All EPA Monitory Data/ Info

http://www.epa.gov/ttn/amtic/
Last updated on Wednesday, November 17, 2010

Technology Transfer Network
Ambient Monitoring Technology Information Center

The Ambient Monitoring Technology Information Center (AMTIC) contains information on ambient air quality monitoring programs, monitoring methods, quality assurance and control procedures, and federal regulations related to ambient air quality monitoring. This site is primarily intended for use by air monitoring staff responsible for collecting ambient air monitoring data.

Announcements

New! The 2011 Monitoring Schedule is now available.

SLAMS Networks
- Ozone and Photochemical Assessment Monitoring Stations (PAMS)
- Air Toxics
- NCore
- PM2.5 and Chemical Speciation
- Lead Monitoring
- Meteorological Measurements
- State and Local Monitoring Plans

Other Networks and Partners
- PM Supersites
- Environmental Technology Verification (ETV)

Air Monitoring Methods
- Criteria Pollutants
- National Air Toxics Trends Stations
- Inorganic Compendium
- Toxic Organic Compendium
- Open Path Monitoring
- Passive Monitoring

Quality Assurance
- Quality Assurance Guidance Documents
- National Performance Evaluation Program
- Quality Indicator Assessment Reports
- QA/QC Workgroup Activities (national meeting)
- Newsletters
- Pollutant/Network Specific QA

Regulations and Guidance
- National Air Monitoring Strategy
- Monitoring Regulations
- Policy Memoranda and Technical Guidance
- 40 CFR 50
- 40 CFR 53 and 58

Training and Calendars
- National Ambient Air Monitoring Conference
- Sampling Schedule Calendar
- NCORE Training
- Meteorological Measurement Training

Program Reviews and Oversight
- Clean Air Scientific Advisory Committee (CASAC)
- Committee on Environment and Natural Resources

Risk Assessment Tool

http://www.epa.gov/ttn/fera/human_hem.html
Last updated on Monday, July 02, 2007

Technology Transfer Network
FERA (Fate, Exposure, and Risk Analysis)

Risk Assessment and Modeling - Human Exposure Model (HEM)

- **General Information.** The Human Exposure Model (HEM) is used primarily for performing risk assessments for major point sources (usually producers or large users of specified chemicals) of air toxics. The HEM only addresses the inhalation pathway of exposure, and is designed to predict risks associated with emitted chemicals in the ambient air (i.e., in the vicinity of an emitting facility but beyond the facility's property boundary). The HEM provides ambient air concentrations, as surrogates for lifetime exposure, for use with unit risk estimates and inhalation reference concentrations to produce estimates of cancer risk and noncancer hazard, respectively, for the air toxics modeled.

  The HEM contains (1) an atmospheric dispersion model, the Industrial Source Complex Model , with included meteorological data: and (2) U.S. Bureau of Census population data at the Census block level. The model utilizes 2000 Census data (Note: the model and it's datasets have been populated for the 50 states, DC, PR, and the VI only). Each source in HEM must be specifically located by latitude and longitude, and its release parameters must be described. These include stack height, exit velocity, emission rate, etc. Based on the inputs for source parameters and the meteorological data, the model estimates the magnitude and distribution of ambient air concentrations in the vicinity of each source. The model is generally used to estimate these concentrations within a radial distance of 50 kilometers (30.8 miles) from the source. Exposure estimates generated by HEM are the ambient air concentrations predicted by the model, in micrograms per cubic meter. These exposure estimates are actually surrogates, as important exposure variables (e.g., duration, human activity patterns, residential occupancy period, etc.) are not explicitly addressed.

  The HEM is available in two versions: HEM-Screen and HEM-3.

  HEM-Screen can generate chronic cancer risk and hazard estimates for multiple facilities nationwide in one run. This model uses a simplified version of the Industrial Source Complex Model (Long-term), Version 2 (ISCLT2), dispersion model. Several simplifications and assumptions are built in to HEM-Screen, and user-supplied data requirements are relatively low. For these reasons, HEM-Screen may be more appropriate for lower-tier or screening-level assessments involving a large number of facilities.

  HEM-3 generates chronic cancer risk and chronic and acute hazard estimates for one facility at a time. This model uses either the Industrial Source Complex Model (Short-term), Version 3 (ISCST3), or the AERMOD dispersion model. Data requirements are somewhat higher for HEM-3 compared to HEM-Screen, however, the results are typically more refined because ISCST3 provides several additional dispersion modeling options.

- **Download Model**

- **User's Guide** Those planning to use HEM are encouraged to carefully review the User's Guides. They describe technical information about the models and the steps involved in running them.
  HEM-Screen User's Guide (PDF) (1.1 MB) HEM-3 User's Guide (PDF) (379 KB)

- **Peer Review and Publications.** The ISC model has undergone review and evaluation as part of the regulatory models process.
  Appendix A (labeled Appendix W in CFR) of Guideline on Air Quality Models provides a summary description of the ISC model. The SCRAM web site provides documentation of ISC, version 2, as well as the current version 3 of ISC.

- **Other Supporting Documents.**

COMPARISON OF 2002 MODEL-PREDICTED CONCENTRATIONS TO MONITORED DATA

As part of the 2002 National-Scale Air Toxics Assessment, EPA compared ASPEN-modeled concentrations with available, but geographically limited, ambient air quality monitoring data for the years 2002 through 2005. For each monitor-pollutant combination, EPA compared the annual average concentration estimated by the ASPEN model in the census tract where the ambient monitor is located to the annual average monitored value to get a point-to-point comparison between the model and monitor concentrations. EPA used an approach similar to that used for comparing the ASPEN model-to-monitor data for the 1996 and 1999 national-scale assessment except that EPA used updated emissions and monitor input data for the 2002 assessment; there were no major changes to model formulation. For more details about the model-to-monitor analysis for the 1996 national-scale assessment, see http://www.epa.gov/ttn/atw/nata/mtom_pre.html. For more details about the model-to-monitor analysis for the 1999 assessment, see http://www.epa.gov/ttn/atw/nata1999/99compare.html. Note that in this assessment, ambient chromium concentrations were compared to the sum of modeled chromium III and chromium IV concentrations. Chromium VI was measured at too few sites to provide a valid comparison for the model.

Table 1 shows the number of monitoring sites used in the 2002 comparison and the median ratio of model-to-monitor annual average concentrations by pollutant, on a point-to-point basis. The number of sites is the number of monitors with valid data. A large number of monitors means that more data are available which, in turn, facilitates an assessment of the degree of agreement between model and monitor data. The $PM_{2.5}$ metals (manganese, lead, arsenic, nickel, and selenium), benzene, toluene, and xylenes have the highest number of monitors. The number of available sites has increased substantially since the 1999 analysis. The median ratio is based on the model-to-monitor ratios for a given pollutant. A median close to 1 implies that the model overestimates the ambient concentrations about as often as it underestimates them. Methyl tert-butyl ether, acetaldehyde, and chloromethane all had median ratios between 0.9 and 1.1. The percent of sites estimated "within a factor of 2" is the percent of sites for which the model estimate is somewhere between half and double the monitor average. The "percent of sites estimated within 30%" is the percent of sites for which the model-to-monitor ratio is between 0.7 and 1.3. The "percent of sites underestimated" is the percent of sites for which the model-to-monitor ratio is below 1.

