, and yet complete enough to adequately characterize complex sources or exposure situations has led to dissatisfaction on both sides. It certainly would have been beneficial to the process if EPA had applied the proposed guidance in a test version to a number of facilities that have been previously assessed (such as in support of the MACT standard) and identified as "potentially problematic" or "below regulatory concern". Then it may have been possible to establish which fate and transport models were admittedly simplified for risk screening purposes, and which were "near-research grade", but most appropriate to use if predicted risks were high enough to warrant detailed investigation.

If the objective of applying the risk characterization model is to screen new combustion sources against either a base hypothetical index, or against each other, it will be most helpful to try to determine how this can be structured as a two (or more) step process. As the guidance stands (and states), there is nothing forbidding a facility from using the current guidance as the "screening" step, and then applying a more complete and/or precise set of algorithms to get a more realistic quantification of risk - except the extraordinary cost of doing so. However, a two-step process which returns to simpler models as a basis for initial screening, may make it easier for both the agency and the external reviewers to agree on the limits to scientific accuracy to maintain for each level of assessment. However, the agency will have to perform enough initial testing of the screening method to determine that a state-of-the-art facility that meets the latest MACT standards has a reasonable chance of passing the screening-level analysis. Otherwise, the risk screening tool has very limited value.

Since the current HHRAP guidance appears to mix both the screening goal and the comprehensive risk characterization goal in the same document, it is bound to accumulate criticisms on both grounds. In concept, it is possible to use a computer program version of the current guidance to reduce the level of effort needed for a "screening" application of the guidance. However, considerable effort is still

expended to gather and verify "site-specific" versions of parameters. The inherent uncertainty in many of the models and default parameters are large enough that it is difficult to determine which ones are best suited to improvement. For that reason, of course, the "External Peer Review" exercise currently undertaken by the agency is potentially a valuable step in attacking this latter problem.

3. Scientific Soundness:

Overall, the HHRAP represents a comprehensive presentation of methodologies and variables reflecting over a decade of interagency research dedicated to the field of risk assessment of combustor emissions. With some key exceptions noted in this set of review comments, the methods and assumptions represent a reasonable position based on scientific consensus and available data. For certain areas with large levels of uncertainty, research is already underway to develop better methods and assumptions (e.g., dioxins in eggs). The HHRAP methodology and assumptions should continue to be revised as new data become available in this broad field.

4. Credibility of Results:

In general, the high degree of documentation that accompanies the HHRAP lends it a greater degree of credibility than was given to earlier versions. In certain areas, such as the air dispersion and deposition modeling, the HHRAP largely reflects what is currently known and thus provides credible results. Certain other areas, specifically fate and transport components, such as those involving mercury in watersheds and dioxin bioaccumulation, are not as scientifically sound. Model predictions are well above levels measured in the environment. Since these pathways and COPCs are driving total risks for virtually all combustion facilities, the inherent problems with some of the fate and transport algorithms diminish the model's credibility.

5. Major Data Gaps and Limitations:

The major data gaps and limitations associated with fate and transport modeling are identified and discussed in this document. The key issues are noted below:

- mercury behavior in watersheds;
- mercury bioaccumulation in fish,
- dioxin bioaccumulation in chicken and eggs; and
- biotransfer into foodstuffs of COPCs with log K_{ow} greater than 5.0.

We believe that these data gaps are sufficiently large so that regulatory decisions should not be made without more detailed evaluations of these issues.

6. Long-Term Research:

We believe that risk assessments of this type would benefit greatly from the study of, and integration with, research being performed on fate and exposure of pesticides. In particular, the agricultural literature contains many efforts at quantifying mass balance of pesticides in various media and in adjacent locations following controlled applications. While significant portions of the existing methodology (e.g., the application of the Universal Soil Loss Equation) derive from the agricultural disciplines, much work has been done since the development of these simple algorithms. A major uncertainty and likely bias toward high exposures, in the existing methods is the efficiency of constituent runoff (contributed by the parameterization of the sediment delivery ratio, the sediment enrichment ratio, and the extremely high efficiency of runoff from impervious surfaces). This uncertainty could very likely be reduced by a careful review of the current literature on pesticide runoff from croplands. Behavior of pesticides in adjacent, uncultivated areas subject to over-spray should also be a productive topic for further review.

The risk assessment methodology should also work to integrate the recent findings, and models, of mercury fate and transport. Of particular relevance are the ongoing research efforts in south Florida as well as the work being pursued by the Electric Power Research Institute. Both of these efforts are seeking to understand the importance of atmospheric loadings, watershed processes, aquatic geochemistry, and the structure of the food chain on the accumulation of mercury in fish tissue. The efficiency of mercury runoff from the watershed to the water body should be evaluated in the context of these studies. As noted below, these issues are the most likely ones to contribute to uncertainty in assessing risks due to mercury emissions.

Specific Technical Issues

Our review has focused on the six specific technical issues contained in the Charge Statement. Based on our experience implementing the guidance at a number of sites and facility types, ENSR concurs that the specific issues identified in the Charge Statement are the principal ones affecting the technical validity and accuracy of the HHRAP. Each of these six issues is summarized below followed by a discussion regarding technical issues and recommendations for one or more alternative assumptions or approaches.

1. Review and Comment on the Technical Validity of Key Elements of Mercury Modeling.

The questions posed in the Specific Technical Issue #1 are addressed following a general discussion of the issue. Specific comments keyed to the text of the HHRAP are also provided.

General Comment:

The fate and transport of mercury in the aquatic and soil environment is very complex. Several different environmental factors can greatly affect the generation of methyl mercury and its subsequent uptake into fish (Watras et al., 1995; Grieb et al., 1990). The HHRAP protocols have attempted to develop a manageable protocol by greatly simplifying the true situation in the environment. The procedures used to estimate the runoff of mercury, its loading to water bodies, and its accumulation into fish are all greatly simplified from the real world. To take only one example, the assumption that the concentration of mercury in fish is simply and directly proportional to the concentration dissolved in water is very likely a weak one. Several studies have shown that the concentration of mercury in fish varies as strongly with the aquatic geochemistry and the nature of the food chain as with the loading of, and concentrations of, mercury to the water body (Bjornberg et al., 1988, Hill et al., 1996).

While this is appropriate for such a screening tool (especially if the analyst is encouraged to go beyond the screening approach if the predicted risks are high and/or the protocols are thought to be unrealistic in the specific setting), there are aspects of the protocol that are very likely to result in the over-prediction of fish body burden and human health risk. The human health risk assessment of mercury is appropriately driven in the HHRAP by consumption of fish containing mercury. As such, the important endpoint is the prediction of fish tissue concentration resulting from the predicted increase in mercury loading to a watershed with the introduction of a new combustion source. Runoff and erosion from the watershed, the equilibration of mercury between its geochemical forms as well as solids and water, and the bioaccumulation of mercury should all be considered in the context of human health risk from fish consumption.

