Draft Roadmap on Benefits Assessment for Air Toxics: Options for Discussion

U.S. Environmental Protection Agency
Office of Air and Radiation
Office of Research and Development
Office of Policy, Economics and Innovation

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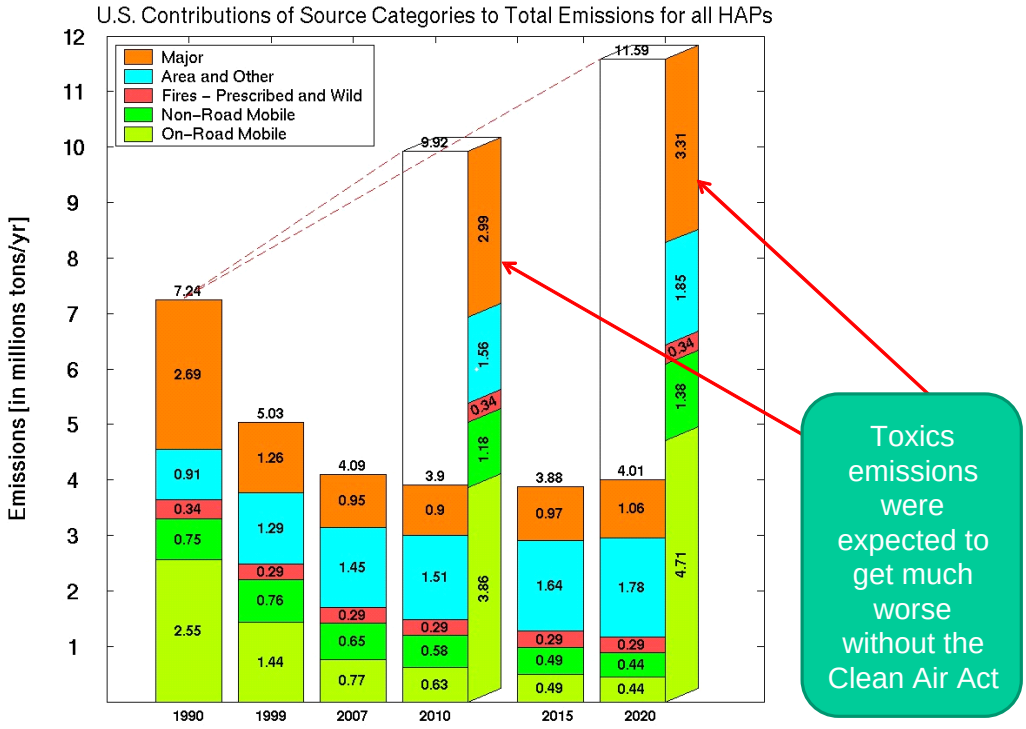
Workshop on Air Toxics Benefits
Washington, DC
April 30 – May 1, 2009

Section I – Introduction

The U.S. Environmental Protection Agency (EPA) regulates hazardous air pollutants (HAPs), also referred to as air toxics, from both stationary and mobile sources. The Clean Air Act Amendments of 1990 (CAAA) list 187 air toxics (e.g., benzene, chromium compounds) and require EPA to set "Maximum Achievable Control Technology" (MACT) standards to reduce emissions of air toxics from major stationary sources, which range from dry cleaners to petroleum refineries. After setting these standards, EPA evaluates the remaining risks to determine if further regulation in warranted. For smaller stationary sources (e.g., gasoline distribution, auto body refinishing), EPA is required to set technology standards known as "Generally Available Control Technology" (GACT). And to control mobile source air toxics, EPA is required to set technology and fuel standards for motor vehicles.

Since 1990, EPA has made significant progress in reducing air toxics. (See Figure 1.) We have set standards for 174 major sources of air toxics, resulting in 1.7 million tons of toxics reduced annually. Through our Mobile Source Air Toxics rule and other efforts, including voluntary programs such as diesel retrofits, we continue to realize major reductions of toxics from mobile sources. We are completing standards to reduce emissions from smaller stationary sources and have voluntary programs to reduce exposures to indoor air toxics.

Figure 1: Progress in Reducing National Air Toxics Emissions from 1990 to 2007 and Beyond
(Source: Strum et al., 2006)



While we can point to this progress in reducing air toxics, EPA does not currently have a peer-reviewed framework for analyzing the direct air-toxic related monetized benefits of our regulatory programs. Section 812 of the CAAA requires EPA to perform periodic, comprehensive analyses of the total costs and total benefits of programs implemented pursuant to the Clean Air Act (CAA). EPA has completed two of these analyses: a retrospective analysis in 1997 of the original CAA covering the period 1970 to 1990, and a prospective analysis in 1999 of the incremental costs and benefits of the CAAA over the period 1990 to 2010. In both of these studies, it has been difficult to estimate benefits of reduced air toxics emissions, for reasons explored in more detail below. In the case of evaluating specific regulations (e.g., in accordance with Executive Order 12866 to estimate the costs and benefits of major rules), we have used peer-reviewed methods to assess the co-benefits from reducing criteria air pollutants (e.g., ozone) along with HAPs. However, this is largely seen as an unsatisfactory accounting of the benefits of air toxics regulations.

In June 2000, EPA and its Science Advisory Board (SAB) jointly held the SAB/EPA Workshop on the Benefits of Reductions in Exposure to Hazardous Air Pollutants. The goal of that meeting was to convene experts from different disciplines and different backgrounds to discuss ideas for dose-response assessment methods for air toxics that would be appropriate for use in assessing benefits associated with air toxics control measures. For an agenda and papers that were presented at that workshop, see http://yosemite.epa.gov/sab/SABPRODUCT.NSF/34355712EC011A358525719A005BF6F6/$File/ecwkshp02001%2Bappa-g.pdf.

The 2000 Workshop also highlighted the complexity of integrating economics and risk assessment data to develop a benefits assessment. No consensus was reached, although participants discussed alternative approaches that could be considered and possible research directions.

This Draft Roadmap has been developed as part of a follow-on meeting to the 2000 workshop. The 2009 Workshop on Air Toxics Benefits on April 30 and May 1 is sponsored by EPA's Office of Air and Radiation (OAR), Office of Research and Development (ORD) and Office of Policy, Economics and Innovation (OPEI). The workshop will bring together experts from EPA and from outside organizations to discuss relevant approaches for air toxics benefits assessment. The Draft Roadmap, which will be discussed at the workshop, is intended to provide background information as well as possible options for moving forward on benefits assessment. It should not be considered final Agency policy.

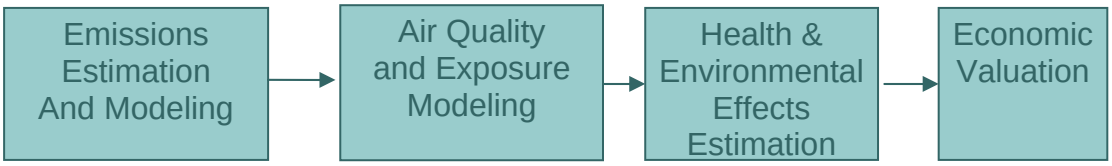
This Draft Roadmap is organized as follows:

- Section I provides an introduction to the Roadmap and background information on benefits assessment as well as specific challenges for air toxics benefits assessment.
- Section II briefly summarizes relatively recent research methods and studies that will be discussed at the 2009 workshop;
- Section III provides EPA's proposed approach for moving forward in the near term on air toxics; and
- Section IV proposes considerations for a longer-term approach on air toxics benefits.

