A Closer Look at Air Pollution in Houston:
Identifying Priority Health Risks

Report of the **Mayor's Task Force**
on the Health Effects of Air Pollution

Convened by the
INSTITUTE FOR HEALTH POLICY

THE UNIVERSITY of TEXAS
SCHOOL OF PUBLIC HEALTH

Under the auspices of
The University of Texas Health Science Center at Houston
and the
City of Houston

INSTITUTE FOR HEALTH POLICY REPORT ES-001-006

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Photo by **Heidi Bethel**

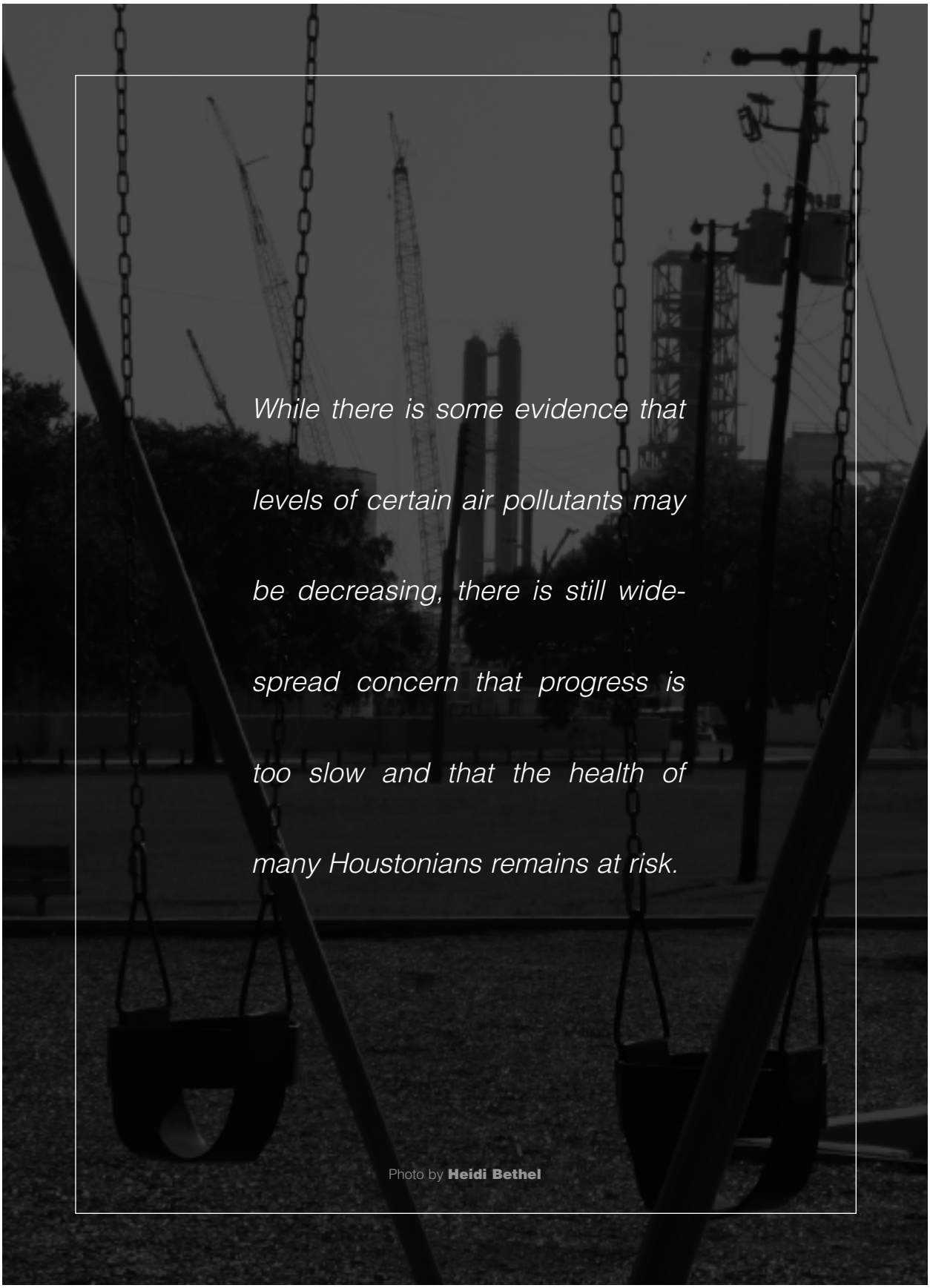

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Photo by **Heidi Bethel**

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Disclaimer

The opinions and interpretations expressed herein are the sole responsibility of the Mayor's Task Force on the Health Effects of Air Pollution and do not necessarily reflect the official views of their respective organizations or the views of the individuals and organizations who contributed their technical expertise. The authors have attempted to provide the most accurate information and analysis according to accepted research standards at the time of publication.

Abstract

Thousands of tons of potentially harmful chemicals are discharged each day into Houston's atmosphere as a result of human activities, substances, and technologies. Consequently, people living in Houston are exposed routinely to a myriad of pollutants in the air they breathe. Estimated and/or measured concentrations of some of these airborne chemicals in ambient air are high enough to cause illness or injury in exposed individuals, especially those in our society who are most vulnerable, such as children and seniors. Although the available data are incomplete and uneven, the Task Force surveyed information on 179 air pollutants and identified 12 substances in Houston's air that are definite risks to human health, 9 that are probable risks, and 24 that are possible risks. Sixteen substances were found to be unlikely risks to Houstonians at current ambient levels, and 118 substances were labeled uncertain risks because there was inadequate or insufficient information to determine whether they presently pose a health threat to Houston residents.

Introduction

It is no secret that ambient (outdoor) air pollution is a problem in Houston. So much so, in fact, that the city has, rightly or wrongly, been referred to as the smog capital of the U.S., and is widely perceived to be one of the most polluted cities in the country. Houston's air pollution predicament has been the subject of frequent media reports, the topic of numerous scientific articles, and the focus of public debate and political wrangling. And if Houstonians need any further reminding, they have only to venture outside during a pollution episode to see and smell the problem for themselves. While there is some evidence that levels of certain air pollutants may be decreasing, there is still widespread concern that progress is too slow and that the health of many Houstonians remains at risk.

Today, provisions of the federal Clean Air Act are forcing cities and states to find ways to reduce airborne levels of two virtually ubiquitous urban pollutants - ozone and particulate matter - or face severe penalties. The Act also mandates technology-based standards for many industrial processes to limit emissions of numerous chemicals and chemical classes, such as benzene, 1,3-butadiene, and polycyclic organic matter, referred to as hazardous air pollutants (HAPs). In addition, the Act limits emissions of many of these same chemicals and their precursors from mobile sources, including both on-road (e.g., cars, trucks, buses) and off-road (e.g., marine engines, construction equipment, aircraft, locomotives) sources. More recently, attention has also been directed towards reducing emissions from so-called 'area' sources, such as the collective air releases from dry cleaners, service stations, and restaurants.

Yet despite three decades of progressively more extensive and stringent regulatory controls, there remains a broad-based consensus among knowledgeable experts and the general public that air pollution concentrations in Houston are by and large unacceptable, that some Houstonians are likely to suffer from air pollution-related health effects, and that something must be done to rectify this unfortunate situation. An important first step in any attempt to improve the healthfulness of ambient air quality in Houston is to identify those pollutants liable to pose serious risks to human health so that more attention and resources can be directed towards mitigation efforts. In that spirit, the Mayor of Houston, the Honorable Bill White, asked the President of the University of Texas Health Science Center at Houston, Dr. James T. Willerson, to help answer a critical science-policy question.

> ***"Which ambient air pollutants are most likely to cause significant health risks for current and future residents of Houston?"***

In response, the Task Force on the Health Effects of Air Pollution (the Task Force) was formed under the auspices of the Institute for Health Policy based at the University of Texas School of Public Health. It is composed of environmental health experts from The University of Texas School of Public Health, The University of Texas Medical Branch at Galveston, The University of Texas M.D. Anderson Cancer Center, Baylor College of Medicine, and Rice University. These scientists surveyed available information on air pollution-related health risks relevant to the Greater Houston[1] area, and used scientific judgment to distinguish among different levels of chronic risk likely to be experienced by Houston residents.

The challenges confronting the Task Force as it worked to answer the Mayor's question reinforced the old adage, "If it were easy, somebody would already have done it." For example, although there are quantitative data on health risk values, exposure levels, and emission amounts for some air pollutants, they tend to be incomplete, uneven in quality, and uncertain. There is, moreover, a scarcity, and in some cases a total lack, of risk-related information for many potentially important chemicals and pollutants. Consequently, although the Task Force examined much quantitative information, the comparative assessment of air pollution-related health risks for Houstonians ultimately must rely on informed judgment rather than precise calculation. This lack of precision is due not only to a general insufficiency of relevant Houston-specific information, but also to deficits in our scientific understanding of exposure-response relationships and the etiology of many environmentally-influenced health outcomes.

[1] For purposes of this report, Greater Houston consists of the 10 county, Houston-Sugar Land-Baytown metropolitan statistical area (MSA) defined by the U.S. Census Bureau as of 2003.

Just because a task is difficult, however, does not necessarily mean that it is not worth doing. Members of the Task Force acknowledge that this exercise in comparative risk assessment involves unavoidably imprecise, uncertain, and incomplete data. Nevertheless, they believe strongly that the Mayor's question is the right question to ask, and that scientists should not shy away from responding, even when limited knowledge and inadequate understanding limit them to only partial or approximate answers.

The risk rankings provided in this report represent the consensus judgment of a group of objective, academic experts. They are meant to draw the attention of decision makers to those air pollutants that, after taking account of all available evidence, appear to constitute a real health threat to Houstonians. The results should be used as a direction finder, a compass if you will, to help guide decision makers as they struggle with difficult choices about how best to allocate limited resources among an overabundance of air pollution problems. In that context, findings of the Task Force should not be taken as the final word or absolute truth, but rather as an initial attempt to look comprehensively across the entirety of air pollution problems in Houston and set some provisional priorities. It is our intent that the conclusions of the Task Force be subject to continuous refinement and modification as new knowledge becomes available. Ultimately, we hope that the findings presented here will encourage constructive debate over better options for reducing health risks, as well as stimulate further research and continual re-examination of air pollution issues.

Background

Houston and Los Angeles are probably the two cities in the U.S. most associated in the public mind with air pollution. Over the past decade it was not unusual to see headlines like “Houston passes L.A. in smog” or “Los Angeles retakes lead in air pollution.” Houston, with a population of more than 2 million living in an area of more than 600 square miles, is the largest city in Texas and the fourth largest city in the U.S. (Los Angeles is second). It is the county seat of Harris County, which is the third most populous in the country. The Greater Houston area is the seventh largest metropolitan area in the U.S. with a population of more than 5 million residing in 10 counties.

As one would expect, there are numerous sources of air pollution in Houston. Tailpipe emissions from cars, trucks, and buses are a significant source of airborne pollutants owing to the fact that Houstonians drive on average more than 140,000,000 miles every day. A plethora of toxic pollutants are emitted into Houston's air by more than 400 chemical manufacturing facilities, including 2 of the 4 largest refineries in the U.S. The huge petrochemical complex along the Houston Ship Channel is the largest in the country, and the Port of Houston, which is the largest in the U.S. in terms of foreign tonnage and second in total tonnage, is the sixth-largest in the world. Adding to the city's air pollution are aggregate airborne emissions from many small operations spread geographically across Greater Houston, such as surface coating processes, dry cleaners, gas stations, printing processes, restaurants, charcoal barbecues, and gasoline-fueled lawn maintenance equipment.

Meteorology - Meteorological conditions and patterns also contribute to the air pollution problem in Houston. Between April and October there tends to be a high number of warm sunny days with stagnant winds, which causes ground-level buildup of air pollutant concentrations, especially photochemical oxidants such as ozone. Most air pollution episodes in Houston occur as the wind direction rotates continuously over a 24-hour period trapping a mass of stagnant, unmoving air over the city. In these situations elevated levels of air pollution occur in combination with high temperatures and humidity, making the air in Houston hazy, malodorous, and oppressive.

Pollutants and Sources - The pollution that sometimes degrades Houston's air quality is made up of thousands of airborne agents, including biological (e.g., ragweed pollen), chemical (e.g., benzene), and physical (e.g., noise) stressors, which individually and in combination may have an adverse effect on human health. Our focus in this report is on a subset of all chemical pollutants (or classes of pollutants) likely to be present in urban airsheds and known or suspected to harm people at sufficiently elevated concentrations. National Ambient Air Quality Standards (NAAQS) have been promulgated for six substances. In this report we focus on two of these pollutants - ozone and particulate matter. Another 188 substances are listed in the Clean Air Act as Hazardous Air Pollutants (HAPs) based on concerns about their toxicity, and

we focus on 176 of these and diesel particulate matter, which was recently designated as a Toxic Air Contaminant (TAC) by the State of California.

Most of the air pollutants are emitted directly into the air from one or more of four, major source categories: mobile sources, including both (1) on-road emissions from motor vehicles and (2) off-road emissions from ships, trains, airplanes, and heavy construction equipment; (3) industrial point sources, such as petroleum refineries along the Ship Channel; and (4) area sources, for example, aggregate airborne releases from all of the gas stations in Harris County. A few chemicals, such as ozone, are secondary pollutants not emitted directly by technological activities, operations and processes, but formed subsequently from complex reactions among chemical precursors in the atmosphere.

Air Monitoring - Air pollution levels in Houston have been monitored in one form or another since the early 1970s. It has been reported that in Greater Houston there are currently more than 140 air pollution monitors, owned by the Texas Commission on Environmental Quality (TCEQ), local governments, or private industry, operating at more than 20 locations and screening for more than 130 chemical pollutants. According to the TCEQ, "The air quality in Houston is monitored more closely and analyzed with more intensity than perhaps anywhere in the country - if not the world" (TCEQ, 2005).

The Houston air monitoring network is designed primarily to measure levels of six so-called 'criteria' pollutants - ozone, particulate matter, carbon monoxide, sulfur dioxide, nitrogen dioxide, and lead - for which the U.S. Environmental Protection Agency (EPA) has established health-based National Ambient Air Quality Standards (NAAQS). Houston air meets the standards for 5 of the criteria pollutants (all except ozone), and it is the largest metropolitan area in the country that meets the existing standard for fine (PM 2.5) particulate matter. However, Houston routinely exceeds the NAAQS standard for ozone. Moreover, monitors in the region have recorded some of the highest ozone readings in the nation. Consequently, eight counties - Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery, and Waller - have been designated by the EPA as a 'severe ozone nonattainment area'. Under provisions of the Clean Air Act, Houston must achieve attainment with the 8-hour ozone standard by June 15, 2010 (TCEQ, 2006; U.S. EPA, 2006a) or face severe penalties, including loss of federal highway funds. Because volatile organic compounds (VOCs) and nitrogen oxides (NOx) are the main precursors for photochemical ozone formation, substantial monitoring efforts have also been devoted to measuring these pollutants in Houston.

Growth and Air Quality - Over the past two decades, the City of Houston has experienced steady growth as illustrated by the consistently rising trends in population, vehicle miles traveled, employment, and gross area product shown in **Figures 1 and 2** on pg. 10 (Greater Houston Partnership, 2005). At the same time, reported emissions of many ozone precursors have decreased, and the number of days that ozone levels exceed the federal ozone standard has decreased by more than 50%. Similarly, since the early 1980s the number of days that any monitor in the ten-county Greater Houston area records 1-hour ozone concentrations ≥ 0.165 ppm, a level designated 'unhealthy' according to the EPA Air Quality Index, has decreased by more than 20%. However, in the last few years, ozone exceedances for Greater Houston (as opposed to the City of Houston as represented in **Figures 1 and 2**) have increased from a low of 40 days in 2002 to 51 days in 2005 (U.S. EPA, 2006e).

Identifying Priority Health Risks

To answer the Mayor's question, "Which ambient air pollutants are most likely to cause significant health risks for current and future residents of Houston?" it is necessary to distinguish the most serious health threats among a diverse mix of substances. Conceptually this exercise is straightforward, but in practice it is complicated by inadequate information on emissions, ambient concentrations, actual exposures, and linked health consequences, as well as incomplete scientific understanding of risk-related processes and mechanisms.

A fundamental principle in environmental toxicology is that "the dose makes the poison," which is to say that there is a set of exposure conditions for every chemical that makes it toxic and, conversely, there is another set of exposure conditions that makes it either non-toxic or without significant effects. Thus, hypothetically, even a minimally toxic chemical like table salt can cause harm at elevated exposures, while even a highly toxic chemical like asbestos can be harmless at negligible exposures. Among the variables affecting dose are the dose-response relationship, the magnitude, duration, frequency, tim-

Figure 1

Gross Area Product, Employment and Ozone Exceedances, City of Houston

1980-2004

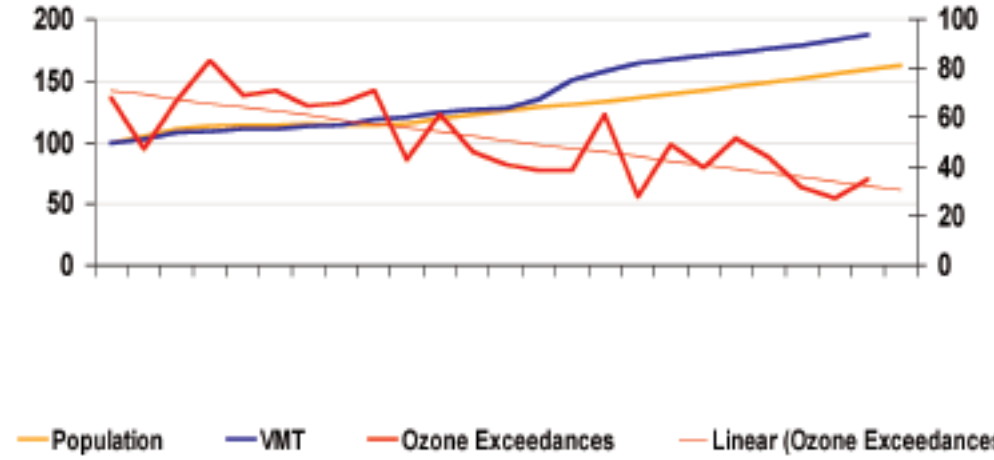
(1 HOUR STANDARD)

Source: The Perryman Group, Fall 2004. GAP – Gross Area Product. From *Clearing the Air on Clean Air for the Greater Houston Region* – Greater Houston Partnership, 2005.