Application of the HHRAP has been performed on a mercury-emitting combustion facility that has been in operation for approximately five years and has greatly overestimated the concentration of mercury in fish tissue. In fact, the HHRAP-predicted concentration was approximately a factor of ten higher than that measured in fish tissue despite the fact that the local mercury source is only a portion of the total mercury loading to the watershed. The cause of this overestimation is likely to be a combination of several factors:

- The rate of total mercury loading from the watershed is overestimated. This was clearly the case as the mass of mercury predicted to pass through the pond was in excess of the mass predicted to be deposited to the entire watershed on an annual basis. Other watersheds in the area had mass fluxes through the water of slightly less than the entire mass flux to the watershed. In either case, not only do the runoff algorithms predict very efficient mercury runoff but in one case the rate of runoff is physically impossible. Part of the explanation of the high rate of runoff is likely the method for consideration of erosion of soils containing mercury (see the discussion of erosion losses under Specific Technical Issue #5). An average seasonal rate of mercury runoff has been measured to be 33% in a number of catchments (Hurley et al., 1995), although the rate varies substantially due to storage in winter and release in spring runoff.

- The concentration of dissolved methyl mercury is likely to be overestimated. This over-estimation stems from an assumption of a fraction of methyl mercury that is too high (15% versus a more likely 9%) as well as the potential for too high a prediction of mercury concentration in water due to inclusion of bed sediment-sorbed mercury in the water column.

- The bioaccumulation factor for methyl mercury is likely to be overestimated for several types of water bodies. Selection of a trophic level 4 fish as the default is likely inappropriate for several small water bodies in which the fish consumed by humans is more likely to be trophic level 3. A mistake in the definition of the equations describing mercury bioaccumulation would also, if uncorrected, greatly overestimate mercury body burden in fish (see below).

Specific Questions:

1) The percent of divalent mercury assumed to speciate to methyl mercury in various media.

Mercury loaded to the watershed is greatly dominated by Hg^{2+}. A small proportion of mercury in soil is assumed to be converted to methyl mercury. Fifteen percent of inorganic mercury loaded to water bodies is assumed to be converted to methyl mercury. This proportion is based on a review of the methyl mercury proportion of the total mercury in various water bodies. Thus, slightly more than 15% of the mercury loaded to a water body is assumed to take the methylated form.

This approach raises two important issues:

- The assumption that 15% of mercury is methylated is at the high end of the data reviewed by the Mercury Study Report to Congress (see Table D-15). The values reported vary from 0.046 to 0.15. The later value is an "aggregate" of 22 lakes evaluated in Wisconsin. These lakes are generally small acid lakes in which the fraction of methyl mercury would be expected to be relatively high (Watras et al., 1995). A value more typical of those reported in Table D-15 would be approximately 0.09.

- The HHRAP neglects the importance of wetlands in generating methyl mercury. The literature contains numerous references (St. Louis et al., 1994; Hurley et al., 1995) to the major contribution of wetlands to water body methyl mercury. It is a relatively simple matter, similar in scope to estimating the area of impervious surface, to include the proportion of wetlands in a watershed when evaluating the loading of methyl mercury to a water body.

2) The quantitative modeling of mercury species concentrations in appropriate water body compartments.

Once in the water column, mercury is assumed to partition between three distinct compartments: dissolved in the water column, associated with suspended particles, and associated with particles in the bed of the water body. Appropriately, the HHRAP assumes that only the dissolved phase mercury is available for uptake in the aquatic food chain. This is consistent with the literature (Boethling and MacKay, 2000) as well as previous regulatory precedent. The quantification of sorption between water and suspended solids is also consistent with the literature and is generally reasonable given the simplified approach. The partition coefficients selected for mercury also appear to be reasonable ones.

Of more subtle effect are the assumed concentrations of suspended particles and the mass of bed sediment that is involved in sorption of mercury. The HHRAP recommends that these model inputs be developed on a site-specific basis and advocates for 10 mg solids/liter in the water column and for a sediment depth of 3 cm. Together with the water column depth, these two parameters specify the mass of solids available to absorb any mercury loaded to the water column. At first inspection, the 10 mg solids/liter seems relatively low for many aquatic systems. It is not uncommon for rivers to have concentrations 10 times this high or higher. Increasing the solids concentration would tend to lower the dissolved phase concentration and reduce the availability of mercury to the food chain.

More important is the depth of aquatic sediment and the assumption that it is in equilibrium with the water column. In fact, there is a good possibility that mercury associated with eroded soil will not

equilibrate with the water column but will deposit directly to the bed where it can become unavailable (Gilmour et al., 1992). It is difficult to test this hypothesis without specialized, site-specific data but given the high partition coefficients of mercury as well as its slow diffusion, it represents a likely explanation for potential over-prediction of fish body burdens.

3) The assumption that mercury bioaccumulation is dominated by uptake of methyl mercury.

The literature on mercury accumulation in the aquatic food chain supports the assumption that methyl mercury dominates the contribution of mercury to fish tissue (Grieb et al., 1990). Generally, the bioaccumulation factor (BAF) for methyl mercury is greater than the BAF for inorganic mercury by approximately a factor of ten (Boethling and MacKay, 2000). In addition, the Mercury Study Report to Congress discusses "hybrid" approaches, such as that of the Great Lakes Water Quality Initiative, that account for both methyl mercury and inorganic mercury. If one assumes a constant ratio of methyl mercury to total in solution, proper accounting for the relative contributions of the two forms can be made to capture the same outcome in a single BAF.

Having said this, it is important to track the loading of other species of mercury as they contribute to the generation of methyl mercury in aquatic systems. The HHRAP, as amended, assumes that 15% of dissolved inorganic mercury is converted to methyl mercury upon reaching the water body. As discussed above, this fraction appears to be high for several water bodies that may be of concern in the risk assessment of combustion sources.

It is important at this point to note an error in Table B-4-27 of the HHRAP:

$$C_{fish(MHg)} = C_{dw\ (Hg2+ + MHg)} * BAF_{fish(MHg).}$$

As written, this equation combines both the dissolved inorganic and methyl mercury into one pool and subjects both to the BAF appropriate for methyl mercury. This would clearly result in the overestimation of the mercury concentration in fish tissue. That this is the case is made clear by several lines of evidence in the Mercury Study Report to Congress. Most notably, the BAF for methyl mercury alone is considerably higher than the BAF accounting for the sum of inorganic and methyl mercury. The errors in the equation and accompanying text were not addressed in the Addendum to the HHRAP but should be corrected.

