MINUTES FROM THE EPA SCIENCE ADVISORY BOARD
Risk and Technology Review Consultative Panel
Public Teleconference
December 19, 2006

PURPOSE: The Risk and Technology Review Consultative Panel of the EPA Science Advisory Board (SAB) met on December 19, 2006 via teleconference. The purpose of this consultation was to review and respond to questions concerning the Agency's proposed Risk and Technology Review (RTR) Assessment plan. As a part of the technical basis for rulemaking in the EPA's RTR effort, EPA sought input on whether its proposed assessment plan (emission data; dispersion and exposure modeling; risk characterization) is adequate to provide the basis for regulatory decisions concerning specific source categories. The Agency sought this consultation as one of several outreach efforts to identify the needs of the user community and the major relevant technical issues that should be incorporated into the update. Attachment A is the Federal Register notice announcing the meetings (71 FR 219, November 14, 2006). A meeting agenda is included as Attachment B.

LOCATION: By telephone only

DATE AND TIME: December 19, 2006 from 1:00 – 5:00 PM Eastern Time.

PARTICIPANTS: The following individuals participated in this meeting: SAB Committee and Board Members - Drs. Rogene Henderson (Chair), Timothy Buckley, Robert Schnatter, Richard Fenske, Randy Maddalena, Maria Morandi, Bryan Shaw, Mark Rood, Jeffrey Fisher and Lauren Zeise. The Consultative Panel roster is included as Attachment C and a set of biographical sketches is included in Attachment D. SAB Staff - Dr. Sue Shallal, Designated Federal Officers (DFO); EPA Presenters – Dave Guinnup, Ted Palma and Roy Smith; Other Participants – Other EPA staff and members of the public listened in to the discussions. A partial list of names is attached (Attachment E).

MEETING SUMMARY: The meeting followed the agenda (Attachment B). A summary of the meeting follows.

Convene the Meeting and Introductory Remarks **–** Dr. Suhair Shallal, Designated Federal Officer (DFO), opened the meeting at approximately 1:10 PM after allowing time for panel members to dial in to the teleconference. She reminded the members of the panel and the audience that the panel had met previously via teleconference on December 7, 2006 and that background materials including the charge questions (Attachments F), can be found on the SAB website. Preliminary comments submitted by panel members since the last teleconference have been shared with members of the response group as shown on the agenda.

Welcome - Dr. Henderson welcomed panel members and then stated that time is short and there are many question that need to be discussed. She reminded panel members that she had assigned individuals as lead discussants for each of the charge questions. She began the meeting with presentations from the public commenters and asked Dr. Sue Shallal to introduce them. Mr. Caffey Norman of Patten Boggs, LLP was the only registered public commenter. His comments

are attached (Attachment G). Dr. Buckley asked for clarification regarding the inclusion of uncertainty in the RTR II assessment methodology.

Charge Question 1

Dr. Henderson called on Dr. Morandi and Dr. Maddalena to present their comments on charge question 1. They were followed by other members of the panel who also offered comments and suggestions for improving the Agency's document.

Some of the recommendations included a sensitivity analysis should be performed on the model used and a list of caveats be included for when a risk number should be used and when it should NOT be used. The introduction should be revised to make it clearer by providing a flow chart of the process and a glossary of acronyms. It was also suggested that the authors should try to link the methodology to previous uses of the model.

Panel members commented that considering the numbers of sources that were being addressed, it is understandable that EPA's methodology focused on the easy ones first in order to finish them to meet legal requirements. However, the focus should be on source categories that have the greatest residual risk. There should also be a way to screen multipathway toxicants and focus on those that are very problematic. Finally, the rationale used to select this process should be articulated.

Charge question 2

Dr. Rood and Dr. Shaw began the discussion on charge question 2. They commented that the description of the methodology is good. The panel expressed some concern that most data were provided on a voluntary basis. It was recommended that the sources of information be clarified. The panel was agreed that an engineering review would add value, but the criteria that are used to decide what is accurate are needed. A public review of the rule is a good idea and the approach presented is reasonable for reconciling comments.

Panel members wondered if EPA has considered that NEI (National Emissions Inventory) reporting might change if it is used for regulatory purposes. A method for insuring that data are accurate and reliable should be established.

Charge Question 3

Dr. Fenske and Dr. Schnatter presented their comments on Charge questions 3. The panel stated that using the worst case scenario was protective for the general population. The proposed screening process would provide an adequate margin of safety. Panel members wondered if assumptions used in RTR III will differ from those being employed in RTR II. The panel noted that there are three subcategories of sources being created: 1) those that have no non-inhalation risk are considered not significant; 2) those that have non-inhalation risk are of limited potential; and 3) those that exceed non-inhalation risk are of significant concern. The panel was concerned with the fact that for many of the subcategories there is only one source facility on which to base a determination. The Agency could improve their explanation of the process by providing the criteria used to sort source categories into "significant" and "limited potential".

The panel stated that there are several lists being used to classify sources as persistent and/or bioaccumulative and this should be better explained. There may also be other types of chemicals included not just those that are Persistent, Bioaccumulative and Toxic (PBT).

Charge Question 4

Dr. Shaw and Dr. Rood responded to charge question 4. Panel members noted that the Agency's use of a stochastic Gaussian dispersion modeling may not be adequate if the deposition and/or chemical properties of sources are taken into consideration.

Panel members also expressed concern about the size of the modeling domain (i.e., 50 km). Meteorological data and the census data used in the model are from 1991 and 2000, respectively; it is unclear as to why these years were selected. Sensitivity analysis will be helpful to strength confidence in the output of the model.

Charge Question 5

Dr Fisher and Dr. Fenske addressed charge question 5. Panel members stated that the ramifications of using a factor of 10 to adjust emissions data to predict exposure and health risks are unclear. A more thorough explanation of the rationale for using a factor of ten to adjust emissions data is needed and may be supported by monitoring data to confirm the model predicted exposures. The panel also noted that AEGL values reflect very short exposures and may not be the most appropriate.

Charge Question 6

Dr. Maddelana and Dr. Fisher lead the discussion on Charge Question 6. Panel members suggested that census blocks be better defined, i.e., how is a workplace defined? The panel also suggested that the document should clearly articulate the limitations of calculating risks for individuals who may have previous exposures prior to entering a region of concern. There should be some discussion of indoor exposure vs ambient exposure levels. The panel noted that including short term activity patterns and the variability of short term emissions may lead to miscalculation and possibly lower the estimates of acute exposure.

Charge Question 7

Dr. Zeise and Dr. Buckley presented their comments on charge question 7. Panel members stated that for some hazardous air pollutants (HAPS) there are no unit risk estimates (URE) available. Members also noted that intermittent short-term exposures are important and should be assessed. A screening level assessment should be conducted and if a concern is found, a further dose response assessment should be conducted, e.g., an exposure screen can be conducted and if there are no exposures then further analysis is not needed.

Panel members agreed that IRIS, ATSDR, CalEPA are the appropriate data sets; however, these datasets are not entirely populated with HAPS. In addition, panel members affirmed the need for

the Agency to continue to update and to assess chemicals that are currently not assessed in the IRIS database.

Charge Question 8

Dr. Schnatter and Dr. Zeise addressed charge question 8. Panel members agreed that the methodology attempts to present an expedited approach that conserves resources and is easy to understand. The weakness of this approach is the use of a single value (slope factor / reference concentration) to characterize potency. Panel members noted that aggregating chemicals based on a similar mode of action (MOA) is appropriate. Aggregating chemicals that affect the same target organ, i.e., target specific chemicals, may not be correct. Panel members also expressed the concern that using models may bring in bias inherent to the models and doing a sensitivity analysis may help to reduce these concerns.

Charge Question 9

Dr. Buckley and Dr. Morandi presented their comments on charge question 9. The panel noted that the lack of data is the main problem. Overall, it is a good, reasonable, and scientifically sound method to protect public health. Many of the issues with how it is being conducted is associated with lack of data

The panel concluded their discussions with commending the Agency on putting forth a good process that uses information that is available in an effective manner. They supported the work of the Agency in this effort.