Figure 2

Population, Vehicle Miles Traveled and Ozone Exceedances, City of Houston

1980-2004



(1 HOUR STANDARD)

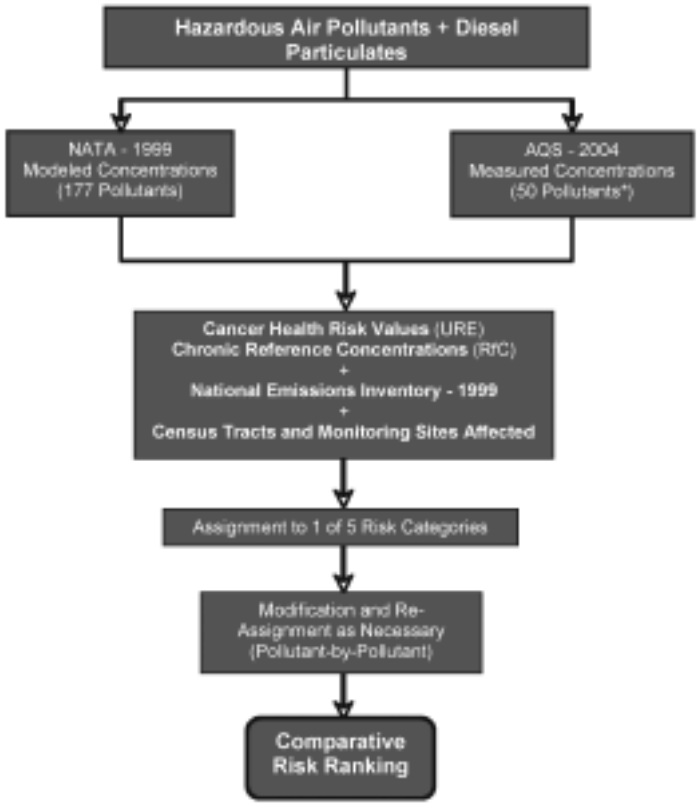
Source: The Perryman Group, Fall 2004 (Population); Texas Department of Transportation (VMT; some years interpolated). VMT - Vehicle Miles Traveled. From *Clearing the Air on Clean Air for the Greater Houston Region* – Greater Houston Partnership, 2005.

ing, and route of exposure, and other factors like nutrition, health status, age, sex, and genetic makeup.

The health risk posed by a particular air pollutant is usually thought of as a combination of both the likelihood and severity of harm that may be experienced by people exposed to typical ambient concentrations present in the indoor and outdoor air in their communities. A "screening" or approximate estimate of health risk can be calculated by comparing a measured or modeled ambient concentration against an established health risk value - a threshold level based on the probability that an individual (or members of a defined population) exposed to that airborne concentration for a lifetime will develop cancer.

Theoretically, at least, this approach produces a rough numerical estimate of chronic risk for each pollutant, which can then be used to sort individual chemicals into appropriate risk categories. But in reality there are numerous complications. For example, there are no established (consensus-based, government-sanctioned) health risk values for over half of the HAPs. Furthermore, most HAPs are not measured routinely at urban monitoring sites so there is a scarcity of actual measurements to either estimate ambient concentrations or verify models used to predict ambient concentrations. As a result, comparative assessment of air pollution-related health risks is unavoidably an exercise in scientific judgment based on incomplete and imperfect data.

Figure 3
Overview of the Risk Ranking Approach Used by the Task Force



*Note that all pollutants in the AQS dataset where also included in the NATA dataset.

Ranking Process - The Task Force used a systematic process to survey the available information and compare relative risks among air pollutants in Houston. There are health-based standards (NAAQS), as well as abundant health effects information and extensive exposure data for the two criteria pollutants (ozone and particulate matter) included in this analysis. Therefore, assignment of ozone to a particular risk category was based on how often, and by how much, ambient concentrations exceeded the NAAQS. No such ambient concentration exceedances were found for PM 2.5 concentrations in 2000 through 2005 so the ranking was based on the weight of the evidence indicating that exposures at or below the existing standard may contribute to increased morbidity and mortality. The task of assigning HAPs to particular risk categories was more difficult for three reasons: there are currently no health-based standards, as there are for ozone and PM 2.5; there tends to be less data on linkages between exposure and effects; and measurements of ambient concentrations are generally spotty or completely lacking. The approach used by the Task Force to compare relative risks among these substances is summarized graphically in **Figure 3** and explained more fully in **Appendix 1**.

To obtain estimates of ambient concentrations for as many HAPs as possible, the Task Force used modeled annu-

al average concentrations for 1999 from EPA's National-scale Air Toxics Assessment (NATA) (U.S. EPA, 2006d). A description of NATA 1999 is presented in **Appendix 2**. Results from the NATA provided estimated ambient concentrations for 177 substances (176 HAPs and diesel particulate matter) in 895 census tracts (each with approximately 4,000 inhabitants) included in the 10-county Greater Houston area. The NATA values were derived by EPA using a computerized air dispersion model that combined 1999 airborne emissions data from outdoor sources, including point, mobile (on-road and non-road), area, and background sources with Houston-specific meteorological variables. The model also took into consideration the breakdown, deposition and transformation of pollutants in the atmosphere after their release. The Task Force supplemented these data with measured 2004 annual concentrations for 50 pollutants (49 HAPs plus a diesel particulate matter surrogate) from 20 monitoring sites in and around Houston - 14 in Harris County, 4 in Galveston, 1 in Brazoria, and 1 in Montgomery. These data were obtained from EPA's Air Quality System (AQS); for a description of the AQS dataset see **Appendix 2**. All AQS data used for risk ranking was from 2004 (U.S. EPA, 2006e), the most recent year for which complete data were available.


Photo by Heidi Bethel

To get a sense of relative health risks associated with estimated ambient concentrations of HAPs, the Task Force used health-related toxicity values developed for health risk assessments by either the U.S. EPA or the California Office of Environmental Health Hazard Assessment (OEHHA), whichever value was the more stringent (health protective) (California EPA & OEHHA, 2002; California OEHHA, 2005; U.S. EPA, 2005, 2006h, 2006i). In instances when no value was developed by US EPA or California OEHHA, health values from other available sources were used. A detailed table of health values is presented in **Appendix 3, Table A3.1**. For carcinogens, estimates were based on their respective unit risk values (UREs), which represent the excess lifetime cancer risk estimated to result from continuous lifetime exposure to an average concentration of 1 microgram per cubic meter (µg/m3) of a certain pollutant in the air. For noncarcinogens, estimates were based on comparison of estimated ambient concentrations with their respective chronic non-cancer inhalation health values: reference concentrations (RfC) - used by U.S. EPA; reference exposures levels (REL) - used by California OEHHA; or minimum risk levels (MRL) - used by the Agency for Toxic Substances and Disease Registry (ATSDR).

Each HAP was assigned initially to a specific risk category contingent on how measured or modeled annual-average concentrations translated into comparative risk estimates using established UREs (carcinogens) and/or RfCs, RELs, or MRLs (noncarcinogens). Initial risk-category assignments were adjusted, as necessary, based on evaluation of additional information about relative emission quantities and number of census tracts or monitoring stations affected. See **Appendix 1** for a thorough explanation on the ranking process.

Final Risk Categories - Using the process outlined above, the Task Force assigned each of the 179 air pollutants (176 HAPs modeled and/or monitored, ozone, fine particulate matter, and diesel particulate matter) to one of five comparative risk categories. Substances were designated "**Unlikely Risks**" when there was suggestive evidence of negligible or insignificant risk to the general population and vulnerable subgroups. Substances were deemed "**Uncertain Risks**" when there was inadequate or insufficient evidence to ascertain whether they posed a significant risk to the general population

and vulnerable subgroups. Substances were designated "**Possible Risks**" when there was partial or limited evidence that suggested they might constitute a significant risk under certain circumstances, and "**Probable Risks**" when there was substantial corroborating evidence that they were likely to represent a significant risk under the right conditions. Those substances for which there was compelling and convincing evidence of significant risk to the general population or vulnerable subgroups at current ambient concentrations were labeled "**Definite Risks**."

As shown in **Table 1**, 12 air pollutants were classified as "Definite Risks". The Task Force found that existing and projected ambient concentrations of two criteria pollutants - ozone and fine particles (PM 2.5) - are almost certainly causing respiratory and cardiopulmonary effects in some individuals as well as contributing to premature death. It was also determined that airborne concentrations of seven carcinogens - diesel particulate matter (see **Appendix 4** for more detail on this pollutant), 1,3-butadiene, chromium VI (see **Appendix 4** for more detail on this pollutant), benzene, ethylene dibromide, formaldehyde, and acrylonitrile - pose an unacceptable increased cancer risk. In addition, it was concluded that five substances -- 1,3-butadience (reproductive effects in addition to being a carcinogen), formaldehyde (respiratory effects), acrolein (respiratory effects), chlorine (respiratory effects), hexamethylene diisocyanate (pulmonary and respiratory effects). -- are present at ambient concentrations that represent an unacceptable increased risk for chronic disease in Houston.

The evidence is not as strong but nevertheless persuasive that an additional 9 air pollutants are likely to pose unacceptable health risks at concentrations measured or modeled in Houston air. These substances were designated as "Probable Risks," and included eight carcinogens - vinyl chloride, acetaldehyde, ethylene dichloride, naphthalene, arsenic compounds, carbon tetrachloride, ethylene oxide, 1,1,2,2-tetrachloroethane - and one pollutant - acrylic acid - that has chronic non-cancer effects. These are shown in **Table 2** on pg. 14.

Table 1. Definite Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint	Point	Mobile		Area
				On Road	Off Road	
Ozone		Respiratory /Cardiovascular/Immune	N/A	N/A	N/A	N/A
Fine Particulate Matter (PM 2.5)	Yes	Respiratory /Cardiovascular	X	X	X	X
Diesel Particulate Matter	Yes	Respiratory		X	X	
1,3-Butadiene	Yes	Female reproductive	X	X	X	X
Chromium VI	Yes	Respiratory	X	X	X	X
Benzene	Yes	Immune	X	X	X	X
Ethylene Dibromide (Dibromoethane)	Yes	Male reproductive	X			X
Acrylonitrile	Yes	Respiratory	X			X
Formaldehyde	Yes	Respiratory; Eyes	X	X	X	X
Acrolein	No	Respiratory	X	X	X	X
Chlorine	No	Respiratory	X			X
Hexamethylene Diisocyanate	No	Respiratory	X			X

[1]Emissions taken from the National Emission Inventory (NEI), 1999; (U.S., EPA, 2006c)

[2]Only chronic health effects associated with chronic health value used in the analysis are depicted in the table.

Table 2. Probable Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint	Point	Mobile		Area
				On Road	*Off Road*	
Vinyl Chloride	Yes	Alimentary (liver)	X			X
Acrylic Acid	No	Respiratory	X			X
Acetaldehyde	Yes	Respiratory	X	X	X	X
Ethylene Dichloride (1,2-Dichlorcethane)	Yes	Alimentary (liver)	X			X
Naphthalene	Yes	Respiratory	X	X	X	X
Arsenic Compounds (Inorganic may including Arsine)	Yes	Development	X			X
Carbon Tetrachloride	Yes	Alimentary (liver)	X			X
Ethylene Oxide	Yes	Nervous	X			X
1,1,2,2-Tetrachloroethane	Yes		X			X

[1]Emissions taken from the National Emission Inventory (NEI), 1999; (U.S., EPA, 2006c)
[2]Only chronic health effects associated with chronic health value used in the analysis are depicted in the table.

The evidence available for another 24 air pollutants was even more limited, but still suggestive that Houstonians might, in certain situations, experience negative health consequences from exposure to plausible concentrations in ambient air. Twenty-two of these substances are carcinogens and, as summarized in **Table 3** on pg. 15, the Task Force classified them as "Possible Risks".

The Task Force deemed 16 air pollutants to be "Unlikely Risks" (See **Table 4** on pg. 16) because available evidence suggests that they probably create no significant threat of harm for Houstonians. Two of these substances - coke oven emissions and nitrosodimethylamine - have zero reported emissions; two have negligible modeled ambient concentrations; and 12 have unknown emissions in the Greater Houston Area.

The Task Force labeled 118 air pollutants as "Uncertain Risks". The complete listing appears in **Appendix 5**. Pollutants were assigned to this category because there was inadequate or insufficient information to determine whether they currently pose a significant health threat to the residents of Houston. There are almost twice as many substances assigned to this risk category as to the other four classifications combined. Of these 118 air pollutants, 16 are carcinogens emitted in Greater Houston for which UREs are available; 45 are noncarcinogens emitted in Greater Houston for which RfCs are available; 17 are emitted here and have both a URE and RfC; and finally, 27 are emitted here but have neither a URE nor an RfC. Another 13 pollutants of the 118 do not appear in the emissions inventory for the Greater Houston Area, 1 of which (1,2-diphenylhydrazine) is a carcinogen with a URE (see **Appendix 5**).

In summary, the Task Force surveyed data on ambient concentrations (from the U.S. EPA and the Houston monitoring network) for 179 air pollutants that might potentially affect the health of Houstonians. Of these 179 pollutants, 137 HAPs have related health-based benchmarks (from the U.S. EPA and California OEHHA) and 2 pollutants (ozone and fine particulate matter) are regulated by National Ambient Air Quality Standards. After reviewing the evidence, it was the collective opinion of Task Force members that, currently and into the foreseeable future, 12 substances are definite risks, 9 are probable risks, 24 are possible risks, 118 are uncertain risks, and 16 are unlikely risks. The most appropriate focus for additional public health concern and effort is initially on the 21 substances ranked as either definite or probable risks. As shown in Tables 1 and 2, they represent a combination of carcinogens and non-carcinogens emitted by a diversity of source categories.

Caveats - It is critical to understand that assessment of air pollution-related health risks is not an exact science. For example, annual fatalities in a particular city from car accidents, homicides, or lightning strikes can be determined quite

Table 3. Possible Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint	Point	Mobile		Area
				On Road	Off Road	
Nickel Compounds	Yes	Respiratory; Immune	X	X	X	X
Manganese Compounds	No	Nervous	X	X	X	X
Cadmium Compounds	Yes	Kidney; Respiratory	X		X	X
Titanium Tetrachloride	No	Respiratory	X			X
2,4-Dinitrotoluene	Yes	Alimentary (liver)	X			X
Methyl Tert-Butyl Ether	Yes	Alimentary (liver); Kidney; Eyes	X	X	X	X
1,3-Dichloropropene	Yes	Respiratory	X			X
Chloroform	Yes	Alimentary (liver)	X			X
Methylene Chloride (Dichloromethane)	Yes	Cardiovascular; Nervous	X			X
p-Dichlorobenzene	Yes	Alimentary (liver)	X			X
Propylene Oxide	Yes	Respiratory	X			X
Tetrachloroethylene (Perchloroethylene)	Yes	Kidney; Alimentary (liver)	X			X
Trichloroethylene	Yes	Nervous; Eyes	X			X
1,1,2-Trichloroethane	Yes	Alimentary (liver)	X			X
Bis(2-Ethylhexyl)Phthalate (Dehp)	Yes	Respiratory	X			X
Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	Yes	Respiratory	X			X
Lead Compounds	Yes	Development	X		X	X
1,2-Dibromo-3-Chloropropane	Yes	Male reproductive				X
1,4-Dioxane	Yes	Alimentary (liver) /Kidney/Hematologic	X			X
2,4-Toluenediamine	Yes		X			X
Acrylamide	Yes	Nervous	X			X
Benzidine	Yes	Nervous				X
Dichloroethyl Ether (Bis[2-Chloroethyl]Ether)	Yes		X			X
Polycyclic Organic Matter (POMs)	Yes		X	X	X	X

[1]Emissions taken from the National Emission Inventory (NEI), 1999; [2]Only chronic health effects associated with chronic health value used in the analysis are depicted in the table.

accurately from death certificates. But the number of fatalities related to air pollution cannot be so easily and precisely ascertained, except when exceptional pollution episodes cause significant and proximal increases in mortality, as in the Meuse Valley in 1930, Donora, Pennsylvania in 1948, and London in 1952. Today, improved air quality in most American cities, and the fact that cause-and-effect relationships are less well-defined at lower ambient concentrations, make it necessary to use statistical techniques, along with appropriate scientific assumptions and approximations to estimate the number of "theoretical" deaths from air pollution likely to occur under artificial (but hopefully realistic) exposure scenarios.

Efforts to measure air pollution-related risks (both morbidity and mortality) directly are stymied by an array of problems that make it difficult to establish causality between typical levels of urban air pollution and connected adverse health effects. Among the common obstacles that normally confront risk assessors are the following:

- Incomplete understanding of disease etiology;
- Wide range of non-environmental causes for most diseases to which environmental agents contribute;
- Environmental pollutants often enhance or exacerbate, rather than only cause disease or dysfunction;

Table 4. Unlikey Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint	Point	Mobile		Area
				On Road	Off Road	
1,3-Propane sultone	Yes					
2,4-Dinitrophenol						X
2,4,6-Trichlorophenol	Yes					
2-Chloroacetophenone		Respiratory				
3,3-Dimethoxybenzidine	Yes					
3,3-Dimethyl benzidine	Yes					
Arsine	Yes	Hematological	X			X
Chlorobenzilate	Yes					
Coke Oven Emmissions	Yes					
Ethyl carbamate	Yes					
Ethylene thiourea	Yes	Endocrine				
Lindane	Yes	Kidney				
Nitrosodimethylamine	Yes					
N-Nitrosomorpholine	Yes					
p-Dimethylaminoazobenzene	Yes					
Toxaphene	Yes					

[1]Emissions taken from the National Emission Inventory (NEI), 1999; [2]Only chronic health effects associated with chronic health value used in the analysis are depicted in the table.

- Lack of suitable methods, measurements, and models to a) estimate exposure, dose, and effects, and b) characterize variability over individuals, time, and space;
- Deficiency of surveillance and reporting systems for exposure and environmentally-related health outcomes;
- Long latency period from exposure to negative health consequences for many environmentally-induced diseases (e.g., lung cancer);
- Real-world exposures occur not to a single pollutant, but to complicated mixtures of environmental agents that vary both temporally and spatially;
- Observed health endpoints (e.g., lung damage) may not be the primary target of the environmental agent (e.g., immune system); and
- Inherent variability among individuals in terms of biological (e.g., genetic) susceptibility to environmentally-induced illness and injury.

It is also important to keep in mind that the Task Force considered only a specific and narrowly defined type of risk - namely the harmful chronic (long-term) effects of human inhalation exposure to estimated annual-average outdoor concentrations of 179 chemical pollutants. Air pollution can also cause acute (short-term) effects in people, as well as serious impairment to ecological resources (e.g., fish, wildlife) and damage to social welfare (e.g., poor visibility, degraded property values). People are exposed to other chemical, biological, and physical agents in the air they breathe, and real-life exposures are not just to outdoor air pollutants but also to airborne contaminants inside residences, cars, workplaces, restaurants, and other settings. Also, certain substances in Houston's ambient air, including photochemical degradation products and short-lived intermediates, may pose significant health risks, and are not well understood because of their complex photochemistry. Consideration of these and other potentially noteworthy factors, such as cumulative effects from simultaneous or sequential exposure to multiple stressors by various pathways and routes, were explicitly excluded from this initial assessment to make the task manageable and feasible within time and resource constraints.

