

Air Toxics

<http://www.epa.gov/oar/aqtrnd97/chapter5.pdf>

BACKGROUND

Hazardous air pollutants (HAPs), commonly referred to as air toxics or toxic air pollutants, are pollutants known to cause or suspected of causing cancer or other serious human health effects or ecosystem damage. Section 112 of the CAA now lists 188 pollutants or chemical groups as hazardous air pollutants and targets sources emitting them for regulation.¹ Examples of air toxics include heavy metals like mercury and chromium; organic chemicals like benzene, 1,3-butadiene, perchloroethylene (PERC), dioxins, and polycyclic organic matter (POM).

HAPs are emitted from literally thousands of sources including: large stationary industrial facilities or major point sources (such as electric power plants or utilities), smaller area sources (such as neighborhood dry cleaners), and mobile sources (such as automobiles). Adverse effects to human health and the environment due to HAPs can result from exposure to air toxics from individual facilities, exposure to mixtures of pollutants found in urban settings, or exposure to pollutants emitted from distant sources that are transported through the atmosphere over

regional, national or even global airsheds. Exposures of concern to HAPs can be either short-term or long-term in nature. In addition to breathing air contaminated with air toxics, people can also be exposed to some HAPs through other pathways such as through the ingestion of contaminated food from waters polluted from the deposition of HAPs. Some HAPs can bioaccumulate in body tissues. When a predator feeds on contaminated prey, concentrations of these bioaccumulative HAPs can build up in the predator's tissues, magnifying the toxic burden. Presently, over 2,299 U.S. water bodies are under fish consumption advisories (for particular species of fish), representing approximately 16.5 percent of the nation's total lake acreage, and 8.2 percent of the nation's river miles.²

Health and Environmental Effects

Compared to information for the criteria pollutants described in previous chapters, less information is available concerning potential health and environmental effects of the HAPs. Most of the information on potential health effects of these pollutants is derived from experimental animal data. The different health effects which may be caused

by HAPs include cancer, neurological, cardiovascular, and respiratory effects, effects on the liver, kidney, immune system and reproductive system and effects on fetal and child development. The timing of effect and the severity (e.g. minor or reversible vs serious, irreversible, and life-threatening) may vary among HAPs and with the exposure circumstances. In some rare cases, effects can be seen immediately. Rare cases involve the catastrophic release of lethal pollutants, such as the 1984 incident in Bhopal, India where more than 2,000 people were killed from the release of methyl isocyanate into the atmosphere. In other cases, the resulting effects (e.g. liver damage or cancer) are associated with long-term exposures and may not appear until years after exposure. About half of the HAPs have been classified by EPA as "known", "probable" or "possible" human carcinogens. Known human carcinogens have been demonstrated to cause cancer in humans. Examples of these include benzene, which has been shown to cause leukemia in workers exposed over several years to certain amounts in their workplace air, and arsenic, which has been associated with lung cancer in workers at metal smelters. Probable and possible

human carcinogens include chemicals that we are less certain cause cancer in people, yet for which laboratory animal testing indicates carcinogenic effects.

Some HAPs pose particular hazards to people of a certain age or stage in life (e.g. as a young child, adolescent, adult, or elderly person). Available data indicate that about a third of HAPs are developmental or reproductive toxicants (e.g. mercury). This means that exposure during the development of a fetus or young child may prevent normal development into a healthy adult. Other such critical exposures may affect the ability to conceive or give birth to a healthy child. Ethylene oxide, for example, has been associated with increased miscarriages in exposed workers, and has affected reproductive ability in both male and female laboratory animals.

Toxic air pollutants can have a variety of environmental impacts in addition to the threats they pose to human health. Animals, like humans, may experience health problems if they breathe sufficient concentrations of HAPs over time, or ingest HAPs through contaminated food (e.g. fish). Apart from the laboratory testing results on animal species that make up a large portion of the human health effects database, and aquatic toxicity criteria for some HAPs, little quantitative information currently exists to describe the nature and scope of the effects of air toxics on non-human species.

One of the more documented ecological concerns associated with toxic air pollutants is the potential

for some HAPs to damage aquatic ecosystems. For example, a number of studies suggest that deposited air toxics contribute to deleterious effects such as reproductive failures, developmental disorders, disease, and premature death in fish and wildlife species native to the Great Lakes. Deposited air pollutants can be significant contributors to overall pollutant loadings entering water bodies (especially for persistent chemicals which continue to move among air, water, and sediments). For the Great Lakes, international programs have examined the importance of deposition of air toxics, relative to other loadings such as direct discharge. While data are presently insufficient for many quantitative estimates comparing air deposition and other loading pathways, deposition of air toxics to the Great Lakes is considered significant and continues to be investigated with a binational monitoring network, the Integrated Atmospheric Deposition Network (IADN).³

Persistent air toxics are of particular concern in aquatic ecosystems, as toxics levels can magnify up the food chain resulting in exposures greater than those expected based solely on the levels in water or air. For example, the Florida panther, an endangered species which inhabits the Everglades, has experienced adverse effects because of the high levels of mercury that have accumulated in the panther's tissues from eating fish contaminated with mercury. Such "bioaccumulation" and "biomagnification" (where the

levels of a toxic substance increase as you go higher in the food chain) are also seen in breeding loons in New England, a bird which feeds on fish in waters contaminated by airborne mercury. Studies are showing that an estimated 30 percent of the breeding loons have mercury levels that put them at risk of behavioral, reproductive and other effects.

AIR TOXICS CONTROL PROGRAM

The Regulatory Response

In 1990, Congress amended Section 112 of the CAA by adding a new approach to the regulation of HAPs. This new approach is divided into two phases. The first requires the development of technology-based emissions standards for sources of the 188 HAPs. The second phase is to evaluate remaining problems or risks, and develop additional regulations to address sources of those problems, as needed.

The first phase is comprised of the technology-based standards, known as MACT (Maximum Achievable Control Technology) and GACT (Generally Achievable Control Technology) regulations, under Sections 112(d). All large, or major, sources of the 188 HAPs must be addressed by such regulations, as well as the smaller, area, sources found to carry significant risk or identified as important under the Specific Pollutants Strategy [Section 112(c)(6)] or the urban program [Sections 112(c)(3) and 112(k)]. Some combustion sources, such as municipal waste combustors, are regulated under equivalent requirements in Section 129. The purpose of this technology-based approach is to use

Figure 5-1. National total HAP emissions by source type, based on 1993 NTI.