Panel members had the opportunity to ask clarifying questions through the presentation period and individual panel comments have been appended to these minutes. Dr. Henderson reminded panel members that final comments should be sent to her and Dr. Shallal by January 12 or 15, 2007, if extra time is needed. Dr. Shallal reiterated that panel members should include her as a recipient on any correspondence with other panel members.

The meeting adjourned at approximately 4:00 PM.

Respectfully Submitted:

/Signed/

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Dr. Suhair Shallal
Designated Federal Officer,
EPA SAB PFOA Review Panel

I certify that these minutes are accurate to the best of my knowledge:

/Signed/

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Dr. Rogene Henderson
Chair, EPA SAB RTR Consultative Panel

Appendix A	Individual Comments
Attachment A	Federal Register notice (71 FR 219, November 14, 2006)
Attachment B	Meeting agenda- December 19, 2006
Attachment C	Consultative Panel roster
Attachment D	Biographical sketches
Attachment E	List of participants
Attachment F	Charge Questions
Attachment G	Oral presentation by Mr. Caffey Norman of Patten Boggs, LLP

APPENDIX A

Comments from Dr. Timothy Buckley

1. Scope: Is the scope of the assessment appropriate for the stated purpose? Is the overall approach clearly and adequately explained for review?

The Risk and Technology Review (RTR) Assessment Plan (Draft 11/20/2006) is a well written report. The methods and plans are well organized and clearly described. The proposed risk assessment approach is appropriate for meeting the regulatory needs under 112(f)(2) for assessing source category residual risks.

7. Dose-Response Values: Is the plan for using available dose-response information (e.g., sources of information, prioritization scheme) appropriate for the purposes of this assessment? If not, can you suggest ways to improve it?

The tiered approach for selection of hazard ID and dose-response values for use in the RTR is appropriate and reasonable. The development of dose-response values is a complex and challenging process that requires distillation and synthesis of an always evolving primary literature in a systematic and transparent way. IRIS provides an appropriate 1st priority database because it contains a large number of relevant chemicals and is scientifically credible, in recent years including a process of peer review. The use of ATSDR and CalEPA dose response findings as secondary and tertiary sources appears reasonable in evolving from federal to state agencies. This section of the RTR Assessment Plan can be strengthened by more completely describing the differences between the three databases (e.g. peer review, # of chemicals, staff and resource allocation) that support priority selection. It would also be of interest to know what the overlap is across the three data sets and a general assessment of how values differ (e.g. is one more conservative than another).

The hazard identification and dose-response approach for selecting acute effects is not as clear cut in that not all HAPs have values listed and selection is individual chemical specific based on professional judgement. The criteria for selection including conceptual consistency and level of peer review are appropriate.

On pg 16 it is stated that dose-response values for chronic oral exposure were obtained from OAQPS rather than IRIS but this selection is not justified. Why wouldn't IRIS be the appropriate selection as is the case for inhalation exposure?

9. Overall: Has any important scientific information been omitted from this assessment plan that could impact a subsequent regulatory decision? In your opinion, will the overall approach for the 51 source categories provide results that will be sufficient to support regulatory decision-making in the context of EPA's residual risk program?

The report is comprehensive in its consideration of the science that underlies the RTR Assessment Plan.

The report's scientific credibility can be strengthened by including the extensive peer review literature that relates to NATA risk and ambient concentration estimates. Studies comparing/validating ambient concentration measurements to modeled estimates are particularly relevant and provide assurance of the model estimates.

The scope of the RTR II is stated to be based on practical considerations including the availability of emissions, complexity of the assessment, etc (pg 4). Although it is understandable that feasibility is considered, a stronger rationale for inclusion under RTRII would be an assessment of what source categories are believed to pose the greatest risk and therefore where will the greatest public health benefit be gained.

Section 1.2 RTR II Process could benefit from a flow chart that shows the process elements and how they relate to one another.

Comments on RTR-II Charge Questions 3&8- A.R. Schnatter
December 18, 2006

Charge question 3
Are the methods planned for selecting source categories with potentially significant ecological risks or multi-pathway human health risks for a separate, more refined ecological and multi-pathway assessment sufficiently health-protective? Are there ways that you might suggest for improving such screening techniques that can make them less conservative and still scientifically defensible?

The screening risk assessments for these compounds appear, in most cases, to be sufficiently health protective, but the selection of the compounds should be scrutinized more closely to assure that compounds and sources that may have such effects are not missed. Comments consider both the selection methods and the procedures used in the screening risk assessments.

Selection of PB compounds
The proposed method uses existing Agency tools/policies that are not intended to prioritize ecological risks associated with HAPs.

The proposed method assumes that the 14 HAPs designated as "persistent and bioaccumulative" (Appendix 5) may pose an adverse environmental effect and are thus the focus of the screening procedure proposed. This list of priority HAPs were selected based on previous EPA priority lists/policies:

- PBT profiler
- Great Waters pollutants of concern
- TRI PBT rule

Based on information provided on EPA's website, the PBT Profiler is a screening tool, and PBT estimations rendered by it are not sufficient for definitive PBT determinations. The profiler is a research rather than a regulatory tool, and is used to identify chemicals that may need further evaluation for potential Persistence, Bioaccumulation and Toxicity characteristics. The use of the PBT profile for prioritizing HAPs that pose ecological risks is can be questioned.

Pollutants of concern for the Great Waters policy were selected on the basis of available data on effects and deposition. These pollutants were known to be persistent and/or bioaccumulative and cause adverse effects in humans and the environment. All pollutants were known to occur specifically in Great Waters with atmospheric deposition being one potential source. It should be pointed out that not all the compounds listed in this policy were considered persistent and bioaccumulative (e.g. cadmium, BAP). For example, BAP, and PAHs (POMs) in general, were designated as chemicals that were not bioaccumulative chemicals of concern in EPAs Great Lakes Water Quality Initative.

The TRI PBT rule required reporting thresholds for substances designated as persistent bioaccumulative toxic (PBT) chemicals. These chemicals were judged to be of particular concern not only because they are toxic but also because they remain in the environment for long periods

of time, are not readily destroyed, and build up or accumulate in body tissue. While substances that are persistent and/or bioaccumulative will have higher exposure potential, HAPs that do not possess these attributes may still pose ecological risks.

The approach proposed by EPA also does not differentiate differences in effect endpoints that are relevant to human health versus ecological risk assessment. For example, in developing initial PB-HAP emissions thresholds for POM, facility specific emissions were converted to toxic equivalents of BAP using inhalation cancer risk estimates of 8 categories. However, cancer is not the relevant endpoint for POM's ecological effects. Rather, POM ecologic risks are based on survival, growth and reproduction due to narcosis.

An alternative approach is to develop a prioritization strategy that focuses on a relative human vs. ecological risk assessment process. For most HAPs, protection of human health will likely be protective of the environment. A transparent decision framework should be developed that (1) identifies HAPs where this is not the case (ecological risks rather than human health endpoints drive derivation of air emission thresholds) and (2) addresses relative importance of facility emission relative to background sources for naturally occurring HAPs in prioritization.

Multi-pathway screening risks

Initial review of the preliminary assessment of non-inhalation human exposure to HAPs using a multi-pathway modeling approach raises two general concerns. Firstly, there is a lack of transparency in the model algorithms used to calculate exposure via non-inhalation routes. The selection and use of particular model input parameters is unclear and undefended. Without greater transparency, it is difficult to offer a comprehensive review of the modeling aspects. Second, a simpler modeling approach may help reduce conservatism, while maintaining a high degree of health protection and scientific integrity, by focusing attention on the most important processes driving chemical fate and human exposure relative to the non-inhalation pathway.

The text and attachments of Appendix 5 do not clearly identify the model equations used to estimate human exposure by multimedia pathways. While there are a number of model parameters listed in Attachment A, a description of how these inputs are used is not included. It is also important to note that there is no explanation as to why the listed values have been selected. For example, on page 5-16, a soil mixing zone depth of 20 cm is selected, while a value of 2 cm is used in the HHRAP default. There also appears to be two separate sets of food consumption rate data (page 5-17), however, it is not indicated which (or if these) apply to adults versus children. Moreover, a listed value of 0.074 kg kg^{-1} day^{-1} for milk consumption seems unlikely (5.2 kg day^{-1} for a 70 kg adult or 1.1 kg day^{-1} for a 15 kg child).