Finally, it should be remembered that the Task Force used only data that were on hand or easily obtainable to complete its assessment. Ambient concentration estimates by census tract were only available for one year (1999) from NATA's most recent

assessments, and monitoring data from 20 stations in Houston were only available for a small fraction of HAPs, and only analyzed in depth for 2004, the most recent complete year. The Task Force used "off-the-shelf" health values (UREs and RfCs/RELs/MRLs) from the U.S. EPA (U.S. EPA, 2005, 2006h, 2006i), the California OEHHA (California EPA & OEHHA, 2002; California OEHHA, 2005) and the Agency for Toxic Substances and Disease Registry (ATSDR) to estimate health risks, implicitly assuming that these unmodified risk values were uniformly applicable to the Houston situation and population.

SUMMARY OF AIR POLLUTION-RELATED HEALTH EFFECTS

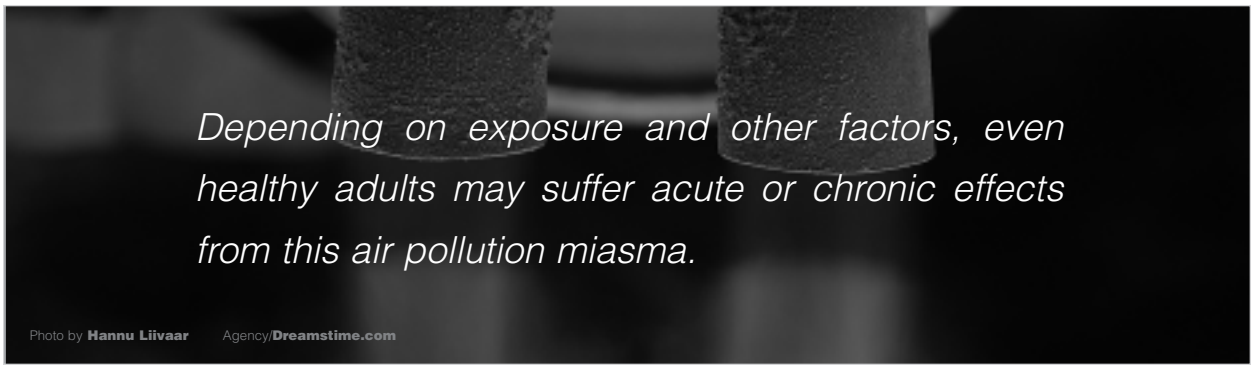
Thousands of epidemiologic (human) and toxicologic (animal) studies conducted over the past 35 years have documented the fact that urban air pollution at sufficiently elevated concentrations can adversely affect human health. Poor air quality can potentially cause or contribute to a variety of harmful outcomes, ranging from subtle biochemical and physiological changes, to symptoms like headaches, eye and throat irritation, wheezing and coughing, difficulty breathing, aggravation of existing respiratory and cardiovascular conditions, chronic respiratory disease, cancer, and premature death. Although the most obvious effects are typically on the respiratory and cardiovascular systems, many air pollutants can harm development processes and be toxic to other systems, including, among others, nervous, reproductive, immune, digestive, urinary and endocrine systems. In addition, numerous air pollutants are known or suspected human carcinogens.

Ozone-related health effects are of special interest because Houston currently exceeds the NAAQS standard. Ozone is a strong oxidizing agent, and short-term exposures on the order of minutes to hours can impair pulmonary function, decrease lung volumes and flows, and increase airway responsiveness, resistance, and irritation. Evidence indicates that a substantial fraction of summertime hospital visits and admissions for respiratory problems are associated with elevated short-term ozone levels. Repeated daily short-term exposure to ozone can cause an increased response to bronchial allergen challenges in subjects with preexisting allergic airway disease, with or without asthma. Long-term exposure to ozone over months to years can cause structural changes in the respiratory tract, and may play a role in causing irreversible lung damage. Ozone exposure can also impair the immune system so that people are more susceptible to respiratory infections, like colds and pneumonia.

Although Houston does not exceed the current NAAQS for either of the regulated fractions of particulate matter (PM 2.5 and PM 10), it is likely to exceed the new fine (PM 2.5) particle standard if and when it is promulgated. Particulate matter is a combination of solid, liquid, and solid-liquid particles suspended in air, and typically is composed of a complex mixture of organic and inorganic constituents. Fine particles, with aerodynamic diameters ≤ 2.5 microns, are taken into the deepest part of the lungs, where they tend to remain trapped among millions of tiny alveoli. Short-term exposures (minutes to hours) to elevated levels of PM 2.5 have been linked with physiological changes, biomarkers of cardiac changes, decreased lung function, increased respiratory symptoms, emergency room visits and hospitalization for cardiopulmonary diseases, and mortality from cardiopulmonary diseases. Longer-term exposures (months to years) have been causally associated with effects on the respiratory system, such as decreased lung function, development of chronic respiratory disease, and mortality from cardiopulmonary diseases and lung cancer.

There is no NAAQS for diesel particulate matter, however, concerns about human health effects recently prompted California to list it as a Toxic Air Contaminant (TAC) (California ARB, 1998; California ARB & OEHHA, 1998). Diesel exhaust, which is ubiquitous in urban environments, is a complex mixture of hundreds of toxic substances, including gaseous and particulate constituents. The particles in diesel exhaust are mostly 2.5 microns, and are composed of an elemental carbon core with adsorbed organic compounds and small amounts of sulfate, nitrate, metals, and other trace elements. Short-term exposures (minutes to hours) may cause eye, throat, and bronchial irritation, lightheadedness, nausea, cough, and phlegm, as well as exacerbation of allergic responses and asthma-like symptoms. Long-term exposures (months to years) may play a role in chronic respiratory disease, and are likely to increase the risk of developing lung cancer.

Short-term, high-level exposure (minutes to hours) to many of these substances, like benzene, toluene, and



formaldehyde, can cause headaches, difficulty breathing, nausea, confusion, and seizures. Long-term, lower-level exposure (months to years) to HAPs may cause many different adverse health effects, including cancer and damage to respiratory, circulatory (cardiovascular), nervous, reproductive, digestive (GI tract), endocrine, and immune systems, as well as kidney, blood and developmental effects. Despite the fact that many HAPs are ever-present in urban atmospheres, few cities or communities have extensive monitoring networks for this diverse concoction of air pollutants.

A recently released study by the U.S. EPA, the National-scale Air Toxics Assessment or NATA, examined the effect of 1999 emissions on ambient concentrations and related exposures across the U.S. (U.S. EPA, 2006b). They found that nationally, benzene accounted for almost 25 percent of the estimated lifetime cancer risk from the HAPs studied, and that together with six other pollutants -- carbon tetrachloride, chromium VI, polycyclic organic matter (POM), 1,3-butadience, formaldehyde, and coke oven emissions -- accounted for over 90% of the estimated HAP-related cancer risk. Acrolein (respiratory effects), formaldehyde (respiratory effects), and diesel particulate matter (variety of effects) were found to pose the top three non-cancer health risks among HAPs. Acrolein alone contributed 91 percent of the risk for respiratory effects nation-wide.

Although air pollutants are typically identified, studied, assessed, and regulated one at a time, this is obviously not the way they are encountered as part of everyday urban life. On a "smoggy" day in Houston, or a typical day for that matter, residents are simultaneously exposed to a complicated mix of ozone, particulate matter, carbon monoxide, sulfur dioxide, nitrogen oxides, lead, diesel exhaust, benzene, POM, 1,3-butadiene, formaldehyde, and hundreds of other airborne chemicals. Depending on exposure and other factors, even healthy adults may suffer acute or chronic effects from this air pollution miasma. But those most likely to be affected are the elderly, particularly those with lung and heart disease, children and adults with asthma, chronic obstructive pulmonary disease or other respiratory illnesses, individuals with cardiovascular disease, pregnant women and their fetuses, and children in general because, compared to adults, they inhale more air per kilogram of body weight, breathe more rapidly, and tend to breathe through their mouth more often.

For more information on health effects of pollutants in the Definite Risk category, see **Appendix 6**.

VULNERABLE POPULATIONS

A diversity of factors may affect the nature and magnitude of health risks associated with breathing a specific concentration of polluted air. Suppose, for example, that ambient air pollution levels in a large city in the upper Midwest are equivalent to those in Houston. Related chronic health risks for residents in one city may, nevertheless, differ dramatically from the other because of differences in climate (e.g., temperature, relative humidity), meteorology (e.g., wind speed, mixing heights), building characteristics (e.g., air exchange rates), commuting modes and patterns (e.g., use of public transportation, time spent in traffic), activity patterns and lifestyles (e.g., percentage of time indoors versus outdoors, exercise and nutritional habits), smoking prevalence (e.g., proportion of children living in homes with smokers), and socio-demographic and occupational characteristics of the population (e.g., age distribution, genetic makeup, median household income and education).

The reality is that, even at similar ambient pollutant levels, air pollution-related health risks can diverge considerably not only from city to city, but also from community to community, neighborhood to neighborhood, street to street, house to house, and person to person.

Just as different individuals may respond dissimilarly to the same dose of a particular prescription medicine, so too can different individuals be affected dissimilarly by equal concentrations (or doses) of air pollution. The nature, likelihood, and severity of air pollution-related health effects are directly related to the vulnerability of exposed individuals and populations. In this context, vulnerability is used to mean the conditions determined by physical, social, economic, and environmental factors or processes, which increase the susceptibility of a community or an individual to the impact of hazards. There are four general types of vulnerability that influence air pollution-related health effects: inter-individual differences in biological susceptibility; differential exposure; disparities in preparedness to cope with air pollution exposure; and divergence in the ability to recover from air pollution exposure. It is important to note that these categories are not mutually exclusive, and that populations with disproportionate numbers of vulnerable individuals will be more likely to suffer air pollution-related discomfort, dysfunction, disability, disease, and death (U.S. EPA, 2003).

Biological Susceptibility - Some people are genetically predisposed to experience adverse effects from air pollution because they have genetic polymorphisms that change the level of expression of a gene or the activity of gene product, such as an enzyme. Life stage can also affect susceptibility, and it is well established that pregnant women, fetuses, children, and the elderly tend to be more prone to air pollution-related effects. Furthermore, those with preexisting medical conditions, such as asthma or heart disease, are also more likely to endure adverse effects from air pollution exposure.

Differential Exposure - When two individuals or populations have different exposures to air pollution, they are at different points on the dose-response curve, which means that they may have dissimilar likelihoods of suffering adverse effects. This can be true for contemporaneous exposure (e.g., two individuals are exposed to different air pollution levels at the same time), historical exposure (e.g., two individuals are exposed to the same level now but had different exposures in the past), background exposure (e.g., two individuals have the same exposure now to ambient (outdoor) air pollution but have different current exposure to indoor (or background) air pollution), and body burden (e.g., two individuals have the same exposure now to air pollution but have different levels of environmental chemicals, their metabolites, or reaction products in their bodies).

Another important factor that may affect disparities in exposure to ambient air pollution is the presence and use of air conditioning (cooling and heating systems). The use of air conditioning isolates indoor from outdoor air, and decreases the infiltration of ambient pollutants into residences and other buildings. Residents of economically disadvantaged neighborhoods may either not have air conditioning, or limit its use, resulting in dependence on natural ventilation, and thus greater exposure to outdoor pollutants.

Disparities in Preparedness to Cope - Differences in the quality and quantity of coping systems and resources available to an individual or population can affect their ability to withstand the effects of air pollution exposure. For example, two children may be exposed to the same concentration of air pollution, but one may suffer no ill effects because her parents could afford disease immunizations, routine medical and dental checkups, daycare, a healthy diet, and vitamin supplements, while the other may get sick because she did not have these same advantages - and thus was less able to withstand the air pollution insult.

Divergence in Ability to Recover - Differences in the quality and quantity of coping systems and resources available to an individual or population can affect their ability to recover from the effects of air pollution. For example, two children with air pollution-induced respiratory problems may be exposed to the same concentration of air pollution, but one may have fewer symptoms, less severe symptoms, less frequent disease episodes, slower progression of the disease, and a better prognosis for full recovery because his parents are more health conscious, more knowledgeable about environmentally-induced disease, more in control of their home environment and, most importantly, more affluent, which means they can afford health insurance, better medical care, prescription medicine, and more nutritious food (U.S. EPA, 2003).

CUMULATIVE RISKS FROM EXPOSURE TO MULTIPLE AIR POLLUTANTS

Vulnerable groups as well as the general public are exposed every day during normal activities to a varied array of thousands of environmental pollutants in the air they breathe, the water and beverages they drink, the food they eat, the surfaces they touch, and the products they use. The cumulative effects of this complex and ever-changing brew of environmental stressors, including biological (e.g., Mycobacterium tuberculosis), chemical (e.g., 1,3-butadiene), physical (e.g., heat, noise), and psychosocial (e.g., job- or family-related stress) agents, may be critically important for accurate assessment of environmentally-induced risks, including those related to air pollution. We know, for example, that exposure to tobacco smoke and asbestos or radon increases the risk of developing lung cancer over what would be expected from simple addition of individual effects. Moreover, there is evidence that exposure to noise and toluene results in higher risk of hearing loss than from either stressor alone, that exposure to polycyclic aromatic hydrocarbons and ultraviolet radiation increases toxicity to aquatic organisms, and that adults with increased perceived stress and children of parents experiencing stress are more susceptible to viral infections.

Thus, it is essential to keep in mind that the health risk of any particular chemical in outdoor air is just a lone contributor to the cumulative risk from the sum of all chemicals breathed in ambient air, which, in turn, is merely a share of the cumulative risk associated with aggregate airborne chemical exposures that occur in all indoor and outdoor environments and for all occupational and non-occupational activities. Even this is only part of the story, however, because to estimate cumulative inhalation risk it is also necessarily to take account of the effects from concurrent exposure to biological, physical, and psychosocial stressors. In the end, a realistic estimate of cumulative health risks from total air pollution exposure would have to incorporate not only consideration of the variables described above, but also of the contemporaneous risks from all pertinent routes of exposure (i.e., inhalation, ingestion, and dermal absorption) over all applicable temporal and spatial dimensions.

In reality, comprehensive assessment of cumulative, air pollution-related health risk is presently precluded by the lack of appropriate methods, measurements, and models to estimate relevant exposures and related health effects. We are, for example, unsure in most cases whether the combined consequences of inhalation exposure to multiple air pollutants are likely to be independent (substances cause separate, unrelated effects), additive (effect of one substance adds to the other), synergistic (effects are more than additive), or antagonistic (effects are less than additive). In the absence of better information, it is common practice to assume that risks are additive for all airborne carcinogens (regardless of type of cancer), and for all systemic toxicants (i.e., causing chronic effects other than cancer, such as injury to the respiratory or nervous systems) that affect the same organ system (e.g., respiratory, cardiopulmonary, neurologic, reproductive).

The bottom-line message is that the risk categories discussed earlier are based solely on consideration of the health effects caused by ambient (outdoor) concentrations of each individual substance or group of substances acting alone. Risk rankings might change, for instance, if we took account of actual exposures, which are determined by combining information about (a) airborne concentrations in various indoor and outdoor locations, (including both occupational and non-occupational settings) through which people move, and (b) the time they spend in each place (or microenvironment). Further modifications could occur if the rankings factored in other cumulative risk issues, such as interactions among multiple pollutants that cause similar effects or the combined vulnerabilities of highly exposed populations.

A CASE STUDY - CUMULATIVE RISKS IN A VULNERABLE COMMUNITY

At this point, it is useful to illustrate how the characteristics of populations and neighborhoods can relate to sources of hazardous air pollutants and put some people's health at much greater risk. An earlier section introduced the notion that people may be more vulnerable to pollution's health effects for a variety of reasons including whether they live closer to high concentrations of pollutants, already suffer from disease or dis-

ability, have inadequate means to cope with stresses, or fewer resources to recover. The neighborhoods of East Houston share many of these characteristics and provide a concrete example of how different risks can add up when they are concentrated in a few areas.

About half of the point sources for air pollution in the Greater Houston area are concentrated on the eastern side of Harris County. Over twenty of the largest industrial sources are located in East Houston. The Port of Houston, and the Ship Channel that feeds it, passes through the middle of this area and generates a variety of hazardous pollutants, adding to those from the nearby industrial sources. Four major highways intersect this area including, Interstate Highways 10, 610 and 45 and State Highway 225; each generating substantial pollution from high traffic density. Within the City of Houston, there are nine super-neighborhoods that span this area: Denver Harbor/Port Houston, Pleasantville, Clinton Park/Tri-Community, Magnolia Park, Lawndale/Wayside, Harrisburg/Manchester, Pecan Park, Park Place, and Meadowbrook/Allendale. On the basis of location alone these neighborhoods appear far more vulnerable to health risks than others in Greater Houston.

More detail can be provided by the National-scale Air Toxics Assessment (NATA) 1999 (U.S. EPA, 2006d), since it has modeled ambient concentrations of pollutants at the level of the census tract. There are 895 census tracts in the Greater Houston area, and 28 of these are located in the nine super-neighborhoods in East Houston. If we consider only the 12 pollutants whose concentrations and toxicity put them in our highest risk category, most census tracts have one or two pollutants present at this high level. Ozone, for example is relatively pervasive. The revealing contrast comes in the comparison between the total picture of the 895 census tracts and a closer look at the 28 that make up our super-neighborhoods.

Figure 4 shows the tally of how many census tracts register harmful ambient concentrations of HAPs (that is, at the level of a definite health risk) for one or more pollutants in the Greater Houston area. Over 80 percent of all census tracts show three or fewer pollutants at a level that high.