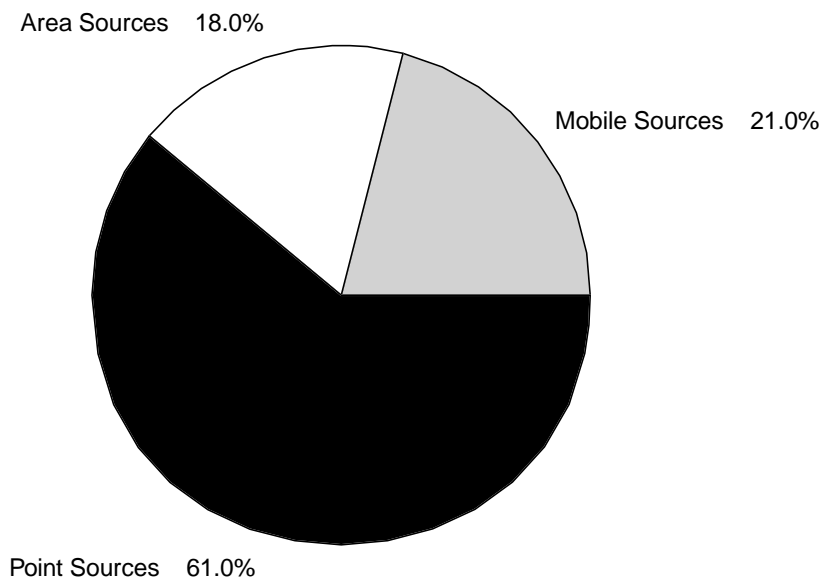
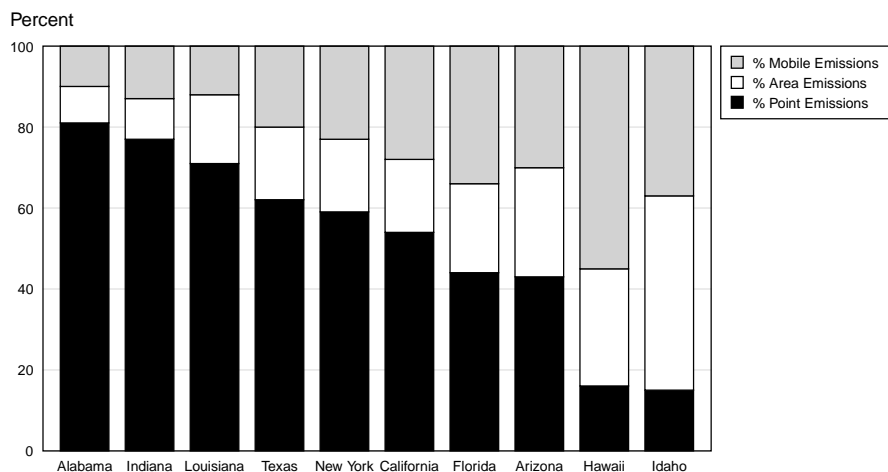


Figure 5-2. Source category contributions for selected states, based on 1993 NTI.



available control technologies or changes in work practices to get emission reductions for as many of the listed HAPs as possible. Although there is no health test in this phase, it is intended that effective MACT standards will reduce a majority of the HAP emissions and, with it, potential risks from regulated sources.

After application of the technology-based standards comes Phase Two, which consists of strategies and programs for evaluating remaining risks and ensuring that the overall program has achieved a sufficient reduction in risks to public health and the environment. This phase will be implemented primarily through the integrated urban air toxics strategy, which will evaluate risks in urban areas from the mix of source types present, and the residual risk program for MACT-controlled sources (Section 112(f)). The integrated urban strategy will identify at least 30 HAPs which have the greatest health risk in urban areas, and assure that area sources accounting for 90% of the total emissions of those urban HAPs are subject to MACT or GACT regulations. In addition, the strategy will target long-term actions to substantially reduce risks from urban air toxics from all sources, to include a 75 percent reduction in cancer risk from major and area sources.

The second phase will also use information generated through the special studies required in the Clean Air Act --the Great Waters program [Section 112(m)], and the Mercury and Utility Studies [Sec-

tion 112(n)]. The Great Waters program contains an ongoing examination of atmospheric deposition of air toxics to aquatic ecosystems, and the effects of those toxics when concentrated through the food web. The Mercury Study examined the adverse effects of, and possible controls for, mercury from all sources. The Utility Study examined health hazards of, and possible controls for, the numerous toxics from electric utilities.

The components of this two phase approach are described in more detail later in this chapter.

The CAA recognizes that not all problems are national problems or have a single solution. Authority for national emission standards are complemented by authorities to examine problems on other scales in order to address specific concerns. The Act also provides mechanisms for increasing partnerships among EPA, States and local programs in order to address problems specific to these regional and local environments. As we move toward the 21st century, EPA's air toxics program is beginning to progress from the more technologically-based approach for regulating toxics to the more risk-based approach. This shift will require more and better information about all emission sources of HAPs, ambient levels of HAPs and human and ecosystem exposure to HAPs. The development of an "information infrastructure" to inform the risk-based decisions has been a priority for the EPA over the last few years. The next part of this chapter summarizes the resulting activities and data from major

Figure 5-3. HAP Emissions by state, based on 1993 NTI.

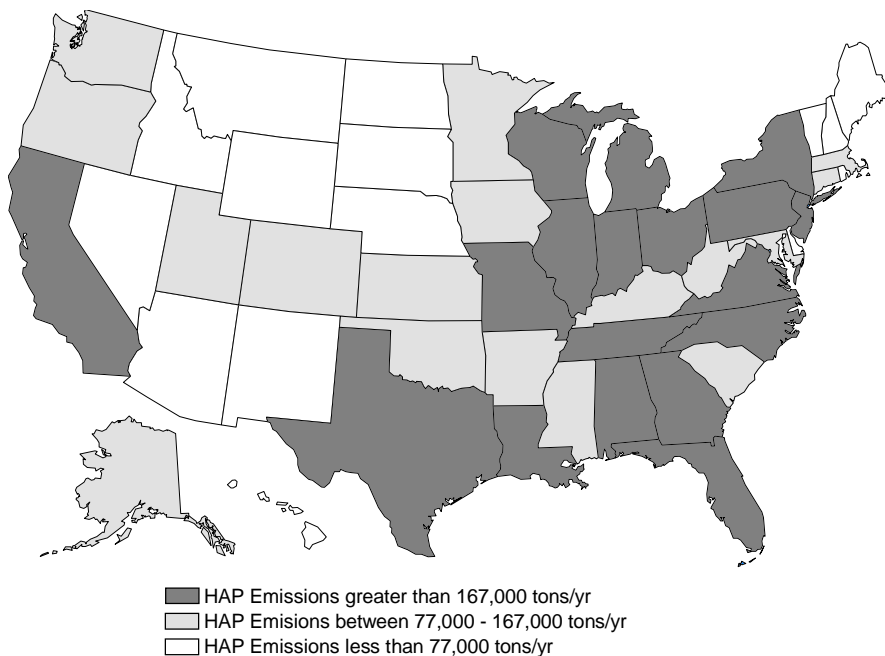


Figure 5-4. State Data Summary for 1996 NTI.