Background on Cost-Benefit Analysis and Air Toxics

EPA has developed extensive tools for cost-benefit analysis, which OAR uses primarily for analyzing the economic impacts of regulations on criteria pollutants. As mentioned briefly above, we estimate benefits for Regulatory Impact Analyses (RIAs) for rules that have a significant impact on the economy, based on the general analytical steps outlined in Figure 2. For major air toxics rules, we have estimated co-benefits based on reductions in fine particulate matter (referred to as $PM_{2.5}$) or ozone rather than those based directly on reductions of air toxics. For example, in the analysis for the final Mobile Source Air Toxics Rule, we estimated the $PM_{2.5}$-related co-benefits associated with the vehicle standards, even though $PM_{2.5}$ is not considered an "air toxic." Also, in the first retrospective Section 812 Study looking at benefits from 1970-1990, EPA estimated benefits from vinyl chloride reductions, but was criticized by the SAB for developing unrealistic estimates. In the second report, looking prospectively from 1990-2010, EPA did not include any benefits assessment for air toxics, but provided a cost assessment of the program. Plans for the third report, which will estimate costs and benefits of the CAA from 1990-2020, include a case study on benzene reductions in Houston (discussed in Section II).

Figure 2. Typical Analytical Steps for Estimating Benefits



Challenges for Air Toxics Benefits Assessment

Benefits analysis is necessarily based on many assumptions and is therefore subject to significant limitations. Some aspects can be easily quantified while others can be discussed qualitatively. This section provides a brief summary of some of the specific challenges related to air toxics benefits assessment. By outlining these challenges, we hope to provide a common understanding of the impediments and opportunities for addressing these issues. For example:

- *Emissions.* EPA maintains an inventory most of the 187 air toxics listed in Section 112 of the CAA, although the data are often less reliable than the inventory for the six criteria pollutants. The National Emissions Inventory (NEI) is updated every three years and provides information from major stationary sources, smaller "area" stationary sources, on-road and non-road mobile sources and other sources (e.g., wildfires). EPA also publishes an annual Toxics Release Inventory (TRI) that requires facilities above thresholds to report pollution releases (including most of the air toxics) to air land, and water. In addition, EPA has a national program for air toxics monitoring, with a limited number of national trend sites for some HAPs, community and local-scale monitoring projects and grants to states and local governments for discretionary monitoring. Finally, regions and states may have additional, more detailed air toxics data. For some source

sectors, there are limited toxic emission data. In sum, while we have several sources of air toxics data, the data are often incomplete and less reliable than criteria air pollutant emissions data. There are also greater uncertainties when projecting future emissions, given the challenges in predicting activity levels using economic data and limited information on likely impacts of advanced technology

- *Air quality and exposure modeling.* EPA's current National Air Toxics Assessment (NATA) (with 2002 data) uses the Assessment System for Population Exposure Nationwide (ASPEN) to estimate ambient concentrations at the census tract level due to emissions from mobile and area sources, the AMS/EPA Regulatory Model (AERMOD) to estimate ambient concentrations at the census block level due to emissions from point sources, and the Hazardous Air Pollutant Exposure Model (HAPEM) to estimate inhalation exposures at the census block level, taking into account ambient exposures in various microenvironments. NATA uses these exposure modeling results to estimate lifetime inhalation risks for both cancer and non-cancer effects from outdoor exposure to air toxics. It does not estimate ingestion risks (which are important for certain air toxics like mercury or lead), nor does it include the impacts of indoor sources, accidental releases, natural disasters, or transport of pollution from countries outside the United States.

  EPA is currently looking to improve NATA-type modeling applications in the future by using state-of-the-art photochemical models for reactive air toxics and state-of-the-art deposition modeling for pollutants that deposit from the air and persist in the soil and water. For many individual air toxics regulations, such as residual risk assessments, a Gaussian dispersion model such as AERMOD is used to estimate ambient levels near the sources that are subject to the specific regulation. For criteria pollutants, we have national-scale air quality models (e.g., Community Multiscale Air Quality (CMAQ) model) that predict ambient pollutant concentrations, considering atmospheric chemistry, generally at a relatively coarse resolution (e.g., 36 km, 12 km), with the option of incorporating finer grid resolution (down to 1 km). The coarse level of resolution is generally suitable for national-level regulatory support since criteria pollutant benefits are calculated based on grid-cell averaged concentrations applied to the population within a modeled grid-cell. The same grid-based models can be used to predict concentrations of some air toxics at the same grid resolution, which may or may not be suitable depending upon the risk characterization that is desired. While the Gaussian models used for current air toxic assessments (ASPEN and AERMOD) capture near-field gradients in concentrations, they do not accurately represent the complex chemistry that can be significant for some pollutants. In many cases, finer-resolution modeling may be more appropriate for air toxics benefits analysis, particularly when addressing equity concerns, although such modeling is generally not feasible for national-level analyses. For example, local hot spots may lead to small groups being exposed to high concentrations of air toxics, even if, at the standard resolutions, the average exposures are estimated to be low. As a result, large-scale modeling may indicate far fewer health effects than finer-scale modeling. Thus, future air toxic assessments, such as the 2005 NATA will employ models such as CMAQ to capture the chemistry associated with highly reactive

pollutants, yet maintain Gaussian models such as AERMOD to capture the local gradients of ambient concentrations and exposures.

Also, as noted below, community epidemiology studies based on ambient concentrations of air toxics are rare, and their interpretation is complicated by the presence of numerous pollutants with similar health endpoints. Given limitations in characterizing dose-response relationships based on analyses of ambient concentrations and health endpoints, assessing changes in risk or disease incidence rate for individual regulations of particular air toxics may require an assessment of exposures in terms similar to available health studies (e.g., ppm-years, mg/kg-day). For many air toxics, there are several factors that influence this relationship between ambient concentrations and exposure:

- Air toxics vary widely in their sources, photochemical reactivity, and scale of spatial variability. Therefore, different modeling techniques to estimate exposures may be required for different pollutants or classes of pollutants (e.g., aldehydes vs. polycyclic aromatic hydrocarbons). For some pollutants, a simple Gaussian plume model may be an appropriate method of estimating ambient levels, whereas for others, it may be important to consider including other complex phenomena, such as atmospheric chemistry (e.g., photochemical reactions) or deposition. Thus, it may be difficult, given the current state of understanding of the health effects of toxics, to estimate the benefits of reducing a subset of multiple toxics because of their interactions with each other, and because of limitations in our understanding of their interactions with remaining toxics.

