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Section 812 Prospective Study of the Benefits and Costs of the Clean Air Act:

Air Toxics Case Study - Health Benefits of Benzene Reductions in Houston, 1990-2020

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LIST OF ACRONYMS

AEO	Annual Energy Outlook
AERMET	AERMOD Meteorological Preprocessor
AERMOD	American Meteorological Society/Environmental Protection Agency Regulatory Model
ALL	Acute Lymphocytic Leukemia
AML	Acute Myelogenous Leukemia
ANLL	Acute Non-lymphocytic Leukemia
AQM	Air Quality Modeling
ATP	Anti-Tampering Program
BEIR	Biological Effects of Ionizing Radiation
CAA	Clean Air Act
CAAA	Clean Air Act Amendments of 1990
CalEPA	California Environmental Protection Agency
CAMD	Clean Air Markets Division
CHAD	Consolidated Human Activity Database
CI	Compression Ignition
CLL	Chronic Lymphocytic Leukemia
CMAQ	Community Multi-scale Air Quality
CML	Chronic Myelogenous Leukemia
COI	Cost-of-Illness
DOE	U.S. Department of Energy
EC	Exposure Concentration
EGU	Electricity Generating Unit
EIA	U.S. Department of Energy's Energy Information Administration
EMS-HAP	Emissions Modeling System for Hazardous Air Pollutants
EPA	U.S. Environmental Protection Agency

ESD	Emission Standards Division
HAP	Hazardous Air Pollutant
HAPEM	Hazardous Air Pollutant Exposure Model
HL	Hodgkin's Lymphoma
I/M	Inspection and Maintenance
IEc	Industrial Economics, Incorporated
IRIS	Integrated Risk Information System
IUR	Inhalation Unit Risk
MACT	Maximum Achievable Control Technology
ME	Microenvironment
MOA	Mode of Action
MSAT	Mobile Source Air Toxics
NAAQS	National Ambient Air Quality Standards
NATA	National Air Toxics Assessment
NEI	National Emissions Inventory
NESHAP	National Emissions Standards for Hazardous Air Pollutants
NHL	Non-Hodgkin's Lymphoma
NLEV	National Low-Emission Vehicle
NMIM	National Mobile Inventory Model
NPV	Net Present Value
NRC	National Research Council
NWS	National Weather Service
OAR	Office of Air and Radiation
OGWDW	Office of Ground Water and Drinking Water's
OTAQ	Office of Transportation and Quality
PM	Particulate Matter
POTW	Publicly Owned Treatment Works
RfC	Reference Concentration
RFG	Reformulated Gasoline
RIA	Regulatory Impact Analysis
RR	Relative Risk

RVP	Reduced Vapor Pressure
SAB	Science Advisory Board
SAB Council	Science Advisory Board Advisory Council for Clean Air Compliance Analysis
SAB EEAC	Science Advisory Board Environmental Economics Advisory Committee
SAB HES	Science Advisory Board (SAB) Health Effects Subcommittee
SCCs	Source Classification Codes
SI	Spark Ignition
SOCMI HON	Synthetic Organic Chemical Manufacturing Industry Hazardous Organic NESHAP
TCEQ	Texas Council on Environmental Quality
TPY	Tons Per Year
TRI	Toxics Release Inventory
TTI	Texas Transportation Institute
VMT	Vehicle Miles Traveled
VOC	Volatile Organic Compound
VSL	Value of Statistical Life
WSC	West South Central
WTP	Willingness to Pay

EXECUTIVE SUMMARY

Section 812 of the Clean Air Act Amendments of 1990 (CAAA) requires the U.S. Environmental Protection Agency (EPA) to perform periodic, comprehensive analyses of the total costs and total benefits of programs implemented pursuant to the Clean Air Act (CAA). EPA has completed two of these analyses: a retrospective analysis in 1997 of the original CAA covering the period 1970 to 1990, and a prospective analysis in 1999 of the incremental costs and benefits of the CAAA over the period 1990 to 2010. In both of these studies, estimation of the benefits of reduced concentrations of hazardous air pollutants (HAPs) has proved difficult, due to gaps in the toxicological database; difficulty in designing population-based epidemiological studies with sufficient power to detect health effects; limited ambient and personal exposure monitoring data; limited data to estimate exposures in some critical microenvironments; and insufficient economic research to support valuation of the types of health impacts often associated with exposure to individual HAPs.

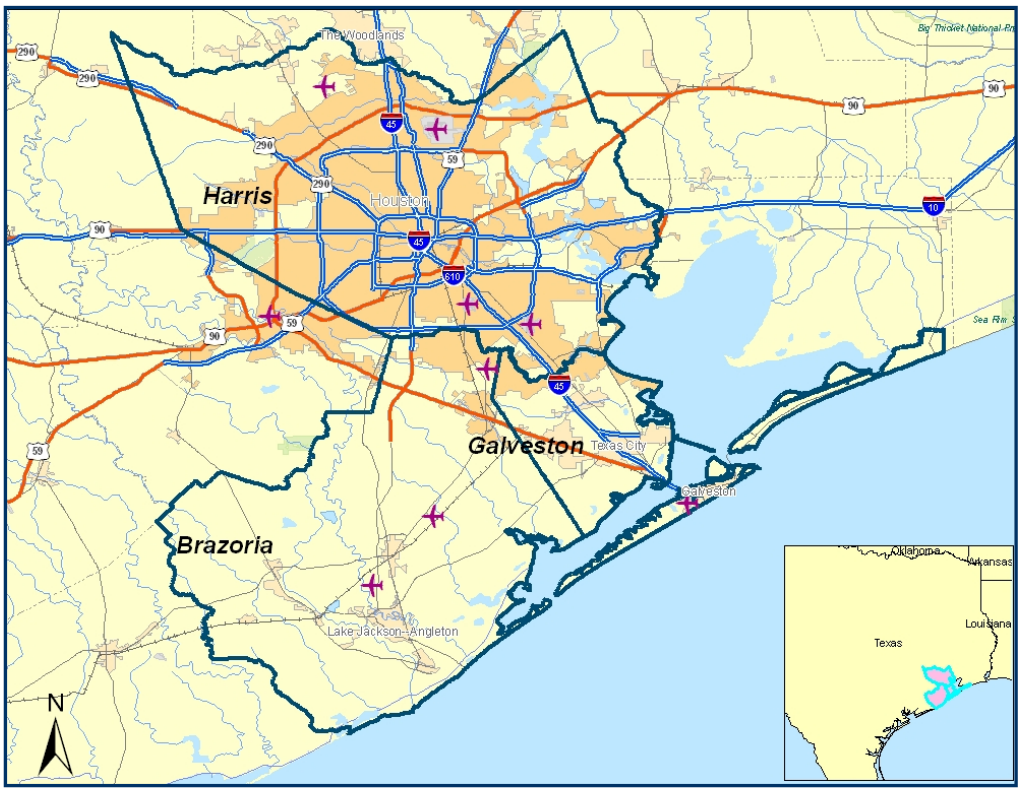
In 2001, EPA's Science Advisory Board Advisory Council for Clean Air Compliance Analysis (SAB) proposed that EPA undertake a HAP benefits case study of a well-studied HAP such as benzene to accompany EPA's second prospective cost-benefit analysis of the CAAA. The SAB indicated that such a study should identify limitations and data gaps; provide an estimate of uncertainties; and provide a scientific basis for deciding whether there is merit in pursuing a greater ability to address air toxics. In response to these comments, EPA developed a methodology for estimating the health benefits of benzene reductions and has applied it in a metropolitan-scale case study of the benefits of CAAA controls on benzene emissions to accompany the main 812 analysis. The results of this study are described in this report.

This case study has two main objectives. The first is to demonstrate a methodology that generates human health benefits resulting from CAAA controls on a single HAP in an urban setting, while highlighting key limitations and uncertainties. The second is to provide a basis for considering more broadly the value of such an exercise for HAP benefits characterization nationwide. This case study is not intended to provide a comprehensive assessment of the benefits of benzene reductions due to the Clean Air Act.

We selected the Houston-Galveston area for the case study (Figure ES-1), a metropolitan area with a large population (a total of 3.4 million in 2000, with nearly 3 million people in Harris County alone) and significant benzene emissions from both on-road mobile sources and large industrial point sources such as petroleum refineries. The study area includes Harris, Galveston, and Brazoria counties – the three counties responsible for

99% of the point source emissions in Houston metropolitan area, according to EPA's 1999 National Emissions Inventory (NEI).

FIGURE ES-1: BENZENE CASE STUDY AREA



The timeframe for this analysis, 1990 through 2020, matches that used in the criteria pollutant analysis of the second prospective Section 812 study. In addition to the base year, 1990, we model results for three target years, 2000, 2010, and 2020.

We conducted this benefits analysis using the standard approach applied in the main 812 criteria pollutant analysis, which includes the following five steps:

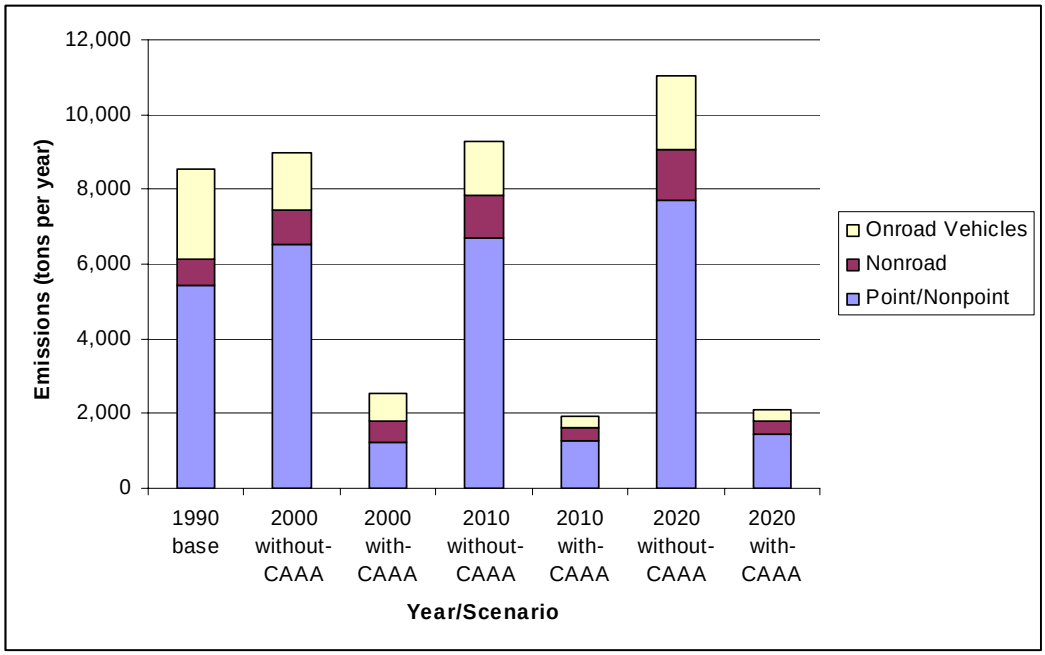
1. Scenario Development
2. Emissions Estimation
3. Air Quality and Exposure Modeling
4. Health Effects Modeling
5. Valuation

We model benzene exposures and health impacts under two scenarios, one reflecting the impacts of all regulatory programs affecting benzene that were enacted in response to the 1990 CAAA (the *With-CAAA* scenario), and one assuming no additional benzene pollution control activity beyond the regulatory requirements existing in 1990 (the

Without-CAAA scenario).[1] The difference between the two scenarios reflects the impact of the CAAA on benzene concentrations and benzene-related health effects in the study area.

We estimated benzene emissions in the Houston-Galveston study area for four source categories: point, non-point (formerly "area sources"), on-road, and non-road. Exhibit ES-2 illustrates emissions changes in each category due to CAAA programs, with significant reductions observed in all categories compared to the *Without-CAAA* case.

FIGURE ES-2: MAJOR, AREA AND OTHER, ON-ROAD, AND NON-ROAD EMISSIONS (TONS) FOR EACH YEAR AND SOURCE TYPE



We applied EPA's American Meteorological Society/U.S. EPA Regulatory Model (AERMOD) dispersion modeling system (U.S. EPA 2004b) to convert emissions estimates to ambient benzene concentrations in the Houston-Galveston study area. Following completion of the AERMOD runs, we applied EPA's Hazardous Air Pollutant Exposure Model, Version 6 (HAPEM6) to the hourly ambient benzene concentration output from AERMOD to generate time-weighted average benzene exposure concentrations for the study population. The HAPEM results reflect the average benzene concentrations likely to be experienced by the study population as they carry out their daily activities.

[1] Our modeling does not include indoor sources of exposure.

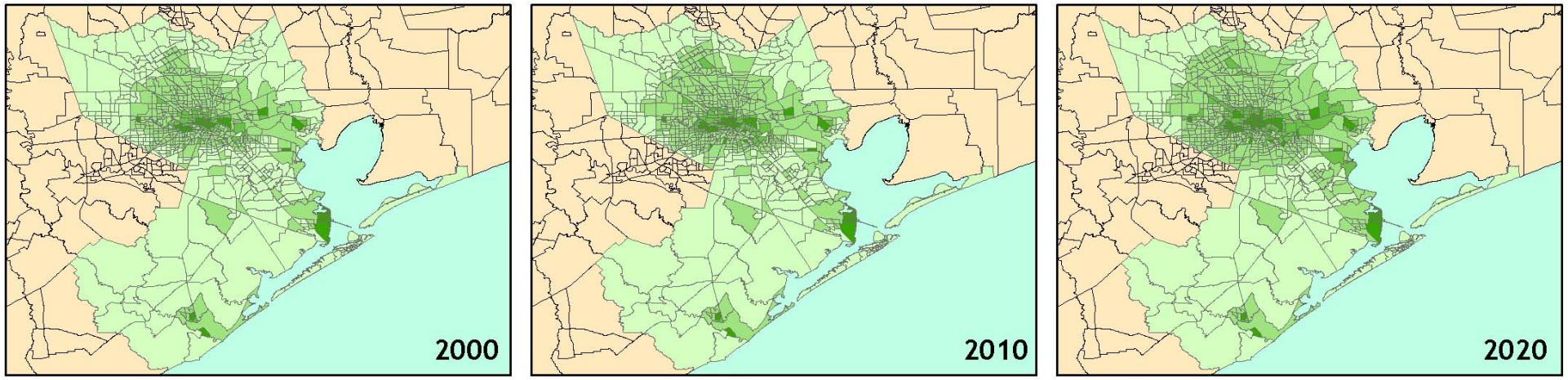
Figure ES-3 presents maps showing the spatial distribution of benzene reductions across the study area. The top row of maps shows the AERMOD estimates of the reduction in annual average ambient benzene levels due to CAAA programs in 2000, 2010, and 2020. The bottom row shows the same progression using the exposure concentration results from the HAPEM model. The maps show the greatest reductions (in excess of 5 $\mu g/m^3$) occur in Harris County in the downtown Houston area, within the rings of the interstate; in the Texas City area of Galveston County where a number of refineries and chemical facilities are located; and in southeastern Brazoria County, which also features major chemical manufacturing and petroleum refining facilities. Mobile source emission controls are a significant contributor to the reductions in Harris County, and we observe an increase over time in the extent and magnitude of reductions in that area, as mobile source controls become more effective over time. In general, HAPEM tends to smooth and spread out the AERMOD concentration changes; this reflects both aggregating results to the census tract level and incorporating the impact of commuting and other activities on the concentration experienced by the population in each census tract.

We focused our health benefits analysis on quantifying avoided cases of leukemia (all types), based on an extensive review of the available health effects literature. To estimate the avoided cases associated with benzene reductions in the study area, we constructed a life-table based risk assessment model. The life-table model assessed age-specific risks within each census tract in each year of the study, based on county-specific background rates of leukemia mortality and morbidity, age-specific benzene exposure data generated by HAPEM (and interpolated for unmodeled years) and a dose-response function from Crump (1994) relating benzene exposure with leukemia.

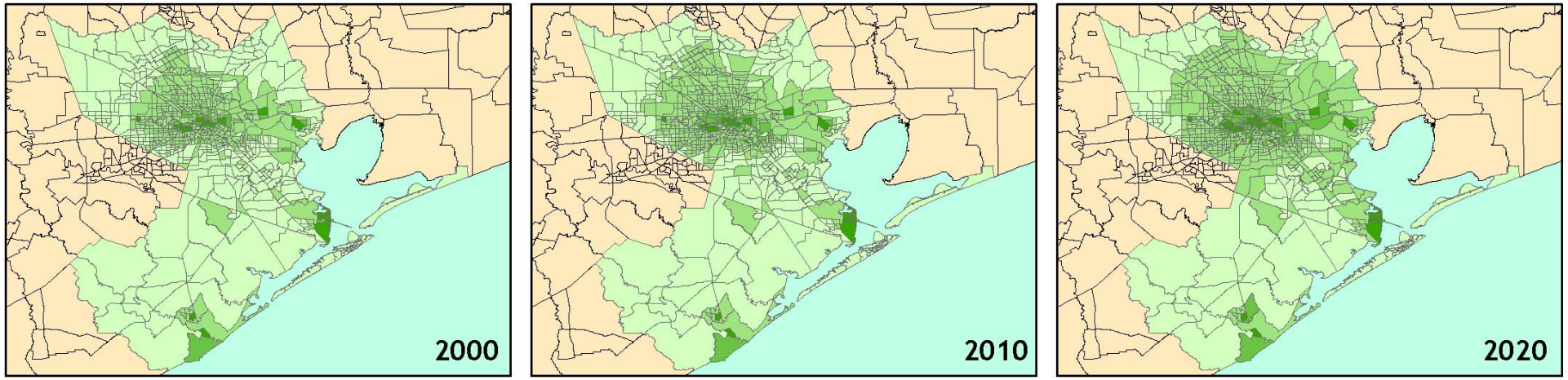
We applied valuation methods that are consistent the current economic literature and SAB advice concerning valuation of cancer-related outcomes. We valued fatal cancers using a value of statistical life (VSL) estimate, with an adjustment for medical costs associated with the period of cancer illness leading up to death. We valued non-fatal cancer cases using a per-case value based on SAB advice in a 2001 consultation on EPA's arsenic in drinking water rule (USEPA, 2001a).

FIGURE ES-3: ESTIMATED CAAA-RELATED REDUCTIONS IN BENZENE CONCENTRATIONS IN THE HOUSTON METROPOLITAN AREA (*WITHOUT-CAAA* MINUS *WITH-CAAA*) – AERMOD AND HAPEM RESULTS

AERMOD RESULTS



HAPEM RESULTS



Reductions in Concentration >2.5 µg/m³ 1.5 to 2.5 µg/m³ 0.5 to 1.5 µg/m³ <0.5 µg/m³

Note: HAPEM results represent the estimated exposure concentration reduction for the median exposed individual in each census tract.

Tables ES-1 and ES-2 present our primary estimate for avoided fatal and non-fatal cases of leukemia due to CAAA-related changes in ambient benzene levels in the Houston area. Table ES-1 presents the number of expected annual cases avoided in each study year as well as the total cumulative avoided cases throughout the study period and the total cumulative avoided cases expected to occur after 2020, due to changes in benzene occurring within the study period. Table ES-2 shows the monetary value of the benefits of the avoided leukemia cases in the study period. Figure ES-4 illustrates the sensitivity of our results to alternative assumptions about the dose-response model.