Figure 4 Greater Houston Area Census Tracts by Number of Definite Risk Pollutants

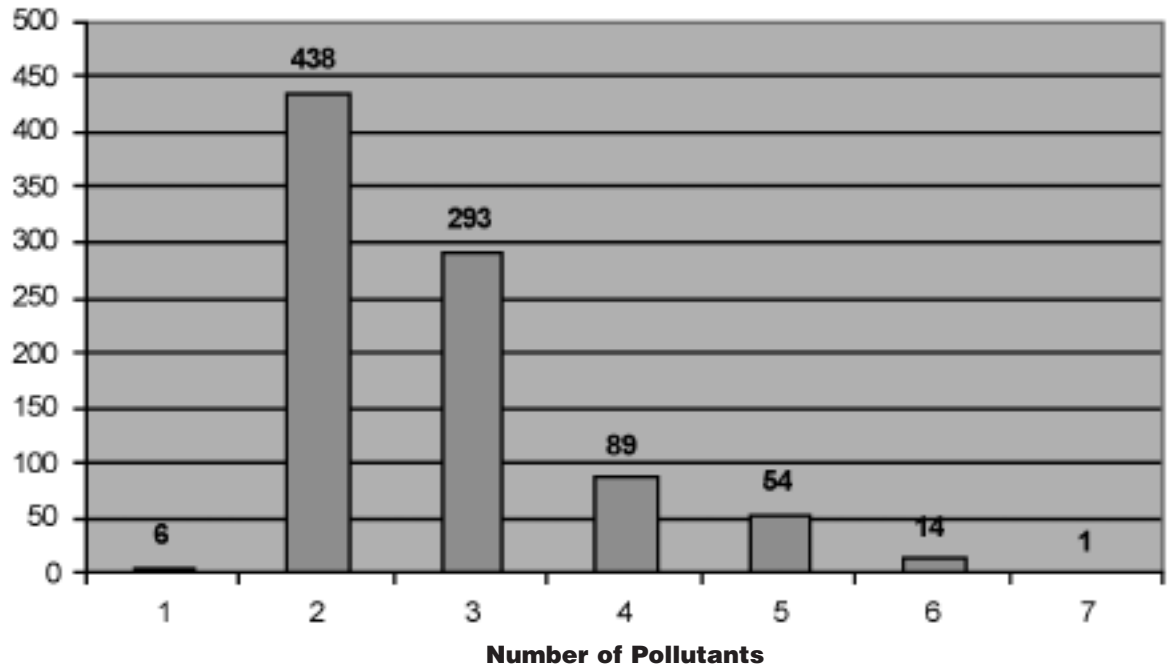


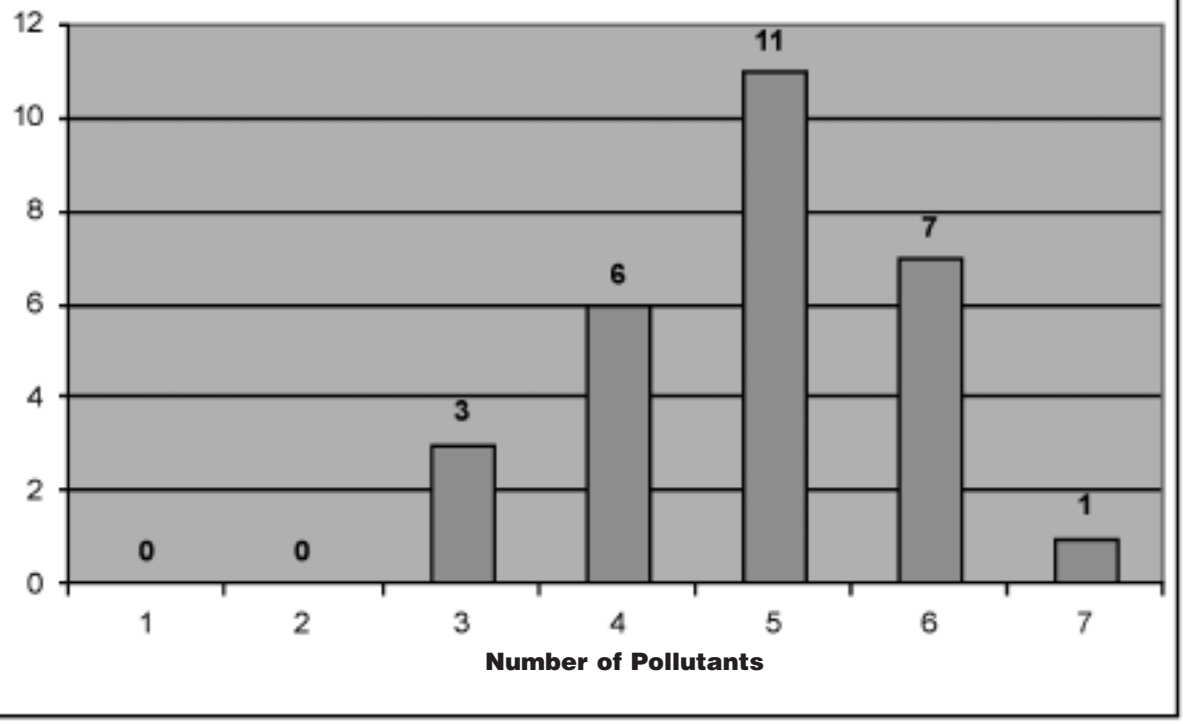
Figure 5 gives the corresponding tally for our East Houston neighborhoods. None of the East Houston census tracts have fewer than 3 pollutants in the highest risk category. Almost 90 percent of the census tracts located here have four or more pollutants present. Further, the one tract in the entire Houston area that has seven pollutants present at our highest risk level falls in one of these neighborhoods. Of the tracts throughout Greater Houston that have 6 or more pollutants, fully half of them appear in East Houston.

The way these pollutant concentrations are distributed disproportionately in East Houston neighborhoods suggests a greater burden of exposure for residents there, as compared to those living in other parts of the city. If we consider that the effects of exposure to each different pollutant can be cumulative, then neighborhoods with 5 or more pollutants present will face a higher lifetime risk of cancer or chronic disease than those where only one or two of these pollutants are found.

If we factor in some of the other dimensions of vulnerability mentioned above, then the overall risks to health increase still further. The median level of family income in our 9 super-neighborhoods is more than 30 percent lower than for the City of Houston; over a quarter of the residents fall below the poverty level. Almost 20 percent of the residents have less than a ninth grade education. These neighborhoods have some of the highest uninsured rates for health coverage in Harris County.

Consider the census tracts that have 6 or 7 of the 12 pollutants found at levels that pose a definite risk to health. These tracts appear in orange and red on the map in **Appendix 7**. Two super-neighborhoods account for the majority of these tracts: Clinton Park/Tri-Community and Harrisburg/Manchester, the latter containing the tract in red with 7 pollutants. Harrisburg/Manchester is the poorer of the two; the median per capita income (drawn from the U.S. Census for 2000) is $8,820. For Clinton Park, it is $9,529. As a reference point, the City of

Figure 5 East Houston Census Tracts by Number of Definite Risk Pollutants



Houston reaches $21,701. These are neighborhoods where residents live on less than half of the income of their fellow Houstonians.

In Harrisburg/Manchester, 37 percent of the residents have less than a high school education, and 32 percent fall below the Federal poverty level - double the rate for the surrounding county. In Clinton Park, 27 percent have less than a high school education, and the same percent fall below the poverty level. The residents in these neighborhoods are also segregated by race or ethnicity. Clinton Park is over 90 percent African-American. Harrisburg/Manchester is 88 percent Hispanic. Further, the pattern of land use shows pockets of residences surrounded by industrial sites, either disposal lagoons for dredged material from the Ship Channel at Clinton Park or fence lines behind heavy industry for Harrisburg/Manchester. The conditions necessary for healthy lifestyles, economic sustenance and quality of life for residents are fewer here than in most neighborhoods.

Aside from vulnerability, there is also the question of whether the sources of the pollutants posing the highest risks are the same in East Houston as in the rest of the Greater Houston Area. As it turns out, they are typically not the same. For East Houston, NATA attributes the ambient modeled concentrations of 7 of the top 12 pollutants to point sources; for the Greater Houston Area, this number drops to 3. East Houston had no pollutants where area sources dominated among those in the definite risk category; Greater Houston had 1. Between on-road and non-road mobile sources, the most dramatic difference is for diesel particulate matter: over 90 percent of the ambient modeled concentrations in East Houston neighborhoods are attributed to non-road mobile sources compared to three-quarters of the total in Greater Houston.

The map in **Appendix 7** also shows several monitoring sites where one or more of the pollutants in the definite risk category are currently being measured (The supplemental **Table A8.1** in **Appendix 8** shows which pollutants are monitored). Since these sites record ambient concentrations, the levels present in any given census tract cannot be accurately determined without considering factors such as wind direction and temperature. Nonetheless, the sites that appear in **Appendix 7** recorded annual average concentrations for 2004 that exceeded our health value thresholds for posing definite health risks. Three of these sites are contained in or adjacent to the neighborhoods that also had the largest number of definite risk pollutants, based on NATA modeled estimates for 1999.

In sum, East Houston neighborhoods that face a number of vulnerabilities based on their marginal social and economic standing also carry a heavier burden of health risks from breathing pollutants in their air. They tend to be located closer to major point sources than most other neighborhoods in the Greater Houston area and to be nearer to major transportation corridors. The burden of these risks taken together poses special needs in these neighborhoods.

CONCLUSIONS AND RECOMMENDATIONS

Substantial efforts have been devoted over the years to scrutinizing air pollution levels in Houston, and considerable resources have been expended on mitigation measures. Although the success of these endeavors is difficult to quantify, it appears that levels of some air pollutants, like ozone, have decreased since the early 1980s even though Houston's population, economy, and traffic have grown steadily. Much of the progress over the past 35 years can be attributed to regulatory controls mandated by the 1970 Clean Air Act and subsequent amendments. But air quality improvements in Houston appear to have slowed or even stalled recently, and there is legitimate concern that matters will only get worse. A critical first step in finding cost-effective solutions is to identify those airborne pollutants that represent the most serious health risks so that control strategies can be designed to focus on the worst risks first.

Historically, federal and state regulatory efforts have been directed primarily toward meeting National Ambient Air Quality Standards for the 6 criteria pollutants commonly found in urban air. Most of the attention in Houston has been on ozone -- the only criteria pollutant for which the city is not in compliance -- because of harsh penalties mandated by the Clean Air Act if ambient ozone concentrations do not meet the 8-hour standard by June 2010 (an unlikely prospect). There is also a growing body of evidence indicating that fine particulate matter causes significant health effects at ambient concentrations below the

existing NAAQS. Consequently, it is possible that the standard will be lowered, thereby putting Houston in noncompliance and making it subject to further penalties. Regardless of the statutory issues surrounding ozone and PM 2.5, and despite longstanding and ongoing control programs, the Task Force determined that current outdoor concentrations of both ozone and fine particulate matter represent a real and present threat to the health of Houston residents.

Diesel exhaust is a complicated chemical mixture that contributes to ambient levels of both gaseous and particulate air pollution in urban and rural environments. It contains many known or suspected cancer-causing substances as well as other harmful pollutants that may cause acute and chronic health effects. The widespread use of diesel engines means that diesel exhaust and its by-products are ubiquitous in urban atmospheres, and exposure is virtually unavoidable for city dwellers. Among those most likely to experience higher-than-average exposures are commuters, including children riding school buses, bus and truck drivers, operators of heavy equipment, and people living near busy streets and roadways, port facilities, industrial plants, and truck loading and unloading operations. Although direct measurements of ambient concentrations are unavailable, indirect estimates of diesel particle levels in Houston suggest that residents are experiencing increased risk of illness and premature death from current exposures.

The identification of ozone, PM 2.5, and diesel particulate matter as definite health risks is relatively straightforward owing to the comparatively large data base on adverse health effects that exists for each substance, along with clear evidence that people are exposed to outdoor levels considered unsafe. The picture is generally less certain and more problematic for the HAPs, which include a diverse mix of carcinogens and systemic toxicants. These air pollutants historically have received less regulatory attention, and ambient concentrations and exposure-effect relationships tend to be less well characterized. Accordingly, unambiguous assignment of these substances to a particular risk category is often hindered by incomplete and inadequate data, making it necessary in many instances to use scientific judgment as a basis for extrapolating beyond the limited or nonexistent data base.

Despite these difficulties, the Task Force found convincing evidence that 12 HAPs are definite health risks for Houstonians - 4 carcinogens, 4 systemic toxicants, 2 substances that are both, ozone, and fine particulate matter (See **Appendix 8, Table A8.1**). Another 9 (7 cancer-causing agents, 1 systemic toxicant, and 1 that is both) were designated probable risks because the Task Force deemed there was sufficient, although less compelling evidence that they currently pose significant health risks for people living in Houston (see **Appendix 8, Table A8.2**). Although available data were partial and uneven, the Task Force also decided there was sufficient suggestive evidence to justify labeling an additional 24 substances - 20 carcinogens, 2 systemic toxicants, and 2 that are both - as possible health risks at ambient concentrations in Houston air. A further 16 substances, all carcinogens, were found to represent unlikely health risks because there are no known emissions in the Houston area and/or modeling suggested that ambient levels are likely to be negligible (see **Table 4**).

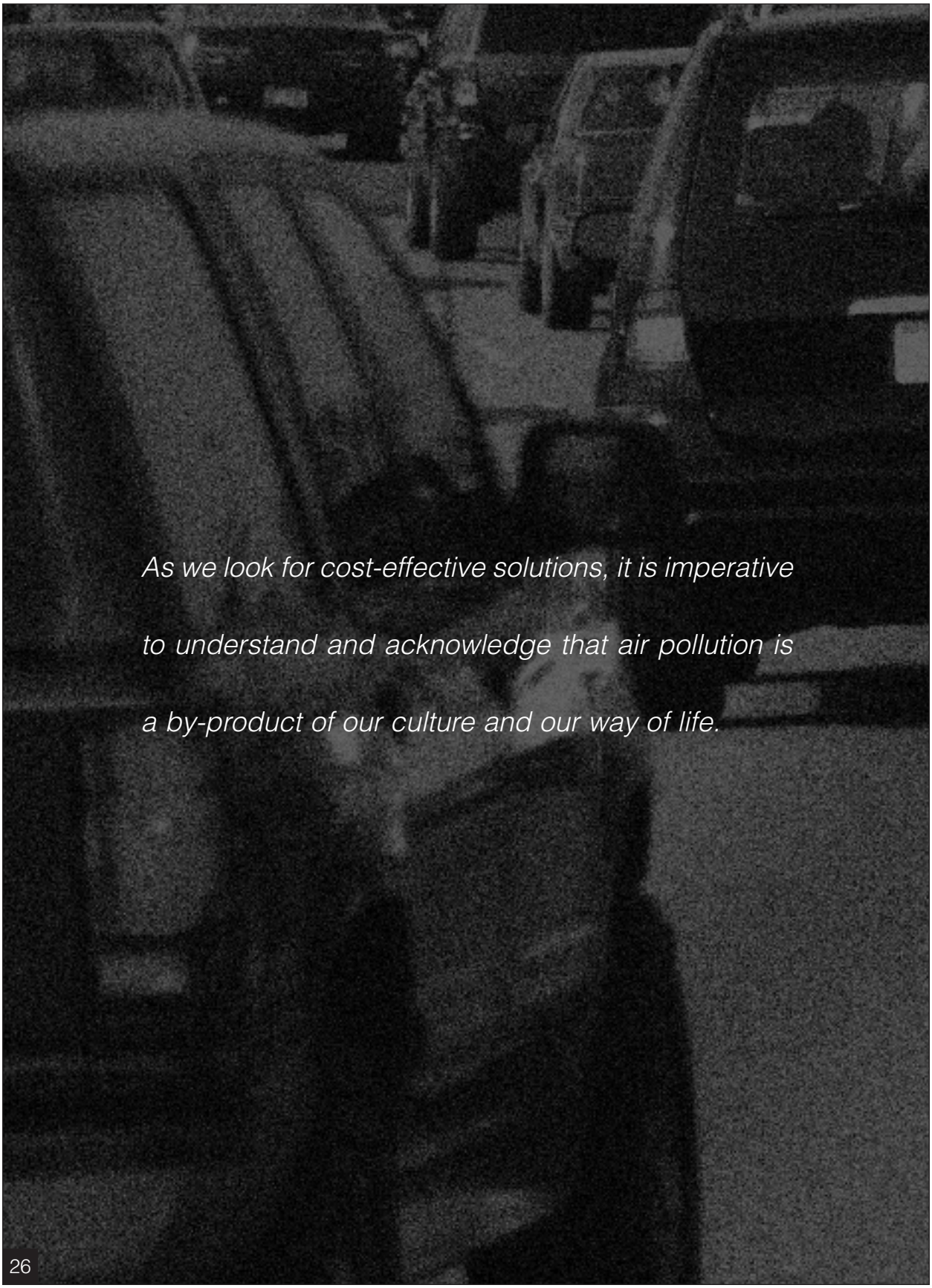
The intrinsic challenges of comparing HAPs-related health risks are illustrated by the fact that 118 (67%) of the 176 HAPs examined by the Task Force were assigned to the uncertain risk category. This decision was based on our collective judgment that there is insufficient evidence on hand to ascertain whether these substances currently pose a significant threat to the health and well-being of Houston residents. In short, it was not possible to say, with an acceptable degree of certainty, whether these pollutants are a health risk. Obviously, from a public health perspective this leaves us in an unsatisfying situation, wherein we lack the necessary scientific information to distinguish among definite, probable, possible, and unlikely health risks. Only targeted research aimed at filling critical data gaps and resolving crucial uncertainties will allow us eventually to (a) determine the appropriate risk category for HAPs presently listed as uncertain risks, and (b) verify the risk assignments for HAPs in other categories.

Notwithstanding the inherent scientific uncertainties, the results of our assessment further reinforce the prevailing opinion of many experts that ambient air pollution in Houston is harmful to exposed individuals and populations. Furthermore, we know that air pollution-related health risks disproportionate-

ly affect those most vulnerable - the young, the elderly, the sick, the pregnant, the unborn, and the poor. Cumulative health risks from combined effects of concurrent exposure to multiple air pollutants are a particular concern in vulnerable populations. Socio-economically disadvantaged groups, for example, are more likely to live near industrial facilities and busy roadways, where air pollution levels are typically elevated. Moreover, they are also more likely to work in hazardous occupations, to reside in dilapidated housing with inadequate air conditioning, to eat a substandard diet, to smoke cigarettes and drink alcohol, and to generally live more stressful and less healthful lifestyles. It therefore makes sense from a public health perspective to direct attention and resources toward high-risk groups so as to anticipate and prevent adverse effects, if possible. Failing that, emphasis should be placed on stopping or limiting exposures that damage the health and well-being of the most vulnerable in our society.

As we look for cost-effective solutions, it is imperative to understand and acknowledge that air pollution is a by-product of our culture and our way of life. It is produced as a direct result of choices we make, both individually and collectively, about energy sources, technologies, economic activities, and lifestyles. While the relative contribution of a particular source or source category may vary from place to place, it is the blending together of combined emissions from numerous point, mobile, and area sources that makes Houston's air quality unhealthful. Thus, focusing on a single type of source, no matter how obvious or obnoxious, is unlikely, by itself, to solve the problem.

In summary, we view the comparative risk process as a decision tool for organizing and analyzing information about air pollution in a manner that will aid decision makers as they choose among competing priorities. It is not, in our opinion, a decision rule that automatically and inevitably leads to a specific conclusion about resource allocation. We hope our risk rankings will be a useful adjunct to other relevant information, and that results will contribute to informed decisions not only about how to use available resources more effectively and efficiently, but also about how to justify the need for additional funding. We recommend that decision makers avoid using our findings as a detailed road map that provides precise directions about how to move forward; instead, we recommend that they use results as a compass to help determine appropriate directions for the development of an overarching strategy to address Houston's air pollution problem.



As we look for cost-effective solutions, it is imperative to understand and acknowledge that air pollution is a by-product of our culture and our way of life.