- Exposure assumptions may differ significantly from actual individual exposures. The typical assumption that people are exposed to the air outside their residences for 24 hours a day for a lifetime simplifies the analysis but introduces error in the effects estimates, particularly when considered at the individual level. As most individuals move about their communities throughout the day (and in wider areas throughout a lifetime), actual individual exposures may differ significantly from modeled ones. Indoor sources may be important considerations, depending on the choice of a dose metric and the shape of a dose-response curve. While most of EPA's regulations result in reductions of outdoor air toxics, some indoor air toxics are influenced by emission regulations (e.g., lawn equipment in attached garages). Current modeling of exposure based on ambient measure may miss the effects of these sources. On the other hand, some exposure to some outdoor pollutants is significantly attenuated indoors.

- Finally, addressing acute exposures may be difficult, as extreme exposure events may occur periodically resulting from numerous low-probability events occurring at once (e.g., toluene exposure while operating recreational equipment such as snowmobiles). As such, different approaches to exposure assessment may be required among different air toxics. Looking only at average or cumulative exposures may miss what may be significant acute exposures.

In summary, the connection between emissions of toxics and human exposure is complex and often difficult to estimate. The gaps in our understanding may lead to significant underestimation of the effects of air toxics.

- *Health and environmental effects estimation.* For several criteria pollutants, we have extensive human clinical or epidemiological data on health effects – such as hospital admissions and heart attacks in the population as a whole – at ambient exposure levels. This information obviates the need to extrapolate from high exposure levels to environmental exposure levels, from one exposure route to another, or from animals to humans. For the large majority of air toxics, most toxicity information from EPA's Integrated Risk Information System (IRIS) and other sources is based on animal studies, with substantial uncertainty in how to interpret those data for humans given differences in toxicokinetics and modes of action. The relatively smaller epidemiological data set for air toxics is largely based on relatively high occupational exposures (e.g., for benzene). There is also a wide range of cancer and non-cancer effects associated with the various air toxics, with a resulting wide range of (uncertain) time scales over which different air toxics may affect health. The shape of the dose-response curve and choice of a dose-metric may also vary by pollutants and health endpoint. Moreover, partial lifetime probabilities of cancer for different age groups, changes in survival rates over time, and cessation lag estimates do not exist for most air toxics. Finally, available cancer dose-response estimates are often upper-bound estimates, as it is difficult if not impossible to define a "best" estimate dose-response curve. For non-cancer, EPA and other agencies typically identify a concentration level that is thought to be without appreciable risk; such an approach does not easily lend itself to economic analysis. For all these reasons, characterizing the health effects of air toxics at ambient levels can be subject to a high level of uncertainty and difficult to adapt to economic benefits assessments.

- *Economic valuation.* Standard economic analysis for pollution reduction starts with estimates of reduced mortality and morbidity effects (and other non-health effects as appropriate). The "deaths avoided" are multiplied by a "value of a statistical life" to estimate the benefits of reduced mortality; reduced morbidity is, similarly, multiplied by either an estimate of willingness to pay (WTP) to avoid the illness or an estimate of the cost associated with the treatment of that illness (COI). Applying this method to air toxics leads to a number of potentially significant omissions of benefits. First, many air toxics tend to vary significantly from area to area; often a relatively small number of people can be exposed to high levels of air toxics while the average exposure to the general population is much lower; as discussed above, health effects may therefore be underestimated. Additionally, an individual's views about the risks from toxics may differ from what is known about the health and epidemiological effects. As a result, a person's WTP to reduce risks may differ from the standard value of a statistical life. Also, the standard value of a statistical life, usually based on risk-wage studies, does not necessarily measure accurately the WTP for risk reduction for children and seniors, two groups likely to be disproportionately affected by exposures to toxics (or other pollutants, for that matter). Finally, individuals may have greater WTP to reduce risk from some causes of mortality than others (e.g., a possible "cancer premium"). For any of these reasons, a person's WTP for a risk reduction may differ from the value derived in

conventional ways and may result in potential underestimation of benefits. For air toxics, we would like to identify methods for estimating WTP for risk reductions when distributional concerns and subjective beliefs are major components of the benefits to be achieved.

- *Efficiency vs. Distributional/Equity Considerations*. As noted above, as opposed to NAAQS which are focused on overall public health objectives, regulations focused on air toxics seek not only to improve overall public health, but also to address disproportionate risk in a segment of the population. In fact, the Clean Air Act specifically states that if the initial set of air toxics standards issued by EPA does not "reduce lifetime excess cancer risks to the individual most exposed to emissions from a source in the category or subcategory to less than one in one million," the EPA Administrator must issue additional standards for sources to protect public health with an adequate margin of safety [see CAA §112 (f)(2)]. In these cases, protecting the most-affected individuals rather than maximizing efficiency may be the guiding objective. Existing cost-benefit techniques are not well-suited to addressing distributional and/or equity considerations. Economic efficiency analysis serves the purpose of identifying the opportunity costs associated with achieving this protection goal, but it cannot substitute for concerns over the distribution of those effects. There have been some recent efforts to develop methods to address tradeoffs between equity and total health benefits (see Levy et al, 2007; Levy et al, in press) but these methods have not been adopted in EPA regulatory analyses to date.

Section II – Sample Research Methods for Air Toxics Benefits Assessment

EPA and others have been working on creative ways to assess benefits related to reductions of air toxics. This section describes some of the case studies that will be discussed at the workshop. This is not a comprehensive summary.

Benzene Case Study – Health Benefits of Benzene Reductions in Houston, 1990-2020.

Industrial Economics, Inc. (IEc) (2008), as part of EPA's upcoming 812 Study, developed a methodology for estimating the benefits of reducing benzene for a three-county area around Houston, Texas. This case study comes out of EPA's Science Advisory Board (SAB) suggestion that EPA look at one data-rich air toxic to begin developing a methodology that could possibly be applied more widely. The methodology includes quantification of changes in individual risk using air quality and exposure models, with and without the CAAA controls, and translating these changes into monetized benefits. Modeling was done at a high level of resolution to capture extremes in the exposure distribution. Highway vehicle emissions were modeled at the individual link level, and air quality modeling was done with American Meteorological Society/U.S. EPA Regulatory Model (AERMOD) with receptors in each census block. Time-weighted average benzene exposure concentrations for the study populations were generated using HAPEM. This case study was reviewed by the SAB in March 2008 and will be included in EPA's Section 812 Report *The Benefits and Costs of the Clean Air Act, 1990-2020* to be released later in 2009.

The case study considered the change in estimated leukemia cases associated with reductions in benzene emissions using a life-table approach. The life-table approach assessed age-specific leukemia and leukemia mortality risks within each census tract in each year of the study, based on county-specific background rates of leukemia mortality and morbidity, age-specific benzene exposure data from HAPEM, and a dose-response function derived from epidemiological studies of benzene-exposed workers in Pliofilm manufacturing plants. IEc modeled benzene exposures and health impacts under two scenarios – one assuming all regulatory programs affecting benzene emissions were enacted in response to the 1990 CAA Amendments (With-CAAA) and one assuming no additional benzene controls beyond the requirements existing in 1990 (Without-CAAA). The difference in emissions between these two scenarios provided the basis for the reduction in benzene concentrations and related health benefits due to the CAAA. This model also took into account assumptions regarding latency and cessation lags for benzene-induced leukemias (e.g., the number of years after exposure during which leukemia risk remains elevated before reaching a new, lower steady-state in the exposed population).