TABLE ES-1: AVOIDED ANNUAL LEUKEMIA CASES (FATAL AND NON-FATAL) BY STUDY YEAR DUE TO CAAA-RELATED BENZENE EXPOSURE CHANGES IN THE HOUSTON AREA

STUDY YEAR	ANNUAL AVOIDED CASES OF LEUKEMIA		
	AVOIDED FATAL CASES	AVOIDED NON-FATAL CASES	TOTAL AVOIDED CASES
2000	0.03	0.02	**0.05**
2010	0.09	0.07	**0.2**
2020	0.2	0.1	**0.3**
Cumulative Cases Occurring Within the Study Period	**2**	**2**	**4**
Additional Cumulative Cases Occurring After 2020*	**1**	**1**	**2**
Total Cumulative Cases	**3**	**3**	**6**

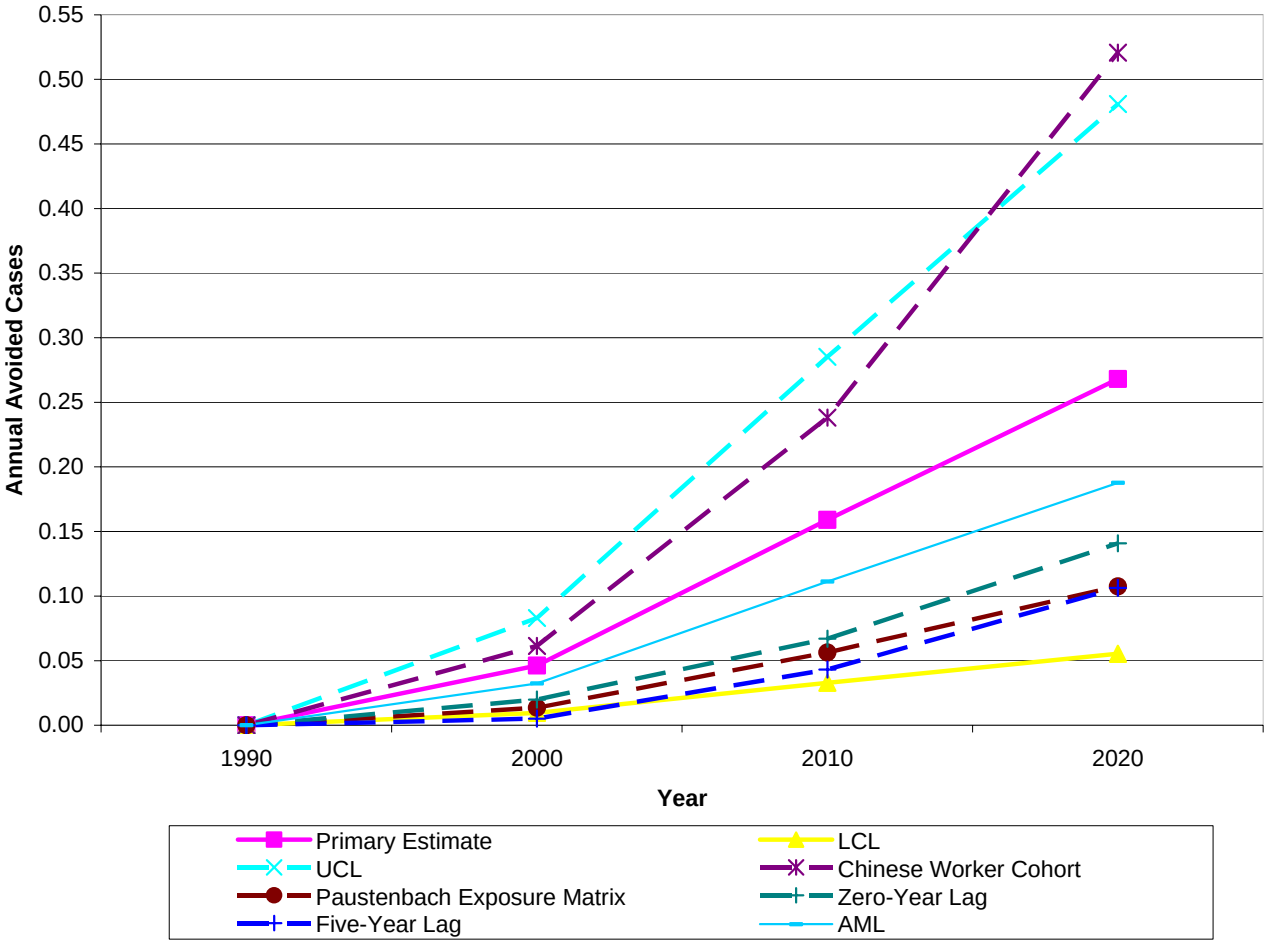
*Note: These avoided cases are due to changes in benzene exposure that occurred within the study period.

TABLE ES-2: TOTAL ANNUAL MONETARY BENEFITS THROUGH 2020 DUE TO CAAA-RELATED CHANGES IN BENZENE EXPOSURE IN THE HOUSTON AREA

STUDY YEAR	TOTAL BENEFITS (1990 NPV, MILLIONS OF 2006$, 5% DR)		
	BENEFITS FROM FATAL CASES OF LEUKEMIA	BENEFITS FROM NON-FATAL CASES OF LEUKEMIA	TOTAL BENEFITS
2000	$0.12	$0.01 – 0.06	**$0.13 - 0.18**
2010	$0.27	$0.01 – 0.13	**$0.28 - 0.40**
2020	$0.31	$0.01 – 0.15	**$0.32 - 0.46**
Cumulative Cases Occurring Within the Study Period	**$6.7**	**$0.32 - 3.3**	**$7.0 - 10**
Additional Cumulative Cases Occurring After 2020*	**$1.6**	**$0.08 - 0.8**	**$1.7 - 2.4**
Total Cumulative Cases	**$8.3**	**$0.40 - 4.1**	**$8.7 - 12**

*Note: These avoided cases are due to changes in benzene exposure that occurred within the study period, but occurred after 2020 due to lagging effects of these changes on leukemia risks.

FIGURE ES-4: ANNUAL AVOIDED CASES OF LEUKEMIA DUE TO CAAA-RELATED REDUCTIONS IN BENZENE IN THE HOUSTON AREA – PRIMARY ESTIMATE AND SENSITIVITY ANALYSES RESULTS



Note: We have linearly interpolated between the avoided leukemia estimates for each target year; however, the true shape of the curve between each of these points is uncertain.

In addition to the leukemia analysis, we evaluated the numbers of individuals likely to be exposed to benzene at levels exceeding EPA's chronic reference concentration (RfC) for benzene, which is based on changes in white blood cell counts, under the *With*-and *Without-CAAA* scenarios. We found no individuals exposed to benzene at concentrations exceeding the RfC in either the *With*- or *Without-CAAA* scenarios. We also conducted illustrative analyses of exposure and risk reductions to highly exposed subpopulations in the study area, and found potentially significant individual risk reductions due to the CAAA for individuals in these groups.

In summary, this case study demonstrates that the 1990 CAAA controls on benzene emissions are expected to result in reductions in the incidence of leukemia in the greater Houston area over the period 1990 to 2020. Key findings include:

- CAAA programs are expected to reduce benzene emissions across all source categories in the study area by thousands of tons per year, with the largest reductions in the point and non-point source category, followed by on-road and non-road sources;
- The largest reductions in benzene exposures are expected to occur in downtown Houston and the surrounding area, and in two areas with significant point sources: the Texas City area of Galveston County and southeastern Brazoria county;
- Reductions in benzene levels are expected to continue, and hence benefits are expected to increase in the latter decades of the study period, as engine and other capital stock turns over and the impact of CAAA controls on on-road and non-road mobile sources in the area increases;
- Primary benefit estimates indicate four fewer cases of leukemia would occur in the three-county area in the study period, two of which we expect would have been fatal. We also expect benefits from the benzene changes that occur between 1990 and 2020 will continue accruing through at least 2030, potentially avoiding another two leukemia cases between 2020 and 2030. We estimate the net present value (NPV) in 1990 of the two fatal and two non-fatal leukemia cases avoided is between $7 and 10 million in 2006 dollars, based on a five percent discount rate.
- 1990 CAAA controls on benzene are expected to significantly reduce individual leukemia risk levels for those living in census tracts with the highest estimated benzene levels by one to two orders of magnitude. For example, some risks in Brazoria County drop from an increased lifetime leukemia risk of 2 in ten thousand (i.e., 2×10^{-4}) to 3 in a million (3×10^{-6}). In four of the six census tracts with the highest risks, individual lifetime leukemia risks are reduced by at least 80 percent.
- Additional health benefits may accrue to individuals living in homes with attached garages. Back-of-the-envelope estimates of the benefits of CAAA-related benzene reductions in the garages of these homes suggest these benefits may be similar in magnitude to our primary estimate. Therefore, these results suggest that adding attached garage-related benefits to our primary estimate could result in an approximate doubling of our primary estimate.

To place these results in context, we note that this air toxics case study focuses only on a subset of the health effects associated with benzene exposure and does not include the total benzene emissions reductions achieved in the Houston area by the CAAA. As such, the case study does not provide a comprehensive assessment of current health effects resulting from benzene exposures in the Houston area; nor does it provide a full measure of the benefits that could be achieved by reducing current benzene emissions affecting

the area. Additional caveats to consider when interpreting the results of this case study include:

- Recent studies in the Houston area suggest that emissions inventories such as the ones used in this case study may significantly underestimate local emissions of VOCs such as benzene from large point sources (e.g., refineries). To the extent that CAAA programs would reduce these emissions, we would not capture these benefits in the case study.
- The case study results include only overall leukemia effects associated with reductions in benzene emissions achieved by a subset of new controls implemented pursuant to the 1990 amendments to the Clean Air Act.
- Reductions from new programs established since we began this case study, especially the Mobile Source Air Toxics Rule, are not included in the analysis.
- Additional health effects that may be associated with benzene exposure but were not included in the quantitative results include other cancers, such as Hodgkin's Lymphoma, and non-Hodgkin's Lymphoma, multiple myeloma, and myelodysplastic syndrome; and potential non-cancer effects related to various hematological abnormalities, including aplastic anemia.
- Co-benefits of reducing air toxics, including reductions in ozone and particulate matter levels, are captured in the overall section 812 study but are not incorporated in the case study.

Despite the limitations of this case study, it successfully demonstrates a methodology that can serve as a useful tool in EPA's evolving HAP benefits assessment strategy. It can provide a comprehensive assessment of the impact of benzene controls from multiple CAAA Titles on cancer incidence in an urban population, using a combination of national and local data to conduct urban-scale modeling of air quality and health impacts. Further, the life-table model allows for more careful assessment of risk changes over time at the census tract level, incorporating local, age-specific baseline incidence data with age-specific exposure data and information on the lag between exposure changes and risk reductions.

Determining where this approach best fits within EPA's HAP benefits assessment strategy will require additional analysis and evaluation to determine the added value of the detailed, urban-scale approach, as well as the potential pool of HAPs suitable for assessment via the damage-function approach for cancer and/or non-cancer effects.

CHAPTER 1 | INTRODUCTION

Section 812 of the Clean Air Act Amendments of 1990 (CAAA) requires the U.S. Environmental Protection Agency (EPA) to perform periodic, comprehensive analyses of the total costs and total benefits of programs implemented pursuant to the Clean Air Act (CAA). The first analysis required was a retrospective analysis, addressing the original CAA and covering the period 1970 to 1990. The retrospective was completed in 1997. Section 812 also requires prospective cost-benefit analyses, the first of which was completed in 1999. The prospective analyses address the incremental costs and benefits of the CAAA. The first prospective analysis covered implementation of the CAAA over the period 1990 to 2010.

EPA's Office of Air and Radiation (OAR) began work on the second prospective study in 2003 with the drafting of an analytical plan for the study. One of the objectives of the analytical plan was to address past comments from EPA's Science Advisory Board Advisory Council for Clean Air Compliance Analysis (SAB Council) concerning treatment of hazardous air pollutants (HAPs) in the previous 812 studies. Assessing the benefits of Clean Air Act controls on the 188 HAPs listed in Title III of the CAAA is much more challenging than analyzing the benefits associated with criteria pollutant reductions, which are the focus of the main 812 benefit/cost analysis. Difficulties include gaps in the toxicological database; difficulty in designing population-based epidemiological studies with sufficient power to detect health effects; limited ambient and personal exposure monitoring data; limited data to estimate exposures in some critical microenvironments; and insufficient economic research to support valuation of the types of health impacts often associated with exposure to individual HAPs. As a result, EPA's efforts to characterize the benefits of HAP reductions in prior 812 analyses have been only partially successful. The SAB Council criticized an analysis of National Emissions Standards for Hazardous Air Pollutants (NESHAP) regulations conducted for the retrospective analysis as substantially overstating benefits, with particular note made of the use of "upper bound" dose-response relationships (i.e., the cancer potency factor used for standard setting).

EPA made a second attempt to incorporate air toxics benefits, in the first prospective analysis (USEPA, 1999a), but the SAB Council found that the national air quality and exposure model proposed would not yield estimates suitable for benefits analysis. In 2001, the SAB Council proposed that EPA undertake a HAP benefits case study, and suggested benzene as a candidate pollutant. The SAB recommended benzene in part because of the wealth of available national ambient concentration data from monitors. The SAB believed that an 812 analysis using the available benzene data would:

- Identify limitations and gaps in the database of air toxics health impact functions;
- Provide an estimate of the uncertainties in the analyses and perhaps provide a reasonable lower bound on potential health benefits from control; and
- Provide a scientific basis for deciding whether there is merit in pursuing a greater ability to assess the benefits of air toxics (USEPA, 2001b).

In response to these comments, EPA conducted a metropolitan scale case study of the benefits of CAAA controls on benzene emissions to accompany the main 812 analysis. This report describes the methodology and results of that analysis.

1.1. PURPOSE AND SCOPE

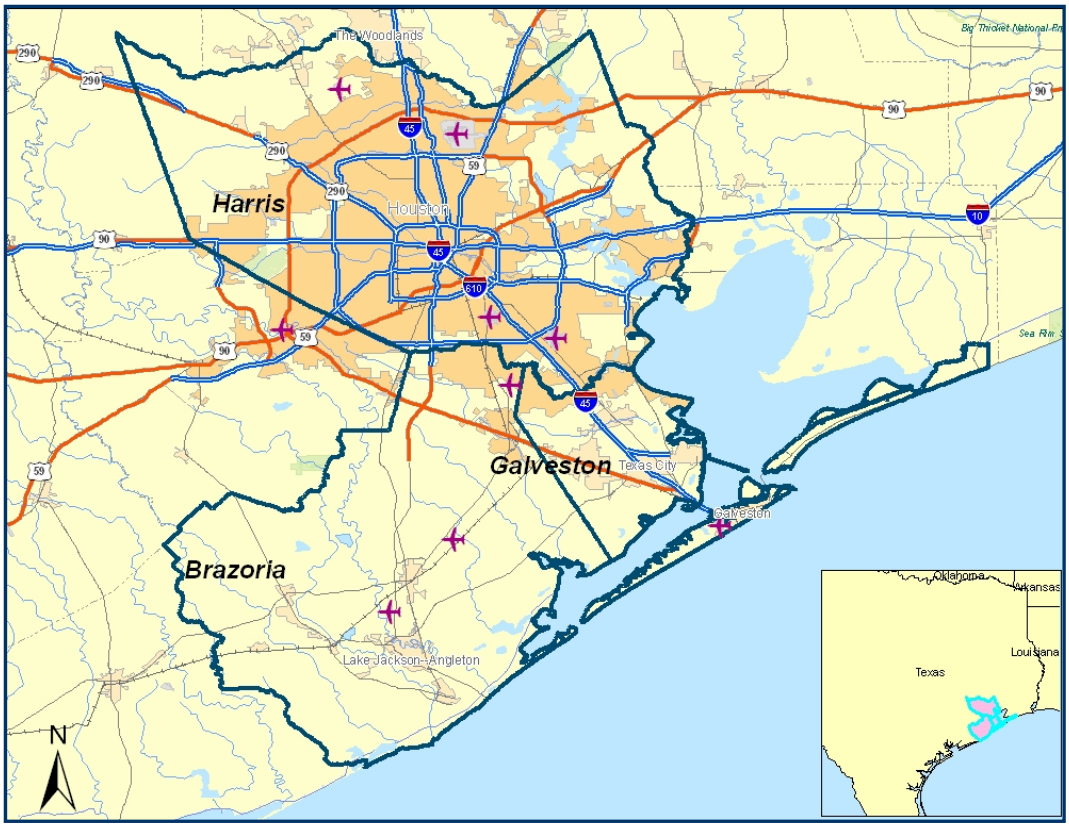
This case study has two main objectives. The first is to demonstrate a methodology that generates human health benefits resulting from CAAA controls on a single HAP in an urban setting, while highlighting key limitations and uncertainties. The second is to provide a basis for considering more broadly the value of such an exercise for HAP benefits characterization nationwide. This case study is not intended to provide a comprehensive assessment of the benefits of benzene reductions due to the Clean Air Act.

We selected the Houston-Galveston area for the case study, a metropolitan area with a large population (a total of 3.4 million in 2000, with nearly 3 million people in Harris County alone) and significant benzene emissions from both on-road mobile sources and large industrial point sources such as petroleum refineries.

Figure 1 displays the study area for this analysis. The study area encompasses three counties – Harris, Galveston, and Brazoria. The selection of these counties represents a balance of resource concerns with comprehensiveness – according to EPA's 1999 National Emissions Inventory (NEI), these three counties contribute 99% of the point source emissions in Houston metropolitan area. The study area also captures significant major roadways, including Interstate 45 and the loops surrounding downtown Houston, Houston's major airports (Bush/Intercontinental and Hobby International), the Port of Houston and the Houston Ship Channel, the refineries of Texas City, and major chemical manufacturing and petroleum refining facilities located in southeastern Brazoria county.

The timeframe for this analysis, 1990 through 2020, matches that used in the criteria pollutant analysis of the second prospective Section 812 study. In addition to the base year, 1990, we model results for three target years, 2000, 2010, and 2020. For each of the target years, we model benzene exposures and health impacts under two scenarios, one reflecting the impacts of regulatory programs enacted in response to the 1990 CAAA, and one assuming no additional benzene pollution control activity beyond the regulatory requirements existing in 1990.

FIGURE 1: BENZENE CASE STUDY AREA



1.2 ORGANIZATION OF THIS DOCUMENT

The remainder of this document is divided into three chapters. Chapter 2 describes our analytical approach to the benefits analysis. Chapter 3 presents the results for the various steps in the analytical chain (i.e., emissions, air quality, exposure concentrations, and health benefits). Chapter 4 presents our conclusions and a discussion of the usefulness of the methods described in this report for broader HAP benefits analysis. The report also includes five appendices. Appendix A provides a detailed description of the emissions modeling conducted by E.H. Pechan and Associates (Pechan). Appendix B describes in detail the air quality modeling performed by EPA. Appendix C presents a health effects literature review memo conducted in support of the health benefits modeling approach, Appendix D presents additional details about the health benefits model, and Appendix E provides the equations used in an analysis of attached garage benzene exposures.

CHAPTER 2 | ANALYTICAL APPROACH

This chapter describes the approach we applied to estimate the health benefits of reduced benzene emissions in Houston between 1990 and 2020 resulting from the Clean Air Act Amendments of 1990 (CAAA). We conducted this benefits analysis using the standard approach applied in the main 812 criteria pollutant analysis, which includes the following five steps:

1. Scenario Development
2. Emissions Estimation
3. Air Quality and Exposure Modeling
4. Health Effects Modeling
5. Valuation

We provide in the following sections a description of our methods for each step in the analytical chain. More detailed information for steps 2 through 4 may be found in Appendices A through D of this document.

2.1 SCENARIO DEVELOPMENT

To assess benefits of CAAA-related benzene reductions, we adopted an approach consistent with the main criteria pollutant analysis in the 812 study. Our benefit analysis is based on construction and comparison of two distinct scenarios: a *Without-CAAA* scenario and a *With-CAAA* scenario. This case study then estimated the differences between the health outcomes associated with these two scenarios.

The *Without-CAAA* scenario essentially freezes federal, state, and local air pollution controls at the levels of stringency and effectiveness that existed in 1990. This scenario is consistent with the baseline for the main 812 analysis. The *With-CAAA* scenario assumes that all federal, state, and local rules promulgated pursuant to, or in support of, the 1990 CAAA were implemented. This scenario includes all current and currently anticipated regulations that affect benzene emissions resulting from the amended clean air act issued in 1990. It includes the regulations listed in Table 1. We note that the Mobile Source Air Toxics (MSAT) rule issued by EPA on February 26, 2007, was not included in this scenario, because the rulemaking was still in progress at the time of this analysis. We expect the MSAT rule to further reduce benzene emissions under the *With-CAAA* scenario beyond what we report in this case study.[2]

[2] Other planned control programs on small spark ignition engines, including locomotive and marine engines, may also reduce benzene further (http://www.epa.gov/otaq/regs/nonroad/marinesi-equipld/420f07032.htm).

