

# Health Consultation

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SUMMARY OF RESULTS OF THE DUWAMISH VALLEY  
REGIONAL MODELING AND HEALTH RISK ASSESSMENT

SEATTLE, WASHINGTON

JULY 14, 2008

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Agency for Toxic Substances and Disease Registry  
Division of Health Assessment and Consultation  
Atlanta, Georgia 30333

## **Health Consultation: A Note of Explanation**

An ATSDR health consultation is a verbal or written response from ATSDR to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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HEALTH CONSULTATION

SUMMARY OF RESULTS OF DUWAMISH VALLEY  
REGIONAL MODELING AND HEALTH RISK ASSESSMENT

SEATTLE, WASHINGTON

Prepared By:

Washington Department of Health Services  
Office of Environmental and Occupational Toxicology  
Under Cooperative Agreement with the  
The U.S. Department of Health and Human Services  
Agency for Toxic Substances and Disease Registry

## Foreword

The Washington State Department of Health (DOH) has prepared this health consultation in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR is part of the U.S. Department of Health and Human Services and is the principal federal public health agency responsible for health issues related to hazardous waste. This health consultation was prepared in accordance with methodologies and guidelines developed by ATSDR.

The purpose of this health consultation is to identify and prevent harmful human health effects resulting from exposure to hazardous substances in the environment. Health consultations focus on specific health issues so that DOH can respond to requests from concerned residents or agencies for health information on hazardous substances. DOH evaluates sampling data collected from a hazardous waste site, determines whether exposures have occurred or could occur, reports any potential harmful effects, and recommends actions to protect public health. The findings in this report are relevant to conditions at the site during the time of this health consultation, and should not necessarily be relied upon if site conditions or land use changes in the future.

For additional information or questions regarding DOH or the contents of this health consultation, please call the health advisor who prepared this document:

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For persons with disabilities this document is available on request in other formats. To submit a request, please call 1-800-525-0127 (voice) or 1-800-833-6388 (TTY/TDD).

For more information about ATSDR, contact the ATSDR Information Center at 1-888-422-8737 or visit the agency's Web site: [www.atsdr.cdc.gov/](http://www.atsdr.cdc.gov/).

## Glossary

<b>Acute</b>	Occurring over a short time [compare with <b>chronic</b> ].
<b>Agency for Toxic Substances and Disease Registry (ATSDR)</b>	The principal federal public health agency involved with hazardous waste issues, responsible for preventing or reducing the harmful effects of exposure to hazardous substances on human health and quality of life. ATSDR is part of the U.S. Department of Health and Human Services.
<b>Cancer Slope Factor</b>	A number assigned to a cancer causing chemical that is used to estimate its ability to cause cancer in humans.
<b>Carcinogen</b>	Any substance that causes cancer.
<b>Chronic</b>	Occurring over a long time (more than 1 year) [compare with <b>acute</b> ].
<b>Contaminant</b>	A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.
<b>Dermal Contact</b>	Contact with (touching) the skin (see route of exposure).
<b>Dose (for chemicals that are not radioactive)</b>	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
<b>Environmental Protection Agency (EPA)</b>	United States Environmental Protection Agency.
<b>Epidemiology</b>	The study of the occurrence and causes of health effects in human populations. An epidemiological study often compares two groups of people who are alike except for one factor, such as exposure to a chemical or the presence of a health effect. The investigators try to determine if any factor (i.e., age, sex, occupation, economic status) is associated with the health effect.
<b>Exposure</b>	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [ <b>acute exposure</b> ], of intermediate duration, or long-term [ <b>chronic exposure</b> ].

<b>Ingestion</b>	The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].
<b>Ingestion rate</b>	The amount of an environmental medium that could be ingested typically on a daily basis. Units for IR are usually liter/day for water, and mg/day for soil.
<b>Inhalation</b>	The act of breathing. A hazardous substance can enter the body this way [see <b>route of exposure</b> ].
<b>Inorganic</b>	Compounds composed of mineral materials, including elemental salts and metals such as iron, aluminum, mercury, and zinc.
<b>Lowest Observed Adverse Effect Level (LOAEL)</b>	The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
<b>No Observed Adverse Effect Level (NOAEL)</b>	The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.
<b>Oral Reference Dose (RfD)</b>	An amount of chemical ingested into the body (i.e., dose) below which health effects are not expected. RfDs are published by EPA.
<b>Organic</b>	Compounds composed of carbon, including materials such as solvents, oils, and pesticides that are not easily dissolved in water.
<b>Plume</b>	A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.
<b>Route of exposure</b>	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
<b>Volatile organic compound (VOC)</b>	Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

## Summary and Statement of Issues

Residents of Georgetown and South Park neighborhoods in south Seattle asked the Washington State Department of Health (DOH) to conduct an assessment of pollutant impacts on their health. DOH hired a consultant to model air emissions from multiple sources in south Seattle. The objective of the multiple source air modeling project in the Duwamish Valley was to identify air pollutants, key air pollution sources impacting residential areas of south Seattle, and the geographic areas of south Seattle that are impacted by air pollutants. This effort was not intended to be the final step of the assessment process, but an initial step to identify priorities for future work in the area. The purpose of this health consultation is to summarize key findings of the modeling effort while acknowledging the limitations of the work, and make recommendations for future actions.

## Background

The Duwamish Valley is an industrial area located south of downtown Seattle, Washington along the Lower Duwamish Waterway. The Lower Duwamish Waterway was created by the U.S. Army Corps of Engineers by straightening and dredging the Duwamish River to enable barge traffic. The majority of land use along the Duwamish Waterway and in the Duwamish Valley is commercial or industrial with the exception of the two residential communities of Georgetown and South Park (Figure 1).

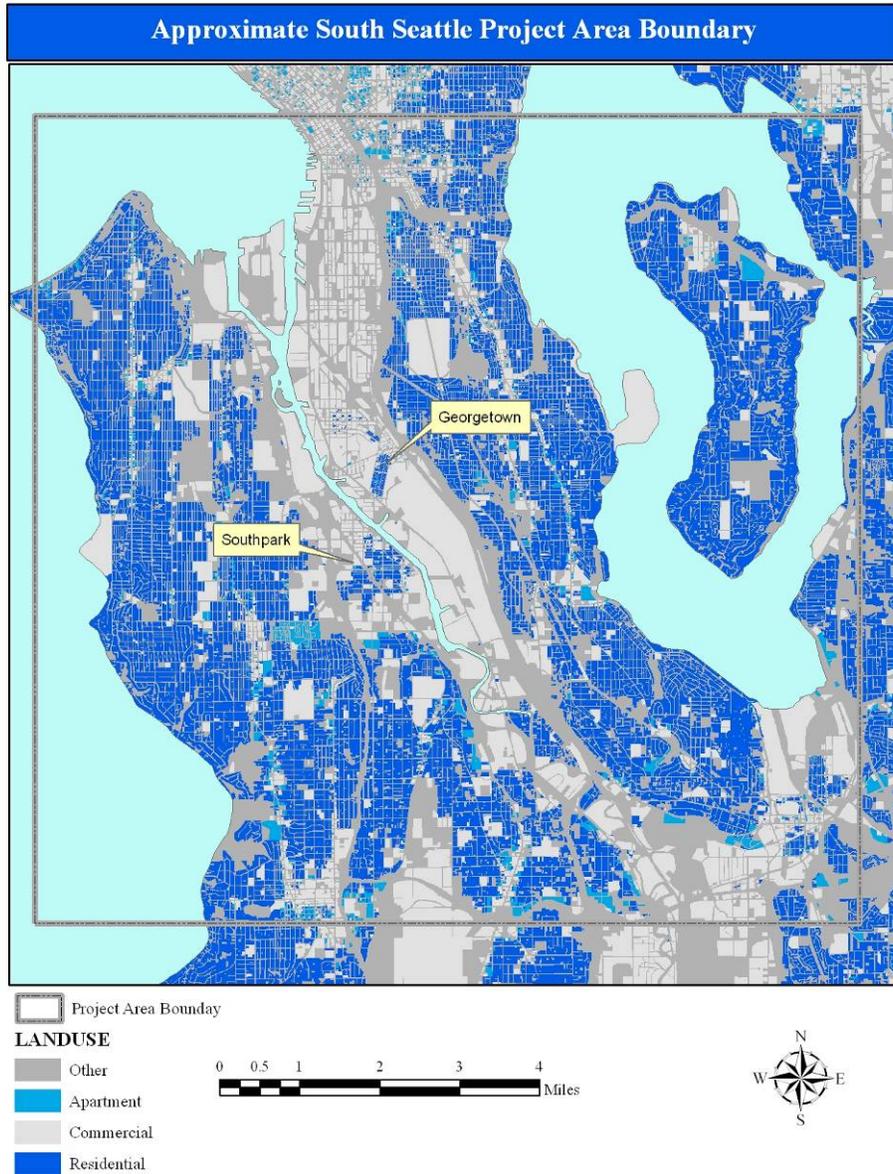
Residents of South Park and Georgetown have voiced numerous concerns related to chemical pollution and air quality including odors, headaches, respiratory problems, miscarriage, nausea, blurry vision, fatigue, cancer and environmental justice.<sup>a</sup> In 1996, a community group, 98108 We Are Ready Now, or 98108 WARN (98108 is the zip code for South Park and Georgetown), petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) to conduct an area-wide assessment of hazardous pollutants in south Seattle.<sup>b</sup> ATSDR was unable to conduct the area-wide assessment, but negotiated with the community to select eight sites perceived by the community to potentially impact health. DOH, ATSDR's cooperative agreement partner, evaluated potential health impacts posed by these eight sites and concluded that sites by themselves may not impact residential areas at levels of health concern, but may contribute in concert with other sources to create an unhealthy environment. This is especially true in areas where multiple sources are densely located in one area.

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<sup>a</sup> Environmental justice is a term used to describe the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies

<sup>b</sup> The community group 98108 WARN became inactive, and the petition was later made by the Community Coalition for Environmental Justice

**Figure 1.** Land use in South Seattle



### **Air pollution – general**

Air pollution is/ or can be associated with a variety of health effects including respiratory tract irritation, asthma, heart and lung diseases, decreased immunity, and increased risk of cancer. The very young and very old are particularly sensitive to air pollution. Most healthy people recover from the effects of air pollution when air quality improves. However, people with existing lung and heart diseases, (such as asthma, chronic obstructive pulmonary disease, previous heart attack, angina, coronary heart disease, or congestive heart failure) and diabetics are at health risks from either short-term or long-term exposure to air pollution.

Air pollution in Washington comes primarily from local sources, not from sources in other states. The Washington State Department of Ecology (Ecology) estimated that motor vehicles accounted for about 55% of air pollution (Washington State Department of Ecology, 2003).

Other significant sources of air pollution included industrial emissions (13%), woodstoves and fireplaces (9%), and outdoor burning (4%). Lawnmowers, boats and other recreational vehicles, aircraft, and trains accounted for the remaining 19%.

Statewide, there are more than 60 active monitoring stations designed to measure regional air quality (EPA, 2007a). The majority of these monitors collect information about particulate matter (e.g., dust and soot) levels in air. Currently, three monitors are sited in south Seattle.

### Air monitoring in south Seattle

In south Seattle, air monitors are or have been located at Beacon Hill, South Park, Georgetown, Duwamish, and Harbor Island (Figure 2). Monitors are mostly designed to analyze or detect criteria air pollutants, but air toxics have been monitored on occasion. Criteria air pollutants and air toxics are defined below.

