

# Ranking and Selection of Hazardous Air Pollutants For Listing Under Section 112(k) of the Clean Air Act Amendments of 1990

## Technical Support Document

*Roy L. Smith, Ph.D.*

*Charles L. French*

*Deirdre L. Murphy, Ph.D.*

*Rhonda Thompson*

*EPA Office of Air Quality Planning and Standards*

*July 28, 1999*

### 1. Introduction

EPA has published numerous guidelines for risk assessment that support the development of qualitative and quantitative estimates of risk to health. These guidelines are widely used and understood, and EPA considers them an appropriate basis for ranking and selecting hazardous air pollutants (HAPs) for the purposes of section 112(k) of the Clean Air Act (CAA) Amendments of 1990. This document describes EPA's use of risk assessment tools and information in selecting HAPs posing the greatest health risk in urban areas ("urban HAPs"), and a subset of urban HAPs that pose health risks as a result of emissions from area sources ("area source HAPs").

The essence of the Agency's model for risk assessment lies in a combination of two types of information. The first type of information concerns the nature of adverse effects caused by a substance (the "hazard identification"), and specific exposure levels at which the effects occur (the "dose-response assessment"). This information is based on human or animal studies of high quality, usually obtained from scientific journals. The second type of information concerns the amount, or dose, of the substance that receptors get from the environment. This "exposure assessment" is developed from actual measurements, mathematical models, or a combination of both. These two types of evidence--the dose that causes harm and the dose actually received--are combined in a "risk characterization" that describes the potential for real-world exposure to cause harm and the uncertainties surrounding this potential.

If it were possible to do so, the selection of urban and area source HAPs could reasonably be based on a quantitative national risk assessment for all HAPs in all urban areas. Such an assessment would include evidence of (1) doses of each HAP known to cause adverse effects (and the nature of those effects) and (2) estimated doses of each HAP that receptors in urban areas may actually receive from the environment. However, such a comprehensive risk assessment is not yet possible. The limitation is not that EPA does not know how to do a fully quantitative national risk assessment, but rather that we do not yet have some of the information needed to do it.

EPA's list of HAPs currently contains 188 substances and "categories" of substances. Many of these HAPs have not yet been subjected to toxicological testing, and existing test results for others have not yet been developed into dose-response assessments. Although 188 HAPs might seem to be a reasonably sized group to address, in fact it is much larger. Some HAP categories (*e.g.*, polycyclic organic matter, or POM) are broadly defined, containing thousands of individual chemical compounds with widely varying toxic potential. The scientific community is working hard to collect new toxicity data, and EPA and other regulatory agencies are working equally hard to develop these data into dose-

response assessments. However, given realistic research and resource constraints, the sheer size of the HAP list precludes a complete understanding of HAP toxicity at this time.

To address exposure to HAPs, we would prefer to use measured personal exposures or ambient concentrations from monitoring stations. However, personal monitoring data are still rare, and EPA's ambient air monitoring activity focuses on criteria pollutants such as particulate matter and ozone. Some States and local governments fund and operate HAP monitoring stations, but these are based on the priorities of the funding agencies. For this reason, sampling strategies, lists of substances monitored, and analytical methods vary substantially from place to place. Many HAPs, and many locations, are not monitored at all. Consequently, ambient monitoring information provides important but limited evidence of exposure potential.

EPA's data for amounts of HAPs emitted from various sources is more complete than our ambient monitoring databases, but these emission data also have important limitations. EPA developed many of the national emissions estimates by applying an emission factor, or series of factors, to activity data thought to represent source categories nationally. Emission factors were developed from information from a small number of sources within a source category, or by professional judgment. Applying emission factors and activity estimates across all emission sources in a source category carries substantial uncertainties.

Furthermore, an emission rate does not equal an exposure. Before a receptor can be affected, the substances must be diluted and dispersed through the atmosphere, where some may be transformed to other substances or deposited before exposure occurs. To provide a more meaningful indicator of exposure, emission data can be input to a dispersion model capable of estimating ambient concentrations. Although our current national emissions inventory data do not include sufficient location data to support dispersion modeling, our inventory for 1996 (currently under state review) will support such modeling.

For these reasons, neither the dose-response nor the exposure database can currently support a direct, quantitative national risk assessment for HAPs in urban areas. Nevertheless, the Act requires EPA to select 30 or more HAPs posing the greatest threat to health and the environment in urban areas. Recognizing the above limitations, EPA is obligated to make decisions based on the best available information. EPA has based its proposal on the results of three separate analyses of information concerning HAPs in urban areas. These analyses were for the most part developed independently, although they are by necessity based on much of the same data. They were prepared by three different groups of scientists, although these groups communicated and exchanged ideas during their work. The three analyses arrived at conclusions that are in some ways similar, while varying significantly in others. EPA has endeavored to combine the results in a way that takes advantage of concordance among these groups and makes reasonable judgments in areas where opinions vary.

## 2. Methods

In 1997, EPA conducted an initial screening evaluation to develop a list of 40 candidate urban HAPs. The evaluation used three independent ranking analyses (a review of existing studies, an urban analysis conducted by the EPA Cumulative Exposure Project team, and calculation of risk-based ranking indices). Two of these analyses are summarized briefly in Sections 2.1 and 2.2 below, and presented fully as appendices to this document. The third analysis is described in detail in Section 2.3 below.

Interested parties were invited to submit emission data to augment or replace information used to develop the list of candidate HAPs. EPA also subjected the screening evaluation methodology itself to peer-review by independent experts in air toxics and risk assessment. In early 1998, EPA held a full-day session of the peer-review panel to discuss the methodology and underlying data. The reviewers evaluated all facets of the methodology and its suitability for identifying HAPs for the urban HAPs list, the relative value of various data sources, the availability of additional data sources, the scientific validity of assumptions, consistency across the methodology, and appropriate presentation formats. Reviewers provided oral comments at the meeting, and written comments before and after the meeting. EPA substantially revised the HAP selection methodology in response to the reviewers' comments.

EPA also received comments from the public in response to our publication of the draft list of urban HAPs [1]. Consideration of issues raised by some commentors led us to modify certain aspects of both the identification methodology and the underlying data inputs. None of these changes, described in the sections below, conflicted with recommendations made earlier by the 1998 peer review panel.

In finalizing the HAP selection methodology, EPA also took the opportunity to update once again all data on emissions, ambient concentrations, health effects, and bioaccumulation potential to ensure that the selection process has relied on the most recent available information. Nevertheless, it is important to realize that the methodology is based on databases that are far from complete, and that contain information of widely varying quality. EPA believes that this information is the best available for this purpose, and that basing its ranking on these data is reasonable. However, readers must keep in mind that substantial uncertainty surrounds this analysis. Results should be considered only relative estimates of potential hazard of various HAPs, and not construed as quantitative estimates of actual risks.