HHRAP p. B-197: The document estimates that approximately 2% of deposited inorganic mercury is methylated in soils. This is defined by modifying Ds (the total mercury deposition rate) by 0.98 for Ds

(Hg^{2+}) and 0.02 for Ds (MHg). Aside from any technical issues with these assumptions (see above), the terminology is confusing as it suggests that the Hg2+ to MHg conversion occurs in air. In reality, methylation occurs most commonly in aquatic sediments (Ramlal et al., 1993).

HHRAP p. B-309: As written, the second equation on this page will greatly overestimate the concentration of mercury in fish (see above). The equation should be corrected to the following:

$$C_{fish(MHg)} = C_{dw\ (MHg)} * BAF_{fish\ (MHg).}$$

The text accompanying this equation should also be corrected.

2. Modeling of Biotransfer Processes

EPA requested in particular review and comment on the technical validity of various Ba values, including the validity of using Travis and Arms (1988) for determining biotransfer values, especially for compounds with log Kow greater than 6.0. EPA requested, in particular, comments on the validity of the Ba_{egg} and $Ba_{chicken}$ for dioxins and furans.

The special review of this specific topic is motivated by the fact that, based upon the latest updates to the HHRAP model, concentrations predicted for several "high log K_{ow}" COPCs in the diet of subsistence farmers can dominate final risk results. This is especially true for the chicken and egg exposure pathway and the overall risk from dioxins and furans. This particular result differs from those of virtually every multi-pathway risk assessment performed prior to the release of the "Errata" document on the EPA Region VI Web Site in September 1999. The Stephens et al., 1995 article cited as the basis for the change in the biotransfer factor, Ba, may not have been explicitly considered in many of those studies. However, the apparent increase in importance of this particular food source is somewhat surprising. This is especially true when one considers the overall characterizations of sources of dioxins and furans from the U.S. diet. For example, the very recent paper presented by Dwain L. Winters (John Schaum, et al., 1999) at the Dioxin '99 meeting in Venice indicated the average concentrations of TCDD-TEQ for chicken were 0.11 pg/g, about 1/2 those for pork or beef and similar to the average dairy product, based on fat content, except milk and eggs, which were each listed as close to 0.03 pg/g.

Clearly, these large-scale average values can be quite different (and lower) than the maximum

concentrations which might be found in the vicinity of a source with contaminated soil. However, when the calculations include both cattle, which ingest soil as well as more heavily contaminated grasses with their diet, and chickens consuming the same soil, care should be taken to assure that the expected dose to the cattle and chickens are appropriately proportional to each other.

There are several key issues that have arisen during the review of the HHRAP modeling assumptions for the calculation of uptake from ingestion of chicken or eggs. Specifically, there are significant questions about the selection of the specific default values for Ba (chicken) and Ba(egg) for dioxin/furan congeners; and about the details of the calculation and use of the resultant Ba values in the human exposure dose calculations. Of primary concern are the following questions:

a) The meaning of the BCF measured vs. the application of the Ba for HHRAP;

b) Whether to adjust the EPA-calculated transfer coefficients by a factor of 10 to represent the 10% diet fraction for soil documented in the Stephens, et al. reference;

c) Which data set is most representative of the circumstances anticipated for the vicinity of hazardous waste combustors; and

d) Disparities in BCFs reported in the literature, including contaminated soil cases and average concentrations of dioxin/furans in the food supply.

The sections below address each of these issues.

2.1 Derivation of $Ba_{chicken}$ and BA_{egg}

The two equations that predict the tissue concentration of dioxin and furan congeners in eggs (A_{egg}) and chicken ($A_{chicken}$) are presented in Tables B-3-13 and B-3-14 of HHRAP, respectively. As previously mentioned, current default values recommended by the U.S. EPA for parameters in these equations frequently result in the calculation of unacceptable risks to the subsistence farmer due specifically to the emission of dioxins and furans. The derivation and application of default values for two of these parameters, the egg and chicken biotransfer/bioaccumulation factors (Ba_{egg} and $Ba_{chicken}$, respectively), is incorrect and results in an overestimation of dioxin and furan congener concentrations in eggs (A_{egg}) and chickens ($A_{chicken}$).

What follows is a discussion of why the current U.S. EPA derivation of default values for Baegg and

Bachicken requires revision. Although the derivations presented below were performed independently, the analysis and results closely parallel those performed separately by Dr. Lucy Frasier (1999).

The HHRAP estimation of dioxin and furan concentrations in eggs and chickens is based upon on the standard toxicological equation that expresses the COPC concentration in tissue (A) as a function of the ingested dose and the propensity of that chemical to be accumulated in the tissue. For A_{egg} and $A_{chicken}$, this equation can be written as follows:

$$A_{egg} = \text{exposure dose} \times Ba_{egg} \qquad \text{<Equation 1>}$$

and

$$A_{chicken} = \text{exposure dose} \times Ba_{chicken} \qquad \text{<Equation 2>}$$

where,

Ba_{egg} = the bioaccumulation factor for congener in eggs
$Ba_{chicken}$ = the bioaccumulation factor for congener in chickens

For the exposure dose term of these equations, the U.S. EPA makes the following assumptions (presented in Tables B-3-13 and B-3-14):

- A chicken raised by a subsistence farmer consumes a diet that consists of 90% plants and 10% soil;
- A chicken consumes 0.20 kg of plants and 0.022 kg of soil per day for a total of 0.222 kg/day;
- All dietary plants are grown on contaminated soil;
- The dose of each congener is solely from the diet;
- 100% of each congener in soil is bioavailable; and
- There is no significant difference in bioavailability from plant matter and soil.

These assumptions result in the rewriting of the exposure dose portion of equations 1 and 2 above to their present form in HHRAP:

$$A_{egg} = [(F_i \times Qp_i \times P_i) + ((Qs \times Cs \times Bs)] \times Ba_{egg} \qquad \text{<Equation 3>}$$

and

$$A_{chicken} = [(F_i \times Qp_i \times P_i) + ((Qs \times Cs \times Bs)] \times Ba_{chicken} \quad \text{<Equation 4>}$$

where,

F_i = fraction of plant type i (grain) grown on contaminated soil and ingested by the animal (default value = 1.0)
Qp_i = quantity of plant type i (grain) ingested by the animal (default value = 0.2 kg plant/day)
P_i = concentration of congener in plants (site-specific)
Qs = quantity of soil ingested by the animal (default value = 0.022 kg/day)
Cs = average congener soil concentration over exposure duration (site-specific)
Ba_{egg} = congener biotransfer factor for chicken eggs (congener-specific)
$Ba_{chicken}$ = congener biotransfer factor for chicken
Bs = ratio of the soil bioavailability to grain bioavailability, assumed = 1.0