**Table 1.** Air monitor locations and pollutants measured in south Seattle (EPA, 2007).

Monitor Location	Pollutants	Active years
Beacon Hill	CO	1979-present
	PM <sub>2.5</sub>	
	PM <sub>10</sub>	
	SO <sub>2</sub>	
	NO <sub>x</sub>	
	O <sub>3</sub>	2000-current
	PM <sub>2.5</sub> speciation Select Air Toxics	2000-current
Duwamish	PM <sub>2.5</sub>	1971-present
	PM <sub>10</sub>	
	PM <sub>2.5</sub> speciation	2002-2007
Georgetown	CO, NO <sub>x</sub> , Select Air Toxics	2000-2006
	PM <sub>2.5</sub> speciation	2000-2004
South Park	PM <sub>2.5</sub> , PM <sub>10</sub>	1977-present
Harbor Island	PM <sub>10</sub> Lead	1974-1997

CO – carbon monoxide

PM<sub>2.5</sub> – particulate matter less than 2.5 microns in diameter

PM<sub>10</sub> – particulate matter less than 10 microns in diameter

SO<sub>2</sub> – sulfur dioxide

NO<sub>x</sub> – oxides of nitrogen

O<sub>3</sub> – ozone

PM<sub>2.5</sub> speciation – metals, ions, and carbon constituents of particulate matter less than 2.5 microns in diameter (<http://www.epa.gov/tnamti1/files/ambient/pm25/spec/spec997.pdf>)

**Figure 2.** Air monitor locations (past and current) in south Seattle (EPA, 2007a)



### *Criteria Air Pollutants*

The U.S. EPA classifies six pollutants, carbon monoxide (CO), lead (Pb), nitrogen dioxide (NO<sub>2</sub>), ozone (O<sub>3</sub>), particulate matter (PM), and sulfur dioxide (SO<sub>2</sub>), as criteria air pollutants because they are common, come from many different sources, and may cause harm to human health, the environment, and property. Under the Federal Clean Air Act, EPA establishes health-based standards called National Ambient Air Quality Standards (NAAQS) for criteria air pollutants. Ecology regularly monitors criteria pollutants at numerous locations in King County and the state of Washington. The Puget Sound Clean Air Agency (Clean Air) monitors for fine particulate matter (PM<sub>2.5</sub>) at several locations in its 4-county jurisdiction (King, Snohomish, Pierce, and Kitsap) (Puget Sound Clean Air Agency 2007a).

Currently, three active monitoring stations are located near the Duwamish Valley. In the past, monitors were also located at Georgetown and Harbor Island. Table 1 shows station names and pollutants measured at each monitoring station in south Seattle. Prior to 2005, the Puget Sound region had been in attainment for all criteria pollutants for nearly a decade (Puget Sound Clean Air Agency, 2006). EPA strengthened its daily PM<sub>2.5</sub> standard in 2006. A few areas in the Puget Sound region have concentrations close to violating this stricter standard. These include the Duwamish area in south Seattle as well as the Marysville area in Snohomish County. Concentrations at a monitor in Tacoma's south end violate the stricter PM<sub>2.5</sub> standard, and the area will be designated in non-attainment by EPA (EPA, 2007b).

### *Air Toxics*

Washington State identifies over 400 chemicals as air toxics (Washington Administrative Code, 173-460). These same air toxics are acknowledged by Clean Air (Puget Sound Clean Air Agency 2007b) Examples of air toxics include benzene, which is found in gasoline; perchlorethylene, which is emitted from some dry cleaning facilities; and methylene chloride, which is used as a solvent and paint stripper by a number of industries. Examples of other listed air toxics include dioxin, asbestos, toluene, and metals such as cadmium, mercury, chromium, and lead compounds. These pollutants are known or suspected to cause cancer and other serious health effects, such as reproductive effects or birth defects. People exposed to toxic air pollutants at sufficient concentrations and durations may have an increased chance of getting cancer or experiencing other serious health effects.

Ecology currently monitors for a subset of air toxics at the Beacon Hill station, which is a national air toxics trends site. From 2000 to 2003, Ecology monitored air toxics at Georgetown as part of a special study.

Unlike criteria air pollutants, there are no ambient air quality standards (NAAQS) for air toxics. Clean Air is working with state, local, and tribal governments to reduce air toxics releases. While there are no ambient standards, there are several regulatory tools that are used to reduce air toxics emissions. These include: national regulations on industrial sources that require emission-reducing technology, "new source review" for sources in Washington State, local regulations for specific industries that require specific technology, and national regulations to reduce emissions from mobile sources (including cars, trucks and buses as well as marine vessels and locomotives.<sup>c,d,e,f</sup> Local regulations for specific industries include those that Clean Air developed for perchloroethylene (also know as tetrachloroethylene) dry cleaners and ethylene oxide sterilizers (Puget Sound Clean Air 2007b). In addition to regulatory tools, Clean Air and Ecology run voluntary incentive programs to reduce air toxics emissions from priority air toxics. These priority air toxics include diesel exhaust and wood smoke which were identified in Clean Air's Air Toxics Assessment.

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<sup>c</sup> EPA. National Emissions Standards for Hazardous Air Pollutants (NESHAP).

<http://www.epa.gov/ttn/atw/mactfnlalph.html>.

<sup>d</sup>EPA. Area Source Standards. <http://www.epa.gov/ttn/atw/area/arearules.html>.

<sup>e</sup> EPA. Control of Hazardous Air Pollutants from Mobile Sources. <http://www.epa.gov/fedrgstr/EPA-AIR/2007/February/Day-26/a2667a.htm>.

<sup>f</sup> EPA. Clean Diesel Campaign – Regulatory Rules. <http://www.epa.gov/cleandiesel/index.htm#regulatory>.

## *Puget Sound Clean Air Agency's Air Toxics Assessment*

Clean Air, the agency responsible for regulating emissions from stationary sources in King, Pierce, Kitsap and Snohomish Counties, conducted an assessment of air toxics in the region based on monitoring of 17 pollutants at six locations from 2000-2003 (Puget Sound Clean Air Agency, 2003). In addition to the measurement of these pollutants, wood smoke and diesel particulate matter (DPM) concentrations were estimated based on PM<sub>2.5</sub> monitoring data from the Beacon Hill monitor.

The results of the assessment indicated that stationary sources (e.g., factories, cement plants) make up only about 4% of the overall long-term health risk associated with air pollution in the region. Mobile sources (i.e., cars, trucks, buses, ships, planes, trains) and wood stove/fireplace emissions are thought to make up the bulk of air pollution health risk in the region. Diesel particulate, benzene and formaldehyde from car and truck emissions, and wood smoke were identified as being the toxic air pollutants that make up the bulk of risk.

While these pollutants were identified on a regional scale, the residential communities that border industrial areas expressed concerns that their neighborhoods may be situated in “hotspots,” or areas of increased impact from pollutants. For example, of the six air toxics monitoring sites in King County, risks from the 17 pollutants were greatest at the Georgetown site (located in the Duwamish Valley). Given that there are residential communities in and around the Duwamish Valley, there may be “hotspots” located in residential areas where monitoring is not occurring or has not occurred.

Since it is cost-prohibitive and technically infeasible to monitor hundreds of different pollutants at all locations through out the Duwamish Valley, DOH contracted with Dillingham Software Engineering Inc. (DSE) and Bluescape Environmental to conduct a multiple source air model to help determine:

- Which air pollutants are key contributors to acute and/or chronic health risk in the Duwamish Valley?
- What sources are the key contributors to acute and/or chronic health risk in the Duwamish Valley?
- To what degree are different geographic areas in south Seattle impacted by air emissions from sources in the Duwamish Valley.

## **Methods**

The Air Toxics Hot Spots Program Risk Assessment Guidelines developed by the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) was the guiding document for this project (California Environmental Protection Agency, 2003). DOH chose to use California's Hotspots Program guidance because there is not currently an equivalent program in Washington. The Hotspots Analysis and Reporting Program (HARP) developed by DSE for the California Air Resources Board (ARB) was the primary tool

used for this project. In general, an emissions inventory (i.e., an estimate of the amount and types of pollutants emitted from each source) was compiled. The dispersion and ground level impact of these emissions were then modeled (i.e., how pollutants might travel is calculated or estimated depending on weather and the type of landscape) using local meteorological and terrain conditions. Finally, non-cancer hazards and cancer risks were calculated based on modeled ground level pollutant concentrations at locations throughout the project area. The full report and methodology prepared by DSE and Bluescape Environmental is posted at <http://www.doh.wa.gov/NOTpostedYET> or available upon request. Portions of the following description of the methodology were taken directly from the Duwamish Valley Regional Modeling and Health Risk Assessment report (Dillingham Software Engineering, Inc., 2005). For a detailed description of the methodology, refer to the report posted at <http://www.doh.wa.gov/NOTpostedYET>.

## **Project Area**

The focus of the project was initially intended to estimate conditions in the communities of Georgetown and South Park. The project area was expanded to a 10 kilometer by 10 kilometer (i.e., approximately 36 square miles) area that encompasses these neighborhoods to account for impacts from sources outside the valley. Figure 1 shows the extent of the project area.

## **Emissions Inventory**

Air emissions sources in the Duwamish Valley include point (or stationary) sources, mobile sources and wood stove sources. A full description of the emissions inventory can be obtained from the Duwamish Valley Regional Modeling and Health Risk Assessment report posted at <http://www.doh.wa.gov/NOTpostedYET>.

An emissions inventory, or an estimation of the amount and type of pollutants emitted from each source in the project area, was compiled by DSE and Bluescape Environmental for the project. Point source emissions were estimated from information obtained from Clean Air. However, the emissions inventory maintained by Clean Air was not intended for modeling. As a result, many assumptions were made to fill in data gaps.

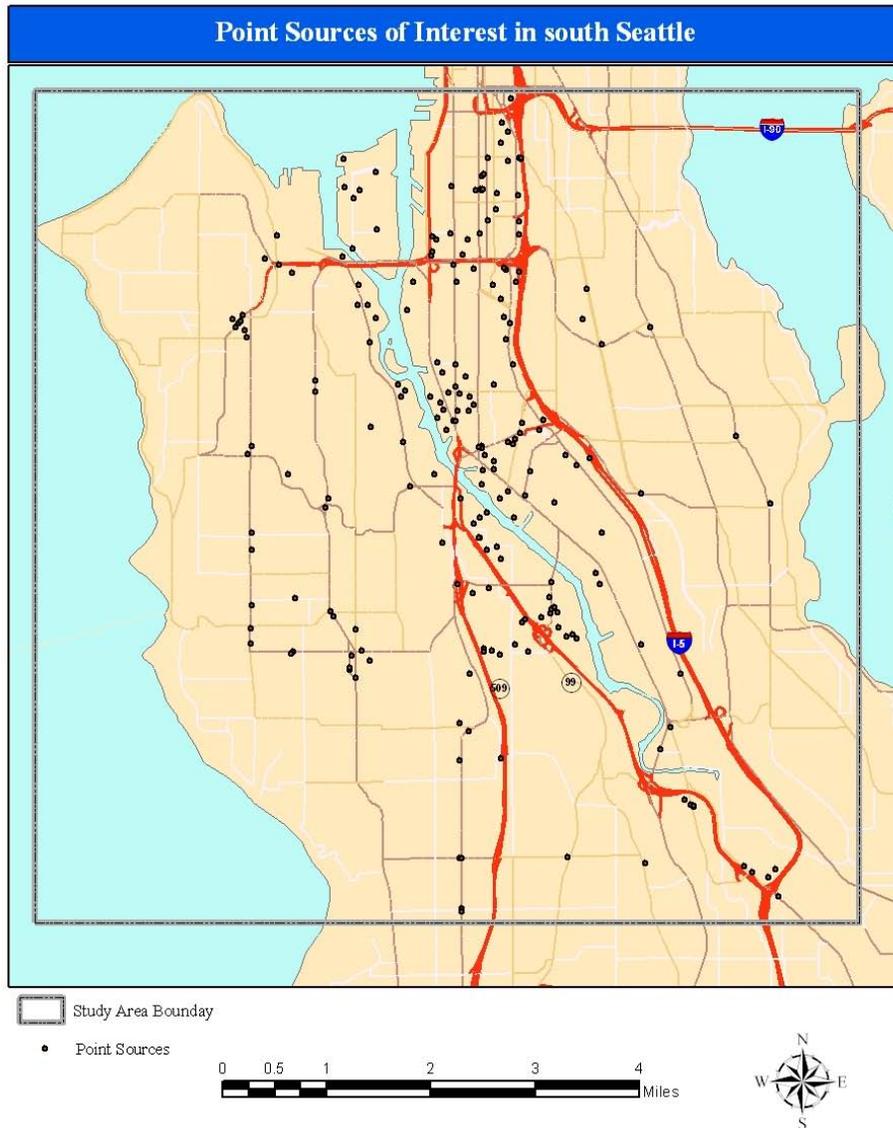
Mobile sources included motor vehicle traffic on highways, traffic arterials and local roads throughout the project area. Traffic estimates were obtained from Ecology. Rail, air, and ship emissions were not included because emission estimates from these sources were not available when this project was initiated. Port activities are expected to be significant contributors to air pollution in areas south Seattle.

