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Washington State Toxic Air Pollutants Priorities Study

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Foreword

Writing of this report was assigned to an Air Quality Program staff toxicologist. The report was critically reviewed by several technical experts: An USEPA toxicologist reviewed the risk-based concentrations before they were used in the pollutant ranking step. A number of Air Quality Program staff, including two other toxicologists, two engineers, two emissions inventory specialists, a physicist and four non-technical, staff reviewed subsequent drafts. Other reviewers, not affiliated with the Department of Ecology, were invited to review the semifinal inter-departmental review draft. These included two toxicologists and two epidemiologists. In addition, a private consulting environmental engineer also provided critical review.

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Abstract

Many potentially toxic chemicals are emitted into the air each year in Washington. With increasing human population and activities, most kinds of toxic air pollutant emissions are growing. At the same time evidence is mounting that exposure to these chemicals is sufficient to cause serious illnesses and premature deaths in some people. Widespread exposure probably accounts for some of the occurrence of various types of cancers within our population. The Air Quality Program screened many of these toxic air pollutants to identify ones likely to pose the greatest health risks. This report documents the evolution of this process over the past six years, and explains our methods, results, and associated uncertainties. Toxic air pollutants were ranked by risk potential through examination of emissions, toxicity and estimated adverse health effects based on monitoring and concentration modeling. Integration of risk rank and modeling in a single framework allowed estimates of the relative significance of each of the 178 air pollutants

we were able to examine. Chemicals from mobile sources (motor vehicle transportation) were by far the largest contributors to potential cancer risks among all source categories. Other source categories (area, major and background) contributed significantly to potential risks, too. Emissions of particulate matter from diesel engines and residential wood-burning (uncertified fireplaces and wood stoves) are our greatest concern due their carcinogenic potency and role in cardiopulmonary (heart, circulatory and respiratory systems) illnesses and deaths, high emission volumes and long-term, widespread human exposure. Additionally, 18 other toxic air pollutants may pose increased cancer risks, and another (acrolein) may be causing significant respiratory irritation to sensitive people. These comprise a list of 21 priority toxic air pollutants. In order of importance they are: Diesel Particulate Matter; Wood Smoke; Benzene; Carbon Tetrachloride; Formaldehyde; Polycyclic Organic Matter; Chromium; Chloroform; Ethylene Dichloride; 1,3-Butadiene; Ethylene Dibromide; Acetaldehyde; Tetrachloroethylene; Trichloroethylene; Nickel; Arsenic; 1,4-Dichlorobenzene; 1,3-Dichloropropene; Ethylene oxide; Acrolein; and possibly Selenium. The estimates of the plausible upper limits to the true values of cancer risk of from exposure to these pollutants (except acrolein) together are hundreds of times higher than one-in-a-million for typical Washingtonians.

Executive Summary

Background

Hundreds of toxic chemicals, totaling millions of pounds, are emitted into the air each year in Washington. No ambient standards and few emission limits have been established for these chemicals. Currently there are 187 chemicals or classes of chemicals listed as hazardous air pollutants (HAPs) by USEPA in the 1990 Clean Air Act Amendments Section 112(b). These are chemicals for which special standards and risk assessments are required (at present most have yet to be completed). Their listing is based on estimated release volumes and toxicity. HAPs are distinct from the USEPA's seven "criteria" pollutants, which are CO, NO₂, SO₂, Pb, PM_{2.5}, PM₁₀ and O₃, which do have health-based ambient standards.

The Washington Department of Ecology's Air Quality Program (AQP) has a stated goal of reducing public health risks from toxic air pollutants by 50%, from year 2000 levels, by 2010. A milestone in achieving this goal was the development of this toxic air pollutants ranking: A screening and data review effort to identify the air pollutants posing the highest potential health risk for people in Washington performing normal daily activities. The AQP's ongoing work includes evaluation, selection and implementation of appropriate strategies to reduce emissions of priority toxic air pollutants as well as improvement of toxic air pollutants emissions inventories.

In this report, we examine release volumes and toxicity of air pollutants, and human health risks based on ambient monitoring and on toxic air pollutants concentration modeling. This examination reveals exposures to some toxic air pollutants may be high enough to cause serious illnesses and premature deaths among some residents of Washington. This examination shows that based on estimates of the plausible upper limits of the true values of unit cancer risks for several chemicals, cancer risks are not trivial: They exceed the *de minimis* level of one-in-one million.

Purpose

The purpose of this report is to detail the screening and data review processes and results by which the AQP identified the toxic air pollutants most likely to pose unreasonable risks. This report also summarizes results of the USEPA's 1996 and 1999 National-Scale Toxic air pollutants Assessments (NATA), and of air monitoring of toxic chemicals done in Washington. Lastly, it outlines recommendations based on its findings.

Methods

We developed a *toxicity-weighted emission inventory (TWEI)* and a *cancer potency-weighted emission inventory (CPWEI)*. We combined these with the USEPA's NATAs and with toxic air pollutants monitoring data to build statewide and regionally-specific toxic air pollutants assessments. NATA involved modeling of both concentrations and human inhalation exposure patterns to estimate health risks. Our use of NATA risk estimates – available for some of the toxic air pollutants – allowed our effort to more closely approach quantitative risk estimates for the toxic air pollutants considered.

Priority Toxic Air Pollutants

A toxic air pollutant was considered a *priority toxic air pollutant (PTAP)* in our study if NATA calculated excess cancer risk for it greater than one in a million, or if its non-cancer health risk reference concentration was exceeded. Also, a few toxic air pollutants that were not considered in the NATA were added to the list of PTAPs based on their rank in the CPWEI or other information.

NATA indicated the risk of developing cancer from exposure to certain toxic air pollutants may be hundreds of times higher than *de minimis* (greater than one-in-a-million) for average people living in the more densely populated areas of Washington. The aggregate cancer risk from all toxic air pollutants together is greater than one-in-a-million for all populated census tracts in Washington. Our screening, together with the 1996 NATA and with toxic air pollutants monitoring, indicate as many as 20 toxic air pollutants pose excessive cancer risks directly

through inhalation at estimated exposure levels, in all or part of Washington. These carcinogenic toxic air pollutants, along with acrolein (which may be causing respiratory and eye irritation), comprise the complete list of 21 PTAPs. In order of importance they are:

- | | | | |
|-----|---------------------------|-----|---------------------|
| 1. | Diesel Particulate Matter | 12. | Acetaldehyde |
| 2. | Wood Smoke | 13. | Tetrachloroethylene |
| 3. | Benzene | 14. | Trichloroethylene |
| 4. | Carbon Tetrachloride | 15. | Nickel |
| 5. | Formaldehyde | 16. | Arsenic |
| 6. | Polycyclic Organic Matter | 17. | 1,4-Dichlorobenzene |
| 7. | Chromium | 18. | 1,3-Dichloropropene |
| 8. | Chloroform | 19. | Ethylene oxide |
| 9. | Ethylene Dichloride | 20. | Selenium |
| 10. | 1,3-Butadiene | 21. | Acrolein |
| 11. | Ethylene Dibromide | | |

The 1996 NATA identified 16 HAPs as being present in one or more census tracts in Washington at levels high enough to result in plausible upper limits of excess cancer risks greater than one-in-a-million. Subsequently, the USEPA published the 1999 NATA, which assessed additional HAPs but used partially different methods and reporting formats (see page 132). Both the 1996 NATA and the 1999 NATA indicated benzene and carbon tetrachloride pose higher risk than the other HAPs they assessed, but the rank order of HAPs posing lesser risks in the NATAs did not match between years. The top ranked HAPs from the 1996 NATA are listed in table 1, along with toxic air pollutants not included in NATA but ranked high in the CPWEI and other analyses in this report.

Table 1. Toxic air pollutants of cancer risk concern in Washington State

Pollutant	Average lifetime excess cancer risk (plausible upper limit per million)	% of Census tracts with excessive cancer risk (plausible upper limit > 1 per million)	Carcinogenicity weight of evidence	Emissions	Notes
Diesel Particulate Matter	253	100%	IARC designated DPM as a probable (Group 2A) carcinogen in humans based on	30% of emissions were from on-road vehicles; 70% was emitted from non-road engines.	Estimated by Positive Matrix Factorization (PMF). DPM is the

			sufficient evidence in experimental animals and limited evidence in humans.		air toxic of highest concern.
Wood Smoke	ND	ND	Some wood smoke constituents are known (Class A) human carcinogens; a few other components are probable (Classes B1 and B2) human carcinogens or possible (Class C) human carcinogens.	Emissions are from residential wood combustion and open burning of logging debris.	Estimated by PMF
Benzene 71-43-2	11.6	100%	USEPA designated it a known (A) human carcinogen.	Mobile sources were 71% of emissions; area sources were 23% of emissions.	Monitored
Carbon Tetrachloride 56-23-5	9.5	100%	USEPA designated it a probable (B2) human carcinogen.	Over 99.9% is from historic sources. Area sources in all counties continue to emit small quantities.	Monitored
Formaldehyde 50-00-0	8.8	100%	USEPA designated it a probable (B1) human carcinogen.	Mobile sources constituted 49% of emissions; area sources (mainly wildfires and prescribed burns) constituted 49%; and major industrial sources comprised the remaining ~2%. Formaldehyde also arises from natural sources and atmospheric reactions.	Monitored
Polycyclic Organic Matter	3.8	78%	USEPA designated a few of the many POM chemicals as probable (B2) human carcinogens or possible (C) human carcinogens.	Major sources, such as paper mills, wood products manufacture, and petroleum refining, comprised nearly 72% of all emissions; area combustion sources (e.g. waste incinerators, crematoria, etc.) constituted 28% of emissions.	All POM except 7-PAH in NATA. Monitoring limited to a few compounds
Chromium Compounds	2.3	59%	USEPA designated Cr VI as a known (A) human carcinogen; and Cr III as not classifiable	Area sources (electroplating, residential heating, wood preserving, etc.)	One-third of emissions were assumed to be Cr VI. Total Cr

			as to its human carcinogenicity (Class D).	accounted for over 45% of emissions; Major sources accounted for 39% of emissions; mobile sources comprised the remaining 16%.	monitored
Chloroform 67-66-3	1.6	100%	USEPA designated it a probable (B2) human carcinogen.	Background sources contributed 94% of the ambient concentration; major sources constituted 5%; and area sources constituted 1%.	Monitored
Ethylene Dichloride 107-06-2	1.6	100%	USEPA designated it a probable (B2) human carcinogen.	Background sources contributed 99.95% of the ambient concentration; major sources constituted 0.03%; area sources constituted 0.02%.	Not monitored
1,3-Butadiene 106-99-0	1.4	60%	USEPA designated it a known (A) human carcinogen.	46% of emissions were from incomplete combustion of motor fuels; 46% were area sources such as residential wood, agricultural burning, wildfires and prescribed burns; major sources contributed 0.1% of the total emissions.	Monitored
Ethylene Dibromide 106-93-4	1.3	100%	USEPA designated it a probable (B2) human carcinogen.	47% of emissions were area sources; 53% were major sources; EDB is persistent and nearly all exposure results from the background level.	Monitored
Acetaldehyde 75-07-0	1.0	50.5%	USEPA designated it a probable (B2) human carcinogen.	On-road and non-road internal combustion engines comprised 68% of emissions; area sources contributed 25%; and major sources contributed 7%.	Monitored
Tetrachloroethylene (Perchloroethylene) 127-18-4	1.0	38%	IARC designated it as a probable (2A) carcinogen in humans based on based on equivocal evidence in animals and on several	Area sources contributed 98.5% of emissions; major sources contributed 1.46%; The atmospheric lifetime is	Monitored

			human epidemiological studies showing elevated risks of certain types of cancer.	~ 3 months resulting in 61% of public exposure from background sources.	
Trichloroethylene 79-01-6	0.6	24%	IARC (2A) probably a human carcinogen based on limited evidence in humans but sufficient evidence in animals.	97% of emissions were from area sources; 3% were from major sources.	Monitored
Nickel Compounds	0.5	14%	USEPA designated nickel refinery dust and nickel subsulfide as known (A) human carcinogens. USEPA designated nickel carbonyl a probable (B2) human carcinogen.	56% of emissions were from area sources, such as heating oil combustion; motor fuel combustion, especially non-road, contributed 22%; major sources accounted for 22%.	Rank based on assumption all emissions were Ni subsulfide. Total Ni monitored
Arsenic Compounds (Inorganic, including arsine)	0.2	0.95%	USEPA designated inorganic arsenic a known (A) human carcinogen.	Major sources accounted for 74% of emissions; area sources accounted for about 20%; mobile sources contributed 6% .	Total As monitored
1,4-Dichlorobenzene 106-46-7	ND	ND	IARC (2B) possibly carcinogenic to human based on animal evidence.	Area sources accounted for 99.95% of emissions; major sources for the rest. Atmospheric half-life is ~1 month.	Ranked just below arsenic compounds in the CPWEI. Cancer risk might be greater than one in a million in some area(s). Monitored
1,3-Dichloropropene 542-75-6	0.2	0.09%	USEPA designated it a probable (B2) human carcinogen.	All known emissions were from the area source category, including consumer products usage and miscellaneous organic chemical process emissions.	Monitored
Ethylene oxide 75-21-8	<0.1	0.09%	IARC designated it carcinogenic to humans (Group 1) based on limited evidence in humans, sufficient evidence in experimental animals.	All emissions reported were from area sources, which include hospital sterilizers and miscellaneous organic chemical processes.	Not monitored
Selenium and Compounds	ND	ND	USEPA designated selenium and compounds as not classifiable as to human carcinogenicity (D); and selenium sulfide as a probable (B2) human	99% of emissions were from the “miscellaneous manufacturing coating” area source category; major and mobile sources also	Assuming all emissions were selenium sulfide, selenium ranked just below chromium compounds in the

carcinogen.	contributed to man-made emissions slightly.	CPWEI. Total selenium was monitored
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ND – Not determined

IARC – International Agency for Research on Cancer

Average lifetime excess cancer risk (plausible upper limit per million) based on the 1996 NATA results

Non-cancer illness risks to Washingtonians may be significant as well: NATA indicates levels of one pollutant - acrolein - may be high enough in some areas to cause respiratory irritation, especially in sensitive subpopulations such as people with asthma. In most counties in Washington, the acrolein level exceeds the reference concentration promulgated by USEPA in the Integrated Risk Information System (IRIS), indicating potential risk. Acrolein originates largely from area sources - prescribed burns and forest fires, gasoline and diesel exhausts, wood smoke (the largest contributors to air borne particulate mater which in turn is associated with cardiopulmonary illnesses and deaths) and from cigarettes.

The NATA reported that for at least 50% of the population of Washington, the inhalation hazard index (HI) was nearly four in 1996. The HI for the most exposed 5% (in urban areas) was approximately seven. This means exposure to the combination of these respiratory irritants was at a level higher than the level at which irritation might occur in sensitive persons.

This assessment indicates mobile (transportation) emission sources of toxic air pollutants are by far the largest contributors to potential cancer risks among any type of toxic air pollutant source. Mobile source emissions are addressed primarily by the *Clean Air Act Title II Emission Standards for Mobile Sources*. Each of the other source categories (area, major and background) contribute significantly to potential cancer risks, too.

Limitations and Uncertainties

There are limitations and uncertainties with each component of this assessment (i.e., the emissions inventories, the toxicity data, the NATA results, and toxic air pollutants monitoring data). However, by bringing these components together in a single framework, we made the best estimates of the relative rank and significance of each of the air pollutants possible.

It is important to remember that our ranked toxicity-weighted and cancer potency-weighted emission inventories are only relative estimates of possible risk levels from each toxic air pollutant: The rankings cannot be interpreted as quantitative risk estimates. The reason for this is that the emission inventory data (National Toxics Inventory), although indirectly related to human exposure, are not exposure estimates. Further, the inventories are out-of-date, thus do not reflect increases or reductions in toxic air pollutant emissions that have occurred subsequently. It follows that the CPWEIs and TWEIs, as well as the NATAs, are not based on current emissions data. In basing recommendations on these data, we assume the 1996 and 1999 emissions inventories are like current year emissions, except as noted.

The NATA's key limitations and uncertainties are its assumptions in air concentration models, and the assumptions needed to cover gaps in the large database used as model input. The ASPEN model used in the NATA to estimate outdoor toxic air pollutants concentrations has a 50-mile limit. Modeled major source influences are believed to be more reliable than modeled area sources influences. Another limitation is that on-road emissions were not modeled as line sources but as area sources. Further, the reporting of toxic air pollutant emissions by major sources is voluntary and thus incomplete since some sources do not report and others have underreported their emissions at times. However, USEPA attempted to fill-in missing parts of the inventory. Because of this and because USEPA did not design NATA to characterize risks at local levels, NATA results are most reliable when viewed at the state or national level. USEPA has stated that for smaller geographic areas, these limitations may be significant.

Cancer potency assessments for some toxic air pollutants lack sufficient information to verify their carcinogenicity to humans; however, a substantial number of toxic air pollutants have sufficient evidence of carcinogenicity in animal tests to be of concern. In the absence of sufficient human data, animal toxicity data provide plausibility for extrapolation to humans and allow carefully defined quantitative estimates of risk. Animal studies have proven ability in predicting adverse responses in humans; however, some uncertainty about the potential for induction of cancer in humans remains. In addition, for some potentially carcinogenic metals, we lack knowledge about how much of the carcinogenic forms are actually emitted. Lack of specification of metal-chemical forms in the air emissions inventory is of consequence because it adds to uncertainty.

More uncertainty comes from awareness that some toxic air pollutants may counteract each other's effects resulting in less than additive toxic effects and lower than expected threats to public health. Conversely, super-additive or synergistic effect interactions among toxic air pollutants are possible too. Synergistic interactions could be posing unquantified additional threats to public health; however, we lack much of the basic information we would need to predict either positive or negative interactions.

The NATA risk analysis does not estimate individual extremes – only typical average/median exposures. A population's air pollution-associated cancer risk over 70 years (an assumed lifetime, with the more likely exposure duration of around 30 years) does not address a particular individual's lifetime risk. There are susceptibility differences among people due to such factors as age, sex, race, ethnicity and state of health, but the nature and magnitude of these differences is not well understood. Uncertainties in the risk model apply to the model as a whole, and pertain equally to individual and population risk estimates.

Several toxic air pollutants would have substantially higher ranks in our assessment if their chemical persistence and biomagnification potentials were considered. Environmental persistence and food chain biomagnification amplifies exposures in humans and other top consumer species. In this assessment, we account only for inhalation exposure, but not dietary and skin absorption exposure. Similarly, the NATA included only inhalation exposure health risks, leaving out risks associated with other routes of exposure. Inclusion of environmental persistence and biomagnification potentials in the ranking would require analysis and incorporation of these potentials. Such an analysis was beyond the scope of the current effort. The HAPs that are listed as PBTs in the Federal Resource Conservation and Recovery Act (RCRA) *Draft Waste Minimization PBT Chemical List* are noted in table 2.

Table 2. Potential and known PBT air pollutants

Air Pollutant	CAS
PCDDs (Dioxins) and PCDFs (Furans)	Several
Polychlorinated biphenyls (Arochlors)	..
Polycyclic Organic Matter	..
Chlordane	57-74-9
DDT, DDD, DDE	several

Heptachlor	76-44-8
Heptachlor epoxide	1024-57-3
Pentachlorophenol	87-86-5
Trifluralin	1582-09-8
1,2,4-Trichlorobenzene	120-82-1
1,4-Dichlorobenzene	106-46-7
Bis-(2-ethylhexyl) phthalate	117-81-7
Chloroform	67-66-3
Cyanide	57-12-5
Dibenzofuran	132-64-9
Dibutyl phthalate	84-74-2
Hexachlorobenzene	118-74-1
Hexachlorobutadiene	87-63-3
Pentachloronitrobenzene	82-68-8
Phenol	108-95-2
Antimony and compounds	several
Arsenic and compounds	..
Beryllium and compounds	..
Cadmium and compounds	..
Chromium and compounds	..
Lead and compounds	..
Mercury and compounds	..
Nickel and compounds	..
Selenium and compounds	..
Zinc and compounds	..

Despite the unavoidable uncertainty imposed by the scarcity of information and the limitations of study methods, the AQP believes that toxic air pollutants are an important concern for public health in Washington, and that procedures for reducing emissions (and associated health risks) should be further developed and implemented.

Conclusions

Toxic air pollutants pose excessive public health risks in Washington, and new measures to reduce these risks are necessary. Concerning potential economic and environmental effects of increasing control of toxic air emissions: some transportation, goods and services are necessary for protecting public health and prosperity, and it is clear vast systems of transport, and a wide variety of goods and services are integral to most people's lifestyles; however, there is still plenty of room for improvement in controlling toxic air emissions for the benefit of public health.

Recommendations

1. Our greatest opportunity for reducing public health risks from toxic air pollutants appears to be limitation of diesel engine emissions.
2. Reduction of airborne particulate matter emissions from residential wood burning could greatly improve public cardiopulmonary health.
3. We recommend further scrutiny be placed on the priority toxic air pollutants in order to identify emission sources for which additional controls are needed.
4. We also recommend residual risk analyses be done periodically to better determine trends in how much harm ambient toxic air pollutants pose over time.

Acronyms

ACGIH	American College of Governmental And Industrial Hygienists
AQP	Air Quality Program
ASPEN	Assessment System for Population Exposure Nationwide
ATSDR	Agency for Toxic Substances and Disease Registry
BCAA	Benton Clean Air Authority
BMF	Biomagnification Factor
CAA	Clean Air Act
CAPCOA	California Air Pollution Control Officers Association
CARB	California Air Resources Board
	Comprehensive Environmental Response, Compensation, and Liability Act
CERCLA	Act
COPD	Chronic Obstructive Pulmonary Disease
CPWEI	Cancer-Potency Weighted Emissions Inventory
CRO	Central Regional Office, Ecology
DPM	Diesel Particulate Matter
EI	Emission Inventory
ERO	Eastern Regional Office, Ecology
GMAP	Government Management Accounting and Performance
HAD	Health Assessment Document for Diesel Engine Exhaust
HAP	Hazardous Air Pollutant
HAPEM	Hazardous Air Pollutant Exposure Model
HEAST	Health Effects Assessment Tables
HI	Hazard Index
HQ	Hazard Quotient
HWEI	Hazard-Weighted Emissions Inventory
IMPROVE	Interagency Monitoring of Protected Visual Environments
IRIS	Integrated Risk Information System
LAA	Local Air Agency
MACT	Maximum Achievable Control Technology
MRL	Minimal Risk Level
NATA	National-scale Air Toxics Assessment
NESHAP	National Emission Standards For Hazardous Air Pollutants
NOAEL	No-Observed-Adverse-Effect-Level
NTI	National Toxics Inventory
NWCAA	Northwest Clean Air Agency
NWRO	Northwestern Regional Office, Ecology
OEHHA	Office of Environmental Health Hazard Assessment, California
ORCAA	Olympic Region Clean Air Agency
PAH	Polycyclic Aromatic Hydrocarbon
PBT	Persistent Biomagnifying Toxicant

PCB	Polychlorinated Biphenyl
PCCD	Polychlorinated Dibenzodioxin
PCDF	Polychlorinated Dibenzofuran
POM	Polycyclic Organic Matter
PSCAA	Puget Sound Clean Air Agency
PUL	Plausible Upper Limit
RBC	Risk-Based Concentration
REL	Reference Exposure Level
RfC	Reference Concentrations
RfD	Reference Dose
RL	Risk Level
SRCAA	Spokane Regional Clean Air Agency
SRP	Scientific Review Panel, California Air Resources Board
SWCAA	Southwest Clean Air Agency
TAP	Toxic Air Pollutant
TCE	Trichloroethylene
TEF	Toxicity Equivalency Factor
TLV	Threshold Limit Value
TWA	Time-Weighted Average
UCL	Upper Confidence Limit
UF	Uncertainty Factor
URE	Cancer Potency Unit Risk Estimate
USDOE	United States Environmental Protection Agency
USEPA	United States Department of Energy
WAC	Washington State Administrative Code
WDOE	Washington Department of Ecology
WOE	Weight of Evidence
WWTP	Wastewater Treatment Plant
YRCAA	Yakima Regional Clean Air Agency

1. Introduction

Toxic air pollutants and public health

A number of toxic pollutants are emitted in quantities sufficient to threaten public health in the United States. Indeed, the National Cancer Institute has estimated that exposure to environmental pollutants accounts for about 2% of the total cancer cases.^{1,2} The USEPA's Cumulative Exposure Project and their National-Scale Air Toxics Assessment (NATA) have raised concerns about the public health effects of toxic air pollutants. Studies of personal exposure employing the sampling and analysis of certain toxics in air have corroborated USEPA modeling.³ Widespread population exposure to (toxic air pollution probably results in higher rates of serious illnesses and deaths than would occur if toxic air pollutants were absent.

HAPs are toxic air pollutants listed by USEPA in the 1990 Clean Air Act Amendments Section 112(b) for which special standards and risk assessments are required (some have been completed for certain large sources but most have yet to be completed). The Federal Clean Air Act (CAA) Amendments list 187 HAPs for which emission sources would be identified and technology-based emissions standards would be developed. However, no ambient standards and few emission limits have been established for these chemicals. HAPs are distinguished legally from the "criteria pollutants"⁴ by USEPA, which has established National Ambient Air Quality Standards (NAAQS) for seven criteria-pollutants, which are ubiquitous across the United States.

¹ National Cancer Institute. 1992. Cancer statistics review: 1973-1989, Division of Cancer Prevention and Control. Publication No. 92-2789. Bethesda, MD: National Cancer Institute

² Möller, L. *et al.* 1994. Future research needs associated with the assessment of potential human health risks from exposure to toxic ambient air pollutants. *Environ Health Perspect* 102(4):193-210

³ Payne-Sturges, D. *et al.* 2004. Personal exposure meets risk assessment: a comparison of measured and modeled exposures and risks in an urban community. *Environ Health Perspect* 112(5):589-598

⁴ The air pollutants for which USEPA has established national ambient air quality standards are CO, NO₂, SO₂, Pb, PM_{2.5}, PM₁₀ and O₃.

Air Quality Program toxic air pollution goal

The Air Quality Program (AQP) seeks to reduce public health risks from toxic air pollutants in Washington by 50% by 2010 relative to 2000 levels. In this report, the AQP examines different kinds of air pollutant data to determine whether ambient concentrations present excessive human health risks. More specifically, we estimate health risks based on ambient monitoring and air toxics concentration modeling in Washington. Towards achieving the goal of reducing public health risks from toxic air pollutants, the AQP is also working to improve toxic air pollutants emissions inventories; and is evaluating various strategies that will reduce emissions of priority toxics.

This report does not propose control strategies for toxic air pollutants, but is intended to assist in proposing improved air toxics regulations and voluntary programs and to provide more information for allocating resources. Washington clean air agencies may use the results from this study to evaluate their own air toxics regulations, to focus on compounds of greatest concern, and to identify areas of potential improvements in existing air toxics programs. These results are intended to provide direction to planners and managers within the state's various air pollution control authorities who are working to develop or improve toxics programs and regulations.

Within the limitations of the available data, this study identifies the toxic air pollutants representing the highest potential health risks. Future regulatory and scientific activities can begin to focus on these pollutants to address and further evaluate their public health significance. As is the case for most urban areas in the United States,⁵ better control of toxic air pollutants exposures is necessary in most areas of Washington.

⁵ Woodruff, Tracey J., Daniel A. Axelrad, Jane Caldwell, Rachel Morello-Frosch, and Arlene Rosenbaum. 1998. Public Health Implications of 1990 Air Toxics Concentrations across the United States. *Environ Health Perspect* 106:245-251.

2. Emissions Inventory Toxicity Screening

Methods overview

This section describes the methods used to screen the toxic air pollutants listed in the emissions inventory by relative health hazard. As the first step in assessment, as many toxic air pollutants as possible were screened by quantitatively loading emission amounts with cancer potency and non-cancer reference concentrations. The resulting cancer potency weighted emission inventory and the toxicity weighted emissions inventory were then sorted from high to low magnitude.

This involved the following steps:

- Obtaining 1996 NTI emissions inventory data and MOBILE6-based emissions inventories.
- Obtaining toxicity data included chemical exposure limits from IRIS, ATSDR, OEHHA, and ACGIH, current at the time of this ranking.
- Dividing NTI emissions by allowable cancer or non-cancer risk level concentrations (the best available cancer or non-cancer risk level concentration).
- Sorting the resulting hazard-weighted emission quantities of each air toxic by relative contribution to the total air pollution-associated cancer or non-cancer risk.

These screening and ranking methods are similar to recently published methods for ranking and prioritizing toxic air pollutants including the USEPA Office of Air Quality and Planning NATA⁶ and Cumulative Exposure Project,⁷ the California Air Resources Board *Proposed Update to the Toxic Air Contaminant List*,⁸ the Minnesota Pollution Control Agency *Assessment of Air Toxics*,⁹ the and Eastern Research Group¹⁰ methods.

⁶ <http://www.epa.gov/ttn/atw/nata/>

⁷ <http://www.epa.gov/CumulativeExposure/air/air.htm>

⁸ <http://www.arb.ca.gov/toxics/toxupd.htm>

⁹ <http://www.pca.state.mn.us/air/pubs/at-report.pdf>

¹⁰ Eastern Research Group. 2000. *Documentation for the 1996 Base Year National Toxics Inventory for Onroad Sources*, for the U.S. Environmental Protection Agency. June 2, 2000

Toxic air pollutant screening process

A database with emissions inventory and toxicity data was compiled. Pollutants were included from the NTI and MOBILE6 emissions inventories if they also had any published estimates of health risk or guideline concentration limits. Division of the emission estimates for each toxic air pollutant by the best available cancer and/or non-cancer risk level concentration provided a crude estimate of the relative contribution the toxic air pollutants to the total cancer or non-cancer risk. We prepared one hazard-weighted toxic air pollutant ranking for the whole state and separate rankings for each of the state's 10 local air pollution control regions.

For the prioritization analysis, toxic air pollutants with the potential to cause cancer were scored using the following equation:

$$\text{Cancer potency weighted EI} = \frac{\text{Emissions (tons/year)}}{10^{-6} \text{ excess cancer risk concentration } (\mu\text{g}/\text{m}^3)} \quad \text{Eq. 2-1}$$

Where the 10^{-6} excess cancer risk (*de minimis*) concentration level was $10^{-6} \div \text{Unit Risk Estimate}$.

Similarly, to score air pollutants with non-cancer type health risks, the hazard-weighted EI were divided by non-cancer risk level concentrations (published health criterion or guideline concentration limit¹¹ obtained from the sources listed in section 3). Specifically non-cancer hazard weighted EIs were calculated using the following equation:

$$\text{Non cancer toxicity weighted EI} = \frac{\text{Emissions (tons/year)}}{\text{Reference concentration } (\text{mg}/\text{m}^3)} \quad \text{Eq. 2-2}$$

¹¹ Health criterion or guideline concentration limits are defined as concentrations of a pollutant in the ambient air below which there is likely to be no non-cancer type public health concern over a lifetime of exposure, or for cancer causing pollutants, defined as concentrations posing a 1E-6 upper-bound excess lifetime inhalation cancer risk for a 70-Kg adult.

Where non cancer toxicity-weighted EI is the estimate of the quantity of an air pollutant released from a source during a one year period, and RfC is the estimate of a continuous inhalation exposure to the humans likely to be without an appreciable risk of deleterious effects during a lifetime.

Source category apportionment

Emissions inventory data were detailed enough to allow separate accounting for different source categories i.e., on-road and non-road mobile, major, and area and other. After calculating the hazard-weighted emissions of each toxic air pollutant, we partitioned the total by relative contribution of each source category according to the amounts contributed by each source category.

Limitations

It is important to emphasize that this screening was only a toxic air pollutant prioritization effort and that no estimates of actual hazards were made. The rankings indicate relative potential for health impacts that could result from people's inhalation exposure to them. Although the absolute magnitude of the risks posed by the toxic air pollutants cannot be determined in this way, the analysis allowed a ranking of most of the toxic air pollutants in the combined emissions inventories.

Because of limited resources, we did not continue with comprehensive risk assessment, which would include more detailed analyses of exposure, toxicity, and risk characterization. The hazard ranking does not account for additional risks posed from chemicals that have a strong tendency to bioaccumulate and that are very persistent¹² or that are likely to biomagnify (i.e. to

¹² Resistance of a chemical to degradation determines the property of persistence. For organic chemicals released into air, water and soil, the passage of time results chemical decomposition. For metals, which do not decompose, the property of persistence is analogous to the length of time they remain in a biologically available, toxic form. In air, water, sediment and soil, bioavailability is a complex function of many factors including total concentration and speciation (physical-chemical forms) of metals, mineralogy, pH, redox potential, temperature, total organic content, and suspended particulate content, as well as volume of water and water velocity. In addition, wind transport and removal from the atmosphere by rainfall must be considered (See *Bioavailability of Metals*. 1995. John, D. and Leventhal, J., in *Preliminary compilation of descriptive geoenvironmental mineral deposit models*. du Bray, E. Ed. Open-File Report

become increasingly concentrated in increasingly higher-level animal consumers within ecological food webs). The tendency of some toxic air pollutants to biomagnify indicates that their release into the air ultimately poses greater risks for top consumers (such as humans, orca whales, raptors, etc.) than other toxic air pollutants of equal toxicity. The converse is also true: Toxic air pollutants with shorter environmental half-lives pose lower risk than longer lived pollutants of equal toxic potency.

Emissions Inventory Information

One of the main parts of our toxic air pollutants assessment was collection and evaluation of emission inventory data. We used the USEPA's 1996 National Toxics Inventory (NTI) and Mobile6 estimates for this ranking (Some of the NTI and Mobile6 data were also used by USEPA for the 1996 NATA). The inventories contain estimates of emissions of the toxic air pollutants that are regulated as HAPs under federal rules¹³.

Available emissions inventories

USEPA's Office of Air Quality and Planning Standards (OAQPS) prepared the NTI from five primary sources of data:

- State and local air agency emissions data
- OAQPS Maximum Achievable Control Technology databases
- Toxics Release Inventory
- USEPA Office of Transportation and Air Quality data
- USEPA emission factors and activity data

The NTI was then reviewed and finalized by OAQPS. States were given an opportunity to review the draft NTI data. Due to resource constraints, only a general review was possible. Corrections were made to prescribed burning emissions allocations and to the volatile organic

95-831, U.S. DEPARTMENT OF THE INTERIOR, U.S. GEOLOGICAL SURVEY. pp. 10-18
[http://pubs.usgs.gov/of/1995/ofr-95-0831/Ctoxic air pollutant2.PDF](http://pubs.usgs.gov/of/1995/ofr-95-0831/Ctoxic%20air%20pollutant2.PDF)).

¹³ Title III, Section 112(b) of the Clean Air Act (HAP list).

compound speciation for gasoline distribution. Additional review and modification were done by some of the local air agencies (LAAs) for counties within their jurisdictions.

Though not part of the NTI, highway source (diesel and gasoline engine) emissions were estimated using Mobile6.¹⁴ The ranking includes those Mobile6 estimated emissions. In its final form, the inventory for this evaluation contains HAP emissions estimates for major¹⁵, area¹⁶, non-road mobile, and on-road mobile sources. Except for DPM, most of the toxic air pollutants from highway sources were part of the 1996 NTI.

Emissions inventory methods

Data from the NTI estimates of toxic air pollutants emissions occurring in 1996 and diesel PM emission estimates was organized into a single database. We included the names of the chemicals and chemical mixtures as given in the NTI and their Chemical Abstracts Service (CAS) registration numbers. We numbered all substances that did not have CAS numbers. We put the itemized emissions from area, mobile and major point sources into each record in the database. We also noted the location of each emission in each record by documenting both the county and local LAA where the emissions occurred.¹⁷ To facilitate organization of the emissions inventory data, we used Federal Information Processing Standard (FIPS) location codes for each county.

¹⁴ MOBILE6 is USEPA's emission factor model for estimating pollution from highway vehicles. MOBILE6 calculates emissions from passenger cars, motorcycles, light- and heavy-duty trucks.

¹⁵ "Major" sources are those stationary sources that emit 10 or more tons per year of any of the listed toxic air pollutants, or 25 or more tons per year of a mixture of toxic air pollutants.

¹⁶ "Area" sources are those smaller sources that have not been inventoried as a specific point, mobile or biogenic source. They emit less than 10 tons per year of a single air toxic, or less than 25 tons per year of a combination of toxic air pollutants. In most cases, area sources are more numerous than major sources.

¹⁷ There are 10 regional local air agencies in Washington, as noted in section 1 of this report.

Toxic air pollutant sources

The main emission sources of the PTAPs, in the order of their priority, are listed below. The order is based on the ranking results at the end of this in section, and on NATA risk estimates in section 3 of this report.

1. Diesel PM

In Washington, non-road mobile sources (motors not typically used on roads and highways such as airplanes, trains, lawn mowers, construction vehicles, and farm machinery) constituted 69% of the DPM emissions on average in 1996, as listed in the NTI. The remainder came from on-road sources¹⁸ (figure 2-1). USEPA used these NTI estimates for the 1996 NATA DPM assessment.

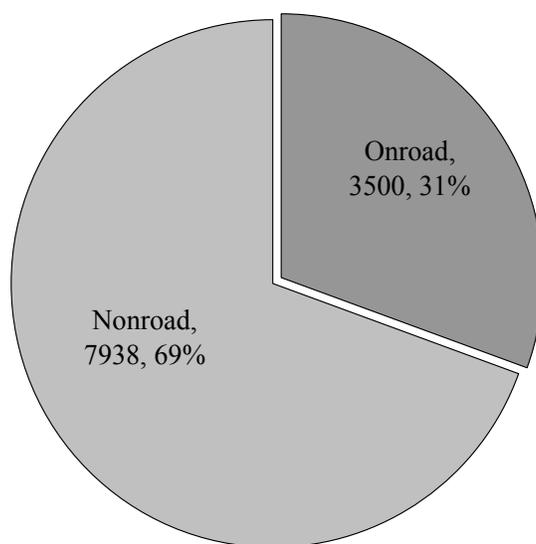


Figure 2-1. NTI 1996 DPM emissions (tons, source category percentage)

Models were used to estimate 1996 DPM₁₀ emissions. The on-road sources were estimated using EPA's PART5 model¹⁹ and WSDOT estimates of vehicle miles traveled. The non-road estimates were derived from 1996 population data and from reports by Energy and

¹⁸ <http://www.epa.gov/ttn/atw/nata/>

¹⁹ *PART5. Model and User's Guide*. EPA-AA-AQAB-94-2. Environmental Protection Agency. Office of Mobile Sources. National Motor Vehicle and Fuels Emission Laboratory. 2565 Plymouth Road. Ann Arbor, MI 48105. February 1995.

Environmental Analysis, Inc.²⁰; the US Environmental Protection Agency, Office of Air and Radiation²¹; and the California Air Resources Board²².

The Visibility Study inventory of DPM estimated total statewide on-road emissions of 4191.1 tons in 1996. It was based on USEPA's PART5 model²³ and WSDOT estimates of vehicle miles traveled. The non-road estimate of 2278.1 tons was derived from 1996 population data, and from reports by Energy and Environmental Analysis, Inc.²⁴; the U.S. Environmental Protection Agency, Office of Air and Radiation²⁵; and from knowledge of chemical speciation and size fractions reported by the California Air Resources Board²⁶. The results are shown in figure 2-2.

²⁰ *Nonroad Engine Emission Inventories for CO and Ozone Nonattainment Boundaries Seattle-Tacoma*. Energy and Environmental Analysis, Inc. Arlington, Virginia. Inventory (A+B)/2. Spreadsheets dated Aug. 25 and 26, 1992.

²¹ *Nonroad Engine and Vehicle Emissions Study - Report*. US Environmental Protection Agency, Office of Air and Radiation (ANR-433), Washington DC, 20460. 21A-2001, November 1991.

²² *Proposed Update of ARB Particulate Matter (PM) Chemical Speciation and Size Fraction Data for Diesel Truck/Bus (#118) Profile*. California Air Resources Board. 2002.
<http://arbis.arb.ca.gov/emisinv/speciate/pmtbl.htm>.

²³ *PART5. Model and User's Guide*. EPA-AA-AQAB-94-2. Environmental Protection Agency. Office of Mobile Sources. National Motor Vehicle and Fuels Emission Laboratory. 2565 Plymouth Road. Ann Arbor, MI 48105. February 1995.

²⁴ *Nonroad Engine Emission Inventories for CO and Ozone Nonattainment Boundaries Seattle-Tacoma*. Energy and Environmental Analysis, Inc. Arlington, Virginia. Inventory (A+B)/2. Spreadsheets dated Aug. 25 and 26, 1992.

²⁵ *Nonroad Engine and Vehicle Emissions Study - Report*. US Environmental Protection Agency, Office of Air and Radiation (ANR-433), Washington DC, 20460. 21A-2001, November 1991.

²⁶ *Proposed Update of ARB Particulate Matter (PM) Chemical Speciation and Size Fraction Data for Diesel Truck/Bus (#118) Profile*. California Air Resources Board. 2002
<http://arbis.arb.ca.gov/emisinv/speciate/pmtbl.htm>

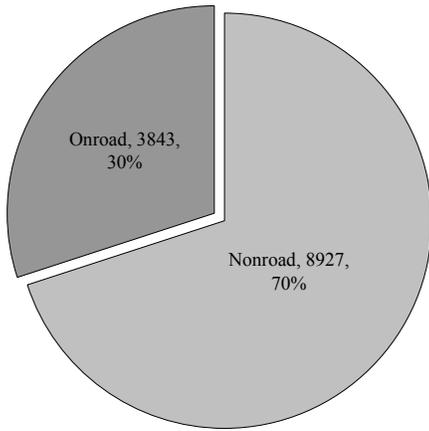


Figure 2-2. Visibility study DPM₁₀ (tons, source category percentage)

The National Emissions Inventory (NEI)²⁷ also provided DPM estimates. The initial 1996 NEI statewide non-road diesel emission estimate diesel emissions inventory was 11,801 tons: much higher than the more recent, revised estimate of 8320 tons. The NEI provides quantitative details on several subcategories that comprise the non-road contributions. The results — using the current non-road estimate, and with the on-road NEI estimate — are shown in figure 2-3.

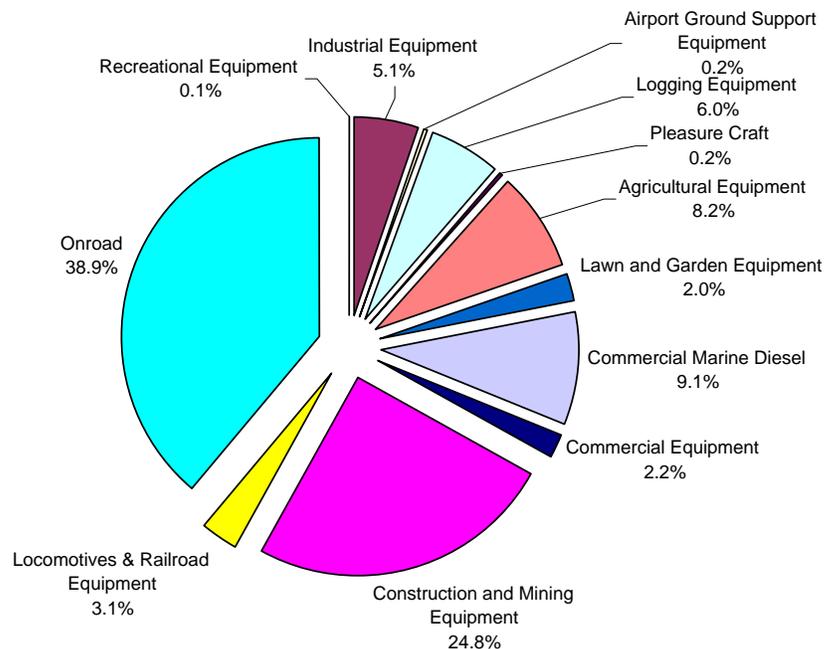


Figure 2-3. NEI 1996 DPM₁₀ subcategories contributions (11,156 tons total).

²⁷ U.S. Environmental Protection Agency. October 2002. 1999 National Emission Inventory, Version 2.0.

Items counted among non-road diesels in USEPA's NONROAD emissions model are listed in table 2-1, below.

Table 2-1. Specific sources listed in the USEPA NONROAD emissions model

Recreational equipment	Specialty vehicles/carts
Construction and mining equipment	Pavers
	Plate compactors
	Rollers
	Scrapers
	Paving equipment
	Surfacing equipment
	Signal boards/light plants
	Trenchers
	Bore/drill rigs
	Excavators
	Concrete/industrial saws
	Cement and mortar mixers
	Cranes
	Graders
	Off-highway trucks
	Crushing/processing equipment
	Rough terrain forklifts
	Rubber tire loaders
	Rubber tire tractor/dozers
	Tractors/loaders/backhoes
	Crawler tractor/dozers
	Skid steer loaders
	Off-highway tractors
Dumpers/tenders	
Other construction equipment	
Industrial equipment	Aerial lifts
	Forklifts
	Sweepers/scrubbers
	Other general industrial equipment
	Other material handling equipment
	A.C.\refrigeration
Lawn and garden equipment	Terminal tractors
	Leafblowers/vacuums (commercial)
	Snowblowers (commercial)
	Front mowers (commercial)
	Lawn and garden tractors (commercial)
	Chippers/stump grinders (commercial)
	Turf equipment (commercial)
	Other lawn and garden equipment (commercial)
Agricultural equipment	2-wheel tractors
	Agricultural tractors
	Combines

	Balers Agricultural mowers Sprayers Tillers > 6 hp Swathers Hydro-power units Other agricultural equipment Irrigation sets
Commercial equipment	Generator sets Pumps Air compressors Gas compressors Welders Pressure washers
Logging equipment	Shredders > 6 hp Forest equipment - feller/bunch/skidder
Airport ground support equipment	Airport ground support equipment
Underground mining equipment	Other underground mining equipment
Industrial equipment	Other oil field equipment
Diesel pleasure craft	Inboard/sterndrive Outboard
Diesel railroad equipment	Railway maintenance

USEPA NONROAD Emissions Model: Core Model, Version 2.1, June 2000 draft

Note that some of the variation in the NEI DPM inventories is attributable to real differences in emissions. However, significant inter-year variation is believed to be due to changes in inventory method. Comparison of the 1996 non-road inventory (used in figure 4-4) with both the recently revised and reissued NEI diesel inventory and Ecology's Visibility Study inventory further demonstrates the degree of uncertainty in quantifying diesel emissions.

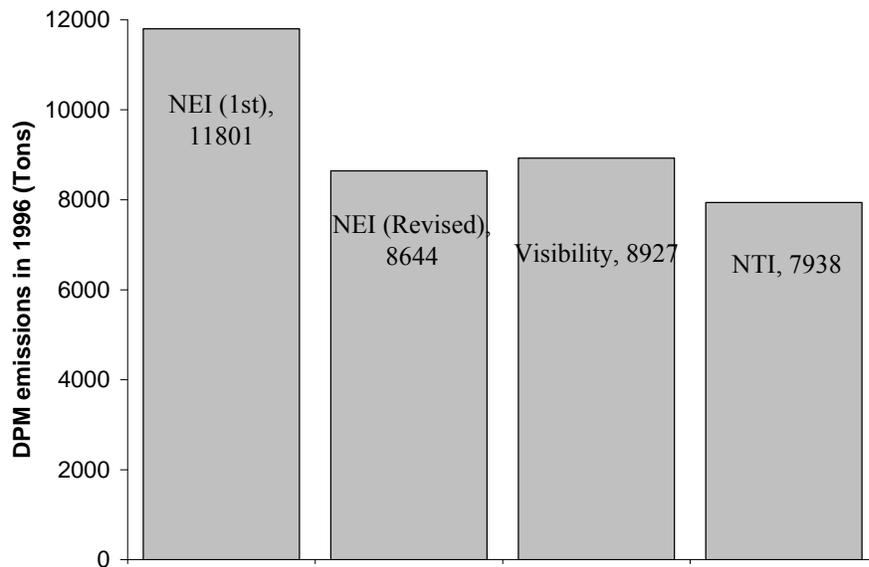


Figure 2-4. Comparison of four non-road DPM emissions inventories for 1996.

The NEI DPM inventory is also noteworthy in that it provides information both on particles 2.5- μm and less in diameter, and on particles 10- μm and less. Neither a unit risk estimate nor RfC have been issued for the smaller particles; however there is substantial evidence that DPM_{2.5} is more toxic than an equal weight of DPM₁₀. The NEI DPM_{2.5} non-road emissions inventory for 1996 is shown in figure 2-5.

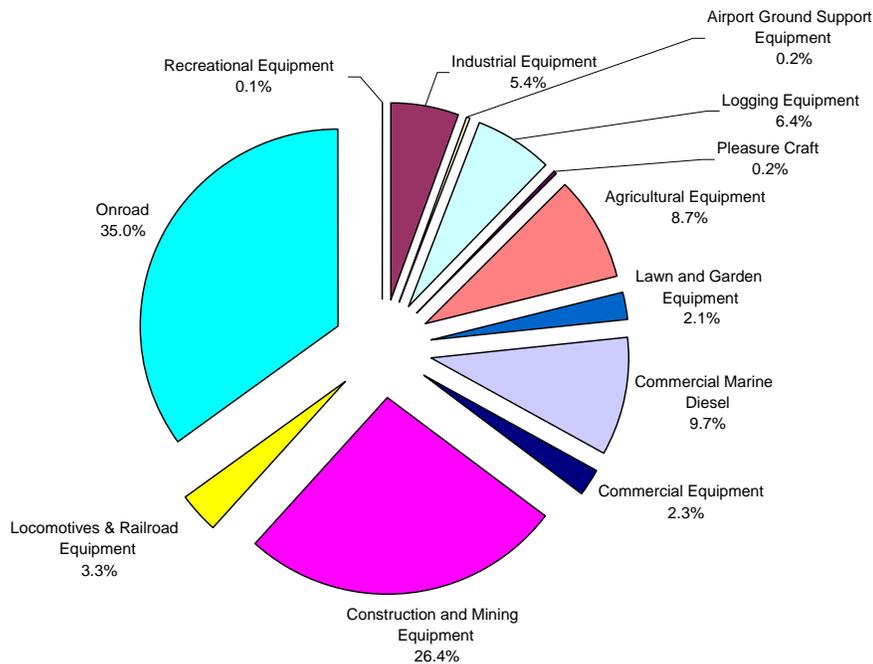


Figure 2-5. NEI 1996 DPM_{2.5} subcategories contributions (12,241 tons total).

The two most reliable estimates are the 1996 NEI inventory (3500 tons on-road, 8624 tons non-road) and the last Visibility SIP Review inventory (3843 tons on-road, 8927 tons non-road). The two inventories are not far apart, and both are well documented; however, between these, the best choice is the Visibility inventory because it was prepared with more local data.

2. Wood smoke

Residential wood smoke (RWS) is counted as an area source. Emissions estimates for RWS were developed using methods specified in the Emission Inventory Improvement Program series²⁸ by conducting surveys of wood burning habits and applying activity data to AP-42 factors for the category. The activity data was obtained from a regional survey of wood burning habits conducted by Washington State University.²⁹ Emissions estimates employ the same

²⁸ U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. January, 2001. Emission Inventory Improvement Project (EIIP) Document Series - Volume III Area Sources. Chapter 2 – Residential Wood Combustion.

²⁹ Tarnai, John. Wood Burning Stove Survey for Idaho, Oregon and Washington State. Washington State University. Social and Economic Sciences Research Center. August 2001.

emission factors used to derive RWC estimates for the NEI. Material use rates at the county level (in tons of wood burned by device type) were derived.

RWS emissions are at least partly accounted for as area emissions of the following chemicals and mixtures of chemicals: acetaldehyde, benzene, chlorinated dioxins and furans, formaldehyde, hydrochloric acid (gas), methyl ethyl ketone, polycyclic organic matter, toluene, xylenes, and free and compounded arsenic, cadmium, chromium, lead, manganese, mercury and nickel.

Aggregated emissions estimates for residential wood combustion have been found to be four to five times higher than estimates in the NEI.³⁰ This may have profound significance for all of Washington since it suggests the NEI greatly underestimates RWS and therefore RWS levels.

Emissions from open burning of logging debris (slash burning) were not estimated in Version 2.0 of the NEI, but Version 3 of the NEI draft includes an estimate of emissions for open burning-prescribed burnings, which presumably include slash burning as a subset.

3. Benzene

Mobile sources comprise the majority of benzene emissions statewide, followed by area sources. The statewide background concentration of 0.5- $\mu\text{g}/\text{m}^3$ benzene contributes about 50% or less of the ambient level in most Washington counties. However, in Clark, Cowlitz, King, Kitsap, Pierce, Snohomish and Thurston counties, an average annual benzene concentration greater than 1- $\mu\text{g}/\text{m}^3$ exists because on-road mobile, and to lesser degrees area and non-road mobile sources, combine with the background sources. Primary emissions of benzene that occurred in 1996 are shown in figure 2-6.

³⁰ Kelly, J. ORCAA. (Note that point and area source emissions are for emission year 2000. Other source category emissions are for 1999).

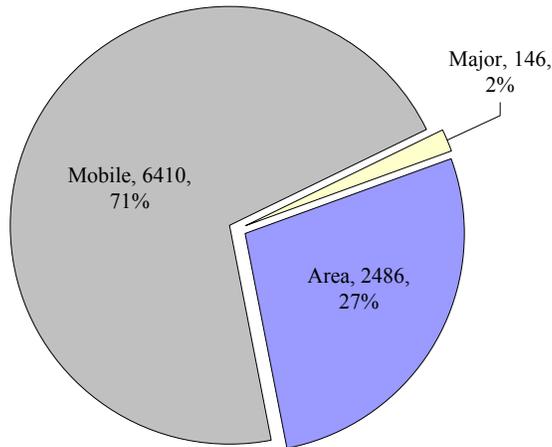


Figure 2-6. NTI 1996 Benzene emissions (Tons, percentage)

Area sources of benzene are largely wildfires and prescribed burns, and residential and commercial heating by wood burning. Other area benzene sources are listed in table 2-2.

Table 2-2. Area sources of benzene.

Asphalt concrete manufacturing
Asphalt roofing manufacturing
Aviation gasoline distribution: stages I & II
Consumer products usage
Gasoline distribution: stages I & II
Industrial boilers: distillate oil
Industrial boilers: natural gas
Industrial boilers: residual and waste oil
Institutional/commercial heating: anthracite, bituminous and lignite coal
Institutional/commercial heating: distillate oil
Institutional/commercial heating: natural gas
Institutional/commercial heating: POTW digester gas
Institutional/commercial heating: residual oil
Miscellaneous organic chemical processes
Municipal landfills
Natural gas transmissions and storage
Oil and natural gas production
Open burning: forest and wildfires
Open burning: prescribed burnings
Open burning: scrap tires
Publicly owned treatment works (POTWs)
Residential heating: anthracite, bituminous and lignite coal
Residential heating: distillate oil
Residential heating: natural gas
Residential heating: wood/wood residue

Soil and groundwater remediation
Stationary internal combustion engines - diesel
Stationary internal combustion engines - natural gas
Stationary turbines
Structure fires
Surface coatings: architectural
Treatment, storage, disposal facilities
Vessel loading/unloading

4. Carbon tetrachloride

Carbon tetrachloride (CCl₄) is very stable in the troposphere - with residence times of 30-50 years - it is now present in air at a relatively constant global background level. Over 99.9% of the carbon tetrachloride in Washington air is from historic sources here and elsewhere.

U.S. production of carbon tetrachloride began in about 1907. World production ranged from 850 to 960 kilotons over the years 1980-1988.³¹ Since 1990 the production of carbon tetrachloride has dropped. The Montreal Protocol of 1990 and its subsequent amendments established the phase-out by 1996 of production and use of carbon tetrachloride and of chlorofluorocarbons (CFCs) by major manufacturing countries. Special conditions were allowed for developing countries, where consumption of controlled substances under Annex B (including carbon tetrachloride) was required to be reduced by 85% of its 1998-2000 average level (or a calculated consumption level of 0.2-kg per capita, whichever is lower) by 2005, and completely stopped by 2010.³²

³¹ ECDIN. 1992. On-line search in the environmental chemicals data and information network. ECDIN/copyright Joint Research Centre/European Commission, Ispra.

and

BUA. 1990. [Tetrachloromethane. Report of the German Chemical Society-Advisory Committee on Existing Chemicals of Environmental Relevance.] Stuttgart, S. Hirzel Wissenschaftliche Verlagsgesellschaft (BUA Report 45).

³² UNEP. 1996. The 1987 Montreal protocol on substances that deplete the ozone layer as adjusted and amended by the Second, Fourth and Seventh Meeting of the Parties: *Handbook For The International Treaties For The Protection Of The Ozone Layer, 4th ed.* Nairobi, Kenya, United Nations Environment Programme, pp 18-39.

Area sources within all of Washington’s counties continue to emit small quantities. Examples of area source contributors, in order of descending significance, are publicly owned treatment works, traffic markings, miscellaneous organic chemical processes, municipal landfills and consumer products. There were also major point sources in Clark, Whatcom, Cowlitz, Skagit, Mason, Spokane, Yakima, Wahkiakum, Island, Skamania, and Pacific counties. Statewide emissions of CCl₄ during 1996 are shown in figure 2-7, below.

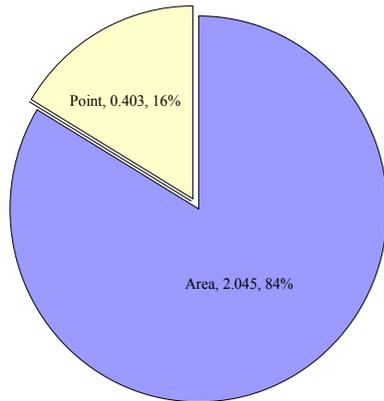


Figure 2-7. 1996 Carbon tetrachloride emissions (Tons, percentage).

5. Formaldehyde

Primary formaldehyde is a product of incomplete combustion. It is emitted into the atmosphere from mobile, major point and area sources. The largest primary source of formaldehyde is vehicular exhaust. Together on-road, non-road, and area sources make up the most of formaldehyde exposure in all but the most rural of Washington’s counties. The largest area sources are wildfires and prescribed burns. Statewide primary formaldehyde emissions listed in NTI as occurring in 1996 are summarized in figure 2-8.

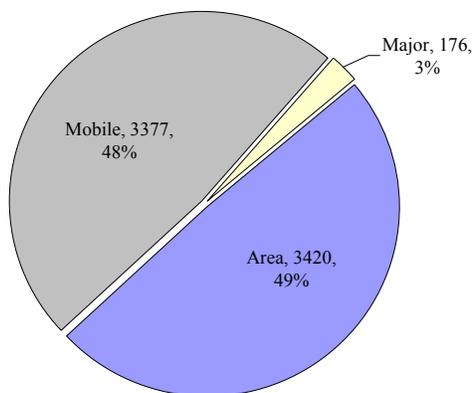


Figure 2-8. NTI 1996 Formaldehyde emissions (tons, percentage)

Nationally, other area sources of formaldehyde include the chemicals listed in table 2-3.³³

Table 2-3. Area sources of formaldehyde

Softwood drying kilns
Structure fires
Residential heating: wood/wood residue
Residential heating: natural gas
Consumer products usage
Residential heating: distillate oil
Industrial boilers: natural gas
Institutional/commercial heating using natural gas or distillate oil
Industrial boilers: residual oil
Institutional/commercial heating: residual oil
Industrial boilers using waste oil or distillate oil
Publicly owned treatment works
Miscellaneous organic chemical processes
Institutional/commercial heating: POTW digester gas
Residential heating: bituminous and lignite coal
Institutional/commercial heating: bituminous and lignite
Residential heating: anthracite coal
Institutional/commercial heating: anthracite coal
Cremation
Amino and phenolic resins production
Asphalt roofing manufacturing
Cathode ray television picture tube manufacturing
Chemical preparations
Industrial boilers: wood/wood residue
Municipal landfills
Municipal waste combustors
Open burning: forest and wildfires, prescribed burnings
Paper coating
Refractories manufacturing
Stationary internal combustion engines using diesel or natural gas
Stationary turbines
Treatment, storage, disposal facilities

Secondary formaldehyde forms as a result of photochemical oxidation in the presence ozone, nitrogen oxides and reactive organic gases. Resulting background levels are significant in all Washington counties. The atmospheric lifetime of formaldehyde is less than one day; thus long-range transport is not significant.

³³ As indicated in the NTI, some of these sources may not be present in Washington.

6. Polycyclic organic matter

The term polycyclic organic matter (POM) defines a broad class of compounds that generally includes all organic structures having two or more fused aromatic rings, and that have a boiling point greater than or equal to 100°C. POM has been identified with up to seven fused rings. Theoretically, millions of POM compounds could be formed; however, only about 100 species have been identified and studied. The most common category is the polycyclic aromatic hydrocarbons (PAHs), also known as polynuclear aromatics. The PAHs are primarily planar, nonpolar compounds with melting points considerably over 100°C. USEPA has classified seven PAHs as probable human carcinogens: benzo[a]pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene. POM is present in the atmosphere predominantly in particulate form.³⁴ POM is formed primarily during incomplete combustion of fossil fuels and vegetable matter. POM has been detected in motor vehicle exhaust, smoke from residential wood combustion, and fly ash from coal-fired electric generating plants. Estimates of statewide POM emissions by source category occurring in 1996 are shown in figure 2-9 below.

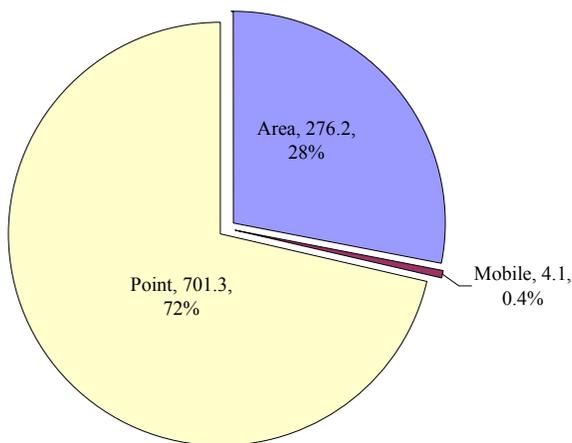


Figure 2-9. NTI 1996 POM emissions (by source category: tons, percentage)

In Washington, the primary major point sources reporting emissions during 1996 were paper mills, manufacturers of miscellaneous wood products, and petroleum refiners, as shown in figure 2-10.

³⁴ U.S. Environmental Protection Agency. *Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter*. EPA-454/R-98-014. Office of Air Quality Planning and Standards, Research Triangle Park, NC. 1998

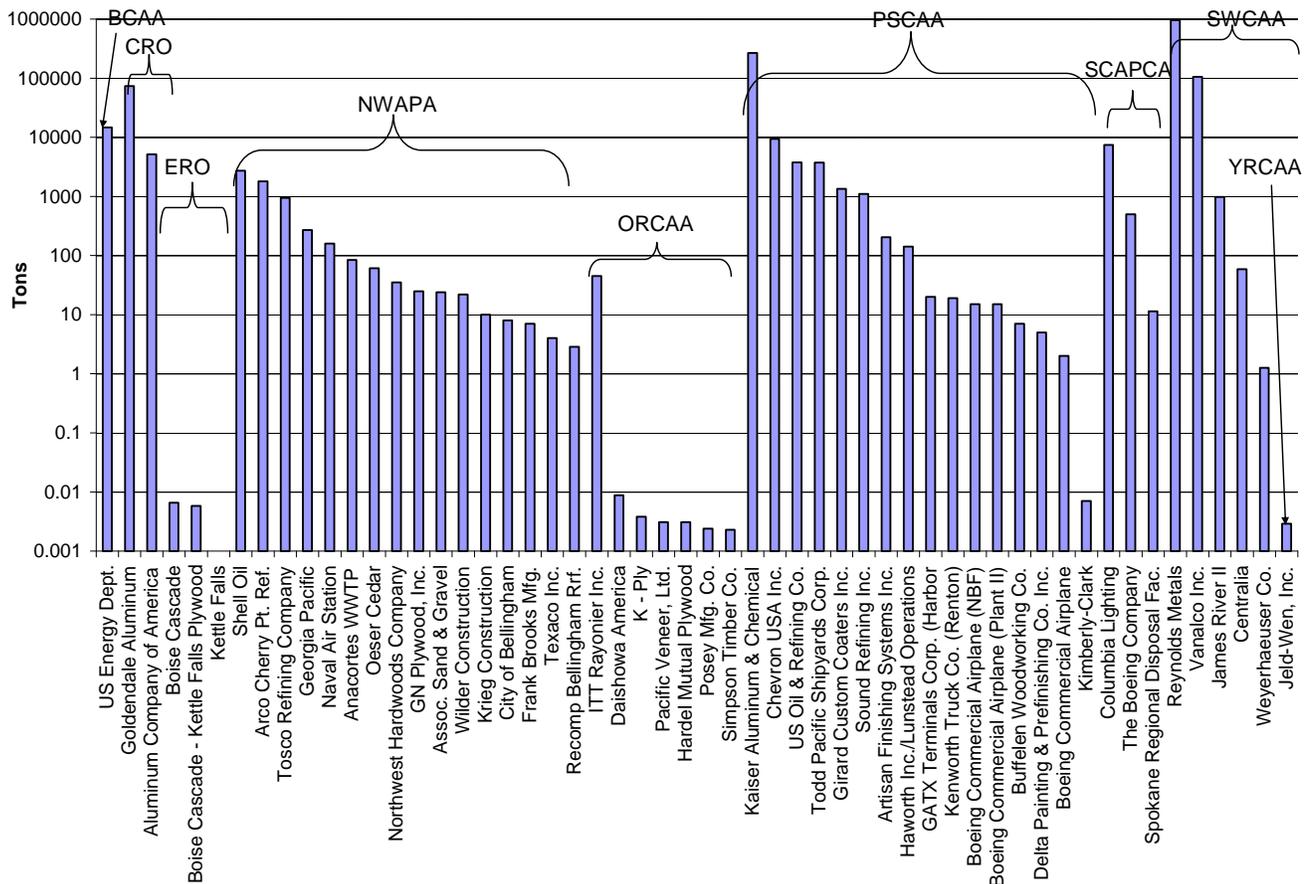


Figure 2-10. 1996 Major point source POM emissions by facility

The major point sources shown in figure 2-10 voluntarily reported POM emissions during 1996; however, similar facilities did not report emissions. The significance of this discrepancy is discussed in detail later in this section.

Area source POM emissions may include the activities listed in table 2-4.

Table 2-4. Area sources of POM and subset chemical categories

Polycyclic Organic Matter group	Area Source
POM, total ^a	Asphalt roofing manufacturing Industrial boilers: distillate oil; natural gas; residual and waste oil; and wood/wood residue Institutional/commercial heating: anthracite, bituminous and lignite coal; POTW digester gas Municipal waste combustors Open burning: forest and wildfires; and prescribed burnings Refractories manufacturing Residential heating: anthracite, bituminous and lignite coal; distillate oil; and wood/wood residue Stationary internal combustion engines - diesel and natural gas Stationary turbines
POM as 16-PAH ^b	Aerospace industries Animal cremation Asphalt concrete manufacturing Cold cleaning (miscellaneous) Consumer products usage Dry cleaning (petroleum solvent) Gasoline distribution stages I and II Human cremation Industrial boilers: wood/wood residue Industrial inorganic chemical manufacturing Institutional/commercial heating: distillate oil; natural gas; and residual oil Miscellaneous organic chemical processes Open burning: forest and wildfires; prescribed burnings; and scrap tires Paints and allied products manufacturing Publicly owned treatment works (POTWs) Reinforced plastic composites production Residential heating: anthracite, bituminous and lignite coal; distillate oil; natural gas; and wood/wood residue Structure fires Surface coatings: industrial maintenance and traffic markings Surface coatings:
POM as 7-PAH	Human and animal cremation Industrial boilers: wood/wood residue Institutional/commercial heating: distillate oil and residual oil Open burning: forest and wildfires; prescribed burnings; and scrap tires Residential heating: anthracite, bituminous and lignite coal; distillate oil; and wood/wood residue

^a All POM defined as aromatic molecules with two or more fused rings.

^b 16-PAH includes 9 PAHs in addition to the carcinogenic 7 PAHs.

7. Chromium

Chromium and chromium compound emissions in Washington are primarily from activities listed as area sources. There are also major point sources of chromium in several counties.

Mobile sources contribute to atmospheric chromium levels across all Washington regions. The source category distribution of statewide chromium emissions is shown in figure 2-11, below.

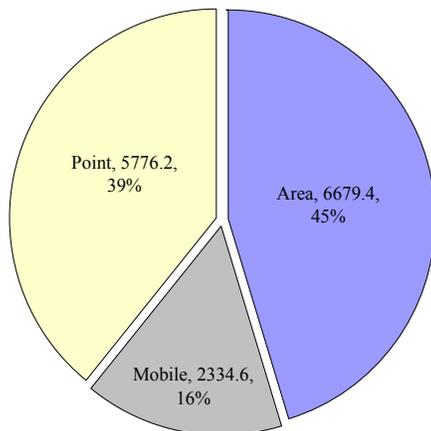


Figure 2-11. NTI 1996 Chromium emissions (lbs., percentage).

As mentioned, area sources contribute the most to chromium levels on average across Washington. In order of decreasing contribution, these area sources are noted in table 2-5.

Table 2-5. Area sources of chromium and chromium compound emissions

Fabricated plate work (boiler shops)
Hard chromium electroplating
Residential heating: wood/wood residue
Wood preserving
Residential heating: distillate oil
Industrial boilers: residual oil
Chromic acid anodizing
Institutional/commercial heating: distillate oil
Industrial inorganic chemical manufacturing
Institutional/commercial heating: residual oil or bituminous and lignite coal
Industrial boilers: waste oil
Industrial boilers: distillate oil
Miscellaneous organic chemical processes
Cremation
Decorative chromium electroplating
Fabricated pipe and fittings
Glass containers
Industrial boilers using natural gas or wood/wood residue
Industrial machinery
Institutional/commercial heating using anthracite coal or natural gas
Municipal waste combustors
National security
Open burning: scrap tires
Plastic parts and products (surface coating)
Refractories manufacturing
Residential heating using anthracite or bituminous or lignite coal, or natural gas

Emissions from major sources were the second largest category of contributors to chromium emissions across Washington. Reported major point source chromium emissions were listed in the NTI 1996 report. These are shown in figure 2-12.

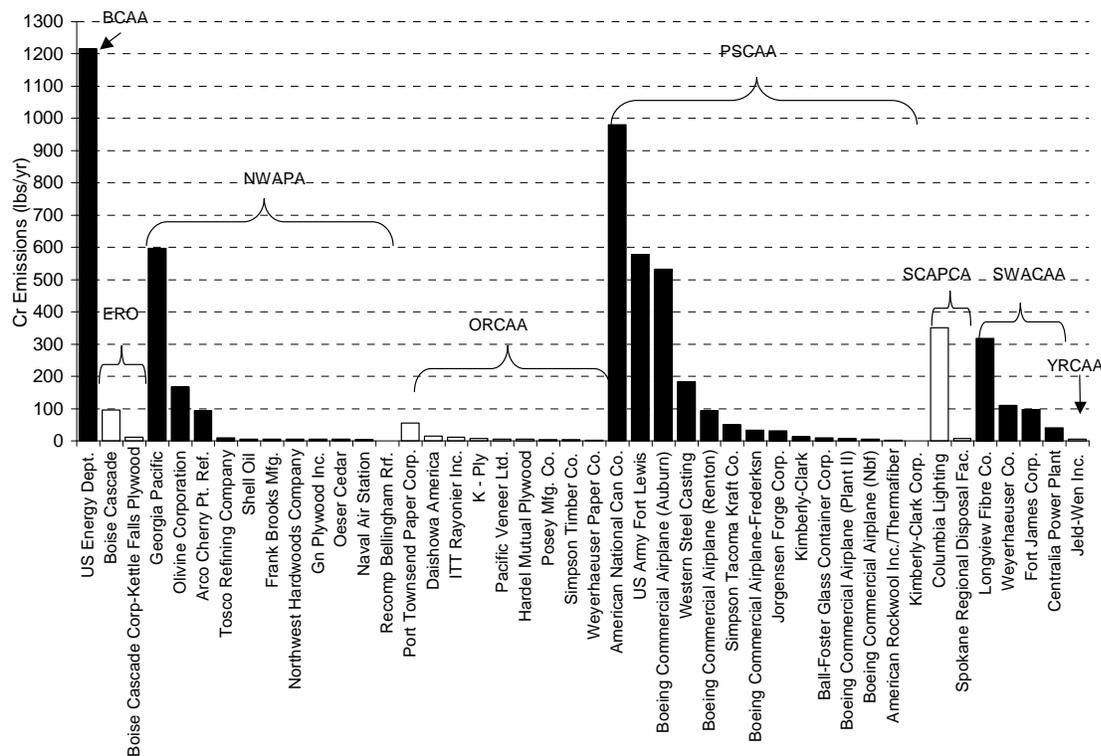


Figure 2-12. 1996 Major point source chromium emissions by facility.

8. Chloroform

Chloroform has an estimated atmospheric lifetime of 4.6 months, resulting in its global distribution from sources in and outside of Washington. 126 tons of chloroform emissions were reported in Washington during 1996 – mainly major sources (116 tons, resulting in ~5% of ambient concentrations) but also area sources (11 tons, resulting in ~1% of ambient concentrations). The 1996 NTI indicates that publicly owned wastewater treatment works constitute the largest portion of area source emissions, as listed in table 2-6, below.

Table 2-6. Statewide area source category chloroform emissions in 1996

Area Source	Lbs
Publicly owned treatment works	18,055
Consumer products usage	3,612
Municipal landfills	31
Miscellaneous organic chemical processes	22
Structure fires	4
Asphalt roofing manufacturing	1
<i>Total</i>	<i>21,725</i>

9. Ethylene dichloride

Ethylene dichloride (EDC) has an estimated atmospheric half-life of 45 days. Because of its persistence, the majority of EDC exposure is from background sources. Less than 0.1% of exposure to Washingtonians results from the small area sources emissions (528 lbs., statewide total). Area sources include consumer products, institutional/commercial and residential heating with anthracite or bituminous or lignite coal, municipal landfills, and miscellaneous organic chemical processes. The source category distribution of primary EDC emissions in 1996 is shown in figure 2-13.

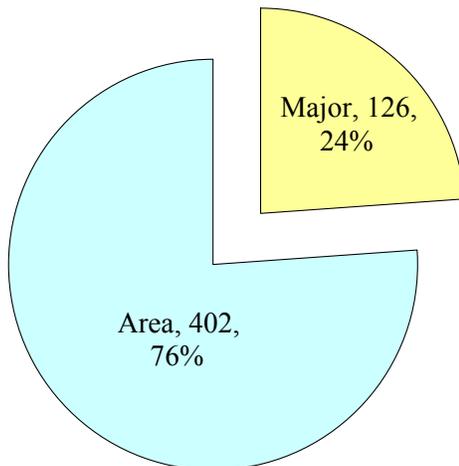


Figure 2-13. NTI 1996 Ethylene dichloride emissions (lbs, percentage).

The major point source facilities that reported EDC emissions in 1996 are listed in table 2-7, below.

Table 2-7. Major point sources reporting EDC emissions.

Facility	County	Lbs.
Centralia	Lewis	123
Weyerhaeuser Co.	Cowlitz	2.6

10. 1,3-Butadiene

The NTI estimated primary 1,3-butadiene emissions of 1150 tons in Washington occurring in 1996. Mobile source category emissions of 1,3-butadiene from incomplete combustion of gasoline and diesel fuels account the greatest portion of total emissions. Nearly all the remaining 1,3-butadiene emissions are from area sources, such as asphalt roofing manufacturing, miscellaneous organic chemical processes, wildfires and prescribed burns, open burning of scrap tires, polyvinyl chloride and copolymers production and publicly owned wastewater treatment works. Major sources reported approximately 0.1% of the total 1,3-butadiene emissions in 1996. The source category distribution is shown in figure 2-14.

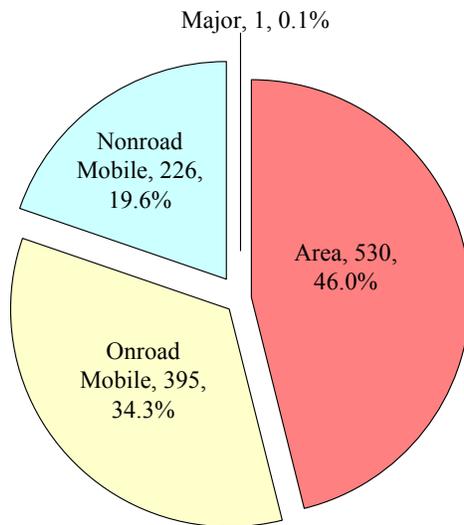


Figure 2-14. NTI 1996 Primary 1,3-butadiene emissions (tons, percentage).

The major point source facilities that reported 1,3-Butadiene emissions in 1996 are listed in table 2-8.

Table 2-8. Major point sources reporting 1,3-Butadiene emissions.

County	Facility	Lbs.
Whatcom	TOSCO Refining Company	1278
	ARCO Cherry Pt. Ref.	294
Skagit	Texaco Inc.	134
	Shell Oil	66
Island	Naval Air Station	20

11. Ethylene dibromide

Ethylene dibromide (EDB) has a calculated atmospheric half-life estimated to be 40 days. Because it is persistent and little is emitted in Washington, nearly all EDB exposure is from the globally present background level. Primary sources in Washington are responsible for less than one one-hundredth of one percent of exposure. Area sources of an estimated 2.6 lbs. of EDB constituted the total emissions occurring statewide in 1996. Area sources include miscellaneous organic chemical processes and municipal landfills. No major point sources reported emissions of EDB.

12. Acetaldehyde

Primary emissions of acetaldehyde in Washington occurring in 1996 were estimated at 2180 tons as reported in the NTI. Mobile sources of acetaldehyde were the largest category. Acetaldehyde is also a product of incomplete combustion of wood (in residential fireplaces and woodstoves), wildfires, and agricultural burning. It is used in the production of a range of chemicals. Area sources like these accounted for the next greatest portion of the total emissions. Major sources, such as large boilers, process heaters and other activities comprised the remaining emissions. Photochemical oxidation may also be a significant source of acetaldehyde concentrations in the ambient air.

The source category distribution of primary acetaldehyde emissions occurring in 1996 is shown in figure 2-15.

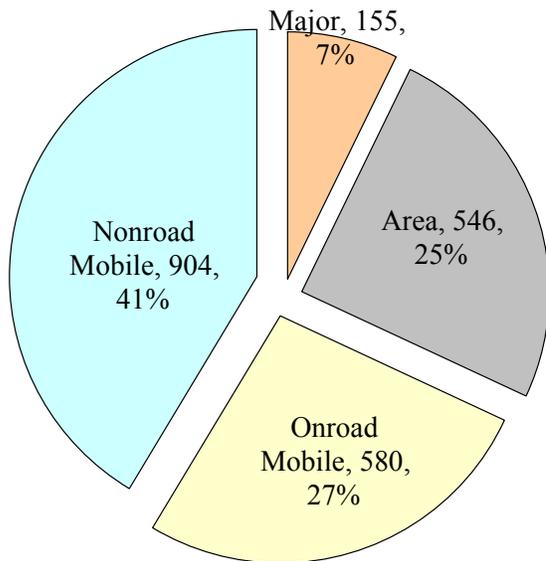


Figure 2-15. NTI 1996 Acetaldehyde emissions (tons, percentage).

Area source emissions of acetaldehyde include a number of activities listed in table 2-9.

Table 2-9. Area sources emitting acetaldehyde

Industrial boilers: distillate oil / residual oil / waste oil
Industrial boilers: natural gas
Institutional/commercial heating: anthracite, bituminous and lignite coal
Institutional/commercial heating: distillate oil / natural gas /residual oil
Institutional/commercial heating: POTW digester gas
Miscellaneous organic chemical processes
Open burning: forest and wildfires
Open burning: prescribed burnings
Publicly owned treatment works (POTWs)
Residential heating: anthracite, bituminous and lignite coal
Residential heating: distillate oil
Residential heating: natural gas
Residential heating: wood/wood residue
Softwood drying kilns
Stationary internal combustion engines - diesel
Stationary internal combustion engines - natural gas
Stationary turbines

The major point source facilities that reported acetaldehyde emissions in 1996 are listed in table 2-10, below.

Table 2-10. Major point sources reporting acetaldehyde emissions.