The degree of agreement between model-to-monitor data can be attributed to the following five uncertainties (which are the same identified in the 1996 and 1999 model-to-monitor comparison):

1. emission characterization uncertainties (e.g., specification of source location, emission rates, and release characterization);
2. meteorological characterization uncertainties (e.g., representativeness);
3. model formulation and methodology uncertainties (e.g., characterization of dispersion, plume rise, deposition,);
4. monitoring uncertainties; and

5. uncertainties in background concentrations.

ASPEN's limited ability to address the complex chemical transformation mechanisms needed to estimate ambient concentrations for highly reactive pollutants results in additional uncertainty for acetaldehyde and formaldehyde concentrations.

Figures 1 and 2 are box plots showing the distribution of the model-to-monitor ratios shown in Table 1. For example if there are 284 monitors measuring benzene, there are 284 model-to-monitor ratios to compute. EPA then computed the median of these 284 ratios as well as the percentiles to create the plot. The bottom of the box is the 25^{th} percentile, the top of the box is the 75^{th} percentile, and the horizontal line in the middle of the box is the median (i.e., 50^{th} percentile). If the model consistently agrees with the monitored data for the pollutant, the boxes will be narrow and centered at 1. Pollutants are organized alphabetically in two groups according to whether they are gases or embedded in particles. This side-by-side display of pollutants facilitates comparison to indicate which pollutants are being overestimated and underestimated, and which are estimated consistently. As in the 1996 comparison, the box plots do not show extreme percentiles (e.g., 10^{th} and 90^{th}) of the ratios because the extreme percentiles were far from the center of the distribution.

In this comparison, several assumptions about the monitoring data were made. Pollutants measured by fewer than 50 monitors and in limited geographical coverage (located in only one state) were excluded from the comparisons because the ability to assess model-to-monitor agreement is limited to that state or geographical area and does not extend nationwide. If annual average concentrations (e.g., >85% of the data were below the method detection limit) were not quantifiable using the monitor data, EPA also excluded the pollutant.

These results show that the interquartile range of model-to-monitor comparisons was within a factor of two for 1,3-butadiene, formaldehyde, acetaldehyde, chloromethane, carbon tetrachloride, benzene, toluene, xylenes, lead $PM_{2.5}$, and nickel $PM_{2.5}$. The remainder of the pollutants show various degrees of agreement. These results are an improvement over those found in the 1996 and 1999 national-scale assessment comparisons. However, the model is still underestimating several pollutants, most noticeably, acrylonitrile, chlorobenzene, isopropylbenzene, antimony, arsenic $PM_{2.5}$, manganese TSP, mercury $PM_{2.5}$, and selenium $PM_{2.5}$ all have 75^{th} percentile median ratios below 0.5. There are five possible reasons that ASPEN underestimates pollutant concentrations; these reasons also applied to the 1996 and 1999 assessments):

1. The National Emissions Inventory (NEI) may be missing specific emissions sources (for many of the sources in the NEI some of the emissions parameters are missing).

2. The emission rates may be underestimated. EPA believes the ASPEN model itself contributed only in a minor way to the underestimation. The modeled results from the ASPEN predecessor compared favorably to monitoring data in cases where the emissions and meteorology were accurately characterized and the monitors made more frequent readings.

3. There is uncertainty in the accuracy of the monitor averages, which, in turn, have their own sources of uncertainty. Sampling and analytical uncertainty, measurement bias, and temporal variation can all cause the ambient concentrations to be inaccurate or imprecise representations of the true atmospheric averages.

4. Model-to-monitor spatial comparisons are imprecise. The results suggest that the model estimates are uncertain on a local scale (i.e., at the census tract level). EPA believes that the model estimates are more reliably interpreted as being a value likely to be found within 30 km of the census tract location.

5. Background concentrations are poorly characterized. Most of the pollutants for which the model underestimated ambient concentrations were those for which background concentrations were not estimated. If background concentrations are a large fraction of ambient concentrations, the result would be large underestimations in model predictions.

Table 1. Agreement of 2002 model-predicted concentrations and ambient monitored concentrations on a point-by-point basis. Pollutants listed were monitored in at least 50 locations in several states.

Parameter	Number of Sites	Median of Model: Monitor	Percent Within Factor of 2	Percent Within 30%	Percent Underestimated
Manganese $PM_{2.5}$	343	0.73	64%	30%	67%
Lead $PM_{2.5}$	339	0.67	70%	32%	71%
Benzene	284	1.47	69%	29%	23%
Toluene	270	1.53	66%	28%	22%
Arsenic $PM_{2.5}$	260	0.09	12%	4%	92%
Xylenes	256	1.21	65%	32%	39%
Chloromethane	251	1.02	97%	81%	45%
Chromium $PM_{2.5}$	230	0.51	63%	32%	67%
Nickel $PM_{2.5}$	228	0.75	48%	23%	61%
Selenium $PM_{2.5}$	226	0.02	0%	0%	100%
Carbon Tetrachloride	224	1.17	97%	74%	17%
Styrene	217	0.46	35%	16%	76%
1,3-Butadiene	191	0.78	69%	29%	63%
Dichloromethane	187	0.75	65%	43%	79%
Formaldehyde	165	0.65	75%	32%	84%
Acetaldehyde	164	0.97	84%	52%	52%
N-Hexane	163	0.60	47%	23%	70%
Lead TSP	147	0.32	27%	12%	90%
Mercury $PM_{2.5}$	142	0.01	0%	0%	100%
Tetrachloroethylene	125	0.63	59%	26%	77%
Propionaldehyde	122	0.81	61%	29%	59%
2,2,4-Trimethylpentane	122	1.48	58%	25%	34%
1,4-Dichlorobenzene	120	0.41	29%	7%	74%
Chlorobenzene	115	0.05	12%	7%	81%
Methyl Tert-Butyl Ether	109	0.94	49%	20%	54%
Methyl Chloroform	102	1.99	46%	10%	14%
Isopropylbenzene	94	0.03	4%	4%	98%
Chloroform	86	0.82	63%	31%	60%
Chromium TSP	85	0.20	25%	12%	88%
Manganese TSP	80	0.14	8%	3%	94%
Trichloroethylene	76	0.48	37%	20%	71%
Methyl Isobutyl Ketone	75	2.57	28%	13%	25%
Antimony $PM_{2.5}$	63	0.07	0%	0%	100%
Nickel TSP	56	0.37	41%	18%	84%
Acrylonitrile	50	0.03	0%	0%	100%