Wood stove emission estimates were based on estimates derived from the 2000 census. Of an estimated 8600 households in King County that use wood as heating fuel, more than 400 are located within the project area. It is not clear how accurate these data are or how much they differ from surveys conducted by Clean Air to gauge wood stove and fireplace use.

As described in the Duwamish Valley Region Modeling report, there are many emission inventory data gaps. Therefore, surrogate assumptions, or estimates based on knowledge from

similar sources, were needed to complete the emissions inventory. For this reason, the modeled estimates of the ground level pollutant concentrations and health risks are also uncertain and should be viewed with some caution.

**Figure 3.** Point (or Stationary) Sources in south Seattle



### Air Model

Air dispersion modeling is a tool used to estimate the level of a pollutant downwind from an air pollution source. HARP uses EPA's Industrial Source Complex Short-term 3 (ISCST3) air model to estimate ground level concentrations of pollutants across the project area. Typically, ISCST3 is used as a tool to predict impacts from a source or sources for permit purposes. HARP compiles the results of numerous ISCST3 model iterations from numerous sources to estimate

ground level concentrations. Deposition of pollutants is also factored into the model so that other pathways of exposure (e.g., soil ingestion) in addition to inhalation can be assessed.

The meteorological information put into the air model for this project were based on data from Seattle-Tacoma International Airport (Sea-Tac). Local meteorological conditions in the Duwamish Valley and south Seattle could vary from those measured at Sea-Tac. Any discrepancies, such as prevailing wind direction, would add to existing factors affecting precision and accuracy of air modeling (which influences how correct the model will be in providing information).

## **Risk Assessment**

Once modeling has been accomplished to determine the ground level concentration of pollutants, HARP calculates non-cancer hazards (e.g., risks from chronic diseases such as asthma or heart disease) and cancer risk at locations throughout the project area. HARP uses guidelines established by OEHHA for conducting risk analysis. For this project, inhalation (breathing in), soil ingestion (taking in soil by mouth) and dermal (skin) contact were the exposure routes and pathways (how contaminants or pollutants get into the body) assessed.

In order to evaluate the potential for non-cancer adverse health effects that may result from exposure to air pollutants, a dose (how much of something a person takes into the body) is estimated for each pollutant. These doses are calculated for situations (scenarios) in which residents might breathe in air pollutants or come into contact with the contaminated soil. The estimated dose for each contaminant under each scenario is then compared to a reference exposure level (REL). RELs are doses below which non-cancer adverse health effects are not expected to occur (so called "safe" doses). They are derived from toxic effect levels obtained from human population and laboratory animal studies. Due to uncertainty in these data, the toxic effect level is divided by "safety factors" giving a lower and more protective REL. If a dose exceeds the REL, this indicates only the potential (possibility) for adverse health effects. The magnitude of this potential can be inferred from the degree to which this value is exceeded (the higher the number is above the REL the greater the possibility there might be a health risk). If the estimated exposure dose is only slightly above the REL, then that dose will fall well below the toxic effect level (i.e., the level of exposure where health effects were observed in animal or epidemiological studies). The higher the estimated dose is above the REL, the closer it will be to the actual toxic effect level. This comparison is typically known as a hazard quotient.<sup>8</sup> For the purposes of this report, the term hazard index (HI) is used in place of hazard quotient.

$$\text{HI (inhalation)} = \frac{\text{Estimated average daily exposure concentration } (\mu\text{g}/\text{m}^3)}{\text{REL } (\mu\text{g}/\text{m}^3)}$$

$$\text{HI (soil contact)} = \frac{\text{Estimated average daily dose (mg/kg/day)}}{\text{REL (mg/kg/day)}}$$

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<sup>8</sup> In the Duwamish Valley Regional Modeling and Health Risk Assessment report, the term health hazard index (HHI) is used in place of hazard quotient. Typically, the term hazard index is used to define the sum of hazard quotients.

Theoretical cancer risk is estimated by calculating a dose similar to that described above and multiplying it by a cancer potency factor, also known as the cancer slope factor. Each chemical has a different cancer potency factor which is based on the best studies available of either cancer in people or animals. Laboratory animal studies involve doses much higher than what would be encountered in the environment. Use of animal data requires extrapolation of the cancer potency obtained from these high dose animal studies down to real-world human exposures. This process involves much uncertainty.

Current regulatory practice assumes there is no "safe dose" of a carcinogen and that a very small dose of a carcinogen will give a very small cancer risk. Cancer risk estimates are, therefore, not yes/no answers but measures of chance (probability). Such measures, however uncertain, are useful in determining the degree or amount of a cancer threat assuming that any level of a carcinogenic contaminant carries some risk, and are a helpful tool for prioritizing pollutants to target for emissions reduction. Some people are more likely to develop cancer than others depending on a number of factors including: their age, genetic make up, sex, or the amount of chemical exposures they have received in a lifetime.

<b>Theoretical Cancer Risk</b>		
Cancer risk estimates do not reach zero no matter how low the level of exposure to a carcinogen. Terms used to describe this risk are defined below as the number of excess cancers expected in a lifetime:		
<u>Term</u>		<u># of Excess Cancers</u>
moderate	is approximately equal to	1 in 1,000
low	is approximately equal to	1 in 10,000
very low	is approximately equal to	1 in 100,000
slight	is approximately equal to	1 in 1,000,000
insignificant	is less than	1 in 1,000,000

The following equations in Table 2a show how theoretical cancer risk and hazard indices are calculated for each pathway. Table 2b defines each parameter.

**Table 2a.** Equations to calculate theoretical cancer risk and non-cancer hazard indices.

<b>Exposure Route/ Pathway</b>	<b>Theoretical Cancer Risk</b>	<b>Chronic non-cancer hazard index</b>	<b>Acute hazard index</b>
Inhalation	$\frac{C_{air} \times IR \times EF \times ED \times CF \times CPF}{AT}$	$\frac{C_{air} \times IR \times EF \times ED \times CF}{AT \times REL}$	$\frac{C_{air}}{Acute REL}$
Soil ingestion	$\frac{C_s \times BAF \times SIR \times EF \times ED \times CF \times CPF}{AT}$	$\frac{C_s \times BAF \times SIR \times EF \times ED \times CF}{AT \times REL}$	NA
Dermal Absorption	$\frac{C_s \times SA \times SL \times EF \times ABS \times CF \times ED \times CPF}{BW \times AT}$	$\frac{C_s \times SA \times SL \times EF \times ABS \times CF \times ED}{BW \times AT \times REL}$	NA

**Table 2b.** Exposure parameters defined

<b>Exposure Route / Pathway</b>	<b>Parameter</b>	<b>Value</b>	<b>Units</b>	<b>Source / comments</b>
Inhalation	Cair = Concentration in Air	Modeled concentration: average concentration for chronic and one-hour maximum concentrations for acute exposures	micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ )	Based on assumed emission rates.
	IR = Inhalation Rate	393	Liters per kilogram body weight per day	95 <sup>th</sup> percentile inhalation rate
	EF = Exposure Frequency	350	days/year	
	ED = Exposure duration	70	years	
	CF = Conversion Factor	0.000001	$\mu\text{g}/\text{mg}$ Liters/ $\text{m}^3$	Converts $\mu\text{g}$ to mg Converts liters to $\text{m}^3$
	AT = Averaging Time	25,550	days	70 years
	REL = Reference Exposure Level	Contaminant-specific	$\mu\text{g}/\text{m}^3$	Available for chronic and acute non-cancer hazards
	CPF = Cancer Potency Factor	Contaminant - Specific	$(\text{mg}/\text{kg}/\text{day})^{-1}$	
<b>Soil Ingestion</b>				
Soil Ingestion	Cs = Soil concentration	Modeled	mg/kg	Based on modeled deposition
	BAF = Bioavailability factor	1.0 (all other) 0.43 (dioxin)	unitless	Fraction of pollutant in soil that is absorbed in the gut
	SIR = Soil ingestion Rate	1.7	mg/kg BW per day	Approximately 100 mg/day for an adult
	EF = Exposure Frequency	350	days/year	
	ED = Exposure duration	70	years	
	AT = Averaging Time	25,550	days	
	REL = Reference Exposure Level	Contaminant-specific	mg/kg/day	
	CPF = Cancer Potency Factor	Contaminant - Specific	$(\text{mg}/\text{kg}/\text{day})^{-1}$	
<b>Dermal Absorption</b>				
Dermal Absorption	Cs = Soil Concentration	Modeled	mg/kg	Based on modeled deposition
	SA = Surface Area	5,500	$\text{cm}^2$	Exposed skin
	SL = Soil Loading	1.0	$\text{mg}/\text{cm}^2$	Amount of soil that adheres to an area of skin

**Table 2b (continued).** Exposure parameters defined

<b>Exposure Route / Pathway</b>	<b>Parameter</b>	<b>Value</b>	<b>Units</b>	<b>Source / comments</b>
Dermal Absorption	ABS = Absorption fraction	Contaminant-specific	unitless	Based on a contaminants properties (relative ease with which it passes through the skin)
	BW = Body weight	63	kg	
	EF = Exposure Frequency	350	days/year	
	ED = Exposure duration	70	years	
	AT = Averaging Time	25,550	days	
	REL = Reference Exposure Level	Contaminant-specific	mg/kg/day	
	CPF = Cancer potency factor	Contaminant - Specific	(mg/kg/day) <sup>-1</sup>	

Many of the assumptions used in the risk assessment portion of the project are designed to be especially protective of the public. For example, many of the exposure parameters (i.e., inhalation rate and dermal soil loading) are intended to be high-end estimates. Cancer potency factors used to estimate a chemical’s cancer risk are generally based on the theoretical upper bound (the 95% upper confidence limit) probability of extra cancer cases occurring in an exposed population, assuming a lifetime exposure. These assumptions, because they are intended to be conservative and not underestimate exposure and risk, are likely to result in overestimation of the actual risk. Additionally, OEHHA has defined cancer potency factors for many contaminants that U.S. EPA has not. The uncertainty with many of these factors may tend to over-estimate risk as opposed to underestimate. HARP risk assessment guidance points out that the risks estimated using this methodology are meant to err on the side of public health protection, so they are on the cautious side in order to protect as many people as possible. An individual’s true risk is likely to be lower than the estimates provided using this methodology. The results are not intended to predict disease rates in the community, but to prioritize concerns for potential public health actions (or figure out which chemicals or exposures we should be most concerned about). The following statement from the HARP guidance document summarizes the issue:

“risk estimates generated by a health risk analysis should not be interpreted as the expected rates of disease in the exposed population but rather as estimates of potential risk, based on current knowledge and a number of assumptions. Additionally, the uncertainty factors integrated within the estimates of non-cancer reference exposure levels (RELs) are meant to err on the side of public health protection in order to avoid underestimation of risk. Risk assessment is best used as a ruler to compare one source with another and to prioritize concerns. Consistent approaches to risk assessment are necessary to fulfill this function.”

## Results

The following sections describe the chronic cancer and non-cancer risks associated with point, mobile, and woodstove sources in Seattle. Theoretical cancer risks are presented as probabilities of a person developing cancer related to exposure to air pollutants in south Seattle, or as the number of cancers in population of exposed people. Cancer estimates are expressed in scientific notation, for example  $1e^{-6}$  or  $1 \times 10^{-6}$ , is interpreted as 1 excess cancer per million individuals exposed, or an individual's probability of getting cancer from exposure to air pollutants is 1 in 1,000,000. As noted previously, these risks should not be interpreted as estimates of disease in the community, only as a tool to define potential risk. Non-cancer risks are presented according to hazard indices where hazard indices greater than one represent exposure levels of concern.

### Point Sources

#### *Theoretical cancer risk*

Figure 3 shows theoretical cancer risks associated with point sources in south Seattle. In general, risks attributable to point source emissions are highest in two areas on the east side of the Duwamish River near the Georgetown neighborhood. These areas of higher risk are associated with chromium compound emissions. If chromium compounds are excluded from the analysis, the higher risk areas near Georgetown and South Park would be minimized. When excluding chromium compounds, benzene becomes the contaminant that contributes most to cancer risk at areas impacted by point sources.

The two areas of higher risk, corresponding to theoretical cancer risks of  $1.1 \times 10^{-3}$  and  $5.4 \times 10^{-4}$  (approximately 1 excess cancer per 1000 people exposed and 5 excess cancers per 10,000 people exposed) are near North Boeing Field and Capital Industries. A Clean Air inquiry into the chromium emissions used in the air model showed that control technologies were not adequately factored into the emissions estimate. Both facilities had submitted conservative emissions estimates to Clean Air that did not fully take into account the filters they have in place to reduce emissions. Consequently, cancer risk related to these sources is likely over-estimated.