## 2.1 Review of Existing Studies

The first analysis of HAPs in urban areas, prepared by an EPA contractor, reviewed twenty-three existing studies of exposure, risk, or hazard associated with HAPs. These studies were conducted during recent years by EPA, state agencies, and others. Of these original twenty-three, fourteen studies were deemed appropriate for comparative ranking of HAPs. (Six assessments were dropped from consideration because they were conducted partly or entirely in rural locations, and three more were omitted because they covered fewer than ten HAPs.) Hazard ranking scores (*e.g.*, quantitative risk estimates, percent contribution to risk, ranks) from each study were normalized to the same scale, then aggregated to make a combined total score for each HAP. Carcinogens and non-carcinogens were ranked separately. Separate analyses were done for all sources combined (*i.e.*, major, area, and mobile sources), and for area sources alone. The combined analysis was the one used in the HAP selection process. HAPs that ranked above obvious breakpoints in the frequency distribution graphs from each of the four analyses were assigned highest priority. The full analysis of existing studies is presented in Appendix A.

## 2.2 Cumulative Exposure Project Urban Analysis

The second HAP ranking analysis was performed as part of the Cumulative Exposure Project (CEP) conducted by the EPA Office of Policy, Planning and Evaluation. The CEP urban analysis compared modeled yearly average ambient concentrations of HAPs in urban areas for 148 HAPs against risk-based concentrations (RBCs, termed "health benchmarks" by the authors) at the census tract level. A long-term Gaussian dispersion modeling approach was used, with emission rates drawn from the

Toxics Release Inventory and other EPA databases, addressing major, area, and mobile sources. In the original analysis prepared by the CEP team, contributions from distant emissions of persistent pollutants and from non-anthropogenic sources were addressed with background values drawn from measurements in remote locations. The CEP compared these estimated ambient concentrations to RBCs corresponding to: (1) a one in a million upper bound lifetime cancer risk (assuming continuous exposure for 70 years), or (2) a concentration considered to have no significant risk of adverse non-cancer effects in continuously exposed populations<sup>1</sup>. HAPs were ranked according to the number of urban census tracts in which the modeled concentration was above the RBC. HAPs estimated to exceed their respective RBC in 50 or more urban census tracts were marked for consideration as urban HAPs.

Following the September 14, 1998 proposal on the draft integrated strategy for urban air toxics, EPA received numerous comments objecting to the CEP's use of (1) background concentrations in the HAP selection process, and (2) outdated RBCs for specific substances. To address these comments, we compared predicted ambient concentrations (omitting background) for specific HAPs with our current RBCs. These recalculations were done only for HAPs to which a background concentration was assigned in the original CEP analysis, or for which an RBC had changed.

The original CEP analysis is presented in Appendix B, and the recalculated results are presented in Appendix C.

## 2.3 Risk-Related Ranking Analysis

The third relative hazard analysis, prepared by EPA staff, ranked HAPs by combining surrogates for toxicity and exposure into ranking indices. The surrogates for toxicity were the risk-based concentration (RBC) for inhalation or the risk-based dose (RBD) for ingestion. For effects other than cancer, the RBC or RBD represented an exposure considered to have no significant risk of adverse non-cancer effects. For carcinogenic HAPs, RBCs or RBDs represented exposures associated with fixed levels of upper-bound predicted lifetime cancer risk. Two sets of RBCs and RBDs for carcinogens were calculated, the first at a one in ten thousand risk level and the second at one in one million. Surrogates for exposure included measured ambient concentrations, and emission rates from area, major, and mobile sources. Seven separate ranking indices were calculated, then combined into a single ranking. The risk-related ranking indices, and the process by which they were combined with results of the review of existing studies and the CEP analysis, are described below. The lists of urban HAPs and area source HAPs were developed from the results of all three analyses by considering (1) how many of the analyses identified the HAP and (2) the contribution of emissions from area sources.

### 2.3.1 Surrogates for Toxicity

Toxicity information used in the risk-related ranking analysis consisted of dose-response assessments developed by various regulatory agencies for protection of human health. A wide variety of these assessments were incorporated, many of which were performed at different times, intended for different purposes, and subjected to varying levels of review. EPA believes this to be defensible practice for the purpose of selecting urban and area source HAPs, because the alternative to using

---

<sup>1</sup>An example of an estimate of "a concentration considered to have no significant risk of adverse non-cancer effects" is the EPA reference concentration (RfC). The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious non-cancer effects during a lifetime.

potentially inconsistent dose-response information from non-EPA sources would be a *de facto* assumption of zero toxic potential for some substances. This practice would create false negatives that EPA considers unacceptable in this context.

All 189 HAPs originally listed under Section 112(b) of the CAAA (with the exception of radionuclides, asbestos, and fine mineral fibers) were carried through the index calculations. The remaining 186 substances and substance categories were included in the detailed calculations, even those that lacked dose-response, emission, or ambient data, and for which no indices could be calculated. (Caprolactam, recently deleted from the list of HAPs, was also included in the calculations.) EPA believes that this full presentation will allow readers to see data gaps more clearly, and may serve as a guide for future efforts to upgrade data collection for the air toxics program.

Dose-response assessments for health effects of HAPs were obtained from various sources, and prioritized according to (1) applicability, (2) conceptual consistency with EPA risk assessment guidance, and (3) level of review received. The following dose-response assessment sources were used in this analysis.

#### 2.3.1.1 US Environmental Protection Agency (EPA)

EPA has developed chronic dose-response assessments for many of the HAPs. These assessments typically specify a reference concentration (to protect against effects other than cancer) and a unit risk (to estimate the probability of contracting cancer). A reference concentration (RfC) is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) likely to be without an appreciable risk of deleterious non-cancer effects during a lifetime. The unit risk (UR) is the upper bound excess lifetime probability of contracting cancer per microgram of HAP per cubic meter of air, assuming constant inhalation exposure over a lifetime.

EPA also publishes analogous dose-response values for oral exposure, called the reference dose (RfD) and carcinogenic potency slope (CPS). The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) likely to be without an appreciable risk of deleterious effects during a lifetime. The CPS is the upper bound excess lifetime risk of contracting cancer per milligram of HAP per kilogram body weight per day, assuming constant oral exposure over a lifetime.

In assessing a substance's carcinogenic potential, EPA evaluates various types of toxicological data and develops a "weight-of-evidence" determination. EPA's present weight-of-evidence categories are Group A (carcinogenic in humans), Group B (probably carcinogenic), Group C (possibly carcinogenic), Group D (not classifiable), and Group E (probably not carcinogenic). EPA is in the process of changing to a text-based descriptive weight-of-evidence procedure that is less categorical, but few EPA assessments reflect this change so far.