An ambient air temperature of 11 ^{o}C is used in this analysis while the HHRAP default is 25 ^{o}C. It is not clear why this value has been used and more importantly, whether or not physical-chemical properties have been adjusted accordingly (i.e. physical-chemical properties are commonly measured at 25 ^{o}C and need to be adjusted for application at different temperatures). There is also concern regarding the selected environmental parameters representing the worst case scenario. For example, the water flow rate from the lake has been set to zero, which is probably not truly realistic.

The current approach for assessing multipathway exposure to HAPs requires a large number of model input parameters. At a screening level, the goal should be to reduce these inputs to the least number possible. One method may include the use of sensitivity analyses; which help to highlight the parameters that drive chemical exposure. By indicating the most sensitive parameters, efforts can be focused on obtaining the best estimates or measurements for these inputs. This approach can be used to reduce model conservatism. Furthermore, and maybe most useful, would be to use a multimedia fate model to help identify the environmental and physical-chemical properties for which non-inhalation exposure pathways become most important (i.e. the ratio of the intake fraction in water, food or both to the intake fraction by inhalation is greater than 1). Not only are models, such as EQC or RAIDAR more transparent and widely used and accepted, they offer simpler approaches to determine important environmental and physical-chemical parameters.

Charge question 8

8a)*What are the strengths and weaknesses of the overall conceptual approach to risk characterization planned for this assessment?*
Strengths:

1. attention is given to using validated emissions data as the basis for estimating exposure.
2. an attempt is made to provide a consistent approach for assessing risks for different sources.
3. an attempt is made at estimating the effect of uncertainties in risk characterization

Weaknesses:

1. use of a single value (e.g. slope factor or reference concentration) to characterize potency. The uncertainty in these values is not made transparent in final decision making
2. use of potency values from different agencies
3. non-transparent methods to incorporate uncertainties or variabilities into final steps of the process.

Overall, many of the techniques proposed seem most appropriate for a screening risk assessment – i.e. to produce a ranking of source categories that would require more in-depth risk assessments that account more directly for many of the scientific uncertainties inherent in use of slope factors, population estimates, exposure modelling, emission estimates, etc.

8b) Does the characterization plan adequately cover sensitive populations and early life exposures?

It is difficult to answer this question generically. For some HAP's (and therefore some source categories), the cancer slope factors or reference concentrations may have uncertainty factors or other health protective assumptions that would be expected to adequately cover sensitive sub-populations. Indeed, in some cases, the dose-response information could have been derived from these sensitive sub-populations, and therefore would cover them. There is always the possibility

that a more sensitive, unknown subgroup exists. However, in the absence of plausible scientific evidence that these subgroups exist, it would be difficult to account for their "possible" existence.

When known sensitive subgroups exist of a reasonable size, the RTR should account for this sensitivity. On page 24, the RTR-II document states that risk characterizations will assess physiologically susceptible demographic groups or life stages, if known. This is certainly appropriate and should be encouraged.

Page 24 also states that for HAP's acting by a mutagenic mode of action, extra factors of 10 (for children aged 0-1), 3 (for children aged 2-15), or 1.6 (for 70 years beginning at birth) will be applied. This is likely to be overly conservative, since cancer is primarily a disease of the elderly and the longest latent periods (e.g. for asbestos and mesothelioma) are on the order of 40 years. The extra factors proposed for "early life sensitivity" are likely to result in higher risk estimates than are truly present. They may be justified in a screening exercise, (to perhaps make them somewhat comparable to the conservative uncertainty factors used to derive RfC's), but are probably not justified in final risk assessments of source categories.

8c)Does the risk characterization plan appropriately aggregate (cancer and non-cancer) risks?

Page 25 correctly notes that non-cancer HQ's that act by similar modes of action can be aggregated. It's also noted that when this information is absent, HAP's that affect the same target organ will be aggregated to form a target organ specific hazard index. However, some HAP's are likely to act on the same target organ through different modes of action. This situation (same target organ/different MOA) is not a possibility for all unknown MOA's. Since a large number of TOSHI's could be made up of unknown, aggregated MOA's, EPA should consider balancing this conservative assumption with relaxed judgments on the evidence needed to apply a known MOA. The strategy of developing target organ specific hazard indices when an MOA is not known is better justified in the screening assessments rather than the final risk characterizations.

8d) What are the strengths and weaknesses of the planned approach for characterizing important uncertainties, variabilities and limitations? *Given the underlying science and the intended purposes of the assessment, can you suggest ways that the characterization of uncertainty and variability could be improved, made more transparent, or integrated more effectively into the risk characterization?*

This is perhaps the most important, but least clear aspect of the RTR-II assessment plan. Table 2 begins to provide a good framework for assessing various uncertainties (and variabilities) in the RTR-II plan. The outputs listed in section 4.5 include a table of "generic sources of uncertainty and variability for all source categories and for each specific source category. Both of these are strengths. While this is good practice, it is still unclear how uncertainties are factored into final decisions such as whether a new control technology is indicated for a given source, or for a given set of HAP's. This is the primary weakness.

The characterization of uncertainty and variability could be improved and made more transparent by including estimates, where possibly quantifiable in the outputs mentioned in section 4.5. EPA should consider regarding the 'first pass' of the RTR-II plan a screening assessment to rank high

priority sources and/or HAP's. Here, uncertainty could be characterized as proposed by the agency. Then, a second step could be made for higher priority sources and/or HAP's identified through the first pass. In this step, a more complete and transparent assessment of variability and uncertainty could be conducted for the sources or HAP's that are ranked high by the screening assessment.

A couple of examples of what this more complete assessment of uncertainty and variability might entail follow. In the exposure assessment area, centroid locations for receptors should be supplemented minimal and maximal distances within blocks. Exposure estimates with and without plume depletion assumptions should be made.

Another example involves dose response values. Rather than point estimates of dose response values, ranges for these should be given. For example, the IRIS URE for benzene is 2.2 to 7.8 x 10^{-6}, and within that range any calculated URE has equal scientific validity. Yet only the 7.8 value is used in risk characterization. Uncertainty in reference concentrations are thought to cover one order of magnitude. Thus a reference concentration that shows this uncertainty explicitly should be provided.

While this process may ultimately be more resource intensive, it involves a better scientific assessment, which should focus attention on the sources and/or HAP's that may be truly affecting population health.

There is one issue in the Table 2, where the magnitude and direction of uncertainties are mentioned. It is suggested that the largest uncertainty is that resulting from not considering background exposures. Yet, as the agency has stated in other places, the goal of the RTR-II plan is to estimate the additional risk from specific sources. Thus, background exposures are appropriately not included. While this could result in underestimation of total exposure, it does not result in underestimation of exposure or risk from the sources included in RTR-II. Since Table 2 includes the influence on risk estimates from sources or HAP's, "background risk" should not be included, or if it is, the influence on risk estimates (from sources) should be "mixed".

Final Comments from Dr. Jeff Fisher

Charge Question 5. Plan describes screening methods for identifying important short term exposures. Is the method protective? Can the method be refined to support acute exposure assessments?

ACUTE EXPOSURE APPROACH
Page 7, fourth paragraph, **'It will be assumed that the maximum one-hour emission rate from a source is ten times the average annual emission rate for that source.'** The factor of 10 is based on a paper by Allen et al. (2004) using VOC data. More specific information may be considered during the ANRPM comment period, especially if the screening identifies acute exposures of concern.

ACUTE EXPOSURE DATA BASE FOR TOXICITY SCREENS
Appendix 6 contains a list of AEGL-1 and AEGL-2 values, ERPG-1 and ERPG-2, MRL, and REL values.

Comments
It is unclear to me what the relationship between increasing the emission rate by a factor of ten and the resultant model predicted exposures or the health risks. The authors need to edit their text to include more information about the nature of the data they receive for use in dispersion/risk modeling and explain how they perform exposure simulations after using the 10x factor. The authors do more computational work than is described in the text of this document. My questions below reflect some of my uncertainty about what is actually carried out with the calculations.