Appendices

Photo by Heidi Bethel

Appendix 1

The Risk Ranking Procedure: An Illustration for Benzene

Consider the key question addressed in this report: "Which ambient air pollutants are the most likely to pose significant health risks for current and future residents of Houston?" For purposes of this report, ambient air pollutants include: HAPs and diesel particulates, as well as two criteria pollutants, ozone and fine particulates. The task was to assign priority among these contaminants based on the relative health risk that each poses to the residents of the Greater Houston area. Although a full quantitative risk assessment was not possible, we were able to screen the pollutants by comparing estimates of their ambient concentrations against authoritative health risk values for cancer and reference values for chronic disease, whenever these were available. Health risk values were calculated from inhalation, unit risk estimates. The reference values were based on inhalation, reference concentrations; while not a direct estimate of risk, these specify levels at or below which adverse health effects are not likely to occur. The full set of unit risk estimates and reference concentrations, as well as their respective sources, appears in **Appendix 3**. As a rule, we relied on current, EPA-sanctioned (final, peer-reviewed) values and concentrations, unless more stringent levels had been promulgated by California EPA. Estimates of ambient concentrations were drawn from two, independent sources: the NATA modeled averages for 1999 (available in Spring of 2006) (U.S. EPA, 2006d) and the monitoring averages for 2004 drawn from EPA's Air Quality System (U.S. EPA, 2006e) (See **Appendix 2** for a description of these data).

Since our purpose is to establish an ordering among pollutants in terms of relative risks, we created 4 ranked categories - unlikely, possible, probable and definite -- each designating a particular level of risk. A fifth category, uncertain, was added to cover instances when ambiguity or a lack of information kept us from determining an appropriate risk level. Using categories permits us to accommodate a range of numerical values at each risk level and to allow for imprecision in our estimates. We are also able to take advantage of some widely-used qualitative distinctions among risk levels, making the categories more meaningful.

The assignment of pollutants to these 5 risk categories works in three rounds. In the first and longest round, data on the ambient concentration of each pollutant are collected from NATA's modeled estimate for each census tract and from the measured estimates from AQS monitors. These data are then screened relative to selected threshold levels for each unit risk estimate and reference concentration corresponding to the pollutant under consideration.

There were 4 threshold concentrations computed from each available unit risk estimate; these formed the boundaries of 5 risk groupings, each corresponding to added lifetime cancer risk to the population - "Below 1/1,000,000" "Between 1/100,000 and 1/1,000,000" "Between 1/10,000 and 1/100,000" "Between 1/1,000 and 1/10,000" "1/1,000 and Greater". Similarly, there were 3 percentile thresholds computed for each reference concentration, also leading to 5 groupings - "Below 50% RfC" "Between 75% and 50% RfC" "Between 100% and 75% RfC" "Between 150% and 100% RfC" and "150% and Above".

Pollutants are then assigned to the appropriate grouping based on their modeled NATA concentrations and their measured AQS concentrations, taken separately. As a result, there are four distinctive orderings: a unit risk estimate grouping for NATA concentrations and one for AQS concentrations, together with a reference concentration grouping for each. Within each of these groupings, pollutants are sorted first by their relative emissions masses reported in the National Emissions Inventory (NEI) for 1999 (U.S. EPA, 2006c). Four percentile categories were used: "90th Percentile and Above" "89th to 75th Percentile" "74th to 50th Percentile" and "Below 50th Percentile". Within each of these categories, pollutants are then sorted by the number of census tracts or monitors yielding concentrations above the threshold risk or reference levels for that grouping; this provides a rough indication of the relative extent of exposure in the population. The mass and location factors become important in the third round. Those pollutants with neither a unit risk estimate nor a reference concentration are assigned to a residual group, as are those with either no concentrations reported or modeled concentrations of zero.

In the second round, we apply a decision rule to take us from the 4 elaborate orderings developed in the first round to our 5 overall risk categories. In effect, the rule assigns each

grouping to a particular risk category and, thereby, creates a default assignment for each pollutant. Pollutants with concentrations that place them in the two highest groupings - "Between 1/1,000 and 1/10,000" and "1/1,000 or Greater" for unit risk thresholds and "Between 150% RfC and 100% RfC" and "150% RfC and Above" for reference thresholds -- go to the "Definite Risk" category. Those in the next highest grouping go to Probable Risk; and those in the grouping below that one go to Possible Risk. Those in the lowest grouping - "Below 1/1,000,000" and "Below 50% RfC" - are assigned to the Uncertain category, along with the pollutants in the residual group without unit risk estimates or reference concentrations. Pollutants with evidence of no emissions in the Greater Houston Area, modeled concentrations of zero, or no measured concentrations reported, go to the Unlikely category. Note that both AQS and NATA concentrations are combined in the same categories at this point. Although preference is given to the measured over the modeled data, only just over 20 pollutants with a health risk or reference value have both kinds of data. Although these pollutants may appear in multiple categories as a result, in every case, we assign them to the highest category in which they appear.

In the third round, adjustments are made to improve the reliability of the default assignments. Here, the emissions and location factors come into play. If there is evidence that emissions levels have changed dramatically, the pollutant can be moved to a lower risk category (1 pollutant). Pollutants whose assignments are based on a modeled concentration in only a single census tract can be moved to a lower risk category (7 pollutants). Those whose unit risk estimates are based on oral rather than inhalation evidence can be moved to a lower category (3 pollutants). A total of 11 pollutants were moved in this round to produce the final assignments to our 5 risk categories.

To illustrate this process, consider the pollutant, benzene; it has both a unit risk estimate and a reference concentration and appears in both modeled and measured concentration estimates.

Using the Cal/EPA unit risk estimate for benzene, 2.9×10^{-5} per ug/m3, we calculate the threshold risk concentrations as follows. Take a particular, lifetime, cancer risk level, say, 1 in 10,000 expressed as 1/10,000, and then divide it by the unit risk estimate. This means that the air concentration at each of these threshold levels changes by a factor of 10, the same as the change in the risk levels (1/10,000: 3.4 ug/m3; 1/100,000: 3.4×10^{-1} ug/m3; 1/1,000,000: 3.4×10^{-2} ug/m3). The highest modeled concentration for benzene from NATA is 9.04; the concentrations for 66 census tracts exceed 3.4, our threshold for the "Between 1/1,000 and 1/10,000" cancer risk grouping. The highest measured concentration is 5.51. Two monitors show annual averages exceeding 3.4 ug/m3. This places benzene in the same grouping for both measured and modeled concentrations. The reference value thresholds are based on percentages of the reference concentration, ranging from 150% to below 50%. For benzene, the reference value is 30 ug/m3. Again, measured and modeled concentrations lead to the same grouping, "Below 50% RfC".

In the second round, benzene is assigned to the Definite Risk category, since the top two risk groupings have been combined. Its lowest grouping on the reference concentration does not affect this assignment. Finally, in the third round, benzene's emissions mass above the 90th percentile and its appearance above threshold levels in 66 census tracts and at two monitors reinforce the default assignment.

Appendix 2

Data Source Descriptions: NATA and AQS

A national-scale assessment of air toxics for 1999 was prepared by EPA and made public at the beginning of 2006. (U.S. EPA, 2006b) It included 176 HAPs drawn from the 188 named in Section 3 of the Clean Air Amendments of 1990, and added diesel particulate matter. The assessment included a national inventory of air toxics emissions from outdoor sources, and estimates of ambient concentrations and population exposure.

Two databases were used. First, the National Emission Inventory 1999 reported information on each pollutant in tons released per year, categorized their emission source, and reported by county (U.S. EPA, 2006c). Second, the Assessment System for Population Exposure Nationwide (ASPEN) (U.S. EPA, 2006d) provided annual pollutant concentrations modeled by means of a computer simulation.

ASPEN used estimates of air toxics emissions and meteorological data from National Weather Service Stations, as well as took into consideration other determinants such as rate, location, and height from which they are released, reactive decay, deposition and secondary formation. The smallest geographic unit, for which the concentrations were modeled, was a census tract, typically containing 4000 inhabitants. The ten county area of interest comprised a total of 895 census tracts. East Houston analysis included an area of 28 census tracts.

Concentrations of each pollutant were expressed in ug/m3 and were presented by source of emission, such as point, area, mobile (on road and non-road), and for some pollutants as background. Background concentrations are based on monitored data, estimates from the technical literature and reported emissions during 1999. They are attributable to unidentified and natural sources, previous emissions persisting in the environment, and to long-range transport. Additional information on background concentrations is available at:
http://www.epa.gov/ttn/atw/nata1999/background.html.

Extensive information on ASPEN model can be found at:
http://www.epa.gov/ttn/atw/nata1999/aspen99.html.

The Air Quality System (AQS) database, as compiled by the U.S. Environmental Protection Agency (EPA), provides air monitoring data -- ambient concentrations of criteria and hazardous air pollutants -- from monitoring sites throughout the United States. These concentrations are reported to the EPA by various agencies throughout the United States (e.g. the Texas Commission on Environmental Quality). The data can be obtained directly from the EPA in ASCII format, or can be accessed online. The data that we obtained were in three forms labeled as: DM_300 (exceedance data for ozone, PM 2.5 and PM 10); DM_350 (raw data for HAPs, VOCs, speciated carbon, ozone, PM 2.5 and PM 10); and DM_AMP450 (annual means for HAPs, VOCs, speciated carbon, ozone, PM 2.5 and PM 10). Datasets were received for years 1970 through 2005. We selected 2004 as the year of analysis, since it had the most complete recent data. The ten county area was used as a filter for the data request since “COUNTY_NAME” is a variable in the AQS dataset.

There were a total of 49 HAPs that were monitored and included in our analysis. Of those 49, 25 had cancer risk values and 41 had non-cancer reference values. Several VOCs that are also defined as HAPs were duplicated in both datasets. This was confirmed by analysis of both the raw and summary data.

For 2004, there were 19 “species” of carbon monitored and included in the AQS dataset for the 10 county area. Only two of these were included in the final analysis as surrogates for diesel particulate matter, based on the recommendation of EPA officials. Our final analysis of raw and summary data for HAPs and speciated carbons included 51 pollutants. Ozone, PM 2.5 and PM 10 were reviewed for daily exceedances for 2000-2005.

Pollutants are represented as “parameters” in the AQS dataset and are assigned a five digit code. Site IDs in the database represent geographic locations of monitoring sites. A parameter occurrence code (POC) is used to distinguish between multiple monitors at the same site that are measuring the same parameter. So, in effect, a POC represents a monitor. Monitoring duration is also included in the data set per POC at individual site IDs. For example, a duration code of 7 represents 24 hours, whereas a duration code of 1 represents 1 hour. The particular method for collecting and analyzing samples for

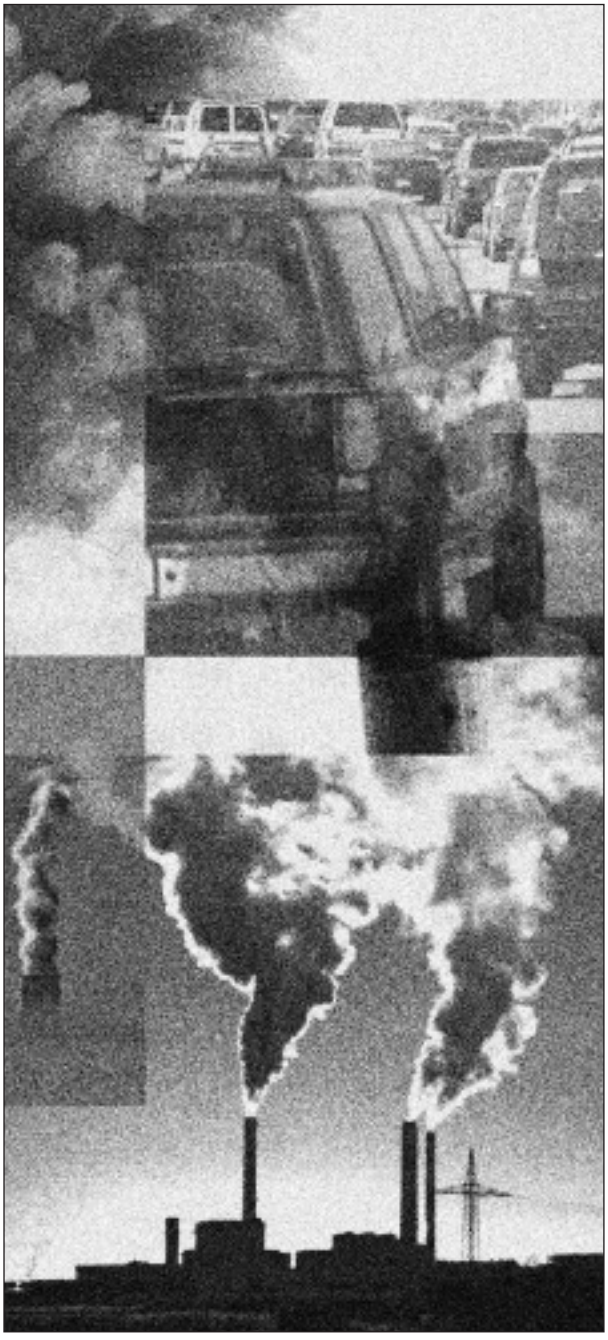
Appendix 2

the time period is represented by a three digit "method" code. For example, a method code of 175 represents a passivated canister. Collection frequency is also included and was used to calculate the percentage of scheduled readings that were actually recorded. "Unit_Code" represents the unit of measurement. Although unit risk estimates and reference concentrations are reported in µg per m^3; most HAPs are measured in parts-per-billion carbon (ppbC). In order to convert these values to µg per m^3, the molecular weight and number of carbons of these pollutants were used in the following equation:

$$\frac{ug}{m^3} = \frac{(ppbC \times MW \times 273K)}{(22.4 \times 298K \times \#C)}$$

This formula was obtained from EPA (http://www.epa.gov/oar/oaqps/pams/analysis/receptor/rectxtsac.html) and confirmed through our calculations. Finally, in the raw dataset, sample dates and recorded values for those dates (depending on the collection frequency) are included.

The dataset, entitled DM_AMP450, contains all of the variables mentioned above; however, instead of multiple readings per day for all days of collection, an arithmetic mean is reported. In order to obtain this mean, the EPA simply added all monitored values and divided by the total number present. No readings are excluded. For verification, arithmetic means were calculated using the "raw" dataset and confirmed against the "summary" dataset.



Appendix 3

Inventory of Health Values and Reference Concentrations

Chronic inhalation dose-response information for this for this assessment was obtained from various sources. It was a Task Force decision to give first priority to the most protective of the final, peer-reviewed health values for cancer and non-cancer endpoints between those values reported by the US EPA's Integrated Risk Information System (IRIS) (U.S. EPA, 2006i) and California EPA's Office of Environmental Health Hazard Assessment (OEHHA) (California OEHHA, 2006). California OEHHA values were found in two tables: a table entitled *All Chronic Reference Exposure Levels (RELs) adopted by OEHHA* as of February 2005 (for health risks other than cancer) (California OEHHA, 2005); and a table entitled *Hot Spots Unit Risk and Cancer Potency Values* found in *Appendix A* of the *Air Toxics Hot Spots Program Risk Assessment Guidelines (Part II) Technical Support Document for Describing Available Cancer Potency Factors* (California EPA & OEHHA, 2002). Where values from IRIS or OEHHA do not exist, a consolidated list of health risk values from EPA's Office of Air Quality Planning and Standards (OAQPS) entitled *Prioritized Chronic Dose Response Values for Screening Risk Assessments* (U.S. EPA, 2005) which was updated in February 2005 was used. This table consolidated health risk values from externally peer reviewed EPA IRIS draft assessments, final peer-reviewed EPA IRIS assessments, the Agency for Toxic Substances and Disease Registry (ATSDR), California OEHHA and US EPA's Health Effects Assessment Tables (HEAST) values, prioritized in that order. The Task Force's prioritization of health values followed that of the OAQPS table with the exception that first priority was given to the most protective of the final, peer-reviewed health values for cancer and non-cancer endpoints between values reported by IRIS and California OEHHA. Where values do not exist from any of the above sources, we consulted and crosschecked a table entitled *Health Effects Information Used in Cancer and Noncancer Risk Characterization for the 1999 National-Scale Assessment* (U.S. EPA, 2006h). This table was used in EPA's 1999 National-Scale Air Toxics Assessment (NATA) (U.S. EPA, 2006b) and was updated on November 7, 2005.

US Environmental Protection Agency (EPA)

The US EPA has developed dose-response assessments for chronic exposure to many of the pollutants in this report. EPA reports reference concentrations, or RfCs (to protect against effects other than cancer), and/or a unit risk estimate, or URE (to estimate the probability of contracting cancer). Reference concentrations are estimates of an inhalation exposure to the human population (including sensitive subgroups) that are likely to be without appreciable risks of deleterious effects during a lifetime. The URE is the upper-bound excess cancer risk estimated to result from a lifetime of continuous exposure to an agent at a concentration of 1 µg/m3 in air.

California Office of Environmental Health Hazard Assessment (OEHHA)

Dose-response assessments have been developed for many substances for both cancer and non-cancer endpoints by the California OEHHA. The process for developing these values is similar to that use by the EPA to develop IRIS values. Non-cancer inhalation health risk values are expressed as chronic inhalation reference exposure levels (RELs). OEHHA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the URE, defined similarly to EPA's URE.

Agency for Toxic Substances and Disease Registry (ATSDR)

Minimum Risk Levels (MRLs) are developed and published by ATSDR, which is part of the US Department of Health and Human Services. The MRL is defined as an estimate of daily human exposure to a substance that is likely to be without

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an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure. MRLs are considered to be concentrations below which contaminants are unlikely to pose a health threat. Concentrations above an MRL do not necessarily represent a threat. The concept, definition and derivation of inhalation MRLs is philosophically consistent (though not identical) with the basis for EPA's RfCs. MRLs are published and updated on the world wide web (ATSDR).

US EPA Health Effects Assessment Tables (HEAST)

HEAST is a listing of provisional UREs and RfCs developed by EPA. HEAST assessments have undergone review by individual EPA program offices and are supported by agency references, but are not considered to be high-quality, EPA-wide consensus information. HEAST was last updated in 1997 and exists only in hard copy (PB97-921199). HEAST values are only used when no values from other sources exist.

Conversions of Oral Unit Risk Estimates into Inhalation Estimates

For eleven carcinogenic substances (benzotrichloride, captan, dichlorvos, 3,3'-dimethoxybenzidine, 3,3'-dimethyl benzidine, ethyl acrylate, isophorone, pentachloronitrobenzene, propylene dichloride, quinoline, trifluralin) that lack inhalation assessments from these sources, inhalation UREs are derived from oral carcinogenic potency estimates and are reported in the OAQPS or NATA tables.