The study does not include the full scope of reductions in benzene emissions (e.g., for programs established after the case study control scenario was fixed, especially the Mobile Source Air Toxics Rule). Potential health effects associated with benzene exposure that were not included in the quantitative results include other cancers, such as Hodgkin's Lymphoma, and non-Hodgkin's Lymphoma, and multiple myeloma; and potential non-cancer effects related to various hematological abnormalities, including aplastic anemia. Co-benefits of reducing toxics, including reductions in ozone and particulate matter levels, are captured in the overall section 812 study but are not incorporated in the case study.

In their review of the case study, the SAB "laud[ed] the OAR's air quality and exposure modeling, the life table approach for estimating health benefits and supplemental analyses of individuals in high-exposure environments. Given these achievements, the case study offers a reasonable, if qualified, estimate of health benefits." While the SAB believed the case study successfully synthesized best practices and implemented the standard damage function approach to estimating the benefits of reduced benzene, the members were not optimistic that such an approach could be repeated on a national scale due to the high level of spatial resolution or extended to many of the other 187 air toxics due to insufficient epidemiological data. Rather, they urged EPA to consider alternative approaches to estimate the benefits of air toxics regulations considering multi-pollutant approaches.

Acrolein Dose-Response

Woodruff et al. (2007) conducted an analysis of acrolein risks for respiratory effects, based on dose-response modeling of animal data. The authors selected two endpoints from a study by Costa et al. (1986) of rats exposed to acrolein in air for 62 days. Each endpoint displayed reductions in lung function significantly related to dose of acrolein. Acrolein concentrations were converted into human equivalent concentrations using standard methods, and the data for each endpoint were modeled using EPA's Benchmark Dose Software.

A key feature of the animal lung function data is that they are measured on a continuous scale – similar to other measures from the toxicological and medical fields such as blood pressure, organ weight, or hormone levels. Thus, the dose-response models derived from the Costa et al. data estimate the change in the lung function parameter of a 1 microgram per cubic meter (ug/m^3) change in acrolein concentration.

To use the models for estimation of effects, a level for each parameter that is considered to be adverse is needed. In this analysis, the baseline adverse outcome prevalence was defined as values ≥ 90th percentile of the distribution in control animals. Therefore, 10% of unexposed individuals would, by definition, experience adverse outcomes. The analysis then estimates the increase in the proportion of the population experiencing adverse outcomes above the baseline 10%. Sensitivity analyses examined alternate definitions of the adverse level.

At median acrolein concentrations (NATA estimates for 1999), the dose-response modeling provided an estimated incidence of 2.5 additional cases per 1,000 exposed people for one of the selected lung function measures. Estimated risks for the second modeled lung function measure were much lower.

The endpoints selected for modeling from the Costa et al. study in rats are consistent with human lung function changes seen in obstructive lung diseases. However, these lung function measures in the rat are not interpreted to strictly correspond to specific effects in human lungs, but rather are considered more general indicators of potential for decrements in human lung function. Further analysis and consultation with lung function experts may be able to establish a stronger understanding of the potential human health consequences of the lung function decrements observed in the animal study.

Lead

EPA estimated the human health and welfare benefits associated with attaining alternative lead National Ambient Air Quality Standards (NAAQS). The principal goal of a NAAQS Regulatory Impact Analysis (RIA) is to provide the public with a best estimate of the expected benefits and costs of attaining a new standard. While EPA followed the same basic analytical steps as an ozone or $PM_{2.5}$ benefits analysis, the analysis was complicated by key data gaps described below.

This analysis followed four basic steps. First EPA estimated the change in ambient lead resulting from attainment of alternate NAAQS levels, relative to baseline ambient lead levels in 2016. The environmental Benefits Mapping and Analysis Program (BenMAP) was used to develop an air quality surface by interpolating lead monitor values. BenMAP then applied Woods and Poole population projections to estimate census-block level ambient lead population exposures. Having estimated ambient exposure, the second analytic step was to estimate the change in blood lead, which is the biomarker of interest. EPA applied air-to-blood ratios to quantify the change in blood lead as a function of exposure to ambient lead. The third step was to apply epidemiological studies found in the Criteria document (U.S. EPA, 2006) to quantify the population-level change in IQ points. The fourth and final step was to monetize the change in IQ points by using economic valuation functions that measured the foregone lifetime earnings per lost IQ point.

As part of a sensitivity analysis to test the importance of key model inputs, the analysis concluded that total benefits were highly sensitive to the air quality estimates, the discount rate and the air-to-blood ratio. In particular, the selection of the air-to-blood ratio was a point of contention during interagency review. These ratios predict geometric blood lead levels due to direct lead exposure via inhalation as well as indirect exposures via ingestion of dust and soils contaminated by lead deposition, based on comparisons of historical data on lead in ambient air and measured or modeled geometric mean blood lead levels in an exposed population. Because these ratios may vary based on historical lead exposure, a key question was whether EPA selected the air-to-blood ratio that was most appropriate for the exposed (future) population.

This approach to estimating ambient lead-related benefits may serve as a useful model for future analyses of non-inhalation air toxics. To the extent that sufficient ambient air quality modeling or monitoring data are available, and the historical exposure of populations of interest are well characterized, this method may be viable.

Using Dose-Equivalence Relationships to Estimate Costs of Intoxication

Evaluating the economic benefits of reducing exposure to a pollutant requires estimating the economic damages associated with exposure to the pollutant. Economic damages are not known for most chemicals, but may in some cases be estimated from the economic damages related to exposure to well-studied chemicals. For example, a great deal is known about the health effects and economic damages associated with ethanol ingestion. Because many air toxics have effects on the nervous system that are very similar to those of ethanol, the benefits of reducing exposure to those air toxics may be estimated from knowledge of the dose-effect functions for the air toxics and for ethanol and from data on the economic damages associated with consumption of alcoholic beverages.

To determine the feasibility of this approach, scientists in ORD's National Health and Environmental Effects Research Laboratory (NHEERL) analyzed published data on the effects of ethanol and toluene, a HAP with a rich toxicological database. ORD developed a procedure to estimate the monetary cost of toluene exposure, based upon its acute behavioral effects in relation to effects of ethanol, and then compare quantitative dose-effect functions for the two chemicals.

Behavioral effects of toluene and ethanol in human subjects were quantified by using a meta-analysis of studies from the peer-reviewed literature that measured the effects of the chemicals on choice reaction time (CRT). CRT is measured in tests that require a subject to choose among two or more alternatives and make a discrete choice response as quickly as possible. Because the acute effects of ethanol and toluene are best predicted by the internal dose (concentration in blood or brain at the time of behavioral measurement), effects on CRT were related to the internal doses of the two chemicals. Physiologically-based pharmacokinetic (PBPK) models were used to estimate these doses from exposure parameters provided in the published studies. Behavioral effects were converted to a common metric (proportion of baseline) to make them quantitatively comparable.