TABLE 1: BENZENE CASE STUDY *WITH-CAAA* SCENARIO SUMMARY, BY TITLE

Title I	Any effects of Title I will be expressed through state implementation plan (SIP) requirements, such as (enhanced) I/M programs, transportation control measures, and other VOC controls. These requirements are dependent on the ozone non-attainment status of the case study area(s).
Title II	**Tailpipe standards** **On-road** Tier 1 Standards (phased in 1994 to 1997) National Low-Emission Vehicle (NLEV) program – voluntary bridge between Tier 1 and Tier 2 Tier 2 Standards take effect in 2004 Heavy Duty Engine/Diesel Fuel Rule - New emission standards – 2007 model year, new fuel standards 2006 **Non-road** Federal Phase I and II compression ignition (CI) engine standards Federal Phase I and II spark ignition (SI) engine standards Federal locomotive standards Federal commercial marine vessel standards Federal recreational marine vessel standards **Evaporative Emissions** Stage II Vapor Recovery Systems (Section 182) Onboard Refueling Vapor Recovery (Section 202; 1998 model year and on) Evaporative Test Procedure **Fuel Regulations** Reformulated Gasoline (RFG) Standards (1995 on) Phase II – (2000 – present) – benzene requirements essentially unchanged Summertime Volatility Requirements for Gasoline (Phase II – 1992 on) Fuel Sulfur Limits *2007 Mobile Source Air Toxics (MSAT) Rule NOT included.
Title III	**Maximum Achievable Control Technology (MACT) Standards** The *With-CAAA* scenario included MACT standards that would be expected to have a significant effect on future-year benzene emissions in the Houston area. These standards include: Oil and Natural Gas Production: 7-Year MACT Petroleum Refineries: 4-Year MACT Gasoline Distribution: 4-Year MACT Pulp and Paper Production: 7-year MACT Municipal Landfills: 10-year MACT Natural Gas Transmission and Storage: 10-year MACT Publicly Owned Treatment Works (POTW) Emissions: 7-year MACT Coke Ovens: Pushing, Quenching, & Battery Stacks: 4-year MACT Synthetic Organic Chemical Manufacturing Industry Hazardous Organic NESHAP (SOCMI HON): 2-year MACT

This approach requires two simplifying assumptions. First, we assumed, as noted above, that under the *Without-CAAA* scenario regulations are frozen at 1990 levels, and that no additional voluntary, state, or local pollution control activities occur in the Houston area beyond regulations "on the books" as of 1990. Second, we assumed that the distribution of population and economic activity is the same under both scenarios. Thus, for example, as air quality worsens under the *Without-CAAA* scenario, we did not attempt to model the movement of individuals out of the study area. While migration might in fact occur in response to a degradation in air quality, estimation of the extent of this migration would have required speculation that would not necessarily have been superior to our chosen approach.

Although this is a HAP case study, we did not analyze benefits specific only to Title III of the CAAA (the Title that specifically focuses on HAPs), because doing so would have ignored significant benefits related to reductions of benzene emissions from mobile and stationary sources. Instead, the difference between the *With-* and *Without-CAAA* scenarios for benzene in Houston reflects all CAAA regulations that affect benzene emissions.

2.2 EMISSIONS

We estimated benzene emissions in the Houston-Galveston study area for four source categories: point, non-point, on-road, and non-road. For each category, we generated emissions estimates for the 1990 base year and for three target years (2000, 2010, and 2020) under both the *With-CAAA* and *Without-CAAA* scenarios.

Our overall approach involves estimating emissions by backcasting or forecasting historical emissions data based on the expected growth in emissions-generating activities over time, adjusted for the impact of future year control assumptions under each scenario. For the *With-CAAA* scenario we estimate emissions for the three target years by adjusting benzene emissions from EPA's 2002 National Emissions Inventory (NEI). For the *Without-CAAA* scenario, we generate projections for the three target years by adjusting the 1990 benzene emissions from EPA's 1990 NEI.

The emissions estimates thus depend primarily on the following three elements: 1) the data and methods used to establish the historical year inventories; 2) the indicators used to forecast growth in emissions activities and emission control effectiveness; and 3) the specific regulatory programs modeled under the *With-* and *Without-CAAA* scenarios. We have included a detailed report (E.H. Pechan and Associates, 2006) describing the approach to each of these elements for each source category in Appendix A. We provide an overview of the methods used for each sector below.

2.2.1 POINT SOURCES

We estimated point source emissions in 1990 in the study area using EPA's 1990 National Emissions Inventory (NEI) for HAPs, which was recently revised by EPA.[3] This inventory also served as the base year file for estimating *Without-CAAA* scenario emissions for 2000, 2010, and 2020. We estimated point source benzene emissions for the study area for the 2000 *With-CAAA* scenario by backcasting from the EPA 2002 NEI, and this served as the base year emissions file for preparing 2010 and 2020 *With-CAAA* scenario emission estimates.[4]

For the 1990 NEI, EPA established a hierarchy of preferred data sources in order to prepare the 1990 NEI for HAPs, listed below in order of preference:

- Maximum Achievable Control Technology (MACT) data from EPA's Emission Standards Division (ESD);
- Data developed by state and local air agencies;
- Data from inventories developed by EPA's Emission Inventory Group to support requirements of Sections 112(c)(6) and 112(k); and
- Emissions reported in the Toxics Release Inventory (TRI), and emissions that the Emission Inventory Group generated using emission factors and activity factors.

Nearly 90 percent of the 1990 point source emissions data for the study area came from the first two categories.

The sources of data in the NEI for benzene in the Houston-Galveston area are as follows:

- State data,
- Industry data;
- Data gathered by EPA's Emission Standards Division while developing MACT standards;
- TRI data; and

[3] The original baseline 1990 NTI was a county-level inventory for all source categories. The newly released 1990 NEI for HAPs was created by converting the county-level emission estimates to facility-specific estimates for as many sources as possible. Locational data and stack parameters were added, and additional estimates were developed for missing MACT source categories and HAPs.

[4] We also considered basing the 2000 *With-CAAA* scenario benzene emissions estimates on Texas Council on Environmental Quality (TCEQ) emissions data generated for use in the Texas Air Quality Study (AQS) 2000 study. EPA's Science Advisory Board encouraged EPA to investigate these data as an alternative to the 1999 NEI, which we had originally proposed to use. We chose not to use the Texas AQS data for several reasons. First, it would have required significant adjustments to generate year round emissions estimates, as it only provided data for an August-September 2000 Houston area modeling episode. Second, it lacked the control device information that assists in making emission forecasts to future years. Furthermore, subsequent to the SAB consultation, the 2002 NEI was issued, and the TCEQ submittal for the 2002 NEI reflected improved point source emissions estimates over the 1999 submission. The availability of this improved inventory, combined with the limitation of the Texas AQS data led us to select the 2002 NEI.

- Electricity generating unit (EGU) data developed from information by the U.S. Department of Energy (DOE) and EPA's Clean Air Markets Division (CAMD).

State data comprised over 87 percent of the 2002 point source emissions data, with the rest primarily coming from TRI.

2.2.2 EMISSIONS ACTIVITY FACTORS

When estimating point source category emissions for future years, we applied emission activity factors that reflect the projected ratios of 2000, 2010, and 2020 emission activity to 1990 emission activity (for *Without-CAAA* case emissions estimation) and the ratios of 2000, 2010, and 2020 emission activity to 2002 emission activity (for *With-CAAA* case emissions backcasting and forecasting).[5] We developed emission activity levels for energy-producing and energy-consuming source categories from historical/forecast energy production/consumption data. Because it is not feasible to develop estimates of actual emission activity levels for every non-energy related source category, we derived growth factors for these source categories from surrogate socioeconomic indicator data that are more readily available than emission activity data.

In keeping with past EPA practice, we relied on energy data from DOE's Energy Information Administration (EIA) to backcast and forecast energy consumption and energy production emission source categories. To reflect the 1990 to 2000 trend in energy consumption for source categories, Pechan generally relied on historical time-series energy data for Texas from an EIA energy consumption database (EIA, 2005a). For Crude Oil and Natural Gas Production source categories, Pechan obtained 1990 and 2000 Texas relevant activity data from another EIA source that provided the number of operating oil well days (used for Crude Oil Production) and the number of operating gas well days (used for Natural Gas Production) (EIA, 2005b). For source categories that describe railroad and marine distillate fuel consumption emission processes, Pechan obtained 1990 and 2000 consumption estimates for Texas from an EIA distillate fuel data resource (EIA, 2005c).

When projecting activity for future years, we relied on projections of U.S. energy supply, demand, and prices through 2025, which are published by EIA in its *Annual Energy Outlook 2005 (AEO 2005)* (EIA, 2005d). We applied *AEO 2005* West South Central (WSC) region forecasts to project changes in Houston-Galveston area emissions activity (Texas is included in the WSC region). For example, Stage II (Gasoline Vehicle Refueling) emission activity is projected using *AEO 2005* projections of WSC region transportation sector motor gasoline consumption. We relied on national energy forecasts when *AEO 2005* only provided national projections for the energy growth indicator of interest.

[5] We also applied this approach for projecting non-road source categories that are not incorporated into EPA's NONROAD emissions model.

Because population growth and the performance of the U.S. economy are two of the main determinants of energy demand, the EIA also prepares socioeconomic projections. This study relies on *AEO 2005* historical and forecast socioeconomic data as surrogates for emission activity level changes for most non-energy source categories.[6]

2.2.3 NON-POINT SOURCES

Non-point (formerly "area source") emissions were projected for both the *With-CAAA* scenario (2010 and 2020) and the *Without-CAAA* scenario (2000, 2010, and 2020). The draft 2002 NEI was used as the initial base for the *With-CAAA* scenario, while the 1990 NEI for HAPs inventory was used as the initial base for the *Without-CAAA* scenario.

We conducted a ranking of non-point and non-road benzene emitting categories (i.e., Source Classification Codes (SCCs)) for the 3-county Houston-Galveston area based on benzene emissions reported for EPA's draft 2002 NEI. Based on this ranking, we identified five priority SCCs on which to focus this analysis: gasoline marketing, commercial marine loading, bulk terminals, pipeline facilities, and commercial marine diesel engines.[7] Adjustments to these emissions data to generate projections for future years in each of the two scenarios followed the procedure described in the point source section. Detailed descriptions of additional adjustments to NEI data for use in this analysis are provided in Appendix A.

2.2.4 ON-ROAD SOURCES

For the on-road source category, we calculated emissions estimates that are linked to specific roadway segments (i.e., link-level estimates) for the 1990 base year and all three target years under the *With-* and *Without-CAAA* scenarios. Link-level values have been applied in other urban-scale air quality analyses (e.g., EPA, 2002, Stein et al., 2002, Hao et al, 2002) and can provide improved emissions resolution over grid-based methods for air quality modeling at the census block group level. We generated hourly link-level emissions data by season for each year/scenario combination.

The inputs to the on-road emissions estimation process include estimates of vehicle miles traveled (VMT) and emissions factors (e.g., grams of benzene per mile traveled) for specific vehicle types and driving conditions. We prepared link-level VMT data from VMT data files prepared in 2005 for the Houston area by the Houston-Galveston Area Council and further processed by the Texas Transportation Institute (TTI). We obtained VMT data sets for 2002, 2009, and 2012, and adjusted the data as necessary to match the temporal needs of the Section 812 study. For example, we adjusted the VMT data,

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[6] For four source categories – Residential Wood Fireplaces and Wood Stoves, Aircraft, Forest Wildfires, and Prescribed Burning for Forest Management - we opted to use non-*AEO 2005* surrogates for projecting emissions activity. We applied methods to derive growth factors for these categories that are consistent with those used in past EPA analyses, such as the Clean Air Interstate Rule Regulatory Impact Analysis (RIA). The specific approaches we applied are described in Appendix A.

[7] Portable fuel containers are another significant source of non-point/non-road benzene emissions that were not included in this assessment.

originally developed for an August/September ozone modeling episode, from the specific modeling period to the four seasons, using adjustment factors provided by TTI. We then allocated the VMT by vehicle type and adjusted the data to the study years for this analysis. We conducted the study year adjustment by calculating the average annual VMT growth rate between the two years of VMT data nearest to the year of interest (e.g., 2002 and 2012 for 2020) and then applying that rate to interpolating or extrapolating VMT for that year.

Emission factors were calculated using EPA's MOBILE6.2 model.[8] Where possible, local input data for the Houston area, as provided by the Texas Commission on Environmental Quality (TCEQ), were used in the development of the MOBILE6.2 input files. Emissions factor inputs include registration distributions of vehicles by age, diesel sales fractions, inspection and maintenance (I/M) and Anti-Tampering Program (ATP) inputs, temperature, and fuel characteristics and properties. Details on the specific data used for each of these input categories can be found in Appendix A. Once the input files were prepared, we ran MOBILE 6.2 for the 1900 base year and for 2000, 2010, and 2020 under the *With-* and *Without-CAAA* scenarios. For the *With-CAAA* scenarios, MOBILE6.2 generated emissions factors that reflect the impact of I/M programs and ATPs instituted in the study area after 1990, as well as emissions requirements and fuel programs in place in the year being modeled. For the *Without-CAAA* scenarios, we ran MOBILE 6.2 using 1990 fuel characteristics and the "NO CAAA" command, which excluded the effects of national CAAA programs on emission factors.

2.2.5 NON-ROAD SOURCES

To develop non-road benzene emission estimates from in the Houston, Texas area, we first used EPA's NONROAD2004 model to generate volatile organic compounds (VOC) exhaust and evaporative VOC emissions output from non-road sources for the 1990 base year and future years under both the *With-* and *Without-CAAA* scenarios (USEPA, 2004a).[9] We obtained VOC emissions estimates for the following model equipment categories: recreational vehicles, farm and construction machinery, lawn and garden equipment, aircraft and rail support equipment, and other industrial and commercial applications. Aircraft, commercial marine and locomotive emissions, which are not modeled by NONROAD, were included in the non-point area source portion of the emissions inventory.

[8] Analysis for the recent MSAT rule found that cold start emissions for Tier 1 and later vehicles are much larger than estimated by MOBILE6, suggesting a potential downward bias on emission reduction estimates for this category; however, the impact of these emissions in Houston is likely smaller than in colder climates.

[9] The NONROAD2004 model was released by EPA's Office of Transportation and Quality (OTAQ) in May 2004. This version of the model incorporates all Federal engine standards, with the exception of the large spark-ignition evaporative standards. VOC reductions from this standard were applied outside of the NON-ROAD model, as described in Appendix A. A recent revision to NONROAD (NONROAD2005) includes new evaporative emission categories, such as tank and hose permeation, and revised hot soak emission estimates, which increase the inventory. These revisions are not included in our analysis.

To estimate the specific benzene emissions associated with NONROAD's various categories of VOC emissions, we compiled engine-specific benzene speciation factors for exhaust and evaporative emissions from EPA's National Mobile Inventory Model (NMIM) (USEPA, 2005a).[10] We then multiplied the SCC-level VOC emissions estimates by these factors to produce estimates of benzene emissions from non-road sources.[11] The specific benzene speciation factors applied can be found in Appendix A.

We employed a revised NONROAD model growth file with region-specific growth rates, consistent with the main criteria pollutant analysis of the Section 812 Prospective study.[12] Input files were prepared for Brazoria, Galveston, and Harris counties to reflect the appropriate temperature and fuel inputs for the *With-CAAA* scenario runs.[13] In addition, fleet emission rate inputs were modified to remove the effect of CAAA-related standards for the *Without-CAAA* runs. Using county-specific input files, NONROAD model runs were performed to generate seasonal emission estimates for each scenario year. Seasonal emissions were then summed to estimate annual emissions at the county and SCC level for each scenario/year.

2.3 AIR QUALITY AND EXPOSURE MODELING

The air quality modeling (AQM) step links emissions changes within the three-county study area to changes in atmospheric concentrations of benzene. It replicates dispersion and transport of emitted benzene through the atmosphere to generate a set of estimated ambient benzene concentrations at the census tract level. When combined with information about the time-activity patterns of an exposed population, the ambient AQM estimates can be converted to estimates of individual exposure concentrations for that population.

We applied EPA's American Meteorological Society/U.S. EPA Regulatory Model (AERMOD) dispersion modeling system (U.S. EPA 2004b) to convert emissions estimates to ambient benzene concentrations in the Houston-Galveston study area in the base and target years under the *With-* and *Without-CAAA* scenarios.[14] AERMOD is a state-of-the-art steady-state Gaussian plume model that is one of EPA's preferred models

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[10] Evaporative hydrocarbon emissions as calculated by NONROAD are comprised of crankcase, diurnal, spillage, and vapor displacement components.

[11] No benzene emission factors were available (or applied) for Liquified Petroleum Gas or Compressed Natural Gas-fired equipment.

[12] The procedures used to develop the regional growth rates are described in the Section 812 Prospective report (Pechan, 2005a).

[13] Input parameters for Brazoria, Galveston, and Harris counties were developed that reflected local and national fuel programs for the *With-CAAA* scenario runs for 2000, 2010, and 2020. Local inputs, including seasonal reduced vapor pressure (RVP) limits, oxygenated fuel specifications for reformulated gasoline, and Stage II programs were available from EPA's NMIM county database (USEPA, 2005a). Federal gasoline and diesel fuel sulfur levels were incorporated as well.

[14] We also considered using the Community Multiscale Air Quality (CMAQ) model to estimate ambient benzene concentrations. However, benzene is a relatively stable compound and therefore the ability of CMAQ to account for photochemical processes was not necessary. In addition, the AERMOD model is able to provide finer spatial scale resolution.

for regulatory analyses of this scale; it handles multiple sources, incorporates building downwash, has flexibility in receptor location choices, and also includes the option to vary emissions by season and hour of day. We fed the AERMOD output into EPA's Hazardous Air Pollutant Exposure Model, Version 6 (HAPEM6) to generate benzene exposure concentrations.

The next three sections describe the AERMOD modeling approach, and the fourth covers the HAPEM exposure modeling.

2.3.1 AQM MODEL INPUTS

Inputs to the model included a receptor grid (i.e., the geographical locations at which concentrations are to be estimated); the emissions data from the previous step, which were processed to conform with AERMOD requirements; meteorological data; land use and elevation data; and information on background levels. Detailed information about the development of each input can be found in Appendix B (note that Appendix B uses the term "area and other" to refer to non-point source emissions). We present below a brief overview of how each of these inputs was handled:

- **Modeling Domain/Receptors.** The modeling domain matched the three-county study area; we located receptors at census block group centroids.[15] We also placed some receptors at benzene monitoring locations for the purpose of model evaluation.
- **Emissions Data.** We employed EPA's Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP, Version 3.0, USEPA, 2004d) to process the seven emissions inventories developed by Pechan (2006) into the emissions input files required by AERMOD. The emissions processing required two steps. First, some of the emissions data required additional modifications prior to input into EMS-HAP, such as development of some source characteristics needed by AERMOD. Details of the emissions pre-processing can be found in Chapter 3 of Appendix B. Once the pre-processing was complete, we ran the emissions profiles through EMS-HAP to generate spatially and temporally allocated emissions input files appropriate for use with AERMOD. Additional information about EMS-HAP processing can be found in Chapter 4 of Appendix B.
- **Meteorological Data**. We prepared meteorological data for two years, 1990 and 2000. We input the 1990 meteorological data for the 1990 AERMOD simulation. We input the year 2000 data for all the other simulations. We used the AERMOD Meteorological Preprocessor (AERMET) (U.S. EPA, 2004c) to process the National Weather Service (NWS) data for both 1990 and 2000.

[15] A census block is a subdivision of a census tract. It is the smallest geographic unit for which the Census Bureau tabulates sample data. A block group consists of all the blocks within a census tract with the same beginning number (U.S. Census Bureau, 2007). For the 1990 simulation, the receptors were the 1990 census block group centroids, giving a total of 2,429 receptors. For all other AERMOD simulations in the study (2000, 2010, and 2020), the 2000 census block group centroids were chosen as the receptors, for a total of 2,285 receptors.

- **Land Use and Elevation Data.** We used data on land use to designate sources as urban or rural for dispersion modeling purposes. The urban/rural designation is important for AERMOD modeling when assigning deposition parameters. For non-point and non-road sources, excluding airport emissions, we assigned sources the land use designation of the census tracts to which the emissions were assigned during spatial allocation in EMS-HAP. We assigned each point source the land use designation of the closest tract. We modeled link-level on-road emissions as rural sources. This is consistent with previous studies in Houston (U.S. EPA, 2002a). We also modeled non-point and non-road airport related emissions as rural sources. Because the terrain is relatively flat over the Houston area, we ran the AERMOD simulations using the flat terrain option (i.e., we assumed sources and receptors are at the same elevation).
- **Background.** We added background concentrations to AERMOD modeled concentrations at each receptor (block group centroids) in a post-processing step to account for benzene contributions from sources outside the study area. We assigned background concentrations of benzene for all years and modeling scenarios based on the 1999 county specific background concentrations as used for the 1999 National Air Toxics Assessment (NATA, USEPA 2001b).[16] We applied the same background concentration to every receptor in a given county.

2.3.2 AQM MODEL RUNS

We performed seven model runs using AERMOD Version 04300 (one for the 1990 base year and two for each target year – one under the *With-CAAA* scenario and one under the *Without-CAAA* scenario). (The control options used for each run can be found in Appendix B, Table 12.).[17] For each model run, we generated hourly, daily, and annual average concentration output files for each source category (major, non-point, on-road, non-road, and total).[18] The hourly concentrations from AERMOD were then input into the HAPEM6 model (described in the next section), to generate exposure concentrations that reflect the influence of the activity patterns of the exposed population.