Photo by Heidi Bethel

Table A3.1. Health Values and Reference Concentrations

Prioritized Chronic Inhalation Dose-Response Values for Task Force Risk Assessment				
		Non-Cancer		Cancer
Hazardous Air Pollutant (HAP) Name	CAS No.	Chronic RfC, REL or MRL in (µg/m3)	Source	URE (Unit Risk Estimate) in 1/(ug/m^3)	Source
1,1,2,2-Tetrachloroethane	79-34-5			5.80E-05	IRIS & CAL
1,1,2-Trichloroethane	79-00-5	400.00	P-CAL (EPA OAQPS & NATA)	1.60E-05	IRIS & CAL
1,2,4-Trichlorobenzene	120-82-1	200.00	HEAST (NATA)		
1,2-Dibromo-3-chloropropane	96-12-8	0.20	IRIS	2.00E-03	CAL
1,2-Diphenylhydrazine	122-66-7			2.20E-04	IRIS
1,2-Epoxybutane	106-88-7	20.00	IRIS & CAL		
1,3-Butadiene	106-99-0	2.00	IRIS	1.70E-04	CAL
1,3-Dichloropropene	542-75-6 1120-71-	20.00	IRIS	4.00E-06	IRIS
1,3-Propane sultone	4			6.90E-04	CAL
1,4-Dioxane	123-91-1	3000.00	CAL	7.70E-06	CAL
2,4,6-Trichlorophenol	88-06-2			2.00E-05	CAL
2,4-Dinitrotoluene	121-14-2	7.00	P-CAL(OAQPS & NATA)	8.90E-05	CAL
2,4-Toluene diamine	95-80-7 26471-			1.10E-03	CAL
2,4-Toluene diisocyanate	62-5	0.07	IRIS & CAL	1.10E-05	CAL
2-Chloroacetophenone	532-27-4	0.03	IRIS		
2-Nitropropane	79-46-9	20.00	IRIS	5.60E-06	OAQPS & NATA
3,3-Dichlorobenzidene	91-94-1			3.40E-04	CAL
3,3'-Dimethoxybenzidine	119-90-4			4.00E-06	Conv.Oral^{AD} (OAQPS & NATA)
3,3'-Dimethyl benzidine	119-93-7			2.60E-03	Conv.Oral^{AD} (OAQPS & NATA)
4,4'-Methylene bis(2-chloroaniline)	101-14-4			4.30E-04	CAL
4,4'-Methylenedianiline	101-77-9	20.00	CAL	4.60E-04	CAL
Acetaldehyde	75-07-0	9.00	IRIS & CAL	2.70E-06	CAL
Acetamide	60-35-5			2.00E-05	CAL
Acetonitrile	75-05-8	60.00	IRIS		
Acrolein	107-02-8	0.02	IRIS		
Acrylamide	79-06-1	0.70	P-CAL(OAQPS & NATA)	1.30E-03	IRIS & CAL
Acrylic acid	79-10-7	1.00	IRIS		
Acrylonitrile	107-13-1	2.00	IRIS	2.90E-04	CAL
Allyl chloride	107-05-1	1.00	IRIS	6.00E-06	CAL
Aniline	62-53-3	1.00	IRIS	1.60E-06	CAL
Antimony compounds	7440-36-0	0.20	IRIS(NATA)		

Table A3.1. Health Values and Reference Concentrations

Prioritized Chronic Inhalation Dose-Response Values for Task Force Risk Assessment				
	Non-Cancer		Cancer	
Hazardous Air Pollutant (HAP) Name	CAS No.	Chronic RfC, REL or MRL in (µg/m3)	Source	URE (Unit Risk Estimate) in 1/(ug/m3)	Source
Arsenic compounds (inorganic, may include arsine)	7440-38-2	0.03	CAL	4.30E-03	IRIS
Arsine	7784-42-1	0.05	IRIS		
Asbestos	1332-21-4			6.30E-02	CAL
Benzene	71-43-2	30.00	IRIS	2.90E-05	CAL
Benzidine	92-87-5	10.00	P-CAL(OAQPS & NATA)	1.40E-01	CAL
Benzotrichloride	98-07-7			3.70E-03	Conv.Oral[#3] (OAQPS & NATA)
Benzyl chloride	100-44-7			4.90E-05	IRIS & CAL
Beryllium compounds	7440-41-7	0.01	CAL	2.40E-03	IRIS & CAL
Bis(2-ethylhexyl)phthalate	117-81-7	10.00	P-CAL(OAQPS & NATA)	2.40E-06	CAL
Bis(chloromethyl)ether	542-88-1			6.20E-02	IRIS
Bromoform	75-25-2			1.10E-06	IRIS
Cadmium compounds	7440-43-9	0.02	CAL	4.20E-03	CAL
Captan	133-06-2			1.00E-06	Conv.Oral[#3] (OAQPS & NATA)
Carbon disulfide	75-15-0	700.00	IRIS		
Carbon tetrachloride	56-23-5	40.00	CAL	4.20E-05	CAL
Chlordane	57-74-9	0.70	IRIS	1.00E-04	IRIS
Chlorine	7782-50-5	0.20	CAL		
Chlorobenzene	108-90-7	1000.00	CAL		
Chlorobenzilate	510-15-6			7.80E-05	HEAST (OAQPS & NATA)
Chloroform	67-66-3	98.00	ATSDR (OAQPS & NATA)	5.30E-06	CAL
Chloroprene	126-99-8	7.00	HEAST (NATA)		
Chromium VI[c]	18540-29-9	0.10	IRIS	1.50E-01	CAL
Cobalt compounds	7440-48-4	0.10	ATSDR (NATA)		
Coke Oven Emissions	8007-45-2			6.20E-04	IRIS
Cresols_Cresylic acid (isomers and mixture)	1319-77-3	600.00	CAL		
Cumene (Isopropyl benzene)	98-82-8	400.00	IRIS		
Cyanide compounds	57-12-5	3.00	IRIS(NATA)		
Dichloroethyl ether	111-44-4			7.10E-04	CAL
Dichlorvos	62-73-7	0.50	IRIS	8.30E-05	Conv.Oral[#3] (OAQPS & NATA)

Table A3.1. Health Values and Reference Concentrations

Prioritized Chronic Inhalation Dose-Response Values for Task Force Risk Assessment				
	Non-Cancer		Cancer	
Hazardous Air Pollutant (HAP) Name	CAS No.	Chronic RfC, REL or MRL in (µg/m3)	Source	URE (Unit Risk Estimate) in 1/(ug/m3)	Source
Diesel exhaust	Diesel Emis	5.00	IRIS &CAL	3.00E-04	CAL
Diethanolamine	111-42-2	3.00	CAL		
Dimethyl formamide	68-12-2	30.00	IRIS		
Epichlorohydrin	106-89-8	1.00	IRIS	2.30E-05	CAL
Ethyl acrylate	140-88-5			1.40E-05	Conv.Oral[A,E] (NATA)
Ethyl benzene	100-41-4	1000.00	IRIS		
Ethyl carbamate	51-79-6			2.90E-04	CAL
Ethyl chloride	75-00-3	10000.00	IRIS		
Ethylene dibromide	106-93-4	0.80	CAL	6.00E-04	IRIS
Ethylene dichloride	107-06-2	400.00	CAL	2.60E-05	IRIS
Ethylene glycol	107-21-1	400.00	CAL		
Ethylene oxide	75-21-8	30.00	CAL	8.80E-05	CAL
Ethylene thiourea	96-45-7	3.00	P-CAL(OAQPS & NATA)	1.30E-05	CAL
Ethylidene dichloride	75-34-3	500.00	HEAST (OAQPS & NATA)	1.60E-06	CAL
Formaldehyde[G]	50-00-0	3.00	CAL	6.00E-06	CAL
Glycol ethers[H]	7529-27-3	20.00	OAQPS(NATA)		
Heptachlor	76-44-8			1.30E-03	IRIS
Hexachlorobenzene	118-74-1	3.00	P-CAL(OAQPS & NATA)	5.10E-04	CAL
Hexachlorobutadiene	87-68-3	90.00	P-CAL(OAQPS & NATA)	2.20E-05	IRIS
Hexachlorocyclopentadiene	77-47-4	0.20	IRIS		
Hexachloroethane	67-72-1	80.00	P-CAL(OAQPS & NATA)	4.00E-06	IRIS
Hexamethylene-1,6-diisocyanate	822-06-0	0.01	IRIS		
Hexane	110-54-3	700.00	IRIS		
Hydrazine	302-01-2	0.20	CAL	4.90E-03	IRIS & CAL
Hydrochloric acid	7647-01-0	8.00	CAL		
Hydrofluoric acid	7664-39-3	14.00	CAL		
Isophorone	78-59-1	2000.00	CAL	2.70E-07	Conv.Oral[A,E] (OAQPS & NATA)
Lead compounds	7439-92-1	1.50	OAQPS & NATA	1.20E-05	CAL
Lindane (all isomers)	58-89-9	0.30	PCAL (EPA OAQPS & NATA)	3.10E-04	CAL
Maleic anhydride	108-31-6	0.70	CAL		
Manganese compounds	7439-96-5	0.05	IRIS		

Table A3.1. Health Values and Reference Concentrations

Prioritized Chronic Inhalation Dose-Response Values for Task Force Risk Assessment

Hazardous Air Pollutant (HAP) Name	CAS No.	Non-Cancer		Cancer	
		Chronic RfC, REL or MRL in (µg/m3)	Source	URE (Unit Risk Estimate) in 1/(ug/m3)	Source
Mercury compounds	7439-97-6	0.09	CAL		
Methanol	67-56-1	4000.00	CAL		
Methyl bromide	74-83-9	5.00	IRIS & CAL		
Methyl chloride	74-87-3	90.00	IRIS		
Methyl chloroform	71-55-6	1000.00	CAL		
Methyl ethyl ketone	78-93-3	5000.00	IRIS		
Methyl isobutyl ketone	108-10-1	3000.00	IRIS		
Methyl isocyanate	624-83-9	1.00	CAL		
Methyl methacrylate	80-62-6	700.00	IRIS		
Methyl tert butyl ether	1634-04-4	3000.00	IRIS	2.60E-07	CAL
Methylene chloride	75-09-2	400.00	CAL	1.00E-06	CAL
Methylene diphenyl diisocyanate	101-68-8	0.60	IRIS		
Naphthalene	91-20-3	3.00	IRIS	3.40E-05	CAL
Nickel compounds	7440-02-0	0.05	CAL	2.60E-04	CAL
Nitrobenzene	98-95-3	30.00	PCAL (EPA OAQPS & NATA)		
Nitrosodimethylamine	62-75-9			1.40E-02	IRIS
N-Nitrosomorpholine	59-89-2			1.90E-03	CAL
o-Toluidine	95-53-4			5.10E-05	CAL[C] (EPA OAQPS & NATA)
p-Dichlorobenzene	106-46-7	800.00	IRIS & CAL	1.10E-05	CAL
p-Dimethylaminoazobenzene	60-11-7			1.30E-03	CAL
Pentachloronitrobenzene	82-68-8			7.40E-05	Conv.Oral[AB] (OAQPS & NATA)
Pentachlorophenol	87-86-5	100.00	PCAL (EPA OAQPS & NATA)	5.10E-06	CAL
Perchloroethylene	127-18-4	35.00	CAL	5.90E-06	CAL
Phenol	108-95-2	200.00	CAL		
Phosgene	75-44-5	0.30	IRIS		
Phosphine	7803-51-2	0.30	IRIS		
Phthalic anhydride	85-44-9	20.00	CAL		
Polychlorinated biphenyls (PCBs)[E]	1336-36-3			1.00E-04	IRIS & CAL
POM[F]					
POM Group 1: Unspeciated				5.50E-05	OAQPS (NATA)[D]
POM Group 2: no URE data				5.50E-05	OAQPS (NATA)[D]
POM Group 3: 5.0E-2<URE<=5.0E-1				1.00E-01	OAQPS (NATA)[D]

Table A3.1. Health Values and Reference Concentrations

Prioritized Chronic Inhalation Dose-Response Values for Task Force Risk Assessment				
	Non-Cancer		Cancer	
Hazardous Air Pollutant (HAP) Name	CAS No.	Chronic RfC, REL or MRL in (µg/m3)	Source	URE (Unit Risk Estimate) in 1/(ug/m3)	Source
POM Group 4: 5.0E-3<URE<=5.0E-2				1.00E-02	OAQPS (NATA)[D]
POM Group 5: 5.0E-4<URE<=5.0E-3				1.00E-03	OAQPS (NATA)[D]
POM Group 6: 5.0E-5<URE<=5.0E-4				1.00E-04	OAQPS (NATA)[D]
POM Group 7: 5.0E-6<URE<=5.0E-5				1.00E-05	OAQPS (NATA)[D]
POM Group 8: Unspeciated 7-PAH				2.00E-04	OAQPS (NATA)[D]
Propylene dichloride	78-87-5	4.00	IRIS	1.90E-05	Conv.Oral[A,B] (OAQPS & NATA)
Propylene oxide	75-56-9	30.00	IRIS & CAL	3.70E-06	IRIS & CAL
Quinoline	91-22-5			3.40E-03	Conv. Oral[A,B] (NATA)
Selenium Compounds	7782-49-2	20.00	CAL		
Styrene	100-42-5	900.00	CAL		
Styrene oxide	96-09-3	6.00	PCAL (EPA OAQPS & NATA)		
Titanium tetrachloride	7550-45-0	0.10	ATSDR (NATA)		
Toluene	108-88-3	300.00	CAL		
Toxaphene	8001-35-2			3.20E-04	IRIS
Trichloroethylene	79-01-6	600.00	CAL	2.00E-06	CAL
Triethylamine	121-44-8	7.00	IRIS		
Trifluralin	1582-09-8			2.20E-06	Conv.Oral[A,B] (OAQPS & NATA)
Vinyl acetate	108-05-4	200.00	IRIS & CAL		
Vinyl bromide	593-60-2	3.00	IRIS	3.20E-05	HEAST (OAQPS & NATA)
Vinyl chloride	75-01-4	100.00	IRIS	7.80E-05	CAL
Vinylidene chloride	75-35-4	70.00	CAL		
Xylenes (isomers and mixture)	1330-20-7	100.00	IRIS		

Source Key:

CAL, California Office of Environmental Health Hazard Assessment (OEHHA); P-CAL, Pre-CAL, under consideration, but not yet adopted by California; IRIS, US EPA Integrated Risk Information System; IRIS & CAL, IRIS values which have been adopted by California OEHHA; OAQPS, Consolidated Health Risk Values Table from US EPA's Office of Air Quality Planning and Standards (OAQPS); NATA, Consolidated Health Risk Values Table from the US EPA's National Air Toxics Assessment; HEAST, US EPA Health Effects Assessment Tables (EPA PB97-921199); ATSDR, The Agency for Toxic Substances and Disease Registry.

Table A3.1. Health Values and Reference Concentrations

Footnotes:

The URE is the upper-bound excess cancer risk estimated to result from a lifetime of continuous exposure to an agent, at a concentration of 1 µg/m3

CAS No. - chemical abstracts services number for compound; RfC - reference concentration; REL - reference exposure level; MRL - minimal risk level; URE - unit risk estimate (the URE is the upper-bound excess cancer risk estimated to result from a lifetime of continuous exposure to an agent, at a concentration of 1 µg/m3).

A, Conversion of carcinogenic oral potency (CPS) slope to inhalation unit risk estimate was based on the following assumptions: (1) whole-life, continuous exposure, (2) inhalation rate of 20 cubic meters of air per day, and (3) body mass of 70 kg. Further details are provided in the text, above.

B, Oral carcinogenic potency slope (CPS) factors and their respective source agencies used for conversions to an inhalation Unit Risk Estimates (UREs).

Compound	*Oral Carcinogenic Potency Slope Factor (CPS) (mg/kg-day)[1] (Source Agency)*
Benzotrichloride	1.30 E+1 (IRIS)
Captan	No source found
Dichlorvos	2.90 E-1 (IRIS)
3,3-Demethoxybenzidine	No source found
3,3-Dimethyl benzidine	No source found
Ethyl acrylate	No source found
Isophorone	9.50 E-4 (IRIS)
Pentachloronitrobenzene	No source found
Propylene dichloride	No source found
Quinoline	3.00 E+1 (IRIS)
Trifluralin	7.70 E-3 (IRIS)

C, Reported as a California OEHHA value on OAQPS and NATA consolidated tables, but not found on California OEHHA tables, possibly an old value.

D, Value derived by OAQPS for use in NATA evaluation of Polycyclic Organic Matter (POM).

E, Low risk for use with unspeciated PCB mixtures.

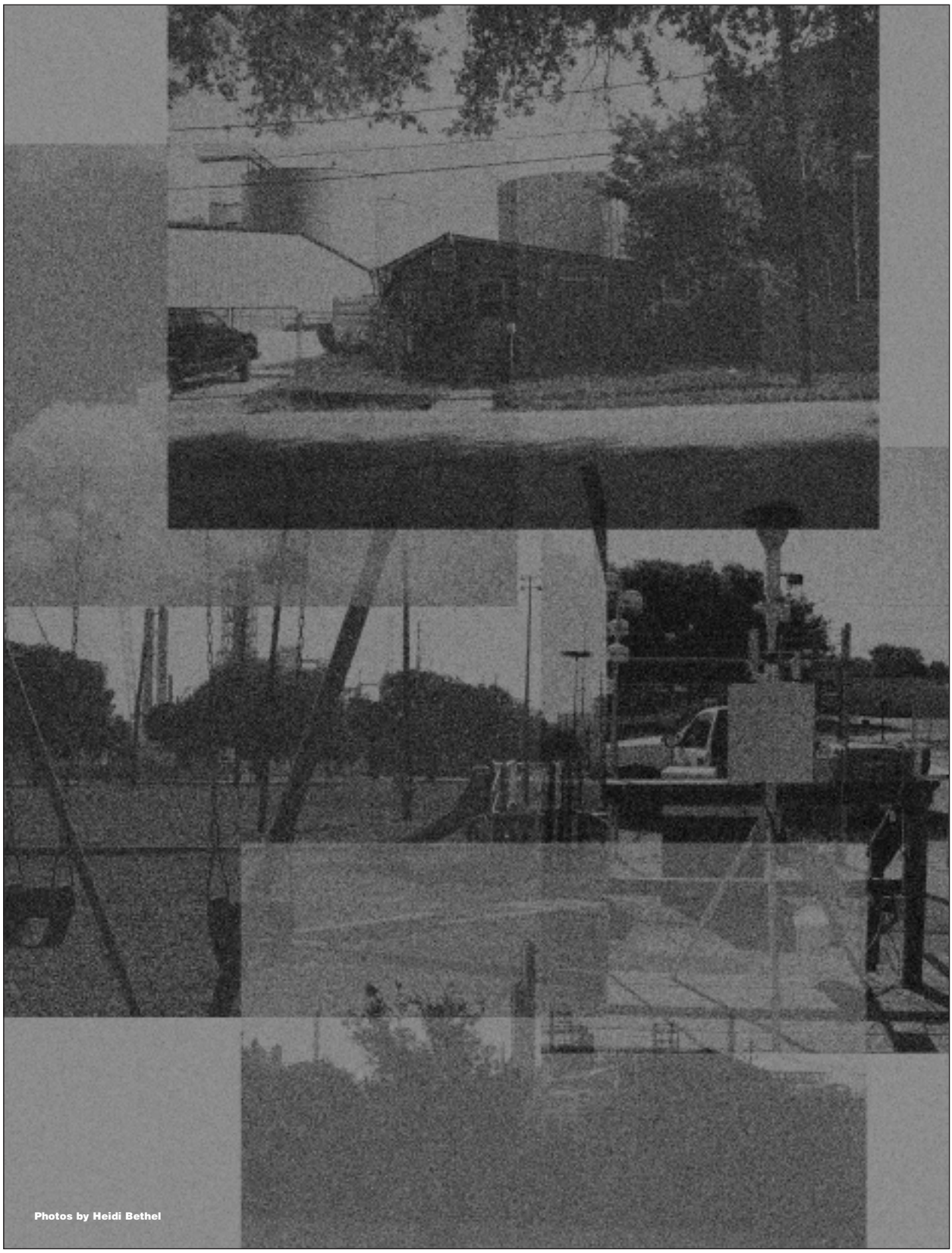
F, The IRIS RfC for particulate hexavalent chromium was used in preference to the RfC for chromic acid mists and dissolved aerosols.