Figure 1 - Model-to-Monitor Ratios for Gaseous HAPs

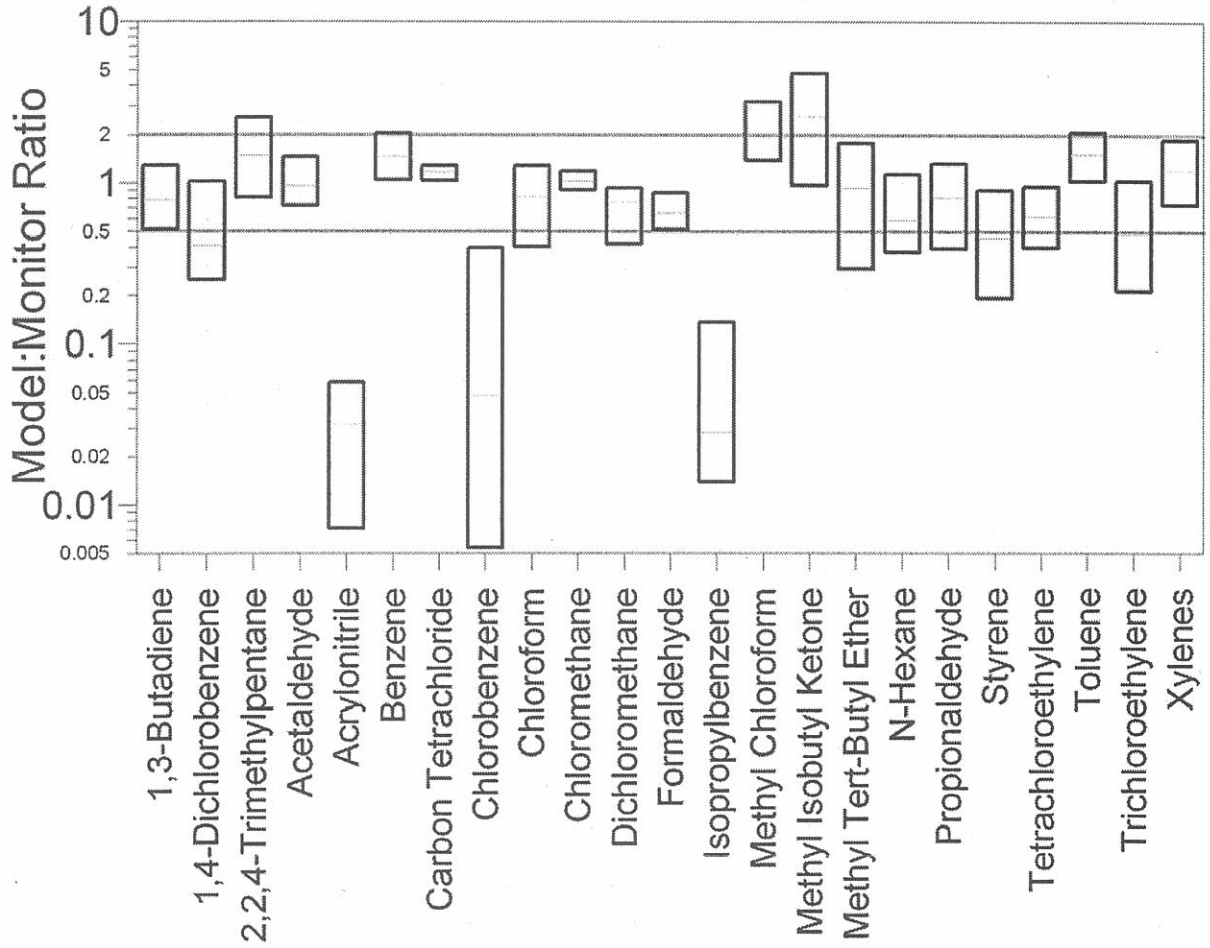
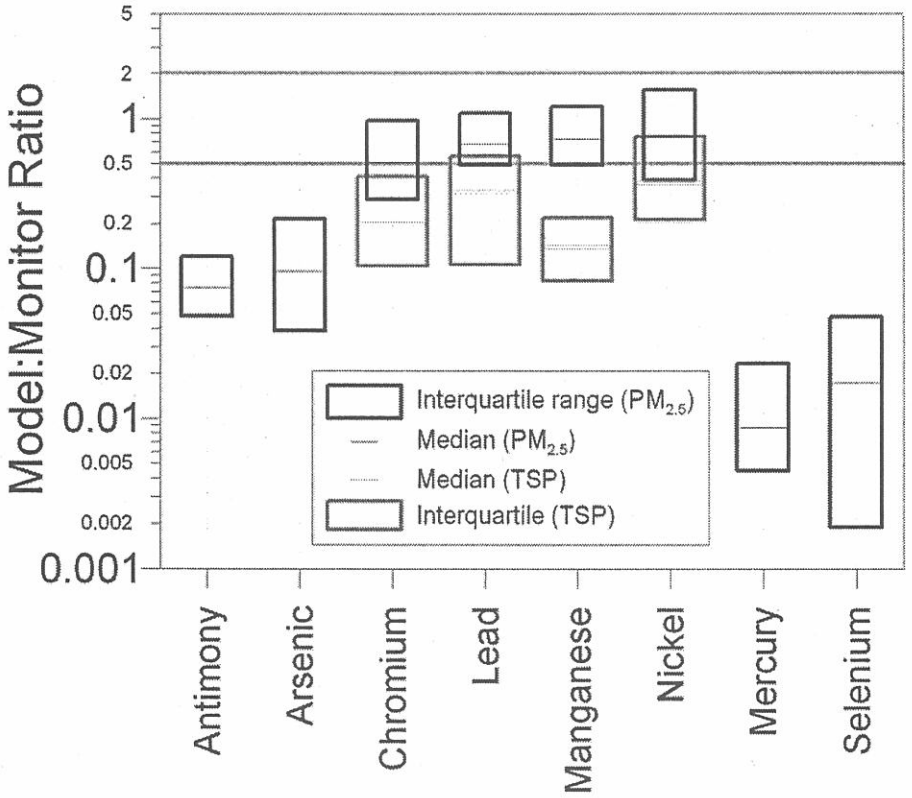


Figure 2 - Model-to-Monitor Ratios for Particulate HAPs



NATA-

http://www.epa.gov/ttn/atw/natamain/index.html
Last updated on Tuesday, November 02, 2010

Technology Transfer Network
Air Toxics Web Site

National Air Toxics Assessments

What is NATA?

The National-Scale Air Toxics Assessment (NATA) is EPA's ongoing comprehensive evaluation of air toxics in the U.S. EPA developed the NATA as a state-of-the-science screening tool for State/Local/Tribal Agencies to prioritize pollutants, emission sources and locations of interest for further study in order to gain a better understanding of risks. NATA assessments do not incorporate refined information about emission sources, but rather, use general information about sources to develop estimates of risks which are more likely to overestimate impacts than underestimate them. NATA provides estimates of the risk of cancer and other serious health effects from breathing (inhaling) air toxics in order to inform both national and more localized efforts to identify and prioritize air toxics, emission source types and locations which are of greatest potential concern in terms of contributing to population risk. This in turn helps air pollution experts focus limited analytical resources on areas and or populations where the potential for health risks are highest. Assessments include estimates of cancer and non-cancer health effects based on chronic exposure from outdoor sources, including assessments of non-cancer health effects for Diesel Particulate Matter (PM). Assessments provide a snapshot of the outdoor air quality and the risks to human health that would result if air toxic emissions levels remained unchanged.

How do I access NATA assessments?

EPA has completed three assessments that characterize the nationwide chronic cancer risk estimates and noncancer hazards from inhaling air toxics. The latest, the 2002 NATA, was made available to the public in June of 2009. You can access any of the NATA assessments by clicking below on the specific year of interest.

- 2002 National-Scale Air Toxics Assessment
- 1999 National-Scale Air Toxics Assessment
- 1996 National-Scale Air Toxics Assessment

Why was NATA developed?

The NATA assessments were designed to help guide efforts to cut toxic air pollution and build upon the already significant emissions reductions achieved in the US since 1990.

NATA was developed as a tool to inform both national and more localized efforts to collect air toxics information, characterize emissions, and help prioritize pollutants/geographic areas of interest for more refined data collection and analyses.

The goal is to identify those air toxics which are of greatest potential concern in terms of contribution to population risk. Ambient and exposure concentrations, and estimates of risk and hazard for air toxics in each State are typically generated at the census tract level.