According to a recent U.S. EPA response to comments memo supplied to the review team, default values for Ba_{egg} and $Ba_{chicken}$ were derived using the following general toxicological equation presented by Travis and Arms (1988):

$$Ba \equiv \frac{Concentration\ of\ COPC\ Tissue}{Daily\ Intake\ of\ COPC}$$

Because the Daily Intake of COPC will equal the product of the concentration of COPC in all ingested items and the daily intake of those ingested items, the Travis and Arms equation can be rewritten as follows:

$$Ba \equiv \frac{Concentration\ of\ COPC\ in\ Tissue}{Daily\ Intake\ of\ All\ Ingested\ Items \times Concentration\ of\ COPC\ in\ All\ Ingested\ Items}$$

or

$$Ba \equiv \frac{\left[\frac{Concentration\ of\ COPC\ Tissue}{Concentration\ of\ COPC\ in\ All\ Ingested\ Items}\right]}{Daily\ Intake\ of\ All\ Ingested\ Items}$$

or

$$Ba \equiv \frac{BCF}{DI_{all}} \qquad \text{<Equation 5>}$$

where,

$$BCF \equiv \frac{Concentration\ of\ COPC\ in\ Tissue}{Concentration\ of\ COPC\ in\ All\ Ingested\ Items}$$

DI_{all} = Daily Intake of All Ingested Items

According to the U.S. EPA, values for Equation 5 were obtained from a study by Stephens et. al. (1995), which measured uptake of dioxin congeners in chickens and eggs following a diet of plants (90%) and soil (10%). In selecting BCF and DI_{all} values from the Stephen's paper, it is very important to keep the same definition of "All Ingested Items" for both parameters so that Equation 5 remains algebraically sound.

- BCF: Values for the BCF part of Equation 5 are presented in Table 3 of the Stephens paper. In calculating the BCF for Table 3, Stephens and coworkers divided the concentration of congener measured in each tissue by the concentration of congener in feed and soil combined.[1] Therefore, "All Ingested Items" in Equation 5 are defined as feed and soil.

- DI_{all}: Consistent with the definition of "All Ingested Items" used in the BCF parameter, the value for DI_{all} is equal to combined feed and soil consumption.

Based on the Stephens study, Equation 5 can therefore be rewritten by replacing the terms Ba, BCF, and DI_{all} with measured units:

[1]In the Stephens study, the overall concentration of dioxin in these "All Ingested Items" (feed and soil) was equal to one-tenth of the dioxin concentration in the soil, because

- unlike that for soil, the concentration of dioxins in plants was below the detection limit (and assumed to be zero), and
- the diet consisted of 90% plants and 10% soil

$$\left(\frac{day}{kg-tissue}\right) \equiv \frac{\left[\frac{\left(\frac{mg-congener}{kg-tissue}\right)}{\left(\frac{mg-congener}{kg-feed\ and\ soil}\right)}\right]}{\left(\frac{kg-feed\ and\ soil}{day}\right)}$$ <Equation 6>

The problem with U.S. EPA's current derivation of A_{egg} and $A_{chicken}$ is that, for DI_{all}, the HHRAP defines "All Ingested Items" as soil alone (0.022 kg/day), making the definition of "All Ingested Items" in the denominator of Equation 5 inconsistent with its definition in the numerator. This inconsistency in the definition of "All Ingested Items" not only makes Equation 5 algebraically untenable, it overestimates the HHRAP predictions for A_{egg} and $A_{chicken}$. This can be demonstrated by rewriting Equation 6 using the current HHRAP definition of "All Ingested Items":

$$\left(\frac{day}{kg-tissue}\right) \equiv \frac{\left[\frac{\left(\frac{mg-congener}{kg-tissue}\right)}{\left(\frac{mg-congener}{kg-feed\ and\ soil}\right)}\right]}{\left(\frac{kg-soil}{day}\right)}$$ <Equation 7>

Careful examination of the measurement units in Equation 7 reveal that the units on the right side of the equation do not simplify to equal the units on the left side of the equation. In order to make the units in Equation 7 agree, the measurement units for DI_{all} must be corrected to equal that in Equation 6:

$$\left(\frac{kg-feed\ and\ soil}{day}\right)$$

This correction means that the DI_{all} value selected from the Stephens study will be that for feed and soil

combined (0.22 kg/day) rather than that for soil alone (0.022 kg/day) and will result in a A_{egg} or $A_{chicken}$ for any congener 10-fold lower than what the current HHRAP procedure assumes.

To demonstrate that the EPA assumes Ba's are ten-fold too high, we include a sample calculation for Ba_{egg}, directly applying results presented by Stephens et al. (1995) to Equation 5 for 2,3,4,7,8-penta-CDF:

$$Ba_{egg} = \frac{2.55\left(\frac{kg-tissue}{kg-feed\ and\ soil}\right)}{0.22\left(\frac{kg-feed\ and\ soil}{day}\right)} = 11.6\left(\frac{day}{kg-tissue}\right)$$

This value is 1/10th that of the Ba_{egg} of 116 published by the U.S. EPA in the HHRAP Errata (August 1999).

2.2 Assumptions of Linearity and Diet Composition

Unfortunately the literature is not complete enough to provide the detailed information needed to resolve this issue. There are laboratory tests on the ingestion of contaminated (and sometimes spiked) soils, such as Stephens (1995) and Petreas et al (1991), and there are measurements on chickens and eggs that may be presumed to have consumed some unknown ratio of feed (at some difficult-to-detect COPC concentration) and soil (which may have a detectable concentration of several of the important COPCs), such as Schuler et al. (1997). However, studies where both variables are measured are not available.