The Allen et al. (2004) paper, as cited by the authors of this Plan, suggest that short term emissions are greater than annualized emissions by a factor of 2-9 fold. It appears that the resulting exposures can vary many fold, depending on assumptions such as weather conditions. I think a better description of the consequences of the 10x factor is needed. If this assumption holds for many source categories, this appears to be a public health protective measure. It would be better to obtain stronger justification for this phenomenon. Are there monitoring data that can support this? Is the under-prediction of short term exposures by the modeling programs a function of the assumptions or model parameters? It appears that the data used in the model is in units that do not lend itself to short term analysis? Please explain this for people who do not do this type of computational work. Why do you need to stay with hourly intervals and annualized information? Can the models be changed to predict an 8 hr exposure with 5 min increments?

The implementation of the acute exposure toxicity data base is not well defined. I think some effort is needed to determine best how to use existing data bases (Appendix 6), a priori. I have copied and pasted definitions of the toxicity values for the various data bases (see below). There are several considerations for using these data bases, such as how current is the chemical value in the data base, does the toxicity value makes sense relative to what you are doing (professional judgment) and how is the toxicity value interpreted relative to how it will be used in the Risk and Technology Review Assessment Plan. I am on the NAS AEGL subcommittee and most familiar with this program. I think the REL values, by definition, may be most useful for this situation.

These toxicity values represent hot spots from which the public may have one hour intermittent exposures, where AEGL and ERPG values represent emergency situations and one time exposure (in theory). MRL values may be useful if you are interested in longer term exposures. I am unsure if much is known about temporal aspects of atmospheric exposures. AEGL values for carcinogens contain a cancer risk calculation to ensure that the theoretical risks of cancer are not exceeded for a short term high exposure. I believe this rarely happens.

AEGLs are developed for *emergency situations and **one time exposure.*** Thus, when these values are finally approved by the NAS/AEGL subcommittee, single exposure data is much preferred over repeated exposure data.

> AEGLs represent threshold exposure limits for the general public and are applicable to emergency exposure periods ranging from 10 minutes to 8 hours. Three levels — AEGL-1, AEGL-2 and AEGL-3 — are developed for each of five exposure periods (10 and 30 minutes, 1 hour, 4 hours, and 8 hours) and are distinguished by varying degrees of severity of toxic effects. The three AEGLs are defined as follows:
>
> AEGL-1 is the airborne concentration (expressed as parts per million or milligrams per cubic meter [ppm or mg/m^3]) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic, non-sensory effects. However, the effects are not disabling and are transient and reversible upon cessation of exposure.
>
> AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape.
>
> AEGL-3 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience life-threatening health effects or death.
>
> Airborne concentrations below the AEGL-1 represent exposure levels that could produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic, non-sensory effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold levels for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to unique or idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL.

California Air Toxic Hot Spots Program (REL)

The objective is to present a method for deriving acute (one-hour) inhalation Reference Exposure Levels (RELs) for hazardous airborne substances. The acute REL is an exposure that is not likely to cause adverse effects in a human population, including sensitive subgroups, exposed to that concentration for one hour **on an intermittent basis**. These health based acute RELs are applicable to risk characterization of air releases, defined in Health and Safety Code Section 44303, as: “including actual or potential spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing of a substance into the ambient

air and that results from routine operation of a facility or that is predictable, including, but not limited to continuous and intermittent releases and predictable process upsets or leaks."

1.1.1 Definition of Reference Exposure Level (REL)
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.

ERPG

The Emergency Response Planning Guidelines (ERPGs) were developed by the ERPG committee of the American Industrial Hygiene Association. The ERPGs were developed as planning guidelines, to anticipate human adverse health effects caused by exposure to toxic chemicals. The ERPGs are three-tiered guidelines with one common denominator: a 1-hour contact duration (Figure 1). Each guideline identifies the substance, its chemical and structural properties, animal toxicology data, human experience, existing exposure guidelines, the rationale behind the selected value, and a list of references.

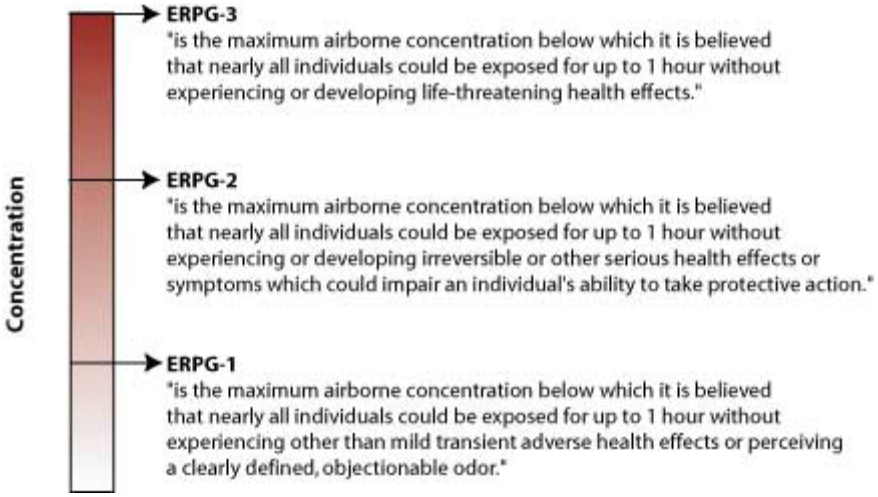


FIGURE 1. The three-tiered ERPG public exposure guidelines. The definitions and format are from the ERPG publication.

The ERPG guidelines do not protect everyone. Hypersensitive individuals would suffer adverse reactions to concentrations far below those suggested in the guidelines. In addition, ERPGs, like other exposure guidelines, are based mostly on animal studies, thus raising the question of applicability to humans. The guidelines are focused on one period of time: 1 hour. Exposure in the field may be longer or shorter. However, the ERPG committee strongly advises against trying to extrapolate ERPG values to longer periods of time.

The most important point to remember about the ERPGs is that they do not contain safety factors usually incorporated into exposure guidelines such as the TLV. Rather, they estimate how the general public would react to chemical exposure. Just below the ERPG-1, for example, most people would detect the chemical and may experience temporary mild effects. Just below the ERPG-3, on the other hand, it is estimated that the effects would be severe, although not life-threatening. The TLV, on the other hand, incorporate a safety factor into their guidelines, to prevent ill effects. **The ERPG should serve as a planning tool, not a standard to protect the public.**

MRL

MRLs are derived when ATSDR determines that reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure to the substance. MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. Inhalation MRLs are exposure concentrations expressed in units of p ATSDR uses the no observed adverse effect level/uncertainty factor (NOAEL/UF) approach to derive MRLs for hazardous substances. They are set below levels that, based on current information, might cause adverse health effects in the people most sensitive to such substance induced effects. MRLs are derived for **acute (1- 14 days)**, intermediate (>14 364 days), and chronic (365 days and longer) exposure durations, and for the oral and **inhalation** routes of exposure. ….Parts per million (ppm) for gases and volatiles, or milligrams per cubic meter (mg/m3) for particles. ATSDR does not use serious health effects (such as irreparable damage to the liver or kidneys, or birth defects) as a basis for establishing MRLs. Exposure to a level above the MRL does not mean that adverse health effects will occur.

Charge Question 6. I think the idea of using dispersion models and risk assessment procedures for technology control is an important step toward national methods and procedures. Keep up the good work! The question of providing sufficient details to refine the risk estimates by accounting for human daily activity/terrain, may go beyond the screening level. I would prefer to say, for a given location, that the emission restrictions are sufficiently protective to protect some who resides in the area of concern without leaving the region. If this is the 'worst case exposure' calculation, then this becomes a bench mark, which is probably not realistic, but provides a worst case scenario for the protection of public health. The use of population movement patterns is desirable. The text should articulate that the calculations do not account for risks achieved from living in other regions of the US or world and vise versa, when someone leave the region of concern.

Response to SAB Charge Question Two
Responses Provided by Dr. Mark J. Rood, Primary Reviewer
December 19, 2006

Overall, the "Risk and Technology Review Assessment Plan" (RTR II) is well written when discussing the emissions and source data analyses.