What NATA is not.

NATA results provide answers to questions about emissions, ambient air concentrations, exposures and risks across broad geographic areas (such as counties, states and the Nation) at a moment in time. As such, they help the EPA identify specific air toxics compounds, and specific source sectors such as stationary sources or mobile sources, which generally produce the highest exposures and risks in the country. These assessments are based on assumptions and methods that limit the range of questions that can be answered reliably. The results cannot be used to identify exposures and risks for specific individuals, or even to identify exposures and risks in small geographic regions such as a specific census block, i.e., hotspots.

These assessments use emissions data for a single year as inputs to models which will yield concentration and risk estimates. These estimates reflect chronic exposures resulting from the inhalation of the air toxics emitted and do not consider exposures which may occur indoors or as a results of exposures other than inhalation, i.e., dermal or ingestion.

These limitations, or caveats, must always be kept in mind when interpreting the results, and the results should be used only to address questions for which the assessment methods are suited.

How should I use NATA results?

The results of assessments are best used to focus on geographic patterns and ranges of risks across the country. You can use NATA to do all of the following:

- Prioritize pollutants and emission sources
- Identify locations of interest for further investigation
- Provide a starting point for local-scale assessments
- Focus community efforts
- Inform monitoring programs
- To prioritize schools for monitoring outdoor air toxics

For example, assessments made at the community level, have relied on assessments to prioritize data and research needs to better

assess the local risk from air toxics. Communities have found that accessing NATA data helps inform and empower citizens to make local decisions concerning the health of their communities. In some cases, local projects can achieve environmental improvements sooner than federal regulations alone.

EPA uses the results of assessments to do all of the following:

- Set priorities for improving data in emission inventories
- Direct priorities in expanding EPA's air toxics monitoring network
- More effectively target risk reduction activities
- Identify pollutants and industrial source categories of greatest concern
- Help set priorities for the collection of additional information
- Improve understanding of the risk from air toxics
- Work with communities in designing their own assessment
- Link Air Toxics to Criteria Pollutant Program

NATA assessments should not be used for any of the following:

- As a sole means for identifying localized hotspots*
- As a definitive means to pinpoint specific risk values within a census tract
- To characterize or compare risks at local levels such as between neighborhoods
- As the sole basis for developing risk reduction plans or regulations
- To control specific sources or pollutants
- To quantify benefits of reduced air toxic emissions

*For analysis of air toxics in these smaller areas, other tools such as monitoring and local-scale assessments should be used to evaluate potential hot spots using more refined and localized data.

Can I compare data across assessments?

For each assessment, EPA has improved its methodology by doing all of the following:

- Use a better and more complete inventory of emission sources
- Increase the number of air toxics evaluated
- Improve upon health data information used in assessments

Due to the extent of improvements in methodology, it is not meaningful to compare the assessments. This is because any change in emissions, ambient concentrations, or risks maybe due to either improvement in methodology or to real changes in emissions or source characterization.

How are NATA assessments developed?

NATA assessments generally include a four step process including:

1. Compile a national emissions inventory from outdoor sources.
2. Estimate ambient concentrations of air toxics across the United States.
3. Estimate population exposures across the United States.
4. Characterize potential public health risks due to inhalation of air toxics.

Is NATA a collaborative process?

EPA collaborated with State, local and Tribal agencies to develop the information that is contained in the assessment. Communities have been actively involved in partnerships with local governments to use NATA data to develop local toxics inventories and to provide the basis for developing a community-supported plan for reducing toxic emissions. The National Research Council (NRC) in their review of the 1996 NATA ,emphasized in their 2004 report on "Air Quality Management in the United States" Exit Disclaimer that "NATA has provided a tool for exploring control priorities and has served as a preliminary attempt to establish a baseline for tracking progress in reducing HAP emissions".(See p.247 of that report).

Aside from interactions with other environmental agencies, EPA has sought to collaborate with EPA's Science Advisory Board which provided helpful comments through their peer review process. The methods used for these assessments were peer-reviewed and endorsed by EPA's Science Advisory Board in 2001. (See http://www.epa.gov/ttn/atw/sab/sabrev.html). The SAB review concluded that NATA represents "an important step toward characterizing the relationship between sources and risk of hazardous air pollutants".

Table 1. Prioritized Chronic Dose-Response Values (4/27/2010). Revisions since 06/12/07 are shown in red. **CAS NO.** = Chemical Abstracts Services number for the compound. **HAP NO.** = Position of the compound on the HAP list in the Clean Air Act (112[b][2]). "999" denotes substances under consideration for listing.	**Sources:** IRIS = Integrated Risk Information System; ATSDR = US Agency for Toxic Substances and Disease Registry; D-ATSDR = draft ATSDR; CA = California EPA; P-CAL = Proposed CAL; HEAST = EPA Health Effects Assessment Tables; Conv. Oral = Oral unit risk converted to inhalation.	**IARC WOE** = weight-of-evidence for carcinogenicity in humans (1 - carcinogenic; 2A - probably carcinogenic; 2B - possibly carcinogenic; 3 - not classifiable; 4 - probably not carcinogenic).	**EPA WOE** (1986 guidelines) = weight-of-evidence for carcinogenicity under the 1986 EPA cancer guidelines: A - human carcinogen; B1 - probable carcinogen, limited human evidence; B2 - probable carcinogen, sufficient evidence in animals; C - possible human carcinogen; D - not classifiable E - evidence of noncarcinogenicity.	**EPA WOE** (1999 guidelines) = weight-of-evidence for carcinogenicity under the 1999 EPA cancer guidelines: CH - carcinogenic to humans; LH - likely to be carcinogenic; SE - suggestive evidence for carcinogenicity; Inl - inadequate information to determine carcinogenicity; NH - not likely to be carcinogenic).

Table 1. Prioritized Chronic Dose-Response Values for Screening Risk Assessments (4/27/2010)

				CHRONIC INHALATION					CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Acetaldehyde	75-07-0	1	2B	0.009	IRIS	B2	0.0000022	IRIS					
Acetamide	60-35-5	2	2B				0.00002	CAL					
Acetonitrile	75-05-8	3		0.06	IRIS	Inl							
Acetophenone	98-86-2	4				D							
Acrolein	107-02-8	6	3	0.00002	IRIS	Inl							
Acrylamide	79-06-1	7	2A	***0.006***	***IRIS***	***LH***	***0.0001***	IRIS					
Acrylic acid	79-10-7	8		0.001	IRIS								
Acrylonitrile	107-13-1	9	2A	0.002	IRIS	B1	0.000068	IRIS					
Allyl chloride	107-05-1	10	3	0.001	IRIS	C	0.000006	CAL					
Aniline	62-53-3	12	3	0.001	IRIS	B2	0.0000016	CAL					
Antimony compounds	7440-36-0	173											
Antimony pentoxide	1314-60-9	173											
Antimony potassium tartrate	304-61-0	173											
Antimony tetroxide	1332-81-6	173											
Antimony trioxide	1309-64-4	173	2B	0.0002	IRIS								
Arsenic compounds	7440-38-2	174	1	***0.000015***	CAL	A	0.0043	IRIS					
Arsenic pentoxide	1303-28-2	174											
Arsine	7784-42-1	174		0.00005	IRIS								
Benzene	71-43-2	15	1	0.03	IRIS	CH	0.0000078	IRIS					
Benzidine	92-87-5	16		0.01	P-CAL	A	0.067	IRIS					
Benzotrichloride	98-07-7	17	2B			B2	0.0037	Conv. Oral					
Benzyl chloride	100-44-7	18	2B			B2	0.000049	CAL					
Beryllium compounds	7440-41-7	175	1	0.00002	IRIS	LH	0.0024	IRIS					
Beryllium oxide	1304-56-9	175		0.000007	CAL								
Biphenyl	92-52-4	19				D							
Bis(2-ethylhexyl)phthalate	117-81-7	20	2B	0.01	P-CAL	B2	0.0000024	CAL					
Bis(chloromethyl)ether	542-88-1	21	1			A	0.062	IRIS					
Bromoform	75-25-2	22	3			B2	0.0000011	IRIS					
1,3-Butadiene	106-99-0	23	2A	0.002	IRIS	CH	0.00003	IRIS					
Cadmium compounds	7440-43-9	176	1	***0.00001***	***D-ATSDR***	B1	0.0018	IRIS	0.0005	IRIS	B1		