This data gap has led to a number of toxicological assumptions. The EPA is effectively assuming that the Stephens experiment concentrations of chicken and eggs (when compared to the concentration of the same congener in the ingested soil) produces a BCF which is linear with COPC intake. That is, if the Stephens chickens had eaten twice as much soil, they would be expected to have twice the concentration of each congener in their tissue, regardless of the concentration in the soil-and regardless of the presence or absence of the grain portion of their diets. This EPA model assumes that fasting chickens and overfed chickens would have identical BCFs and only the concentration of COPC in the soil portion of the diet is important. Given that the Stephens experiment also includes results obtained for a lower COPC concentration (closer to the maximum levels that are often calculated for the vicinity of combustion sources), and those tests produced LOWER BCFs, the assumption of linearity and

independence of diet composition appear to be unrealistic. At the least, the EPA should have given more consideration to the lower dose results. They should have also considered other literature that reflects the averages that have been measured in chickens with varied diets, since they cannot obtain the specific Ba components for each specific element in the chicken's diet. The equation is using an AVERAGE Ba, therefore its default value should be based upon a (conservatively drawn) AVERAGE Ba value. {Dr. Lucy Frasier's paper presents a number of alternatives which should be given due consideration.}

2.3 Adjustment for Soil Fraction in Experimental Diet

Another factor in the confusion may stem from Stephens explanation of the adjustment made in the stated BCF values for the fact that all of the COPC concentration in the chickens and eggs came from the experimental input in only the soil fraction of the diet. Thus the BCF reported was "adjusted" effectively to represent the "projected concentration" (expected) in the chicken or egg if the mass of COPC input were 10 x higher. That is the BCF results published would be the "expected values" if either 100% of the diet were soil contaminated at the given level, OR the chicken attained an identical rate of uptake and retention of COPC from grain as that which would occur if the COPC input came entirely from soil (i.e., 100% soil, linearly extrapolated from the 10% soil in diet experiment).

Ultimately the scientific problem with the EPA approach does not appear to be its division of the measured BCF by an intake rate, per se. The problem is that the model representation assumed for the projected concentrations in chickens for a set of dietary conditions quite different from those of the experiment.

Granted, the agency attempted to simplify the matter by stating that the experiment might only represent soil behavior, and then taking a BCF (extrapolated upward to a value assumed to occur for a soil diet of 100%, and assumed to be independent of soil concentration range) and then dividing it by the assumed soil intake rate in the entire diet. Knowing the when the Ba was next used in the HHRAP model, that it would be again multiplied inside the model by the soil intake rate, they were confident that these two intake rates would "cancel" and the concentration assumed to be in the farmers intake would be the concentration measured in the laboratory, "adjusted for the ratio of the concentration in the soil at the farm to the concentration utilized in the experiment.

2.4 Summary of Difficulties with EPA Assumptions

The difficulties outlined above boil down to several problematic assumptions, rather than a pure calculational error:

1) The highest soil concentration laboratory experiment may not be appropriate for making predictions for field situations with typical maximum soil concentration predictions are one to two orders of magnitude lower. This is especially important when other portions of the same laboratory study point out a dose-rate (soil concentration) dependence.

2) The Bioconcentration Factor, BCF, and subsequent estimate of the value of the Biotransfer factor, Ba, when evaluated for a diet with 10% soil, may not be an adequate representation of the best default value for situations in which the remaining diet is not similar to soil in its concentration, nor in its transfer characteristics.

3) The tacit assumption that all future chickens in the vicinity of combustor facilities (for the next 30 to 40 years) will be "free range chickens that consume contaminated local soils as 10% of their diets, rather than the 2% to 3% fraction that has been previously documented as more typical for chickens kept by current-day farmers may not be credible. (Major chicken and egg production farms certainly would not allow their broods to consume anything but uncontaminated grains and grit that are imported from outside the local area).

4) The percentage of grain (and total grain quantity) is assumed to have no appreciable effect upon the BCF (which is unlikely due to changes in metabolism).

5) All other studies of BCFs in poultry and eggs, which have been undertaken in more realistic exposure conditions (though sometimes less precisely documented) and produce BCFs which are more consistent with global measurement experience seem to have been ignored.

2.5 Recommendations for Ba for Chickens and Eggs

The "weight of evidence" approach should be used to establish a more credible default value for the Ba parameter until a more appropriate set of experiments that specifically address the sensitivities to dose rate and diet variation can be accomplished.

It is proposed, based upon review of the available articles (see reference list) that the default values for $Ba_{e, ch}$ should, in the interim be lowered by at least one order of magnitude to produce projections that are more consistent with global measurement experience on the ratios of concentrations typically found as maximum values in eggs and poultry vs. those found in beef and dairy products to date. Depending upon the final definitions of Ba and Ba_{egg} and their use in HHRAP, the Stephens experiment itself would support a factor of 3 to 10 reduction, and another factor of 3 to 4 can be readily justified on the basis of smaller percentages of contaminated soil comprising the typical diet of even those chickens with

access to outdoor pens or runs that qualify them as "free range" chickens.

2.6 Technical Validity of Ba Value Determination

1) Ba_{egg} values for di-n-octylphthalate, polycyclic aromatic hydrocarbons (PAHs), and hexachlorophene.

More data are available in the peer reviewed literature on the subject of biotransfer of PCDDs and PCDFs than other bioaccumulative compounds from environmental media to animal products. In fact, for chickens and eggs, there are few references beyond those cited here (and in Dr. Frasier's paper) that present data pertinent to calculating BCFs and Ba's for PCDDs and PCDFs. It is thus understandable that in the HHRAP, Baegg values for all organics (except PCDDs and PCDFs) were derived from a correlation equation developed by California Environmental Protection Agency (CEPA, 1993). Interestingly the correlation equation developed by CEPA was derived from experimental studies conducted on PCDDs and PCDFs using fat-soil and fat-diet partitioning factors in chickens and eggs. The correlation coefficient (r2) of linear regression for the CEPA algorithm is cited in the HHRAP as being 0.61. This correlation coefficient is low enough to suggest that there is considerable variability in this model for prediction of biotransfer factors for hens' eggs.

In preparing the latest HHRAP guidance the U.S. EPA has conducted extensive reviews of available state and federal regulatory guidance and of the peer reviewed literature. This would appear to indicate that the CEPA algorithm is the only method available for estimating Ba_{egg} for organic compounds. Our own quick literature review confirmed this fact.

The lack of measurement data in this area represents a large knowledge gap in the combustion risk assessment process. Due to the lack of data, it may be difficult to prove that the recommended approach for determination of Ba_{egg} is conservative (i.e., protective of human health). The potential for bioaccumulation of organic compounds with large K_{ow} values in eggs is highly uncertain.

2) Estimation of Ba_{beef} and Ba_{milk} values for highly lipophilic compounds (i.e., high log Kow compounds).

A review of the data presented by Travis and Arms (1988) for log Kow, Ba_{beef} and Ba_{milk} reveals that for both Ba_{beef} and Ba_{milk} the relationship between increasing log Kow and increasing Ba becomes highly variable. In fact, if one analyzes the relationship between log Kow and Ba for only those compounds evaluated by Travis and Arms which have a log Kow greater than 5 there is no obvious relationship between log Kow and Ba. For Ba_{beef} the correlation coefficient is 0.12 and for Ba_{milk} is

0.0008. These weak correlations indicate the relationship between log Kow and Ba may not be linear for compounds having a log Kow greater than 5. The relationship may actually be curve-linear and may even be parabolic.