The estimated effects of toluene were then compared to the estimated effects of ethanol by deriving a dose-equivalence equation (DEE). The DEE defines a function relating equivalent doses of toluene and ethanol on the basis of equal effect magnitude (of CRT in this case). Because the DEE is a continuous, quantitative relationship (with confidence limits), it can be used to calculate an internal dose of ethanol that slows CRT by an amount equal to the slowing produced by any internal dose of toluene. Because the economic damages associated with an internal dose of ethanol (blood alcohol) can be monetized (for example, by its relationship to the probability of an automobile crash), the cost of that internal dose toluene can be monetized as well. Finally, because of the well-known relationship between inhalation of toluene and its internal dose, PBPK models can be used to generate exposure scenarios of toluene (concentration and duration of exposure) that will lead to that internal dose.

In sum, this method provides a way to monetize the benefits of any acute toluene exposure reduction scenario in the blood. Of course, there are several uncertainties that must be considered when using this method, including the quality of data and the extent of mechanistic similarity between the observed effects of the two compounds. That is, whereas the computations involved in the DEE are empirical, interpretation of the predictions of effect in the 'target' chemical depend upon knowledge of the modes of action of the two chemicals in the DEE (Benignus, et al., 2005). There is strong evidence that toluene and ethanol act on the nervous system in a similar manner, and that the suite of effects of the two chemicals are similar, so estimates by the DEE for these two chemicals can be made with relatively high confidence. Given that several other HAPs (e.g., perchloroethylene, trichloroethylene, and 1,1,1-trichloroethane) act via a similar mode of action to ethanol and toluene, and cause similar acute effects, this method may generalize well within this class of HAPs. In other cases, where mode of action is less well known, or unknown, confidence in predictions from a completely empirical DEE will necessarily be lower. It may also be possible to use this method for other health endpoints and for comparison of HAPs to compounds other than ethanol, but EPA would have to investigate this further.

Section III. Options for Near-Term Approaches to Air Toxics Benefits

In this section, we cover potential near-term approaches for assessing benefits from air toxics reductions. First, we discuss focusing on a limited number of HAPs based on available dose-response and exposure information, which will hopefully provide concrete examples for discussion and lay the foundation for considering various approaches to benefits assessment. Second, we outline four potential methods for assessing air toxics benefits and discuss the pros and cons to each of them.

Focus on Limited Number of HAPs

OAR has always focused on a limited number of HAPs, partly because of the requirements of the CAA and partly because a "worst first" approach supports a more efficient use of resources. In order to choose which HAPs to focus on in the near term from a benefits perspective, we continue to rely on existing dose-response and exposure information, evaluating as many HAPs as possible.

Dose-response assessments. OAR looks to ORD, the Agency for Toxic Substances and Disease Registry (ATSDR), and other agencies to develop and review dose-response assessments. OAR participates in reviews of these assessments and packages and disseminates dose-response information in the form of HAP information web pages and dose-response tables (e.g., see http://www.epa.gov/ttn/atw/hlthef/hapindex.html and http://www.epa.gov/ttn/atw/toxsource/summary.html). These tables are a product of a much more complete system, the Air Toxics Health Effects Database (ATHED), developed by OAR's Office of Air Quality Planning and Standards (OAQPS) about ten years ago. OAQPS and ORD have cooperated in expanding ATHED to include more substances and acute dose-response values, and we're currently discussing how to use it to produce more useful outputs and disseminate it more widely.

Exposure assessments. OAQPS has combined dose-response values with exposure estimates in several ways to inform our prioritization of HAPs and sources. As part of the Integrated Urban Air Toxics Strategy submitted to Congress under the CAAA, for example, EPA had to determine the "30 hazardous air pollutants that present the greatest threat to public health in the largest number of urban areas." We used a toxicity-weighted scoring system that included both inhalation and indirect exposures to select the list of ultimately 33 urban HAPs of greatest concern (Table 1). This list was later validated by the 1999 NATA, which considered the full HAP list and modeled both dispersion and receptor behavior, but confirmed that our urban HAP list was well-considered.

We have also used a toxicity-weighted scoring system, optimized for ingestion exposure, to select 14 persistent and bioaccumulative HAPs (Table 2) recommended for detailed multipathway risk assessments in the Risk and Technology Review (RTR) program. We are in the process of developing *de minimis* emission limits for these HAPs.

Table 1. Urban HAPs	
Acetaldehyde	Ethylene oxide
Acrolein	Formaldehyde
Acrylonitrile	Hexachlorobenzene
Arsenic compounds	Hydrazine
Benzene	Lead compounds
Beryllium compounds	Manganese compounds
1, 3-Butadiene	Mercury compounds
Cadmium compounds	Methylene chloride
Carbon tetrachloride	Nickel compounds
Chloroform	Polychlorinated biphenyls (PCBs)
Chromium compounds	Polycyclic organic matter (POM)
Coke oven emissions	Quinoline
Dioxin	1, 1, 2, 2-Tetrachloroethane
Ethylene dibromide	Perchloroethylene
Propylene dichloride	Trichloroethylene
1, 3-Dichloropropene	Vinyl chloride
Ethylene dichloride	

Table 2. Persistent and Bioaccumulative HAPs
Cadmium compounds
Chlordane
Chlorinated dibenzodioxins & furans
DDE
Heptachlor
Hexachlorobenzene
Hexachlorocyclohexane (all isomers)
Lead compounds
Mercury compounds
Methoxychlor
Polychlorinated biphenyls
Polycyclic organic matter
Toxaphene
Trifluralin

HAPs lacking dose-response assessments. There are still 47 HAPs for which we have emission data but no dose-response assessment. To estimate the possible relative importance of these HAPs, we prepared a toxicity-weighted emission analysis that assigned median and 95th percentile dose-response values and compared the emission scores with those of HAPs that have dose-response values. At least five HAPs (Table 3) without a dose-response assessment appear to have the potential, depending on their toxicity, to be major risk drivers. However, IRIS assessments either have not been done or cannot be done due to lack of toxicity data. EPA has not done an IRIS assessment for ethyl acrylate, nor determined the carcinogenic potential of

carbonyl sulfide. IRIS determined there were inadequate human and animal data to assess the carcinogenic potential from inhalation exposure for propionaldehyde, quinoline, and 2,2,4-trimethylpentane. There were also inadequate data to derive inhalation reference concentrations for propionaldehyde, quinoline, 2,2,4-trimethylpentane, and carbonyl sulfide.

Table 3. Potentially Important Unassessed HAPs (percentage of average national risk or hazard index assuming 95th percentile toxicity and 2002 NEI emissions)
Ethyl acrylate (~15% of total cancer risk)
Quinoline (~1 % of total risk)
2,2,4-Trimethylpentane (~80% of total HI)
Carbonyl sulfide (~5% of total HI)
Propionaldehyde (~4% of total HI)
Hazard Index (HI) is the sum of hazard quotients for substances that affect the same target organ or organ system. The hazard quotient is the ratio of the potential exposure to the substance and the level at which no adverse effects are expected.