Table 1. Prioritized Chronic Dose-Response Values (4/27/2010). Revisions since 06/12/07 are shown in red. **CAS NO.** = Chemical Abstracts Services number for the compound. **HAP NO.** = Position of the compound on the HAP list in the Clean Air Act (112[b][2]). "999" denotes substances under consideration for listing.

Sources: IRIS = Integrated Risk Information System; ATSDR = US Agency for Toxic Substances and Disease Registry; D-ATSDR = draft ATSDR; CA = California EPA; P-CAL = Proposed CAL; HEAST = EPA Health Effects Assessment Tables; Conv. Oral = Oral unit risk converted to inhalation.

IARC WOE = weight-of-evidence for carcinogenicity in humans (1 - carcinogenic; 2A - probably carcinogenic; 2B - possibly carcinogenic; 3 - not classifiable; 4 - probably not carcinogenic).

EPA WOE (1986 guidelines) = weight-of-evidence for carcinogenicity under the 1986 EPA cancer guidelines: A - human carcinogen; B1 - probable carcinogen, limited human evidence; B2 - probable carcinogen, sufficient evidence in animals; C - possible human carcinogen; D - not classifiable E - evidence of noncarcinogenicity.

EPA WOE (1999 guidelines) = weight-of-evidence for carcinogenicity under the 1999 EPA cancer guidelines: CH - carcinogenic to humans; LH - likely to be carcinogenic; SE - suggestive evidence for carcinogenicity; Inl - inadequate information to determine carcinogenicity; NH - not likely to be carcinogenic).

Table 1. Prioritized Chronic Dose-Response Values for Screening Risk Assessments (4/27/2010)

				CHRONIC INHALATION					CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Captan	133-06-2	26	3			B2	0.000001	Conv. Oral					
Carbaryl	63-25-2	27											
Carbon disulfide	75-15-0	28		0.7	IRIS								
Carbon tetrachloride	56-23-5	29	2B	*0.1*	*IRIS*	*LH*	*0.000006*	IRIS					
Chloramben	133-90-4	32											
Chlordane	57-74-9	33	2B	0.0007	IRIS	LH	0.0001	IRIS	0.0005	IRIS	LH	0.35	IRIS
Chlorine	7782-50-5	34		*0.00015*	*D-ATSDR*								
Chloroacetic acid	79-11-8	35											
2-Chloroacetophenone	532-27-4	36		0.00003	IRIS								
Chlorobenzene	108-90-7	37		1	CAL	D							
Chlorobenzilate	510-15-6	38				B2	0.000078	HEAST					
Chloroform	67-66-3	39	2B	0.098	ATSDR	LH							
Chloroprene	126-99-8	41		0.007	HEAST								
Chromium (III) compounds	16065-83-1	177				Inl							
Chromium (VI) compounds	18540-29-9	177	1	0.0001	IRIS	CH	0.012	IRIS					
Chromium (VI) trioxide, chromic acid mist	11115-74-5	177	1	0.000008	IRIS								
Cobalt compounds	7440-48-4	178		0.0001	ATSDR								
Coke Oven Emissions	8007-45-2	179				A	0.00062	IRIS					
m-Cresol	108-39-4	44				C							
o-Cresol	95-48-7	43				C							
p-Cresol	106-44-5	45				C							
Cresols (mixed)	1319-77-3	42		0.6	CAL	C							
Cumene	98-82-8	46		0.4	IRIS	Inl							
Cyanazine	21725-46-2	180				C	0.00024	Conv. Oral					
Cyanide compounds	57-12-5	180				D							
Acetone cyanohydrin	75-86-5	180		0.01	HEAST								
Calcium cyanide	592-01-8	180											
Copper cyanide	544-92-3	180											
Cyanogen	460-19-5	180											
Cyanogen bromide	506-68-3	180											

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IARC WOE = weight-of-evidence for carcinogenicity in humans (1 - carcinogenic; 2A - probably carcinogenic; 2B - possibly carcinogenic; 3 - not classifiable; 4 - probably not carcinogenic).

EPA WOE (1986 guidelines) = weight-of-evidence for carcinogenicity under the 1986 EPA cancer guidelines: A - human carcinogen; B1 - probable carcinogen, limited human evidence; B2 - probable carcinogen, sufficient evidence in animals; C - possible human carcinogen; D - not classifiable E - evidence of noncarcinogenicity.

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Table 1. Prioritized Chronic Dose-Response Values for Screening Risk Assessments (4/27/2010)

				CHRONIC INHALATION					CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Cyanogen chloride	506-77-4	180											
Ethylene cyanohydrin	109-78-4	180											
Hydrogen cyanide	74-90-8	180		0.003	IRIS								
Potassium cyanide	151-50-8	180											
Potassium silver cyanide	506-61-6	180											
Silver cyanide	506-64-9	180											
Sodium cyanide	143-33-9	180											
Thiocyanic acid, 2-(benzothiazolylthio) methyl est	21564-17-0	180											
Zinc cyanide	557-21-1	180											
2,4-D, salts and esters	94-75-7	47											
DDE	72-55-9	48				B2	0.000097	Conv. Oral			B2	0.34	IRIS
1,2-Dibromo-3-chloropropane	96-12-8	51		0.0002	IRIS	B2	0.002	CAL					
Dibutylphthalate	84-74-2	52				D							
p-Dichlorobenzene	106-46-7	53	2B	0.8	IRIS	C	0.000011	CAL					
3,3'-Dichlorobenzidine	91-94-1	54	2B			B2	0.00034	CAL					
Dichloroethyl ether	111-44-4	55				B2	0.00033	IRIS					
1,3-Dichloropropene	542-75-6	56	2B	0.02	IRIS	LH	0.000004	IRIS					
Dichlorvos	62-73-7	57	2B	0.0005	IRIS	B2	0.000083	Conv. Oral					
Diesel engine emissions	DIESEL EMIS.	999		0.005	IRIS	LH							
Diethanolamine	111-42-2	58		0.003	CAL								
3,3'-Dimethoxybenzidine	119-90-4	61	2B			B2	0.000004	Conv. Oral					
p-Dimethylaminoazobenzene	60-11-7	62	2B				0.0013	CAL					
3,3'-Dimethylbenzidine	119-93-7	63				B2	0.0026	Conv. Oral					
Dimethyl formamide	68-12-2	65	2B	0.03	IRIS								
N,N-dimethylaniline	121-69-7	59	3										
1,1-Dimethylhydrazine	57-14-7	66	2B			B2							
2,4-dinitrophenol	51-28-5	70											
2,4-Dinitrotoluene	121-14-2	71	2B	0.007	P-CAL	B2	0.000089	CAL					
2,4/2,6-Dinitrotoluene (mixture)	25321-14-6	71	2B			B2	0.00019	Conv. Oral					
1,4-Dioxane	123-91-1	72	2B	3.6	D-ATSDR	B2	0.0000077	CAL					