G, The EPA no longer considers the formaldehyde URE reported in IRIS, which is based on a 1987 study, to represent the best available science in the peer-reviewed literature. A new URE has been developed for formaldehyde which is based on a dose-response value developed by the CIIT Centers for Health Research (formerly the Chemical Industry Institute of Toxicology). This assessment developed a URE using approaches that are consistent with EPA's guidelines for carcinogenic risk assessment. EPA is currently reviewing the CIIT analysis and other recent information, including recently published epidemiological studies, in their reassessment of the formaldehyde unit risk estimate. At the time of the Task Force evaluation, this URE had not been fully peer reviewed and it was a Task Force decision not to use this value. The new value was being used by EPA in their National Air Toxics assessment. EPA's new URE for formaldehyde predicts permissible air concentrations for cancer risks which are much higher than previously thought, therefore predicting that formaldehyde represents a much lower cancer threat. The unit risk estimate used in the Task Force evaluation was from California OEHHA and is in between the current IRIS peer reviewed value and the new value currently being evaluated by EPA. Therefore, this case is an exception to the rule that the Task Force established to use the most protective of the health risk values between California OEHHA and IRIS. The IRIS, peer reviewed value, is the most protective value for cancer endpoints of the peer reviewed values that currently exist.

H, Most of the reporting information for glycol ether compounds reports only the total mass for the entire group and does not distinguish between individual glycol ether compounds. The RfC for ethylene glycol methyl ether (the most toxic for which an assessment exists) was applied in this assessment in order to avoid underestimating the health hazard associated with glycol ethers.

I, The assessment used a URE of 5.6E-6 (ug/m3)-1. This value was derived in 1999 by the Health Council of the Netherlands (available at http://www.gr.nl/pdf.php?ID=423&p=1) and is consistent with weight-of-evidence determinations by the U.S. National Toxicology Program ("reasonably anticipated to be a human carcinogen") and IARC ("possibly carcinogenic to humans").

J, The EPA's 1999 National Air Toxics Assessment divided POM emissions into eight categories. Each of the eight has a URE. The first two categories were assigned a URE equal to 5%of that for pure benzo[a]pyrene. Categories 3-7 were composed of emissions that were reported as individual compounds. These compounds were placed in the category with an appropriate URE. Category 8, composed of unspeciated carcinogenic polynuclear aromatic hydrocarbons (a subset of POM called 7-PAH), was assigned a URE equal to 18% of that for pure benzo[a]pyrene. Details on the development of the 5% and 18% URE estimates are available at: http://www.epa.gov/ttn/atw/sab/appendix-h.pdf. For purposes of the Clean Air Act these POMs are treated as a single group among the 188. Our tallies in the main text reflect this practice.



Photos by Heidi Bethel

Appendix 4

Metals, Diesel PM Conversions and Polycyclic Organic Matter

All Metals Analyses

The U.S. EPA's 1999 National Air Toxics Assessment (NATA) (U.S. EPA, 2006b) modeling and analyses used fine and course PM data in modeling metal concentrations. The Mayor's Task Force analyses used only fine PM metal concentrations from PM 2.5 speciated metals data files from the U.S. EPA's Air Quality System database (U.S. EPA, 2006e) to compare to reference concentrations (RfCs) and unit risk estimates (UREs). The choice to eliminate the PM 10 speciated metals data was based on the assumption that PM 2.5 particles would penetrate further into the lungs and therefore represent greater health consequences than PM 10 speciated metals. After completing the analyses, it was found that metal concentrations from ambient PM 2.5 speciated metals files for 2004 were consistently lower than the modeled NATA concentrations from 1999. The inconsistency between the modeled and ambient data analyses is further complicated by the fact that the time frame for these analyses is separated by five years. The NATA Model-to-Monitor Comparison (U.S. EPA, 2006g) reported that measured ambient metal concentrations were typically higher than modeled concentrations for chromium, lead, manganese and nickel. Recalculating our ambient metal concentrations to include both fine and course speciated metals may lead to higher risk categories reported for metals from ambient datasets.

Chromium Compounds

Chromium compounds, which are a group of pollutants, are listed in the Clean Air Act (U.S. EPA, 1990) as one of the 188 Hazardous Air Pollutants (HAPs). Chromium sources of emissions include the combustion of coal and oil, electroplating, vehicles, iron and steel plants, and metal smelters. Chromium occurs in the environment primarily in two valence states, trivalent chromium (Cr III), which occurs naturally and is an essential nutrient, and hexavalent chromium (Cr VI), which along with the less common metallic chromium (Cr 0), is most commonly produced by industrial processes. Air emissions of chromium are predominantly of trivalent chromium, and in the form of small particles or aerosols. Chromium forms a large number of compounds, in both the chromium (III) and chromium (VI) forms (ATSDR, 1998). The Mayor's Task Force assessed chromium (VI) compounds as a group of pollutants using modeled concentrations from the U.S. Environmental Protection Agency's 1999 National Air Toxics Assessment (U.S. EPA, 2006d). Modeled chromium VI compound concentrations were assessed for carcinogenic and non-carcinogenic endpoints. Currently, no UREs or RfCs exist from the sources consulted for chromium III compounds, and chromium III is much less toxic than chromium VI.

The emissions of chromium compounds reflected in the 1999 NATA assessment are based on state and local agency reporting of chromium as "chromium and compounds," individual chromium compounds and chromium ions. In the EPA's 1996 National Air Toxics Assessment (U.S. EPA, 1999), because of the inconsistent reporting, all of the chromium was lumped together for dispersion modeling as "Chromium VI." EPA then based quantitative risk estimates on an assumption that 34 percent of the chromium is hexavalent chromium based on information from past inventorying efforts. For 1999, EPA used a more refined approach to estimate emissions of hexavalent chromium. Individual compounds of chromium reported in the inventory were identified as either hexavalent or trivalent based upon their chemical formulae. Any compounds reported as either "chromium" or "chromium and compounds" were then speciated using source category specific speciation data (U.S. EPA, 2004). For source categories where speciation data were not available, EPA assumed that 34 percent of the chromium is hexavalent.

Elemental Carbon to Diesel PM Conversion Factor

In order to assess diesel particulate matter concentrations the Task Force used ambient elemental carbon (EC) concentrations for 2004 (U.S. EPA, 2006f) as a surrogate for diesel particulate matter (PM) concentrations. This technique used the relative contribution of diesel combustion to all sources of ambient EC to determine a scaling factor for diesel PM concentrations. Sources of EC in the Houston area include gas and diesel vehicles, road dust, vegetative detritus, wood combustion, meat cooking and fuel oil combustion. A study by Fraser,

et al. (Fraser et al., 2003) used organic molecular markers specific to the above sources to apportion fine particulate matter at four sites in Houston. The samples used in this study were collected between March 1997 and February 1998. Two sites from this study, Clinton (adjacent to the Houston Ship Channel in the vicinity of a high concentration of industrial emission sources) and Bingle (located in a suburban neighborhood in north-west Houston) were used to develop an appropriate scaling factor to relate ambient EC levels to diesel PM concentrations. Raw data from this study were obtained from Fraser, and the ratio of elemental carbon attributed to diesel exhaust to total apportioned elemental carbon at each site was determined to be 0.775 at Clinton and 0.887 at Bingle.

In a second study by Fraser, et al. (Fraser *et al.*, 2002) samples of fine particle emissions from four heavy-duty diesel vehicles were analyzed for chemical and molecular composition. Particle emissions were sampled for vehicles under load and idling. Ratios of elemental carbon to total carbon (EC/TC) for diesel emissions from two tractor-trailer trucks from the fleet of the HEB Grocery Company of San Antonio, Texas running at an Heavy-Duty Chassis Cycle (HDCC), designed to simulate urban and highway operation, were measured in a range of 0.66 - 0.72. The mean and median of this range is 0.69.

In order to calculate conversion factors for ambient measured EC to diesel (PM) concentrations, the data mentioned from the two studies by Fraser et al. were used. Factors for conversion were calculated for both the Clinton and Bingle sites by dividing the ratio of fine elemental carbon mass attributed to diesel PM in the Houston atmosphere at each site (0.775 at Clinton and 0.887 at Bingle) by the EC/TC ratio of 0.69 for diesel engine emissions from the two representative tractor-trailer trucks. This calculation assumes that these two diesel trucks provide an accurate representation of the diesel vehicle fleet as a whole in Houston. This assumption was required because source apportionment of elemental carbon to diesel sources other than diesel truck engines at our monitoring sites in the Houston area was not directly available.

Our conversion factors were calculated as follows:

Clinton:	*Bingle:*
(0.775)/(0.69) = 1.12	(0.887)/(0.69) = 1.29

Estimates of ambient diesel PM concentrations can then be made by multiplying the elemental carbon concentrations measured at a local air quality monitoring location by one of the conversion factors above. An explanation of which of these factors was used in the Mayor's Task Force evaluation can be found below.

Several other conversion factors used by the California Air Resources Board (ARB) (California ARB, 1998; California ARB & OEHHA, 1998) in their identification of particulate emissions from diesel fueled engines as a Toxic Air Contaminant and by the U.S. EPA (U.S. EPA, 2002) were also found. The ARB used a study by Gray (Gray, 1986) which showed that the ratio of fine elemental carbon mass attributed to diesel engine emissions to total elemental carbon in the Los Angeles atmosphere was approximately 0.67. The EC/TC ratio for all diesel exhaust particles emitted was 0.64. Therefore, diesel particulate concentrations are estimated by multiplying the elemental carbon concentrations by 1.04 (0.67/0.64 = 1.04).

The U.S. EPA also calculated elemental carbon to diesel PM conversion factors for various areas in the United States using seven different studies (U.S. EPA, 2002), as well as raw data obtained from various researchers involved in the studies. For the Western United States, which encompasses the state of Texas, an average EC to diesel PM conversion factor of 1.6 was calculated for elemental carbon measurements using the thermal optical transmittance (TOT) method and an average value of 0.8 was calculated for elemental carbon measurements using the thermal optical reflectance (TOR) measurement method during winter months in the Eastern and Western United States.

Table A4.1. Elemental Carbon to Diesel Particulate Matter Conversions

Study	**Conversion Factor**
Fraser - Clinton	1.12
Fraser - Bingle	1.29
Cal. Air Resources Board	1.04
EPA - TOT Method	1.60
EPA - TOR Method	0.80

The conversion factor chosen to be used in this analysis was 1.12 calculated from the local Houston data obtained by Fraser et al. (Fraser et al., 2002; 2003). This value was within the range of the other values that were calculated from various sources (1.04 - 1.60) and was chosen because it was calculated using data representing the Houston area at a monitoring site located in East Houston. The East Houston area has also been identified by the Task Force as an area of specific interest in evaluating the health impacts from air pollution sources.

The ambient monitoring data obtained from EPA had various measurements for elemental carbon making it necessary to determine which monitors were appropriate for our analyses.

Under advice from staff at the U.S. Environmental Protection Agency, parameter code 88307 - Elemental Carbon Stn PM 2.5 and parameter code 88321 - EC Improve PM 2.5 LC (U.S. EPA, 2006f) were used for analyses. These two data types represent different monitoring procedures and different monitoring locations, but can both act as a surrogate for diesel PM conversions. Parameter code 88307 - Elemental Carbon Stn PM 2.5 monitoring sites are in urban areas and use the Thermal Optical Transmittance (TOT) method. Parameter code 88321 - EC Improve PM 2.5 LC monitoring sites are in rural areas and use the Thermal Optical Reflectance (TOR) method. It was decided to use both types of measurements in our analyses.
Concentrations of elemental carbon from the following Houston monitoring sites were used in our analyses:

Site Name	**Parameter Code**
Galveston Airport	88307
Houston Aldine	88307
Channelview	88307
Houston Bayland Park	88307
Houston East	88307
Houston Deer Park 2	88307
Houston Deer Park 2	88321
Conroe (Relocated)	88307

Cancer evaluations of diesel emissions vary between the U.S. Environmental Protection Agency (U.S. EPA) and the California Office of Environmental Health Hazard Assessment (OEHHA). The U.S. EPA has determined that diesel exhaust is likely to be carcinogenic to humans but has judged that toxicological data are not yet sufficient to develop a unit risk estimate for cancer evaluations. The California (OEHHA) Diesel Exhaust Toxic Air Contaminant (TAC) document (California ARB & OEHHA, 1998) stated that the results of epidemiological analyses are consistent with a positive association between occupational exposure to diesel exhaust and an increased risk of developing lung cancer and has developed a cancer unit risk estimate (URE) for diesel exhaust. The Mayor's Task Force analysis has used the URE developed by OEHHA for the cancer assessment for diesel exhaust. Diesel emissions have been assessed for effects other than cancer by the U.S. EPA's Integrated Risk Information System (IRIS) program and this Reference Concentration (RfC) value has also been adopted by the OEHHA.

It should be noted that in the evaluation done by the Mayor's Task Force, the cancer unit risk estimate from the California OEHHA was for diesel exhaust, which includes both particulate and vapor phases. This number was applied to the diesel particulate matter concentrations estimated by the NATA and the Task Force. Diesel particulate matter does not include the vapor phase chemicals.

Polycyclic organic matter (POM)

The EPA's 1999 National Air Toxics Assessment divided POM emissions into eight categories. The first two categories were assigned a URE equal to 5% of that for pure benzo[a]pyrene. Categories 3-7 were composed of emissions that were reported as individual pollutants. These pollutants were placed in the category with an appropriate URE. Category 8, composed of unspeciated carcinogenic polynuclear aromatic hydrocarbons (a subset of POM called 7-PAH), was assigned a URE equal to 18% of that for pure benzo[a]pyrene (U.S. EPA, 2001). The POM placement into the possible risk category (as determined by the Mayor's Task Force) was based on the placement of POM groups 1-3 into this risk category and is based on their NATA modeled concentrations. POM group 2 ranked as the highest risk of the three groups in the possible risk category based on its modeled concentration in the greatest number of census tracts and its probability of causing cancer. More information about the ranking of pollutants into risk categories can be found in **Appendix 1** of this document.


Photo by Heidi Bethel

Appendix 5

Appendix 5: Table of Uncertain Risks

Table A5.1. Uncertain Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint[2]	Point	Mobile		Area
				On Road	*Off Road*	
1,2-Dichloropropane (propylene dichloride)	Yes[3]	Respiratory	X			X
Ethyl Acrylate	Yes[3]		X			X
Quinoline	Yes[3]					X
Aniline	Yes	Spleen	X			X
Hexachloroethane	Yes	Kidney	X			X
Isophorone	Yes[3]	Development	X			X
2,4-Toluene Diisocyanate	Yes	Respiratory	X			X
2-Nitropropane	Yes	Alimentary (liver)				X
3,3-Dichlorobenzidene	Yes		X			X
4,4'-Methylene bis(2-Chloroaniline)	Yes					X
4,4'-Methylenedianiline	Yes	Eyes	X			X
Acetamide	Yes		X			X
Allyl Chloride	Yes	Nervous	X			X
Asbestos	Yes					X
Benzotrichloride	Yes[3]					X
Benzyl Chloride	Yes					X
Beryllium Compounds	Yes	Respiratory	X		X	X
Bis(Chloromethyl) Ether	Yes					X
Bromoform	Yes		X			X
Captan	Yes[3]					X
Chlordane	Yes	Alimentary (liver)				X
Dichlorvos	Yes[3]	Nervous				X
Heptachlor	Yes					X
Hexachlorobenzene	Yes	Alimentary (liver)	X			X
Hexachlorobutadiene	Yes	Reproductive	X			X
o-Toluidine	Yes		X			X
Pentachloronitrobenzene (Quintobenzene)	Yes[3]					X
Pentachlorophenol	Yes	Alimentary (liver)				X
Trifluralin	Yes[3]					X
Vinyl Bromide	Yes	Alimentary (liver)				X
Ethylbenzene		Development	X	X	X	X
Hexane (N-Hexane)		Nervous	X	X	X	X
Methyl Chloroform (1,1,1-Trichloroethane)		Nervous	X			X
Methyl Ethyl Ketone (2-Butanone)		Development	X			X
Styrene		Nervous	X	X	X	X
Toluene		Nervous (some)	X	X	X	X
Acetonitrile		Mortality (whole body)	X			X
Chlorobenzene		Alimentary (liver); Kidney; Male Repro Sys	X			X
Cyanide Compounds		Nervous	X			X
Methyl Bromide (Bromomethane)		Respiratory	X			X
Methyl Chloride (Chloromethane)		Nervous	X			X
Methyl Isobutyl Ketone (Hexone)		Development	X			X
Vinyl Acetate		Respiratory	X			X
1,2,4-Trichlorobenzene		Alimentary (liver)	X			X

Appendix 5

Table A5.1. Uncertain Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint [3]	Point	Mobile		Area
				On Road	*Off Road*	
Antimony Compounds		Respiratory	X			X
Maleic Anhydride		Respiratory	X			X
Methyl Methacrylate		Respiratory	X			X
Selenium Compounds		Alimentary (liver), Hematological, Nervous	X		X	X
Vinylidene Chloride (1,1-Dichloroethylene)		Alimentary (liver); Kidney	X			X
1,2-Epoxybutane		Respiratory	X			X
4,4'-Methylenediphenyl Diisocyanate (MDI)		Respiratory	X			X
Chloroprene		Respiratory	X			X
Ethylidene Dichloride (1,1-Dichloroethane)	Yes	Kidney	X			
Hexachlorocyclopentadiene		Respiratory	X			X
Hydrazine	Yes	Alimentary (liver); Endocrine	X			X
Mercury Compounds		Nervous	X			X
Methyl Isocyanate		Respiratory; Decreased weight gain	X			X
Phosphine		Whole body (decreased weight)				X
Styrene Oxide		Respiratory	X			X
Triethylamine		Respiratory	X			X
Isopropylbenzene (Cumene)		Kidney and Endocrine	X	X	X	X
2,2,4-Trimethylpentane			X	X	X	X
Polychlorinated Biphenyls (Aroclors)	Yes		X			X
1,2-Diphenylhydrazine	Yes					
Propoxur						
Hydrochloric Acid (Hydrogen Chloride [Gas Only])		Respiratory	X			X
Methanol		Development	X			X
Xylenes (Mixed Isomers)		Nervous	X	X	X	X
Carbonyl Sulfide			X			X
Cresol/Cresylic Acid (Mixed Isomers)		Nervous	X			X
Ethylene Glycol		Respiratory	X			X
Glycol Ethers		Reproductive	X			X
Hydrogen Fluoride (Hydrofluoric Acid)		Skeletal flurosis (increased bone density)	X			X
Propionaldehyde			X	X	X	X
Acetophenone			X			X
Biphenyl			X			X
Carbon Disulfide		Nervous	X			X
Diethanolamine		Respiratory	X			X
Diethyl Sulfate			X		X	X
Ethyl Chloride		Development	X			X
Methyl Iodide (Iodomethane)			X			X
Dimethylformamide		Alimentary (liver)	X	X	X	X