2.3.3 AQM MODEL EVALUATION

We performed an evaluation of the AERMOD results by comparing modeled concentrations to observed concentrations. In addition to the census block group centroids, we estimated daily and annual average model concentrations at monitor locations. We performed model-to-monitor comparisons for the year 2000 AERMOD

[16] For details about the 1999 background values see http://www.epa.gov/ttn/atw/nata1999/background.html or Battelle (2003).

[17] Receptors were the census block group centroids (the 1990 census block group centroids for the 1990 and the 2000 census block group centroids for all other years).

[18] Appendix B refers to non-point emissions as "area and other."

results using monitor observations obtained from EPA's Air Toxics Archive.[19] We identified 15 monitor locations available for comparison, mostly in southern Harris County (See Appendix B). We were unable to conduct a comparison for 1990, because only one benzene monitor existed in the study area at that time.

2.3.4 BENZENE EXPOSURE CONCENTRATION MODELING

Following completion of the AERMOD runs, we estimated time-weighted average benzene exposure concentrations for the study populations using the Hazardous Air Pollutant Exposure Model, Version 6 (HAPEM6) and the hourly ambient benzene concentration output from AERMOD. HAPEM assesses average long-term inhalation exposures of the general population, or a specific sub-population, over spatial scales ranging from urban to national. HAPEM6 tracks representatives of specified demographic groups as they move among indoor and outdoor MEs and among geographic locations.[20] The estimated pollutant concentrations in each ME visited are combined into a time-weighted average concentration, which is assigned to members of the demographic group (ICF, 2007). The model uses four main sources of information to calculate exposure: population data from the 2000 US Census; population activity data from the Consolidated Human Activity Database (CHAD) (Glen et al., 1997); commuting data from the 2000 Census; air quality data from AERMOD; and data on concentrations levels in MEs versus ambient levels. As part of the ME evaluation, algorithms accounting for the gradient in concentrations of primary mobile source air toxics within 200 meters of major roadways are used, which is an addition since the previous version of HAPEM (Version 5).

The HAPEM6 output from the runs performed for this study consisted of average annual exposure for an individual at the census tract level in each of six demographic groups. The demographic groups were determined by age (0-1; 2-4; 5-15; 16-17; 18-64; and ≥65 years). Contributions to ambient concentrations were calculated for the following source sectors: point ("major" in Appendix B), non-point ("area and other" in Appendix B), on-road, non-road, and background (USEPA, 2007a). Concentrations were provided for the 1st, 5th, 10th, 25th, 75th, 90th, 95th, and 99th percentiles, average, and median concentration for each source category, age group, and census tract in each of the target years for this study (1990, 2000, 2010, 2020).

2.4 HUMAN HEALTH EFFECTS ESTIMATION

This section presents our approach for estimating avoided adverse health effects in humans resulting from reductions in exposures to benzene in ambient air and in various MEs in the Houston area. We first review the epidemiological evidence evaluating potential health effects of benzene exposure and present the health endpoints included in

[19] EPA's Air Toxics Archive (http://vista.cira.colostate.edu/atda) contains multiple years of monitor observations for multiple HAPs across the U.S. The Archive contains a program that performs quality assurance on daily monitor observations and calculates an annual average concentration for each valid monitor.

[20] The model includes a total of 14 MEs, such as residential, school, office, public transit, and service station.

the human heath effects estimation. Based on the available evidence, we have focused our evaluation on the epidemiological evidence examining the link between benzene and leukemia. We next describe our selection of dose-response model for our analysis and review the exposure modeling conducted for the study population. We then describe our leukemia risk model, which employs a life-table approach to risk analysis. We close the section by describing our approach for estimating non-cancer health effects and describe ancillary illustrative analyses of high-exposure subpopulations, including residents living in high exposure census tracts, residents living near roadways, and residents with attached garages.

2.4.1 SELECTION OF HEALTH ENDPOINTS

Benzene is a very well studied chemical with a substantial database of epidemiological data associating it with leukemia. There is also limited evidence supporting a link between benzene and other health effects, such as other cancers and non-cancerous effects. IEc conducted an extensive literature review of the heath effects of benzene exposure to identify health endpoints for which the benefits of benzene reductions could be estimated. Note that this literature review was completed in early 2005. Therefore, our results do not reflect the findings of additional studies completed since that date. This section describes the health endpoints selected for the human health effects analysis as a result of that review and our rationale for including them. Additional details may be found in Appendix C.

CANCER

Leukemia

We selected leukemia as the primary health endpoint for our health benefits analysis. Significantly increased risks of leukemia have been consistently reported in benzene-exposed workers of various industries, leading EPA to classify inhaled benzene as a "known/likely" human carcinogen under its 2005 cancer guidelines (USEPA, 2005b). In the EPA document *Carcinogenic Effects of Benzene: An Update* (USEPA, 1998), it states "[e]pidemiologic studies and case studies provide clear evidence of a causal association between exposure to benzene and leukemia" (page 4).

Two groups of benzene-exposed workers have been extensively studied and peer-reviewed. The first consists of a group of 1,717 white male workers employed between 1940 and 1972 in Pliofilm manufacturing plants located in Ohio (hereafter, the "Pliofilm Cohort").[21] The second consists of nearly 75,000 workers in a variety of industries in China employed between 1972 and 1987 (hereafter, the "Chinese Worker Cohort"). Results from retrospective analyses of these workers indicate an association between exposure to a range of benzene concentrations and an elevated risk of leukemia (all types). Recent analyses comparing exposed workers to unexposed workers in the

[21] Pliofilm is a glossy membrane made from rubber hydrochloride and used chiefly for water-resistant materials and packaging (Crump, 1994).

Chinese Worker Cohort show that exposed workers were roughly two and a half times more likely to develop leukemia than the unexposed workers (Yin et al., 1996, Hayes et al., 1997). Pliofilm Cohort analyses have found similar results comparing the observed cases of leukemia in the cohort to an expected number of cases based on US sex- and age-specific rates (Crump 1994, 1996; Rinsky, 2002). Appendix C provides information on other recently published epidemiologic studies that have found an overall increase in risk of leukemia (all types) with exposure to benzene, or a trend of increasing relative risks (RRs) with increased exposure to benzene (Ireland et al., 1997; Costantini et al., 2003; Adegoke et al., 2003; Sorahan et al., 2005; Guenel et al., 2002; Bloemen et al., 2004; Glass et al., 2003; Collins et al., 2003).

There are four subtypes of leukemia: Acute Myelogenous Leukemia (AML), Acute Lymphocytic Leukemia (ALL), Chronic Myelogenous Leukemia (CML), and Chronic Lymphocytic Leukemia (CLL). The strength of evidence supporting a link between benzene and specific types of leukemia varies. AML has the most evidentiary support for a link with benzene exposures, including associations found in both of the major cohort studies.[22] However, other recent studies identified through the literature search have only found non-significantly elevated risks of AML with benzene exposure or suffer from methodological limitations, such as small numbers of cases or possible exposure misclassification, making the results difficult to interpret.

Based on evidence gathered by EPA in the Integrated Risk Information System (IRIS) support document for benzene carcinogenicity as well as the results of the literature review on the health effects of benzene exposure performed by IEc, we chose to quantify the avoided cases of leukemia due to changes in benzene exposure through a dose-response analysis. We decided to use the outcome of all leukemias for the primary estimate, since this endpoint is the most data rich, compared to the limited evidence for a link with benzene and the specific leukemia types (i.e., AML, ALL, CML and CLL). However, because AML was the subtype with the most evidentiary support, we performed a sensitivity analysis to estimate the number of avoided cases of AML.

Other Cancers

In addition to leukemia, benzene exposure has been associated with other cancerous health endpoints in epidemiologic studies, such as Hodgkin's Lymphoma (HL) and non-Hodgkin's Lymphoma (NHL) (Hayes et al., 1997), multiple myeloma (Rinsky et al., 1987 & 2002; Wong et al., 1995), and myeodysplastic syndrome (MDS) (Hayes et al., 1997). However, data on these endpoints are inconsistent and do not yet support a quantitative evaluation of avoided cases due to benzene exposure.

[22] The Chinese Worker Cohort found an elevated RR of acute non-lymphocytic leukemia (ANLL) incidence of 3.0 (95% CI: 1.0, 8.9) and 3.1 (95% CI: 1.2, 10.7) (Hayes et al., 1997; Yin et al., 1996) and the Pliofilm Cohort identified a RR of AML deaths of 5.03 (95% CI: 1.84, 10.97) (Wong, 1995).

NON-CANCER

Benzene exposure at high concentrations has been associated with various hematological abnormalities, including aplastic anemia; however, these adverse non-cancer health effects are unlikely to occur at levels expected to be found in ambient air (less than 10 $\mu g/m^3$, based on EPA's NATA study).

EPA developed a chronic reference concentration (RfC) of 0.03 mg/m^3, based on decreases in lymphocytes (a type of white blood cell) reported in a cross-sectional study of a subset of the Chinese Worker Cohort (Rothman et al. 1996a).[23] This study found blood cell effects at exposure concentrations of about 8 parts per million (ppm).[24] The IRIS profile states that decreased lymphocyte count is a biomarker of exposure and is also thought to have a potential role as a "sentinel" effect (i.e., an early sign of toxicity in the bone marrow), but the effect itself is of uncertain clinical significance to the average population (USEPA, 2007b). The significance of the effect depends both on the magnitude of the decrease in lymphocytes and an individual's baseline lymphocyte level.[25]

2.4.2 DOSE-RESPONSE EVALUATION

The following section describes our evaluation of the existing epidemiologic evidence examining the link between benzene and leukemia and how that informed our selection of a leukemia dose-response function for our health benefits model. Specifically, it describes the major cohort studies, the shape of the dose-response relationship, and the cessation lag, which is the estimate of how quickly cancer risks in a population will decline to a new steady-state level following a reduction in exposure.

Choice of Epidemiologic Data

EPA's IRIS identifies the Pliofilm cohort results as the best available data for dose-response evaluation (Rinsky et al., 1981, 1987). IRIS reports a range of inhalation unit risk (IUR) estimates for benzene-induced leukemia (2.2×10^{-6} to 7.8×10^{-6} per $\mu g/m^3$ benzene in air; USEPA, 1998) based on a reanalysis of the Pliofilm Cohort data by Crump (1994).[26]

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[23] An RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (USEPA, 2007c).

[24] More recent epidemiological and animal studies have found decreased lymphocyte counts at lower exposure levels (Turtletaub and Mani, 2003; Lan et al., 2004; Qu et al., 2002).

[25] For example, the effect of reduced lymphocytes might be more significant for individuals whose immune systems were compromised (e.g., those suffering from HIV/AIDS).

[26] An IUR represents the excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air. While these are typically upper bound estimates, the range of IUR estimates reported for benzene are best statistical estimates derived from observable dose responses using a linear extrapolation model to estimate low environmental exposure risks (USEPA, 2007c).

Strengths of this cohort study include the lack of confounding by other chemicals since workers were exposed primarily to benzene and it is likely that increased risks found in these analyses were due to benzene exclusively; the exposure experienced by this cohort has a wide range, consisting of both high and low exposures; and two sets of exposure estimates were used, Crump and Allen (1984), and Paustenbach et al. (1992), providing a range of estimates. However, the Pliofilm cohort has relatively small number of leukemia cases (14) and some uncertainty in the exposure estimates because there is limited monitoring data in the early years of the study (before 1946).

After reviewing the analytical plan for this case study presented by IEc in 2003, the Science Advisory Board (SAB) Health Effects Subcommittee (HES) recommended that EPA take a closer look at the Chinese Worker Cohort analysis as well as other available data as a possible replacement for the risk estimates of Crump, since this study includes a small number of cases of leukemia in its analysis (USEPA, 2004e).

We found the Chinese Worker Cohort to have a much larger sample size and number of cases (47) than the Pliofilm Cohort. In addition, researchers of this cohort found positive associations between benzene and leukemia at levels closer to ambient (e.g., for workers with <10 ppm average exposure). However, limitations of this study include possible confounding by occupational exposures to chemicals other than benzene and potential problems with exposure assessment, in that only 38 percent of exposures were based on actual measurements (Dosemeci et al., 1994).

In addition to the Pliofilm and Chinese Worker Cohort studies, we examined a number of cohort and case-control studies linking benzene and leukemia, including two studies of petroleum workers known to have low exposures specifically mentioned by the SAB HES in their recommendations (Rushton and Romanieuk (1997) and Schnatter (1996)). We found that these studies suffer from a variety of methodological weaknesses, such as small cohort size, insufficient exposure assessment, and potential confounding of other exposures that limit the usefulness of these studies for our analysis.

For the purposes of our analysis, we ultimately chose to use dose-response slope factors reported by Crump (1994) for our primary estimate of avoided leukemias because the IRIS profile for benzene currently supports the use of data from the Pliofilm cohort for calculating potency estimates. However, despite its limitations, the Chinese Worker Cohort data has certain advantages over the Pliofilm Cohort, such as large sample size and benzene exposure levels that are more consistent with ambient exposures. Therefore, we performed a sensitivity analysis using the results of the Chinese Worker Cohort.

Shape of the Dose-Response Relationship

The shape of the dose-response function for leukemia and benzene is uncertain, with different studies suggesting one or more possible functional forms in the observable range (e.g., linear, supralinear). This makes extrapolating the dose-response function to low levels, such as those found in this study, uncertain as well. Linear models in the observable range were found to be the best fit in the Crump (1994) analysis of the

Pliofilm Cohort.[27,28] The author concluded that "[t]here was no indication of either [cumulative exposure]-dependent or intensity-dependent nonlinearity in the dose responses for any model based on the Crump and Allen exposure matrix" (Crump, 1994, page 234).[29] We also found evidence supporting a supralinear dose-response relationship between observed benzene concentrations and leukemia. For example, an analysis of the Chinese Worker Cohort found that effect estimates tended to plateau at higher levels of benzene (Hayes et al., 1997).[30]

In addition, conflicting information exists regarding the possibility of a threshold in the dose-response function. In our literature search, we found some evidence of a potential threshold in that statistically significant increases in leukemia are not seen at lower exposures levels in the Pliofilm Cohort studies. However, these analyses are uncertain due to minimal statistical power at low benzene levels (see Appendix C for more information).

We chose to use a linear model throughout the range of exposure concentrations in our analysis for several reasons.[31] First, we did not find current evidence on potential thresholds for benzene-induced leukemia to be persuasive. Furthermore, the best fitting models from our chosen epidemiological dataset, the Pliofilm Cohort, were linear in the observable range. Finally, EPA (1998) concludes that "[t]oo many questions remain about the mode of action for benzene-induced leukemia for the shape of the dose-response function to be known with certainty" (page 34). According to EPA's *Guidelines for Carcinogen Risk Assessment*, linear extrapolation to low doses should be used when there is insufficient data to establish a mode of action (MOA) as a default approach because linear extrapolation "generally is considered to be a health-protective approach" (USEPA, 2005b, page 3-21).

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[27] Specifically, linear multiplicative risk models, where the leukemia mortality rate is proportional to both the change in exposure and the baseline rate of dying from leukemia, were the best fit.

[28] Crump (1994) did not investigate supralinear models; the linear model was the best fit when compared to sublinear models.

[29] We explored the possibility of performing a sensitivity analysis using an intensity-dependent quadratic function reported in the Crump (1994) analysis. In this case, the intensity of the exposure was given greater weight than the duration of exposure. Only borderline significant results were found for this model, using the Paustenbach exposure estimates. Ultimately, we decided not to include this sensitivity analysis because it would have required substantial revisions to the life-table model's exposure processing routing, which we felt were not justified, given the borderline significance of fit of this model.

[30] Additional Chinese Worker Cohort analyses found that benzene metabolite levels plateau at higher benzene exposures, potentially suggesting the existence of an enzyme-mediated process for benzene toxicity that could involve saturation of the enzyme at higher doses (Rothman et al., 1996b & 1997).

[31] We selected a linear dose-response relationship assumption for the observable range as well as for extrapolation to low doses.

Cessation Lag

The term "cessation lag" refers to the estimate of how quickly cancer risks in a population will decline to a new steady-state level following a reduction in exposure.[32] In the original analytical plan, we proposed to use a five-year "cessation lag" for benzene-induced leukemias. The SAB HES, in their review of the report, suggested that we re-examine whether our lag approach was consistent with the epidemiologic literature on this subject.

The ideal data for modeling cessation lag would come from studies that follow the pattern of changes in risk in a study population over time following an exposure reduction. Where such data are limited or unavailable, information on the distribution of latency in a population can be useful for bounding potential cessation lag periods, because it indicates a period of time over which latent cases of disease at the time of the exposure change may continue to be diagnosed, while the population risk moves to a new steady-state level. During our review, we found only one study that explicitly modeled the cessation lag concept, using an analysis stratified on time since last exposure (Silver et al., 2002). This study found that exposures five to ten years prior to the cessation of exposure have the maximum impact on risk, and that exposures between ten and 15 years prior to cutoff may also contribute to a lesser degree. All of the other studies we reviewed included an estimate of latency in their models (i.e., the delay between the critical exposure and manifestation of disease or death). While not the same as the cessation lag, information about latency can also help inform our estimate for a cessation lag. Of the studies examining latency, most found that latency periods of 10 years or less were the best fit for the data. A few found latency periods as long as 15 years.

Rather than incorporating a cessation lag into the benefits as a post-processing step, as EPA has done with other pollutants, such as fine particles ($PM_{2.5}$), we instead chose to select a dose-response slope factor from the Crump analysis that directly incorporates assumptions about the differential impacts of past exposures on current risks. See the section entitled "Incorporating Weighted Exposure" in Section 2.4.3 for more information.

2.4.3 RISK MODEL

Overview of the Model

The purpose of the risk model is to calculate the expected number of fatal and non-fatal cases of benzene-induced leukemia avoided as a result of the implementation of the 1990 CAAA regulations affecting benzene emissions in three counties in the Houston area (Brazoria, Galveston, and Harris). The approach used to estimate these benefits is based on the model used to estimate risks due to radon exposure in the National Research Council's (NRCs) Biological Effects of Ionizing Radiation (BEIR) IV report (1988). The

[32] See *Arsenic Rule Benefits Analysis: An SAB Review* Science Advisory Board. EPA-SAB-EC-01-008, August 30, 2001 (USEPA, 2001a) for more information about the concept of cessation lag.

approach consists of a life-table analysis that calculates the probability of contracting (or dying from) leukemia for a given age cohort in a given time period, conditional on the probability of surviving to that period. Figure 2 provides an overview of the life-table model, including the inputs and calculations.

The model first takes the difference between the *With-CAAA* and *Without-CAAA* benzene exposure estimates in each study year to calculate the CAAA-related exposure changes in each year. It calculates these changes for each census tract in the study area and for every five-year age group (e.g., 65 to 69 year olds) residing in each census tract. The model then takes these data to construct a cumulative exposure history for each age group in each census tract, reflecting a total impact of benzene changes, both current and past. Thus, for example, in 2020, the model would calculate an exposure history based on exposure changes experienced from 2020 all the way back to 1990 (or birth, whichever was more recent). Because studies of benzene-exposed workers suggest that exposures may have different effects on leukemia risk depending on when they occur, the model incorporates this information when calculating a cumulative exposure value. For example, in our main model, exposures occurring roughly five years in the past are the most influential for developing leukemia in the current year. Therefore, exposures in that year are given the most weight and exposures occurring before or after that year are given less.