Options for Estimating Benefits for Air Toxics

Below are options that EPA is considering on how to proceed in the near term to estimate benefits from air toxics reductions. We recognize that these options are not mutually exclusive and there may be other approaches worth considering. We also acknowledge the importance of keeping in mind how such information would be used by decision makers and the appropriateness of using such information in evaluating the air toxics program or in estimating benefits for particular regulatory actions. In addition to the discussion on the strengths and limitations of each approach, Table 4 provides a summary comparison of these approaches.

- *Describe air toxics benefits qualitatively with no attempt at quantification.* This approach gives program offices the flexibility to make a qualitative case for air toxics control, while not exposing the analysis (or the regulation it supports) to the drawbacks and uncertainties associated with estimating air toxics benefits based on the current state-of-the-science.

  Strengths of this approach: For air toxics rules where the case can be made without quantified air toxics benefits (due to regulatory mandates, court-orders, technological-feasibility, or a compelling co-pollutant case), the estimation of highly uncertain, incomplete and partially unquantified air toxics benefits may undermine the overall case for regulation.

  Weaknesses of this approach: It does not build upon existing work to estimate air toxics benefits and continues to leave air toxics benefits as an unquantifed estimate, leading to an overall underestimate of the benefits associated with the control of air toxics.

- *Use the existing frameworks to sketch out minimum benefits from a national perspective.* This approach would build on work that is ongoing regarding benefits analysis looking pollutant-by-pollutant. For example, we could:

- Use the benzene methodology to estimate benefits of reducing benzene on a wider geographic scale and/or to estimate benefits of reducing other carcinogens with human data on inhalation exposures (e.g., 1,3-butadiene, vinyl chloride).
- Extend the analysis of acrolein effects for application in the benefits context (e.g., more specific characterization of lung function decrements that may occur in humans).
- Adapt the acrolein methodology to estimate effects of other priority inhalation noncancer pollutants (e.g., manganese). Conduct case studies applying the dose-response methods recommended in the NAS *Science and Decisions* report. And conduct willingness-to-pay research on specific noncancer effects associated with the HAPs being modeled.
- Use the lead methodology to estimate benefits from other non-inhalation risks (e.g., mercury, other persistent, bioaccumulative toxics)
- Expand the toluene methods to estimate benefits from reducing acute neurological effects of other similar pollutants (e.g., perchloroethylene).
- Acknowledge which priority air toxics described above would not be evaluated through these various frameworks at this point and other gaps in this approach.

Strengths of this approach: This approach would build on work that has been peer reviewed and shown to be applicable to some pollutants. It would give an assessment of fatal and non-fatal cancer effects, using standard benefits methods (multiplying reductions in the incidence of mortality and morbidity avoided by the value of a statistical incidence to estimate benefits). It would focus on the pollutants that we believe are driving the majority of the risks from a national perspective.

Limitations of this approach: It would not address some of the challenges laid out earlier, such as possible underestimation of health effects from hot spots and effects of interactions among pollutants on health. As a result, it is possible that people would misinterpret this minimum estimate to be, in fact, the actual estimate. It would also not consider estimates of WTP for risk reductions where equity concerns and individual values of reducing risk in the face of uncertainty are important components of the benefits. In addition, existing limitations in the valuation of mortality risks, such as the lack of differentiation between values for reductions in cancer-related mortality risks relative to other mortality risks, may lead to increased uncertainty or biases in the estimates of the values of air toxics risk reductions.

- *Use NATA or other existing modeling tools to pursue national or regional or local analysis focusing on reduction of individual risk levels.* For example, EPA used this approach with the 1999 NATA framework in its 2007 Mobile Source Air Toxics Rule. In that rule, air toxics modeling was done for 1999 and several future years, with and without controls. The 1999 NATA approach was significantly modified to use an enhanced version of the exposure model, the Hazardous Air Pollutant Exposure Model (HAPEM), which accounted for the pollutant gradients near roads. Impacts of the rule on individual risk were modeled for 19 mobile source air toxics. The rule also included estimates of monetized benefits from PM reductions that also occurred as a result of the rule. However, reductions in toxics risk were not monetized for the following reasons:

- The Science Advisory Board (SAB) specifically commented in their review of the 1996 NATA that these tools were not yet ready for use in national-scale benefits analysis, because they did not consider the full distribution of exposure and risk, or address sub-chronic health effects.
- We were unable to estimate cessation lag.
- We had not resolved analytical challenges with quantifying lifetime partial probabilities of cancer for different age groups or estimating changes in survival rates over time.
- Modeling did not account for indoor sources, and a variety of relatively unique microenvironments/exposure scenarios that could be tremendously important. As an example, the MSAT rule's analysis found that exposure from attached garages could be significantly more important than exposure to ambient benzene.

As discussed above, the benzene case study made progress in addressing the above issues, but the modeling approach used to capture distributions of exposure and risk cannot feasibly be extrapolated to the entire U. S. or extended to many other pollutants.

Strengths of this Approach: With NATA or other national-scale models, we can look at multiple pollutants together and look across various geographic areas to estimate impacts on individual risk levels.

Limitations of this Approach: With existing tools, we are unable to quantify the full distribution of exposure and risk nation-wide, quantify exposures in key microenvironments, or address sub-chronic health effects.

- *Estimate benefits of air toxics in conjunction with the criteria air pollution program.* Many regulations and implementation actions to meet criteria air pollution goals affect the same sources that emit significant air toxics. And in some cases, the emissions that contribute to ambient concentrations of ozone, PM, and other criteria air pollutants are also HAPs. For example, volatile organic compounds (VOCs) are an important precursor to ozone formation, and many VOCs are also HAPs. Likewise, many metallic HAPs also contribute to ambient $PM_{2.5}$ concentrations. To date, with the significant exception of mobile source regulations of VOC and PM precursor emissions, including diesel emissions, EPA regulatory impact analyses for criteria air pollutant programs have not assessed the air toxics impacts of the programs. Because these criteria pollutant programs are often very broad in nature, covering many sources and geographic areas, the cumulative impact on air toxics may be large. The newest one atmosphere atmospheric chemistry model, the Community Multiscale Air Quality model (CMAQ), is capable of estimating concentrations of many HAPs as well as criteria pollutants. It may thus be possible to use the results from CMAQ to inform a benefits analysis of reductions in air toxics that is consistent with the analysis of criteria pollutant-related benefits. At the very least, changes in population-weighted concentrations of air toxics could be assessed (using a program such as BenMAP), even in the absence of appropriate concentration-response functions linking concentrations with health endpoints.

  Strengths of this approach: It makes use of EPA's state of the art air quality modeling system and provides a consistent air quality framework for integration with other benefits

analyses. This approach would also have the benefit of providing information that could be used in later assessments as exposure-response functions become available.

Limitations of this approach: Without concentration-response functions, this approach still does not provide quantified health impacts or economic benefits. Also, the scale of the CMAQ modeling may not capture hotspots well and may therefore underestimate population weighted concentrations of air toxics.

