1990 – 2002 NEI HAP Trends: Success of CAA Air Toxic Programs in Reducing HAP Emissions and Risk

Anne Pope and Madeleine Strum
Office of Air Quality Planning and Standards (OAQPS), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711
pope.anne@epa.gov
strum.madeleine@epa.gov

ABSTRACT
Requirements of the Clean Air Act (CAA) established the need for a more comprehensive hazardous air pollutant (HAP) emissions inventory effort that can be used to track progress by the Environmental Protection Agency (EPA) over time in reducing HAPs in ambient air. In response to these needs, the EPA developed the National Air Toxics Assessment (NATA) to estimate the magnitude of HAP emissions reductions and demonstrate reduced public risk from HAP emissions attributable to CAA toxics programs. EPA also developed toxicity weighted emissions data trends to track progress in reducing risk of HAPS to Americans. To estimate risk and HAP emission reductions, EPA compiles the National Emissions Inventory (NEI).

This paper briefly discusses the compilation of the 2002 NEI for HAPs, highlights data sources and quality, and describes the methodology used to toxicity-weight NEI HAP emissions. This paper focuses on 2002 emissions data summaries and trends in HAP emissions and toxicity-weighted emissions trends from 1990 to 2002. The toxicity-weighted emission trends shows the success of CAA air toxic programs in reducing HAP emissions and risk.

INTRODUCTION
Requirements of the CAA established the need for a more comprehensive HAP emissions inventory effort that can be used to track progress by the EPA over time in reducing HAPs in ambient air. The CAA requires EPA to promulgate standards that reduce emissions and risks of HAPs. Section 112 (d) requires EPA to promulgate technology-based emission standards, known as maximum achievable control technology (MACT) standards, for major sources of HAPs. Section 112(f) requires EPA to promulgate standards to address risks remaining after implementation of MACT standards, known as residual risks standards. Section 112(c)(3) and Section 112(k) of the CAA requires EPA to address emissions and risks of HAPs from area sources and to show a 75% reduction in cancer incidence of emissions from stationary sources of HAPs since 1990.

In order to determine if CAA programs are successful in reducing emissions and human health and environmental risk due to HAPs emissions, EPA compiles the NEI for HAPs. The EPA previously compiled a baseline 1990 and 1996 National Toxics Inventory (NTI) and 1999 NEI for HAPs and has recently completed version 3 of the 2002 NEI. The NEI

includes point major and area sources, nonpoint area and other sources, and mobile source estimates of emissions. Stationary major sources of HAPs are defined as sources that have the potential to emit 10 tons per year or more of any single HAP or 25 tons per year or more of any combination of HAPs. Stationary area sources of HAPs are defined as sources that have the potential to emit less than 10 tons per year or more of any single HAP or less than 25 tons per year or more of any combination of HAPs. Mobile sources include onroad vehicles, nonroad equipment, and aircraft/locomotive/commercial marine vessels (ALM).

EPA has developed the National Air Toxics Assessment (NATA) to estimate the magnitude of HAP emissions reductions and demonstrate reduced public risk from HAP emissions attributable to CAA toxics programs. NATA consists of the following steps.

- Compile the NEI
- Process the emissions using EMS-HAP to group/partition HAPs belonging to compound groups, spatially allocate county-level emissions to tracts and temporally allocate emissions diurnally
- Estimate ambient concentrations using ASPEN at census tract resolution
- Compare modeled ambient concentrations to available air toxics monitoring data
- Estimate population exposures using HAPEM at census tract resolution
- Assess public health risks (cancer risks and noncancer respiratory and neurological effects) due to inhalation of air toxics

The 2002 NATA will employ newer modeling tools for point sources to use an AERMOD dispersion algorithm and to allow for finer (census block) resolution for these sources. 1996 and 1999 NATA results are available at: **www/epa.gov/ttn/atw/nata/**.

The NATA has been a useful tool to assess inhalation risks from HAP emissions when ambient air toxics monitoring and more finer scale modeling using more locally resolved HAP emissions are not available . The 1990 NEI has not been modeled in NATA. To assess the reduction in risks from CAA programs, an alternative less-desired approach to NATA is to toxicity-weight the 1990 and 2002 NEI and analyze the relative potential cancer risk and noncancer respiratory hazard posed by the inhalation of the pollutants emitted by particular source sectors. This approach is simple to apply, and accounts for differences in toxicity among pollutants. The approach has the limitations of not considering fate, transport, or location and behavior of receptor populations, and it is capable only of estimating relative risks. The absolute value of a toxicity-weighted emissions value has no meaning. The value of toxicity-weighting emissions is in comparing toxicity-weighted emissions across source sectors, for prioritization, and comparing across time periods to show progress in reducing particular health risks. The toxicity-weighting technique is a partial risk analysis tool that has proven valuable for screening-level analyses.