Facility	County	Emissions	Units
Longview Fibre Co.	Cowlitz	73.3	
James River II	Clark	19.3	
Simpson Tacoma Kraft Co.	Pierce	15.7	
Georgia Pacific	Whatcom	14.8	
Fort James Corp. (Sulfite & Kraft)	Clark	8.0	Tons
Rayonier Inc.	Clallam	6.9	
Weyerhaeuser Co.	Cowlitz	6.4	
Boise Cascade Corp.	Walla Walla	6.1	
Daishowa America	Clallam	5.0	
Port Townsend Paper Corp.	Jefferson	3.7	
Centralia	Lewis	1753	
Arco Cherry Pt. Ref.	Whatcom	493	
Nw Pipeline Corp (Plymouth Plant)	Benton	390	
Rainier Veneer Inc.	Pierce	320	
Shell Oil	Skagit	193	
Naval Air Station	Island	170	
Buffelen Woodworking Co.	Pierce	114	Lbs
Lakeside Industries	Skagit	91	
Whatcom Builders, Inc.	Whatcom	52	
GN Plywood, Inc.	Whatcom	33	
Northwest Hardwoods Company	Skagit	25	
Frank Brooks Mfg.	Whatcom	9	
US Army Fort Lewis	Pierce	8	
Lakeside Industries (Oak Harbor 053)	Island	5	
Oeser Cedar	Whatcom	4	
Kettle Falls	Stevens	0.00002	

13. Tetrachloroethylene (Perchloroethylene)

Tetrachloroethylene is a solvent used primarily in dry-cleaning operations. It is also used in degreasing operations, and in paints, coatings, adhesives, aerosols, specialty chemical production, printing inks, silicones, rug shampoos, and laboratory solvents. Tetrachloroethylene has an atmospheric lifetime of approximately three months. Most of the exposure to Washingtonians in 1996 was from background sources.

The NTI documents 539 tons of emissions in Washington during 1996. The total is shown by source category in figure 2-16.

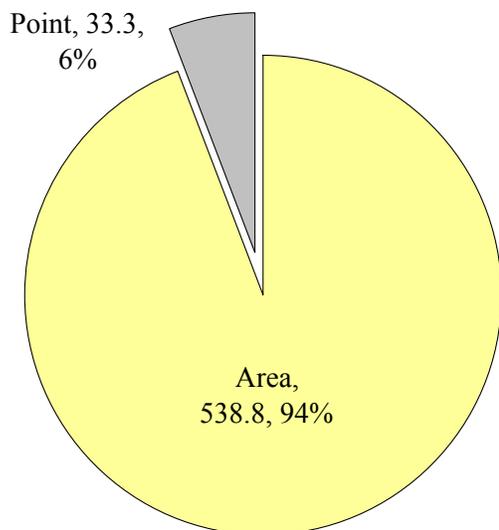


Figure 2-16. NTI 1996 Tetrachloroethylene emissions (tons, percentage)

A number of activities that release tetrachloroethylene are included in the area source emissions category: These activities are listed in table 2-11.

Table 2-11. Area sources emitting tetrachloroethylene

Cold cleaning (miscellaneous)
Consumer products usage
Halogenated solvent cleaners
Institutional/commercial heating: anthracite coal
Institutional/commercial heating: bituminous and lignite
Miscellaneous organic chemical processes
Municipal landfills
Tetrachloroethylene dry-cleaning
Plastic parts and products (surface coating)
Publicly owned wastewater treatment works
Residential heating: anthracite coal
Residential heating: bituminous and lignite coal

Area sources may contribute significantly to tetrachloroethylene exposure in the two-thirds of Washington counties that have the greatest population density. For example, in King County, area sources contribute more to exposure than do background sources. Whereas 39% of

exposure was from area sources, and 0.2% of exposure was from major sources. Among these major point sources (shown in table 2-12), the largest ones that reported emissions in 1996 are located in King County.

Table 2-12. Reported major point sources of tetrachloroethylene emissions

Facility	County	Emissions (1996)	Units
Wescor Graphics Corp.	King	7.24	Tons
Corry's Fine Drycleaning	King	4.94	
Rubingh Enterprises	King	4.74	
Leathercare Inc.	King	4.08	
Simon & Son Drycleaning	King	3.9	
Tacoma Rubber Stamp Co.	Pierce	3.4	
Boeing Commercial Airplane (Auburn)	King	1.89	
Bakker's Fine Dry Cleaning	King	1.35	
James River II	Clark	1782	Lbs
Shell Oil	Skagit	1000	
Sun Sportswear ^a	King	560	
Centralia	Lewis	132	
Gaco Western Inc.	King	12	
Weyerhaeuser Co.	Cowlitz	3	

^a Closed

14. Trichloroethylene

The 1996 NTI lists total trichloroethylene (TCE) emissions of 1390 tons. The source category contributions of these emissions are shown in figure 2-17.

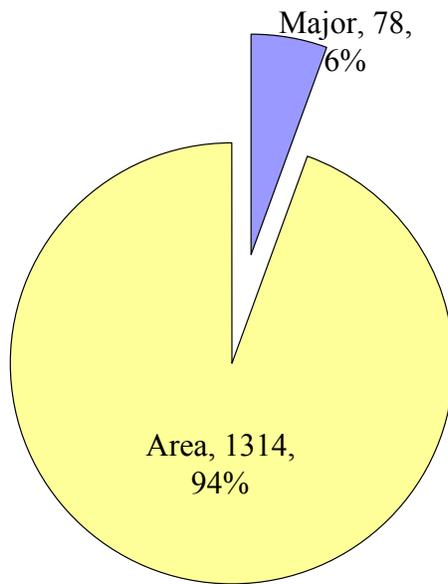


Figure 2-17. NTI 1996 Trichloroethylene emissions (tons, percentage)

Area sources, such as degreasing operations are the largest sources of TCE emissions to the atmosphere. Other significant area TCE emissions include paints and coatings, adhesive formulations, publicly owned wastewater treatment works, PVC production, distribution facilities, solvent reclamation and other activities listed in the table 2-13.

Table 2-13. Area sources emitting trichloroethylene

Cold cleaning (miscellaneous)
Consumer products usage
Fabricated plate work (boiler shops)
Halogenated solvent cleaners
Miscellaneous manufacturing coating
Miscellaneous organic chemical processes
Municipal landfills
Publicly owned treatment works

The major point source facilities that reported TCE emissions occurring 1996 are listed in table 2-14.

Table 2-14. Trichloroethylene emissions reported by major point sources

Site Name	County	Emissions	Units
Western Pneumatic Tube Company	King	23.283	Tons
Red Dot Corporation	King	16.5	
Art Brass Plating Inc.	King	8.03	
Protective Coatings Inc.	King	4.9615	
Travis Pattern	Spokane	4.92	
Asko Processing Inc.	King	4.8835	
HTB Inc.	King	3.8945	
Exotic Metals Forming Co.	King	3.3	
US Naval Shipyard Puget Sound	Kitsap	3	
Color Tech. Div. of Asko Processing	King	3771	
Mamco Mfg. Inc.	King	3286	
Universal Brass Inc.	King	2640	
WSDOT Materials Lab	Thurston	270	
Georgia Pacific	Whatcom	166	
Queen City Plating Co. Inc.	Snohomish	64	
Raytheon Systems Co.	Snohomish	12	
James River II	Clark	9.2	
ARCO Cherry Pt. Ref.	Whatcom	6	

15. Nickel

The NTI lists estimated emissions of nickel and nickel compounds in Washington of 14.9 tons during 1996. The source category contributions to total emissions are shown in figure 2-18.

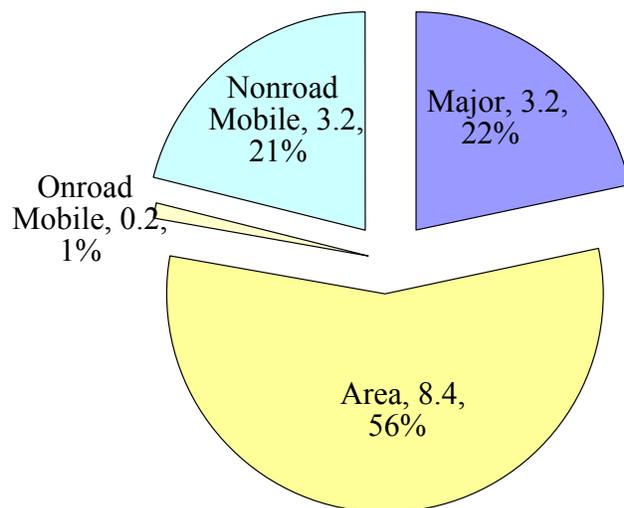


Figure 2-18. NTI 1996 Nickel emissions (by source category tons, percentage).

Area source activities with nickel emissions are listed in table 2-15.

Table 2-15. Area sources known to emit nickel and/or nickel compounds

Aerospace industries
Asphalt roofing manufacturing
Fabricated pipe and fittings
Fabricated plate work (boiler shops)
Cremation
Industrial boilers: distillate oil
Industrial boilers: natural gas
Industrial boilers: residual oil
Industrial boilers: waste oil
Industrial boilers: wood/wood residue
Institutional/commercial heating: anthracite coal
Institutional/commercial heating: bituminous and lignite
Institutional/commercial heating: distillate oil
Institutional/commercial heating: residual oil
Miscellaneous organic chemical processes
Municipal waste combustors
National security
Open burning: scrap tires
Residential heating: anthracite coal
Residential heating: bituminous and lignite coal
Residential heating: distillate oil
Residential heating: natural gas
Residential heating: wood/wood residue
Special industry machinery
Valves and pipe fittings

Major source facilities that reported nickel and nickel compound emissions during 1996 are listed in table 2-16.

Table 2-16. Reported major point source emissions of nickel compounds

Site Name	County	Lbs
Shell Oil	Skagit	3543
US Energy Dept.	Benton	1566
Arco Cherry Pt. Ref.	Whatcom	1018
Georgia Pacific	Whatcom	613.5
Longview Fibre Co.	Cowlitz	493.7
Tosco Refining Company	Whatcom	197.8
Fort James Corp. (Sulfite & Kraft)	Clark	161.0
Weyerhaeuser Co.	Cowlitz	147.5
Olivine Corporation	Whatcom	146.0
Boise Cascade	Walla Walla	132.2

Simpson Tacoma Kraft Co.	Pierce	111.8
Port Townsend Paper Corp.	Jefferson	82.6
US Army Fort Lewis	Pierce	59.0
Puget Power	Island	29.4
Centralia	Lewis	20.0
Spokane Regional Disposal Facility	Spokane	13.7
Naval Air Station	Island	9.4
Birmingham Steel Corp (West Seattle)	King	5.0
Daishowa America	Clallam	2.0
Kimberly-Clark	Snohomish	1.6
Boise Cascade Corp-Kettle Falls Plywood	Stevens	1.3
American Rockwool, Inc./Thermafiber *	Pierce	1.3
K - Ply	Clallam	0.9
Pacific Veneer, Ltd.	Grays Harbor	0.7
Hardel Mutual Plywood	Thurston	0.7
Frank Brooks Mfg.	Whatcom	0.7
Northwest Hardwoods Company	Skagit	0.7
GN Plywood, Inc.	Whatcom	0.7
Oeser Cedar	Whatcom	0.7
Jeld-Wen Inc.	Yakima	0.7
Posey Mfg. Co.	Grays Harbor	0.5
Simpson Timber Co.	Mason	0.5
Rayonier Inc.	Clallam	0.1

* Each facility reported amounts of actual annual emissions except American Rockwool, Inc./Thermafiber, which reported potential emissions.

Natural sources of nickel in the atmosphere include volcanoes and wind erosion of soils.

16. Arsenic

Total emissions of 2844 pounds of arsenic and its compounds occurring in 1996 were reported in the NTI. The source category distribution of these emissions is shown in figure 2-19.

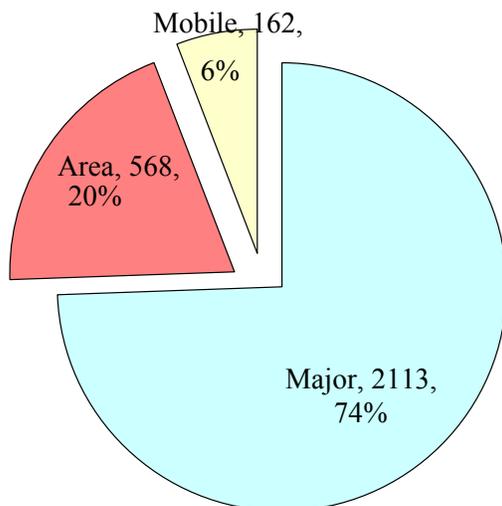


Figure 2-19. NTI 1996 Arsenic emissions (lbs, percentage)

Diesel engines are the mobile sources primarily responsible for emitting arsenic. Area sources of arsenic emissions are cremation; industrial boilers using distillate oil, natural gas, or residual oil, or waste oil, or wood/wood residue; industrial gases manufacturing; industrial inorganic chemical manufacturing; institutional/commercial heating using anthracite, bituminous or lignite coal, or distillate oil, or natural gas, or residual oil; miscellaneous organic chemical processes; municipal waste combustors; open burning of scrap tires; residential heating using anthracite or lignite coal, or distillate oil, or natural gas, or wood/wood residue; and wood preserving. Major point sources that reported of arsenic and or arsenic compound emissions in Washington in 1996 are listed in table 2-17.

Table 2-17. Reported major point sources of arsenic emissions

Facility	County	Emissions (1996) (lbs)
Longview Fibre Co.	Cowlitz	614.74
US Energy Dept.	Benton	437.00
Fort James Corp. (Sulfite & Kraft)	Clark	210.76
Weyerhaeuser Co.	Cowlitz	206.08
Simpson Tacoma Kraft Co.	Pierce	190.47
Boise Cascade	Walla Walla	166.38
Port Townsend Paper Corp.	Jefferson	98.63
Centralia	Lewis	40.00
Arco Cherry Pt. Ref.	Whatcom	33.00
Georgia Pacific	Whatcom	24.15
James River II	Clark	19.97
Spokane Regional Disposal Facility	Spokane	11.29
Daishowa America	Clallam	8.02
Kimberly-Clark	Snohomish	6.43
Recomp of Washington	Whatcom	6.00
Boise Cascade Corp-Kettle Falls Plywood	Stevens	5.35
Olivine Corporation	Whatcom	4.00
K - Ply	Clallam	3.46
Recomp Bellingham Rrf.	Whatcom	2.84
Pacific Veneer, Ltd.	Grays Harbor	2.80
Hardel Mutual Plywood	Thurston	2.80
Frank Brooks Mfg.	Whatcom	2.71
Northwest Hardwoods Company	Skagit	2.68
GN Plywood, Inc.	Whatcom	2.68
Oeser Cedar	Whatcom	2.68
Jeld-Wen Inc.	Yakima	2.68
Posey Mfg. Co.	Grays Harbor	2.15
Simpson Timber Co.	Mason	2.13
ITT Rayonier Inc.	Clallam	0.69

Weyerhaeuser Paper Co.	Grays Harbor	0.36
Kimberly-Clark Corp.	Snohomish	0.29
American Rockwool, Inc. / Thermafiber*	Pierce	0.24

* Each facility reported amounts of actual annual emissions except American Rockwool, Inc./Thermafiber, which reported potential emissions.

17. 1,4-Dichlorobenzene

Total emissions of more than 229 pounds of 1,4-dichlorobenzene were reported in 1996. The only major point source that reported emissions was the Anacortes wastewater treatment plant (WWTP) in Skagit County. They reported emissions of 0.1065 tons, as shown in figure 2-20.

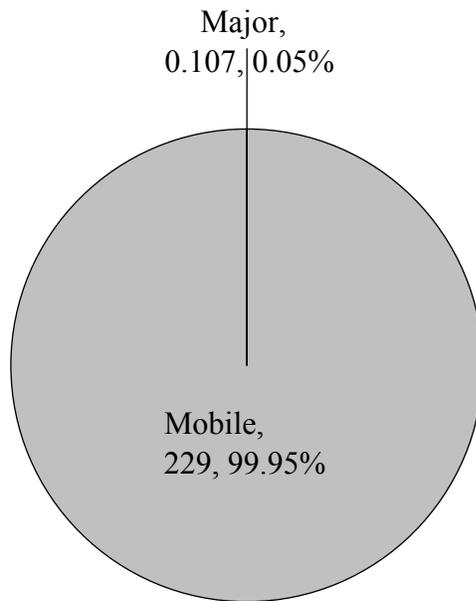


Figure 2-20. NTI 1996 1,4-Dichlorobenzene emissions (source category contributions: tons, percentage)

Although no area sources emissions were listed in the NTI, area sources of 1,4-dichlorobenzene may include consumer products usage, miscellaneous organic chemical processes, municipal landfills, and publicly owned treatment works.

18. 1,3-Dichloropropene

All 441 tons of 1,3-dichloropropene emissions occurring in 1996, as reported in the NTI, were from area sources. These area source emissions were from consumer products usage and miscellaneous organic chemical processes.

19. Ethylene oxide

All 14.9 tons of ethylene oxide emissions occurring in 1996, as reported in the NTI, were from area sources. Area sources include hospital sterilizers and miscellaneous organic chemical processes such as in the production of detergents, ethylene glycol, and glycol ethers; and publicly owned treatment works for wastewater.

20. Selenium

Selenium is a naturally occurring element. As shown in figure 2-21, area sources constitute 99% of human-made emissions. Major point sources account for the remaining anthropogenic emissions. Selenium is also released to the atmosphere as selenious acid and elemental selenium with the combustion of fossil fuels.³⁵

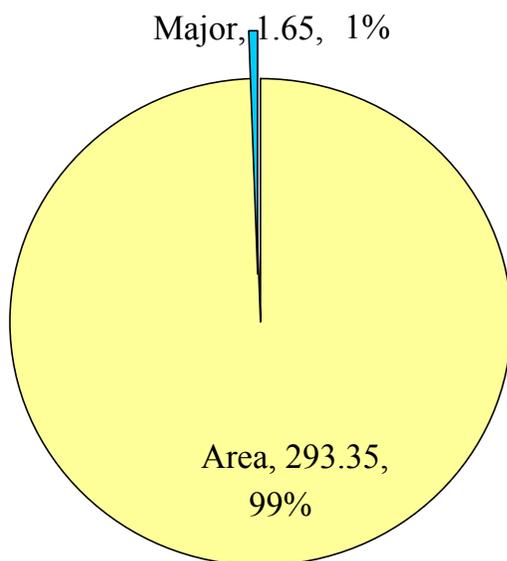


Figure 2-21. NTI 1996 Selenium emissions (tons, percentage)

³⁵ Hazardous Substance Databank. 1991. Information from the Hazardous Substance Databank. Toxicology Data Network System. National Library of Medicine. Washington, D.C.

Area source selenium emissions may include the activities listed in Table 2-18.

Table 2-18. Area sources of selenium emissions.

Industrial boilers: distillate, residual and waste oil
Institutional/commercial heating: anthracite, bituminous and lignite coal
Institutional/commercial heating: distillate and residual oil
Miscellaneous manufacturing coating
Miscellaneous organic chemical processes
Open burning: scrap tires
Prepared feeds manufacturing
Residential heating: anthracite, bituminous and lignite coal
Residential heating: distillate oil

The major sources that reported selenium emissions occurring in 1996 are listed in table 2-19.

Table 2-19. Reported major point sources of selenium

Facility	County	Lbs (1996)
Centralia	Lewis	1540
Longview Fibre Co.	Cowlitz	620
Weyerhaeuser Co.	Cowlitz	256
Fort James Corp. (Sulfite & Kraft)	Clark	212
Simpson Tacoma Kraft Co.	Pierce	192
Boise Cascade	Walla Walla	182
US Energy Dept.	Benton	158
Port Townsend Paper Corp.	Jefferson	100
Arco Cherry Pt. Ref.	Whatcom	19
Georgia Pacific	Whatcom	11
American Rockwool, Inc./Thermafiber	Pierce	0.6 ^a

^aEach facility reported amounts of actual annual emissions except American Rockwool, Inc./Thermafiber, which reported potential emissions.

21. Acrolein

In 1996, the estimated primary emissions of acrolein reported in the NTI in Washington totaled 833 tons. Prescribed burns, forest fires, gasoline and diesel exhausts were the largest emissions

sources. As shown in figure 2-22, acrolein is also emitted from major sources (including paper mills and wood product manufactures). Notably, it is used as an intermediate for glycerin, methionine, glutaraldehyde, and other organic chemicals; it is a registered aquatic algicide and herbicide in Washington. Acrolein is also a photooxidation product of various hydrocarbons including 1,3-butadiene.³⁶

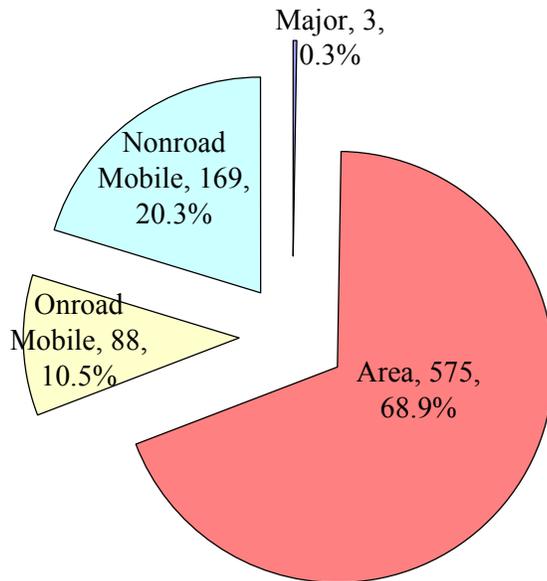


Figure 2-22. NTI 1996 Primary acrolein emissions (tons, percentage).

Emissions inventories limitations and uncertainties

The NTI was prepared using two approaches: In one, emission estimates were based on emission factors that were developed from experimentally-obtained data. Estimates were scaled to the county-level using data on some measure of activity related to emissions. For example, population density was used to estimate the number of woodstoves in use. This scaling approach is less time consuming than the other approach: collecting and compiling emissions data from all local individual sources. However, the accuracy of scaled emission estimates suffers from

³⁶ See <http://www.arb.ca.gov/toxics/tac/factshts/acrolein.pdf>

inaccuracies in emission factors³⁷ or activity estimates, which in some cases may be significant. The area category, on-road mobile category, and non-road mobile category emissions included in the NTI were developed by USEPA, mainly using the emission factor scaling approach. Only major source inventories were prepared using individual source emissions estimates.

Adding together the estimates developed specifically for each member of a group of source activities is one way to inventory toxic air pollutants. Because some NTI estimates (primarily those in the major source category) were developed specifically for individual sources, the inventory results are believed to be more accurate than those that were estimated by emission-factor-scaling approach (primarily those in area and mobile source categories). However, it is important to remember that major point source reporting is not uniform throughout the state. While there is regulatory authority to require toxic air pollutants reporting, it has not specifically been required. This has resulted in inconsistent reporting requirements throughout the state ranging from no required reporting to fairly comprehensive reporting programs. Further, since there is no federal toxics data reporting rule (other than TRI), an air pollution agency may choose not to submit toxics data even if they collect them.

The USEPA supplemented major point source data in NTI with Maximum Achievable Control Technology (MACT) and TRI data in order to correct the problem of inconsistent reporting, at least in part. The USEPA and local air agencies have attempted to fill-in parts of the inventory that are missing. Despite this, at the regional level, these limitations may be significant. The emissions inventories are incomplete. Further, natural emissions (e.g. formaldehyde) and historical emissions (e.g. EDB) are not counted in the NTI.

The emission factor scaling approach is a population-based model of emissions. The mobile and area source inventory models are “top down” and may overlook a few air emission sources altogether. It is probably not as reliable for prediction of emission inventories in smaller geographic areas, but probably more reliable for larger geographic areas. We concur with USEPA that caution should be exercised when interpreting county-level emission inventories,

³⁷ For example, technical limitations of acrolein measurement result in a high level of uncertainty in emissions factors. It is doubtful the air sampling and analysis method is as accurate as the methods developed for other HAPs. This might have affected the accuracy of acrolein emission factors.

and that the state-level emission inventories are reasonably more accurate. More detailed information on emissions of toxic air pollutants is needed to adequately assess risks at the local level.

Emissions inventories recommendations

The AQP and Washington's Local air agencies should continue to develop and improve procedures for estimating and tracking toxic air pollutant emissions. Continued efforts will lead to refined estimates of regionally important sources and toxic air pollutants risks. To date, detailed emissions estimates for many sources have not been developed. More comprehensive, facility and process-specific emissions data for sources in each LAA jurisdiction are needed.

Part of the NTI was derived from specific emission estimates from individual sources. The remainder of the inventory, including the most significant source category - mobile sources, was developed using the emission factor scaling approach. Because of the uncertainty stemming from this limitation, we recommend that any proposed decisions based solely on emissions inventory data be reviewed with emphasis on the reliability of the inventory itself. In other words, extra caution is needed when considering the implications of emissions inventory-based decisions. The NTI is most reliable at a state-wide scale as a guide for new monitoring and risk assessment efforts.

Air pollutant toxicity information

We used quantitative toxicity estimates for weighting TAP emissions inventories. The need for this is illustrated in the following example: 1,3-dichloropropene emissions were more massive than arsenic emissions during 1996 (441 tons of DCP versus 1.4 tons of arsenic); however, 1,3-dichloropropene is less potent a carcinogen than arsenic (lifelong average exposure to $0.2\text{-}\mu\text{g}/\text{m}^3$ 1,3-dichloropropene may raise cancer risk by 1/1,000,000 as compared to the same increase in cancer risk by exposure to $0.0002\text{-}\mu\text{g}/\text{m}^3$ arsenic), it is evident arsenic may be of greater public

health concern than 1,3-dichloropropene. Thus the knowledge of respective toxic potencies of each air pollutant is needed for comparing hazard-weighted emissions.

For estimating risks, carcinogenicity was considered separately from non-cancer toxicity risk assessment. Toxicity data were used for determining the types and degree of health risks. The key health problems are both chronic (long-term, annual average to lifetime), which include cancer, developmental effects, asthma induction, allergic sensitization; and acute (short-term, hours to days), which include primarily eye irritation and respiratory irritation (including asthma exacerbation). Note that diesel particulate matter, wood smoke, and several of the chemical air pollutants considered in this report exist in the atmosphere mostly as microscopic particles (larger than molecular gasses), and that exposure to these respirable particles is associated with cardiopulmonary illness and death. However, quantification of particulate-associated health impairments was beyond the scope of this report.

Risk-based concentrations

We collected published cancer and non-cancer health-protective exposure concentration guideline limits, which we term: risk-based concentrations (RBCs), published by the USEPA; the California Office of Environmental Health Hazard Assessment (OEHHA); the Agency for Toxic Substances and Disease Registry (ATSDR); and the American College of Governmental and Industrial Hygienists (ACGIH).

Cancer risk from chemical exposure is treated as a non-threshold phenomenon. In other words: there is some finite risk from any exposure, although it may be quite small at low exposures. The cancer potency unit risk estimate (URE) can be used to quantify excess cancer risk using equation 2-3.

$$\text{Excess cancer risk} = \text{Average exposure concentration} \times \text{URE} \quad \text{Eq. 2-3}$$

Where exposure concentration ($\mu\text{g}/\text{m}^3$) is provided by measurement or air concentration modeling, and URE [units are $(\mu\text{g}/\text{m}^3)^{-1}$] is a health assessment value that provides an estimate of the slope of the exposure-response curve at low exposures. UREs are estimated from human

epidemiological data, if available or otherwise, experimental animal data. For expressing the aggregate toxic air pollutants cancer risk, the risks from individual toxic air pollutants are summed in this report. The NATA also sums cancer risks from different toxic air pollutants.

Non cancer health risk from chemical exposure is a considered a threshold phenomenon, for most toxic air pollutants: A level of exposure is thought to exist below which there is no health risk. The threshold is estimated from human data when available, or else from animal data. The no-effect exposure concentration level, established by human and/or animal data, is divided by necessary uncertainty or safety factors to derive a non cancer RBC. Uncertainty factors are used to account for sensitive subpopulations, extrapolation from animal data to human populations, and other assumptions about the data. In the Hazard Quotient (HQ) risk screening approach, toxic air pollutants with HQs less than one are deemed to present no risk, whereas toxic air pollutants with HQs greater than one may pose health risks:

$$\text{Hazard Quotient} = \frac{\text{Estimated average exposure concentration}}{\text{RBC}} \quad \text{Eq. 2-4}$$

Where the RBC is the concentration in air at or below which no adverse non-cancer health impacts are anticipated. Additivity of risks from more than one chemical can be assumed if the chemicals affect the same target tissue or organ, especially if they act by the same physiological mechanism. In such cases, HQs may be added together. Such a sum is called the hazard index (HI).

Selection of health risk-based concentrations

We used risk-based concentrations to weight toxic air pollutants emission inventories by cancer and non-cancer health hazard. RBCs from the highest preferred data source were used. The RBCs for some toxic air pollutants differ between issuing authority. In order to choose one RBC over another, we applied decision hierarchies. In table 2-20, the sources for cancer potency RBC are listed, in order of preference, with accompanying descriptions.

Table 2-20. Sources of cancer risk-based concentration values

Preference	Authority
1	Integrated Risk Information System inhalation (IRIS) UREs. We collected the IRIS upper bound concentration estimate for carcinogens at which an excess cancer risk of 1 in 100,000 may result for an average person exposed at this level over an average lifetime of 70 years. We also noted the USEPA cancer classification or weight of evidence (WOE) for each air toxic, i.e., groups A, B1, B2, C, D and E, for carcinogenicity. This system is for characterizing the extent to which available data support the hypothesis that each chemical causes cancer in humans (see glossary).
2	California Office of Environmental Health Hazard Assessment UREs. OEHHA's quantitative concentration-response information on chemical carcinogenicity is developed and defined similarly to USEPA's UREs.
3	American College of Governmental and Industrial Hygienists Threshold Limit Value (TLV)-Time-Weighted Average (TWA) concentrations for workplace exposures. TLV-TWA definition: the concentration for a conventional 8-hour workday and 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed, day after day, without excessive cancer risk.
4	USEPA IRIS oral UREs. We converted ingested dose quantities to inhalation concentrations if they were the only cancer potency estimates available.
5	Peer reviewed primary scientific literature

The sources for reference concentrations (RfCs) and similar non cancer RBCs are listed in order of preference in the table 2-21:

Table 2-21. Sources of non-cancer risk-based concentration values

Preference	Authority
1	USEPA IRIS inhalation Reference Concentration. Estimates of exposure levels below which the population, including sensitive subpopulation groups, are protected from adverse non cancer health effects.

Agency for Toxic Substances and Disease Registry chronic inhalation Minimal Risk Level. Similar to the USEPA's RfCs for non-cancer endpoints, MRLs are estimates of the daily human exposure to hazardous substances likely to be without appreciable risk of adverse non-cancer health effects for chronic (365 days or longer) exposures. MRLs are based on non-cancer health effects only and are not based on a consideration of cancer effects. MRLs are intended to serve as screening levels to identify contaminants that may be of concern. The ATSDR uses the no-observed-adverse-effect-level/uncertainty factor (NOAEL/UF) approach to derive MRLs. MRLs are set below levels that, based on current information, might cause adverse health effects in the people who are most sensitive to such effects. However, exposure to a level above the MRL does not necessarily mean that adverse health effects will occur. MRLs are intended to serve as a screening tool.

3 OEHHA chronic inhalation Reference Exposure Levels. OEHHA defines a REL as a concentration level at or below which no health effects are anticipated. This is similar to USEPA's non-cancer exposure-response assessment.³⁸

4 ATSDR intermediate (>14 -364 days) duration inhalation MRLs. We preferred to use MRLs derived for long-term exposure but also collected MRLs derived for intermediate exposure. Intermediate MRLs were used to weight the EIs of six of the toxic air pollutants. We divided the intermediate MRLs by five to simulate long-term exposure (365 days and longer). When examining the ATSDRs database, we also recorded the critically affected organ system; respiratory, neural, hepatic, etc. of each air toxic chemical listed.

³⁸ OEHHA URE and RELs are widely used in risk assessments, for example USEPA used OEHHA UREs in the 1996 NATA, see NATA's appendix H.

5 ACGIH Threshold Limit Values Time Weighted Average for workplace exposures. TLV-TWAs are defined as concentrations for a conventional 8-hour workday and 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed, day after day, without adverse non cancer health effects. We modified TLV-TWAs for use in this assessment as noted below.

6 USEPA IRIS oral Reference Dose. We estimated inhalation concentrations from ingested reference doses (RfDs) if they were the only non cancer toxicity estimates available.

Excess cancer risk exposure concentrations unit conversions

We used the air concentration potentially associated with a 1/100,000 increased cancer risk level (the 10^{-5} excess cancer risk level was selected for ranking for the sake of consistency. Use of some other risk level, such as 10^{-6} or 10^{-4} , would not have changed the rank order of the toxic air pollutants or our conclusions, as long as the risk level is the same for all the conversions) to weight the emission inventory of potentially carcinogenic toxic air pollutants. The OEHHA does not publish 1/100,000 excess risk level concentration estimates expressed as such. Instead they list carcinogenicity UREs. We converted the UREs published by OEHHA to the equivalent 10^{-5} excess cancer risk level concentration using equation 2-5.

$$10^{-5} \text{ Risk level (mg/m}^3\text{)} = 10^{-5} \div \text{URE (mg/m}^3\text{)}^{-1} \quad \text{Eq. 2-5}$$

MRLs and TLV-TWAs for gasses

The ATSDR and ACGIH express some MRLs and TLV-TWAs in ppm units (for toxic air pollutants that are normally gasses). We converted these to units of mg/m^3 assuming standard temperature and pressure conditions (760 torr, 25° C) and including a term for molecular weight:

$$\frac{\text{TLV (ppm)} \times \text{Molecular Weight}}{24.45} = \text{TLV (mg/m}^3\text{)} \quad \text{Eq. 2-6}$$

TLV-TWA scaling

We evaluated using ACGIH TWA-TLVs for use in weighting emissions of some toxic air pollutants. We could not use the TWA-TLVs without first adjusting them from occupational exposure to continuous exposure conditions. Below, we describe this adjustment of the TLV-TWAs — as published — to scaled RBCs. Using scaled TWA-TLVs for some toxic air pollutants allowed us to screen more toxic air pollutants than would have been possible otherwise. When the hazard-weighted emissions inventories were completed, we found that none of the toxic air pollutants whose inventories we weighted with scaled TWA-TLVs were among those presenting apparent public health threats. Our process for determining comparison (scaling) factor ranges is noted here in the interest of making our ranking methods transparent.

To make TLV-TWAs more directly comparable to RBC's from other authorities, we divided them by a range of factors. The analysis that led to the selection of the factor range was one of comparing the TLV-TWAs to corresponding RBCs among those toxic air pollutants that, in addition to TLV-TWAs from ACGIH, had RBCs from one or more of the other authorities. These toxic air pollutants' TLV-TWAs were divided by corresponding RBCs from other authorities. We examined the distributions of the resulting quotients sets. Results of the comparison of potentially carcinogenic toxic air pollutants are displayed in figure 2-23.

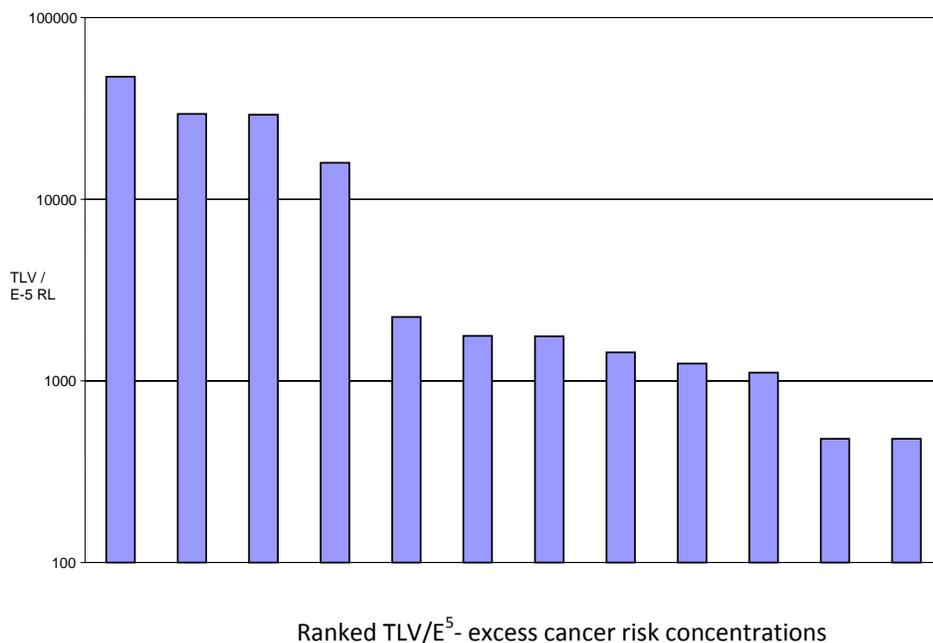


Figure 2-23. Distribution of quotients of TLVs to $1E^{-5}$ excess cancer risk concentrations.

The summary statistics of the distribution of quotients are: Median 1764; Mean 11,019; Low 480; High 47,190; N = 12. We used the median of the distribution as a factor by which to divide those TLV-TWAs listed as carcinogenic, that did not have matching RBCs from preferred sources. The median; high and low of the resulting quotients range were determined and then substituted into equation 2-7 to get estimates of the concentration that might be associated with an 10^{-5} excess cancer risk.

$$0.1 \oint TLV \cong 1/1E^{-5} \text{ Risk level estimate} \qquad \text{Eq. 2-7}$$

Where TLV is the TLV-TWA for an air toxic without a matching preferred cancer RBC.

In order to derive scaling factors for TLV-TWAs for toxic air pollutants that exhibit non-cancer toxicity, we examined the distribution of quotients from TLV-TWAs divided by RfCs or other preferred RfC-like values. The results are shown in figure 2-24.

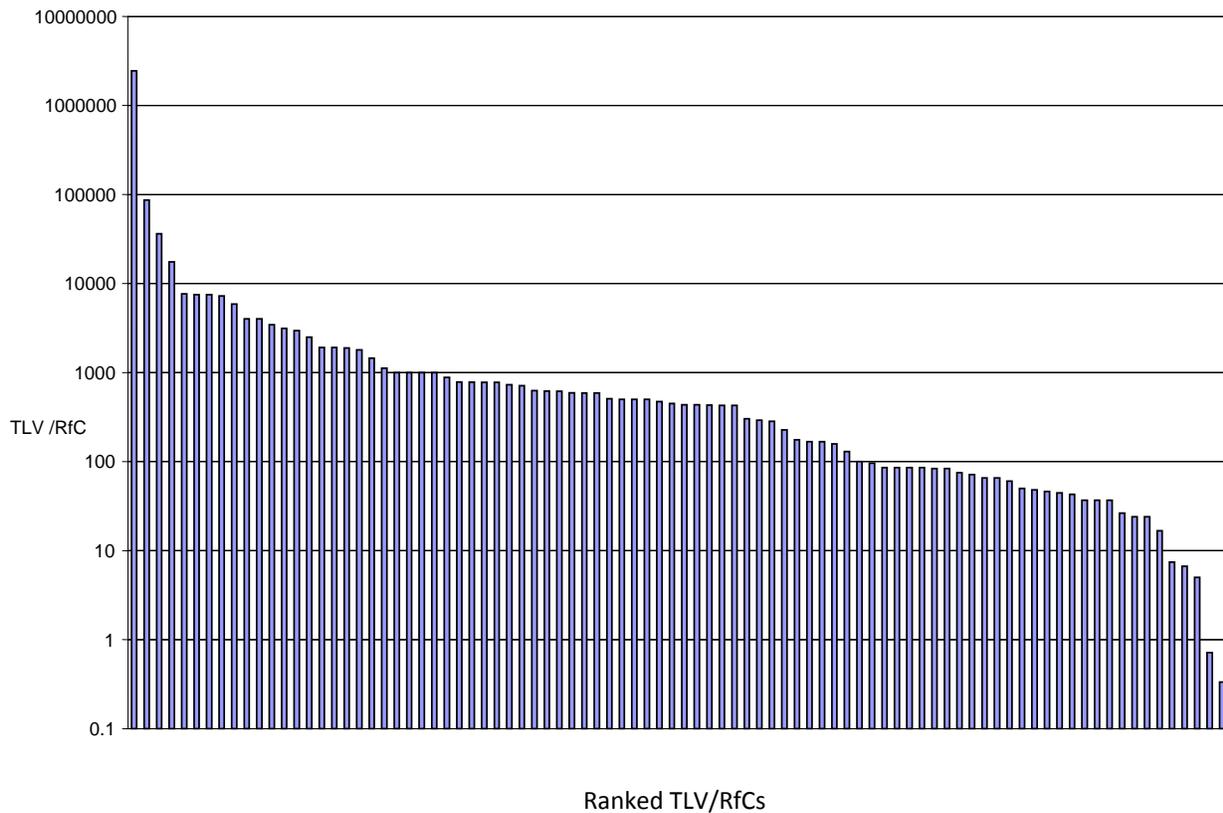


Figure 2-24. Distribution of quotients of TLVs to RfCs or other preferred RfC-like values.

The summary statistics of the distribution of quotients are: Median 441; Mean 30,509; Lowest 0.33; Highest 2,457,055; N = 88. The median of the distribution was used as a factor by which to divide non-cancer TLVs without matching toxic air pollutant criteria from preferred sources.

$$\frac{\text{TLV}}{\text{Median}} \cong \text{RfC-like value} \quad \text{Eq. 2-8}$$

Where TLV is the TLV-TWA for an air toxic without a matching preferred RfC-like value.

Oral exposure route RBCs

A few of the toxic air pollutants had oral-route UREs, but did not have inhalation-route UREs. For these, an inhalation concentration roughly equivalent to the oral-route exposure cancer RBC was estimated using the slope factor. We converted RfDs to RfCs assuming standard adult body weight and inhalation rate variables using the following algorithm.

$$\begin{aligned} & \text{RfD (mg/kg/day)} \times 70 \text{ kg} \times 20 \text{ m}^3/\text{day} \times 100\% \text{ Absorption Factor} \\ & \cong \text{RfC-like value (mg/m}^3\text{)} \quad \text{Eq. 2-9} \end{aligned}$$

National Ambient Air Quality Standard for lead

Finally, for lead (Pb) the National Ambient Air Quality Standard was used. All of the RBCs we used in this effort, along with their derivations (if any), are detailed for each toxic air pollutant in appendix A.

Priority toxic air pollutants risk-based concentrations

The RBCs used for the toxic air pollutants we identified as being significant priorities — the Priority Toxic air pollutants — are detailed below.

Diesel Particulate Matter Emissions.

The toxicity of DPM is determined by the particle size and composition. DPM consists of a solid core composed mainly of carbon, a soluble organic fraction (14~35 carbon alkanes, alkyl-substituted benzenes, PAH derivatives), sulfates, and trace elements. Some of the carcinogenicity of DPM may be due to the nitro-PAHs it contains. Biochemical and cellular assays have demonstrated DPM mutagenicity. Exposure to DPM in controlled laboratory animal studies has demonstrated its carcinogenicity. Further, epidemiological evidence among occupationally exposed people, although lacking in well quantified exposure levels, suggests diesel exhaust may cause lung and bladder cancer. In the *Health Assessment Document for Diesel Engine Exhaust (HAD)* the USEPA ORD states that diesel exhaust is a probable human carcinogen; they have stated the possible range of upper-bound risk is $1E^{-3}$ to $1E^{-5}$ per $\mu\text{g}/\text{m}^3$ for lifetime exposure,³⁹ but have not promulgated a specific URE.

The HAD reviews numerous epidemiologic studies and concludes that many have shown increased lung cancer risks among workers in certain occupations. The relative risks or odds ratios in this systematic review ranged from 1.2 to 2.6. The HAD also notes two independent meta-analyses that show smoking-adjusted relative risk increases of 1.35 and 1.47. Taking this information together, the EPA analysts selected a relative risk of 1.4 as a reasonable estimate of risk in these DE-exposed workers, which is equivalent to an additional excess lifetime lung cancer risk of 2% beyond the risk among the whole U.S population. The HAD summarized the estimated possible unit risk ranges (10^{-5} to 10^{-3} per $\mu\text{g}/\text{m}^3$ “as well as lower and zero risk”) to provide a perspective of the potential significance of the lung cancer hazard. It went on to state

“Lower risks are possible and one cannot rule out zero risk. The risks could be zero because (a) some individuals within the population may have a high tolerance to exposure from DE and therefore not be susceptible to the cancer risk from environmental exposure, and (b) although evidence of this has not been seen, there could be a threshold of exposure below which there is no cancer risk.”

³⁹ *Health Assessment Document for Diesel Engine Exhaust*. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, Washington, DC, EPA/600/8-90/057F, 2002. Available at <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060>

In ranking toxic air pollutants, the AQP is concerned with population-wide effects as opposed to individuals within the population may have a high tolerance some carcinogens. All available evidence indicates DPM is carcinogenic and we are not aware of evidence of an exposure threshold for DPM below which there is no cancer risk. For these reasons we consider the HAD's limited suggestion of zero risk to be unrealistic and irrelevant in the context of this toxic air pollutant ranking process.

The IARC has also evaluated diesel exhaust concluding it is probably carcinogenic to humans (Group 2A) because there is sufficient evidence for carcinogenicity in experimental animals of whole diesel engine exhaust, and there is sufficient evidence for the carcinogenicity in experimental animals of extracts of diesel engine exhaust particles. Also there is limited evidence for carcinogenicity in humans of diesel engine exhaust, and there is limited evidence for the carcinogenicity in humans of engine exhausts (unspecified as from diesel or gasoline engines). The California Air Resources Board Scientific Review Panel estimates the unit risk as $3E^{-4}$ per $\mu\text{g}/\text{m}^3$ (range $1.3E^{-4}$ to $2.4E^{-3}$ per $\mu\text{g}/\text{m}^3$). Although this URE is still controversial, the specific issues in question have been addressed in detail by CARB and OEHHA⁴⁰. In addition to carcinogenicity, DPM contributes to $\text{PM}_{2.5}$ levels and has a demonstrated potential to induce of pulmonary inflammation and histopathology. Both the USEPA RfC and the California OEHHA REL for DPM are $0.005\text{-mg}/\text{m}^3$. The AQP is currently preparing a separate evaluation of DPM carcinogenic potency and non-cancer toxicity. It will detail the basis for the AQP's use of the OEHHA's DPM cancer potency unit risk, which we used in the current TAP ranking effort.

Wood Smoke

None of the authorities cited in this report have established toxicity criteria for wood smoke; however, it contains known and suspected carcinogens. In fact, both human epidemiological investigations and experimental animal research have indicated wood smoke is carcinogenic, and IARC has stated there is sufficient evidence to conclude wood smoke is probably carcinogenic to

⁴⁰ Part B: *Health Risk Assessment for Diesel Exhaust of the Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*. Published by the California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, in May 1998. Available at <http://www.arb.ca.gov/regact/diesltac/diesltac.htm>.

humans (placing it in group 2A)⁴¹. To rank wood smoke with the other potentially carcinogenic air pollutants, we applied unit risk estimates developed by Lewtas and by Anderson. Lewtas (1988) proposed a cancer URE of $1.0E^{-5}$ per $\mu\text{g}/\text{m}^3$.⁴² Anderson (1989) derived a residential wood smoke URE of $3.0E^{-5}$ per $\mu\text{g}/\text{m}^3$.⁴³

Benzene

USEPA has designated benzene a known (Class A) human carcinogen based on convincing human evidence as well as supporting evidence from animal studies. The excess cancer risk level inhalation concentration listed in IRIS is a range of $2.2E^{-6}$ to $7.8E^{-6}$ is the increase in the lifetime risk of an individual who is exposed for a lifetime to $1\text{-}\mu\text{g}/\text{m}^3$ in air (a range from 0.0013 to $0.0045\text{-mg}/\text{m}^3$). The more conservative limit ($1.3E^{-3}\text{-mg}/\text{m}^3$) was used for cancer potency-weighting the emission inventory in this report. The RfC and REL are based on critical impairment effects on hematopoiesis; the nervous system; and development. The USEPA listed RfC of $0.03\text{-mg}/\text{m}^3$ was used for toxicity-weighting the EI.

Carbon Tetrachloride

USEPA designates CCl_4 a probable (Class B2) human carcinogen, based on observed carcinogenicity in rats, mice, and hamsters. However, there is inadequate evidence for its carcinogenicity in humans. The IRIS inhalation $1E^{-5}$ cancer risk level concentration is $6.7E^{-4}\text{-mg}/\text{m}^3$. The USEPA has not established an RfC; however, the OEHHA chronic REL is $0.04\text{-mg}/\text{m}^3$.

Formaldehyde

USEPA has designated formaldehyde a probable (B1) human carcinogen, based on limited

⁴¹ Straif, K ; Baan, R ; Grosse, Y; Secretan, B; El Ghissassi, F; Coglianò, V; International Agency for Research on Cancer Monograph Working Group. 2006. Carcinogenicity of household solid fuel combustion and of high-temperature frying. *The Lancet Oncology*, Vol. 7, Iss. 12, pp 977 – 978. Available at <http://oncology.thelancet.com>.

⁴² Lewtas, J. 1988. Genotoxicity of complex mixtures: Strategies for the identification and comparative assessment of airborne mutagens and carcinogens from combustion sources. *Fund. & Appl. Tox.* 10:571-589

⁴³ Anderson, N. Final Report: Risk assessment document for residential wood combustion emissions. Maine Department of Health Services, Environmental Toxicology Program, Environmental Health Unit, Division of Diseases Control, Bureau of Health. October 1989.

evidence in humans, and sufficient evidence in animals (the International Agency for Research on Cancer lists formaldehyde as a human carcinogen, too). Human data include nine studies that show statistically significant associations between site-specific respiratory neoplasms and exposure to formaldehyde or formaldehyde-containing products. An increased incidence of nasal squamous cell carcinomas was observed in long-term inhalation studies in rats and in mice. The designation is supported by *in vitro* genotoxicity data and formaldehyde's structural relationships to other carcinogenic aldehydes such as acetaldehyde. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $8E^{-4}$ -mg/m³ used for toxicity-weighting the emission inventory. The OEHHA lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $1.67E^{-3}$ -mg/m³. USEPA has not issued an RfC; however, the ATSDR chronic inhalation MRL is 0.0098-mg/m³. It was used in preference to the OEHHA REL, which is $3E^{-3}$ -mg/m³. MRL and REL are based on critical effects on the eye and on respiratory irritation.

Polycyclic Organic Matter

Lung cancers are the critical effect possible from exposure to POM.⁴⁴ The OEHHA URE for total-POM when expressed as the equivalent $1E^{-5}$ risk level concentration is $1.8E^{-4}$ -mg/m³. No RfC or similar non-cancer criterion for POM was available. In some cases, the emissions inventory lists specific POM chemicals and subset mixtures separately from the larger POM group. The POM group contains the polycyclic aromatic hydrocarbons (PAHs), also known as polynuclear aromatics. The OEHHA URE for total-PAH, when expressed as the equivalent $1E^{-5}$ risk level concentration, is $1.8E^{-4}$ -mg/m³. No RfC or similar non-cancer criterion for total-PAH was available. USEPA has classified the 7-PAHs (benzo[a]pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene) as probable (B2) human carcinogens. For the 7-PAHs together, the USEPA provides a URE equivalent to $3E^{-5}$ mg/m³ at the $1E^{-5}$ excess cancer risk level. Examining the 7-PAHs separately, USEPA designated benz[a]anthracene a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate however; OEHHA lists an inhalation URE equivalent to $9.1E^{-5}$ -mg/m³ at the $1E^{-5}$ excess cancer risk level. USEPA designated benzo[a]pyrene a probable (B2) human carcinogen based on sufficient evidence of

⁴⁴ USEPA. Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter. EPA-454/R-98-014. Office of Air Quality Planning and Standards, Research Triangle Park, NC. 1998.

carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate, however OEHHA lists an inhalation URE equivalent to $9.1E^{-6}$ - mg/m^3 at the $1E^{-5}$ excess cancer risk level. In some cases, the emission inventory reported benzo[b]fluoranthene and benzo[k]fluoranthene together as "benzofluoranthenes" or "benzo[b+k]fluoranthene" We assumed these mixtures have the same toxic potency as either benzo[b]fluoranthene or benzo[k]fluoranthene alone. USEPA has designated benzo(b)fluoranthene a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate, however OEHHA lists an inhalation URE equivalent to $9.1E^{-5}$ - mg/m^3 at the $1E^{-5}$ excess cancer risk level. USEPA has designated benzo[k]fluoranthene a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate, however OEHHA lists an inhalation URE equivalent to $9.1E^{-5}$ - mg/m^3 at the $1E^{-5}$ excess cancer risk level. USEPA has designated chrysene a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate, however OEHHA lists an inhalation URE equivalent to $9.1E^{-4}$ - mg/m^3 at the $1E^{-5}$ excess cancer risk level. USEPA has designated dibenzo[a,h]anthracene — synonymous with dibenz(a,h)anthracene — a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate for dibenzo[a,h]anthracene, however OEHHA lists an inhalation URE equivalent to $8.3E^{-6}$ - mg/m^3 at the $1E^{-5}$ excess cancer risk level. USEPA has designated indeno[1,2,3-cd]pyrene a probable (B2) human carcinogen based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate, however OEHHA lists an inhalation URE equivalent to $9.1E^{-5}$ - mg/m^3 at the $1E^{-5}$ excess cancer risk level. No RfC or similar non-cancer criterion is currently available for the 7-PAHs group or any of its individual chemicals.

In some instances, POM chemicals were listed in the emissions inventory under the heading "Polycyclic Organic Matter as 16-PAH". These 16-PAHs include acenaphthene, acenaphthylene, anthracene, benzo[ghi]perylene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene and each of the "carcinogenic 7-PAHs". The NATA estimates of toxic

equivalence to BaP of 16-PAH emitted from different sources are for 16-PAH from residential wood burning, 3.57% BAP equivalents; for aluminum smelting, 5.14%; and for wildfires and utility emissions, 6.70%.⁴⁵ Based on these, the average BaP_{eq}/16-PAH is 5.53%, which is equivalent to $1.6E^{-4}$ -mg/m³ at the $1E^{-5}$ excess cancer risk level.

A summary of the toxicity criteria of each of the nine PAHs in the 16-PAH group not yet mentioned (the 16 PAHs excluding the 7-PAH subset) is as follows: USEPA designated naphthalene as a possible (Class C) human carcinogen based on the inadequate data of carcinogenicity in humans exposed via the oral and inhalation routes, and limited evidence of carcinogenicity in animals via inhalation. No quantitative cancer potency estimates have been published by any of the sources referenced in this report. USEPA lists an RfC of $3E^{-3}$ -mg/m³ of naphthalene in IRIS. Due to a lack of data, USEPA has not published carcinogenicity or inhalation RfC assessments for most of the 16-PAHs that are not part of the 7-PAH group, however, they have issued RfDs (in units of mg/kg/day) for most of these.

We estimated long-term RfCs (in units of mg/m³) by transforming the available RfDs under exposure assumptions of a 70-kg adult breathing 20 m³/d, with a factor of 0.2 to account for variations in the average daily concentration. This yielded the following RfC estimates: for acenaphthene, 0.042-mg/m³; anthracene, 0.21-mg/m³; fluoranthene, 0.028-mg/m³; fluorene, 0.028-mg/m³; and pyrene, 0.021-mg/m³. USEPA and the other authorities referenced in this report have not published carcinogenicity, RfC-like, or RfD-like assessments for acenaphthylene, benzo[g,h,i]perylene, or phenanthrene due to a lack of toxicity data.

Chromium

USEPA has designated hexavalent chromium - Cr(VI) - a known (Class A) human carcinogen based on convincing human evidence by the inhalation route of exposure as well as supporting evidence from animal studies. Its IRIS cancer URE is equivalent to $8.3E^{-7}$ -mg/m³ at the $1E^{-5}$ excess risk level exposure. IRIS states “Hexavalent chromium is known to be carcinogenic in humans. Results of occupational epidemiological studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been

⁴⁵ See Appendix H, Estimating Carcinogenic Potency for Mixtures of Polycyclic Organic Matter for the 1996 National-Scale Assessment available at <http://www.epa.gov/ttn/atw/sab/appendix-h.pdf>

established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both Cr(III) and Cr(VI) compounds. Because only Cr(VI) has been found to be carcinogenic in animal studies; however, it was concluded that only Cr(VI) should be classified as a human carcinogen.” The IRIS RfC for chromic acid mists and dissolved Cr(VI) aerosols: is $8E^{-6}$ -mg/m³. The IRIS RfC for Cr(VI) particulates is $1E^{-4}$ -mg/m³. In NATA, USEPA used the IRIS RfC for particulate hexavalent chromium in preference to the RfC for chromic acid mists and dissolved aerosols. As in NATA, both the URE and the RfC for hexavalent chromium were adjusted to reflect an assumption that 34% of all atmospheric chromium is hexavalent: The remaining 66% assumed to be trivalent, which USEPA has designated not classifiable as to its human carcinogenicity (Class D) due to insufficient data. None of the other authorities referenced in this report have published carcinogenicity assessments of Cr(III) either. The ACGIH TWA-TLV was used to estimate an average RfC of $2.3E^{-5}$ -mg/m³ for Cr(III). Calcium, strontium and zinc chromates were reported in the emissions inventory. These were toxicity-weighted using OEHHA chronic REL criteria (none of these have been specifically assessed by USEPA). The OEHHA chronic REL for Strontium chromate is $2E^{-4}$ -mg/m³. Zinc chromate toxicity has not been specifically assessed by USEPA or any of the authorities referenced in this report.

Selenium

USEPA designates selenium and compounds as not classifiable as to carcinogenicity (Class D) based on inadequate human data and inadequate evidence of carcinogenicity in animals. The only selenium compound that has been shown to be carcinogenic in animals is selenium monosulfide, to which oral exposure has resulted in an increase in liver tumors. USEPA notes that evidence for selenium sulfide is sufficient for a probable human carcinogen (B2) classification, but they have not provided a quantitative potency estimate. However, the California Air Pollution Control Officers Association (CAPCOA) recommended a preliminary selenium sulfide cancer URE of $1.4E^{-4}$ /μg/m³. This is equivalent to $7.14E^{-5}$ -mg/m³ at the $1E^{-5}$ excess cancer risk level. Likewise, the USEPA has not established an RfC for selenium and compounds; however, the CAPCOA *Revised 1992 Risk Assessment Guidelines* for selenium lists a chronic REL of $5E^{-4}$ -mg/m³ for selenium compounds, based on respiratory irritation.⁴⁶

⁴⁶ CAPCOA, 1993. The CAPCOA Air Toxics “Hot Spots” Program; Revised 1992; Risk Assessment Guidelines, California Air Pollution Control Officer’s Association, October 1993.

Chloroform

USEPA has designated chloroform a probable (B2) human carcinogen, because it is likely to be carcinogenic to humans by all routes of exposure under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissues. It is not likely to be carcinogenic to humans by any route of exposure under exposure conditions that do not cause cytotoxicity and cell regeneration. The IRIS inhalation $1E^{-5}$ cancer risk-based concentration is $4E^{-4}$ -mg/m³. The USEPA has not established an RfC; however, the ATSDR chronic inhalation MRL is 0.098-mg/m³.

1,3-Butadiene

USEPA has designated 1,3-butadiene a known (Class A) human carcinogen based on sufficient evidence from epidemiological studies of the majority of U.S. workers occupationally exposed to either the monomer or polymer by inhalation, showing increased lymphohematopoietic cancers and a dose-response relationship for leukemias in polymer workers; sufficient evidence in laboratory studies showing tumors at multiple sites in mice and rats by inhalation; and numerous studies consistently demonstrating that 1,3-butadiene is metabolized into genotoxic metabolites by experimental animals and humans. The IRIS inhalation $1E^{-5}$ cancer risk-based concentration is $3E^{-4}$ -mg/m³. The RfC and REL are based on critical effects on the reproductive system (ovarian atrophy). The RfC was used for toxicity-weighting the emission inventory.

Ethylene Dichloride

The USEPA has classified ethylene dichloride as a probable (B2) human carcinogen based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $4E^{-4}$ -mg/m³. The OEHHA URE is $2.1E^{-5}$ /μg/m³. The USEPA has not published an RfD or RfC; however, the ATSDR chronic inhalation MRL is 2.4-mg/m³. It was used in preference to the OEHHA REL (0.4-mg/m³).

Ethylene Dibromide

The USEPA has classified EDB as a probable (B2) human carcinogen based on increased incidences of a variety of tumors in rats and mice in both sexes by three routes of administration at both the site of application and at distant sites. EDB is mutagenic in various *in vitro* and *in*

vivo assays. EDB is structurally similar to DBCP and to ethylene dichloride, both of which are probable human carcinogens. IRIS lists an $1E^{-5}$ excess cancer risk inhalation exposure level of $5E^{-5}$ -mg/m³. The USEPA has not published an RfD or RfC; however, the OEHHA REL is $8E^{-4}$ -mg/m³.

Acetaldehyde

USEPA has designated acetaldehyde a probable (B2) human carcinogen, based on increased incidence of nasal tumors in male and female rats and laryngeal tumors in male and female hamsters after inhalation. The IRIS inhalation $1E^{-5}$ cancer risk level concentration is listed as 0.005-mg/m³. The IRIS RfC is 0.009-mg/m³.

Tetrachloroethylene (Perchloroethylene)

No carcinogenicity assessment of tetrachloroethylene is available from USEPA; however, the IARC lists it as probably (Group 2A) a human carcinogen: based on equivocal evidence in animals (some induction of peroxisome proliferation in mouse liver; mutations in proto-oncogenes in liver tumors from mice treated with tetrachloroethylene; and induction of leukemia in rats) and several human epidemiological studies showing elevated risks for esophageal cancer, non-Hodgkin's lymphoma and cervical cancer.⁴⁷ The OEHHA has issued an inhalation URE for tetrachloroethylene of $5.9E^{-6}$ /μg/m³, which is equivalent to a $1E^{-5}$ excess cancer risk level concentration of $1.7E^{-3}$ -mg/m³. Its ATSDR chronic inhalation MRL is 0.3-mg/m³.

Trichloroethylene

TCE has been shown cause cancer in mice, and is suspected to cause cancer in humans. According to the IARC, trichloroethylene is probably carcinogenic to humans (Group 2A) based on limited evidence in humans but sufficient evidence in experimental animals. Several epidemiological studies have shown elevated risks for cancer of the liver and biliary tract and for non-Hodgkin's lymphoma. Formation of mouse liver tumors with peroxisome proliferation is plausible; and trichloroethylene has induced tumors at other sites in mice and rats.⁴⁸ No carcinogenicity assessment of trichloroethylene is available from the USEPA; however, the

⁴⁷ <http://www-cie.iarc.fr/htdocs/monographs/vol63/tetrachloroethylene.htm>

⁴⁸ <http://www-cie.iarc.fr/htdocs/monographs/vol63/trichloroethylene.htm>

OEHHA issued an inhalation URE of $2E^{-6}/\mu\text{g}/\text{m}^3$, which is equivalent to a $1E^{-5}$ excess cancer risk level concentration of $5E^{-3}\text{-mg}/\text{m}^3$. USEPA has not published an RfC; however, the OEHHA chronic inhalation REL is $0.6\text{-mg}/\text{m}^3$. The OEHHA values were used for toxicity-weighting the emission inventory.

Nickel

USEPA designates nickel refinery dust and nickel subsulfide as known (Class A) human carcinogens based on the observed increases in lung and nasal cancer in humans exposed to nickel refinery dust, most of which was believed to have been nickel subsulfide; also increased tumor incidences in animals by several routes of administration in several animal species and strains; and positive results in genotoxicity assays. Nickel refinery dust and nickel subsulfide are listed in IRIS as posing a $1E^{-5}$ excess cancer risk at a concentration of $4E^{-5}\text{-mg}/\text{m}^3$ (similar to the OEHHA URE), which is equivalent to a $1E^{-5}$ risk level concentration of $3.9E^{-5}\text{-mg}/\text{m}^3$. The USEPA classifies nickel carbonyl a probable (B2) human carcinogen based on the observation of pulmonary carcinomas and malignant tumors at various sites in rats after inhalation or intravenous injection. Administered nickel carbonyl binds nickel to DNA. However USEPA concluded that these data are not sufficient to derive an inhalation unit risk. USEPA has not issued RfDs for any form of nickel; however, the ATSDR chronic inhalation MRL for all forms is $2E^{-4}\text{-mg}/\text{m}^3$.

Arsenic

The USEPA has designated inorganic arsenic a known (Class A) human carcinogen, based on sufficient evidence from human data. Increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. The IRIS inhalation exposure $1E^{-5}$ excess cancer risk level of inorganic arsenic is $2E^{-6}\text{-mg}/\text{m}^3$. USEPA has not assessed non-cancer effects of inhaled inorganic arsenic. The RfD-based RfC estimate for inorganic arsenic is $2.1E^{-4}\text{-mg}/\text{m}^3$. No assessment or quantitative cancer risk estimate has been published for arsine. The IRIS RfC for arsine is $5E^{-5}\text{-mg}/\text{m}^3$.

1,4-Dichlorobenzene

The USEPA has not completed an evaluation of 1,4-dichlorobenzene for evidence of human carcinogenic potential; however, OEHHA published a URE of $1.1E^{-5}/\mu\text{g}/\text{m}^3$, which is equivalent

to $9.1E^{-4}$ -mg/m³ at the $1E^{-5}$ excess cancer risk exposure level. The IARC states that para-dichlorobenzene is possibly carcinogenic to humans (Group 2B). An IARC Working Group noted supporting evidence that its mechanism of carcinogenesis may be relevant for humans because of evidence of DNA damage in liver and spleen of mice, and weak binding to DNA in mouse liver.⁴⁹ The USEPA lists its RfC as 0.8-mg/m³.

1,3-Dichloropropene

USEPA lists 1,3-dichloropropene as a probable (B2) human carcinogen because of sufficient evidence of carcinogenicity in animals but the lack of data in humans. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $2E^{-3}$ -mg/m³. The IRIS RfC is 0.02-mg/m³.

Ethylene Oxide

No carcinogenicity assessment for ethylene oxide is currently available from the USEPA. The IARC has stated that ethylene oxide is carcinogenic to humans (Group 1) because there is limited evidence of its carcinogenicity in humans, and because there is sufficient evidence for carcinogenicity in experimental animals. Ethylene oxide is an alkylating agent that: induces chromosomal aberrations in peripheral lymphocytes and micronuclei in bone-marrow cells of exposed workers; has been associated with malignancies of the lymphatic and haematopoietic systems in both humans and experimental animals; increases in the frequency of haemoglobin adducts in exposed humans and dose-related increases in adducts in both DNA and haemoglobin in exposed rodents; induces gene mutations and heritable translocations in germ cells of exposed rodents; and is a powerful mutagen and clastogen at all phylogenetic levels.⁵⁰ The OEHHA cancer potency estimate ($8.8E^{-5}$ /μg/m³) is equivalent to $1.1E^{-4}$ -mg/m³ at the $1E^{-5}$ excess risk level concentration. The USEPA has not published an RfC; however, the OEHHA chronic REL is 0.03-mg/m³.

Acrolein

USEPA and the other authorities cited in this report have not classified acrolein according to its carcinogenic potential. The IARC evaluation of acrolein states it is not classifiable as to its

⁴⁹ <http://www-cie.iarc.fr/htdocs/monographs/vol73/73-08.html>

⁵⁰ <http://www-cie.iarc.fr/htdocs/monographs/vol60/m60-02.htm>

carcinogenicity to humans (Group 3) because there is inadequate evidence of carcinogenicity in humans and experimental animals. The toxicological endpoints considered for acute and chronic toxicity are histopathological changes in the nasal cavity, lung, larynx, and trachea.⁵¹ Inhalation exposure to acrolein at higher existing ambient concentrations may cause irritation of the eyes, nose, throat, and respiratory tract,⁵² and possibly inflammation within airways,⁵³ which could be is a specific threat to people with asthma or chronic obstructive pulmonary disease (COPD).⁵⁴

Limitations and uncertainties of risk-based concentrations

Inherent uncertainty in UREs and RfCs

The RBCs of many toxic air pollutants are based on data from studies using animals exposed to relatively high concentrations for limited amounts of time under controlled conditions, that bear limited resemblance to those experienced by humans exposed to these chemicals. Both the cancer risk level and non-cancer RBCs are based on a combination of average and reasonably conservative (health-protective) assumptions. Our use of these data allowed us to screen and sort as many toxic air pollutants as possible. However, our confidence in estimates of how many people are actually adversely affected by the toxic air pollutants we assessed is currently quite limited.

Chemical mixture composition and toxic potency

In addition to numerous specific chemical species and compounds, the Federal list of 187 HAPs includes 17 compound groups (e.g., POM, glycol ethers, nickel and its compounds, etc.). A limitation encountered when hazard-weighting emissions inventories was the lack of information on grouped metals and organic toxic air pollutants. In emissions reported as groups or mixture, there are various individual compounds that have substantially different toxicity characteristics

⁵¹ USEPA IRIS

⁵² Toxic Air Contaminant Identification. List Summaries. Acrolein - ARB/SSD/SES. September 1997.

⁵³ Miller, LA. 1997. Effects of tobacco on inflammatory cell responses. Tobacco-Related Disease Research Program Grant #: 6KT-0411 University of California, Davis.

⁵⁴ The USEPA has established a Reference Concentration (RfC) of 0.02 $\mu\text{g}/\text{m}^3$ for acrolein. They estimate that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-cancer effects.

relative to the others. We made generalizations about these groups we following guidance on the toxicity of pollutants reported as mixtures in the NATA. For example, with chromium compounds, the IRIS RfC for particulate hexavalent chromium was used in preference to the RfC for chromic acid mists and dissolved aerosols. Both the RfC and cancer risk estimate for hexavalent chromium were adjusted to reflect an assumption that 34% of all atmospheric chromium is hexavalent.⁵⁵ Another example comes from the compounds within the POM group: Some are relatively non-toxic, while others are highly potent carcinogens.⁵⁶ Assumed potencies for mixtures of POM were based on the known or estimated potency of each mixture component relative to that of benzo[a]pyrene, according to the Toxicity Equivalency Factor (TEF) approach. As shown appendix A, we applied TEFs for certain POMs to a URE for benzo[a]pyrene, which was given by the OEHHA. Further details and assumptions about each mixture group's toxicity are noted in appendix A.

Intermediate exposure duration MRL conversion uncertainty

ATSDR promulgated intermediate MRLs for exposure durations lasting 15 to 364 days. We used these intermediate inhalation exposure MRLs reduced by 80% to weight the emissions of a few toxic air pollutants. Since the toxicity data used to derive intermediate MRLs is from less than life-long exposure-response studies it may not be appropriate to assume chronic exposure outcomes, even using a reduction factor as we did. More toxicity studies of the toxic air pollutants involved are needed in order to evaluate this question.

TLV-TWA conversion uncertainty

An ACGIH Threshold Limit Value–Time Weighted Average is an estimate of the concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed day after day, without adverse effects. ACGIH states “These limits are intended for use in the practice of industrial hygiene as guidelines or recommendation in the control of potential health hazards and for no other use, e.g., in evaluation or control of community air pollution nuisances, in estimating the toxic potential of continuous uninterrupted exposures...” Therefore, using TLV-TWAs to derive air quality criteria is inconsistent with the

⁵⁵ USEPA, 2001. Appendix G of the 1996 NATA

⁵⁶ <http://www.epa.gov/ttnchie1/conference/ei11/toxics/pope.pdf>

use intended by ACGIH. It is important to distinguish this inappropriate usage from the way we used this information: Our goal was to make large hazard-weighted toxic air pollutants inventories, not to develop air quality criteria. Nevertheless, there is a great deal of uncertainty in the practice of weighting emissions inventories with TLV-TWAs. The huge variations in the scaling factor ranges shown in figures 2-23 and 2-24 add to the level of uncertainty in our TLV-TWA standardization procedures. Due to these uncertainties and limitations, using the TLV-TWAs in this manner may have resulted in inappropriate exclusion one or more toxic air pollutants from our list of priority toxic air pollutants.

Oral exposure route RBC uncertainty

We converted ingested dose quantities to inhalation concentrations in order to weight the emissions of some of the toxic air pollutants. In such cases, the cancer potency data and/or non-cancer toxicity data for these toxic air pollutants were not extensive enough to directly develop inhalation RBCs. In order to complete the screening of as many toxic air pollutants as possible, we were forced to make these conversions with the assumption that inhalation exposure-responses would be quantitatively the same as oral ingestion exposure-responses. We do not have specific information supporting or refuting this assumption for any of the toxic air pollutants involved, however.

Hazard-Weighted Emissions Inventories Results

In this section, the statewide and region-by-region hazard-weighted air pollutant emissions inventory ranking results are shown. The ranked hazard-weighted EI of each air toxic is proportional to the estimated relative magnitude of its public health risk. The figures below show the proportional quantity of each as a percentage of the sum of all the hazard-weighted toxic air pollutants emissions.

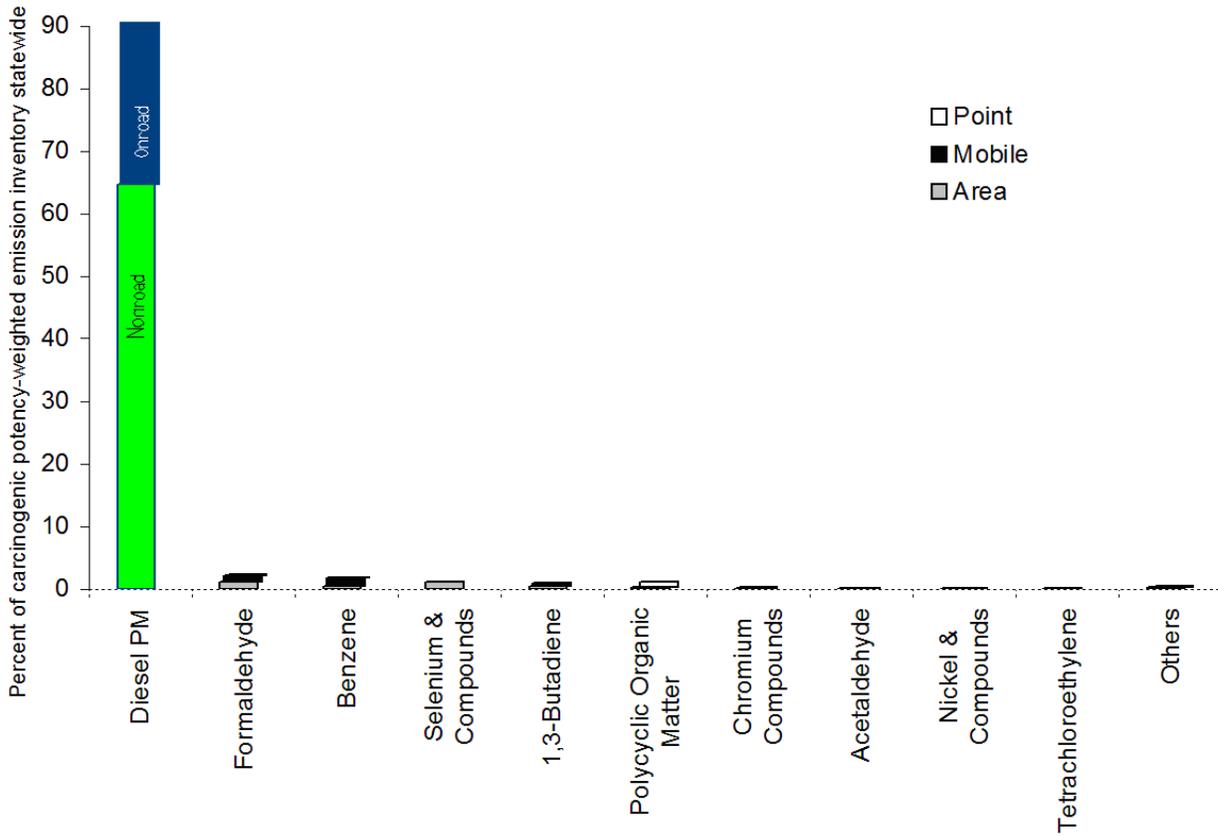


Figure 2-25. Statewide carcinogenic potency weighted-emissions inventory.

All DPM emissions may be regarded as originating from mobile sources in either on-road or non-road categories; However, in the following figures, non-road mobile diesel PM₁₀ emissions are shown as area emissions, and on-road emissions are shown as “mobile” sources.

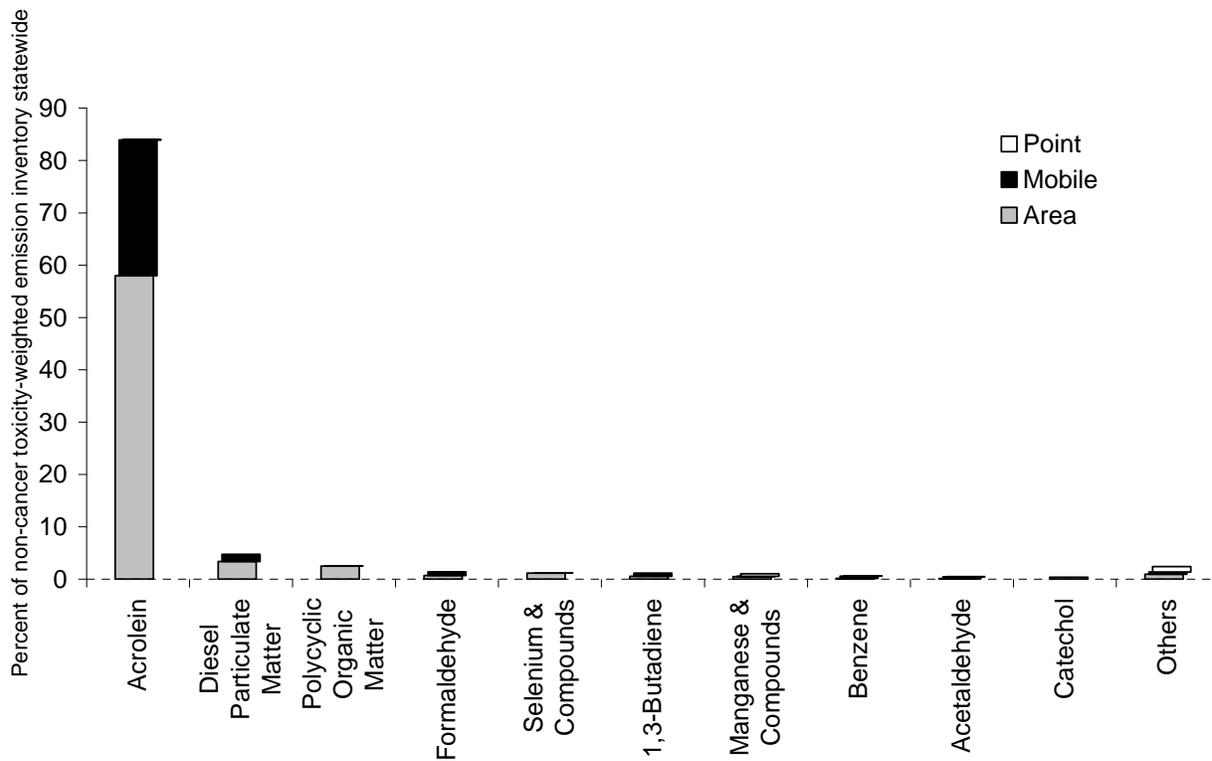


Figure 2-26. Statewide non-cancer toxicity weighted-emissions inventory.

As shown, statewide, diesel PM is the leading carcinogen, and acrolein is the leading non-carcinogen. We also prepared hazard-weighted emission inventories for Washington’s seven Local Air Agencies and Ecology’s three Regional Offices. When considering the results it is important to note that (1) the reporting efficiency may be better in some LAA jurisdictions⁵⁷; and (2) some of the regions are small, especially in Benton, Spokane and Yakima counties, each of which have LAAs unto themselves. The accuracy of toxic air pollutants rankings in these one-county LAA regions, are less definite than those in larger regions. The rankings of the top scoring toxic air pollutants in each region are summarized in the figures below.

⁵⁷ See Section 3 of this report for a discussion of the NATA.

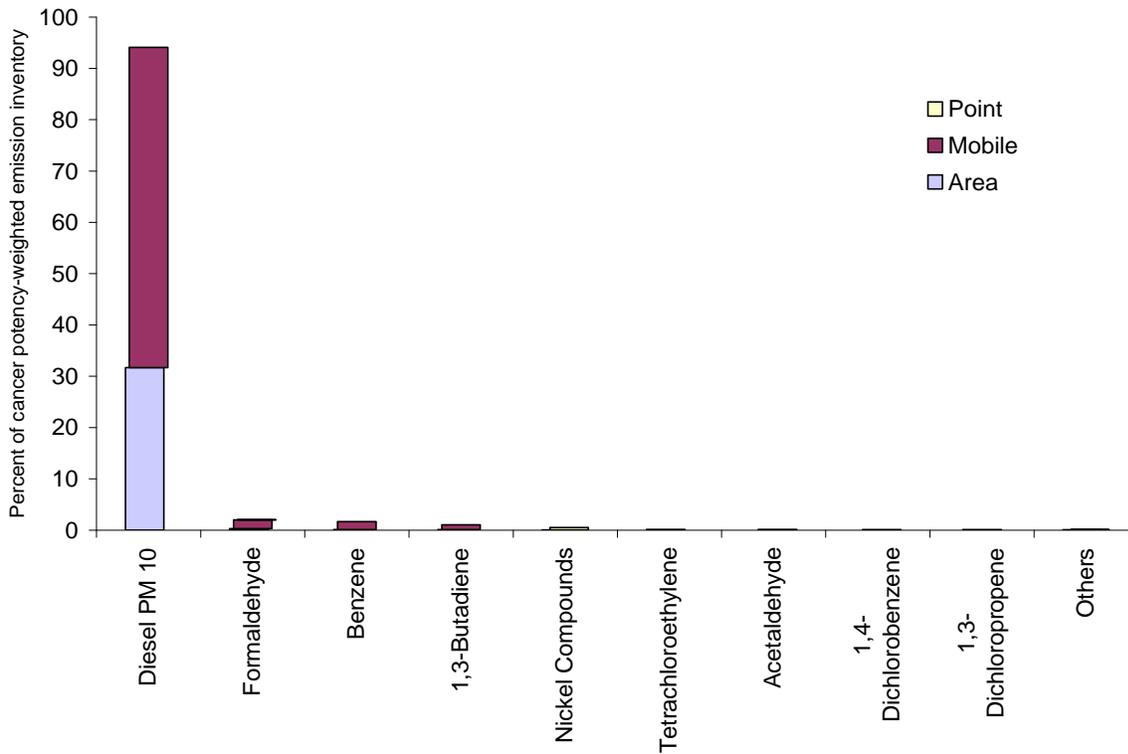


Figure 2-27. Benton County Air Agency carcinogenic potency weighted-emissions inventory.