The following chemicals are the dominant (main) contributors to theoretical cancer risk, in descending (decreasing) order of risk.

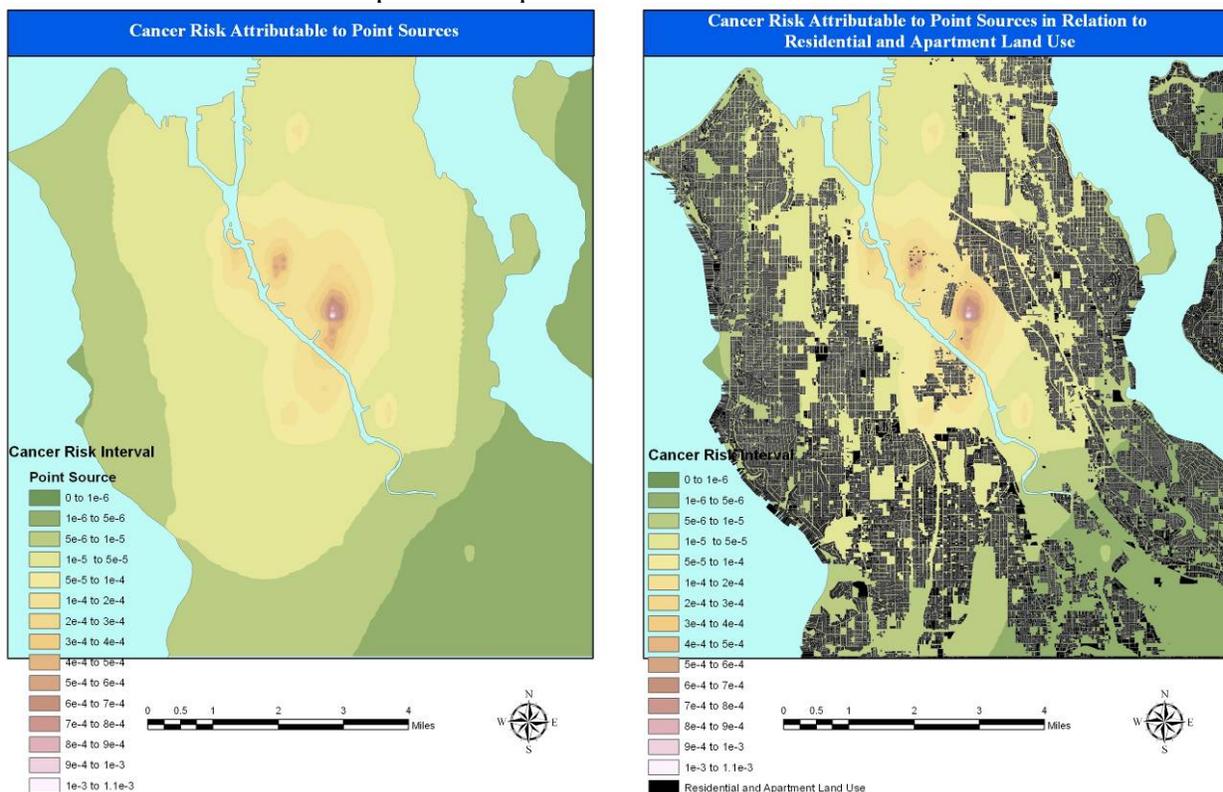
- Strontium Chromate
- Lead Chromate
- Hexavalent Chromium
- Benzene
- Lead
- Naphthalene

Eliminating chromium compound emissions would greatly reduce theoretical cancer risk in areas of south Seattle attributable to point sources. As mentioned previously, due to the limitations of the emissions inventory, chromium compound emission rates are highly uncertain. Follow-up

work conducted by Clean Air revealed that most chromium emission rates were over-estimated (Puget Sound Clean Air Agency, 2006b). Furthermore, two sources listed as emitting chromium are no longer operating (Veteran’s Affairs Hospital Medical Incinerator and Precision Engineering). These sources are no longer a factor with regard to chromium emission. With risks from chromium sources being the driver at hotspots in south Seattle, it is important to obtain clear and accurate estimates of chromium emissions from all sources so we can better focus priorities.

When we looked at which areas of south Seattle are residential areas and which are industrial areas, we found that areas most impacted by point sources were areas where no or few people live, but theoretical cancer risks attributable to chromium sources impact Georgetown and South Park resulting in theoretical cancer risks ranging from  $1 \times 10^{-4}$  to  $4 \times 10^{-4}$ . As mentioned previously, the two areas of highest impact were related to chromium emissions from North Boeing Field and Capital Industries. It was also noted that the emission rates reported by these sources did not fully consider engineering controls. It is clear that reducing chromium emissions (or considering engineering controls) would greatly reduce theoretical cancer risk attributable to these point sources.

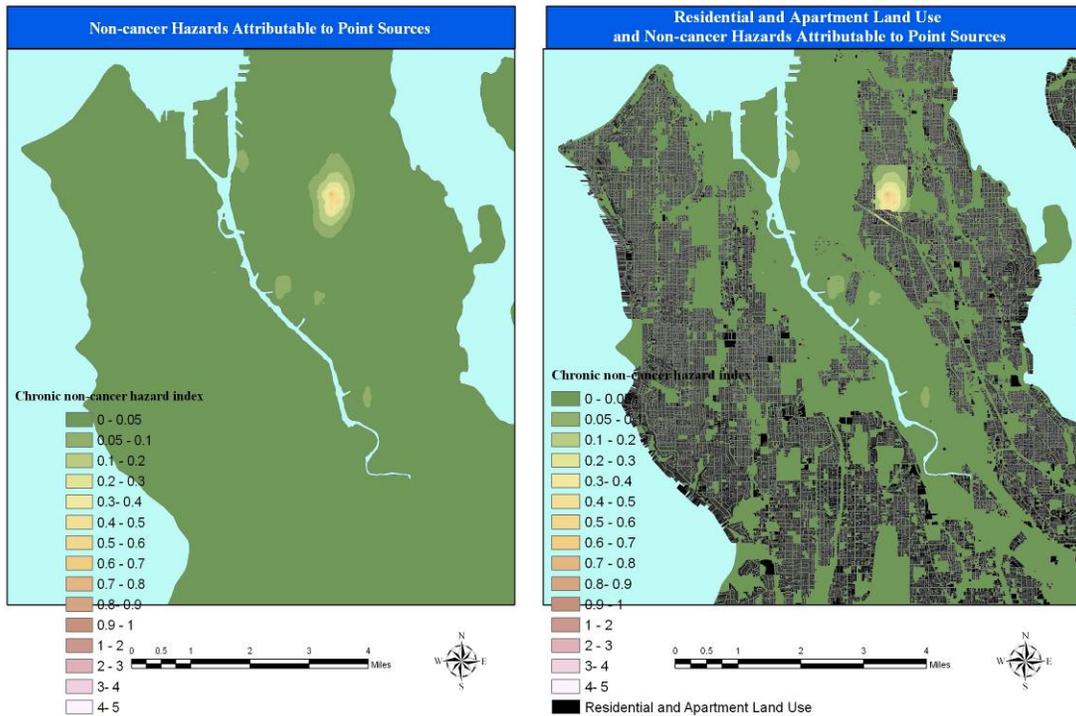
**Figure 3.** Theoretical cancer risk impacts from point sources in south Seattle. Residential land use overlaid on cancer risk impacts from point sources in south Seattle.



## Non-cancer hazard

Non-cancer hazards are quantified using a hazard index. A hazard index less than one is considered to be acceptable from a human health point of view. Non-cancer hazard indices associated with point source emissions in south Seattle were generally below a level of concern. The highest hazard index (0.8) was associated with mercury emitted from the Veterans Affairs Hospital incinerator on Beacon Hill. This incinerator was shut down in the 1990s and no longer emits pollutants. Figure 4 shows a map of chronic hazard indices.

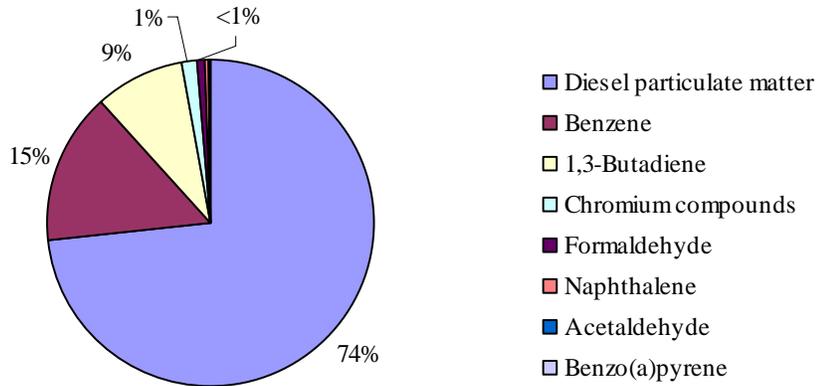
**Figure 4.** Non-cancer hazard impacts from point sources in south Seattle. Residential land use overlaid on non-cancer hazard impacts from point sources in south Seattle.



## On-Road Sources

### *Theoretical Cancer risk*

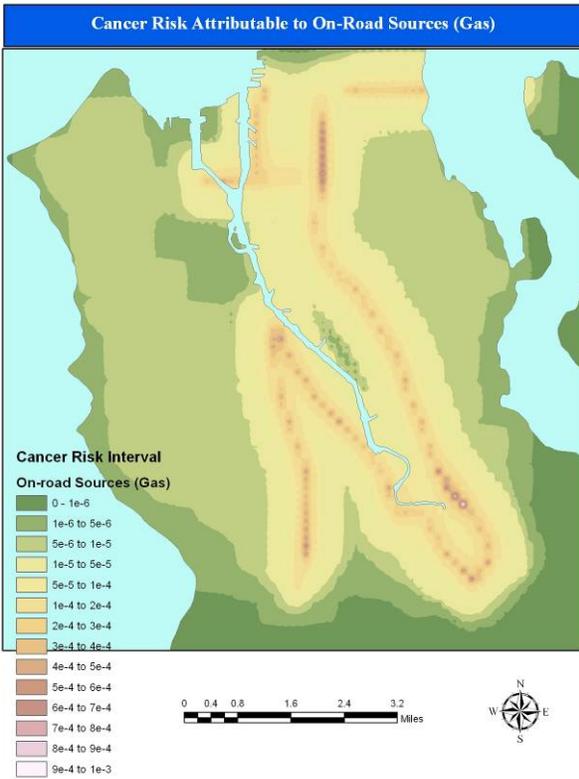
The following graphic shows the pollutants that dominate cancer risk from on-road sources, in descending order of cancer risk. Diesel particulate matter, benzene, and 1,3-butadiene are the chemicals that contribute to the bulk of risk.



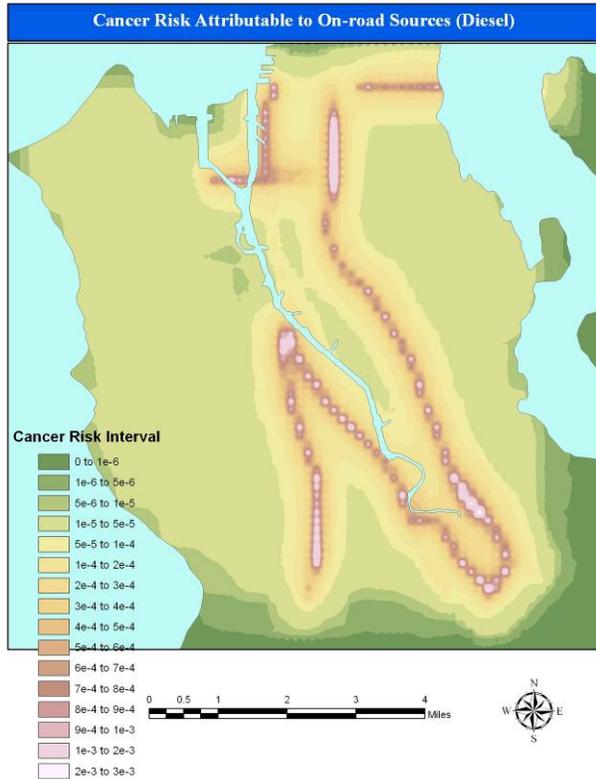
Estimated theoretical cancer risk is greatest near the center of major highways such as Interstate-5 and Highway 99. Figures 5a-5b illustrate the individual contributions of gasoline and diesel sources to cancer risk. Although there are fewer vehicles with diesel engines than gasoline-powered vehicles, risks attributable to diesel are about three times greater (about  $3 \times 10^{-3}$  for diesel and  $1 \times 10^{-3}$  for gas sources) at the highest impacted point (at the center of Interstate-5). Figure 5c shows the risks of the diesel and gas combined. Estimated risks drop dramatically a few hundred meters on either side of major roadways. Figure 5d shows that many residential parcels are located adjacent to major traffic corridors in south Seattle, and thus located in areas of higher exposure and risk.