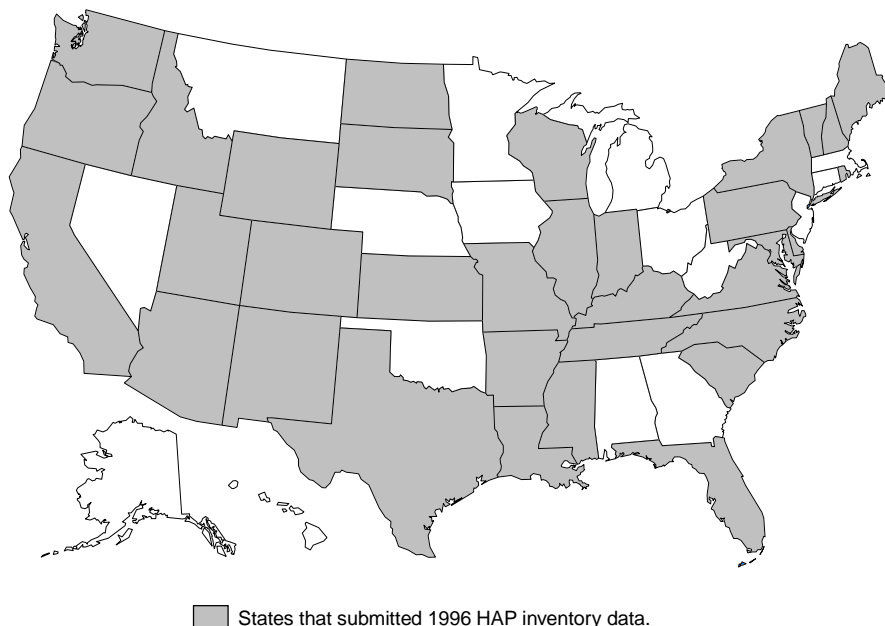


Table 5-1. Emission Reductions from Full Implementation of MACT Standards

Compliance Date	MACT Source Category	HAPs Emitted		Total Baseline Pre-MACT Emissions ^b	Emissions Reduction ^b	Total Post-MACT Emissions ^b
11/15/93	Coke Ovens: Charging, Top side, and Door leaks ^a	Benzene Coke oven gases Polycyclic Organic Matter		1,760 tpy	80% = 1,408 tpy	352 tpy
9/23/96	Perchloroethylene Dry Cleaning Facilities	Perchloroethylene		95,700 tpy	56% = 53,592 tpy	42,108 tpy
3/8/96	Industrial Process Cooling Towers	Chromium & compounds		25 tpy	>99%	0
12/15/96 (w/o new control device), 12/15/97 (w/ new control device)	Magnetic Tape Manufacturing	Methyl ethyl ketone Methyl isobutyl ketone Toluene		4,470 tpy	51% = 2,300 tpy	2,170 tpy
1/25/96 (decorative) 1/25/97 (hard & anodizing)	Chrome Electroplating: – Decorative – Hard – Anodizing	Chromium & compounds		11.5 160 3.9 = 175.4 tpy	99% = 173 tpy	2 tpy
4/22/97	Hazardous Organic NESHAP (HON)	Total unspesiated HAPs		573,000 tpy	90%= 515,700 tpy	57,300 tpy
11/2/97	Halogenated Solvent Cleaning	Methyl chloroform Methylene chloride	Tetrachloroethylene Trichloroethylene	142,000 tpy	60% = 85,200 tpy	56,800 tpy
12/15/97	Gasoline Distribution	Benzene Cumene Ethyl benzene Ethylene dichloride Hexane Lead & compounds	Methyl tert-butyl ether Polycyclic Organic Matter Toluene 2,2,4-Trimethylpentane Xylenes (o,m,p)	44,200 tpy	5% = 2,210 tpy	41,990 tpy
12/16/97	Shipbuilding and Ship Repair Facilities	Acrylonitrile Chlorine Chromium & compounds Diethanolamine Ethylbenzene Ethylene dichloride Ethylene glycol Glycol ethers Lead & compounds Manganese & compounds	Methyl chloroform Methyl ethyl ketone Methyl isobutyl ketone Methylene chloride Nickel & compounds Polycyclic Organic Matter Toluene Trichloroethylene Xylenes (o,m,p)	7,890 tpy	24% = 1,894 tpy	5,996 tpy
12/23/97	Secondary Lead Smelting	Acetaldehyde Acetophenone Acrolein Acrylonitrile Antimony & compounds Arsenic & compounds Benzene Biphenyl Bis (2-ethylhexyl)phthalate 1,3-Butadiene Cadmium & compounds Carbon disulfide Chlorobenzene Chloroform Chromium & compounds Cumene Dibutyl phthalate 1,3-Dichloropropene Dioxins/Furans Ethyl carbamate	Ethylbenzene Formaldehyde Hexane Lead & compounds Manganese & compounds Mercury & compounds Methyl bromide Methyl chloride Methyl ethyl ketone Methyl iodide Methylene chloride Nickel & compounds Phenol Polycyclic Organic Matter Propionaldehyde Styrene 1,1,2,2-Tetrachloroethane Toluene Trichloroethylene Xylenes(o,m,p)	2,030 tpy	72% = 1,421 tpy	609 tpy

^a Due to the various criteria for implementation dates for coke ovens, the date shown here is the Effective Date.

^b tons per year is abbreviated as tpy.

sectors of that infrastructure -- emissions inventories, ambient monitoring, and modeling. Data from these three areas are already assisting the EPA in more quantitatively characterizing the air toxics problems. As the information continues to improve, it will provide the base for air toxics decisions in the future.

AIR TOXICS CHARACTERIZATION

Emissions Data - National Toxics Inventory

In 1993, there were approximately 8.1 million tons of air toxics released to the air according to EPA's National Toxics Inventory (NTI). These emissions came from all types of manmade sources, including large industrial sources, small stationary sources, and mobile sources. As shown in Figure 5-1, 1993 NTI estimates reveal that point, or major, sources (sources of hazardous air pollutants (HAPs) emitting more than 10 tons per year of an individual HAP or 25 tons per year of aggregate emissions of HAPs) account for approximately 61 percent of the total HAP emissions, nationally, while area sources (smaller stationary sources) contribute approximately 18 percent, and mobile sources contribute 21 percent. Since these estimates vary from State to State, Figure 5-2 illustrates the range in percent contributions of point, area, and mobile source emissions for selected states. Figure 5-2 shows that point source contributions ranged from 81 percent (Alabama) to 16 percent (Hawaii), area source contributions ranged from 48

Table 5-2. Comparison of 1993 to 1996 Emission Reductions for Mobile On-Road Gasoline Vehicles

1993 Total HAP Emissions (tons per year)	1996 Total HAP Emissions (tons per year)	Emissions Reduction (tons per year)
1,571,000	1,313,000	258,000 = 16%

Table 5-3. HAPs Emitted From On-Road Gasoline Vehicles

Acetaldehyde	Manganese and compounds
Acrolein	Mercury and compounds
Arsenic and compounds	Methyl tert-butyl ether ^a
Benzene	Nickel and compounds
1,3-Butadiene	Polycyclic Organic Matter (defined as 16-PAH)
Chromium and compounds	Propionaldehyde
Dioxins/Furans (defined as TEQ)	Styrene
Ethylbenzene	Toluene
Formaldehyde	Xylenes (ortho-,meta-,para-)
n-Hexane	
Lead and compounds	

^a not available for the 1993 inventory year

percent (Idaho) to 9 percent (Alabama), and mobile source contributions ranged from 55 percent (Hawaii) to 10 percent (Alabama). Figure 5-3 presents the geographic distribution of 1993 emissions of HAPs by mass at the State level. While this figure shows total emissions of HAPs for each State, it does not imply relative health risk from exposure to HAPs. The categorization of pollutant emissions as high, medium, and low only provides a rough sense of the geographic distribution of emissions. In viewing these figures it is important to note that some states may show relatively high emissions totals as a result of very large emissions from a few facilities. Likewise, relatively large

emissions totals may also result from numerous small point sources.

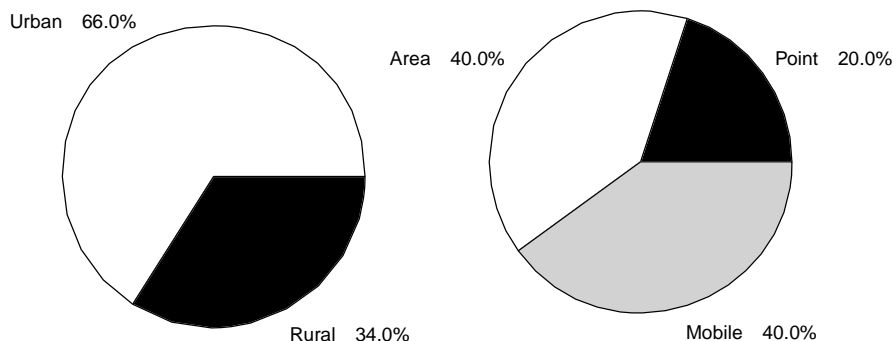
The EPA periodically updates the NTI and is currently compiling the 1996 NTI. The 1993 NTI includes emissions information for 166 of the 188 HAPs from 958 point-, area-, and mobile-source categories. Emissions data from the Toxic Release Inventory (TRI) were used as the foundation of the 1993 NTI. However, the TRI's lack of emission estimates from mobile and area sources severely limit its utility as a comprehensive air toxics emissions database.⁴ Analysis of the 1993 NTI suggests that the TRI data alone represent less than 10 percent (760,000 tons/year) of the total NTI emissions. The NTI, therefore,