EPA disseminates dose-response assessment information in several forms, based on the level of internal review. EPA publishes dose-response assessments that have achieved full intra-agency consensus on its Integrated Risk Information System (IRIS)[2]. Assessments prepared by the EPA Office of Research and Development (ORD), but that have not been approved by all EPA program offices, are often published by ORD as individual health effects assessment documents. The results of many such assessments have been assembled in EPA's Health Effects Assessment Summary Tables (HEAST)[3]. EPA updates HEAST regularly.

### 2.3.1.2 Agency for Toxic Substances and Disease Registry (ATSDR)

ATSDR, which is part of the US Department of Health and Human Services, regularly publishes Health Guidelines Comparison Values (CVs) for many toxic substances. ATSDR describes CVs as media-specific concentrations to be used by health assessors to select environmental contaminants for further evaluation. They are presented with only 1 significant figure, and are considered concentrations below which contaminants are unlikely to pose a health threat. Concentrations above a CV do not necessarily represent a threat, and CVs are therefore not intended for use as predictors of adverse health effects or for setting cleanup levels.

For this analysis, the ATSDR CV of choice was the minimum risk level (MRL). An MRL is an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure. MRLs can be derived for acute, intermediate, and chronic duration exposures by the inhalation and oral routes. MRLs were chosen for use in this HAP analysis because their concept, definition, and derivation are philosophically consistent (though not identical) with the basis for EPA's RfC and RfD.

ATSDR publishes MRLs as part of toxicological profile documents, one per substance. MRLs are also collected in a table of CVs [4], regularly updated and distributed by ATSDR.

### 2.3.1.3 California Environmental Protection Agency (CalEPA).

The CalEPA Air Resources Board has developed dose-response assessments for many HAPs, based both on carcinogenicity, and health effects other than cancer resulting from chronic and acute exposure.

The non-cancer information includes available inhalation health risk guidance values developed by USEPA or CalEPA, expressed as acute or chronic reference exposure levels (RELs). CalEPA defines the REL as a concentration level or dose at (or below) which no health effects are anticipated. Because this concept is substantially similar to EPA's non-cancer dose-response values, this analysis has used chronic RELs in the same way as RfCs and RfDs.

CalEPA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the unit risk, defined similarly to EPA's unit risk. This analysis has used specific CalEPA URs in the same way as EPA's URs.

### 2.3.1.4 National Advisory Committee for Acute Exposure Guideline Levels (NAC)

USEPA's Office of Prevention, Pesticides and Toxic Substances established the NAC in 1995 to develop Acute Exposure Guideline Levels (AEGLs) and supplementary information on hazardous substances for federal, state, and local agencies and organizations in the private sector concerned with emergency planning, prevention, and response. The NAC/AEGL Committee is a discretionary Federal advisory committee that combines the efforts of stakeholders from the public and private sectors to promote efficiency and utilize sound science.

The NAC published an initial priority list of 85 chemicals for AEGL development in May 1997 and proposed AEGLs for 12 substances in October 1997 [5]. The AEGLs for a substance take the form of a matrix, with separate ambient levels for mild, moderate, and severe effects. Each of these three effect levels are provided for as many as four different exposure periods, typically 0.5, 1, 4, and 8 hours. Although still under public review, those proposed AEGLs for which substantial issues have not been in public comment have been considered in this analysis. AEGL values used for the HAP ranking

analysis were 1-hour concentrations for the mildest available effect level.

#### 2.3.1.5 International Agency for Research on Cancer (IARC)

The International Agency for Research on Cancer (IARC) was established in 1965 by the World Health Organization. IARC's mission is to coordinate and conduct research on the causes of human cancer, and to develop scientific strategies for cancer control. The Agency sponsors both epidemiological and laboratory research, and disseminates scientific information through meetings, publications, courses and fellowships.

As part of its mission, the IARC assembles evidence that substances cause cancer in humans and issues judgments on the strength of evidence. IARC's weight-of-evidence categories are Group 1 (carcinogenic in humans), Group 2A (probably carcinogenic), Group 2B (possibly carcinogenic), Group 3 (not classifiable), and Group 4 (probably not carcinogenic). The rankings may be applied to either single chemicals or mixtures.

IARC's weight-of-evidence for HAPs have been included in the supporting information of this analysis as a backup to EPA's weight-of-evidence determinations, which do not cover all HAPs and in some cases may be out of date.

#### 2.3.1.6 American Industrial Hygiene Association (AIHA)

AIHA has developed emergency response planning guidelines (ERPGs) for acute exposures at three different levels of severity of health effects [6]. These guidelines represent concentrations for exposure of the general population for up to 1 hour associated with effects expected to be mild or transient (ERPG-1), irreversible or serious (ERPG-2), and potentially life-threatening or lethal (ERPG-3). ERPG values used for the HAP ranking analysis were for the mildest available effect level.

#### 2.3.1.7 National Institute for Occupational Safety and Health (NIOSH)

As part of its mission to study and protect worker health, NIOSH determines concentrations of substances that are immediately dangerous to life or health (IDLHs). IDLHs were originally determined for 387 substances in the mid-1970's as part of the Standards Completion Program (SCP), a joint project by NIOSH and the Occupational Safety and Health Administration (OSHA), for use in assigning respiratory protection equipment. NIOSH is currently evaluating the scientific adequacy of the criteria and procedures used during the SCP for establishing IDLHs. In the interim, the IDLHs have been reviewed and, (if appropriate) revised. NIOSH maintains an on-line database [7] of IDLHs, including the basis and references for both the current and original IDLH values (as paraphrased from the SCP draft technical standards). For this HAP ranking, IDLH values were divided by 10 to more closely match the mild-effect levels developed by other sources, consistent with methodology used to develop levels of concern under Title III of the Superfund Amendments and Reauthorization Act [8].

#### 2.3.1.8 Prioritization of Sources

The risk-related ranking analysis relied on separate dose-response assessments for inhalation and oral exposure. Inhalation RBCs were developed for chronic and acute time scales, but oral RBDs were developed only for chronic exposure.

Some HAPs have been subjected to dose-response assessments by several of the regulatory agencies used as sources for this analysis. Because these assessments were done by different agencies at different times, for purposes which were similar but not identical, it is inevitable that results will not be totally consistent. To resolve inter-agency discrepancies for this analysis, EPA applied a consistent

priority scheme to the universe of dose-response information.

RfCs and URs for chronic inhalation exposure obtained from EPA's IRIS database were given first priority. For HAPs lacking IRIS data, ATSDR MRLs for effects other than cancer received next preference, followed by RfCs and URs published in EPA's HEAST, then by CalEPA RELs and URs. Sources for oral RBDs were prioritized in the same order used for chronic inhalation RBCs.

For carcinogenic HAPs having no chronic inhalation assessments from any of these sources, oral CPSs were converted to URs to simulate inhalation exposure. Oral-to-inhalation conversion was not done for non-carcinogenic HAPs. EPA understands that conversion of oral dose-response information to inhalation exposure is not optimal risk assessment practice. However, the alternative to this is to omit such HAPs from the analysis altogether, based on a *de facto* assumption of zero toxicity. EPA regards this alternative as unacceptable for the purposes of urban HAP selection. This procedure carries some risk of inappropriate rankings for some HAPs.