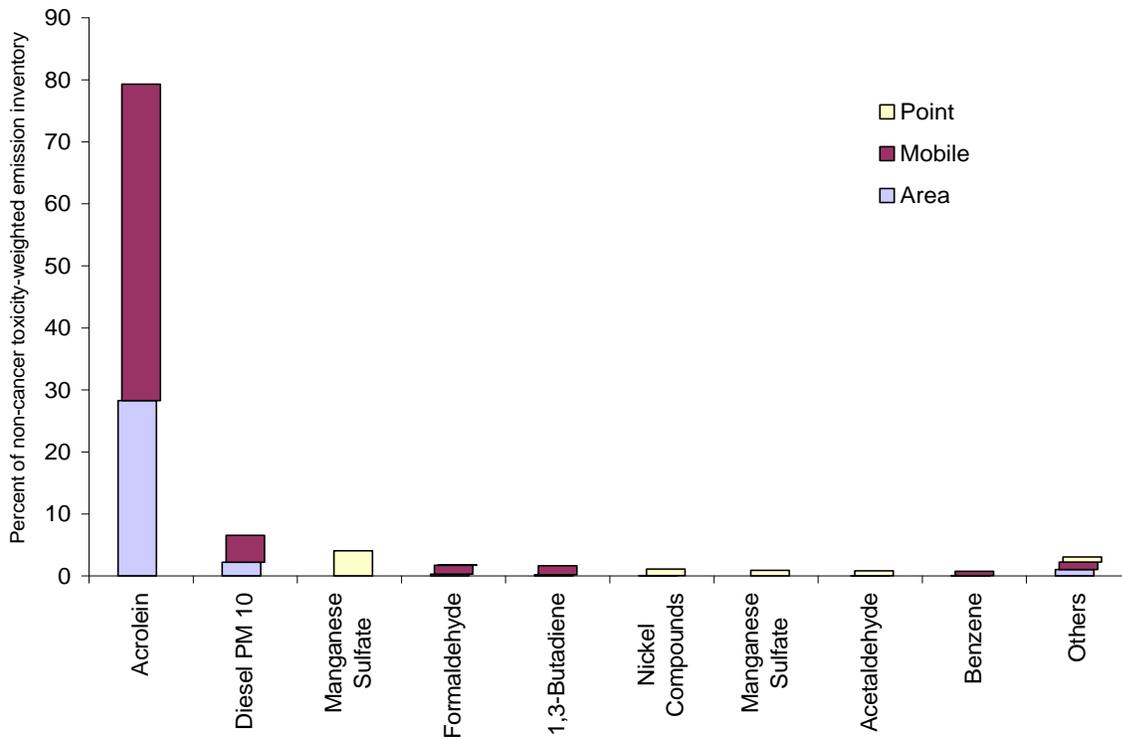


Figure 2-28. Benton County Air Agency Non-cancer toxicity weighted-emissions inventory.

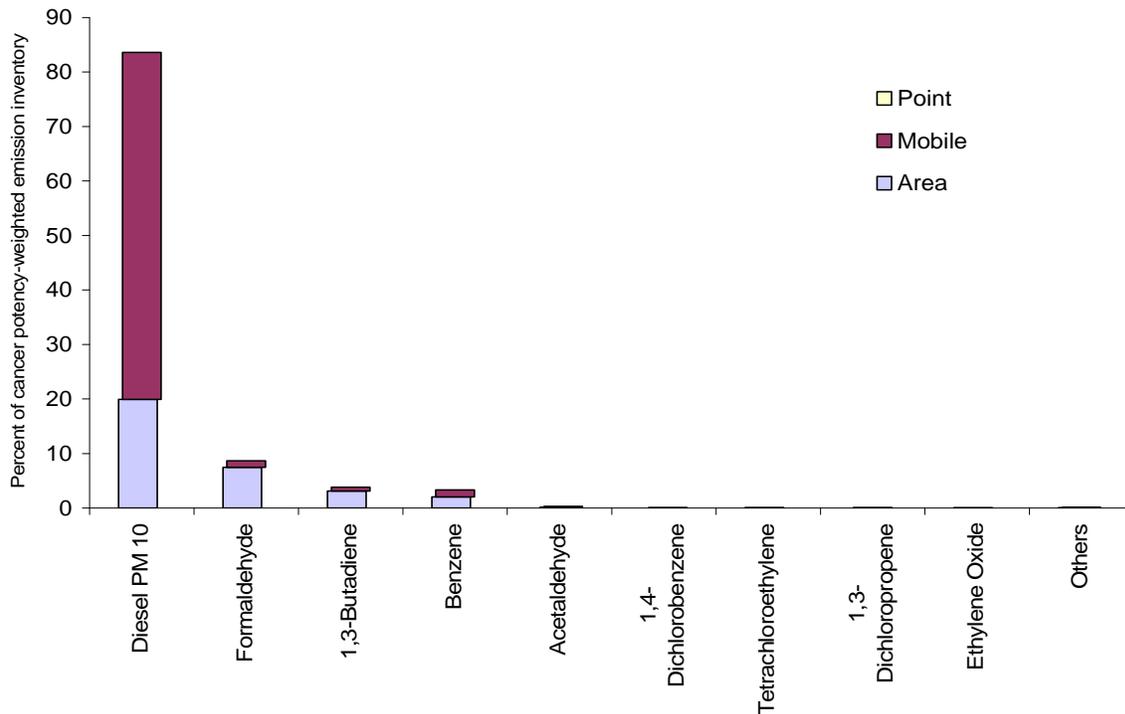


Figure 2-29. Ecology – Central Regional Office carcinogenic potency weighted-emissions inventory.

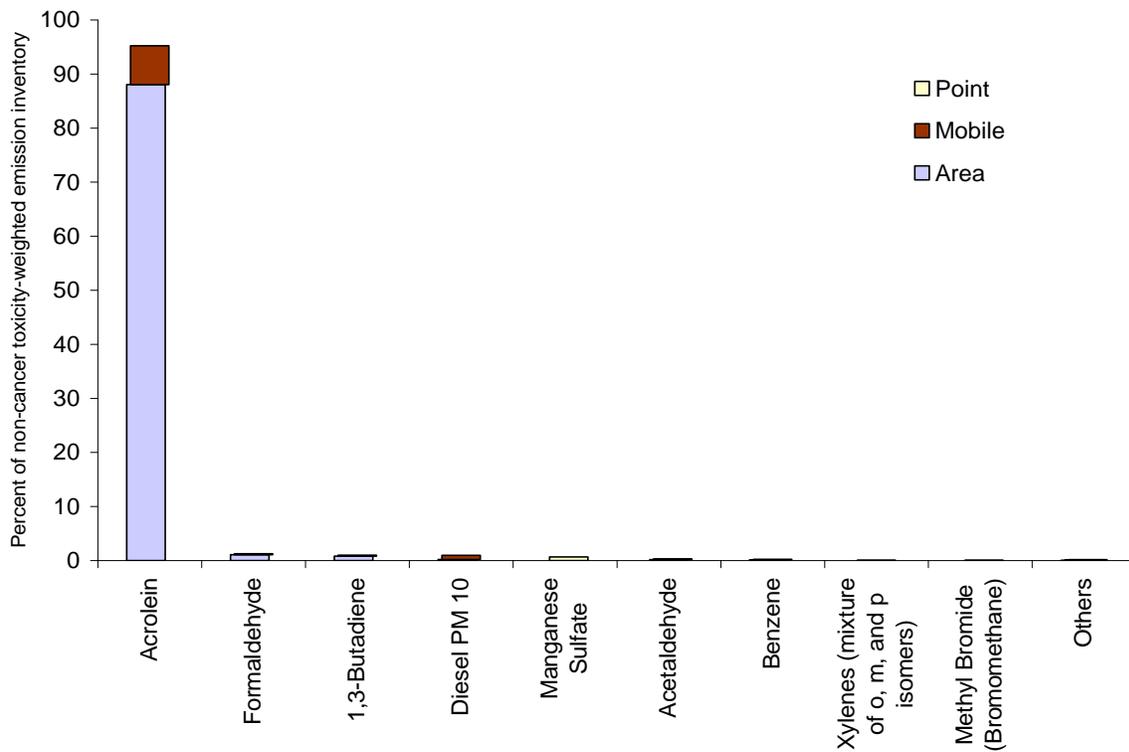


Figure 2-30. Ecology – Central Regional Office non-cancer toxicity weighted-emissions inventory.

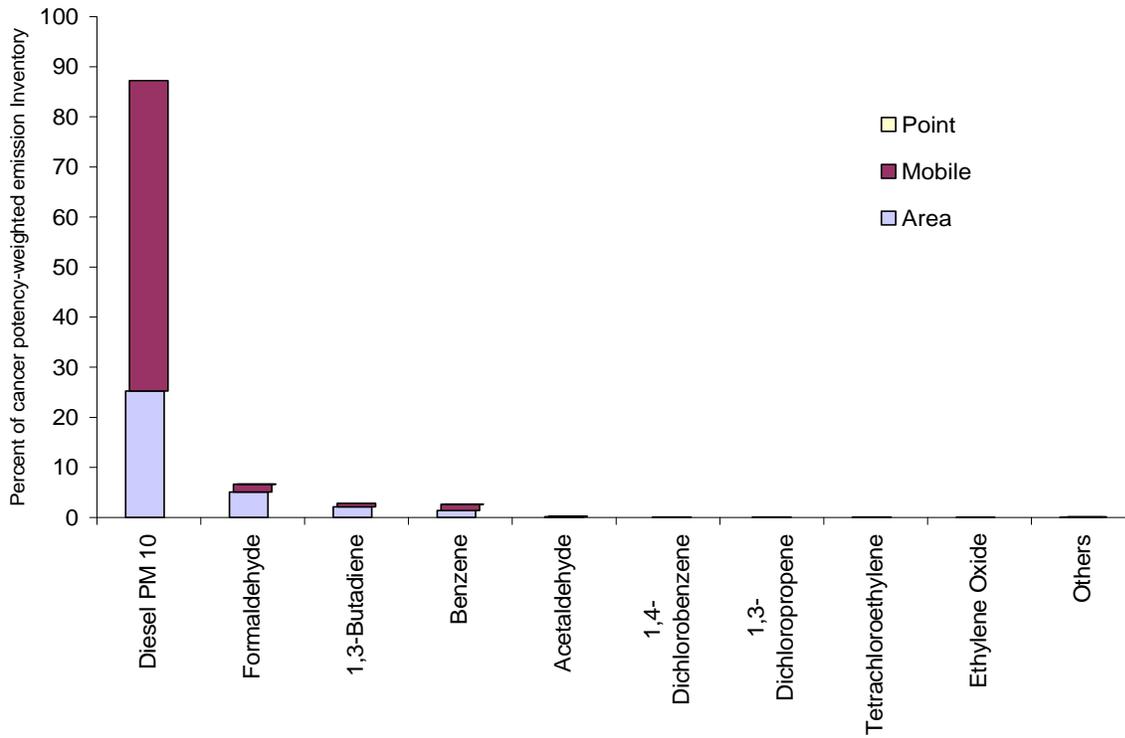


Figure 2-31. Ecology – Eastern Regional Office carcinogenic potency weighted-emissions inventory.

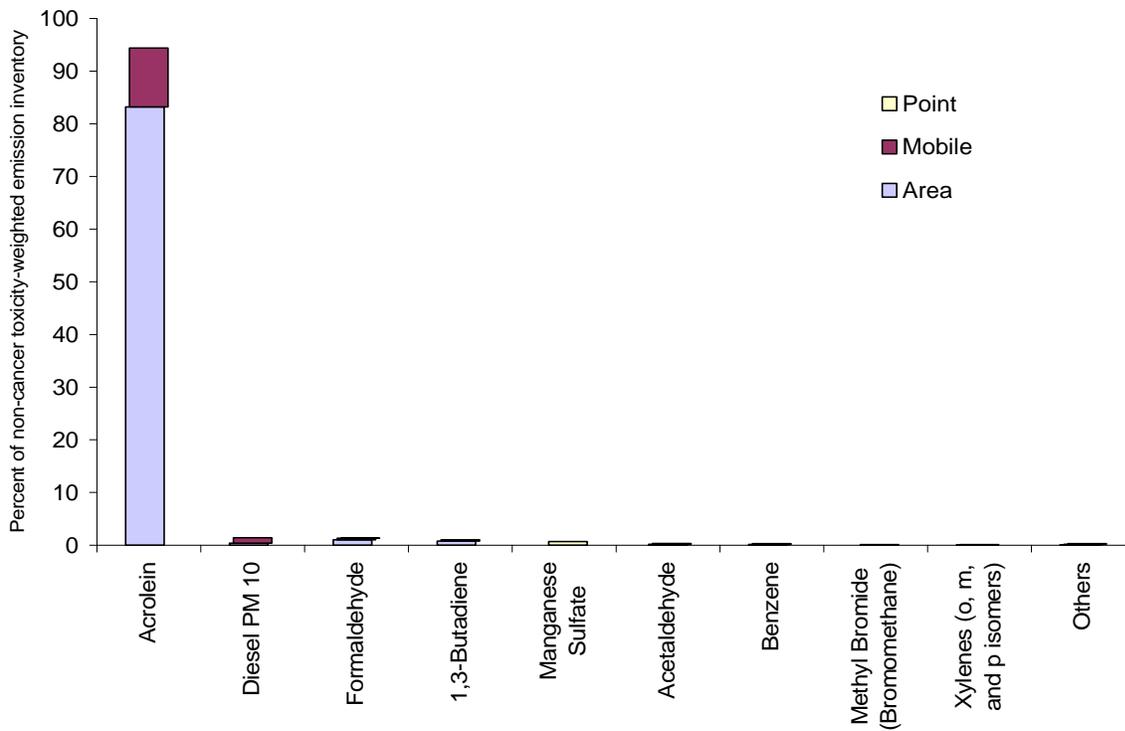


Figure 2-32. Ecology – Eastern Regional Office non-cancer toxicity weighted-emissions inventory.

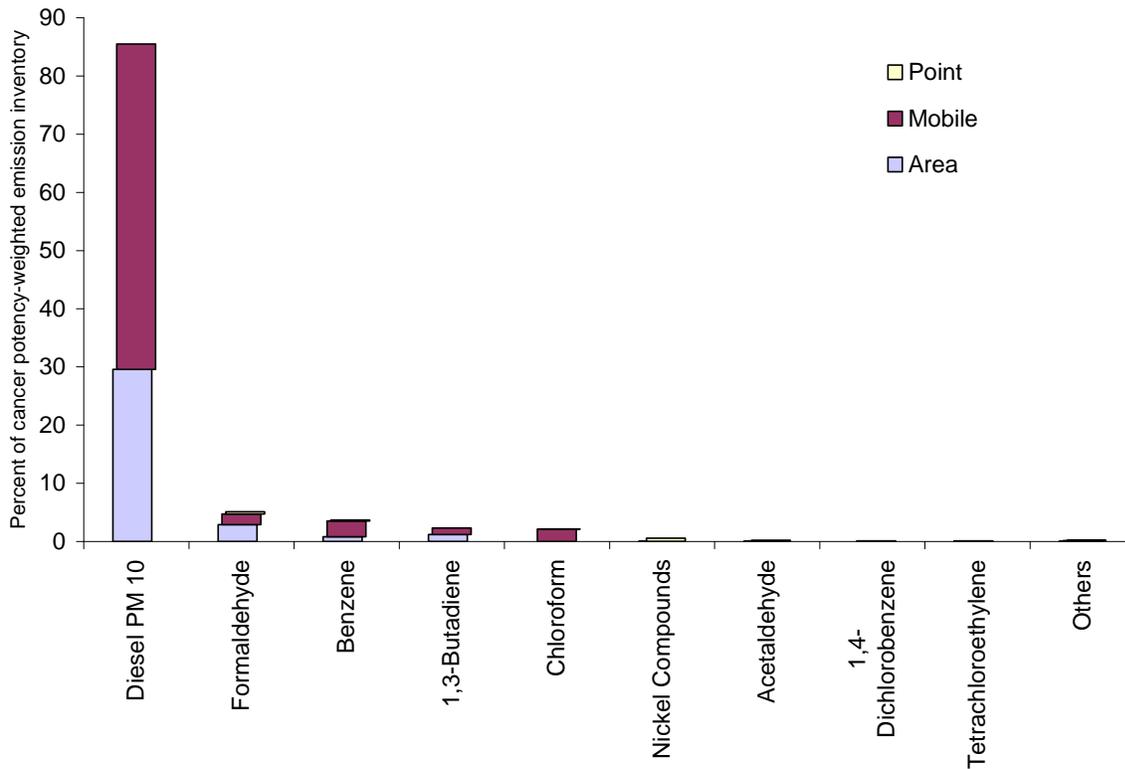


Figure 2-33. Northwest Clean Air Agency carcinogenic potency weighted-emissions inventory.

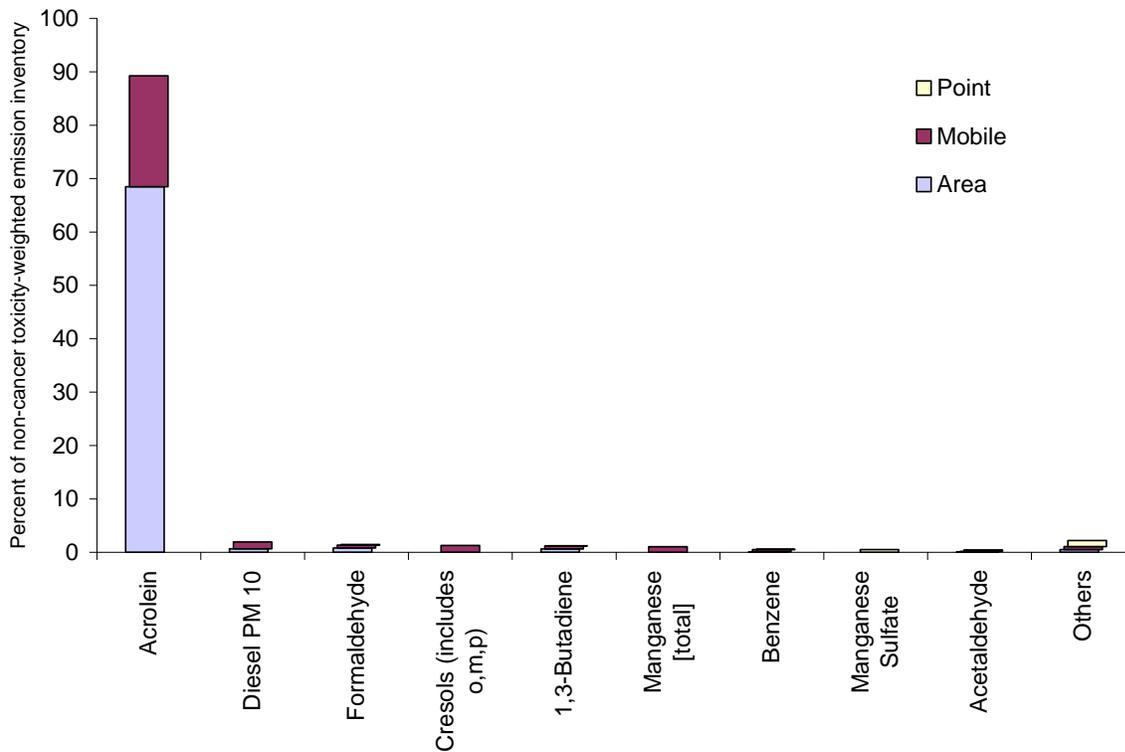


Figure 2-34. Northwest Clean Air Agency non-cancer toxicity weighted-emissions inventory.

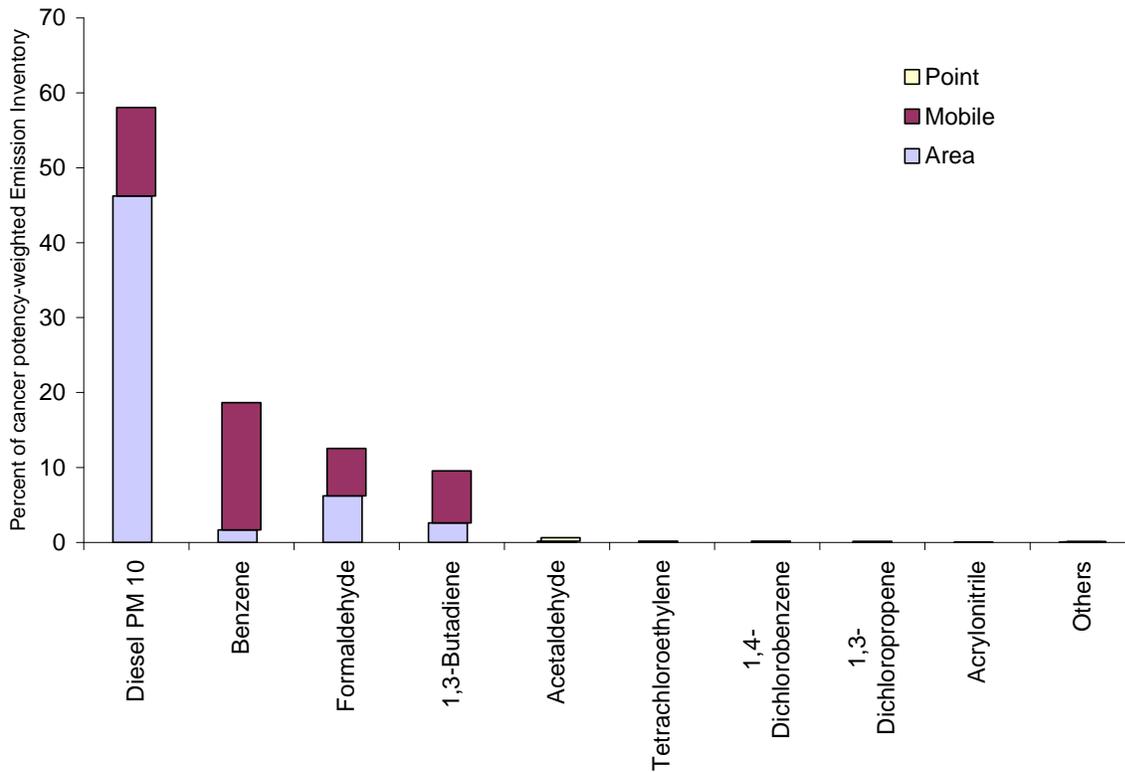


Figure 2-35. Ecology - Northwest Regional Office carcinogenic potency weighted-emission inventory.

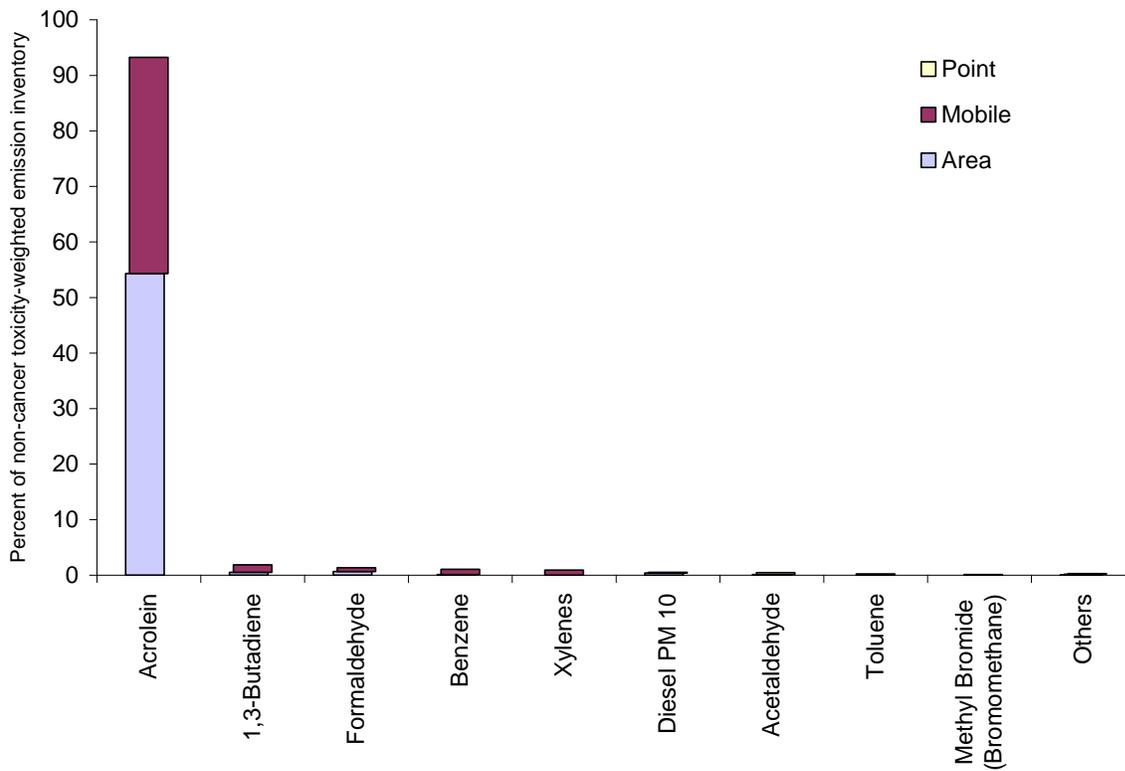


Figure 2-36. Ecology - Northwest Regional Office non-cancer toxicity weighted-emission inventory.

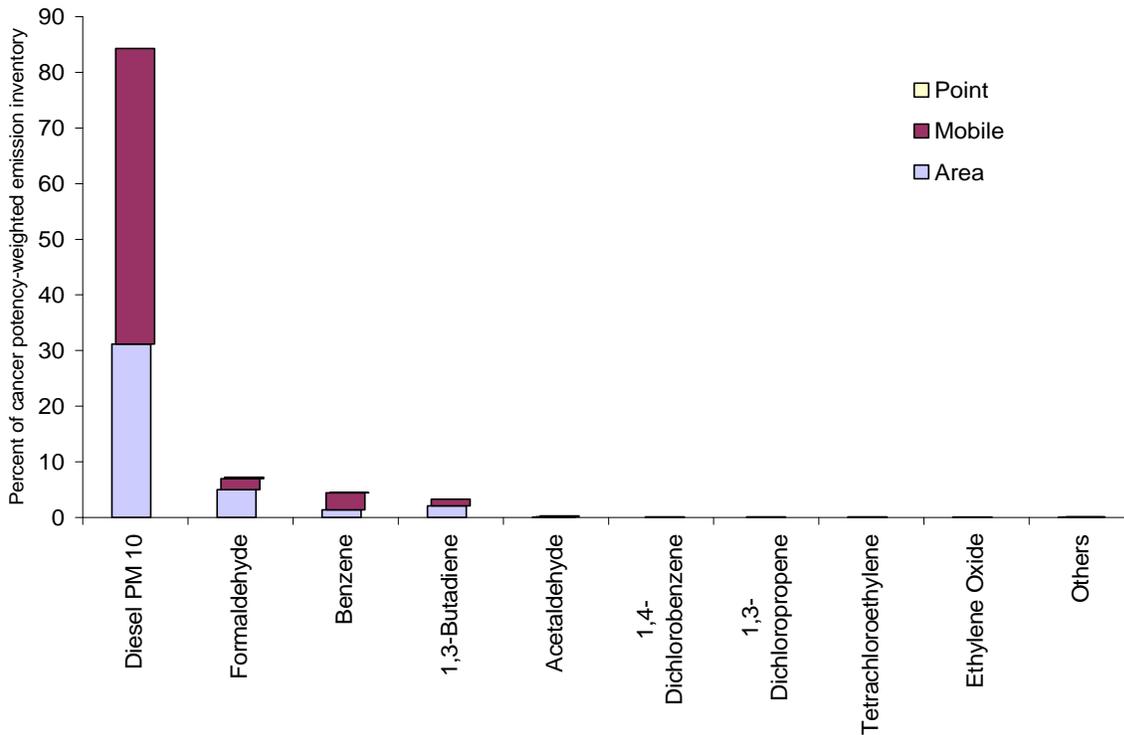


Figure 2-37. Olympic Region Clean Air Agency carcinogenic potency weighted-emission inventory.

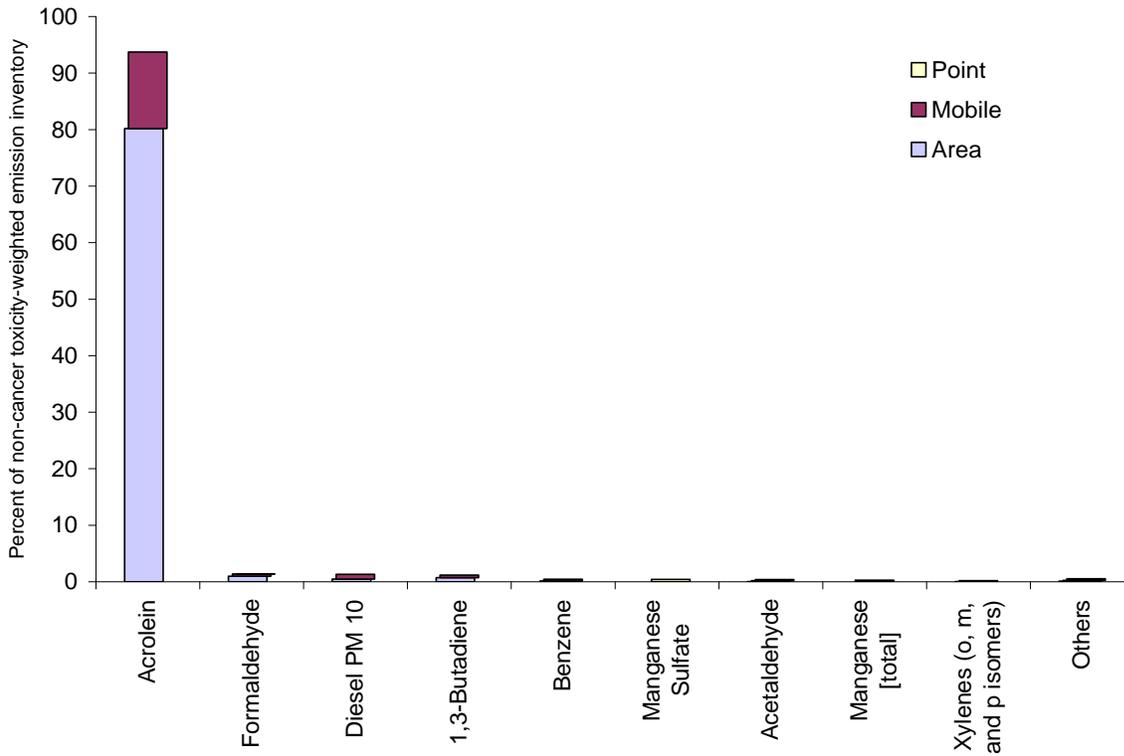


Figure 2-38. Olympic Region Clean Air Agency non-cancer toxicity weighted-emission inventory.

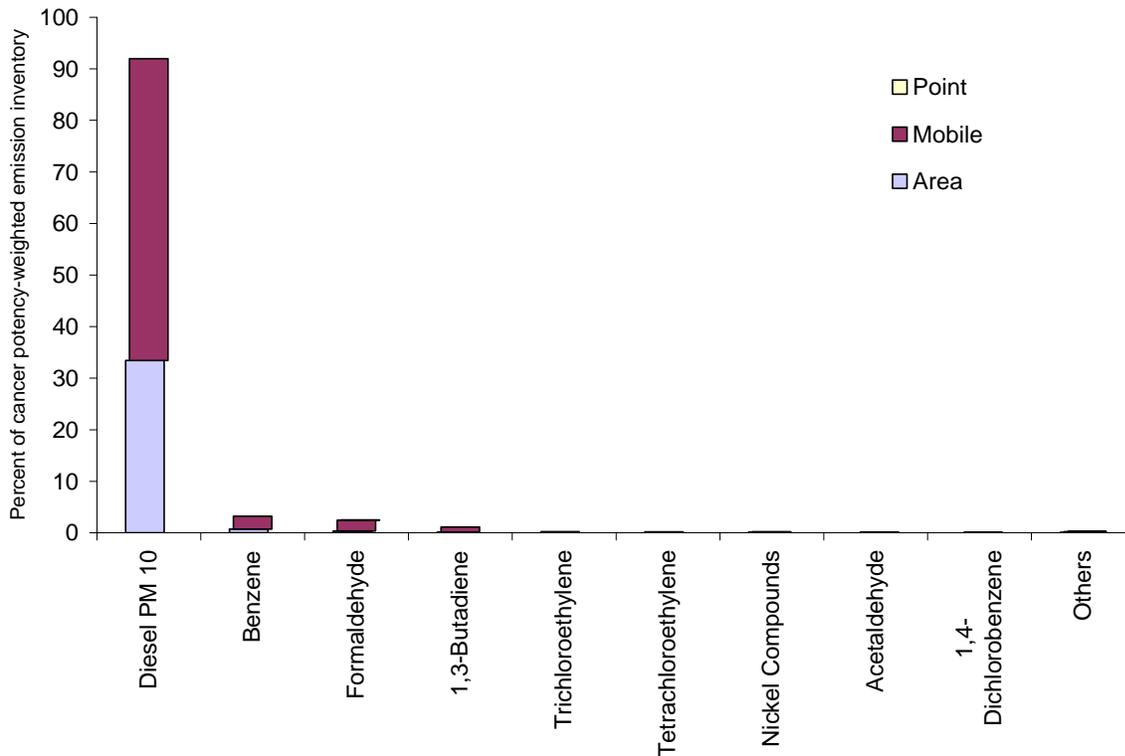


Figure 2-39. Puget Sound Clean Air Agency carcinogenic potency weighted-emission inventory.

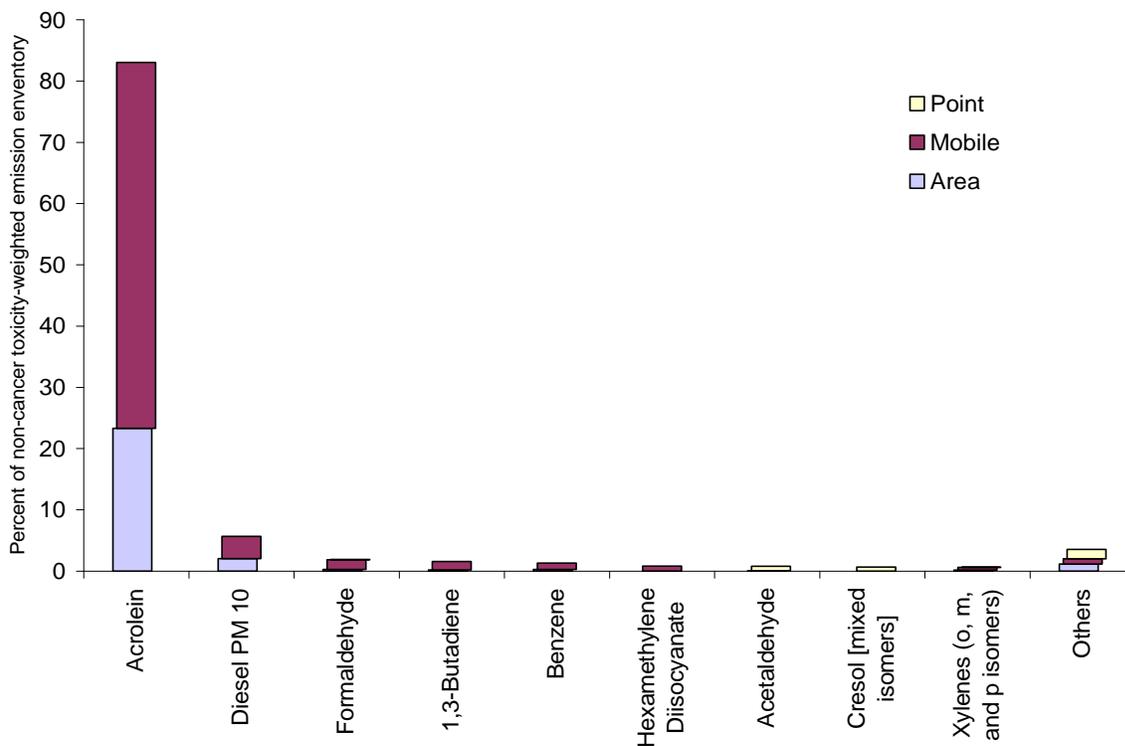


Figure 2-40. Puget Sound Clean Air Agency non-cancer toxicity weighted-emission inventory.

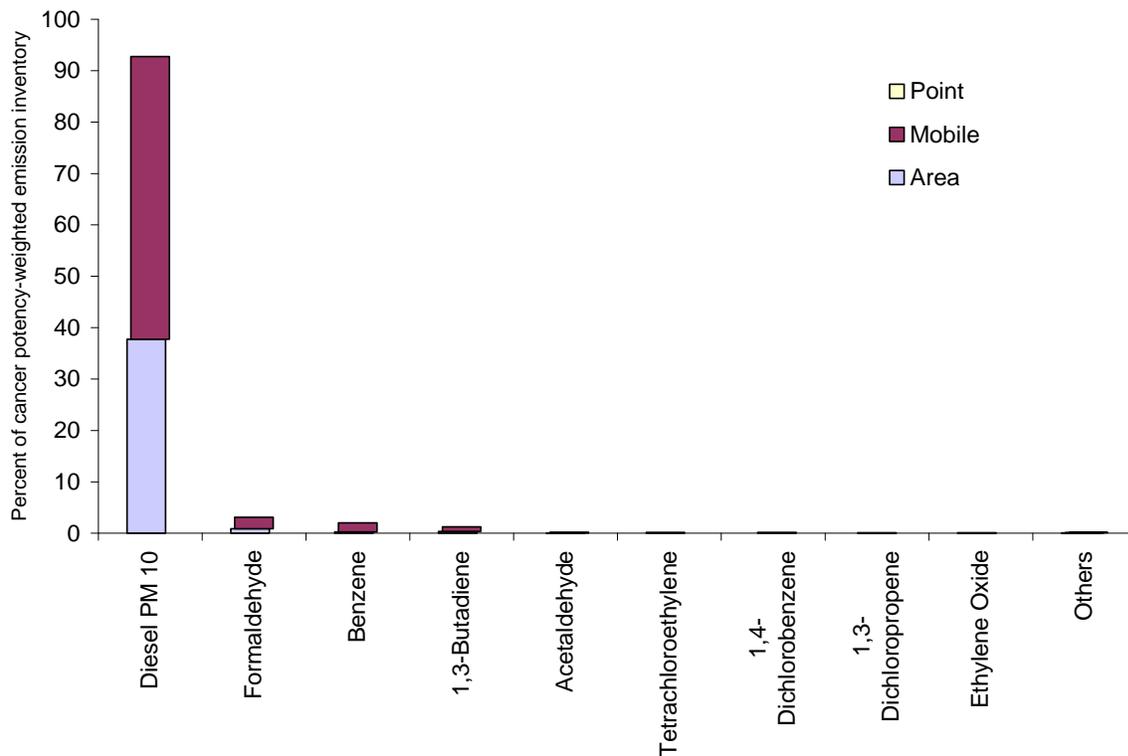


Figure 2-41. Spokane Regional Clean Air Agency carcinogenic potency weighted-emission inventory.

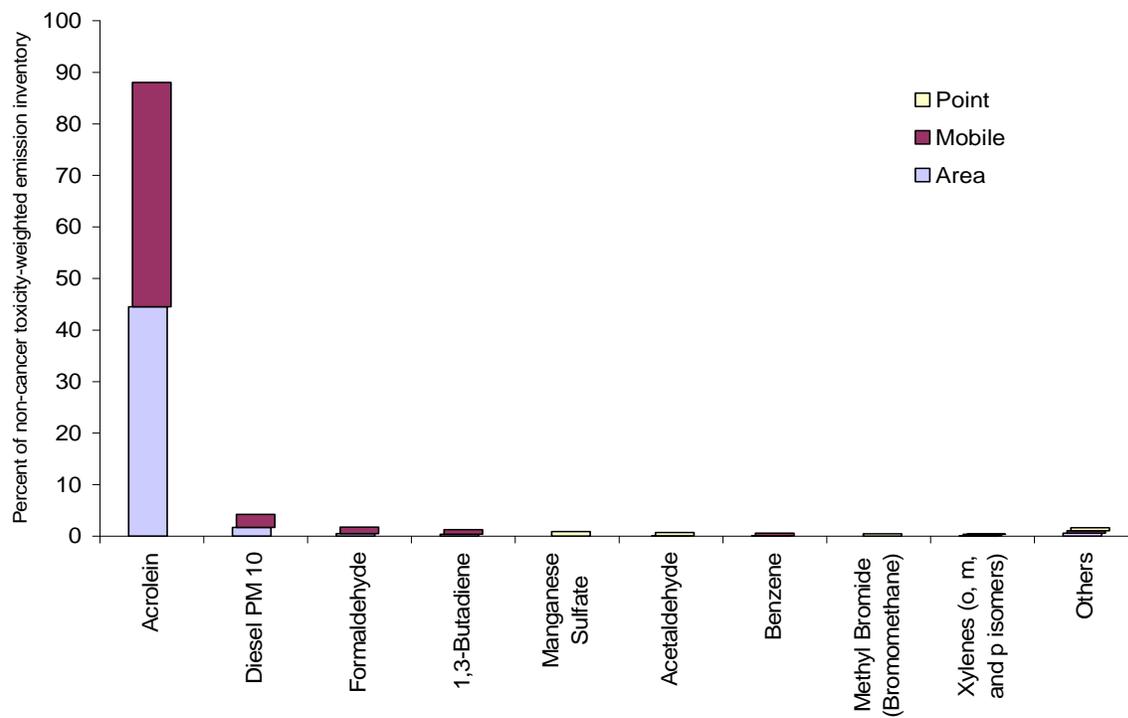


Figure 2-42. Spokane Regional Clean Air Agency non-cancer toxicity weighted-emission inventory.

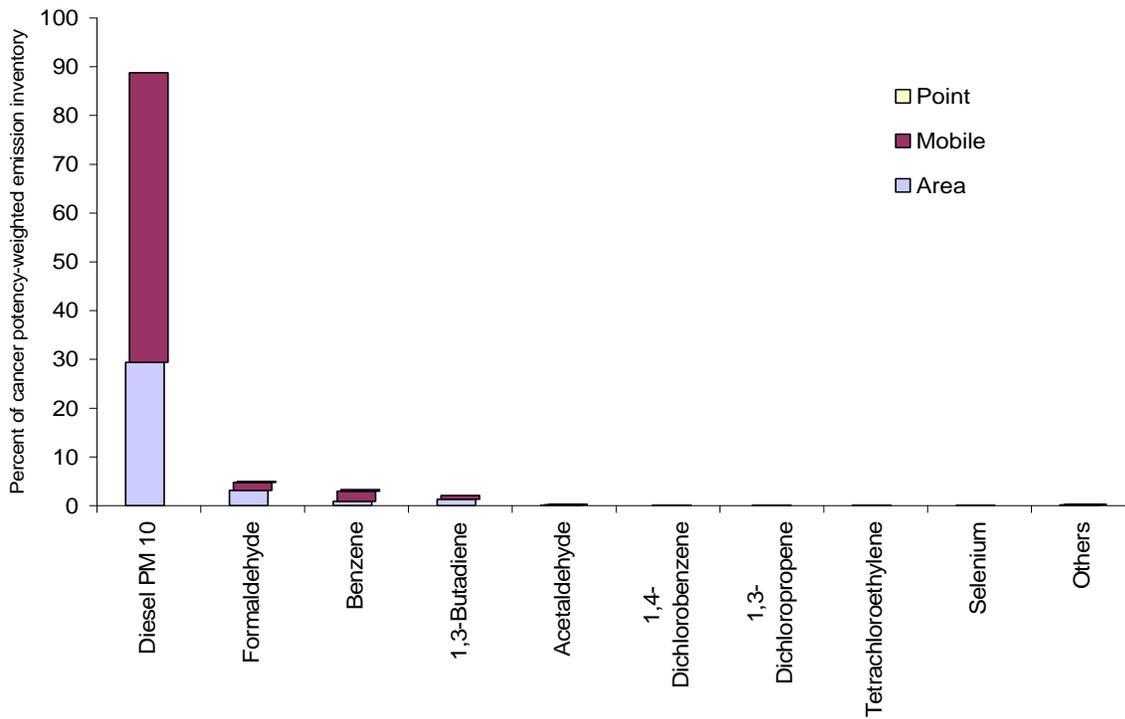


Figure 2-43. Southwest Clean Air Agency carcinogenic potency weighted-emission inventory.

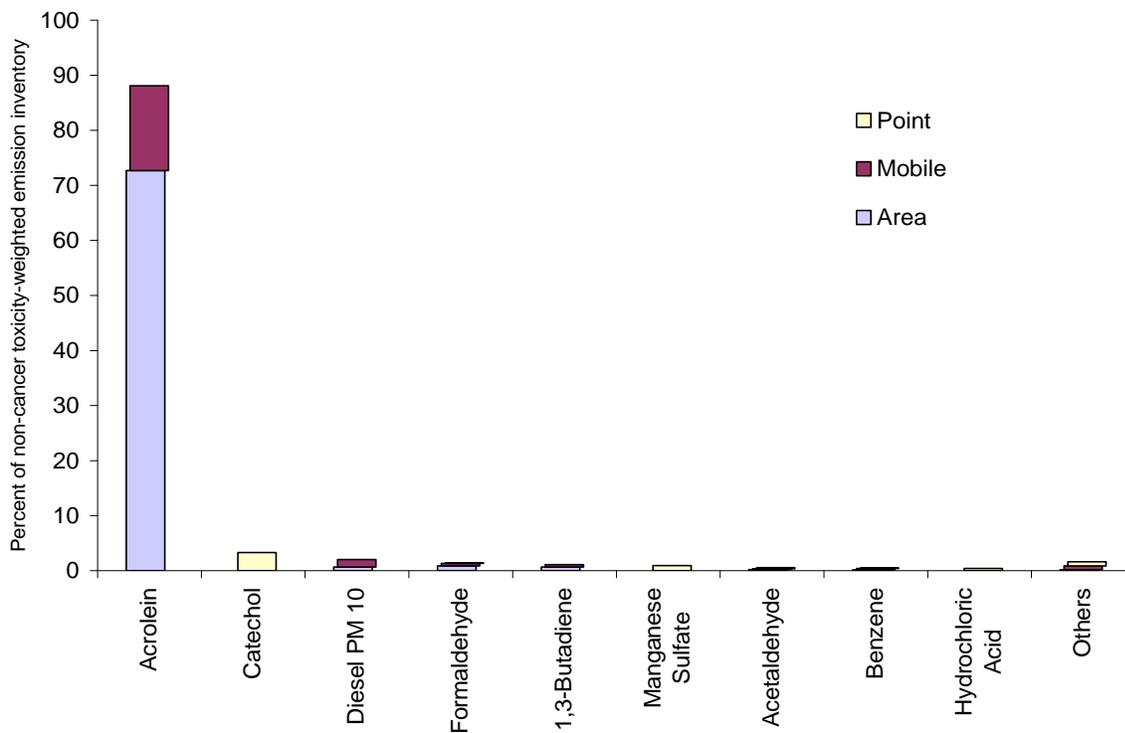


Figure 2-44. Southwest Clean Air Agency non-cancer toxicity weighted-emission inventory.

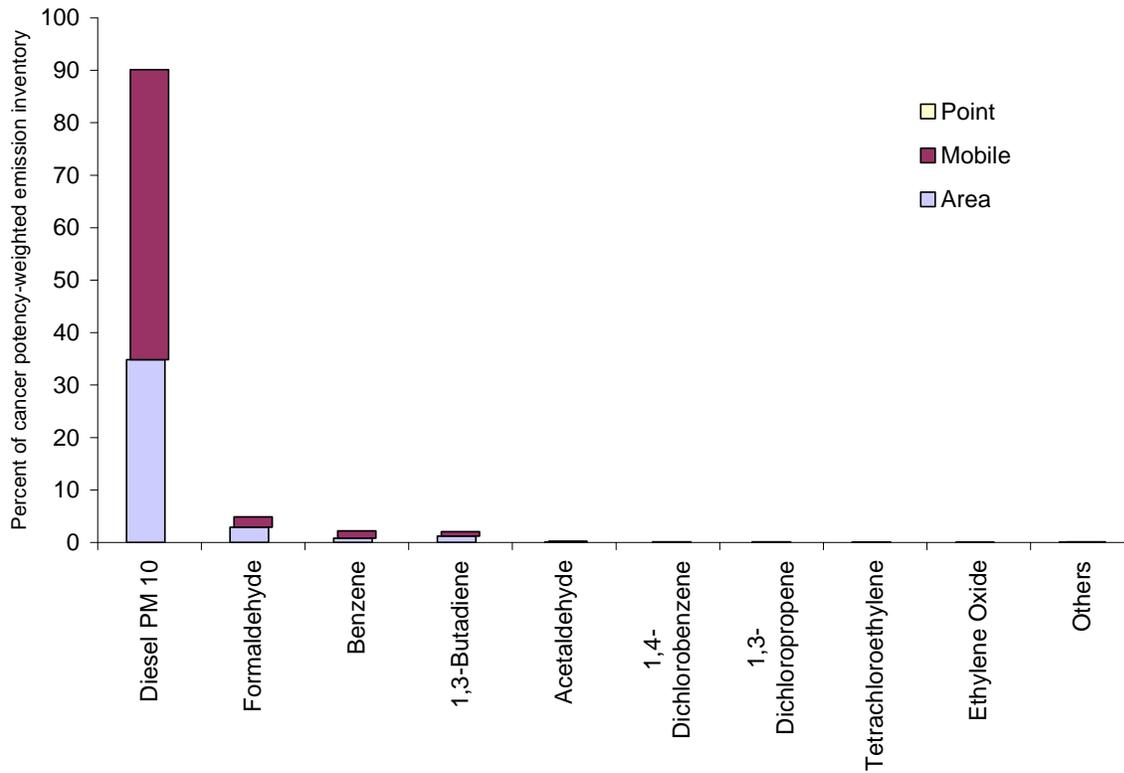


Figure 2-45. Yakima Regional Clean Air Agency carcinogenic potency weighted-emission inventory.

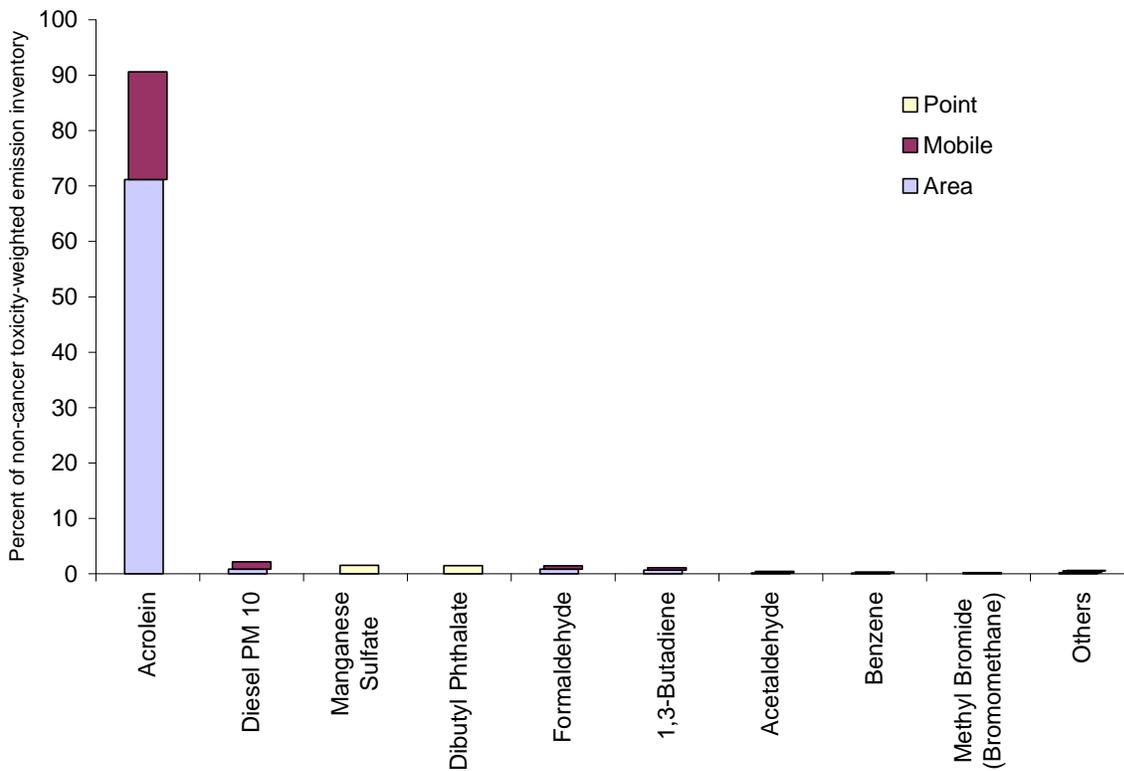


Figure 2-46. Yakima Regional Clean Air Agency non-cancer toxicity weighted-emission inventory.

Discussion

The cancer-potency weighted emissions inventory (CPWEI) suggests diesel PM is the most hazardous air pollutant in Washington. DPM's CPWEI top rank is corroborated by the NATAs DPM exposure estimates. Further, the CPWEI suggests that several other toxic air pollutants may be of concern. Among these are formaldehyde, benzene, chromium, 1,3-butadiene, polycyclic organic matter, arsenic and its compounds, acetaldehyde, nickel and its compounds, tetrachloroethylene, chloroform, trichloroethylene, 1,4-dichlorobenzene, 1,3-dichloropropene, and ethylene oxide. Lower ranking pollutants are not listed because their NATA median exposure estimates suggested they posed less than one-in-a-million excess cancer risk anywhere in Washington. Acrolein ranked first in the non-cancer TWEI. The NATA exposure estimates for acrolein indicated an eye and respiratory tract irritation that hazards may also exist in many counties in Washington.

The regional-scale rankings are similar to the state-wide rankings, with diesel PM and acrolein emissions dominating the cancer-potency and non-cancer toxicity-weighted EI rankings, respectively.

Limitations

The HWEI ranking approach has several important limitations. These are numbered below:

1. The emission inventory we used (NTI) is only indirectly related to human exposure. Emissions quantities cannot by themselves be construed as exposure estimates; therefore the hazard weighted EI does not address the magnitude of risk. This is a screening effort only, not a risk assessment. In other words, the estimates of proportionate health effect risks cannot be viewed as actual cancer or non-cancer incidences resulting from air pollution, but as an estimate of relative impact of the evaluated toxic-air pollutants to support the prioritization of exposure reduction efforts.
2. Emissions data were from 1996. Since 1996, there have been changes that might alter modeled estimates of pollutant concentrations. In general, we expect emissions from major point sources and several area source categories to have declined as sources closed

or came into compliance with the National Emissions Standards for Hazardous Air Pollutants. Since population has increased in most places, emissions from area source categories are likely to have increased. For mobile sources, both vehicles and fuels have gotten cleaner, but vehicle-miles and fuel-use have increased.

3. The USEPA tried to compensate for missing emissions inventory data. Still the reporting is incomplete and therefore portions of the NTI are subject to doubt.
4. Because of the limited knowledge for the ambient air speciation of metals and the composition of mixtures of POM, chlorinated dioxins, etc., it is unknown how much the relative risk of these heterogenous materials may have been miscalculated. For example, we do not know the proportion of insoluble Cr(VI) relative to soluble Cr(VI) compounds are emitted because they were not itemized in the emissions inventory. The actual amount to Cr(VI) in each form in the mixture reported in the inventory is probably not equal. Further the toxicity profiles are different as noted in the table below.

Table 2-22. ACGIH TWA-TLVs for Chromium⁵⁸

Substance	TWA-TWA (mg/m³)	Notations	TLV-Basis-Critical Effects
Chromium, and inorganic compounds, as Cr - Metal and Cr(III) compounds	0.5	not classifiable as a human carcinogen	Irritation; dermatitis
Chromium, and inorganic compounds, as Cr - Insoluble Cr(VI) compounds	0.01	confirmed human carcinogen	Cancer; irritation
Chromium, and inorganic compounds, as Cr – Water-soluble Cr(VI) compounds	0.05	confirmed human carcinogen; Biological Exposure Indices: Monitoring should be instituted to determine exposure from all routes	Liver; kidney; respiratory

⁵⁸ ACGHI 2001

Substantial uncertainty arises from lack of confidence in the appropriateness of some of the toxicity values used for weighting the emissions inventories: Most notably from our use of a range of factors for modifying the TWA-TLVs of a few of the toxic air pollutants. Some toxic air pollutants had TWA-TLVs for cancer and/or non-cancer endpoints, but did not have toxicity criteria from any of the preferred sources (IRIS, ATSDR, OEHHA, etc.), whereas other toxic air pollutants had TWA-TLV and preferred source criteria. We determined the range of differences between TLVs and preferred criteria for the latter group, separately for non-cancer and cancer endpoints, then applied these ranges as adjustment factors to the TWA-TLVs of toxic air pollutants in the former group. For example, n,n-dimethylaniline, whose total emissions in 1996 were reported as 0.409 tons, and whose TWA-TLV (25-mg/m³) divided by the median of the range of preferred RfC-like criteria (441) equals 0.0562. Likewise, the lowest in the range of differences between TWA-TLVs and preferred criteria is 1E⁻⁵, while the highest in the range is 74.3. Dividing the tons-per-year emissions by the median, lowest and highest of this observed uncertainty factor-adjusted TWA-TLV range yields a toxicity-weighted inventory of 7.3, 0.0055, and 40,555 (in units of [tons/year]/[mg/m³]), respectively. This uncertainty comprises a range spanning almost seven orders of magnitude. Compared to the rank order at the median of the uncertainty range, the uncertainty factor does not affect the order of the top ranked toxic air pollutants in the cancer-potency weighted emissions inventory. However, the rank order of the toxic air pollutants with significant non-cancer risk was influenced: specifically, if the high end of the uncertainty range of TLV derived RfC-life factors for certain chromium compounds is used for toxicity-weighting the emission inventory, they rank as high as acrolein and DPM. Conversely these chromium compounds may be much less important than acrolein and diesel if the appropriate toxicity value at the lower range of the range.

5. There may be some combinations of toxic air pollutants that are additive or super-additive or that counteract each other's toxicity. These combinations have not been identified.

6. The pre-existing toxic air pollutants background levels were not accounted for in any way using the HWEI approach resulting in under-prediction of toxicity-weights for some of the persistent toxic air pollutants.
7. The HWEI does not take into account pollutant dispersion, deposition, hotspots, transport and fate, or secondary chemical reactions.
8. The results pertain to outdoor air; however, people spend a large fraction of time indoors. Indoor concentrations of some toxic air pollutants tend to be higher than outdoor concentrations and lower for other toxic air pollutants.

Conclusions

The TWEI and CPWEI may be summarized as in the following tables.

Table 2-23. Non-cancer toxicity-weighted EI rank

Pollutant	Non-Cancer TWEI Rank	Assessed in the NATA
Acrolein	1	yes
Diesel PM	2	yes
Total POM *	3	no

* Rank based of combined toxicity weight of the few chemicals in this group reported in the NTI that have published non-cancer exposure limits.

Table 2-24. Cancer potency-weighted EI rank

Pollutant	CPWEI Rank	Assessed in the NATA
Diesel Particulate Matter	1	no
Formaldehyde	2	yes
Benzene	3	yes
Chromium & Compounds	4	yes
1,3-Butadiene	5	yes
Polycyclic Organic Matter	6	yes
7-PAH	7	yes
Arsenic Compounds	8	yes
Acetaldehyde	9	yes
Benzo[b+k] Fluoranthene	10	yes
Nickel & Compounds	11	yes
Tetrachloroethylene (Perchloroethylene)	12	yes

Chloroform	13	yes
Trichloroethylene	14	yes
1,4-Dichlorobenzene	15	no
1,3-Dichloropropene	16	yes*
Ethylene Oxide	17	yes
Benzo[a]Pyrene	18	yes
PAH, Total	19	yes

* NATA median exposure estimate poses <E-6 excess cancer risk throughout Washington.

Note: For the for lower ranking pollutants not listed in table 2-24, the NATA median exposure estimate poses less than one in a million excess cancer risk throughout Washington, but a few people may have an additional cancer risk greater than $1E^{-6}$.

3. USEPA National-Scale Air Toxics Assessment

Introduction to NATA

USEPA’s 1996 and 1999 National-Scale Air Toxics Assessment (NATA)⁵⁹ reports are reviewed here with emphasis on Washington state findings. The NATA was an assessment of health risks from exposure to toxic air pollutants based on sums of risks of census tracts in each county within the contiguous 48 states, Puerto Rico and the U.S. Virgin Islands. The first report was based on 1996 air emissions data, and looked at 33 air pollutants (diesel particulate matter and a subset of 32 toxic air pollutants from the Clean Air Act list of 187 hazardous air pollutants). The second report differed from the first in that it was based on 1999 toxic air emission data and examined 176 federal hazardous air pollutants plus diesel particulate matter. Given the reduced level of access to local data provided for the 1999 NATA results, most of this report refers to the 1996 NATA results except where noted.

In general, the NATA results show the following: Inhalation of some of the toxic air pollutants may be associated with significant carcinogenic risk or respiratory tract irritation, at the national scale or across broad regions of the country. Three toxic air pollutants (chromium, benzene, and formaldehyde) appear to pose the greatest carcinogenic risk nationwide. Another (acrolein) is estimated to pose the highest potential for significant chronic non-cancer effects. In addition, four toxic air pollutants (arsenic, 1,3-butadiene, coke oven emissions, and polycyclic organic

⁵⁹ The National-Scale Air Toxics Assessment website is <http://www.epa.gov/ttn/atw/nata>

matter) appear to pose carcinogenic health threats in some regions. Five (acetaldehyde, arsenic, 1,3-butadiene, formaldehyde, and manganese) have a potential to pose chronic non-cancer effects in some regions.

The potential risk from diesel exhaust emissions was not addressed in NATA in the same way as were other toxic air pollutants. This was because USEPA considers existing health data on DPM to be inadequate to develop a numeric estimate of its cancer potency.⁶⁰ In NATA however, USEPA made the contradictory conclusion that diesel exhaust is a likely human carcinogen and that it ranks with the other substances that pose the “greatest relative risk”.⁶¹ Our examination of published epidemiological and experimental studies leads to the conclusion that diesel exhaust particulate matter is carcinogenic and that the California Air Resources Board Science Advisory Panel’s unit risk estimate is appropriate for assessing these cancer risks.

USEPA states that NATA is not designed to be used as the basis for regulatory action. Indeed they have not done so. As noted, the AQP relied on the NATA and other information to identify priority toxic air pollutants primarily for subsequent evaluation. In the final section of this report, we compare and contrast NATA to our statewide and regional toxic air pollutants rankings.

NATA used emissions inventories and the sparse monitoring data to estimate ambient toxic air pollutant concentrations. With its subsequent model-based estimates of human health risks, NATA greatly adds to the Washington toxic air pollutants assessment. Consideration of the NATA provided us with health risk cutoff points for our HWEIs ranking.

⁶⁰ See the report on diesel at <http://toxnet1.nlm.nih.gov>

⁶¹ <http://www.epa.gov/ttnatw01/nata/perspect.html>

NATA methods

Overview

The NATA consisted of four phases. In Phase I, USEPA calculated emissions of hazardous air pollutants and diesel particulate matter in mobile, area, and major point source categories. The pollutants they considered are listed in table 3-1.

Table 3-1. Toxic air pollutants included in the 1996 and 1999 NATAs

Air pollutant	CAS Number	1996	1999
Acetaldehyde	75070	x	x
Acetamide	60355		x
Acetonitrile	75058		x
Acetophenone	98862		x
2-Acetylaminofluorene	53963		x
Acrolein	107028	x	x
Acrylamide	79061		x
Acrylic acid	79107		x
Acrylonitrile	107131	x	x
Allyl chloride	107051		x
4-Aminobiphenyl	92671		x
Aniline	62533		x
Antimony compounds	–		x
Arsenic compounds (inorganic, may include arsine)	–	x	x
Arsine	7784421		x
Asbestos	1332214		x
Benzene	71432	x	x
Benzidine	92875		x
Benzotrichloride	98077		x
Benzyl chloride	100447		x
Beryllium compounds	–	x	x
beta-Propiolactone	57578		x
Biphenyl	92524		x
Bis(2-ethylhexyl)phthalate	117817		x
Bis(chloromethyl)ether	542881		x
Bromoform	75252		x
1,3-Butadiene	106990	x	x
Cadmium compounds	–	x	x
Calcium cyanamide	156627		x
Captan	133062		x
Carbaryl	63252		x

Carbon disulfide	75150		X
Carbon tetrachloride	56235	X	X
Carbonyl sulfide	463581		X
Catechol	120809		X
Chlordane	57749		X
Chlorine	7782505		X
Chloroacetic acid	79118		X
2-Chloroacetophenone	532274		X
Chlorobenzene	108907		X
Chlorobenzilate	510156		X
Chloroform	67663	X	X
Chloromethyl methyl ether	107302		X
Chloroprene	126998		X
Chromium compounds	–	X	X
Cobalt compounds	–		X
Coke Oven Emissions	–	X	X
Cresols - Cresylic acid (isomers and mixture)	1319773		X
Cumene	98828		X
Cyanide compounds	–		X
2,4-D, salts and esters	94757		X
Diazomethane	334883		X
Dibenzofurans	132649		X
1,2-Dibromo-3-chloropropane	96128		X
Dibutylphthalate	84742		X
3,3-Dichlorobenzidene	91941		X
Dichloroethyl ether	111444		X
1,3-Dichloropropene	542756	X	X
Dichlorvos	62737		X
Diesel particulate matter (DPM)	–	X	X
Diethanolamine	111422		X
Diethyl sulfate	64675		X
3,3-Dimethoxybenzidine	119904		X
3,3-Dimethyl benzidine	119937		X
Dimethyl carbamoyl chloride	79447		X
Dimethyl formamide	68122		X
1,1-Dimethyl hydrazine	57147		X
Dimethyl phthalate	131113		X
Dimethyl sulfate	77781		X
4,6-Dinitro-o-cresol, and salts	534521		X
2,4-Dinitrophenol	51285		X
2,4-Dinitrotoluene	121142		X
1,4-Dioxane	123911		X
1,2-Diphenylhydrazine	122667		X
Epichlorohydrin	106898		X

1,2-Epoxybutane	106887		x
Ethyl acrylate	140885		x
Ethyl benzene	100414		x
Ethyl carbamate	51796		x
Ethyl chloride	75003		x
Ethylene dibromide (1,2 dibromoethane)	106934	x	x
Ethylene dichloride (1,2 dichloroethane)	107062	x	x
Ethylene glycol	107211		x
Ethylene imine (Aziridine)	151564		x
Ethylene oxide	75218	x	x
Ethylene thiourea	96457		x
Ethylidene dichloride	75343		x
Fine mineral fibers	–		x
Formaldehyde	50000	x	x
Glycol ethers	–		x
Heptachlor	76448		x
Hexachlorobenzene	118741	x	x
Hexachlorobutadiene	87683		x
Hexachlorocyclopentadiene	77474		x
Hexachloroethane	67721		x
Hexamethylene-1,6-diisocyanate	822060		x
Hexamethylphosphoramide	680319		x
Hexane	110543		x
Hydrazine	302012	x	x
Hydrochloric acid	7647010		x
Hydrofluoric acid	7664393		x
Hydroquinone	123319		x
Isophorone	78591		x
Lead compounds	–	x	x
Lindane (all isomers)	58899		x
Maleic anhydride	108316		x
Manganese compounds	–	x	x
Mercury compounds	–	x	x
Methanol	67561		x
Methoxychlor	72435		x
Methyl bromide	74839		x
Methyl chloride	74873		x
Methyl chloroform	71556		x
Methyl ethyl ketone	78933		x
Methyl hydrazine	60344		x
Methyl iodide	74884		x
Methyl isobutyl ketone	108101		x
Methyl isocyanate	624839		x
Methyl methacrylate	80626		x

Methyl tert butyl ether	1634044		x
4,4'-Methylene bis(2-chloroaniline)	101144		x
Methylene chloride	75092	x	x
Methylene diphenyl diisocyanate	101688		x
4,4'-Methylenedianiline	101779		x
N,N-Dimethyl aniline	121697		x
Naphthalene	91203		x
Nickel compounds	—	x	x
Nitrobenzene	98953		x
4-Nitrobiphenyl	92933		x
4-Nitrophenol	100027		x
2-Nitropropane	79469		x
Nitrosodimethylamine	62759		x
N-Nitrosomorpholine	59892		x
N-Nitroso-N-methylurea	684935		x
o-Anisidine	90040		x
o-Toluidine	95534		x
Parathion	56382		x
p-Dichlorobenzene	106467		x
p-Dimethylaminoazobenzene	60117		x
Pentachloronitrobenzene	82688		x
Pentachlorophenol	87865		x
Phenol	108952		x
Phosgene	75445		x
Phosphine	7803512		x
Phthalic anhydride	85449		x
Polychlorinated biphenyls (PCBs)	1336363	x	x
Polycyclic Organic Matter (POM)	—	x	x
p-Phenylenediamine	106503		x
1,3-Propane sultone	1120714		x
Propionaldehyde	123386		x
Propoxur	114261		x
Propylene dichloride (1,2 dichloropropane)	78875	x	x
Propylene oxide	75569		x
1,2-Propylenimine	75558		x
Quinoline	91225	x	x
Quinone	106514		x
Selenium Compounds	—		x
Styrene	100425		x
Styrene oxide	96093		x
1,1,2,2-Tetrachloroethane	79345	x	x
Tetrachloroethylene (perchloroethylene)	127184	x	x
Titanium tetrachloride	7550450		x
Toluene	108883		x

2,4-Toluene diamine	95807		x
2,4-Toluene diisocyanate	584849		x
Toxaphene	8001352		x
1,2,4-Trichlorobenzene	120821		x
1,1,2-Trichloroethane	79005		x
Trichloroethylene	79016	x	x
2,4,5-Trichlorophenol	95954		x
2,4,6-Trichlorophenol	88062		x
Triethylamine	121448		x
Trifluralin	1582098		x
2,2,4-Trimethylpentane	540841		x
Vinyl acetate	108054		x
Vinyl bromide	593602		x
Vinyl chloride	75014	x	x
Vinylidene chloride	75354		x
Xylenes (isomers and mixture)	1330207		x

In Phase II, USEPA used an air dispersion model, along with measurements of background concentrations of some air toxics, to estimate ambient concentrations in each census tract. In Phase III, USEPA used the predicted ambient concentrations as input for an exposure model that predicted human exposure concentrations. Finally, in Phase IV, they used these estimated human exposure concentrations to calculate potential cancer risks and non-cancer hazards. Greater detail of the methods of the phases in NATA is provided below.

Phase I: Emissions Inventories

In Phase I, USEPA used the emissions estimates reported in the 1996 National Toxics Inventory (NTI), along with the National Air Pollutant Emission Trends report⁶² for chemicals formed from precursors in the atmosphere. The NTI includes major toxic air pollutant sources such as larger waste incinerators, factories, and smaller sources, such as dry cleaners, small manufacturers, and wildfires. It also includes emissions from roadway and non-road mobile sources, such as cars, trucks and boats.⁶³ USEPA took several steps to fill in data for sources

⁶² <http://www.epa.gov/ttn/chief/trends/trends98/>

⁶³ See section 3: Emissions Inventories.

that were missing or poorly reported, and made other corrections as noted previously in this report. They also took steps to verify the quality of the emissions estimates.

Phase II: Estimating Ambient Air Concentrations

After emissions estimates were calculated, they were entered into a computer model. Estimates of average concentrations of toxics in the outdoor air were developed using this model, which analyzed total emissions, the number of emissions sources in a particular area, weather patterns, pollution source characteristics and other factors. The model, called the Assessment System for Population Exposure Nationwide (ASPEN), was used to predict annual average concentrations. It combined a Gaussian dispersion model with weather information, for each census tract across the United States. ASPEN used available information or assumptions about the rate and location of release of each chemical, the release height, wind speed and direction from the nearest meteorological station, wet and dry deposition rates, and atmospheric chemical transformations data. If monitoring data were available for specific pollutants in remote background areas (having little influence from distant modeled sources), these “background” concentrations were added to the values predicted by ASPEN. USEPA added background concentrations for 13 of the pollutants. These concentrations accounted for the long-range transport of toxic air pollutants that originated from natural sources (e.g., windblown soils, volcanic eruptions, etc.) and for sources not included in the emissions estimates. The background levels of toxic air pollutants without background concentration data were assumed to be zero (except in the case of diesel particulate matter). For DPM, background concentrations were estimated through modeling, which is described in the NATA Science Advisory report.⁶⁴

Phase III: Estimating Human Exposures

The ambient concentrations estimates from phase II were entered into another model – the Hazardous Air Pollutant Exposure Model (HAPEM4 for the 1996 NATA and HAPEM5 for the 1999 NATA) – to account for personal exposures and variations among the population in terms of daily activities. HAPEM allowed evaluation of long-term inhalation exposures by simulating the movement of representative individuals of various demographic groups through different types of locations. Each of these locations was referred to as a “microenvironment”.

⁶⁴ USEPA, 2001. Appendix F. <http://www.epa.gov/ttn/atw/sab/appendix-f.pdf>

Microenvironments generally include spaces such as outdoor (near source), indoor, and in-vehicle (while traveling along roadways), inside homes located with differing proximities to major and area sources, etc. HAPEM predicts concentrations in different microenvironments and calculates a time-weighted average, depending on the amount of time spent in each microenvironment.⁶⁵ The HAPEM includes both population activity pattern data and commuting pattern data.⁶⁶ Exposures were calculated for a range of activity patterns in microenvironments. Activity patterns include the amount of time spent in each microenvironment, along with activities during those times (sleeping, eating, sitting, etc.). Various demographic groups were defined by age, gender, or race, etc.⁶⁷

Phase IV: Estimating Potential Health Risks

Characterization of potential public health risks involved integrating available toxicity information and the population exposures to outdoor sources of toxic air pollutants that were estimated⁶⁸ in phase III using the current USEPA risk assessment and risk characterization guidelines.

⁶⁵ Pollutant concentrations within each microenvironment were estimated using ambient concentrations multiplied by a penetration factor, which is a ratio of indoor to outdoor concentration. A time-weighted average exposure concentration was predicted using these factors and the ambient concentration data for specified amounts of time.

⁶⁶ The commuting pattern data was based on a 1990 U.S. census that reports the number of individuals who work within the census tract where they live.

⁶⁷ USEPA selected 40 demographic population groups based on different combinations of characteristics (e.g., age, race, gender). For each of these groups, 365 activity patterns were randomly selected. The amount of time spent in each microenvironment (for eight separate time blocks in 24-hours) for each demographic group was then averaged for the entire set of 365. This process was repeated 100 times for each demographic group to derive typical exposure ranges. (USEPA. *Development of Microenvironmental Factors for the HAPEM4 in Support of the National-scale Air Toxics Assessment (NATA)*. External Review Draft. Prepared for the Office of Air Quality Planning and Standards by ICF Consulting and TRJ Environmental Inc., Research Triangle Park, NC, May 8, 2000).

⁶⁸ Although the NATA looked only at outdoor sources of air toxics, USEPA also is concerned about the risks to the public from toxic air pollutants indoors. The 1996 NATA stated intent to include an indoor emissions component in future NATAs but one was not included in the 1999 NATA.

NATA results

As noted, the NATA derived estimates of air toxics-associated health hazards for a range of demographic groups across most U.S. census tracts. The risk ranges are displayed primarily at county level geographic resolution as risk maps showing the medians of risk ranges. Note that the median is derived for each county in its entirety. For those toxic air pollutants whose concentrations are not uniform but instead follow concentration gradients, the display of the county median tends to dilute apparent risk in some areas and overstate it in others.

Non-cancer health hazards

NATA presents hazard indexes (HI), which are calculated using the sum of hazard quotients for substances that affect the same target organ or organ system, since hazard quotients may be combined for pollutants that cause adverse effects by the same toxicity mechanism. Because detailed information on toxic mechanisms was not available for most of the substances in NATA, USEPA combined the HQs of only eight pollutants (acetaldehyde, acrolein, acrylonitrile, arsenic, 1,3-dichloropropene, ethylene dibromide, formaldehyde, and trichloroethylene) as shown in figures 3-1, 3-2 and 3-3. The HI for respiratory irritation is only an approximation of the aggregate effect on the respiratory system (i.e., lungs and airways) because it is possible that some of the substances cause irritation by different mechanisms.

As with the hazard quotient, aggregate exposures below a HI of one will likely not result in adverse health effects over a lifetime of exposure. However, an HI greater than one (1.0) does not necessarily indicate the likelihood of adverse effects. Furthermore, the HI cannot be translated to a probability that adverse effects will occur, and may not be proportional to risk. A respiratory HI greater than one (1.0) can best be described as indicating that a potential may exist for irritation to the respiratory system.

The hazard indices based on median exposure concentrations in U.S. counties are shown in the map below (figure 3-1).

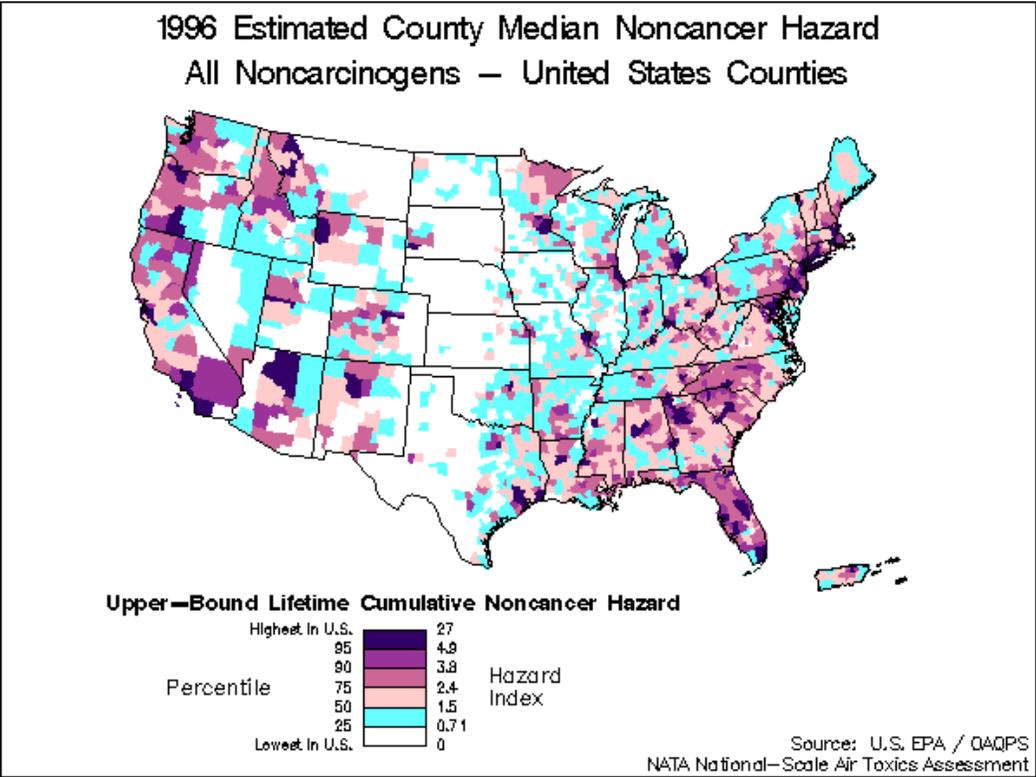


Figure 3-1. 1996 Median non-cancer hazards from all toxic air pollutants considered in NATA nationally.

Looking at Washington alone (Figure 3-2), we see much of the Interstate 5 corridor has elevated aggregate toxic air pollutant-associated irritation risk, lead by King and Clark Counties. Clark County has a hazard index in the worst 5% nationally.