**Figure 5a-5d. Theoretical cancer risk impacts from on-road sources in south Seattle.**  
 Residential land use overlaid on cancer risk impacts from on-road sources in south Seattle.

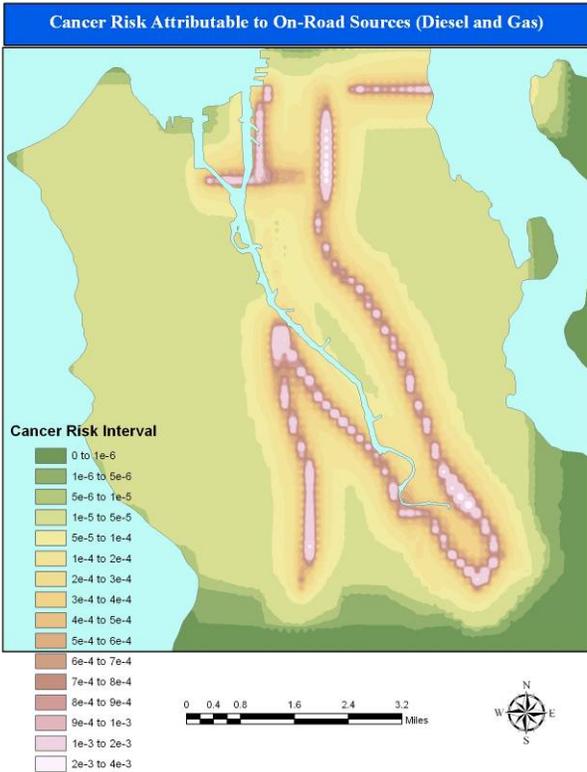
**5a. Gas only**



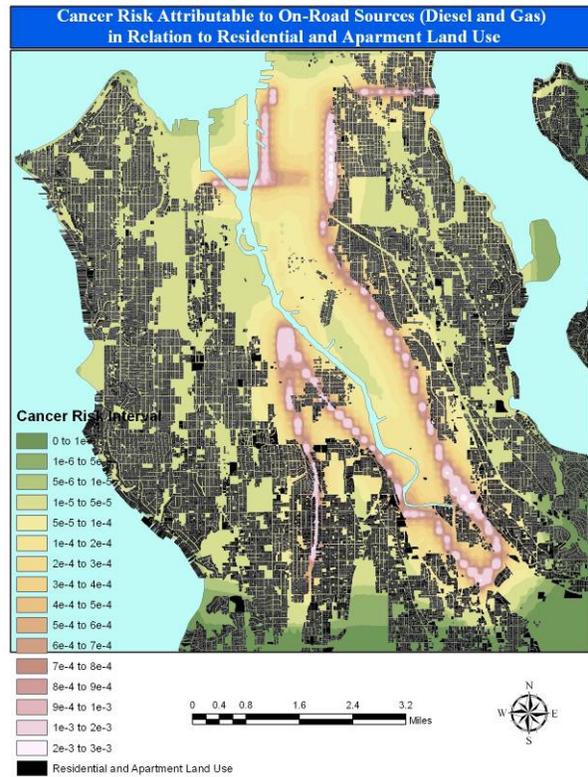
**5b. Diesel only**



### 5c. Gas and Diesel



### 5d. Land use overlay

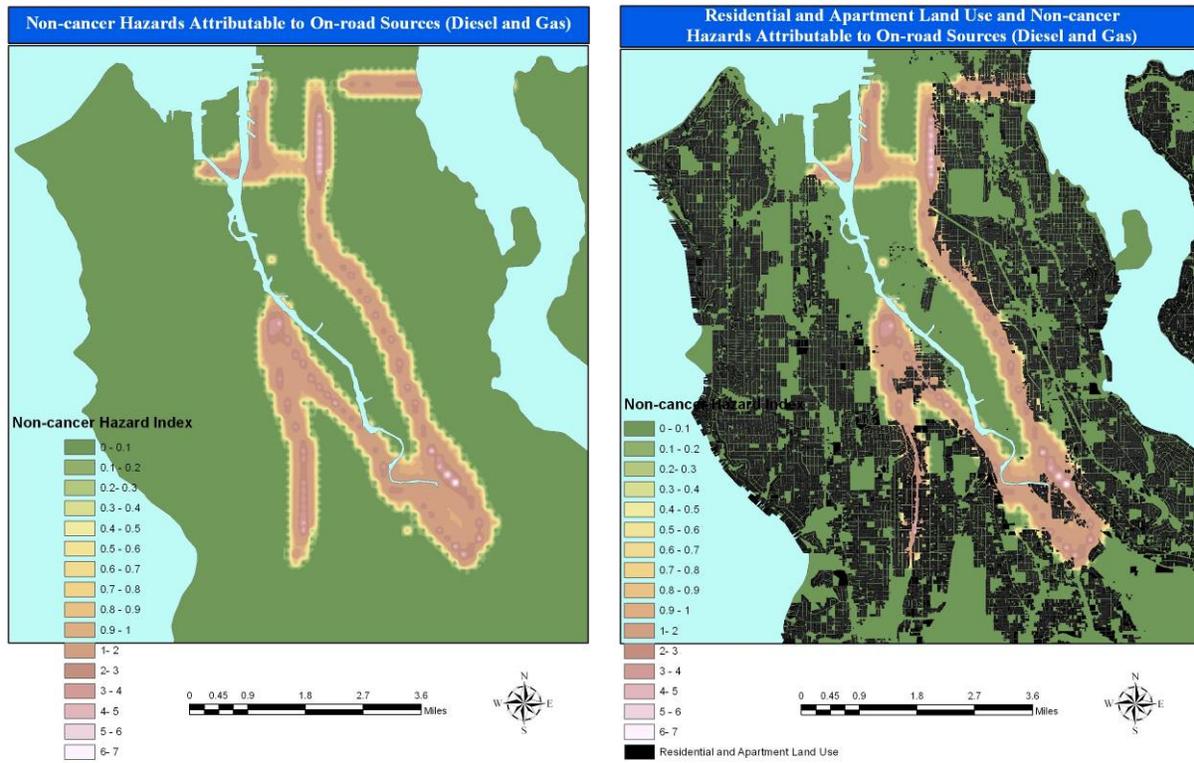


### *Non-cancer*

Similar to cancer risk, non cancer hazards are highest near major roadways. Hazard indices decrease with distance from the center of highways, but exceed one up to a few hundred meters on either side of major highways. Acrolein is the primary contaminant associated with non-cancer hazards from road sources. The following four pollutants are the main contributors to non-cancer hazards from mobile sources in descending order.

- Acrolein
- Formaldehyde
- Diesel Engine Exhaust, Particulate Matter
- Nitrogen Dioxide

**Figure 6.** Non-cancer hazard impacts from on-road sources in south Seattle. Residential land use overlaid on non-cancer hazard impacts from on-road sources in south Seattle.

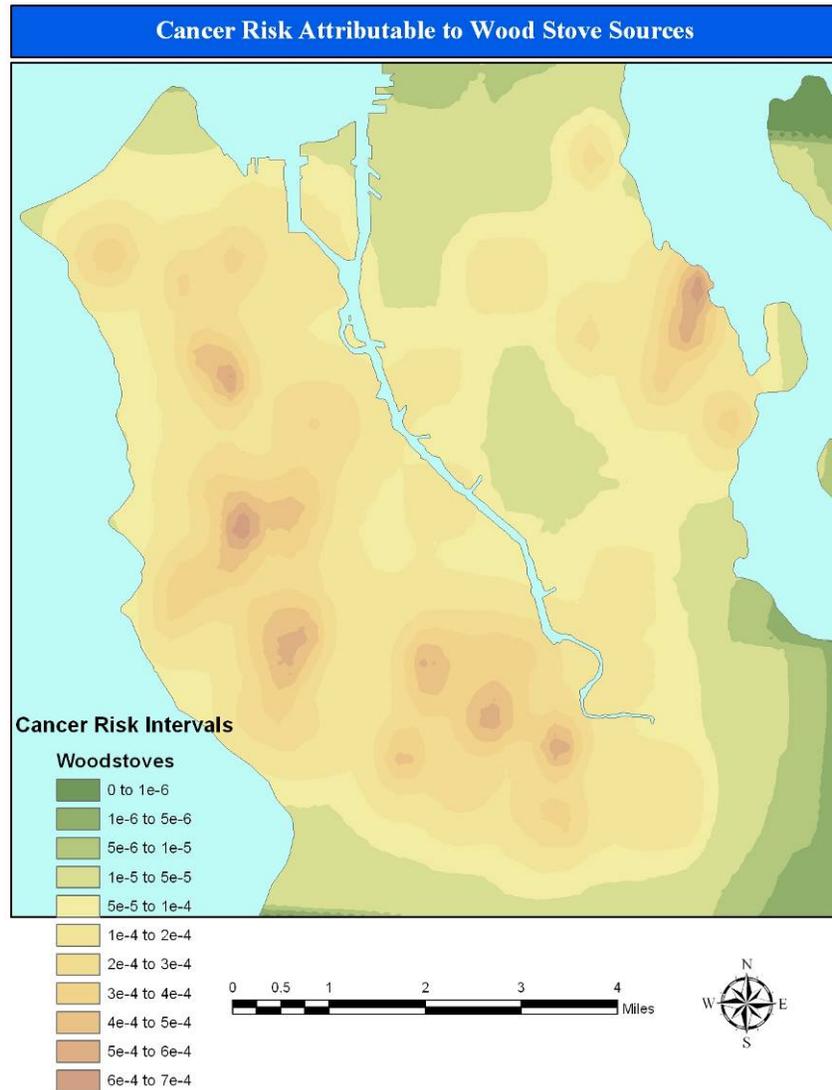


## Wood Stove Sources

### *Theoretical cancer risk*

Estimated theoretical cancer risks attributable to wood stove sources in south Seattle are roughly in the  $1 \times 10^{-4}$  to  $5 \times 10^{-4}$  (1 to 5 excess cancers per 10,000 people exposed) range throughout most of the project area. It should be noted that theoretical cancer risks from wood stove sources were calculated somewhat differently than point or mobile sources. Deposition (settling) of smoke particles (and accompanying polycyclic aromatic hydrocarbons which are probable human carcinogens) was calculated so that soil ingestion and dermal absorption exposures could also be factored into risk. Risks attributable to inhalation exposures represent only about 10% of the total presented in Figure 7. In this case, the dermal pathway dominated theoretical cancer risks attributable to numerous polycyclic aromatic hydrocarbons from wood stove sources. As mentioned previously, many of the assumptions used to quantify risks are very conservative (i.e., tend to overestimate exposure and risk), so these estimates are to be viewed with some caution.

**Figure 7.** Theoretical cancer risk impacts from wood stove/fireplace sources in south Seattle (note: risks include consideration of deposition and soil contact in addition to inhalation).