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Table 1. Prioritized Chronic Dose-Response Values for Screening Risk Assessments (4/27/2010)

				CHRONIC INHALATION						CHRONIC ORAL				
				NONCANCER		CANCER				NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE		mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
1,2-Diphenylhydrazine	122-66-7	73				B2	0.00022	IRIS						
Epichlorohydrin	106-89-8	74	2A	0.001	IRIS	B2	0.0000012	IRIS						
1,2-Epoxybutane	106-88-7	75		0.02	IRIS									
Ethyl acrylate	140-88-5	76	2B			B2								
Ethyl benzene	100-41-4	77		1	IRIS	D	*0.0000025*	CAL						
Ethyl carbamate	51-79-6	78	2B				0.00029	CAL						
Ethyl chloride	75-00-3	79		10	IRIS									
Ethylene dibromide	106-93-4	80	2A	0.009	IRIS	LH	0.0006	IRIS						
Ethylene dichloride	107-06-2	81	2B	2.4	ATSDR	B2	0.000026	IRIS						
Ethylene glycol	107-21-1	82		0.4	CAL									
Ethylene oxide	75-21-8	84	1	0.03	CAL	B1	0.000088	CAL						
Ethylene thiourea	96-45-7	85	2B	0.003	P-CAL	B2	0.000013	CAL						
Ethylidene dichloride (1,1-Dichloroethane)	75-34-3	86		0.5	HEAST	C	0.0000016	CAL						
Formaldehyde	50-00-0	87	2A	0.0098	ATSDR	B1	*0.000013*	*IRIS*						
Diethylene glycol monobutyl ether	112-34-5	181		0.02	HEAST									
Diethylene glycol monoethyl ether	111-90-0	181												
Ethylene glycol ethyl ether	110-80-5	181		0.2	IRIS									
Ethylene glycol ethyl ether acetate	111-15-9	181		0.3	CAL									
Ethylene glycol methyl ether	109-86-4	181		0.02	IRIS									
Ethylene glycol methyl ether acetate	110-49-6	181		0.09	CAL									
Heptachlor	76-44-8	88	2B			B2	0.0013	IRIS		0.0005	IRIS	B2	4.5	IRIS
Hexachlorobenzene	118-74-1	89	2B	0.003	P-CAL	B2	0.00046	IRIS		0.0008	IRIS	B2	1.6	IRIS
Hexachlorobutadiene	87-68-3	90	3	0.09	P-CAL	C	0.000022	IRIS						
Hexachlorocyclopentadiene	77-47-4	91		0.0002	IRIS	NH								
Hexachlorodibenzo-p-dioxin, mixture	19408-74-3	187				B2	1.3	IRIS				B2	6200	IRIS
Hexachloroethane	67-72-1	92	3	0.08	P-CAL	C	0.000004	IRIS						
Hexamethylene-1,6-diisocyanate	822-06-0	93		0.00001	IRIS									
n-Hexane	110-54-3	95		0.7	IRIS	Inl								
Hydrazine	302-01-2	96	2B	0.0002	CAL	B2	0.0049	IRIS						
Hydrochloric acid	7647-01-0	97	3	0.02	IRIS									

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Table 1. Prioritized Chronic Dose-Response Values for Screening Risk Assessments (4/27/2010)

				CHRONIC INHALATION					CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Hydrofluoric acid	7664-39-3	98		0.014	CAL								
Hydrogen sulfide	7783-06-4	999		0.002	IRIS	Inl							
Hydroquinone	123-31-9	99											
Isophorone	78-59-1	100		2	CAL	C	2.7E-07	Conv. Oral					
Lead compounds	7439-92-1	182	2B	*0.00015*	EPA OAQPS	B2					B2		
Tetraethyl lead	78-00-2	182							0.0000001	IRIS			
Lindane (gamma-HCH)	58-89-9	101	2B	0.0003	P-CAL	B2-C	0.00031	CAL	0.0003	IRIS	B2-C	1.1	CAL
alpha-Hexachlorocyclohexane (a-HCH)	319-84-6	101	2B	0.02	P-CAL	B2	0.0018	IRIS	0.008	D-ATSDR	B2	6.3	IRIS
beta-Hexachlorocyclohexane (b-HCH)	319-85-7	101	2B	0.002	P-CAL	C	0.00053	IRIS			C	1.8	IRIS
technical Hexachlorocyclohexane (HCH)	608-73-1	101	2B			B2	0.00051	IRIS			B2	1.8	IRIS
Maleic anhydride	108-31-6	102		0.0007	CAL								
Manganese compounds	7439-96-5	183		0.00005	IRIS	D							
Mercuric chloride	7487-94-7	184				C			0.0003	IRIS	C		
Mercury (elemental)	7439-97-6	184		0.0003	IRIS	D					D		
Methyl mercury	22967-92-6	184				C			0.0001	IRIS	C		
Phenylmercuric acetate	62-38-4	184							0.00008	IRIS			
Methanol	67-56-1	103		4	CAL								
Methoxychlor	72-43-5	104	3			D			0.005	IRIS	D		
Methyl bromide	74-83-9	105		0.005	IRIS	D							
Methyl chloride	74-87-3	106		0.09	IRIS	Inl							
Methyl chloroform (1,1,1-Trichloroethane)	71-55-6	107		*5*	*IRIS*	*Inl*							
Methyl isobutyl ketone	108-10-1	111		3	IRIS	Inl							
Methyl isocyanate	624-83-9	112		0.001	CAL								
Methyl methacrylate	80-62-6	113		0.7	IRIS	E							
Methyl tert-butyl ether	1634-04-4	114		3	IRIS		2.6E-07	CAL					
4,4'-Methylene bis(2-chloroaniline)	101-14-4	115	2A			B2	0.00043	CAL					
Methylene chloride	75-09-2	116	2B	1	ATSDR	B2	4.7E-07	IRIS					
Methylene diphenyl diisocyanate	101-68-8	117		0.0006	IRIS	Inl							
4,4'-Methylenedianiline	101-77-9	118	2B	0.02	CAL		0.00046	CAL					
Naphthalene	91-20-3	119		0.003	IRIS	C	0.000034	CAL					