Table 5-4. 33 Draft Urban HAPs^a

HAP Name	CAS Number
Acetaldehyde	75070
Acrolein	107028
Acrylonitrile	107131
Arsenic and compounds	
Benzene	71432
Bis(2-ethylhexyl)phthalate	117817
1,3-Butadiene	106990
Cadmium and compounds	
Carbon tetrachloride	56235
Chloroform	67663
Chromium and compounds	
1,4-Dichlorobenzene	106467
1,3-Dichloropropene	542756
Dioxins/Furans (defined as TEQ)	
Ethylene dibromide (dibromoethane)	106934
Ethylene dichloride (1,2-dichloroethane)	107062
Ethylene oxide	75218
Formaldehyde	50000
Hydrazine	302012
Lead and compounds	
Manganese and compounds	
Mercury and compounds	
Methylene chloride (dichloromethane)	75092
Methylene diphenyl diisocyanate (MDI)	101688
Nickel and compounds	
Polycyclic organic matter (defined as 16-PAH)	
Propylene dichloride (1,2-dichloropropane)	78875
Tetrachloroethylene (perchloroethylene)	127184
Trichloroethylene	79016
Vinyl chloride	75014
Coke oven emissions	
Methyl chloride	
Quinoline	

^a The first 30 HAPs in this list have significant contributions from area source categories, and thus, required listing under sections 112(c)(3) and 112(k). The last 3 HAPs, coke oven emissions, methyl chloride, and quinoline are predominantly emitted by major sources.

Figure 5-5. National emissions of 30 draft urban area source HAPs, 1990.



sought other sources to fill in the gaps. Data from EPA studies, such as the Mercury Study,⁵ inventories for Clean Air Act sections 112c(6) and 112(k), and data collected during development of Maximum Achievable Control Technology (MACT) Standards under section 112(d), supplement the TRI data in the NTI.

The 1993 and 1996 NTIs also incorporate data from State and local HAP inventories. The 1996 NTI currently includes 38 HAP inventory data from State and local agencies (representing 34 States). Figure 5-4 shows the States that have submitted 1996 HAP inventory to EPA. Thus, the State and local HAP inventories will form the new foundation of the 1996 NTI. One other important distinction between the 1993 and 1996 NTI is that 1993 data are allocated at the county level, whereas, the 1996 NTI data will be allocated at the facility level for point (major) sources. This allows for greater spatial detail in the 1996 NTI. The complete 1996 NTI is targeted for completion in the Fall of 1999.

As a result of the implementation of MACT standards, point source emissions are estimated to have decreased by over 660,000 tons from 1993 through 1997. Table 5-1 presents a summary of estimated emission reductions from the full implementation of MACT standards with compliance dates through 1997.

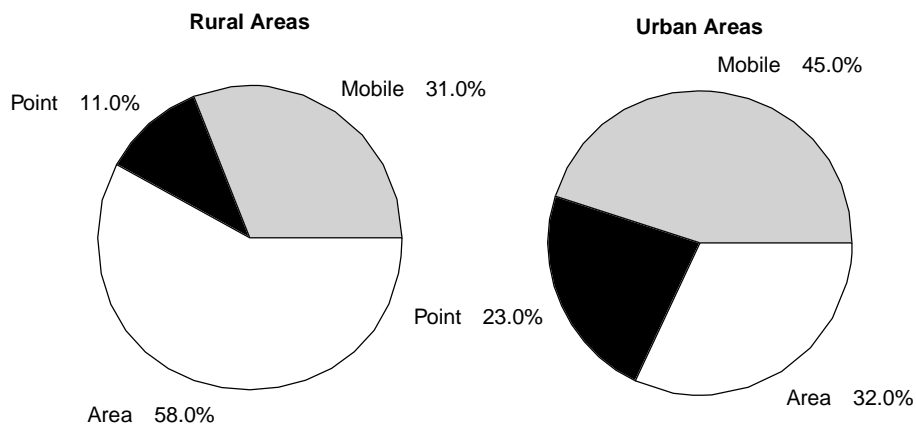
Table 5-2 compares 1993 and 1996 mobile on-road source emissions. Mobile on-road emissions decreased by 258,000 tons over this time period, primarily as a result of

regulations requiring the use of reformulated fuels. (See the Ozone section in Chapter 2: National Trends for Criteria Pollutants.) Table 5-3 lists HAPs emitted from on-road gasoline vehicles that have emission estimates in the 1993 and 1996 NTIs. Although the EPA addresses stationary and mobile sources under separate regulatory authorities and through separate offices, these emissions are being evaluated together in EPA's air toxics strategies. Section 202(l) requires EPA to regulate the emissions of HAPs from motor vehicles. EPA's reformulated gasoline program requires a 15 percent year-round reduction in the total mass of toxic emissions. EPA's Office of Mobile Sources has provided estimation methodologies for the mobile source-emitted HAPs included in the NTI.

The EPA is compiling the NTI every three years (1993, 1996, 1999, etc.) The emissions estimates in the NTI, regardless of base year, have several limitations. The NTI is a repository of HAP emissions data from various sources, and it varies in quality and completeness among source categories, geographic location, and estimation methods. As the process of compiling these data is evolving, estimates will likely improve. However, as new base year inventories are compiled and source category and emissions calculation methods change, emissions estimates are likely to change over time because of these factors as well as because of actual changes in emissions.

EMISSIONS DATA

Figure 5-6. National urban and rural emissions of 30 draft urban area source HAPs, 1990.



Section 112(k) Interim Inventory for Urban HAPs

To support the regulation of area sources in urban areas under CAA Section 112(k), which requires selecting at least 30 HAPs that pose the greatest threat to public health in the largest number of urban areas, EPA compiled an interim 1990 emissions inventory for proposed urban HAPs. The EPA conducted an analysis to comprehensively identify the pollutants that pose the largest public health threat in urban areas. The result was a draft list of 33 urban HAPs. Of these, 3 are emitted predominantly from major sources and 30 have emissions from multiple source types, but with significant contributions from area sources. Table 5-4 lists the 33 draft urban area source HAPs. Figures 5-5 and 5-6 present summary data from the interim urban area emissions inventory for the 30 HAPs significantly associated with area sources. Figure 5-5 indicates that area sources account for 40 percent of emissions of these 30 HAPs, mobile sources account for 40 percent, and

point (major) sources account for 20 percent. Further, Figure 5-5 also shows that urban emissions of these 30 HAPs account for 66 percent of total emissions while rural emissions account for 34 percent. Figure 5-6 summarizes source type contributions of emissions of these 30 HAPs by rural and urban geographic areas. Urban geographic areas have higher proportions of mobile and point sources than rural areas. The 1990 interim urban emission inventory has undergone extensive external and internal review and has subsequently been incorporated into the 1993 NTI. The urban inventory is available at the following website.

www.epa.gov/ttn/uatw/112k/riurban.html

It should be noted that the percentage contributions (mobile, point, area) shown in Figure 5-5 for the interim urban inventory differ significantly from those shown in Figure 5-1 for the 1993 NTI. The primary reason for this difference is that the interim urban inventory includes data for only the 30 urban HAPs while the 1993 NTI incorpo-

Table 5-5. Summary of Changes in Mean Concentration for HAPs Measured as a Part of the PAMS Program (24-hour measurements), 1995-1997*

HAP	1995 to 1996			1996 to 1997		
	# Sites	# Up	# Down	# Sites	# Up	# Down
Acetaldehyde	2	0	0	4	1	1
Benzene	5	1	2	12	0	4
Ethylbenzene	5	0	2	12	0	2
Formaldehyde	2	0	0	4	1	1
Hexane	4	0	0	10	0	2
Toluene	5	0	1	12	0	1
Styrene	5	1	2	11	1	5
m/p-Xylene	5	0	0	10	0	3
o-Xylene	5	0	1	12	1	3
2,2,4-Trimethyl-pentane	5	0	3	11	0	4

* Note that the terms #Up and #Down refer to the number of sites in which the change in annual mean concentration between 1995 and 1996, or 1996 and 1997, is a statistically significant increase or decrease. The total number of sites (# sites) may not necessarily equal the sum of the corresponding #Up and #Down categories.