2. Emissions and Source Data: The NEI for hazardous air pollutants represents an ongoing voluntary national effort whose creation results from the collaborative efforts of State, local, and tribal air agencies with EPA Regional and Headquarters staff.

a. Short of creating a federal mandate for reporting emissions to the EPA, do the methods by which the NEI was developed, reviewed, and compiled result in a technically-credible database that can support regulatory assessment and action? If not, can you suggest ways to improve it?

Yes, the methods by which the NEI were developed, compiled, and reviewed will result in a technically-credible database that can support regulatory assessment and action

The report could be strengthened by describing the sources of "voluntary" data and what is done when the data are not provided for relevant sources. There is also a reference to "EPA's National Emissions Inventory (NEI) contain[ing] 2002 emissions data and source characterization information for sources of HAP emissions[3]" in the RTRII on page 4. However, reference 3 refers to a report about "industrial VOC emissions and their impact on ozone formation." It would be useful to include a brief description of the source(s) of the emission data that were used for the draft 2002 national inventory and a list of the relevant references that describe those sources, instead of referring to the 1999 NEI and the TRI as mentioned in the RTR II (page 9).

b. Do the plans for conducting an engineering review and incorporating currently-available refined emissions and source data into the inventory add value to the assessment?

Completion of a careful engineering review of the parameters used as inputs to dispersion models (e.g. existence of the sources, emission inventories, physical stack parameters, stack gas properties, temporal variability of the source strength, and location of stack with respect to plant boundary) is an important part of RTR II. The reported intent of the review is to screen the data to readily identify short-comings and problems. However, the example RTR questionnaire and summary spreadsheet, that is located in Appendix 1, refers to "looks accurate," "looks correct," and "looks reasonable." There is no guidance as to how to interpret those qualitative assessments for a quantitative emission inventory. There is also reference to significant discrepancies between two inventories (i.e. > 50%) that will initiate additional actions. It would be constructive to justify the magnitude of such difference. Inclusion of select sensitivity analyses will strengthen the report when describing criteria used to make decisions about the database.

The maximum one hour emission rate is assumed to be ten times the annual average emission rate, and such assumption is based on reference 3 (Allen et al., 2004). Short-term emissions of pollutants related to ozone formation (e.g. NOx, VOC, HRVOC, 1,3-butadiene, butene, propene, and ethene) were compared to their annual average emissions in that report. It was unusual (e.g. a few events per year) for the short-term emission rate to be greater than 10 times the annual

average emission rate for the four county region. However, the actual short-term emissions could have local impact on air quality over limited durations, but overall it appears that such short-term emissions do not add significantly impact to the *annual based* inventories *for the studied region* (four counties around Galveston TX).

The draft RTR II reports that the highest peak emission event was $\leq$ 8.5 times the annual average. However, Figures 9, 10 (i.e. 1,500 lb 1,3-butadiene/event compared to 97 lb/hr annual average), 12, and 13 appears to report events where short term emission rates were more than 10 times the annual averaged emission rate. Overall, the assumption of using 10 times the annual average for short term events appears reasonable, assuming that the acute risk assessments are applicable for a region, not a specific source.

Does the plan for soliciting public comment through an advanced notice of rulemaking add scientific credibility to the inventory?

Soliciting public comment through the advanced notice of proposed rulemaking (ANPRM) is an important aspect of the draft RTR II, especially due to the voluntary nature of the emission inventory that is used as an input to the dispersion models. Potential oversights that could occur during the development and review of the emission inventories by USEPA could be overcome by taking advantage of information provided during the public comment period, and such effort is expected to improve the scientific credibility of the emission inventory. Facilities and States that have not provided emission data could be more receptive to providing such information during the public comment period.

Is the plan for reconciling comments on the inventory adequate?

The methodology used to reconcile differences between comments provided during the ANPRN and the draft 2002 national emission inventory are described in detail in Section 2.1.3, on p. 8 of the RTR II Plan. The Plan includes descriptions of how USEPA will review the comments about the emission inventory, update the database, and provide responses from the ANPRN to the original?/new? data providers. There is reference to a detailed Quality Assurance document that describes quality assurance issues for the development of the draft 2002 national emission inventory. Such report complements the discussion about how to resolve differences between the comments obtained during the ANPRN and the national emission inventory, but it is not apparent how the quality assurance issues will be considered when updating the emission inventory based on comments from the ANPRN.

If not, can you suggest other approaches for reconciling such comments?

The approach proposed by USEPA appears to be reasonable.

Response to SAB Charge Question Four
Responses Provided by Dr. Mark J. Rood, Secondary Reviewer
December 19, 2006

4. Dispersion Modeling: Does the coupling of the AERMOD dispersion model with the census block human exposure modeling (HEM) approach to estimating individual and population exposures represent a credible approach for this goal?

AERMOD is a Gaussian based dispersion model that is very useful, but also has its limitations (e.g. estimation of plume dispersion coefficients, treatment of chemically reactive species during transport, extent of modeling domain (50 km radius), surface roughness, and plume reflection). However, I am unaware of the current status of regional or global models that could be used for the same purposes as AERMOD. Use of modeled short-term (e.g. 1 hr) exposures to estimate actual short-term exposures could provide a wide range of differences in those values. Text in the RTR II indicates that pollutant concentrations will be over-estimated for materials that exhibit transformations during transport, but such transformations should be very small during transport in AERMOD's modeling domain. Such approach does not consider reactions such as the formation of pollutants (e.g. ozone, gas to particle conversion, and oxygenated organic species). These pathways will result in elevated concentrations of pollutants when compared to the results provided by AERMOD, especially outside of AERMOD's modeling domain, or within AERMOD's modeling domain when stagnant meteorological conditions exist. Comparison of measured field results to modeled results will help to better understand how well the models predict actual conditions for HAPs.

Effort to couple AERMOD with census block human exposure modeling (HEM) appears reasonable, with sufficient precision (finest resolution is comprised of about 40 people or 10 households). However, the input meteorology is based on 1991 calendar year data and the census data are from 2000. Averaging of multiple years of meteorological data before during and after 2000 appears to be a more representative approach. The report would be strengthened to provide a few sentences for the justification of the time period used for the meteorological data compared to the census data.

Overall, the description of using AERMOD, AERMET, and HEM is well written to describe their trade-offs. The approach described in the RTR II appears credible, but the errors caused by invoking the assumptions in the models are difficult to quantify.

Are there other more credible approaches available for the estimation of inhalation risks from the types of source categories being examined?

Not to my knowledge.

Is the level of accuracy of this approach acceptable for the purposes of residual risk decision making?

I am not able to locate quantitative analyses about the accuracy of using AERMOD with AERMET, and HEM. However, there are useful discussions describing qualitative issues

that will influence the accuracy of this approach (p. 12-13). Hence, I am not able to comment on the level of accuracy of this approach as it pertains to residual risk decision making.

Are there any specific source categories, sources, or pollutants for which this approach might be considered inadequate?

Pollutants that experience chemical reactions (e.g. SO_2, photochemical reactants (e.g. NO_x, VOCs)), exhibit gas to particle conversion (e.g. H_2SO_4, NH_3), hygroscopic materials that are removed from the gas phase by clouds or precipitation (e.g. alcohols, organic acids, SO_2 and HNO_3), and are lost from the atmosphere due to dry deposition (e.g. particulate material) are examples of materials that appear to be inadequately considered by AERMOD. There does not appear to be adequate treatment of materials that experience transformations once they are deposited from the atmosphere (e.g. elemental and ionic Hg), but such multi-pathway considerations are expected to be treated in a follow-up RTR.

Comments from Dr. M. Morandi. Charge Question 1.

Charge Question 1. Scope: Is the scope of the assessment appropriate for the stated purpose? Is the overall approach clearly and adequately explained for review?