No-effect (or minimal-effect) concentrations for acute exposure were taken first from the proposed NAC AEGLs (using the 1-hour concentration for the mildest severity level), then CalEPA acute RELs, next the AIHA ERPG (at the mildest severity), followed by the NIOSH IDLH (divided by 10). ATSDR acute MRLs were the source of last resort because they are based on 15-day exposure periods and no-adverse-effect levels, a derivation that should produce results that are fundamentally more protective than acute values from the other sources.

#### 2.3.1.9 Assumptions on Speciation and Other Adjustments to Dose-Response Information

Following the prioritization of dose-response information, the following revisions and decisions were made, based on professional judgment:

1. *1,3-Butadiene*. On April 29, 1999, EPA's Office of Research and Development informed the Office of Air Quality Planning and Standards via memo that the UR for 1,3-butadiene currently on IRIS ( $2.8 \times 10^{-4} \text{ [mg/m}^3\text{]}^{-1}$ ) was no longer supportable. The memo recommended an interim UR ( $2.08 \times 10^{-6} \text{ [mg/m}^3\text{]}^{-1}$ ) that was more than two orders of magnitude lower (*i.e.*, less potent). Although it was too late to revise the tables and index calculations supporting the ranking to reflect this change, we confirmed that the status of 1,3-butadiene as an urban HAP would be unaffected by the revised UR.
2. *Chromium*. For chromium VI compounds, the IRIS RfC for Cr(VI) particulates was used in preference to the RfC for chromic acid mists and dissolved Cr(VI) aerosols.
3. *Chlorine*. Emissions of chlorine gas undergo a complex series of reactions in the atmosphere that rapidly deplete the parent compound. Although this analysis was not able to consider the intricate chemistry of atmospheric chlorine, it was necessary at least to consider the lack of persistence of parent Cl<sub>2</sub> gas. For this reason, the IRIS RfC for hydrogen chloride was also used to represent emissions of Cl<sub>2</sub>, which otherwise would have been over-represented in the ranking.
4. *Cobalt*. Cobalt emissions exist mostly as oxide, but the CalEPA REL and the ATSDR MRL are based on cobalt sulfate heptahydrate aerosol. These dose-response values were deemed not to match the environmental data, and were dropped.
5. *1,4-Dichlorobenzene*. In response to public comments, EPA reviewed the toxicological databases for compounds that EPA has designated as class "C" carcinogens, and for which URs are available.

Data for one of these compounds, 1,4-dichlorobenzene (p-DCB), indicate that (1) metabolic activation is probably necessary for tumor formation, (2) humans metabolize p-DCB much more slowly than do mice (in which tumors were observed), and (3) normal detoxification mechanisms effectively remove low levels of carcinogenic p-DCB metabolites such as humans might produce. Because of these uncertainties this analysis did not use a UR for p-DCB. Available URs for other class “C” carcinogens were retained.

6. *Glycol Ethers.* Five different glycol ether compounds had available dose-response assessments that provided recommended RfCs or equivalent levels. The lowest of these (*i.e.*, the most protective) was applied to the entire category.
7. *Lead.* For lead and compounds, the CalEPA UR was used for carcinogenic effects and the EPA national ambient air quality standard was used in lieu of an RfC for non-cancer effects.
8. *Mercury.* The IRIS RfC for elemental mercury was applied to inhalation of mercury and compounds, based on the finding of EPA’s Mercury Report [9] that the dominant form of mercury in the atmosphere is elemental (although divalent Hg may exist near some sources.) The IRIS RfD for methyl mercury was used for food chain calculations, to reflect that compound’s bioaccumulation potential.
9. *Nickel.* The IRIS unit risk for nickel inhalation was based on carcinogenic effects of insoluble nickel compounds in crystalline form. Soluble nickel species, and insoluble species in amorphous form, do not appear to produce genotoxic effects by the same mechanism as insoluble crystalline nickel. Available nickel speciation information for some of the largest nickel-emitting sources (including oil combustion, coal combustion, and others) suggests that at least 35% or more of total nickel emissions are soluble compounds. Of the insoluble nickel emissions, 17% is thought to be oxides, 3% or more sulfidic, and the rest is unknown. Based on these data, this analysis has assumed that 50% of emitted nickel is insoluble, and that 50% of insoluble nickel is crystalline. On this basis, the UR for nickel subsulfide (representing pure insoluble crystalline nickel) was divided by 4 and applied to all nickel compounds.
10. *Phosphorus.* Dose-response assessment values for white phosphorus, which can exist only momentarily in the presence of oxygen, were deemed inappropriate to apply to phosphorus emission or monitoring data, and were dropped.
11. *Polycyclic Organic Matter.* The analysis used a group of 7 carcinogenic PAH compounds (benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) to represent the entire polycyclic organic matter (POM) HAP category. A weighted UR of  $3.3e-4$  ( $\text{ug}/\text{m}^3$ )<sup>-1</sup> was developed for these carcinogenic PAH compounds tracked as a group by EPA’s National Toxics Inventory (described below). The UR was based on a combination of compound-specific UR values [10], and the inventory emissions for each of the compounds.
12. *Selenium.* The CalEPA chronic REL for hydrogen selenide was deemed inappropriate to apply to all selenium compounds, based on ATSDR’s judgment [11] that fossil fuel combustion is the primary source of atmospheric Se, which is emitted predominantly as SeO<sub>2</sub>. No inhalation RBC was used.

13. *Vinyl Chloride*. The IRIS UR for vinyl chloride is also currently under review. Although this analysis uses the older UR currently on IRIS, we confirmed that the status of vinyl chloride as an urban HAP would not be affected by the draft reassessment.

The complete set of regulatory dose-response information used in the risk-related ranking analysis is presented in Table 1, together with the EPA and IARC weight-of-evidence determinations for carcinogenicity and the source of each regulatory value. All HAPs (plus caprolactam) appear in this table, with blanks showing where dose-response assessments were not available. Ranking indices could not be calculated for these substances.

#### 2.3.1.10 Development of Risk-Based Concentrations (RBCs) and Risk-Based Doses (RBDs)

RBCs [12] and RBDs are a simple device by which dose-response information for cancer and non-cancer effects can be reduced to a single type of information—an ambient air concentration (or oral dose) of a substance that defines an insignificant health risk over a specified exposure period. Concentrations or doses lower than the RBC/RBD can usually be ignored. Higher concentrations or doses do not necessarily equate to a significant threat, but may deserve a closer look.