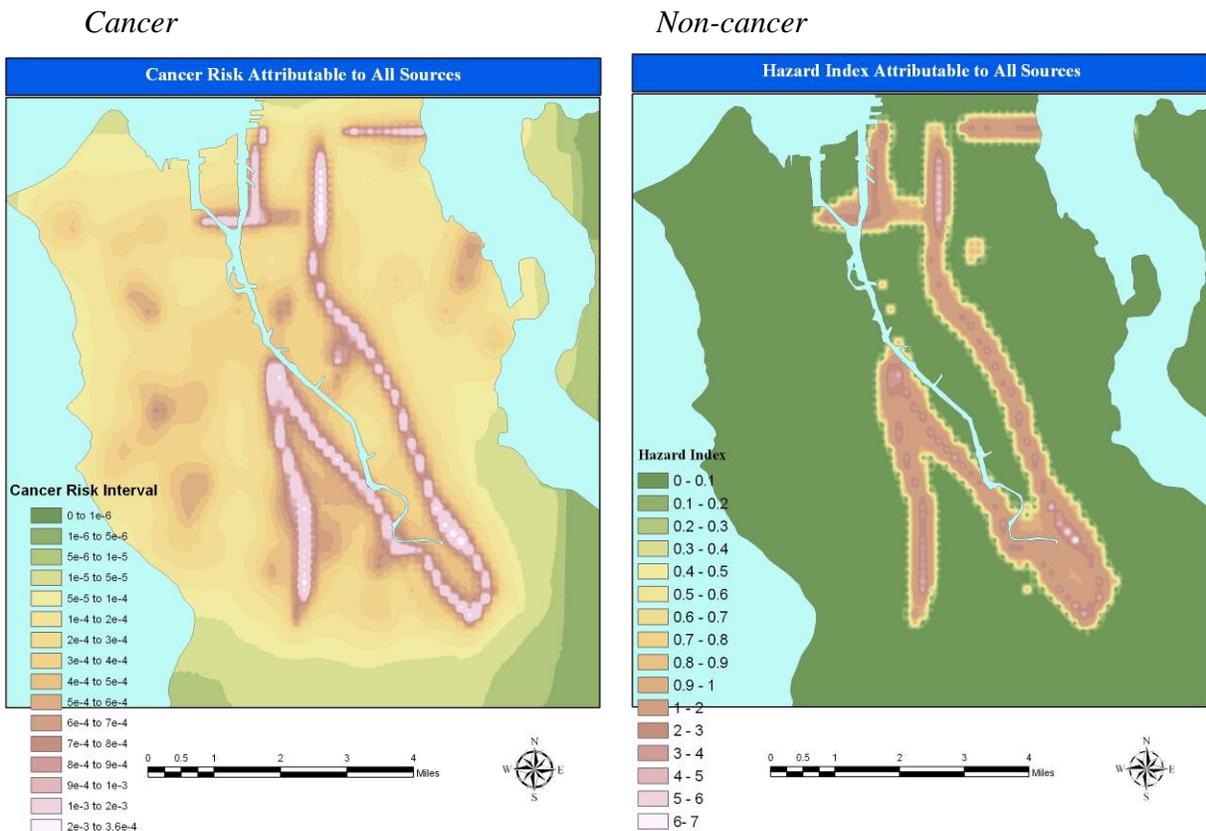
*Non-cancer*

Woodstove sources modeled in the project area did not result in a hazard index greater than one for any contaminant. However, acute hazards related to CO and NO<sub>2</sub> approached a hazard index of 1 (0.95 and 0.77 respectively) at a point of maximum impact. It is important to note that although woodstoves emit a large amount of particulate matter, HARP does not calculate a hazard index for PM because OEHHA does not have an available chronic or acute REL value for PM. This is a significant limitation of the risk analysis portion of the project as PM is expected to be of health concern in the community and across Washington State.

## All sources

Figure 8 shows the combined theoretical cancer risk and non-cancer hazard from all sources. In general, the impacts from mobile sources near major roadways are most apparent, but when looking back at the previous figures in this document, hotspot impacts from point sources (primarily theoretical cancer risk attributable to chromium compound emitters) can be seen near Georgetown on the east bank of the Duwamish River. Estimated theoretical cancer risks attributable to woodstove/fireplace sources are also evident over portions of the project area.

**Figure 8.** Theoretical cancer and non-cancer impacts from all sources in south Seattle.



## Discussion

Air modeling is useful in that it can estimate contaminant concentrations across a wide geographic area. Although monitoring would be preferred, it is neither practical nor possible to sample continuously for numerous pollutants at an infinite amount of points in an area.

Nonetheless, it is useful to compare modeled pollutant concentrations with actual monitored observations. As mentioned, some air toxics monitoring occurs at the Beacon Hill monitor and occurred in 2000-2003 at a monitoring location in Georgetown.

Table 3 shows the comparison between modeled and monitored average concentrations for the Georgetown and Beacon Hill sites. The estimated concentration of pollutants at the grid point nearest the actual monitor was selected for each comparison. Average monitored concentrations over the duration of air toxics monitoring were then compared to modeled average ground level concentrations.

The ratio between the modeled concentrations to the monitored concentration is also presented. Ratios greater than one indicate the modeled concentration is greater than the monitored level (overestimates), and ratios less than one indicate the modeled concentration is less than the monitored levels (underestimates). Exact agreement between the modeled and monitored levels is not expected because of many factors including the accuracy of emissions inventory from all sources, the fact that air models are not expected to be exact, and monitors have analytical limitations.

Modeled concentrations tended to be more than an order of magnitude (10 times) lower than monitored values for many contaminants. The underestimates may be a factor of gaps in emissions inventory used in the model or perhaps some combination of emissions inventory data gaps, modeling uncertainties, and analytical capabilities of the monitoring instruments.

Contrarily, ethylbenzene and toluene are overestimated at the Beacon Hill monitor. Mercury was also overestimated at Beacon Hill when compared to the PM<sub>10</sub> mercury measurement. This might be attributable to the fact that the air model assumed the medical waste incinerator was active when in reality it has been shut down since the 1990s.

Modeled average concentrations reasonably match monitored average concentrations for benzene, 1-3, butadiene styrene, trichloroethylene, dichloromethane, o-xylene, lead, and hexavalent chromium (Beacon Hill monitor only).

Since the predicted hotspots estimated in the model attributed to point sources were primarily from chromium compound emitters, it is unfortunate that hexavalent chromium monitoring was not available at Georgetown (an area closest to chromium "hotspots"). This would have served to help determine to what degree risk attributed to chromium sources are over or underestimated by the model. However, even if monitoring from these points was available, the results would need to be qualified as there is not currently a dependable ambient air monitoring method available with which to measure hexavalent chromium.

**Table 3.** Monitored air toxics levels at Georgetown and Beacon Hill monitors versus modeled concentrations at points nearest the monitors ( $\mu\text{g}/\text{m}^3$ )

Parameter	Monitored Average (Georgetown 2000-2003)	Modeled Average	Ratio of Modeled / Monitored	Monitored Average (Beacon Hill 2000-2007)	Modeled	Ratio of Modeled / Monitored
Diesel Particulate	NA	0.13	NA	1.4	0.11	0.1
Acetaldehyde	0.51	0.033	0.065	0.42	0.028	0.066
Benzene	0.25	0.41	1.6	0.12	0.32	2.6
1,3-Butadiene	0.044	0.028	0.63	0.023	0.023	1
Dichloromethane	NA	0.33	NA	0.035	0.014	0.4
Ethylbenzene	NA	0.23	NA	0.013	0.11	8.2
Formaldehyde	1.43	0.096	0.067	1.1	0.082	0.077
Styrene	NA	0.048	NA	0.016	0.026	1.6
Tetrachlorethylene	0.010	0.00043	0.043	0.0047	0.00027	0.057
Toluene	NA	1.35	NA	0.10	0.67	6.6
Trichloroethylene	0.018	0.042	2.3	0.006	0.019	3.2
o-xylene	NA	0.0072	NA	0.014	0.0084	0.6
Arsenic (PM10)	NA	0.0000029	NA	0.00089	0.0000039	0.004
Arsenic(PM2.5)	0.0015		0.002	0.0011		0.004
Beryllium (PM10)	NA	0.0000002	NA	0.000018	0.0000003	0.17
Cadmium (PM10)	NA	0.00023	NA	0.00013	0.000019	0.14
Cadmium(PM2.5)	0.0023		0.10	0.0039		0.005
Chromium (PM10)	NA	NA	NA	0.0017	NA	NA
Chromium (PM2.5)	0.0035		NA	0.0017	NA	NA
Chromium VI (TSP)	NA	0.00005	NA	0.00007	0.000034	0.48
Lead (PM10)	NA	0.0057	NA	0.0044	0.0012	0.28
Lead (PM2.5)	0.0092		0.62	0.0038		0.32
Manganese (PM10)	NA	0.00032	NA	0.0091	0.00037	0.04
Manganese(PM2.5)	0.013		0.026	0.0046		0.08
Mercury(PM10)	NA	0.000068	NA	0.00003	0.00040	13
Mercury(PM2.5)	0.00096		0.071	0.0016		0.25
Nickel (PM10)	NA	0.000025	NA	0.0025	0.000028	0.01
Nickel(PM2.5)	0.0039		0.006	0.0024		0.01

Note: PM 2.5 metals were analyzed using X-Ray Fluorescence, and PM10 metals were analyzed using ICP-MS.  
TSP – total suspended particulate

### Usefulness of the model

The purpose of air modeling was to try to answer three questions about pollutants and sources in south Seattle. These questions will be restated and answered below.

- Which air pollutants are key contributors to acute and/or chronic health risk in the Duwamish Valley?

Based on the model, diesel particulate matter, compounds found in wood smoke, chromium compounds, benzene, and 1-3 butadiene contribute to higher theoretical cancer risks in areas of south Seattle. Acrolein, formaldehyde, diesel particulate matter, and  $\text{NO}_2$  are the pollutants that make up the bulk of the non-cancer hazard. The primary pollutants of concern determined through this project largely match those highlighted in Clean Air's air toxics assessment.

- What sources are the key contributors to acute and/or chronic health risk in the Duwamish Valley?

Mobile sources, particularly diesel engines contribute the most to theoretical cancer risk near roadways. Some point sources that emit chromium compounds can contribute to theoretical cancer risk near the source. It should be noted that the actual chromium emissions from point sources is uncertain and likely overestimated because engineering controls were not fully factored into the equation when the emission estimates were reported. Better understanding of chromium emissions in south Seattle will provide better understanding of cancer risk from point sources.

Woodstoves and fireplaces contribute to theoretical cancer risk in south Seattle. This is based on evaluating individual compounds found in wood smoke, and considering that these chemicals can be inhaled and or deposited in soil where additional exposure can occur. The impact of wood smoke particulate matter (treated as a single chemical) on non-cancer hazards, however, was not quantified because OEHHA does not have a REL for particulate matter. For this reason, impacts of wood smoke particulate matter on acute or chronic non-cancer health effects was not evaluated in this project and represents a significant limitation. Woodsmoke is a current concern for acute health effects especially when meteorological conditions do not allow smoke to be dispersed (temperature inversions).

- To what degree are different geographic areas in south Seattle impacted by air emissions from sources in the Duwamish Valley?

The results of the air model clearly demonstrate the potential health impacts of vehicle emissions on residents living near major roadways. While many of the major traffic corridors run through industrial or commercial areas, many residential areas are within 200 meters of the roadways. Impacts are greatest in these areas (refer to land use overlays in Figures 5 and 6).

Because woodstove emissions are spread somewhat uniformly throughout south Seattle, these impacts are not focused heavily on a single or specific area. Impacts from woodstoves are likely experienced somewhat equally by all living in south Seattle. Woodstove use was estimated based on census data from 2000. In general, an estimated 8600 homes in King County, and 420 homes in south Seattle use wood to heat their homes. The number of households that burn wood in fireplaces is not known.

One important health endpoint not evaluated in the model is the potential for wood smoke particulate matter to contribute to chronic and acute non-cancer hazards. OEHHA does not have a REL for wood smoke particulate matter, and therefore hazards associated with it were not quantified. While theoretical cancer risk estimates associated with environmental pollution are often highlighted as major public health issues, it is important to understand that acute non-cancer health effects related to PM such as aggravation of asthma and triggering heart attacks are serious public health concerns.

Finally, although uncertain, emissions from point sources have the potential to heavily impact areas of south Seattle. Given that the emissions inventory used to create the model was uncertain,

the results should be viewed with caution. However, the need for more accurate reporting of emissions from facilities, transportation, and home sources in south Seattle is important for prioritizing air pollution reduction efforts in areas of south Seattle and the region.

### **Broader Implications**

Since the clean air act was signed into law, improvements in air quality have occurred all across the U.S. Significant reduction in criteria pollutants and volatile organic compounds has been documented (EPA, 2007c). Regulatory requirements on point sources and cleaner vehicles have greatly reduced air pollution. From a public health point of view, these reductions have been beneficial (Graham, 2004); unfortunately, health effects attributable to air pollution are still being documented (Peters et al. 2004, Norris et al. 1999, Yu et al. 2000, Tolbert et al. 2000, Schwartz 1999). Brief summaries of some of the health effects associated with the primary pollutants of concern identified during this project are presented in Appendix A.