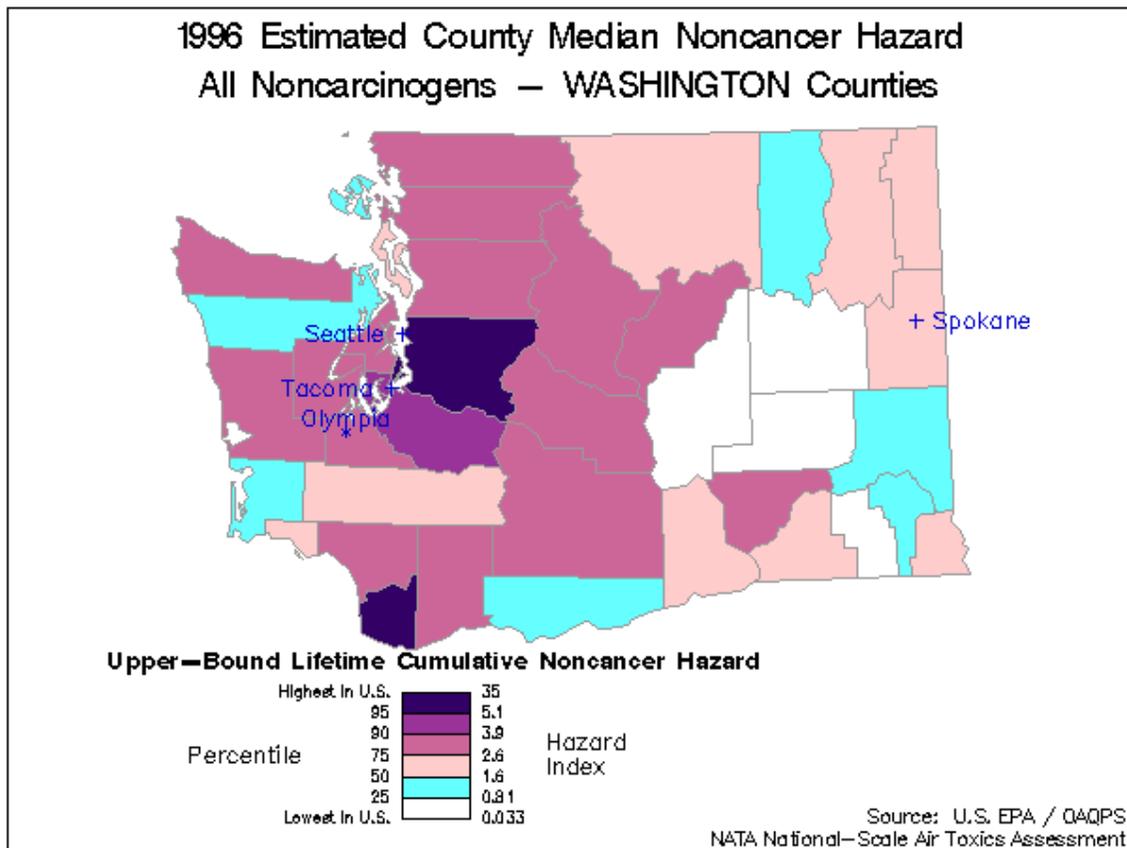


Figure 3-2. 1996 Median non-cancer hazards from all toxic air pollutants considered in NATA in Washington.

The figure 3-2 was revised by USEPA following the initial release of the NATA report. In the revised figure, apparent health hazards were lowered in some counties and the arbitrary hazard interval cut points used were lowered. The new map (reproduced below as figure 3-3) does not agree with the spreadsheet data of percentile distribution of hazard indexes across census tracts available on the 1996 NATA website.⁶⁹ A possible explanation is that in preparing the new map USEPA may have removed from the hazard index those toxic air pollutants whose primary health hazards were other than irritation.

⁶⁹ *non-cancer-hi-tract.zip* available at <http://www.epa.gov/ttn/atw/nata/ed/exporisk.html#aggb>

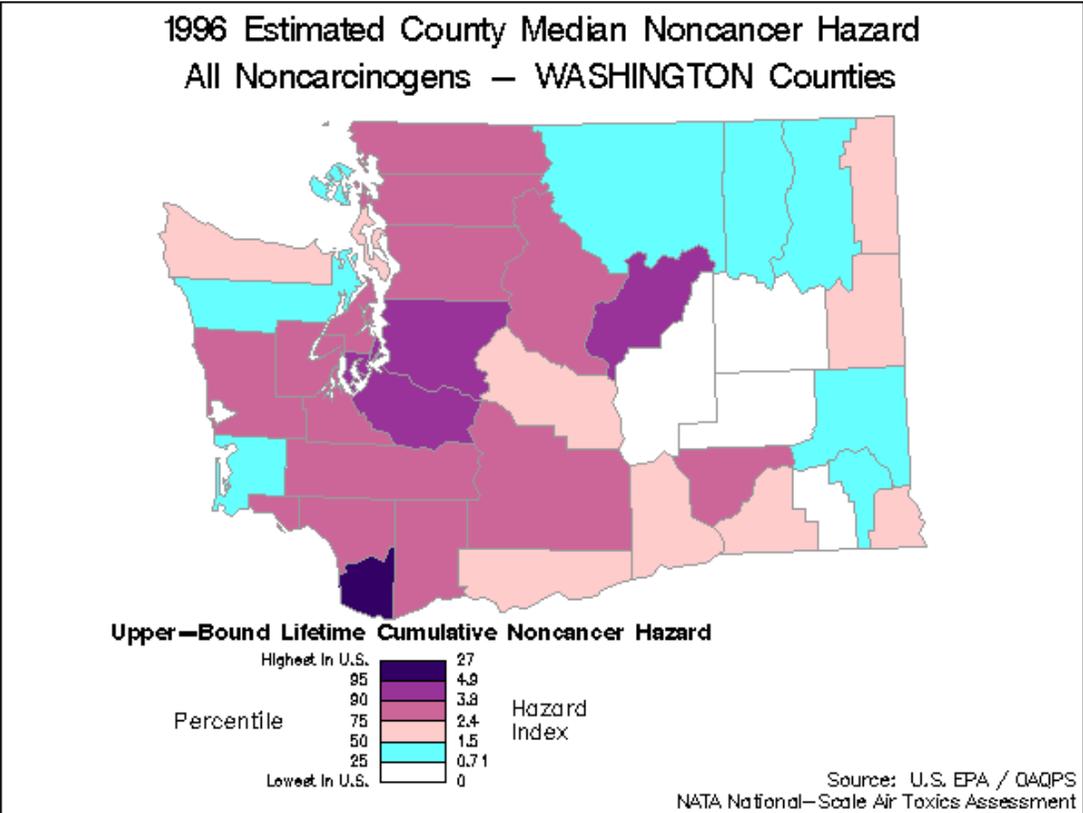


Figure 3-3. 1996 Median non-cancer hazards from all toxic air pollutants considered in NATA in Washington with revised intervals.

As noted, the non-cancer health hazards summed in the previous figures are the sum of HQs of each of the respiratory irritant chemicals. The only air pollutant that appears to present potential non-cancer health risks is acrolein. Acrolein accounts for ~86% of the hazard index followed by DPM at ~3%. Each of the other toxic air pollutants accounted for < 2% of the hazard.

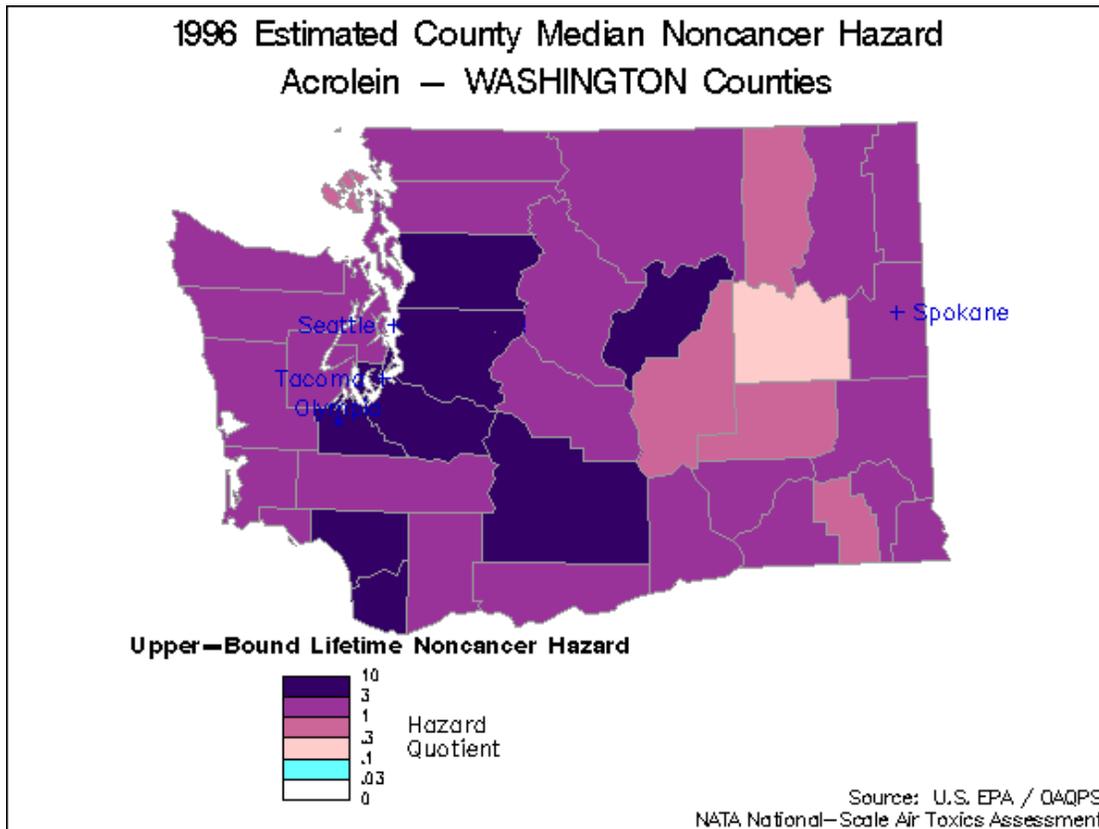


Figure 3-4. 1996 County median acrolein hazards in Washington.

Table 3-2. Regional and statewide acrolein hazard quotient distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	2.1	3.5	3.6	4.7	1152
BCAA	1.4	1.6	1.6	1.9	26
CRO	1.3	2.3	2.6	4.3	40
ERO	0.6	1.1	1.7	1.8	86
NWCAA	1.4	2.0	2.2	2.9	77
NWRO	0.7	0.9	1.0	1.0	6
ORCAA	1.6	2.9	2.9	3.8	101
PSCAA	3.5	4.2	4.6	5.1	579
SRCAA	1.8	2.0	2.0	2.1	99
SWCAA	3.1	4.6	4.7	5.9	105
YRCAA	2.0	3.0	2.8	3.6	33

None of the other toxic air pollutants assessed had hazard quotients of one or greater, including our calculations of those for DPM. USEPA presented the county-wide median DPM exposure in the map reproduced below (Figure 3-5) but did not calculate DPM HQs.

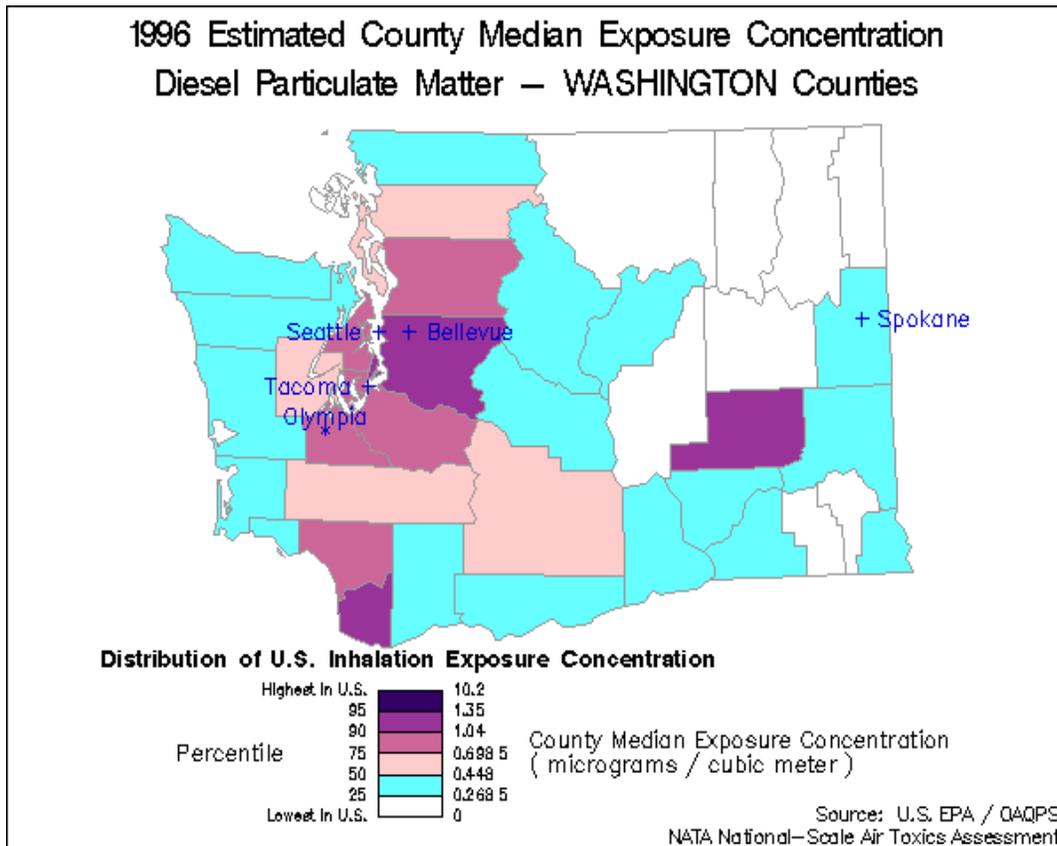


Figure 3-5. 1996 County median DPM exposures in Washington.

We therefore divided the concentration ranges expressed in this figure by California OEHHA's chronic reference exposure level for DPM ($5\text{-}\mu\text{g}/\text{m}^3$). This yielded chronic inhalation hazard quotients shown in the legend in figure 3-6.

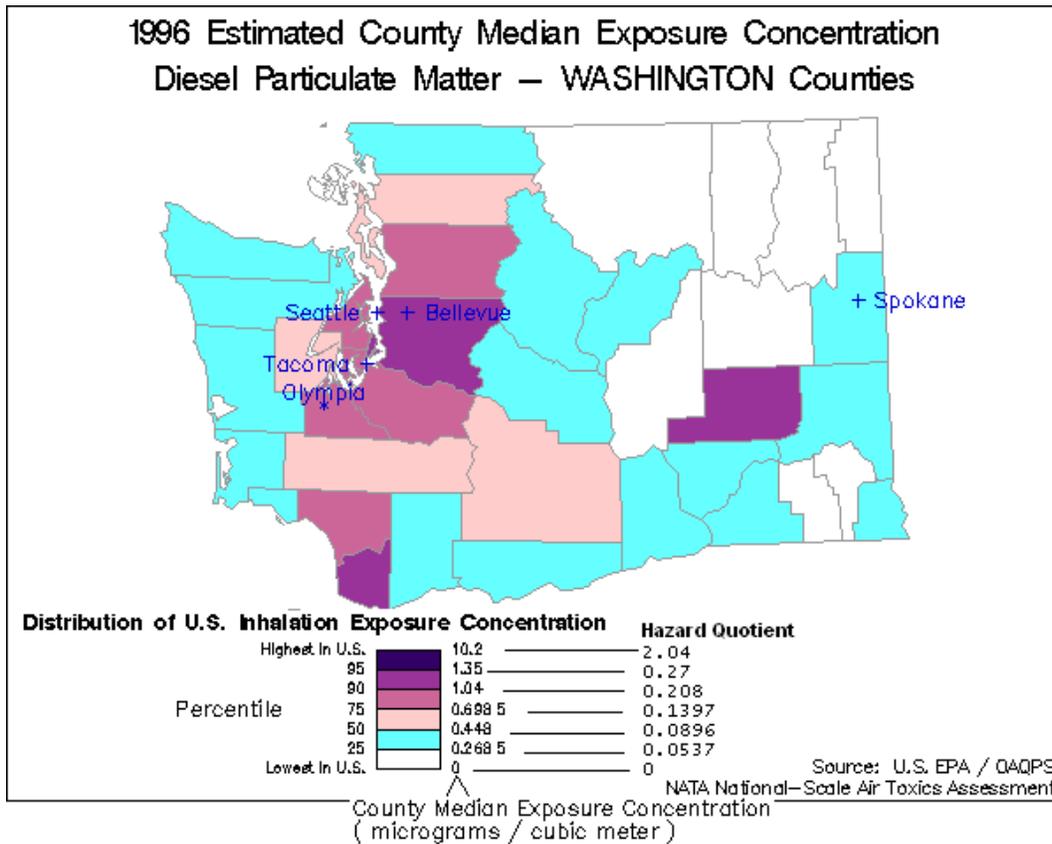


Figure 3-6. 1996 County median DPM hazard quotients.

All median HQs were below one (1.0). The non-cancer health hazards of diesel particulate matter are best viewed as only part of the fine particulate matter health threat complex.

Cancer risks

To numerically express potential cancer risk, exposure to a toxic air pollutant at a given ambient concentration may be compared to the concentration associated with some specific excess cancer risk. However, because cancer potency estimates incorporate protective assumptions in the face of uncertainty, exposure to a given concentration is an estimate of individual risk at that level. The cancer potency estimates used in NATA are generally at the upper bound of the plausible cancer potency range. This means they are likely to be more protective than corresponding mid-range estimates would be, but that they are within the likely range of true cancer potencies.

Actual exposures of most toxic air pollutants vary widely depending on wind direction, the location of people in the exposed population relative to toxic air pollutants sources, and other factors. Further, USEPA has stated that NATA probably underestimates actual exposures for most of the pollutants studied. Taking the possible over-protectiveness of the upper bound cancer potencies and the under-protectiveness of the exposure estimates together, NATA's median risk estimates are probably good enough to serve as guidance for toxic air pollutants prioritization efforts.

Because risk estimates are probabilities, cancer risks associated with different toxic air pollutants can be added together as long as each of the substances cause cancer by similar mechanisms. Addition of cancer risk estimates is appropriate only if the toxic air pollutants being added do not interact in ways that enhance or inhibit each other's carcinogenic potency. Had it been available, information on such non-additive interactions could have been considered in the NATA. Because no such information was identified, USEPA used the default assumption⁷⁰ that cancer risks from different toxic air pollutants could be added. The resulting median aggregate cancer risks from exposure to potentially carcinogenic toxic air pollutants (not including DPM), across the USA, are shown in figure 3-7.

⁷⁰ As recommended in USEPA (1986) *Guidelines for the Health Risk Assessment of Chemical Mixtures*, 52 FR 34014-34025.

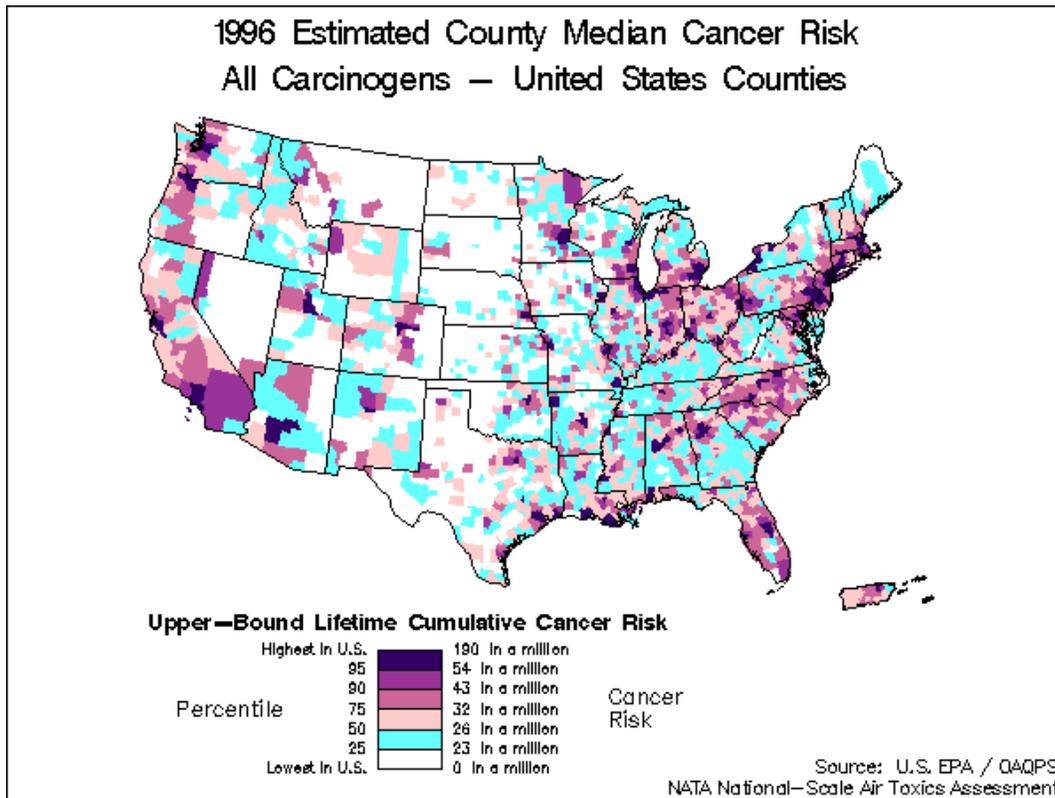


Figure 3-7. 1996 Aggregate county median cancer risks from the 29 potentially carcinogenic air pollutants in NATA nationally.

County-by-county median aggregate cancer risks from inhalation exposure to potentially carcinogenic toxic air pollutants, excluding DPM, are shown in figure 3-8. Interquartile risk ranges are shown in table 3-3.

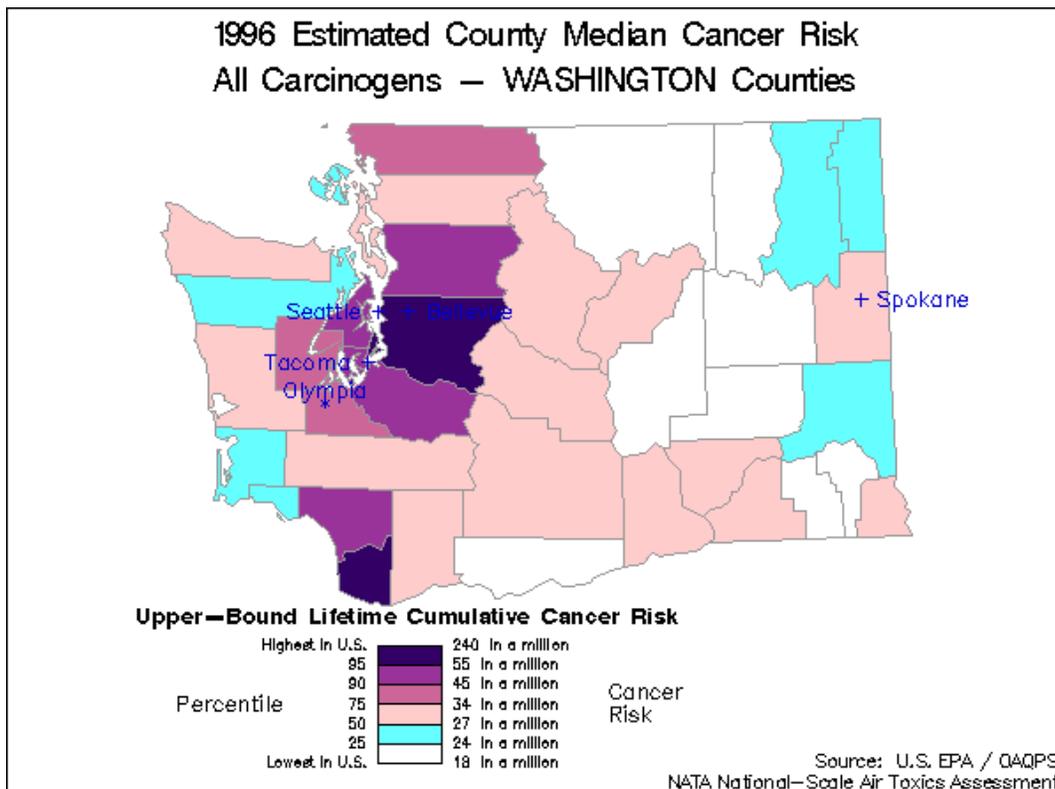


Figure 3-8. 1996 Aggregate county median cancer risks from the 29 potentially carcinogenic air pollutants in NATA in Washington.

Table 3-3. Regional air pollutants-associated (without DPM) cancer risk distributions.

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	2.9E ⁻⁵	4.2E-5	4.5E ⁻⁵	5.7E ⁻⁵	1152
BCAA	2.6E ⁻⁵	2.9E-5	2.8E ⁻⁵	3.1E ⁻⁵	26
CRO	2.3E ⁻⁵	2.5E-5	2.7E ⁻⁵	3.1E ⁻⁵	40
ERO	2.2E ⁻⁵	2.4E-5	2.6E ⁻⁵	2.7E ⁻⁵	86
NWCAA	2.7E ⁻⁵	3.0E-5	3.1E ⁻⁵	3.5E ⁻⁵	77
NWRO	2.3E ⁻⁵	2.4E-5	2.4E ⁻⁵	2.5E ⁻⁵	6
ORCAA	2.5E ⁻⁵	3.1E-5	3.1E ⁻⁵	3.6E ⁻⁵	101
PSCAA	4.5E ⁻⁵	5.4E-5	5.7E ⁻⁵	6.5E ⁻⁵	579
SRCAA	3.0E ⁻⁵	3.2E-5	3.2E ⁻⁵	3.4E ⁻⁵	99
SWCAA	3.3E ⁻⁵	4.6E-5	5.1E ⁻⁵	6.0E ⁻⁵	105
YRCAA	2.6E ⁻⁵	3.1E-5	3.1E ⁻⁵	3.5E ⁻⁵	33

Estimated plausible upper limit cancer risks

NATA calculations for individual toxic air pollutants are shown in the following text and figures, arranged in decreasing order of risk.

Diesel particulate matter

NATA did not include cancer risk estimates for DPM exposure; however, we applied the California ARB SRB’s URE for DPM ($3E^{-4}/\mu\text{g}/\text{m}^3$) to the NATA estimates of DPM exposure. The cancer risk estimates that correspond to the exposure concentration medians are shown in the legend of figure 3-9.

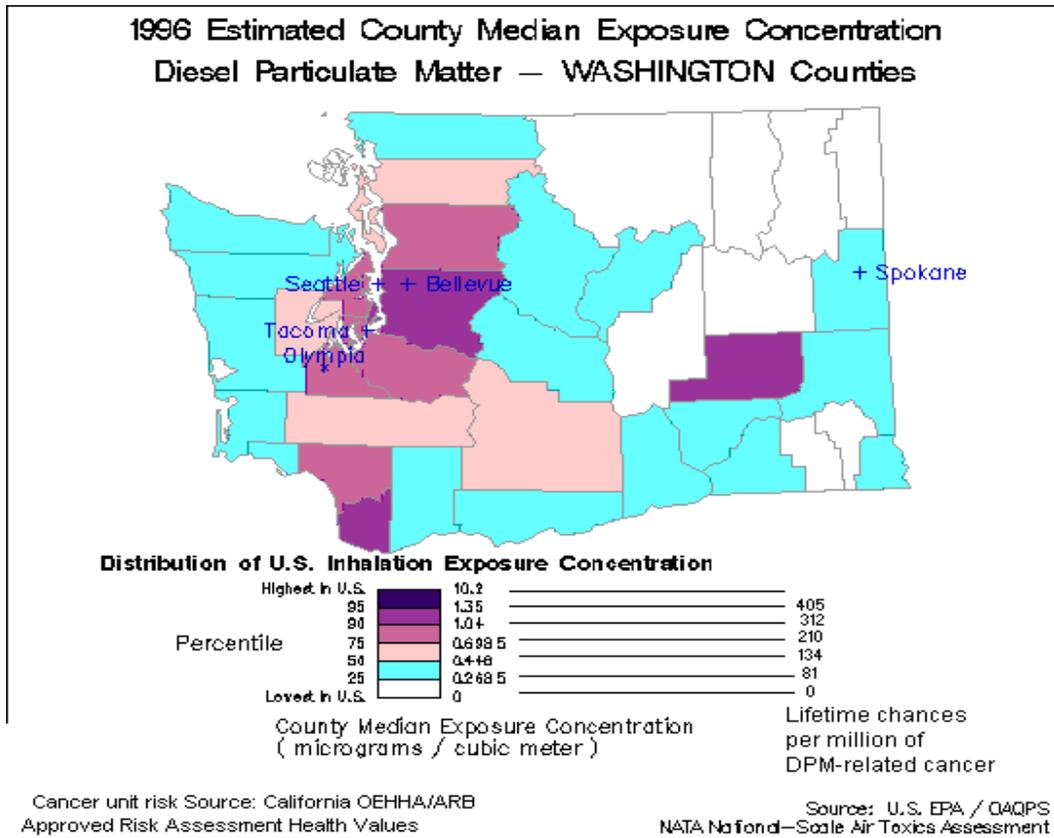


Figure 3-9. 1996 DPM median county exposure concentrations with corresponding upper-bound lifetime excess cancer risks (based on the CARB SRB’s URE for DPM).

The cancer risk statistics we calculated using NATA DPM exposure estimates and SAB’s DPM URE are shown in Table 3-4.

Table 3-4. Regional and statewide DPM cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	1.3E ⁻⁴	2.57E ⁻⁴	2.53E ⁻⁴	3.38E ⁻⁴	1152

BCAA	1.0E ⁻⁴	1.1E ⁻⁴	1.2E ⁻⁴	1.3E ⁻⁴	26
CRO	5.9E ⁻⁵	8.8E ⁻⁵	9.6E ⁻⁵	1.2E ⁻⁴	40
ERO	4.7E ⁻⁵	6.9E ⁻⁵	9.3E ⁻⁵	1.2E ⁻⁴	86
NWCAA	1.2E ⁻⁴	1.6E ⁻⁴	1.4E ⁻⁴	1.7E ⁻⁴	77
NWRO	7.0E ⁻⁵	7.6E ⁻⁵	7.7E ⁻⁵	8.6E ⁻⁵	6
ORCAA	1.1E ⁻⁴	1.5E ⁻⁴	1.7E ⁻⁴	2.1E ⁻⁴	101
PSCAA	2.7E ⁻⁴	3.2E ⁻⁴	3.4E ⁻⁴	3.8E ⁻⁴	579
SRCAA	1.1E ⁻⁴	1.2E ⁻⁴	1.2E ⁻⁴	1.3E ⁻⁴	99
SWCAA	2.2E ⁻⁴	3.4E ⁻⁴	3.4E ⁻⁴	4.2E ⁻⁴	105
YRCAA	1.2E ⁻⁴	1.6E ⁻⁴	1.6E ⁻⁴	1.9E ⁻⁴	33

Estimated plausible upper limit cancer risks

As noted previously, UREs are intended to be used for calculating upper bound cancer risks for an average individual. Limiting the interpretation of risk in this way is intended to cope with uncertainties in UREs. However, for the sake of estimating public health benefits that might be gained by reducing DPM emissions, we used the CARB and USEPA ORD DPM URE ranges to calculate the plausible upper limit (PUL) possible number of cancer cases resulting from DPM exposures. The results are shown in table 3-5.

Table 3-5. DPM unit risk estimate and ranges

	CARB SRB			USEPA ORD	
	URE	Range		Range	
	3E ⁻⁴ per µg/m ³	1.3E ⁻⁴ per µg/ m ³	2.4E ⁻³ per µg/ m ³	1E ⁻⁵ per µg/ m ³	1E ⁻³ per µg/ m ³
Average WA Risk (estimated PUL cancer risk per million)	253	110	2023	8	843
Number of Washington census tracts with PUL risk over 1E ⁻⁶	100%	100%	100%	98.6%	100%
Excess PUL risk per million in the most exposed census tract *	906	393	7248	30	3020

*The census tract with maximum risk is located in Pierce County.

Table 3-6 shows the results on the ranking of DPM relative to the other air pollutants by using the different DPM UREs presented in table 3-5. The proportion of cancer risk from DPM (as a percentage relative to the total toxic air pollutant-associated cancer risk) is shown. In addition, the table shows the estimate of the plausible upper limit to the true value of cancer risk using the CARB URE: as many as 1274 people in the state may develop cancer during their lifetimes

because of continuous DPM exposure, at 1996 levels. The range of UREs proposed by the CARB SRB was $1.3E^{-4}$ per $\mu\text{g}/\text{m}^3$ to $2.4E^{-3}$ per $\mu\text{g}/\text{m}^3$. Applying these UREs to the NATA exposure estimates across Washington's census tracts yields plausible upper limit excess cancer case estimates of 552 to 10,188 resulting from lifetime DPM exposure. Similarly, the $1E^{-5}$ per $\mu\text{g}/\text{m}^3$ to $1E^{-3}$ per $\mu\text{g}/\text{m}^3$ URE range proposed by USEPA ORD yields plausible upper limit estimates ranging from 42 to 4245 Washingtonians possibly developing cancer during their lifetimes as a result of DPM exposure. To put this in context of the current causes of death in Washington, keep in mind that DPM is associated mainly with lung cancer, which has a five-year survival rate of only 15%.⁷¹ Thus for comparison, the possible plausible upper limit of the range of numbers of new lung cancer cases could be from 1 to 146 per year (based on NATA exposure estimates in Washington and the overlapping USEPA ORD and CARB risk ranges), leading to plausible upper limits of between 0.85 to 124.1 deaths per year within five years of diagnosis.

Table 3-6. DPM cancer risk range (based on the 1996 NATA exposure estimates and CARB or USEPA URE estimate ranges).

	DPM URE	Proportion of Risk from DPM	Rank of DPM among the all of the studied air pollutants
CARB SRB high end of unit risk range	0.0024	98%	1
CARB SRB point URE	0.0003	85%	1
CARB SRB low end of unit risk range	0.00013	71%	1
USEPA ORD high end of unit risk range	0.001	95%	1
USEPA ORD low end of unit risk range	0.00001	16%	4*

*At the lowest end of the USEPA ORD risk range, DPM would rank 4th behind 1st place benzene; 2nd place carbon tetrachloride; and 3rd place formaldehyde.

NATA results are most meaningfully interpreted when viewed over population covering large geographic areas. The accuracy and resolution of the data is uncertain and can be contradictory or at the county/local level, as evidenced by the unexpectedly high DPM concentration estimate in Adams County and the unexpectedly low estimate for Spokane County. In addition, confidence in the accuracy of DPM cancer risk estimates would be greater if they were based on more certain exposure cancer potency estimates. Further, estimates of new cancer cases assume

⁷¹ http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_are_the_key_statistics_for_lung_cancer_26.asp

continuous levels of exposure to DPM; however, the levels are not expected to remain as high as in 1996 due to planned changes in fuel and emissions controls. Thus, the number of people who develop cancer resulting from DPM exposure in Washington between 1996 and 2066 (70 years) is likely to be less than 10,188.

Estimated additional cancer risk from exposure to DPM decreased slightly between 1996 and 1999 due to diminished emissions from on-road and non-road engines and consequently lower population exposures as estimates in the NATAs published for these years. Statewide distribution of excess cancer risk is shown in figure 3-10.

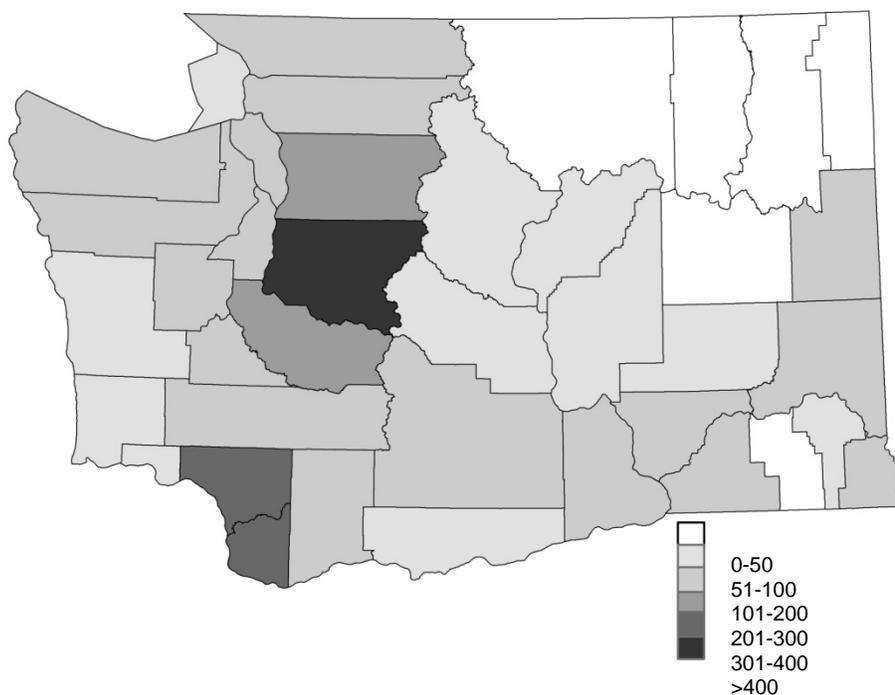


Figure 3-10. DPM exposure-associated additional cancer risks (estimated PUL cancer risk per million) by county at 1999 exposure levels.

Benzene

Inhalation exposure to benzene accounts for 3 to 4% of the calculated total toxic air pollutants cancer risk to Washington citizens. The USEPA's 1996 NATA estimate of cancer risk from median benzene inhalation exposure indicates an excess cancer risk of 11 per million in Washington: ranging from 4 to 17 per million across regions. Additional cancer risk exceeds the *de minimis* level in all Washington census tracts (figure 3-11 and table 3-7). Conversely, in

terms of non-cancer health risks, the NATA estimates of benzene population exposure suggest insignificant inhalation-route health hazards throughout the state.

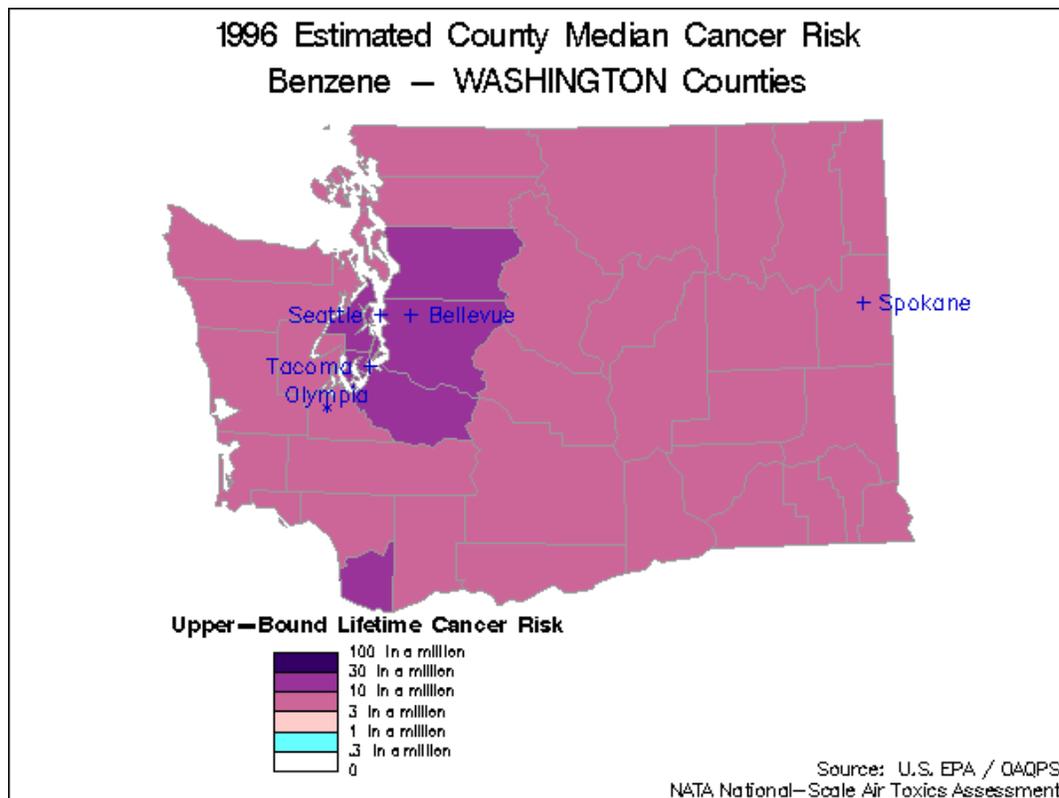


Figure 3-11. 1996 County median cancer risks from benzene in Washington.

Table 3-7. Regional and statewide benzene cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	5.9E ⁻⁶	1.1E ⁻⁵	1.2E ⁻⁵	1.7E ⁻⁵	1152
BCAA	4.9E ⁻⁶	5.75E ⁻⁶	5.6E ⁻⁶	6.3E ⁻⁶	26
CRO	3.7E ⁻⁶	4.3E ⁻⁶	4.7E ⁻⁶	5.6E ⁻⁶	40
ERO	3.6E ⁻⁶	4.1E ⁻⁶	4.4E ⁻⁶	4.6E ⁻⁶	86
NWCAA	5.4E ⁻⁶	6.1E ⁻⁶	6.4E ⁻⁶	7.6E ⁻⁶	77
NWRO	4.4E ⁻⁶	4.6E ⁻⁶	4.6E ⁻⁶	4.7E ⁻⁶	6
ORCAA	4.7E ⁻⁶	6.3E ⁻⁶	6.7E ⁻⁶	8.5E ⁻⁶	101
PSCAA	1.4E ⁻⁵	1.7E ⁻⁶	1.7E ⁻⁵	1.9E ⁻⁶	579
SRCAA	5.8E ⁻⁶	6.5E ⁻⁶	6.4E ⁻⁶	6.9E ⁻⁶	99
SWCAA	6.5E ⁻⁶	1.0E ⁻⁵	1.0E ⁻⁵	1.4E ⁻⁵	105
YRCAA	4.6E ⁻⁶	5.8E ⁻⁶	5.7E ⁻⁶	6.4E ⁻⁶	33

Estimated plausible upper limit cancer risks

Carbon tetrachloride

The USEPA's 1996 NATA estimate of inhalation median exposure cancer risk from CCl₄ indicates an excess cancer risk of 9.6 per million in Washington (Figure 3-12 and Table 3-8). In terms of non-cancer health risks, NATA estimates that the median exposure to CCl₄ presented is an insignificant hazard throughout the state.

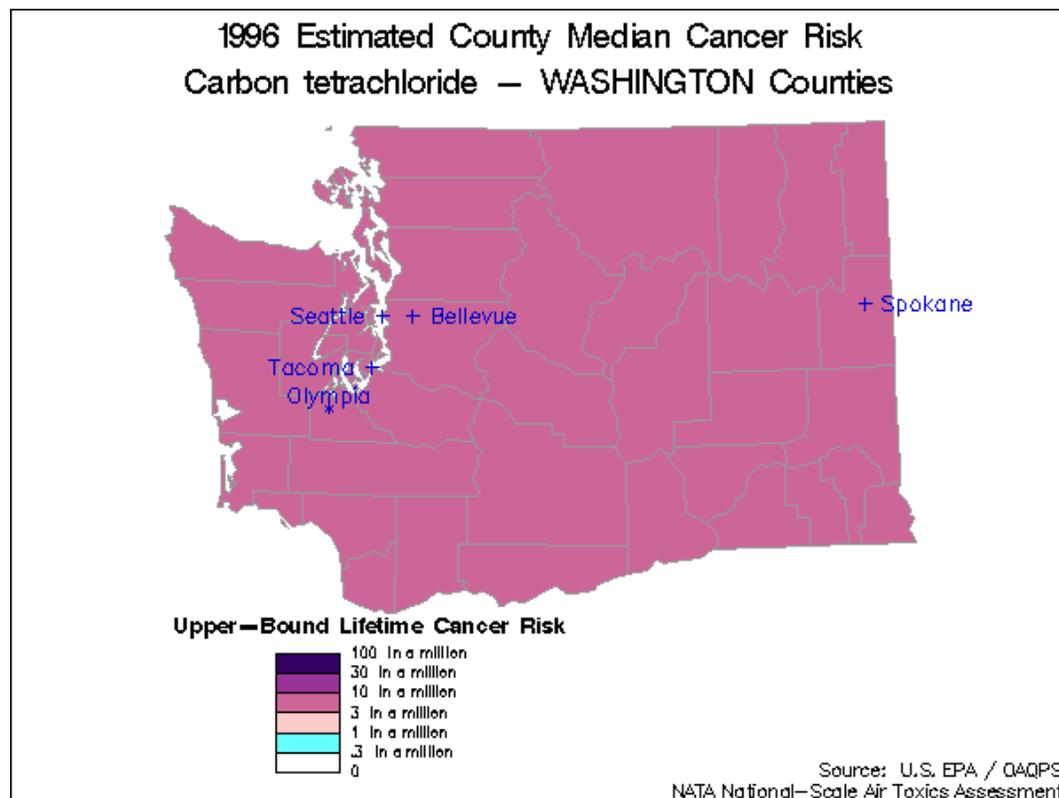


Figure 3-12. 1996 County median cancer risks from carbon tetrachloride in Washington.

Table 3-8. Regional and statewide carbon tetrachloride cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	9.5E ⁻⁶	9.6E ⁻⁶	9.5E ⁻⁶	9.7E ⁻⁶	1152
BCAA	9.5E ⁻⁶	9.6E ⁻⁶	9.2E ⁻⁶	9.7E ⁻⁶	26
CRO	9.6E ⁻⁶	9.6E ⁻⁶	9.6E ⁻⁶	9.7E ⁻⁶	40
ERO	9.5E ⁻⁶	9.6E ⁻⁶	9.5E ⁻⁶	9.7E ⁻⁶	86
NWCAA	9.5E ⁻⁶	9.6E ⁻⁶	9.1E ⁻⁶	9.7E ⁻⁶	77
NWRO	9.6E ⁻⁶	9.6E ⁻⁶	9.6E ⁻⁶	9.6E ⁻⁶	6
ORCAA	9.5E ⁻⁶	9.6E ⁻⁶	9.4E ⁻⁶	9.7E ⁻⁶	101
PSCAA	9.5E ⁻⁶	9.6E ⁻⁶	9.5E ⁻⁶	9.7E ⁻⁶	579
SRCAA	9.5E ⁻⁶	9.6E ⁻⁶	9.5E ⁻⁶	9.6E ⁻⁶	99
SWCAA	9.6E ⁻⁶	9.6E ⁻⁶	9.6E ⁻⁶	9.6E ⁻⁶	105
YRCAA	9.5E ⁻⁶	9.6E ⁻⁶	9.6E ⁻⁶	9.7E ⁻⁶	33

Estimated plausible upper limit cancer risks

Formaldehyde

The USEPA's 1996 NATA estimates of cancer risks from the formaldehyde inhalation exposure level implies a median excess cancer risk of 8.6 per million throughout Washington (Figure 3-13). Risk exceeds the *de minimis* level in all Washington census tracts (Table 3-9). The risk is highest in Clark, King and Pierce Counties. The NATA estimates of inhalation exposure to formaldehyde yielded low HQs statewide and in each LAA region. Thus, ambient level exposures throughout the state appeared unlikely to result in non-cancer health risks.

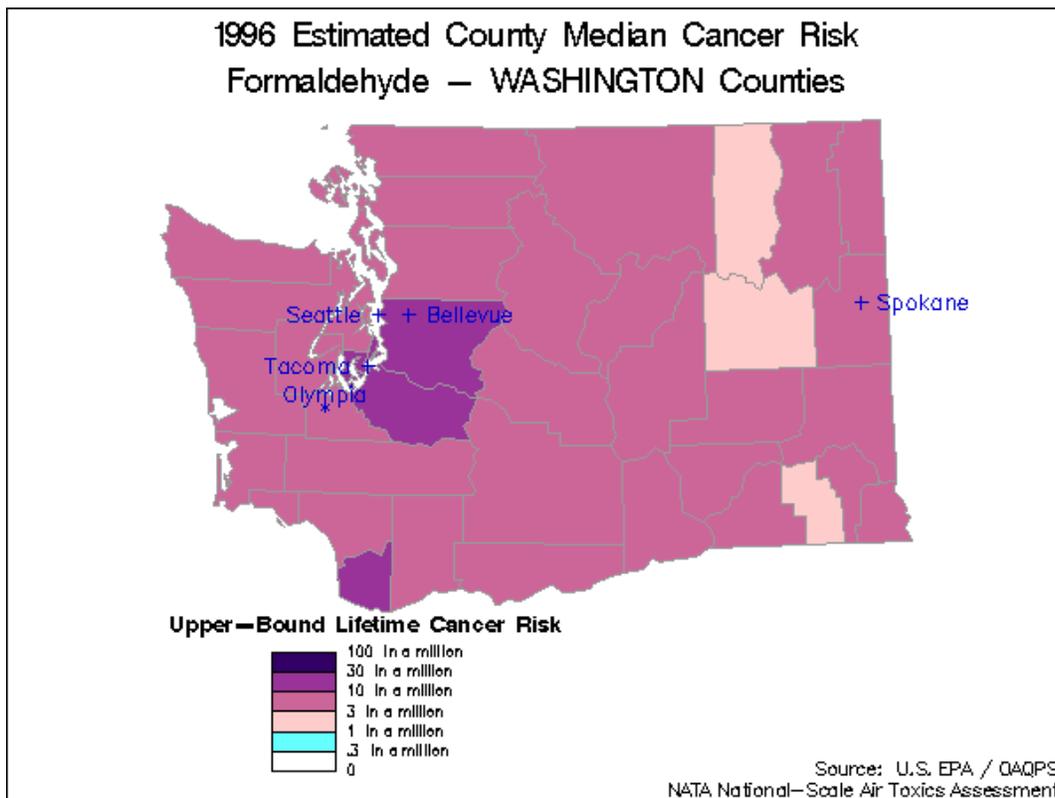


Figure 3-13. 1996 County median cancer risks from formaldehyde in Washington.

Table 3-9. Regional and statewide formaldehyde cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	5.9E ⁻⁶	8.6E ⁻⁶	8.8E ⁻⁶	1.1E ⁻⁵	1152
BCAA	4.5E ⁻⁶	4.8E ⁻⁶	4.7E ⁻⁶	5.4E ⁻⁶	26
CRO	3.6E ⁻⁶	4.7E ⁻⁶	5.2E ⁻⁶	6.7E ⁻⁶	40
ERO	3.2E ⁻⁶	3.8E ⁻⁶	4.5E ⁻⁶	4.9E ⁻⁶	86

NWCAA	4.8E ⁻⁶	5.7E ⁻⁶	5.7E ⁻⁶	7.0E ⁻⁶	77
NWRO	3.5E ⁻⁶	3.7E ⁻⁶	4.0E ⁻⁶	4.0E ⁻⁶	6
ORCAA	4.5E ⁻⁶	6.8E ⁻⁶	6.5E ⁻⁶	8.2E ⁻⁶	101
PSCAA	9.0E ⁻⁶	1.0E ⁻⁵	1.1E ⁻⁵	1.2E ⁻⁵	579
SRCAA	5.7E ⁻⁶	6.0E ⁻⁶	6.1E ⁻⁶	6.3E ⁻⁶	99
SWCAA	6.6E ⁻⁶	1.1E ⁻⁵	1.1E ⁻⁵	1.5E ⁻⁵	105
YRCAA	4.9E ⁻⁶	6.6E ⁻⁶	6.5E ⁻⁶	7.4E ⁻⁶	33

Estimated plausible upper limit cancer risks

Polycyclic organic matter

On average POM inhalation exposure accounts for 1.3% of the calculated toxic air pollutant-associated cancer risk to Washington citizens. The median statewide POM-associated excess cancer risk is 2.6 per million (figure 3-14). Total POM-associated cancer risk exceeds the *de minimis* level in 78% of Washington's census tracts (table 3-10). In terms of non-cancer health risks, no RfC or RfC-like criterion was available for the total-POM group so the NATA did not evaluate its non-cancer health hazards.

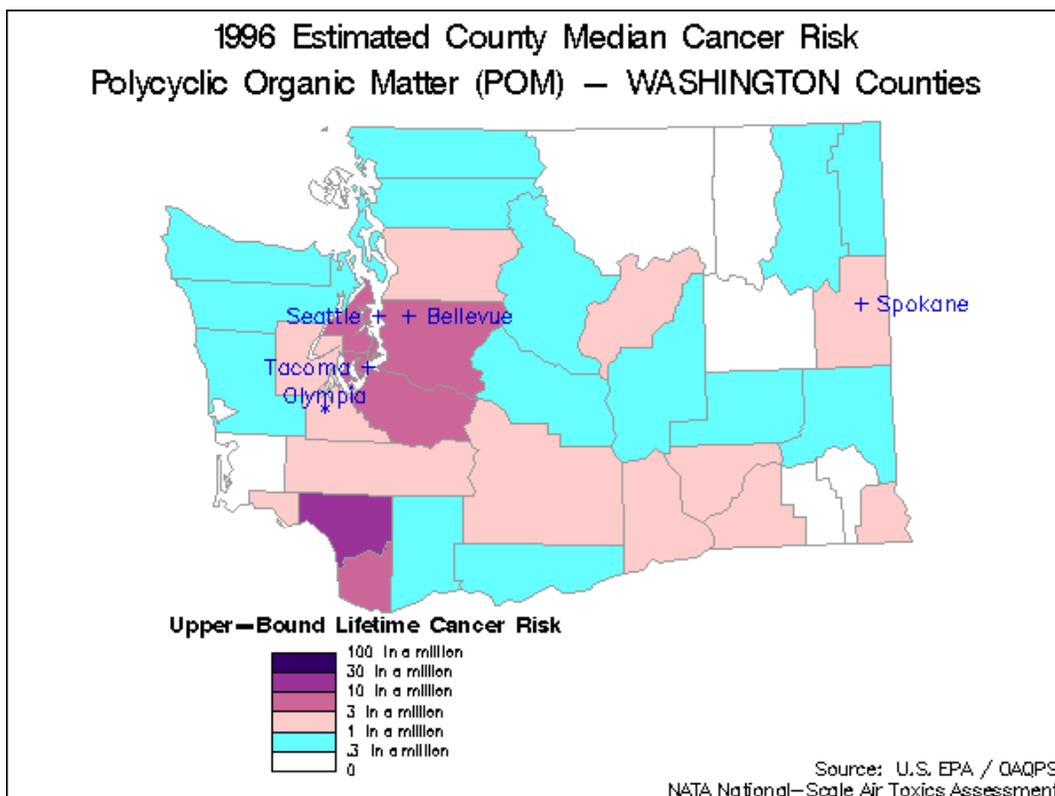


Figure 3-14. 1996 County median cancer risks from POM in Washington.

Table 3-10. Regional and statewide polycyclic organic matter cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	1.2E ⁻⁶	2.6E ⁻⁶	3.8E ⁻⁶	4.7E ⁻⁶	1152
BCAA	8.0E ⁻⁷	1.5E ⁻⁶	1.6E ⁻⁶	2.0E ⁻⁶	26
CRO	2.5E ⁻⁷	4.9E ⁻⁷	1.1E ⁻⁶	1.9E ⁻⁶	40
ERO	1.8E ⁻⁷	4.4E ⁻⁷	8.7E ⁻⁷	1.2E ⁻⁶	86
NWCAA	5.4E ⁻⁷	7.3E ⁻⁷	1.1E ⁻⁶	1.8E ⁻⁶	77
NWRO	1.5E ⁻⁷	2.0E ⁻⁷	2.3E ⁻⁷	2.4E ⁻⁷	6
ORCAA	4.4E ⁻⁷	1.1E ⁻⁶	1.3E ⁻⁶	1.8E ⁻⁶	101
PSCAA	2.6E ⁻⁶	4.0E ⁻⁶	5.2E ⁻⁶	7.0E ⁻⁶	579
SRCAA	1.4E ⁻⁶	2.0E ⁻⁶	1.9E ⁻⁶	2.5E ⁻⁶	99
SWCAA	2.1E ⁻⁶	4.6E ⁻⁶	7.5E ⁻⁶	7.5E ⁻⁶	105
YRCAA	7.2E ⁻⁷	1.9E ⁻⁶	2.1E ⁻⁶	3.0E ⁻⁶	33

Estimated plausible upper limit cancer risks

Excess cancer risk estimates in individual census tracts for 7-PAH were not published by USEPA on the 1996 NATA website, unlike the other toxic air pollutants studied. Because this information was missing, no statewide average risk estimate is available. Also we could not report risk percentiles for the LAAs; however, a figure in the 1996 NATA (figure 3-15) does give estimates of cancer risk from median 7-PAH inhalation exposure by county.

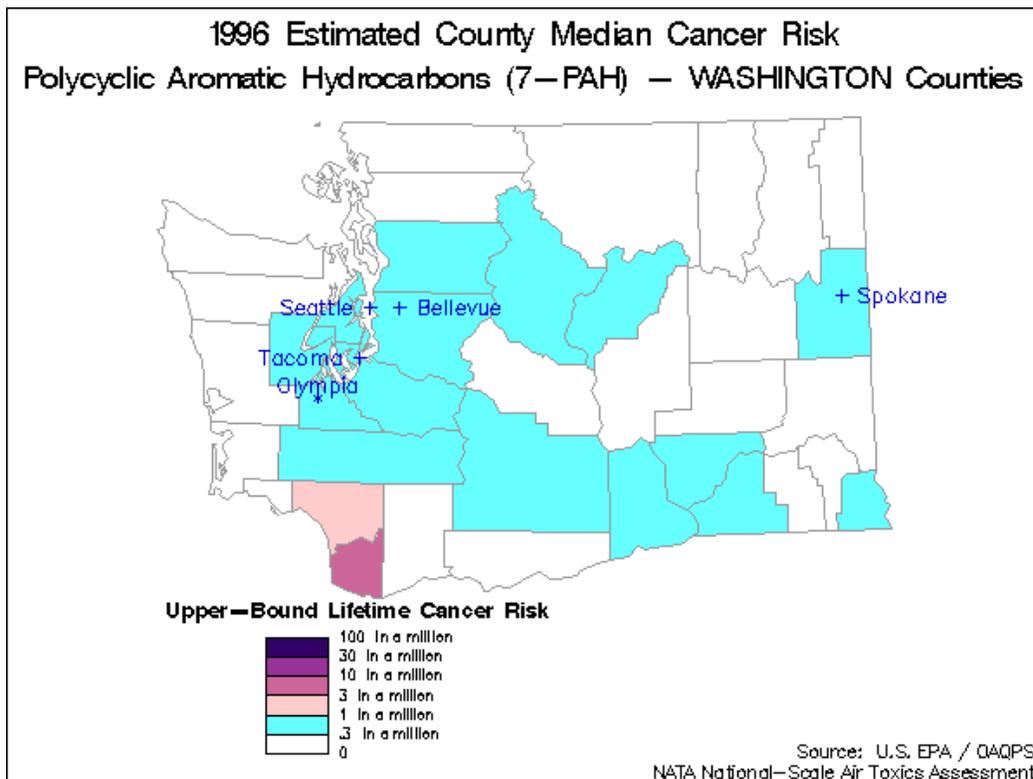


Figure 3-15. 1996 County median cancer risks from polycyclic aromatic hydrocarbons (7-PAH) in Washington.

At the time this report was drafted, 7-PAH did not have an RfC (or RfC-like) value so non-cancer effects hazards were not evaluated in the NATA.

Chromium and chromium compounds

Inhalation exposure to chromium accounts for ~0.8% of the calculated total toxic air pollutants cancer risks for an average Washington citizen. In the 1996 NATA, the estimates of cancer risk from exposure in Washington indicate a median excess cancer risk of 1.5 per million (table 3-11). The risk was lower than one-in-a-million in most of Washington's counties but greater one-in-a-million in PSCAA and SWCAA (figure 3-16). In terms of non-cancer health risks, the NATA estimates exposure to chromium suggested insignificant inhalation health hazards throughout the state.

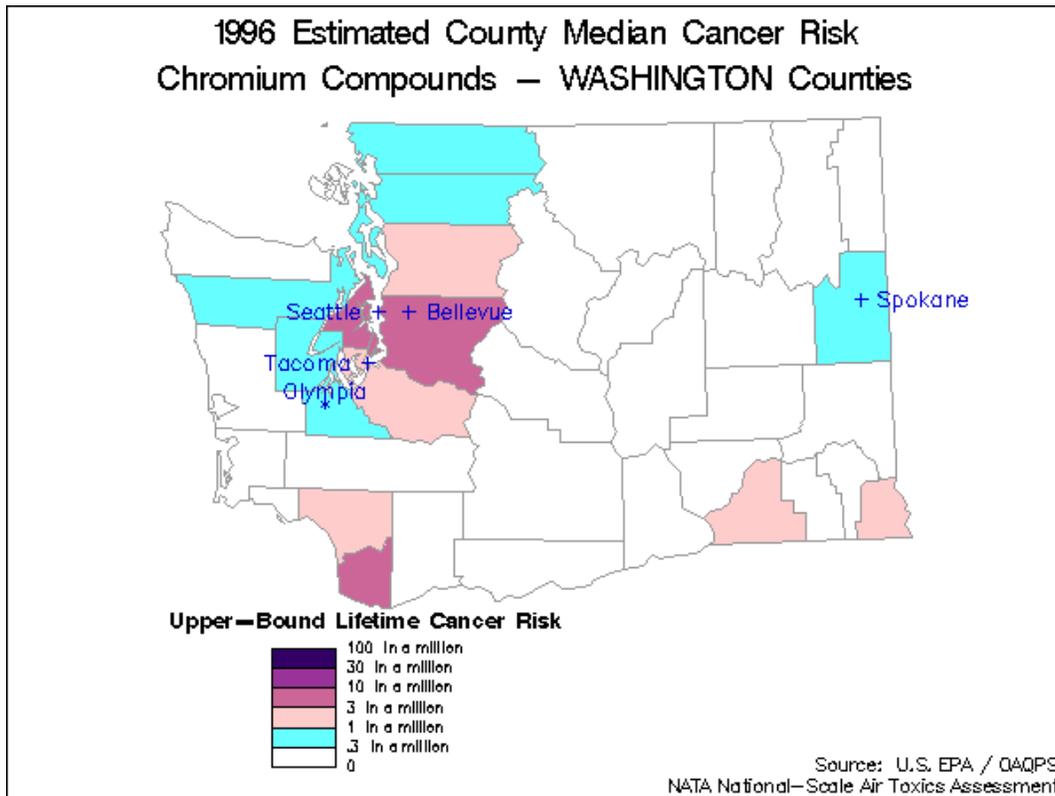


Figure 3-16. 1996 County median cancer risks from chromium and its compounds in Washington.

Table 3-11. Regional and statewide chromium cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	3.7E ⁻⁷	1.5E ⁻⁶	2.3E ⁻⁶	3.1E ⁻⁶	1152
BCAA	2.0E ⁻⁷	2.7E ⁻⁷	2.4E ⁻⁷	3.0E ⁻⁷	26
CRO	2.0E ⁻⁸	5.5E ⁻⁸	1.2E ⁻⁷	2.2E ⁻⁷	40
ERO	2.6E ⁻⁸	10.0E ⁻⁸	4.6E ⁻⁷	2.8E ⁻⁷	86
NWCAA	3.4E ⁻⁷	5.3E ⁻⁷	8.5E ⁻⁷	7.8E ⁻⁷	77
NWRO	4.9E ⁻⁸	8.3E ⁻⁸	7.3E ⁻⁸	9.4E ⁻⁸	6
ORCAA	1.0E ⁻⁷	2.9E ⁻⁷	3.2E ⁻⁷	4.0E ⁻⁷	101
PSCAA	1.9E ⁻⁶	2.8E ⁻⁶	3.6E ⁻⁶	4.0E ⁻⁶	579
SRCAA	5.8E ⁻⁷	8.3E ⁻⁷	9.58E ⁻⁷	1.2E ⁻⁶	99
SWCAA	8.9E ⁻⁷	2.2E ⁻⁶	2.9E ⁻⁶	3.8E ⁻⁶	105
YRCAA	1.0E ⁻⁷	2.0E ⁻⁷	2.5E ⁻⁷	3.7E ⁻⁷	33

Estimated plausible upper limit cancer risks

Chloroform

Inhalation exposure to chloroform accounts for less than 0.6% of the calculated toxic air pollutants cancer risk to Washington citizens. At its nearly constant background global concentration of $0.083\text{-}\mu\text{g}/\text{m}^3$, chloroform presents an individual excess risk of 1.6 per million. The risk is slightly higher than average in NWCAA where the median was 1.8 per million (table 3-12). All census tracts exceeded the *de minimis* risk level (figure 3-17). In terms of non-cancer health risks, the NATA estimates of exposure suggested insignificant chloroform inhalation health hazards throughout the state.

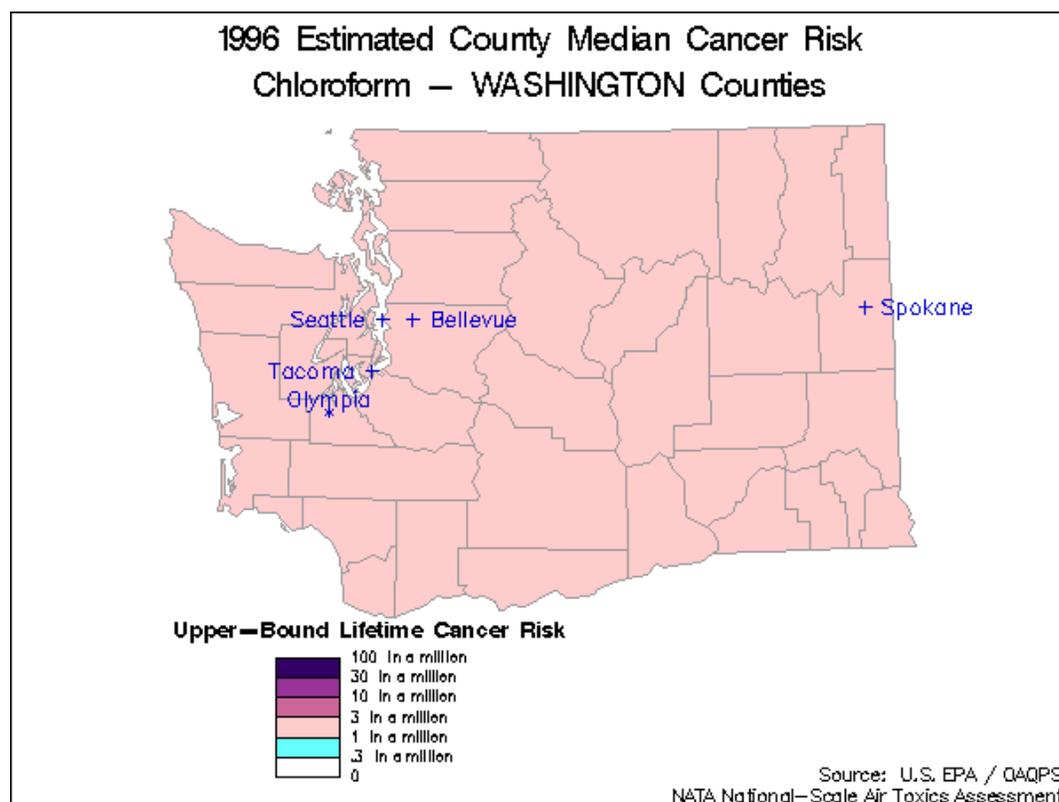


Figure 3-17. 1996 County median cancer risks from chloroform in Washington.

Table 3-12. Regional and statewide chloroform cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	$1.6\text{E-}6$	$1.6\text{E-}6$	$1.6\text{E-}6$	$1.6\text{E-}6$	1152
BCAA	$1.6\text{E-}6$	$1.6\text{E-}6$	$1.5\text{E-}6$	$1.6\text{E-}6$	26
CRO	$1.6\text{E-}6$	$1.6\text{E-}6$	$1.6\text{E-}6$	$1.6\text{E-}6$	40
ERO	$1.6\text{E-}6$	$1.6\text{E-}6$	$1.5\text{E-}6$	$1.6\text{E-}6$	86

NWCAA	1.6E ⁻⁶	1.8E ⁻⁶	2.8E ⁻⁶	2.1E ⁻⁶	77
NWRO	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	6
ORCAA	1.6E ⁻⁶	1.6E ⁻⁶	1.5E ⁻⁶	1.6E ⁻⁶	101
PSCAA	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	579
SRCAA	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	99
SWCAA	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	105
YRCAA	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	1.6E ⁻⁶	33

Estimated plausible upper limit cancer risks

Ethylene dichloride

The NATA estimate of median EDC inhalation exposure indicated a uniform excess cancer risk of 1.3 per million throughout Washington (figure 3-18 and table 3-13). EDC accounts for 0.45% of the total statewide toxic air pollutants cancer risk. In terms of non-cancer health risks, the NATA estimates of the EDC inhalation exposures suggest these hazards were insignificant throughout the state.

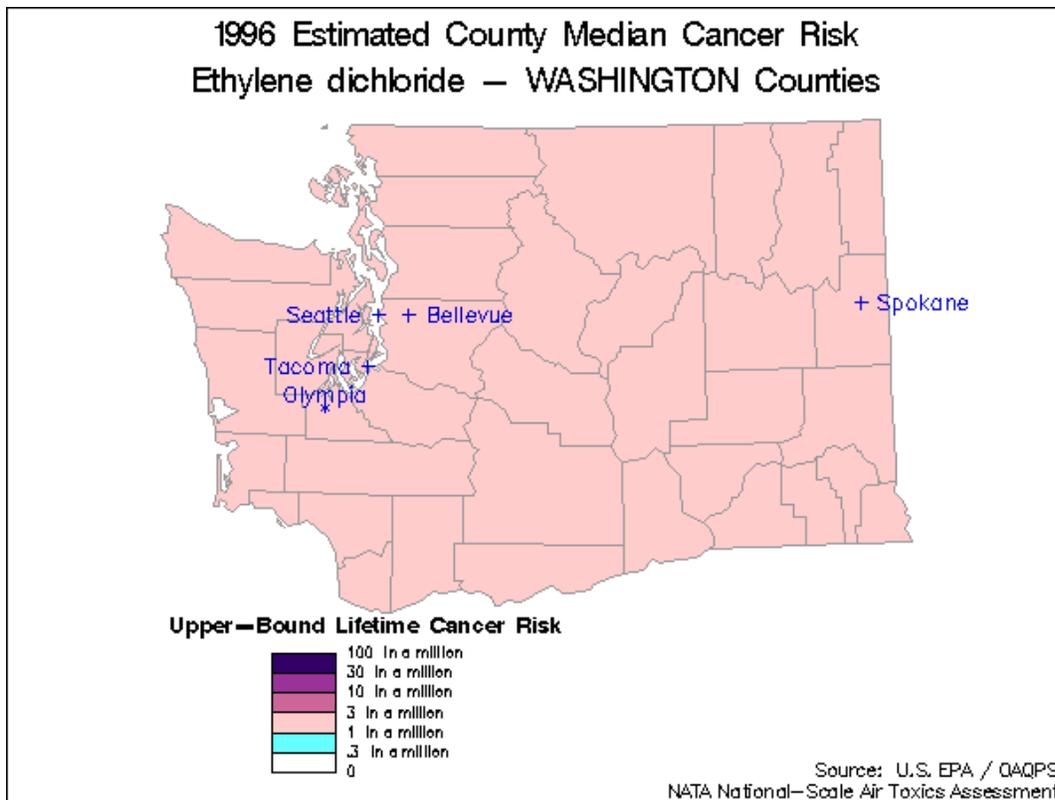


Figure 3-18. 1996 County median cancer risks from ethylene dichloride in Washington.

Table 3-13. Regional and statewide ethylene dichloride cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	1.4E ⁻⁶	1.4E ⁻⁶	1.3E ⁻⁶	1.4E ⁻⁶	1152
BCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.3E ⁻⁶	1.4E ⁻⁶	26
CRO	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	40
ERO	1.4E ⁻⁶	1.4E ⁻⁶	1.3E ⁻⁶	1.4E ⁻⁶	86
NWCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.3E ⁻⁶	1.4E ⁻⁶	77
NWRO	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	6
ORCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.3E ⁻⁶	1.4E ⁻⁶	101
PSCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	579
SRCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	99
SWCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	105
YRCAA	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	1.4E ⁻⁶	33

Estimated plausible upper limit cancer risks

1,3-Butadiene

The USEPA's 1996 NATA estimates median 1,3-butadiene inhalation exposure in Washington indicated an excess cancer risk of 1.35 per million. Approximately 60% of the census tracts in Washington had estimates of excess cancer risk of one-in-a-million or greater (table 3-14). In the PSCAA region, the median estimated risk was 1.5 per million; in the SWCAA region, the median estimated risk was 1.3 per million (figure 3-19). In terms of non-cancer health risks, the NATA estimates of 1,3-butadiene inhalation exposure suggested such health hazards are negligible throughout the state.

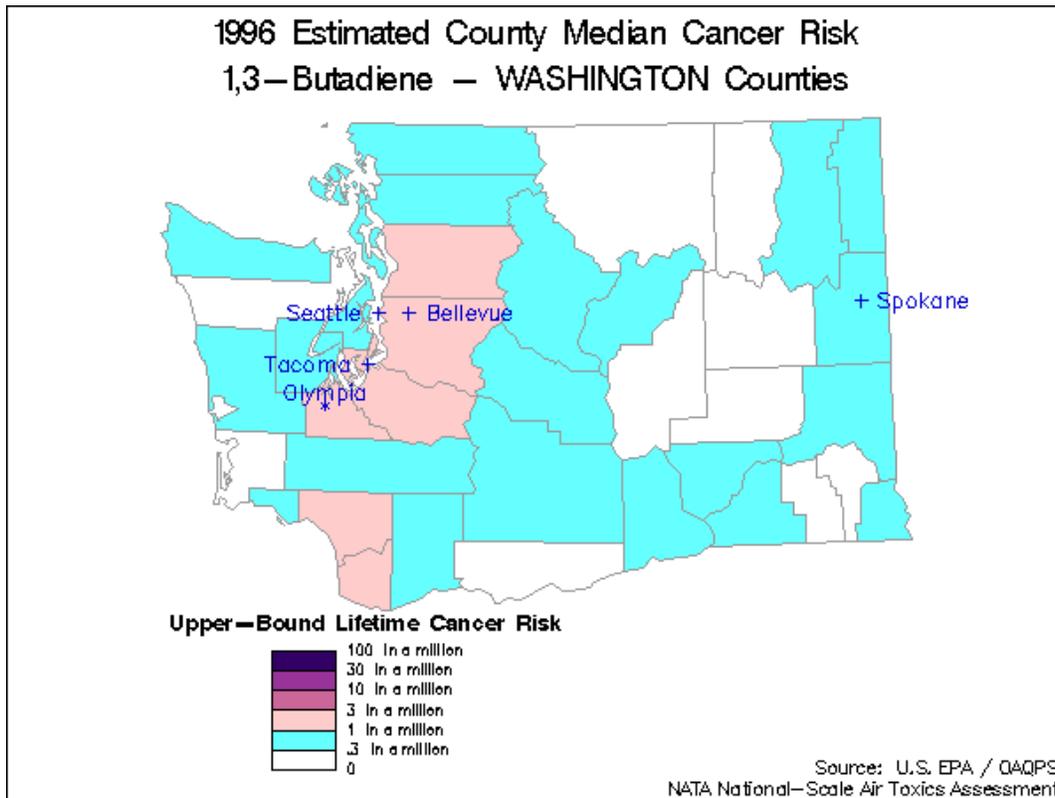


Figure 3-19. 1996 County median cancer risks from 1,3-butadiene in Washington.

Table 3-14. Regional and statewide 1,3-butadiene cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	7.0E ⁻⁷	1.2E ⁻⁶	1.4E ⁻⁶	1.8E ⁻⁶	1152
BCAA	5.3E ⁻⁷	9.0E ⁻⁷	8.7E ⁻⁷	1.1E ⁻⁶	26
CRO	2.5E ⁻⁷	4.9E ⁻⁷	7.0E ⁻⁷	1.0E ⁻⁶	40
ERO	1.4E ⁻⁷	2.8E ⁻⁷	6.4E ⁻⁷	6.8E ⁻⁷	86
NWCAA	3.7E ⁻⁷	5.7E ⁻⁷	8.4E ⁻⁷	1.3E ⁻⁶	77
NWRO	3.5E ⁻⁷	4.2E ⁻⁷	4.9E ⁻⁷	4.8E ⁻⁷	6
ORCAA	4.2E ⁻⁷	7.6E ⁻⁷	9.9E ⁻⁷	1.5E ⁻⁶	101
PSCAA	1.2E ⁻⁶	1.6E ⁻⁶	1.8E ⁻⁶	2.1E ⁻⁶	579
SRCAA	7.0E ⁻⁷	8.7E ⁻⁷	8.9E ⁻⁷	1.0E ⁻⁶	99
SWCAA	8.3E ⁻⁷	1.3E ⁻⁶	1.4E ⁻⁶	1.8E ⁻⁶	105
YRCAA	4.9E ⁻⁷	7.84E ⁻⁷	7.9E ⁻⁷	1.1E ⁻⁶	33

Estimated plausible upper limit cancer risks

Ethylene dibromide

The estimated median excess cancer risk from EDB exposure was 1.3 million throughout

Washington (figure 3-20 and table 3-15). EDB accounted for 0.44% of the total statewide toxic air pollutants cancer risk. In terms of non-cancer health risks, the NATA estimates of EDB inhalation exposure suggested such health hazards were insignificant throughout the state.

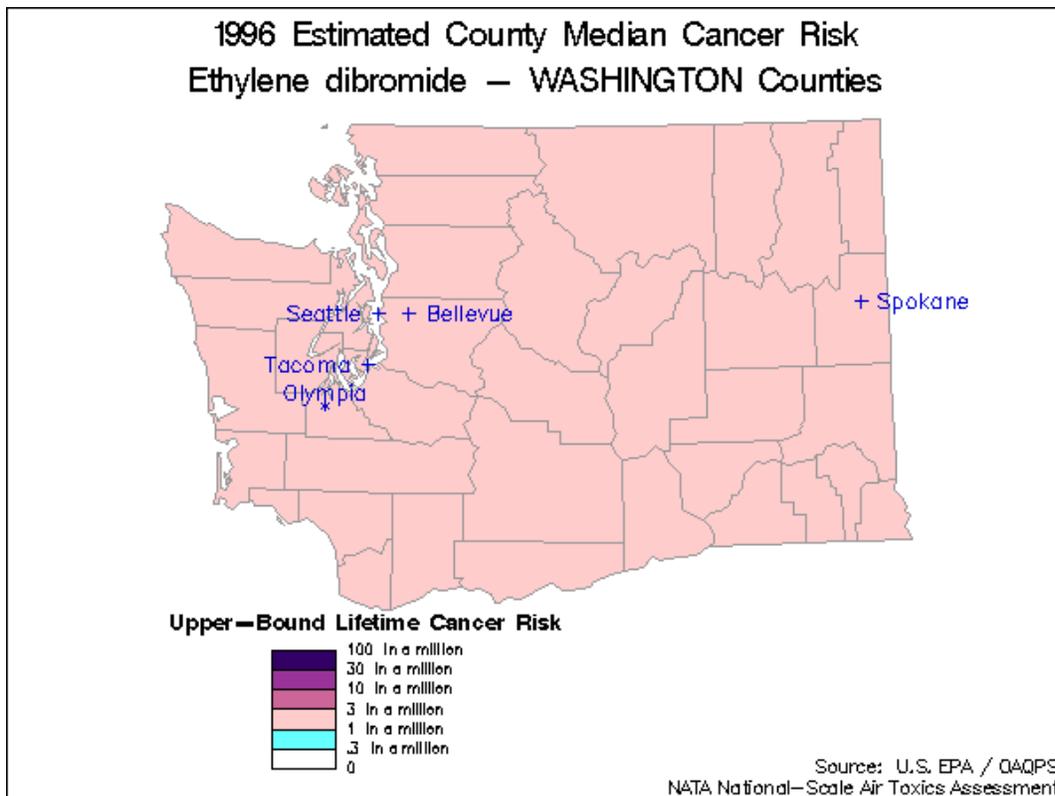


Figure 3-20. 1996 County median cancer risks from ethylene dibromide in Washington.

Table 3-15. Regional and statewide ethylene dibromide cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1152
BCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	26
CRO	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	40
ERO	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	86
NWCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	77
NWRO	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	6
ORCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	101
PSCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	579
SRCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	99
SWCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	105
YRCAA	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	1.3E ⁻⁶	33

Estimated plausible upper limit cancer risks

Acetaldehyde

The USEPA's 1996 NATA estimates of lifetime cancer risk from acetaldehyde inhalation exposure had a statewide median of 1.03 per million (figure 3-21, table 3-16). Acetaldehyde posed 0.35% of the total estimated air toxics-associated cancer risk. Approximately 50.5% of Washington census tracts had excess cancer risks equal to or exceeding one-in-a-million. PSCAA had a median excess cancer risk of 1.5 per million. SWCAA had a median excess cancer risk of 1.2 per million. In terms of non-cancer health risks, the NATA estimates of the acetaldehyde inhalation exposure suggested its related health hazards were insignificant throughout the state.