RBCs and RBDs are products of risk assessments run in reverse. Instead of beginning with environmental concentrations and applying an exposure scenario to calculate a risk, the risk assessor begins with a fixed level of risk and inverts the calculations to determine the environmental concentration of a substance that will produce it. Such inverted calculations, when performed in accordance with EPA's national risk assessment guidelines, are no less valid than the usual forward computation of risk. The selection of a fixed risk level, however, may appear to imply a policy choice that is not intended.

For non-cancer effects, the RBC/RBD is simply the reference concentration or reference dose (or similar value from another source). For non-threshold carcinogens, the RBC/RBD is based on a fixed, nonzero level of risk, implying a risk management decision that this fixed risk level is "not significant," or *de minimis*. If the *de minimis* cancer risk were set at 1 in 1 million ( $1e-6$ ) upper bound lifetime cancer risk, the list of 30 substances selected likely would be dominated by carcinogens. This would in effect give non-cancer effects "second-class" status. If, on the other hand, *de minimis* cancer risk were considered to be, for example, 1 in 10,000 ( $1e-4$ ), readers might assume that EPA was willing to accept risks up to that level for single HAPs.

This issue was addressed by calculating two sets of chronic RBC/RBDs, called "case 1" and "case 2". The case 1 concentration or dose was that yielding a  $1e-6$  upper-bound lifetime cancer risk, or the RfC for chronic non-cancer effects, whichever was lower. The case 2 concentration or dose represented a  $1e-4$  upper-bound lifetime cancer risk, or the RfC, whichever was lower. For HAPs having only a UR and no RfC, there was a 100-fold difference between case 1 and case 2. For HAPs having only an RfC and no UR, case 1 and case 2 were identical. For HAPs with both a UR and RfC, case 1 was often (though not always) based on cancer and case 2 on non-cancer effects.

Exposure assumptions were deliberately kept simple and minimal. Inhalation RBCs for chronic exposure were based on an assumption of continuous lifetime exposure. Inhalation RBCs for acute exposure were based on episodic 1-hour exposures with enough recovery time between exposures to preclude lingering adverse effects. RBDs for chronic oral exposure, expressed as mg of HAP ingested per kg of body mass per day (mg/kg/d), were used directly without exposure assumptions. RBCs and RBDs for case 1 and case 2 are presented in Table 2.

EPA recognizes that actual exposures to HAPs are far more complex, and that these minimalist exposure scenarios, if used for quantitative risk assessment, could produce misleading results. Readers are reminded that this analysis is not intended to quantify absolute levels of risk, but rather to rank HAPs according to *relative* hazard. Applying a more detailed and realistic exposure assessment to this analysis would drastically increase the complexity of the ranking analysis, but whether this additional complexity would greatly alter the overall list of priority HAPs is unclear.

#### 2.3.1.11 Uncertainties in Use of Dose-Response Surrogates

##### 2.3.1.11.1 Carcinogens

EPA's methods for deriving URs and oral potency slopes were intentionally designed to avoid underestimation of cancer risk. This protectiveness was incorporated into several steps of the process. First, potency estimates for most HAPs were based on a mathematical model (the linearized multistage model) that assumes a straight-line dose-response all the way from administered doses in animals to zero dose. In effect, the model predicts that any dose of a carcinogen, however small, carries some minute lifetime cancer risk. EPA uses this model as its protective default in the absence of information supporting a different model for a substance. Use of other less conservative models would produce lower ranks for many carcinogens relative to non-carcinogens.

Carcinogenic potency estimates for many HAPs also incorporate protective extrapolations from test animals to humans, based on relative surface area (assumed to be the 0.67 power of body mass) as a surrogate for metabolic rate. It can also be argued, for example, that animal data can be converted to human equivalence using body mass itself (i.e., the 1.0 power of body mass), which is less protective. EPA itself is changing to a conversion based on relative basal metabolic rate (assumed to be the 0.75 power of body mass). Use of a higher power of body mass would produce lower ranks for carcinogens relative to non-carcinogens.

Third, carcinogenic potency estimates for most HAPs are 95% upper confidence limits rather than best estimates. The true potencies may be less, but are unlikely to be greater.

##### 2.3.1.11.2 Non-carcinogens

RfCs and oral RfDs define continuous lifetime exposures, with uncertainty spanning perhaps an order of magnitude, that EPA expects to be safe for human populations. RfCs and RfDs often must be based on limited data, and may be well below the actual human threshold for adverse effects, for two reasons. First, EPA favors the most sensitive species and the adverse effect to that species which occurs at the lowest dose. Although extrapolations from animals to humans are based on the best available data, in some cases EPA assumes that humans may be up to ten times more sensitive than the tested species, and that sensitive humans may be up to ten times more sensitive than the average human. These assumptions, designed to give the benefit of uncertainty to the exposed public, may produce RfCs and RfDs that are well below the true human adverse-effect thresholds for some HAPs.

Second, EPA has based some RfCs and RfDs on the no observed adverse effect level (NOAEL). The NOAEL is the highest dose at which test animals did not exhibit adverse effects relative to controls. Because most toxicological studies are designed with considerable gaps between test doses, the true threshold for adverse effects may be substantially higher than the experimental NOAEL. Use of the NOAEL instead of the true threshold for effects provides an additional level of protectiveness in reference doses.

##### 2.3.1.11.3 Adaptation of Oral Dose-Response Assessments to Inhalation

Additional uncertainty was introduced for 15 carcinogenic HAPs and HAP categories (out of the total 188) that lacked dose-response assessments for inhalation, but had oral values. For these HAPs, EPA judged that a converted oral value was preferable to the alternative *de facto* assumption of zero carcinogenic potential. Conversion from oral to inhaled exposure was based on an assumed body mass of 70 kg and inhalation rate of 20 m<sup>3</sup>/d. No adjustment was applied to account for differences in absorption through the GI tract and the lung, or for possible direct adverse effects to the lung. There is no way of knowing if “quasi” RfCs and URs derived by oral-to-inhalation conversions are more or less protective than fully-developed ones.

#### 2.3.1.11.4 Prioritizing Dose-Response Assessments

While dose-response assessments developed by EPA, ATSDR, CalEPA, and others share substantially the same purpose and philosophy, these factors are not identical. If EPA were to develop a complete set of RfCs and URs for all HAPs, it is possible that some would be significantly different than the non-EPA values actually used.

CalEPA has proposed URs for six HAPs or HAP categories that lack both an EPA and IARC weight-of-evidence determination. This ranking analysis has used these URs. Leaving them out would move these substances lower in the ranking, and would eliminate some entirely. Use of these six URs in this analysis does not constitute a recommendation by EPA that they are necessarily appropriate to use in quantitative risk assessments.

This analysis used a somewhat different prioritization scheme than did the EPA Cumulative Exposure Project (CEP). The major differences were that the CEP (1) did not use EPA Superfund Technical Support values at all, (2) did not extrapolate from oral to inhalation exposure for noncarcinogens, (3) used older CalEPA assessments, and (4) included assessments from unpublished 1994 draft EPA guidance for determining *de minimis* risk levels.