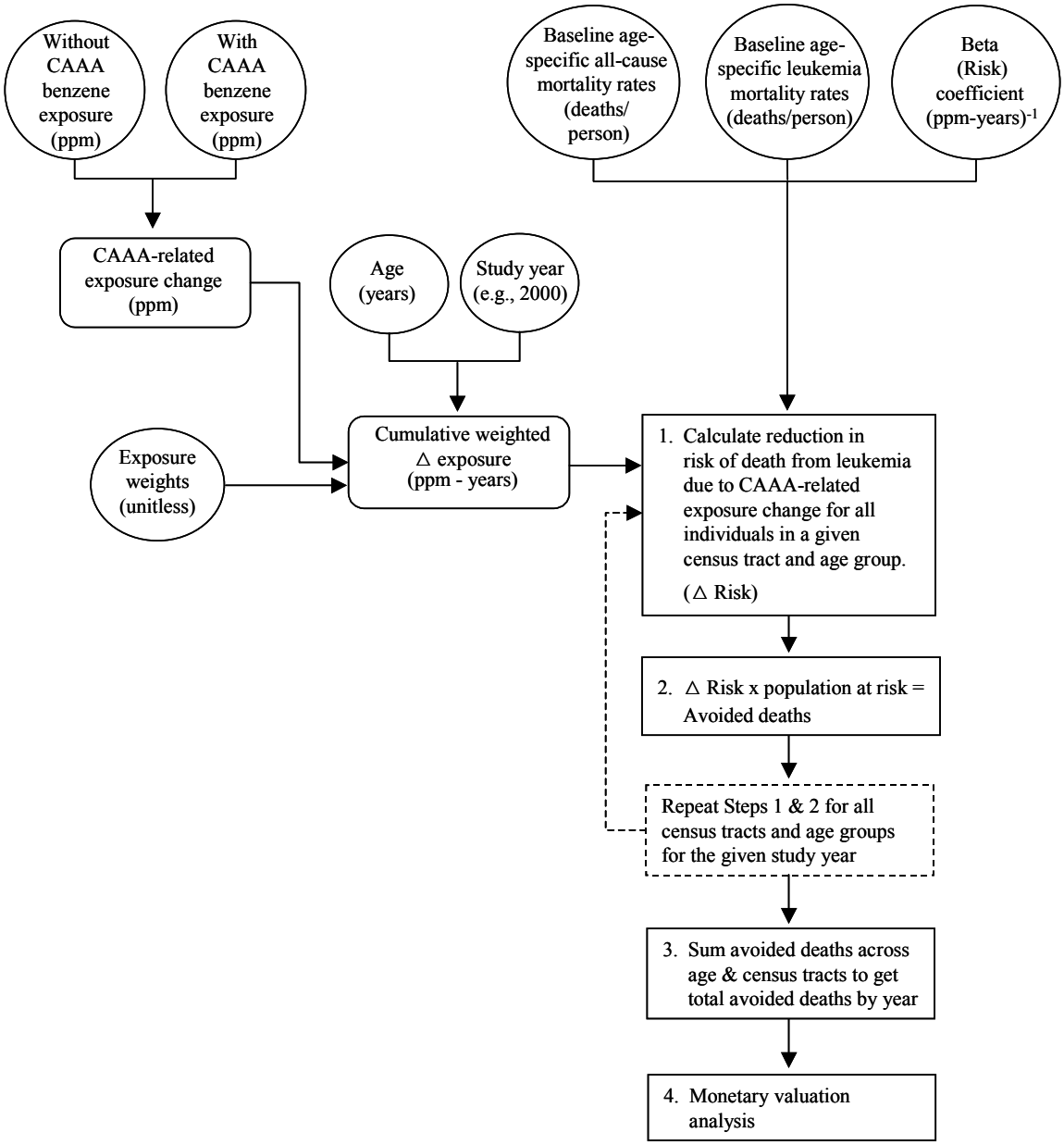
For each census tract, the model uses this cumulative weighted exposure estimate to determine changes in each age group's risk of dying from leukemia in the current period. This size of this effect depends on the size of the cumulative change in benzene exposure; the size of the mortality risks (both all-cause and leukemia-specific) faced by each age group in the baseline; and the assumed relationship (derived from studies of worker exposures) between changes in benzene exposure and changes in leukemia risk. (See equations in Appendix D for further detail). Changes in risk for each age group are then multiplied by the population in that age range in that census tract in that year to calculate the number of avoided leukemia deaths expected to occur among that group in the current year. The model then repeats this process until avoided leukemia deaths are calculated for each five-year age group, census tract, and study year combination. Once completed, the model sums across all age groups and census tracts to produce estimated reductions in avoided leukemia deaths in each county in each study year, and also sums avoided deaths across the whole study area and study period. The final step in the model is to apply an economic value to the avoided leukemia deaths, which is discussed in Section 2.5.

The life-table approach allowed us to estimate benefits to age-specific cohorts, taking into account age-specific mortality rates, both for all-causes and leukemia. This approach also allowed us to explicitly integrate an expected latency period into our model. That is, exposures that were expected to be responsible for initiating the development of leukemia were weighted more heavily and exposures occurring after initiation were weighted less.[33] This approach allows us to estimate a delay in the realization of benefits, but it is

[33] This process assumes that once the benzene-induced cancer has been initiated, the time from that occurrence until the resulting mortality is benzene-independent (Crump, 1994).

not necessarily the same as the "cessation lag" effect previously cited by the SAB (USEPA, 2001a). The "cessation lag" refers to the estimate of how fast cancer risks in a population will decline to a new steady-state level following a reduction in exposure. The latency represents the period before any benefits begin to be observed. However, this may provide a reasonable approximation of the cessation lag. See the section entitled "Incorporating Weighted Exposure" below for more information.

FIGURE 2: LIFE-TABLE MODEL OVERVIEW



Note: This flowchart assumes the model is being run with leukemia mortality data. The model can also be run with leukemia incidence data. The difference between the model results for these two runs represents an estimate of avoided non-fatal cases of leukemia.

We calculated a partial lifetime risk of dying from leukemia, focusing on the study period. We estimated the risk change due to the difference in exposure between the *With-* and *Without-CAAA* exposure scenarios for five-year age cohorts at the census tract level.[34] The basic risk equation we used for calculating the partial lifetime probability of dying from leukemia (R) is below. (See Appendix D for a more in depth description of the risk model, including more detailed exposure and risk equations.)

$$R = h/h^* \times S \times (1-q)$$

Where:	R	=	risk of dying from leukemia in the current year, given survival up to that year;
	h	=	leukemia mortality rate;
	h*	=	all-cause mortality rate;
	S	=	probability of surviving up to the current year;
	q	=	probability of surviving through the current year; and
	1-q	=	probability of dying during the current year.

We then multiplied these partial lifetime probabilities of leukemia by the population of the specific age cohort in that census tract in that year to estimate the number of avoided cases.

Survival rates for leukemia have improved since the time of the Pliofilm cohort, suggesting that an increased percentage of leukemia incidence in the study period (1990-2020) will be non-fatal. Non-fatal leukemia cases represent a separate health endpoint in our benefits analysis. Therefore, we ran the risk model using both leukemia mortality and incidence rates with the same dose-response slope factor. The difference between these results represents our estimate of avoided non-fatal cases of leukemia.[35]

Model Inputs

This section describes the various sources of data that were used in the model. Because the model required large amounts of data, we used a Microsoft Access™ database to perform all calculations. Each of these datasets were constructed in Microsoft Excel™ spreadsheets and uploaded in the Access database. In some cases, we manipulated the original data so that it would be consistent with the parameters of our model. For instance, for mortality and incidence rates, if age cohorts reported in the original data differed from those in our model, we calculated weighted average rates for the model age cohorts, using population data from the same year or years as the rate data.

[34] The age cohorts started at 0-4 and ended with 95-99.

[35] Note that we compared the resulting split between fatal and non-fatal cases of leukemia against 10-year survival rates for 1988-2004 from the from the Surveillance Epidemiology and End Results (SEER) website to ensure that our methodology was reasonable (http://seer.cancer.gov/). See Section 3.3.1 for further information.

Population Data

For study years between 1990 and 2000, we used population data from the 2000 US Census.[36] For the remaining study years, we used Woods and Poole population projections (2001), consistent with the main 812 analysis. We extracted the relevant population projection data from EPA's BenMAP model at the census tract level by single year of age. We then aggregated the data to match the five-year age intervals in our life-table model.

Health Data

We acquired county level all-cause background mortality rates from the Texas Department of State Health Services, Center for Health Statistics.[37] We used data from the year 1990, which was the base year of the study period. We procured background leukemia mortality and incidence data from the Texas Department of State Health Services, Cancer Epidemiology and Surveillance Branch, Texas Cancer Registry.[38] These were both county-level rates and were only available as an average over several years (1990-2003 for mortality and 1999-2003 for incidence), due to small numbers of cases in each county.[39]

Exposure Data

The HAPEM6 median concentration representing "total" exposure was used for each age group in each census tract as the exposure values for our primary estimate.[40] However, we first adjusted the HAPEM6 output so that it would be consistent with the structure of our life-table model. For instance, our model assessed risk for 20 five-year age cohorts (e.g., 0-4, 5-9, 10-14), whereas the HAPEM6 output contained only six age groups of differing lengths. Therefore, in order to convert the HAPEM6 data to a format consistent with our model, we did one of the following: 1) if a given five-year age cohort was entirely covered by a HAPEM6 age group, we assigned that cohort the exposure concentration for that HAPEM6 age group; or 2) if the five-year age cohort spanned more than one HAPEM6 age group, we calculated a weighted average exposure concentration, based on the number of years spent in each of the HAPEM6 age groups.[41] In addition, HAPEM6 results were only available for the base year (1990) and target years (2000,

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[36] http://www.census.gov/.

[37] http://www.dshs.state.tx.us/tcr/default.shtm.

[38] Data supplied by Dr. David Risser of the Texas Department of State Health Services.

[39] Since leukemia is a relatively rare disease, in order to get reliable estimates, it is necessary to average over several years of incidence data. It is possible that the later years included migrants, which could introduce uncertainty in the estimates (if these individuals had been exposed to different benzene levels than other residents).

[40] Total exposure consists of a sum of the ambient air concentrations due to the four source sectors (point, non-point, on-road, non-road, and background).

[41] For example, HAPEM6 estimated exposure for 0-1 year olds and 2-4 year olds separately. To calculate exposure for the 0-4 age group in our risk model, we took a weighted average of the two HAPEM6 exposure concentrations, weighting the first concentration with a value of 2 years, the second concentration with a value of 3 years.

2010, 2020). In order to estimate exposure for each year, we linearly interpolated the exposure concentrations between target years. Because our model calculated risk at five-year intervals, we then took an average over the previous five years (e.g., we averaged the concentrations for 1991-1995 for the 1995 concentration).

In order to be consistent with the epidemiological data used in the life-table model, we then converted the output from $\mu g/m^3$ to ppm.[42] In addition, the dose-response slope factor from Crump (1994) is based on occupational exposures. Therefore, we multiplied the output concentration by a conversion factor so that it would be consistent with a typical occupational exposure scenario.[43]

To reduce model computations, we subtracted the exposure concentrations for the *With-CAAA* scenario from the *Without-CAAA* scenario to obtain a "delta exposure" value representing the change in exposure due to the CAAA for each year, age cohort, and census tract. These delta exposure values were then used in the risk calculations, rather than calculating partial lifetime risk of leukemia for each of the two scenarios separately and then subtracting to obtain the difference in risk.

Incorporating Weighted Exposure

EPA's SAB has defined "cessation lag" as the period it takes for risk to decline to a steady state level following a reduction in exposure (USEPA, 2001a). As described in Section 2.4.2, we identified only one epidemiological study specifically estimating the length of the cessation lag. Therefore, in order to develop a temporal stream of benefits, we relied on data that attempts to characterize the latency period (the time between a critical exposure and the development of symptomatic disease or death). We used these data to create a cumulative exposure value in each year for each age group/census tract combination that reflects the differential weighting of past exposures based on their expected importance for developing leukemia, as described below.

Crump (1994) evaluated benzene risk using several models based on data from the Pliofilm cohort. The dose-response models used in the analysis required that a person's prior exposure to benzene be condensed to a single summary measure (Crump, 1994). The author considered two exposure metrics to calculate this single exposure estimate. The first method, "cumulative exposure," employed a "lag," L, and assigned a weight of zero to the last L years of an individual's exposure, assuming that exposures during the most recent L years do not affect mortality rate. The second method, "weighted

[42] In order to convert benzene concentrations from $\mu g/m^3$ to ppm, we first converted $\mu g/m^3$ to mg/m^3 by multiplying by 1×10^{-3}. We then multiplied the concentration by 24.45 (a constant) and divided by 78.11 g (the molecular weight of benzene).

[43] The conversion factor consisted of the following: (7 days/week / 5 days/week) × (24 hr/day / 8 hr/day) × (0.833 m^3/hr / 1.25 m^3/hr). The value of 0.833 m^3/hr is an average breathing rate for the general population. Subjects in the occupational studies on which the risk estimates are based are assumed to exhibit a faster breathing rate of 1.25 m^3/hr during an eight-hour work day.

exposure," utilized a weighting function that increases from zero to a maximum for exposures that occur K years in the past, if K represents the best estimate of latency.[44]

A cumulative exposure value can then be calculated for each age cohort in each year in each census tract by weighting previous exposures based on one of the two methods described above and then summing them.

For the purposes of our life-table model, we chose to use the dose-response slope factor from the linear multiplicative model for all leukemia that incorporated the "weighted exposure" method from the Crump analysis. The value of K for this model was 5.3 years. We selected this model because the latency estimate was consistent with the literature, most of which reported latency estimates between 5 and 10 years. In addition, unlike the "cumulative exposure" model, which applies equal weight to all exposures that occur before the latency estimate, the "weighted exposure" model applies lower weights to exposures far in the past, which is more consistent with the literature, where no studies found latency to be greater than 15 years. Because of the uncertainty in the true latency period for benzene-induced leukemia, we performed sensitivity analyses using the "cumulative exposure" model with a five-year lag and a zero-year lag.

Model Output

The model output consisted of the number of deaths from leukemia that were avoided due to the presence of the CAAA for each age cohort in each census tract over a five-year period. We first divided the estimate of avoided deaths by five to obtain an annual avoided deaths value for each year in the study period. We also summed the avoided deaths across all age groups in all census tracts, resulting in an overall cumulative sum of avoided deaths for each county for the entire study period. In addition, we estimated the number of cases expected to occur after the end of the study period that are due to CAAA-related benzene changes within the study period. See the "Expected Total Benefits" subsection of Section 3.3.1 for further information.

2.4.4 ADDRESSING HIGH-EXPOSURE SUBPOPULATIONS

The life-table model used in this case study used median benzene concentrations to estimate avoided cases of leukemia. Therefore, to provide a more complete illustration of the effects of reducing benzene exposures to populations in the Houston area, we performed supplemental calculations of risk reductions to three high-end exposure groups: residents living in census tracts with the highest benzene exposures, residents living near major roadways, and residents with attached garages.

Residents Living In Census Tracts With High Exposure

As part of our assessment of highly exposed subpopulations, we examined CAAA-related reductions in the risk of leukemia for individuals living in census tracts with the highest

[44] The weighting function took on the following form: $w(t) = (t/K^2) \exp(-t/K)$. Where: t = the number of years prior to the current year; and K = number of years prior to the current year when the weight reaches its maximum (this also represents the latency estimate).

levels of benzene. We first selected the two tracts in each of the three counties included in this case study with the highest exposure concentrations from HAPEM6 under the *Without-CAAA* scenario in 2020. These tracts also exhibited the highest changes in exposure between the *With-* and *Without-CAAA* scenarios. We then calculated an estimate of individual lifetime risk of leukemia in each of the six tracts under both the *With-* and *Without-CAAA* scenarios, assuming continuous lifetime exposure to median 2020 levels, using the following equation:

$$\text{Individual Lifetime Risk of Leukemia} = EC \times IUR$$

Where: EC = median 2020 exposure concentration from HAPEM6 ($\mu g/m^3$); and

IUR = benzene inhalation unit risk estimate from IRIS $(\mu g/m^3)^{-1}$.[45]

We then subtracted the individual lifetime risks of leukemia under the *Without-CAAA* scenario from the *With-CAAA* scenario to estimate the CAAA-related risk reduction for each of the six tracts.

Note that this assessment of risk is different than the method employed by our main life table model and therefore, the results are not directly comparable. In the calculations above, we are assessing lifetime risk of leukemia, assuming constant lifetime exposure of an individual born today to median 2020 levels of benzene exposure under the *With-CAAA* and *Without-CAAA* scenarios. The life table model calculates partial lifetime risks of leukemia retrospectively over the study period from estimates of cumulative weighted previous exposures.

Residents Living Near Roadways

Another highly exposed subpopulation in the study area includes individuals living in close proximity to major roadways, such as the interstate loops in Harris County that surround downtown Houston. A substantial number of studies have demonstrated increased concentrations of benzene and other mobile source related HAPs near roadways. For example, Kwon (2005) analyzed ambient VOC measurements in Elizabeth, New Jersey from the Relationship among Indoor, Outdoor, and Personal Air (RIOPA) study and found that ambient concentrations of benzene, toluene, ethylbenzene, and xylenes measured near homes within 200 m of roadways are 1.5 to 4 times higher than urban background levels. Several other studies have found that concentrations of benzene and other mobile source air toxics are significantly elevated near busy roads compared to "urban background" concentrations measured at a fixed site (e.g., Skov et al., 2001; Jo et al., 2003; Fischer et al., 2000; Ilgen et al., 2001; and Sapkota et al., 2003).

Version 6 of the HAPEM model, which was applied in this study, includes algorithms that account for the gradient in concentrations of primary (directly emitted) mobile source air toxics within 200 meters of major roadways (ICF, 2007). HAPEM6 adjusts ambient concentrations generated by AERMOD for each census tract using concentration

[45] Note that the IRIS profile for benzene presents a range of values for the IUR ($2.2 \times 10^{-6} - 7.8 \times 10^{-6}$). We calculated values using both ends of the range.

gradients developed with the CALPUFF dispersion model (Cohen et al., 2005). For locations within 75 meters and from 75 to 200 meters from major roads, HAPEM6 adjusts ambient concentrations upward, while concentrations at locations further from major roadways are adjusted downward. These adjustments are consistent with results from prior modeling studies that explicitly accounted for concentration gradients around major roads within census tracts (Cohen et al., 2005; Stein et al., 2007). HAPEM6 then applies the adjusted concentrations in its microenvironmental concentration calculations.

To assess the impact of HAPEM's near-roadway algorithm on our primary results and on exposures to highly exposed individuals, we conducted an additional HAPEM run for 2020, turning off the near-roadway feature. We then compared the difference in the annual average benzene concentration in 2020 between the *With*- and *Without-CAAA* scenarios for these two HAPEM runs.

Residents With Attached Garages

We also performed supplemental calculations of risk reductions to residents living in homes with attached garages. Studies of benzene levels in homes with attached garages suggest that residents in these homes may be exposed to higher indoor benzene concentrations than residents in other types of homes (Gordon et al., 1999; Schlapia and Morris, 1998). While the population living in homes with attached garages may benefit from reductions in benzene emissions that occur in-garage, we were unable to identify data on local benzene concentrations in attached garages in the Houston area with which to estimate those benefits. Therefore, we performed an illustrative, back-of-the-envelope calculation to assess the rough magnitude of additional potential benefits that may result from CAAA-related reductions of in-garage benzene emissions in 2020. Appendix E contains a detailed description of the calculations we performed, including the equations used. We provide a brief overview of the process below.

Our approach involved the following three steps:

1) We assessed the CAAA-related percent reduction in total emissions in the non-road and on-road categories that are expected to occur within attached garages in 2020. The percent reduction was based on the difference in the in-garage emissions between the *With*- and *Without-CAAA* scenarios in 2020. We used slightly different approaches for determining the non-road and on-road portions of the total emissions under the *With*- and *Without-CAAA* scenarios, due to the available data for each of these categories (See Appendix E for more information);

2) We applied the percent reduction in emissions to an estimate of average benzene exposure attributable to attached garages based on previous U.S. studies;[46] and

[46] We used an estimate of average indoor benzene exposure attributable to attached garages from Appendix 3A of the MSAT Regulatory Impact Analysis (RIA) (USEPA, 2007d). We selected a value that excluded studies conducted in Alaska, due to a number of differences expected in attached garage-related exposures between Alaska and Houston (see Appendix E for additional information).

3) We calculated the annual number of avoided cases of leukemia in the Houston area in 2020 that would be expected based on the CAAA-related reduction in attached garage-related exposures using the value calculated in step 2, the benzene IUR from IRIS, and an estimate of the size of the affected population.

2.4.5 ESTIMATING NON-CANCER HEALTH EFFECTS

We considered extrapolating the dose-response function based on the data supporting the RfC in order to estimate "cases" of reduced lymphocyte counts expected at environmental exposure levels. However, the data set supporting the RfC is limited (2 data points) and does not support an extrapolation beyond the benchmark concentration down to ambient levels. We identified in our 2005 literature search two additional studies linking reduced lymphocyte count to occupational benzene exposure, both of which had lower exposure concentrations (below 1 ppm) and larger number of data points (3 and 4). Ultimately, extrapolating these studies to low doses proved to be too time and resource intensive for this case study. Therefore, we assessed this endpoint using the approach outlined in the original analytic plan (IEc, 2003), reporting the difference in the number of individuals experiencing benzene concentrations above the RfC under the *With-CAAA* and *Without-CAAA* scenarios.

2.5 BENEFIT VALUATION

This section describes our approach to assigning economic value to the estimated benefits of reductions in ambient benzene concentrations. The scope of the valuation methodology is determined by the prior steps in the case study, which necessarily limits monetization to those health effects for which dose-response functions are available. Therefore, other benefits of reductions in benzene likely exist and have value (e.g., non-cancer health effects, cancers other than leukemia), but we were unable to quantify them in the framework of this case study.