The scope of the assessment appears appropriate for the purpose of estimating source category-specific residual risks as a tool for prioritizing the Agency's rule making activities. While there are considerable uncertainties inherent in the proposed approach - some of them resulting from limitations of the underlying data (e.g., source emissions) but others deriving from the state of scientific knowledge (e.g., health impact from pollutant mixes) - unless the uncertainties impact differentially the estimates of residual risk for some source categories compared to others, the proposed assessment method will not result in misclassification of the source-specific residual risks in terms of priorities (i.e., misclassifying a source as not having a significant impact on residual risk compared to others, when it fact it does). Given the process proposed, which is conservative, it is unlikely that misclassification of this type will occur, but the Agency should undertake a sensitivity analysis to determine which inputs are the main drivers of the RR estimates and if differences in level of uncertainties for those inputs (for example, uncertainties in emissions from some sources compared to others) could potentially result in such misclassification.

There is some concern that this type of effort, as it has happened with NATA to some extent, acquires a life of its own once "number" are available to the general public and even the broader scientific community, so that they may be misinterpreted and applied for inappropriate purposes. While this is not the fault of the Agency, it is important that the Agency clearly and repeatedly states in any communication to the public that the assessment is intended only for the purpose intended by the Agency and that the estimates should not be construed as absolute estimates of residual risk for use in population-based studies. The current documentation contains caveats, but these should be listed up-front, and perhaps with some further elaboration on the limitations of the approach for uses other than the intended purpose.

The narrative of the approach is presented in a clear manner but it may be useful to add one or more diagrams with decision-making nodes clearly indicated so that it is easier to follow the text. It would also be advisable to add a statement regarding what the Agency proposes to do if and when input data may be insufficient in quantity or quality to make the estimates sufficiently reliable for a specific source type (see the comments about the potential for misclassification.) Performing the sensitivity analysis suggested above may also help in the prioritization in cases where rankings are very close.

Comments from Dr. M. Morandi: Charge question 9.

9. Overall: Has any important scientific information been omitted from this assessment plan that could impact a subsequent regulatory decision? In your opinion, will the overall approach for the 51 source categories provide results that will be sufficient to support regulatory decision-making in the context of EPA's residual risk program?

In general, the methodology incorporates current scientific understanding. One area that needs further discussion is how fugitive emissions, especially those from large facilities, will be treated and what the impact of uncertainties in the emission estimates or the identification/location of a source of fugitive emissions might have in potentially misclassifying sources or strongly underestimating exposures for some categories. It may be possible to identify some of these situations and determine if this is the case. Overall, the methodology will be useful for supporting decision making, but it is not clear at this time if that will be the case for all 51 source categories.

Comments from Dr. Randy Maddalena

Charge question #1:

Scope: Is the scope of the assessment appropriate for the stated purpose?

The scope of the assessment is stated in Section 1.1 of the RTR Assessment Plan in terms of the choice of source categories. Essentially, the scope of the RTR II will be limited to source categories that already have readily available post-MACT emission data in the most recent National Emissions Inventory (NEI) where the primary/dominant route of exposure is inhalation.

The purpose of the assessment is not stated very clearly but the last paragraph of the introduction implies that the purpose of this "new approach" is to save time and money while improving the consistency of the residual risk process and to ultimately satisfy the regulatory requirements of the CAA. This will be done by grouping together source categories and assessing the residual risks concurrently using available emissions data. If this is the "stated purpose" then the scope of the study as indicated in Section 1.1 makes a lot of sense.

Beyond the need for streamlining the residual risk process, the RTR assessment plan (and the residual risk process in general) seems to have two more overarching purposes. The first is to determine whether additional risk reductions are necessary to protect public health and the environment from industrial source categories after they have come into compliance with Maximum Achievable Control Technology (MACT). The second, if necessary, is for the RTR assessment to provide the basis for regulatory decisions concerning specific source categories. It may be that these two overarching purposes are implied by stating that the approach will satisfy the regulatory requirements of the CAA but it would be helpful if the purpose of the assessment were stated clearly stated in the introduction.

Is the overall approach clearly and adequately explained for review?

The report is well written and reasonably concise.

As indicated above, I think it would be helpful if there was a section before the current section 1.1 that clearly stated the purpose of the new approach in the context of the overall residual risk process. This is currently in the introduction but it does not come across very clearly.

Section 1.2, describes the RTR II process but it is difficult to pull out the specific steps and chronology of events/tasks. For example, the section talks a lot about the collection and evaluation of emissions data but does not talk about the screen for PBTs and multipathway HAPS, which seems to be an important part of the assessment. It would be helpful if this section were written to present each step or task in the process and identify key decision points along the way but without necessarily focusing on the details. A time-line or flow chart might be useful here.

I would like to see a list of acronyms.

In addition to the model summaries provided in the text and appendices, it might be helpful for reviewers if each of the individual models used in the assessment were linked to the relevant page in the EPA's CREM Models Knowledge Base.

FINAL COMMENTS- Richard A. Fenske, Ph.D., MPH

#3. Identifying Source Categories with Significant Non-inhalation Risk Potential: *This assessment is only designed to include source categories whose risks are dominated by the inhalation pathway. Are the methods planned for selecting source categories with potentially significant ecological risks or multi-pathway human health risks for a separate, more refined ecological and multi-pathway assessment sufficiently health-protective? Are there ways that you might suggest for improving such screening techniques that can make them less conservative and still scientifically defensible?*

The Agency indicated that it has already completed Part 1 of the screening process. This initial screening was based primarily on a "modeling assessment of emissions of 13 PB-HAPs (excluding dioxins/furans) from a hypothetical facility emitting into a domain that included a 'worst-case' subsistence receptor population, constituting a conservative exposure group." The screening process used an approach that back-calculated the emission rate of each PB-HAP that produced either a lifetime cancer risk of 1 in one million, or a hazard quotient (HQ) of 1 for non-cancer effects at the worst-case modeled receptor. The Agency has referred to this emission rate as the "threshold emission rate" for the PB-HAP.

For the estimation of ingestion exposures, in lieu of site-specific data, the Agency incorporated a health-protective farming/fishing scenario that was believed to represent individuals most exposed to HAPs emitted from the model facility. The estimated exposures were combined with dose-response values for chronic oral exposure recommended by QAQPS for screening-level risk assessments.

Since this was a screening process, the use of "worst-case" assumptions, standard factors, and scenarios would appear to provide an ample margin of safety to protect public health.

The Part 2 screening process will take place after the ANPRM, and will employ a screening version of the Total Risk Integrated Methodology (TRIM) model. The Agency indicates that this screening process will be similar to the Part 1 process, but will take advantage of an improved emission inventory, and will be using an "improved modeling platform". Presumably, this improved modeling platform is the TRIM model. The TRIM model appears to be a fate and transport model that will produce estimates of pollutant concentrations in soil, water, and biota that may be ingested.

The process described in the RTR-II document describes a later, more refined multi-pathway assessment for those source categories deemed to hold the potential for significant non-inhalation risks. However, this second-tier analysis appears to be focused primarily on refined fate and transport modeling (TRIM model). It is not clear how the Agency will refine the human exposure aspects of the estimates; e.g., the receptor population, intake rates.

The Agency should ensure that risk estimates would not be overly conservative in regard to assumptions and scenarios. It would be helpful for the Agency to provide a more detailed discussion as to whether/how it plans to modify the assumptions, standard factors, and scenarios in the second-tier analysis.

Appendix 5 refers to 51 source categories that were subjected to non-inhalation screening thresholds. However, Attachment B in the appendix lists 33 source categories, and 51 sub-categories.

Presumably the authors have applied their analysis to sub-categories rather than categories. This point should be clarified.

The criteria used to separate the five source 'categories' with "a significant number of exceedances" from the 17 source 'categories' that indicated a "limited potential" for non-inhalation risks were not clear. And again, the terminology is confusing. For example, the authors list "pulp and paper" as one of the 5 source categories in the exceedance group, but the "pulp and paper MACT I and III" sub-category has only 1 of 127 facilities that exceed the threshold (<1%), whereas "pulp and paper MACT II has 13 of 134 facilities that exceed the threshold (9.7%), and the third pulp and paper sub-category has 8 of 348 facilities in exceedance (2.3%). Are distinctions across these sub-categories important in this screening process? If not, then why is the analysis conducted at the sub-category level? And what should we conclude about the source category "ferroalloys" that has an exceedance rate of 36% (4 of 11), or several other source categories that have double-digit exceedance rates? The Agency could improve their explanation of the Part I process by providing the criteria used to sort source categories into "significant" and "limited potential".