In assessing acute hazards, the CEP divided SARA LOCs by a factor of 1000 to simulate no-effect levels, whereas the risk-related ranking analysis used ATSDR acute RELs, followed by NAC AEGLs, with unaltered LOCs serving only as a last resort. As a result of its treatment of LOCs (and their subsequent comparison to yearly average concentrations, rather than short-term averages) the CEP produced more protective acute results for some HAPs than did the risk-related ranking indices. EPA has determined that the outcome of the analysis—the proposed list of 30 substances—was not influenced by the CEP’s high level of protectiveness for acute effects.

These differences in assessment prioritization resulted partly from the fact that the CEP had somewhat different goals than did the present analysis. Mostly, however, these variations arose from the fact that there is no clear “best” way to prioritize dose-response assessments. Two groups of scientists independently addressed a fuzzy issue, and arrived at somewhat different answers. EPA believes that the HAP selection process will be strengthened, rather than weakened, by this dichotomy of opinion.

### 2.3.2 Surrogates for Exposure

The second major part of the HAP ranking indices (the first part being the dose-response data described in the previous section) was information on exposure. Actual data describing human exposure to HAPs are limited, and lack the comprehensive geographic, temporal, and multi-contaminant coverage that this ranking exercise requires. Therefore, EPA chose to base the ranking on exposure surrogates—data related to, but not identical with, exposure. The two types of exposure

surrogates chosen were (1) long- and short-term ambient air quality measurements in urban areas, and (2) estimated annual masses of HAPs released in urban areas by major, area, and mobile sources.

#### 2.3.2.1 Measured Concentration Data

The ambient air quality dataset used in this analysis was created by combining all available monitoring data from EPA's Aerometric Information Retrieval System (AIRS) and Toxics Data Archive (9/30/98 version) databases for the 188 compounds defined in the Clean Air Act as hazardous air pollutants. The analysis was restricted to data collected in urban areas from 1988 through 1997. Data were expressed in units of micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ). Concentration data that were below the minimum detection limit (MDL) were replaced by  $\frac{1}{2}$  the MDL before averaging. When the MDL was missing, the lowest reported value was assumed a plausible value for the MDL.

For input to the chronic exposure indices, selected ambient air quality data were first averaged arithmetically for each combination of day, HAP, and monitoring site. Annual averages were then calculated from the daily averages. Data were selected for inclusion where (1) short-term measurements for at least 75% of the hours in a day, and (2) daily averages for at least 75% of the days in a year, were available. The expected number of daily measurements corresponding to 100% completeness was estimated by determining the frequency distribution of sampling intervals (days) and dividing 365 by the mode of the distribution.

Annual average concentrations from 1988 to 1997 for each site-pollutant combination were next averaged across years. Finally, the resulting multi-year average concentrations were averaged across monitoring sites into a single long-term multi-city average concentration for each HAP for which data met the selection criteria. The criterion for multiyear statistics was 75% completeness for 75% of the years. HAPs for which more than 90% of reported results were below the MDL were dropped from the analysis. Ambient data for individual compounds in the "7-PAH" group (*i.e.*, benz[a]anthracene, benzo[a]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and indeno(1,2,3-cd)pyrene) were summed and entered on the 7-PAH line.

To simulate acute exposure for each HAP, the 95<sup>th</sup> percentile of the original dataset was selected. EPA judged that this concentration represented a reasonable maximum short-term exposure, while avoiding potential problems with outliers that could result if higher percentiles were used.

The ambient concentration data used in the ranking analysis are presented in Table 3. All HAPs were included in the table, with missing ambient concentration data shown as blanks. Ranking indices based on ambient concentrations could not be calculated for these substances lacking these data.

#### 2.3.2.2 Emission Mass Data

The second type of data used in this ranking analysis as a surrogate for exposure were estimated emitted masses of individual HAPs. These data were obtained from several EPA emission data sources, for the period from 1990 to 1993 (the "baseline year" for measuring risk reductions). Data were retrieved for counties that contained a metropolitan statistical area (MSA) of 250,000 people ("urban-1"), or (for counties lacking an MSA of 250,000) a population designated as more than 50% urban by the Bureau of Census ("urban-2"). Data for counties classified as "rural" were excluded. Retrievals contained emissions from all types of sources, including major, area, and mobile sources.

Emission data were retrieved from the four sources described in Exhibit 1, below.

Exhibit 1. Emission data sources used in HAP ranking analysis, in order of preference. Data from lower-priority sources were used only if information from a higher-priority source was not available.

Inventory Data Source	Date Available	HAP Estimates Used in Urban Analyses	Comments
1. 1990 Emissions Inventory of Forty Potential Section 112(k) Pollutants [13]	March 1999	<ul style="list-style-type: none"> <li>- 40 candidate urban HAPs</li> <li>- National level emissions split into urban/rural county designations</li> </ul>	<ul style="list-style-type: none"> <li>- Best source for 40 HAP emissions, estimation technique documentation, urban/rural splits and definitions</li> <li>- Publicly available.</li> </ul>
2. Updated inventory for two section 112(c)(6) HAPs [14]	March 1999	<ul style="list-style-type: none"> <li>- PCB and HCB estimates were updated from the 4/97 112(c)(6) inventory</li> <li>- Urban/rural splits not included in database, but developed by EPA contractor for this analysis.</li> </ul>	<ul style="list-style-type: none"> <li>- Most recent data set for these 2 HAPs</li> <li>- Not documented or publicly available</li> <li>- Changes primarily reflect new data from MACT standard development</li> </ul>
3. 1993 NTI version 9801 (revised)	February 1999	<ul style="list-style-type: none"> <li>- 188 individual HAPs and category totals</li> <li>- Urban/rural splits not included in database, but developed by EPA staff for this analysis.</li> </ul>	<ul style="list-style-type: none"> <li>- Most recent compiled data set for HAPs not in 40-HAP inventory or 112(c)(6) update.</li> <li>- Publicly available on CD by written request.</li> </ul>
4. 1993 NTI version 9702 [15]	October 1997	<ul style="list-style-type: none"> <li>- Any included speciated HAPs (e.g., individual POM compounds)</li> <li>- Urban/rural splits not included in database, but developed by EPA staff for this analysis.</li> </ul>	<ul style="list-style-type: none"> <li>- Only compiled data for individual species within HAP categories.</li> <li>- Individual species estimates are artifacts of primary data sources (e.g., States or TRI). Estimates for these individual species are not reported consistently and are likely to under-represent national totals.</li> <li>- Superseded by version 9801, which lacks speciated data; no longer available.</li> </ul>

Emission data used in ranking index calculations are shown in Table 3. HAPs for which information was not available from the emission databases described above were included in this table as blanks, and emission-based indices for these substances were not calculated.