Table 5-6. Comparison of Loading Estimates for the Great Lakes

Chemical	Year	Superior (kg/yr)	Michigan (kg/yr)	Huron (kg/yr)	Erie (kg/yr)	Ontario (kg/yr)
PCBs (wet+dry)	1988	550	400	400	180	140
	1992	160	110	110	53	42
	1994	85	69	180	37	64
	1996	50	42	na	34	na
DDT (wet+dry)	1988	90	64	65	33	26
	1992	34	25	25	12	10
	1994	17	32	37	46	16
	1996	4	12	na	2	na
B(a)P (wet+dry)	1988	69	180	180	81	62
	1992	120	84	84	39	31
	1994	200	250	na	240	120
	1996	77	117	na	160	na
Pb (wet+dry)	1988	230,000	540,000	400,000	230,000	220,000
	1992	67,000	26,000	10,000	97,000	48,000
	1994	51,000	72,000	100,000	65,000	45,000
	1996	na	na	na	na	na

rates data for 166 HAPs. Further, the subset of 30 pollutants included in the interim urban inventory are those with significant contributions from area sources, as indicated above. An analysis of the 1993 NTI inventory for the subset of 30 urban HAPs indicates similar percentage contributions to those shown in Figure 5-5.

Ambient Air Quality Data

Presently, there is no national ambient air quality monitoring network designed to perform routine measurements of air toxics levels. Therefore, ambient data for individual air toxic pollutants is limited (both spatially and temporally) in comparison to the data available from the long-term, nationwide monitoring for the six criteria pollutants. EPA has several efforts underway which, although less effective than a comprehensive and routine HAPs network, will provide some information useful to assessing the toxics issue.

The Agency's Photochemical Assessment Monitoring Stations (PAMS) collect data on concentrations of ozone and its precursors (VOCs and NO_x) in 21 areas across the nation classified as serious, severe or extreme nonattainment areas for ozone. Because a number of ozone precursors are also air toxics, ambient data collected from PAMS sites can be used for limited evaluations of toxics problems in selected urban areas as well as assessment of the tropospheric ozone formation. Despite some limitations, the PAMS sites will provide consistent, long-term measurements of selected toxics in major metropolitan areas. The

PAMS program requires routine measurement of 10 HAPs: acetaldehyde, benzene, ethyl benzene, formaldehyde, hexane, styrene, toluene, m/p-xylene, o-xylene and 2,2,4-trimethylpentane.

Preliminary analysis of measurements of selected HAPs in PAMS areas indicate that concentrations of certain toxic VOCs in those areas appear to be declining. Table 5-5 shows 2-year comparisons for 24-hour measurements for the ten parameters measured at PAMS sites for the periods 1995-1996 and 1996-1997.⁶ For a more detailed discussion of the PAMS program, see the Ozone section in Chapter 2: National Trends of Criteria Pollutants.

In addition to the PAMS program, EPA continues to administer and support voluntary programs through which states may collect ambient air quality measurements for suites of toxics. These programs include the Urban Air Toxics Monitoring Program (UATMP), as well as the Non-Methane Organic Compound (NMOC) and Speciated Non-Methane Organic Compound (SNMOC) monitoring programs. The UATMP is the program dedicated to toxics monitoring which involves measurements of 37 VOCs and 13 carbonyl compounds.⁷ In the current programs, five states are participating and operating 15 ambient measurement sites for toxics.⁸ The EPA is currently working to incorporate data from these measurement programs into national and local air toxics assessments. Results from these analyses

will be discussed in future editions of this report.

In addition to ambient concentration monitoring, EPA also participates in efforts to measure the atmospheric deposition of toxic pollution. In 1990 the U.S. and Canada initiated a joint measurement program, the Integrated Atmospheric Deposition Network (IADN), to assess the relative importance of atmospheric deposition to the Great Lakes, and to provide information about sources of these pollutants.⁹ The network consists of master (research-grade) stations on each lake, with additional satellite stations. Two master stations in Canada and three in the United States were chosen to be representative of regional deposition patterns. In addition to collecting data on precipitation rates, temperature, relative humidity, wind speed and direction, and solar radiation collected at each site, IADN measures concentrations of target chemicals in rain and snow (wet deposition), airborne particles (dry deposition), and airborne organic vapors.¹⁰

Table 5-6 presents the results of a comparison of deposition estimates from IADN studies performed between 1988 and 1996 (1996 loadings estimates for Lake Huron and Lake Ontario are not yet available). These estimates reveal a significantly large decline in loadings from the atmosphere of PCBs and DDT, which would be expected considering that use of DDT has been restricted since 1972 and banned since 1988, and PCBs are no longer manufactured and are being phased out of use. The vast

majority of the PCBs and DDT being recorded by the monitoring network results from emissions (via chemical volatility) from soil and water contaminated with these chemicals and transport to the lakes. On the other hand, benzo(a)pyrene (B(a)P), a by-product of combustion processes and one of the most toxic polycyclic aromatic hydrocarbons yet characterized, is being emitted by a number of current sources around the Great Lakes.

Loadings to the lakes show some variability at each site from year to year, probably due in part to the range of error inherent in the B(a)P loadings calculations and to fluctuations in ambient concentrations.¹¹

Mercury's adverse effects on ecological and public health have raised the level of awareness regarding its persistence in the environment. As a result, there has been a concerted effort by local, State, and national environmental agencies to accurately measure the annual progress of regulations and technologies aimed at reducing mercury. The Mercury Deposition Network (MDN) is a key element of these efforts. A subsidiary of the National Atmospheric Deposition Program (NADP), the MDN currently consists of nearly 40 sites located in 16 States and two Canadian provinces. The MDN monitors the presence of mercury and methyl mercury in precipitation and has enabled scientists to compile a national database of weekly precipitation concentrations. As a result, State and federal air regulators can monitor progress in reducing

mercury and amend policy decisions accordingly. There are plans to expand the network in the near future, pending availability of new funds. Additional information about the network is available on the Internet at <http://nadp.sws.uiuc.edu/mdn/>.

Concurrent with the monitoring efforts discussed in this section, EPA has recently initiated a program to identify, compile and catalogue all previously collected monitoring data for air toxics which is not now centrally archived. This effort is focusing presently on the compilation of measurements previously made by state and local agencies. These data will contribute to the development of an expanded and enhanced information infrastructure for air toxics.¹² All data completed as a result of this effort will be made universally accessible to all interested programs and analysts.