2.5.1 OVERVIEW OF APPROACH

We applied valuation methods that are largely consistent with those employed to value the benefits of the Second 812 Prospective analysis of criteria pollutants (see Chapter 8). That analysis employed a Value of Statistical Life (VSL) estimate to assign economic value to avoided deaths from air pollutants. In the benzene exposure case, however, there is the additional consideration of medical costs associated with the period of cancer illness (the morbidity increment) leading up to death (hereafter, "pre-mortality morbidity"). In addition, we have also valued non-fatal cancer cases, which are not reflected in the criteria pollutant analysis. In order to value these non-fatal cancer cases, we followed recent SAB advice on this topic given during a consultation in 2001 regarding the arsenic in drinking water rule-making by EPA's Office of Water, discussed in more detail below.

2.5.2 VALUATION OF CANCER ENDPOINTS

Fatal Cancers

Value of Statistical Life

Fatal cancers were valued on a per-case basis using a VSL estimate presented in a meta-analysis of several U.S. wage-risk studies by Viscusi and Aldy (2003). We used a mean value of $7.4 million at 1990 income levels (in 2006 dollars).[47] We then applied income elasticity values for premature mortality to account for the projected growth in willingness-to-pay (WTP)-based VSL estimates that is associated with real income growth.[48] This results in an adjusted VSL value for each year subsequent to 1990. The resulting VSL estimate for 2020, for example, was $8.9 million in 2006 dollars.

Pre-Mortality Morbidity

For this analysis, in addition to using VSL to estimate the benefits of avoided cancer deaths, we also provide an estimate of the value of avoided morbidity associated with deaths from cancer. The procedure we apply is consistent with EPA SAB advice delivered as part of prior reviews of both a cancer valuation white paper in 2000 and an economic analysis of more stringent standards for arsenic in drinking water.[49]

To summarize the SAB advice, a special panel of the SAB Environmental Economics Advisory Committee (EEAC), in its review of the EPA Office of Ground Water and Drinking Water's (OGWDW) Arsenic in Drinking Water Rule, endorsed adding estimates of the medical costs of treatment and amelioration for fatal cancers to the VSL as a lower bound on the true (total) value of avoiding fatal cancers (USEPA, 2001a).[50] As a preface to this endorsement, the panel had acknowledged that, as a general recommendation, there was insufficient evidence to support a broad "cancer premium" for the avoidance of fatal cancer risk relative to other types of fatal risk reflected in the VSL typically applied

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[47] This value is from Table 8 of Viscusi and Aldy (2003) and represents the mean predicted VSL for the U.S. sample using Huber Weights (model 5). This estimate was selected because it was the best model for the data, had relatively tight confidence bounds and reduced non-normality in the error term by using Huber weighting. We adjusted the reported value ($6.3 million in 2000 dollars) for inflation to 2006 dollars using the standard inflators reported in BenMAP (USEPA, 2008).

[48] The specific income elasticity values and per-capita income growth estimates combine to yield annual adjustment factors for the growth in WTP over time. The annual adjustment factors were taken from BenMAP (USEPA, 2008a) for all years up to 2024. For years after 2024, we estimated an approximate income adjustment factor growth rate and applied that rate to generate annual adjustment factors through 2030.

[49] See USEPA (2001a). *Arsenic Rule Benefits Analysis: An SAB Review*. Science Advisory Board. EPA-SAB-EC-01-008, August 30, 2001; and USEPA (2000). *An SAB Report on EPA's White Paper Valuing the Benefits of Fatal Cancer Risk Reductions*. Science Advisory Board. EPA-SAB-EEAC-00-013, July 27, 2000.

[50] Note that this specific adjustment has been subsequently applied in several economic analyses supporting final OGWDW rules. It was most recently applied in the *Economic Analysis for the Final Stage 2 Disinfectants and Disinfection Byproducts Rule*, USEPA Office of Water (4606-M), EPA 815-R-05-010, December 2005. See page 6-83 for a brief description of the procedure applied in that RIA, which closely follows the procedure we have used here.

by EPA.[51] Just prior to the issuance of this report, the larger EEAC had also concluded that, while a cancer premium for morbidity, dread, and fear was valid in principle, there was insufficient evidence to apply any specific WTP adjustment to the standard VSL to reflect a cancer premium. This finding was reflected in subsequent SAB review of the benefits of the arsenic rule; that panel, however, did not consider the option of adjusting the VSL to reflect the medical cost of illness for cancers (USEPA, 2000).[52]

A few additional studies since this time have further tested the idea of a cancer premium for VSL. Most recently, Van Houtven, Sullivan, and Dockins (2008), found that WTP to reduce cancer risk with a five year latency period is three times larger than WTP to reduce current automobile-accident risks, although the cancer premium declined as respondents considered cancers with longer latency periods. In addition, Hammitt and Liu (2004) found respondents in a Taiwanese stated preference survey were willing to pay about 30 percent more to reduce their risk of contracting a fatal cancer versus a similar non-cancer illness, though the results were only weakly significant. Finally, Tsuge, Kishimoto, and Takeuchi (2005) identified a small but significant preference for avoiding cancer risks in Japan. While this literature is growing, we believe it is premature at this time to develop risk-attribute-based adjustment factors for VSLs that specifically address WTP to reduce cancer mortality risks, and instead apply only the pre-

[51] The full quote from EPA, 2001a reads as follows, "We believe that the central estimate of $6.1 million for the value of a statistical life (VSL) is appropriate. On the question of whether to add a value for cancer morbidity before death, we do not believe that there is an adequate basis in the literature for doing this. But we can endorse adding estimates of the medical costs of treatment and amelioration for fatal cancers to the VSL as a lower bound on the true value of avoiding fatal cancers" (from page 5-6 in the referenced SAB report).

[52] The full quote from USEPA (2000) reads as follows, "The Committee supports the principle that the morbidity, fear, or dread associated with cancer is a valid component of the cost that individuals attribute to the incidence of cancer. Thus, in principle, the value of reductions in cancer risks should include both the value of the reduced risk of death and the value of reduced risk of the morbidity, fear, and dread that precedes the death incident. To the extent that cancer victims typically suffer greater morbidity, fear, or dread than the victims of the causes of death involved in VSL studies, it would be appropriate to attach a "cancer premium" to the value of an avoided death from cancer. The Committee finds, however, that existing studies provide little reliable information as to the magnitude of this premium, and concludes that until better information becomes available, it is best not to assign such a premium.

The white paper cites studies by Savage (1993) and by Jones-Lee, Hammerton, and Philips (1985) as evidence that people are willing to pay a "cancer premium" to avoid fatal cancers relative to other fatal risks. The paper cites a suggestion from Revesz (1999) that the VSL for an immediate fatality be adjusted by "at least a factor of two" to capture the morbidity, fear, and dread associated with cancer.

The Committee disagrees with this suggestion for two reasons. First, the articles by Savage and Jones-Lee et al. do not measure an individual's willingness-to-pay (WTP) to avoid fatal cancer; hence they cannot be used to justify the proposed adjustment. Jones-Lee et al. ask respondents if they could reduce deaths from one of three causes — motor accidents, heart disease and cancer — by 100 persons annually, which cause would they select. The respondent is then asked how much he or she would pay for this reduction. This question measures WTP to reduce risks to others as well as to oneself, whereas the VSL values private risk reductions. Similarly, the Savage article does not elicit private WTP but asks the respondent to allocate $100 among "commercial airplane accident research," "household fires research," "automobile accident research," and "stomach cancer research." Second, the appropriate way to determine whether a "cancer premium" is required is to value reductions in the risk of a fatal cancer directly. There is only one study (Magat et al. 1996) that has attempted to value reductions in fatal cancer risk directly. For the case of fatal lymphomas it suggests that no cancer premium is warranted. Clearly, further research is called for in this area. The Committee believes that until empirical work clearly establishes the value of this premium, it is best not to attempt to apply one " (from page 5-6 of the referenced SAB report).

mortality morbidity adjustment described above. This approach is consistent with recent advice from the SAB Council provided as part of its review of this case study (USEPA, 2008b).

Based on the available literature and SAB advice, we conclude that the VSL applied to value avoided fatal leukemia risks represents the value of avoiding a premature death, but it does not explicitly take into account the medical costs associated with the period of illness (the morbidity increment) leading up to death. Based on estimates presented in EPA's Cost of Illness Handbook (USEPA 1999b) for a "typical" cancer case, we estimate the medical costs for a fatal leukemia case to be $98,971 at 1996 price levels.[53] This cost can be updated to 2006 price levels using the Consumer Price Index (CPI) for Medical Care (see USGPO 2009); the result is $145,810, which for our purposes we round to $150,000 and apply as a point estimate to each fatal case of leukemia in the benefits model.[54]

Non-Fatal Cancers

To our knowledge, EPA's Office of Air and Radiation (OAR) has not previously developed or published an estimate to value non-fatal cancers. In addition, the overall EPA Guidelines for Economic Analysis provides only general guidance on valuation of non-fatal morbidity; in summary, WTP values are preferable, but cost-of-illness values are also acceptable.

EPA's OGWDW, however, has applied existing valuation estimates to non-fatal cancers. Prior to 2001, valuation of non-fatal cancer in OGWDW economic analyses was based on application of a WTP value for chronic bronchitis, based on the assumption that the severity of a chronic but non-fatal cancer case and a case of chronic bronchitis are roughly similar. That approach was reviewed by the SAB EEAC in 2001. At that time, the SAB recommenced that the chronic bronchitis value be supplemented by a value from

[53] Estimate derived from EPA's Cost of Illness Handbook, Chapter II.1, entitled, "Introduction to the Costs of Cancer." See Table II.1-4 - Incremental Undiscounted Direct Medical Costs for a Typical Cancer, on page II.1-26. The estimates presented in that table were for a typical case with a 50 percent mortality rate. We adjusted the reported value for the component attributed to terminal care to reflect a certain fatal case. The result is an estimate of $98,970.84 in 1996 dollars. Cost of Illness Handbook available on EPA's website at: http://www.epa.gov/oppt/coi/ (downloaded July 2005).

[54] CPI-Medical Care series taken from Table B-60 in USGPO 2009, *Economic Report of the President*. (http://www.gpoaccess.gov/eop/tables09.html).

the one study that values a non-fatal cancer, Magat et al. (1996).[55] The Magat et al. study is a stated preference, health risk tradeoff study that evaluated the marginal rate of substitution for risks of non-fatal lymphoma and risk of accidental death from a car accident. The resulting risk-risk tradeoff value can be applied to an estimate of the VSL to generate a value for avoiding a statistical case of non-fatal lymphoma.

In this study, we have followed the previous SAB EEAC advice to estimate the value of a non-fatal cancer case using the chronic bronchitis value and a value from the Magat et al. work to bracket a range of possible values. To generate the endpoints of this range, we derived a WTP value for chronic bronchitis from EPA's September 2006 PM NAAQS RIA ($410,000, 2006$, 1990 income levels)[56], and used the VSL for our primary estimate from Viscusi and Aldy (2003) along with the risk-risk ratio estimated by Magat et al. (0.583) to calculate a non-fatal lymphoma value ($4.3 million, 2006$, 1990 income levels).

Based on SAB advice provided during a review of this case study (USEPA, 2008b), we further examined the appropriateness of using estimates of WTP from chronic bronchitis and non-fatal lymphoma to value cases of non-fatal leukemia by comparing the symptoms, severity, duration, and treatment of these illnesses.

Symptom data for these conditions show significant overlap between leukemia and lymphoma (e.g., fever, weight loss, night sweats, fatigue, enlargement of spleen, loss of appetite, and swollen lymph nodes) and to a lesser extent between leukemia and chronic bronchitis (e.g., shortness-of-breath, fatigue, and headaches) (MedLine, 2008).[57]

The duration and treatment of leukemia varies by subtype. For instance, acute leukemia is usually treated immediately with options such as chemotherapy, targeted drug therapy, biological therapy, radiation therapy or stem cell transplant (NCI, 2008). It is possible for acute leukemia to go into remission or even be cured. Chronic leukemia, however,

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[55] The full text of the SAB advisory states:

"To value non-fatal bladder cancers, the Agency used a value for avoiding a statistical case of chronic bronchitis obtained by Viscusi, Magat, and Huber (1991). We have two reservations about this. First, this study used a small sample obtained in a shopping mall in North Carolina and thus may not be representative of either the U.S. population as a whole or the population of individuals at risk of bladder cancer. Second, we have no basis for determining that avoiding a case of chronic bronchitis has the same value as avoiding a non-fatal case of bladder cancer.

On this second point, there is one study of willingness to pay to avoid a non-fatal case on one type of cancer. Magat, Viscusi, and Huber estimated the willingness to pay to avoid a case of non-fatal lymphoma to be $3.6 million (Magat, et al. 1996). This value was obtained from a similar shopping mall intercept survey with a substantially larger sample size. So, although the endpoint being valued more nearly corresponds to non-fatal bladder cancer, there is still the question of the representativeness of the sample. We also note that the value obtained is at least 20 times larger than the cost of illness for non-fatal bladder cancer cited in Exhibit 5-10. Thus we do not have a lot of confidence in this number. Therefore, we recommend that the value used in the report and the alternative discussed here be used as bounds in an uncertainty analysis. However, this range should be clearly identified as displaying the two extreme estimates available in the literature so it is not misconstrued as a confidence interval" (from EPA, 2001a, page 5 and subsequent text).

[56] See U.S. EPA 2006, *Regulatory Impact Analysis for the 2006 National Ambient Air Quality Standards for Particle Pollution*, available for download at: http://www.epa.gov/ttn/ecas/ria.html.

[57] We also attempted to locate health-related quality of life (HRQL) scores (such as the health utilities index (HUI) or the EuroQoL EQ-5D) for these specific conditions, but were unsuccessful.

can be asymptomatic and treatment is sometimes delayed. Chronic leukemia can often be controlled but it is difficult to cure (MedLine, 2008).

The duration and treatment of lymphoma also varies by type. Hodgkin's lymphoma is one of the most curable forms for cancer. Treatment generally includes chemotherapy and/or radiation therapy. Non-Hodgkin's lymphoma is more likely to be cured if it is fast growing. Treatments vary depending on the type of non-Hodgkin's lymphoma and include chemotherapy, radiation therapy, and stem cell transplant (Leukemia and Lymphoma Society, 2008). Chronic bronchitis symptoms can be treated with medication and oxygen therapy. However, this disease is long-term and cannot be cured completely (MedLine, 2008).

In conclusion, based on the data we have gathered on symptoms, duration, and treatment of leukemia, lymphoma, and chronic bronchitis, it appears as though employing a range of WTP values is appropriate for non-fatal leukemia. Leukemia includes several subtypes, each of which varies significantly. Acute non-fatal leukemia likely is a better match with non-fatal lymphoma in terms of symptoms, duration, and treatment. However, chronic non-fatal leukemia is unlikely to be cured and could be seen as similar in duration to chronic bronchitis. Therefore, bounding the WTP estimates for non-fatal leukemia with estimates for these two illnesses appears to be reasonable.

As a crosscheck on these values, we estimated cost-of-illness (COI) values for a non-specific case of non-fatal cancer. Our estimates rely on estimates of the direct medical cost of illness from EPA's *Cost-of-Illness Handbook*. Assuming three months of initial treatment followed by 16 years of follow-up treatment, consistent with the median age of diagnosis for all leukemias of 67 and an approximate average life expectancy at 67 of 16 years, we estimated the net present value in 2006$ of the direct medical cost of illness as $116,000 (using a 7 percent discount rate) to $200,000 (using a 3 percent discount rate). As expected, the direct medical costs are significantly less than a comparable WTP estimate - the direct medical costs exclude such factors as lost earnings, implicit value of lost caregiver time, and pain and suffering of the patient over the period of illness.

We also identified a value for a case of non-fatal cancer used by the European Commission countries in their "ExternE" study of the external costs of energy generation. The value of 450,000 (1995 European Currency Units or ECU)[58] was based on a U.S. COI study that included indirect costs of illness in the form of lost wages.[59] Converting to 2006$, we obtain a value of $700,000. The ratio of the WTP used in this case study to this estimate of COI ranges from 0.5 at the low end of the WTP range for non-fatal leukemia to 5.4 at the upper end of the range. A review of studies estimating both WTP and COI for various illnesses in the *Handbook for Non-Cancer Health Effects Valuation* (SPC, 2000) found that WTP/COI ratios ranged from 2 – 31.5 with a median of 3.9 and a

[58] The ECU was a currency used by the member states of the European Union (EU) prior to introduction of the euro on January 1, 1999.

[59] See Table 5.2, page 35 in, Common Annexes of the ExternE National Implementation Reports (1998), downloaded 6/1/07 from: http://externe.jrc.es/reports.html.

mean of 8.1 (see Appendix B, Table B-1). Therefore, the estimates used in this case study fall outside this range on the low end for the WTP estimate derived from the chronic bronchitis study but are between the median and mean of values for the non-fatal leukemia WTP.

Cessation Lag

As discussed previously, reduction in exposure to benzene leads to reduction in cancer cases after a period of cessation lag. In economic terms, it is plausible to assume that individuals would prefer avoidance of immediate health effects relative to avoidance of health effects with a delay, suggesting that their WTP to avoid delayed heath effects is affected. Because the underlying VSL estimates are largely for immediately manifest risks of death, the VSL estimate needs to be adjusted to account for the effect of the cessation lag on WTP.

We made this adjustment by discounting the VSL estimate by the period of cessation lag using four alternative discount rates. We used a discount rate of 5 percent for our primary estimate and used discount rates of 0, 3 and 7 percent as sensitivity analyses.

CHAPTER 3 | RESULTS

This chapter presents the results of the emissions, air quality/exposure, and health effects modeling steps in the analytical chain. We present the health benefit results both as avoided cases of leukemia, and as monetized benefits valued as described in the Valuation step in Chapter 2.

3.1 EMISSIONS

Figure 3 illustrates the difference in emissions of benzene in the Houston-Galveston study area in 2000, 2010, and 2020 under the *With-CAAA* and *Without-CAAA* scenarios. Table 2 provides the specific modeled emission estimates by sector. Both exhibits show that the CAAA have resulted in significant benzene emission reductions in the Houston-Galveston study area since 1990. We first discuss the emissions trends under the *With-CAAA* scenario, and then compare the results for the *With-* and *Without-CAAA* scenarios. For additional details concerning these results, please consult Appendix A.

3.1.1 EMISSIONS UNDER THE *WITH-CAAA* SCENARIO

A significant fraction of the reductions in benzene from the CAAA occurred within the first decade following passage of the amendments.[60] Under the CAAA in 2000, total emissions decreased 70 percent from 1990 levels, with the bulk of this reduction occurring in the combined point and non-point sector.[61] For these sources, the benzene emission reductions during this 1990 to 2000 period are largely attributable to Federal maximum achievable control technology (MACT) emission standards, and local VOC measures in the 1-hour ozone attainment plan that required the petrochemical facilities in the area to reduce hazardous air pollutant (HAP) and/or VOC emissions. The chemical manufacturing and petroleum refining industries achieved the most significant benzene emission reductions in these sectors in this period. Mobile sources also exhibit significant reductions in this period, due in part to existing pre-1990 Tier 1 regulations reducing exhaust and evaporative VOC emissions and in part to CAAA-related

[60] These results do not include the impact of the 2007 MSAT rule, which was promulgated too late to be included in the *with-CAAA* scenario.

[61] We have chosen to combine point and non-point emissions into a single category because of a discrepancy in the way that the 1990 and 2000 NEIs treat fugitive emissions from the synthetic organic chemical manufacturing industry ("SOCMI fugitives"). The 1990 NEI includes these emissions in the non-point source category while the 2000 NEI reports them as point source emissions. Because we project *Without-CAAA* emissions for point and non-point sources from 1990 NEI data and project *With-CAAA* emissions for these sources from 2002 NEI data, SOCMI fugitives end up categorized differently under the two scenarios. SOCMI fugitive emissions are a significant source of emissions, contributing nearly 2,400 tpy in 1990; therefore, we have combined the two categories to accurately reflect the combined impact of CAAA measures on point and non-point emissions sources in Houston.

reformulated gasoline requirements and inspection and maintenance (I/M) programs in each county.