2.3.2.3 Speciation Assumptions for Inventory and Ambient Monitoring Data

The following decisions were made regarding the use of NTI emission data, based on staff judgment:

1. *Antimony*. Emission and ambient data for antimony were assumed to represent the carcinogenic

trioxide, which is thought to be the predominant form of atmospheric antimony [16].

2. *Arsenic*. Emission and ambient data for arsenic, which is released to the air mainly as arsenic trioxide and is usually found in the atmosphere as a mixture of particulate arsenite and arsenate [17], were evaluated as inorganic arsenic.
3. *Chromium*. Emission data for total chromium, which did not distinguish between the III and VI valences, were apportioned to reflect a 35% reported proportion of chromium VI [18].
4. *Lead*. Emission and ambient data for total lead were assumed to be inorganic, and paired with health RBC/RBDs for inorganic lead. Emission data for alkylated lead were paired with RBC/RBDs for tetraethyl lead in the index calculations. Alkylated and inorganic lead were scored separately.
5. *Mercury*. Emissions and ambient air concentrations of mercury were presumed to be elemental mercury, the dominant form of mercury in the atmosphere [9].
6. *Polycyclic Organic Matter*. Emission and ambient data for a group of 7 carcinogenic PAH compounds (benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) were used to represent the entire polycyclic organic matter (POM) HAP category. These data were paired with a weighted UR developed for these compounds, described in section 2.3.1.9.

#### 2.3.2.4 Bioconcentration Data

The bioaccumulation factor (BAF) and bioconcentration factor (BCF) are estimates of the ratio of the concentration of a substance that an organism will accumulate in its tissues relative to the concentration of the substance in the environment, at equilibrium. The previous draft of the risk-related ranking analysis used a database of these values obtained from the 1997 beta test version of EPA's Waste Management Prioritization Tool (WMPT). EPA received several comments noting that these BAFs and BCFs were incomplete and of inconsistent quality, and further comments requesting a more complete treatment of bioaccumulative HAPs in general. We have partially addressed these concerns by replacing the 1997 WMPT data with the database of BAFs and BCFs from EPA's recently-released 1999 version of the WMPT [19], which has been substantially expanded and improved.

The WMPT is intended to allow EPA to rank relative hazards from the list of hazardous substances regulated under the Resource Conservation and Recovery Act, and was judged to be the most comprehensive source of high-quality information for the purpose of HAP ranking. The present analysis follows the WMPT's preferences for BAFs over BCFs, and for measured values over predicted values. Among the 7 PAH compounds grouped as the POM surrogate for this analysis, measured BAFs were available only for chrysene and benz[a]anthracene. EPA assigned this measured BAF value (800 for both compounds) to the entire 7-PAH group.

BCF/BAFs used in this ranking analysis are presented in Table 3.

#### 2.3.2.5 Uncertainties in Use of Exposure Surrogates

This analysis has the following important limitations: (1) the ranking is relative rather than absolute, (2) the results cannot be interpreted as quantitative risk estimates, and (3) the emission and ambient concentration data bear some relation to human exposure, but cannot themselves be construed as exposure estimates.

The ambient monitoring database had many gaps, shown as blanks in Table 3. No measurements exist for many urban locations, and locations that were monitored were usually sampled for only a few HAPs. Measurements that do exist were taken only at specific locations and times, and cannot represent the whole spectrum of ambient concentrations. Furthermore, even perfectly accurate ambient concentrations cannot fully explain human exposure, which is influenced by complex behaviors. Finally, the ambient air measurements are subject to the same limitations as all measured data—detection limits that may be too high, and potential for errors in sampling, analysis, and reporting of data.

Most NTI emission data are from 1990, with updated information for some HAPs in some locations for 1993. This database was used to reflect a 1990 baseline, the year the Act was passed, as a baseline from which to measure future improvements, and it should not be interpreted as representing current conditions. Most emission data are predicted from emission factors and activity levels, both of which are subject to error. Even perfectly accurate emission data would be a substantially inaccurate predictor of ambient concentrations, which are also influenced by factors such as proximity of populations, site-specific parameters like stack height, meteorological conditions, atmospheric transformation of HAPs, and non-source-related background concentrations.

### 2.3.3 The HAP Ranking Process

Four distinct ranking indices were calculated, data permitting. Three of these indices were based on chronic exposure, and one on acute exposure. The three chronic indices were calculated using case 1 and case 2 dose-response information (described above). The total number of calculated “sub-indices” was seven.

As discussed above, each of these calculated indices represents only a simple surrogate measure of relative hazard that cannot be translated to absolute risk. Index values can be most accurately described as ambient concentrations and emission masses that have been adjusted to account for relative differences in the toxicity of various HAPs. They provide no information about whether emissions, ambient levels, or risks are acceptable or unacceptable.

#### 2.3.3.1 Index 1: Ambient/Acute

The ambient acute index was calculated by dividing the 95<sup>th</sup> percentile 24-hour concentration of each HAP by its risk-based concentration for acute effects. The purpose of this index was to rank HAPs by relative short-term inhalation hazard.

#### 2.3.3.2 Index 2: Ambient/Chronic

The ambient chronic index was calculated by dividing the long-term average ambient concentration of each HAP by its risk-based concentration for chronic effects. This was done separately for case 1 (RBC set at 1e-6 risk or the RfC, whichever was lower) and case 2 (RBC set at 1e-4 risk or the RfC, whichever was lower). The purpose of this index was to rank HAPs by relative long-term inhalation hazard.

#### 2.3.3.3 Index 3: Emission/Chronic/Inhalation

The NTI emission rate, in tons per year, was adjusted by dividing it by the RBC for chronic effects. As with the ambient chronic index, this was done separately for case 1 (RBC set at 1e-6 risk or the RfC, whichever was lower) and case 2 (RBC set at 1e-4 risk or the RfC, whichever was lower). The purpose of this index was to rank HAPs by relative long-term inhalation hazard. Although emission

data represent a less direct surrogate for exposure than ambient data do, this index is valuable because the emission database is far more complete in terms of numbers of HAPs and locations considered.

#### 2.3.3.4 Index 4: Emission/Chronic/Oral

The NTI emission rate, in tons per year, was adjusted by multiplying it by the bioconcentration factor and dividing it by the oral risk-based dose (RBD) for chronic effects. As with the other chronic indices, this was done separately for case 1 (RBD set at  $1e-6$  risk or the RfD, whichever was lower) and case 2 (RBD set at  $1e-4$  risk or the RfD, whichever was lower). The purpose of this index was to rank HAPs by relative potential hazard due to food-chain bioaccumulation.

## 2.4 Combination of Individual Ranking Indices

The seven sub-indices described above were calculated for each HAP, to the extent that data permitted. Raw scores (Table 4) were normalized to a scale of 0-100 within each sub-index (Table 5), with 100 representing the most hazardous score and 0 representing no hazard. Scores that could not be calculated because of missing data were treated as blanks, not as zeros.