This paper discusses the compilation of the 2002 NEI and the methodology used to toxicity-weight the NEI, and summarizes trends in HAP emissions and toxicity weighted emissions trends from 1990 to 2002.

COMPILATION OF THE 2002 NEI

Complete source category coverage is needed, and the NEI contains estimates of emissions from stationary point and nonpoint and mobile source categories. The stationary point source inventory contains estimates of facility-specific HAP emissions and their source-specific parameters necessary for modeling such as location and facility characteristics (stack height, exit velocity, temperature, etc.). The major steps involved in compiling the 2002 NEI include:

- Submittal of 2002 inventory data by state and local agencies, tribes, industry, and EPA offices;
- Blending/Merging of data from multiple data sources;
- Augmentation of data for missing data elements;
- QC/QA of data;
- Preparation of draft 2002 NEI for external and internal review;
- Incorporation of external and internal review comments on the draft 2002 NEI and incorporation of new inventory data submitted during review period; and
- Preparation of final 2002 NEI.

Important steps in preparing NEI data for use in air quality and risk and exposure modeling are the quality assurance (QA) and augmentation of data. The draft 2002 NEI was available for a period of 90 days for external review. For more information about the compilation of the 2002 NEI, please refer to the documentation reports for point, nonpoint and mobile sources and to the report, NEI Quality Assurance and Data Augmentation for Point Sources, found at: http://www.epa.gov/ttn/chief/net/2002inventory.html#documentation.

The sources of data in the 2002 NEI are:

- state and local agencies,
- regional program organizations (RPO's)* CAP only,
- tribes,
- industry,
- EPA's MACT staff (MACT, residual risk and area source standards data),
- EPA's Clean Air Market Division,
- Toxic Release Inventory (TRI)
- EPA's Office of Transportation and Air Quality,
- EPA's Emission Inventory and Assessment Group (EIAG) augmented chromium emissions
- EIAG augmented HAP boiler estimates from criteria air pollutant (CAP) boiler estimates, and
- EIAG augmented non-point emissions for sources not included in the state, local and tribal data.

The EIAG developed hexavalent chromium or trivalent chromium estimates from reported chromium/chromium and compounds and hexavalent or trivalent chromium emission estimates. The EIAG developed boilers estimates for HAPs when CAP emission estimates were reported, but HAP emission estimates were not reported.

METHODOLOGY TO TOXICITY-WEIGHT THE 2002 NEI and 1990 NTI

Unit Risk Estimates (UREs) and Reference Concentrations (RfCs) are used to express dose responses for cancer and noncancer effects. Cancer risks across pollutants are additive, and noncancer risks by target organ across pollutants are additive. Cancer and noncancer risks are not additive and cannot be summed together.

For cancer, EPA assumes a linear relationship between the level of exposure and the lifetime probability of cancer from an air toxics compound. For cancer, the URE is an upper bound estimate of an individual's probability of contracting cancer over a lifetime of exposure to a concentration of one microgram of the pollutant per cubic meter of air. If an URE is 1.5 x 10-6 per ug/m3, 1.5 excess tumors are expected to develop per 1,000,000 people if they are exposed daily for a lifetime to 1 ug of chemical in 1 cubic meter of air. EPA considers UREs to be upper bound estimates, meaning they represent a plausible upper limit to the true value. Cancer risks associated with different substances can be added together as long as the substances cause cancer by (1) similar mechanisms, or (2) completely independent mechanisms.