- *Expand benefits assessment efforts to include equity considerations*. As noted earlier, there is an emerging literature on addressing equity considerations in addition to traditional analyses of economic efficiency. Two recent articles (Levy et al, 2007; Levy et al, in press) provide a framework for examining tradeoffs between health inequality and total public health benefits in evaluating reductions in power plant and mobile source emissions, respectively. These approaches use statistical measures of inequality, such as Gini coefficients, to determine the level of response in health inequality to a change in the population distribution of air toxic risks. These changes in equity can then be compared to changes in total public health benefits to identify possible complementarities in health benefits and equity, or the tradeoffs between health benefits and equity. This may either be an outcome of interest in itself, or may provide an input into assessments of the complementaries of efficiency and equity, once the costs of reducing exposure under different approaches are estimated.

  Strengths of this Approach: It provides a more detailed explanation of who is affected by exposure to air toxics, where the potential gains in health benefits are likely to be greatest, and the relationship between these metrics. It also provides alternative equity-based metrics that can be compared against costs.

  Limitations of this Approach: It looks only at health effects, not the net benefits (i.e., benefits less costs) of toxics reduction. Additionally, the Levy et al. studies use ambient concentrations of pollutants; as discussed above, indoor air quality or other exposure mechanisms, which appear to be important for health risks, might be difficult to include via this approach.

Table 4: Comparing Toxics Benefits Assessment Needs and Applicability

	Lead Case Study (single CAP)	Acrolein Case Study	Benzene Case Study	Toluene Case Study	Multi-pollutant Approach (NATA)	Co-benefits from Criteria Pollutants	Equity
Economic Valuation Considerations							
Scale	National	National, Regional or Local	Regional or Local	National, Regional or Local	National or Regional	National, Regional or Local	Regional or Local
Data Issues and Needs							
Emissions (# pollutants)	Single pollutant	Groups of pollutants with similar health endpoints	Single pollutant, could be expanded if data available	Single pollutant, could be expanded if data available	All	Organics & metals	All
Health Metrics & Endpoints	Focus on neurological effects associated with blood lead levels, but could include others	Lung function indicators, but could include others (non-cancer)	Cancer	Acute exposures and neurotoxicological effects , but could be expanded	Separate carcinogen, non-carcinogen, and acute assessments for inhalation exposures	Mortality and morbidity effects (both chronic and acute)	Investigating equity measures (e.g., Gini coefficients)
Toxicity Data Needs	Human, with animal data to support; need to consider if animal data is useful in absence of human data	Animal data; significant assumptions needed for interspecies extrapolation	Human cancer data; life-table data generally unavailable	PBPK modeling or measurement; human data	Data based on IRIS and other peer-reviewed values; some pollutants lack necessary data	Have good human data	Relatively fine spatial resolution of data, including baseline health and differential exposures
Exposure Data Needs	Concentration-dose relationship (e.g., blood lead)	Depending on pollutant, indoor levels as well as outdoor would be useful	Depending on pollutant, indoor levels as well as outdoor would be useful	Concentration-dose relationship (e.g., blood alcohol)	Depending on pollutant, indoor levels as well as outdoor would be useful	Typically outdoor	Depending on pollutant, indoor levels as well as outdoor would be useful; susceptibility and vulnerability data
Key Strengths	Follows same analytic steps as PM or ozone benefits analysis	Endpoint impacts measured on a continuous scale	Captures high-end exposures; accounts for age-specific effects and cessation lag	Enables estimation of costs of exposure for pollutants where costs unknown	Would give an estimate of fatal and non-fatal cancer effects, using standard benefits methods	Would provide perspective on toxics reductions from criteria pollutant regulations	Information on exposures that different populations face is useful for looking for discriminatory impacts
Key Limitations	Air-to-blood ratios based on estimates of historical exposure	Significant uncertainty in animal to human extrapolation	Resource intensive; difficult for national scale; life-table data not available for most pollutants; not demonstrated for non-cancer	Cost data must be available for a pollutant with similar effects; internal dose information must be available.	Possible underestimation of health effects from hot spots; cannot account for interaction or willingness-to-pay; provides only a lower bound on benefits	Would not provide quantifiable benefits estimates for HAPs to use in economic analysis.	Does not estimate net benefits; may not estimate health endpoints.

Section IV. Considerations for Longer-Term Approaches to Air Toxics Benefits

In Section III, EPA presents near-term options for estimating benefits. The approach or approaches that EPA chooses in the near term, combined with longer-term priorities for the overall toxics program, can help inform our research needs for benefits analysis. The SAB, as mentioned earlier, provided feedback on the benzene case study, which included recommendations to move away from a pollutant-by-pollutant approach for benefits assessment. In addition, the National Academy of Sciences has recently made recommendations on unification of cancer and noncancer risk assessment approaches as well as on developing comprehensive air quality management plans, which consider both criteria pollutants and air toxics. And as the toxics program has matured, EPA is moving toward a sector-based approach for stationary sources of air toxics and looking more cumulatively at risks from all air toxics.

Issues for Discussion on Benefits Methods

What steps do we need to take to refine or develop benefits assessment methods over the longer-term? Some options include:

Expand case study efforts (e.g., benzene, acrolein) and NATA to improve methodologies (e.g., including multiple pollutants, more extensive health endpoints).

Develop alternative estimation approaches for willingness to pay for risk reduction. Research on the benefits of risk reduction could examine, for instance, differences in willingness to pay for reducing different risks, ways to consider objective vs. subjective risk, or variations in willingness to pay in different age cohorts. It could also involve developing approaches that incorporate multiple sources and kinds of exposure. It might even be possible to examine willingness to pay for programs that reduce toxics in the absence of strong health information, by presenting people with tradeoffs involving poor risk information.

Develop language for regulatory analyses to provide a qualitative assessment of the benefits associated with toxics reduction. Regulatory analyses often focus on quantitative information with less emphasis on qualitative information that would be useful for making decisions and informing the public. EPA could develop a template for describing what is known about the benefits of toxics reduction and how that qualitative information could be used for decision-making.

Identify opportunities for conducting epidemiologic studies of HAPs at general population exposure levels. For example, NATA has been used for an epidemiology study of breast, lung, and colorectal cancer in Long Island (Jacquez and Greiling, 2003) and autism in San Francisco (Windham et al., 2006), and we have begun sharing data with a team of University of Texas epidemiologists studying links between mercury exposure and autism (e.g., Palmer et al., 2009). We could continue to pursue this kind of collaboration.

Invest in methodologies to better understand equity considerations. Research could examine different forms of inequities related to toxics, such as whether some demographic groups face more exposure than others and whether different regulatory approaches lead to differences in exposure. In addition, NATA risks are expressed on a Census tract basis, so they can easily be linked to demographic variables in the Census. Some investigators have already taken this approach to test correlations between economic hardship and inhalation risk. We could use NATA as a platform for environmental justice studies targeting the most sensitive populations.

Improve efforts to quantify co-benefits looking at air toxics from the standpoint of co-benefits from PM and ozone reductions.