As demonstrated in this project and by Ecology's air quality trends publications, transportation sources and wood smoke (includes outdoor burning) make up the bulk of air pollution (and health risk) in the region and the state. Clean Air has the authority to regulate and control point source emissions in the region, and to a lesser degree, wood stove and fireplace burning, but they do not have the authority to regulate mobile sources. So instead of regulating, Clean Air is actively engaged in encouraging transportation options that reduce the amount of pollutants released into the air. They also sponsor an innovative program called Diesel Solutions which partners with government and private vehicle fleets to voluntarily retrofit vehicles with pollution control devices, use cleaner fuels, and encourage vehicle operators to avoid idling. In addition to these efforts, Ecology and air agencies from different regions of Washington have been conducting anti-idling campaigns especially around schools. Furthermore, in 2003, the Washington State legislature provided funding to retrofit more than 5,000 school buses with clean diesel technology (Washington State Department of Ecology, 2004). Additional funding has also been made available to retrofit other public fleets.

New diesel fuel and diesel engine standards are now in effect and are expected to reduce emissions, but diesel engines can run for a long time, so there will be many pre-standard engines on the road for many years to come. Also, population in the region is expected to increase; therefore, additional vehicle (diesel and gas) traffic will be expected to increase. This may offset many of the improvements made by new fuel and engine standards. Transportation planning will need to be an integral part of maintaining a decline in mobile sources contribution to air pollution into the future.

Port operations were not evaluated in this project, but they are expected to contribute significantly to air pollution. Recent goals set by the ports of Seattle, Tacoma, and Vancouver, British Columbia aim to reduce PM emissions from ships by 70% while they are in port, and to reduce emissions from land-based equipment by 30% (Port of Seattle, Port of Tacoma, and Vancouver Port Authority, 2007). Providing power plug-ins to ships is an example of measures being taken to reduce emissions while ships are in port.

Wood stove change-out programs have been implemented in the region to reduce the amount of

particulate emitted from homes using wood heat. It is not clear how effective these programs have been to date, but the concept is clear: replacing uncertified devices with cleaner ones has the potential to reduce the amount of wood smoke that is released into the ambient air. Uncertified woodstoves can emit more than two times the PM<sub>2.5</sub> as certified woodstoves, and pellet stove PM<sub>2.5</sub> emissions are roughly 10 times lower than uncertified woodstoves, while gas stoves are the cleanest burning. Continued or enhanced change-out programs would be expected to further reduce wood smoke PM emissions. Recently, House Bill 2261, an amendment to Washington State law (Revised Code of Washington 70.94.473), was passed by the legislature in 2007 and approved by governor Gregoire. This amendment established a Wood Smoke Work group that is studying ways to reduce exposure to wood smoke to protect public health. Recommendations should be presented to the governor and the legislature by December 2007.

### **Children's Health Concerns**

DOH recognizes that infants and children may be more vulnerable to exposures than adults when faced with contamination of air, water, soil, or food (Agency for Toxic Substances and Disease Registry, 1998). This vulnerability is a result of the following factors:

- Children are more likely to play outdoors.
- Children are shorter and their breathing zone is closer to the ground, resulting in a greater likelihood to breathe dust, soil, and heavy vapors.
- Children are smaller and receive higher doses of chemical exposure per body weight.
- Children's developing body systems are more vulnerable to toxic exposures, especially during critical growth stages in which permanent damage may be incurred.

The Children's Health Study in southern California documented numerous acute and chronic health outcomes in children related to common air pollutants (i.e., particulate matter, NO<sub>2</sub>, ozone, and CO) (Peters et al. 2004). Children with existing respiratory illness such as asthma are more susceptible to air pollutants. Other recent studies have shown that children living closer to major roadways are more likely to have respiratory symptoms or develop asthma (Gauderman et al. 2005, Kim et al. 2004, Brauer et al. 2002).

Evidence exists that air pollution at levels that meet current standards may still harm children's health. Work to reduce emissions from all sources should be continued along with a framework that recognizes children's susceptibilities to air pollution. For example, California's Senate Bill 352 enacted in 2003 requires that public school districts incorporate air quality concerns when siting new schools. The bill states that new schools in California should not be sited within 500 feet of major highways due to air pollution concerns from mobile sources. Some suggestions have been made that similar legislation should apply to child care facilities. Currently, there are roughly 168 child care facilities and 34 schools in King County, and 13 childcare facilities and 3 schools in south Seattle located within 500 feet of major highways.

## Conclusions

In summary:

- On-road mobile sources contribute to the highest risks near major roadways over a large area of south Seattle.
- Risks and hazards are greatest near major highways and drop dramatically about 200 meters from the center of highways.
- Point sources, especially those that emit chromium compounds, have the potential to impact residential areas in south Seattle resulting in increased theoretical cancer risk.
- Wood stove / fireplace use in the winter season contributes to health risk.

Limitations of the study:

- The point source emissions inventory used in this project is highly uncertain.
- Reported chromium emissions are likely over-estimated resulting in over-estimated health risks.
- Emissions from other mobile sources (i.e., planes, trains, and ships) were not quantified. These sources are likely to contribute to air pollution in the region.
- Although it is unclear how many woodstoves/fireplaces are used in south Seattle, wood smoke is clearly a pollutant of concern not only in south Seattle, but state-wide.
- A major limitation of the methodology used in this project was that the impact of wood smoke particulate matter relative to non-cancer hazards was not assessed. While theoretical cancer risk estimates are often highlighted with respect to particulate matter, it is important to understand that acute health effects related to particulate matter such as aggravation of asthma and triggering heart attacks are important public health concerns.

Although modeling and risk assessment cannot accurately predict the occurrence of air pollution related disease in the community, it can be used to prioritize efforts for reducing health risk. DOH concludes that long-term exposures to air toxics from multiple sources result in increased theoretical cancer risk (risk greater than  $1 \times 10^{-4}$ ) and non cancer hazard (hazard quotients greater than one). Overall, the lifetime increase of cancer risk associated with exposure to air toxics from multiple sources is low. The level of risk or hazard varies across the region depending in part on proximity to sources emitting toxic compounds. The primary air toxics contributing to theoretical

cancer risk at “hotspots” are diesel particulate, chromium compounds, benzene, 1-3 butadiene, and formaldehyde. Acrolein is the primary chemical resulting in elevated non-cancer hazards.

Mobile (on-road) source emissions result in elevated theoretical cancer risk near major highways with cancer risks approaching  $4 \times 10^{-3}$  at the point of maximum impact (near the center of Interstate-5). The lifetime increase of cancer risk associated with exposure to air toxics from mobile source emissions is considered moderate. Risk associated with diesel vehicles is 3 times higher than gasoline-powered vehicles ( $3 \times 10^{-3}$  vs.  $1 \times 10^{-3}$  at the point of maximum impact). Mobile (on-road) sources are also responsible for the bulk of non-cancer hazard with hazard quotients approaching 7 at the point of maximum impact (attributable primarily to acrolein exposure).

Woodstove sources result in a theoretical cancer risk that ranges from about  $1 \times 10^{-4}$  to  $5 \times 10^{-4}$ . The lifetime increase of cancer risk associated with exposure to air toxics from wood stoves is considered low. In addition to inhalation exposure, dermal absorption and soil ingestion were also considered when quantifying risks to wood smoke. Chronic and/or acute non-cancer health effects for wood smoke and diesel PM were not evaluated in this study. DOH acknowledges that PM is of great concern for non-cancer health effects.

Theoretical cancer risks attributable to chromium were estimated to be as high as  $1 \times 10^{-3}$  near chromium sources, however, it was noted that the emissions inventory for chromium emitters may be inaccurate. The lifetime increase of cancer risk associated with exposure to chromium emissions is considered moderate. In general theoretical cancer risks from point sources are lower if chromium compound emissions are reduced. Estimated theoretical cancer risks for on-road sources are lower within few hundred meters on either side of major roadways, and estimated theoretical cancer risks from wood stove sources can drop significantly if wood smoke emission is reduced.

## **Recommendations**

1. Refine the point source emissions inventory so that priorities can be identified and pollutant control strategies can be developed.
  - Chromium sources have the potential to impact residential areas of south Seattle, so all chromium sources should be located and inventoried.
  - Clean Air should consider inventorying emissions from sources not currently required to report their emissions.
2. Determine wood stove and fireplace usage in the south Seattle area and the need for a focused wood stove change-out program.
3. Determine impacts from other mobile sources that were not included in this assessment.
  - Port operations
  - Rail
  - King County Airport
4. Clean Air and other regulatory air agencies (and Ecology) should consider cumulative impacts from all emission units at each facility when issuing air permits.
5. Health agencies (e.g., DOH, Public Health-Seattle and King County, and ATSDR) should partner with regulatory agencies to increase education about air pollution and steps

individuals can take to reduce their impact and exposure.

6. Land use decisions should be made with an attempt to protect sensitive individuals from areas where air pollution is expected.
  - Consider proximity to highways or other known air pollution sources when siting new schools, daycares, retirement homes, and assisted living facilities.
  - Public Health-Seattle and King County's Built Environment and Land Use program could work to develop land use strategies and policies to help minimize the impacts of air pollution on sensitive individuals.

## **Public Health Action Plan**

### *Completed actions*

1. Clean Air has worked to identify chromium sources and refine chromium compound emissions rates from point sources in south Seattle.
2. Clean air has active programs aimed to reduce wood stove, diesel, and ship emissions.

### *Planned actions*

1. DOH will work with Clean Air and other agencies to conduct follow-up assessments and analyses once complete emission inventories are compiled.
2. DOH will work to explore and enhance the role of public health agencies with regard to air toxics and air pollution.
  - Public Health - Seattle and King County has drafted an air quality primer to educate people about air quality issues. PH-SKC intends to work with focus groups to ensure the product is useful to the community. Pending the outcome of focus groups and funding, translation, printing, and distribution would be the next steps.
3. DOH will identify land use zoning or designations that could result in increased exposure of sensitive individuals to air pollutants. Strategies will need to be developed to provide education or alternatives for reducing sensitive individuals exposure to air pollutants

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## References

- Agency for Toxic Substances and Disease Registry. 1998. Interim guidance on including child health issues in Division of Health Assessment and Consultation Documents. Atlanta: US Department of Health and Human Services, Public Health Service, July 1998.
- Brauer M, Hoek G, Van Vliet P, Meliefste K, Fischer PH, Wijga A, Koopman LP, Neijens HJ, Gerritsen J, Kerkhof M, Heinrich J, Gellander T, and B Brunekreef. 2002. Air Pollution from Traffic and the Development of Respiratory Infections and asthmatic and Allergic Symptoms in Children. *American Journal of Respiratory and Critical Care Medicine* 166(8): 1092-1098.
- California Environmental Protection Agency. 2003. The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments. August 2003. Available at URL [http://www.oehha.ca.gov/air/hot\\_spots/pdf/HRAguidefinal.pdf](http://www.oehha.ca.gov/air/hot_spots/pdf/HRAguidefinal.pdf)
- Dillingham Software Engineering Inc. and Bluescape Environmental. 2005. Duwamish Valley Regional Modeling and Health Risk Assessment. August 2005. Available at URL <http://www.doh.wa.gov/NOTpostedYET>
- EPA. 2007a. Query of Air Data Monitor Locator Map. Tool available at URL <http://www.epa.gov/air/data/monloc.html?st~WA~Washington>
- EPA. 2007b. PM Standards Revision - 2006. Available at URL <http://www.epa.gov/pm/naaqsrev2006.html>
- EPA. 2007c. Air Trends Basic Information. Available at URL <http://www.epa.gov/airtrends/sixpoll.html>
- Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, and R McConnell. 2005. Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide. *Epidemiology* 16(6):737-743.
- Graham, John D. 2004. Statement of John D. Graham, Ph.D. Administrator, Office of Information and Regulatory Affairs Before the Subcommittee on Energy Policy, Natural Resources and Regulatory Affairs, United State House of Representatives. February 25, 2004. Available at URL [http://www.whitehouse.gov/omb/legislative/testimony/graham/040225\\_graham\\_reg-know-act.html](http://www.whitehouse.gov/omb/legislative/testimony/graham/040225_graham_reg-know-act.html)
- Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, and B Ostro. 2004. Traffic-related Air Pollution near Busy Roads: the East Bay Children's Respiratory Health Study. *American Journal of Respiratory and Critical Care Medicine* (170) p. 520-526.
- Norris G, YoungPong SN, Koenig JQ, Larson TV, Sheppard L, and JW Stout. 1999. An Association Between Fine Particles and Asthma Emergency Department Visits for Children in Seattle. *Environmental Health Perspectives* 107: 489-493.