If feasible, a probabilistic analysis of the exposure scenario and other factors would provide more transparency regarding variability and uncertainty in the exposure and risk estimates, and would allow a more informed judgment regarding the extent of conservatism included in the calculation/model.

#5. Acute Exposure Screening: *The plan describes a screening methodology for identifying potentially significant acute exposures from routine emissions. Is this method appropriately protective? Can you suggest ways to refine the proposed acute exposure assessment process to enable it to support decision-making?*

Section 3.2 of plan (pp. 20-22) describes sources of acute dose-response information. It is not clear why, on pages 21 and 22, there are discussions of URE values for formaldehyde, nickel, 2-nitropropane, and POM. Perhaps this information belongs elsewhere.

The authors' judgment that the multiple sources of acute hazard information are probably not comparable seems correct. Each of the four sources have used different criteria for calculating hazardous air concentrations, and within each source values have probably been generated over time with different methodologies. All of these sources are reputable, and the information they provide is probably the "best available" science or professional judgment. The Agency has not delved into these sources to determine what value would be most appropriate for a specific pollutant. It is hard to judge whether these values are appropriately protective without a more careful examination of how the Appendix 6 acute exposure values were derived by the individual sources.

Response to Charge Question 2
Bryan W. Shaw, Secondary Reviewer

2. Emissions and Source Data: The NEI for hazardous air pollutants represents an ongoing voluntary national effort whose creation results from the collaborative efforts of State, local, and tribal air agencies with EPA Regional and Headquarters staff.

a. Short of creating a federal mandate for reporting emissions to the EPA, do the methods by which the NEI was developed, reviewed, and compiled result in a technically-credible database that can support regulatory assessment and action? If not, can you suggest ways to improve it?

Development of accurate NEI estimates is always challenging. Incorporating a framework for improving the NEI as more accurate data becomes available is critical.

b. Do the plans for conducting an engineering review and incorporating currently-available refined emissions and source data into the inventory add value to the assessment? Does the plan for soliciting public comment through an advanced notice of rulemaking add scientific credibility to the inventory? Is the plan for reconciling comments on the inventory adequate? If not, can you suggest other approaches for reconciling such comments?

The plans for conducting an engineering review adds value. However, to maximize the benefit, the assessment must be conducted in a thoughtful manner. Furthermore, the subjective nature of the assessment may not provide the level of accuracy or precision needed to be useful.

Public Comment – Yes this is crucial and adequate.

Response to Charge Question 2
Bryan W. Shaw, Primary Reviewer

4. Dispersion Modeling: Does the coupling of the AERMOD dispersion model with the census block human exposure modeling (HEM) approach to estimating individual and population exposures represent a credible approach for this goal?

There are inherent shortcomings associated with any model. In this instance the model is a Gaussian based model. The challenges are primarily associated with dispersion parameters and chemical reactions/deposition. The chemical reactions can be critical as the concentration of pollutant of interest at the receptor can change dramatically due to chemical change or deposition (this can be an increase or decrease). As models are improved to better reflect the atmospheric chemistry, this will improve models usefulness for estimating potential health/environmental impacts associated with emissions of interest.

Attempt to couple AERMOD and HEM seems to be appropriate so long as effort is made to ensure compatibility of data.

Are there other more credible approaches available for the estimation of inhalation risks from the types of source categories being examined?

Not that I am aware of at this time.

Is the level of accuracy of this approach acceptable for the purposes of residual risk decision making?

I do not have quantitative assessment of the accuracy of this approach. However, this does seem to be a reasonable approach so long as the uncertainty and/or bias of the model and disparities between AERMOD and HEM data compatibility are addressed.

Are there any specific source categories, sources, or pollutants for which this approach might be considered inadequate?

Potential source categories that may not be well addressed by this approach include any source where significant chemical reaction is not well defined or where deposition is not well described by the model. I have not identified any such categories.

Lauren Zeise
Preliminary responses to charge questions

7. Dose Response Values: Is the plan for using available dose-response information (e.g., sources of information, prioritization scheme appropriate for the purposes of this assessment? If not, can you suggest ways to improve it?

The plan for adopting chronic values (unit risk estimates and RfDs) is a reasonable one. The adjustments to the dose response values discussed on pages 21-22 are reasonable, although the formaldehyde adjustment should be reconsidered in light of new evidence. The prioritization scheme to select from available peer reviewed values is reasonable. Reliance on sources that utilize methods similar to those embodied in EPA's risk assessment guidelines, and that are thoroughly peer reviewed, provide sufficient scientific justification for their use. A major limitation in this approach, however, is that the toxicity data base is not as fully populated with values. This is important and desirable for a comprehensive and reliable assessment.

- 24% of HAPs have no toxicity measures
- Several carcinogenic HAPs lack unit risk values.
    - Several carcinogens in Appendix 6 have no unit risk estimates (antimony trioxide, chloroprene, cobalt compounds (cobalt metal powder and certain other compounds identified by IARC), diesel engine emissions, dimethyl sulfate, 1,1-dimethylhydrazine, ethyl acrylate, ethylbenzene, ethylene imine, methyl hydrazine, methyl iodide, nickel carbonyl, nickel oxide, nitrobenzene, styrene oxide.
    - Other HAPs with carcinogenic activity have no unit risk estimates and no other toxicity measures (2-acetoaminofluorene, 4-aminobiphenyl, o-anisidine, catechol, 4-nitrobiphenyl, N-nitroso-N-methylurea, p-phenylenediamine, beta-propiolactone, propoxur, quinoline).
    - In addition to developmental neurotoxicity, the carcinogenicity of lead should at least be considered, if only for the purpose of uncertainty or sensitivity analyses.
- The majority of HAPs have no measures for acute toxicity potential.
- A number of HAPs have the potential to induce reproductive toxicity (e.g., dibutylphthalate, bis(2-ethylhexylphthalate [DEHP], and a number of the carcinogens are also developmental reproductive toxicants). The potential for high, intermittent exposures is therefore a concern. It does not appear that such effects will be considered in the absence of subchronic or acute toxicity values.

The approach is to assume that the HAP produces no risk in the absence of a health value. The question is what difference does this make in the assessment and whether facilities will be falsely found to have no significant residual risk. As a suggested improvement in the approach, sensitivity analyses could be performed to understand the importance of these omissions for the different source categories. Where found to be important, additional, more careful analyses to develop more reliable health values would follow. Here are some possible approaches:

- Gain an understanding of which HAPs with missing health values are emitted from the sources under review. Develop health values for these HAPs.
    - For reproductive and developmental toxicity, the reviews of the National Toxicology Program's Center for the Evaluation of Reproductive Health Risks

could be utilized to obtain toxicity values for developmental/reproductive endpoints. Health values from other government sources outside the US could also be considered.

- For chemicals where reliable and sensitive acute and subacute non-cancer health values data are not available, crude lower bound estimates can be obtained from compilations of effect and no effect toxicity values such as the NIOSH Registry of Toxic Effects of Chemical Substances (RTECS). Values for sensitive studies could be selected, and relatively large uncertainty factor applied. It is anticipated that it would be a rare case that facilities would have exposures that would fall above these crude health values. When exposures exceed these crude estimates further work will be needed, unless they reinforce what is already observed for chemicals with reliable peer reviewed estimates.
- For cancer endpoints, crude estimates could also be developed. This could be done probabilistically, or by developing rough estimates of ranges of values that unit risk could potentially take for these compounds. Expedited assessments could develop rough estimates for the purpose of this sensitivity analysis. Oral unit risk values can be the basis for estimates of inhalation unit risk for certain compounds. In addition, EPA rules for study selection for unit risk estimation could be applied to the Bruce Ames/Lois Gold Cancer Potency Database to select sensitive data sets. Unit risk could be estimated from their TD50s or could be developed by applying standard EPA procedures to the EPA rule based data set selections. Alternative values available in the literature or from other state or federal governmental agencies could be also be used. Unit risk estimates found to suggest risks near the significant risk threshold could be scrutinized more carefully.