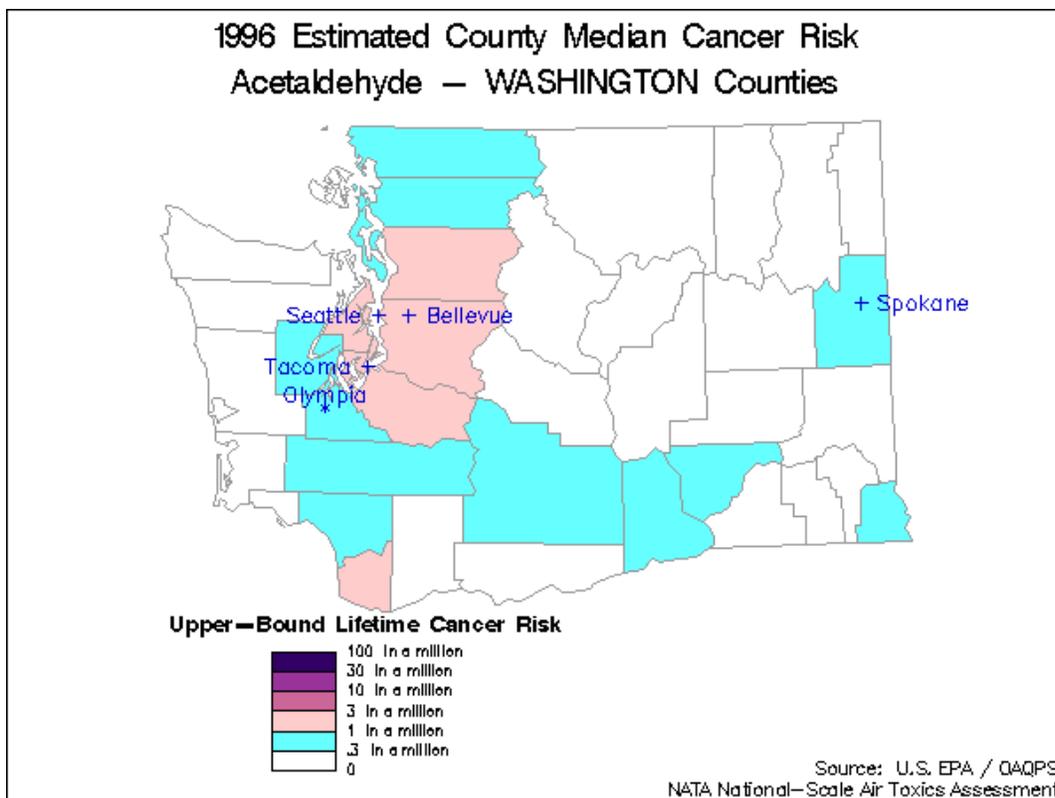


Figure 3-21. 1996 county median cancer risks from acetaldehyde in Washington.

Table 3-16. Regional and statewide acetaldehyde cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	4.4E ⁻⁷	1.0E ⁻⁶	1.0E ⁻⁶	1.60E ⁻⁶	1152
BCAA	3.7E ⁻⁷	4.0E ⁻⁷	3.8E ⁻⁷	4.6E ⁻⁷	26
CRO	7.2E ⁻⁸	1.5E ⁻⁷	2.0E ⁻⁷	3.0E ⁻⁷	40

ERO	7.9E ⁻⁸	1.7E ⁻⁷	1.9E ⁻⁷	2.5E ⁻⁷	86
NWCAA	3.7E ⁻⁷	4.3E ⁻⁷	4.3E ⁻⁷	5.5E ⁻⁷	77
NWRO	1.4E ⁻⁷	1.9E ⁻⁷	1.8E ⁻⁷	2.2E ⁻⁷	6
ORCAA	1.9E ⁻⁷	4.1E ⁻⁷	4.7E ⁻⁷	7.2E ⁻⁷	101
PSCAA	1.2E ⁻⁶	1.5E ⁻⁶	1.5E ⁻⁶	1.8E ⁻⁶	579
SRCAA	5.3E ⁻⁷	5.8E ⁻⁷	5.7E ⁻⁷	6.4E ⁻⁷	99
SWCAA	4.5E ⁻⁷	1.2E ⁻⁶	1.2E ⁻⁶	1.9E ⁻⁶	105
YRCAA	3.5E ⁻⁷	6.0E ⁻⁷	5.5E ⁻⁷	6.8E ⁻⁷	33

Estimated plausible upper limit cancer risks

Tetrachloroethylene

Tetrachloroethylene (perchloroethylene) posed 0.32% of the total toxic air pollutant-associated cancer risk. Cancer risk was one-in-a-million or higher in about 38% of census tracts across Washington (table 3-17 and figure 3-22). Some of these census tracts were in SWCAA's Clark County, where the median excess risk was estimated to be 0.8 per million. In PSCAA, the estimated median risk was 1.1 per million. In terms of non-cancer health risks, the NATA estimates of exposure suggested insignificant inhalation health hazards.

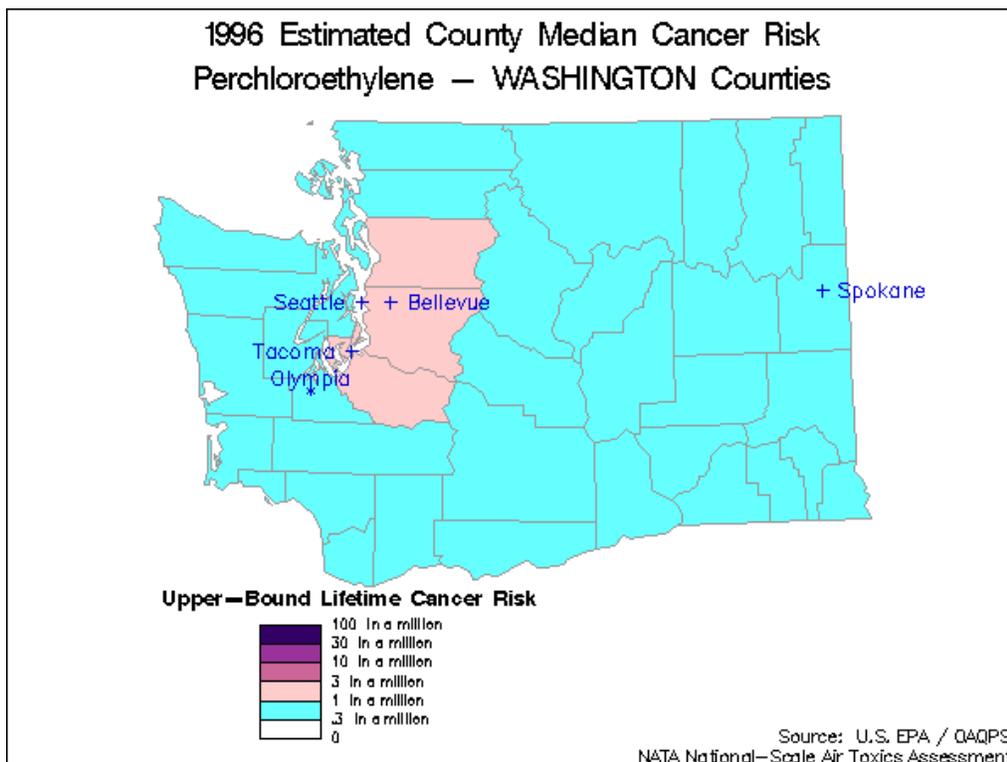


Figure 3-22. 1996 County median cancer risks from tetrachloroethylene in Washington.

Table 3-17. Regional and statewide tetrachloroethylene cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	7.0E ⁻⁷	8.8E ⁻⁷	9.6E ⁻⁷	1.1E ⁻⁶	1152
BCAA	6.7E ⁻⁷	7.7E ⁻⁷	7.6E ⁻⁷	8.9E ⁻⁷	26
CRO	6.0E ⁻⁷	6.1E ⁻⁷	6.5E ⁻⁷	6.9E ⁻⁷	40
ERO	6.0E ⁻⁷	6.2E ⁻⁷	6.6E ⁻⁷	6.8E ⁻⁷	86
NWCAA	6.4E ⁻⁷	6.6E ⁻⁷	6.7E ⁻⁷	7.4E ⁻⁷	77
NWRO	6.1E ⁻⁷	6.1E ⁻⁷	6.1E ⁻⁷	6.1E ⁻⁷	6
ORCAA	6.2E ⁻⁷	6.8E ⁻⁷	7.0E ⁻⁷	7.6E ⁻⁷	101
PSCAA	9.4E ⁻⁷	1.1E ⁻⁶	1.2E ⁻⁶	1.4E ⁻⁶	579
SRCAA	7.7E ⁻⁷	8.2E ⁻⁷	8.1E ⁻⁷	8.5E ⁻⁷	99
SWCAA	6.7E ⁻⁷	8.4E ⁻⁷	8.7E ⁻⁷	1.0E ⁻⁶	105
YRCAA	6.3E ⁻⁷	7.4E ⁻⁷	7.4E ⁻⁷	8.3E ⁻⁷	33

Estimated plausible upper limit cancer risks

Trichloroethylene

Estimated statewide median inhalation exposure to TCE poses an excess cancer risk of 0.25 per million (table 3-18 and figure 3-23). It accounted for 0.214% of the total estimated cancer risk to Washington citizens. Results of the ASPEN modeled annual average concentrations from different source categories indicated that area sources accounted for 0.299- $\mu\text{g}/\text{m}^3$ of outdoor concentrations. TCE's atmospheric half-life is approximately 27 to 272 hours. This characteristic leads to lingering background levels. Background sources were estimated to account for 0.081- $\mu\text{g}/\text{m}^3$ of the average outdoor concentration (21% of exposure). Major sources were estimated to account for 0.00758- $\mu\text{g}/\text{m}^3$ (2% of exposure) on average. The census tract with the highest excess cancer risk from TCE (7.93 per million) was located in King County. The median risk across all of PSCAA was 0.95 per million. In all, there were 277 census tracts within King, Pierce and Snohomish counties where the estimated excess cancer risk was greater than one-in-a-million in 1996 Table 3-18. In terms of non-cancer health risks, the NATA estimates of exposure to TCE suggested insignificant inhalation health hazards throughout the state.

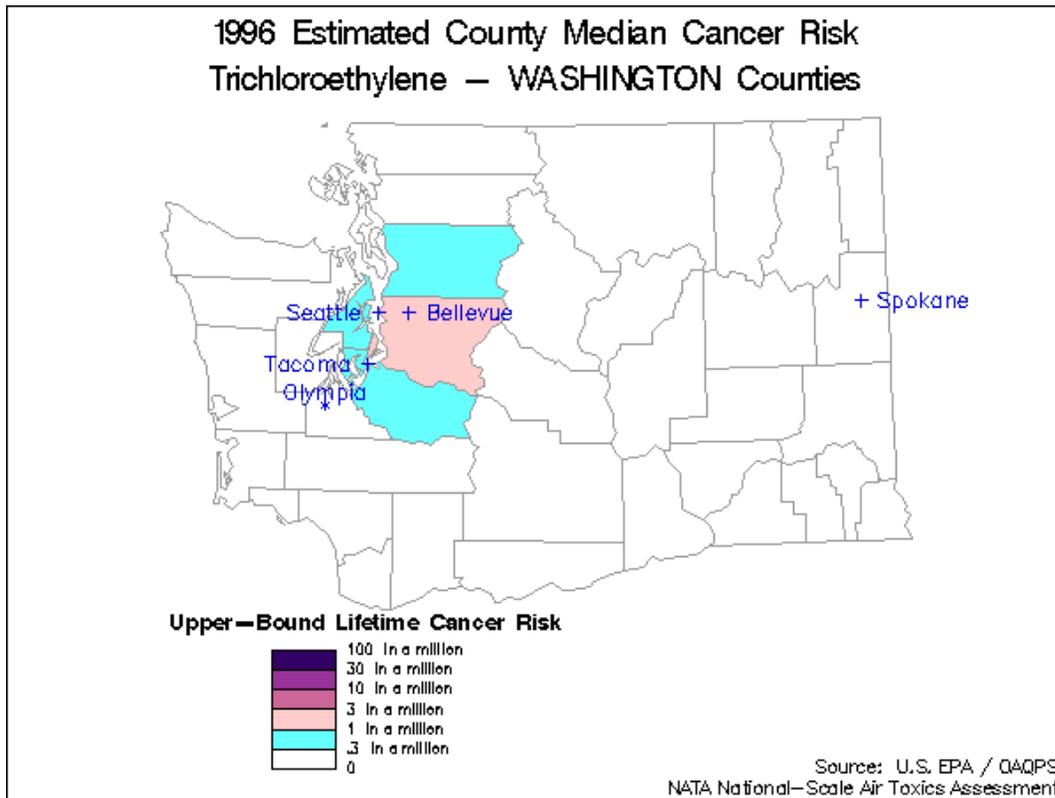


Figure 3-23. 1996 County median cancer risks from trichloroethylene in Washington.

Table 3-18. Regional and statewide trichloroethylene cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	1.5E ⁻⁷	2.5E ⁻⁷	6.4E ⁻⁷	9.8E ⁻⁷	1152
BCAA	1.4E ⁻⁷	1.5E ⁻⁷	1.5E ⁻⁷	1.6E ⁻⁷	26
CRO	1.4E ⁻⁷	1.4E ⁻⁷	1.4E ⁻⁷	1.5E ⁻⁷	40
ERO	1.4E ⁻⁷	1.4E ⁻⁷	1.4E ⁻⁷	1.5E ⁻⁷	86
NWCAA	1.5E ⁻⁷	1.6E ⁻⁷	1.7E ⁻⁷	1.9E ⁻⁷	77
NWRO	1.4E ⁻⁷	1.4E ⁻⁷	1.4E ⁻⁷	1.4E ⁻⁷	6
ORCAA	1.4E ⁻⁷	1.4E ⁻⁷	1.5E ⁻⁷	1.7E ⁻⁷	101
PSCAA	5.4E ⁻⁷	9.8E ⁻⁷	1.1E ⁻⁶	1.3E ⁻⁶	579
SRCAA	1.6E ⁻⁷	1.7E ⁻⁷	1.7E ⁻⁷	1.8E ⁻⁷	99
SWCAA	1.5E ⁻⁷	1.7E ⁻⁷	1.8E ⁻⁷	2.0E ⁻⁷	105
YRCAA	1.4E ⁻⁷	1.6E ⁻⁷	1.6E ⁻⁷	1.7E ⁻⁷	33

Estimated plausible upper limit cancer risks

Nickel and nickel compounds

The statewide median inhalation exposure to nickel and compounds pose an excess cancer risk of 0.27 per million (figure 3-24), in many census tracts (table 3-19): Approximately 0.1% of the total calculated toxic air pollutants cancer risk to Washington citizens.

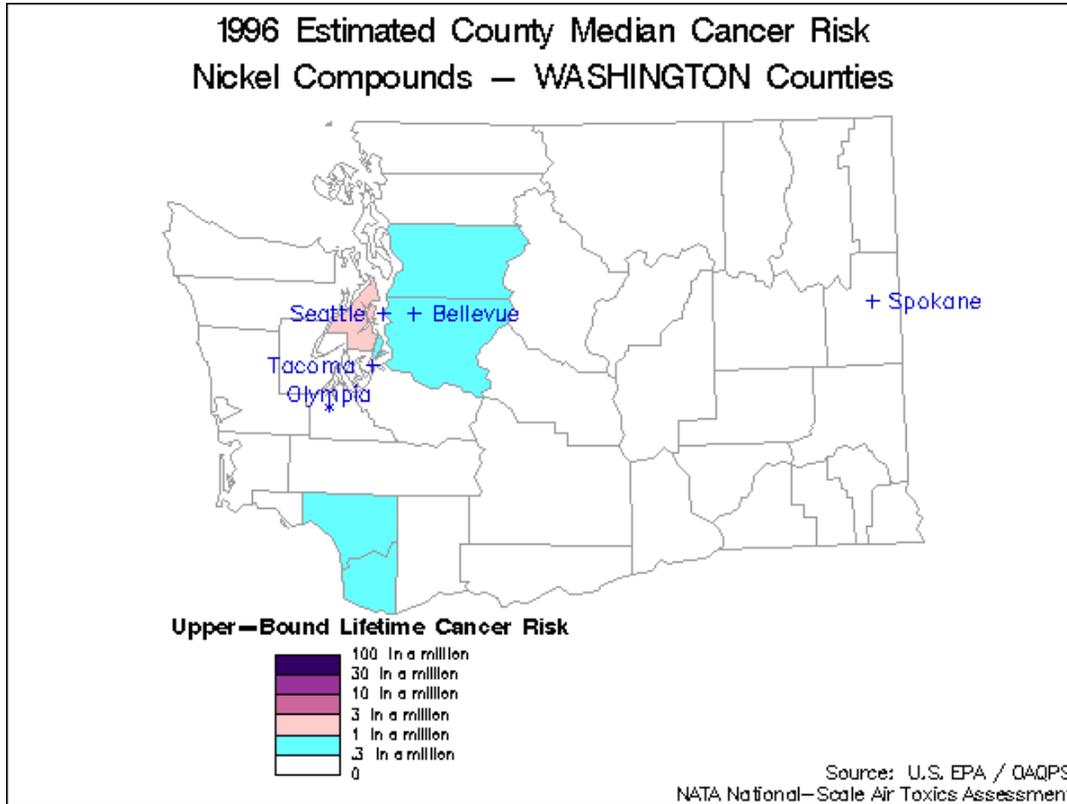


Figure 3-24. 1996 County median cancer risks from nickel and its compounds in Washington.

Table 3-19. Regional and statewide nickel cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	6.3E ⁻⁸	2.7E ⁻⁷	4.9E ⁻⁷	6.8E ⁻⁷	1152
BCAA	2.9E ⁻⁸	5.0E ⁻⁸	6.0E ⁻⁸	7.0E ⁻⁸	26
CRO	1.9E ⁻⁹	6.0E ⁻⁹	1.9E ⁻⁸	3.1E ⁻⁸	40
ERO	2.8E ⁻⁹	1.2E ⁻⁸	2.8E ⁻⁸	3.7E ⁻⁸	86
NWCAA	1.0E ⁻⁷	1.5E ⁻⁷	2.1E ⁻⁷	2.0E ⁻⁷	77
NWRO	2.6E ⁻⁸	3.5E ⁻⁸	3.7E ⁻⁸	4.7E ⁻⁸	6
ORCAA	2.2E ⁻⁸	5.7E ⁻⁸	6.6E ⁻⁸	9.0E ⁻⁸	101
PSCAA	3.49E ⁻⁷	6.4E ⁻⁷	8.0E ⁻⁷	10.0E ⁻⁷	579
SRCAA	5.6E ⁻⁸	7.2E ⁻⁸	8.2E ⁻⁸	10.0E ⁻⁸	99
SWCAA	2.3E ⁻⁷	4.4E ⁻⁷	6.2E ⁻⁷	8.5E ⁻⁷	105
YRCAA	1.2E ⁻⁸	5.9E ⁻⁸	6.4E ⁻⁸	9.6E ⁻⁸	33

Estimated plausible upper limit cancer risks

Estimates of excess cancer risk from nickel exposure exceeded one-in-a-million in 164 census tracts located across five counties in Washington (table 3-19). These counties are noted in table 3-20.

Table 3-20. Census tracts where nickel cancer risk estimates exceeded one-in-a-million.

County	Number of census tracts where PUL of cancer risk estimates exceeded one-in-a-million
King	112
Kitsap	29
Clark	17
Cowlitz	4
Pierce	2

In terms of non-cancer health risks, the NATA estimates of exposure to nickel and its compounds suggested insignificant inhalation health hazards throughout the state.

Arsenic and arsenic compounds

The statewide median excess cancer risk from inhalation of arsenic was 0.16 per million. Arsenic and compounds posed 0.074% of the total median cancer risk. Although no counties had countywide excess cancer risks exceeding one-in-a-million (figure 3-25), risk estimates exceeded one-in-a-million in 11 census tracts in Clark, King and Pierce Counties (table 3-21).

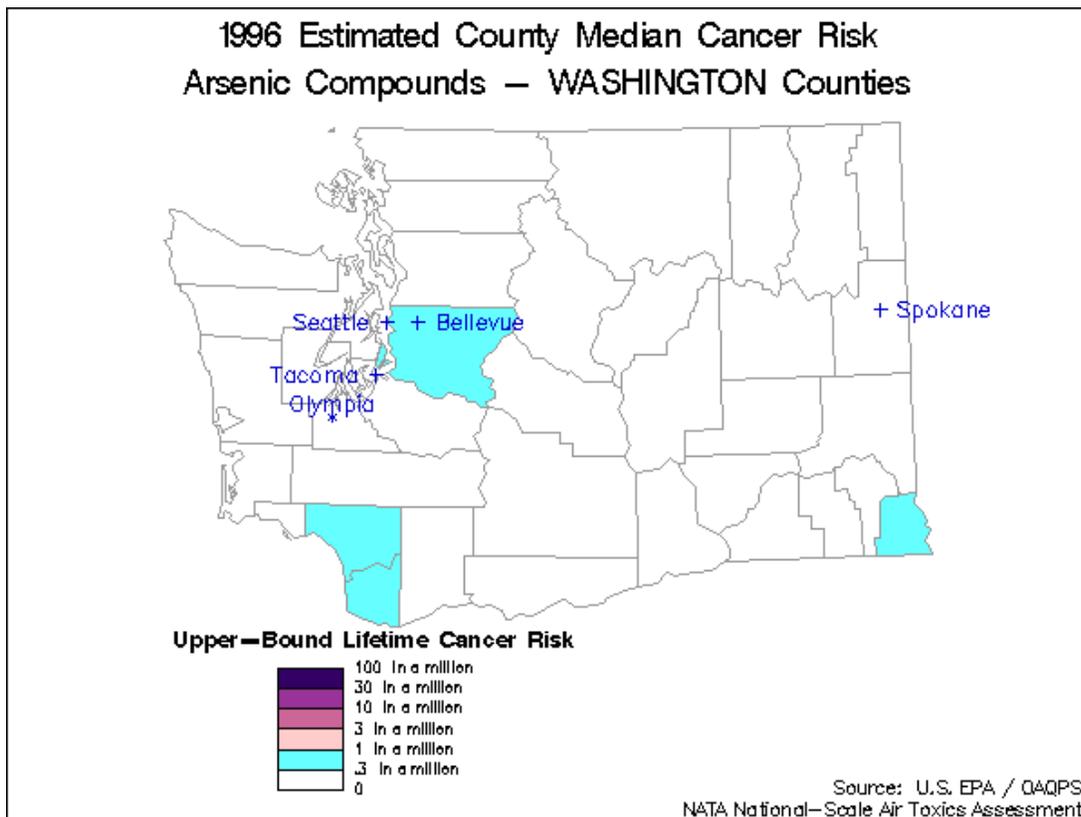


Figure 3-25. 1996 County median cancer risks from arsenic and its compounds in Washington.

Table 3-21. Regional and statewide arsenic inhalation cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	7.4E ⁻⁸	1.6E ⁻⁷	2.2E ⁻⁷	3.2E ⁻⁷	1152
BCAA	8.4E ⁻⁸	1.1E ⁻⁷	1.1E ⁻⁷	1.4E ⁻⁷	26
CRO	6.0E ⁻⁹	1.9E ⁻⁸	5.2E ⁻⁸	7.1E ⁻⁸	40
ERO	7.6E ⁻⁹	3.0E ⁻⁸	7.4E ⁻⁸	9.1E ⁻⁸	86
NWCAA	4.5E ⁻⁸	7.9E ⁻⁸	1.0E ⁻⁷	1.2E ⁻⁷	77
NWRO	1.2E ⁻⁸	1.9E ⁻⁸	1.8E ⁻⁸	2.1E ⁻⁸	6
ORCAA	2.8E ⁻⁸	5.1E ⁻⁸	6.5E ⁻⁸	9.9E ⁻⁸	101
PSCAA	1.7E ⁻⁷	2.6E ⁻⁷	3.0E ⁻⁷	3.9E ⁻⁷	579
SRCAA	6.7E ⁻⁸	8.4E ⁻⁸	8.3E ⁻⁸	10.0E ⁻⁸	99
SWCAA	2.4E ⁻⁷	3.6E ⁻⁷	4.2E ⁻⁷	5.5E ⁻⁷	105
YRCAA	3.6E ⁻⁸	1.5E ⁻⁷	1.7E ⁻⁷	2.4E ⁻⁷	33

Estimated plausible upper limit cancer risks

1,3-Dichloropropene

1,3-Dichloropropene comprised 0.073% of the total statewide toxic air pollutants cancer risk.

The statewide median excess cancer risk was 0.21 per million (figure 3-26). However, the

excess cancer risk exceeded one-in-a-million (at 1.06 per million) in one census tract (located in King County). Estimated exposure levels were slightly below the *de minimis* risk level in several other census tracts (table 3-22). Non-cancer health hazards appeared insignificant throughout Washington.

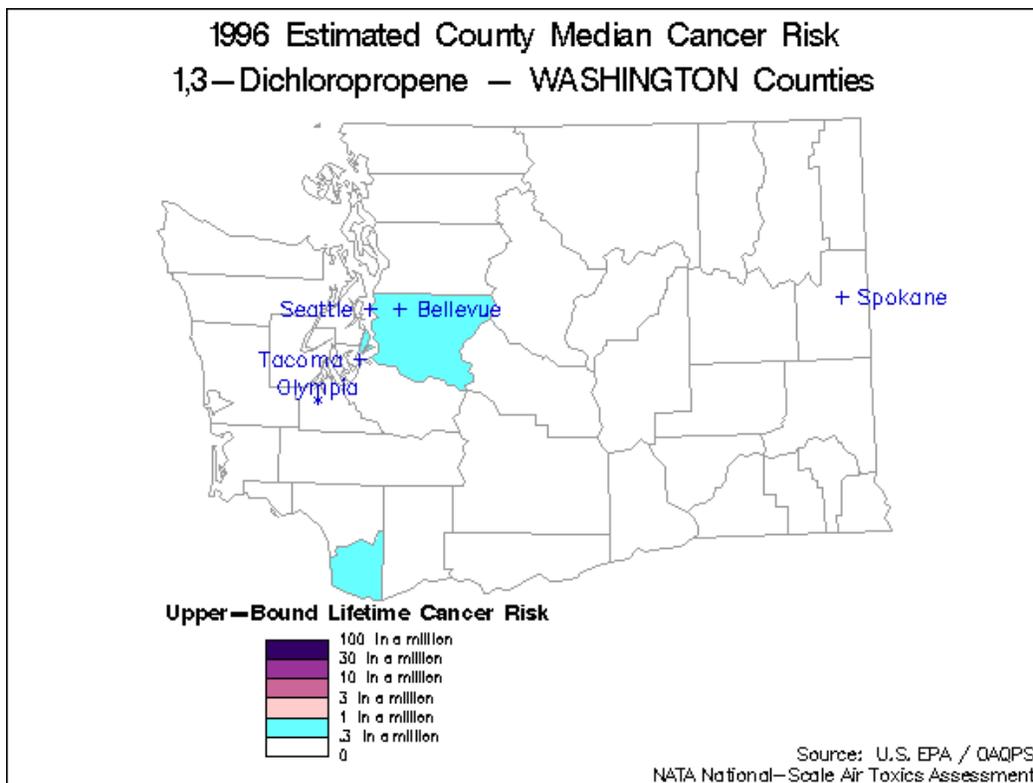


Figure 3-26. 1996 County median cancer risks from 1,3-dichloropropene in Washington.

Table 3-22. Statewide and regional 1,3-dichloropropene cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	8.6E ⁻⁸	2.1E ⁻⁷	2.2E ⁻⁷	3.2E ⁻⁷	1152
BCAA	7.1E ⁻⁸	1.5E ⁻⁷	1.6E ⁻⁷	2.1E ⁻⁷	26
CRO	3.8E ⁻⁹	1.6E ⁻⁸	7.3E ⁻⁸	9.6E ⁻⁸	40
ERO	6.8E ⁻⁹	2.9E ⁻⁸	7.3E ⁻⁸	1.0E ⁻⁷	86
NWCAA	4.2E ⁻⁸	6.1E ⁻⁸	10.0E ⁻⁸	1.6E ⁻⁷	77
NWRO	1.1E ⁻⁸	1.2E ⁻⁸	1.6E ⁻⁸	1.5E ⁻⁸	6
ORCAA	2.6E ⁻⁸	7.1E ⁻⁸	1.1E ⁻⁷	1.7E ⁻⁷	101
PSCAA	2.1E ⁻⁷	2.9E ⁻⁷	3.0E ⁻⁷	3.8E ⁻⁷	579
SRCAA	1.1E ⁻⁷	1.7E ⁻⁷	1.6E ⁻⁷	2.2E ⁻⁷	99
SWCAA	9.5E ⁻⁸	2.1E ⁻⁷	2.3E ⁻⁷	3.5E ⁻⁷	105
YRCAA	5.1E ⁻⁸	1.6E ⁻⁷	1.9E ⁻⁷	2.7E ⁻⁷	33

Estimated plausible upper limit cancer risks

Ethylene oxide

The statewide median excess ethylene oxide-associated cancer risk was 0.07 per million (table 3-23 and figure 3-27), comprising 0.029% of the total air toxics-associated cancer risk. However, excess cancer risk exceeded one-in-a-million (1.07 per million) in one census tract, located in Pierce County. Estimated exposure levels were slightly below the *de minimis* risk level in several other census tracts in Pierce and King Counties. Non-cancer health hazards appeared insignificant throughout Washington.

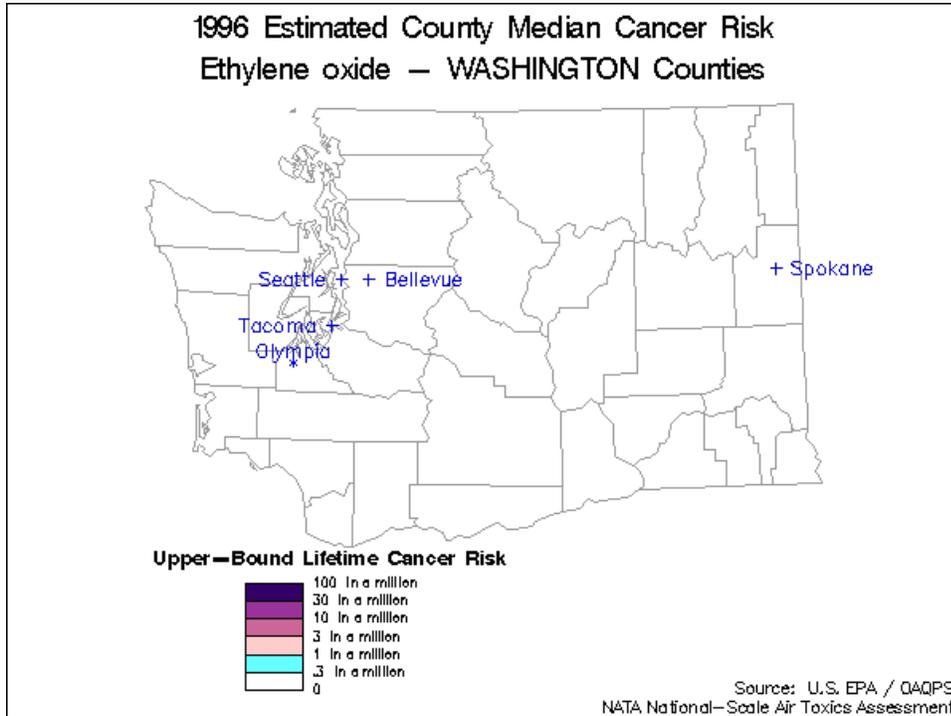


Figure 3-27. 1996 County median cancer risks from ethylene oxide in Washington.

Table 3-23. Statewide and regional ethylene oxide cancer risk distributions

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	2.3E ⁻⁸	6.8E ⁻⁸	8.7E ⁻⁸	1.2E ⁻⁷	1152
BCAA	1.4E ⁻⁸	2.7E ⁻⁸	3.8E ⁻⁸	5.4E ⁻⁸	26
CRO	9.8E ⁻¹⁰	3.9E ⁻⁹	1.6E ⁻⁸	2.0E ⁻⁸	40
ERO	2.2E ⁻⁹	6.2E ⁻⁹	2.3E ⁻⁸	2.4E ⁻⁸	86
NWCAA	1.0E ⁻⁸	1.8E ⁻⁸	3.5E ⁻⁸	4.3E ⁻⁸	77
NWRO	8.6E ⁻¹⁰	1.5E ⁻⁹	1.5E ⁻⁹	2.0E ⁻⁹	6
ORCAA	4.4E ⁻⁹	1.4E ⁻⁸	4.5E ⁻⁸	5.5E ⁻⁸	101
PSCAA	6.4E ⁻⁸	1.1E ⁻⁷	1.3E ⁻⁷	1.5E ⁻⁷	579

SRCAA	4.7E ⁻⁸	6.8E ⁻⁸	8.1E ⁻⁸	9.5E ⁻⁸	99
SWCAA	1.7E ⁻⁸	4.7E ⁻⁸	6.2E ⁻⁸	8.8E ⁻⁸	105
YRCAA	9.9E ⁻⁹	3.8E ⁻⁸	4.9E ⁻⁸	6.8E ⁻⁸	33

Estimated plausible upper limit cancer risks

Total toxic air pollutants-associated cancer risks

USEPA’s data include individual risk estimates for 29 potentially carcinogenic toxic air pollutants in each county. Once we calculated DPM cancer risks for each census tract using the 1996 NATA exposure estimates and the CARB SRB’s URE, we added them to the risks from the 29 other carcinogenic air pollutants as a measure of total toxic air pollutant-associated cancer risk. The results are in shown in table 3-24.

Table 3-24. Regional and statewide carcinogenic air pollutants + DPM cancer risk distributions.

	25 th %ile	50 th %ile	Mean	75 th %ile	Number of census tracts
Statewide	1.6E ⁻⁴	3.0E ⁻⁴	3.0E ⁻⁴	4.0E ⁻⁴	1152
BCAA	1.3E ⁻⁴	1.4E ⁻⁴	1.5E ⁻⁴	1.6E ⁻⁴	26
CRO	8.1E ⁻⁵	1.1E ⁻⁴	1.2E ⁻⁴	1.5E ⁻⁴	40
ERO	6.9E ⁻⁵	9.2E ⁻⁵	1.2E ⁻⁴	1.4E ⁻⁴	86
NWCAA	1.5E ⁻⁴	1.8E ⁻⁴	1.7E ⁻⁴	2.0E ⁻⁴	77
NWRO	9.3E ⁻⁵	1.0E ⁻⁴	1.0E ⁻⁴	1.1E ⁻⁴	6
ORCAA	1.3E ⁻⁴	1.8E ⁻⁴	2.0E ⁻⁴	2.5E ⁻⁴	101
PSCAA	3.2E ⁻⁴	3.7E ⁻⁴	3.9E ⁻⁴	4.5E ⁻⁴	579
SRCAA	1.3E ⁻⁴	1.5E ⁻⁴	1.5E ⁻⁴	1.6E ⁻⁴	99
SWCAA	2.5E ⁻⁴	3.8E ⁻⁴	3.9E ⁻⁴	4.8E ⁻⁴	105
YRCAA	1.4E ⁻⁴	1.9E ⁻⁴	1.9E ⁻⁴	2.2E ⁻⁴	33

Estimated plausible upper limit cancer risks

The NATA presented source category distinctions between toxic air pollutants contributions to cancer health risks at the county level. We added these data to the DPM-associated cancer risks by source category to derive estimates of local aggregate risks by each source category. The county median risks are grouped together by LAA region and sorted highest to lowest in figure 3-28.

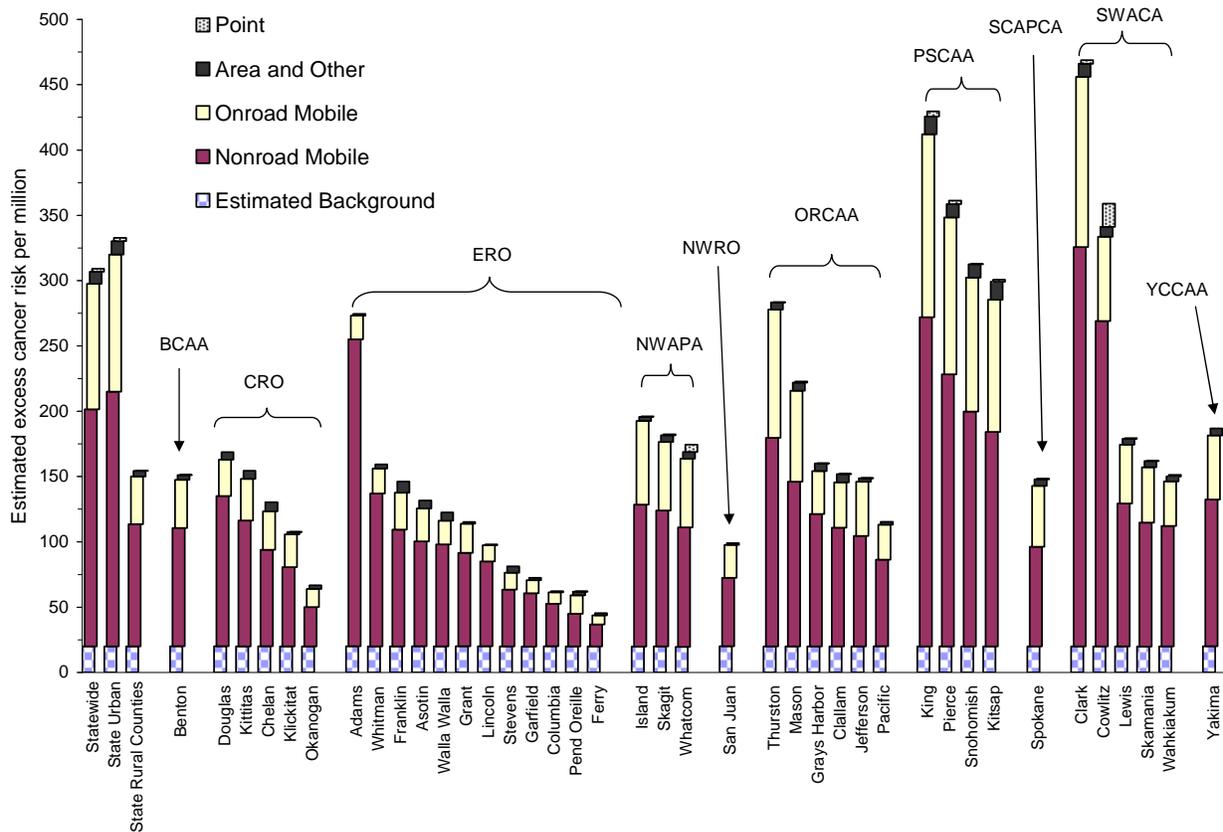


Figure 3-28. 1996 Exposure levels estimated lifetime inhalation cancer risks for 29 hazardous air pollutants plus diesel particulate matter by source category and county (Estimated plausible upper limit cancer risks).

NATA's limitations

A USEPA risk assessment guidance document⁷² states "Risks may be characterized either in terms of the excess individual lifetime risks, the excess number of cancers produced per year in the exposed population or both". USEPA also discusses individual and population risks in its 1992 *Guidelines for Exposure Assessment*,⁷³ providing an equation for calculating potential population risks. In that document, they emphasize that risk estimates are predictions, and should not be interpreted as actual cases. Others have suggested that the risk estimates apply

⁷² USEPA. 1986. *Risk Assessment Guidelines*. EPA/600/8-87/045

⁷³ USEPA 1992 *Guidelines for Exposure Assessment* (see pages 47-48).

only to a theoretical individual, and cannot be interpreted over a population. In other words, such a cancer risk estimate would only apply to a person, and should not be viewed as fifty cancer cases for every million people exposed. In NATA, USEPA uses the term "lifetime cancer risk per million" suggesting an individual risk perspective. For the purpose of toxic air pollutants prioritization, the population- or individual-risk debate is overshadowed by the need for estimation of relative risks in general.

In our evaluation of the NATA, we cautiously interpret unit risk estimates as population risk estimators. Keeping in mind, the regional-level uncertainties are inherent in using ambient modeled concentrations and monitored concentrations, which vary based largely on proximity and number of sources. We also keep in mind that exposure durations and times are variable among different populations. These factors worsen the large innate uncertainty in multiplying the estimated exposure concentration-associated risks by U.S. census population data.

NATA's other limitations and uncertainties that are relevant to the toxic air pollutants prioritization are:

- Risk estimates reflected average population exposures rather than most exposed individuals. Risk analysis does not estimate individual extremes – only typical average/median exposures. The individual risk over 70 years may not address population risk over 70 years).
- NATA did not include risk estimates for diesel particulate matter.
- NATA did not reflect significant reductions or increases in toxic air pollutant emissions that have occurred since 1999.
- NATA likely underestimated the health risks from wood smoke because only a few of the toxic chemicals in wood smoke were estimated individually and because the emission factors for wood smoke were found to be too low in at least one region. They are probably too low in other regions of Washington as well.

- The emissions inventories for mobile and area sources were “top-down”: Some emissions were estimated using census data with activity factors rather than actual counts of real individual sources.
- The AQP and Washington’s local air agencies supplied information about major sources to USEPA, but the statewide consistency of the data is not assured. Reporting may have varied greatly among jurisdictions so that similar sources may have been reported in different formats and with varying completeness. Results should be used cautiously for ranking areas because some areas have done a better job of reporting/monitoring than others. Higher risk could merely reflect better reporting.
- The spatial resolution of ASPEN results may not be good enough to derive firm conclusions about ambient concentrations at county or local levels (areas smaller than a county).⁷⁴ The accuracy and resolution of the data is indistinct and can be contradictory and unexplainable at the county/local level, as evidenced by the anomalously high median DPM concentration estimate in Adams County in the 1996 NATA.
- The annual average concentrations were derived for each census tract with respect to the location of the receptors by calculating concentrations in one or five dispersion grids per tract, depending on the size of the tract.⁷⁵
- The ASPEN modeling system treated area sources and motor vehicles as a pseudo-point sources located within each census tract. It used an assumption that these sources were vented point sources with an effective stack height of five meters and no plume rise behavior. Therefore, actual ambient concentrations near most area and mobile sources may in fact have been higher than those predicted in NATA.

⁷⁴ Some states are smaller than some LAA regions in Washington; however, NATA did not specify how small an area could be reliably interpreted.

⁷⁵ <http://www.epa.gov/ttnatw01/nata/appendix-e.pdf>

- ASPEN predictions are more reliable in areas where wind patterns can be accurately forecast. Thus, ASPEN was probably more reliable in the areas from the Puget Sound to the Columbia River because the predominant wind patterns are more stable in that area. Other areas, such as along the coast, the Columbia River gorge, and around the Cascades and Blue Mountains are more difficult to evaluate with ASPEN. ASPEN is relatively unreliable in the windy northern part of the Puget Sound, especially near the Strait of Juan de Fuca.
- ASPEN has a 50-km range limit. Sources that affect more distant locations were not included in ambient concentration calculations.
- NATA risk analysis is based on chronic inhalation exposure only: Some food chain-biomagnifying persistent air pollutants (e.g., mercury, PCBs) may pose significant risks by ingestion as well, because a substantial portion of total exposure to these air pollutants is by ingestion.
- For some air toxics, indoor sources are responsible for more of the exposure than are outdoor sources; however, sources of indoor origin were excluded from consideration in the NATA.
- HAPEM only estimates toxic air pollutants exposures from outdoor sources, which are assumed to penetrate into various microenvironments by certain factors. Penetration factors are not available for most toxic air pollutants and therefore had to be estimated. Most factors were likely overestimated.
- There are differences among people in susceptibility to toxic air pollutants due to such factors as age, sex, race, health status and ethnicity, but the nature and magnitude of these susceptibility differences is not well understood.
- Uncertainty about carcinogenicity of the toxic air pollutants also contributes to the overall uncertainty in NATA. Some of the toxic air pollutants considered in NATA are known

human carcinogens. These were presented in a NATA map of the aggregate cancer risks across Washington, reproduced in figure 3-29.

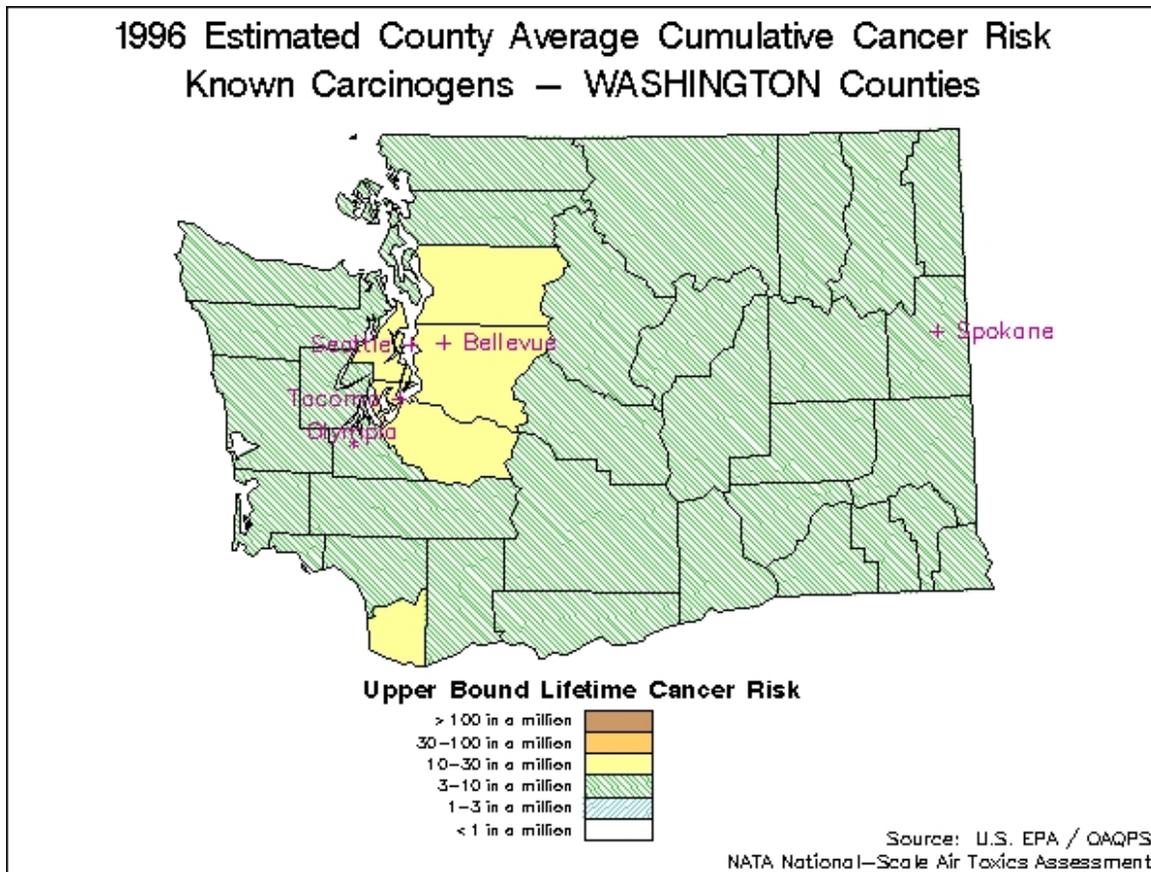


Figure 3-29. 1996 Cumulative county-wide averages of cancer risks of known human carcinogens.

The median of the cancer risk range posed by the other air toxics, having more limited evidence of carcinogenicity (excluding DPM), was also mapped in NATA (figure 3-30).

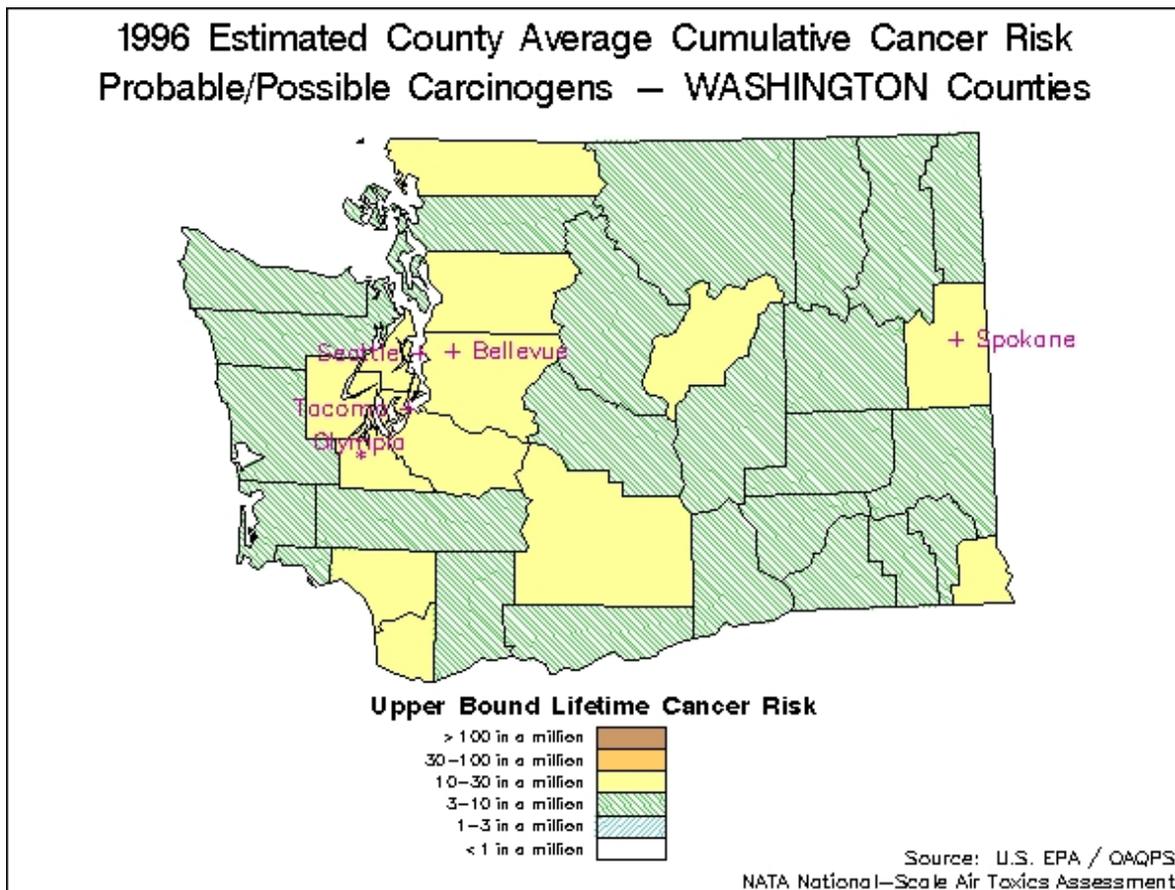


Figure 3-30. 1996 Cumulative county-wide averages of cancer risks of probable and possible carcinogens.

1999 National-Scale Air Toxics Assessment

Overview

USEPA released the 1999 NATA in February, 2006. In this part of the report, we compare the 1999 NATA to the 1996 NATA, and make a few modifications the new NATA results to make them comparable with the previous NATA and more scientifically defensible. As in the 1996 NATA, applying the California OEHHA's diesel exhaust particulate URE to the HAPEM exposure estimates bring to light that diesel engine emissions pose the greatest risk for air pollution-associated cancer risk. The 1999 NATA directly shows that several other toxic air pollutants, most significantly formaldehyde and benzene, also pose excessive cancer risks in Washington. Acrolein appears to pose a significant respiratory toxicity hazard throughout most

of the state. Uncertainties in the methods used in the 1999 NATA suggest that these risks and hazards are underestimates and that true air pollution threats to public health are higher than estimated.

Although there is substantial evidence showing that respirable particulate air pollution is of much greater concern to public health than toxic air pollution-associated cancer risks and non-cancer hazards assessed in the report, the NATA does not account for all of the health impacts of particulate air pollutants (including notably diesel PM). There are more children and more people with cardiac and pulmonary diseases (which are exacerbated by exposure to PM) than the number of people who develop air pollution-associated cancers and assessed non-cancer effects. It is scientifically established that PM exposure is linked to adverse health effects among young children, older people, and especially in people with lung and heart conditions, but this threat is not assessed in the NATA.

ASPEN validation

In the 1999 NATA, data from toxic air pollutants monitoring stations was compared to ASPEN model toxic air pollutants concentration estimates. USEPA stated that these model-to-monitor comparisons for 14 toxic air pollutants suggested a general tendency for the model to underestimate measured ambient levels. On average, modeled concentrations ranged from 95% of monitored levels for benzene to 14% for 1,4-dichlorobenzene. Only one of the 14 pollutants (methylene chloride) had average model-estimated ambient levels above the monitored levels (143%). Thus, the model-to-monitor comparison results suggest that the ASPEN model may systematically underestimate ambient concentrations for 13 of the 14 pollutants that were evaluated. Given that air monitoring data are usually more reflective of actual ambient conditions, and given the apparent tendency of the ASPEN model to underestimate ambient concentrations, it is possible that ambient concentrations for other pollutants are underestimated as well.

PSCAA extracted ASPEN model toxic air pollutants concentration estimates reported in the 1999 NATA for the census tract where the Seattle Beacon Hill toxic air pollutants monitoring station (Table 3-25) is located and compared them to data collected in 2000 from the Beacon Hill toxic air pollutants monitoring station. Although this was not a direct comparison of the same-

year data, if we assume changes in emissions of were negligible between 1999 and 2000, the comparison suggests reasonable agreement between ASPEN estimates and monitored concentrations for most of the toxic air pollutants.

Table 3-25. Concentration ratios of 1999 ASPEN estimates to 2000 Beacon Hill monitor.

	[ASPEN Model] / [Beacon Hill Monitor]
Diesel Particulate Matter ^a	0.8
Formaldehyde	1.0
Benzene	1.3
1,3-Butadiene	0.8
Chloroform	0.5
Carbon Tetrachloride	0.4
Acetaldehyde	1.4
Chromium ^b	0.7
Tetrachloroethylene	1.3
Trichloroethylene	1.7

^a Diesel Particulate Matter from PMF Toxic air pollutants Evaluation (1996 through 1999). This is the inter-year difference in NATA's diesel PM emissions inventories.

^b Total Beacon Hill Chromium from Toxic air pollutants Evaluation (2001).

Despite apparent agreement of a local monitor data with ASPEN estimates, we cannot rule out the possibility that the majority of risk estimates based on the ASPEN model might be underestimates.

Toxicity values

In the 1996 and 1999 NATAs, USEPA did not apply a cancer unit risk factor for diesel exhaust. In addition, in the 1999 NATA, USEPA used a different approach to acquire toxicity values than the approach established in the 1996 NATA. The key discrepancy is that, in the 1999 NATA but not the 1996 NATA, USEPA gave highest priority to their draft Integrated Risk Information System (IRIS) followed by the published IRIS database. For substances lacking IRIS assessments, ATSDR MRLs (available only for non-cancer effects) received next preference, followed by OEHHA RELs (estimates of non-cancer effects toxicity thresholds) and UREs (unit risk estimates of cancer potency). Except for formaldehyde, which had an URE in IRIS, the 1999 NATA used Health Effects Assessment Tables (HEAST) only when no values from the other sources described above were available, despite the fact that previous guidance from USEPA has

said not to use HEAST values since they have not been verified by USEPA's Risk Assessment Group. The most significant effect of this new approach is that the URE used for formaldehyde was far less protective than the one published in IRIS.

Diesel exhaust

The 1999 national-scale assessment, like the 1996 assessment, did not include quantitative cancer risk estimates for diesel exhaust because USEPA judged that toxicological data are not yet sufficient to develop a URE. Since there is no USEPA URE, we applied the California ARB's cancer URE ($0.0003/\mu\text{g}/\text{m}^3$) to NATA exposure model results. In addition, as noted above, PM (including diesel PM) exposure raises risks of cardiac and pulmonary diseases, especially among young children and people with lung and heart conditions; however the NATA does not account for such health risks and hazards. We believe that despite only partially established cause and effect relationships, exposure to diesel exhaust poses significant risk to public health.

Formaldehyde

USEPA selected a recent limited analysis of formaldehyde carcinogenicity for use in the 1999 NATA. USEPA used the Chemical Industry Institute of Toxicology Centers for Health Research cancer unit risk estimate of 5.9E^{-9} per $\mu\text{g}/\text{m}^3$, for use in the assessment. This URE is 2275-fold more tolerant of cancer risk than the URE published in IRIS, which is 1.3E^{-5} per $\mu\text{g}/\text{m}^3$).

USEPA explained that they are currently updating the IRIS file for formaldehyde to consider new science published in the peer-reviewed literature including risk estimates developed by the CIIT and epidemiologic studies published by the National Cancer Institute and others. Although the CIIT's formaldehyde unit risk estimate used a nonlinear low-dose extrapolation method and was based only on animal experiments without accounting for occupational epidemiological findings, USEPA no longer considers the formaldehyde URE reported in IRIS to represent the best available science in the peer-reviewed literature. In addition to concern over the limited information CIIT used, the AQP declined use of the new cancer potency factor because it is a "maximum likelihood" estimate rather than a 95% upper confidence level estimate. Further, the results of a thorough review of available data were published by the International Agency for Research on Cancer in June 2004 concluding the new information from recent epidemiologic studies increases the overall weight of evidence of formaldehyde carcinogenicity. The IARC

states “formaldehyde is carcinogenic to humans.”⁷⁶ For these reasons, we chose to apply the current formaldehyde URE published in IRIS to 1999 NATA exposure model results.

Cancer risks estimates

Recalculating cancer risks using appropriate UREs for diesel PM and formaldehyde along with UREs for other toxic air pollutants chosen by NATA’s authors, we found the statewide sum of average plausible upper limit excess cancer risks (based HAPEM5 median exposure concentration of each toxic air pollutant in each census tract) was 242.7 per million. Thus, calculated plausible upper limit of air toxics-associated cancer risk is close to 243-fold higher than the *de minimis* level on average in the state. Toxic air pollutants with average excess cancer risks over *de minimis* are shown in figure 3-31. Note that these risks are unevenly distributed: Some parts of the population spend significantly more time in more polluted areas and consequently have higher toxic air pollutant-associated cancer risks than parts of the population in areas with lower toxic air pollution.

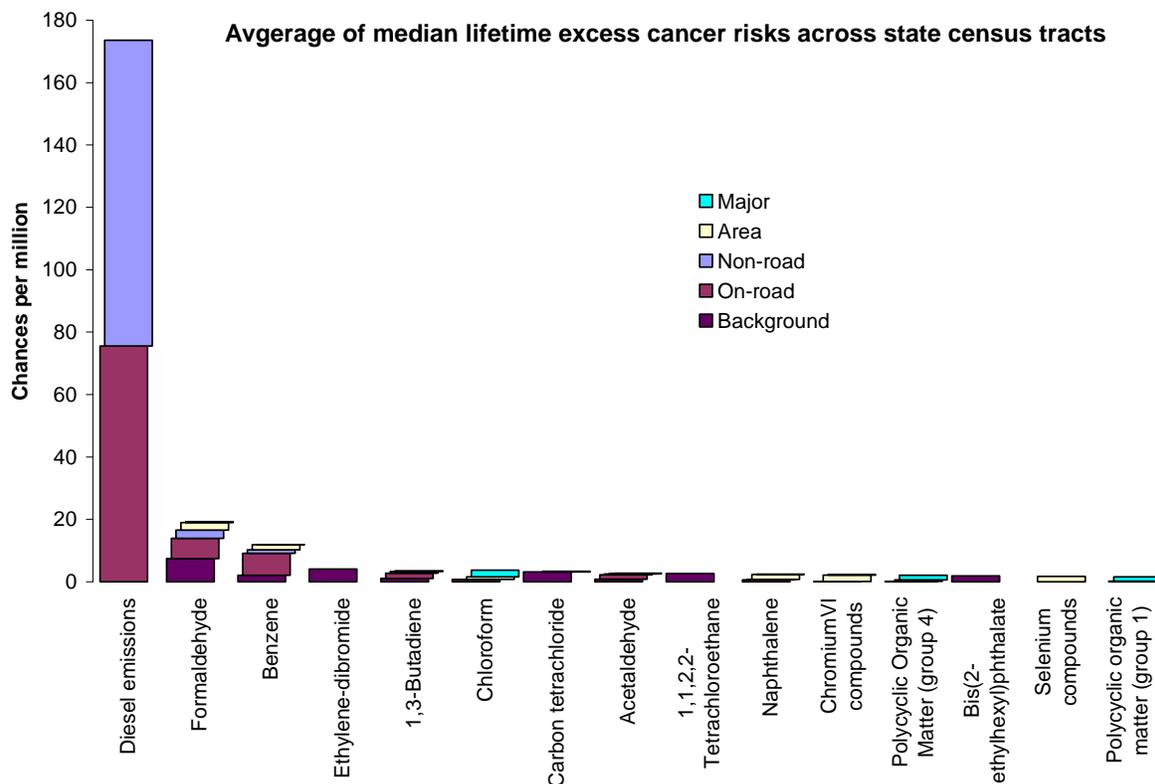


Figure 3-31. Average of median lifetime excess cancer risks at 1999 exposure levels across Washington census tracts.

⁷⁶ http://www.iarc.fr/ENG/Press_Releases/archives/pr153a.html

There are some discrepancies between the 1996 NATA and the 1999 NATA results. The statewide sums of median plausible upper limit cancer risks in each census tract posed by the top three toxic air pollutants are shown in figure 3-32. Each risk is shown as the product of the unit cancer risk estimate (for life-time excess risk) times the exposure estimate (from HAPEM5 modeling of each source category's contribution to human exposure) times the 1990 census tract population divided by 70 years. For DPM the URE used is the California OEHHA value, and for formaldehyde, the UREs used are the ones published in the USEPA IRIS.

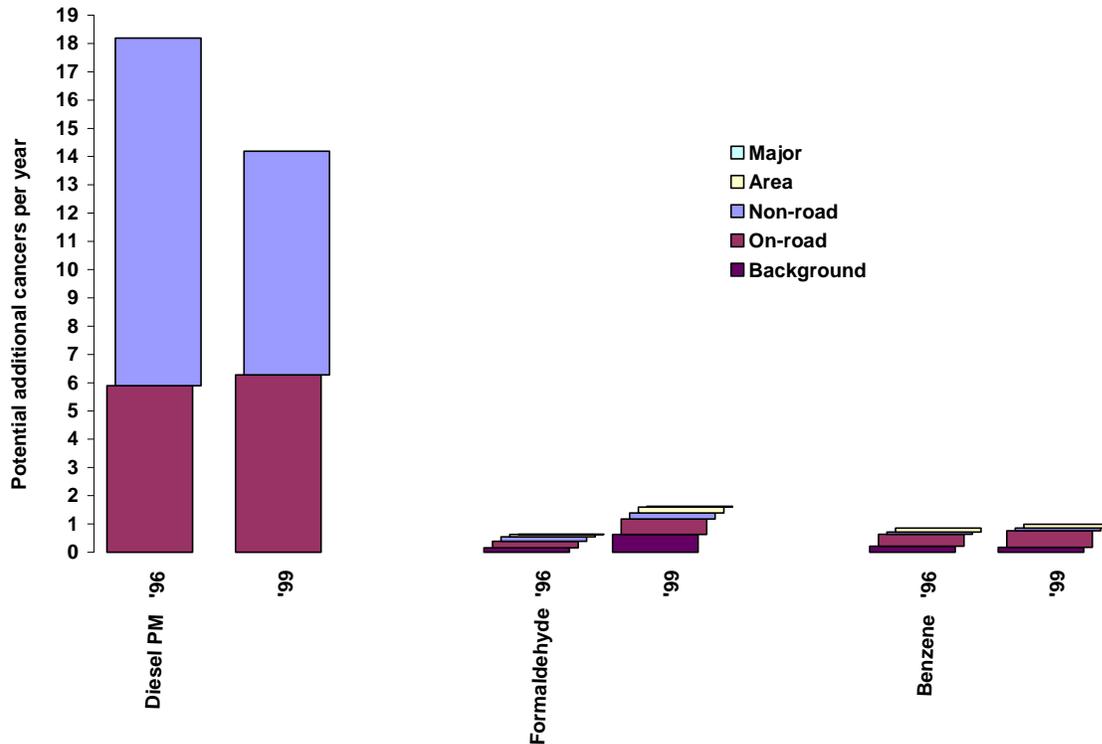


Figure 3-32. Comparison of statewide median cancer risks posed by the top three toxic air pollutants based on NATA human exposure model data.

At these exposure concentrations, there is some possibility that several people, out of the approximately six million in Washington could contract cancer if exposed continuously for 70 years (an assumed lifetime) within the census tracts in which they spend time. This would be in addition to those cancer cases that would normally occur in an unexposed population of six million people. The NATA looks at lifetime cancer risks, which should not be confused with

annual cancer risk estimates. To compare annual cancer risk estimates with the results in NATA, we divided the lifetime risk by a factor of 70.

Why did reported diesel cancer risk decline between 1996 and 1999?

The most significant difference between the 1999 NATA and the 1996 NATA in Washington is the contribution of diesel PM to air toxics-associated cancer risk. Some of the difference seems to be because USEPA started with much lower diesel emissions in 1999 than in 1996. Table 3-26 shows statewide and county comparisons as a percent of emissions counted in 1999 versus those in 1996.

Table 3-26. Inter-year difference of the 1996 and 1999 NATAs diesel PM emissions inventories.

	On-road	Non-road	Total
	(%)		
Adams	286	117	140
Columbia	41	120	103
Garfield	40	121	113
Grant	151	93	105
Kittitas	393	72	183
Lincoln	140	118	120
Skagit	99	127	119
Whitman	52	116	110
Asotin	83	89	87
Benton	128	67	84
Chelan	67	57	61
Clallam	54	53	53
Clark	75	58	64
Cowlitz	93	67	72
Douglas	79	101	96
Ferry	55	53	54
Franklin	57	91	83
Grays Harbor	61	39	42
Island	52	31	39
Jefferson	69	15	28
King	69	47	53
Kitsap	64	39	52
Klickitat	57	73	68
Lewis	79	40	51
Mason	66	21	41
Okanogan	56	45	49
Pacific	53	21	31
Pend Oreille	63	82	72
Pierce	67	53	57
San Juan	29	24	26
Skamania	46	91	70
Snohomish	71	41	51

Spokane	72	55	61
Stevens	63	52	56
Thurston	70	40	53
Wahkiakum	45	26	29
Walla Walla	79	93	91
Whatcom	78	47	56
Yakima	89	62	72

* If less than 100%, less was reported in 1999 than in 1996; If more than 100%, more was reported in 1999 than in 1996; e.g., the statewide on-road diesel emissions counted in the 1999 NATA are only 75% as much what were counted in the 1996 NATA.

In most counties, the reported emissions from on-road and non-road diesel engines were much less in the 1999 NATA as compared to 1996 NATA. The difference between the years is expressed in pounds of diesel PM per capita by county in figure 3-33.

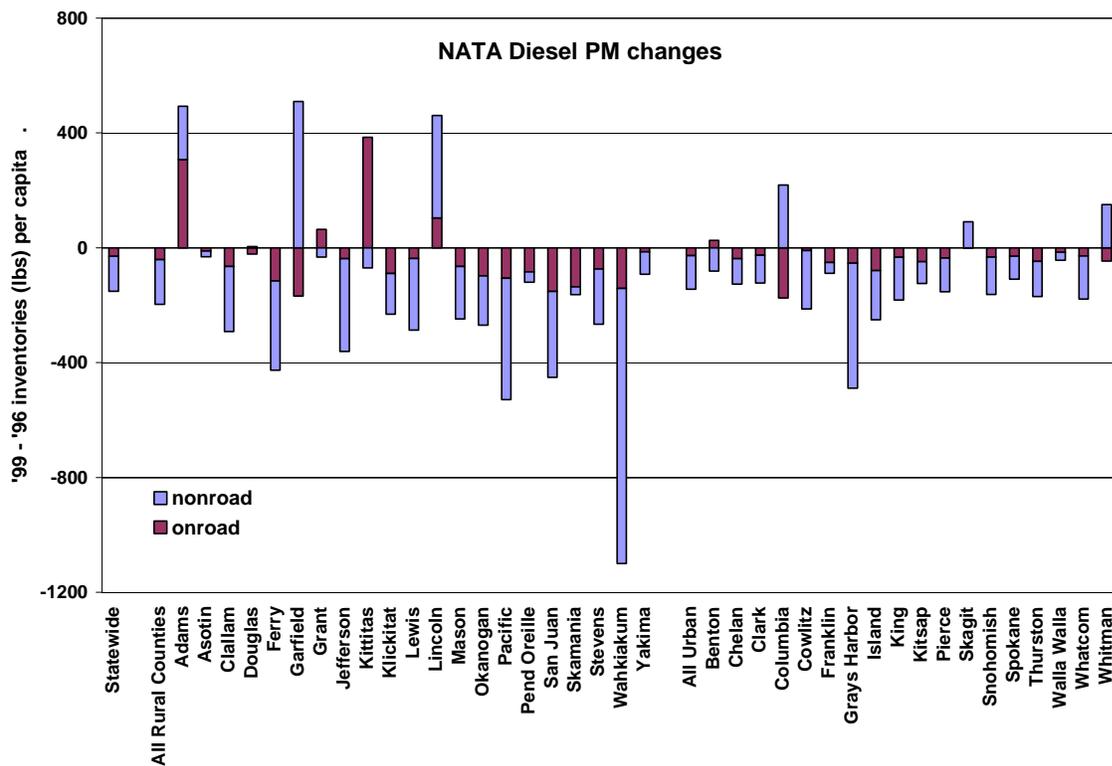


Figure 3-33. Difference between NATA 1999 and 1996 diesel PM emissions per capita by county. Note: if > 0, more was reported in 1999 than in 1996; if < 0, less was reported in 1999 than in 1996.

These between-year differences in diesel emissions are a main factor in the change of relative ranks of the top toxic air pollutants, but cannot account for all of the reported reduction in cancer

risk: There also appear to be changes between HAPEM4 (used in the 1996 NATA) and HAPEM5 (used in the 1999 NATA). These changes, along with changes in the inventoried amounts of diesel PM seem to account for most of the between-year change in exposure levels. In addition, some of the decline in exposure is probably real, as evident when examining the AQP's inventories of diesel PM, which were conducted as part of the Government Management, Accounting and Performance (GMAP) system, shown in figure 3-34.

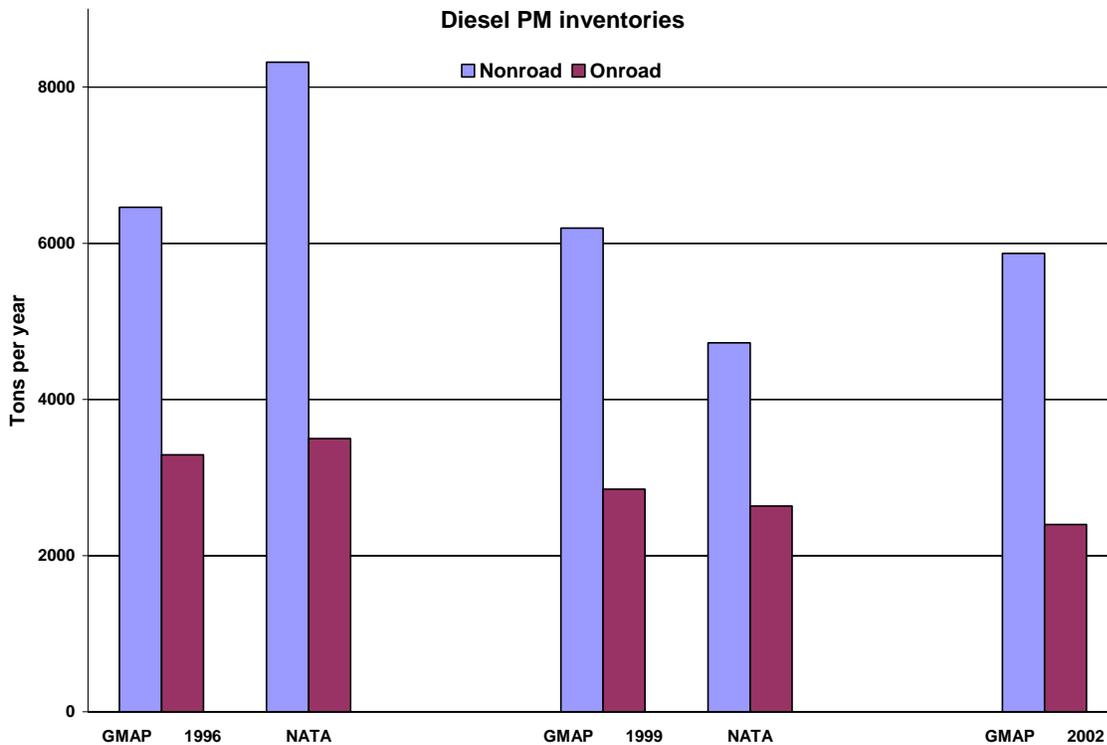


Figure 3-34. Between-year differences in GMAP and NATA diesel emissions inventories.

The data prepared for GMAP show emissions steadily trending downwards. These appear to be real changes; however, when GMAP inventories are compared to the NATA inventories, an overestimate of emissions in the 1996 NATA is suggested, and an underestimate of emissions in the 1999 NATA is suggested. Relative to GMAP inventories in 1996, 1999 and 2002, the NATA 1996 inventory was higher than expected and the 1999 inventory was lower than expected; in both cases the differences in non-road emissions are more pronounced than on-road inventory differences. Another observation is that the on-road figures for NATA and GMAP are close for

both 1996 and 1999. The non-road figures show more difference. Some reasons for this could include:

- The NONROAD model was updated post-1999 NATA effort. The AQP used the updated model to generate the 1999 GMAP figures.
- USEPA did not have all of our local data with which to tailor both the MOBILE and NONROAD models, although NONROAD does not have many local data inputs.
- The AQP did not have 1996 and 1999 ship emissions data, so substituted 2002 data. Since an economic slowdown occurred in 2002, this might result in biasing 1996 and 1999 AQP figures low.
- The AQP substituted 2002 data for 1996 and 1999 for locomotives. Therefore, the same concern stated above for ships also applies to locomotives.

Formaldehyde

Another difference between the 1996 and the 1999 NATA reports is the increased cancer risk associated with formaldehyde exposure. The combined increases in exposure from area, on-road and background sources have doubled average formaldehyde air pollution-associated cancer risk across Washington.

Cancer risk conclusions

We conclude that, despite differences between the 1999 and 1996 NATA reports, diesel PM remains the toxic air pollutant of highest concern. Formaldehyde and benzene emissions are of next highest concern. Several other toxic air pollutants exceed *de minimis* risk levels, as well.

Respiratory toxicity hazards

Exposures to three air pollutants (acrolein, nickel and hexamethylene1,6-diisocyanate) with potential to cause respiratory tract toxicity were reported to be excessive in one or more census tracts in Washington. As in 1996, the most significant hazard in 1999 was from acrolein. Most acrolein exposure can be traced to its formation from 1,3-butadiene that is emitted by mobile sources but NATA did not account for this secondary formation process. In the 1999 NATA, 28 of Washington's 33 counties had one or more census tracts with acrolein HQs greater than one.

The range and average of its HQs greater than one across tracts in each affected county is shown in figure 3-35. The county sums of populations in each affected tract are reported next to the county names on the lower axis.

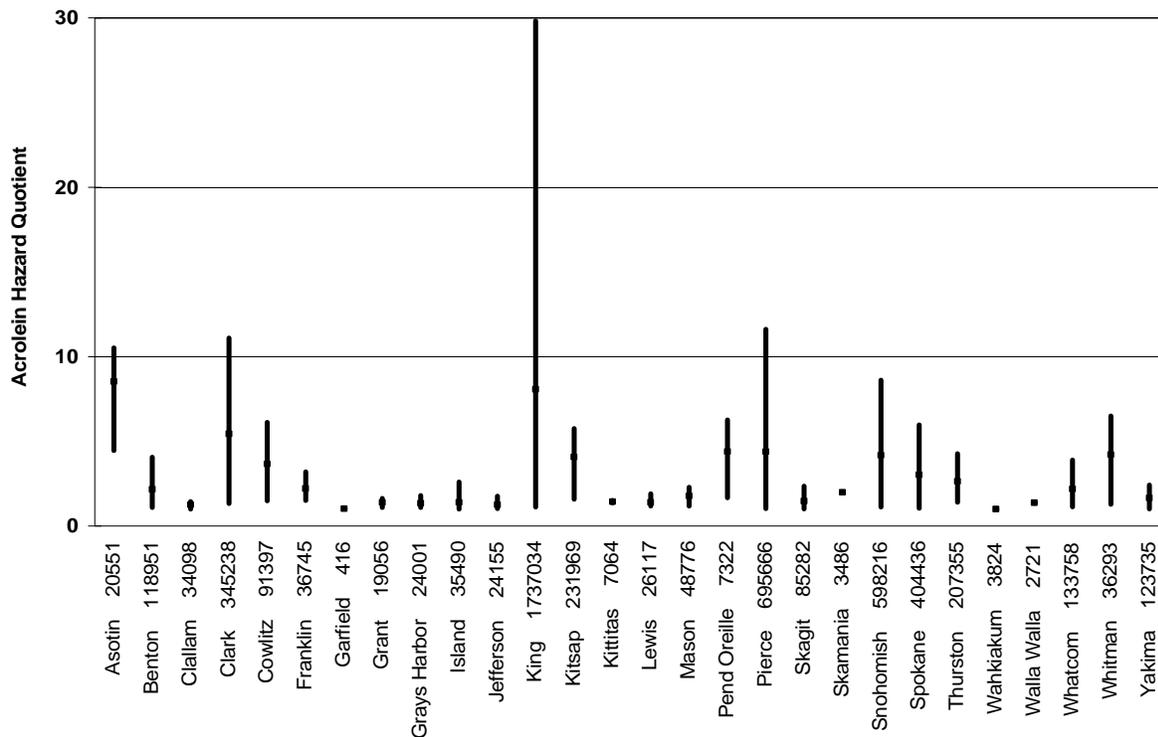


Figure 3-35. Range and average of acrolein hazard quotients greater than one across census tracts in affected counties.

Also, there was one census tract in King County with a HQ for hexamethylenel,6-diisocyanate of 4.492. The affected tract had a population of 6316. There was one census tract in Whatcom County with a HQ for nickel compounds of 1.104. The affected tract had a population of 754.

Neurological toxicity hazards

Exposure to mercury compound air pollution, which has the potential to cause neurological toxicity, was reported to be excessive in one census tract in Whatcom County. The HQ was 1.678. The affected tract had a population of 754.

Conclusions about NATA and recommendations

The 1996 and 1999 NATA findings reinforce our conclusions about the relative importance of each toxic air pollutant, in terms of health risks. Unlike our hazard-weighted emissions inventory rankings, NATA provides an understanding of the level of risks from inhalation exposure to air toxics, because it employs exposure estimates, and in so doing, indicates which toxic air pollutants pose excessive health risks. NATA also helps us identify research needs, and may provide a baseline for comparing future trends.

The key messages from USEPA in NATA regarding Washington are:

- Urban areas generally have higher toxic air pollutants risks than rural areas.
- The median aggregate plausible upper limit of the cancer risk from the studied toxic air pollutants in Washington was about 243- to 320-in-a-million.
- These risks were primarily due to chemicals emitted from mobile sources such as cars and trucks (on-road); and busses, ships, trains, construction equipment, etc. (non-road). Excluding the risk posed by DPM, the aggregate cancer and non-cancer health hazards by source category are as shown in figure 3-36.

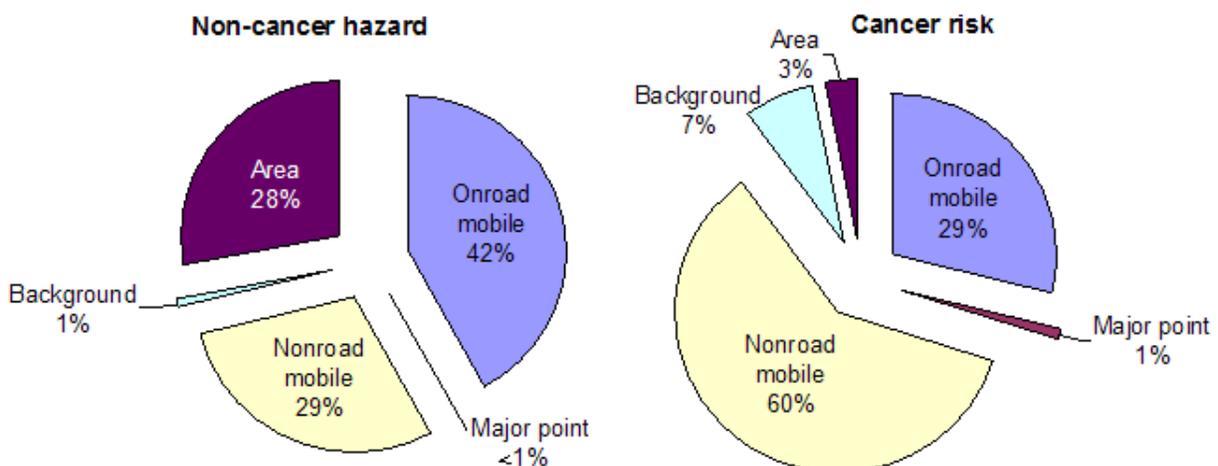


Figure 3-36. Aggregate cancer and non-cancer health hazards from toxic air pollutants (excluding DPM): Relative importance of source categories.