In addition, the Agency is also sponsoring a related project to develop environmental indicators based on air quality monitoring data, emissions data, modeling data, and administrative/programmatic data that can effectively demonstrate the extent and severity of the air toxics problem, and any progress made toward solving it in future years through regulatory or voluntary programs. Indicators will be included that consider population exposure and health risk, as well as ambient concentrations and emissions. Such indicators will be used to make geographic comparisons and assess temporal trends in subsequent trends reports.¹³

Modeled Air Quality Levels

The EPA has recently developed and demonstrated a national air toxics modeling system, as part of its Cumulative Exposure Project (CEP), which has been designed to provide a broad screening-level quantitative perspective on outdoor air toxics concentrations. The CEP utilized an atmospheric dispersion model, called ASPEN (the Assessment System for Population Exposure Nationwide), in conjunction with a preliminary national emissions inventory, including both human-made and natural sources of emissions, to estimate the ambient air toxics concentrations for the year 1990. The outputs generated by the model were annual average outdoor concentrations for 148 air toxics, estimated at each census tract in the continental U.S. (There are 60,803 census tracts in the continental U.S. which vary in physical size but generally have about 4,000 residents each.) Given the inherent uncertainties involved in computer modeling and emission inventories, the CEP results are most reliable when analyzed on a national or State scale, but less so at the county and census tract levels.

The results of this preliminary application of the model for 1990, although considered to include uncertainties, indicate that outdoor HAP concentrations may be significantly elevated in many urban areas when compared to rural areas, and that many predicted levels are relatively high, suggesting potential public health concerns. The results thus lend support to the decision of Congress in 1990 to strengthen the air toxics provisions of the Clean Air Act. It is important to note,

however, that these preliminary CEP estimates for 1990 do not reflect the reality that, since that time, the EPA and State and local governments have issued regulations that have reduced and will continue to significantly reduce air toxic emissions from major industries, such as chemical plants and oil refineries, as well as from motor vehicles. Specific results from the 1990 run of the model can be reviewed on the Internet at <http://www.epa.gov/CumulativeExposure>.

Further development and demonstration of the CEP modeling approach, now referred to as CEP-II, will represent a significant evolution in the ability of the EPA to assess the nature and magnitude of the air toxics problem on a national scale. In its CEP-II efforts, the EPA is currently using model performance evaluations and improved emissions information from State and local agencies to update and refine the methods employed in the original CEP analysis. Later this year, the EPA plans to use the CEP-II model based on the improved 1996 NTI emissions inventory. In future years, EPA plans to continue to refine the modeling approach and utilize results from these model simulations to help track air toxics problems and the success of efforts to address them. Results from these simulations will be included in future Trends Reports to provide a national perspective on air toxics trends.

AIR TOXICS REGULATION AND IMPLEMENTATION STATUS

The CAA greatly expanded the number of industries affected by national air toxics emissions controls. Large industrial complexes (major sources) such as chemical plants, oil refineries, marine tank vessel loading, aerospace manufacturers, steel mills, and a number of surface coating operations are some of the industries being controlled for toxic air pollution. Where warranted, smaller sources (area sources) of toxic air pollution such as dry cleaning operations, solvent cleaning, commercial sterilizers, secondary lead smelters, and a chrome plating are have also been regulated. EPA estimates that over the next 10 years the technology-based phase of the air toxics program will reduce emissions by 1.5 million tons per year.¹⁴

The MACT Program

The technology-based regulation of air toxics emissions is already beginning to achieve significant emissions reductions of HAPs. As of September 1997, Maximum Achievable Control Technology (MACT) standards have been promulgated for 52 source categories. Sixteen major- and eight area-source categories have begun to take action toward complying with the standards required by the 2- and 4-year MACT regulations. Sources are required to comply with these standards within three years of the effective date of the regulation, with some exceptions. In October 1997, to comply with Section 112(s), EPA released a report to Congress describing the status of the HAP program under the CAA. EPA estimates that the 2-

and 4-year standards will reduce HAP emissions by approximately 980,000 tons/year when fully implemented.¹⁴ In addition, EPA has promulgated regulations on municipal waste combustors and hospital/medical/infectious waste incinerators under Section 129 of the CAA. These regulations will significantly reduce emissions of the listed Section 129 pollutants, including particulate matter, sulfur dioxide, hydrogen chloride, oxides of nitrogen, carbon monoxide, lead, mercury, dioxins and dibenzofurans. For example, mercury emissions from municipal waste combustors are estimated to be reduced in the year 2000 by about 98 percent from 1990 levels. When the regulations become fully effective, mercury emissions from hospital/medical/infectious waste incinerators are estimated to be reduced by 93-95 percent from 1995 levels.

The air toxics program and other air pollution programs, such as the NAAQS program, complement each other. Many air toxics are also particulate matter (PM) or VOCs which can be ozone precursors. As such, many control efforts to meet the NAAQS for ozone and PM10 also reduce air toxic emissions. Furthermore, as air pollution control strategies for automobiles become more stringent, air toxic emissions from vehicles also are reduced. Requirements under the air toxics program can also significantly reduce emissions of some of the six NAAQS pollutants. For example, MACT standards are predicted to reduce approximately 1.8 million tons per year in combined emissions of particulate

matter and VOC as ozone precursors. EPA's final air toxics rule for organic chemical manufacturing alone is expected to reduce VOC emissions by nearly 1 million tons annually.

The Specific Pollutants Strategy

Section 112(c)(6) of the CAA requires EPA to identify sources of seven specific compounds (alkylated lead compounds, POM, mercury, hexachlorobenzene, PCBs, 2,3,7,8-tetrachlorodibenzo-p-dioxin, and 2,3,7,8-tetrachlorodibenzofuran), and to regulate sources accounting for at least 90 percent of the emissions of each pollutant.²⁰ MACT standards must be developed by EPA for sources of these HAPs that are not subject to current MACT standards. In order to meet the requirements of Section 112(c)(6), EPA compiled national inventories of sources and emissions of each of the seven HAPs, and identified source categories for regulation.¹⁵

The Integrated Urban Air Toxics Strategy

To address the problem of exposure to air toxics in urban areas, EPA is developing an integrated urban air toxics strategy that addresses the urban air toxics risks from both stationary and mobile sources. This strategy is expected to produce a set of actions that will be more responsive to the cumulative risks presented by multiple sources of toxics and combined exposures to multiple toxics. By considering urban air toxics emissions from all sources, EPA will better respond to the relative risks posed by any one pollutant and/or source category. Thus, integration of the activities

under both the air toxics and mobile source sections of the Act will more realistically address the total exposure and will better allow us and the States to develop activities to address risks posed by toxic pollutants where the emissions and risks are most significant and controls are most cost effective.

Under the urban program, EPA's first phase regulatory task is to identify at least 30 HAPs that are of particular concern when emitted in urban areas, especially from area sources. Then, EPA is to reduce risks from these pollutants, first by regulating sources that account for 90 percent of the emissions of the HAPs of concern. Regulations of area sources under this program can be with MACT or GACT regulations. In September of 1998, EPA released a draft of the Integrated Urban Air Toxics Strategy to the public for comment. In the draft strategy, EPA provided a draft list of 33 HAPs that are of particular concern when emitted in urban areas, including a subset of 30 particularly relevant to area sources. EPA also provided a draft list of area source categories that may require regulation in the future to meet the "90% requirement". This list may change before it is published as final.