- Peters JM, Avol E, Berhane K, Gauderman J, Gilliland F, Jerrett M, Kunzli N, London S, McConnell R, Navidi B, Rappaport E, Duncan T, Lurmann F, Roberts P, Alcorn S, Funk T, Gong H Jr., Linn WS, Cass G, and H Margolis. 2004. Epidemiologic Investigation to Identify Chronic Effects of Ambient Air Pollutants in Southern California. Report Prepared for the California Air Resources Board and the California Environmental Protection Agency. Available at <http://www.arb.ca.gov/research/apr/past/94-331a.pdf>
- Port of Seattle, Port of Tacoma, Vancouver Port Authority, U.S EPA, Puget Sound Clean Air Agency. 2007. Joint News Release: PNW Port Join Forces to Reduce Maritime Emissions. May 16, 2007. Available at URL [http://maritimeairforum.org/news/Release\\_NW\\_Ports\\_CleanAirStrategy.pdf](http://maritimeairforum.org/news/Release_NW_Ports_CleanAirStrategy.pdf)
- Puget Sound Clean Air Agency. 2003. Final Report: Puget Sound Air Toxics Evaluation. October 2003. Available at URL [http://www.pscleanair.org/airq/basics/psate\\_final.pdf](http://www.pscleanair.org/airq/basics/psate_final.pdf)
- Puget Sound Clean Air Agency. 2006a. 2005 Air Quality Data Summary. Available at URL <http://www.pscleanair.org/news/library/reports/2005AQDSFinal.pdf>
- Puget Sound Clean Air Agency. 2006b. Memorandum: Revisions to Duwamish Valley Regional Modeling and Health Risk Assessment Table 6-3 a, b, c. December 2006.
- Puget Sound Clean Air Agency. 2007a. 2006 Air Quality Data Summary. Available at URL <http://www.pscleanair.org/news/library/reports/2006AQDSFinal.pdf>
- Puget Sound Clean Air Agency. 2007b. Regulation III. Last Revised March 22, 2007. Available at URL <http://www.pscleanair.org/regulated/reg3/reg3.pdf>
- Schwartz J. 1999. Air Pollution and Hospital Admissions for Heart Disease in Eight U.S. Counties. *Epidemiology* 10:17-22.
- Tolbert PE, Mulholland JA, MacIntosh DD, Xu F, Daniels D, Devine OJ, Carlin BP, Lein M, Dorley J, Butler AJ, Nordenberg DF, Frumkin H, Ryan PB, and MC White. 2000. Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia. *American Journal of Epidemiology* 151: 798-810.
- Washington Administrative Code, 173-460. Controls for New Sources of Toxic Air Pollutants. Last updated July 21, 1998. Available at URL <http://apps.leg.wa.gov/WAC/default.aspx?cite=173-460>
- Washington State Department of Ecology. 2003. 2000-2002 Air Quality Trends Report. Report # 03-02-008. Available at URL <http://www.ecy.wa.gov/pubs/0302008.pdf>
- Washington State Department of Ecology. 2004. Washington State Clean School Bus Program: Report to 2005 Legislature. Publication Number 04-02-029. December 2004. Available at URL <http://www.ecy.wa.gov/pubs/0402029.pdf>

Yu O, Sheppard L, Lumley L, Koenig JQ, and GG Shapiro. 2000. Effects of Ambient Air Pollution on Symptoms of Asthma in Seattle-Area Children Enrolled in the CAMP Study. *Environmental Health Perspectives* 108(12): 1209-1214.

## Appendix A: Summary of health effects associated with key air pollutants in south Seattle

Brief summaries of health effects of selected chemicals of concern in south Seattle air are provided in the following pages and were obtained in part from the sources listed below. For more detailed information of each chemical, follow the link provided.

- ATSDR's toxicological profiles
  - 1, 3- butadiene - <http://www.atsdr.cdc.gov/toxprofiles/tps28.html>
  - benzene – <http://www.atsdr.cdc.gov/toxprofiles/tp3.html>
  - chromium compounds - <http://www.atsdr.cdc.gov/toxprofiles/tp7.html>
  - formaldehyde - <http://www.atsdr.cdc.gov/toxprofiles/tp111.html>
  - acrolein - <http://www.atsdr.cdc.gov/toxprofiles/tp124.html>
- California EPA's Office of Environmental Health Hazard Assessment and the American Lung Association of California's sheet on the health effects of diesel [http://www.oehha.ca.gov/public\\_info/facts/pdf/diesel4-02.pdf](http://www.oehha.ca.gov/public_info/facts/pdf/diesel4-02.pdf)
- Ecology's fact sheet on the health effects of wood smoke <http://www.ecy.wa.gov/pubs/92046.pdf>

### 1, 3 - Butadiene

1,3- butadiene is always present at very low levels in the air around cities and towns. The average amount of 1,3-butadiene in the air is 0.3 parts of 1,3-butadiene per billion parts of air (ppb) (or 0.7 micrograms per cubic meter) in cities and suburban areas of the United States. In south Seattle, monitored average levels are much lower.

Short-term exposure to high levels of 1,3-butadiene causes eye, nose, and throat irritation. Studies of rubber industry workers suggested possible harmful effects such as more cases of heart diseases, blood diseases, lung diseases, and even cancer from the long-term exposure to low levels of 1,3-butadiene. These rubber industry workers were also exposed to other chemicals along with 1,3-butadiene, so we do not know for sure which chemical (or a combination of them) caused these effects. In addition, the effect of harmful habits like smoking was not considered in the evaluation of health risks of occupational exposure to 1,3-butadiene. Rats and mice exposed to 1,3-butadiene for a long time developed cancer in many organs.

### Benzene

Long-term exposure to benzene can cause cancer of the blood-forming organs. This condition is called leukemia. Exposure to benzene has been associated with development of a particular type of leukemia called acute myeloid leukemia (AML).

Breathing benzene for long periods may cause harmful effects in the tissues that form blood cells, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components. A decrease in red blood cells can lead to anemia.

Reduction in other components in the blood can cause excessive bleeding. Blood production may return to normal after exposure to benzene stops. Excessive exposure to benzene can be harmful to the immune system, increasing the chance for infection and perhaps lowering the body's defense against cancer.

### **Chromium compounds**

Chromium (VI) and compounds (calcium chromate, chromium trioxide, lead chromate, strontium chromate, and zinc chromate) are known human carcinogens. Long-term exposure to chromium has been associated with lung cancer in workers exposed to levels in air that were 100 to 1,000 times higher than those estimated in air in south Seattle. Chromium (VI) is believed to be primarily responsible for the increased lung cancer rates observed in workers who were exposed to high levels of chromium in workroom air. It is not clear how low levels of exposure typical in urban air contribute to health effects, but because Chromium VI is a known human carcinogen, it contributes to cancer risk when assessing risks in exposed populations.

### **Diesel Particulate Matter**

Diesel engines emit a complex mixture of air pollutants, composed of gaseous and solid material. The visible emissions in diesel exhaust are known as particulate matter or PM, which includes carbon particles or "soot." Diesel exhaust also contains a variety of harmful gases and over 40 other probable cancer-causing substances. In 1998, California identified diesel PM as a toxic air contaminant based on its potential to cause cancer, premature death, and other health problems. Those most vulnerable are children whose lungs are still developing and the elderly who may have other serious health problems. Each year in California, diesel PM is estimated to contribute to 2000 premature deaths and thousands of hospital admissions, asthma attacks and other respiratory symptoms, and lost workdays. Overall, diesel engine emissions are responsible for the majority of California's known cancer risk from outdoor air pollutants. In addition, diesel soot causes visibility reduction and is a potent greenhouse agent involved in global warming.

### **Formaldehyde**

Formaldehyde is irritating to tissues when it comes into direct contact with them. Some people are more sensitive to the effects of formaldehyde than others. The most common symptoms include irritation of the eyes, nose, and throat, along with increased tearing, which occurs at air concentrations of about 0.4–3 parts per million (ppm). One large study of people with asthma found that they may be more sensitive to the effects of inhaled formaldehyde than other people; however, many studies show that they are not more sensitive.

Several studies of laboratory rats exposed for life to high amounts of formaldehyde in air found that the rats developed nose cancer. Some studies of humans exposed to lower amounts of formaldehyde in workplace air found more cases of cancer of the nose and throat (nasopharyngeal cancer) than expected, but other studies have not found nasopharyngeal cancer in other groups of workers exposed to formaldehyde in air. The Department of Health and Human Services (DHHS) has determined that formaldehyde may reasonably be anticipated to be a human carcinogen (NTP). The International Agency for Research on Cancer (IARC) has

determined that formaldehyde is probably carcinogenic to humans. This determination was based on specific judgments that there is limited evidence in humans and sufficient evidence in laboratory animals that formaldehyde can cause cancer. The Environmental Protection Agency (EPA) has determined that formaldehyde is a probable human carcinogen based on limited evidence in humans and sufficient evidence in laboratory animals.

## **Wood Smoke**

The biggest health threat from wood smoke comes from fine particles (also called particulate matter or PM). These small particles can get into your eyes and respiratory system, where they can cause health problems such as burning eyes, runny nose, and illnesses such as bronchitis. Fine particles also can aggravate chronic heart and lung diseases—and are linked to premature deaths in people with these chronic conditions.

Some people with heart or lung disease (e.g., congestive heart failure, chronic obstructive pulmonary disorder, emphysema, asthma) may experience health effects earlier and at lower smoke levels than healthy people. Older adults are more likely to be affected by smoke, possibly because they are more likely to have chronic heart or lung diseases than younger people. Children also are more susceptible to smoke because their respiratory systems are still developing; they breathe more air (and air pollution) per pound of body weight than adults; and they're more likely to be active outdoors.

Particle exposure can lead to a variety of health effects. For example, numerous studies link particle levels to increased hospital admissions and emergency room visits—and even to death from heart or lung diseases. Both long- and short-term particle exposures have been linked to health problems.

*Long-term exposures*, such as those experienced by people living for many years in areas with high particle levels, have been associated with problems such as reduced lung function and the development of chronic bronchitis—and even premature death.

*Short-term exposures* to particles (hours or days) can aggravate lung disease, causing asthma attacks and acute bronchitis, and may also increase susceptibility to respiratory infections.

If you have lung disease, and you are exposed to particles, you may not be able to breathe as deeply or vigorously as normal. You may have respiratory symptoms including coughing, phlegm, chest discomfort, wheezing and shortness of breath.

In people with heart disease, short-term exposures have been linked to heart attacks and arrhythmias. If you have heart disease, particles may cause you to experience chest pain, palpitations, shortness of breath, and fatigue. Healthy children and adults have not been reported to suffer serious effects from short-term exposures, although they may experience temporary minor irritation when particle levels are elevated.

## **Acrolein**

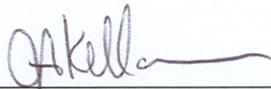
Acrolein can enter the environment as a result of burning wood, tobacco, vehicle fuels; overheating or burning of cooking oils; and accidental release from chemical plants or release

from a hazardous waste site. Acrolein that enters the air as a vapor changes into other chemicals within days.

Acrolein is very irritating to the eyes, nose, throat, lungs, stomach, and skin. In general, children are not likely to be affected by acrolein more than adults. However, children who are sensitive to irritants in the air (such as children with asthma) may be more sensitive to lung irritation from acrolein.

## Certification

This health consultation is a summary and interpretation of the Duwamish Valley Regional Modeling and Health Risk Assessment prepared by Dillingham Software Engineering Inc., for the Washington State Department of Health. The public health consultation was prepared by the Washington State Department of Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodology and procedure existing at the time the health consultation was initiated. Editorial review was completed by the cooperative agreement partner.



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Technical Project Officer, CAT, SPAB, DHAC

The Division of Health Assessment and Consultation (DHAC) ATSDR, has reviewed this health consultation and concurs with the findings.



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Team Lead, CAT, SPAB, DHAC, ATSDR