HAPs are associated with a wide variety of noncancer adverse health effects that include neurological, cardiovascular, liver, kidney, and respiratory effects as well as effects on the immune and reproductive systems. For noncancer effects, EPA expresses dose-response relationships for effects other than cancer in terms of the RfC. The RfC is a concentration (ug/m3) of the compound in air thought to be without adverse effects even if a person is exposed continuously during a lifetime. In other words, exposures below the RfC will probably not cause adverse noncancer health effects. To express noncancer hazards the EPA uses the RfC as part of a calculation called the hazard quotient (HQ), which is the ratio between the concentration to which a person is exposed and the RfC. A value of the HQ less than one indicates that the exposure is lower than the RfC and that no adverse health effects would be expected. A value of the HQ greater than one indicates that the exposure is higher than the RfC. However, because many RfCs incorporate protective assumptions in the face of uncertainty, an HQ greater than one does not necessarily suggest a likelihood of adverse effects. An HQ greater than one can best be described as indicating that a potential exists for adverse health effects. Because different pollutants may cause similar adverse health effects, it is often appropriate to combine HQs associated with different substances. HQs should be combined for pollutants that cause adverse effects by the same toxic mechanism. The NATA combines noncancer hazards associated with respiratory irritation and neurological effects using the hazard index (HI). The HI is defined as the sum of hazard quotients for individual air toxics compounds that affect the same organ or organ system. The HI is only an approximation of the combined effect because some of the substances may affect the target organs in different (i.e., non-additive) ways. A value of the HI below 1.0 will likely not result in adverse effects over a lifetime of exposure. An HI greater than one can be best described as indicating that a potential may exist for adverse effects to respiratory or nervous system.

More information on cancer risks, UREs, noncancer effects, and RfCs can found at: http://www.epa.gov/ttn/atw/nata1999/riskbg.html.

In order to estimate the magnitude of toxicity-weighted emissions reductions, emissions of individual speciated HAPs are needed because the toxicity associated with an individual compound within a compound group varies widely. The NEI contains speciated emissions reported by individual species if reported. More than 500 pollutants are reported in the NEI. The CAA lists 188 compounds including 20 compound groups. For these 20 groups, the EPA requests speciated data from NEI data submitters. Knowledge of the particular chemical compound of a metal compound group allows you to estimate the mass of the metal, which is the emissions value that is toxicity-weighted. It is critical that emissions be reported by individual compound for certain key compound groups list in the CAA such as chromium, mercury, and polycyclic organic matter (POM). Mercury speciation is not necessary for toxicity-weighting for inhalation risks, but it is essential in chemical transport models which utilize the species in chemical reactions. For these other compounds, the risks depend upon the particular species. Hexavalent chromium poses the cancer risk as opposed to trivalent chromium and the URE's for specific POM can vary by orders of magnitude. However, not all emissions are reported in the NEI by individual pollutant and assumptions must be made to toxicity-weight emissions data. For chromium and mercury, EPA maintains default speciation files. If chromium emissions are reported as "elemental chromium" or "chromium and compounds", then chromium speciation default profiles are used to speciate emissions into hexavalent and trivalent chromium emissions. If mercury emissions are not reported as elemental gaseous mercury, particulate divalent mercury, and gaseous divalent mercury emissions, then mercury speciation default profiles are used. Emissions by source category are needed in order to use chromium and mercury speciation files. For POM, NEI HAPs are assigned to 8 different POM "risk"groups that are used to toxicity-weight emissions and in risk assessments.

The file, Toxicity Weighting Factors, provides data and steps for assigning HAPs reported in the file contains, the following data fields.

- NEI pollutant code and description
- NEI HAP category name (CAA name)
- Chemical formula
- Molecular weight
- Modeling pollutant description – used in ASPEN
- Metal_CN speciation factor – factors used to estimate the metal portion of a metal compound or to convert the mass of cyanide (CN) compound to HCN equivalents.
- Tox Table chemical name – used in HAPEM and dose response
- Tox Table CAS number – used in HAPEM and dose response
- Tox Table HAP number – used in HAPEM and dose response
- URE – unit risk estimate, dose response value for cancer risks
- RfC – reference concentration, dose response value for non-cancer effects
- Urban 33 HAP – indicates if HAP is on Section 112k list of urban area source HAPs
- VOC constituent
- PM constituent
- TEQ factors – factors used to estimate TEQ from individual congeners
- Coarse PM factors and Fine PM factors for HAPs – needed for dispersion modeling

In order to toxicity-weight the NEI, the reported HAPs in the NEI must first be assigned to dose response values. The NEI pollutant code is matched to Tox Table chemical name, and Tox Table CAS number using the Toxicity Weighting Factors file. The steps for toxicity-weighting emission inventories include the following.