FIGURE 3: MAJOR, AREA & OTHER, ON-ROAD, AND NON-ROAD EMISSIONS (TONS) FOR EACH YEAR AND SOURCE TYPE

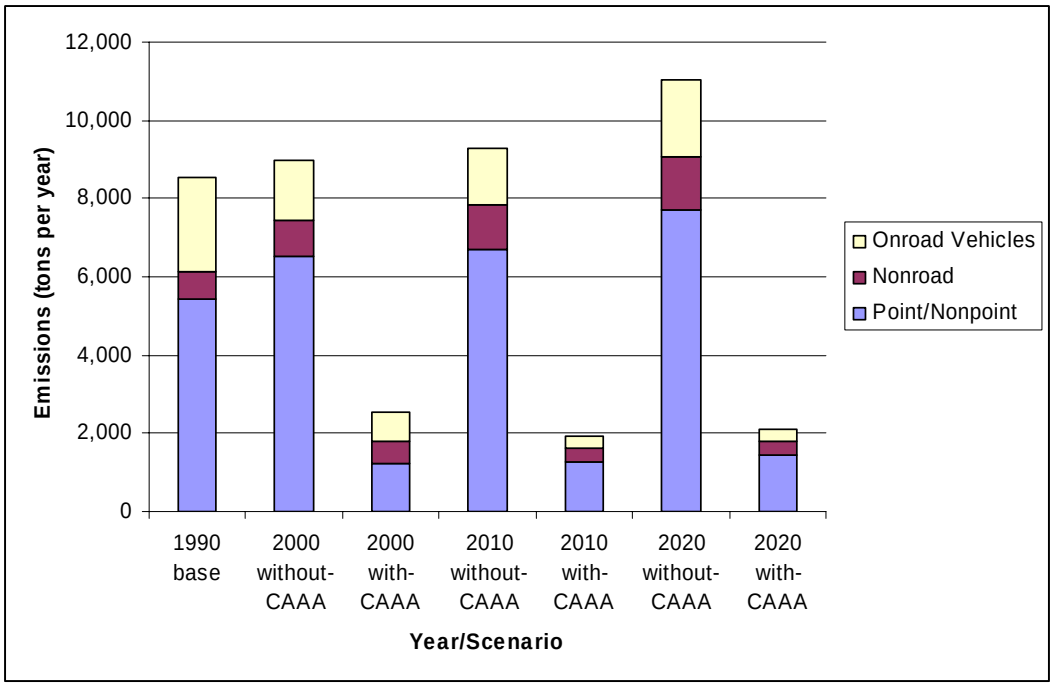


TABLE 2: HOUSTON-GALVESTON BENZENE EMISSIONS SUMMARY (TONS PER YEAR [TPY])

YEAR	1990	2000		2010		2020	
SCENARIO		WITHOUT-CAAA	WITH-CAAA	WITHOUT-CAAA	WITH-CAAA	WITHOUT-CAAA	WITH-CAAA
SECTOR							
Point/Non-point	5,409	6,532	1,230	6,699	1,258	7,702	1,440
Non-road	740	900	567	1,127	354	1,351	360
On-road Vehicles	2,375	1,541	762	1,449	328	1,988	282
Total	8,524	8,973	2,559	9,275	1,940	11,041	2,082

Total emissions continue to decrease in 2010 under the CAAA (77 percent below 1990 levels) and increase slightly between 2010 and 2020. Point and non-point source emissions are essentially stable from 2000 to 2010 and slightly increase from 2000 through 2020. The addition of 7- and 10-year MACT rules in the second decade mitigate emissions growth in this category the first period, but we see a slight increase from 2010 to 2020 in part because the analysis is not applying any new point or non-point source

VOC or benzene-related control programs post-2010.[62] We do observe additional reductions in the mobile source category in this period due to Tier 2 emission standards and associated requirements that lower the sulfur content of gasoline. Reductions in non-road emissions are due largely to the implementation of spark-ignition engine standards.

3.1.2 DIFFERENCE IN EMISSIONS BETWEEN THE *WITH-* AND *WITHOUT-CAAA* SCENARIOS

When we compare the *With-CAAA* scenario to the counterfactual *Without-CAAA* scenario, we observe substantial and increasing differences in each of the three target years – approximately 6,500 fewer tons of benzene in 2000, 7,300 fewer tons in 2010, and nearly 9,000 fewer in 2020. These changes represent reductions in benzene emissions of 71, 79, and 82 percent, respectively, over the *Without-CAAA* scenario. Most of this difference is due to emission controls on point and non-point sources, which emit thousands fewer tons per year under the CAAA; however, reduced emissions from motor vehicles also contribute significantly, particularly in the later years, as the Tier II emissions standards begin to have an impact.[63] Emissions reductions from the non-road sector are a relatively small contributor, because the base year emissions are relatively low; its contribution to overall reductions is greatest in 2010 and 2020.

3.2 AIR QUALITY/EXPOSURE MODELING

The air quality modeling step produced both estimated ambient concentrations of benzene in the study area, using AERMOD, and estimates of age-specific exposure concentrations using EPA's HAPEM that reflect the influence of individuals' activity patterns on the benzene exposure they are likely to experience during their daily activities. Detailed results for both study elements may be found in Appendix B; we provide an overview and comparison of the results from both models below.

Figure 4 summarizes the distribution of benzene concentrations predicted in the study area in the base year and each target year under the *With-* and *Without-CAAA* scenarios. The distributions reflect the variation in concentrations across census block groups in the three counties studied. The yellow *With-CAAA* distributions show both lower median (center line) concentrations under the *Without-CAAA* scenario and tighter distributions with less variation than the green *Without-CAAA* distributions. The difference in medians widens with time, both due to additional CAAA-related benzene decreases (particularly

[62] While there may be regulations added in this area in the next few years to meet new nonattainment obligations, based on the current set of Federal and State regulations affecting this area, benzene emission rates for this category have no expected declines in the 2010 to 2020 period other than for woodstoves.

[63] Our model indicates that some benzene emissions reductions from mobile sources occur between 1990 and 2000 even in the absence of the 1990 CAAA, due to fleet turnover enhancing the effects of pre-1990 CAA emissions regulations. As a result, growth in emissions in the first decade of the *Without-CAAA* scenario is less than might be expected, and the percentage reduction in total emissions between the with- and *Without-CAAA* scenarios is not much larger than the percent difference between the 2000 *With-CAAA* scenario and 1990. This effect lessens in 2010 as fewer older cars remain on the road and vehicle miles traveled (VMT) increase. By 2020, the VMT effect dominates and emissions increase in the *Without-CAAA* scenario. Meanwhile, CAAA mobile source provisions such as Tier II emission regulations have an increasing impact from 2000 to 2020, widening the difference between the two scenarios for mobile sources during that time.

between 2000 and 2010) and due to projected emissions growth without the CAAA (particularly between 2010 and 2020).

FIGURE 4: BLOCK GROUP LEVEL TOTAL CONCENTRATION (μg/m³) DISTRIBUTIONS FOR 1990, 2000, 2010, AND 2020 FOR *WITH-CAAA* (YELLOW) AND *WITHOUT-CAAA* (GREEN) SCENARIOS

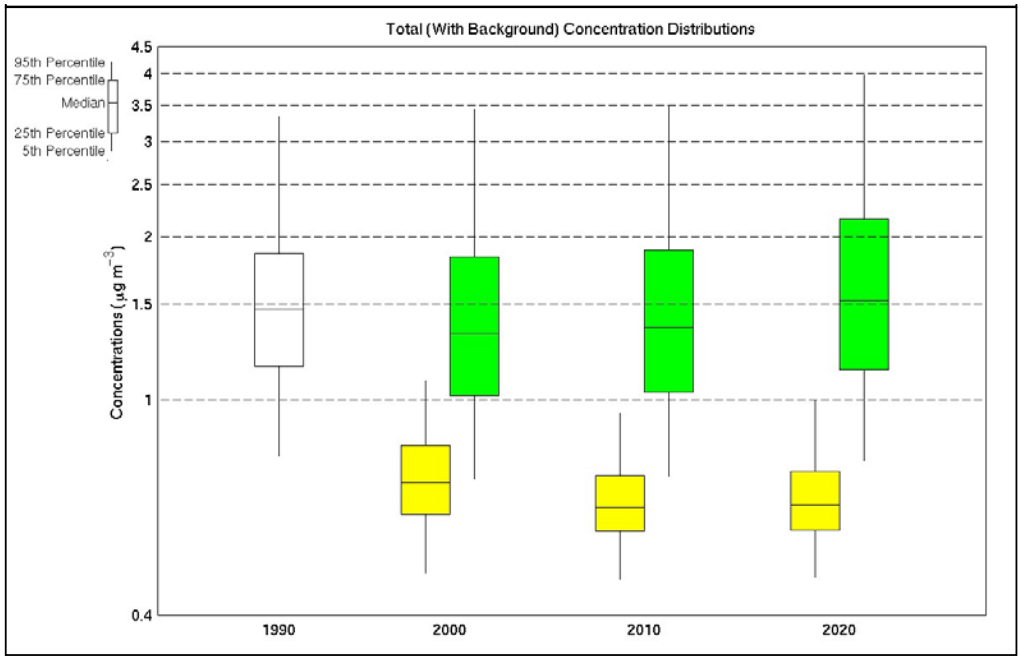
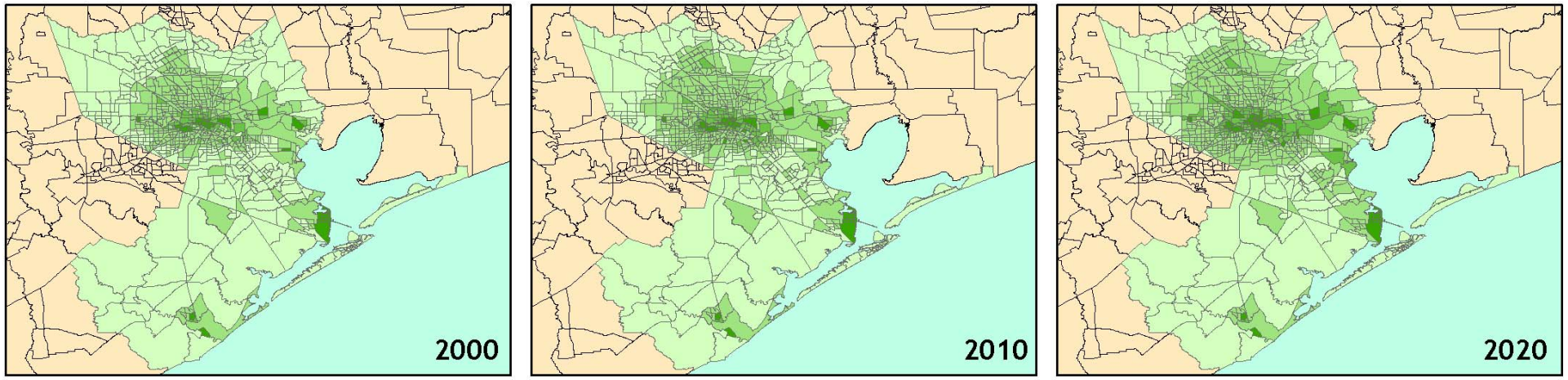


Figure 5 presents maps showing the spatial distribution of benzene reductions across the study area. The top row of maps shows the AERMOD estimates of the reduction in annual average ambient benzene levels due to CAAA programs in (from left to right) 2000, 2010, and 2020. The bottom row shows the same progression using the exposure concentration results from the HAPEM model. On all six maps, the darker shades of green represent greater benzene reductions.

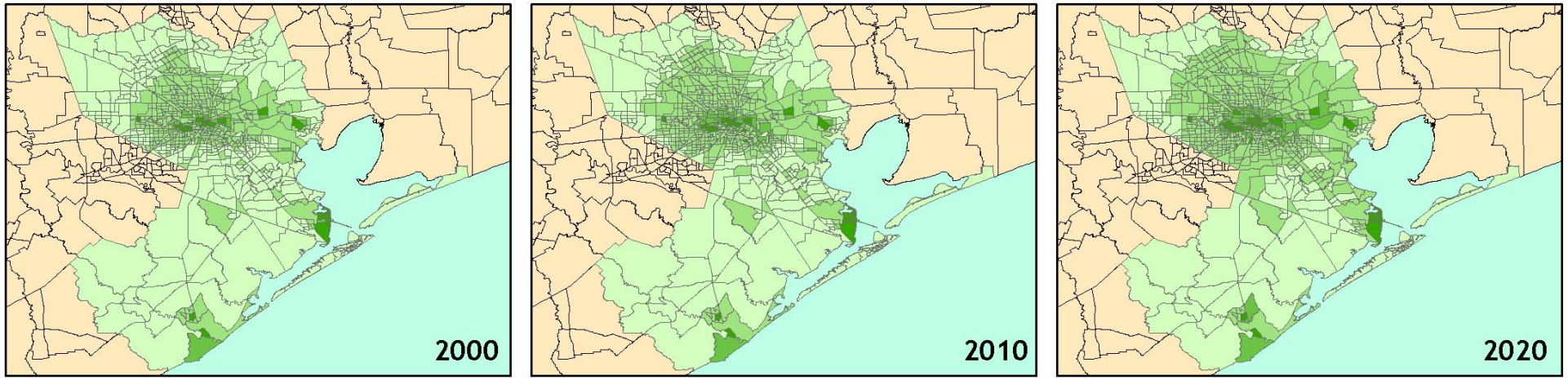
The AERMOD maps show the greatest reductions (in excess of 5 μg/m^3) occur in Harris County in the downtown Houston area, within the rings of the interstate, in the Texas City area of Galveston County where a number of refineries and chemical facilities are located; and in southeastern Brazoria County, which also features major chemical manufacturing and petroleum refining facilities. Mobile source emission controls are a significant contributor to the reductions in Harris County, and thus we can see an increase in the areas experiencing larger reductions in that area, as mobile source controls become more effective over time. The major reductions in Galveston and Brazoria are primarily driven by controls on major point and non-point sources, which tend to be realized earlier in our analysis; thus, we see less change over time in the reductions in these areas. There are some additional reductions gained in the Texas city vicinity, however, most likely due to controls on on-road and non-road sources.

FIGURE 5: ESTIMATED CAAA-RELATED REDUCTIONS IN BENZENE CONCENTRATIONS IN THE HOUSTON METROPOLITAN AREA (*WITHOUT-CAAA* MINUS *WITH-CAAA*) - AERMOD AND HAPEM RESULTS

AERMOD RESULTS



HAPEM RESULTS



Reductions in Concentration >2.5 µg/m³ 1.5 to 2.5 µg/m³ 0.5 to 1.5 µg/m³ <0.5 µg/m³

Note: HAPEM results represent the estimated exposure concentration reduction for the median exposed individual in each census tract.

As we compare the maps from top to bottom, we can see the changes in exposure estimates as we process the ambient data through HAPEM to incorporate time-activity patterns of the exposed populations. The exposure changes reflected in the bottom maps represent the change in concentration that we expect would be experienced by the median individual in a given census tract. In general, HAPEM tends to smooth and spread out the AERMOD concentration changes; this reflects both aggregating results to the census tract level and incorporating the impact of commuting and other activities on the concentration experienced by the population in each census tract.

Tables 3 and 4 present mean reductions in annual average benzene from AERMOD and HAPEM, respectively, for each county in each year. In addition, these tables indicate the minimum and maximum reductions estimated for a census block group (AERMOD) or census tract (HAPEM) in that county in that year. To facilitate comparison between the air quality modeling and exposure modeling results, we have calculated population-weighted mean benzene reductions from AERMOD in Table 3. That is, the mean estimates in Table 3 have been adjusted to give more weight to reductions in areas with large populations and less weight to reduction in areas with small populations. The population-weighted mean reductions tend to be around 1 $\mu g/m^3$, though the range of reductions can be significant, in several cases exceeding 20 $\mu g/m^3$. The results for HAPEM tend to be slightly lower than the AERMOD results. The average ratio of HAPEM to AERMOD concentrations is about 90 percent (see Table 19 in Appendix B), suggesting that much of the population may be commuting from census tracts with higher benzene levels to census tracts with lower levels.

TABLE 3: POPULATION-WEIGHTED MEAN REDUCTION IN AMBIENT ANNUAL AVERAGE BENZENE CONCENTRATION DUE TO CAAA, BY YEAR AND COUNTY

STUDY YEAR	MEAN CHANGE IN BENZENE CONCENTRATION, $\mu g/m^3$ (RANGE)		
	BRAZORIA	GALVESTON	HARRIS
2000	1.0 (0.04 - 25)	0.8 (0.04 - 18)	0.8 (-3 - 34)*
2010	1.1 (0.08 - 25)	0.9 (0.05 - 17)	1.0 (-4 - 33)*
2020	1.3 (0.09 - 28)	1.0 (0.06 - 20)	1.2 (-4 - 37)*

* Seven of the 1,911 census block groups in Harris County showed dis-benefits under the *With-CAAA* scenario. Of these, five reported increases of 0.3 $\mu g/m^3$ or less. The smallest reductions estimated were between 0.02 and 0.1 $\mu g/m^3$.

TABLE 4: HAPEM-ESTIMATED MEAN REDUCTION IN ANNUAL BENZENE EXPOSURE CONCENTRATION DUE TO CAAA, BY YEAR AND COUNTY

STUDY YEAR	MEAN CHANGE IN BENZENE CONCENTRATION μg/m³* (RANGE)		
	BRAZORIA	GALVESTON	HARRIS
2000	0.9 (0.07 - 19)	0.7 (0.08 - 14)	0.8 (-1 - 11)**
2010	0.9 (0.1 - 19)	0.7 (0.09 - 14)	0.9 (-1 - 12)**
2020	1.1 (0.1 -21)	0.9 (0.1 - 16)	1.1 (-1 - 14)**

* The HAPEM results in this table represent the exposure change for the median individual in a census tract (i.e., they are neither highly nor minimally exposed in terms of their activities and characteristics). The exposure change is an average change in exposure across all age categories.

* *One of the 649 census tracts in Harris County reported dis-benefits under the *With-CAAA* scenario. The smallest reductions estimated were between 0.07 and 0.1 $\mu g/m^3$.

3.2.1 MODEL TO MONITOR COMPARISONS

The results of the model-to-monitor comparisons are presented in Appendix B. As can be seen in Figures 32 and 33 of that document, many of the AERMOD predicted values fall within a factor of 0.5 to 2 of the monitored values, which is considered good agreement. However, a significant fraction of the *With-CAAA* estimates are less than half of the monitor values, suggesting the model may be underestimating benzene levels.

3.3 HEALTH EFFECTS

This section presents the health effects results and the associated monetary benefits results. We first present the life-table model results for our primary estimate of avoided fatal and non-fatal cases of leukemia (all types) and the monetized value of those cases. We then discuss the results of our assessment of the non-cancer effects of benzene. The next section presents our analysis of CAAA-related individual leukemia risk reductions for individuals that are part of highly exposed populations in the case study area. Finally, we describe the additional life-table model runs we conducted to assess the sensitivity of the model to alternative assumptions.

3.3.1 CANCER

Avoided Cases

Table 5 below presents the results of our primary estimate for avoided fatal and non-fatal cases of leukemia due to CAAA-related changes in ambient benzene levels in the Houston area (including Brazoria, Galveston, and Harris counties). The results are presented for the base year (1990) as well as the three study years (2000, 2010, and 2020). The values in Table 5 represent the annual number of avoided cases in each target

year as well as a total number of expected cases avoided from 1990 through 2020. We expect the benefits of the benzene reductions that occur in the study period will continue accruing to the study population beyond the end of the study period. Therefore, we also estimated a total number of cases expected to occur past 2020 that are a result of CAAA-related changes in benzene occurring within the study period. We discuss the derivation of this estimate further in the section entitled "Expected Total Benefits."

TABLE 5: ANNUAL AVOIDED LEUKEMIA CASES (FATAL AND NON-FATAL) BY STUDY YEAR DUE TO CAAA-RELATED BENZENE EXPOSURE CHANGES IN THE HOUSTON AREA

STUDY YEAR	ANNUAL AVOIDED CASES OF LEUKEMIA		
	AVOIDED FATAL CASES	AVOIDED NON-FATAL CASES	TOTAL AVOIDED CASES
1990	0	0	**0**
2000	0.03	0.02	**0.05**
2010	0.09	0.07	**0.2**
2020	0.2	0.1	**0.3**
Cumulative Cases Occurring Within the Study Period	**2**	**2**	**4**
Additional Cumulative Cases Occurring After 2020*	**1**	**1**	**2**
Total Cumulative Cases	**3**	**3**	**6**

*Note: These avoided cases are due to changes in benzene exposure that occurred within the study period.

Our results indicate that by the year 2020, a total of four cases of leukemia would be avoided due to the 1990 CAAA programs in the Houston area, with three of those occurring in Harris County. We estimate two of the four cases to be fatal and two to be non-fatal.[64]

Monetary Valuation

We applied the valuation methods described in Section 2.5.2 to determine the economic value of these avoided leukemia cases. The results of the valuation analysis are presented below in Table 6.

[64] The composition of fatal and non-fatal cases is consistent with data from the SEER website for 1988-2004, which indicates that ten year-survival rates for leukemia are approximately 40 percent (http://seer.cancer.gov/).