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				CHRONIC INHALATION						CHRONIC ORAL				
				NONCANCER		CANCER				NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE		mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Nickel compounds	7440-02-0	186	2B	0.00009	*ATSDR*	A								
Nickel oxide	1313-99-1	186		0.0001	CAL									
Nickel refinery dust	NI_DUST	186				A	0.00024	IRIS						
Nickel subsulfide	12035-72-2	186		0.00005	CAL	A	0.00048	IRIS						
Nitrobenzene	98-95-3	120	2B	*0.009*	*IRIS*	*LH*	*0.00004*	IRIS						
2-Nitropropane	79-46-9	123	2B	0.02	IRIS	B2	0.0000056	OAQPS						
Nitrosodimethylamine	62-75-9	125	2A			B2	0.014	IRIS						
N-Nitrosomorpholine	59-89-2	126	2B				0.0019	CAL						
Parathion	56-38-2	127	3			C								
Polychlorinated biphenyls	1336-36-3	136	2A			B2	0.0001	IRIS				B2	2	IRIS
Aroclor 1016	12674-11-2	136								0.00007	IRIS			
Aroclor 1254	11097-69-1	136								0.00002	IRIS			
Pentachloronitrobenzene	82-68-8	128	3			C	0.000074	Conv. Oral						
Pentachlorophenol	87-86-5	129	2B	0.1	P-CAL	B2	0.0000051	CAL						
Phenol	108-95-2	130	3	0.2	CAL	Inl								
p-Phenylenediamine	106-50-3	131												
Phosgene	75-44-5	132		0.0003	IRIS	Inl								
Phosphine	7803-51-2	133		0.0003	IRIS	*Inl*								
Phosphorus, white	7723-14-0	134		0.00007	P-CAL	D								
Phthalic anhydride	85-44-9	135		0.02	CAL									
Polybrominated biphenyls	59536-65-1	187				B2	0.0025	Conv. Oral		0.000007	HEAST	B2	8.9	HEAST
Acenaphthene	83-32-9	187				D				0.06	IRIS	D		
Acenaphthylene	206-96-8	187				D						D		
2-Aminoanthraquinone	117-79-3	187					0.0000094	CAL					0.033	CAL
Anthracene	120-12-7	187	3			D				0.3	IRIS	D		
Benzo(a)anthracene	56-55-3	187	2A			B2	0.00011	CAL				B2	1.2	CAL
Benzo(b)fluoranthene	205-99-2	187	2B			B2	0.00011	CAL				B2	1.2	CAL
Benzo[j]fluoranthene	205-82-3	187	2B				0.00011	CAL					1.2	CAL
Benzo(k)fluoranthene	207-08-9	187	2B			B2	0.00011	CAL				B2	1.2	CAL
Benzo(g,h,i)perylene	191-24-2	187	3			D						D		

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				CHRONIC INHALATION					CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Benzo(a)pyrene	50-32-8	187	2A			B2	0.0011	CAL			B2	7.3	IRIS
Benzo(e)pyrene	192-97-2	187	3										
Carbazole	86-74-8	187	3			B2	0.0000057	Conv. Oral			B2	0.02	HEAST
beta-Chloronaphthalene	91-58-7	187							0.08	IRIS			
Chrysene	218-01-9	187	3			B2	0.000011	CAL			B2	0.12	CAL
Dibenz[a,h]acridine	226-36-8	187	2B				0.00011	CAL				1.2	CAL
Dibenz[a,j]acridine	224-42-0	187	2B				0.00011	CAL				1.2	CAL
Dibenz(a,h)anthracene	53-70-3	187	2A			B2	0.0012	CAL			B2	4.1	CAL
7H-Dibenzo[c,g]carbazole	194-59-2	187	2B				0.0011	CAL				12	CAL
Dibenzo[a,e]pyrene	192-65-4	187	2B				0.0011	CAL				12	CAL
Dibenzo[a,h]pyrene	189-64-0	187	2B				0.011	CAL				120	CAL
Dibenzo[a,i]pyrene	189-55-9	187	2B				0.011	CAL				120	CAL
Dibenzo[a,l]pyrene	191-30-0	187	2B				0.011	CAL				120	CAL
7,12-Dimethylbenz(a)anthracene	57-97-6	187					0.071	CAL				250	CAL
1,6-Dinitropyrene	42397-64-8	187	2B				0.011	CAL				120	CAL
1,8-Dinitropyrene	42397-65-9	187	2B				0.0011	CAL				12	CAL
Fluoranthene	206-44-0	187	3			D			0.04	IRIS	D		
Fluorene	86-73-7	187	3			D			0.04	IRIS	D		
Indeno(1,2,3-cd)pyrene	193-39-5	187	2B			B2	0.00011	CAL			B2	1.2	CAL
3-Methylcholanthrene	56-49-5	187					0.0063	CAL				22	CAL
5-Methylchrysene	3697-24-3	187	2B				0.0011	CAL				12	CAL
1-Methylnaphthalene	90-12-0	187							0.07	*ATSDR*			
2-Methylnaphthalene	91-57-6	187				Inl			*0.04*	*ATSDR*	Inl		
5-Nitroacenaphthene	602-87-9	187	2B				0.000037	CAL				0.13	CAL
6-Nitrochrysene	7496-02-8	187	2B				0.011	CAL				120	CAL
2-Nitrofluorene	607-57-8	187	2B				0.000011	CAL				0.12	CAL
1-Nitropyrene	5522-43-0	187	2B				0.00011	CAL				1.2	CAL
4-Nitropyrene	57835-92-4	187	2B				0.00011	CAL				1.2	CAL
Octabromodiphenyl ether	32536-52-0	187				D			0.003	IRIS	D		
Phenanthrene	85-01-8	187				D					D		

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				CHRONIC INHALATION						CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Pyrene	129-00-0	187				D			0.03	IRIS	D		
1,3-Propane sultone	1120-71-4	137	2B				0.00069	CAL					
Propionaldehyde	*123-38-6*	*139*		*0.008*	*IRIS*	*Inl*							
Propoxur	114-26-1	140				B2							
Propylene dichloride	78-87-5	141		0.004	IRIS	B2	0.000019	Conv. Oral					
Propylene oxide	75-56-9	142	2B	0.03	IRIS	B2	0.0000037	IRIS					
Quinoline	91-22-5	144				LH							
Selenium compounds	7782-49-2	189		0.02	CAL	D							
Hydrogen selenide	7783-07-5	189		0.00008	P-CAL								
Selenious acid	7783-00-8	189				D							
Selenium dioxide	7446-08-4	189		0.02	CAL								
Selenium disulfide	7488-56-4	189		0.02	CAL								
Selenium sulfide	7446-34-6	189		0.02	CAL	B2							
Selenourea	630-10-4	189											
Styrene	100-42-5	146	2B	1	IRIS								
Styrene oxide	96-09-3	147	2A	0.006	P-CAL								
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746-01-6	148		4E-08	CAL	B2	33	EPA ORD	1E-09	ATSDR	B2	150000	EPA ORD
1,1,2,2-Tetrachloroethane	79-34-5	149	3			C	0.000058	IRIS					
Tetrachloroethene	127-18-4	150	2A	0.27	ATSDR	B2-C	0.0000059	CAL					
Titanium tetrachloride	7550-45-0	151		0.0001	ATSDR								
Toluene	108-88-3	152	3	5	IRIS	Inl							
2,4-Toluene diamine	95-80-7	153				B2	0.0011	CAL					
2,4/2,6-Toluene diisocyanate mixture (TDI)	26471-62-5	154	2B	0.00007	IRIS		0.000011	CAL					
o-Toluidine	95-53-4	155	2B			B2	0.000051	CAL					
Toxaphene	8001-35-2	156	2B			B2	0.00032	IRIS			B2	1.1	IRIS
1,2,4-Trichlorobenzene	120-82-1	157		0.2	HEAST	D							
1,1,2-Trichloroethane	79-00-5	158	3	0.4	P-CAL	C	0.000016	IRIS					
Trichloroethylene	79-01-6	159	2A	0.6	CAL	B2-C	0.000002	CAL					
2,4,5-Trichlorophenol	95-95-4	160											
2,4,6-Trichlorophenol	88-06-2	161				B2	0.0000031	IRIS					