- USEPA has not yet recommended a unit risk estimate for evaluating potential cancer risks associated with exposures to DPM; nonetheless, there is both human and experimental animal evidence that DPM poses a risk of lung cancer to humans at common levels of exposure.⁷⁷ Even the lower end of the plausible upper limit of cancer risk range is above the one-in-a-million excess risk level. At the higher exposure levels found in a number of urban areas, there is an overlap with the occupational exposure levels observed in epidemiological studies.
- For DPM and the toxic air pollutants studied in the NATA, the median of the plausible upper limit of the cumulative excess cancer risk, which ranges from ~50 to 470 per million across Washington counties, is well above the *de minimis* risk level. Most of this risk is associated with exposure to DPM.
- Background levels of a few persistent toxic air pollutants exceed the *de minimis* risk level in Washington.

USEPA warns readers that results of the NATA should not be used as absolute measures to determine whether risks are acceptable, but should target further measurement and assessment activities. For further analysis of toxic air pollutants in Washington, we should work toward reducing uncertainties with (1) better inventory data, (2) more complete atmospheric chemistry, and (3) comparisons of modeled estimates with ambient measurements. Along these lines, we should develop emission inventories with better spatial resolution and accuracy than those used in NATA. We should then use these to make refined risk estimates. We should also conduct more monitoring. Lastly, we should begin to do more refined local-scale assessments to evaluate potential hot spots.

⁷⁷ Limited evidence exists for a casual relationship between risk of lung cancer and occupational exposure to DPM. In addition, laboratory studies have shown unequivocally that DPM can cause benign and malignant lung tumors in rats in an exposure-related manner following chronic inhalation exposure to sufficiently high concentrations. (USEPA 2002. *Health Assessment Document For Diesel Engine Exhaust*. USEPA EPA/600/8-90/057F. May 2002. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, Washington, DC. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060>)

4. Discussion and Recommendations

This report ranks toxic air pollutants by cancer risk and non-cancer health hazard potential. In this final section, the results from different toxic air pollutants evaluation methods are summarized and compared, and reasons for their similarities and differences are discussed. In addition, general recommendations for resource allocation are provided.

We found that ambient toxic air pollutants monitoring and modeled toxic air pollutants concentrations data suggest that the levels and geographic extent of these pollutants in air cause a significant number of premature deaths and serious illnesses among the people of Washington State. Many of the chemicals evaluated in this study have the potential to induce various types of cancer. Lung cancer, leukemia, nasal and liver cancers are associated with chemicals that rank high. These are discussed individually later in this section.

The toxic air pollutant of greatest concern is diesel PM due to long-term widespread human exposure at levels potentially capable of inducing lung cancer. On average in Washington, diesel PM accounts for somewhere between 77 to 89 percent of the cancer risk posed by the top toxic air pollutants, in total. Diesel PM also appears to reduce lung function through pulmonary inflammation and histopathology, even with short-term exposures. DPM and wood smoke PM comprise a significant portion of all PM air pollution, especially fine PM, which is associated with a range of cardiopulmonary diseases and death in human populations. The cardiopulmonary effects associated with diesel PM and the particle fraction of wood smoke are not quantified in this report but are likely to be large with respect to public health impacts.

Toxicity data

The primary uncertainty in the toxicity data that we used in TWEI and NATA is due to our lack of certainty in how well high-dose experimental animal data can be extrapolated to low-level exposure responses in humans. Standardized toxicity criteria, published by USEPA, ATSDR, OEHHA and similar institutions, include numeric quantification of this uncertainty expressed as “uncertainty” and “modifying” factors. An examination of the potency factors of the toxic air

pollutants reveals that confidence is higher for some and lower for others. This generally reflects the quality and applicability of the experimental data to use in human risk assessments. Further, comparing the toxicity risk-based concentrations (RBC) databases of each institution to the other institutions reveals that the RBCs for certain toxic air pollutants are higher or lower, depending on the institution, despite the similarity of the RBCs' purposes. We tried to compensate for these differences by employing an authority preference hierarchy. Use of these criteria for weighting the emission inventory (EI) allows inter-comparison of the pollutants with as much confidence as possible. Known sources of uncertainty in the cancer potency estimates are the following:

- The carcinogenic classification of many of the toxic air pollutants included in this assessment is qualified as “probable” because the existing data are not sufficient to prove these chemicals definitely cause cancer in humans. It is possible but unlikely that some are not human carcinogens at environmentally relevant exposure levels, and that the true excess cancer risk posed by these toxic air pollutants is zero.
- Cancer potency estimates for most of these toxic air pollutants were developed from animal data using conservative (protective) methods to extrapolate between species. In most cases, airborne levels have not directly been shown to cause cancer in human beings. No one knows exactly how much our exposure to these chemicals affects the overall cancer rate, despite any observed epidemiological associations. Epidemiological studies cannot, by themselves, prove that exposure to something caused the correlated response. There are many unmeasured factors that also influence cancer rates.
- All cancer potency estimates used in this assessment were based on linear extrapolation from high to low doses. To the extent that true exposure-response relationships for some toxic air pollutants are non-linear, this assumption may result in significant overestimates of risk.
- Assessing potential health impacts using the TWEI analysis is further limited by the lack of toxicity data for some of the toxic air pollutants. Approximately 20% of the toxic air pollutants with a weight-of-evidence indicating potential carcinogenicity do not have a quantitative cancer potency estimate, and half of the toxic air pollutants do not have a

benchmark concentration for non-cancer health effects more applicable than a TWA-TLV. Even for some of the ubiquitous pollutants identified in this analysis, there is incomplete toxicity information. For example, benzene and 1,3-butadiene have both been associated with reproductive and developmental effects,⁷⁸ but they currently have no benchmark concentrations for such effects.

- Concerning mixture effects and other uncertainties: Although the uncertainty and modifying factors are large, there may be biological interactions among air contaminants of health significance. However, except for acrolein, none of the toxic air pollutants' hazard quotients exceed one.

Emissions inventories

The EI covers about 220 pollutants. The exact number is unknown because there is some overlap in reporting of certain specific chemicals and mixtures that contain them. The completeness and consistency of emissions reporting varies from region-to-region (in general, reporting is better in more populous areas). To compensate for missing information in the inventory, USEPA applied activity factors based upon prior knowledge acquired through more thorough inventories of emissions taken in other places.⁷⁹ Such population-activity-based emission modeling has low geographic resolution. Its reliability is proportional to the population and area of the region being considered. The activity-based emission modeling is “top down-type” modeling, which has certain limitations. An example of the result of this drawback is that the non-road DPM emissions are predicted to be higher than on-road DPM emissions in San Juan County because traffic estimates from WDOT are low and population-based modeling of non-road diesel may be inaccurate for such a small area.

⁷⁸ USEPA. Technical Background Document to Support Rulemaking Pursuant to Clean Air Act Section 112(g): Ranking of Pollutants with Respect to Human Health. EPA-450/3-92-010. Research Triangle Park, NC: U.S. Environmental Protection Agency, 1994

⁷⁹ For example “1990 Emissions Inventory of Forty Potential Section 112(k) Pollutants Supporting Data for EPA’s Section 112(k) Regulatory Strategy: Final Report.” May, 1999. Emission Factors and Inventory Group (MD-14) Emissions, Monitoring and Analysis Division, U.S. EPA and Emission Standards Division (MD-15), U.S. EPA. Research Triangle Park, NC 27711. <http://www.epa.gov/ttnatw01/urban/112krpt.pdf>

Incompleteness of the emissions inventories may not have been adequately corrected in some cases, leading to biased risk estimates. For example, in ORCAA’s re-evaluation of the 1999 NEI found that model-based risk estimates appeared to greatly underestimate the cancer risks from wood smoke. EIs for wood smoke were four to five-fold too low in some cases.⁸⁰ Another limitation of the 1996 and 1999 EIs is the data they contain are out of date. There may be significant differences between these EIs and current conditions of importance.

Ranking methods

Toxicity-weighting the toxic air pollutants emissions inventory and then sorting the results from highest to lowest allowed a screening to identify pollutants needing further examination.

Ranking the toxicity-weighted emissions inventories does not quantify the health risks posed.

The estimates of cancer and non-cancer weights of the EI are not estimates of actual cancer or non-cancer cases resulting from air pollution but are estimates of relative impact of the toxic air pollutants that can be used to prioritize research efforts. Table 4-1 shows the CPWEI rankings of the top 50 toxic air pollutants in each region. The priority toxic air pollutants are in bold font.

Table 4-1. The 50 top-ranked toxic air pollutants in each LAA region’s CPWEI.

	BCAA	CRO
1	Diesel Particulate Matter	Diesel Particulate Matter
2	Chromium & Compounds	Formaldehyde
3	Arsenic Compounds	1,3-Butadiene
4	Formaldehyde	Benzene
5	Benzene	Polycyclic Organic Matter
6	1,3-Butadiene	7-PAH
7	Polycyclic Organic Matter	Chromium & Compounds
8	Nickel & Compounds	Acetaldehyde
9	Cadmium Compounds	Arsenic Compounds
10	Tetrachloroethylene (Perchloroethylene)	1,4-Dichlorobenzene
11	Acetaldehyde	Tetrachloroethylene (Perchloroethylene)
12	Beryllium Compounds	1,3-Dichloropropene
13	1,4-Dichlorobenzene	Ethylene Oxide
14	1,3-Dichloropropene	Methylene Chloride (Dichloromethane)
15	7-PAH	Benzo[a]Pyrene
16	Ethylene Oxide	Nickel & Compounds
17	Selenium Compounds*	Acrylonitrile
18	Methylene Chloride (Dichloromethane)	Cadmium Compounds

⁸⁰ Kelly, J. A Comparison of Local and National Air Toxics Emissions Estimates: Regional Importance of Selected Source Categories. Olympic Region Clean Air Agency, 2940-B Limited Lane NW, Olympia, WA 98502

19	Trichloroethylene	Chloroform
20	Chloroform	Trichloroethylene
21	Lead Compounds	Lead Compounds
22	Acrylonitrile	1,1,2,2-Tetrachloroethane
23	Carbon Tetrachloride	Beryllium Compounds
24	1,1,2,2-Tetrachloroethane	Selenium Compounds*
25	Propylene Oxide	Dioxins/Furans as 2,3,7,8-TCDD TEQ
26	2,4-Dinitrotoluene	Carbon Tetrachloride
27	Dioxins/Furans as 2,3,7,8-TCDD TEQ	Ethylene Dichloride (1,2-Dichloroethane)
28	Hydrazine	Propylene Oxide
29	Ethylene Dichloride (1,2-Dichloroethane)	Hexachlorobenzene
30	Hexachlorobenzene	2,4-Dinitrotoluene
31	Bis (Chloromethyl) Ether	Ethylidene Dichloride (1,1-Dichloroethane)
32	Acrylamide	Benzyl Chloride
33	Benzyl Chloride	2,4-Toluene Diisocyanate
34	Ethylidene Dichloride (1,1-Dichloroethane)	Ethylene Dibromide (Dibromoethane)
35	p-Dioxane	p-Dioxane
36	Ethylene Dibromide (Dibromoethane)	Hydrazine
37	Allyl Chloride	Allyl Chloride
38	Chloromethyl Methyl Ether	Bis (Chloromethyl) Ether
39	1,1,2-Trichloroethane	Acrylamide
40	o-Toluidine	o-Toluidine
41	Aniline	Methyl tert-Butyl Ether
42	4,4'-Methylenedianiline	1,1,2-Trichloroethane
43	bis(2-Ethylhexyl)phthalate	Hexachlorobutadiene
44	2,4-Toluene Diisocyanate	Chloromethyl Methyl Ether
45	Methyl tert-Butyl Ether	Aniline
46	Hexachlorobutadiene	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)
47	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	bis(2-Ethylhexyl)phthalate
48	Dichlorethyl Ether	Acetamide
49	Acetamide	4,4'-Methylenedianiline
50	Benzotrighloride	Dichlorethyl Ether

	ERO	NWCAA
1	Diesel Particulate Matter	Diesel Particulate Matter
2	Formaldehyde	Formaldehyde
3	1,3-Butadiene	Benzene
4	Benzene	1,3-Butadiene
5	Chromium & Compounds	Chloroform
6	7-PAH	Chromium & Compounds
7	Arsenic Compounds	Nickel & Compounds
8	Polycyclic Organic Matter	7-PAH
9	Acetaldehyde	Arsenic Compounds
10	1,4-Dichlorobenzene	Acetaldehyde
11	1,3-Dichloropropene	Polycyclic Organic Matter
12	Tetrachloroethylene	1,4-Dichlorobenzene

	(Perchloroethylene)		Tetrachloroethylene (Perchloroethylene)
13	Ethylene Oxide		1,3-Dichloropropene
14	Nickel & Compounds		Benzo[a]Pyrene
15	Cadmium Compounds		Cadmium Compounds
16	Lead Compounds		Ethylene Oxide
17	Acrylonitrile		Methylene Chloride (Dichloromethane)
18	Methylene Chloride (Dichloromethane)		PAH, Total
19	Chloroform		Lead Compounds
20	Selenium Compounds*		Beryllium Compounds
21	Beryllium Compounds		Trichloroethylene
22	Trichloroethylene		Methyl tert-Butyl Ether
23	1,1,2,2-Tetrachloroethane		Acrylonitrile
24	Carbon Tetrachloride		Carbon Tetrachloride
25	Dioxins/Furans as 2,3,7,8-TCDD TEQ		Selenium Compounds*
26	Hexachlorobenzene		1,1,2,2-Tetrachloroethane
27	Ethylene Dichloride (1,2-Dichloroethane)		1,1,2-Trichloroethane
28	Propylene Oxide		Dioxins/Furans as 2,3,7,8-TCDD TEQ
29	2,4-Dinitrotoluene		Dioxins/Furans as 2,3,7,8-TCDD TEQ
30	Ethylidene Dichloride (1,1-Dichloroethane)		Propylene Oxide
31	Dioxins/Furans as 2,3,7,8-TCDD TEQ		2,4-Dinitrotoluene
32	Hydrazine		Ethylene Dichloride (1,2-Dichloroethane)
33	Bis (Chloromethyl) Ether		Ethylidene Dichloride (1,1-Dichloroethane)
34	Acrylamide		bis(2-Ethylhexyl)phthalate
35	Benzyl Chloride		Benzyl Chloride
36	Ethylene Dibromide (Dibromoethane)		p-Dioxane
37	p-Dioxane		Hydrazine
38	Allyl Chloride		Hexachlorobenzene
39	o-Toluidine		Bis (Chloromethyl) Ether
40	1,1,2-Trichloroethane		Acrylamide
41	Chloromethyl Methyl Ether		Allyl Chloride
42	Aniline		Ethylene Dibromide (Dibromoethane)
43	Methyl tert-Butyl Ether		o-Toluidine
44	4,4'-Methylenedianiline		Pentachlorophenol
45	Hexachlorobutadiene		Chloromethyl Methyl Ether
46	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)		Hexachlorobutadiene
47	Acetamide		2,4-Toluene Diisocyanate
48	bis(2-Ethylhexyl)phthalate		Aniline
49	Dichlorethyl Ether		2,3,4,7,8-Pentachlorodibenzofuran
50	Benzotrichloride		

	NWRO	ORCAA
1	Diesel Particulate Matter	Diesel Particulate Matter
2	Benzene	Formaldehyde
3	Formaldehyde	Benzene
4	1,3-Butadiene	1,3-Butadiene
5	7-PAH	Chromium & Compounds

6	Chromium & Compounds	7-PAH
7	Acetaldehyde	Polycyclic Organic Matter
8	Polycyclic Organic Matter	Acetaldehyde
9	Tetrachloroethylene (Perchloroethylene)	Arsenic Compounds
10	1,4-Dichlorobenzene	1,4-Dichlorobenzene
11	1,3-Dichloropropene	1,3-Dichloropropene
12	Lead Compounds	Tetrachloroethylene (Perchloroethylene)
13	Arsenic Compounds	Nickel & Compounds
14	Acrylonitrile	Ethylene Oxide
15	Nickel & Compounds	Cadmium Compounds
16	Methylene Chloride (Dichloromethane)	Methylene Chloride (Dichloromethane)
17	Chloroform	Chloroform
18	Trichloroethylene	Acrylonitrile
19	1,1,2,2-Tetrachloroethane	Trichloroethylene
20	Cadmium Compounds	Lead Compounds
21	Beryllium Compounds	Beryllium Compounds
22	Selenium Compounds*	Selenium Compounds*
23	Ethylene Oxide	1,1,2,2-Tetrachloroethane
24	Carbon Tetrachloride	Benz[a]Anthracene
25	Dioxins/Furans as 2,3,7,8-TCDD TEQ	Carbon Tetrachloride
26	Ethylene Dichloride (1,2-Dichloroethane)	Dioxins/Furans as 2,3,7,8-TCDD TEQ
27	Propylene Oxide	Propylene Oxide
28	2,4-Dinitrotoluene	Ethylene Dichloride (1,2-Dichloroethane)
29	Ethylidene Dichloride (1,1-Dichloroethane)	2,4-Dinitrotoluene
30	Methyl tert-Butyl Ether	Dioxins/Furans as 2,3,7,8-TCDD TEQ
31	Hydrazine	Methyl tert-Butyl Ether
32	Bis (Chloromethyl) Ether	Ethylidene Dichloride (1,1-Dichloroethane)
33	Acrylamide	Hydrazine
34	Ethylene Dibromide (Dibromoethane)	Benzyl Chloride
35	Benzyl Chloride	p-Dioxane
36	p-Dioxane	Bis (Chloromethyl) Ether
37	Allyl Chloride	Ethylene Dibromide (Dibromoethane)
38	o-Toluidine	Acrylamide
39	Chloromethyl Methyl Ether	Allyl Chloride
40	1,1,2-Trichloroethane	Hexachlorobenzene
41	Aniline	o-Toluidine
42	Hexachlorobenzene	2,4-Toluene Diisocyanate
43	4,4'-Methylenedianiline	1,1,2-Trichloroethane
44	Hexachlorobutadiene	Chloromethyl Methyl Ether
45	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	Aniline
46	Acetamide	Hexachlorobutadiene
47	bis(2-Ethylhexyl)phthalate	4,4'-Methylenedianiline
48	Dichlorethyl Ether	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)
49	Benzotrichloride	bis(2-Ethylhexyl)phthalate
50	Heptachlor	Acetamide

	PSCAA	SRCAA
1	Diesel Particulate Matter	Diesel Particulate Matter
2	Selenium Compounds*	Formaldehyde
3	Benzene	Benzene
4	Chromium & Compounds	1,3-Butadiene
5	Formaldehyde	Chromium & Compounds
6	1,3-Butadiene	Acetaldehyde
7	Polycyclic Organic Matter	Polycyclic Organic Matter
8	Trichloroethylene	7-PAH
9	Tetrachloroethylene (Perchloroethylene)	Tetrachloroethylene (Perchloroethylene)
10	Nickel & Compounds	1,4-Dichlorobenzene
11	Acetaldehyde	1,3-Dichloropropene
12	Arsenic Compounds	Ethylene Oxide
13	1,4-Dichlorobenzene	Arsenic Compounds
14	1,3-Dichloropropene	Nickel & Compounds
15	7-PAH	Methylene Chloride (Dichloromethane)
16	Ethylene Oxide	Trichloroethylene
17	Methylene Chloride (Dichloromethane)	Acrylonitrile
18	Lead Compounds	Chloroform
19	Chloroform	Lead Compounds
20	Cadmium Compounds	Cadmium Compounds
21	Benzo[a]Pyrene	Beryllium Compounds
22	Beryllium Compounds	1,1,2,2-Tetrachloroethane
23	Acrylonitrile	Selenium Compounds*
24	Benz[a]Anthracene	Carbon Tetrachloride
25	Propylene Oxide	Ethylene Dichloride (1,2-Dichloroethane)
26	Carbon Tetrachloride	Propylene Oxide
27	Chrysene	Methyl tert-Butyl Ether
28	Methyl tert-Butyl Ether	2,4-Dinitrotoluene
29	1,1,2,2-Tetrachloroethane	Dioxins/Furans as 2,3,7,8-TCDD TEQ
30	bis(2-Ethylhexyl)phthalate	Ethylidene Dichloride (1,1-Dichloroethane)
31	2,4-Toluene Diisocyanate	Dioxins/Furans as 2,3,7,8-TCDD TEQ
32	2,4-Dinitrotoluene	Hydrazine
33	Dioxins/Furans as 2,3,7,8-TCDD TEQ	Bis (Chloromethyl) Ether
34	Ethylene Dichloride (1,2-Dichloroethane)	Hexachlorobenzene
35	Polychlorinated Biphenyls (Aroclors)	Acrylamide
36	Hydrazine	Benzyl Chloride
37	Bis (Chloromethyl) Ether	p-Dioxane
38	p-Dioxane	Ethylene Dibromide (Dibromoethane)
39	Ethylidene Dichloride (1,1-Dichloroethane)	Allyl Chloride
40	Acrylamide	o-Toluidine
41	Benzyl Chloride	1,1,2-Trichloroethane
42	p-Dioxane	Chloromethyl Methyl Ether
43	Dioxins/Furans as 2,3,7,8-TCDD TEQ	Aniline
44	Pentachlorophenol	4,4'-Methylenedianiline
45	Allyl Chloride	Hexachlorobutadiene

46	Ethylene Dibromide (Dibromoethane)	2,4-Toluene Diisocyanate
47	o-Toluidine	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)
48	1,1,2-Trichloroethane	bis(2-Ethylhexyl)phthalate
49	Chloromethyl Methyl Ether	Acetamide
50	Aniline	Dichlorethyl Ether

	SWCAA	YRCAA
1	Diesel Particulate Matter	Diesel Particulate Matter
2	Polycyclic Organic Matter	Formaldehyde
3	Formaldehyde	Benzene
4	Benzene	1,3-Butadiene
5	Chromium & Compounds	7-PAH
6	1,3-Butadiene	Chromium & Compounds
7	Benzo[b+k] Fluoranthene	Acetaldehyde
8	Arsenic Compounds	Polycyclic Organic Matter
9	7-PAH	1,4-Dichlorobenzene
10	Benzo[a]Pyrene	1,3-Dichloropropene
11	PAH, Total	Arsenic Compounds
12	Acetaldehyde	Tetrachloroethylene (Perchloroethylene)
13	Dibenzo[a,h]Anthracene	Ethylene Oxide
14	Nickel & Compounds	Nickel & Compounds
15	Cadmium Compounds	Acrylonitrile
16	Benzo[b]Fluoranthene	Methylene Chloride (Dichloromethane)
17	1,4-Dichlorobenzene	Chloroform
18	Benz[a]Anthracene	Cadmium Compounds
19	1,3-Dichloropropene	Lead Compounds
20	Selenium Compounds*	Trichloroethylene
21	Tetrachloroethylene (Perchloroethylene)	1,1,2,2-Tetrachloroethane
22	Benzo[k]Fluoranthene	Beryllium Compounds
23	Hexachlorobenzene	Selenium Compounds*
24	Indeno[1,2,3-c,d]Pyrene	Carbon Tetrachloride
25	Beryllium Compounds	Ethylene Dichloride (1,2-Dichloroethane)
26	Ethylene Oxide	Propylene Oxide
27	Lead Compounds	Dioxins/Furans as 2,3,7,8-TCDD TEQ
28	Methylene Chloride (Dichloromethane)	2,4-Dinitrotoluene
29	Chrysene	Ethylidene Dichloride (1,1-Dichloroethane)
30	Acrylamide	Hydrazine
31	Chloroform	Bis (Chloromethyl) Ether
32	Trichloroethylene	Acrylamide
33	Acrylonitrile	Dioxins/Furans as 2,3,7,8-TCDD TEQ
34	Ethyleneimine (Aziridine)	Hexachlorobenzene
35	1,1,2,2-Tetrachloroethane	Ethylene Dibromide (Dibromoethane)
36	Ethylene Dichloride (1,2-Dichloroethane)	Benzyl Chloride
37	Carbon Tetrachloride	p-Dioxane
38	Dioxins/Furans as 2,3,7,8-TCDD TEQ	Allyl Chloride
39	1,1,2-Trichloroethane	2,4-Toluene Diisocyanate
40	Propylene Oxide	Chloromethyl Methyl Ether

41	2,4-Dinitrotoluene	1,1,2-Trichloroethane
42	Methyl tert-Butyl Ether	o-Toluidine
43	bis(2-Ethylhexyl)phthalate	Aniline
44	Hydrazine	4,4'-Methylenedianiline
45	Dioxins/Furans as 2,3,7,8-TCDD TEQ	Methyl tert-Butyl Ether
46	Bis (Chloromethyl) Ether	Hexachlorobutadiene
47	Ethylidene Dichloride (1,1-Dichloroethane)	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)
48	Benzyl Chloride	bis(2-Ethylhexyl)phthalate
49	p-Dioxane	Acetamide
50	Ethylene Dibromide (Dibromoethane)	Dichloroethyl Ether

* Selenium ranked high in the initial screenings of some of the regional emissions inventories because all emissions were counted as selenium sulfide (a probable human carcinogen); however, there is no indication that any of the selenium emitted was this form.

PBTs

Mercury, PCBs, dioxins/furans, and a few other toxic air pollutants would rank higher if persistence and biomagnification potential factors were included. In the current effort, we only accounted for inhalation exposure, not the chemicals' environmental persistence and biomagnification potentials. Similarly, NATA included only inhalation exposure risks, not total exposure. Inclusions of environmental persistence and biomagnification potentials in the ranking would require analysis of persistence and biomagnification potential of each toxic air pollutant to obtain Toxic Equivalency Potentials, which would then be ranked.

Toxic air pollutants listed in Table 5 of the Draft RCRA PBT list⁸¹ were matched to air pollutants listed as HAPs in the CAA amendments. The pollutants found on both lists are noted in the table 4-2.

Table 4-2. Potential and known PBT HAPs

Air Pollutant	CAS
PCDD (Dioxins) and PCDF (Furans)	
1,2,3,4,7,8,9-Heptachlorodibenzofuran	
1,2,3,6,7,8-Hexachlorodibenzofuran	
2,3,4,7,8-Pentachlorodibenzofuran	
2,3,7,8-Tetrachlorodibenzofuran	
Polychlorinated biphenyls (Arochlors)	
7-PAH	

⁸¹ <http://www.epa.gov/fedrgstr/EPA-WASTE/1998/November/Day-09/f29952.htm>

16-PAH	
Acenaphthene	83-32-9
Acenaphthylene	208-96-8
Fluoranthene	206-44-0
Fluorene	86-73-7
Naphthalene	91-20-3
Phenanthrene	85-01-8
Pyrene	129-00-0
Chlordane	57-74-9
DDT, DDD, DDE	
Heptachlor	76-44-8
Heptachlor epoxide	1024-57-3
Pentachlorophenol	87-86-5
Trifluralin	1582-09-8
1,2,4-Trichlorobenzene	120-82-1
1,4-Dichlorobenzene	106-46-7
Bis-(2-ethylhexyl) phthalate	117-81-7
Chloroform	67-66-3
Dibenzofuran	132-64-9
Dibutyl phthalate	84-74-2
Hexachlorobenzene	118-74-1
Hexachlorobutadiene	87-63-3
Pentachloronitrobenzene	82-68-8
Phenol	108-95-2
Antimony and compounds	
Arsenic and compounds	
Beryllium and compounds	
Cadmium and compounds	
Chromium and compounds	
Lead and compounds	
Mercury and compounds	
Nickel and compounds	
Selenium and compounds	
Zinc and compounds	

In the future, we may apply an index that is based on both the inherent toxicity of each chemical and the potential exposure by the different possible routes.⁸² Considering multi-pathway

⁸² Hertwich, E.; Mateles, S.; Pease, W.; McKone, T. 2001. Human Toxicity Potentials for Life Cycle Assessment and Toxics Release Inventory Risk Screening. Environmental Toxicology and Chemistry 20(4). Manuscript available at http://design.ntnu.no/ansatte/hertwich/HTP_ETC.pdf

exposure will better reflect the potential harm of a chemical in the environment. Note that DPM contains certain PAHs but is not listed as a whole in the Draft RCRA PBT list.

NATA

The USEPA's NATAs are risk assessments in that they consider risks associated with estimated exposures to people in different activities. The NATAs' risk estimates place King, Clark and Spokane Counties in the worst 5% of counties in the nation for health risk from airborne toxic chemicals. Because the risk estimates are based on annual average or median exposure concentrations combined with conservative toxicity estimates, they are expected to be high-end but not maximum risk estimates. The 1996 NATA the estimates of plausible upper limits of additional cancer risks range from 19-per-million in the least exposed residents of rural Washington, to 820-per-million in the most exposed residents of urban Washington. Thus, even the least exposed people have a PUL risk 19-fold higher than one-in-a-million (set by USEPA and the courts as a non-trivial "*de minimis*" risk level that could be confidently assessed).

For some toxic air pollutants, and some individuals, the assessment process may underestimate potential cancer risks. Further, the concentrations used in the risk calculations are county-wide averages that may not reflect local hotspots. For example, individuals who spend more of their time near road traffic or large point sources experience higher risks than those who spend less time in these areas.

Monitoring

Compared to the 1996 NATA, the monitoring of certain toxic air pollutants in Seattle has provided more current data with which to screen pollutant risks but of fewer pollutants, in only a small area of the state, and not of actual exposure. In their analysis of toxic air pollutants monitoring data from Seattle, Keill and Maykut concluded "The highest risk estimates based on monitored data are 560 in one million, using the full year (2000) of data in the Georgetown area. This risk estimate includes the 15 toxic air pollutants, *plus* wood smoke and diesel particulate matter. All risk estimates reflect a 70-year exposure period."⁸³

⁸³ Keill and Maykut, 2003

It is noteworthy that Keill and Maykut estimated that wood smoke accounted for a significant portion of the total toxic air pollutant-associated risk. In terms of percent contribution to potential cancer risks, wood smoke contributed approximately 6%, whereas 70% was from DPM and 23% was due to other toxic air pollutants, primarily from mobile sources. Their results are summarized and compared to the cancer potency-weighted emissions inventory ranking and the NATA's ASPEN modeled ambient concentrations in table 4-3.

Table 4-3. Comparison of CPWEI to ASPEN and monitor data (per cent of contribution to the sum of assessed toxic air pollutant risks).

Pollutant	Contributions (%) to Estimated Cancer Risks at Beacon Hill (2000 monitor data)	PSCAA TWEI (%) (1996 modeled data)	King County Cancer Risks (%) (NATA ASPEN, 1996 modeled data)
Estimated Diesel PM Risk	73	92	86
Formaldehyde	7	3	3
Wood smoke	6	NA	NA
Benzene	5	3	3
Carbon Tetrachloride	3	0.001 ^a	2
Chromium	2	0.20	3
Polycyclic Organic Matter	NA	1	1
Chloroform	-	0.10	< 1
Tetrachloroethylene	1	0.10	< 1
Acetaldehyde	1	0.10	< 1
Arsenic	1	No reported emissions	< 1

^a Low because it counts only current emissions not ambient levels

Note: Both the TWEI and the NATA are based on the EI which is a partial account of industrial emissions supplemented with population-based activity emission estimates and VMT-based mobile emission estimates.

PSCAA's analysis is instructive in that it suggests what the concerns in other urban areas of Washington might be if such complete efforts were done in those places.

Toxic air pollutants of greatest concern

Whereas the state-wide cancer potency-weighted EI ranking suggested that 19 toxic air pollutants were potentially of concern (table 4-4), the subsequently released NATAs refined this estimate.

Table 4-4. State-wide cancer potency-weighted EI ranks

Pollutant	Rank
Diesel Particulate Matter	1
Formaldehyde	2
Benzene	3
Chromium & Compounds	4
1,3-Butadiene	5
Polycyclic Organic Matter	6
7-PAH	7
Arsenic Compounds	8
Acetaldehyde	9
Benzo[b+k] Fluoranthene	10
Nickel & Compounds	11
Tetrachloroethylene (Perchloroethylene)	12
Chloroform	13
Trichloroethylene	14
1,4-Dichlorobenzene	15
1,3-Dichloropropene	16
Ethylene Oxide	17
Benzo[a]Pyrene	18
PAH, Total	19

Note: For the for lower ranking pollutants not listed, NATA median exposure estimate poses less than one-in-a-million excess cancer risk throughout Washington.

The 1996 NATA reported the aggregate cancer risk from all the known and potentially carcinogenic toxic air pollutants exceeded 19 in-a-million throughout Washington. It identified 15 toxic air pollutants as being present in one or more counties in Washington at levels high enough to result in exposures with excess cancer risk greater than one-in-a-million. These toxic air pollutants are listed in table 4-5.

Table 4-5. Ranks of state-wide cancer risk estimate by census tract averages in the 1996 NATA ^a

Pollutant	Rank
Diesel Particulate Matter ^b	1
Benzene	2
Carbon Tetrachloride ^c	3
Formaldehyde	4
POM (Total) (excluding 7-PAH only in NATA)	5
Chromium Compounds	6
Chloroform	7
1,3-Butadiene	8
Ethylene Dichloride ^d	9

Ethylene Dibromide ^e	10
Acetaldehyde	11
Tetrachloroethylene (Perchloroethylene)	12
Trichloroethylene	13
Nickel Compounds	14
Arsenic Compounds (Inorganic, Including Arsine)	15
1,3-Dichloropropene	16

^a The state-wide average estimated cancer risks

$$\sum \text{risks in state's tracts} \div \text{number of tracts in the state} = \text{state average risk}$$

of the other toxic air pollutants examined in the NATA were less than one-in-a-million;

^b Based on NATA exposure estimates and the CARB cancer potency URE;

^c Ranked 33rd in the CPWEI

^d Ranked 40th in the CPWEI

^e Ranked 50th in the CPWEI.

The 1999 NATA estimated that 26 toxic air pollutants were at levels in one or more census tract in Washington that would pose excess cancer risk of greater than one-in-a-million if population exposure continued for 70 years. The toxic air pollutants identified are listed in table 4-6.

Table 4-6. 1999 NATA cancer risk estimate summary by census tract maxima and averages.

Pollutant	Rank	Maximum excess cancer risk tract*	Average excess cancer risk tract*
Benzene	1	46.4	27.8
Carbon tetrachloride	2	41.3	10.3
Ethylene dibromide	3	20.4	9.8
Acetaldehyde	4	23.3	7.0
Bis 2 ethylhexyl phthalate	5	29.5	7.0
Butadiene	6	17	6.8
1,1,2,2-Tetrachloroethane	7	12.7	6.7
Naphthalene	8	10.4	4.6
Chromium VI	9	38.5	3.9
Ethylene dichloride	10	7.3	2.1
Tetrachloroethylene	11	10.8	1.9
1,3-Dichloropropene	12	6.8	1.1
Trichloroethylene	13	8.4	0.9
p-Dichlorobenzene	14	2.9	0.9
Vinyl chloride	15	2	0.9
Propylene dichloride	16	1.5	0.8
POM	17	≤ 75.3 ^a	0.6 ^b
Ethylene oxide	18	11.3	0.6
Methylene chloride	19	3	0.5
Quinoline	20	38.4	0.4
Lindane (all isomers)	21	2.5	0.3
Acrylonitrile	22	7.2	0.1
Nickel compounds	23	20.1	0.1

Arsenic compounds (including arsine)	24	6.6	0.1
Cadmium compounds	25	1.6	0.1
Acrylamide	26	4.5	<0.1

^a POM group 1 maximum

^b Average of 8 POM groups

*Excess cancer risk of greater than one-in-a-million

All excess cancer risk values are plausible upper limit estimates of the true value

Alignment of the top ranked pollutants in the CPWEI with those pollutants that had PUL estimates of excess cancer risk greater than one-in-a-million in the 1996 NATA yield figures 4-1 and 4-2, from which DPM is excluded in order to improve visual resolution of the remaining air pollutants in the figures.

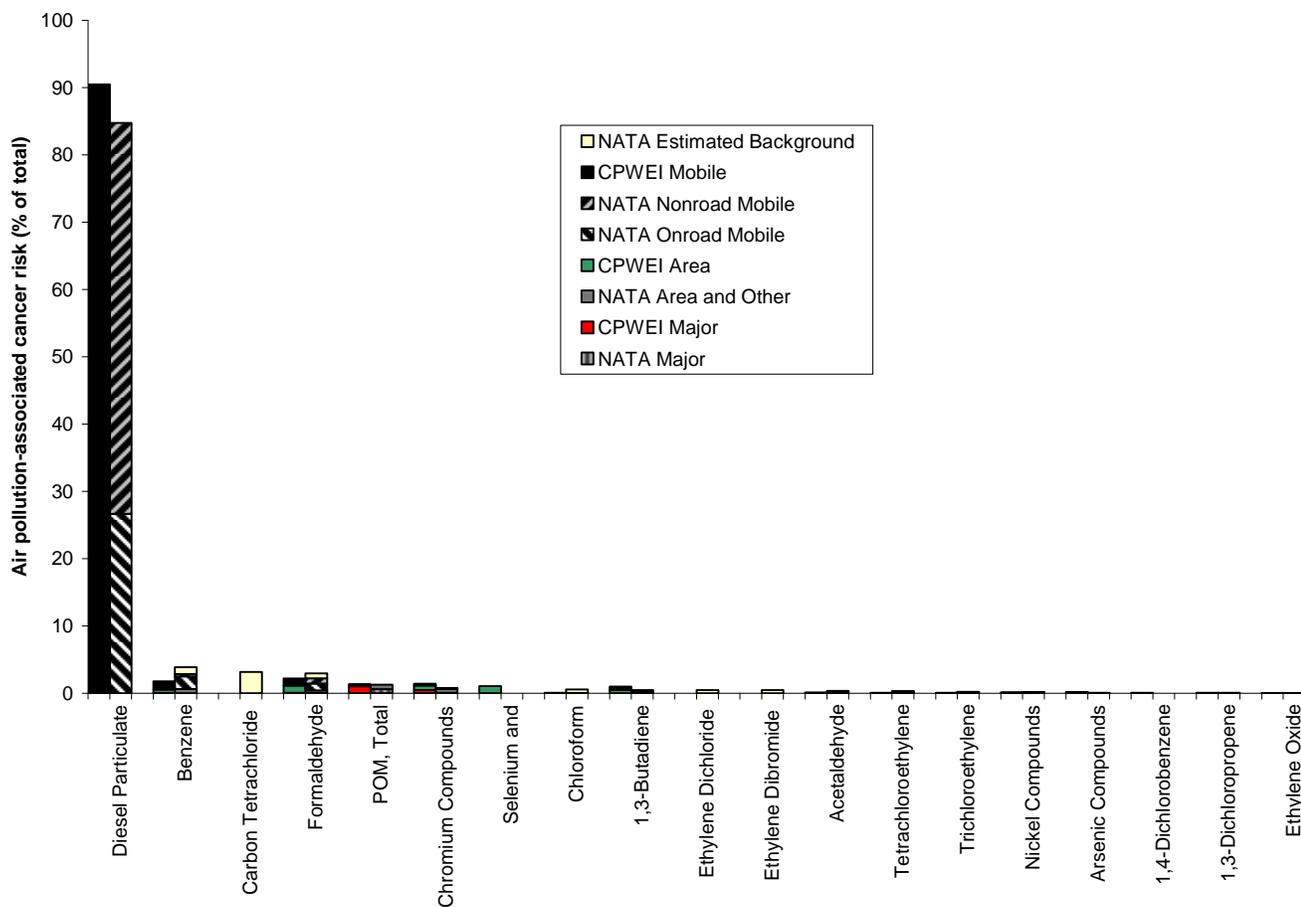


Figure 4-1. Top ranked pollutants in the CPWEI and in the 1996 NATA with estimates of excess cancer risk greater than one-in-a-million.

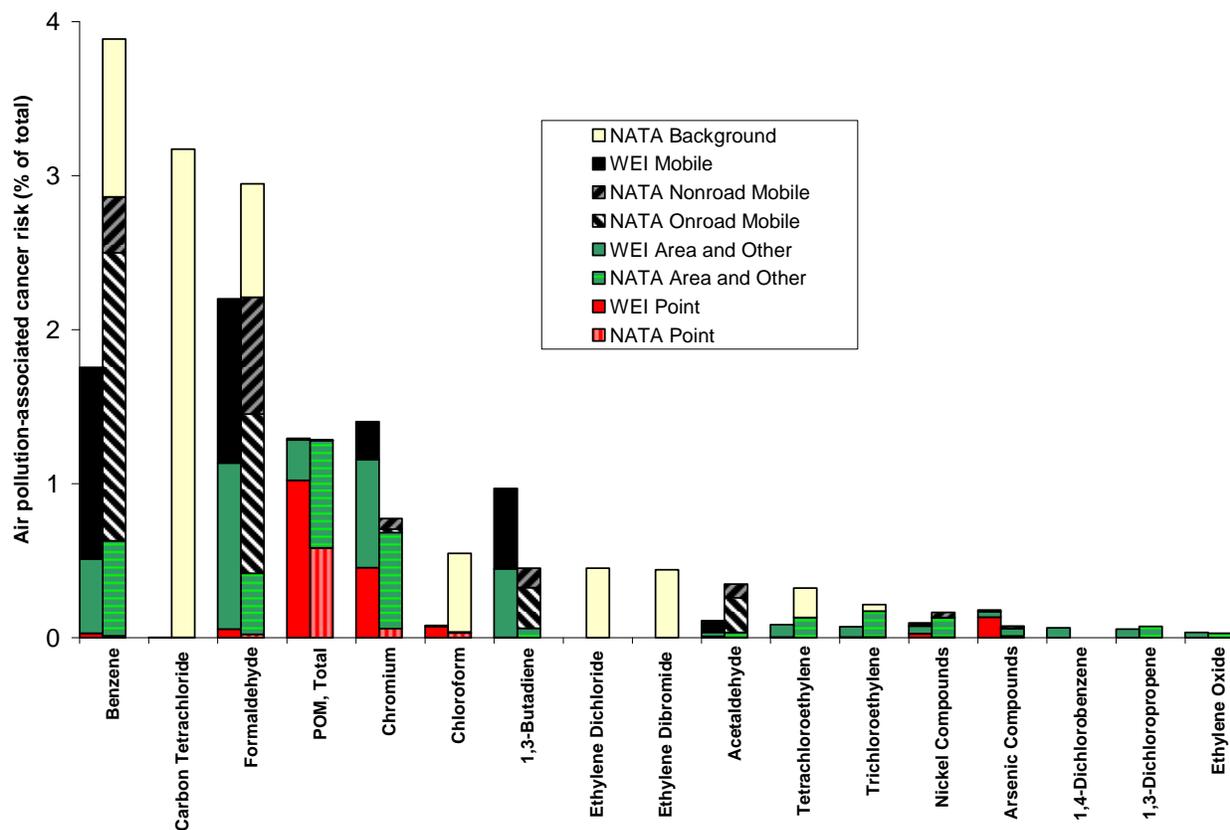


Figure 4-2. Top ranked pollutants excluding DPM, in the CPWEI and in the 1996 NATA with estimates of excess cancer risk greater than one-in-a-million.

PSCAA analyzed toxic air pollutants monitor data from six locations around western King County collected between 2000 and 2004.⁸⁴ In addition, PSCAA analyzed toxic air pollutants monitor data from Beacon Hill, in 2005, which was (and still is) the one site still operating after the initial six-site monitoring study ended.⁸⁵ Both reports conclude that diesel particulate matter and wood smoke particulate matter present the majority of potential health risk in the Puget Sound area. The reports' conclusions about toxic air pollutant ranks are summarized in Table 4-7.

⁸⁴ Keill and Maykut, 2003

⁸⁵ 2005 Air Quality Data Summary. Puget Sound Clean Air Agency. Seattle. July 2006

Table 4-7. Ranks of estimated air pollutants-associated cancer risks based on average levels monitored in Seattle.

Pollutant	Rank	
	2005	2003
Diesel particulate matter	1	1
Wood Smoke	2	2
Formaldehyde	3	4
Carbon tetrachloride	5	3
Chloroform	6	6
Benzene	7	5
Chromium ^a	4	9
Arsenic	8	8
Acetaldehyde	9	7
1,3-Butadiene	10	11
Perchloroethylene (Tetrachloroethylene)	12	10
Trichloroethylene	13	12
Dichloromethane (Methylene chloride)	^b	13
Nickel	11	15
Cadmium	14	14
Lead	15	16

^a Estimated chromium exposure-associated cancer risks are 0.5 to 10.1 per million. At the high end of this range, the estimate is based on PM₁₀ total chromium and USEPA’s 1999 NATA assertion that 66% of total chromium (hexavalent and trivalent) is hexavalent in the Beacon Hill Census Tract. (USEPA 1999 National Air Toxic Assessment. <http://www.epa.gov/ttn/atw/nata1999/>). The low end of the excess risk range estimates is based on results of the hexavalent chromium pilot monitoring conducted in 2005 that shows hexavalent forms are only 3% of total chromium, with resulting risk less than one in a million.

^b Dichloromethane was not included in the 2005 report.

Summary by pollutant

In the following discussion, we summarize and integrate available information on the 21 pollutants of concern identified in this report.

Diesel particulate matter

Sources Diesel PM is the toxic air pollutant posing the largest toxic air pollutant-associated cancer risk to the people of Washington. Approximately 30% of DPM₁₀ emissions are from on-road vehicles such as heavy and light trucks and cars. The other ~70% of DPM₁₀ is emitted from non-road engines. These non-road engines are used in agricultural, construction and mining, logging, marine, and other non-road vehicles and powered equipment.

State and regional WEI ranks Diesel particulate matter ranks first in the statewide cancer potency weighted EI. It accounts for ~90% of the calculated toxic air pollution CPWEI on average statewide. Likewise, DPM ranks highest in each LAA region’s cancer potency weighted EI, ranging from 85% to 95% of the total across the regions. DPM ranks second (after acrolein) in the statewide non-cancer TWEI.

NATA state and regional findings NATA did not include health risk estimates for DPM exposure; however, we applied the California ARB SAB’s URE for DPM to the USEPA’s 1996 NATA estimates of DPM exposure. As noted previously, the range of plausible upper limit UREs proposed by California ARB SAB was $1.3E^{-4}$ to $2.4E^{-3}$ per $\mu\text{g}/\text{m}^3$. Applying these UREs to the NATA exposure estimates across Washington’s census tracts yields plausible upper limit estimates of 552 up to 10,188 excess cancer cases assuming population lifetime DPM exposure at 1996 levels. Similarly, the $1E^{-5}$ to $1E^{-3}$ per $\mu\text{g}/\text{m}^3$ URE range for DPM proposed by USEPA ORD yields plausible upper limit estimates ranging from 42 to 4245 people in the state possibly developing cancer during their lifetimes (risk standardized to 70-years) as a result of DPM exposure. To put this in context of the current causes of death in Washington, keep in mind that DPM is associated mainly with lung cancer, which has a 5-year survival rate of 15%.⁸⁶ The estimate of the plausible upper limit to the true range of numbers of new cancer cases is from 1 to 146 per year (based on NATA exposure estimates and the overlapping USEPA ORD and California EPA risk ranges – see table 4-8).

Table 4-8. DPM risk based on NATA exposure estimates and CARB and USEPA ORD URE ranges: Effects of relative risk and ranking

	DPM URE	Risk of DPM	Rank of DPM
Upper end of unit risk range used by CARB	0.0024	98%	1
URE point CARB	0.0003	85%	1
Lower end of unit risk range used by CARB	0.00013	71%	1
USEPA ORD upper end of unit risk range	0.001	95%	1
USEPA ORD lower end of unit risk range	0.00001	16%	4 ^a

^a At the lowest end of the ORD risk range, DPM would rank 4th behind 1st place Benzene; 2nd place Carbon Tetrachloride; and 3rd place Formaldehyde.

⁸⁶ http://www.cancer.org/docroot/CRI/content/CRI_2_4_1X_What_are_the_key_statistics_for_lung_cancer_26.asp

Confidence in the accuracy of DPM risk estimates is hindered because they are based on uncertain exposure estimates and uncertain cancer potency. Further, the estimate of new cancer cases assumes a continuous level of exposure to DPM; however, the levels are not expected to remain as high as in 1996 due to changes in emissions controls and fuel composition. The number of people who develop cancer resulting from DPM exposure in Washington between 1996 and 2066 (70 years) is not likely to be higher than 10,188 – see table 4-9.

Table 4-9. 1996-level DPM exposure-associated risk summary.

	URE $3E^{-4}$ per $\mu\text{g}/\text{m}^3$	CALEPA Range		USEPA ORD Range	
		$1.3E^{-4}$ per $\mu\text{g}/\text{m}^3$	$2.4E^{-3}$ per $\mu\text{g}/\text{m}^3$	$1E^{-5}$ per $\mu\text{g}/\text{m}^3$	$1E^{-3}$ per $\mu\text{g}/\text{m}^3$
Average risk (per million) in Washington	253	110	2023	8	843
Census tracts over <i>de minimis</i> risk ($>1E^{-6}$)	100%	100%	100%	98.6%	100%
Census tract with maximum risk (Located in Pierce County)	906	393	7248	30	3020

NATA results are most meaningfully interpreted when viewed over large geographic areas, such as national or state levels. The accuracy and resolution of the data is uncertain and can be contradictory or at the county/local level, as evidenced by the unexpectedly high DPM concentration estimate in Adams County and the unexpectedly low estimate for Spokane County in 1996. County-by-county DPM exposure-associated excess cancer risks at sustained 1996 levels are shown in table 4-10. Note that for counties with small populations, the uncertainty in risk projection accuracy is greater than for projections in counties with larger populations.

Table 4-10. Estimates of excess cancer risk from DPM exposure by county and region.

		Average lifetime PUL cancer risk per million	Potential number of associated cancers based on 1990 census data
BCAA	Benton	120	14
	Douglas	145	4
	Kittitas	119	3
CRO	Chelan	97	5
	Klickitat	86	1
	Okanogan	43	1
ERO	Adams	229	3
	Whitman	123	5

	Franklin	108	4
	Asotin	100	2
	Walla Walla	95	5
	Lincoln	92	1
	Grant	89	5
	Stevens	65	2
	Garfield	56	< 1
	Columbia	42	< 1
	Pend Oreille	39	< 1
	Ferry	24	< 1
NWCAA	Island	165	10
	Skagit	142	12
	Whatcom	122	17
NWRO	San Juan	77	1
ORCAA	Thurston	257	40
	Mason	186	7
	Grays Harbor	124	8
	Clallam	115	7
	Jefferson	109	2
	Pacific	81	2
PSCAA	King	372	553
	Pierce	324	181
	Snohomish	276	124
	Kitsap	249	48
SRCAA	Spokane	121	42
SWCAA	Clark	444	98
	Cowlitz	324	25
	Lewis	155	9
	Wahkiakum	124	< 1
	Skamania	122	1
YRCAA	Yakima	159	29
STATEWIDE		253	Total 1273

Estimates by applying the California ARB SAB's plausible upper limit cancer URE of $3E^{-4}$ per ug/m^3 . Source: 1996 NATA

In terms of non-cancer health risk, statewide, diesel particulate matter ranks second after acrolein. DPM accounts for (~3%) of the non-cancer health hazard. Each of the other non-cancer causing toxic air pollutants we examined accounts for less than 2% of the hazard.

Monitoring Although methods for identifying and quantifying DPM in air samples are being developed, ambient DPM levels have not been directly monitored anywhere in Washington; however, given a sufficiently large number of speciated air particulate samples,⁸⁷ PM source

⁸⁷ Speciation of air particulates is quantification of individual particulate elements and various fractions of the particulate organic and elemental carbon.

apportionment is possible by using multivariate receptor model analysis. The analysis procedures involve the UNMIX and PMF models. Using these approaches with PM samples taken at Beacon Hill from 4/1996 to 2/1999, Keill and Maykut⁸⁸ estimated that the average concentration of diesel particulate matter at the Beacon Hill site $1.4\text{-}\mu\text{g}/\text{m}^3$. At this level, they concluded that DPM posed the greatest cancer risk among the 17 toxic air pollutants they studied – accounting for 78% of the air toxic exposure cancer risk. The finding of $1.4\text{-}\mu\text{g}/\text{m}^3$ is similar to the 1996 NATA DPM median concentration estimate for King County (where Beacon Hill is located) of $1.66\text{-}\mu\text{g}/\text{m}^3$.

Recommendations The 1996 and 1999 NATA estimates of diesel PM levels throughout much of Washington are greater than the *de minimis* risk level. Using the CARB SAB cancer potency estimate of DPM to obtain the upper-bound lifetime excess cancer risks at the median county-wide exposure concentrations reveals an increase population lifetime cancer risk from this probable carcinogen of approximately 253-per-million at 1996 levels and 174-per-million at 1999 levels. Reducing DPM exposure by cutting emissions should be given high priority. Even using the least protective extreme of the risk range stated by the USEPA ORD along with the exposure levels calculated in the NATA, DPM would pose an average excess risk of six to eight per million. Diesel engine particulate emissions are a severe drawback to an otherwise superior engine technology; however, application of currently available low sulfur fuel formulation and emission control equipment, such as oxidation catalysts and particulate filters, could significantly reduce this problem if applied to all engines.

Wood smoke

Sources Wood smoke sources, such as residential wood combustion and open burning of logging debris, occur throughout much of Washington and are a significant source of toxic air pollution.

WEI state and regional ranks ORCAA's emissions estimates for residential wood combustion were developed by conducting a local survey of wood burning habits in the study area, and by

⁸⁸ Keill and Maykut (2003)

applying this activity data⁸⁹ to AP-42 factors. ORCAA found that aggregated emissions estimates for residential wood combustion were four to five times higher than NEI estimates for this category.⁹⁰ No statewide wood smoke emission inventory is available; however, based on the reasonable extrapolation of results of a wood smoke inventory by ORCAA, if a statewide inventory were available and Lewtas' cancer potency estimate was used to weight it, wood smoke would rank high among the toxic air pollutants of concern. Based on PMF of particulate matter collected in Seattle, wood smoke poses less excess cancer risk than DPM does but more risk than other toxic air pollutants examined.⁹¹

NATA state and regional findings Although the NATA has not specifically assessed wood smoke, it has partly accounted for it by incorporating estimates of wood burning to calculate emissions of certain wood smoke components. As already noted in this report, there is evidence that the amount of wood burned is significantly more than the estimate used in the NATA. Therefore, we believe NATA underestimates the contribution of wood smoke to certain toxic air pollutant concentrations and consequently underestimates the health risks from exposure to those toxic air pollutants.

Monitoring No direct monitoring of wood smoke concentrations has been done; however, source apportionment analysis of PM_{2.5} by Maykut and others identified vegetative burning (wood-burning fireplaces and yard waste combustion), as the greatest source of PM_{2.5} in Seattle.⁹¹ Based on this information, Keill and Maykut estimated the excess cancer risk from ambient wood smoke exposure at Beacon Hill to be approximately 30 per million (about 6% of the toxic air pollutants exposure risk), placing it 2nd in rank in the *Puget Sound Air Toxics Evaluation*.⁹²

⁸⁹ Tarnai, J. Wood burning stove survey for Idaho, Oregon and Washington State. Washington State University. Social and Economic Sciences Research Center. August 2001

⁹⁰ Kelly, J. (Note that point and area source emissions are for emission year 2000. Other source category emissions are for 1999.

⁹¹ Maykut, N., J. Lewtas, E. Kim, and T. Larson. 2003. Source Apportionment of PM_{2.5} at an Urban IMPROVE Site in Seattle, Washington. *Environ. Sci. Technol.* 37(22): 5135-5142

⁹² Keill and Maykut (2003).

Recommendations To better understand the magnitude of cancer and other adverse health effect risks from wood smoke exposure in Washington, we should expand wood smoke emission inventory efforts to the entire state, and supplement our knowledge with continued source apportionment analysis of PM_{2.5}. Monitoring of atmospheric levoglucosan could be used to indicate wood smoke levels. We also recommend reducing health risks, with focus on reducing maximum individual risk among households with residential wood combustion.

Benzene

Sources Gasoline vapor emissions from motor vehicles constitute a significant source of benzene. The statewide background concentration 0.5- $\mu\text{g}/\text{m}^3$ benzene comprises 50% or less of the ambient level in most Washington Counties. However, in Clark, Cowlitz, King, Kitsap, Pierce, Snohomish, and Thurston Counties, an average annual benzene concentration greater than 1- $\mu\text{g}/\text{m}^3$ exists primarily because mobile (71% of emissions) and area (23% of emissions) sources add significantly to the background level.

State and regional WEI ranks Benzene ranks third in the statewide cancer potency- weighted EI after DPM and formaldehyde. Benzene accounts for ~1.76% of the calculated toxic air pollution cancer potency weighted emissions on statewide average. In terms of non-cancer health risks, the TWEI ranking of benzene is low: the exposure levels estimated in the NATA place its HQ well under one (acrolein is the only toxic air pollutant with an HQ greater than one). The low benzene HQs statewide and in each LAA region indicate ambient level exposures are not likely to result in significant non-cancer health risks.

NATA state and regional findings Inhalation exposure to benzene accounted for 3 to 4 % of the calculated toxic air pollutants exposure-associated cancer risk to Washington citizens. The USEPA's 1996 NATA estimate of cancer risk from median benzene inhalation exposure indicates an excess cancer risk of 11.6 per million, on average, in Washington: 4 to 7 per million in each region except in the PSCAA region where the average estimated risk is 15 per million. Risk exceeds the *de minimis* level in all Washington census tracts. Conversely, in terms of non-cancer health risks, the NATA estimates of the median exposure to benzene suggest an insignificant benzene inhalation health hazard throughout the state.

Monitoring The average and median concentrations measured in Vancouver, WA during 2001-2002 were 1.85- and 1.23- $\mu\text{g}/\text{m}^3$, respectively. If exposure continued for 70 years at these levels, lifetime excess cancer risks would be 14.4 and 9.6 per million, respectively. Benzene comprised about 2% of the toxic air pollutants exposure-associated risk estimated by Keill and Maykut in their evaluation of the air Seattle toxics monitoring data.⁹³

Recommendations Modeled benzene risk and monitoring data closely agree. Further, human carcinogenicity is well established. We recommend reducing risks by intensifying the effort to reduce all combustion engine emissions, and continuing implementation of and support for benzene emission control regulations and low emission transportation alternatives. Further, we anticipate that increasing stringency of federal emission standards and replacement of older motor vehicles with newer ones will reduce ambient benzene concentrations.

Carbon tetrachloride

Sources Carbon tetrachloride (CCl_4) is very stable in the troposphere - with residence times of 30 to 50 years - it is now present at a high, but slowly declining, global background level.⁹⁴ Over 99.9% of the CCl_4 in Washington air is from historic sources. However, area sources within all counties continue to emit small quantities (~2 tons/year, 84% of emissions in Washington). Examples of area source contributors, in order of descending significance, are wastewater treatment plants, traffic markings, miscellaneous organic chemical processes, municipal landfills and consumer products. Larger facilities (in the “major source” category), in Clark, Whatcom, Cowlitz, Skagit, Mason, Spokane, Yakima, Wahkiakum, Island, Skamania, and Pacific counties, continue to release small amounts of CCl_4 (~0.4 tons/year, 16% of emissions in Washington).⁹⁵

⁹³ Keill and Maykut, 2003

⁹⁴ Walker S., Weiss R., Salameh P. 2000. Reconstructed histories of the annual mean atmospheric mole fractions for the halocarbons CFC-11, CFC-12, CFC-113 and carbon tetrachloride. *Journal of Geophysical Research* 105(C6): 14285—14296.

⁹⁵ USEPA. 1996 NATA

State and regional WEI ranks CCl₄ ranks 34th in the statewide cancer potency weighted EI. It accounts for much less than 1% of the calculated toxic air pollution cancer potency weighted emissions on statewide average. CCl₄ ranks 69th in the TWEI suggesting ambient level exposures are not likely to result in significant non-cancer health risks.

NATA state and regional findings The USEPA's 1996 NATA estimate of inhalation exposure cancer risk from median CCl₄ inhalation exposure indicates an excess cancer risk of 9.5 per million in Washington. In terms of non-cancer health risks, NATA estimates that the median exposure to CCl₄ presents an insignificant hazard throughout the state because the HQ of CCl₄ is much less than one in all counties.

Monitoring The average concentration at the six monitoring sites in Bellingham during 2001-2002 was 0.67- $\mu\text{g}/\text{m}^3$. The lifetime excess cancer risk at this level (if exposure continued for 70 years) is 3.6 per million. CCl₄ appears to comprise about 2% of the toxic air pollutants exposure-associated cancer risk. It placed 3rd in rank in the *Puget Sound Air Toxics Evaluation*.⁹⁶

Recommendations Nearly all CCl₄ exposure occurs due to the high background levels prevalent worldwide. Although data are insufficient to completely establish carcinogenic potency in humans, the available information indicates that risk is greater than the *de minimis* level throughout the state. To do our part in solving this problem, we must explore and implement solutions for reducing the ~2.5 tons/year emitted from area and major point sources here in Washington.

Formaldehyde

Sources Formaldehyde is a product of incomplete combustion. It is emitted into the atmosphere from mobile, area and major industrial point sources. The largest primary source of formaldehyde is vehicular exhaust. Mobile and area sources each constitute about 49% of the 6970 tons/year emissions; major point sources comprise the remaining ~2%. The largest area sources are wildfires and prescribed burns. Area sources also include softwood drying kilns;

⁹⁶ Keill and Maykut, 2003

structure fires; residential and commercial heating/boilers (with all types of fuels); consumer products; WWTPs and crematoria. Together on-road, non-road, and area sources make up the majority of formaldehyde exposure in all but the most rural Washington Counties.

Formaldehyde also forms as a result of photochemical oxidation in the atmosphere. The resulting background levels are significant throughout Washington.

State and regional Weighted Emission Inventory Formaldehyde ranks second in the statewide CPWEI. It accounts for ~2.2% of the calculated statewide toxic air pollution cancer potency weighted emissions. In terms of the non-cancer health risks, its fourth-place rank in the statewide non-cancer hazard weighted-emission inventory rank (from 2nd to 5th across the LAA regions) suggest it is insignificant as a regional non-cancer hazard.

NATA state and regional findings The USEPA's 1996 NATA estimate of cancer risk from the median formaldehyde inhalation exposure level implies an excess cancer risk of 8.8 per million throughout Washington. Risk exceeds the *de minimis* level in all Washington census tracts. The risk is highest in Clark, King and Pierce Counties. The NATA estimates of the median inhalation exposure to formaldehyde yield low HQs statewide and in each LAA region. Thus, ambient level exposures throughout the state are not likely to result in significant non-cancer health risks.

Monitoring The average and median formaldehyde concentrations at the Vancouver monitoring site during 2001 were 2.44- and 1.94- $\mu\text{g}/\text{m}^3$, respectively. The lifetime excess cancer risk at this level is 24- and 19 per million respectively – about 4% of the toxic air pollutants exposure-associated cancer risk. Formaldehyde placed 3rd in rank in the *Puget Sound Air Toxics Evaluation*.⁹⁷

Recommendations Limited evidence of formaldehyde's carcinogenicity in humans and sufficient evidence in animals, along with modeled human exposure and ambient monitoring data, strongly suggest that exposure to formaldehyde carries an excess cancer risk greater than one-in-a-million throughout Washington. We recommend looking for solutions for reducing

⁹⁷ Keill and Maykut, 2003

risks by intensifying the effort to reduce all combustion engine emissions, and improvement of controllable area source emissions (softwood drying kilns; residential and commercial heating/boilers; WWTPs; and crematoria). Further, because exposure from indoor sources is probably greater than from outdoor sources, outreach efforts should also focus on steps people can take to reduce individual risk.⁹⁸

Polycyclic organic matter

Sources Polycyclic organic matter (POM) is formed primarily during incomplete combustion of fossil fuels and plant matter. POM has been detected in motor vehicle exhaust, smoke from residential wood combustion (area sources make up about 28% of emissions) and fly ash from coal-fired electricity generating plants. In Washington, nearly 1000 tons/year are emitted. Major point sources, such as paper mills, manufacturers of miscellaneous wood products, and petroleum refining, comprise nearly 72% of all emissions. Table 4-11 shows aluminum mills as the largest major point sources in 1996; however, the aluminum industry has changed a since then: Some of these mills are no longer operating due to higher energy costs.

Table 4-11. Highest reported Polycyclic Organic Matter major point sources in 1996

County	Site Name	Emissions (tons/year)
Cowlitz	Reynolds Metals	477
Pierce	Kaiser Aluminum & Chemical	131
Clark	VANALCO Inc.	53
Klickitat	Goldendale Aluminum	37
Benton	US Energy Dept. Hanford site	7

WEI state and regional ranks The CPWEI of each of the POM toxic air pollutants (table 4-12) was added together giving a total POM cancer potency weighted emissions inventory sum.

Table 4-12. POM chemicals cancer potency-weighted emissions inventories

	Portion of CPWEI (%)	CPWEI Rank
POM	0.907	7
7-PAH	0.196	8

⁹⁸ Using outdoor plywood (containing phenyl-formaldehyde binder) for indoor items would reduce formaldehyde off-gassing relative to indoor plywood, which uses urea-formaldehyde binder.

Benzo[b+k] Fluoranthene	0.102	11
Benzo[a]Pyrene	0.031	19
PAH, Total	0.023	20
Dibenzo[a,h]Anthracene	0.015	23
Benzo[b]Fluoranthene	0.007	25
Benz[a]Anthracene	0.007	26
Benzo[k]Fluoranthene	0.003	29
Indeno[1,2,3-c,d]Pyrene	0.002	31
Chrysene	0.001	32
Benzo[b+k]Fluoranthene	8.3E ⁻¹⁰	72

The combined carcinogenic potency weighted-emission inventory of chemicals in the POM category put this category near fourth place statewide: Altogether accounting for about 1.3% of the CPWEI.

We considered CPWEI ranks of POMs at the regional level. The ranks of POM vary from region-to-region. For the PAHs that had RfCs or similar RBCs, the TWEIs suggest that non-cancer health risks are insignificant.

NATA state and regional findings On average POM inhalation exposure accounts for 1.3% of the calculated toxic air pollutants-associated cancer risks to Washington citizens (the average statewide POM excess cancer risk is 3.83 per million). Total POM-associated cancer risk exceeds the *de minimis* level in 78% of Washington's census tracts. The NATA estimates of median POM inhalation exposure indicated an excess cancer risk of 5.5 per million in SWCAA; 4.2 per million in PSCAA; 2.1 per million in YRCAA; 1.9 per million in SRCAA; 1.6 per million in BCAA; 1.1 per million in NWCAA; 1 per million in CRO and ORCAA; 0.7 per million in ERO; and 0.2 per million in NWRO. In terms of non-cancer health risks, no RfC or RfC-like criterion is available for the total-POM group so the 1996 NATA did not evaluate its non-cancer health hazards.

Excess cancer risk estimates in individual census tracts for 7-PAH were not published by USEPA in their 1996 NATA website, unlike other toxic air pollutants in that study. Because this information was missing, no statewide average risk estimate is available; however, a figure in the NATA gives estimates of cancer risk from median 7-PAH inhalation exposure by county. It indicates an excess risk in BCAA (Benton County) of 0.3 to 1 per million; in CRO counties the range was 0 to 0.3 per million except in Chelan and Douglas where it was 0.3 to 1 per million; in

the ERO counties the range was 0 to 0.3 per million except in Asotin, Franklin and Walla Walla counties where it was 0.3 to 1 per million; in NWCAA and NWRO counties the risk range was from 0 to 0.3 per million; in ORCAA counties, the risk range is from 0 to 0.3 per million except in Mason and Thurston counties, where the range was 0.3 to 1 per million; in PSCAA, SRCAA and YRCAA counties, the risk rank was from 0.3 to 1 per million; and in SWCAA's Skamania and Wahkiakum counties, the range was 0 to 0.3 per million, in Lewis County it was 0.3 to 1 per million, in Cowlitz County, 1 to 3 per million, and in Clark County the estimated risk range was from 3 to 10 per million.

Monitoring 23 different PAHs, including some that are probable human carcinogens, were monitored in the Vancouver study in 2001. Lifelong exposure to the carcinogenic PAHs at the median levels of would have an associated excess cancer risk slightly greater than one-in-a-million. The non-cancer HQs for the PAHs detected that have RfC-like criteria were orders of magnitude below one.

Recommendations The POM chemical group is present in the atmosphere predominantly in particulate form and contains probable and possible carcinogens, which account for 1.3% of the estimated toxic air pollutant cancer risk to Washington citizens. Although there is limited evidence for carcinogenicity of POM in humans, there is sufficient evidence in animals. Further, the true risk from POM may be higher than indicated in 1996 NATA because only a few of the PAHs were included in that assessment. At the median POM exposure levels estimated in the NATA, excess cancer risks exceed one-in-a-million in 8 of the 10 LAA regions in Washington. The risks are greatest (19 per million) in SWCAA's Cowlitz County where major point sources contribute significantly to levels of POM and its subset 7-PAH. Effort to reduce emissions should be made to reduce the excess cancer risks in the affected areas. Current controls should at least be maintained in the compliant areas.

Chromium and chromium compounds

Sources Area sources are the largest source category of chromium and chromium compounds in Washington accounting for over 6600-lbs (45%) of emissions in 1996. These area sources include fabricated plate workshops, hard chromium electroplating, residential heating with wood,

wood preserving, residential/institutional/commercial heating with distillate oil, industrial boilers using residual oil, chromic acid anodizing, industrial inorganic chemical manufacturing, institutional/commercial heating with residual oil and coal, industrial boilers using waste oil and distillate oil, miscellaneous organic chemical processes and cremation.

The major point sources present in several regions are the second largest source category of chromium and chromium compounds in Washington statewide. They account for over 5700-lbs (39%) of emissions. The 44 major facilities (including the USDOE-Hanford site in Benton County, the American National Can Company in King County and other facilities in several counties) that reported chromium emissions listed in the NTI, were the second largest category of contributors to chromium emissions across Washington in 1996. The regions with major source emissions were BCAA, PSCAA, NWCAA, SRCAA, SWCAA, ERO, ORCAA and YRCAA. Mobile sources also contribute significantly atmospheric chromium levels. The 2300-lbs emitted by mobile sources comprised the remaining 16% of chromium emissions in Washington in 1996.

State and regional WEI ranks Chromium compounds rank 4th in the statewide cancer potency weighted EI, accounting for ~1.4% of the calculated carcinogenic air pollution total. The TWEI ranking of chromium and chromium compounds is unimportant because the exposure levels estimated in the NATA place its HQ far below one, indicating ambient level exposures are not likely to result in non-cancer health risks.

NATA state and regional findings Inhalation exposure to chromium accounts for ~0.8% of the calculated toxic air pollutant cancer risk to Washington citizens. The USEPA's estimate of cancer risk from median chromium inhalation exposure in the 1996 NATA indicates an average excess cancer risk of 2.3-per-million in Washington. The risk is lower than one-in-a-million in most of Washington's counties but between one-in-a-million and 8.6 in a million in two of ERO's counties, Asotin and Walla Walla, NWCAA's Whatcom County, all four of PSCAA's Counties, and SWCAA's Clark and Cowlitz counties. In terms of non-cancer health risks, the NATA estimate of the median exposure to chromium suggests an insignificant inhalation health hazard throughout the state.

Monitoring The median chromium concentration monitored in urban and suburban areas of Washington in studies done in the late 1990s through the early 2000s was approximately $1.4E^{-3}$ - $\mu\text{g}/\text{m}^3$. Lifelong exposure at this level would pose an excess cancer risk of 17-per-million. Levels measured by monitors in rural areas have consistently been lower.

Recommendations Chromium is an essential nutrient at low levels but its toxic potential appears to pose an added risk to the general population due to widespread Cr(VI) atmospheric contamination. If the distribution of emissions, 1/3 Cr(VI) and 2/3 Cr(III), is accurate, exposure reduction is needed, especially in more affected regions. Because a large portion of chromium emissions occur at major industrial facilities, a focus on reducing maximum individual risk is advised. On average, chromium and chromium compounds account for 0.8% of the calculated toxic air pollutant cancer risk to Washington citizens, but the risk must be substantially higher for people spending much of their lifetime in close proximity to these point sources. A critical need is improved inventory and monitoring to distinguish between Cr(VI), which is a known human carcinogen, and Cr(III), which is not classifiable as to carcinogenicity. We also recommend reducing risks by intensifying the effort to reduce all combustion engine emissions, and continuing implementation and support for particulate emission control regulations and low emission transportation alternatives.

Chloroform

Sources Chloroform has an estimated atmospheric lifetime of 4.6 months. Thus, its global distribution is nearly homogenous. Sources outside of Washington ultimately contribute ~94% of the ambient concentrations and most of the population's exposure.⁹⁹ Nonetheless, there are significant chloroform sources in Washington – mainly the 116 tons per year (in 1996) from major point sources (~5% of ambient concentrations) but also the 11 tons per year from area sources (~1% of ambient concentrations). The 1996 NTI indicates that the largest area sources of chloroform are WWTPs (~83% of area emissions) and consumer products (~17% of area emissions). The NTI also states that more densely populated counties tend to have the highest area source category emissions.

⁹⁹ <http://www.epa.gov/ttn/atw/nata/taiblconc.html>

State and regional WEI ranks Chloroform accounts for < 0.08% of the average statewide toxic air pollution CPWEI. In terms of non-cancer health risks, the TWEI ranking of chloroform is negligible because the exposure levels estimated in the NATA place its HQ much less than one. The low chloroform HQs statewide and in each LAA region indicate ambient level exposures are not likely to result in significant non-cancer health risks.

NATA state and regional findings Inhalation exposure to chloroform accounts for less than 0.6% of the calculated toxic air pollutant cancer risk to Washington citizens. The NATA estimate of cancer risk from median chloroform inhalation exposure indicates an excess risk of ~1.63-per-million throughout Washington. The risk was higher than average in NWCAA where the three county average was 2.7-per-million. All Washington census tracts exceeded the *de minimis* risk level. In terms of non-cancer health risks, the NATA estimate of the median exposure suggests insignificant chloroform inhalation health hazard throughout the state.

Monitoring The average chloroform concentration at Bellingham monitoring sites was 0.43- $\mu\text{g}/\text{m}^3$ during 2001-2002. The lifetime excess cancer risk at this exposure level is 3.8-per-million. Levels were consistently below the detection limit (0.5- $\mu\text{g}/\text{m}^3$) in the 2001 Vancouver monitoring study.

Recommendations Toxicological evidence suggests that this atmospherically long-lived pollutant is carcinogenic to humans at higher exposure levels. Emissions to ambient air may contribute to attainment of high concentrations. Furthermore, exposure from indoor sources may be even greater than from outdoor sources. For example, showering produces significant concentrations of chloroform when using chlorinated water supplies. Significant reductions in chloroform risk will come only if worldwide efforts to reduce emissions occur. Washington needs to do its share to reduce emissions. The largest source reported in the 1996 NTI is the now partially closed Georgia Pacific facility in Whatcom County, which did not report any chloroform emissions in 2002. As for area sources, the most effective change would be adopting non-chlorine sterilization methods for controlling pathogens in drinking and wastewater treatment facilities. More monitoring and more thorough emissions inventories are also needed to better assess the health risk from chloroform inhalation.

1,3-Butadiene

Sources The NTI estimated 1150-tons of primary emissions of 1,3-butadiene in Washington during 1996. Nearly 620-tons (54%) of these emissions were from incomplete combustion of gasoline and diesel fuels. Approximately 530-tons (46%) of 1,3-butadiene emissions were from area sources such as residential wood combustion, agricultural burning, wildfires and prescribed burns. Major point sources reported approximately 0.1% of the total emissions. Background levels of 1,3-butadiene are insignificant due to its short atmospheric half-life.

State and regional WEI ranks 1,3-Butadiene ranks 6th in the statewide, accounting for just under 1% of the total cancer potency weighted EI. In terms of non-cancer health risks, the TWEI ranking of 1,3-butadiene indicates negligible hazard because exposure levels estimated in the 1996 NATA place its HQ well under one. Low HQs statewide and in each LAA region, calculated in 1996 NATA, indicated ambient level exposures are not likely to pose significant non-cancer health risks.

NATA state and regional findings The USEPA's 1996 NATA estimates of cancer risk from median 1,3-butadiene inhalation exposure indicate an excess risk of 1.35-per-million in Washington. Approximately 60% of the census tracts in Washington have cancer risk estimates of one-in-a-million or greater. Some counties fare worse than others: in CRO's Chelan County, the estimated risk is 1.09 per million; in ERO's Franklin and Stevens counties, the risk is 1.55- and 1.11-per-million respectively; in NWCAA's Whatcom County, the estimated risk is 1.11; in ORCAA's Thurston, Grays Harbor and Clallam counties, the risk is 1.28-, 1.09- and 1.07-per-million, respectively; in PSCAA's King, Pierce and Snohomish counties, the estimated risk is 2.14-, 1.46- and 1.21-per-million, respectively; and in SWCAA's Clark County, the average risk is 1.82-per-million. In terms of non-cancer health risks, the 1996 NATA estimate of the median 1,3-butadiene inhalation exposure suggests these health hazards are nearly negligible throughout the state.

Monitoring The average 1,3-butadiene concentration at the Bellingham monitoring sites was $0.1\text{-}\mu\text{g}/\text{m}^3$ during the 2001-2002 study. The excess cancer risk associated with a lifelong exposure at this level is 3-per-million. This is similar to the 1,3-butadiene cancer risk level

predicted in the 1996 NATA. 1,3-Butadiene was also detected at 1.125- $\mu\text{g}/\text{m}^3$ in one of the 57 samples taken in the 2001 Vancouver study.

Recommendations The upper-bound excess cancer risk from exposure to 1,3-butadiene – a known human carcinogen - is over one-in-a-million in 60% of the state’s census tracts. At these levels, we recommend reducing risks by intensifying efforts to reduce emissions from gasoline and diesel engines, and by continuing implementation and support for emission control regulations and low-emission transportation alternatives. We also recommend reducing emissions from residential wood combustion, agricultural burning and prescribed burning.

Ethylene dichloride (1,2-Dichloroethane)

Sources Ethylene dichloride is an additive in leaded gasoline, which is still used in the U.S. as an aviation fuel. In 1986, the USEPA banned EDC as a grain and food fumigant. However, EDC is still used in the manufacture of paints, coatings, adhesives and solid fuel; solvent bonding of polycarbonate products; solvent extraction of seeds, animal fats, and pharmaceutical materials; cleaning polyvinyl chloride manufacturing equipment; preparation of polysulfide compounds; leaching of copper ore; and the manufacture of film. Most of these uses do not occur in Washington but do affect us because EDC has an estimated atmospheric half-life for its gas-phase reaction with the hydroxyl radicals of 45-days. Because of this persistence, background sources comprise nearly all ambient concentrations, as shown in table 4-13.

Table 4-13. Estimated annual average ambient concentrations and percent source contributions of ethylene dichloride

	Major	Area and Other	Mobile	Estimated Background
Concentration ($\mu\text{g}/\text{m}^3$)	1.76E ⁻⁵	1.46E ⁻⁵	0	6.10E ⁻²
Contribution by source category	0.03%	0.02%	0%	99.95%

Source: 1996 NATA

An estimate of ethylene dichloride area source emissions in Washington in 1996 was 402.3-lbs. These area source emissions, occurring in all Washington counties, may include consumer

products, heating with coal, miscellaneous organic chemical processes, and municipal landfills. Reported major point sources of EDC reported were 3-lbs from Weyerhaeuser Co. in Cowlitz County and 123-lbs from Transalta Centralia power plant in Lewis County in 1996.

State and regional WEI ranks Ethylene dichloride ranks 42nd in the statewide cancer potency weighted EI - accounting for 0.00017% of the total. The apparent discrepancy in regional ranks compared to the statewide rank results from differing chemicals being reported by each region, especially of specific PAHs, and from fewer chemicals being reported by each LAA relative to the state as a whole. In terms of non-cancer health risks, the TWEI ranking of EDC is insignificant. The low HQs statewide and in each LAA region calculated in the 1996 NATA indicate ambient level exposures are not likely to result in significant non-cancer health risks.

NATA state and regional findings The NATA estimate of average EDC inhalation exposure indicates a uniform excess cancer risk of 1.6-per-million throughout Washington. EDC accounts for 0.45% of the total toxic air pollutant-associated cancer risk estimated in the 1996 NATA, placing it in 10th rank statewide. In terms of non-cancer health risks, the NATA estimate of the average EDC inhalation exposure suggests these health hazards are insignificant throughout the state.

Monitoring There is no record that atmospheric EDC concentrations have ever been monitored in Washington.

Recommendations Sufficient evidence of carcinogenicity in animals but inadequate human data heightens the uncertainty in the EDC risk screening. Notwithstanding, it appears that cancer risk may exceed the *de minimis* level. Significant reductions in EDC-associated cancer risk will come only if worldwide efforts to reduce emissions occur. As for outdoor EDC sources, Washington needs to do its share to reduce emissions. Monitoring and better emissions inventories are also needed to better assess the health risk from EDC inhalation exposure in Washington.