1. For all NEI pollutants except chromium and compounds (pollutant code 136) and chromium (pollutant code 7440473):
   Multiply emissions by Metal_CN Speciation Factor to extract metal and cyanide mass for tox weighting.

2. For NEI pollutant codes 136 and 7440473:
   Use chromium speciation file to speciate source category emissions into hexavalent chromium (Cr6) and trivalent chromium (Cr3) emissions.

3. For Cancer Toxicity-Weighting:
   Multiply emissions generated from steps 1 and 2 by URE.

4. For Noncancer Toxicity-Weighting:
   Divide emissions generated from steps 1 and 2 by RfC for each target organ.

These default speciation and toxicity weighting factor files can be found at: http://www.epa.gov/ttn/chief/net/2002inventory.html#documentation.

The main limitations of toxicity-weighting include:

- Toxicity-weighting does not consider environmental fate and exposure potential, and therefore does not reflect the risks posed by releases.
- Toxicity-weighting tools are typically designed to address only one or at most a few toxicity end points. If the user is concerned with other effects that are not accounted for in the weighting scheme, the resulting information will not be particularly useful.
- Toxicity-weighting generally does not account for multiple chemical releases from the same source or nearby sources, the combined effect of which could differ from the sum of individual chemical releases.
- Variation in the amount and quality of toxicity data available for different chemicals introduces uncertainty into the toxicity-weighting based on such data.
- Multiple toxicity-weighting systems may be required to address different hazards (e.g., acute versus chronic affects) posed by different chemicals.
- Toxicity-weighted releases may not be how a community or other stakeholder wishes to view pollutant releases, particularly for a chemical that has been shown to present a risk based on other attributes, e.g. persistence and/or ability to bioaccumulate. Non-inhalation routes of exposure such as ingestion are important for HAPs such as mercury and dioxins. A chemical that appears to be of less concern based solely on a toxicity-weighting may have a much higher risk potential based on its other inherent attributes.

1990 – 2002 TRENDS IN EMISSIONS AND TOXICITY-WEIGHTED EMISSIONS

The 1990 NEI was compiled originally from several HAP emission inventories, such as the 112(k), 112(c)(6), pre-MACT baseline emissions inventory, and TRI. The 112(k), 112(c)(6), and pre-MACT baseline emissions inventory were primarily developed as top-down emission inventories. Emissions were calculated at the national-level, and then allocated to the county-level using surrogates (county business patterns, county population, etc.). Additionally, HAP emission inventories provided by the Great Lakes Commission (GLC) for 8 states, California's Air Resources Board (CARB), Harris County, TX and Maricopa County, AZ were integrated into the 1990 NEI.

Version 3 of the 2002 NEI was used for the trends in this paper. Inventory methodologies have improved greatly between the compilation of the 1990 NEI and the 2002 NEI for HAPs. Improvements to the 2002 NEI for HAPs include higher resolution of data to support modeling, improved speciation of HAPs, participation by most state and local agencies and tribes, increased the number of source categories, inclusion of facility and stack specific data, electronic QA/QC of data, and external review of inventory by a large number of individuals within state and local agencies, tribes, industry, and EPA.

National Emission and Toxcity-Weighted Emission Trends

Significant decreases in HAP emissions have occurred in the 1990s due to the implementation of MACT standards and Mobile Source Onroad regulations. Area and other source emissions have increased because EPA has not yet fully implemented its area source program as required by Section 112c(3) and 112(k) of the CAA. Figure 1 and Table 1 presents emissions trends for the sum of 188 HAPs by source sectors. The methodology for fires is not comparable in 1990 and 2002.

Toxicity-weighted emissions have also declined between 1990 and 2002 for cancer and noncancer respiratory and neurological effects. Figures 2 – 4 and Tables 2 – 4 present toxicity-weighted emissions scaled to the sum of 7.24 million tons for 1990 total emissions. 86 HAPs have UREs. 18 HAPs have noncancer neurological RfCs. 39 HAPs have noncancer respiratory RfCs.

Figure 5 compares contributions of specific HAPs to 2002 NEI emissions and toxicity-weighted emissions for carcinogens, noncancer neurological HAPs and noncancer respiratory HAPs. Pollutants such as hexavalent chromium, arsenic, and manganese have very low emissions, but have high toxicity-weighted emissions. Other pollutants such as toluene have high emissions, but no toxicity-weighted emissions. This type of analysis can be used to prioritize efforts in quality assuring data. Table 5 identifies the sectors with the largest contribution for the pollutants shown in Figure 5.