After identifying the HAPs and their sources, EPA will examine the MACT, vehicle fuels and emissions program, and other air toxics-related programs, including State and local programs, to assess the risk reduction that can be reasonably expected once these other programs have been fully implemented. Then EPA will

identify what additional controls may be needed. Since point, area, and mobile sources in urban areas emit many of the same pollutants EPA believes this integrated approach is the most efficient and cost-effective way to address the health risks attributable to exposure to air toxics in urban areas. In the draft strategy published in September of 1998, EPA included schedules for potential actions to address HAP emissions from mobile sources and stationary sources (including consideration of the requirements to reduce cancer risk from these sources by at least 75 percent and to substantially reduce other risks). Schedules for developing tools and databases to better understand cumulative exposures to HAPs were also included in the draft strategy.¹⁶

Residual Risk

To determine whether risks are acceptable after the application of MACT standards, Congress added, in Section 112(f), a human health risk and adverse environmental effects criteria to the second regulatory phase. In this phase, referred to as "residual risk" standard setting, EPA is required to promulgate additional standards for those source categories that are emitting HAPs at levels that present an unacceptable risk to the public or the environment. Congress directed that residual risk standards should "provide an ample margin of safety to protect public health." Risks of cancer and other health effects, as well as the potential for adverse environmental effects will be considered in setting residual risk standards. Using a risk man-

agement framework, EPA is currently conducting residual risk assessments to determine whether technology-based emission standards are sufficient to protect human health. The first rules, if necessary, are due in 2002.

EPA is also required by Section 112(f)(1) of the Act to provide a report to Congress describing the methodology of approaches assessing these residual risks, the public health significance of any remaining risks, and technical and economic issues associated with controlling the risks. The report is currently scheduled for publication in early 1999.

SPECIAL STUDIES/PROGRAMS

The Great Waters Program

Section 112(m) of the CAA requires the Agency to study and report to Congress every two years on the extent of atmospheric deposition of HAPs and other pollutants to the Great Lakes, Chesapeake Bay, Lake Champlain, and coastal waters, and the need for new regulations to protect these water bodies. The pollutants of concern to this effort include mercury, chlorinated organics and other persistent, bioaccumulating HAPs, as well as nitrogen compounds. This program coordinates with extensive research programs to provide new understanding of the complicated issue of atmospheric deposition of air pollution to water bodies. New scientific findings are incorporated into each required biennial report to Congress and appropriate regulatory recommendations are made as warranted by those findings. This statute provides the authority to

introduce new regulations or influence those under development in order to prevent adverse effects from these pollutants to human health and the environment.

The Mercury Study

The Mercury Study is a comprehensive study of mercury emissions from anthropogenic sources in the United States, an assessment of the public health and ecological effects of such emissions, and an analysis of technologies to control mercury emissions, and the costs of such control. The study is mandated by Section 112(n)(1)(B) of the CAA because mercury is, as an element, eternally persistent, as well as bioaccumulative and the cause of fish consumption advisories in more than 39 States. A number of observations can be made regarding trends in mercury use and emissions. The overall use of mercury by industrial and manufacturing source categories has significantly declined. Industrial use of mercury declined by nearly 75 percent between 1988 and 1995. Much of this decline can be attributed to the elimination of mercury as a paint additive and the phase-out of mercury in household batteries. Reducing mercury in manufactured products is important because emissions of mercury are most likely to occur when these products are broken or discarded. Based on trends in mercury use, EPA predicts that manufacturing use of mercury will continue to decline. Chlorine production from mercury cell chlor-alkali plants will continue to account for most of the use in, and emissions from, the manufacturing sector. This industry has indicated it will voluntarily reduce mercury

use by 50 percent by 2006. Secondary production of mercury may increase as more recycling facilities begin operations to recover mercury from discarded products and wastes. A significant decrease will occur in mercury emissions from municipal waste combustors and medical waste incinerators when the final regulations promulgated by EPA for these source categories are fully implemented. Emissions from both categories will decline by at least 90 percent from 1995 levels; to roughly 6 tons per year from municipal waste combustors and 1 ton per year from medical waste incinerators. In addition, EPA has proposed mercury emission limits for hazardous waste combustors. Based on 1995 estimates, coal-fired utility boilers are the largest source category at 52 tons per year. Future mercury emissions from utility boilers depend on a number of factors including the nation's energy needs, fuel choices, industry restructuring and other requirements under the CAA (e.g., the acid rain program). A recent EPA analysis also predicted mercury emissions will decline at least 11 tons per year as a result of implementation of the ambient standards for fine particulate matter. International efforts to reduce greenhouse gases will also reduce mercury emissions. The Mercury Study Report to Congress was completed in December 1997.⁵

The Utility Air Toxics Study

As mandated by Section 112(n)(1)(A) of the CAA, the Agency has completed a study of HAP emissions from fossil fuel-fired (coal, oil, and gas) electric utilities, the associated hazards to

public health, as well as an assessment of alternative emissions control strategies. EPA released a final report from this study in February, 1998.¹⁷ The report identifies 67 HAPs emitted from electric utilities, and predicts that by the year 2010 a 30 percent increase in HAP emissions from coal-fired utilities while emissions from oil-fired utilities are estimated to decline by 50 percent. Though substantial uncertainties exist in these future emission estimates, they are based on projected energy demands, changes in fuel mix at utilities, and expected emission reductions from the Acid Rain Program. While significant uncertainty exists regarding the risks posed from HAP emissions, the main pollutants of concern from electric utilities include: mercury, arsenic, nickel, and dioxin. Due to the uncertainties mentioned, the Agency has deferred a final decision on the need for additional control of HAP emissions from electric utilities until more information on this industry is available.

REFERENCES

1. This list originally included 189 chemicals. The CAA allows EPA to modify this list if new scientific information becomes available that indicates a change should be made. Using this authority, the Agency modified the list to remove caprolactam in 1996, reducing the list to 188 pollutants (Hazardous Air Pollutant List; Modification, 61 FR 30816, June 18, 1996).
2. "Update: Listing of Fish and Wildlife Advisories," announcing the availability of the 1996 update for the database: Listing of Fish and

Wildlife Advisories (LFWA); U.S. EPA Fact Sheet, EPA-823-97-007, June 1997.

3. Hillery, B.R., Hoff, R.M., and Hites, R.A. 1997. "Atmospheric contaminant deposition to the Great Lakes determined from the Integrated Atmospheric Deposition Network." Chapter 15 in *Atmospheric Deposition of Contaminants to the Great Lakes and Coastal Waters*. 1997, Joel E. Baker, Editor. SETAC Press. (Society of Environmental Toxicology and Chemistry.)

4. In addition to the absence of emissions estimates for area and mobile source categories, there are other significant limitations in the TRI's portrayal of overall HAP emissions. First, facilities with Standard Industrial Classification (SIC) codes outside the range of 20 to 39 (the manufacturing SICs) are not required to report. Therefore, HAP emissions from facilities such as mining operations, electric utilities, and oil and gas production operations are not represented in the TRI. Further, TRI data are self-reported by the emitting facilities, and TRI does not require facilities to perform any actual monitoring or testing to develop their reported estimates. Consequently, the accuracy of the reported data may vary from facility to facility and from year to year. Finally, the original TRI list only required reporting for 173 of the 188 HAPs identified in the CAA.

5. Mercury Study Report to Congress. Volume II. *An Inventory of Anthropogenic Mercury Emissions in the United States*. EPA-452/R-97-004b. The report can also be access via the internet at: [http://](http://www.epa.gov/ttnuatw1/112nmerc/mercury.html)

www.epa.gov/ttnuatw1/112nmerc/mercury.html

6. Summaries of the health effects associated with the compounds included in this analysis are provided below:

Acetaldehyde: The primary effects on humans, reported from short-term exposure to low to moderate levels of acetaldehyde, are irritation of eyes, skin, and respiratory tract. Short-term exposure effects on animals also include slowed respiration and elevated blood pressure. Effects on humans from long-term acetaldehyde exposure resemble those of alcoholism. Long-term exposures of animals have resulted in changes in respiratory tract tissues, as well as growth retardation, anemia, and kidney effects. While no information is available on acetaldehyde effects on human reproduction or development, both such effects have been observed in animal tests. Based on evidence of tumors in animals, EPA has classified acetaldehyde as a probable human carcinogen.