This system of normalizing sub-index scores to the same 0-100 scale was adopted in response to comments received on the September 1998 proposed HAP selection protocol. The earlier method ranked HAPs within each sub-index, then averaged the ranks. Commentors noted that this method obscured quantitative differences in magnitude among HAPs, and artificially increased the importance of sub-indices having the fewest calculated results.

EPA agreed with these comments, and revised the methodology to use normalized scoring. The normalized 0-100 scale preserves differences in relative magnitude of hazards. For example, if the highest-scoring HAP has a raw index score ten times higher than the second HAP, the two HAPs would have been ranked 1 and 2 under the old system. Under the new system, their normalized scores would be 100 and 10. The system also treats all sub-indices equally, regardless of how many HAPs are scored. For example, under the old system only about 20 HAPs could be scored for the ambient/acute index<sup>2</sup>, so the least hazardous HAP had a rank of about 20. However, more than 150 HAPs were scored for the emission/chronic/inhalation index. Thus, the HAP that ranked 20<sup>th</sup> out of 150 in this index was probably much more important than the HAP ranking 20<sup>th</sup> of 20 in the ambient/acute index. This system artificially deflated the importance of data-rich sub-indices for which many HAPs were scored. The new scoring system removes this artificial bias.

Normalized scores were averaged across the seven sub-indices, for each HAP. Average scores and the overall HAP rank are shown in Table 5. Figures 1 and 2 show the 60 HAPs that ranked highest in this exercise, sorted in order of average score. Individual sub-index scores appear as points on these figures, except for blanks caused by data gaps.

## 3. Results and Selection of HAPs Proposed for Listing

Results for all three ranking analyses—(1) the risk-related ranking indices, (2) the CEP urban analysis, and (3) the review of existing risk assessments and hazard rankings—are combined and summarized in Table 6. In selecting the urban HAPs for the integrated strategy, we compared the results of the three separate analyses, and selected those HAPs for which a publicly reviewed baseline national emissions

---

<sup>2</sup> Note: In the revised HAP ranking, we have been able to score over 50 HAPs for the ambient/acute index.

inventory was available (under CAA section 112(k) or 112(c)(6)), and which was either:

1. Identified by at least two of the three analyses (regardless of area source contribution), or
2. Identified by at least one of the three analyses, with an area source contribution to total emissions of at least 25 percent.

This second criterion was set in recognition of the area source emphasis of this integrated strategy. These criteria produced an integrated list of 33 “urban HAPs” (Table 6). Section 112(k) of the CAA requires us to identify not less than 30 “area source HAPs” that pose the greatest threat to public health in the largest number of urban areas, as the result of emissions from area sources.

To identify these 30 area source HAPs, we ranked the list of 33 urban HAPs by percent contribution to national urban emissions from area sources and selected the 30 urban HAPs with the greatest area source contributions. The remaining three urban HAPs (coke oven emissions, 1,2-dibromoethane, and carbon tetrachloride) have less significant emissions contributions from area sources, and are not among the 30 area source HAPs considered for area source category listing.

---

#### 4. References

1 Environmental Protection Agency (1998). Draft Integrated Urban Air Toxics Strategy to Comply With Section 112(d), 1129c)(3) and Section 202(l) of the Clean Air Act; Notice. 63 FR 49240, September 14, 1998.

2 Environmental Protection Agency (1998). Integrated Risk Information System. Office of Research and Development. Updated regularly, available on-line at <http://www.epa.gov/iris/>.

3 Environmental Protection Agency (1997). Health effects assessment summary tables. FY 1997 update. Office of Solid Waste and Emergency Response. Document No. EPA-540-R-97-036.

4 Agency for Toxic Substances and Disease Registry (1998). Minimal risk levels for hazardous substances. Updated regularly, available on-line at <http://www.atsdr1.atsdr.cdc.gov:8080/mrls.html>.

5 Environmental Protection Agency (1997). Proposed acute exposure guideline levels. National Advisory Committee for Acute Exposure Guideline Levels for Hazardous Substances. Federal Register 62: 58839-58851.

6 American Industrial Hygiene Association (1998). Emergency Response Planning Guidelines and Workplace Environmental Exposure Level Guides Handbook. AIGH, Fairfax, VA.

7 National Institute for Occupational Safety and Health (1994). Documentation for Immediately Dangerous To Life or Health Concentrations (IDLHs). National Technical Information Service Publication No. PB-94-195047, available on-line at <http://www.cdc.gov/niosh/idlh/intridl4.html>.

8 US Environmental Protection Agency, Federal Emergency Management Agency, and Department of Transportation (1987). Technical guidance for Hazards Analysis: Emergency planning for extremely hazardous substances. EPA-OSWER-88-0001.

9 Environmental Protection Agency (1997). Mercury study report to Congress. Office of Air Quality

---

Planning and Standards. Document No. EPA-452/R-97-003.

10 Environmental Protection Agency (1994). Interim draft toxicity equivalence factors for polynuclear aromatic hydrocarbons. National Center for Environmental Assessment, Cincinnati, OH.

11 Agency for Toxic Substances and Disease Registry (1998). Toxicity profile for selenium (update). ATSDR, Atlanta, GA.

12 Smith, R.L. ((1996). Risk-based concentrations: prioritizing environmental problems using limited data. *Toxicology* 106: 243-266.

13 Environmental Protection Agency (1997). Section 112(k) - Urban air toxics program development of air emissions inventory. Available on-line at <http://www.epa.gov/ttn/uatw/112kfacs.html>.

14 Environmental Protection Agency (1998). 1990 Emissions Inventory of Section 112 (c)(6) Pollutants: Final Report. Available on-line at <http://www.epa.gov/ttn/uatw/112c6/112c6fac.html>.

15 Environmental Protection Agency (1997). National Air Quality and Emissions Trends Report, 1996. Office of Air Quality Planning and Standards. Document No. EPA-454/R-97-013.

16 Agency for Toxic Substances and Disease Registry (1992). Draft toxicological profile for antimony. U.S. Dept. of Health & Human Services, Public Health Service, ATSDR, Atlanta, GA.

17 Agency for Toxic Substances and Disease Registry (1998). Draft toxicological profile for arsenic. U.S. Dept. of Health & Human Services, Public Health Service, ATSDR, Atlanta, GA.

18 Agency for Toxic Substances and Disease Registry (1998). Draft toxicological profile for chromium. U.S. Dept. of Health & Human Services, Public Health Service, ATSDR, Atlanta, GA.

19 Environmental Protection Agency (1998) Waste Minimization Prioritization Tool Spreadsheet Document for the RCRA Waste Minimization PBT Chemical List Docket (# F-98-MMLP-FFFFF). Office of Solid Waste and Emergency Response. Available on-line at [www.epa.gov/wastemin](http://www.epa.gov/wastemin).