In 2002, benzene accounts for 28% of cancer risks in the toxicity-weighted NEI. Benzene was a national cancer risk driver in the 1999 and 1996 NATA. In the 1999 NATA, benzene accounted for 22% of the average cancer risk. In 2002, manganese accounts for 77% of noncancer neurological effects in the toxicity-weighted NEI, and

acrolein accounts for 90% of noncancer respiratory effects in the toxicity-weighted NEI. Acrolein was a national driver for noncancer effects and manganese was a regional driver in the 1999 and 1996 NATA. In the 1999 NATA, acrolein accounted for 86% of the average noncancer effects. A discussion of specific source categories for these 3 HAPs follows.

- Benzene: Stationary sources of benzene accounted for 22% of the total benzene emissions in 1990 and 41% in 2002. Overall benzene emissions decreased by nearly 15% between 1990 and 2002. The mobile sources portion of benzene decreased substantially during that time period (36%); however, the MACT source categories exhibited the highest percentage decline (62%). The MACT categories that emit benzene having the largest decrease in emissions during this time period include: Synthetic Organic Chemical Manufacturing (HON), Petroleum Refineries, Oil & Natural Gas Production, and Gasoline Distribution (Stage I).

- Acrolein: Stationary sources of acrolein accounted for 83% of the total acrolein emissions in 1990 and 82% in 2002. Overall acrolein emissions decreased by over 51% between 1990 and 2002. The mobile sources portion of acrolein decreased substantially during that time period (47%); the MACT source categories exhibited a 42% decrease in emissions. The MACT categories that emit acrolein having the largest decrease in emissions during this time period include: Synthetic Organic Chemical Manufacturing (HON), Plywood and Composite Wood Products, and Pulp & Paper Production.

- Manganese: Stationary sources of manganese accounted for 99.9% of the total acrolein emissions in 1990 and 99.7% in 2002. Overall manganese emissions decreased by more than 16% between 1990 and 2002. The MACT source categories exhibited over 37% decrease in emissions. The MACT categories that emit manganese having the largest decrease in emissions during this time period include include: Integrated Iron & Steel Manufacturing, Iron and Steel Foundries, and Ferroalloys Production.

Emissions and toxicity-weighted emissions of source categories emitting HAPs vary geographically. Figure 6 presents maps of county-level 2002 cancer toxicity-weighted emissions for benzene and arsenic using version 1.0 of the 2002 NEI. These figures will be replaced in the final presentation of this paper when mobile source emissions of HAPs are revised. The distribution of risks in counties differs for each pollutant. Benzene risks are located in counties with high population due to the large contribution of mobile sources and in counties where the primary stationary source categories are chemical manufacturing/petroleum refineries. Arsenic risks are not associated as much with population because arsenic is not dominated by mobile source emissions. The primary sources of arsenic in the 2002 NEI are utility coal boilers, primary metal industries, and industrial boilers. Figure 7 compares county-level maps of 1990 and 2002 NEI version 1 cancer toxicity-weighted benzene emissions. The success of EPA programs in addressing risks associated with benzene from mobile sources and from MACT sources can be seen when comparing the two maps.

CONCLUSIONS

Significant decreases in HAP emissions, risks and noncancer effects have occurred in the 1990s due to the implementation of MACT standards and Mobile Source regulations. Inventory methodologies have improved greatly between the compilation of the 1990 NTI and 2002 NEI for HAPs

Toxicity-weighting of emission inventories is simple to apply and accounts for differences in toxicity among HAPs. However this approach has the limitations of not considering fate, transport, or location and behavior of receptor populations. Toxicity weighting of inventories is a useful tool to compare relative risks associated with pollutants in order to focus future inventory development efforts. Toxicity-weighting allows agencies to focus resources on the HAPs with the highest potential risks.