This lack of linearity between Kow and Ba for compounds having a log Kow greater than approximately 6, is also supported by observations within the U.S. EPA and the peer reviewed literature. U.S. EPA (1994) states that: "Ba values for compounds with a log K_{ow} from 6.5 to 8.0 decrease with increasing Kow." This is similar to observations in aquatic environments for the relationship between log Kow and log BCF (Boethling and MacKay, 2000). Boethling and MacKay cite the work of Bintein (1993) and Meylan et al. (1999), which present data indicating an apparent breakdown in the linear correlation between BCF and Kow for chemicals with a log Kow greater than approximately 6.

Given the evidence supporting nonlinear relationships between Ba and log Kow for chemicals having a log Kow greater than approximately 6, it would appear to be inappropriate to apply the Travis and Arms (1988) algorithm to these compounds. However, if this approach is applied strictly as a screening tool it can at least be said to be conservative (i.e. protective of human health). If high log Kow compounds prove to be risk drivers through the beef and milk consumption pathways, it would be inappropriate to base risk management or regulatory decisions on such a simplified screening tool. Further research is necessary to be able to make informed decisions in this arena.

3) Ba Pork values

It may be imprecise to assume the physiological process of pigs and cows are essentially the same, and that the potential for bioaccumulation in pork only varies from that in beef based on relative fat content. However, there remains a large knowledge gap in the combustion risk assessment process due to the lack of measurement data in this area. Given the importance of pork in the diet, research efforts to improve knowledge of bioconcentration factors in pork should be included with efforts to improve data on chickens, beef, and milk. In the meantime, the EPA's proposed methods seem to be the best available.

3. Technical Validity of Model for Dry Deposition of Vapors to Water Body

The specific question references Sections 3.1.1; 3.5.1.7; and 5.7.1.2 all relate to the dry deposition transport process, but the different focus of each section requires separate comments on each.

3.1 Dry Deposition as a Function of Model Choice

The several historical generations of atmospheric transport and dispersion models are identified in Section 3.1.1 of the HHRAP. This section provides the reader of the HHRAP with an updated perspective on the long process that has been needed to develop, validate, and determine appropriate applicability of each of the many air dispersion models to the type of regulatory planning and decision-making required for the RCRA Combustor sources under present consideration.

ENSR is involved with U.S. EPA regulatory model development and planning activities, and thus we are aware of future developments that are on the "drawing boards" for continuing improvements in the air transport and dispersion modeling tools that may be used for these RCRA-regulated sources. The same models are being applied for other U.S. EPA Federal and delegated state air quality management programs under the 1990 Clean Air Act Amendments. Technically there is a real advantage in retaining the use for the HHRAP of the latest "mainstream" air dispersion models, even if they compromise to some degree the precision of their simulation of air concentrations and deposition patterns. They are the best understood set of models and that makes it easier to communicate with all interested parties about both the pluses and the minuses of the results that they are able to produce.

In the Federal Register of April 21st, the EPA announced that two newer versions of air transport and dispersion models, similar to ISCST3 are now tested and available for broader application. The first is ISC-PRIME (which includes a new refined plume downwash model, as well as the original ISCST3 (96113 version) of the complex terrain model, Complex I). This model version still does not address dry deposition of vapor, a problem that ISCST3 shares.

The second model is AERMOD. Although this model maintains much of the external file handling format of ISCST3, making it a relatively straightforward upgrade for HHRAP for the near future, it contains a largely reworked core of transport algorithms. These algorithms are still consistent with the general Gaussian model format that has worked so effectively in the regulatory applications, but it include better submodels for a number of physical processes (e.g., it includes a probability distribution function submodel to address convective conditions in a more realistic manner). AERMOD does not at this time include the new plume downwash submodel from ISCPRIME nor does it include a vapor deposition submodel. Thus each includes certain improvements over ISCST3, but neither yet has all of the features that would necessarily lead to its complete replacement of the model referenced in HHRAP. As before, the guidance portion of HHRAP has to allow for judgement in the selection of modeling tools for certain site specific cases for which the generally recommended model is not necessarily best.

The lack of a dry deposition submodel for the recommended air transport and dispersion model continues to force the choice between models to remain open to variation from the basic recommendation.

3.2 Dry Vapor Deposition Submodels

Section 3.5.1.7 describes the use of solar radiation data for dry vapor deposition submodels, such as the one used in the EPA ADOM code. ENSR agrees that the inclusion of this data would be necessary to more accurately represent the form of dry deposition that is related to the behavior of the leaf surface for those indirect pathways involving the uptake by plant leaves from airborne COPCs which are in vaporous form. This will, of course further complicate the processing of meteorological data for every site. It may be worthwhile for EPA to consider a research study in which the ADOM submodel is run under a wide variety of conditions for real or hypothetical sites in several geographic regions to develop a statistical parameterized model that is only dependent upon time of day and stability class, for example; and then use a set of "adjustment factors" for a conventional ISCST3 or successor model with a simpler deposition algorithm. In either case, some additional research is likely to be required before the dry deposition modeling question for a "plant uptake" model can be confirmed.

3.3 Adequacy of Model for Deposition to Water Body

In response to the specific question about the equation used to estimate dry deposition of vapors to a water body, our team has reviewed that equation (5-30 in the guidance) and the several related equations (e.g., 5-28, 5-38 through 5-43) and found the equations to be conceptually consistent, with no apparent errors. The question implies concern about a "one-way" transport of material into a water body. However, the formats of the related equations do not pre-suppose that all parameters have positive signs, and overall there is the conceptual possibility that the net transport of COPC material could be "out" of the water body. Since that is not the generally expected result for a long-term average, it is likely that the EPA did not consider it important to clarify sign conventions for the various equations. It may be helpful to future readers if the possibility of short-term negative fluxes of COPCs (from the surface) were recognized in the documentation of these equations.

4. Estimation of Organic Compound Concentrations in Plants

EPA requested review and comment on the equations used to estimate concentrations of some organic compounds (e.g., dioxin/furans) in plants, with particular consideration to conservation of mass of contaminants emitted from a source. EPA also requested review and comment on whether the

equations used to estimate contaminant deposition to plants and deposition to soil result in double-counting total contaminant mass concentrations.

The specific question focuses upon the potential for failing to conserve mass for the COPCs assumed to deposit upon vegetation, and whether this is due to the manner in which the guidance addresses the determination and application of Ba values. In the attached article (Smith et al., 1995) presented at DIOXIN 1995, the authors identified this concern, and presented an explanation of a potential alternative solution. The first problem with the Ba approach is that it is assumed to utilize vapor concentration and deposition rate estimates that are not corrected for upwind loss of material. That alone leads to a mass conservation error. If the key receptor is very close to the source, however, this portion of the error may not be especially significant. The larger uncertainty (biased conservatively high) is due to the fact that the "transfer rate" implied by the transfer coefficient is not necessarily bounded. It can be extrapolated from the air concentration, without consideration of physical limits to transfer imposed by meteorological (fluid flow) boundary layers, and their variation with atmospheric stability.