Ethylene dibromide (1,2-Dibromoethane)

Sources Before it was banned by the USEPA in 1984, ethylene dibromide was used as a

fumigant in soil and on grain, fruits, and vegetables and as a lead scavenger in leaded gasoline, which has been mostly phased out in the US. EDB is still used in miscellaneous organic chemical process as a solvent for resins, gums, waxes, and as a chemical intermediate in the manufacture of dyes, pharmaceuticals and other organic compounds. It also enters the atmosphere from municipal landfills. In Washington in 1996, source category contribution estimates are 2.6 lbs/year (47%) from area sources and 2.9 lbs/year (53%) from major point sources. Major point sources make small contributions to local exposure and to the global background of EDB. Less than one one-hundredth of one percent of EBD exposure is attributable to area and major point sources within Washington. Because EDB is persistent (estimated atmospheric half-life of 40 days) and only small amounts of emissions are reported in Washington, it appears nearly all exposure results from the globally present background level, as shown in Table 4-14.

Table 4-14. Estimated annual average ambient concentrations and percent source contributions of ethylene dibromide

	Major	Area and Other	Mobile	Estimated Background
Concentration ($\mu\text{g}/\text{m}^3$)	7.93E ⁻⁸	1.77E ⁻⁷	0	7.70E ⁻³
Contribution by source category	0.001%	0.002%	0%	99.997%

Source: 1996 NATA

State and regional WEI ranks Ethylene dibromide ranks 51st in the statewide cancer potency weighted EI - accounting for about 7E⁻⁶% of the total CPWEI. The apparent discrepancy in regional ranks compared to the statewide rank results from differing chemicals being reported by each region, especially of specific PAHs, and from fewer chemicals being reported by each LAA relative to the state as a whole. In terms of non-cancer health risks, the TWEI ranking of EDB is negligible because the exposure levels estimated in the NATA place its HQ well under one. The low HQs statewide and in each LAA region calculated in NATA indicate ambient level exposures are not likely to result in significant non-cancer health risks.

NATA state and regional findings The median potential excess cancer risk estimate from EDB exposure is 1.3-per-million throughout Washington. EDB accounted for 0.44% of the total toxic

air pollutant cancer risk estimated in the 1996 NATA placing it in 11th rank statewide. In terms of non-cancer health risks, the NATA estimate of the median EDB inhalation exposure suggests its health hazard is insignificant throughout the state.

Monitoring Atmospheric EDB concentrations were monitored but below detection limits in the 2001 Vancouver toxic air pollutants study. EDB has not been monitored elsewhere in Washington.

Recommendations Universal exposure to this potentially carcinogenic pollutant raises concern. Although there is sufficient evidence of carcinogenicity in animals, human data are inadequate, increasing uncertainty in the EDB risk screening. Significant reductions in EDB risk will come only if worldwide efforts to reduce emissions occur. Washington could do its share as well. More monitoring and better emissions inventories are also needed to better assess our health risk from EDB inhalation.

Acetaldehyde

Sources The NTI estimate of primary emissions of acetaldehyde in Washington in 1996 was 2180 tons. Sources of acetaldehyde include emissions from on-road and non-road internal combustion engines (68% of all emissions). Acetaldehyde is used as an intermediate in the production of certain chemicals. It is also a product of incomplete combustion of wood (in fireplaces and woodstoves), wildfires, and agricultural burning; and it is emitted from boilers, process heaters and other area (25% of emissions) and major point sources (7% of emissions).

State and regional WEI ranks Acetaldehyde ranks 10th in the statewide cancer potency weighted EI - accounting for approximately 0.11% of the total. In terms of non-cancer health risks, the TWEI ranking of acetaldehyde is negligible because the exposure levels estimated in the 1996 NATA place its HQ well under one. The low HQs statewide and in each LAA region calculated in NATA indicate ambient level exposures are not likely to result in significant non-cancer health risks.

NATA state and regional findings The USEPA's 1996 NATA estimate of lifetime cancer risk from acetaldehyde inhalation exposure indicates a statewide average of 1.04-per-million. It ranks 11th, having 0.35% of the total cancer risk estimated in NATA. Approximately 50.5% of the census tracts in Washington have acetaldehyde-associated cancer risks that greater than or equal to one-in-a-million. These counties include PSCAA's King, Pierce, Kitsap and Snohomish, where the estimated excess cancer risks were 1.7-, 1.5-, 1.11- and 1.06-per-million, respectively. In SWCAA's Clark County, the average excess cancer risk from acetaldehyde inhalation was estimated as 1.95-per-million. In terms of non-cancer health risks, the NATA estimate of the median acetaldehyde inhalation exposure suggests these health hazards are insignificant throughout the state.

Monitoring The average acetaldehyde concentration in Bellingham in 2001-2 was 4.5- $\mu\text{g}/\text{m}^3$. If this exposure level persists lifelong, it would have an associated excess cancer risk of 2-per-million. Average and median concentrations observed in Vancouver in 2001 were 2.1- and 1.52- $\mu\text{g}/\text{m}^3$, respectively. The estimated excess cancer at these exposure levels, if persisting throughout lifetime, would be 4.6 and 3.3-per-million, respectively.

Recommendations Estimated excess cancer risks from inhalation of this probable human carcinogen are greater than one-in-a-million in more densely populated areas in Washington, mostly due to on-road and non-road mobile sources, which constitute the largest source category. Area sources also make significant contributions to acetaldehyde cancer risk. Major point sources – primarily wood products facilities in Clallam, Clark, Cowlitz, Jefferson, Pierce, Walla Walla and Whatcom counties, while small in their contribution relative to mobile and area sources, may nonetheless create risk hotspots in these locations. Improvements in internal combustion engine emission controls as well as in area source and major point source controls should be used to reduce acetaldehyde inhalation exposure cancer risks.

Tetrachloroethylene (Perchloroethylene)

Sources Tetrachloroethylene is a solvent used primarily in dry-cleaning operations. It is also used in degreasing operations, and in paints, coatings, adhesives, aerosols, specialty chemical production, printing inks, silicones, rug shampoos, and laboratory solvents. According to the

1996 NATA, 568 tons were emitted from area sources (98.5% of the emissions in Washington) and 8.43 tons were emitted from major point sources (1.46% of emissions). Tetrachloroethylene has an atmospheric lifetime of approximately three months. According to the NATA, an estimated 61% of the public's tetrachloroethylene exposure in Washington was from background sources on average in 1996. Area sources contribute significantly to exposure in the two-thirds of Washington counties that have the greatest population density. For example, in King County, area sources contributed more to exposure than did background sources. Whereas 39% of exposure was from area sources, and 0.2% of exposure was from major point sources. Among these major sources, the largest ones that reported emissions in 1996 were in King County.

State and regional WEI ranks Tetrachloroethylene ranks 13th in the statewide cancer potency weighted EI, accounting for 0.085% of the total. In terms of non-cancer health risks, the TWEI ranking of tetrachloroethylene is insignificant because the exposure levels estimated in the 1996 NATA place its HQ well under one, an indication that ambient level exposures are unlikely to result in significant non-cancer health risks.

NATA state and regional findings Tetrachloroethylene ranked 12th among the cancer causing air pollutants, having 0.32% of the total estimated cancer risk. Tetrachloroethylene cancer risk was one-in-a-million or higher in about 38% of census tracts across Washington. These census tracts are in SWCAA's Clark County, where the average excess risk from tetrachloroethylene exposure was estimated as 1.02 per million, and in PSCAA's King, Pierce and Snohomish counties, where the estimated average risks were 1.34, 1.02 and 1.00-per-million, respectively. In terms of non-cancer health risks, the NATA estimate of the median exposure to tetrachloroethylene suggests an insignificant inhalation health hazard throughout the state.

Monitoring Tetrachloroethylene was detected in 6 of 57 samples in the 2001 Vancouver monitoring study. The average and median concentrations were 0.35 and 0.41- $\mu\text{g}/\text{m}^3$, respectively. The lifetime excess cancer risk at these levels would be 1.9 and 2.3-per-million, respectively.

Recommendations Better monitoring and emissions inventory efforts for tetrachloroethylene are needed to more definitively identify sources. Existing information points to the

recommendation that we look for solutions to reduce risks by intensifying the effort to reduce all tetrachloroethylene emissions, particularly those from area sources. Ultimately, tetrachloroethylene should be phased-out of production and chemical(s) with less carcinogenic potential and other adverse environmental effects should be substituted for it as necessary.

Trichloroethylene

Sources Degreasing operations are the largest sources of trichloroethylene (TCE) emissions to the atmosphere. TCE was used primarily between the 1940s and 1970s to clean machine parts and in the semiconductor industry. Other significant TCE emissions include paints and coatings, adhesive formulations, publicly owned treatment works, PVC production, distribution facilities, and solvent reclamation. Statewide trichloroethylene emissions estimates (totaling 1390-tons) were reported in the 1996 NATA. According to the NATA, 1350 tons (97% of emissions in Washington) were from area sources and 42.9-tons (3% in Washington) were from major point sources (mostly facilities in King County).

State and regional WEI ranks TCE ranks 15th in the statewide cancer potency-weighted EI, accounting for 0.07% of the total. In terms of non-cancer health risks, the TWEI ranking of TCE is negligible because the exposure levels estimated in the NATA place its HQ well under one.

NATA state and regional findings Inhalation exposure to TCE, at 1996 levels, accounted for a statewide average excess cancer risk of 0.639-per-million. It ranked 13th accounting for 0.214% of the total estimated cancer risk to Washington citizens. Results of ASPEN modeled annual average concentrations from different source categories were: Area sources accounted for 0.299- $\mu\text{g}/\text{m}^3$ of outdoor concentrations (~ 77% of the public's exposure); TCE's atmospheric half-life of approximately 27 to 272 hours results in an estimate of background sources accounting for 0.081- $\mu\text{g}/\text{m}^3$ of the average outdoor concentration (21% of exposure); Major point sources were estimated to account for 0.0076- $\mu\text{g}/\text{m}^3$ (2% of exposure) on average. The census tract with the highest excess cancer risk from TCE (7.93 per million) was located in King County. The average across all of King County was 1.41 per million. Also, in Snohomish County, the estimated risk was 1.11 per million. In all, there are 277 census tracts within the King, Pierce and Snohomish counties where the estimated excess cancer risk was greater than one-in-a-million. In terms of non-cancer health risks, the NATA estimate of the median TCE exposure

suggests an insignificant inhalation health hazard throughout the state.

Monitoring The average concentration at the six Seattle monitored in Bellingham during 2001-2002 was $\sim 0.045\text{-}\mu\text{g}/\text{m}^3$. The lifetime excess cancer risk at this exposure level is 0.9 per million. TCE was not detected in the 2001 Vancouver toxic air pollutants monitoring study.

Recommendations Area sources are believed to account for most outdoor exposure; however, the 1996 NATA noted that only in King County did exposure to this possibly carcinogenic air pollutant exceed the level where cancer risk was greater than the one-in-a-million *de minimis* level. Monitoring found negligible levels in Bellingham and undetectable levels in Vancouver. It is noteworthy that indoor TCE exposure may be greater than outdoor exposure where TCE containing consumer products - such as typewriter correction fluid, adhesives, paint removers, and spot removers - are used. Current monitoring in the Seattle area should ultimately help answer the questions of how concerned should we be, and what costs are justified in order to reduce TCE exposure-associated cancer risk.

Nickel compounds

Sources The NTI total statewide emissions of nickel compounds estimate for Washington were 14.9 tons in 1996. The majority (56%) of the emissions were from area sources, such as fuel (residential oil, distillate oil and coal) combustion. Motor fuel combustion, especially in non-road engines was a significant contributor (22%) to the total emissions. Major point sources accounted for the remaining 22%. These major point sources include several industrial processes such as production of various metal alloys, catalysts and nickel-cadmium batteries, and electroplating. Nickel also occurs in nature from sources such as volcanoes and wind erosion of soils.

State and regional WEI ranks Nickel and compounds ranked 12th in the statewide cancer potency-weighted EI, accounting for about 0.01% of the total. In terms of non-cancer health risks, the TWEI ranking of nickel and compounds was insignificant because the exposure levels estimated in the 1996 NATA place its HQ well under one, indicating that ambient exposures are not likely to result in non-cancer health risks.

NATA state and regional findings Inhalation exposure to nickel and compounds accounted for an average excess cancer risk of 0.49-per-million (approximately 0.1% of the total calculated toxic air pollutants-associated cancer risk to Washington citizens), placing this metal 14th in rank, statewide. The only county with an average estimated exposure level above the *de minimis* cancer risk level was PSCAA's Kitsap County, with nickel-associated excess cancer risk of 1.59-per-million; however, estimates of excess cancer risk from nickel exposure exceeded one-in-a-million in 164 census tracts (14% of the state), located across five counties in Washington. These are summarized in the table 4-15.

Table 4-15. Number of census tracts where nickel exposure-associated additional cancer risk estimates exceed one-in-a-million.

County	Number of census tracts where cancer risk estimates exceeded one-in-a-million
King	112
Kitsap	29
Clark	17
Cowlitz	4
Pierce	2

In terms of non-cancer health risks, the 1996 NATA estimate of the median exposure to nickel and its compounds suggests an insignificant inhalation health hazard throughout the state.

Monitoring Interagency Monitoring of Protected Visual Environments (IMPROVE) studies conducted during 2000-2001 report median nickel concentrations ranging from 0.001 to 0.0015- $\mu\text{g}/\text{m}^3$ at select urban and suburban monitoring sites in Washington.

Recommendations The chemical forms of nickel emissions in Washington were not specified in the 1996 NTI. If we assume that these emissions were in the form of the known human carcinogen nickel subsulfide/refinery dust, then exposure levels apparently were greater than the *de minimis* cancer risk level in several areas in the state. Most notably, higher outdoor ambient concentrations were monitored in parts of King County in 2000-2001. These findings prompt a recommendation to reduce population nickel exposure by intensifying the effort to reduce nickel emissions in areas where excess nickel-associated cancer risk is estimated to be greater than one-in-a-million. As implied, due to lack of EI information on the specific forms of nickel emitted,

the NATA cancer risk estimates may be too high. Therefore, future EIs should itemize nickel forms. In any case, the NTI indicates that most nickel emissions are from combustion of fossil fuels for heating and in engine powered equipment. It follows that changes in fuel-nickel content and improved fuel efficiency would achieve the greatest emissions reductions.

1,4-Dichlorobenzene

Sources The NTI reported a total of over 229-tons of 1,4-dichlorobenzene emissions in Washington during 1996. Area source emissions accounted for 99.95% of this total. These area sources are thought to consist of emissions from coating and engraving procedures used in metal manufacturing processes; also fumigation and landscape maintenance uses. The only major point source that reported emissions in Washington during 1996 was the Anacortes WWTP (213 lbs), located in Skagit County; however, 1,4-dichlorobenzene is probably emitted from other WWTPs, too. 1,4-Dichlorobenzene is not known to occur in nature but it has a calculated half-life of 1 month, therefore background levels probably account for a significant portion of population inhalation exposure.

State and regional WEI ranks 1,4-Dichlorobenzene accounts for ~0.064% of the average statewide toxic air pollution cancer potency weighted emissions inventory. 1,4-dichlorobenzene does not appear to pose non-cancer inhalation health hazards in Washington.

NATA state and regional findings The USEPA's 1999 NATA estimate of inhalation exposure cancer risk from median 1,4-dichlorobenzene inhalation exposure indicates an excess cancer risk of 0.387 per million in Washington (USEPA did not examine 1,4-dichlorobenzene in its 1996 NATA). Although no counties had countywide average excess 1,4-dichlorobenzene-associated cancer risk exceeding one-in-a-million, risk estimates exceeded one-in-a-million in 88 census tracts: 2 in Pierce County, 1 in Snohomish County, the rest in King County. In terms of non-cancer health risks, the 1999 NATA reports exposure to 1,4-dichlorobenzene presents an insignificant hazard throughout the state: The HQ of 1,4-dichlorobenzene is much less than one in all census tracts.

Monitoring 1,4-dichlorobenzene was detected in 18% of samples taken in the Vancouver Toxic air pollutants study of 2001, with median and mean concentrations of 0.3 and 0.42- $\mu\text{g}/\text{m}^3$,

respectively. Lifelong exposure at these levels has an associated additional cancer risk of ~3 and ~5 per million. 1,4-dichlorobenzene has not been an analyte in other monitoring studies in Washington.

Recommendations 1,4-Dichlorobenzene exposure-associated cancer risks are apparently greater than one-in-a-million in a several census tracts. Existing information leads to the recommendation that we look for ways to reduce 1,4-dichlorobenzene exposure by intensifying the effort to reduce all 1,4-dichlorobenzene emissions, particularly those from area sources.

Inorganic arsenic, arsenic compounds and arsine

Sources The NTI lists 2844-lbs of emissions of inorganic arsenic, arsenic compounds and arsine, in Washington during 1996. Major point sources accounted for about 74% (2113-lbs) of the total emissions. The major point source facilities that reported the highest arsenic emissions were paper mills in Cowlitz, Clark, Pierce and Walla Walla counties, and the US Energy Dept. (Hanford) in Benton County. Area sources accounted for about 20% (568-lbs) of the total statewide emissions. These sources of arsenic emissions included crematoria; industrial boilers and institutional/commercial heating using distillate oil, natural gas, residual oil, waste oil, or wood/wood residue; industrial gases manufacturing; industrial inorganic chemical manufacturing;; miscellaneous organic chemical processes; municipal waste combustors; open burning of scrap tires; residential heating using coal, or distillate oil, natural gas, wood/wood residue; stationary diesel engines; and wood preserving. Mobile sources accounted for about 6% (162-lbs) of the total.

State and regional WEI ranks Arsenic and compounds ranked 9th in the statewide cancer potency weighted EI, accounting for about 0.18% of the total. In terms of non-cancer health risks, the TWEI ranking of arsenic and compounds was inconsequential because exposure levels estimated in the 1996 NATA placed its HQ well under one: Exposures were not likely to result in significant non-cancer health risks.

NATA state and regional findings The statewide average lifetime excess cancer risk from arsenic inhalation at 1996 levels was 0.222-per-million. Although no counties had countywide

average excess arsenic-associated cancer risk exceeding one-in-a-million, risk estimates exceeded one-in-a-million in 11 census tracts across Clark, King and Pierce counties.

Monitoring The median and average concentrations of arsenic observed in Vancouver during 2001 were 0.001 and 0.002- $\mu\text{g}/\text{m}^3$ respectively. There were 16 of 56 samples with measurable levels. The average concentration of arsenic observed in a special study of rural western Washington during late June 1990 to early September 1990 was 0.000154- $\mu\text{g}/\text{m}^3$. The median and average concentrations of arsenic observed in Seattle during the early 2000s were 0.00092 and 0.0012- $\mu\text{g}/\text{m}^3$ respectively. The median and average concentrations of arsenic observed in the IMPROVE aerosol study, in populated areas across Washington, during the late 1990s to early 2000s mainly, were 0.00035 and 0.00046- $\mu\text{g}/\text{m}^3$, respectively; and in wilderness areas, mainly during this same time period, were 0.00010 and 0.00014- $\mu\text{g}/\text{m}^3$, respectively. The concentrations in populated areas are significant in terms of associated cancer risk: lifetime population inhalation exposure at these levels leads to arsenic-associated cancer risk of 3 to 5 per million.

Recommendations Both the 1996 NATA and toxic air pollutants monitoring indicate that arsenic and arsenic compound emissions may result in excessive cancer risks in some areas of Washington. To more clearly determine the extent of these risks, we need differential monitoring of various arsenic species as well as reporting of specific chemical forms of arsenic in the emissions inventory. Better determination of exposure to specific arsenic chemical forms will be required because of the differing carcinogenic potencies and non-cancer toxicities of various arsenic species.

1,3-Dichloropropene

Sources The NTI reported over 441-tons of 1,3-dichloropropene from area source emissions in Washington during 1996. Consumer products usage and miscellaneous organic chemical process emissions account for all known emissions.

WEI state and regional ranks The statewide cancer potency weighted EI rank of 1,3-dichloropropene was 17th, with 0.056% of the total. The low rank of 1,3-dichloropropene in the

RfC-weighted EI suggests it does not pose non-cancer health effect hazard by inhalation in Washington.

NATA state and regional findings 1,3-Dichloropropene comprised 0.073% of the total statewide cancer risk estimate in the 1996 NATA, with a median statewide excess cancer risk of 0.218 per million. Excess cancer risk was 1.06 per million in one King County census tract. Estimated exposure levels were slightly below the *de minimis* risk level in several other census tracts.

Monitoring 1,3-Dichloropropene was analyzed but not detected in the Vancouver Toxic air pollutants study of 2001. It has not been an analyte in any other monitoring studies in Washington.

Recommendations There is some evidence to suggest that 1,3-dichloropropene presents a level of cancer risk greater than *de minimis* in one area of King County, although uncertainty about its carcinogenicity to humans and about actual population exposure levels frustrate this risk ranking effort. More monitoring and more complete toxicity data will be necessary for establishing stronger conclusions about the potential health risk of this air pollutant.

Ethylene oxide

Sources All 14.9-tons of ethylene oxide emissions reported in the NTI during 1996 were from area sources, which are listed as hospital sterilizer disinfectants; miscellaneous organic chemical processes, such as in the production of detergents, ethylene glycol, and glycol ethers; and from WWTPs.

WEI state and regional ranks Ethylene oxide comprised 0.034% of the total cancer potency weighted EI weight statewide. The TWEI rank of ethylene oxide suggests it does not pose non-cancer inhalation health hazards in Washington.

NATA state and regional findings Ethylene oxide comprised 0.029% of the total statewide air pollution-associated cancer risk estimated in the 1996 NATA, with associated excess cancer risk

of 0.009 per million. Across Washington, excess cancer risk exceeded one-in-a-million (at 1.07 per million) in only in one census tract, located in Pierce County. Estimated exposure levels were slightly below the *de minimis* risk level in several other census tracts in Pierce and King Counties.

Monitoring Ethylene oxide has not been an analyte in air monitoring studies in Washington.

Recommendations Exposure estimates from NATA suggest that air emissions of ethylene oxide, which is carcinogenic to humans, present a level of cancer risk greater than *de minimis*, although the excessive exposure appears to be limited to one census tract in the state. Ambient air monitoring of this pollutant is needed to develop more definitive conclusions regarding its public health risk.

Selenium and selenium compounds

Sources Approximately 99% of reported selenium emissions in Washington are in the area source category listed as “miscellaneous manufacturing coating”. Most of these miscellaneous manufacturing coating emissions were reported in the PSCAA region (194.92-tons/year in King County; 65.75-tons/year in Snohomish County; 30.18-tons/year in Pierce County; and 2.22-tons/year in Kitsap County). All other reported quantities were less than 0.05-tons/year. Other area sources consist mainly of users (in boilers and heaters) of residual, distillate and waste oil, and coal. Major point and mobile sources also contribute to human-made emissions, in small part.

State and regional Weighted Emission Inventories The NTI does not specify what form(s) of “selenium and compounds” are counted. To avoid underestimating the potential cancer risk we assumed all selenium and compounds were selenium sulfide. With this assumption, “selenium and compounds” would rank fifth in the statewide cancer potency weighted EI, posing a statewide average of ~1% of the calculated toxic air pollution cancer potency-weighted emissions. In terms of non-cancer health risks, the statewide non-cancer hazard weighted-emission inventory rank of selenium and compounds is also 5th statewide.

NATA state and regional findings Selenium and compounds were not assessed in the 1996 NATA.

Monitoring Several monitoring studies in Washington have included selenium as an analyte. In urban and suburban areas the average concentration has always been less than 0.7-ng/m³, and in rural areas, the average concentration has always been less than 0.04-ng/m³. With the available monitoring data, if we assume all the selenium detected is the most toxic form, selenium sulfide, and the exposure remains constant for lifetime, the associated excess cancer risk is far less than one-in-a-million.

Recommendations The 5th place rank of selenium and compounds in the cancer potency-weighted EI follows from the assumption that all selenium containing emissions are selenium sulfide. If, however, a substantial portion of total emissions are not in fact selenium sulfide, the selenium and compounds would have a lower overall rank. The CPWEI rank for selenium and compounds suggest that atmospheric levels probably exceed the level that would carry an excess cancer risk greater than one-in-a-million. Selenium monitoring recently performed in Seattle contradicts this conclusion. If there are excessive exposure levels anywhere, they are likely to occur in densely populated places because more than 99% of emissions are from area sources. At this point, basic information is lacking: We do not have enough data to make a risk criteria-based decision about selenium with much confidence. To resolve this, future EI and monitoring efforts are needed that distinguish chemical forms of selenium.

Acrolein

Sources Most acrolein exposure in Washington results from “area and other” sources. The largest quantities reported in the EI are from structure fires and wildfires. Acrolein is found gasoline and diesel exhausts and other combustion processes, contributing significantly to the total emissions. Acrolein is used extensively as an aquatic herbicide, in some counties in eastern Washington, for control of waterborne weeds in irrigation canals. Lesser contributors include emissions from industries where acrolein is manufactured,¹⁰⁰ and from a variety of microbial and

¹⁰⁰ Acrolein is used as an intermediate for glycerin, methionine, glutaraldehyde, and other organic chemicals such as certain plastics.

vegetative processes.¹⁰¹

WEI state and regional ranks On a statewide basis, and in each region, acrolein accounts for most of the non-cancer toxicity-weighted EI (~86% statewide, 79% to 96% across the regions).

NATA state and regional findings According to EPA's 1996 and 1999 NATA reports, the background level of acrolein was elevated in most places in Washington. For example, the 1996 NATA indicates the potential adverse health effects resulting from exposure to acrolein: The average hazard quotient (HQ) across Washington's population was nearly 3.6. The HQ for the most exposed 5% of the urban area populations was approximately 7. HQs higher than 10 were also estimated to occur in a few census tracts in King, Stevens and Clark counties. In all, 93.7% of census tracts in Washington had HQs of one or higher. The only counties with estimated average exposures below the RfC were Adams, Ferry, Grant, Lincoln, San Juan, and Walla Walla.

Monitoring Ambient air concentrations of acrolein are technically difficult to measure. This has added to uncertainty in the accuracy of the acrolein emission factors used to quantify acrolein for the emissions inventory. Because of this and because we have no actual acrolein monitoring data in Washington, our confidence in the TWEI and the NATA results for acrolein are lower than for most of the other top ranked toxic air pollutants. These results are our best estimates based on very limited emissions data. We need to investigate what ambient acrolein concentrations are at representative sites, using better monitoring technology and refined models.

Recommendations Acrolein ranked first in both TWEI and NATA non-cancer screenings. The HQ calculation provided in the NATA is a decision point and although many toxic air pollutants were examined, only acrolein was found to be present at levels above its RfC. This indicates that there may be an exposure-associated hazard of irritation of the eyes, nose, throat, and respiratory tract, and possibly airway inflammation as a result of acrolein at current levels.¹⁰² In much of

¹⁰¹ Howard, P. 1990. *Handbook of Environmental Fate and Exposure for Organic Chemicals*. Volumes 1-4. Lewis Publishers. Chelsea, Michigan

¹⁰² Acrolein effects and fate information from "<http://www.epa.gov/iris/toxreviews/0364-tr.pdf>"

Washington the acrolein HQ was between 1 and 10 as reported in the 1996 NATA. Little can be changed about the largest sources of acrolein emissions (structure and wildfires); however, reducing other combustion emissions, including engine exhausts, would reduce health hazards proportionally.

Final Recommendations

Preventable air pollution has been a problem since humans began using fire as a tool. Important increases in air pollution also occurred with the development of metal working in prehistoric times. Major increases occurred during the industrial revolution and particularly with the rapid increase in motor vehicle usage beginning in the 1910s and continuing through the present.¹⁰³ The most serious of current toxic air pollution problems has grown as diesel engine usage has increased. At minimum, measures for reducing diesel emissions should be evaluated. Greater expense to reduce DPM exposure is in fact justified. In addition, measures for several other pollutants, most notably residential wood smoke, should be examined. The relative importance of toxic air pollution source categories to protect public health is estimated in figure 4-3.

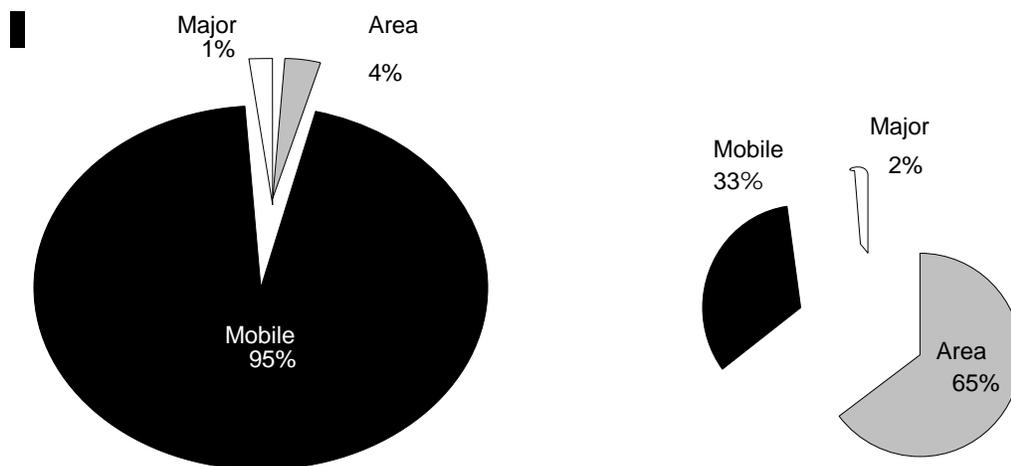


Figure 4-3. Statewide source category contributions to the TWEI. Toxic air pollutant-associated cancer risk (A); Non-cancer toxicity hazard (B) (not to scale)

The most beneficial toxic air pollutants control action for improving public health would be to reduce DPM emissions, which comprise most of the mobile source category risk. The AQP has

¹⁰³ Brimblecombe, P. 1999. Air Pollution and Health History, Ch.2. *Air pollution and Health*. Holgate, S., Samet, J., Koren, H. and Maynard, R. Eds. Academic Press, San Diego. pp 5-18

the authority to set standards and adopt them in rules, but does not have the authority to set motor vehicle emission standards. The authority for regulating diesel engine emission rests with USEPA, which required a phase-in of low sulfur diesel fuel for on-road uses beginning in 2006. There is also technology available to retrofit diesel engines so they release fewer emissions. Retrofitted diesel vehicles and those with new exhaust systems can burn the cleaner low sulfur diesel fuel. In addition, biodiesel may have lower sulfur and lower PAH emissions. Recent state legislation was enacted that will require all state agencies use 20 percent biodiesel blends in their diesel-powered vehicles by 2009.

The AQP and Washington's LAAs do not currently have the resources to conduct complete risk assessments of all of the toxic air pollutants considered in this ranking; however, we should make the effort to determine more precisely the risks from diesel PM, residential wood smoke and the toxic air pollutants that pose excess cancer risks greater than one-in-a-million by continuing and improving EI-based modeling and expanding monitoring to appropriate toxic air pollutants in Seattle and other communities.

Local air agencies should try to inventory emissions of as many as possible of the remaining chemical air emissions, not evaluated here, as possible. Even though such pollutants are neither criteria pollutants nor federal toxic air pollutants, some of them may pose significant public health threats.

Monitoring should be undertaken to better assess at least the top ranked air pollutants. Furthermore, research funds might more efficiently be allocated to reduce some uncertainties, primarily by designing future air toxic monitoring with emphasis on data acquisition for PMF studies, particularly for estimation of diesel PM and RWS exposures.

Future efforts to complete emissions inventories should be done to coincide with toxic air pollutants monitoring. This will allow EI-based toxic air pollutants modeling to be compared with actual toxic air pollutants monitoring results, thereby allowing improvements in the methods of both kinds of efforts. This is a better alternative to the current situation of non-overlapping EIs and toxic air pollutant monitoring periods. Simultaneous EIs and monitoring years would make the best use of information and would decrease the possible consequences of

estimation errors due to various uncertainties.

The AQP and the local air agencies should inform the public and recommend behavior to avoid health risks. Some of the main messages that might be given are as follows:

- Collectively, private citizens can request and support their cities and school districts to purchase cleaner buses and other vehicles. A combination of retrofitting existing diesel engines and burning cleaner diesel fuel could reduce DPM emissions by at least 50 percent.
- To minimize individual risks, effective solutions for private citizens are to support the Ecology AQP and LAAs in their efforts to reduce air pollution by reducing personal actions that generate air pollution and if possible by avoiding personal exposure by keeping away from toxic air pollutants emission sources. For example, individuals can purchase low-emission vehicles; drive less and burn less or not at all; refrain from topping-off when refueling vehicles; and replace uncertified wood stoves and fireplaces with cleaner choices such as natural gas, propane, pellet or USEPA-certified hearth products. In vehicles, individuals can reduce the amount of particulate matter in their vehicles by keeping their windows closed. The car's ventilation system typically removes a portion of the particles coming in from outside. Most cars have the ability to re-circulate the inside air, which will help keep the interior air particulate levels slightly lower.
- For sensitive individuals who must be outdoors during a temperature inversion that traps toxic air pollutants near the ground, wearing a properly fitting activated carbon-fine particulate filtration mask and/or reducing physical activity will lower exposure. Bear in mind that most population exposure occurs indoors because that is where most people spend most of their time, and because there are additional sources of toxic air pollutants indoors. Some sources, such as building materials, furnishings, appliances, like ozone generators, and household products, like "air fresheners", release pollutants more or less continuously. Other sources, related to activities carried out in the home, release pollutants intermittently. These sources include smoking, the use of unvented or

malfunctioning stoves, furnaces, or space heaters; the use of solvents in cleaning and hobbies; the use of paint strippers; and the use of cleaning products and pesticides. High pollutant concentrations can remain in the air for long periods after these activities.

Glossary

Activity Pattern Data:

In an inhalation exposure assessment, activity pattern data depict both the actual physical activity (including an associated inhalation exertion level), the physical location, and the time of the day the activity takes place (e.g., sleeping at home at midnight, jogging in the park at 8 a.m., or driving in a car at 6 p.m.). The HAPEM4 model extracts activity pattern data from the EPA's Comprehensive Human Activity Database.

Air toxics:

Also known as toxic air pollutants or hazardous air pollutants are those pollutants known to or suspected of causing cancer or other serious health problems. Health concerns may be associated with both short and long term exposures to these pollutants. Many are known to have respiratory, neurological, immune or reproductive effects, particularly for more susceptible sensitive populations such as children.

Ambient:

Surrounding, as in the surrounding environment. In this assessment, ambient air refers to the air surrounding a person through which pollutants can be carried.

Area Sources:

Smaller stationary sources: Some smaller facilities submit emissions inventory reports but the majority of area sources are estimated from countywide population risk estimates and assumptions about what the population is doing. In the NATA, USEPA also included other types of area sources such as forest fires and prescribed burning.

Assessment System for Population Exposure Nationwide model (ASPEN):

A computer simulation model used to estimate toxic air pollutant concentrations. The ASPEN model takes into account important determinants of pollutant concentrations, such as: rate of release, location of release, the height from which the pollutants are released, wind speeds and directions from the meteorological stations nearest to the release, breakdown of the pollutants in the atmosphere after being released (i.e., reactive decay), settling of pollutants out of the atmosphere (i.e., deposition), and transformation of one pollutant into another (i.e., secondary formation). The model estimates toxic air pollutant concentrations for every census tract in the continental United States, Puerto Rico and the Virgin Islands. However, the output for the model is presented at the county level. For more detailed information, see ASPEN Model.

Background:

USEPA uses the term to mean air toxics concentrations resulting from natural sources, man-made emissions persisting in the environment from past emissions, and long-range transport from distant sources. To accurately estimate outdoor concentrations, it is necessary to account for the background concentrations by adding them to the modeled concentrations. In the NATA, background concentrations are based on values identified in the Cumulative Exposure Project (a USEPA study that estimated 1990 ambient concentrations of air toxics). In that study, USEPA used the background concentration values reported in technical literature available for 13 toxic air pollutants. For the other toxic air pollutants in the CEP, USEPA assumed a concentration of zero. Further, for diesel PM, instead of using monitored air quality data to estimate background concentrations, a modeling was used.

BCAA: (see *Local Air Agency*)

Bioavailability:

The proportion of a chemical in the environment that can be taken up by an organism.

Biomagnification:

A phenomenon that has been observed of some lipid soluble and persistent chemicals. It is the increase of chemical concentrations at successively higher trophic levels in food chains. A chemical's biomagnification factor (BMF) = $C_{\text{predator}} \div C_{\text{prey}}$

Cancer:

Cancer refers to any of more than 250 different metastatic disorders (uncontrolled increases of cell division, which may occur in almost any part of the body followed by migration of uncontrolled cells to distant sites in the body, forming additional tumors).

Cancer Risk:

A risk level of some number *n* in a million implies a likelihood that up to *n* people, out of one million equally exposed people would contract cancer if exposed continuously (24 hours per day) to the specific concentration over 70 years (an assumed lifetime). This would be in addition to those cancer cases that would normally occur in an unexposed population of one million people. NATA estimates lifetime cancer risks, which should not be confused with annual cancer risk estimates.

Carcinogen:

A chemical or physical agent capable of causing cancer (see glossary weight-of-evidence entry).

Cardiopulmonary:

Having to do with both the heart and lungs. Cardiopulmonary diseases are cardiovascular diseases that also affect the lungs.

Census tracts:

Land areas defined by the U.S. Bureau of the Census that vary in size but typically contain about 4,000 residents each. Census tracts are usually smaller than 2 square miles in size in cities, but much larger in rural areas.

Consolidated Human Activity Database:

Developed by USEPA and based on actual daily diary summaries for more than 22,000 people nationwide, which are coded by age, gender, and race.

Critical Effect:

The first adverse effect, or its known precursor, that occurs to the most sensitive species as the exposure rate to a toxic substance increases.

CRO: (see *Local Air Agency*)

Diesel Particulate Matter:

The mixture of particles that is a component of diesel engine exhaust. USEPA lists diesel exhaust as a mobile source air toxic due to the cancer and non-cancer health effects associated with exposure to it. USEPA states that exposure to whole diesel exhaust is best described, as many researchers have done over the years, by diesel particulate concentrations.

Dispersion model:

A set of mathematical equations that use emissions and meteorological information to simulate the behavior and movement of air pollutants in the atmosphere. The results of a dispersion model are estimated outdoor concentrations of individual air pollutants at specified locations. ASPEN is a dispersion model.

Emission density:

Represents tons per year within a given area on a per square mile basis. In this assessment, total county emissions are divided by the total square mileage of the county. Emission density may be used to show emissions information graphically because it provides a more consistent basis for comparison than emissions totals alone.

Emission inventory:

An estimate of the quantity of an air pollutant released from a source during a one year period. Estimates may be developed using methods ranging from direct reporting by individual facilities in some cases, to generalizations about quantities emitted by certain activities along with estimates of the level of those activities in a specified geographic area.

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ERO: (see *Local Air Agency*)

Exposure assessment:

Identifying the ways in which chemicals may reach individuals (e.g., by breathing); estimating how much of a chemical an individual is likely to be exposed to; and estimating the number of individuals likely to be exposed.

Hazard:

In this report “hazardous chemicals” are defined as any which are a health hazard. A health hazard is any chemical for which there is good evidence that acute or chronic health effects occurs in exposed persons. Hazardous chemicals include carcinogens and other toxic chemicals such as reproductive toxicants; irritants; corrosives; sensitizers; hepatotoxins; nephrotoxicants; neurotoxicants; chemicals that act on the hematopoietic system; and chemicals that damage the lungs, skin, eyes, or mucous membranes.

Hazardous Air Pollutant:

Hazardous air pollutants (HAPs) are air toxics that pose a significant threat to human health and the environment. The pollutants were first regulated by the 1970 Clean Air Act Amendments that instructed USEPA to create a list of HAPs and then issue national emissions standards for those pollutants. By 1990, only eight substances were identified as HAPs, and only seven national emission standards for hazardous air pollutants (NESHAP) were promulgated. Under the new Air Toxics program, found in Title III of the Clean Air Act Amendments of 1990, Congress established an initial list of 189 substances to be regulated as HAPs. Rather than regulating individual pollutants by establishing health-based standards, the new Air Toxics program granted USEPA the authority to regulate specific industrial major source categories with NESHAP based on maximum achievable control technology (MACT) for each source category. Sources not large enough to fall under the major source requirements may still be regulated under the "area" source requirements that will affect smaller facilities. NESHAP and appropriate MACT standards are enforced through the Clean Air Act Title V Operating Permit Program.

Hazard Index:

The Hazard Index (HI) is the sum of hazard quotients of two or more chemicals that affect the same organ or organ system. Because different pollutants may cause similar adverse health effects, it is often appropriate to combine hazard quotients associated with different substances. Ideally, hazard quotients should be combined for pollutants that cause adverse effects by the

same toxic mechanism. However, because detailed information on toxic mechanisms was not available for most of the substances in NATA, USEPA aggregated only the effects of different respiratory irritants. The HI for respiratory irritation is only an approximation of the aggregate effect on the respiratory system (i.e., lungs and air passages) because it is possible that some of the substances cause irritation by different (i.e., non-additive) mechanisms. As with the hazard quotient, aggregate exposures below a HI of 1.0 will likely not result in adverse non-cancer health effects over a lifetime of exposure. However, an HI greater than 1.0 does not necessarily suggest a likelihood of adverse effects. Furthermore, the HI cannot be translated to a probability that adverse effects will occur, and is not likely to be directly proportional to risk. Exposure-response relationships have been established for non-carcinogenic toxic air pollutants, therefore the HI is at least indirectly proportional to risk. A respiratory HI greater than 1.0 can be best described as indicating that a potential may exist for adverse irritation to the respiratory system.

Hazardous Air Pollutant Exposure Model (HAPEM):

A computer model used in the NATA that has been designed to estimate inhalation exposure for specified population groups to air toxics. Through a series of calculation routines, the model makes use of census data, human activity patterns, ambient air quality levels, climate data, and indoor/outdoor concentration relationships to estimate an expected range of inhalation exposure concentrations for groups of individuals.

Hazard Quotient:

The Hazard Quotient (HQ) ratio of the potential exposure to the substance and the level at which no adverse effects are expected. If the HQ is calculated to be less than 1, then no adverse health effects are expected as a result of exposure. If the HQ is greater than 1, then adverse health effects are possible. The HQ cannot be translated to a probability that adverse health effects will occur, and is unlikely to be directly proportional to risk. Exposure-response relationships have been established for non-carcinogenic toxic air pollutants, therefore the HQ is at least indirectly proportional to risk. It is important to understand that an HQ exceeding one does not necessarily mean that adverse effects will occur. When an HQ is one or greater, it is usually treated as a decision factor.

Hazard-Weighted Emission Inventory:

In this report, the cancer potency weighted emission inventory and the toxicity weighted emission inventory are together termed the hazard-weighted emission inventory.

Integrated Risk Information System (IRIS):

An electronic data base containing information on human health effects that may result from exposure to various chemicals in the environment. IRIS was prepared and is maintained by the USEPA as a collection of data focusing on hazard identification and dose-response assessment to support human health risk assessment, decision-making and regulatory activities.

Local Air Agency (LAA):

There are three WDOE regional offices and seven local air agencies in Washington, each encompassing one or more counties of jurisdiction. The LAAs and their counties are:

Benton Clean Air Authority (BCAA)
Benton

Ecology Central Regional Office (CRO)
Chelan
Douglas
Kittitas
Klickitat
Okanogan

Ecology Eastern Regional Office (ERO)
Adams
Asotin
Columbia
Ferry
Franklin
Garfield
Grant
Lincoln
Pend Oreille
Stevens
Walla Walla
Whitman

Ecology Northwestern Regional Office (NWRO)
San Juan

Northwest Clean Air Agency (NWCAA)
Island

Skagit
Whatcom

Olympic Region Clean Air Agency (ORCAA)

Clallam
Grays Harbor
Jefferson
Mason
Pacific
Thurston

Puget Sound Clean Air Agency (PSCAA)

King
Kitsap
Pierce
Snohomish

Southwest Clean Air Agency (SWCAA)

Clark
Cowlitz
Lewis
Skamania
Wahkiakum

Spokane Regional Clean Air Agency (SRCAA)

Spokane

Yakima Regional Clean Air Agency (YRCAA)

Yakima

Major sources:

Defined by the Federal Clean Air Act as those stationary facilities that emit or have the potential to emit 10 tons of any one toxic air pollutant or 25 tons of more than one toxic air pollutant per year.

Median:

The middle value of a set of values (i.e., half the numbers are less than or equal to the median value). A median is the 50th percentile of a data set.

Microenvironment:

A small space in which human contact with a pollutant takes place which can be treated as a well-characterized, relatively homogenous location with respect to pollutant concentrations for a specified time period. In the NATA, HAPEM4 considers cohort activities in 37 microenvironment locations that include: (1) indoor locations (e.g., residence, office, store, school, restaurant, church, manufacturing facility, auditorium, health care facility, service station, other public building, garage); (2) outdoor locations (e.g., parking lot/garage, near road, motorcycle, service station, construction site, residential grounds, school, sports arena, park/golf course); and (3) in-vehicle locations (e.g., car, bus, truck, other, train/subway, airplane).

Minimal Risk Level:

A Minimal Risk Level (MRL) is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. MRLs are based on non-cancer health effects only and not on a consideration of cancer effects. MRLs are accompanied by toxicological profiles, which include an examination, summary, and interpretation of available toxicological information and epidemiologic evaluations. MRLs are derived when Agency for Toxic Substances and Disease Registry (ATSDR) determines that sufficient reliable data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure to the substance. In a practice, MRLs are similar to USEPA's Reference Concentrations (RfCs) and Reference Doses (RfDs) for deriving substance-specific health guidance levels for non-cancer endpoints. The ATSDR and USEPA jointly develop toxicological profiles for the hazardous substances most commonly found at facilities listed on the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) National Priorities List. ATSDR uses the no-observed-adverse-effect-level/uncertainty factor (NOAEL/UF) approach to derive MRLs. They are set below levels that, based on current information, might cause adverse health effects in the people most sensitive to such substance-induced effects (e.g., infants, elderly, and nutritionally or immunologically compromised). MRLs are derived for acute (1-14 days), intermediate (>14-364 days), and chronic (365 days and longer) exposure durations, and for the oral and inhalation routes of exposure. Most MRLs contain some degree of uncertainty because of the lack of precise toxicological information on the people who might be most sensitive to hazardous substances. ATSDR uses a conservative (i.e., protective) approach to address these uncertainties consistent with the public health principle of prevention. Although human data are

preferred, some MRLs have been based on animal studies because relevant human studies are lacking. In the absence of evidence to the contrary, ATSDR assumes that humans are more sensitive than animals to the effects of hazardous substances that certain persons may be particularly sensitive. Thus the resulting MRL may be as much as a hundredfold below levels shown to be nontoxic in laboratory animals. When adequate information is available, physiologically based pharmacokinetic modeling and benchmark dose modeling have also been used as an adjunct to the NOAEL/UF approach in deriving MRLs. Like RfCs, exposure to a level above the MRL does not mean that adverse health effects will occur.

Non-Carcinogenic Effects:

Excessive exposure to non-carcinogenic air pollutant chemicals may have the potential for causing pulmonary, liver, and kidney damage, nervous system changes, birth defects, immune system dysfunction, and other effects, depending on the chemicals involved and other factors.

Non-Road Mobile Sources:

Mobile sources not typically used on roads and highways (e.g., airplanes, trains, lawn mowers, construction vehicles, farm machinery).

NWCAA: (see *Local Air Agency*)

NWRO: (see *Local Air Agency*)

On-Road Mobile Sources:

Vehicles used on roads and highways (e.g., cars, trucks, buses, motorcycles).

Oral Exposure:

Entry of pollutants into the digestive tract by eating and drinking.

ORCAA: (see *Local Air Agency*)

Polycyclic Aromatic Hydrocarbons:

The 7-PAH group includes seven chemicals: Benz[a]anthracene, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Benzo[a]pyrene, Chrysene, Dibenz[a,h]anthracene, and Indeno[1,2,3-cd]pyrene. The 7-PAH are a subset of “16-PAH” (16-PAH is referred to as Polycyclic Organic matter or "POM" in the presentation of results for the NATA). Each of the 7-PAH are probable human carcinogens.

Polycyclic Organic Matter:

The Polycyclic Organic Matter (POM) group is a broad class of chemicals that includes the polycyclic aromatic hydrocarbon compounds (PAHs). POM chemicals are formed primarily from combustion, and are present in the atmosphere in particulate form. Sources of air emissions include, vehicle exhausts, forest and wildfires, asphalt roads, coal, coal tar, coke ovens, agricultural burning, residential wood burning, hazardous waste sites and other sources.

PSCAA: (see *Local Air Agency*)

Reference Concentration (RfC):

The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human populations (including sensitive subgroups which include infants, children, people with asthma and the elderly) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from various types of human or animal data, with uncertainty factors applied to reflect limitations of the data used. It is for continuous lifetime exposure.

Reference Exposure Level (REL):

The California Office of Environmental Health Hazard Assessment defines REL as a concentration level at (or below) which exposed people will have no anticipated health effects. This is similar to the USEPA’s IRIS RfCs and ATSDR MRLs.

Risk:

The probability that damage to life, health, and/or the environment will occur as a result of a hazard (such as exposure to a toxic chemical). Some risks can be measured or estimated in numerical terms (e.g., one chance in a hundred). “Risk” in the context of human health is the

probability of injury, disease, or death from exposure to a chemical agent or a mixture of chemicals. In quantitative terms, risk is expressed in values ranging from zero (representing the certainty that harm will not occur) to one (representing the certainty that harm will occur).” For this ranking, the concentration that might lead to a 1/100,000 lifetime excess cancer risk level (10^{-5} RL) for an individual who is exposed for over their lifetime at this concentration, was used for potential carcinogens. Inhalation RfCs were used for non-carcinogens. These are “an estimate (with uncertainty spanning perhaps an order of magnitude) of daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.” It is for continuous lifetime exposure.

Rural:

A county was that does not contain a metropolitan statistical area with a population greater than 250,000, and where the U.S. Census Bureau did not designate more than 50 percent of the population as "urban."

SRCAA: (see *Local Air Agency*)

SWCAA: (see *Local Air Agency*)

Toxic Air Pollutants:

Toxic Air Pollutants (TAPs) are chemicals regulated under the Washington State Administrative Code (WAC) 173-460 Controls for New Sources of Toxic Air Pollutants. A list of these chemicals is available at <http://www.ecy.wa.gov/pubs/wac173460.pdf>

Unit Risk Estimate (URE):

The Unit Risk Estimate is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to a chemical at a concentration of $1 \mu\text{g}/\text{m}^3$ in air. The interpretation of the URE is as follows: if the $\text{URE} = 1.5 \times 10^{-6}$ per $\mu\text{g}/\text{m}^3$, 1.5 excess tumors are expected to develop per 1,000,000 people exposed daily for a lifetime to $1 \mu\text{g}$ of the chemical per cubic meter of air they breathe. UREs are considered upper bound estimates, meaning they represent a plausible upper limit to the true value. (Note that this is usually not a true statistical confidence limit.) The true risk is likely to be less, but could be greater. In this report, risk values are considered to be

potential population lifetime cancer risk estimates. The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) indicates both individual and population risks as options for presenting numeric risk estimates:

"Risks may be characterized either in terms of the excess individual lifetime risks, the excess number of cancers produced per year in the exposed population or both."

USEPA also discusses the issue in the 1992 *Guidelines for Exposure Assessment* (See pages 47-48, U.S. Environmental Protection Agency. *Guidelines for Exposure Assessment*. FRL-4129-5), and provides an equation for calculating potential population risks. Both documents emphasize that the risk estimates are predictions, and should not be interpreted as actual cases. It is important to be aware that the uncertainties in the risk models apply to the model as a whole, and pertain equally to individual and population risk estimates. The NAS document *Science and Judgment in Risk Assessment* (<http://books.nap.edu/books/030904894X/html/index.html>) makes no statements that risk estimates should or should not be interpreted over an exposed population.

Upper-Bound Lifetime Cancer Risk:

A plausible upper limit to the true probability that an individual will contract cancer over a 70 year lifetime as a result of a given hazard (such as exposure to a toxic chemical). This risk can be measured or estimated in numerical terms (e.g., one chance in a hundred).

Upper Confidence Limit:

The Upper Confidence Limit (UCL):

The upper bound of a confidence interval around any calculated statistic, such as an average. For example, the 95 percent confidence interval for an average is the range of values that will contain the true average (i.e., the average of the full statistical population of all possible data) 95 percent of the time. In other words, with 95 percent certainty, the "true" average will exceed the UCL only 2.5 percent of the time. USEPA has based most Unit Risk Estimates on the Upper Confidence Limit of response data or of fitted curves, to avoid underestimating the true Unit Risk Estimate in the face of uncertainty.

Urban:

A county is considered "urban" in NATA if it either includes a metropolitan statistical area with a population greater than 250,000 or the U.S. Census Bureau designates more than 50 percent of the population as "urban."

Weight-of-Evidence for Carcinogenicity:

USEPA's designation of confidence in the carcinogenic potency of certain chemicals. WOE is a system for characterizing the extent to which the available data support the hypothesis that an agent causes cancer in humans. Under USEPA's 1986 risk assessment guidelines, the weight-of-evidence is described by categories "A through E," Group A for known human carcinogens through Group E for agents with evidence of non-carcinogenicity. Each pollutant may be placed into one of the following five categories:

Group A (Known human carcinogen): Compounds for which human data are sufficient to demonstrate a cause and effect relationship between exposure and cancer incidence (rate of occurrence) in humans. In the national-scale assessment, the 7 air toxics classified as human carcinogens are: arsenic compounds, benzene, 1,3-butadiene, chromium compounds, coke oven emissions, nickel compounds, and vinyl chloride.

Group B (probable human carcinogen):

Group B1 Compounds for which limited human data suggest a cause and effect relationship between exposure and cancer incidence (rate of occurrence) in humans. In the national-scale assessment, the 5 air toxics classified as probable (B1) human carcinogens are: acrylonitrile, beryllium compounds, cadmium compounds, ethylene oxide, and formaldehyde.

Group B2 Compounds for which animal data are sufficient to demonstrate a cause-and-effect relationship between exposure and cancer incidence (rate of occurrence) in animals, and human data are inadequate or absent. In the national-scale assessment, the 15 air toxics classified as probable (B2) human carcinogens are: acetaldehyde, carbon tetrachloride, chloroform, 1,3-dichloropropene, ethylene dibromide, ethylene dichloride, hexachlorobenzene (HCB), hydrazine, lead compounds, methylene chloride, PCBs,

polycyclic organic matter (POM), perchloroethylene, propylene dichloride, trichloroethylene.

Group C (Possible human carcinogen): Compounds for which animal data are suggestive to demonstrate a cause-and-effect relationship between exposure and cancer incidence (rate of occurrence) in animals. In the national-scale assessment, the 4 air toxics classified as possible human carcinogens are: acrolein, mercury compounds, quinoline and 1,1,2,2-tetrachloroethane. Because unit risk estimates have not been developed for acrolein and mercury compounds, EPA has not estimated cancer risk for these pollutants.

Group D (Not classifiable as to human carcinogenicity): Compounds for which human and animal data are inadequate to either suggest or refute a cause-and-effect relationship for human carcinogenicity. In the national-scale assessment, only manganese compounds were considered to be not classifiable as to human carcinogenicity.

Group E (Evidence of non-carcinogenicity): Compounds for which animal data are sufficient to demonstrate the absence of a cause-and-effect relationship between exposure and cancer incidence (rate of occurrence) in animals. In the national-scale assessment, no air toxics were classified as having evidence of non-carcinogenicity.

YRCAA: (see *Local Air Agency*)

Appendix

Names of the 188 HAPs, DPM & other non-HAPs included in the emissions inventory	CAS	Health Risk-Based Concentration weighting factor derivation	Carcinogenicity RBC	Non-cancer health effect RBC
Acetaldehyde	75070	USEPA has designated acetaldehyde a B2: probable human carcinogen, based on increased incidence of nasal tumors in male and female rats and laryngeal tumors in male and female hamsters after inhalation. The IRIS inhalation E-5 cancer risk level concentration is listed as <u>0.005-mg/m³</u> . The IRIS RfC is <u>0.009-mg/m³</u> .	0.005	0.009
Acetamide	60355	Toxicity not assessed by USEPA. The OEHHA URE is <u>2E-5/μg/m³</u> , which is equivalent to a 1E-5 cancer risk level concentration of <u>5E⁻⁴-mg/m³</u> . No RfC-like criterion is available.	5E ⁻⁴	
Acetonitrile	75058	The USEPA has designated acetonitrile as a class D carcinogen: Not classifiable as to human carcinogenicity due to the absence of human evidence and equivocal animal evidence. No quantitative cancer risk assessments are available from the other authorities referenced in this report either. The IRIS RfC is <u>0.06-mg/m³</u>		0.06
Acetophenone	98862	The USEPA has designated acetophenone as a class D carcinogen: not classifiable as to human carcinogenicity due to the absence of human and animal evidence. No quantitative cancer risk assessments are available from other authorities either. The IRIS RfC is <u>2E⁻⁵-mg/m³</u>		2E ⁻⁵
2-Acetylaminofluorene	53963	No toxicity benchmarks are available for 2-acetylaminofluorene from any of the authorities referenced in this report.		
Acrolein	107028	USEPA has designated acrolein a class C: Possible human carcinogen based on increased incidence of adrenal cortical adenomas to female rats and carcinogenic potential of an acrolein metabolite. Acrolein is mutagenic in bacteria and is structurally related to probable or known human carcinogens. No quantitative cancer risk assessment is available from any of the authorities referenced in this report. The IRIS RfC is <u>2E⁻⁵-mg/m³</u> . It is based on critical effects of pulmonary inflammation and histopathology		2E ⁻⁵
Acrylamide	79061	USEPA has designated acrylamide a class B2: Probable human carcinogen based on inadequate human data and sufficient evidence of carcinogenicity in animals; significantly increased incidences of benign and/or malignant tumors at multiple sites in both sexes of rats, and carcinogenic effects in a series of one-year limited bioassays in mice by several routes of exposures. Also, positive genotoxicity data, adduct formation activity, and structure-activity relationships to vinyl carbamate and acrylonitrile. Its IRIS cancer URE is equivalent to <u>7.7E⁻⁶-mg/m³</u> at the 1E ⁻⁵ excess risk level exposure. No RfC is provided however, the OEHHA lists a chronic REL of <u>7E⁻⁴-mg/m³</u>	7.7E ⁻⁶	7E ⁻⁴
Acrylic Acid	79107	No quantitative cancer risk assessment is available from the authorities referenced in this report. The IRIS RfC is <u>1-mg/m³</u>		1

Acrylonitrile	107131	USEPA has designated acrylonitrile a class B1: Probable human carcinogen, based on observed statistically significant increase in incidence of lung cancer in exposed workers and tumors, generally astrocytomas in the brain, in studies in two rat strains exposed by drinking water, gavage, and inhalation routes. The inhalation excess cancer risk level concentration listed in IRIS is <u>1E-4-mg/m³</u> , and the RfC listed is <u>0.002-mg/m³</u>	1E ⁻⁴	0.002
Allyl Chloride	107051	USEPA has designated allyl chloride a class C: Possible human carcinogen, based on a low (but biologically important) incidence of forestomach tumors in female mice and positive results in a variety of genetic toxicity tests. Allyl chloride is an alkylating agent and structurally related to probable human carcinogens. USEPA has not established a quantitative cancer potency estimate; However, the OEHHA cancer URE is equivalent to <u>1.7E⁻³-mg/m³</u> at the 1E ⁻⁵ excess cancer risk level exposure. The RfC listed in IRIS is <u>0.001-mg/m³</u> .	1.7E ⁻³	0.001
4-Aminobiphenyl	92671	No toxicity benchmarks are available for 4-aminobiphenyl from any of the authorities referenced in this report.		
Aniline	62533	USEPA has designated aniline a B2: Probable human carcinogen, based on induction of tumors of the spleen and the body cavity in two strains of rat, and some supporting genetic toxicological evidence. USEPA did not provide a quantitative cancer potency estimate; However, the OEHHA has: the URE is equivalent to <u>0.00167-mg/m³</u> at the E-5 excess cancer risk level concentration. USEPA lists an RfC of <u>0.001-mg/m³</u> in IRIS.	0.00167	0.001
O-Anisidine	90040	USEPA has not completed an evaluation of o-anisidine; However the OEHHA URE is equivalent to <u>2.5E⁻⁴-mg/m³</u> at the E ⁻⁵ excess cancer risk level concentration. The ACGIH TLV-TWA for workplace exposures divided by a range of safety factors to allow for continuous exposure to the general population ranges from 6.0E ⁻⁷ to 0.06 with an average of <u>2.4E⁻⁵-mg/m³</u> as the RfC-like benchmark.	2.5E ⁻⁴	2.4E ⁻⁵
Asbestos	1332214	The USEPA has classified asbestos as a class A: Known human carcinogen. The IRIS inhalation at the 1E ⁻⁵ excess cancer risk level is <u>4E⁻⁵ fibers/mL</u> of air. No RfC has been published. The state-wide emission of asbestos reported in the emission inventory is 1-lb. from area sources. No emissions are reported from other source categories. This information is suspect because it is likely that mobile sources (by vehicular brake pad wear) add to total emissions significantly. Another problem is that converting lbs. into fibers/mL is not feasible without monitoring, or modeling based on information known about the sources to estimate air concentrations. Asbestos is associated with mesothelioma - an otherwise rare form of lung cancer - in occupationally exposed people. It also occurs in with much lower frequency in populations with no such obvious exposure. This is consistent with the theory that ambient concentrations of asbestos are sufficient to induce mesothelioma in a few people; However, given existing information, we cannot calculate a reliable toxicity-weighted asbestos emission inventory.		
Benzene (Including Benzene From Gasoline)	71432	USEPA has designated benzene a class A: Known human carcinogen based on convincing human evidence as well as supporting evidence from animal studies. The excess cancer risk level inhalation concentration listed in IRIS is a range from 0.0013 to 0.0045-mg/m ³ . The more conservative limit (<u>1.3E⁻³-mg/m³</u>) was used for toxicity-weighting the emission inventory in this report, as well as in the NATA.	1.3E ⁻³	0.03

		The USEPA listed RfC of 0.03-mg/m^3 was used in this report. The OEHHA chronic REL is 0.06-mg/m^3 .		
Benzidine	92875	No toxicity benchmarks are available for benzidine from any of the authorities referenced in this report.		
Benzotrichloride	98077	USEPA has designated benzotrichloride as class B2: A probable human carcinogen, on the basis of increased tumors of the lung, skin, and lymphoid tissue observed in mice exposed by inhalation. The IRIS drinking water URE ($3.6\text{E}^{-4}/\mu\text{g/L}$) is equivalent to $2.8\text{E}^{-2}\text{-}\mu\text{g/L}$ at the 1E^{-5} excess cancer risk level. In an average adult, drinking 2L/day, the 1E^{-5} excess cancer risk level is equivalent to $72\text{-}\mu\text{g/day}$ or $72\text{-}\mu\text{g}/20\text{-m}^3$ inhaled air each day, which is equivalent to $3.6\text{E}^{-3}\text{-mg/m}^3$. Allowing for inter-day variability in average exposure concentrations with a factor of 0.2 yields $7.2\text{E}^{-4}\text{-mg/m}^3$. No RfC or similar criterion is available from any of the authorities referenced in this report.	7.2E^{-4}	
Benzyl Chloride	100447	USEPA has designated benzyl chloride a B2: Probable human carcinogen, based on inadequate human data and sufficient evidence of carcinogenicity in animals; namely significantly increased incidences of benign and malignant tumors at multiple sites in both sexes of mice, and a significant increase in thyroid tumors in female rats. There is also evidence of mutagenicity in a variety of test systems. USEPA has not established a quantitative cancer potency estimate; However, the OEHHA URE is equivalent to $2\text{E}^{-4}\text{-mg/m}^3$ at the 1E^{-5} excess cancer risk level concentration. USEPA has not evaluated the non-cancer risk of benzyl chloride; However, the OEHHA lists a chronic REL of 0.012-mg/m^3 .	2E^{-4}	0.012
Biphenyl	92524	The USEPA has designated 1,1-biiphenyl as a class D chemical: Not classifiable as to human carcinogenicity due to the absence of human and animal evidence. No other authorities referenced in this report have published RfC-like criteria for biphenyl either. For this report, we applied a safety factor range to allow for continuous exposure to the general population, to the ACGIH TLV-TWA for workplace exposures. The resulting range, used for toxicity-weighting the emission inventory, was from 1.5E^{-6} to 0.16 with an average of $5.9\text{E}^{-5}\text{ mg/m}^3$. The USEPA IRIS oral chronic RfD ($5\text{E}^{-2}\text{-mg/kg/day}$) was not used for weighting the inventory but was estimated for a 70-kg adult, breathing $20\text{-m}^3/\text{d}$ with an applied factor of 0.2 to account for variations in the average daily concentration. This yielded 0.035-mg/m^3 as an RfC estimate.		5.9E^{-5}
Bis (2-Ethylhexyl) Phthalate (DEHP)	117817	USEPA has designated bis (2-ethylhexyl) phthalate (DEHP) a B2: Probable human carcinogen, based on significant oral dose-related increases in liver tumor responses in rats and mice of both sexes. USEPA has not adopted a quantitative inhalation URE; However, the OEHHA URE is equivalent to 0.004-mg/m^3 at the 1E^{-5} cancer risk level concentration. An RfC for DEHP is not available currently; However, the OEHHA chronic REL is 0.07-mg/m^3 .	0.004	0.07
Bis (Chloromethyl) Ether	542881	USEPA has designated bis (chloromethyl) ether (BCME) a class A: Known human carcinogen, based on statistically significant increases in lung tumors (oat cell carcinomas) observed in six studies of exposed workers and bioassay data from rats and mice. The IRIS inhalation 1E^{-5} cancer risk level concentration is $1.6\text{E}^{-7}\text{-mg/m}^3$. An RfC for BCME is not available currently; However, the ATSDR intermediate duration inhalation MRL was modified for long-term exposure by applying a factor of 0.2. The product is $7\text{E}^{-4}\text{-mg/m}^3$, which was used as an RfC-like	1.6E^{-7}	7E^{-4}

value for this study.

Bromoform	75252	USEPA has designated bromoform as a B2: Probable human carcinogen, $9E^{-3}$ based on inadequate human data and sufficient evidence of carcinogenicity in animals, namely an increased incidence of tumors after oral administration of bromoform in rats and intraperitoneal administration in mice. Bromoform is genotoxic in several assay systems, and is structurally related to carcinogenic trihalomethanes. The IRIS $1E^{-5}$ cancer risk level inhalation concentration is $9E^{-3}\text{-mg/m}^3$. USEPA has not derived an RfC but has issued an RfD of $2E^{-2}\text{-mg/kg/day}$, which is equivalent to $0.07\text{-mg/m}^3\text{/d}$ for an average adult. Accounting for inter-day variation in daily exposure levels by using a factor of 0.2 yields 0.014-mg/m^3 as an RfC estimate.		0.014
1,3-Butadiene	106990	USEPA designates 1,3-Butadiene a class A: known human carcinogen, based on sufficient evidence from epidemiological studies of the majority of U.S. workers occupationally exposed to either the monomer or polymer by inhalation, showing increased lymphohematopoietic cancers and a dose-response relationship for leukemias in polymer workers; sufficient evidence in laboratory animal studies showing tumors at multiple sites in mice and rats by inhalation; and numerous studies consistently demonstrating that 1,3-butadiene is metabolized into genotoxic metabolites by experimental animals and humans. The IRIS inhalation $1E^{-5}$ cancer risk level concentration is $3E^{-4}\text{-mg/m}^3$. The RfC is $2E^{-3}\text{-mg/m}^3$, which was used for toxicity-weighting the emission inventory, as opposed to the OEHHA chronic REL, which is 0.02 mg/m^3 .	$3E^{-4}$	$2E^{-3}$
Calcium Cyanamide	156627	No toxicity benchmarks are available for calcium cyanamide from any of the authorities referenced in this report.		
Captan	133062	USEPA has not completed an evaluation of captan; However, the OEHHA URE is equivalent to 0.015-mg/m^3 at the $1E^{-5}$ risk level exposure concentration. No RfC-like criteria were available except the ACGIH TLV-TWA for workplace exposures, which was divided by a factor range to account for continuous exposure to the general population. The resulting range was from $6E^{-6}$ to 0.625 with an average of $2E^{-4}\text{-mg/m}^3$, which was used for toxicity-weighting the emission inventory in this report. The IRIS RfD for captan is $1.3E^{-1}\text{-mg/kg/day}$.	0.015	$2E^{-4}$
Carbaryl	63252	USEPA has not completed a cancer evaluation of carbaryl. None of the other authorities referenced in this report have published cancer potency estimates for carbaryl either. No RfC-like criteria are provided by USEPA or other referenced authorities; However, the ACGIH TLV-TWA for workplace exposures divided by a range of factors to allow for continuous exposure to the general population ranges from $6E^{-6}$ to 0.625 with an average of $2E^{-4}\text{-mg/m}^3$, which was used for toxicity-weighting the emission inventory in this report. The IRIS RfD for carbaryl is $1E^{-1}\text{-mg/kg/day}$.		$2E^{-4}$
Carbon Disulfide	75150	USEPA has not completed a cancer evaluation of carbon disulfide. None of the other authorities referenced in this report have published cancer potency estimates for carbon disulfide either. The IRIS RfC is <u>0.7</u>		0.7

		<u>mg/m³</u>		
Carbon Tetrachloride	56235	USEPA designates carbon tetrachloride a class B2: probable human carcinogen, based on observed carcinogenicity in rats, mice, and hamsters. The IRIS inhalation 1E ⁻⁵ cancer risk level concentration is <u>6.7E⁻⁴-mg/m³</u> . The USEPA has not established an RfC; However, the OEHHA chronic REL is <u>0.04-mg/m³</u> .	6.7E ⁻⁴	0.04
Carbonyl Sulfide	463581	Carbonyl sulfide is as irritant and CNS depressant, but no toxicity benchmarks are available for it from any of the authorities referenced in this report.		
Catechol	120809	No toxicity benchmarks are available for catechol from any of the authorities referenced in this report; However, the ACGIH TLV-TWA for workplace exposures divided by a range of safety factors to allow for continuous exposure to the general population ranges from 2.7E ⁻⁵ to 2.8 with an average of <u>0.0011-mg/m³</u> , which was used as an RfC-like criterion in this report.		0.0011
Chloramben	133904	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Chlordane	57749	USEPA classifies chlordane as a B2: probable human carcinogen, by all routes of exposure, based on human epidemiology studies showing non-Hodgkin's lymphoma in farmers exposed to chlordane and case reports of aplastic anemia; animal studies in which benign and malignant liver tumors were induced in both sexes of four strains of mice, occurring with an elevated, but not statistically significant, incidence in a fifth strain, as well as liver toxicity in rats of two strains; and structural similarity to other rodent liver carcinogens. IRIS inhalation 1E ⁻⁵ cancer risk level concentration is <u>1E⁻⁴-mg/m³</u> . The IRIS RfC is <u>7E⁻⁴-mg/m³</u> .	1E ⁻⁴	7E ⁻⁴
Chlorine	7782505	No cancer or toxicity criteria are available for chlorine from any of the authorities referenced in this report; However, USEPA staff judgment (in the <i>Ranking and Selection of HAPs Under Section 112(k): Technical support Document</i>) provides an RfC of <u>0.02-mg/m³</u> .		0.02
Chloroacetic Acid	79118	No inhalation WOE or toxicity benchmarks are available for chloroacetic acid from any of the authorities referenced in this report. The USEPA Office of Research and Development <i>Health Effects Assessment Summary Tables</i> (July 1997) provide a provisional ingestion risk value (RfD) of 0.002 mg/kg/day, which was used in this report to estimate a daily exposure rate of 0.007-mg for an average 70-kg adult, breathing 20-m ³ /d. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded <u>0.0014-mg/m³</u> as an annual average RfC estimate for toxicity-weight inventory screening.		0.0014
2-Chloroacetophenone	532274	USEPA has not completed a cancer evaluation of 2-chloroacetophenone. None of the other authorities referenced in this report have published cancer potency estimates for it either. The RfC provided by USEPA in IRIS is <u>3E⁻⁵-mg/m³</u>		3E ⁻⁵
Chlorobenzene	108907	USEPA has designated chlorobenzene in class D: Not classifiable as to human carcinogenicity, on the basis of lack of human data, inadequate animal data, and predominantly negative genetic toxicity data in bacterial, yeast, and mouse lymphoma cells. The OEHHA chronic REL is <u>1-mg/m³</u> . The IRIS RfD is 2E ⁻² -mg/kd/d.		1

Chlorobenzilate	510156	No inhalation toxicity benchmarks are available for chlorobenzilate from any of the authorities referenced in this report except the IRIS RfD: 1E-2-mg/kg/d. For this report, the RfD was used to estimate a daily exposure rate of 0.07-mg for an average 70-kg adult, breathing 20-m ³ /d. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded <u>0.014-mg/m³</u> as an RfC estimate.		0.014
Chloroform	67663	USEPA has designated chloroform in class B2: A probable human carcinogen, because it is likely to be carcinogenic to humans by all routes of exposure under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissues. It is not likely to be carcinogenic to humans by any route of exposure under exposure conditions that do not cause cytotoxicity and cell regeneration. The IRIS inhalation 1E ⁻⁵ cancer risk level concentration is <u>4E⁻⁴-mg/m³</u> . The USEPA has not established an RfC; However, the ATSDR chronic inhalation MRL is <u>0.098-mg/m³</u> .	4E ⁻⁴	0.098
Chloromethyl Methyl Ether	107302	USEPA has designated chloromethyl methyl ether (CMME) a class A: Human carcinogen, on the basis of observed increased incidence of respiratory cancer in exposed workers and of respiratory tumors in mice, rats, and hamsters exposed by inhalation. USEPA has not estimated a quantitative cancer potency value; However, the OEHHA URE is 6.9E ⁻⁴ /μg/m ³ , which is equivalent to <u>1.45E⁻⁵-mg/m³</u> at the 1E ⁻⁵ excess cancer risk level. No RfC or similar criterion is available from any of the authorities referenced in this report.	1.45E ⁻⁵	
Chloroprene	126998	The USEPA has designated chloroprene in Group D: Not classifiable as to human carcinogenicity due to inadequate data. USEPA has not established an RfC or RfD for chloroprene. The OEHHA chronic REL of <u>0.001-mg/m³</u> is listed for chloroprene in the California Air Pollution Control Officers Association <i>Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines</i> .		0.001
Cresols/Cresylic Acid (Isomers and Mixture)	1319773	The USEPA has classified o-cresol (2-Methylphenol CAS 95-48-7), m-cresol (3-Methylphenol CAS 108-39-4), and p-cresol (4-Methylphenol CAS 106-44-5) as Group C: Possible human carcinogens, based on an increased incidence of skin papillomas in mice in an initiation-promotion study. The three cresol isomers produced positive results in genetic toxicity studies both alone and in combination. No data health data on cresylic acid (tr cresol) were available for URE derivation. No UREs have been published for cresols/cresylic acids by any of the authorities referenced in this report USEPA has concluded that data are inadequate for the establishment of an RfC for tricresol and mixed cresols. Cresols are central nervous system depressants, corrosive to the skin and eyes, and may induce methemoglobinemia. The OEHHA chronic REL for mixed cresols is <u>0.6-mg/m³</u> , which was used as the toxicity-weight for the inventory. No REL or other RfC-like criterion has been published for tricresol by the authorities referenced in this report.		0.6
O-Cresol	95487	USEPA lists o-cresol as a class C: Possible human carcinogen, but has not developed a quantitative URE. No URE has been published for o-cresol by any of the authorities referenced in this report. The OEHHA chronic REL for o-cresol is <u>6E⁻⁵-mg/m³</u> , which was used as the toxicity-weight for the inventory. No other RfC-like criterion has been published for o-cresol by any of the authorities referenced in this report.		6E ⁻⁵

M-Cresol	108394	USEPA lists m-cresol as a class C: Possible human carcinogen, but has not developed a quantitative URE. No URE has been published for m-cresol by any of the authorities referenced in this report. The OEHHA chronic REL for m-cresol is $6E^{-5}\text{-mg/m}^3$, which was used as the toxicity-weight for the inventory. No other RfC-like criterion has been published for m-cresol by any of the authorities referenced in this report.		$6E^{-5}$
P-Cresol	106445	USEPA lists p-cresol as a class C: Possible human carcinogen, but has not developed a quantitative URE. No URE has been published for p-cresol by any of the authorities referenced in this report. The OEHHA chronic REL for p-cresol is $6E^{-5}\text{-mg/m}^3$, which was used as the toxicity-weight for the inventory. No other RfC-like criterion has been published for p-cresol by any of the authorities referenced in this report.		$6E^{-5}$
Cumene	98828	The USEPA has designated cumene as class D: Not classifiable as to human carcinogenicity due to the absence of human and animal evidence. No quantitative cancer risk assessments are available from other authorities either. The IRIS RfC is 0.4-mg/m^3		0.4
2,4-D, Salts and Esters	94757	No carcinogenicity assessment and few inhalation toxicity benchmarks are available for 2,4-D, salts and esters from any of the authorities referenced in this report. The ACGIH TLV-TWA (10-mg/m^3) and the IRIS RfD ($1E^{-2}\text{-mg/kg/d}$). For toxicity-weighting the emission inventory, the ACGIH TLV-TWA for workplace exposures divided by a safety factor range, to allow for continuous exposure to the general population, ranges from $1.2E^{-5}$ to 1.25 with an average of $4.7E^{-4}\text{-mg/m}^3$. Also, the RfD was used to derive a daily exposure rate of 0.035-mg for an average 70-kg adult, breathing $20\text{-m}^3/\text{d}$. Then a safety factor of 0.2 was used to account for variations in the average daily concentration. This yielded 0.007-mg/m^3 as an alternate RfC estimate.		$4.7E^{-4}$
DDE	3547044	P,P'-Dichlorodiphenyldichloroethylene (DDE) was not reported in the emissions inventory.		
Diazomethane	334883	No toxicity benchmarks are available from any of the authorities referenced in this report.		
Dibenzofurans	132649	The USEPA IRIS states that dibenzofuran is not classifiable as to human carcinogenicity. The other authorities in this report have established no carcinogenic potency estimates either. Further, no RfC-like or RfD-like criteria have been established for dibenzofuran by the authorities referenced in this report, except for the USEPA CERCLA "Superfund" Technical Assistance Center, who published a provisional RfD is $4E^{-3}\text{mg/kg/day}$. This was used to derive a daily exposure rate of 0.014-mg for an average 70-kg adult, breathing $20\text{-m}^3/\text{d}$. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded a product of 0.0028-mg/m^3 as an RfC estimate.		0.0028
1,2-Dibromo-3-chloropropane	96128	No USEPA carcinogenicity assessment is currently available for 1,2-dibromo-3-chloropropane (DBCP); However, the OEHHA published a URE of $1.9E^{-3}/\mu\text{g/m}^3$, which is equivalent to $5.3E^{-6}\text{-mg/m}^3$ at the $1E^{-5}$ excess cancer risk exposure level. The USEPA published an RfC of $2E^{-4}\text{-mg/m}^3$.	$5.3E^{-6}$	$2E^{-4}$
Dibutylphthalate	84742	The USEPA has designated dibutylphthalate as class D: not classifiable as to human carcinogenicity due to the absence of human and animal evidence. No quantitative cancer risk assessments are available from the other authorities referenced in this report either. The ACGIH TLV-TWA for workplace dibutylphthalate exposures, divided by a safety factor range to allow for continuous exposure to the general population,		$2E^{-4}$

ranges from $6E^{-6}$ to 0.625 with an average of $2E^{-4}$ -mg/m³. It was used in preference to an RfD (0.1-mg/kg/day)-derived RfC estimate.