Figure 1. 1990 – 2002 Trends in HAP Emissions

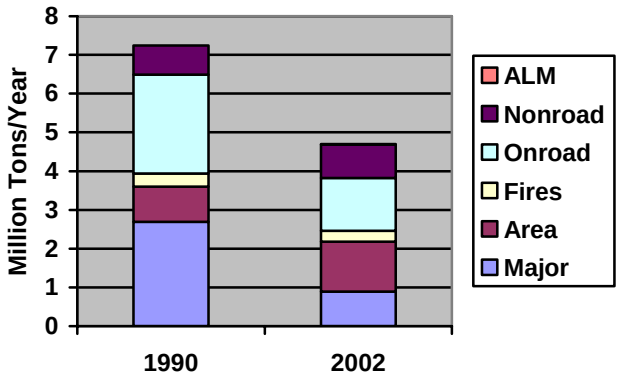


Table 1. 1990 – 2002 Emission Trends – Sum of 188 HAPs

Sector	1990 Emissions	2002 Emissions	% Reduction
TOTAL	**7.24 million tons**	**4.7 million tons**	**35**
Major	2.69 million tons	0.89 million tons	67
Area	0.91 million tons	1.29 million tons	-42
Fires (Wildfires & Prescribed Burns	0.34 million tons	0.28 million tons	18
Onroad Mobile	2.55 million tons	1.36 million tons	47
Nonroad Mobile	0.75 million tons**	0.86 million tons	-15
ALM Mobile*		0.02 million tons	

*ALM – Aircraft Locomotive and Commercial Marine Vessels
** 1990 Nonroad Mobile includes ALM

Figure 2. 1990 – 2002 Trends in Scaled Cancer Toxicity-Weighted Emissions

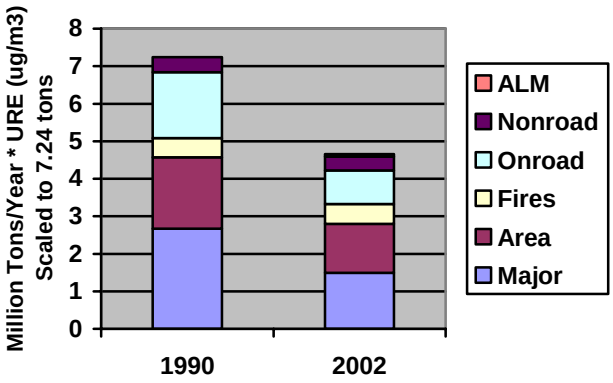


Table 2. 1990 – 2002 Scaled Cancer Toxicity-Weighted Emission Trends

Sector	% Reduction from 1990 to 2002
TOTAL	**36**
Major	44
Area	31
Fires (Wildfires & Prescribed Burns)	-4
Onroad Mobile	49
Nonroad Mobile	7.5

Figure 3. 1990 – 2002 Trends in Scaled Noncancer Neurological Toxicity-Weighted Emissions

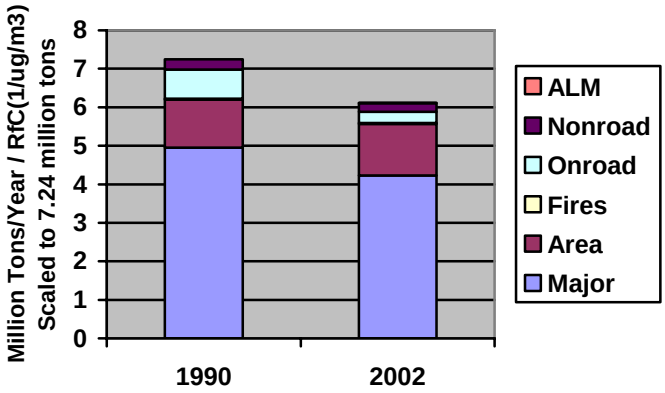


Table 3. 1990 – 2002 Scaled Noncancer Neurological Toxicity-Weighted Emission Trends

Sector	% Reduction from 1990 to 2002
TOTAL	**16**
Major	15
Area	-7
Fires (Wildfires & Prescribed Burns)	0
Onroad Mobile	62
Nonroad Mobile	12

Figure 4. 1990 – 2002 Trends in Scaled Noncancer Respiratory Toxicity-Weighted Emissions

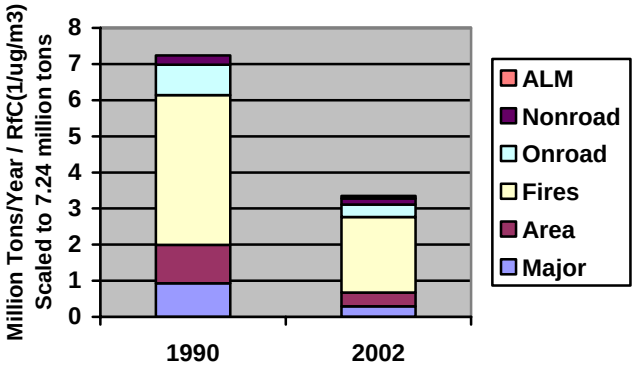


Table 4. 1990 – 2002 Scaled Noncancer Respiratory Toxicity-Weighted Emission Trends