Lorber and Pinsky (1998), in evaluating this suggested approach, had little difficulty with the recommended approach to refining the deposition velocity, but in the case of dioxins and furans, identified a photolytic decay factor that was apparently overestimated on the basis of the existing laboratory data. In this particular case there now exists sufficient field data on dioxin/furan congeners in certain grasses to allow selection of an "adjustment" factor to be integrated into the Ba model.

At present, the refined version of the original EPA model used in the HHRAP for dioxin and furan congeners best fits the available field data. Unfortunately, there are no comparable experimental field data sets for di-n-octyl phthalate or bis-2-hexylethyl ether or the several other generally problematic organic compounds with high log K_{ow} values (>6). These field data are sorely needed to determine whether the same factor approach can be successfully applied to these high log K_{ow} COPCs.

For that reason, we believe that it is better to adopt and calibrate a "mechanistic" deposition model like that used for all other COPC deposition processes, so that the ability to extrapolate to similar, but slightly different COPCs and meteorological cases is supported.

Application of a mechanistic deposition model allows the user to consider setting upper limits on the Ba-model's rate of transfer to the surface, based upon considerations of the "resistance" behavior" of the atmospheric boundary layer. This type of model can be used within the constraints of currently available meteorological preprocessor data to perform a credibility check on maximum deposition rates that can occur under stable conditions, and slightly unstable conditions. We have not yet fully

evaluated what additional advantage might be available from a coupling of this approach with a model like AERMOD and its treatment of unstable conditions.

5. Accounting for Losses to Soil Due to Erosion

In the real world, COPC deposited on soils are subject to several simultaneous and competing processes. As outlined in Section 5 of the HHRAP, these potentially include volatilization, infiltration, degradation, runoff, and movement with eroded soil. Long-term average soil concentrations are important because they affect potential risks associated with direct and indirect exposures to soil as well as acting as a source of COPC to downstream waterbodies. The algorithms defined in the HHRAP set up two different strategies for estimating losses of COPC from soils: one for estimating the soil concentration (Cs) and the other for estimating COPC loadings to waterbodies. The later algorithms use the estimate of soil concentration (Cs) to predict the rate of COPC loading with soil erosion and runoff. Interestingly, the process used to estimate C_s neglects soil erosion as a mechanism of COPC loss. In this way, COPC is assumed to occur both in soils at their point of deposition as well as in eroded materials. The most important outcome is the over-estimation of the water body loading by over-estimation of Cs.

The justification for this omission of soil erosion as a loss term is the concern that soil eroded from a specific location may be replaced by soil (containing COPC) from "uphill" in the watershed. Such a concern has some technical merit and is the basis of the sediment delivery ratio (SD) applied to estimates of soil erosion into a waterbody. Unfortunately, application of soil erosion in the loading algorithms and not in the soil loss procedures assures that too much COPC mass will be assumed to be in the modeled system. This is especially the case for those COPC that have a high affinity for soil solids (e.g., dioxin, mercury, PAHs). In effect, COPC mass can be "double-counted" as occurring in in situ soil as well as in eroded soil. In fact, this phenomenon has been observed in applying the HHRAP protocols when the mass flux of COPC to a water body approximates or, in some cases, exceeds the mass flux to the watershed.

While the accounting of COPC loss due to erosion becomes a significant issue in the HHRAP, this may be due to an artifact of the HHRAP protocols themselves. Due to the employment of the Universal Soil Loss Equation (USLE), it appears likely that loading of sediments themselves to water bodies is over-estimated. The USLE was developed for consideration of agricultural lands (Renard et al., 1997). In many settings, such lands are a minor portion of the watershed and soil losses are likely to be much smaller. A useful check on this phenomenon is the calculation of the suspended solid concentration, implied by the combination of the annual soil loading and the water flow, for comparison to more typical measured concentrations. There are many situations when the measured concentration

is low compared to the predicted ones despite the importance of in situ processes (e.g., erosion of the bed, production of phytoplankton) in generating solids in a water body.

Despite this, three corrections for the double counting of COPC associated with eroded solids are possible:

- Redefine a separate soil concentration that accounts for erosion and that acts as input to water loading term.
- Calculate the COPC lost to the water body and subtract that quantity from mass of COPC contained in watershed soils.
- Develop an approach based on the sediment delivery ratio that would consider the area of the watershed in estimating the efficiency of sediment transport to the water body as well as loss from the soil.

Any of these approaches, alone or in combination, could be made to work as long as the following goals are met:

- COPC mass eroded to water bodies should not be assumed to simultaneously reside in undisturbed soils. The over-prediction of soil concentration results in the over-prediction of water body loading especially for hydrophobic constituents and in untilled soils.
- The potential for redeposition of soils from uphill in the watershed is accounted for so that direct soil exposure scenarios are properly conservative.

As noted above for mercury, there is a very real potential to have COPC mass in the model system exceed the mass estimated to be deposited to the ground surface. This situation should be corrected.

Specific Comments:

HHRAP p. 5-10: It is appropriate to include a conservative estimate of biotic and abiotic degradation of organic constituents in soil (ksg).

HHRAP p. 5-11: Why is SD (sediment delivery ratio) included in the soil loss term? The sediment delivery ratio is an accounting for the reduced efficiency of runoff to a downstream location from a watershed location. The erosion from the location is implied to occur but the sediment is trapped downhill prior to reaching the water body. If this is an attempt to account for subsequent redeposition on downhill locations in a watershed-aggregate fashion, this should be explained. Such consideration should lessen the concern expressed later, and the justification for setting COPC loss due to erosion (kse) to zero, that eroded soil will be replaced from uphill soils. As noted above, if eroded soils are considered to be a source of COPC to waters, the loaded mass of COPC from soils should be fairly accounted.

HHRAP p. 5-13: Again, soil losses due to runoff are accounted for in loadings to water bodies. Failure to account for those same losses from soils clearly results in "double-counting" and an overestimate of COPC exposure.

HHRAP p. 5-17, p. B-226: Volatilization losses from soils (ksv) have been reasonably well studied and several robust models have been used to estimate flux from soils. Inclusion of ksv algorithms in the Addendum to the HHRAP is appropriate. These include those used by California EPA in CALTOX as well as relatively simple algorithms presented in Lyman et al. (1982) and Thibodeaux (1996). In fact, a similar approach is used by U.S. EPA and several states in setting soil clean up level guidelines. Such approaches can be reasonably parameterized to develop conservative estimates of soil losses. Failure to do so will almost certainly overestimate exposure concentrations for volatile COPCs.