Conduct multi-pollutant and multi-media analyses, which could include sector-based analyses.

Options to Improve Underlying Emissions Data and Air Quality and Exposure Modeling

As we develop new economic valuation tools, we should also continue working to improve the underlying data that would be used in any tool. Some suggestions include:

- *Emissions inventory improvements*. . EPA has a number of plans underway to improve the overall inventory for air toxics (primarily by integrating the air toxics inventory with the mandatorily reported criteria pollutant inventory through the forthcoming Emissions Inventory System, or EIS) as well as to address specific weaknesses in particular source categories.

    - For highway mobile sources, collecting more data from State and local sources can improve that capability. Currently, the National Emissions Inventory allocates vehicle activity from the State or Metropolitan Statistical Area (MSA) level to the county using population, then allocates from the county to grid cell or census tract using roadway miles. This approach can result in large errors in correctly estimating local emissions. Collecting and using travel-demand model data from local metropolitan planning organizations can substantially improve activity estimates, although these models often lack detailed temporal resolution or the ability to assess sporadic incidents that affect traffic flow (e.g., car accidents). In addition, States and local governments often have modeling inputs, such as meteorology, fuel properties, and vehicle registration distributions, which can significantly improve the quality of inventories. Finally, completion and implementation of the MOVES model, with improved emission factors for toxics, will improve highway inventories.

    - For nonroad and area sources, there may be opportunities to improve allocation surrogates as well, or even to construct "bottom-up" inventories. These opportunities can include surveys of equipment operators, compiling

more recent land use data to use in developing surrogates, and working with local agencies to improved surrogates.

- For point sources, start-up, shut-down, and malfunction (SSM) emissions often go unreported. EPA is developing a plan for getting better information on these emissions. In addition, some sources that make a relatively small contribution to emissions now are expected to make a much larger contribution in the future (e.g., ethanol production facilities). Identifying these and other sources where we lack data and making a concerted effort to collect more emissions information could substantially improve estimates of future emissions.

- We could improve emissions data by addressing inconsistencies between criteria and toxics inventories for pollutants that are in both. As a specific example, this might include addressing discrepancies between HAP-reported emissions and speciated criteria pollutant emission estimates (using SPECIATE). Additionally, ORD's National Risk Management Research Laboratory (NRMRL) has a dilution sampler system (DSS) that can better characterize aerosols and other condensables.

- Our focus for air toxics is limited to the 187 HAPs in the CAA. We could use TRI data and the urban HAP toxicity-weighting methodology to develop a list of priority substances that are not currently HAPs, and include them in future analyses, including NATA. The NATA results could then be used to select candidates for detailed assessments and possible listing proposals.

- *Ambient monitoring improvements*. EPA is currently initiating a limited ambient monitoring program around schools where children may be exposed to high levels of air toxics and developing a longer-term program for additional monitoring given the limited resources for this initial program. Additional ambient monitoring will help us better understand emissions and exposure in local areas and trends of toxics across the country. Monitoring is an important tool for model evaluation and emissions inventory improvement.

- *Air quality modeling improvements*. EPA is transitioning toward a more integrated approach to air quality modeling, which will include the use of a photochemical model for those air toxics for which such reactions are important. This approach will better allow the simulation of air quality impacts across criteria and toxic air pollutants and will begin by combining current air quality modeling capabilities for air toxics (i.e., AERMOD and ASPEN) with current criteria pollutant photochemical air quality modeling capabilities (i.e., CMAQ). By integrating these models, EPA will create the National Air Pollutant Assessment, or NAPA, which will replace the current NATA and combine assessment methodologies and data presentations for criteria and toxic air pollutants to the extent feasible. However, CMAQ currently is limited in the number of pollutants it can appropriately simulate, and it does not adequately

capture the local concentration gradients that are important for characterizing the impacts of many air toxics around stationary and mobile sources. Longer term, and for purposes of benefits assessment, it is desirable to use the same platform for as many pollutants as possible, and that may necessitate adding chemical and deposition mechanisms for more air toxics to CMAQ. In this regard, EPA could improve and incorporate chemistry information for air toxics, such as mercury, arsenic, and PAHs, and account for secondary toxic and non-toxic product formation. Further, we would need to incorporate an approach that can resolve fine-scale concentrations within the 3-D air quality model, so that the effects of chemistry, transport, and deposition can be addressed at multiple resolutions, particularly near major emission sources of key air toxics. Improved national resolution of CMAQ below 36 square km is also desirable, probably down to 4 square km as the coarsest grid size. However, modeling the U.S. at this level of resolution will probably need to wait until faster computer speeds are available.

In the meantime, we anticipate developing hybrid modeling approaches that combine the benefits of 3-D models (averaged concentrations over grid area) and dispersion models (concentrations near source), such as AERMOD, to estimate exposure near contaminant hotspots (e.g., roadways). For mobile sources, AERMOD can simulate dispersion by representing roadways as a series of volume or area sources. However, for ground-level emissions, AERMOD predictions at receptors near such sources are rather sensitive to the source characterization. Also, there is no direct method to represent the influence of traffic volumes on atmospheric turbulence near roadways within AERMOD. Given the differing capabilities of modeling tools, it is important to communicate with the model developers the scales required for EPA's uses, including health and benefits assessments.

- *Exposure modeling improvements*. Especially given potential localized impacts, it is important to characterize more accurately exposures of populations near sources. The most recent version of HAPEM includes a near-road algorithm to account for elevated exposures people living near roads. This algorithm should be reevaluated, based on data from recent ORD and other research on HAP concentrations along roads. Microenvironmental factors should also be updated with more recent data. In addition, approaches should be developed to account for contributions from indoor sources. As previously mentioned, indoor source contributions may be important considerations, depending on the choice of a dose metric and the shape of a dose-response curve. Finally, for some HAPs, occupational or recreational exposures (e.g., operating lawn and garden equipment, snowmobiles) may be significant in estimating benefits. Approaches should be explored for modeling these exposures.

  Finally, acute exposure assessment may differ substantially from chronic exposure assessment. For example, carbon monoxide poisoning sometimes occurs during boating or power outages, when numerous low-probability events occur simultaneously (e.g., lung ventilation rate, human proximity to emissions).

Assessing acute exposures and health effects for some air toxics may require similar considerations (e.g., intoxication by VOCs).

- *Health and environmental effects characterization improvements*. As acknowledged earlier, we have limited data on many of the 187 air toxics currently on the list of air toxics in the Clean Air Act. While IRIS dose-response values are not designed for benefits assessment, they and other values may be suitable, depending on a clear understanding and alignment of the benefits assessment needs to the nature of the values available. The IRIS dose-response values for cancer are readily amenable to benefits assessment whereas the reference dose/reference concentration values for noncancer endpoints are more challenging for benefits assessment. Moreover, it would be informative to have methodologies accounting for higher risks from exposures during childhood, and thus, greater benefits from reducing these exposures. Similarly, partial lifetime probabilities of cancer for different age groups, changes in survival rates over time, and cessation lag estimates, and a clearer understanding of acute effects would be helpful for air toxics benefits assessment.

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