Appendix 5

Table A5.1. Uncertain Risk Pollutants

Air Pollutant	Health Effects[2]		Emission Source[1]			
	Cancer endpoint	Chronic endpoint [2]	Point	Mobile		Area
				On Road	*Off Road*	
Phenol		Include Liver & Nervous	X			X
Phthalic Anhydride		Respiratory ; Eyes	X			X
1,1-Dimethylhydrazine						X
1,2-Propylenimine (2-Methylaziridine)						X
2,4-Dichlorophenoxyacetic Acid (Including Salts And Esters)						X
4,6-Dinitro-o-Cresol (Including Salts)			X			X
4-Nitrophenol						X
Carbaryl						X
Catechol						X
Chloroacetic Acid						X
Chloromethyl Methyl Ether						X
Cobalt Compounds		Respiratory	X			X
Dibenzofuran						X
Dibutyl Phthalate			X			X
Dimethyl Phthalate			X			X
Dimethyl Sulfate						X
Ethyleneimine (Aziridine)			X			
Fine Mineral Fibers						X
Hydroquinone			X			X
Methylhydrazine						X
Nitrobenzene		Respiratory	X			X
o-Anisidine						X
Phosgene		Respiratory	X			X
p-Phenylenediamine						X
Quinone (p-Benzoquinone)			X			X
2-Acetylaminofluorene						
4-Aminobiphenyl						
4-Nitrobiphenyl						
Beta-Propiolactone						
Calcium cyanamide						
Diazomethane						
Dimethyl carbamoyl chloride						
Hexamethylphosphoramide						
Methoxychlor						
N-Nitroso-N-methylurea						
Parathion						

[1]Emissions taken from the National Emission Inventory (NEI), 1999; [2]Only chronic health effects associated with chronic health value used in the analysis are depicted in the table; (U.S., EPA, 2006c)

Appendix 6

Brief Descriptions of Health Effects for Definite Risks

Some specific health effects of the air toxics labeled as "Definite Risks" have been outlined in several sources including the Agency for Toxic Substances and Disease Registry (ATSDR, 2006) and Scorecard.org (Green Media Toolshed) have been included to elucidate the potential problems associated with these particular hazardous air pollutants. As seen in **Table 1**, the 12 "Definite Risk" pollutants include 9 HAPs, diesel particulate matter, fine particulate matter and ozone. A brief description of the health effects of each of these is presented here.

1,3-Butadiene is colorless gas with a pungent odor. It is used in large volumes for the manufacture of synthetic rubber and other polymers (CEN, 7/11/2005). Human exposure occurs almost entirely through inhalation of contaminated air. It exists in measurable quantities in almost all urban or suburban settings (ATSDR, 1993). The primary sources of butadiene in ambient air are vehicle emissions, combustion products, including tobacco smoke, and emissions from industrial facilities where it is made or used. It is a potent multi-organ carcinogen in mice and to a lesser extent in rats (NTP, 1993; Owen *et al.*, 1987). Occupational exposures to butadiene have been associated in several studies with cancers of the blood forming organs, particularly leukemia in styrene-butadiene rubber workers (Delzell *et al.*, 1996). In mice, low level exposures have been associated with ovarian atrophy. Developmental disorders have been observed in mice at higher concentrations (Hackett *et al.*, 1987). Butadiene is metabolized in the body to intermediate products that are reactive and can bind to DNA, resulting in mutations (permanent genetic changes) (Jackson *et al.*, 2000). Butadiene and its metabolites can induce mutations in mice (Meng *et al.*, 1999). In some studies increased frequencies of mutations were observed in exposed workers (Ammenheuser *et al.*, 2001; Ward *et al.*, 2001). Butadiene is listed as a carcinogen or a probable carcinogen by several organizations including the US National Toxicology Program (NTP, 1993, 2002), the International Agency for Research on Cancer (IARC Working Group, 1999), and the state of California (OEHHA, 2000, 2006).

Although acrolein is often in liquid form, it vaporizes at typical ambient temperatures, and therefore can be present in air. Acrolein may be the result of accidental release from industrial sources or it may be formed by the reactions of pollutants found in outdoor air. It is also produced from the burning of gasoline. Health effects are generally seen in the respiratory system. There is currently no definitive information on the carcinogenicity of acrolein.

According to Scorecard.org, acrylic acid is a suspected immunotoxicant (Hazard Action Mitigation Planning), respiratory toxicant (EPA) and skin toxicant (EPA). Furthermore, it is ranked as one of the most hazardous pollutants to ecosystems and human health. Inhalation is a common route of human exposure to acrylic acid. No information is available on the carcinogenic effects of acrylic acid in humans, and animal studies have shown mixed results.

Like acrolein, acrylonitrile evaporates quickly, and it is most likely to be found in the air around chemical plants where it is made. Although the evidence is not unequivocal, workers exposed to acrylonitrile at low levels for extended periods have a higher-than-average chance of developing lung cancer. In animals, exposure to acrylonitrile in the air or in drinking water has been found to increase the number of tumors occurring in the brain, salivary glands, and intestines. Although birth defects have been seen in animal studies, there is currently no evidence this is an outcome expected in humans.

Benzene comes from both mobile and industrial sources. It is made mostly from petroleum. It has been characterized as having a sweet odor. Exposure to benzene generally occurs through the air, although it can be found in water and soil. Benzene inhalation can lead to problems with blood production. Long-term exposure to benzene can cause cancer of the blood-forming organs (i.e. leukemia). Furthermore, animal studies suggest benzene has harmful effects on the developing fetus. Neurological effects are seen in high level exposure to benzene. The immune system may also be affected by excessive exposure to benzene. This increases one's risk for infection and may even lower the body's defense against cancer.

Appendix 6

Chlorine is considered to be a high volume chemical with production exceeding 1 million pounds annually in the U.S. Inhalation is a probable route of human exposure to chlorine. Low level exposure leads to irritation of the eyes, nose, throat, respiratory tract, and lungs.

Chromium takes several different forms in the environment. The most common are chromium(0), trivalent (chromium III), and hexavalent (chromium VI). Chromium VI is commonly produced by industrial processes. Chromium compounds, mostly in chromium III or chromium VI forms, produced by the chemical industry are used for chrome plating, the manufacture of dyes and pigments, leather tanning, and wood preserving. These chromium compounds are found in the air as fine dust particles. In general, chromium VI is more toxic than chromium III. Chromium VI is believed to be responsible for increased lung cancer rates observed in workers who have chronic exposure to chromium compounds. High levels of chromium VI may also cause respiratory illness such as asthma.

According to Scorecard.com, diesel particulate matter from diesel engines is the predominant source of cancer risk from hazardous air pollutants. It has been determined that for the U.S., the average cancer risk associated with diesel particulate matter is 580 per million. Diesel emissions also pose significant non-cancer health risks. The State and Territorial Air Pollution Program Administrators and the Association of Local Air Pollution Control Officials estimated that 125,000 cancer cases may be due to diesel particulate matter (Green Media Toolshed).

Ethylene dibromide was used primarily as a pesticide and a gasoline additive. Although exposure to ethylene dibromide can occur through the air, the more common routes of exposure are soil and groundwater. Inhalation studies in animals indicate that high concentrations of ethylene dibromide can lead to death, whereas lower concentrations can cause liver and kidney damage. Although no known birth defects are due to ethylene dibromide, it has been linked to decreased sperm production in males.

A major route of exposure for formaldehyde is the air. It is used in many industries and is a ubiquitous part of life. Concentrations, however, are greatest in urban areas. The most common symptoms of high-level formaldehyde exposure are irritation to the eyes, nose and throat. Chronic long-term exposure has been associated with cancer of the nose and throat, although other studies have not confirmed this. The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have concluded that formaldehyde is a potential human carcinogen.

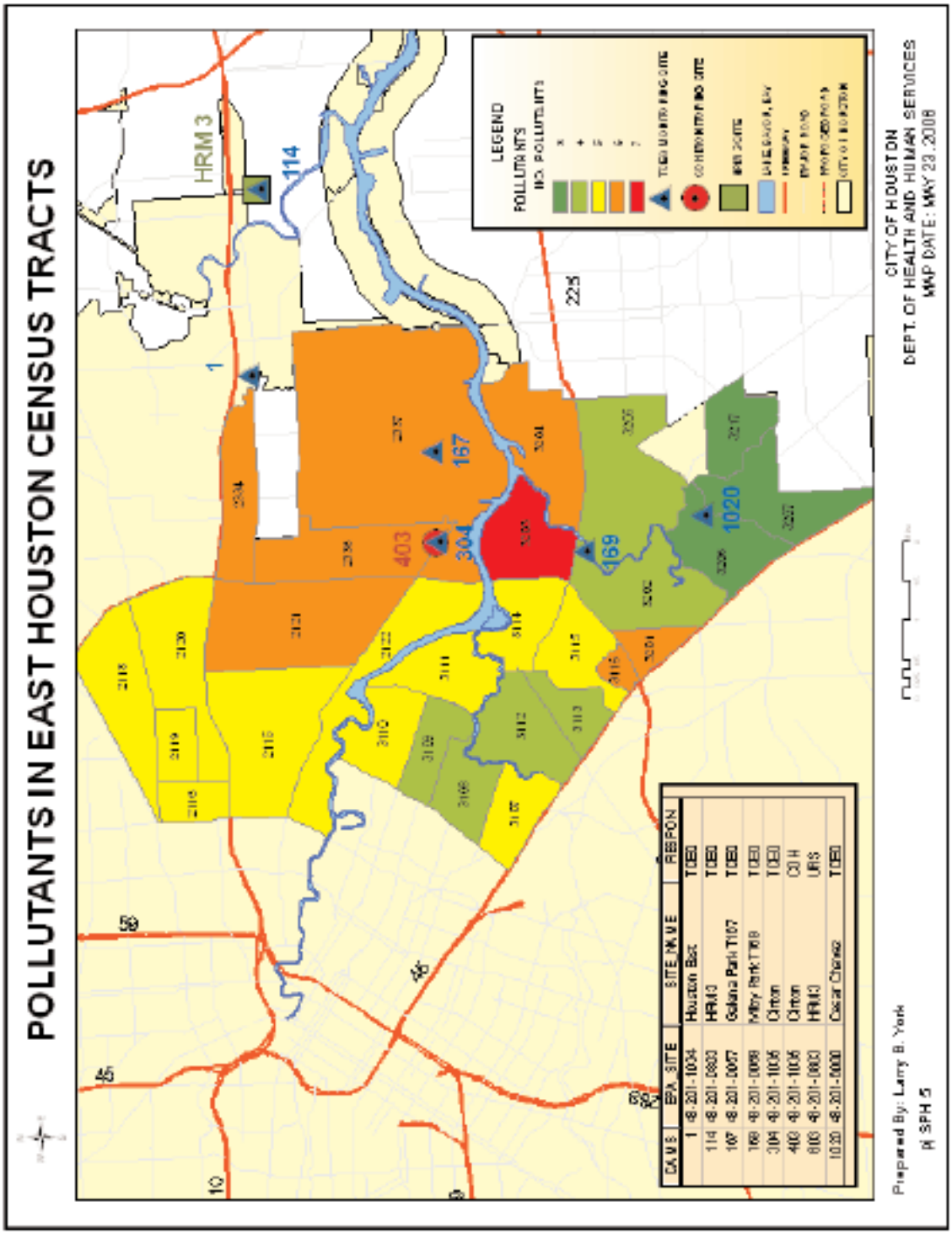
The most common use of hexamethylene diisocyanate is as a hardening agent for automobile paints. The most common route of exposure is through air. Acute high concentrations as well as long-term low levels of hexamethylene diisocyanate are associated with respiratory illnesses.

Ozone is a respiratory irritant which can cause effects which range from mild to severe depending on exposure conditions and individual susceptibility. In general, as concentrations of ground-level ozone increase, more and more people experience health effects and the effects become more severe. Common symptoms of ozone exposure include mild irritation of the throat, difficulty breathing and chest tightness. Ozone aggravates chronic lung disease such as emphysema and bronchitis. Permanent lung damage may be caused through repeated ozone exposures. Repeated exposures by children may lead to reduced lung function in adulthood. In adults, repeated exposures will result in an accelerated decline in lung function. Several groups of people are particularly sensitive to ozone exposure. These groups include: active children, active adults of all ages, people with asthma or other respiratory diseases and people with unusual susceptibility to ozone.

Particulate matter is a multi-component pollutant made up of acids (such as nitrates and sulfates), organic chemicals, metals, soil or dust particles, and allergens (such as fragments of pollen or mold spores). The size of the particles is directly linked to their capacity for causing health problems. Small particles, less than 2.5 micrometers pose the greatest problems because they penetrate deep into the lungs. Particle exposure can lead to a variety of health effects. Short-term exposures to particles (hours or days) can aggravate lung disease, causing asthma attacks and acute bronchitis. Short exposures may increase susceptibility to respiratory infections. Individuals with heart disease may experience heart attacks and arrhythmias. Healthy individuals may experience temporary symptoms after short exposures such as irritation of the eyes, nose and throat; coughing; phlegm; chest tightness; and shortness of breath. Long term exposures to particles have been associated with reduced lung function, the development of chronic bronchitis and premature death. Individuals particularly susceptible to particulate matter exposure include those with lung disease, asthma or heart disease.

Appendix 7

Summary Map and Table for East Houston



Appendix 7: Table 1

East Houston Definite Risk Pollutants

Table A7.1. East Houston Definite Risk Pollutants

Census tract number	Total Pollutants	Acrolein	Chromium VI	Diesel particulate matter	Formaldehyde	Benzene	Chlorine	1,3-Butadiene	Hexamethylene diisocyanate
3203	7	X	X	X	X	X	X	X	
2121	6	X	X	X	X	X	X		
2334	6	X	X	X	X	X	X		
2336	6	X	X	X	X	X	X		
2337	6	X	X	X	X	X	X		
3116	6	X	X	X	X	X		X	
3201	6	X	X	X	X	X		X	
3204	6	X	X	X	X	X	X		
2115	5	X	X	X	X	X			
2116	5	X	X	X	X	X			
2118	5	X	X	X	X	X			
2119	5	X	X	X	X	X			
2120	5	X	X	X	X	X			
2122	5	X	X	X	X	X			
3107	5	X	X	X	X	X			
3110	5	X	X	X	X	X			
3111	5	X	X	X	X	X			
3114	5	X	X	X	X				X
3115	5	X	X	X	X	X			
3108	4	X	X	X	X				
3109	4	X	X	X	X				
3112	4	X	X	X	X				
3113	4	X	X	X	X				
3202	4	X	X	X				X	
3205	4	X	X	X				X	
3206	3	X	X	X					
3207	3	X	X	X					
3217	3	X	X	X					

Appendix 8: Table 1

Supplemental Tables for Definite Risks, Probable Risks, and Possible Risks

Table A8.1. Definite Risk

Air Pollutant	Basis for Classifying as 'Definite Risk'			Data Source for Classifying as 'Definite Risk'				
	Cancer Risk Value	Chronic Reference Value	NAAQS Criteria	AQS 2004			NATA 1999	
				Number of Monitors		Days in Exceedance	Number of Census Tracts	
				Cancer	*Chronic*		*Cancer*	*Chronic*
Ozone			X		20	46		
Fine Particulate Matter			X	0		0		
Diesel Particulate Matter	X	X		6[A]			895	43
1,3-Butadiene	X	X		7	1		9	1
Chromium VI	X						433	
Benzene	X			2			66	
Ethylene Dibromide (Dibromoethane)	X			16				
Acrylonitrile	X						6	
Formaldehyde		X			2			143
Acrolein		X			3			889
Chlorine		X						31
Hexamethylene Diisocyanate		X						6

AQS, Air Quality System; NATA, National-Scale Air Toxics Assessment; NAAQS, National Ambient Air Quality Standards

[A]Diesel estimated using measured ambient elemental carbon concentrations see Appendix 4.

Appendix 8: Table 2

Table A8.2. Probable Risk

Air Pollutant	Basis for Classifying as 'Probable Risk'			Data Source for Classifying as 'Probable Risk'				
	Cancer Risk Value	Chronic Reference Value		AQS 2004			NATA 1999	
				Number of Monitors			Number of Census Tracts	
				Cancer	*Chronic*		*Cancer*	*Chronic*
Vinyl Chloride	X						1	
Acrylic Acid		X						1
Acetaldehyde	X	X		2	1		48	1
Ethylene Dichloride (1,2-Dichloroethane)	X			1			5	
Naphthalene	X						10	
Arsenic Compounds(Inorganic may include Arsine)	X						7	
Carbon Tetrachloride	X			16			895	
Ethylene Oxide	X						9	
1,1,2,2-Tetrachloroethane	X			2				

AQS, Air Quality System; NATA, National-Scale Air Toxics Assessment; NAAQS, National Ambient Air Quality Standards

Appendix 8: Table 3

Table A8.3. Possible Risk

Air Pollutant	Basis for Classifying as 'Possible Risk'		Data Source for Classifying as 'Possible Risk'			
	Cancer Risk Value	Chronic Reference Value	AQS 2004		NATA 1999	
			Number of Monitors		Number of Census Tracts	
			Cancer	*Chronic*	*Cancer*	*Chronic*
Nickel Compounds	X	X			1	1
Manganese Compounds		X				1
Cadmium Compounds	X	X	6		2	1
Titanium Tetrachloride		X				1
2,4-Dinitrotoluene	X				1	
Methyl Tert-Butyl Ether	X		1		61	
1,3-Dichloropropene	X				9	
Chloroform	X		16		41	
Methylene Chloride (Dichloromethane)	X				56	
p-Dichlorobenzene	X				64	
Propylene Oxide	X				8	
Tetrachloroethylene (Perchloroethylene)	X		16		683	
Trichloroethylene	X				2	
1,1,2-Trichloroethane	X		16			
Bis(2-Ethylhexyl)Phthalate (Dehp)	X				895	
Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	X				3	
Lead Compounds	X				1	
1,2-Dibromo-3-Chloropropane	X				3	
1,4-Dioxane	X				2	
2,4-Toluenediamine	X				1	
Acrylamide	X				1	
Benzidine	X				2	
Dichloroethyl Ether (Bis[2-Chloroethyl]Ether)	X				1	
Polycyclic Organic Matter (POMs)	X				76	

AQS, Air Quality System; NATA, National-Scale Air Toxics Assessment; NAAQS, National Ambient Air Quality Standards

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