8. Risk Characterization: What are the strengths and the weaknesses of the overall conceptual approach to risk characterization planned for this assessment? Does the characterization plan adequately cover sensitive subpopulations and early-life exposures? Does the risk characterization plan appropriately aggregate cancer risks? What are the strengths and weaknesses of the planned approach for characterizing important uncertainties, variabilities, and limitations? Given the underlying science and the intended purposes of the assessment, can you suggest ways that the characterizations of uncertainty and variability could be improved, made more transparent, or integrated more effectively into the risk characterization?

The strength of the approach is that it is an expedited, straightforward, resource conserving and fairly transparent method, one that should be readily understood by an interested public and decision makers. The cost of course is scientific accuracy and bias, which may be difficult to rectify without a substantial infusion of funds. While some aspects of the procedure are biased toward overestimation, there are other procedures and assumptions pulling in the opposite direction. The overall bias is unclear, and work is needed to better understand the extent of the potential bias. For example, emissions data are voluntary. Will EPA hear from the heaviest emitters in a category? Also, given geographic variability, the reliance on meteorological data from stations a considerable distance from the facility carries substantial opportunities for mis-estimation. In some cases it will under estimate and in others will over estimate. But given that the goal is to ensure the risks for the most exposed do not exceed certain criteria, the focus

should be on that group, and those cases. The RTR acknowledges that estimation of acute exposure a particularly uncertain; the default use of a factor of 10 across all source categories to characterize acute exposures also provides substantial opportunity for under-characterization of exposures. This in some cases may be an extreme underestimate, for example where there are infrequent batch manufacturing processes. Also, while the assumption that individuals are present at the point of maximum ambient impact is a worse case assumption for human behavior, as pointed out in the RTR Plan, it is not far fetched for the acute case certain subpopulations (newborns, elderly), and indeed may be realistic. The lack of readily available toxicity indices for acute exposure coupled with the uncertainty in exposure estimates means the acute assessment is fairly unreliable.

One possibility would be to study well a few facilities in each source category to evaluate the extent that approach taken to the exposure analysis may result in underestimates of risk/hazard indices. Similarly, an approach to consider the importance of missed health values is given above in response to question 7.

To the extent that the RfCs adequately considered sensitive populations in their derivation, the chronic non-cancer estimates on the dose response side adequately covers sensitive populations. It is beyond the scope of the assessment to redo agency consensus health values. (Of course, where there are no health values, sensitive subgroups (and everyone else) are not considered.) The RTR plan on page 22 acknowledges that health values do not explicitly take into account inter-individual variability in health status and genetic make-up. On the cancer side, they implicitly do not when based on animal data. The underlying assumption is that each and every individual faces the same risk of cancer, not a bad assumption due to the genetic homogeneity in the test animals and the controlled laboratory study conditions. There is no provision for interindividual variability in cancer risk estimation, the animal results just extrapolated to humans using a correction factor to account for differences in body size. (It is unclear for this reason why the box is checked in Table 2 indicating that interindividual variability is likely to bias toward underestimation, ditto for interspecies extrapolation). Also, there is the implicit assumption that in utero exposure poses no cancer risk. When cancer unit risk estimates are based on human data, they only partially address sensitive individuals, since the estimates are typically derived from worker populations (commonly male, healthy workers), do not reflect the general population, and typically are central estimates. These are positions that the agency has taken, and again it is beyond the scope of the RTR to assume otherwise. Where EPA analyses are lacking and the RTR relies on other sources of health values, similar procedures have been applied by these other sources.

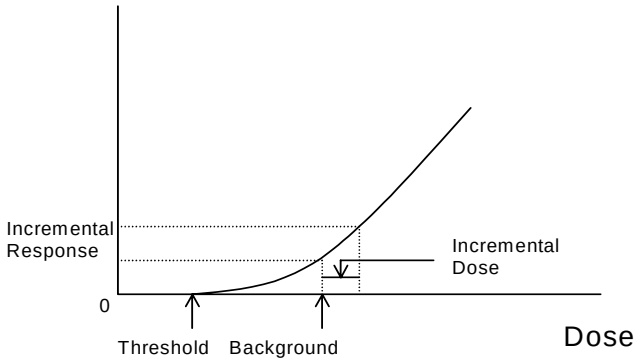
Some of interindividual variability can be characterized. Sometimes risks for certain sensitive subgroup can be described directly, or through modeling and quantification of predisposing factors. Indeed, the SAB has recommended on a number of occasions that the Agency work toward incorporating probabilistic methods in its health value characterizations, to both characterize interindividual variability and uncertainty. It will take considerable effort to develop the methodology and (science permitting) the data to do this in general. Again, it appears beyond the scope of the RTR to perform this work. However, because on the cancer side interindividual variability is assumed not to exist, and on the non-cancer side background health status of the population and related exposures are not typically addressed, the argument that the estimates are biased conservative can be questioned. As pointed out by the RTR on page

22, the magnitude and the direction of the overall bias (from all the factors contributing to a health value assessment) are not known. (There are other places in the document where the estimates are described as biased toward overestimation. For example at the end of the first full paragraph on page 23 – but that lower bound estimate discussed there is dealing with only one of several factors in the analysis.) Finally, use of the EPA Supplemental guidance approach for potency adjustment for early life exposure is warranted, and should be especially important for the analysis where length of residency is less than lifetime.

Generically describing the uncertainty and variability as is done in Table 2 is reasonable for the health endpoints in the absence of material from other parts of the agency. However, an uncertainty/sensitivity analysis to get a better handle on the lack of health values HAPs should be done at minimum (as indicated above in response to question 7). The entries in the table describing uncertainty for some other factors can be questioned. Analyses of human and animal unit risk values for the same chemicals show in some cases that human risks are underestimated and others are overestimated by the animal values. An analysis of cigarette smoke components (Hertz-Picciotto et al.) found that animal based unit risks of components did a fairly good job of predicting the overall cancer impact on humans of smoking. There are also several cases where chemicals are considerably longer lived in humans than is consistent with body size differences in pharmacokinetics would predict. The data would tend to support the influence of interspecies extrapolation procedures on estimation to be mixed, rather than overestimating risk. Similarly as noted above, the procedures for intraspecies extrapolation for cancer are nonexistent, so it is unclear why the overestimation box is checked for this factor. On the non-cancer side, it is mixed and chemical dependent.

Page 15 of the RTR Plan. It is not clear how annual cancer incidence is to be calculated. An explanation is needed.

The RTR plan rightly acknowledges that, for non-cancer effects, not taking into account exposures associated with background sources may result in an understatement of the hazard associated with the incremental emissions form individual facilities. The premise for the application of non-cancer RfDs and guidance levels for carcinogens believed to act by non-linear mechanisms is that there is a threshold dose below which adverse effects should not occur. Typically not considered are the myriad of other exposures from endogenous and exogenous sources that may affect the toxicity pathways or networks by which the chemical operates. The chemical under assessment adds to these exposures, and in some cases these exposures may in toto fall above the population threshold, as illustrated in the figure below. Examples where this appears to be the case include neurodevelopmental effects of lead and methylmercury, particulate matter and asthma induction, the impact of ozone on respiratory health, and perhaps the impact of dioxin-like compounds on a variety of endpoints. This issue of the status of the population in terms of background exposures and disease factors is critical understanding the degree to which a source may pose a (non-cancer) risk to the community.



Not formally evaluating the contributions of background sources in evaluating a facility can, as the EPA RTR plan indicates, result in misestimating the impact of the source in a community. In some cases the source may take a few individuals above threshold levels. In other cases a significant fraction of the population may suffer from subtle or not so subtle effects from combined exposures and the source may increase the severity of the impact susceptible groups or drive more people into a diseased state. Finally in some situations background exposures may be sufficiently low so that the RfC approach does not lead to mischaracterizations. The drinking water program partially addresses the issue by using relative source contribution terms. But here a preferred approach would be to look for cases where aggregate and cumulative exposures to chemicals working like the one emitted from the source is an issue. This could potentially be done through examination of monitoring levels and exposure assessments in various toxicity profiles (e.g., ATSDR, CERHR, the EPA dioxin reassessment). In this way, the HAPs where this is an important consideration can be distinguished and the issue then considered up front in assessing the source category.