Table 1. Prioritized Chronic Dose-Response Values (4/27/2010). Revisions since 06/12/07 are shown in red. **CAS NO.** = Chemical Abstracts Services number for the compound. **HAP NO.** = Position of the compound on the HAP list in the Clean Air Act (112[b][2]). "999" denotes substances under consideration for listing.	**Sources:** IRIS = Integrated Risk Information System; ATSDR = US Agency for Toxic Substances and Disease Registry; D-ATSDR = draft ATSDR; CA = California EPA; P-CAL = Proposed CAL; HEAST = EPA Health Effects Assessment Tables; Conv. Oral = Oral unit risk converted to inhalation.	**IARC WOE** = weight-of-evidence for carcinogenicity in humans (1 - carcinogenic; 2A - probably carcinogenic; 2B - possibly carcinogenic; 3 - not classifiable; 4 - probably not carcinogenic).	**EPA WOE** (1986 guidelines) = weight-of-evidence for carcinogenicity under the 1986 EPA cancer guidelines: A - human carcinogen; B1 - probable carcinogen, limited human evidence; B2 - probable carcinogen, sufficient evidence in animals; C - possible human carcinogen; D - not classifiable E - evidence of noncarcinogenicity.	**EPA WOE** (1999 guidelines) = weight-of-evidence for carcinogenicity under the 1999 EPA cancer guidelines: CH - carcinogenic to humans; LH - likely to be carcinogenic; SE - suggestive evidence for carcinogenicity; Inl - inadequate information to determine carcinogenicity; NH - not likely to be carcinogenic).

Table 1. Prioritized Chronic Dose-Response Values for Screening Risk Assessments (4/27/2010)

				CHRONIC INHALATION					CHRONIC ORAL				
				NONCANCER		CANCER			NONCANCER		CANCER		
CHEMICAL NAME	CAS NO.	HAP NO.	IARC WOE	mg/m3	SOURCE	EPA WOE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	EPA WOE	1/(mg/kg/d)	SOURCE
Triethylamine	121-44-8	162		0.007	IRIS								
Trifluralin	1582-09-8	163	3			C	0.0000022	Conv. Oral	0.0075	IRIS	C	0.0077	IRIS
Uranium compounds	7440-61-1	188		0.0003	ATSDR								
Uranium, soluble salts	URANSOLS	188											
Vinyl acetate	108-05-4	165	2B	0.2	IRIS								
Vinyl bromide	593-60-2	166	2A	0.003	IRIS	B2	0.000032	HEAST					
Vinyl chloride	75-01-4	167	1	0.1	IRIS	CH	0.0000088	IRIS					
Vinylidene chloride	75-35-4	168		0.2	IRIS	SE							
m-Xylene	108-38-3	171											
o-Xylene	95-47-6	170											
Xylenes (mixed)	1330-20-7	169		0.1	IRIS	Inl							

http://www.epa.gov/ttn/atw/toxsource/summary.html
Last updated on 2010年8月24日

Technology Transfer Network
Air Toxics Web Site

Dose-Response Assessment for Assessing Health Risks Associated With Exposure to Hazardous Air Pollutants

The information below presents tabulated dose-response assessments that the Office of Air Quality Planning and Standards (OAQPS) uses for risk assessments of hazardous air pollutants. Two separate tables are provided. Table 1 presents values for long-term (chronic) inhalation and oral exposures; Table 2 presents values for short-term (acute) inhalation exposures. It is important to note that only for the purpose of these tables that the compound categories use the CAS number for the element. However, all compounds having that element in their chemical structure are included in the compound category.

The tables compile assessments from various sources for many of the 188 substances listed as hazardous air pollutants ("air toxics") under the Clean Air Act Amendments of 1990. Sources of chronic dose-response assessments were arranged in priority order according to conceptual consistency with EPA risk assessment guidelines and level of peer review. Table 1 shows only the assessment result from the highest-priority source. The table also reflects decisions we made about several chemicals on the basis of chemical-specific information. For the oral exposure pathway, Table 1 shows only assessment results for persistent and bioaccumulative substances likely to pose important non-inhalation risks when emitted from air sources. Sources of acute dose-response assessments were not prioritized because we judged that many were not directly comparable. Table 2 shows all values from our list of sources.

The numbers in these tables support hazard identification and dose-response assessment, as defined in the National Academy of Sciences (NAS) risk assessment paradigm, for estimating the risk of contracting cancer and the level of hazard associated with adverse health effects other than cancer.

Each assessment in these tables is best visualized as an estimate within a range of possible values, surrounded uncertainty and variability. This range of possible values may change as better data become available. They are generally appropriate for screening-level risk assessments, including assessments to select contaminants, exposure routes, or emission sources of potential concern, or to help set priorities for further research. For more complex, refined risk assessments developed to support regulatory decisions for single sources or substances, we recommend evaluating dose-response in detail for each "risk driver" to incorporate appropriate new toxicological data.

- **Chronic Table 1**  
- **Acute Table 2**  

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http://www.epa.gov/ttn/fera/risk_atra_main.html
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Technology Transfer Network
FERA (Fate, Exposure, and Risk Analysis)

Risk Assessment and Modeling - Air Toxics Risk Assessment Reference Library

EPA is developing an air toxics risk assessment (ATRA) reference library for conducting air toxics analyses at the facility and community-scale. This library provides information on the fundamental principles of risk-based assessment for air toxics and how to apply those principles in different settings as well as strategies for reducing risk at the local level. A more detailed description of each volume of the ATRA library is provided below.

Risk Assessment and Modeling

- General Information
- Air Toxics Risk Assessment
- Criteria Air Pollutant Risk Assessment
- Links to EPA Risk Assessment Policy, Guidelines, and Related Materials

Volume 1: Technical Resource Manual

Volume 1 discusses the overall air toxics risk assessment process and the basic technical tools needed to perform these analyses. The manual, which covers both human health and ecological analysis, also provides a basic overview of risk management and communication. Other tools (such as the public health assessment process) are described to give assessors, risk managers, and other stakeholders a more holistic understanding of the many issues that may come into play during air toxics risk assessment and reduction projects.

Volume 2: Facility-Specific Assessment

Volume 2 builds on the technical tools described in Volume 1 by providing detailed procedures for source-specific or facility-specific risk assessments. Information is also provided on tiered approaches to source- or facility-specific risk analysis.

Volume 3: Community-Scale Assessment

Volume 3 builds on the information presented in Volume 1 to describe how to evaluate and reduce cumulative air toxics risks at the local level. The volume also discusses other multimedia risk factors that may affect communities, and strategies to reduce those risks.

Community Air Screening How-To Manual

The Community Air Screening How-To Manual provides a detailed step-by-by step guide to help community partnerships use one of the screening level approaches described in Volume 3 to understand and improve local outdoor air quality. The Manual explains how to form a partnership, clarify goals, develop a detailed source inventory, use a risk-based screening process to identify priorities, and develop options for reducing risks from priority sources and concentrations. The Manual provides a framework for bringing together technical staff and local residents to share information, deliberate, and build consensus on priorities for improving local air quality. The Manual places special emphasis on sharing information and providing the background education needed to insure that all members of the partnership can participate fully in key partnership decisions.

The ATRA library is an ongoing endeavor and may be revised periodically. EPA welcomes public input on the library at any time. Comments may be sent to Dr. Roy Smith (smith.roy@epa.gov) or Dr. Kenneth Mitchell (mitchell.ken@epa.gov).