1,4-Dichlorobenzene	106467	The USEPA has not completed an evaluation of 1,4-dichlorobenzene for evidence of human carcinogenic potential; However, IARC lists it as a Group 2B chemical (possibly carcinogenic to humans) based on animal evidence. OEHHA has published a URE of $1.1E^{-5}$ -μg/m ³ , which is equivalent to $9.1E^{-4}$ -mg/m ³ at the E-5 excess cancer risk exposure level. The IRIS RfC is 0.8 -mg/m ³ .	$9.1E^{-4}$	0.8
3,3-Dichlorobenzidine	91941	The USEPA has classified 3,3'-dichlorobenzidine as a B2: Probable human carcinogen, based on statistically significantly increased tumor incidences in rats, mice and dogs. Additional support is provided by evidence of genotoxicity and a structural relationship to the known human bladder carcinogen benzidine. The OEHHA URE, when converted to the equivalent $1E^{-5}$ risk level concentration, is $2.9E^{-5}$ -mg/m ³ . USEPA has not published an inhalation cancer risk estimate but has established a $1E^{-5}$ excess cancer risk drinking water exposure level of 0.8-μg/L, which is equivalent to $1.6E^{-5}$ -mg/m ³ at the $1E^{-5}$ excess cancer risk inhalation exposure level. No non-cancer toxicity benchmarks are available for 3,3-dichlorobenzidine from any of the authorities referenced in this report.	$2.9E^{-5}$	
Dichloroethyl Ether (Bis (2-Chloroethyl) Ether)	111444	Dichloroethyl ether is a synonym of bis (2-chloroethyl) ether (BCEE). The USEPA has classified BCEE as a B2: Probable human carcinogen, based on positive carcinogenicity results in two strains of mice and evidence of mutagenicity. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $3E^{-5}$ -mg/m ³ . USEPA has stated that health effects data are inadequate to derive an RfC for BCEE; However, the OEHHA chronic REL is 0.058 -mg/m ³ .	$3E^{-5}$	0.058
1,3-Dichloropropene	542756	USEPA lists 1,3-dichloropropene as a B2: Probable human carcinogen, because of the lack of data in humans but sufficient evidence of carcinogenicity in animals. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $2E^{-3}$ -mg/m ³ . The IRIS RfC is 0.02 -mg/m ³	$2E^{-3}$	0.02
Dichlorvos	62737	OEHHA URE is $1.2E^{-4}$ -mg/m ³ when converted to the equivalent $1E^{-5}$ Risk Level concentration. The IRIS RfC is $5E^{-4}$ -mg/m ³	$1.2E^{-4}$	$5E^{-4}$
Diethanolamine	111422	No carcinogenicity assessment or inhalation toxicity benchmarks are available for diethanolamine from any of the authorities referenced in this report except the ACGIH. The TLV-TWA for workplace exposures to diethanolamine, divided by a factor range to allow for continuous exposure to the general population, ranges from $2.4E^{-6}$ to 0.25 with an average of $9.3E^{-5}$ -mg/m ³ .		$9.3E^{-5}$
N,N-Diethyl Aniline (N,N-Dimethylaniline)	121697	No inhalation carcinogenicity assessment or toxicity benchmarks are available for n-n-dimethylaniline from any of the authorities referenced in this report except the ACGIH. The TLV-TWA for workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $3E^{-5}$ to 3.1 with an average of 0.0012 -mg/m ³ . IRIS lists the RfD as $2E^{-3}$ mg/kg/day.		0.0012

Diethyl Sulfate	64675	No carcinogenicity assessment or inhalation toxicity benchmarks are available for diethyl sulfate from the authorities referenced in this report. Exposure may cause skin, eye, and respiratory tract irritation.	
3,3-Dimethoxybenzidine	119904	No carcinogenicity assessment or toxicity benchmarks are available for 3,3-dimethoxybenzidine from any of the authorities referenced in this report.	
Dimethyl Aminoazobenzene	60117	The USEPA has not completed an evaluation of 4-dimethylaminoazobenzene for evidence of human carcinogenic potential; However, the OEHHA URE is $1.3E^{-3}/\mu\text{g}/\text{m}^3$, which is equivalent to $7.7E^{-6}\text{-mg}/\text{m}^3$ at the $1E^{-5}$ excess cancer risk level. No RfC-like criterion has been published for 4-dimethylaminoazobenzene by any of the authorities referenced in this report.	$7.7E^{-6}$
3,3'-Dimethyl Benzidine	119937	No carcinogenicity assessment or inhalation toxicity benchmarks are available for 3,3'-dimethyl benzidine from the authorities referenced in this report.	
Dimethyl Carbamoyl Chloride	79447	No carcinogenicity assessment or inhalation toxicity benchmarks are available for dimethylcarbamyl chloride (CAS 79-44-7) from the authorities referenced in this report except the OEHHA URE ($3.7E^{-3}/\mu\text{g}/\text{m}^3$), which is equivalent to $2.7E^{-6}\text{-mg}/\text{m}^3$ at the E-5 excess cancer risk level.	$2.7E^{-6}$
Dimethyl Formamide	68122	No carcinogenicity assessment is available for dimethyl formamide. Available inhalation toxicity benchmarks are the RfC ($3E^{-2}\text{-mg}/\text{m}^3$) and the REL ($8E^{-2}\text{-mg}/\text{m}^3$). The former was used for toxicity-weighting the emission inventory in this report.	$3E^{-2}$
1,1-Dimethyl Hydrazine	57147	No inhalation carcinogenicity assessment or toxicity benchmark is available for 1,1-dimethyl hydrazine from any of the authorities referenced in this report except the ATSDR and ACGIH. The ATSDR intermediate duration inhalation MRL was used to derive a daily exposure rate for an average 70-kg adult, breathing $20\text{-m}^3/\text{d}$. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded $2.5E^{-4}\text{-mg}/\text{m}^3$ as an RfC estimate, which was used to toxicity-weight the emission inventory. For comparison, the TLV-TWA to limit health risk from workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $3.5E^{-8}$ to $9.1E^{-4}$ with an average of $8E^{-7}\text{-mg}/\text{m}^3$.	$2.5E^{-4}$
Dimethyl Phthalate	131113	USEPA lists dimethyl phthalate as class D: Not classifiable as to human carcinogenicity, because pertinent data regarding carcinogenicity was not found in available literature. No RfC or RfD has been developed by USEPA. The only available toxicity assessment among the authorities referenced in this report is from the ACGIH. The TLV-TWA for workplace dimethyl phthalate exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $6E^{-6}$ to 0.625 with an average of $2.3E^{-4}\text{-mg}/\text{m}^3$.	$2.3E^{-4}$
Dimethyl Sulfate	77781	USEPA lists dimethyl sulfate as a B2: Probable human carcinogen, based on induction of local carcinomas following inhalation and subcutaneous exposures in rats, tumor induction in rats following prenatal exposure, and evidence of carcinogenicity in hamsters and mice by inhalation. In addition, dimethyl sulfate alkylates cellular macromolecules and is genotoxic. The only available toxicity assessment among the authorities referenced in this report is from the	$2.4E^{-5}$

		ACGIH. The TLV-TWA for exposures to dimethyl sulfate in the workplace, divided by a range of factors to allow for continuous exposure to the general population, ranges from $6E^{-7}$ to 0.065 with an average of $2.4E^{-5}$ -mg/m ³ .		
4,6-Dinitro-o-Cresol, and Salts	534521	No carcinogenicity assessment or toxicity benchmarks are available for 4,6-dinitro-o-cresol and salts from any of the authorities referenced in this report except the ACGIH. The TLV-TWA for exposures to 4,6-dinitro-o-cresol in the workplace, divided by a range of factors to allow for continuous exposure to the general population, ranges from $2.4E^{-7}$ to 0.025 with an average of $9.3E^{-6}$ -mg/m ³ .		$9.3E^{-6}$
2,4-Dinitrophenol	51285	No carcinogenicity assessment or toxicity benchmarks are available for 2,4-dinitrophenol from any of the authorities referenced in this report except the USEPA RfD of 0.002 mg/kg/d. For toxicity-weighting the emission inventory, the RfD was used to derive a daily exposure rate for an average 70-kg adult, breathing 20-m ³ /d. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded 0.0014 -mg/m ³ as an RfC estimate.		
2,4-Dinitrotoluene	121142	No carcinogenicity assessment is available for 2,4-dinitrotoluene from any of the authorities referenced in this report except the OEHHA, who published a URE of $8.9E^{-5}$ -μg/m ³ . At the E-5 excess cancer risk level, this is equivalent to $1.12E^{-4}$ -mg/m ³ . The OEHHA REL is 0.007 -mg/m ³ was used in preference to the IRIS RfD ($2E^{-3}$ -mg/kg/day) for toxicity-weighting the emission inventory. For comparison, the RfD was used to derive a daily exposure rate equivalent to 0.007 -mg/m ³ /d for an average 70-kg adult, breathing 20-m ³ /d. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded 0.0014 -mg/m ³ as an RfC estimate.	$1.12E^{-4}$	0.007
1,4-Dioxane (1,4-Diethyleneoxide)	123911	The USEPA has classified 1,4-dioxane as a B2: Probable human carcinogen, based on induction of nasal cavity and liver carcinomas in multiple strains of rats, liver carcinomas in mice, and gall bladder carcinomas in guinea pigs. USEPA did not derive an inhalation URE but did publish a drinking water E-5 excess cancer risk exposure estimate of 30/μg/L from which an equivalent $1E^{-5}$ cancer risk level inhalation concentration of $6E^{-4}$ -mg/m ³ was estimated. The OEHHA URE at the equivalent $1E^{-5}$ cancer risk level concentration (0.0013 -mg/m ³) was used in preference to the drinking water URE for toxicity-weighting the emission inventory. USEPA has not published RfC or RfD values for 1,4-dioxane; However, the OEHHA chronic REL is 3 -mg/m ³ .	0.0013	3
1,2-Diphenylhydrazine	122667	The USEPA has classified 1,2-diphenylhydrazine (synonymous with hydrazobenzene) as a B2: Probable human carcinogen, based on positive results of studies in both rats and mice. The two apparently negative studies lacked information on compound purity, experimental design, and statistical treatment. IRIS lists an E-5 excess cancer risk inhalation exposure level of $5E^{-5}$ -mg/m ³ . No RfC-like or RfD-like criteria have been published for 1,2-diphenylhydrazine by any of the authorities referenced in this report.		$5E^{-5}$
Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	106898	The USEPA has classified epichlorohydrin (1-chloro-2,3-epoxypropane) as a B2: probable human carcinogen, with inadequate human data but	$8E^{-3}$	$1E^{-3}$

Epoxypropane)		based on multiple positive studies by various routes in rats and mice. As epichlorohydrin is a strong alkylating agent, tumors are produced at the site of application. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $8E^{-3}\text{-mg/m}^3$, which was used in preference to the OEHHA URE-based $1E^{-5}$ excess cancer risk equivalent exposure level of $4.4E^{-4}\text{-mg/m}^3$. Available non-cancer inhalation toxicity benchmarks are the RfC ($1E^{-3}\text{-mg/m}^3$) and the REL ($3E^{-3}\text{-mg/m}^3$). The former was used for toxicity-weighting the emission inventory in this report.	
1,2-Epoxybutane	106887	No carcinogenicity assessment is available for 1,2-epoxybutane from any of the authorities referenced in this report. Non-cancer inhalation toxicity benchmarks are the USEPA RfC and the OEHHA REL: both $2E^{-2}\text{-mg/m}^3$.	$2E^{-2}$
Ethyl Acrylate	140885	No carcinogenicity assessment is available for ethyl acrylate from any of the authorities referenced in this report. The OEHHA REL ($4.8E^{-2}\text{-mg/m}^3$) was used for toxicity-weighting the emission inventory in this report.	$4.8E^{-2}$
Ethyl Benzene	100414	The USEPA has designated ethylbenzene as not classifiable as to human carcinogenicity due to lack of animal bioassays and human studies. None of the other authorities referenced in this report have published cancer potency estimates for ethylbenzene. Inhalation toxicity benchmarks are the RfC (1-mg/m^3) and the REL (2-mg/m^3). The former was used for toxicity-weighting the emission inventory in this report.	1
Ethyl Carbamate (Urethane)	51796	No carcinogenicity assessment is available for ethyl carbamate (synonymous with urethane) except the OEHHA URE $2.9E^{-4}/\mu\text{g/m}^3$, which is equivalent to a $1E^{-5}$ excess cancer risk equivalent exposure level of $3.5E^{-5}\text{-mg/m}^3$. No non-cancer toxicity benchmarks are available from any of the authorities referenced in this report.	$3.5E^{-5}$
Ethyl Chloride (Chloroethane)	75003	No carcinogenicity assessment is available for ethyl chloride (chloroethane) from any of the authorities referenced in this report. Inhalation toxicity benchmarks are the RfC (10-mg/m^3) and the REL (30-mg/m^3). The former was used for toxicity-weighting the emission inventory in this report.	10
Ethylene Dibromide (Dibromoethane)	106934	The USEPA has classified ethylene dibromide (1,2-dibromoethane) as a B2: probable human carcinogen based on increased incidences of a variety of tumors in rats and mice in both sexes by three routes of administration at both the site of application and at distant sites. EDB is mutagenic in various <i>in vitro</i> and <i>in vivo</i> assays. EDB is structurally similar to DBCP and to ethylene dichloride, both probable human carcinogens. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $5E^{-5}\text{-mg/m}^3$. The USEPA has not published an RfD or RfC; However, the OEHHA REL is $8E^{-4}\text{-mg/m}^3$.	$5E^{-5}$ $8E^{-4}$
Ethylene Dichloride (1,2-Dichloroethane)	107062	The USEPA has classified ethylene dichloride (1,2-dichloroethane) as a B2: probable human carcinogen, based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $4E^{-4}\text{-mg/m}^3$. The OEHHA URE is $2.1E^{-5}/\mu\text{g/m}^3$. The USEPA has not published an RfD or RfC; However, the ATSDR chronic inhalation MRL is 2.4-mg/m^3 . It was used in preference to the OEHHA REL (0.4-mg/m^3).	$4E^{-4}$ 2.4
Ethylene Glycol	107211	No carcinogenicity assessment or toxicity benchmarks are available for ethylene glycol from any of the authorities referenced in this report. The USEPA has not published an RfC; However, the OEHHA REL is 0.4-mg/m^3 .	0.4

mg/m³. For comparison, the RfD is 2-mg/kg/day.

Ethylene Imine (Aziridine)	151564	No carcinogenicity assessment is available for ethylene imine (aziridine) from the USEPA, currently. The OEHHA cancer potency estimate ($1.9E^{-2}/\mu\text{g}/\text{m}^3$) is $5.3E^{-7}\text{-mg}/\text{m}^3$ at the E-5 excess risk level concentration. The USEPA and other authorities referenced in this report have not published RfC-like or RfD-like values except for the ACGIH. The TLV-TWA for workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $1.1E^{-6}$ to 0.11 with an average of $4.1E^{-5}\text{-mg}/\text{m}^3$, which was used for toxicity-weighting the emission inventory in this report.	$5.3E^{-7}$	$4.1E^{-5}$
Ethylene Oxide	75218	No carcinogenicity assessment for ethylene oxide is currently available from the USEPA. IARC lists it as a Group 1 carcinogen because of limited evidence in humans, but sufficient evidence in experimental animals. The OEHHA cancer potency estimate ($8.8E^{-5}/\mu\text{g}/\text{m}^3$) is equivalent to $1.1E^{-4}\text{-mg}/\text{m}^3$ at the $1E^{-5}$ excess risk level concentration. The USEPA has not published an RfC; However, the OEHHA chronic REL is $0.03\text{-mg}/\text{m}^3$.	$1.1E^{-4}$	0.03
Ethylene Thiourea	96457	No carcinogenicity assessment for ethylene thiourea is currently available from the USEPA. The OEHHA cancer potency estimate ($1.3E^{-5}/\mu\text{g}/\text{m}^3$) is $7.7E^{-4}\text{-mg}/\text{m}^3$ at the $1E^{-5}$ excess risk level concentration. None of the authorities referenced in this report have published non-cancer criteria for ethylene thioiurea.	$7.7E^{-4}$	
Ethylidene Dichloride (1,1- Dichloroethane)	75343	USEPA has designated ethylidene dichloride (synonymous with 1,1-dichloroethane) a class C: Possible human carcinogen, based on no human data but limited evidence of carcinogenicity in two animal species (rats and mice) as shown by an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas in female rats and an increased incidence of hepatocellular carcinomas and benign uterine polyps in mice. USEPA has not issued a quantitative cancer potency estimate; However, the OEHHA URE for ethylidene dichloride ($1.6E^{-6}/\mu\text{g}/\text{m}^3$) is equivalent to $6.3E^{-3}\text{-mg}/\text{m}^3$ at the $1E^{-5}$ excess cancer risk level concentration. None of the authorities referenced in this report have published non-cancer values for ethylidene dichloride; However, the ACGIH TLV-TWA for workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $4.8E^{-4}$ to 50.6 with an average of $0.0188\text{-mg}/\text{m}^3$	$6.3E^{-3}$	0.0188
Formaldehyde	50000	USEPA has designated formaldehyde a B1: Probable human carcinogen, based on limited evidence in humans, and sufficient evidence in animals. Human data include nine studies that show statistically significant associations between site-specific respiratory neoplasms and exposure to formaldehyde or formaldehyde-containing products. An increased incidence of nasal squamous cell carcinomas was observed in long-term inhalation studies in rats and in mice. The designation is supported by in vitro genotoxicity data and formaldehyde's structural relationships to other carcinogenic aldehydes such as acetaldehyde. IRIS lists a $1E^{-5}$ excess cancer risk inhalation exposure level of $8E^{-4}\text{-mg}/\text{m}^3$ used for toxicity-weighting the emission inventory. The OEHHA URE for formaldehyde is $6E^{-6}/\mu\text{g}/\text{m}^3$. USEPA has not issued an RfC; However, the ATSDR chronic inhalation MRL is $0.0098\text{-mg}/\text{m}^3$. It was used in preference to the OEHHA REL, which is $3E^{-3}\text{-mg}/\text{m}^3$.	$8E^{-4}$	0.0098

Heptachlor	76448	USEPA has designated heptachlor a B2: Probable human carcinogen, based on inadequate human data, but sufficient evidence exist from studies in which benign and malignant liver tumors were induced in three strains of mice of both sexes. Further, several structurally related compounds are liver carcinogens. The IRIS E-5 excess cancer risk level inhalation concentration is $8E^{-6}$ -mg/m ³ . USEPA and other authorities referenced in this report have not issued an RfC-like value for heptachlor. For toxicity-weighting the emission inventory, the ACGIH TLV-TWA for workplace exposures, divided by a factor range to adjust for continuous exposure to the general population, ranges from $6E^{-8}$ to 0.0063, with an average of $2.3E^{-6}$ -mg/m ³ . The adjusted TLV-TWA was used in preference to an RfC estimate derived from the RfD, which is $5E^{-4}$ -mg/kg/d.	$8E^{-6}$	$2.3E^{-6}$
Hexachlorobenzene	118741	The USEPA has designated hexachlorobenzene a B2: Probable human carcinogen, based on inadequate human data but evidence that when administered orally, it induces tumors in the liver, thyroid and kidney in three rodent species. The IRIS and OEHHA $1E^{-5}$ cancer risk level inhalation concentrations are $2E^{-5}$ -mg/m ³ . USEPA has not issued an RfC; However, the OEHHA chronic REL is 0.0028 -mg/m ³ .	$2E^{-5}$	0.0028
Hexachloro-butadiene	87683	The USEPA has designated hexachlorobutadiene a class C: Possible human carcinogen, based on observation of renal neoplasms in male and female rats in one study, but lacking any human data. The IRIS $1E^{-5}$ cancer risk level inhalation concentration is $5E^{-4}$ -mg/m ³ . USEPA and other authorities referenced in this report have not issued an RfC-like value for hexachlorobutadiene. For toxicity-weighting the emission inventory, the ACGIH TLV-TWA for workplace exposures was used. It was divided by a factor range to adjust for continuous exposure to the general population. The results range from $2.5E^{-7}$ to $2.7E^{-2}$ with an average of $9.9E^{-6}$ -mg/m ³ .	$5E^{-4}$	$9.9E^{-6}$
Hexachlorocyclo-Pentadiene	77474	The USEPA has designated hexachlorocyclopentadiene (HCCPD) a group E chemical: Evidently non carcinogenic to humans via inhalation exposure. The apparent inability of HCCPD to cause genotoxic effects, and the lack of evidence for both human and animal carcinogenicity by the inhalation route, justify the conclusion that HCCPD is not likely to present a human cancer risk via inhalation exposure. The RfC and REL are $2E^{-4}$ -mg/m ³ , which was used in preference to the ATSDR chronic inhalation MRL ($2.2E^{-3}$ -mg/m ³).		$2E^{-4}$
Hexachloroethane	67721	The IRIS $1E^{-5}$ cancer risk level inhalation concentration is 0.0025 -mg/m ³ . The OEHHA chronic REL is 29.048 -mg/m ³	0.0025	29.048
Hexamethylene-1,6-Diisocyanate	822060	No carcinogenicity assessment is available for hexamethylene-1,6-diisocyanate from any of the authorities referenced in this report. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the RfC ($1E^{-5}$ -mg/m ³).		$1E^{-5}$
Hexamethyl-Phosphoramidate	680319	Hexamethylphosphoramidate is listed as a HAP, but was not included in the emissions inventory. Ambient concentration data were not available either.		
Hexane	110543	No carcinogenicity assessments are available for hexane from any of the authorities referenced in this report. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the RfC (0.2 -mg/m ³). The REL is 7 -mg/m ³ .		0.2

Hydrazine	302012	The USEPA has designated hydrazine a B2: Probable human carcinogen, based on inadequate human data but evidence of tumor induction in mice, rats and hamsters following oral, inhalation or intraperitoneal administration of hydrazine and hydrazine sulfate. Further, hydrazine is mutagenic in numerous assays. The IRIS $1E^{-5}$ cancer risk level concentration is $2E^{-6}$ -mg/m ³ . The OEHHA $1E^{-5}$ cancer risk level concentration is much lower ($5.9E^{-10}$ -mg/m ³). No RfC is available from USEPA currently; However, the OEHHA chronic REL is $2E^{-4}$ -mg/m ³ .	$2E^{-6}$	$2E^{-4}$
Hydrochloric Acid	7647010	No carcinogenicity assessments are available for hydrochloric acid (hydrogen chloride) from any of the authorities referenced in this report. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the RfC (0.02 -mg/m ³). The REL is $9E^{-3}$ -mg/m ³ .		0.02
Hydrogen Fluoride (Hydrofluoric Acid)	7664393	No carcinogenicity assessments are available for hydrofluoric acid (hydrogen fluoride) from any of the authorities referenced in this report. No RfC is available from USEPA currently. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the OEHHA REL for hydrogen fluoride gas and particulate fluoride salts (0.014 -mg/m ³).		0.014
Hydroquinone	123319	No inhalation carcinogenicity assessment or toxicity benchmarks are available for hydroquinone from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $2.3E^{-6}$ to 0.25 with an average of $9.3E^{-5}$ -mg/m ³ .		$9.3E^{-5}$
Isophorone	78591	The USEPA has designated isophorone a class C: Possible human carcinogen, based on limited evidence of carcinogenicity of one tumor type in one sex of one animal species as shown by an increase of preputial gland carcinomas in male rats. The apparent renal tubular cell tumor in male rats is associated with alpha-2u-globulin, considered to be of questionable relevance to humans. No carcinogenicity data in humans is available. No quantitative carcinogenic potency estimate is available from any of the authorities referenced in this report. No RfC is available from USEPA currently. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the OEHHA REL (2 -mg/m ³).		2
Lindane (All Isomers)	58899	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Maleic Anhydride	108316	No carcinogenicity assessment for maleic anhydride is available from any of the authorities referenced in this report. No RfC is available from USEPA currently. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the OEHHA REL ($7E^{-4}$ -mg/m ³).		$7E^{-4}$
Methanol	67561	No carcinogenicity assessment for methanol is available from any of the authorities referenced in this report. No RfC is available from USEPA currently. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the OEHHA REL (4 -mg/m ³).		4
Methoxychlor	72435	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		

Methyl Bromide (Bromomethane)	74839	USEPA lists methyl bromide (synonymous with bromomethane) as class D: Not classifiable as to human carcinogenicity, because human and animal data are inadequate. There is a single mortality study from which direct exposure associations could not be deduced, and studies in several animal species with too few animals, and too brief exposure or observation time for adequate power. However, bromomethane has shown genotoxicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. The RfC and REL are $5E^{-3}\text{-mg/m}^3$.	$5E^{-3}$
Methyl Chloride (Chloromethane)	74873	USEPA lists methyl chloride as class D: Not classifiable as to human carcinogenicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the RfC ($9E^{-2}\text{-mg/m}^3$). The ATSDR chronic inhalation MRL is 0.1-mg/m^3 .	$9E^{-2}$
Methyl Chloroform (1,1,1-Trichloroethane)	71556	USEPA lists methyl chloroform (1,1,1-trichloroethane) as class D: Not classifiable as to human carcinogenicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. No RfC was available from USEPA. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the REL (1-mg/m^3).	1
Methyl Ethyl Ketone (2- Butanone)	78933	USEPA lists methyl ethyl ketone (2-butanone) as class D: Not classifiable as to human carcinogenicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the IRIS RfC (15-mg/m^3).	15
Methyl Hydrazine	60344	No inhalation carcinogenicity assessment or toxicity benchmarks are available for methyl hydrazine from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $2.2E^{-8}$ to $2.4E^{-3}$ with an average of $8.8E^{-7}\text{-mg/m}^3$.	$8.8E^{-7}$
Methyl Iodide (Iodomethane)	74884	No inhalation carcinogenicity assessment or toxicity benchmarks are available for methyl iodide (iodomethane) from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a safety factor range to allow for continuous exposure to the general population, ranges from $1.4E^{-5}$ to 1.5 with an average of $5.4E^{-4}\text{-mg/m}^3$.	$5.4E^{-4}$
Methyl Isobutyl Ketone (Hexone)	108101	No inhalation carcinogenicity assessment or toxicity benchmark is available for methyl isobutyl ketone (synonymous with hexone) from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $2.4E^{-4}$ to 25.6 with an average of $9.5E^{-3}\text{-mg/m}^3$.	$9.5E^{-3}$
Methyl Isocyanate	624839	No inhalation carcinogenicity assessment is available for methyl isocyanate any of the authorities referenced in this report. No RfC has been established. The non-cancer inhalation benchmark used for toxicity-weighting the emission inventory was the REL $1E^{-3}\text{-mg/m}^3$.	$1E^{-3}$
Methyl Methacrylate	80626	USEPA classified methyl methacrylate (MMA) as a group E chemical: Not likely to be carcinogenic to humans by any route of exposure, because it has been evaluated in four well-conducted chronic inhalation studies in three appropriate animal species without demonstrating	0.7

carcinogenic effects. The IRIS RfC is 0.7-mg/m³

Methyl Tert Butyl Ether	1634044	No carcinogenicity assessment is available for methyl tertiary butyl ether (methyl tert-butyl ether or MTBE) except by the OEHHA. Their URE ($2.6E^{-7}$ - $\mu\text{g}/\text{m}^3$) is equivalent to a $1E^{-5}$ excess cancer risk exposure level of <u>$3.9E^{-2}$-mg/m^3</u> . The IRIS RfC is <u>3-mg/m^3</u> .	$3.9E^{-2}$	3
4,4-Methylene Bis (2-Chloroaniline)	101144	No carcinogenicity assessment is available for 4,4-methylene bis (2-chloroaniline) except by the OEHHA. Their URE ($4.3E^{-4}/\mu\text{g}/\text{m}^3$) is equivalent to a $1E^{-5}$ excess cancer risk exposure level of <u>$2.3E^{-5}$-mg/m^3</u> . No RfC or RfD-like criteria have been published by the authorities referenced in this report.	$2.3E^{-5}$	
Methylene Chloride (Dichloromethane)	75092	The USEPA has designated methylene chloride (dichloromethane) a B2: Probable human carcinogen, based on inadequate human data but sufficient evidence of carcinogenicity in animals; increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, and increased incidence of benign mammary tumors in both sexes of rats, salivary gland sarcomas in male rats and leukemia in female rats. The classification is supported by some positive genotoxicity data, although results in mammalian systems are generally negative. The IRIS $1E^{-5}$ excess cancer risk level inhalation concentration is <u>$2E^{-2}$-mg/m^3</u> . No RfC is currently available; However, the ATSDR chronic inhalation MRL is <u>1-mg/m^3</u> .	$2E^{-2}$	1
Methylene Diphenyl Diisocyanate (MDI)	101688	USEPA lists methylene diphenyl diisocyanate (MDI) as class D chemical: Not classifiable as to human carcinogenicity, stating that the carcinogenic potential of MDI and polymeric MDI cannot be determined, but there is evidence that raises concern for carcinogenic effects. No carcinogenic potency estimates are available from any of the authorities referenced in this report. The non-cancer inhalation toxicity benchmark used for toxicity-weighting the emission inventory was the RfC (<u>$6E^{-4}$-mg/m^3</u>). The REL is $7E^{-4}$ - mg/m^3 .		$6E^{-4}$
4,4'-Methylenedianiline	101779	No carcinogenicity assessment is available for 4,4'-methylenedianiline except by the OEHHA. The OEHHA inhalation URE ($4.6E^{-4}/\mu\text{g}/\text{m}^3$) is equivalent to a $1E^{-5}$ excess cancer risk exposure level of <u>$2.2E^{-5}$-mg/m^3</u> . No RfC has been published by the USEPA; However, the OEHHA lists a chronic REL of <u>0.0019-mg/m^3</u> .	$2.2E^{-5}$	0.0019
Naphthalene	91203	USEPA designates naphthalene a group C: Possible human carcinogen, based on inadequate data of carcinogenicity in humans exposed to naphthalene via oral and inhalation routes, but limited evidence of carcinogenicity in animals via inhalation (observations of benign respiratory tumors and one carcinoma in female mice only exposed to naphthalene by inhalation). USEPA and the other authorities referenced in this report have not developed a quantitative estimate of carcinogenic risk from inhalation exposure. The IRIS RfC for naphthalene is <u>0.003-mg/m^3</u> . The REL is $9E^{-3}$ - mg/m^3		0.003
Nitrobenzene	98953	USEPA lists nitrobenzene as class D chemical: Not classifiable as to human carcinogenicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. No RfC is available from USEPA currently. The IRIS RfD ($5E^{-4}$ - $\text{mg}/\text{kg}/\text{day}$) was used to derive an RfC estimate for toxicity-weighting the emission inventory: The equivalent daily inhalation exposure rate is 0.00175 - $\text{mg}/\text{m}^3/\text{d}$ for an		$3.5E^{-4}$

average 70-kg adult, breathing 20-m³/d. Applying a factor of 0.2, to account for variations in the average daily concentration, yields 3.5E⁻⁴-mg/m³ as an RfC estimate.

4-Nitrobiphenyl	92933	No carcinogenicity assessment or toxicity benchmark for 4-nitrobiphenyl is available from any of the authorities referenced in this report.		
4-Nitrophenol	100027	No carcinogenicity assessment or toxicity benchmark for 4-nitrophenol is available from any of the authorities referenced in this report.		
2-Nitropropane	79469	No carcinogenicity assessment by the authorities referenced in this report is available for 2-nitropropane. The IRIS RfC is 0.02-mg/m ³ .		0.02
N-Nitroso-N-Methylurea	684935	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
N-Nitroso-Dimethylamine	62759	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
N-Nitrosomorpholine	59892	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Parathion	56382	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Pentachloronitrobenzene (Quintobenzene)	82688	No carcinogenicity assessment is available from any of the authorities referenced in this report. The USEPA IRIS RfD for pentachloronitrobenzene (quintobenzene or PCNB) was used to derive an RfC estimate for toxicity-weighting the emission inventory. The RfD is 3E ⁻³ -mg/kg/day based on liver toxicity. The equivalent daily inhalation exposure rate is 0.0105-mg/m ³ /d for an average 70-kg adult, inhaling 20-m ³ /d. Applying a factor of 0.2, to account for variations in the average daily concentration, yields <u>2.1E⁻³-mg/m³</u> as an RfC estimate.		2.1E ⁻³
Pentachlorophenol	87865	The USEPA has designated pentachlorophenol (PeCP) a B2: probable human carcinogen, based on inadequate human data but sufficient evidence of carcinogenicity in animals: statistically significant increases in the incidences of multiple biologically significant tumor types (hepatocellular adenomas and carcinomas, adrenal medulla pheochromocytomas and malignant pheochromocytomas, and/or hemangiosarcomas and hemangiomas) in one or both sexes of B6C3F1 mice using two different preparations of PeCP. In addition, a high incidence of two uncommon tumors (adrenal medulla pheochromocytomas and hemangiomas / hemangiosarcomas) was observed with both preparations. This classification is supported by mutagenicity data, which provides some indication that PeCP has clastogenic potential. No inhalation carcinogenicity potency estimate is available from the USEPA; However, the OEHHA URE (4.6E ⁻⁷ /μg/m ³) was converted to its equivalent 1E ⁻⁵ cancer risk level concentration of <u>2.2E⁻²-mg/m³</u> . No RfC was available, therefore the OEHHA chronic REL (<u>2E⁻⁴-mg/m³</u>) was used for toxicity-weighting the emission inventory.	2.2E ⁻²	2E ⁻⁴
Phenol	108952	USEPA lists phenol as a class D chemical: Not classifiable as to human carcinogenicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. No RfC has been published; However the OEHHA chronic REL is <u>0.2-mg/m³</u> .		0.2

P-Phenylenediamine	106503	No inhalation carcinogenicity assessment or toxicity benchmarks are available for p-phenylenediamine from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a safety factor range to allow for continuous exposure to the general population, ranges from $1.2E^{-7}$ to 0.0125 with an average of $4.7E^{-6}$ -mg/m ³ .	4.7E-6
Phosgene	75445	No carcinogenicity assessment or toxicity benchmarks are available for phosgene from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a safety factor range to allow for continuous exposure to the general population, ranges from $4.8E^{-7}$ to $5.1E^{-2}$ with an average of $1.9E^{-5}$ -mg/m ³ .	1.9E-5
Phosphine	7803512	Not reported in the emissions inventory.	
Phosphorus	7723140	USEPA lists phosphorus as a class D chemical: Not classifiable as to human carcinogenicity. No carcinogenic potency estimates are available from any of the authorities referenced in this report. No RfC-like values have been published by any of the authorities referenced in this report, either. The RfD ($2E^{-5}$ -mg/kg/day) was used to derive an RfC estimate for toxicity-weighting the emission inventory. The equivalent daily inhalation exposure rate is $7E^{-5}$ -mg/m ³ /d for an average 70-kg adult, inhaling 20-m ³ /d. Applying a factor of 0.2, to account for variations in the average daily concentration, yields $1.4E^{-5}$ -mg/m ³ as an RfC estimate.	1.4E-5
Phosphorous Acid	13598362	USEPA has designated phosphorus acid as class D: Not classifiable as to human carcinogenicity. No carcinogenicity assessment is available from any of the authorities referenced in this report. The IRIS RfC is $1E^{-2}$ -mg/m ³	1E-2
Phosphorus Compounds		USEPA has designated white phosphorus a class D chemical: Not classifiable as to human carcinogenicity. No carcinogenicity assessment is available from any of the authorities referenced in this report. USEPA has not established an RfC for any phosphorus compounds; However, the OEHHA lists a REL of $7E^{-5}$ -mg/m ³ for white phosphorus. Using this for toxicity-weighting of any phosphorus emissions was probably overly conservative but still resulted in relatively low and insignificant ranks for the phosphorus compounds.	7E-5
Phthalic Anhydride	85449	No carcinogenicity assessment by the authorities referenced in this report is available for phthalic anhydride. The USEPA has not published an RfC; However, the OEHHA chronic REL is 0.02 -mg/m ³ .	0.02
Polychlorinated Biphenyls (Aroclors)	1336363	The USEPA has designated polychlorinated biphenyls (aroclor) class B2: Probable human carcinogens, based on a 1996 study that found liver tumors in female rats exposed to aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to 1260. These mixtures contain overlapping groups of congeners that together span the range of congeners most often found in environmental mixtures. Earlier studies found high, statistically significant incidences of liver tumors in rats ingesting aroclor 1260 or clophen A 60. Mechanistic studies are beginning to identify several congeners that have dioxin-like activity and may promote tumors by different modes of action. PCBs are absorbed through ingestion, inhalation, and dermal exposure, after which they are transported similarly through the circulation. This provides a reasonable basis for expecting similar internal effects from different routes of environmental exposure. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small. The currently	$1E^{-4}$ 0.0012

		available human evidence is inadequate, but suggestive. The IRIS inhalation E-5 excess cancer risk level concentration is $1E^{-4}\text{-mg/m}^3$. The USEPA has not published an RfC; However, the OEHHA chronic inhalation REL is 0.0012-mg/m^3 .		
1,3-Propane Sultone	1120714	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Beta-Propiolactone	57578	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Propionaldehyde	123386	No carcinogenicity assessment or toxicity benchmarks are available for propionaldehyde from any of the authorities referenced in this report.		
Propoxur (Baygon)	114261	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Propylene Dichloride (1,2-Dichloropropane)	78875	No carcinogenicity assessment is available for propylene dichloride (1,2-dichloropropane) from any of the authorities referenced in this report. The IRIS RfC is $4E^{-3}\text{-mg/m}^3$.		$4E^{-3}$
Propylene Oxide	75569	The USEPA has designated propylene oxide a B2: probable human carcinogen, based on inadequate human data but an increased incidence of benign and malignant tumors at the site of exposure in two species of animals, when exposed by subcutaneous injection, by inhalation, and by gavage. There is also evidence of mutagenicity in a variety of test systems. Propylene oxide is structurally similar to other chemicals that demonstrate carcinogenic activity in animals. The IRIS inhalation $1E^{-5}$ excess cancer risk level concentration is $3E^{-3}\text{-mg/m}^3$. The IRIS RfC is 0.03-mg/m^3 .	$3E^{-3}$	0.03
1,2-Propylenimine (2-Methyl Aziridine)	75558	No carcinogenicity assessment or toxicity benchmarks are available for 1,2-propylenimine (2-methyl aziridine) from any of the authorities referenced in this report except the ACGIH. The TLV-TWA to limit non-cancer risk from workplace exposures, divided by a safety factor range to allow for continuous exposure to the general population, ranges from $5.6E^{-6}$ to 0.58 with an average of $2.2E^{-4}\text{-mg/m}^3$.		$2.2E^{-4}$
Quinoline	91225	USEPA has classified quinoline a group B2: Probable human carcinogen, on the basis of observations of exposure-related increased incidence of an unusual malignant tumor in multiple strains of rats and mice, multiple experiments by several routes, dosing at an early age. The determination is supported by studies that demonstrate that quinoline is genotoxic. Recent evidence from mitogenicity and mutagenicity studies and two dietary studies in rats indicates that sufficient animal evidence of carcinogenicity exists. USEPA and the other authorities referenced in this report have not estimated a quantitative cancer potency. Quinoline causes liver damage in test animals exposed by ingestion. It may be an irritant of the eye and respiratory tract; However, none of the referenced authorities have issued RfC-like or RfD-like values.		
Quinone	106514	No carcinogenicity assessment or toxicity benchmarks are available for quinone from any of the authorities referenced in this report except the ACGIH. The TLV-TWA for exposures in the workplace, divided by a range of safety factors to allow for continuous exposure to the general population, ranges from $5.3E^{-7}$ to $5.5E^{-2}$ with an average of $2.1E^{-5}\text{-mg/m}^3$.		$2.1E^{-5}$
Styrene	100425	No carcinogenicity assessment or toxicity benchmarks are available for styrene from any of the authorities referenced in this report. The IRIS RfC (1-mg/m^3) - based on CNS effects - was used to weight the emission inventory. For comparison, the REL is 0.9-mg/m^3 .		1

Styrene Oxide	96093	No carcinogenicity assessment of styrene oxide is available from USEPA; However, the OEHHA has issued an inhalation URE of $4.6E-5/\mu\text{g}/\text{m}^3$, which is equivalent to $2.2E^{-2}\text{-mg}/\text{m}^3$ at the $1E^{-5}$ excess cancer risk exposure level. None of the authorities referenced in this report have issued RfC-like or RfD-like values for styrene oxide.	$2.2E^{-2}$	
2,3,7,8-Tetrachlorodibenzo-p-Dioxin	1746016	2,3,7,8-Tetrachlorodibenzo-p-dioxin is the only chlorinated dioxin/furan-group chemical listed among the 188 federal HAPs. The chlorinated dioxin/furan-group, itself, is not listed as a HAP. However, in the emissions inventory, chlorinated dioxins/furans are listed in a variety of ways, i.e., as "Dioxins/Furans", "Dioxins/Furans as 2,3,7,8-TCDD TEQ", "Dioxins / Furans {total, non-TEQ}", "1,2,3,4,7,8,9-Heptachlorodibenzofuran", "1,2,3,6,7,8-Hexachlorodibenzofuran", "2,3,7,8-Tetrachlorodibenzofuran", and 2,3,4,7,8-Pentachlorodibenzofuran". These were toxicity-weighted individually to the extent possible, given available information.		
Dioxins		As for the toxicity of dioxins, USEPA lists only the subset mixture of hexachlorodibenzo-p-dioxins, which it designates class B2: Probable human carcinogens. The quantitative estimate in IRIS for a $1E^{-5}$ excess cancer risk from a mixture of hexachlorodibenzo-p-dioxins is $8E^{-9}\text{-mg}/\text{m}^3$. The OEHHA URE for "TCDD and toxic equivalents" is equivalent to a $1E^{-5}$ excess cancer risk level concentration of $2.6E^{-9}\text{-mg}/\text{m}^3$. USEPA has not published an RfC for any chlorinated dioxin/furan. OEHHA lists a chronic REL of $4E^{-8}\text{-mg}/\text{m}^3$ for "chlorinated dibenzo-p-dioxins (as 2,3,7,8-TCDD equivalents)".	$8E^{-9}$	$4E^{-8}$
Dioxins/Furans {Total, Non-TEQ}		Chemicals in the chlorinated dioxins/furans group that are not known to be toxicologically similar to TCDD are nonetheless reported in the emissions inventory. The non-TEQ group was treated as though non-toxic due to the lack of data to the contrary. One specific chemical in the non-TEQ category was reported in the emissions inventory: 1,2,3,6,7,8-Hexachloro-dibenzofuran (CASRN 57117449).		
1,2,3,4,7,8,9-Heptachloro-dibenzofuran	55673897	1,2,3,4,7,8,9-heptachlorodibenzofuran has not specifically been assessed by USEPA; However, the available OEHHA URE is equivalent to a $1E^{-5}$ cancer risk level concentration of $2.6E^{-8}\text{-mg}/\text{m}^3$. The OEHHA chronic REL is $4E^{-6}\text{-mg}/\text{m}^3$.	$2.6E^{-8}$	$4E^{-6}$
2,3,7,8-Tetrachlorodibenzo furan	51207319	2,3,7,8-tetrachlorodibenzofuran has not specifically been assessed by USEPA. The OEHHA URE is equivalent to a $1E^{-5}$ cancer risk level concentration of $2.6E^{-9}\text{-mg}/\text{m}^3$. The OEHHA chronic REL is $4E^{-7}\text{-mg}/\text{m}^3$.	$2.6E^{-9}$	$4E^{-7}$
2,3,4,7,8-Pentachloro-dibenzofuran	57117314	2,3,4,7,8-Pentachlorodibenzofuran has not specifically been assessed by USEPA. For toxicity-weighting the emission inventory, the OEHHA URE was converted to its equivalent $1E^{-5}$ cancer risk level concentration of $5.3E^{-10}\text{-mg}/\text{m}^3$. The OEHHA chronic REL ($8E^{-8}\text{-mg}/\text{m}^3$) was also used for inventory weighting.	$5.3E^{-10}$	$8E^{-8}$
1,1,2,2-Tetrachloroethane	79345	The USEPA has designated 1,1,2,2-Tetrachloroethane a class C: Possible human carcinogen, based on increased incidence of hepatocellular carcinomas in mice but a lack of data on human carcinogenicity. The IRIS inhalation E-5 excess cancer risk level concentration is $2E^{-4}\text{-mg}/\text{m}^3$. The ATSDR intermediate duration inhalation MRL was used to derive a daily exposure rate for an average 70-kg adult, breathing $20\text{-m}^3/\text{d}$ ay. To this a factor of 0.2 was applied to account for variations in the average daily concentration. This yielded	$2E^{-4}$	1.4

1.4-mg/m³ as an RfC estimate.

Tetrachloro-ethylene (Perchloroethylene)	127184	No carcinogenicity assessment of tetrachloroethylene (perchloroethylene) is available from USEPA; However, the IARC lists it as a Group 2A chemical (probably a human carcinogen) based on equivocal evidence in animals and on several human epidemiological studies showing elevated risks of certain types of cancer. The OEHHA has issued an inhalation URE of $5.9E^{-6}/\mu\text{g}/\text{m}^3$, which is equivalent to a $1E^{-5}$ excess cancer risk level concentration of <u>$1.7E^{-3}\text{-mg}/\text{m}^3$</u> . This value was derived from the tumor incidence data for the most sensitive species, sex, and tumor site: male mouse hepatocellular adenomas or carcinomas. Epidemiological data are currently insufficient for establishing a cancer unit risk estimate. The ATSDR chronic inhalation MRL is <u>$0.3\text{-mg}/\text{m}^3$</u> .	$1.7E^{-3}$	0.3
Titanium Tetrachloride	7550450	No carcinogenicity assessment or toxicity benchmarks are available for titanium tetrachloride from any of the authorities referenced in this report except the ATSDR. The ATSDR chronic inhalation MRL for titanium tetrachloride is $1E^{-4}\text{-mg}/\text{m}^3$.		$1E^{-4}$
Toluene	108883	The USEPA has designated toluene a class D chemical: Not classified as to carcinogenicity, based on lack of human data and inadequate animal data. USEPA notes that toluene has not produce positive results in the majority of genotoxic assays. No quantitative carcinogenic potency estimates are available from any of the authorities referenced in this report. The RfC is <u>$0.4\text{-mg}/\text{m}^3$</u> .		0.4
2,4-Toluene Diamine	95807	No carcinogenicity assessment or other toxicity benchmarks are available for 2,4-toluene diamine (toluene-2,4-diamine) from any of the authorities referenced in this report.		
2,4-Toluene Diisocyanate	584849	USEPA has not evaluated 2,4-toluene diisocyanate (CASRN 584849) or 2,4-/2,6-toluene diisocyanate mixture (TDI) (CASRN 26471625) for carcinogenicity; However, the OEHHA lists the inhalation URE for "toluene diisocyanate" as $1.1E^{-5}/\mu\text{g}/\text{m}^3$, which is equivalent to an E-5 excess cancer risk level concentration of <u>$9.1E^{-4}\text{-mg}/\text{m}^3$</u> . Both the chronic inhalation RfC and REL for TDI are <u>$7E^{-5}\text{-mg}/\text{m}^3$</u> .	$9.1E^{-4}$	$7E^{-5}$
O-Toluidine	95534	USEPA has not evaluated o-toluidine for carcinogenicity; However, the OEHHA lists the inhalation URE of $5.1E^{-5}/\mu\text{g}/\text{m}^3$ for o-toluidine, which is equivalent to a $1E^{-5}$ excess cancer risk level concentration of <u>$2E^{-4}\text{-mg}/\text{m}^3$</u> . The ACGIH TLV-TWA for workplace exposures, divided by a factor range to allow for continuous exposure to the general population, ranges from $1E^{-5}$ to 1.1 with an average of <u>$4.1E^{-4}\text{-mg}/\text{m}^3$</u> , which was used like an RfC for toxicity-weighting the emission inventory.	$2E^{-4}$	$4.1E^{-4}$
Toxaphene (Chlorinated Camphenes)	8001352	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
1,2,4-Trichlorobenzene	120821	USEPA has designated 1,2,4-trichlorobenzene as a class D chemical: Not classifiable as to human carcinogenicity due lack of relevant data. A single dermal exposure study in mice was found inadequate for drawing conclusions as to carcinogenicity in humans. No quantitative carcinogenic potency estimates are available from any of the authorities		0.2

		referenced in this report. Except for the ATSDR chronic inhalation MRL (<u>0.2-mg/m³</u>), the USEPA and other authorities referenced in this report have not issued an RfC-like value for 1,2,4-trichlorobenzene.		
1,1,2-Trichloroethane	79005	USEPA has designated 1,1,2-trichloroethane a class C: Possible human carcinogen, based on observed hepatocellular carcinomas and pheochromocytomas in one strain of mice. Carcinogenicity was not shown in rats; However, 1,1,2-trichloroethane is structurally related to 1,2-dichloroethane, a probable human carcinogen. The IRIS inhalation 1E ⁻⁵ cancer risk level concentration is <u>6E⁻⁴-mg/m³</u> . No RfC-like or RfD-like criteria have been published for 1,1,2-trichloroethane by any of the authorities referenced in this report; therefore, the ACGIH TLV-TWA for workplace exposures was used to develop an RfC estimate. It was divided by a factor range, to allow for continuous exposure to the general population. The resulting range was from 6.5E ⁻⁵ to 6.8 with an average of <u>2.5E⁻³-mg/m³</u> .	6E ⁻⁴	2.5E ⁻³
Trichloroethylene	79016	No carcinogenicity assessment of trichloroethylene is available from the USEPA; However, IARC lists it as a Group 2A chemical (a probable human carcinogen) based on limited evidence in humans but sufficient evidence in animals. The OEHHA issued an inhalation URE of 2E ⁻⁶ /μg/m ³ , which is equivalent to a 1E ⁻⁵ excess cancer risk level concentration of <u>5E⁻³-mg/m³</u> . USEPA has not published an RfC; However, the OEHHA chronic inhalation REL is <u>0.6-mg/m³</u> . The OEHHA values were used for toxicity-weighting the emission inventory.	5E ⁻³	0.6
2,4,5-Trichlorophenol	95954	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
2,4,6-Trichlorophenol	88062	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Triethylamine	121448	No carcinogenicity assessment of triethylamine is available from any of the authorities referenced in this report. The IRIS RfC is <u>7E⁻³-mg/m³</u> .		7E ⁻³
Trifluralin	1582098	The USEPA has designated trifluralin a group C: Possible human carcinogen, based on evidence in one study of induction of urinary tract tumors (renal pelvis carcinomas and urinary bladder papillomas) and thyroid tumors (adenomas/carcinomas combined) in F344 rats. Further, trifluralin is structurally similar to ethalfluralin, a carcinogen in the rat. There is no direct evidence of trifluralin carcinogenicity in humans. The USEPA drinking water 1E ⁻⁵ excess cancer risk exposure concentration is 50-μg/L. In an average adult, drinking 2L/day, the 1E ⁻⁵ excess cancer risk level is equivalent to 100-μg/day or 0.005-mg/20-m ³ inhaled air each day. Allowing for inter-day variability in average exposure concentrations with a factor of 0.2 yields <u>0.001-mg/m³</u> . USEPA and other authorities referenced in this report have not established RfC-like or RfD-like criteria for trifluralin.	0.001	
2,2,4-Trimethylpentane	540841	Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Vinyl Acetate	108054	No carcinogenicity assessment of vinyl acetate is available from any of the authorities referenced in this report. The RfC and REL are both <u>0.2-mg/m³</u> .		0.2
Vinyl Bromide	593602	No carcinogenicity assessment of vinyl bromide is available from any of the authorities referenced in this report. The RfC is <u>3E⁻³-mg/m³</u> .		3E ⁻³

Vinyl Chloride	75014	The USEPA has designated vinyl chloride (VC) a class A: Known human carcinogen, based on (1) consistent epidemiological evidence of a causal association between occupational exposure to VC via inhalation and the development of angiosarcoma, an extremely rare tumor; (2) consistent evidence of carcinogenicity in rats, mice, and hamsters by both the oral and inhalation routes; (3) mutagenicity and DNA adduct formation by VC and its metabolites in numerous in vivo and in vitro test systems; and (4) efficient VC absorption via all routes of exposure tested, followed by rapid distribution throughout the body. In light of the very high percentage of angiosarcomas worldwide that are associated with VC exposure, the evidence for VC carcinogenicity is considered strong. The IRIS inhalation $1E^{-5}$ cancer risk level concentration for continuous lifetime exposure during adulthood is 0.0023-mg/m^3 . For continuous lifetime exposure from birth the E-5 cancer risk concentration level is $1.15E^{-3}\text{-mg/m}^3$. The IRIS RfC is 0.1-mg/m^3 .	$1.15E^{-3}$	0.1
Vinylidene Chloride (1,1-Dichloroethylene)	75354	The USEPA evaluation of vinylidene chloride (1,1-dichloroethylene or 1,1-DCE) shows suggestive evidence of human carcinogenicity by the inhalation route of exposure. However USEPA concluded that the weight of evidence is not sufficient to justify deriving an inhalation unit risk. No quantitative estimate of carcinogenic risk from inhalation exposure has been developed the other the authorities referenced in this report either. No non-cancer inhalation benchmarks are available from any of the referenced authorities; therefore, the ATSDR chronic oral MRL (0.009-mg/kg/day) was used to develop an RfC estimate for toxicity-weighting the emission inventory. The equivalent daily inhalation exposure rate is $3.2E^{-2}\text{-mg/m}^3/\text{d}$ for an average 70-kg adult, inhaling $20\text{-m}^3/\text{d}$. Applying a factor of 0.2, to account for variations in the average daily concentration, yields $6.3E^{-3}\text{-mg/m}^3$ as an RfC estimate.		$6.3E^{-3}$
Xylenes (Isomers And Mixture)	1330207	The USEPA has not given xylenes (isomers and mixture) a carcinogenicity WOE class designation: Stating there is inadequate data for determination of carcinogenicity. No quantitative estimate of carcinogenic risk has been developed by the other the authorities referenced in this report either. The IRIS RfC is 0.1-mg/m^3 .		0.1
O-Xylene	95476	The USEPA has not given o-xylene a class designation: stating that there is inadequate data for determination of carcinogenicity. No quantitative estimate of carcinogenic risk has been developed by the other the authorities referenced in this report either. The IRIS RfC is 0.1-mg/m^3 .		0.1
M-Xylene	108383	Not specifically reported in the emissions inventory.		
P-Xylene	106423	Not specifically reported in the emissions inventory.		
Antimony Compounds		"Antimony compounds" are designated as HAPs. The USEPA and the other authorities referenced in this report have not established a carcinogenic potency estimate. OEHHA lists the only non-cancer criterion: the REL for antimony compounds is $2E^{-4}\text{-mg/m}^3$		$2E^{-4}$

Antimony	7440360	In addition to "antimony compounds" the emission inventory reports quantities of "antimony". Due to a lack of data, USEPA has not published inhalation carcinogenicity potency or RfC assessments for "antimony"; However, they have issued an RfD of $4E^{-4}$ -mg/kg-day, which was used to estimate a daily RfC for a 70-kg adult, breathing $20\text{-m}^3/\text{d}$. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded $2.8E^{-4}$ -mg/m ³ as an RfC estimate. For this report, the OEHHA REL for antimony compounds ($2E^{-4}$ -mg/m ³) was used in preference to the RfD-derived RfC estimate because most antimony is in compound rather than in elemental form in atmospheric emissions, and because the OEHHA REL for antimony compounds is similar to the RfD-derived RfC.			$2E^{-4}$
Antimony Oxide	1309644	In addition to "antimony compounds" the emission inventory reports quantities of "antimony oxide" which is synonymous with antimony trioxide. Due to a lack of data, USEPA has not published a carcinogenicity assessment for antimony [tri]oxide. Both the IRIS RfC and OEHHA REL are $2E^{-4}$ -mg/m ³ .			$2E^{-4}$
Antimony Trichloride	10025919	In addition to "antimony compounds" the emissions inventory reports quantities of "antimony trichloride". Due to a lack of data USEPA, has not published carcinogenicity, RfC, or RfD assessments for antimony trichloride. No toxicity criteria were available from the other referenced sources either.			
Arsenic Compounds (Inorganic Including Arsine)		The USEPA has designated inorganic arsenic a class A: Known human carcinogen, based on sufficient evidence from human data. Increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic. The IRIS inhalation exposure $1E^{-5}$ excess cancer risk level of <u>inorganic arsenic is $2E^{-6}$ mg/m³</u> . USEPA has not assessed non-cancer effects of inhaled inorganic arsenic. The IRIS oral RfD for inorganic arsenic is $3E^{-4}$ mg/kg/day, which is equivalent to $0.00105\text{-mg}/\text{m}^3/\text{day}$. Accounting for variations in this average daily level by applying a factor of 0.2 yields $2.1E^{-4}$ -mg/m ³ as the RfD-based RfC estimate for inorganic arsenic. No assessment or quantitative cancer risk estimate has been published for arsine. The IRIS <u>RfC for arsine is $5E^{-5}$ mg/m³</u> .	$2E^{-6}$	$2.1E^{-4}$ $5E^{-5}$	
Arsenic, Inorganic	7440382	The IRIS $1E^{-5}$ excess cancer risk level of inorganic arsenic exposure is <u>$2E^{-6}$-mg/m³</u> . The IRIS oral RfD for inorganic arsenic is $3E^{-4}$ mg/kg/day, which is equivalent to $0.00105\text{-mg}/\text{m}^3/\text{day}$. Accounting for variations in this average daily level by applying a factor of 0.2 yields $2.1E^{-4}$ -mg/m ³ as the RfD-based RfC estimate. USEPA has not assessed non-cancer effects of inorganic arsenic under IRIS. See "Arsenic Compounds (inorganic including arsine)" in this table above.	$2E^{-6}$		$2.1E^{-4}$
Arsine	7784421	No assessment or quantitative cancer risk estimate has been published for arsine. The IRIS RfC for arsine is <u>$5E^{-5}$-mg/m³</u> . See "Arsenic Compounds (inorganic including arsine)" in this table above.			$5E^{-5}$

Beryllium Compounds	7440417	USEPA has designated beryllium a B1: Probable human carcinogen, based on the limited evidence of airborne beryllium carcinogenicity in humans (lung cancer) and sufficient evidence of carcinogenicity in animals. The E-5 excess cancer risk level listed in IRIS is $4E^{-6}$ -mg/m ³ . The IRIS RfC is $2E^{-5}$ -mg/m ³ .	$4E^{-6}$	$2E^{-5}$
Cadmium Compounds		USEPA classifies cadmium as a B1: Probable human carcinogen, based on studies of various cadmium compounds. Limited evidence from occupational epidemiological studies is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection of cadmium. Seven studies in rats and mice, wherein cadmium salts (acetate, sulfate, chloride) were administered orally, have shown no evidence of carcinogenic response. The IRIS inhalation URE ($1.8E^{-3}$ /μg/m ³) is equivalent to a $1E^{-5}$ cancer risk level concentration of $5.6E^{-6}$ -mg/m ³ . USEPA has not published an RfC; However, the OEHHA chronic REL for cadmium and its compounds is $2E^{-5}$ -mg/m ³ .	$5.6E^{-6}$	$2E^{-5}$
Chromium Compounds		Hexavalent chromium - Cr(VI) - is classified as a group A: Known human carcinogen. Its IRIS cancer URE ($1.2E^{-2}$ /μg/m ³) is equivalent to $8.3E^{-7}$ -mg/m ³ at the $1E^{-5}$ excess risk level exposure. IRIS states “Hexavalent chromium is known to be carcinogenic in humans by the inhalation route of exposure. Results of occupational epidemiological studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both Cr(III) and Cr(VI) compounds. Because only Cr(VI) has been found to be carcinogenic in animal studies, however, it was concluded that only Cr(VI) should be classified as a human carcinogen. Animal data are consistent with the human carcinogenicity data on hexavalent chromium. Hexavalent chromium compounds are carcinogenic in animal bioassays, producing the following tumor types: intramuscular injection site tumors in rats and mice, intrapleural implant site tumors for various Cr(VI) compounds in rats, intrabronchial implantation site tumors for various Cr(VI) compounds in rats, and subcutaneous injection site sarcomas in rats. <i>In vitro</i> data are suggestive of a potential mode of action for hexavalent chromium carcinogenesis. Hexavalent chromium carcinogenesis may result from the formation of mutagenic oxidativite DNA lesions following intracellular reduction to the trivalent form. Cr(VI) readily passes through cell membranes and is rapidly reduced intracellularly to generate reactive Cr(V) and Cr(IV) intermediates and reactive oxygen species. A number of potentially mutagenic DNA lesions are formed during the reduction of Cr(VI). Hexavalent chromium is mutagenic in bacterial assays, yeasts, and V79 cells, and Cr(VI) compounds decrease the fidelity of DNA synthesis <i>in vitro</i> and produce unscheduled DNA synthesis as a consequence of DNA damage. Chromate has been shown to transform both primary cells and cell lines.” The IRIS RfC for chromic acid mists and dissolved Cr(VI) aerosols: is $8E^{-6}$ -mg/m ³ . The IRIS RfC for Cr(VI) particulates is $1E^{-4}$ -mg/m ³ . In NATA, USEPA used the IRIS RfC for particulate hexavalent chromium in preference to the RfC for chromic acid mists and dissolved	$8.3E^{-7}$	

aerosols. As in NATA, both the URE and the RfC for hexavalent chromium were adjusted to reflect an assumption that 34% of all atmospheric chromium is hexavalent: The remaining 66% assumed to be trivalent.

USEPA has designated trivalent chromium – Cr(III) - a group D chemical: Not classifiable as to its human carcinogenicity due to insufficient data. None of the other authorities referenced in this report have published carcinogenicity assessments of Cr(III) either. The ACGIH TWA-TLV was used to estimate an RfC for Cr(III) (see the table entry below).

Chromium VI	18540299	The USEPA designated Cr(VI) as a class A: known human carcinogen. The $1E^{-5}$ excess cancer risk level concentration is listed as $8E^{-7}$ -mg/m ³ in IRIS. The IRIS RfC for Cr(VI) particulates is $1E^{-4}$ -mg/m ³ . For chromic acid mists and dissolved Cr(VI) aerosols, the RfC is $8E^{-6}$ -mg/m ³ . See the table entry “Chromium Compounds” above.	$8E^{-7}$	$1E^{-4}$
Chromium III, insoluble salts	16065831	USEPA designated Cr(III) a group D chemical: Not classifiable as to human carcinogenicity. None of the authorities referenced in this report have developed quantitative carcinogenic potency estimates or RfC-like values for Cr(III). The ACGIH TLV-TWA for workplace exposures was used by applying a factor range to account for continuous exposure to the general population. The resulting RfC estimate ranges from $6E^{-7}$ to $6.3E^{-2}$ with an average of $2.3E^{-5}$ -mg/m ³ .		$2.3E^{-5}$
Chromium Compounds		For chromium compounds, the IRIS RfC for particulate hexavalent chromium was used in preference to the RfC for chromic acid mists and dissolved aerosols. Both the RfC and URE for hexavalent chromium were adjusted to reflect an assumption that 34% of all atmospheric chromium is hexavalent. In some cases, specific chromium compounds reported in the emissions inventory. These were toxicity-weighted using the best available toxic potency criteria. See the table entry “Chromium Compounds” above and the entries for individual chromium salts below.		
Calcium Chromate	13765190	Calcium chromate toxicity has not been specifically assessed by USEPA. The OEHHA chronic REL is $2E^{-4}$ -mg/m ³ . See the table entry “Chromium Compounds” above.		$2E^{-4}$
Strontium Chromate	7789062	Strontium chromate toxicity has not been specifically assessed by USEPA. The OEHHA chronic REL is $2E^{-4}$ -mg/m ³ . See the table entry “Chromium Compounds” above.		$2E^{-4}$
Zinc Chromate	13530659	Zinc chromate toxicity has not been specifically assessed by USEPA or any of the authorities referenced in this report. See the table entry “Chromium Compounds”.		
Cobalt Compounds	7440484	Cancer potency values have not been published for cobalt compounds by any of the authorities referenced in this report. Inhalation may cause respiratory effects such as irritation, wheezing, asthma, pneumonia, and fibrosis. Cardiac effects, congestion of the liver, kidneys, conjunctiva, and immunological effects have also been reported. USEPA has not published an RfC; However, the ATSDR draft chronic duration inhalation MRL ($1E^{-4}$ -mg/m ³) was used for toxicity-weighting the emission inventory.		$1E^{-4}$
Coke Oven Emissions		There are no known coke oven emissions in Washington		

Cyanide Compounds (1)		No carcinogenicity assessment of cyanide compounds is available from any of the authorities referenced in this report. USEPA has not published an RfC; therefore, the REL (<u>9E-3-mg/m³</u>) was used to toxicity-weight the emission inventory.		9E ⁻³
Hydrogen Cyanide	74908	No carcinogenicity assessment of hydrogen cyanide is available from any of the authorities referenced in this report. The IRIS RfC is <u>3E-3-mg/m³</u> .		3E ⁻³
Diesel Particulate Matter		The USEPA's diesel assessment report (USEPA,., 2002. <i>Health assessment document for diesel engine exhaust</i> . Washington, DC, National Center for Environmental Assessment) concluded that long-term exposure is likely a lung cancer hazard. It also showed emerging evidence that it exacerbates existing allergies and asthma symptoms. The USEPA ORD states that diesel exhaust is a likely human carcinogen; However, USEPA is presently unable to assign a carcinogenic potency. Nonetheless, they have stated the possible range of upper-bound risk is 10 ⁻³ to 10 ⁻⁵ per µg/m ³ lifetime exposure. The CARB Scientific Review Panel states the unit risk "reasonable estimate" as 3E ⁻⁴ /µg/m ³ in their <i>Particulate Matter from Diesel-Fueled Engines</i> review. They stated a range of UREs of 1.3E ⁻⁴ to 2.4E ⁻³ per µg/m ³ in the TAC document. Converting their "reasonable" URE to an equivalent 1E ⁻⁵ excess cancer risk level exposure yields <u>3.3E⁻⁵-mg/m³</u> . In addition to its carcinogenicity, DPM contributes to PM _{2.5} levels and has a demonstrated potential to induce non-cancer health impairments. The OEHHA lists the REL as <u>0.005-mg/m³</u> .	3.3E ⁻⁵	0.005
Glycol ethers (2)		The USEPA and other authorities referenced in this report have not assessed any glycol ethers on the basis carcinogenicity. Several chemicals in the glycol ether category have chronic RfCs ranging from 0.02 to 13 mg/m ³ . The most conservative (<u>0.02-mg/m³</u>) was used for toxicity-weighting of the glycol ethers emission inventory.		0.02
Ethylene Glycol Methyl Ether	109864	Ethylene glycol methyl ether (EGME) is the only member of the glycol ethers group specifically reported in the emissions inventory. None of the authorities referenced in this report have assessed the carcinogenicity of EGME. Its IRIS RfC is <u>0.02-mg/m³</u> .		0.02
Lead Compounds		The USEPA has established enforceable air and water quality criteria for 8.3E ⁻⁴ lead and has classified lead and inorganic lead compounds as B2: probable human carcinogens. They have not established a quantitative estimate of cancer risk, RfC or RfD. The OEHHA URE, when converted to the equivalent 1E ⁻⁵ risk level concentration is <u>8.3E⁻⁴-mg/m³</u> . The federal air quality criterion is <u>0.0015-mg/m³</u> . The OEHHA cancer risk value and the federal air quality criterion were used to weight the emission inventory.		0.0015
Manganese & Manganese Compounds		The USEPA has classified manganese as a class D chemical: Not classifiable as to human carcinogenicity. None of the other authorities have published cancer risk assessments of manganese. The IRIS RfC for manganese (and manganese compounds) is <u>5E⁻⁵-mg/m³</u> .		5E ⁻⁵

Mercury & Compounds	7439976 and others	The USEPA has designated elemental mercury as class D: Not classifiable as to human carcinogenicity, due to insufficient data. The RfC for <u>elemental Hg</u> is $3E^{-4}$ -mg/m ³ . The OEHHA chronic inhalation REL is $9E^{-5}$ mg/m ³ . IRIS contains an assessment of methyl mercury. It is designated a class C: Possible human carcinogen, but with no quantitative estimate of carcinogenic potency. IRIS does not include an RfC for methyl mercury, but does have an RfD of $1E^{-4}$ mg/kg/day, which is equivalent to $3.5E^{-4}$ -mg/m ³ /day. Adjusting for inter-day concentration variation with a factor of 0.2 yields a long-term RfC estimate of $7E^{-5}$ -mg/m ³ for methyl mercury. IRIS also contains an assessment of mercury chloride. Mercury chloride is designated a class C: Possible human carcinogen but has no published a quantitative cancer risk estimate. IRIS does not include an RfC for mercury chloride, but does have an RfD of $3E^{-4}$ mg/kg/day, which is equivalent to 0.00105 -mg/m ³ /day. Adjusting for inter-day variation with a factor of 0.2 yields an RfC estimate of $2.1E^{-4}$ -mg/m ³ for mercury chloride.		$3E^{-4}$ $7E^{-5}$ $2.1E^{-4}$
Fine Mineral Fibers (3)		Asbestos (as previously noted in this table) is a known human carcinogen; further, the California OEHHA has determined that glass wool and ceramic fibers (of respirable size particles) are carcinogens; However, no mineral fiber emissions inventory was available, and concentration data were not obtained in this study.		
Nickel & Compounds	7440020	USEPA designates nickel refinery dust and nickel subsulfide as known (class A) human carcinogens based on increased risks of lung and nasal cancer in humans exposed to nickel refinery dust, most of which was believed to have been nickel subsulfide; increased tumor incidences in animals by several routes of administration in several animal species and strains; and positive results in genotoxicity assays. These forms of nickel are listed in IRIS as posing a $1E^{-5}$ excess cancer risk at a concentration of $4E^{-5}$ -mg/m ³ : similar to the OEHHA URE, which is equivalent to a $1E^{-5}$ risk level concentration of $3.9E^{-5}$ -mg/m ³ . USEPA classifies nickel carbonyl a probable (B2) human carcinogen based upon the observation of pulmonary carcinomas and malignant tumors at various sites in rats after inhalation or intravenous injection of nickel carbonyl. Nickel administered as nickel carbonyl binds to DNA. However USEPA concluded that these data are not sufficient to derive an inhalation unit risk. USEPA has not issued RfDs for these forms of nickel; however, the ATSDR chronic inhalation MRL for all forms of nickel is $2E^{-4}$ -mg/m ³ .	$4E^{-5}$	$2E^{-4}$
Polycyclic Organic Matter (4)		Polycyclic Organic Matter (POM) comprises a large diverse group of chemicals. The group is listed as a HAP. The OEHHA URE for total POM, when expressed as the equivalent $1E^{-5}$ risk level concentration, is $1.8E^{-4}$ -mg/m ³ . No RfC or similar non-cancer criterion for POM was available. Some members and subset mixtures of the POM group have published toxic potency values. In addition to total POM, the emissions inventory lists specific POM chemicals and subset mixtures separately from the larger POM group. In these cases, the chemicals were toxicity-weighted using the best available toxic potency criteria.		$1.8E^{-4}$
PAH, Total		POM includes a smaller group containing thousands of chemicals known as Polycyclic Aromatic Hydrocarbons (PAH). USEPA has not published a cancer potency estimate for total-PAH. The OEHHA URE for total-PAH, when expressed as the equivalent $1E^{-5}$ risk level concentration, is $1.8E^{-4}$ -mg/m ³ . No RfC or similar non-cancer criterion for total-PAH was		$1.8E^{-4}$

available.

Polycyclic Organic Matter as 7-PAH		The "Carcinogenic 7-PAHs" are a subset of PAHs that includes benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene, which are described separately below. The USEPA staff judgment (in the <i>Ranking and Selection of HAPs Under Section 112(k): Technical support Document</i>) provides a URE of $3.3E^{-4}/\mu\text{g}/\text{m}^3$ for mixtures of these 7-PAHs. When this is expressed as the equivalent $1E^{-5}$ excess cancer risk level concentration, it is <u>$3E^{-5}\text{-mg}/\text{m}^3$</u> . No RfC or similar non-cancer criterion for the 7-PAHs was available.	$3E^{-5}$
Benz[a]Anthracene	56553	USEPA has designated benz(a)anthracene as class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $1.1E^{-4}/\mu\text{g}/\text{m}^3$, which is equivalent to <u>$9.1E^{-5}\text{-mg}/\text{m}^3$</u> at the $1E^{-5}$ excess cancer risk level. No RfC or similar non-cancer criterion is currently available.	$9.1E^{-5}$
Benzo[a]Pyrene	50328	USEPA has designated benzo(a)pyrene as class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $0.0011/\mu\text{g}/\text{m}^3$, which is equivalent to <u>$9.1E^{-6}\text{-mg}/\text{m}^3$</u> at the $1E^{-5}$ excess cancer risk level. No RfC or similar non-cancer criterion is currently available.	$9.1E^{-6}$
Benzo[b] Fluoranthene	205992	USEPA has designated benzo(b)fluoranthene as class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $0.00011/\mu\text{g}/\text{m}^3$, which is equivalent to <u>$9.1E^{-5}\text{-mg}/\text{m}^3$</u> at the $1E^{-5}$ excess cancer risk level. No RfC or similar non-cancer criterion is currently available.	$9.1E^{-5}$
Benzo[k] Fluoranthene	207089	USEPA has designated benzo(k)fluoranthene as class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $0.00011/\mu\text{g}/\text{m}^3$, which is equivalent to <u>$9.1E^{-5}\text{-mg}/\text{m}^3$</u> at the $1E^{-5}$ excess cancer risk level. No RfC or similar non-cancer criterion is currently available.	$9.1E^{-5}$
Benzofluoranthenes		In some cases, the emission inventory reported benzo[b]fluoranthene and benzo[k]fluoranthene together as "Benzofluoranthenes" or "Benzo[b+k]Fluoranthene" In this report, these mixtures were assumed to have the same toxic potency as either benzo[b]fluoranthene or benzo[k]fluoranthene alone.	
Chrysene	218019	USEPA has designated chrysene as class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $0.000011/\mu\text{g}/\text{m}^3$, which is equivalent to <u>$9.1E^{-4}\text{-mg}/\text{m}^3$</u> at the $1E^{-5}$ excess cancer risk level. No RfC or similar non-cancer criterion is currently available for chrysene.	$9.1E^{-4}$

Dibenzo[a,h] Anthracene	53703	USEPA has designated dibenzo[a,h]anthracene [synonymous with dibenz(a,h)anthracene] as a class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $0.0012/\mu\text{g}/\text{m}^3$, which is equivalent to $8.3\text{E}^{-6}\text{-mg}/\text{m}^3$ at the 1E^{-5} excess cancer risk level. No RfC or similar non-cancer criterion is currently available for dibenz[a,h]anthracene.	8.3E^{-6}
Indeno[1,2,3-cd]Pyrene	193395	USEPA has designated indeno[1,2,3-cd]pyrene as class B2: A probable human carcinogen, based on sufficient evidence of carcinogenicity in animals but lack of data in humans. USEPA has not issued a quantitative carcinogenic potency estimate. OEHHA lists an inhalation URE of $0.00011/\mu\text{g}/\text{m}^3$, which is equivalent to $9.1\text{E}^{-5}\text{-mg}/\text{m}^3$ at the 1E^{-5} excess cancer risk level. No RfC or similar non-cancer criterion is currently available for indeno[1,2,3-cd]pyrene.	9.1E^{-5}
Polycyclic Organic Matter as 16-PAH		In some cases, the emissions inventory reports quantities of PAHs as "16-PAH". The 16-PAHs are a subset of PAHs that includes the "carcinogenic 7-PAHs" and acenaphthene, acenaphthylene, anthracene, benzo(ghi)perylene, fluoranthene, fluorene, naphthalene, phenanthrene, and pyrene. EPA method 610 is used to measure 16-PAH. The NATA provided estimates of toxic equivalence to BaP of 16-PAH emitted from different sources. For 16-PAH from residential wood burning, USEPA estimated 3.57% BAP equivalents; for aluminum smelting, 5.14%; for wildfires, 6.70%; and for utility emissions, 6.70%. (See <i>Appendix H, Estimating Carcinogenic Potency for Mixtures of Polycyclic Organic Matter for the 1996 National-Scale Assessment</i> . Available at http://www.epa.gov/ttn/atw/sab/appendix-h.pdf). Based on these, the average BaPeq/16-PAH is 5.53%; thus the average 16-PAH URE is $6.1\text{E}^{-5}/\mu\text{g}/\text{m}^3$, which is equivalent to $1.6\text{E}^{-4}\text{-mg}/\text{m}^3$ at the 1E^{-5} excess cancer risk level.	1.6E^{-4}
Acenaphthene	83329	Due to a lack of data USEPA, has not published carcinogenicity or inhalation RfC assessments. The USEPA IRIS oral chronic RfD is $6\text{E}^{-2}\text{-mg}/\text{kg}/\text{day}$. For this report, the RfD was used to estimate a daily RfC for a 70-kg adult, breathing $20\text{ m}^3/\text{d}$. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded $0.042\text{-mg}/\text{m}^3$ as an RfC estimate.	0.042
Acenaphthylene	208968	Due to a lack of data USEPA, and the other authorities referenced in this report have not published carcinogenicity, RfC-like, or RfD-like assessments for acenaphthylene.	
Anthracene	120127	Due to a lack of data USEPA, has not published carcinogenicity or RfC assessments for anthracene; However, they issued an RfD of $3\text{E}^{-1}\text{-mg}/\text{kg}/\text{day}$. For this report, the RfD was used to estimate a daily RfC for a 70-kg adult, breathing $20\text{-m}^3/\text{d}$. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded $0.21\text{-mg}/\text{m}^3$ as an RfC estimate.	0.21
Benzo[g,h,i] Perylene	191242	Due to a lack of data USEPA, has not published carcinogenicity, RfC, or RfD assessments for benzo[g,h,i]perylene. No toxicity criteria were available from the other referenced sources either.	
Fluoranthene	206440	Due to a lack of data USEPA, has not published carcinogenicity or RfC assessments for fluoranthene; However they issued an RfD of $4\text{E}^{-2}\text{-mg}/\text{kg}/\text{day}$. For this report, the RfD was used to estimate a daily RfC for a 70-kg adult, breathing $20\text{-m}^3/\text{d}$. Then a factor of 0.2 was used to	0.028

		account for variations in the average daily concentration. This yielded <u>0.028-mg/m³</u> as an RfC estimate.		
Fluorene	86737	Due to a lack of data USEPA, has not published carcinogenicity or RfC assessments for fluorene; However, they issued an RfD of 4E ⁻² -mg/kg/day. For this report, the RfD was used to estimate a daily RfC for a 70-kg adult, breathing 20-m ³ /d. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded <u>0.028-mg/m³</u> as an RfC estimate.		0.028
Naphthalene	91203	USEPA designated naphthalene as a Group C: Possible human carcinogen, based on the inadequate data of carcinogenicity in humans exposed via the oral and inhalation routes, and limited evidence of carcinogenicity in animals via inhalation. No quantitative cancer potency estimates have been published by the referenced sources. The IRIS RfC is <u>3E⁻³-mg/m³</u>		3E ⁻³
Phenanthrene	85018	Due to a lack of data USEPA, has not published carcinogenicity, RfC, or RfD assessments for phenanthrene. No toxicity criteria were available from other sources either.		
Pyrene	129000	Due to a lack of data USEPA, has not published carcinogenicity or RfC assessments for fluorene; However, they have issued an RfD of 0.03-mg/kg/day. For this report, the RfD was used to estimate a daily RfC for a 70-kg adult, breathing 20-m ³ /d. Then a factor of 0.2 was used to account for variations in the average daily concentration. This yielded <u>0.021-mg/m³</u> as an RfC estimate.		0.021
Radionuclides (Including Radon) (5)		Although listed as a HAP, not included in the emissions inventory. Ambient concentration data were not available either.		
Selenium & Compounds	7782492	USEPA designates selenium and compounds as class D chemicals: Not classifiable as to carcinogenicity, based on inadequate human data and inadequate evidence of carcinogenicity in animals. The evidence for various selenium compounds in animal and mutagenicity studies is conflicting; However, USEPA notes that evidence for selenium sulfide is sufficient for a B2 (probable human carcinogen) classification, but they did not provide a quantitative risk estimate. The California Air Pollution Control Officers Association (CAPCOA) recommended a selenium sulfide preliminary cancer URE of 1.4E ⁻⁴ /μg/m ³ , which is equivalent to <u>7.14E⁻⁵-mg/m³</u> at the 1E ⁻⁵ excess cancer risk level. The USEPA has not established an RfC for selenium and compounds; However, the CAPCOA Revised 1992 Risk Assessment Guidelines for selenium lists a chronic non-cancer REL of <u>5E⁻⁴-mg/m³</u> for selenium compounds, based on respiratory irritation.	7.14E ⁻⁵	5E ⁻⁴
Butyl Cellosolve	111762	USEPA has designated butyl cellosolve (synonymous with 2-butoxyethanol or ethylene glycol monobutyl ether or EGBE) a class C chemical: Possible human carcinogen, because of the uncertain relevance of tumor increases observed in rats and mice to humans, the fact that EGBE is generally negative in genotoxicity tests, and the lack of human data to support the findings in rodents. No carcinogenicity assessment is available from any of the authorities referenced in this report. The IRIS RfC is <u>13-mg/m³</u> .		13
Wood smoke		None of the authorities cited in this report have established toxicity criteria for wood smoke; however, Lewtas (1988) proposed a cancer		1.0E ⁻³ and

As noted in the CFR: For all listings above which contain the word "compounds" and for glycol ethers, the following applies: Unless otherwise specified, these listings are defined as including any unique chemical substance that contains the named chemical (i.e., antimony, arsenic, etc.) as part of that chemical's structure.

1 $X'CN$ where $X = H'$ or any other group where a formal dissociation may occur. For example KCN or $Ca(CN)_2$

2 Includes mono- and di- ethers of ethylene glycol, diethylene glycol, and triethylene glycol $R-(OCH_2CH_2)_n-OR'$ where

$n = 1, 2, \text{ or } 3$

$R = \text{Alkyl or aryl groups}$

$R' = R, H, \text{ or groups which, when removed, yield glycol ethers with the structure: } R-(OCH_2CH_2)_n-OH.$ Polymers are excluded from the glycol category. (See Modification)

3 Includes mineral fiber emissions from facilities manufacturing or processing glass, rock, or slag fibers (or other mineral derived fibers) of average diameter 1 micrometer or less.

4 Includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100°C .

5 A type of atom which spontaneously undergoes radioactive decay.

6 Lewtas, J. 1988. Genotoxicity of complex mixtures: Strategies for the identification and comparative assessment of airborne mutagens and carcinogens from combustion sources. *Fund. & Appl. Tox.* 10:571-589

7 Anderson, N. *Final Report: Risk assessment document for residential wood combustion emissions.* Maine Department of Health Services, Environmental Toxicology Program, Environmental Health Unit, Division of Diseases Control, Bureau of Health. October 1989.