Sector	% Reduction from 1990 to 2002
TOTAL	**54**
Major	69
Area	64
Fires (Wildfires & Prescribed Burns)	50
Onroad Mobile	58
Nonroad Mobile	35

Figure 5. Percent Contribution of HAPs to 2002 Emissions and Toxicity-Weighted Emissions

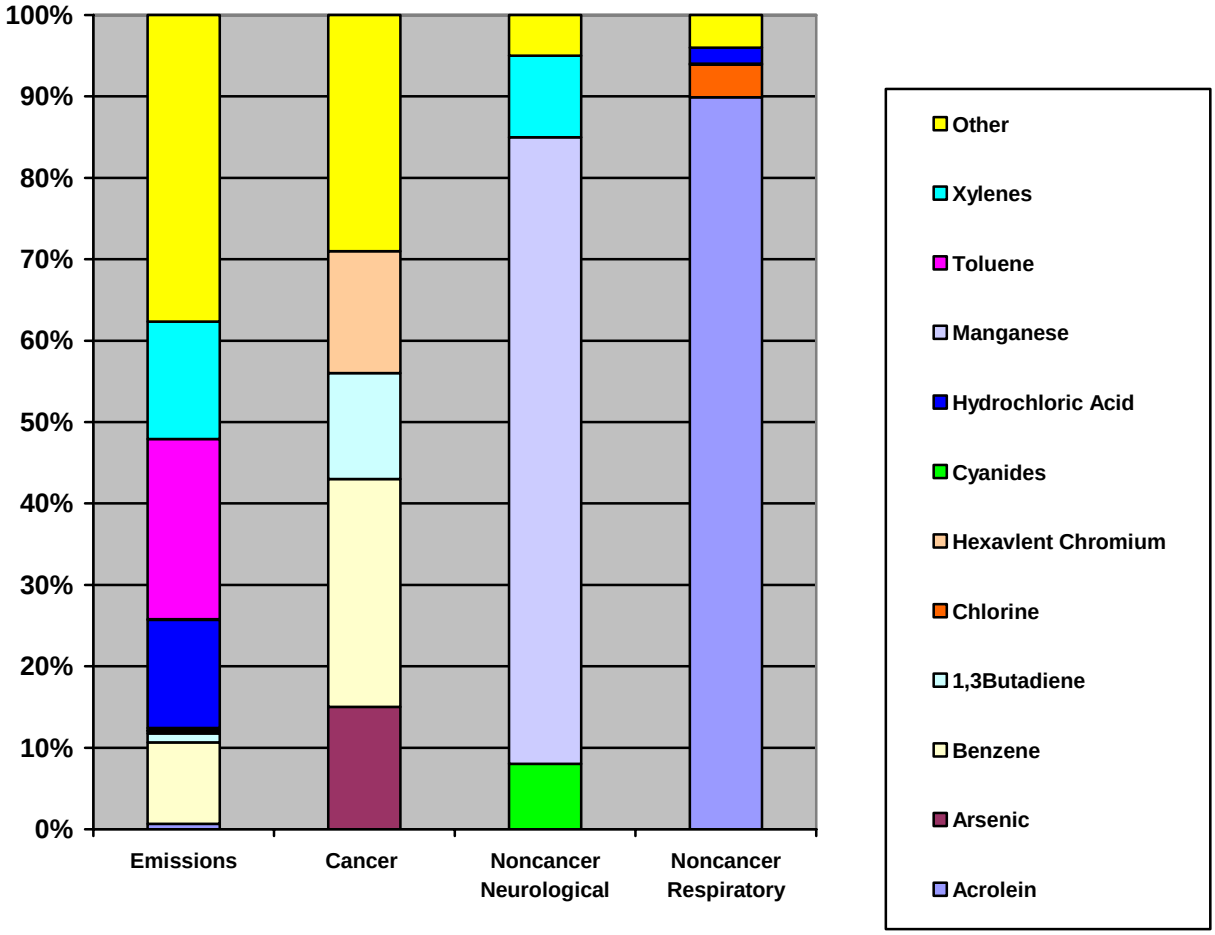
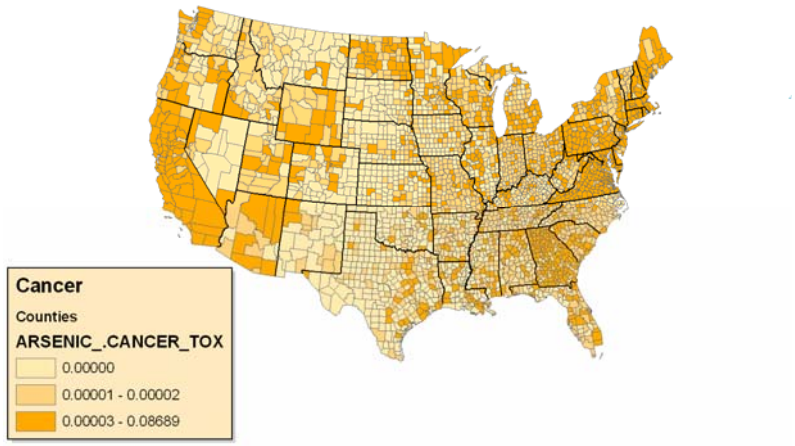