Other Comments on Soil and Water Fate Algorithms:

HHRAP p. 5-64: Runoff from impervious and impervious surfaces as well as soil erosion are considered as loadings to water bodies yet are neglected as losses from soils. This is unreasonable (see above).

HHRAP p. 5-66: Use of an annual average water body temperature of 25oC is unduly high. The annual average water temperature in most parts of the country is more likely to be 10 CE to 15 CE.

HHRAP p. 5-67: Runoff loading from impervious surfaces (LRI) is assumed to occur with perfect efficiency. While this may be appropriate for surface adjacent to surface water bodies, runoff from more distance surfaces are likely to be considerably less efficient. An adjustment for large watersheds, similar to that applied to sediment transport, should be considered.

HHRAP p. 5-75: The overall water body dissipation rate (kwt) could include losses due to biotic and abiotic degradation. While, due to the short residence times implied by the calculations, it may be reasonable to neglect degradation in the water column, it is less appropriate in aquatic sediments where residence times can be significant and losses due to degradation have been documented.

HHRAP p. 5-83: Consideration of the COPC concentration dissolved in water is appropriate given the importance of the freely dissolved constituent to models of both aquatic toxicology and food chain accumulation. Consideration should be given to normalizing the concentration of TSS by its organic carbon content as well as the dissolved organic carbon content. Both of these parameters have been found to be important in predicting the bioaccumulation of hydrophobic organic COPCs and are considered in U.S. EPA evaluations of that phenomenon (U.S. EPA, 1995).

HHRAP p. 5-85: The rules established for use of BCFs, BAFs, and BSAFs appear to be reasonable ones. Care should be used in establishing the specific values of these parameters.

HHRAP p. 5-89: The statement regarding the use of site-specific data is appropriate. Consideration should be given to making the statement more prominent especially with respect to mercury dynamics (e.g., critical aspects such as the fraction of methyl mercury in the water column, potentially the BAF implied by site-specific data).

HHRAP p. B-211: The sediment enrichment ratio (ER) is intended to account for the more ready erosion of light, relatively high organic soils. While this phenomenon has been observed in agriculture, it has not been demonstrated at the low concentrations and diverse watersheds likely to be of interest to the HHRAP. More support should be provided for an estimate of ER of 3 for organic compounds. This might include review of the agricultural literature or a comparison of COPC concentrations on in situ soils versus those being eroded.

HHRAP p. B-212: Inspection of the listed Kd_s suggests that they are generally well considered both for organic and inorganic COPCs.

HHRAP p. B-251: The HHRAP separately considers diffusional loading to the water body and volatilization losses from the water body. Consideration should be given to collapsing these two approaches to estimate their net effect given COPC concentrations in air and water.

HHRAP p. B-256: The USLE cover management factor (C) can be a very important aspect of the estimation of erosion loading to a waterbody (Renard et al., 1997). Application of a C value of 0.1 for an entire watershed is much more appropriate than higher values especially as most row crops are estimated to justify a C of 0.1. Higher values are likely to be overly conservative.

6. Contaminant Mass Losses due to Atmospheric Degradation

6.1 Issues of Mass Balance

The HHRAP makes the assumption vapor or particulate matter is deposited directly to soil. The HHRAP model also includes the process that often strongly affects the food chain fate and transport the dry and we deposition onto plant surfaces. For the beef, dairy cow and pork pathways COPCs falling onto leaves is modeled, but it is also assumed that the same concentration of airborne COPC is susceptible to deposition directly onto soil at every location. When the original deposition is onto the leaves of trees, the COPC (or an organic compound containing it or a derivative) will eventually find their way to the soil through wash-off from leaves and plant decomposition. However, this may take a very long time, particularly in areas with a thick plant litter layer. The transport of compounds from this layer may be quite different than the processes of leaching and erosion considered in the protocol, since they models are generally derived from agricultural erosion models. In agricultural areas, compound migration to water bodies may also be delayed due to mixing through the soil due to plowing, but to a depth considerably larger than the 1-cm value recommended by EPA. All of these reasons suggest that the rates of transport of deposited COPCs to nearest water bodies may generally be exaggerated. The factor of two apparent for the simultaneous deposition assumed for soil and the leaves above it may not be the largest factor in this systematic overestimation.

6.2 The Temporal Issue

This issue of soil concentration is just one of the problems associated with the simplified model framework based upon the assumption of steady-state linear transport processes. In this type of model, compartments such as soil, vegetation and water bodies are filled uniformly and instantaneously. In reality, there is a wide range of transport time scales that must be considered in the risk assessment, especially if the availability of the COPC to a water body and the fish within that water body may be seasonal, rather than almost continuous.

The most rapid transport is associated with the advection, dispersion, chemical transformation and deposition (wet/dry) of air emissions. The next most rapid transport is through water bodies. The slowest transport will be through the soil leaching and erosion. In fact, compounds may be held up the soil or litter layer for years before there is a rainfall event of sufficient magnitude for their mobilization. Although the use of a watershed model to simulate this time-dependency is likely to be impractical to use for each analysis, this technique should be applied by U.S. EPA modelers to a set of several typical cases to develop an understanding of the errors associated with a "simultaneous exposure" compartment modeling approach.

One additional example of the type of comparative validation modeling for the HHRAP fate and transport models, which appears potentially useful, is the application of fugacity models. These are designed primarily to address the ultimate mass balance rather than the exact temporal behavior, but over longer time scales they provide insight as to the ultimate fractions of the total emitted mass in that period which would be expected to wind up in each environmental compartment. (See, e.g., MacKay, 1991).

6.3 Degradation Modeling

A question has arisen regarding how atmospheric degradation of compounds could be incorporated within the protocol. Since these degradation processes will be first order reactions, their effect could be simulated in the dispersion modeling by input of a pollutant half-life. If concentrations of other pollutants such as ozone affect the degradation rate, then separate model run could be performed for each season with a different pollutant half-life specified.

A very recent paper that offers promise for improving screening models for persistent compounds is that of Gouin, MacKay, Webster, and Wania (ES&T, Vol. 34, No. 5, 2000). This paper offers a systematic way to develop a set of "effective" half-lives for a wide range of organic chemicals. In addition, for those chemicals which are relatively more volatile, determination of classes of evaporation rates from the preceding work might be compared with recent experimental and modeling results of research occurring in the field of agricultural pesticides (e.g., Reichman, Wallach and Mahrer, ES&T Vol. 34, No. 7, 2000). Many of these compounds, or related COPCs will eventually find their way to a hazardous waste combustor for "safe disposal".

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