Benzene: Reported effects on humans, from short-term exposure to low to moderate benzene levels, include drowsiness, dizziness, headache, and unconsciousness as well as eye, skin and respiratory tract irritation. Effects on both humans and animals from long-term benzene exposure include blood and immune system disorders. Reproductive effects have been reported for women exposed to high benzene levels and adverse effects on the developing fetus have been observed in animal tests. Changes in human chromosome number and structure have been

reported under certain exposures. EPA has classified benzene as a known human carcinogen.

Formaldehyde: Reported effects on humans, from short-term and long-term exposure to formaldehyde, are mainly irritation of eyes, nose, throat, and, at higher levels, the respiratory tract. Long-term exposures of animals have also resulted in damage to respiratory tract tissues. Although little information is available on developmental effects to humans, animal tests do not indicate effects on fetal development. EPA has classified formaldehyde as a probable human carcinogen.

Toluene: Effects on the CNS of humans and animals have been reported, from short-term exposure to low to moderate levels of toluene, and include dysfunction, fatigue, sleepiness, headaches, and nausea. Short-term exposure effects also include cardiovascular symptoms in humans and depression of the immune system in animals. CNS effects are also observed in long-term exposures of humans and animals. Additional long-term exposure effects include irritation of eyes, throat and respiratory tract in humans and changes in respiratory tract tissue of animals. Repeated toluene exposure has been observed to adversely affect the developing fetus in humans and animals. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider toluene classifiable as to human carcinogenicity.

Xylenes: Reported effects on humans, from short-term exposure to high levels of xylenes, include

irritation of eyes, nose, and throat, difficulty breathing, impairment of the CNS and gastrointestinal effects. Similar effects have been reported in animals in addition to effects on the kidney. Human effects from long-term exposure to xylenes are to the CNS, respiratory and cardiovascular systems, blood, and kidney. Long-term animal exposures to high levels of xylenes have shown effects on the liver. Effects on the developing fetus have been observed in animal studies. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider xylenes classifiable as to human carcinogenicity.

Ethylbenzene: Effects reported, from short-term exposures of humans to high levels of ethyl benzene, include dizziness, depression of the CNS, eye, mucous membrane, nose and respiratory tract irritation, and difficulty breathing. In short-term exposures of laboratory animals, additional effects on the liver, kidney and pulmonary system have also been reported. Long-term exposures of animals have demonstrated effects on blood cells, the liver and kidneys. Effects on fetal development have also been observed in animal exposures. Due to a lack of information for humans and inadequate animal evidence, EPA does not consider ethyl benzene classifiable as to human carcinogenicity.

Styrene: Exposure to styrene vapors can cause irritation of eyes, nose, throat and respiratory tract in humans. Effects on the CNS of humans including dizziness, fatigue, sleepiness, headaches,

nausea, and effects on intellectual function and memory have also been reported from long-term exposure to styrene. Long-term exposures of animals have demonstrated effects on the CNS, liver and kidney as well as eye and nasal irritation. Although the available information for humans is inconclusive, animal tests do not indicate effects on reproduction or fetal development. When styrene is absorbed into the human body, some of it is metabolized into styrene oxide, a direct acting mutagen that causes tumor development in test animals. The carcinogenicity of styrene is currently under review by EPA.

Hexane: Reported effects on humans, from short-term exposure to high levels of hexane, include irritation of eyes, mucous membranes, throat and skin, as well as impairment of the CNS including dizziness, giddiness, headaches, and slight nausea. Long-term human exposure from inhalation is associated with a slowing of peripheral nerve signal conduction which causes numbness in the extremities and muscular weakness, as well as changes to the retina which causes blurred vision. Animal exposures to hexane have resulted in damage to nasal, respiratory tract, lung and peripheral nerve tissues, as well as effects on the CNS. No information is available on hexane effects on human reproduction or development. Limited laboratory animal data indicate a potential for testicular damage in adults, while several animal studies show no effect on fetal development. EPA has not

classified hexane as to human carcinogenicity.

2,2,4-Trimethylpentane: Little information is available on the effects of 2,2,4-trimethylpentane overexposure in humans. Laboratory animals exposed to high levels for short periods have developed irritation, fluid build-up and bleeding in the lungs, as well as depression of CNS function. Kidney and liver effects have been reported from long-term animal exposures. No information is available on the potential for reproductive or developmental effects or on the carcinogenic potential of 2,2,4-trimethylpentane.

7. Twenty-eight of the 37 VOCs, and four of the 13 carbonyls measured as a part of the UATMP are defined as HAPs in section 112(b)(1) of the CAA.
8. The following states are presently participating in the UATMP: Arkansas, Louisiana, New Jersey, Texas, and Vermont.
9. The IADN fulfills legislative mandates in Canada and the United States that address the monitoring of air toxics. An international Great Lakes deposition network is mandated by Annex 15 of the Great Lakes Water Quality Agreement between the United States and Canada. In the United States, the CAA requires a Great Lakes deposition network.
10. The target chemicals include PCBs, pesticides, PAHs and metals. The compounds included as "target chemicals" were selected based on the following criteria: presence on List 1 of Annex 1 of the Great Lakes Water Quality Agreement (substances believed to be toxic and

present in the Great Lakes); established or perceived water quality problem; presence on the International Joint Commission's Water Quality Board's list of criteria

pollutants; evidence of presence in the atmosphere and an important deposition pathway; and feasibility of measurement in a routine monitoring network.

11. Hoff, R.M., Strachan, W.M.J., Sweet, C.W., D.F. Gatz, Harlin, K., Shackleton, M., Cussion, S., Chan, C.H., Brice, K.A., Shroeder, W.H., Bidleman, T.F., Atmospheric Deposition of Toxic Chemicals to the Great Lakes: A Review of Data Through 1994, *Atmos. Environ.*, 1996, 30, 3505-3527. For additional references see A, B, C listed below.

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B. Hillery, B.R., Basu I., Sweet, C.W., Hites, R.A., Temporal and Spatial Trends in a Long-Term Study of Gas-Phase PCB Concentrations near the Great Lakes, *Environ. Sci. Technol.* 1997, 31, 1811-1816.

C. Hillery, B.R., Hoff, R.M., Hites, R. Atmospheric Contaminant Deposition to the Great Lakes Determined from the International Atmospheric Deposition Network, In *Atmospheric Deposition of Contaminants to the Great Lakes and Coastal Water*, Baker, J.E., ed.,

Society for Environmental Toxicology and Chemistry, 1997.

12. Interest in participation in this voluntary effort and/or requests for further information about this data cataloguing effort should be directed to Rhonda Thompson, Office of Air Quality Planning and Standards, Mail Drop 14, Research Triangle Park, North Carolina 27711; 919-541-5538; and thompson.rhonda@epa.gov.

13. The scheduled completion date for this project is April 1999; however, interim products will be released as completed. Additional information on this project is available through Vasu Kilaru, Office of Air Quality Planning and Standards, Mail Drop 14, Research Triangle Park, North Carolina 27711; 919-541-5332; and kilaru.vasu@epa.gov.

14. Second Report to Congress on the Status of the Hazardous Air Pollutant Program Under the CAA, Draft. EPA-453/R-96-015. October 1997.

15. The final inventory report is available at the following Internet address: www.epa.gov/ttn/uatw/112cfac.html.

16. The draft inventory report is available at the following Internet address: www.epa.gov/ttn/uatw/112kfac.html

17. *Study of Hazardous Air Pollutant Emissions from Electric Utility Steam Generating Units - Final Report to Congress: Volume 1*, U.S. EPA 1998. EPA 453/R-98-004a.