Table 5. Source Sector Contribution to Pollutants in Figure 5

HAP	Largest Sector
Acrolein	Fires
Arsenic	Major
Benzene	Mobile
1,3-Butadiene	Mobile
Chlorine	Major
Hexavalent Chromium	Major
Cyanide	Area
Hydrochloric Acid	Major
Manganese	Major
Toluene	Mobile
Xylenes	Mobile

Figure 6. 2002 Cancer Toxicity-Weighted Emissions Map (Emissions (tpy) x URE (ug/m3): Arsenic and Benzene



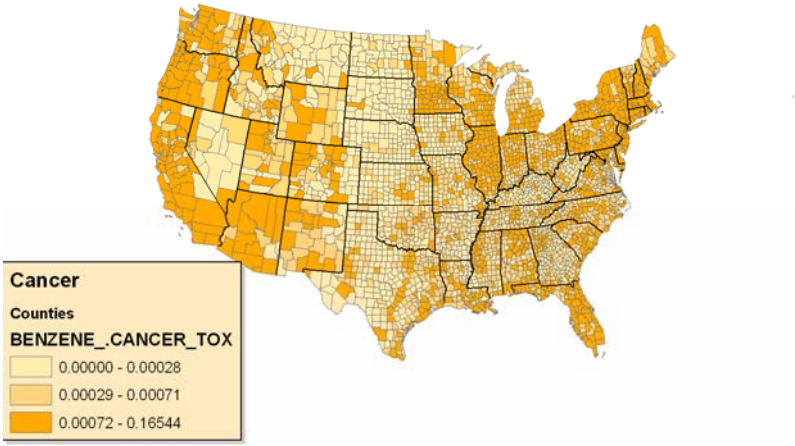
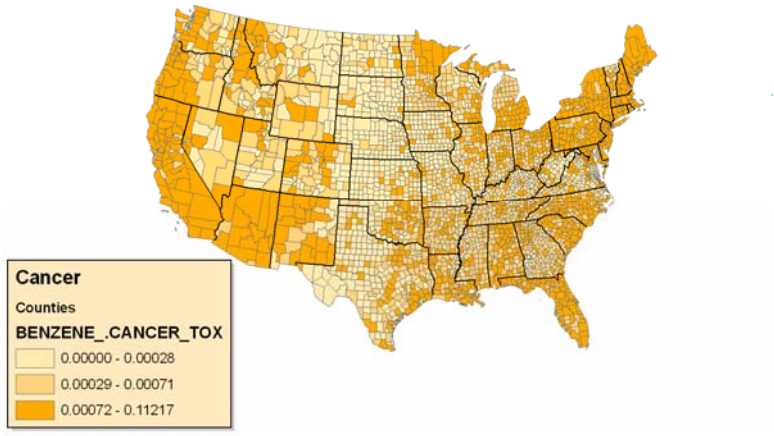


Figure 7. 1990 and 2002 Cancer Toxicity-Weighted Emissions Map (Emissions (tpy) x URE (ug/m3): Benzene

1990:



2002: