PUGET SOUND
Clean Air Agency

# Final Report: Puget Sound Air Toxics Evaluation

October 2003

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This report was released as a draft in 2002 and we received comments on the draft from a variety of reviewers. These reviewers include Dr. Sally Liu from the University of Washington, Dr. Jane Koenig from the University of Washington, Dr. David Solet from the Metro King County Public Health Department, Dr. Matt Kadlec from the Washington Department of Ecology, Dr. Harriet Ammann from the Washington Department of Health, Ms. Julie Wroble from the US Environmental Protection Agency, and Dr. Kay Jones from Zephyr Consulting Company. We also received comments from Dr. Houck of Omni Consulting on behalf of the Hearth Products Association.

The authors addressed many of these comments in this final report. We would like to thank our reviewers, and appreciate the time they took to provide valuable feedback on our draft.

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## Executive Summary

The Puget Sound Clean Air Agency conducted this screening study to identify chemicals and emission sources that pose the greatest potential health risks to citizens in the Puget Sound region. We also hope to better characterize the potential health risks to our three million residents from a group of air contaminants referred to as air toxics. This study is intended to assist the Agency in focusing resources on those emissions and sources that may pose the highest risks. The results should also help improve air toxics regulations and voluntary programs. The estimates of cancer and non-cancer health effects should not be viewed as actual cancer or non-cancer cases resulting from air pollution but as an estimate of relative impact of the evaluated toxic-air pollutants so the Agency can prioritize its efforts to reduce air pollution.

### *Defining Air Toxics*

Air toxics are different from the 6 traditional air pollutants or "criteria pollutants" that have been regulated by environmental regulatory agencies for a number of years. Our agency defines "air toxics" as a broad category of chemicals that covers over 400 air pollutants along with woodsmoke and diesel particles. Similarly, the United States Environmental Protection Agency (USEPA) commonly refers to "air toxics" as a synonym for the 189 hazardous air pollutants listed in the 1990 amendments to the federal Clean Air Act. Because resources are not available to evaluate every chemical, this study evaluates a short list of 17 to 30 air toxics. We hope to expand the list of toxics when more resources become available.

### *Persistent, Bioaccumulative Toxics (PBTs)*

Some persistent, bioaccumulative toxics (PBTs) such as mercury, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), cadmium, and arsenic were included in our study. However, we evaluate potential health risks only from the inhalation pathway, as the ingestion pathway was considered to be beyond the scope of this study.

### *Methods*

This study uses basic risk assessment concepts and models, such as toxicity and exposure assessment, to provide a general overview of the potential health impacts that could be due to air toxics. Because of limited resources, this report does not perform a comprehensive risk

assessment, which would include more detailed analyses and discussion of toxicity and exposure parameters, as well as a more in-depth risk characterization section. More comprehensive information on various details of this study can be found in the technical support documents referenced throughout this report.

***Toxicity***

The toxicity chapter includes dose-response information on the variety of air toxics evaluated in the Puget Sound region. The majority of this information is based on toxicity analyses performed by USEPA and included in their Integrated Risk Information System (IRIS). For some chemicals and mixtures, such as diesel particulate matter, chromium, and woodsmoke, we depart from recommended USEPA IRIS toxicity values. For example, for diesel particulate matter, we use the California Environmental Protection Agency's toxicity evaluation. Our rationale for this and other departures is described in the toxicity chapter.

***Exposure***

The toxicity values described above are combined with exposure assessment information to estimate both cancer and non-cancer potential health risks. We use results from three different exposure assessments to characterize air emissions and to estimate potential exposure concentrations for the residents of the Puget Sound area. These three exposure assessments include a monitoring study conducted in the greater Seattle/King County area, and two modeling assessments conducted as part of USEPA National-scale Air Toxics Assessment (NATA) in the four counties in the Puget Sound Clean Air Agency jurisdiction (King, Kitsap, Pierce, and Snohomish counties).

The monitoring study, which was conducted by the Washington State Department of Ecology in partnership with the Puget Sound Clean Air Agency and USEPA, sampled outdoor air at six different locations throughout the greater Seattle/King County area during 2000 and 2001. These six locations include areas near or in Beacon Hill, Georgetown, Lake Sammamish, Lake Forest Park, the Maple Leaf reservoir in north Seattle, and the city of SeaTac.

In addition to the monitoring study, we used exposure estimates from two models used by USEPA in their nationwide air toxics study entitled the National-scale Air Toxics Assessment (NATA). In this study, USEPA predicts outdoor air concentrations using the ASPEN model for 32 air toxics in counties across the country. We obtained the outdoor air concentrations for the four Puget Sound counties, compared them to monitored concentrations, and calculated potential health risks associated with those concentrations.

The third model used to predict exposure concentrations is also part of the NATA study. This model, entitled the Hazardous Air Pollutant Exposure Model4 (HAPEM4), predicts human exposures to the outdoor air pollutants by considering typical human behaviors and micro-environments where these outdoor pollutants might accumulate or dissipate. For example, this model uses average commute time estimates for a variety of individuals to estimate potential exposures to vehicle exhaust while riding in cars or waiting in traffic. Exposures such as these are combined for multiple activities and locations to estimate an average exposure concentration for each of the 32 air toxics for different population groups.

All exposure concentrations are based on annual averages or medians (the $50^{th}$ percentile), and residents are assumed to be exposed for 70 years, an average lifetime for an individual. We also assumed that these residents are healthy adults. Because of limited resources, we did not include exposure or toxicity adjustments specific to children, such as changes to body weight. Some health-protective assumptions (e.g., assuming a 70-year exposure period) are included in the toxicity estimates to protect sensitive people such as the elderly or diseased individuals. The health risk estimates are based on a combination of average and reasonably conservative or health-protective assumptions. *This is expected to lead to risk estimates that are reasonably high for the chemicals included in the analysis, but not worst case.*

***Results***

The primary health effect of concern from the chemicals evaluated in this study is cancer. More specifically, lung cancer is associated with both diesel soot and woodsmoke, although it is also associated with 1,3-butadiene, a mobile source-related contaminant. In addition to lung cancer, leukemia, nasal, and liver cancers are associated with chemicals that ranked high (e.g., benzene,

formaldehyde) in our study. The majority of the cancer risk estimated in our study is due to diesel soot. On average, diesel soot accounts for somewhere between 70% to 85% of the total cancer risk from air toxics in our area. Of the PBTs, arsenic is the only single compound to appear among the top ranking toxics, however, DPM and woodsmoke include numerous PAHs, so we conclude that these mixtures also contribute PBTs to the air in the Puget Sound region.

Our study found that the significant non-cancer health effects from air toxics in our area are primarily due to acrolein. This chemical is associated with upper respiratory irritation.

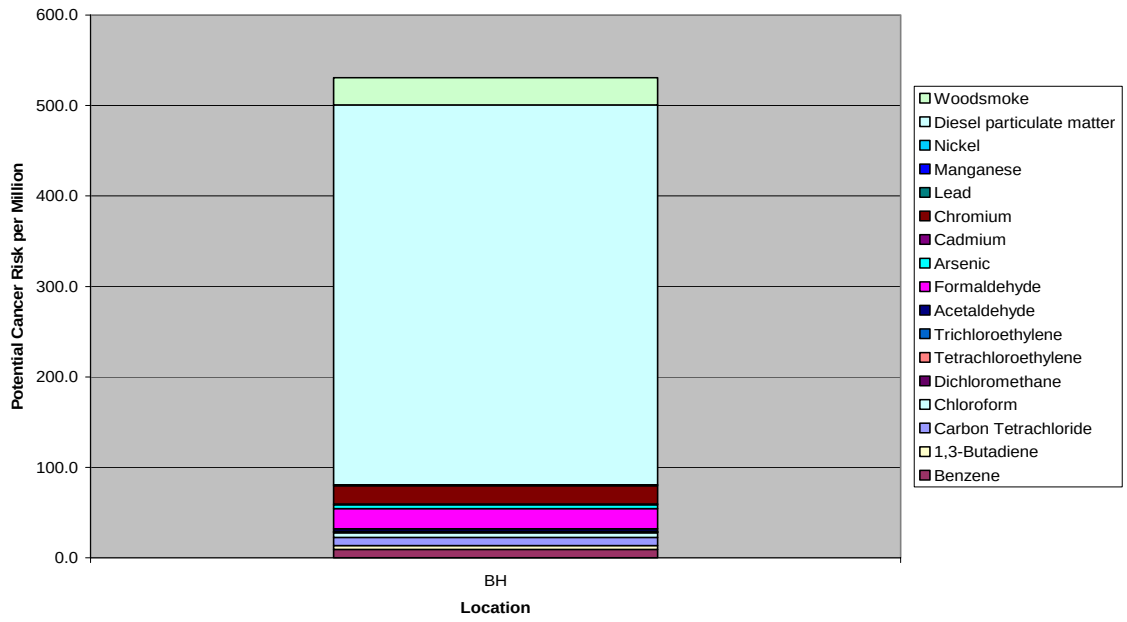
It is important to note, however, that our study does *not* include the serious non-cancer health effects associated with the particle fraction of 2 air toxics: diesel soot and woodsmoke. Non-cancer health effects associated with these particles have been extensively studied and documented in the scientific literature, and a full analysis is beyond the scope of this study.

***Potential Cancer Risks***

The average cancer risk estimates, even when human and pollutant movement/penetration are considered, are similar among the different methods of calculating exposure concentrations, and across different areas of the Puget Sound region. For example, average cancer risk estimates for King County alone range from approximately 400 to 700 in a million, based on 32 air toxics from the human exposure model and outdoor model data, respectively.

The average cancer risk estimates for the monitored data are approximately 550 in a million for the Beacon Hill area (see Figure ES-1). As described above, the monitoring study only looked at a total of 17 air toxics. The total cancer risks associated with the King County modeled estimates are higher because they include more chemicals, not because the estimates of each chemical are higher.

**Figure ES-1: Potential Cancer Risks at Beacon Hill including Diesel Particulate Matter and Woodsmoke**



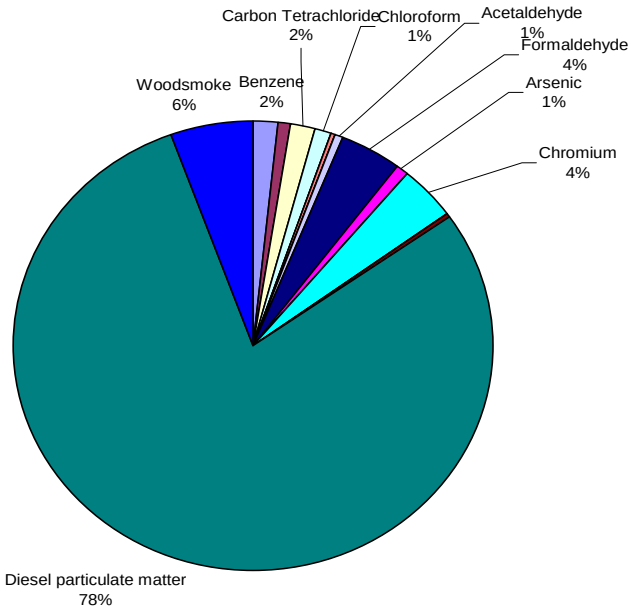

The average cancer risk estimates are also similar in the remaining three counties in the Puget Sound jurisdiction (Kitsap, Pierce, and Snohomish counties), although we do not have monitored information to confirm our findings. The estimated cancer risks range from 400 in a million for all air toxics included in the HAPEM4 model in Snohomish County, including diesel soot, to a high of 600 in a million as an average for 32 ASPEN-modeled ambient concentrations in King County, including diesel soot. All risk estimates reflect a 70-year exposure period. Upper 95$^{th}$ percentile risk estimates based on the modeled ambient concentrations are approximately 980 in a million for King County.

The air toxics that contribute most to the cancer risks are also consistent across the different methods of analysis. **The top toxics for all 3 methods include diesel soot, benzene, formaldehyde, and carbon tetrachloride**. **Woodsmoke** also contributes to the risk estimates based on the monitored data.

In addition, the percent contribution of the top air toxics is also very similar across the different methods of analysis. For example, at Beacon Hill, diesel soot accounts for over 75% of the potential cancer risks (see Figure ES-2) with another 10% or so coming from volatile organic compounds (VOCs) associated with mobile sources. The King County results from the outdoor

NATA model estimate diesel particulate matter at 86%, with other mobile-source-related chemicals at about 8%, and stationary-source-related chemicals at about 6%. Similarly, the NATA human exposure results indicate a diesel soot contribution of 86%, with other mobile-source-related chemicals at 7%, and stationary sources at about 4%. ***This indicates that mobile sources are likely to account for approximately 85% to 95% of the potential cancer risks among outdoor air toxics.***

Figure ES-2: Contributions to Potential Cancer Risk at Beacon Hill (2001)




The only emission source that ranks high in the monitoring data but not in the modeled data is woodsmoke. This is because woodsmoke emissions are estimated differently. The modeled concentrations associated with woodsmoke reflect very few chemicals in the woodsmoke mixture, while the concentrations based on monitored data reflect a greater number of chemicals present in woodsmoke.

### *Uncertainties*

The large number of assumptions necessary in our study reflects the amount of uncertainty and variability associated with the health risk estimates. It is possible that risk is underestimated because (1) not all air toxics are considered in this analysis, and (2) many chemicals have been shown to accumulate in indoor micro-environments, which could increase exposure. In addition,

potential cancer estimates will underestimate risk for those individuals living near large point sources or “hot spots”. Alternatively, risk may be underestimated or overestimated by assuming that the concentration at the monitor accurately reflects lifetime exposure to ambient pollutants. Obviously, chemical concentrations could increase or decrease throughout the lifetime exposure period.

It is important to note that this analysis does not evaluate indoor sources of air pollution (i.e., from paints, home furnishings, cleaning products, building materials, and other indoor sources). Uncertainties in the toxicity information could also serve to over- or underestimate potential risk estimates. These are only a few of the uncertainties associated with this study. A more detailed discussion can be found in Chapter 5.

In summary, we use screening risk estimates as a tool to focus Clean Air Agency attention on those compounds and mixtures that are likely to present the greatest risk of cancer and some non-cancer effects. Concentrations, and corresponding risks, were relatively consistent among areas measured and modeled throughout the Puget Sound region. Although some differences were apparent, overall it is clear that the sites and the region as a whole have similar emission sources of concern (e.g., diesel particulate matter, mobile-source-related VOCs, and probably woodsmoke).

Diesel soot ranks high in potential contributions to cancer risk, higher than other air toxics measured in this study. However, volatile organics associated with mobile sources, such as benzene and formaldehyde, contribute significantly to the potential cancer risks from air toxics. Diesel soot, benzene, 1,3-butadiene, and formaldehyde are classified as class A or B carcinogens under the USEPA cancer rating system. This indicates that USEPA is relatively confident that these chemicals probably cause cancer in humans. These chemicals should have high priority during development of an air toxics reduction program for the Puget Sound area. Finally, acrolein appears to present a potential non-cancer risk as well. As stated earlier, the non-cancer health effects associated with the particulate-matter-related combustion mixtures (e.g., woodsmoke and diesel soot) are not evaluated here, but present serious non-cancer health risks.

# Chapter 1: Introduction

## *1.1 Purpose*

The purpose of this study is to characterize air emissions and to identify those air toxics and sources that may pose the greatest risks to residents of the Puget Sound area. This analysis uses results from a monitoring study conducted in the greater Seattle/King County area and modeling studies conducted in the four counties in the Puget Sound Clean Air Agency jurisdiction (King, Kitsap, Pierce, and Snohomish counties). The United States Environmental Protection Agency (USEPA) performed the modeling in its National-scale Air Toxics Assessment (NATA) project to estimate potential cancer and non-cancer risks associated with the ambient air concentrations of those toxics. In addition, results from a human exposure study provide a general view of the potential exposures and health risks when average or typical human behaviors are considered.

The Puget Sound Clean Air Agency will use the results from this study to evaluate existing air toxics regulations, to focus on compounds of greatest concern, and to identify areas of potential improvement in its air toxics program. These results are intended to provide general direction to planners and managers. These results are *not* intended to provide exact estimates of potential health risk.

The estimates of cancer and non-cancer health effects should not be viewed as actual cancer or non-cancer cases resulting from air pollution, but as an estimate of relative impact of the toxic air pollutants evaluated in order to prioritize Agency efforts at reducing exposures. The estimates are based on a combination of average and reasonably conservative or health-protective assumptions. This is expected to lead to risk estimates that are reasonably high for the chemicals included in the analysis, but not the worst case scenario.

## *1.2 Methods*

Regulatory agencies typically employ risk-based approaches to evaluate potential health impacts from exposures to toxic chemicals. This study uses basic risk assessment concepts and models to provide a general overview of the potential air toxics problems that could be due to air toxics. However, we have not performed a comprehensive risk assessment, which would include more detailed discussions of toxicity and exposure parameters used to calculate risk estimates.

For the purposes of conducting the screening analysis, potential cancer risks are calculated using the following equation:

Cancer Risk = Exposure concentration x Toxicity

where: Exposure concentration = annual average ($\mu g/m^3$)
Toxicity = unit risk for carcinogens (cancer risk/1 $\mu g/m^3$)

Similarly, non-cancer risks are estimated by calculating a hazard index, using the following equation:

Hazard Index (HI) = Exposure concentration/Toxicity

where: Exposure concentration = annual average ($\mu g/m^3$)
Toxicity = reference concentration ($\mu g/m^3$)

Exposure concentrations used to calculate potential cancer and non-cancer health risks were obtained through three different methods. These methods are discussed generally below, and in more detail in Chapters 3 and 4 of this report.

Because resources were not available, a complete risk assessment was not conducted. However, the report includes the primary risk assessment components such as a toxicity or dose-response section, an exposure assessment section, and a risk characterization section. It includes a general discussion of the two major types of exposure models (ASPEN and HAPEM4, discussed later) used to calculate exposure concentrations. More comprehensive descriptions of these models were not included for two reasons. First, adequate resources were not available to the Agency, and second, these models are described and discussed extensively in technical support documents that accompany the NATA project.[1] However, general descriptions of the model

[1] USEPA. ***National-Scale Air Toxics Assessment for 1996***. Office of Air Quality Planning and Standards. EPA-453/R-01-003. January 2001.

assumptions are included when appropriate, and the supporting documentation is referenced accordingly throughout this document.

### *1.3 Exposure*

In this evaluation, three separate methods are used to provide exposure estimates. These include:

1. Monitored ambient concentrations
2. Modeled ambient concentrations
3. Modeled "human exposure concentrations" (where human activities and locations are considered in estimating exposures to air pollutants)

Results from each method of predicting exposure are presented and compared with toxicity values and evaluated for potential or relative risk.

### *1.4 Toxicity*

Although several different methods are used to estimate exposures, essentially one method is used to evaluate toxicity associated with airborne toxics. In most cases, USEPA-recommended toxicity factors are used as the basis for quantitative dose-response information. These values are usually obtained from the USEPA Integrated Risk Information System (IRIS) database. However, in some cases neither IRIS values nor USEPA values in the NATA project were available. In these instances, the alternative values were usually chosen from other sources. The basis for each toxicity factor and rationale for any adjustments are included in Chapter 2.

### *1.5 Conclusions and Recommendations*

In the last chapter, the results from the different methods used in the evaluation are compared. Discrepancies and similarities are discussed. In addition, the uncertainties and limitations of the evaluation and the impact on the results are described. Finally, recommendations for Agency priorities are presented.

## Chapter 2: Toxicity Estimates

Although several different methods are used to evaluate potential exposures, the same toxicity values are used for each of the analyses. Rather than describe toxicity in each section, toxicity estimates and the details associated with them are described in this section.

### *2.1 Separating Carcinogenic and Non-carcinogenic Impacts*

Toxicity estimates for carcinogens and non-carcinogens are derived through different processes and reflect fundamentally different concepts in toxicity. Toxicity values for non-cancer effects are based on the idea that a threshold exists for these health effects. USEPA believes that carcinogenic effects may not have thresholds, and that any exposure is associated with some corresponding (although very low) risk of disease. Physiological changes leading to cancer may occur over many years or decades.

Carcinogenic health effects are presented as a probability or risk of developing cancer. This can be viewed in two ways. First, the risk concept can be viewed as an additional cancer risk for each exposed individual. For example, a risk of one in a million could be added to the existing lifetime cancer risk of one in two to one in three (this excludes consideration of genetic or other susceptibilities) for most individuals.[2] USEPA also interprets risk estimates as potential cancer cases over the population of potentially exposed individuals. For example, a one in a million risk can also be viewed as one additional cancer case for every million people exposed to that concentration.[3,4]

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[2] Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Mariotto A, Fay MP, Feuer EJ, Edwards BK (eds). *SEER Cancer Statistics Review, 1975-2000*, National Cancer Institute. Bethesda, MD, http://seer.cancer.gov/csr/1975_2000, 2003. http://seer.cancer.gov/csr/1975_2000/results_merged/topic_lifetime_risk.pdf

[3] USEPA IRIS Glossary defines the unit risk value as **Unit Risk:** The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m$^3$ in air. The interpretation of unit risk would be as follows: if unit risk = 1.5 x 10$^{-6}$ µg/L, 1.5 excess tumors are expected to develop per 1,000,000 people if exposed daily for a lifetime to 1 µg of the chemical in 1 liter of drinking water.

[4] USEPA also defines "one in a million risk" in the NATA glossary (http://www.epa.gov/ttn/atw/nata/gloss1.html) as follows: **1 in a Million Cancer Risk:** A risk level of 1 in a million implies a likelihood that up to one person, 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. Note that this assessment looks at **lifetime** cancer risks, which should not be confused with or compared to **annual** cancer risk estimates. To compare an annual cancer risk estimate with the results in this assessment, multiply that annual estimate by a factor of 70 or alternatively divide the lifetime risk by a factor of 70. A 1 in a million lifetime risk to the U.S. public in 1996 was 250 cancer cases over a 70-year period.

In contrast to carcinogenic health effect evaluation, non-carcinogenic effects are presented as exceeding (or not exceeding) a particular guideline, referred to as a hazard index. The hazard index is a ratio of the estimated exposure concentration, divided by a concentration deemed to have no adverse effect from a lifetime exposure to that level. This non-carcinogen evaluation does not calculate a probability but instead determines whether a particular exposure is above or below a threshold above which there will be an adverse effect. Levels below the hazard index are deemed to be of no risk. Because of these differences, carcinogenic effects are evaluated separately from non-carcinogenic effects.

### *2.2 Carcinogenic Effects*

Potential carcinogenic effects are measured using unit risk factors. USEPA defines the unit risk factor (URF) as "a measure of the potential cancer risk of exposure to 1 microgram chemical per cubic meter of air over a 70-year period."[5] URFs are typically derived from animal laboratory studies, although human data from epidemiological or clinical studies can sometimes provide appropriate dose-response information. In addition, the URF is considered to be highly conservative or protective of health (it is based on the upper $95^{th}$ percentile of the potency slope). In other words, if we use URFs, it is unlikely that the potential cancer-risk values underestimate the true cancer risk associated with the specified exposure concentrations, and very likely overestimate the true risk.

In addition to the quantitative evaluation, USEPA also assigns each carcinogen a confidence rating based on the certainty associated with the supporting toxicological and health data. The values in this rating are A through E, with Group A being associated with the greatest certainty of evidence for causing cancer in humans and Group E having evidence that the chemical does not cause cancer in humans.

[5] See USEPA IRIS definition in footnote #2 above.

URFs used in this report are listed in Table 2-1 below. We used the same values as those in the NATA project. Most of the unit risk factors were obtained from the USEPA IRIS database, however if alternative sources are used, they are noted.[6]

It is important to note that most chemicals lack sufficient information to develop URFs. For example, adequate health information on which to base risk estimates is not available for the majority of chemicals used in commerce.[7] In addition, synergistic and/or antagonistic effects among the chemicals are not considered in these potency estimates. In other words, we do not know how the toxicity of these chemicals changes when administered in a mixture with other chemicals (except for woodsmoke and DPM which are discussed below). Finally, USEPA typically notes that the cancer risks associated with carcinogens could be as low as zero. Uncertainties associated with the risk estimates are discussed more fully in the last chapter of this document.

Specific URFs are available for two complex chemical mixtures, woodsmoke and diesel particulate matter, although they are not endorsed by USEPA. These two mixtures account for 46% of the total PM2.5 measured in Seattle, and could present potential cancer risk.[8] These values and the supporting documentation for each are also discussed below.

[6] USEPA. ***National-Scale Air Toxics Assessment for 1996***. Office of Air Quality Planning and Standards. EPA-453/R-01-003. January 2001.

[7] National Research Council. ***Toxicity Testing: Strategies to Determine Needs and Priorities***. Steering Committee on Identification of Toxic and Potentially Toxic Chemicals for Consideration by the National Toxicology Program. National Academy Press. Washington DC. 1984.

[8] Maykut N, J Lewtas, E Kim, T Larson. *Source Apportionment of PM2.5 at an urban IMPROVE site in Seattle, WA.* Manuscript accepted to Environmental Science and Technology, August 2003.

## Table 2-1: Unit Risk Factors and Cancer Ratings

| Chemical | Unit Risk Factor (risk /µg/m$^3$) | USEPA Cancer Rating | Reference |
|---|---|---|---|
| 1) Acrylonitrile | 6.8E-05 | B1 | IRIS |
| 2) Benzene | 7.80E-06 | A | USEPA IRIS file, downloaded 10/22/01 |
| 3) 1,3-Butadiene | 3.0E-05 | A | USEPA NATA[9]: EPA NCEA[10] |
| 4) Carbon tetrachloride | 1.50E-05 | B2 | USEPA IRIS file, downloaded 10/22/01 |
| 5) Chloroform | 2.30E-05 | B2 | USEPA IRIS file, downloaded 10/22/01 |
| 6) Dichloromethane | 4.70E-07 | B2 | USEPA IRIS file, downloaded 10/22/01 |
| 7) 1,3-Dichloropropene | 4.0E-06 | B2 | USEPA NATA: IRIS |
| 8) Diesel particulate matter (DPM) | 3.0E-04 | B2 | CALEPA/OEHHA |
| 9) Ethylene dibromide | 2.2E-04 | B2 | USEPA NATA: IRIS |
| 10) Ethylene dichloride | 2.6E-05 | B2 | USEPA NATA: IRIS |
| 11) Ethylene oxide | 8.8E-05 | B1 | CalEPA |
| 12) Hexachlorobenzene | 4.6E-04 | B2 | USEPA NATA: IRIS |
| 13) Hydrazine | 4.9E-03 | B2 | USEPA NATA: IRIS |
| 14) 7-PAHs | 2.0E-04 | B2 | USEPA NATA: OAQPS |
| 15) PCBs | 1.0E-04 | B2 | USEPA NATA: IRIS |
| 16) POM | 5.5E-04 | NA | USEPA NATA: OAQPS |
| 17) Propylene dichloride | 1.9E-05 | C | USEPA NATA: HEAST |
| 18) Quinoline | 3.4E-03 | C | USEPA NATA: HEAST |
| 19) 1,1,2,2-Tetrachloroethane | 5.8E-05 | C | IRIS |
| 20) Tetrachloroethylene | 5.6E-06 | B2 | USEPA NATA: CalEPA |
| 21) Trichloroethylene | 2.00E-06 | B2 | USEPA NATA: CalEPA |
| 22) Acetaldehyde | 2.20E-06 | B2 | USEPA NATA: IRIS |
| 23) Formaldehyde | 1.30E-05 | B1 | IRIS |
| 24) Arsenic | 4.30E-03 | B1 | IRIS |
| 25) Beryllium compounds | 2.4E-03 | B1 | IRIS |
| 26) Cadmium | 1.80E-03 | B1 | IRIS |
| 27) Chromium (VI) | 1.2E-02 | A | USEPA NATA: IRIS |
| 28) Lead | 1.20E-05 | B2 | USEPA NATA: CalEPA |
| 29) Nickel | 4.8E-04 | A | USEPA NATA: IRIS |
| 30) Woodsmoke | 1.0E-05 | NA | Lewtas, 1988 |

[9] USEPA. NATA Appendix G: *Health Effects Information Used In Cancer and Noncancer Risk Characterization for the NATA 1996 National-scale Assessment.* http://www.epa.gov/ttn/atw/nata/nettables.pdf

[10] EPA National Center for Environmental Assessment.

*2.2.1 Woodsmoke Unit Risk Factor*

Woodsmoke is comprised of a variety of chemicals, including but not limited to: particulate matter, nitrogen oxides, carbon monoxide, sulfur oxides, volatile organic compounds, and polycyclic aromatic hydrocarbons (PAHs).[11] Many of the chemicals listed as constituents in woodsmoke have been identified as probable or likely human carcinogens. However, woodsmoke as a mixture has not been thoroughly evaluated for its carcinogenicity by USEPA or other health agencies. Evaluations by the World Health Organization suggest that vegetative burning, primarily woodsmoke, is likely to be carcinogenic, although sufficient data is not yet available.[12,13,14]

The unit risk factor for woodsmoke was developed through a comparative potency method where the mutagenicity and tumor initiating potency from particles emitted from several sources (e.g., diesels, woodsmoke and gasoline-powered automobiles) are systematically evaluated (Lewtas 1988). Lewtas uses bioassay-directed fractionation, a combination of several chemical separation and bioassay techniques, to identify the more toxic elements of several complex mixtures. In the Lewtas study, mutagenicity tests are conducted on different segments of the total mixtures. Segments showing higher mutagenic potencies are further divided into groups and tested until the components or segments with the highest potencies are identified.[15] The unit risk factor calculated for woodsmoke is listed in Table 2-1.

We recognize the Lewtas woodsmoke URF has not undergone the same rigorous evaluation as the other URFs used in our analysis. Although USEPA or CalEPA have not reviewed the

[11] USEPA. *Emission Inventory Improvement Program, Vol. III, Chapter 2: Residential Wood Combustion.* Revised final. January 2001.

[12] World Health Organization (WHO). *Health Guidelines for Vegetation Fire Events.* Edited by DH Schwela, JG Goldammer, LH Morawska, O Simpson (Findings of the WHO-UNEP-WMO expert task force, Lima, Peru) 1999.

[13] WHO. The *Health Effects of Indoor Air Pollution Exposure in Developing Countries*. N Bruce, R Perez-Padilla, R Albalak. WHO/SDE/OEH/02/05. 2002.

[14] WHO. *Health Impacts of Biomass Air Pollution.* M. Brauer. Health Guidelines for Vegetation Fire Events, Lima Peru. Background papers. 1999.

[15] Lewtas J. *Genotoxicity of Complex Mixtures: Strategies for the Identification and Comparative Assessment of Airborne Mutagens and Carcinogens from Combustion Sources.* Funda and Appl Tox 10, 571-589. 1988.

woodsmoke URF, it is developed through a method recommended by the National Academy of Sciences and is published in a respected peer-reviewed journal.[16]

Also, as one reviewer of the draft of this report noted, vegetative burning could include other materials in addition to wood. Therefore, the woodsmoke unit risk factor may not appropriately estimate cancer risk from vegetative burning. As a result of these uncertainties, we use the woodsmoke unit risk as a general indicator of potency and potential risk.

*2.2.2 Diesel Particulate Matter (DPM) Unit Risk Factor*

Combustion of diesel fuel results in hundreds and probably thousands of organic and inorganic compounds in the diesel exhaust mixture. This mixture includes gaseous compounds such as carbon dioxide, carbon monoxide, aldehydes, benzene, and a wide range of PAHs. Dioxins have also been found in trace quantities in diesel exhaust.[17]

DPM is a component of diesel exhaust. DPM contains elemental carbon, organic carbon, and small amounts of nitrate, metals, and unidentified compounds. We focus on the particulate component of diesel exhaust because it is thought to contain the majority of the toxicity associated with the mixture. These particles and their adsorbed toxics penetrate deep into the lung during inhalation.

While specific knowledge of the role of the adsorbed chemicals is not known, it is hypothesized that the presence of such substances may influence particle toxicity. However, relatively little is known about the cumulative toxicity of the multiple toxics present in certain combustion mixtures. For example, it is possible that antagonism or synergism occurs among the chemicals and/or particles. In addition, there may be a variety of carcinogenic or toxic chemicals present in the mixture that have not yet been identified.[18] Therefore, we use unit risk factors for the whole

[16] National Academy of Sciences. ***Complex Mixtures: Methods for In Vivo Toxicity Testing***. National Academy Press. Washington DC, 1988.

[17] USEPA. ***Health Assessment Document for Diesel Engine Exhaust***. Office of Research and Development. EPA/600/8-90/057F. Washington DC, May 2002.

[18] National Academy of Sciences, 1988.

mixture to estimate potential risk for diesel particulate and woodsmoke, rather than unit risk factors for individual carcinogens and summing the individual risks.

The carcinogenicity of diesel particulate matter is widely recognized by a number of health agencies including the USEPA,[19] CalEPA,[20] the US Department of Health and Human Services,[21] and the International Agency for Research on Cancer (IARC).[22] Because USEPA has not yet developed a unit risk factor for diesel particulate matter, the CalEPA value is used in this analysis.[23] CalEPA conducted an extensive literature review and analysis to develop the unit risk factor for DPM.[24] This value is listed in Table 2-1.

We recognize that USEPA has not identified a final unit risk factor for diesel particulate matter. However, USEPA states firmly that diesel particulate matter is a B1 or probable human carcinogen. In the absence of a confirmed URF, USEPA provides a range of potential cancer risks associated with environmental exposures (i.e., exposure levels typically experienced by the general population) in Section 8.4, entitled "Perspectives on Cancer Risk" of their Health Assessment Document. USEPA estimates this risk range to be approximately 6E-05 to 8E-04. This range assumes average environmental exposures of 0.8 - 4.0 $\mu g/m^3$ over a lifetime.[25] The annual estimate for Beacon Hill is 1.4 $\mu g/m^3$, within the range identified by USEPA. Therefore, we believe it is important to characterize potential risks associated with DPM in relation to other air pollutants.

The risk range used by EPA is also comparable to the unit risk estimate calculated by CalEPA. For example, assuming an environmental exposure of 1 $\mu g/m^3$, the range recommended by

[19] USEPA. May 2002.

[20] CalEPA/OEHHA. *For the Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Part B: Health Risk Assessment for Diesel Exhaust*. May 1998.

[21] National Toxicology Program. Public Health Service, US Department of Health and Human Services***. 9th Report on Carcinogens.*** Revised January 2001.

[22] International Agency for Research on Cancer (IARC***). IARC Monograph on the Evaluation of Carcinogenic Risks to Humans. Vol. 46: Diesel and Gasoline Engine Exhausts***. 1989.

[23] CalEPA, 1998.

[24] CalEPA, 1998.

[25] USEPA 2002.

USEPA could be approximately 8E-05 to 2E-02.[26] The unit risk factor recommended by CalEPA, 3E-04, is also within USEPA's range if one assumes an exposure of 1 μg/m$^3$. This suggests that the CalEPA unit risk factor is not as highly conservative as USEPA's high-end estimates. The potential risk could also be zero.

One reviewer suggested that the cancer risks from DPM could be adequately evaluated by using the unit risk factors for individual carcinogenic PAHs and 6 metals. Unfortunately, this approach would only account for a small percentage of the potentially toxic chemicals found in DPM. USEPA and CalEPA state that the mechanism of action for DPM carcinogenesis has not yet been established.[27] In fact, both USEPA and CalEPA suggest that Diesel Particulate Matter (DPM) toxicity is related to a complex combination of factors such as:

- The physical characteristics of fine particles. USEPA states "The carcinogenicity of diesel particles…appears to be related, as least to some extent, to their small size and convoluted shape, which results in a large specific particle surface area."[28] It is possible that this large surface area may act as a carrier for many chemicals.
- Diesel particles may enhance PAH toxicity, suggesting a possible synergistic relationship between PAHs and particles.[29] If so, carcinogenic potency would be underestimated by using PAHs alone to evaluate cancer risk.
- Exposure to both DPM-related organics and carbon particles (i.e., DPM without organics such as PAHs) may produce reactive oxygen species that could result in a cascade of events leading to DNA damage.

In summary, it is simply not clear how DPM causes cancer or what the causative agents might be. Therefore, reliance on the toxicity of a limited number of PAHs to estimate potential cancer risk for a complex mixture such as DPM could dramatically underestimate potential risk.

[26] Assuming an environmental exposure of 1 μg/m$^3$, an EM ratio of 1 to 252 can be calculated using the "broad concentration range" for occupational exposures described in Section 8.4 of the USEPA 2002 report. Multiplying these values by the 2% excess risk due to diesel particulate matter exposures, results in 8E-05 to 2E-02.

[27] USEPA, 2002.

[28] USEPA, 2002.

[29] USEPA, 2002.

The Clean Air Agency uses a more appropriate approach based on accepted California risk numbers that have been widely cited and are the basis for a diesel retrofit program in place for several years in California. This approach evaluates 100% of the highly toxic diesel particulates as a complete and complex mixture. This method is more likely to account for potential interactions (i.e., synergism and antagonism) among the hundreds and/or thousands of chemicals in DPM.

### *2.3 Non-carcinogenic Effects*

Many chemicals also have non-cancer health effects. Non-carcinogenic effects are presumed to have a threshold of exposure below which no effect occurs, although this is not always the case (e.g., fine particulate matter). Non-carcinogenic effects from air exposures are evaluated using reference concentrations. Reference concentrations (RfCs), like unit risk factors, are based on animal or human studies. RfCs are derived by examining the literature to find a critical study, which is defined as a well-designed chronic exposure study that has identified the non-carcinogenic adverse effect that occurs at the lowest level of inhalation exposure. The no-observable-effect-level (NOEL) or a lowest-observable-adverse-effect-level (LOAEL) from animal or human studies is determined. Adjustments for exposure times are made to extrapolate exposures to 24 hours, 7 days per week, and conversion to units of $mg/m^3$ are made. A human equivalent concentration is calculated by considering the nature of the contaminant and its behavior in inhaled air; the region of the respiratory system impacted; and the surface area and respiratory rate of the test organism, relative to the same parameters in humans. This concentration is then divided by factors of 10 to account for uncertainties such as extrapolating from animals to humans, from healthy adult individuals to sensitive individuals, or from sub-chronic to chronic exposures. The RfCs also include confidence statements that speak to the extent and quality of the database, and the certainty of the RfC, based on supporting literature aside from the critical study.

As a result of these types of derivations, the RfC is also considered to be highly conservative or protective of human health. Similar to the unit risk factors used for carcinogens, USEPA considers the RfC to be unlikely to underestimate potential risks to humans. It is important to recognize that many chemicals can have a variety of effects that occur at different levels of

exposure. The RfC only looks at the effect that occurs at the lowest level of exposure. The assumption is made that protection at this level also provides protection at the higher doses as well.

To determine a hazard index for these chemicals, the RfC is compared to the annual average or median concentration for each of the three exposure data sets (e.g., the monitoring results, the ambient modeling results, and the human exposure modeling results). We compare the RfC to the median concentration for the human exposure modeling estimates because they are the only estimate of central tendency available. For the Seattle monitoring data and the ambient exposure modeling exercise, we compare the RfC to the annual average. We also compare the RfC to an upper-bound concentration for the ambient modeling exercise as it was available. However, this value may overestimate exposures over the lifetime of the exposed individual. RfCs used in this evaluation are listed in Table 2-2 below. The name of the chemical is listed with the RfC value, the uncertainty factors and modifying factors used in calculating the RfC, the critical effect, and the source for the information. The information used in the analysis and listed in the table was taken from the USEPA NATA report.

The non-cancer health effects associated with diesel particulate matter and woodsmoke, and the fine particulate fraction of these mixtures specifically are not included in this evaluation. Non-cancer health effects associated with fine particles, such as morbidity related effects such as increased asthma attacks, upper respiratory irritation, and increased mortality are analyzed elsewhere.

**Table 2-2: Reference Concentrations for Air Toxics[30]**

| Chemical | RfC ($mg/m^3$) | UF x MF | Target Organ for Critical Effect | Source |
|---|---|---|---|---|
| 1) Acetaldehyde | 9.0E-03 | 1000 | Nasal epithelium | IRIS |
| 2) Acrolein | 2.0E-05 | 1000 | Nasal epithelium | IRIS |
| 3) Acrylonitrile | 2.0E-03 | 100 | Nasal epithelium | IRIS |
| 4) Arsenic and compounds | 3.0E-05 | 1000 | Teratogenic effects | Cal EPA |
| 5) Benzene | 8.0E-02 | 100 | Blood, bone marrow | IRIS |
| 6) Beryllium compounds | 2.0E-05 | 10 | Lung | IRIS |
| 7) 1,3-Butadiene | 2.0E-03 | 300 | Reproductive system | IRIS |
| 8) Cadmium compounds | 2.0E-05 | 30 | Kidney | Cal EPA |
| 9) Carbon tetrachloride | 4.0E-02 | 300 | Liver | Cal EPA |
| 10) Chloroform | 9.8E-02 | 100 | Liver, kidney | ATSDR |
| 11) Chromium compounds | 1.0E-04 | 90 | Respiratory tract | IRIS |
| 12) 1,3-Dichloropropene | 2.0E-02 | 30 | Nasal epithelium | IRIS |
| 13) Ethylene dibromide | 8.0E-04 | 100 | Reproductive system | Cal EPA |
| 14) Ethylene dichloride | 2.4E-00 | 90 | Kidney | ATSDR |
| 15) Ethylene oxide | 3.0E-02 | 100 | Blood | Cal EPA |
| 16) Formaldehyde | 9.8E-03 | 30 | Respiratory tract | ATSDR |
| 17) Hexachlorobenzene | 3.0E-03 | 100 | Teratogenic effects | Cal EPA |
| 18) Hydrazine | 2.0E-04 | 300 | Liver, thyroid | Cal EPA |
| 19) Lead compounds | 1.5E-03 | 1 | Central nervous system | NAAQS |
| 20) Manganese compounds | 5.0E-05 | 1000 | Central nervous system | IRIS |
| 21) Mercury compounds | 3.0E-04 | 30 | Central nervous system | IRIS |
| 22) Methylene chloride | 1.0E+00 | 30 | Liver | ATSDR |
| 23) Nickel compounds | 2.0E-04 | 30 | Respiratory tract | ATSDR |
| 24) Propylene dichloride | 4.0E-03 | 300 | Nasal epithelium | IRIS |
| 25) Tetrachloroethylene (perc) | 2.7E-01 | 100 | Central nervous system | ATSDR |
| 26) Trichloroethylene | 6.0E-01 | 100 | Central nervous system | Cal EPA |

[30] USEPA. *Health Effects Information Used in Cancer and Noncancer risk Characterization for the NATA 1996 National Scale Assessment.* http://www.epa.gov/ttn/atw/nata/nettables.pdf.

## Chapter 3: Health Risks Based on Air Toxics Monitoring Information

After we identified the toxicity values, we obtained exposure concentrations from three separate studies. The first study provides air monitoring concentrations in the Seattle area for 15 air toxics (Seattle Air Toxics Monitoring Study). The second study models ambient concentrations of woodsmoke and DPM from $PM_{2.5}$ monitored concentrations (Source Apportionment at an Urban IMPROVE Site). The third study provides modeled concentrations for 32 air toxics and DPM in the four counties in our jurisdiction (USEPA NATA study). The first two studies are discussed below, while the third study is discussed in Chapter 4.

### *3.1 Seattle Air Toxics Monitoring Study*

The *Seattle Air Toxics Monitoring Study* was conducted during 2000 and 2001 as a collaborative effort by three agencies: USEPA, the Washington State Department of Ecology, and the Puget Sound Clean Air Agency. The purposes of this study were to provide information on the spatial and temporal variability of ambient air toxics, to evaluate modeling results obtained from the NATA project, and compare results to other urban areas in the United States. The objective of this study was to quantify the urban air toxics such as VOCs, carbonyl, and metal species on a regular basis at several surface sites in Seattle.

USEPA originally selected Seattle for this monitoring study as one of four cities nationwide to take part in the air toxics monitoring component of its overall National-scale Air Toxics Program (NATA).[31] The federal Clean Air Act mandates USEPA to determine a subset of the 189 urban hazardous air pollutants (HAPs) that potentially pose the greatest risks in urban areas. USEPA identified a total of 33 urban HAPs in their 1995 ranking analysis,[32] and developed concurrent monitoring and modeling programs (e.g., NATA) to evaluate potential exposures to these top-ranked 33 HAPs.[33] These 33 are discussed more fully in Chapter 4. Of the 33 HAPs identified

[31] USEPA. Peer Review Draft for the Science Advisory Committee: *Air Toxics Monitoring Concept Paper*. Office of Air Quality Planning and Standards. February 2000.

[32] USEPA. *Ranking and Selection of Hazardous Air Pollutants for Listing Under Section 112(j) of the Clean Air Act Amendments of 1990, Technical Support Document*, July 28, 1999.

[33] USEPA. *National Air Toxics Program: The Integrated Urban Strategy Report to Congress*. Office of Air Quality Planning and Standards. EPA-453/R-99-007. July 2000.

by USEPA, a total of 17 HAPs (see Table 3-1) were monitored at two sites in the Seattle area during calendar year 2000 and at six sites during 2001.

**Table 3-1: Monitored Urban Air Toxic Pollutants (17 total)**

| | CAS No. | VOCs |
|---|---|---|
| 1) | 71432 | Benzene |
| 2) | 7440439 | 1,3-Butadiene |
| 3) | 56235 | Carbon tetrachloride |
| 4) | 67663 | Chloroform |
| 5) | 75092 | Dichloromethane |
| 6) | 78875 | 1,2-Dichloropropane |
| 7) | 127184 | Tetrachloroethene |
| 8) | 79016 | Trichloroethene |
| 9) | 7440382 | Arsenic |
| 10) | Total compounds | Beryllium |
| 11) | Total compounds | Cadmium |
| 12) | Total compounds | Chromium |
| 13) | 7439921 | Lead |
| 14) | Total compounds | Manganese |
| 15) | 7440020 | Nickel |
| 16) | 75070 | Acetaldehyde |
| 17) | 50000 | Formaldehyde |

The remaining 16 HAPs were not monitored because they were considered less stable or lacked approved collection and/or analytical techniques. Every six days at each site, 24-hour integrated air samples were collected.[34] Such collection schedules ensure that every day of the week is sampled over the year. Average concentrations for each monitored chemical were provided by the Washington State Department of Ecology, and are presented in Table 3-2. Because no data were provided for 1,2-dichloropropane and beryllium, these chemicals were removed from further consideration in this analysis.

[34] Washington Department of Ecology. *Urban Air Toxic Measurements in Seattle*. Conducted by the Laboratory for Atmospheric Research, Washington State University, Pullman, WA. Contract #C0000060. Project Officer: John Williamson, Bellevue, WA., May 2001.

**Table 3-2: Monitored Ambient Concentrations at 6 Sites in Greater Seattle, 2000 and 2001 (averages)**

| Chemical | Beacon Hill ($\mu g/m^3$) | George-town ($\mu g/m^3$) | Lake Forest Park ($\mu g/m^3$) | Lake Sam-mamish ($\mu g/m^3$) | Maple Leaf Reservoir ($\mu g/m^3$) | SeaTac ($\mu g/m^3$) | 6-Site Average ($\mu g/m^3$) |
|---|---|---|---|---|---|---|---|
| **Benzene** | 1.18E+00 | 1.80E+00 | 1.64E+00 | 1.15E+00 | 1.13E+00 | 1.02E+00 | 1.32E+00 |
| **1,3-Butadiene** | 1.37E-01 | 1.35E-01 | 1.24E-01 | 1.06E-01 | 8.39E-02 | 9.94E-02 | 1.14E-01 |
| **Carbon tetrachloride** | 6.10E-01 | 6.54E-01 | 6.42E-01 | 6.23E-01 | 6.10E-01 | 6.23E-01 | 6.27E-01 |
| **Chloroform** | 2.30E-01 | 1.42E-01 | 1.47E-01 | 1.27E-01 | 2.30E-01 | 1.27E-01 | 1.67E-01 |
| **Dichloromethane** | 4.55E-01 | 9.13E-01 | 6.53E-01 | 6.98E-01 | 5.49E-01 | 4.69E-01 | 6.23E-01 |
| **Tetrachloroethylene** | 1.56E-01 | 3.66E-01 | 2.44E-01 | 1.76E-01 | 2.10E-01 | 1.42E-01 | 2.16E-01 |
| **Trichloroethylene** | 1.88E-01 | 3.82E-01 | 1.67E-01 | 1.40E-01 | 2.10E-01 | 1.72E-01 | 2.10E-01 |
| **Acetaldehyde** | 1.26E+00 | 1.26E+00 | 1.26E+00 | 1.26E+00 | 1.08E+00 | 1.26E+00 | 1.23E+00 |
| **Formaldehyde** | 1.72E+00 | 1.47E+00 | 1.10E+00 | 9.82E-01 | 1.23E+00 | 1.35E+00 | 1.31E+00 |
| **Arsenic** | 9.70E-04 | 1.40E-03 | 1.63E-03 | 8.63E-04 | 8.67E-04 | 9.69E-04 | 1.12E-03 |
| **Cadmium** | 5.90E-04 | 9.00E-04 | 1.69E-04 | 1.20E-04 | 1.10E-04 | 8.10E-05 | 3.28E-04 |
| **Chromium** | 1.67E-03 | 3.20E-03 | 1.09E-03 | 9.07E-04 | 9.27E-04 | 1.47E-03 | 1.54E-03 |
| **Lead** | 3.49E-03 | 9.30E-03 | 5.27E-03 | 3.26E-03 | 4.28E-03 | 3.24E-03 | 4.81E-03 |
| **Manganese** | 3.61E-03 | 1.08E-02 | 5.15E-03 | 6.99E-03 | 5.57E-03 | 6.54E-03 | 6.44E-03 |
| **Nickel** | 2.39E-03 | 3.40E-03 | 1.16E-03 | 9.21E-04 | 3.60E-04 | 1.42E-03 | 1.61E-03 |
| **Diesel particulate matter*** | 1.40E+00 | | | | | | |
| **Vegetative burning*** | 3.00E+00 | | | | | | |

***calculated using monitoring results and PMF source apportionment model. See discussion below.***

*Site Locations and Selected Pollutants*

A total of six sites were selected to represent the Seattle urban area based on a comprehensive site selection study.[35] Two sites were monitored during calendar year 2000, and four more sites (for a total of six sites) were monitored during calendar year 2001 (see Figure 3-1).

The two sites monitored during 2000 were Beacon Hill and Georgetown. The first site represents a typical urban residential area. Beacon Hill (Fig. 3-1: ❸) was selected to represent this type of area because it has a relatively high population density and is impacted by a mix of urban source categories. For example, it is located near the Interstate 90 and Interstate 5 interchange, and is also impacted by local sources. However, it is more significantly impacted by urban residential sources such as mobile exhaust and woodsmoke. A spatial variation study conducted by UW also verified that Beacon Hill is representative of population exposure.[36]

The second area was selected to represent potentially maximum concentrations near an industrial area. This site is located in the Georgetown neighborhood (Fig. 3-1: ❹). It is impacted by several large industrial sources, as well as an airport. Mobile sources from Highway 99, nearby roadways, and residential wood combustion are also expected to impact this site. This neighborhood is located in the Duwamish industrial valley.

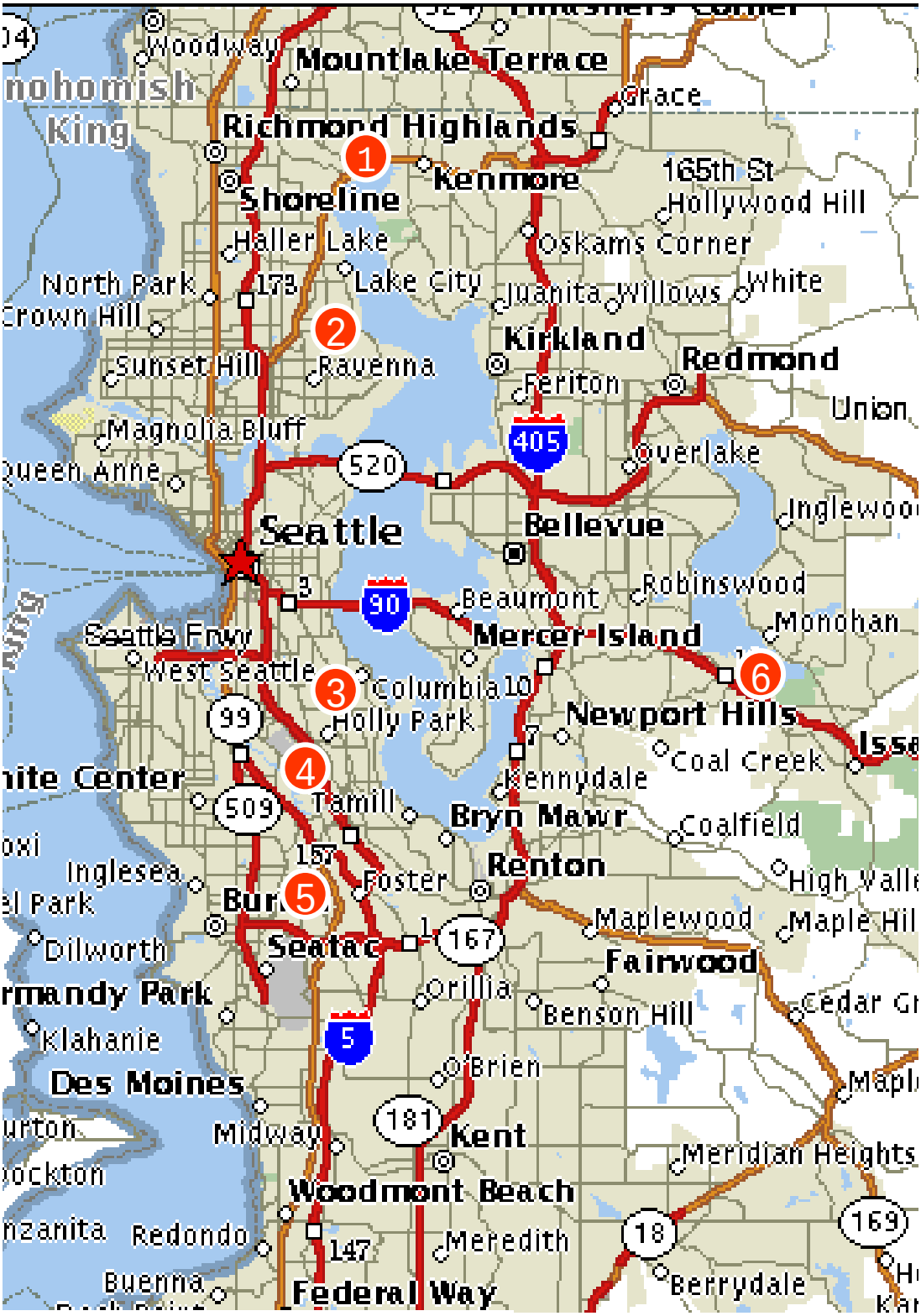
Four more sites were added for the 2001 calendar year. These sites include: Lake Sammamish (Fig. 3-1: ❻) for an urban background site, Maple Leaf (Fig.3-1: ❷) for a typical urban residential site, SeaTac (Fig. 3-1: ❺) for a site that is highly impacted by mobile sources, and Lake Forest Park (Fig.3-1: ❶) for an area affected by woodsmoke and mobile sources.

Two of the six sites are located near airports. The SeaTac monitor is located north of the Seattle-Tacoma International Airport, a major airport that serves the Puget Sound area. The Georgetown site is also located near an airport that serves a number of commercial industries including

[35] Goswami E, T Larson, T Lumley, S Liu. *Spatial Characteristics of Fine Particulate Matter. Identifying Representative Monitoring Locations in Seattle, Washington*. Journal of Air and Waste Management Association. Vol. 52, March 2002.

[36] Goswami et al., 2002.

**Figure 3-1: Air Toxics Monitor Locations**



The Boeing Company, a major aerospace manufacturing site. The potential impact of these airport emissions on the monitored concentrations are discussed in the latter sections of this chapter.

### *3.2 Woodsmoke and Diesel Particulate Concentrations*

In addition to risks from ambient air toxics, ambient concentrations of woodsmoke and DPM have long been recognized as potentially carcinogenic and contribute substantially to ambient particulate matter concentrations in the Puget Sound area.[37,38] To quantify potential risks from these mixtures, ambient concentrations are multiplied by a unit risk factor (see methods in Chapters 1 and 2). We use woodsmoke and diesel particulate concentrations for the Beacon Hill monitoring site as estimated in a recent study conducted by Maykut, Larson, Lewtas, and Kim entitled *Source Apportionment of PM2.5 at an urban IMPROVE site in Seattle, WA.*[39]

*Source Apportionment of PM2.5 at an Urban IMPROVE site in Seattle, WA*

Speciated data from Seattle's Beacon Hill PM2.5 monitoring site were analyzed using two multivariate receptor models, the Positive Matrix Factorization (PMF) and the UNMIX model. EPA's Chemical Mass Balance model was also used to identify the major sources of PM2.5 and organic carbon in Seattle's air. A total of 289 filter samples were obtained with an IMPROVE sampler from 1996 through 1999. These samples were analyzed for 31 particulate "elements" including various fractions of the particulate organic and elemental carbon. All three models predicted the major sources of PM2.5 were wood-burning, mobile sources, and secondary particle formation.

The sources identified by the PMF model are (in descending order of importance): vegetative burning such as wood-burning fireplaces and yard waste (indoor and outdoor), motor vehicles (gasoline and diesel), secondary sulfate, secondary nitrate, soil, and marine sea salt (Fig. 3-2).

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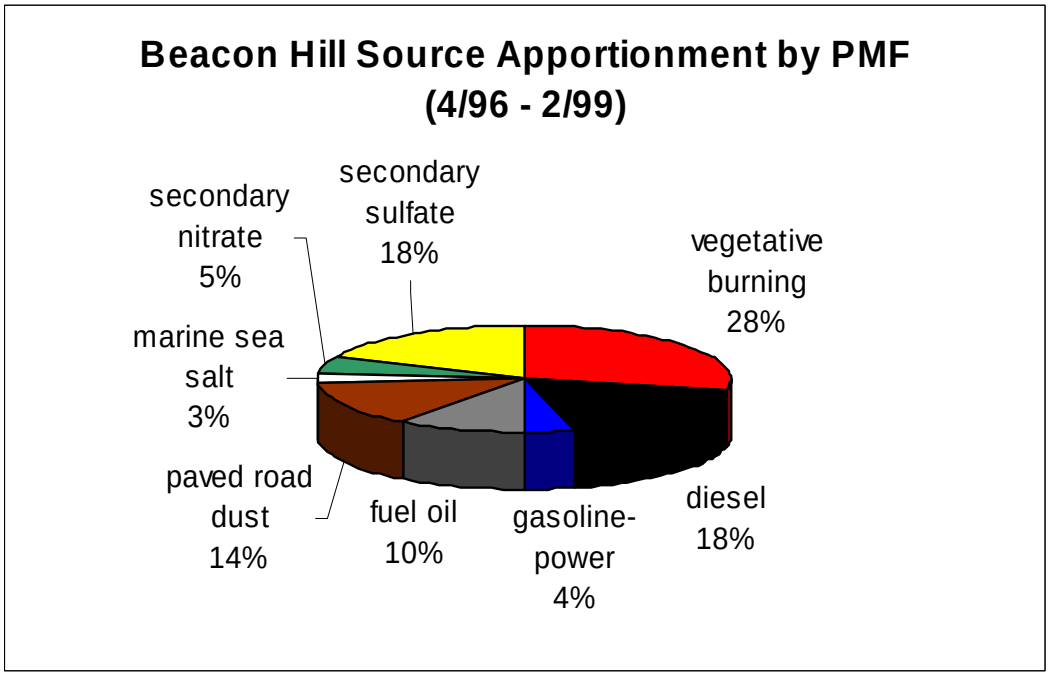
[37] Lewtas J. *Genotoxicity of Complex Mixtures: Strategies for the Identification and Comparative Assessment of Airborne Mutagens and Carcinogens from Combustion Sources.* Fundamental and Applied Toxicology 10, 571-589. 1988.

[38] Yuen and Larson, 1993.

[39] Maykut N, et al. 2003.

**Figure 3-2: Beacon Hill Source Apportionment from Maykut, et al. (2003)**[40]




The average concentration of $PM_{2.5}$ at Beacon Hill from April 1996 through February 1999 was 9 µg/m$^3$. This translates to average annual concentrations of approximately 3 µg/m$^3$ for vegetative burning and 1.4 µg/m$^3$ of diesel particulate. It is important to note that our analysis considers only 46% of the $PM_{2.5}$ present in ambient air (vegetative burning + diesel), while the remaining 54% could contribute to overall cancer risk from particulate matter.[41] Thus our estimates could significantly underestimate potential cancer risk from fine particles.

Diesel particulate matter was estimated from the $PM_{2.5}$ monitor located at the Beacon Hill site for three years using the Positive Matrix Factorization (PMF model).[42] The ambient annual concentration of diesel particulate matter at the Beacon Hill site is estimated to be 1.4 µg/m$^3$. Some reviewers noted that this value could be high due to the proximity of the monitor to Interstate 5 and Interstate 90.

[40] Maykut N et al., 2003

[41] Pope CA, RT Burnett, MJ Thun, EE Calle, D Krewski, K Ito, GD Thurston. *Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution.* JAMA, March 6, 2002, vol. 287, No. 9.

[42] Maykut N et al., 2003.

### *3.3 Estimated Potential Cancer Risks from Six Monitoring Sites*

Potential cancer risk estimates for each chemical at each of the six sites of the Seattle Air Toxics Monitoring Study are presented in Table 3-3 and Figure 3-3. Cancer risks for the average concentrations across all 6 sites are also presented. These values are presented as individual cancer risk per million (over a 70-year lifetime) and potential cancer cases per million people exposed over a 70-year exposure period.

It is important to recognize that these cancer risk estimates are based on the assumption that adults (either one or many in an exposed population) are exposed to this average concentration for their entire lifetime or an exposure period of 70 years. The ambient concentrations may or may not represent actual annual average exposures for individuals throughout the Seattle population. For example, it is highly unlikely that an individual would spend an entire 70-year period outside near a particular monitor. Alternatively, both VOCs and semi-volatile compounds can penetrate indoors. Therefore, it is highly likely that people spending time inside homes or other buildings are exposed to ambient air toxics while indoors.

As indicated on the table and figure, the cumulative cancer risks for the 17 chemicals are similar among the six Seattle sites, ranging from a low of approximately 57 in a million in Lake Sammamish to a high of 100 in a million in Georgetown. Preliminary analyses from the University of Washington indicate that the differences in measured concentrations at various sites are statistically significant.[43] However, from a practical standpoint, these differences are still quite small, particularly when compared with potential risks from DPM and woodsmoke later in this section. We also note that the Beacon Hill risk estimates are similar to the 6-site average (again, 80 to 73 in a million), supporting UW findings that this site is a good indicator for the area.[44]

Georgetown appears to have the highest monitored concentrations, and therefore a higher risk than the other five monitor locations. This appears to be due to higher concentrations of chromium, possibly due to industrial activities near the monitoring site.

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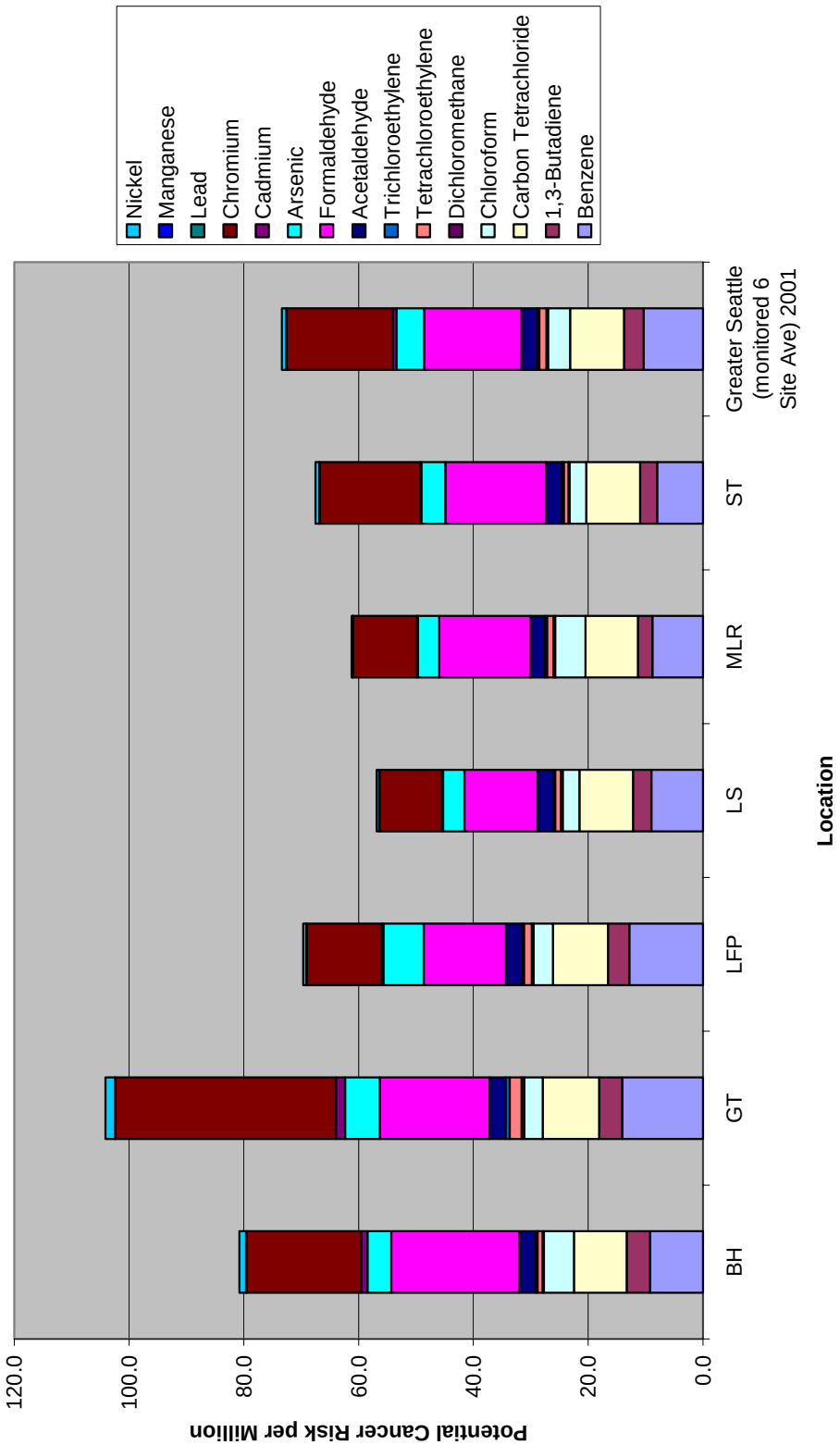
[43] Lui LS, C Wu, A Cullen. *Investigation of Spatial and Temporal Variation in Air Toxics in the Seattle area.* Powerpoint presentation, February 26, 2003.

[44] Goswami et al. 2002.

**Table 3-3: Estimated Cancer Risks Per 1,000,000 Associated with Seattle Air Toxics Monitoring Study Results in 2001 (and 1996-1999 for DPM and Vegetative Burning)**

| Chemical | BH | GT | LFP | LS | MLR | ST | Greater Seattle (monitored 6-site avg.) 2001 |
|---|---|---|---|---|---|---|---|
| **Benzene** | 9.2 | 14.1 | 12.8 | 9.0 | 8.8 | 8.0 | 10.3 |
| **1,3-Butadiene** | 4.1 | 4.0 | 3.7 | 3.2 | 2.5 | 3.0 | 3.4 |
| **Carbon tetrachloride** | 9.2 | 9.8 | 9.6 | 9.3 | 9.2 | 9.3 | 9.4 |
| **Chloroform** | 5.3 | 3.3 | 3.4 | 2.9 | 5.3 | 2.9 | 3.8 |
| **Dichloromethane** | 0.2 | 0.4 | 0.3 | 0.3 | 0.3 | 0.2 | 0.3 |
| **Tetrachloroethylene** | 0.9 | 2.1 | 1.4 | 1.0 | 1.2 | 0.8 | 1.2 |
| **Trichloroethylene** | 0.4 | 0.8 | 0.3 | 0.3 | 0.4 | 0.3 | 0.4 |
| **Acetaldehyde** | 2.8 | 2.8 | 2.8 | 2.8 | 2.4 | 2.8 | 2.7 |
| **Formaldehyde** | 22.3 | 19.1 | 14.4 | 12.8 | 16.0 | 17.5 | 17.0 |
| **Arsenic** | 4.2 | 6.0 | 7.0 | 3.7 | 3.7 | 4.2 | 4.8 |
| **Cadmium** | 1.1 | 1.6 | 0.3 | 0.2 | 0.2 | 0.1 | 0.6 |
| **Chromium** | 20.0 | 38.4 | 13.1 | 10.9 | 11.1 | 17.6 | 18.5 |
| **Lead** | 0.0 | 0.1 | 0.1 | 0.0 | 0.1 | 0.0 | 0.1 |
| **Manganese** | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Nickel** | 1.1 | 1.6 | 0.6 | 0.4 | 0.2 | 0.7 | 0.8 |
| **Diesel particulate matter** | 420.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Woodsmoke** | 30.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| **Total** | 530.8 | 104.1 | 69.7 | 56.9 | 61.2 | 67.5 | 73.4 |

**Figure 3-3: Potential Cancer Risk Estimates per 1,000,000 for 6 Monitoring Sites in Greater Seattle in 2001**



Although carbon tetrachloride is a significant contributor to the cancer risk estimates, it is important to note that this chemical has been banned in the Puget Sound area for some time. These monitored concentrations may reflect emissions that are not currently reported or previous contamination that is extremely persistent.

We also compared risk estimates calculated using modeled and monitored air concentrations. In Figure 3-4, the cancer risks from the 6-site average for Greater Seattle from the air monitoring study are compared to the cancer risks using the USEPA NATA results for King County. Even though the NATA estimates are for 1996 and the monitored estimates are 2001, the cancer risks compare surprisingly well, with cumulative cancer risks of 73 and 78 per million, respectively.

Since Beacon Hill is considered to represent the area, we also compared Beacon Hill estimates to the USEPA NATA estimates and included DPM (see Figure 3-5 and Table 3-4). It is interesting to note that even though the time periods are different for the studies, the modeled estimates compare reasonably well to the monitored estimates.

The cumulative cancer risk for only the chemicals monitored in the 2000 and 2001 studies ranges from approximately 57 to 100 per million over a 70-year exposure period for these chemicals. The total risk for the Seattle average is approximately 73 per million over a 70-year exposure period. These risk estimates are based on the assumption that concentrations observed in this monitoring study will be constant for the assumed 70-year exposure period and that exposures to ambient air reflect the types of exposures that are occurring over the duration.

Emissions from the two airports could impact the SeaTac and Georgetown monitors. However, the results do not reflect significantly higher pollutant levels at these locations when compared with other sites. In fact, SeaTac potential risks appear slightly lower than Beacon Hill. It is possible that the airport emissions do not significantly impact the monitors because the emissions are diluted over the area. It is also possible that the pollutants of concern at the airport are not those included in the monitoring study.

**Figure 3-4: Comparing Cancer Risk between Monitored and Modeled Estimates**

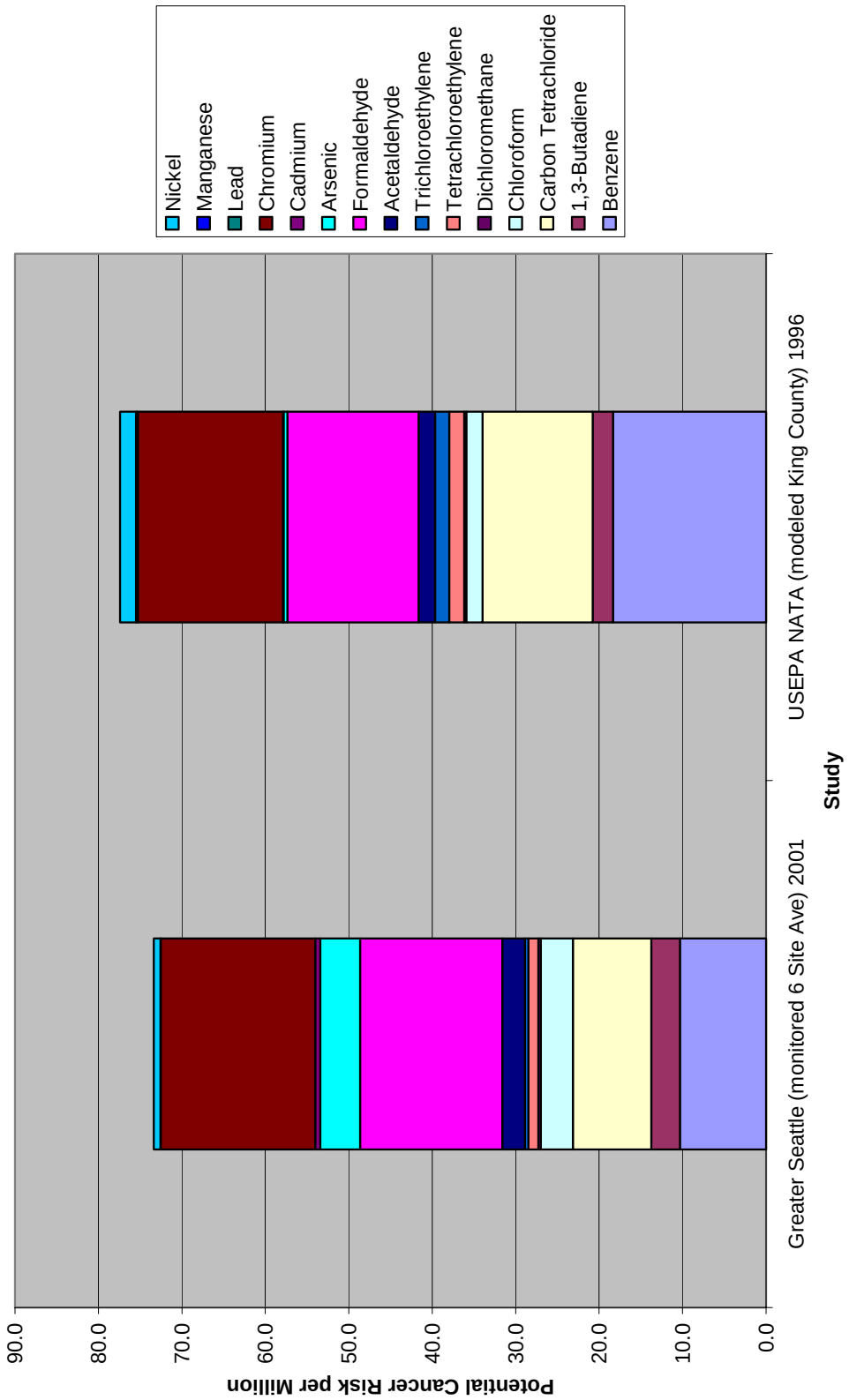


**Figure 3-5: Comparing Beacon Hill (monitored) with NATA (modeled) Cancer Risk Estimates**

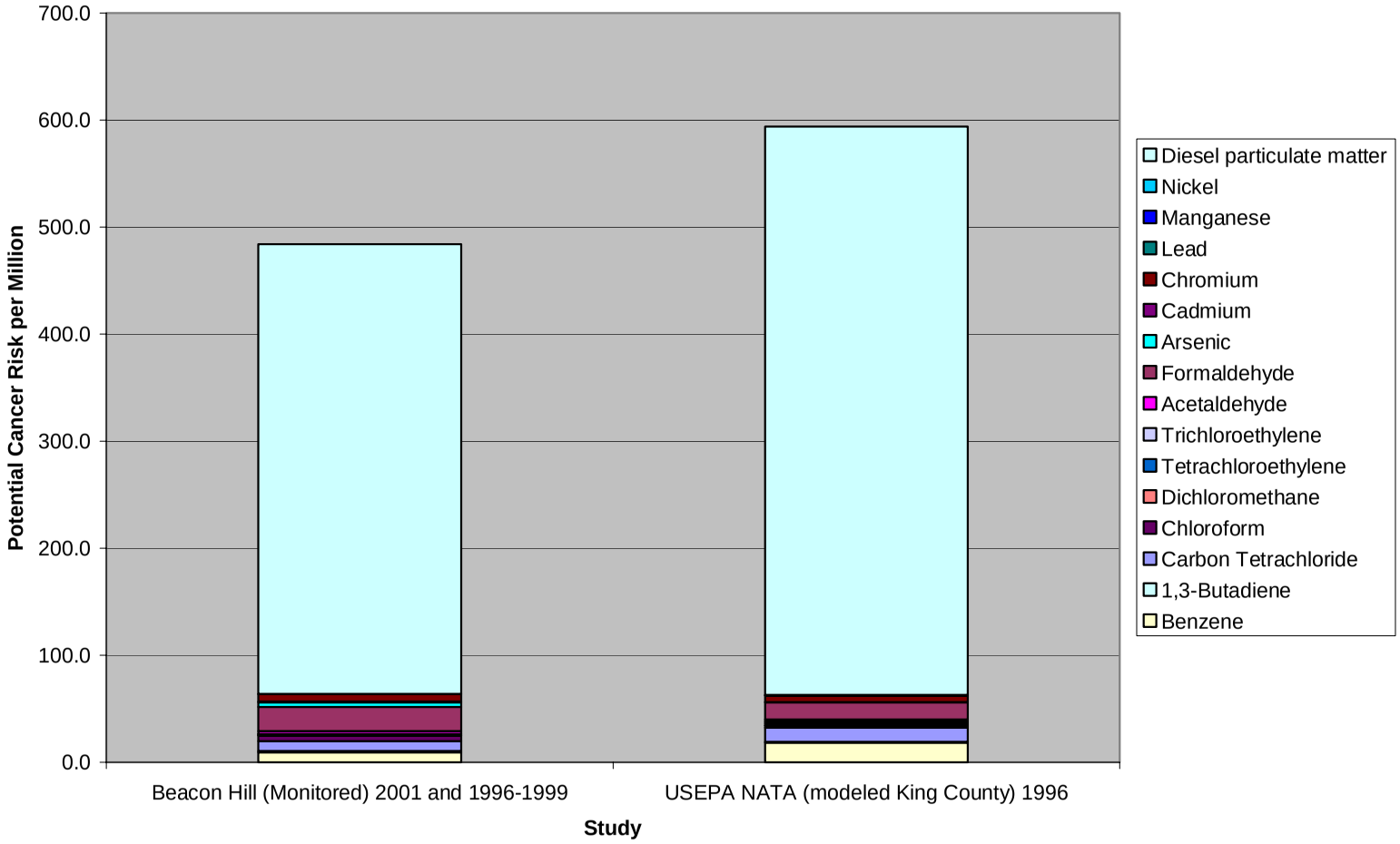


Table 3-4: Comparing Potential Cancer Risks at Beacon Hill (monitoring data) and King County (modeled data)

| Chemical | Beacon Hill 2001 Potential Risk (cancer cases per million) | USEPA NATA 1996 Potential Risk (cancer cases per million) |
|---|---|---|
| **Benzene** | 9.2 | 18.3 |
| **1,3-Butadiene** | 4.1 | 2.5 |
| **Carbon tetrachloride** | 9.2 | 13.2 |
| **Chloroform** | 5.3 | 1.9 |
| **Dichloromethane** | 0.2 | 0.3 |
| **Tetrachloroethylene** | 0.9 | 1.8 |
| **Trichloroethylene** | 0.4 | 1.7 |
| **Acetaldehyde** | 2.8 | 2.0 |
| **Formaldehyde** | 22.3 | 15.7 |
| **Arsenic** | 4.2 | 0.5 |
| **Cadmium** | 1.1 | 0.1 |
| **Chromium** | 20.0 | 17.4 |
| **Lead** | 0.0 | 0.2 |
| **Manganese** | 0.0 | 0.0 |
| **Nickel** | 1.1 | 1.9 |
| **Diesel particulate matter** | 420.0 | 531.0 |
| **Woodsmoke** | 30.0 | 0.0 |
| **Total Cancer Risk** | 530.8 | 608.4 |

Chemicals that pose the greatest risks are primarily associated with mobile sources. Similar to Beacon Hill, the Georgetown risks are dominated by the mobile source chemicals benzene, formaldehyde, and 1,3-butadiene. However, the individual risk estimates from these chemicals are somewhat higher than those estimated at the Beacon Hill site. This may reflect the fact that the Georgetown monitor is located in an industrial area or the Duwamish Valley where contaminants may readily accumulate during winter inversion conditions.

It is important to note that these monitors are placed in areas that are not expected to be heavily impacted by a large industrial source or "hotspot" – except for possibly the Georgetown site, which is located in the Duwamish industrial area. The annual average from the selected monitor locations are expected to reflect general urban settings such as an urban residential area, or an urban industrial area. A few chemicals that are associated with industrial point sources, such as

chromium and trichloroethylene, are higher in the Georgetown area. These chemicals probably reflect more general industrial uses of paints, solvents, and chrome plating rather than one specific industrial source.

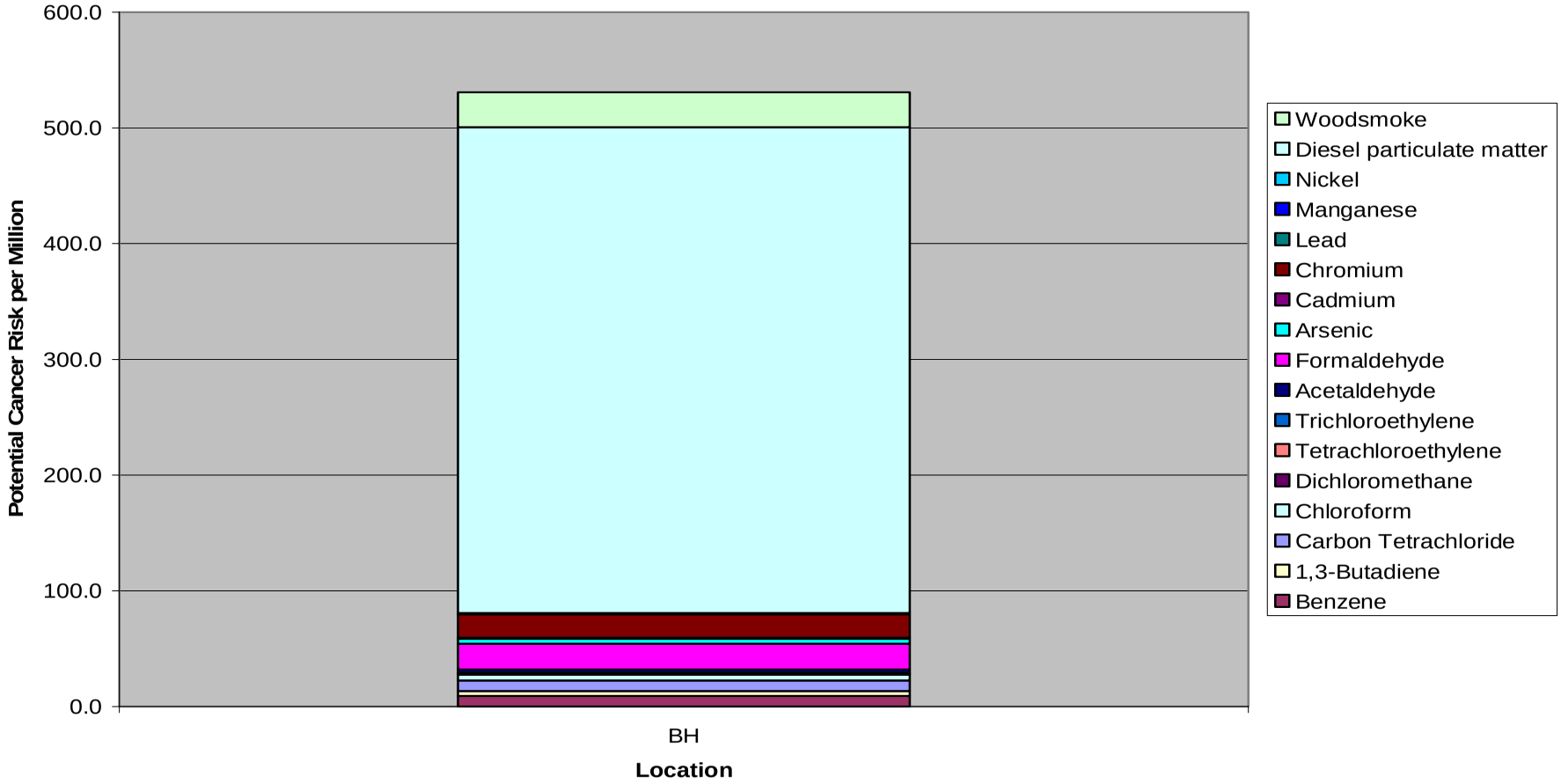
*Woodsmoke and DPM*

In addition to the air toxics measured in the 2000 and 2001 studies, toxics concentrations in many Seattle neighborhoods are heavily impacted by vegetative smoke from residential indoor burning and DPM. We used the vegetative burning and DPM estimates from the Beacon Hill PMF modeling exercise performed by Maykut et. al. (2003)[45]. The annual average vegetative burning and DPM concentrations for the Beacon Hill site are multiplied by a "residential heating wood" and DPM unit risk factor (see Chapter 2 for more discussion on unit risk estimates), respectively, to estimate the potential cancer risk. These estimates are added to the overall estimated cancer risks from the other monitored air toxics to compare the potential impacts. The cumulative cancer risk from air pollution measured at Beacon Hill is shown in Figure 3-6.

One reviewer noted that because vegetative burning and DPM are complex mixtures that include other chemicals already measured (i.e., metals), our methods overestimate risk from these two sources. We recognize that some portion of ambient metals is due to DPM, and may be "double-counted". However, we do not know exactly how much of the ambient metals concentrations are due to DPM. We also know DPM is not the only source of metals in our region. Therefore, we elect to combine metals and DPM estimates, recognizing the results will slightly overestimate risk. We expect the potential impact of "double counting" overall to be quite low since metals are not among the primary risk drivers. Even if the potential risks from metals were reduced to account for DPM, the overall findings would not change. DPM would still rank highest among contributors, with other mobile sources and vegetative burning also among the top sources. Cumulative cancer risks would still approach 500 in a million on average, and could be higher (see Chapter 4).

[45] Maykut N et al. 2003.

**Figure 3-6: Potential Cancer Risks at Beacon Hill including Diesel Particulate Matter and Woodsmoke**



Another reviewer noted that vegetative burning is not synonymous with woodsmoke. We recognize that the unit risk factor for woodsmoke (referred to as "residential heating wood" in the Lewtas study) is uncertain, and could over- or underestimate the toxicity of all vegetative burning included in the Maykut et al (2003) study. However, we believe the limited available data provides a general indication of where vegetative burning might rank in comparison with other air pollution sources in our region.

As indicated in Figure 3-6, DPM is the greatest contributor to potential cancer risk at Beacon Hill. Vegetative burning also contributes significantly to the overall estimated cancer risks from ambient pollution. Although Beacon Hill is considered to represent the area within 20 km,[46] it may underestimate risks in "hot spots" or areas affected by local wood-burning. For example, there are a number of areas, such as Lake Forest Park, Puyallup, and Marysville, where the woodsmoke concentrations may be significantly higher than those measured at Beacon Hill.[47,48] Overall, potential cumulative cancer risks from monitored chemicals approach 500 in a million.

### *3.4 Potential Non-cancer Effects from Six Monitoring Sites*

We evaluated monitored concentrations of toxics for potential non-cancer health effects such as upper respiratory irritation, blood and bone marrow effects, and central nervous system effects. For this exercise, the annual average values from the 6-site average and each individual site were compared to RfCs through ratios referred to as hazard indices (HI).

The HI is a very simple method that compares potential exposure concentrations with health-based guidelines (see Chapter 2 for a discussion of the reference concentrations used in this analysis). HIs for the annual averages (the annual average of the Seattle/King County area) are shown in Table 3-5 and Figure 3-7 below. These results may not be reasonable maximum estimates because upper-bound percentiles were not available for the monitored results.

[46] Goswami et al., 2002.

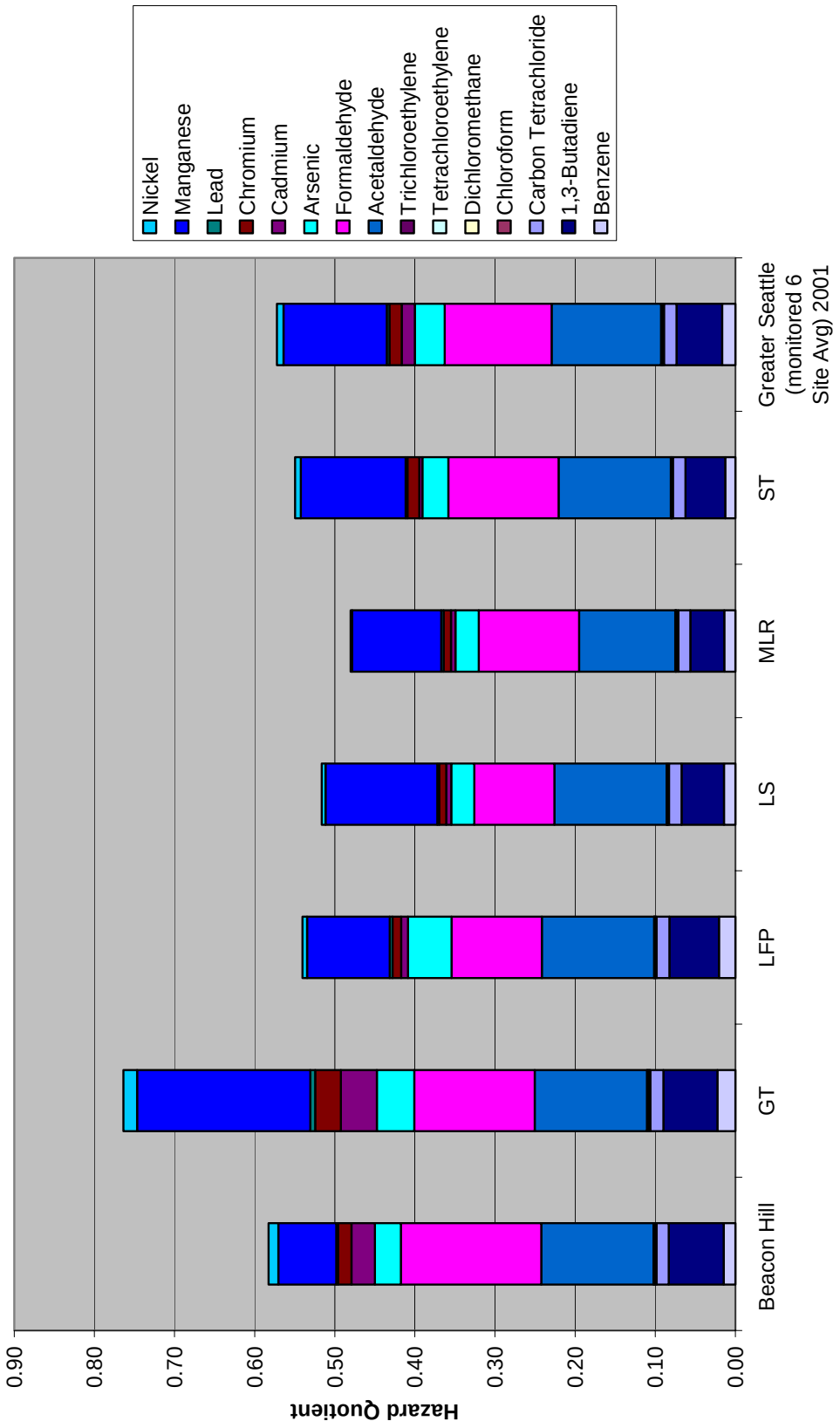
[47] Personal communication, N. Maykut with L. Keill at Puget Sound Clean Air Agency, June 30, 2003.

[48] Yuen and Larson, 1993.

**Table 3-5: Hazard Indices for Monitored Air Toxics in Seattle, 2001**

| | BH | GT | LFP | LS | MLR | ST | Greater Seattle (monitored 6-Site Average) 2001 |
|---|---|---|---|---|---|---|---|
| Benzene | 0.01 | 0.02 | 0.02 | 0.01 | 0.01 | 0.01 | 0.02 |
| 1,3-Butadiene | 0.07 | 0.07 | 0.06 | 0.05 | 0.04 | 0.05 | 0.06 |
| Carbon tetrachloride | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 |
| Chloroform | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| Dichloromethane | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| Tetrachloroethylene | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| Trichloroethylene | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| Acetaldehyde | 0.14 | 0.14 | 0.14 | 0.14 | 0.12 | 0.14 | 0.14 |
| Formaldehyde | 0.18 | 0.15 | 0.11 | 0.10 | 0.13 | 0.14 | 0.13 |
| Arsenic | 0.03 | 0.05 | 0.05 | 0.03 | 0.03 | 0.03 | 0.04 |
| Cadmium | 0.03 | 0.05 | 0.01 | 0.01 | 0.01 | 0.00 | 0.02 |
| Chromium | 0.02 | 0.03 | 0.01 | 0.01 | 0.01 | 0.01 | 0.02 |
| Lead | 0.00 | 0.01 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 |
| Manganese | 0.07 | 0.22 | 0.10 | 0.14 | 0.11 | 0.13 | 0.13 |
| Nickel | 0.01 | 0.02 | 0.01 | 0.00 | 0.00 | 0.01 | 0.01 |
| ***Sum*** | ***0.58*** | ***0.76*** | ***0.54*** | ***0.52*** | ***0.48*** | ***0.55*** | ***0.57*** |

**Figure 3-7: Non-cancer Evaluation (Hazard Quotients) for 6 Monitoring Sites (2001)**



As shown, none of the monitored concentrations exceed a hazard index or hazard quotient of one. Because some chemicals have the same or similar target organ, some hazard indices can be added together. However, even if all chemicals had the same target organ, the resulting hazard index, referred to as a hazard quotient, is approximately 0.76 at the highest location, Georgetown. These results suggest that potential non-cancer health effects associated with the monitored chemicals (with exception of DPM and vegetative burning) alone are not likely to result in significant non-cancer health impacts.

However, these results need to be viewed with caution. The particle-related combustion mixtures, woodsmoke and diesel particulate matter, add a significant amount of $PM_{2.5}$ into the ambient air. The non-cancer health effects associated with fine particles include a wide range of respiratory health effects in humans, and are extensively evaluated elsewhere.[49] More information on non-cancer health impacts are presented in the following chapter on the USEPA NATA project.

[49] USEPA ***Third External Review Draft of Air Quality Criteria for Particulate Matter***. EPA/600/P-99/002ac. Office of Research and Development. Research Triangle Park, NC. April 2002.

# Chapter 4: Air Toxics Modeling: USEPA NATA Project

## *4.1 Overview*

Risks from airborne toxics can also be evaluated and ranked using emission estimates for the primary source categories and dispersion models. USEPA recently completed this type of assessment in their nationwide project entitled the National-scale Air Toxics Assessment (NATA). We use the results from this analysis for King County to evaluate and rank potential risks from airborne toxics. We compare these King County results to those based on the two monitoring studies (see Chapter 3). We also use the results from NATA to evaluate potential cancer risks from the other three counties in the Puget Sound region (Pierce, Kitsap, and Snohomish).

*USEPA NATA Project*

The NATA project consists of four phases. In Phase I, USEPA uses emission factors to calculate emissions for mobile, area, and point source categories for a total of 33 pollutants and DPM. In Phase II, USEPA predicts ambient air concentrations for these pollutants using an air dispersion model (ASPEN, which is explained in more detail later). In Phase III, USEPA predicts human exposure concentrations through the HAPEM4 model, based on the ambient concentrations calculated in Phase II. The HAPEM4 model accounts for individual movements through various micro-environments such as traveling in a vehicle on the highway, living nearer to significant point sources, and remaining indoors for a portion of each day. Finally, in Phase IV, these human exposure concentrations are used to calculate potential cancer risks and non-cancer risks. Details on the methods and results for each of these phases can be found in the USEPA technical support documents for the NATA project. The general approach used in each of the four phases is briefly described below.

## *4.2 Phase I: Emission Inventories*

In Phase I, USEPA calculates emission estimates for each of the 33 pollutants from mobile, area, and point sources. The 33 pollutants are a subset of the 189 hazardous air pollutants (HAPs) listed in the federal Clean Air Act. This subset was determined by an emission inventory

ranking developed by USEPA.[50] This ranking identified 33 chemicals that were expected to contribute the highest risks from airborne toxics. USEPA also added diesel particulate matter to complete the list of 34. Dioxins were originally included in the 33 chemicals, but USEPA recently removed this suite of chemicals. Although coke oven emissions are included in the NATA project, King County does not have any source of this pollutant. Therefore, it is removed from the list. The final list of 32 chemicals used in this analysis is presented in Table 4-1 below:

**Table 4-1: Pollutants Included in the NATA Project**

| Pollutant | CAS Number |
|---|---|
| 1) Acetaldehyde | 75070 |
| 2) Acrolein | 107028 |
| 3) Acrylonitrile | 107131 |
| 4) Arsenic compounds | NA |
| 5) Benzene | 71432 |
| 6) Beryllium compounds | NA |
| 7) 1,3-Butadiene | 106990 |
| 8) Cadmium compounds | NA |
| 9) Carbon tetrachloride | 56235 |
| 10) Chloroform | 67663 |
| 11) Chromium compounds | NA |
| 12) 1,3-Dichloropropene | 542756 |
| 13) Diesel particulate matter (DPM) | NA |
| 14) Ethylene dibromide (1,2-dibromoethane) | 106934 |
| 15) Ethylene dichloride (1,2-dichloroethane) | 107062 |
| 16) Ethylene oxide | 75218 |
| 17) Formaldehyde | 50000 |
| 18) Hexachlorobenzene | 118741 |
| 19) Hydrazine, hydrazine sulfate | 302012 |
| 20) Lead compounds | NA |
| 21) Manganese | NA |
| 22) Mercury compounds | NA |
| 23) Methylene chloride | 75092 |
| 24) Nickel compounds | NA |
| 25) Polychlorinated biphenyls (PCBs) | 1336363 |
| 26) Polycyclic organic matter (POM) | NA |
| 27) Propylene dichloride (1,2-dichloropropane) | 78875 |
| 28) Quinoline | 91225 |
| 29) 1,1,2,2-Tetrachloroethane | 79345 |
| 30) Tetrachloroethylene (perchloroethylene) | 127184 |
| 31) Trichloroethylene (TCE) | 79016 |
| 32) Vinyl chloride | 75014 |

[50] USEPA, July 1999.

In Phase I, USEPA used emission estimates from 1996 inventory reporting and estimates, as listed on the USEPA database referred to as the National Toxic Inventory (NTI). USEPA also used information from the National Emission Trends inventory to supplement information for chemicals that may be formed from pre-cursors in the atmosphere.

In addition, USEPA took several steps to perform quality assurance on the emission estimates. For example, USEPA filled in missing or erroneous information for sources that were missing or poorly reported in the NTI. Emission estimates in NTI are primarily obtained from state and local inventories, USEPA Maximum Achievable Control Technology information, the Toxics Release Inventory, and emissions from USEPA's Office of Transportation and Air Quality. USEPA also requested that individual state and local agencies review emission estimates calculated for the NATA project and submit changes to USEPA before the dispersion-modeling phase was conducted.

USEPA also grouped similar compounds together for more complete evaluation. For example, some chemicals such as various lead or chromium compounds are evaluated together as groups of compounds. In addition, these groups are subdivided according to particle size for more accurate dispersion modeling. Finally, pollutants are assigned to reactivity classes to account for atmospheric decay.

*Source Categories*

Total pollutant emissions are calculated from point sources, mobile sources, and area sources. Major point sources are large stationary sources that emit more than 10 tons per year of any HAP or a cumulative total of 25 tons per year of any combination of the 189 HAPs. Area sources are smaller stationary sources. Some smaller facilities do submit emission inventory reports but the majority of the calculations for area sources are estimated as a ratio to countywide population estimates. USEPA also included other types of area sources such as forest fires and prescribed burning. On-road mobile sources include cars, trucks, buses, etc., while off-road mobile sources include all remaining mobile sources such as trains, boats, lawnmowers, construction vehicles, and aircraft.

### *4.3 Phase II: Predicting Ambient Air Concentrations*

*ASPEN Model*

After the emission estimates are calculated, the information is entered into the USEPA model referred to as the Assessment System for Population Exposure Nationwide (ASPEN) air model. This model essentially combines a Gaussian dispersion model with climatological information for each census tract across the United States. ASPEN considers the rate of release of each chemical, the location of the release, the release height, wind speed, and direction from the nearest meteorological station, weather (e.g., wet and dry deposition), pollutant decay, atmospheric transformation, and general settling.

*Background Concentrations*

USEPA also added a "background concentration" for 13 of the 33 pollutants. These concentrations account for toxics that are due to natural sources (e.g., windblown soils, volcanic eruptions, etc.), sources not included in the emission estimates, and long-range transport. The values included in the analysis as background are typically monitored concentrations in areas that are not heavily impacted by other sources. USEPA refers to these remote areas as "clean air locations." If background concentrations were not available in the literature, the concentrations were assumed to be zero. DPM background concentrations were adopted from modeling exercises. This is described more fully in Appendix F of the NATA Science Advisory report.[51]

### *4.4 Phase III: Predicting Human Exposures*

*HAPEM4 Model*

Predicted ambient concentrations are then entered into another model to account for personal exposures and variation among the population in terms of activities. The model used by USEPA is referred to as the Hazardous Air Pollutant Exposure Model, version 4 (HAPEM4). This model evaluates the long-term inhalation exposures by tracking individuals who are considered to be representative of various demographic groups as they move through different locations. These smaller locations are referred to as "micro-environments." USEPA defines a micro-environment as:

[51] USEPA, 2001. Appendix F.

> *A small space in which human contact with a pollutant takes place, and which can be treated as a well-characterized, relatively homogenous location with respect to pollutant concentrations for a specified time period. MEs include indoors at home, school, work, inside an automobile or bus, outdoors, etc.*

A complete list and more detailed descriptions of each micro-environment are included in the technical support documentation for this model.[52]

The model predicts concentrations in these micro-environments and calculates a time-weighted average depending on the amount of time spent in each micro-location. A total of 37 micro-environments were used in predicting the human exposure concentrations for the NATA project.

The HAPEM4 model includes both population activity pattern data and commuting pattern data. Activity patterns include the amount of time people spend at home, work, or in an automobile along with the activities during that time (e.g., sleeping, eating, etc.). HAPEM4 estimates exposures by activity pattern for various demographic groups as defined by age, gender, or race, etc. The commuting pattern data is based on a 1990 U.S. census tract database that reports the number of individuals who work within the census tract where they live.[53]

Pollutant concentrations within each micro-environment are estimated using ambient concentrations multiplied by a penetration factor, which is a ratio of the indoor to the outdoor concentration. A time-weighted average exposure concentration can be predicted using these factors and the ambient concentration data for a specified amount of time.

In calculating an annual average estimate for the NATA project, USEPA selected 40 demographic 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 micro-environment (for eight separate time blocks for a 24-hour day) for each demographic group was then averaged for the entire set of 365. This process was repeated 100

[52] 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. Prepared by ICF Consulting and TRJ Environmental Inc., Research Triangle Park, NC, May 8, 2000.

[53] USEPA, January 2001.

times for each demographic group so that 100 annual activity patterns were available for each of the 40 groups. For each census tract, 30 of these 100 patterns were randomly selected to represent a typical annual time allocation in each micro-environment for demographic groups in that tract. USEPA notes that this process leads to an annual activity that estimates the average exposure in each group, as opposed to highly sensitive or highly exposed individuals.

### *4.5 Potential Cancer Risks*

Potential cancer risk estimates are presented based on the ASPEN modeling results in Tables 4-2 and 4-3, and Figure 4-1 below. Tables 4-2 and 4-3 present potential cancer risks based on the average concentration and the upper 95$^{th}$ concentration, respectively. We include Figure 4-1 to compare the results more easily. This figure shows the cumulative risks based on the median, average, and upper-bound risk in each county. As shown in Figure 4-1, the median and average risk estimates are very similar, while the upper-bound risk estimates appear greater than the medians and the means. In addition, Kitsap County has the lowest risks, while King County has the highest risks.

In Tables 4-4 and 4-5 we present the exposure concentrations from HAPEM4 and the resulting potential cancer risks, respectively. As noted earlier in this section, the HAPEM4 results only include medians, so we cannot present the range of risks for each chemical. In Figure 4-2, we compare the cumulative potential cancer risks for each county based on the HAPEM4 exposure concentrations. Similar to the results for the ASPEN-based estimates, the potential cancer risks are similar among the four counties, with Kitsap being the lowest and King being the highest.

Finally, in Table 4-6 and Figure 4-3, we compare risks based on the median estimates from ASPEN and HAPEM4 for King County. The cumulative risks for both ASPEN and HAPEM4 are 580 in a million and 419 in a million, respectively. As expected, the risks are reduced when movement among various micro-environments is taken into account, although not dramatically. The only chemicals that appear to present significantly different cancer risks for ASPEN versus HAPEM4 are tetrachloroethylene or perchloroethylene, and PCBs.

**Table 4-2: Average Potential Cancer Risks for Puget Sound Region based on ASPEN (ambient air concentration model) NATA (1996)**

| Chemical | King County | Kitsap County | Pierce County | Snohomish County |
|---|---|---|---|---|
| 1) 1,1,2,2-Tetrachloroethane | 2.35E-09 | 1.26E-08 | 7.13E-09 | 1.21E-08 |
| 2) 1,3-Butadiene | 2.46E-06 | 9.84E-07 | 1.63E-06 | 1.31E-06 |
| 3) 1,3-Dichloropropene | 4.16E-07 | 2.37E-07 | 3.46E-07 | 2.81E-07 |
| 4) 7-PAH | 1.09E-06 | 5.88E-07 | 9.78E-07 | 6.48E-07 |
| 5) Acetaldehyde | 1.96E-06 | 1.27E-06 | 1.70E-06 | 1.16E-06 |
| 6) Acrylonitrile | 6.94E-09 | 3.20E-08 | 1.86E-08 | 2.75E-08 |
| 7) Arsenic Compounds | 4.56E-07 | 1.84E-07 | 4.07E-07 | 1.83E-07 |
| 8) Benzene | 1.83E-05 | 1.15E-05 | 1.58E-05 | 1.34E-05 |
| 9) Beryllium Compounds | 3.05E-08 | 1.45E-08 | 2.42E-08 | 1.73E-08 |
| 10) Cadmium Compounds | 5.81E-08 | 4.14E-08 | 6.59E-08 | 3.78E-08 |
| 11) Carbon Tetrachloride | 1.32E-05 | 1.32E-05 | 1.32E-05 | 1.32E-05 |
| 12) Chloroform | 1.92E-06 | 1.95E-06 | 1.94E-06 | 1.93E-06 |
| 13) Chromium Compounds | 1.74E-05 | 3.92E-05 | 7.04E-06 | 1.10E-05 |
| 14) DPM | 5.31E-04 | 3.54E-04 | 4.62E-04 | 3.84E-04 |
| 15) Ethylene Dibromide | 1.69E-06 | 1.69E-06 | 1.69E-06 | 1.69E-06 |
| 16) Ethylene Dichloride | 1.59E-06 | 1.59E-06 | 1.59E-06 | 1.59E-06 |
| 17) Ethylene Oxide | 1.84E-07 | 7.36E-08 | 2.07E-07 | 7.14E-08 |
| 18) Formaldehyde | 1.57E-05 | 1.08E-05 | 1.35E-05 | 1.08E-05 |
| 19) Hexachlorobenzene | 4.28E-08 | 4.28E-08 | 4.28E-08 | 4.28E-08 |
| 20) Hydrazine | 2.64E-10 | 2.61E-11 | 1.82E-10 | 6.91E-11 |
| 21) Lead Compounds | 1.99E-07 | 4.90E-08 | 4.16E-08 | 3.23E-08 |
| 22) Methylene Chloride | 2.53E-07 | 1.41E-07 | 2.00E-07 | 1.73E-07 |
| 23) Nickel Compounds | 1.93E-06 | 3.16E-06 | 6.67E-07 | 6.14E-07 |
| 24) Perchloroethylene | 1.80E-06 | 1.14E-06 | 1.34E-06 | 1.29E-06 |
| 25) Polychlorinated Biphenyls | 3.88E-08 | 3.86E-08 | 3.81E-08 | 3.82E-08 |
| 26) Polycyclic Organic Matter | 8.64E-06 | 4.15E-06 | 7.15E-06 | 2.97E-06 |
| 27) Propylene Dichloride | 1.36E-10 | 5.09E-10 | 3.10E-10 | 4.62E-10 |
| 28) Quinoline | 7.99E-10 | 1.63E-10 | 5.00E-10 | 2.50E-10 |
| 29) Trichloroethylene | 1.71E-06 | 4.34E-07 | 7.16E-07 | 1.33E-06 |
| 30) Vinyl Chloride | 1.58E-09 | 4.91E-09 | 3.73E-09 | 4.76E-09 |
| Total | 6.22E-04 | 4.47E-04 | 5.32E-04 | 4.48E-04 |

**Table 4-3: 95th Percentile Upper-bound Potential Cancer Risk Estimates based on ASPEN NATA (1996)**

| *Chemical* | King County | Kitsap County | Pierce County | Snohomish County |
|---|---|---|---|---|
| 1) 1,1,2,2-Tetrachloroethane | 3.74E-09 | 1.94E-08 | 1.47E-08 | 2.49E-08 |
| 2) 1,3-Butadiene | 4.74E-06 | 1.69E-06 | 2.66E-06 | 2.04E-06 |
| 3) 1,3-Dichloropropene | 7.80E-07 | 4.72E-07 | 6.12E-07 | 4.92E-07 |
| 4) 7-PAH | 1.85E-06 | 1.01E-06 | 1.55E-06 | 1.00E-06 |
| 5) Acetaldehyde | 2.93E-06 | 1.56E-06 | 2.31E-06 | 1.50E-06 |
| 6) Acrolein | 0.00E+00 | 0.00E+00 | 0.00E+00 | 0.00E+00 |
| 7) Acrylonitrile | 1.02E-08 | 4.92E-08 | 3.73E-08 | 5.66E-08 |
| 8) Arsenic Compounds | 8.86E-07 | 2.71E-07 | 1.06E-06 | 3.71E-07 |
| 9) Benzene | 2.64E-05 | 1.58E-05 | 2.25E-05 | 1.83E-05 |
| 10) Beryllium Compounds | 5.50E-08 | 2.81E-08 | 4.80E-08 | 2.93E-08 |
| 11) Cadmium Compounds | 1.08E-07 | 6.62E-08 | 1.42E-07 | 7.04E-08 |
| 12) Carbon Tetrachloride | 1.32E-05 | 1.32E-05 | 1.32E-05 | 1.32E-05 |
| 13) Chloroform | 1.93E-06 | 1.97E-06 | 1.96E-06 | 1.94E-06 |
| 14) Chromium Compounds | 3.62E-05 | 1.26E-04 | 1.36E-05 | 3.64E-05 |
| 15) Coke Oven Emissions | 0.00E+00 | 0.00E+00 | 0.00E+00 | 0.00E+00 |
| 16) DPM | 8.40E-04 | 4.20E-04 | 8.37E-04 | 6.84E-04 |
| 17) Ethylene Dibromide | 1.69E-06 | 1.69E-06 | 1.69E-06 | 1.69E-06 |
| 18) Ethylene Dichloride | 1.59E-06 | 1.59E-06 | 1.59E-06 | 1.59E-06 |
| 19) Ethylene Oxide | 3.90E-07 | 1.64E-07 | 5.22E-07 | 1.28E-07 |
| 20) Formaldehyde | 2.39E-05 | 1.24E-05 | 1.72E-05 | 1.24E-05 |
| 21) Hexachlorobenzene | 4.28E-08 | 4.28E-08 | 4.28E-08 | 4.28E-08 |
| 22) Hydrazine | 8.04E-10 | 3.90E-11 | 8.58E-10 | 3.18E-10 |
| 23) Lead Compounds | 6.19E-07 | 7.45E-08 | 7.21E-08 | 7.85E-08 |
| 24) Manganese Compounds | 0.00E+00 | 0.00E+00 | 0.00E+00 | 0.00E+00 |
| 25) Mercury Compounds | 0.00E+00 | 0.00E+00 | 0.00E+00 | 0.00E+00 |
| 26) Methylene Chloride | 4.07E-07 | 1.96E-07 | 4.31E-07 | 2.51E-07 |
| 27) Nickel Compounds | 4.18E-06 | 9.89E-06 | 1.57E-06 | 1.32E-06 |
| 28) Perchloroethylene | 2.59E-06 | 1.43E-06 | 2.00E-06 | 1.70E-06 |
| 29) Polychlorinated Biphenyls | 3.95E-08 | 3.92E-08 | 3.82E-08 | 3.84E-08 |
| 30) Polycyclic Organic Matter | 1.50E-05 | 6.77E-06 | 1.40E-05 | 5.02E-06 |
| 31) Propylene Dichloride | 2.20E-10 | 7.77E-10 | 5.91E-10 | 9.69E-10 |
| 32) Quinoline | 1.84E-09 | 2.47E-10 | 1.92E-09 | 6.66E-10 |
| 33) Trichloroethylene | 3.70E-06 | 5.36E-07 | 2.04E-06 | 4.70E-06 |
| 34) Vinyl Chloride | 2.70E-09 | 7.46E-09 | 8.98E-09 | 1.00E-08 |
| Total | 9.83E-04 | 6.17E-04 | 9.38E-04 | 7.88E-04 |

**Figure 4-1: Comparing Ranges of Potential Cancer Risks for Puget Sound based on ASPEN NATA (1996)**

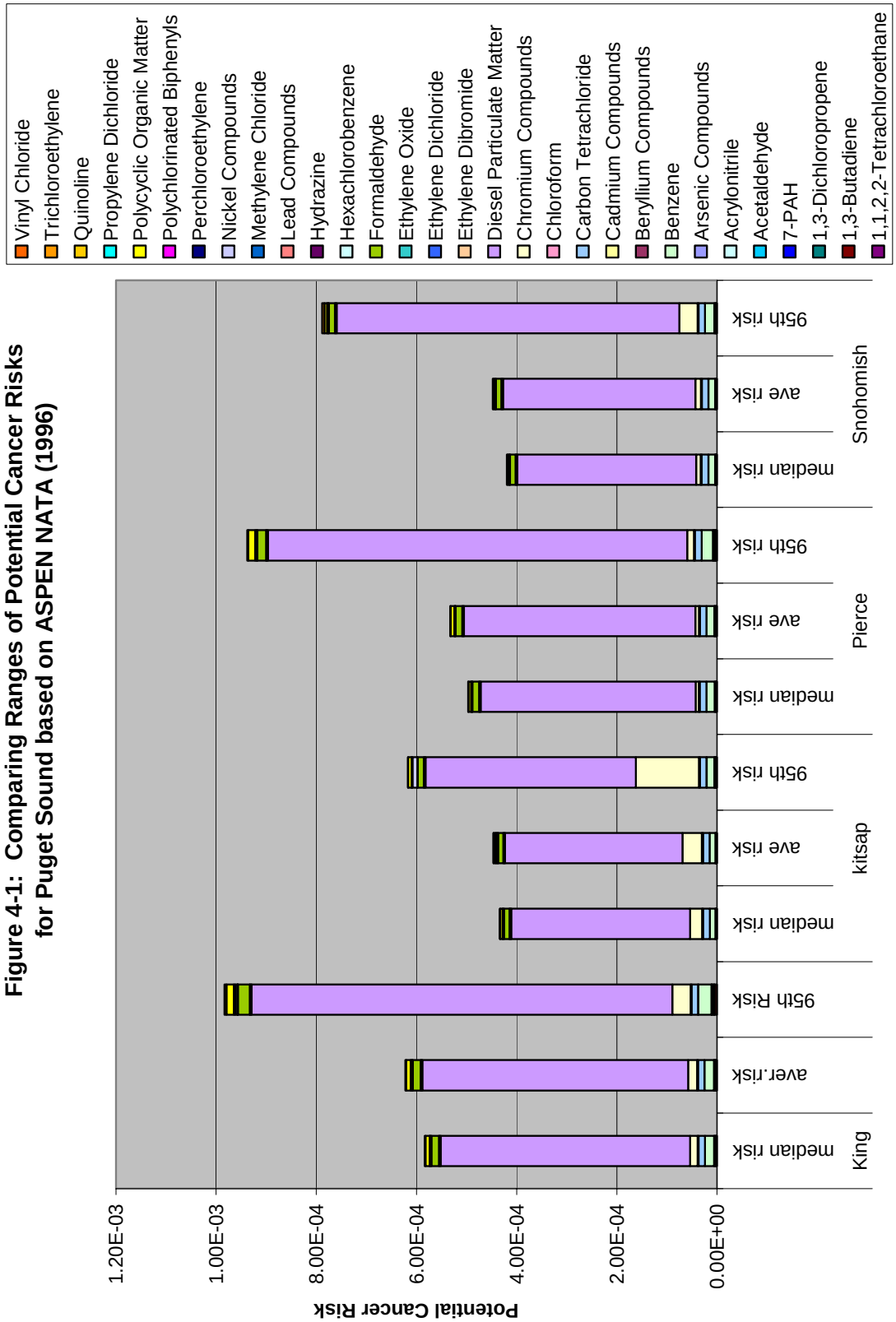


**Table 4-4: Human Median Exposure Concentrations (μg/m$^3$) from HAPEM4**

| Chemical | King County | Kitsap County | Pierce County | Snohomish County |
|---|---|---|---|---|
| | | | | |
| 1) 1,1,2,2-Tetrachloroethane | 3.0E-05 | 1.8E-04 | 4.6E-05 | 1.4E-04 |
| 2) 1,3-Butadiene | 6.4E-02 | 2.8E-02 | 4.7E-02 | 4.1E-02 |
| 3) 1,3-Dichloropropene | 8.0E-02 | 4.6E-02 | 6.9E-02 | 6.1E-02 |
| 4) Acetaldehyde | 7.6E-01 | 5.2E-01 | 6.9E-01 | 4.9E-01 |
| 5) Acrolein | 9.4E-02 | 5.8E-02 | 9.2E-02 | 6.9E-02 |
| 6) Acrylonitrile | 7.3E-05 | 3.7E-04 | 9.6E-05 | 2.5E-04 |
| 7) Arsenic | 7.7E-05 | 3.5E-05 | 5.8E-05 | 3.4E-05 |
| 8) Benzene | 2.3E+00 | 1.4E+00 | 2.0E+00 | 1.8E+00 |
| 9) Beryllium | 9.4E-06 | 4.3E-06 | 7.3E-06 | 5.9E-06 |
| 10) Cadmium | 2.3E-05 | 1.9E-05 | 2.7E-05 | 1.7E-05 |
| 11) Carbon Tetrachloride | 6.4E-01 | 6.4E-01 | 6.4E-01 | 6.4E-01 |
| 12) Chloroform | 6.8E-02 | 6.9E-02 | 6.9E-02 | 6.8E-02 |
| 13) Chromium | 8.4E-04 | 1.4E-03 | 3.7E-04 | 5.3E-04 |
| 14) Diesel PM | 1.2E+00 | 8.4E-01 | 1.0E+00 | 8.7E-01 |
| 15) Ethylene Dibromide | 6.1E-03 | 6.1E-03 | 6.1E-03 | 6.1E-03 |
| 16) Ethylene Dichloride | 5.3E-02 | 5.2E-02 | 5.3E-02 | 5.2E-02 |
| 17) Ethylene Oxide | 1.4E-03 | 5.6E-04 | 1.5E-03 | 6.4E-04 |
| 18) Formaldehyde | 8.9E-01 | 6.7E-01 | 8.2E-01 | 6.7E-01 |
| 19) Hexachlorobenzene | 7.5E-05 | 7.5E-05 | 7.5E-05 | 7.5E-05 |
| 20) Hydrazine | 3.0E-08 | 3.5E-09 | 1.7E-08 | 8.4E-09 |
| 21) Lead | 8.5E-03 | 3.4E-03 | 3.0E-03 | 1.3E-03 |
| 22) Manganese | 9.6E-04 | 4.4E-04 | 7.4E-04 | 7.6E-04 |
| 23) Mercury | 1.3E-03 | 1.4E-03 | 1.3E-03 | 1.3E-03 |
| 24) Methylene Chloride | 4.0E-01 | 2.3E-01 | 3.0E-01 | 3.0E-01 |
| 25) Nickel | 2.7E-03 | 3.4E-03 | 9.3E-04 | 1.0E-03 |
| 26) PCBS | 3.1E-04 | 3.0E-04 | 3.0E-04 | 3.0E-04 |
| 27) Perchloroethylene | 2.3E-01 | 1.5E-01 | 1.8E-01 | 1.8E-01 |
| 28) POM (total) | 1.0E-01 | 6.2E-02 | 7.4E-02 | 4.3E-02 |
| 29) Propylene Dichloride | 5.2E-06 | 2.2E-05 | 6.7E-06 | 1.6E-05 |
| 30) Quinoline | 1.5E-07 | 3.2E-08 | 7.8E-08 | 5.0E-08 |
| 31) Trichloroethylene | 5.9E-01 | 1.8E-01 | 2.5E-01 | 4.6E-01 |
| 32) Vinyl Chloride | 1.2E-04 | 4.4E-04 | 1.9E-04 | 3.4E-04 |

**Table 4-5: Potential Cancer Risks for Puget Sound Clean Air Counties based on HAPEM4 Exposure Estimates (based on median concentrations)**

| Chemical | King County | Kitsap County | Pierce County | Snohomish County |
|---|---|---|---|---|
| | | | | |
| 1) 1,1,2,2-Tetrachloroethane | 1.7E-09 | 1.0E-08 | 2.7E-09 | 8.0E-09 |
| 2) 1,3-Butadiene | 1.9E-06 | 8.3E-07 | 1.4E-06 | 1.2E-06 |
| 3) 1,3-Dichloropropene | 3.2E-07 | 1.8E-07 | 2.8E-07 | 2.4E-07 |
| 4) Acetaldehyde | 1.7E-06 | 1.2E-06 | 1.5E-06 | 1.1E-06 |
| 5) Acrylonitrile | 5.0E-09 | 2.5E-08 | 6.6E-09 | 1.7E-08 |
| 6) Arsenic | 3.3E-07 | 1.5E-07 | 2.5E-07 | 1.5E-07 |
| 7) Benzene | 1.8E-05 | 1.1E-05 | 1.6E-05 | 1.4E-05 |
| 8) Beryllium | 2.2E-08 | 1.0E-08 | 1.8E-08 | 1.4E-08 |
| 9) Cadmium | 4.2E-08 | 3.3E-08 | 4.8E-08 | 3.1E-08 |
| 10) Carbon Tetrachloride | 9.6E-06 | 9.6E-06 | 9.6E-06 | 9.6E-06 |
| 11) Chloroform | 1.6E-06 | 1.6E-06 | 1.6E-06 | 1.6E-06 |
| 12) Chromium | 1.0E-05 | 1.7E-05 | 4.5E-06 | 6.3E-06 |
| 13) Diesel PM | 3.6E-04 | 2.5E-04 | 3.1E-04 | 2.6E-04 |
| 14) Ethylene Dibromide | 1.3E-06 | 1.3E-06 | 1.3E-06 | 1.3E-06 |
| 15) Ethylene Dichloride | 1.4E-06 | 1.4E-06 | 1.4E-06 | 1.4E-06 |
| 16) Ethylene Oxide | 1.2E-07 | 5.0E-08 | 1.3E-07 | 5.6E-08 |
| 17) Formaldehyde | 1.2E-05 | 8.6E-06 | 1.1E-05 | 8.7E-06 |
| 18) Hexachlorobenzene | 3.4E-08 | 3.4E-08 | 3.5E-08 | 3.4E-08 |
| 19) Hydrazine | 1.4E-10 | 1.7E-11 | 8.4E-11 | 4.1E-11 |
| 20) Lead | 1.0E-07 | 4.1E-08 | 3.6E-08 | 1.6E-08 |
| 21) Methylene Chloride | 1.9E-07 | 1.1E-07 | 1.4E-07 | 1.4E-07 |
| 22) Nickel | 1.3E-06 | 1.6E-06 | 4.4E-07 | 4.9E-07 |
| 23) PCBS | 3.1E-08 | 3.0E-08 | 3.0E-08 | 3.0E-08 |
| 24) Perchloroethylene | 1.4E-06 | 8.9E-07 | 1.1E-06 | 1.1E-06 |
| 25) POM (total) | 5.7E-06 | 3.4E-06 | 4.1E-06 | 2.3E-06 |
| 26) Propylene Dichloride | 9.8E-11 | 4.2E-10 | 1.3E-10 | 3.0E-10 |
| 27) Quinoline | 5.0E-10 | 1.1E-10 | 2.7E-10 | 1.7E-10 |
| 28) Trichloroethylene | 1.2E-06 | 3.6E-07 | 4.9E-07 | 9.2E-07 |
| 29) Vinyl Chloride | 1.1E-09 | 3.9E-09 | 1.7E-09 | 3.0E-09 |
| ***Total*** | ***4.3E-04*** | ***3.1E-04*** | ***3.6E-04*** | ***3.1E-04*** |

**Figure 4-2: Potential Cancer Risks in the Puget Sound Region based on HAPEM4 (medians) 1996**

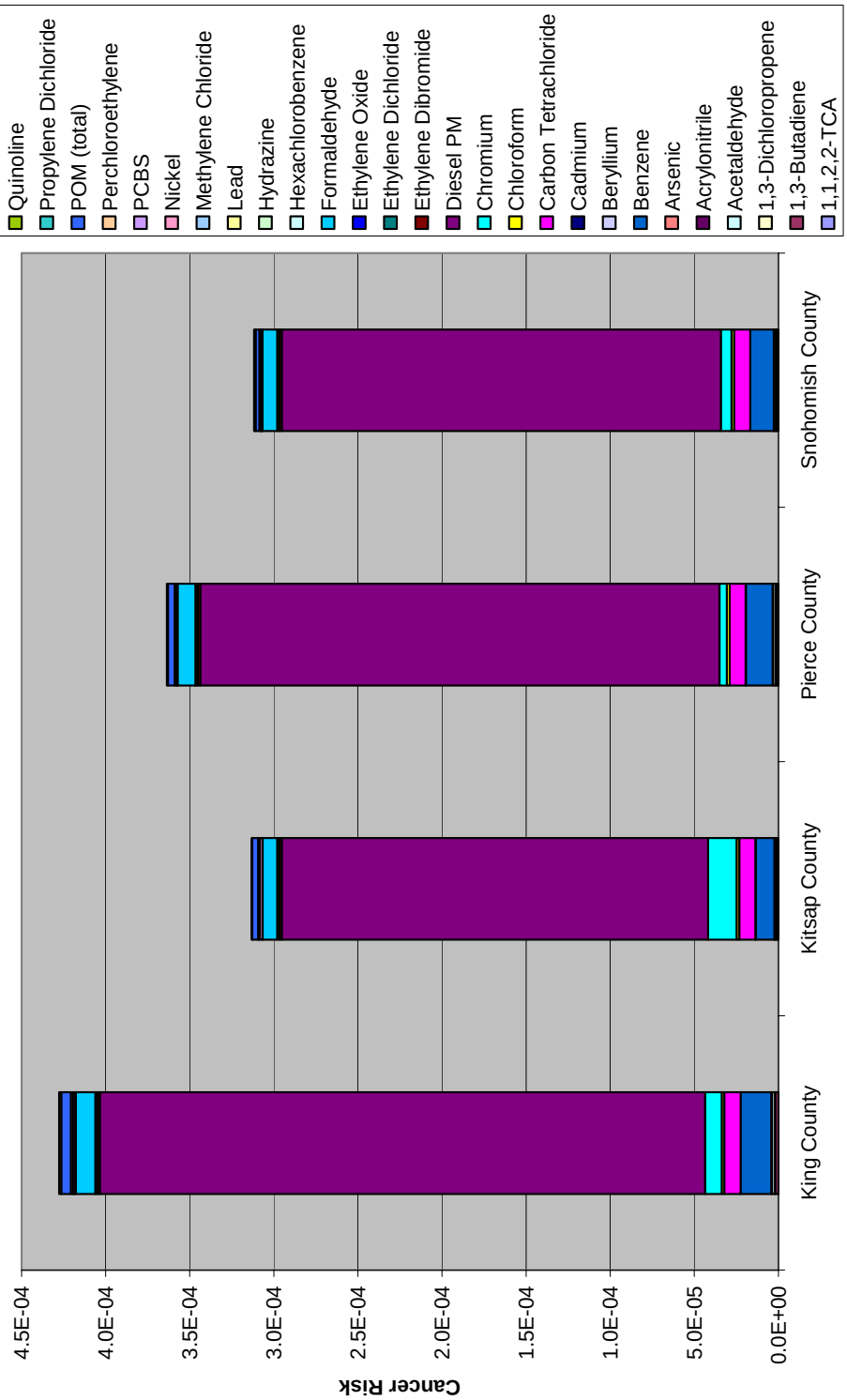
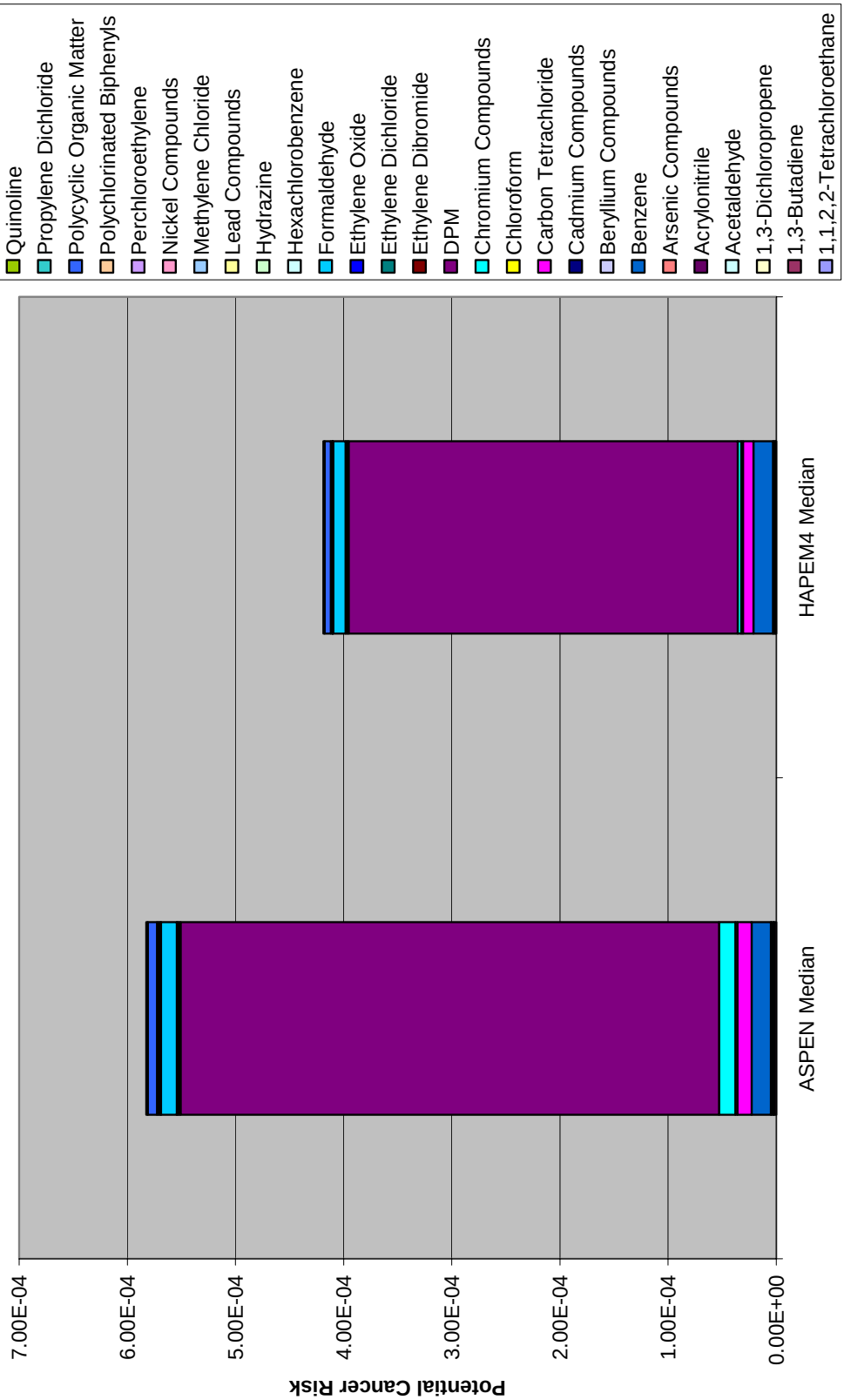


**Table 4-6: Comparing Risks between ASPEN (ambient air) and HAPEM4 (micro-environments) for King County**

| Chemical | ASPEN Median | HAPEM4 Median |
|---|---|---|
| | | |
| 1) 1,1,2,2-Tetrachloroethane | 2.24E-09 | 1.74E-09 |
| 2) 1,3-Butadiene | 2.15E-06 | 6.42E-07 |
| 3) 1,3-Dichloropropene | 3.94E-07 | 3.2E-07 |
| 4) Acetaldehyde | 1.91E-06 | 1.67E-06 |
| 5) Acrylonitrile | 6.69E-09 | 4.97E-09 |
| 6) Arsenic Compounds | 4.20E-07 | 3.31E-07 |
| 7) Benzene | 1.79E-05 | 1.82E-05 |
| 8) Beryllium Compounds | 2.83E-08 | 2.24E-08 |
| 9) Cadmium Compounds | 5.38E-08 | 4.22E-08 |
| 10) Carbon Tetrachloride | 1.32E-05 | 9.6E-06 |
| 11) Chloroform | 1.92E-06 | 1.57E-06 |
| 12) Chromium Compounds | 1.49E-05 | 3.43E-06 |
| 13) DPM | 4.98E-04 | 0.00036 |
| 14) Ethylene Dibromide | 1.69E-06 | 1.34E-06 |
| 15) Ethylene Dichloride | 1.59E-06 | 1.37E-06 |
| 16) Ethylene Oxide | 1.53E-07 | 1.22E-07 |
| 17) Formaldehyde | 1.46E-05 | 1.15E-05 |
| 18) Hexachlorobenzene | 4.28E-08 | 3.45E-08 |
| 19) Hydrazine | 1.70E-10 | 1.45E-10 |
| 20) Lead Compounds | 1.20E-07 | 1.02E-07 |
| 21) Methylene Chloride | 2.32E-07 | 1.9E-07 |
| 22) Nickel Compounds | 1.65E-06 | 3.22E-07 |
| 23) Perchloroethylene | 1.72E-06 | 3.06E-08 |
| 24) Polychlorinated Biphenyls | 3.87E-08 | 1.37E-06 |
| 25) Polycyclic Organic Matter | 8.42E-06 | 5.7E-06 |
| 26) Propylene Dichloride | 1.26E-10 | 9.81E-11 |
| 27) Quinoline | 6.66E-10 | 4.99E-10 |
| 28) Trichloroethylene | 1.39E-06 | 1.18E-06 |
| 29) Vinyl Chloride | 1.43E-09 | 1.07E-09 |
| Total | 5.82E-04 | 4.19E-04 |

**Figure 4-3: Comparing ASPEN Median Risks with HAPEM4 Median Risks for King County based on NATA (1996)**



### *4.6 Potential Non-cancer Risks*

Table 4-7 presents hazard indices for the range of ambient concentrations predicted by ASPEN. These values are presented not only for the average ambient concentration but for the 75$^{th}$, 90$^{th}$, and 95$^{th}$ percentile concentrations as well. The HI associated with the upper percentile concentrations are presented to show the range of potential non-cancer risks. Since we do not have 24-hour average concentrations that are typically compared to the RfC, a conservative or health protective estimate can be derived using the upper-bound concentration. These concentration ranges are not available for the monitored concentrations or the HAPEM4 (human exposure model) results, so they are not presented in those corresponding sections.

As shown in this table, the hazard indices for most of the 32 chemicals are less than 1.0 for the range of concentrations predicted across King County using the ASPEN model. The only chemical that appears to present a potential non-cancer health risk is acrolein, which has an average hazard index of 6 but could be as high as 12 or higher. Ambient concentrations of acrolein could not be verified through monitoring results, so these estimates are considered uncertain. The RfC for this chemical is based on irritation effects in the nasal epithelium, although exposure is also associated with irritation of the larynx, trachea, and lungs.[54]

Although this type of analysis indicates that acrolein is the only chemical of those modeled that should be of concern from a non-cancer perspective, it is important to note that the non-cancer health effects associated with particulate matter (e.g., woodsmoke and diesel particulate matter) have not been adequately evaluated using this method. The association between human health effects, such as increased respiratory effects and increased mortality, and ambient exposures to particulate matter are well documented in the literature.[55] As a result, the hazard index for diesel particulate matter should be viewed as only part of the more complex particulate matter non-cancer risk.

[54] USEPA IRIS file for acrolein. Downloaded February 2002.

[55] USEPA *Air Quality Criteria for Particulate Matter (Second External Review Draft)* EPA 600/P-99/002aB, bB, March 2001.

Hazard indices based on the range of exposure concentrations predicted using the HAPEM4 model could not be calculated at this time. USEPA has indicated that they will provide the range of exposure concentrations from the HAPEM4 model results at a later date. We expect to evaluate these concentrations by calculating hazard indices when this information becomes available.

**Table 4-7: Hazard Indices for ASPEN ambient estimates in King County**

| Pollutant | HI for average | HI for 75th | HI for 90th | HI for 95th |
|---|---|---|---|---|
| 1) Acetaldehyde | 0.1 | 0.1 | 0.1 | 0.1 |
| 2) *Acrolein* | *6.0* | *6.5* | *8.2* | *11.7* |
| 3) Acrylonitrile | 0.0 | 0.0 | 0.0 | 0.0 |
| 4) Arsenic Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 5) Benzene | 0.0 | 0.0 | 0.1 | 0.1 |
| 6) Beryllium Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 7) 1,3-Butadiene | 0.0 | 0.0 | 0.0 | 0.0 |
| 8) Cadmium Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 9) Carbon Tetrachloride | 0.0 | 0.0 | 0.0 | 0.0 |
| 10) Chloroform | 0.0 | 0.0 | 0.0 | 0.0 |
| 11) Chromium Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 12) 1,3-Dichloropropene | 0.0 | 0.0 | 0.0 | 0.0 |
| 13) Ethylene Dibromide | 0.0 | 0.0 | 0.0 | 0.0 |
| 14) Ethylene Dichloride | 0.0 | 0.0 | 0.0 | 0.0 |
| 15) Ethylene Oxide | 0.0 | 0.0 | 0.0 | 0.0 |
| 16) Formaldehyde | 0.1 | 0.1 | 0.2 | 0.2 |
| 17) Hexachlorobenzene | 0.0 | 0.0 | 0.0 | 0.0 |
| 18) Hydrazine | 0.0 | 0.0 | 0.0 | 0.0 |
| 19) Lead Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 20) Manganese Compounds | 0.0 | 0.1 | 0.1 | 0.1 |
| 21) Mercury Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 22) Methylene Chloride | 0.0 | 0.0 | 0.0 | 0.0 |
| 23) Nickel Compounds | 0.0 | 0.0 | 0.0 | 0.0 |
| 24) Perchloroethylene | 0.0 | 0.0 | 0.0 | 0.0 |
| 25) Polychlorinated Biphenyls | 0.0 | 0.0 | 0.0 | 0.0 |
| 26) Polycyclic Organic Matter | 0.0 | 0.0 | 0.0 | 0.0 |
| 27) 7-PAH | 0.0 | 0.0 | 0.0 | 0.0 |
| 28) Propylene Dichloride | 0.0 | 0.0 | 0.0 | 0.0 |
| 29) Quinoline | 0.0 | 0.0 | 0.0 | 0.0 |
| 30) 1,1,2,2-Tetrachloroethane | 0.0 | 0.0 | 0.0 | 0.0 |
| 31) Trichloroethylene | 0.0 | 0.0 | 0.0 | 0.0 |
| 32) Vinyl Chloride | 0.0 | 0.0 | 0.0 | 0.0 |
| ***Total Hazard Quotient*** | ***6.2*** | ***6.8*** | ***8.7*** | ***12.2*** |

## Chapter 5: Summary and Conclusions

We evaluated cancer and non-cancer risks using three different methods of estimating potential exposures.

*Greater Seattle Monitored Ambient Concentrations, and Modeled DPM and Woodsmoke*

Although this method includes the fewest number of compounds, it shows some of the highest potential cancer risks. The average risks range from a low of 57 in a million in Lake Sammamish to a high of 620 in a million at Beacon Hill. The high values at Beacon Hill reflect the fact that we include DPM and woodsmoke in these risk estimates. Excluding DPM and woodsmoke, Lake Sammamish shows the lowest potential cancer risk at 59 in a million and Georgetown the highest at 104 in a million for the greater Seattle area. Although Georgetown is almost twice as high as Lake Sammamish, these differences become less compelling when compared with potential cancer risks that include DPM and woodsmoke, which approach 500 in a million (at Beacon Hill).

Average cancer risks from all monitored sites combined (approximately 73 in a million are comparable to those at Beacon Hill (approximately 80 in a million). Similarly, Beacon Hill average cancer risks including DPM (approximately 500 in a million), are less but within an order of magnitude of cancer risks (approximately 600 in a million) based on NATA ASPEN results for King County. This suggests that the modeled values are reasonable estimates of risk, and could be used when monitored values are not available.

In addition, none of the hazard indices for any of the chemicals monitored in the Seattle Monitoring Study exceed one. However, these results should be viewed with caution because they do not evaluate non-cancer health effects associated with DPM or woodsmoke.

*Modeled ambient concentrations from USEPA NATA*

USEPA presented a range of modeled ambient concentrations from NATA. Similarly, our assessment presents a range of potential cancer risks from air toxics in all four counties in the Puget Sound region. For all toxics combined, average cumulative cancer risks based on ambient concentrations range from approximately 400 in a million for Kitsap and Snohomish Counties to

approximately 600 in a million for King County. Pierce County is approximately 500 in a million. These values include DPM as a whole mixture but only include a small subset of chemicals in the woodsmoke mixture. Although more populated counties such as King and Pierce have somewhat higher potential cancer risks, all four counties are well above the one in a million risk goal for Superfund, and the one-in-a-million to one-in-ten-thousand risk range commonly used by USEPA.[56]

*Modeled human exposure concentrations from USEPA NATA*

The results from the HAPEM4 modeling exercise in NATA show cumulative risks of approximately 400 in a million. Because the NATA project only provides median exposure concentrations from HAPEM4, we can only calculate risks associated with the median values. However, the median values for the ASPEN modeling are approximately 580 in a million. The greatest difference in individual chemical risk estimates between the two appears to be due to DPM. This may be due to the fact that DPM is a particle rather than a gas, and may not penetrate as easily from ambient air to micro-environments.

For all methods, the cumulative cancer risks that include DPM range from a low of 400 in a million (NATA HAPEM4) to a high of 600 in a million for King County (NATA ASPEN). All risk estimates reflect a 70-year exposure period.

### *5.1 Priority Chemicals*

The air toxics that contribute most to the cancer risks are very consistent across the different methods of analysis. The top toxics for all three methods include DPM, benzene, formaldehyde, carbon tetrachloride, and chromium compounds. Woodsmoke also contributed to the cumulative risk estimates based on the monitored data.

In addition, the percent contribution of the top air toxics is also very similar across the different methods of analysis. For example, at Beacon Hill, diesel particulate matter accounts for 79% of the potential cancer risk. ASPEN and HAPEM4 results also show DPM as the top toxic

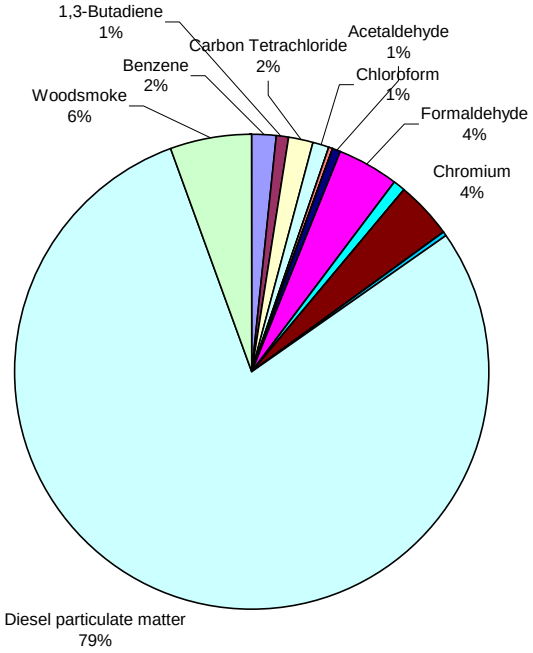
[56] USEPA. ***Residual Risk Report to Congress.*** Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA – 453/R-99-001. March 1999.

comprising approximately 85% of the total risk. The remaining air toxics are primarily formaldehyde, benzene, acetaldehyde, and 1,3-butadiene in all three methods, which are all related to mobile sources. Chromium and carbon tetrachloride also contribute, but appear to be due to area and major sources.[57] Woodsmoke contributes approximately 6% of the risk from Beacon Hill, although it is difficult to say how much it contributes to the NATA estimates because it is not specifically noted. If woodsmoke is included in the POM estimate, NATA results could indicate this category contributes approximately 1% of the total.

*Persistent Bioaccumulative Toxics*

We examined a limited number of PBTs through the inhalation pathway, and only arsenic and cadmium appear to be possible priority chemicals. However, both DPM and woodsmoke contain numerous PAHs, and should be considered potential sources of PBTs in our region. In addition, the Agency may wish to consider further study into possible air emission sources for PBTs and resulting potential risks through the ingestion pathway.

**Figure 5-1: Potential Cancer Risks based on Monitoring/modeling at Beacon Hill (2001)**




[57] USEPA NATA printout for King County, Snohomish County, Pierce County and Kitsap County. See NATA website. http://www.epa.gov/ttn/atw/nata/.

**Figure 5-2: Potential Cancer Risks based on NATA ASPEN results for King County (1996)**

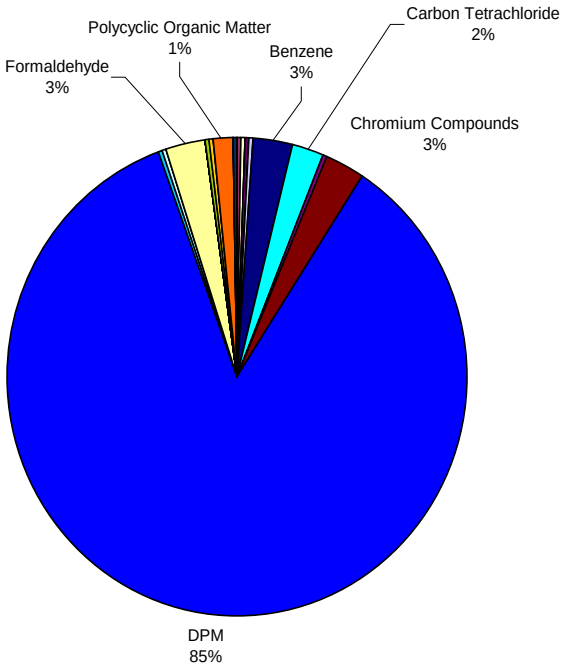
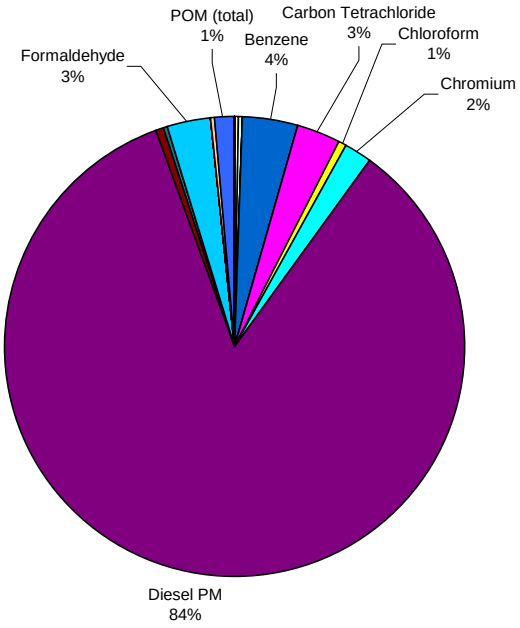



**Figure 5-3: Potential Cancer Risks based on NATA HAPEM4 for Kitsap County (1996)**



### *5.2 Uncertainties*

Although the modeled concentrations provide the highest cancer risk estimates, these values are likely to underestimate the risks from air toxics. The ASPEN model appears to underestimate ambient concentrations in the King County area because the modeling reflects ambient concentrations across the entire county, which includes less impacted areas. In addition, the emissions inventory does not include all sources and may underestimate emissions for those sources that are represented. Finally, the model may not adequately consider production of HAPs from atmospheric transformation reactions. However, the NATA ambient concentrations (ASPEN) result in larger cancer risk estimates than the monitored estimates because they include a larger number of air toxics than the monitoring studies. This suggests that if the list of air toxics were more comprehensive, the overall estimated cancer risks could increase, although it is difficult to say by how much.

Because all the risk values in this assessment are based on annual average or median exposure concentrations, which are combined with conservative toxicity estimates, they are expected to be reasonable high-end risk estimates but not maximum risk estimates. For some chemicals, the values may underestimate potential cancer risks for some individuals. 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 large point sources may experience higher risks due to those emissions.

Alternatively, much of the air monitoring and human behavioral information suggests that potential cancer risks may not vary dramatically across the county. For example, the monitoring results suggest that average ambient concentrations for a variety of toxics do not appear to vary significantly among different areas of the county.

Finally, the cancer risk estimates for diesel particulate matter also have some uncertainties associated with them. Although USEPA has not recommended a final unit risk factor for evaluating potential cancer risks associated with environmental exposures to diesel particulate matter, they state strongly that diesel particulate matter is a probable human carcinogen. In addition, USEPA encourages states to consider further the possible range of potential cancer

risks associated with those levels predicted using the NATA results. In the NATA document, USEPA states[58]

> *Even the lower end of the risk range (presented in the risk perspectives section of the Diesel Exhaust HAD) is above the level that has historically warranted regulatory concern at USEPA for air toxics. The Agency believes that areas of the U.S. that have relatively higher annual exposure levels for diesel exhaust, certainly those counties and States with annual exposure levels above 2 micrograms per cubic meter, should consider the scientific judgments that the Agency has made in the risk perspectives section of the HAD while considering the important limitations in their efforts to compare air toxics risks and set priorities for their programs. At the higher exposure levels found in a number of urban areas in NATA, there is an overlap between what the occupational levels were in the epidemiological studies that EPA considered and the environmentally equivalent exposures.*

Overall, this information suggests that ambient air toxics could contribute significantly to cancer and non-cancer risks in the Puget Sound region. It is possible that these risks are underestimated because (1) not all air toxics are considered in this analysis, and (2) many chemicals have been shown to accumulate in indoor micro-environments, which could increase exposure. Alternatively, risk may be overestimated by assuming that the concentration at the monitor accurately reflects lifetime exposure to ambient pollutants. It is important to note that this analysis does not evaluate indoor sources of air pollution (i.e., from paints, home furnishings, cleaning products, building materials, and other indoor sources). Uncertainties in the toxicity information could also serve to over- or underestimate potential risk estimates.

### *5.3 Conclusion*

The information presented in this report uses screening risk estimates to focus Agency attention on those compounds and mixtures that are likely to present the greatest risk of cancer and some non-cancer effects. DPM ranks high in potential contributions to cancer risk, higher than other air toxics measured in this study. In addition, volatile organics associated with mobile sources such as benzene, formaldehyde, and 1,3-butadiene contribute significantly to the potential cancer risks from air toxics. Woodsmoke could also contribute to the overall potential cancer risk from air toxics in the Puget Sound region.

[58] USEPA 2001 pg 102.

DPM, benzene, 1,3-butadiene, and formaldehyde are all classified as class A or B carcinogens under the USEPA cancer rating system. This indicates that USEPA is relatively confident that these chemicals probably cause cancer in humans. These chemicals should have high priority during development of an air toxics reduction program for the Puget Sound area.

Finally, acrolein appears to present a potential non-cancer risk as well. As stated earlier, the non-cancer health effects associated with the particulate-matter combustion mixtures (e.g., woodsmoke and diesel particulate matter) are not adequately evaluated here, and are extensively evaluated in other analyses.

In addition, these analyses suggest that the ambient concentration estimates from mobile sources are predicted with reasonable accuracy, and that they can be used in the absence of more accurate monitoring data, particularly in urban areas. This conclusion may not apply to model results for more rural areas, particularly if outdoor or agricultural burning could contribute substantially to ambient PM or air toxics concentrations. We also recommend additional review of the HAPEM4 results in future NATA analyses, and further research for exposure models that may more accurately predict potential exposures to air toxics.