TABLE 6: ANNUAL MONETARY BENEFITS BY STUDY YEAR DUE TO CAAA-RELATED CHANGES IN BENZENE EXPOSURE IN THE HOUSTON AREA

STUDY YEAR	TOTAL BENEFITS (1990 NPV, MILLIONS OF 2006$, 5% DR)		
	BENEFITS FROM FATAL CASES OF LEUKEMIA	BENEFITS FROM NON-FATAL CASES OF LEUKEMIA	TOTAL BENEFITS
1990	$0	$0	**$0**
2000	$0.12	$0.01 – 0.06	**$0.13 - 0.18**
2010	$0.27	$0.01 – 0.13	**$0.28 - 0.40**
2020	$0.31	$0.01 – 0.15	**$0.32 - 0.46**
Cumulative Cases Occurring Within the Study Period	**$6.7**	**$0.32 - 3.3**	**$7.0 - 10**
Additional Cumulative Cases Occurring After 2020*	**$1.6**	**$0.08 - 0.8**	**$1.7 - 2.4**
Total Cumulative Cases	**$8.3**	**$0.40 - 4.1**	**$8.7 - 12**

*Note: These avoided cases are due to changes in benzene exposure that occurred within the study period, but occurred after 2020 due to lagging effects of these changes on leukemia risks.

The values in Table 6 represent the annual net present value estimate (discounted to 1990) of the benefits of the CAAA-related benzene controls in Houston in each target year.[65] In addition, we calculated the net present value of benefits over the entire study period and the additional benefits of cases occurring after 2020. Our primary estimate of total benefits due to CAAA-related reductions in benzene are $8.7 - 12 million (in 2006$), $8.3 million of which are due to fatal cases of leukemia, and $0.4 – 4.1 million of which are due to non-fatal cases. Our primary estimate incorporates a discount rate of 5 percent to account for the effect of cessation lag on the distribution of benefits over time.

Expected Total Benefits

The life-table model we applied in this analysis was designed to calculate the change in the number of cases of leukemia likely to be observed in a given year, as a function of a population's current and past exposures. Because of the way we model the lag between exposure reduction and benefits (see Section 2.4.2), the exposure change in the year being modeled contributes little to the observed risk reduction in that year; most of its effects will be realized over the next several years. Similarly, the exposure changes in the years preceding the year being modeled will continue to produce benefits in future years, to a lesser degree over time. As a result, a portion of the benefits that result from exposure changes that occur in the 1990 to 2020 study period will not be observed until after 2020.

[65] Net present value (NPV) calculations facilitate comparison of costs or benefits that may occur at different points in the future by expressing them in terms of their value in a common reference year, using the economic principle of discounting. For example, the value of X dollars received N years from today would be $X/(1 + i)^N$, where i represents the discount rate, a measure of the time value of money. In this case study, we discount the value of all future health benefits back to the first year of the analysis, 1990, and sum them to produce our NPV estimates.

To address this model limitation, we estimated the relative magnitude of the benefits that we expected would occur after the end of the study period (i.e., past the year 2020), assuming that the latency period assumed in our primary estimate is correct. In order to generate an estimate of the size of these benefits, we ran the model using a truncated exposure data set that "turned off" the effect of the CAAA after 2010 (i.e., it assumed no difference in exposure between the *With-* and *Without-CAAA* scenarios after the year 2010) and observed how the benefits decreased following 2010. We found that annual avoided cases peaked in the year 2010 and then decreased to 90 percent of the 2010 level for the first five years (2011-2015) and to 50 percent of the 2010 level for the next 5 years (2016-2020). (Although we did not model past 2020, we believe the benefits after 10 years will likely be minimal, given the exposure weights we used in the model.) We believe the decay in benefits observed in this example 2010 cutoff run represent a reasonable approximation of the results that would be observed after 2020.

We applied the ratios from the 2010 cutoff run to the 2020 estimates of annual avoided cases and calculated estimates of cumulative avoided cases for 2025 and 2030. We estimated less than one additional fatal case and less than one additional non-fatal case avoided in the first five years after the study period. By the year 2030, we estimated another partial fatal and another partial non-fatal case would be avoided, making the cumulative total cases avoided through 2030 due to benzene concentration changes between 1990 and 2020 to be roughly six.

3.3.2 NON-CANCER

As described in Section 2.4.5, in order to assess non-cancer health benefits, we planned to report the difference between the *With-CAAA* and *Without-CAAA* scenarios in the number of individuals experiencing benzene concentrations above the chronic RfC published in EPA's Integrated Risk Information System (IRIS) database. Therefore, we compared the chronic RfC value reported on IRIS (0.03 mg/m^3) with the ambient benzene concentrations from HAPEM6 for each tract under both the *With-* and *Without-CAAA* scenarios. We then calculated the total census population across all of the tracts with benzene concentrations exceeding the RfC under each scenario. We found no individuals exposed to benzene at concentrations exceeding the RfC in either the *With-* or *Without-CAAA* scenarios.

3.3.3 HIGHLY-EXPOSED POPULATIONS

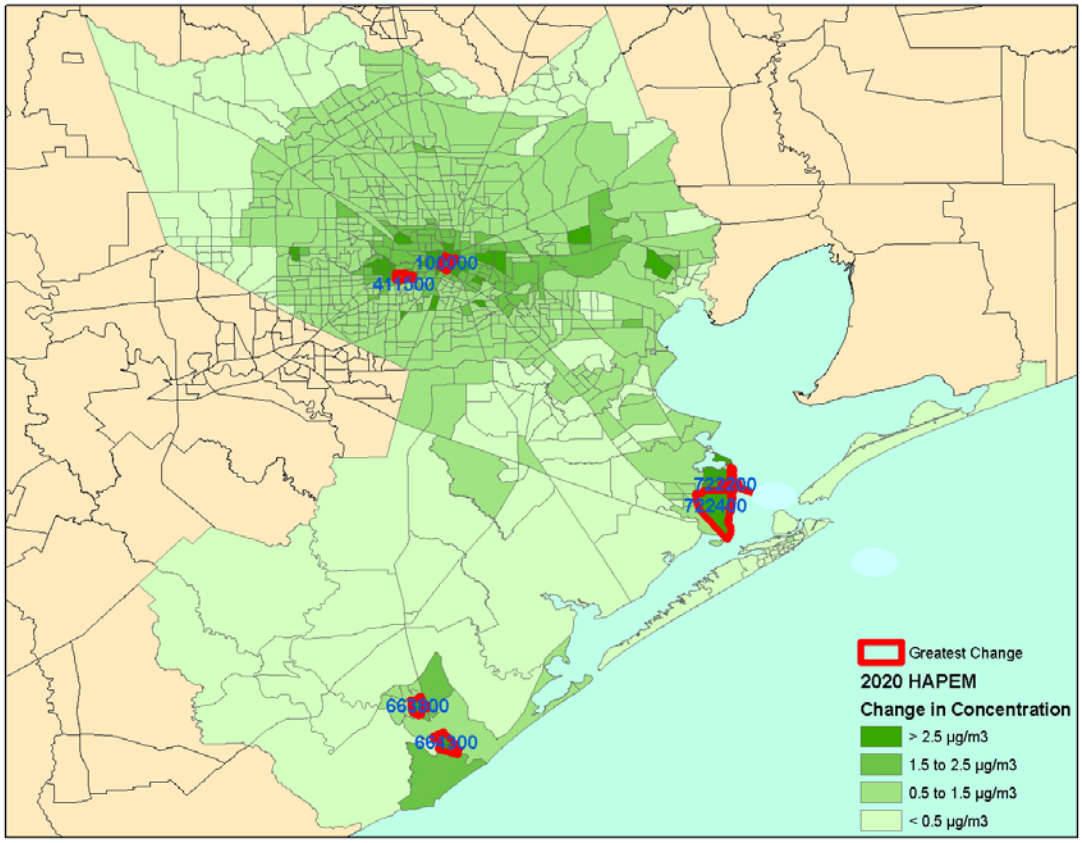
We evaluated risks to three different sets of highly exposed populations: residents living in census tracts with high benzene concentrations, residents living near roadways, and residents living in homes with attached garages.

Residents Living in Census Tracts With High Exposure

As described in Section 2.4.4, we estimated CAAA-related reductions in the lifetime risk of leukemia for individuals living in census tracts with the highest levels of benzene. Figure 6 shows a map that highlights these census tracts. Table 7 below presents the individual lifetime risk of leukemia for a person born in 2020 under both the *With-* and

Without-CAAA scenarios in the two tracts in each county with the highest exposure concentrations. In addition, we report the population of these tracts, who would experience these levels of risk or higher. Risks under the *Without-CAAA* scenario are significantly higher compared to those under the *With-CAAA* scenario. For example, some risks in Brazoria County drop from an increased lifetime leukemia risk of 2 in ten thousand (i.e., 2×10^{-4}) to 3 in a million (3×10^{-6}) as a result of the CAAA, a 98 percent reduction. In four of the six tracts in Table 7, individual lifetime leukemia risks among the highly exposed are reduced by at least 80 percent; the risks in all six counties are reduced by at least 72 percent.[66] For comparison, the estimated average lifetime leukemia risk reduction across the 3-county study area for an individual born in 2020 is 65 percent.

FIGURE 6: CENSUS TRACTS IN THE HOUSTON STUDY AREA WITH THE GREATEST BENZENE EXPOSURE CONCENTRATION CHANGES BETWEEN THE *WITH-* AND *WITHOUT-CAAA* SCENARIOS IN 2020



[66] Risks were calculated using the 7.8×10^{-6} per $\mu g/m^3$ benzene inhalation unit risk (IUR) from the range of IURs reported on IRIS.

TABLE 7: CAAA-RELATED LEUKEMIA RISK REDUCTIONS IN 2020 IN THE HOUSTON AREA FOR INDIVIDUALS LIVING IN CENSUS TRACTS WITH HIGH AMBIENT BENZENE CONCENTRATIONS

COUNTY	CENSUS TRACT	MEDIAN WITHOUT-CAAA RISK	MEDIAN WITH-CAAA RISK	PERCENT REDUCTION IN RISK	POPULATION OF CENSUS TRACT
Brazoria	6643	2×10^{-4}	3×10^{-6}	98	5,452
Brazoria	6638	3×10^{-5}	6×10^{-6}	77	4,470
Galveston	7222	1×10^{-4}	7×10^{-6}	95	3,487
Galveston	7224	5×10^{-5}	8×10^{-6}	82	1,108
Harris	1000	1×10^{-4}	1×10^{-5}	92	6,678
Harris	2523	3×10^{-5}	7×10^{-6}	72	12,686

Note: These risk values were calculated using the 7.8 × 10-6 per $\mu g/m^3$ benzene inhalation unit risk (IUR) from the range of IURs reported on IRIS.

Residents Living Near Roadways

Figure 7 displays boxplots of the results of our 2002 HAPEM runs with and without the near-roadway algorithms. We present results for both the median (50th percentile) and highly exposed (90th percentile) individual.

The boxplots on the left show little change in benzene reductions for the median exposed individual after applying the near-roadway algorithms. Our primary benefit estimates, which are based on the median exposure results, therefore reflect minimal impact of the near roadway adjustment. This is not surprising, because it is unlikely that half of the study population would live within 75 or 200 meters of a major roadway. However, on the right side of Figure 7, we do see an increase in benzene reductions for highly exposed individuals after applying the near-road algorithms. The entire distribution of benzene reductions for the highly exposed group shifts upward, and the median reduction in benzene exposure for this group is about 20 percent larger than the run with the near-roadway algorithm turned off. Thus, overall for the highly exposed group, we observe a moderate impact of incorporating near-roadway effects on benefits. An analysis of the ten census tracts with the highest on-road-related benzene concentrations in 2020 under the *Without-CAAA* scenario (and total population greater than 100) shows more significant impacts in individual locations, with the exposure reduction in one tract in Harris County nearly doubling. On average, the exposure (and hence, risk) reductions in these ten tracts for highly exposed individuals are one and a half times larger when the near-roadway effect is taken into account.

FIGURE 7: BOXPLOTS OF CAAA-RELATED REDUCTIONS IN BENZENE IN THE HOUSTON AREA IN 2020 – IMPACT OF INCORPORATING NEAR-ROADWAY EFFECTS

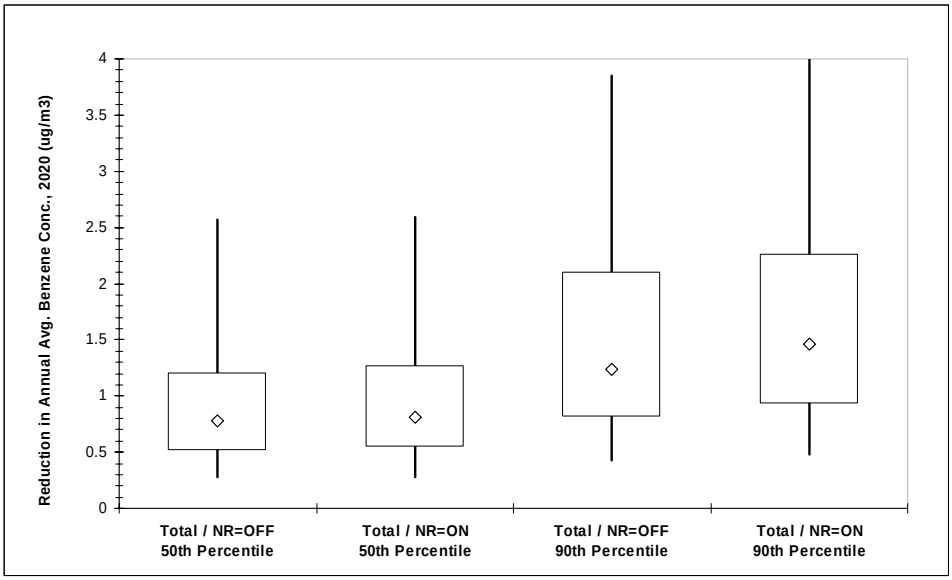


TABLE 8: CAAA-RELATED BENZENE REDUCTIONS IN 2020 INDIVIDUALS LIVING IN CENSUS TRACTS WITH HIGH AMBIENT BENZENE CONCENTRATIONS DUE TO ON-ROAD SOURCES - EFFECT OF HAPEM NEAR-ROADWAY ALGORITHM

COUNTY	CENSUS TRACT	BENZENE REDUCTION NEAR-ROADWAY OFF ($\mu g/m^3$)	BENZENE REDUCTION NEAR-ROADWAY ON ($\mu g/m^3$)	PERCENT CHANGE IN BENZENE DUE TO NEAR ROADWAY EFFECT	POTENTIALLY AFFECTED POPULATION[1]
Harris	321500	1.5	2.6	69	226
Harris	540200	1.3	2.5	89	247
Harris	310700	2.3	3.8	65	457
Harris	541900	2.0	2.5	25	436
Harris	431200	2.4	3.5	44	694
Harris	412100	1.6	2.5	60	98
Harris	450300	2.2	3.1	43	712
Harris	311900	2.0	2.8	42	278
Harris	431900	3.0	3.5	15	206
Harris	410900	2.7	3.3	21	282

[1] Because these values were calculated using 90th percentile exposure concentrations, we assumed that 10 percent of the population in the tracts may be associated with these changes in benzene exposure or higher.

Residents With Attached Garages

We estimated that total emissions in attached garages in the Houston area would be reduced by almost 90 percent. If the average exposure estimate attributable to attached garages (1.2 μg/m^3; see USEPA, 2007d) were reduced by this amount, the expected reduction in exposures due to reductions of in-garage emissions would be 1.1 μg/m^3. We found that this would correspond to an additional estimate of annual avoided cases of leukemia in the Houston area in 2020 that is roughly similar in magnitude to our main benefits estimate. Therefore, these results suggest that adding attached garage-related benefits to our primary estimate could result in an approximate doubling of our primary estimate.[67]

3.3.4 SENSITIVITY ANALYSES

We performed five sensitivity analyses to estimate the range of uncertainty surrounding our primary estimate and to determine how sensitive the health risk model is to various data inputs and assumptions.[68] We first assessed the impact of statistical uncertainty surrounding our primary estimate by running the model with the upper and lower 95 percent confidence limits of the dose-response slope factor from Crump (1994). We then tested the sensitivity of the model to the underlying epidemiological data by substituting the dose-response slope factor used in our primary estimate with that from another major cohort study linking benzene and leukemia. Next, we explored the sensitivity of the model to the health endpoint selected by looking at the differences between incidence rates for all leukemia versus AML. We next substituted a dose-response slope factor derived using different exposure estimates from the same cohort used in our primary estimate, the Pliofilm Cohort. We also ran the model with two alternate lags, a zero-year lag and a five-year lag.

We also explored the range of uncertainty surrounding assumptions made during the valuation of the health effects results. We performed a sensitivity analysis on our primary valuation estimate by altering the discount rate applied. We also substituted alternate values for the VSL used to value fatal cases of leukemia. Finally, we assumed that all of the leukemia cases due to benzene exposure were fatal to get an upper bound benefits estimate.

Statistical Uncertainty

Our primary estimate of avoided cases of leukemia relied on a mean dose-response slope factor from the Crump (1994) paper. To assess the impact of statistical uncertainty on this estimate, we ran the life-table model with both the upper and lower 95 percent

[67] Homes with attached garages may also experience significant short-term spikes in benzene concentrations in the house following cold start or hot soak events (Graham and Noseworthy, 2004). CAAA controls would also be expected to reduce these acute benzene exposures to individuals living in these homes; however estimation of these benefits is beyond the scope of this analysis.

[68] We did not perform a Monte Carlo analysis as part of the sensitivity analysis due to the large amount of data involved and time and resource limitations

confidence bounds (UCL and LCL) around the mean dose-response slope factor, as reported in Crump (1994).

These additional runs indicate that based solely on the statistical uncertainty in the selected dose-response function from the Pliofilm cohort, total cumulative avoided cases of leukemia occurring within the study period could range from a lower bound of 0.8 to an upper bound of seven.

Chinese Worker

Our primary estimate of avoided leukemia cases from the life-table model relied on dose-response slope factors for the relationship between benzene and leukemia from the Pliofilm Cohort, as these are the data currently supported by EPA in the benzene IRIS profile to calculate potency estimates. For our sensitivity analysis, we used a dose-response slope factor from another large, well-studied occupational cohort, the Chinese Worker Cohort. The strengths of this cohort study include a large number of leukemia cases and workers who were exposed to benzene levels similar to ambient levels.

Because the studies examining the Chinese Worker Cohort did not derive dose-response slope factors, we used dose-response slope factors derived by the California Environmental Protection Agency (CalEPA) as part of an analysis to calculate a Public Health Goal for benzene (CalEPA, 2001).[69] We also applied the same lag to our exposure data as was assumed in the Chinese Worker Cohort (1.5 years). The life-table model run with this alternate dose-response slope factor and 1.5-year lag estimated that a total of seven cases of leukemia would be avoided between 1990 and 2020 due to the CAAA.

AML

Our primary estimate was based on a dose-response slope factor derived with all leukemia as the health endpoint. To test the sensitivity of this assumption, we first compared rates for all leukemia to those for AML, the leukemia subtype with the most data supporting its link with benzene, to estimate the proportion of leukemia cases that were AML. We compared national-level age-specific AML incidence rates to national age-specific all leukemia incidence rates.[70] We found that the age-specific all leukemia incidence rates were on average four times higher than the AML rates and ranged from two times higher (for the 25-29 age group) to nine times higher (for the 5-9 age group).

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[69] The CalEPA dose-response slope factors were derived by applying Poisson regression to relative risks presented in Hayes et al. (1997) and were based on an analysis of a subset of the Chinese Worker Cohort (representing approximately 76 percent of the total person-years at risk) for which exposures remained relatively constant over their work experience, making their exposure assignments less uncertain (CalEPA, 2001). We selected the dose-response slope factor that assumed a linear dose-response function for extrapolation to low doses, as the data was not inconsistent with a linear model. In addition, EPA's *Guidelines for Carcinogen Risk Assessment* (USEPA, 2005b) state that linear extrapolation should be used when the mode of action is uncertain, which is the case for benzene. Given the low concentrations that are likely to be experienced in our case study, a linear approximation may be a reasonable fit, even if the overall dose-response function in supralinear, provided the data from which the extrapolation is being made are not in the plateau region of the curve. In this case, the linear slope might be too shallow, underestimating the true dose-response relationship at low doses. To address this, the CalEPA analysis excluded data points expected to be in the plateau region of the curve.

[70] The source of the AML and all leukemia national incidence rates was the Center for Disease Control (CDC) WONDER online database. See http://wonder.cdc.gov/.

To estimate avoided cases of AML, we multiplied the leukemia incidence rates by ¼ and ran the model using the dose-response slope factor derived using AML as the health endpoint in Crump (1994). We found that the incidence results for AML were 70 percent of the all leukemia results. Therefore, we would expect a total of three avoided cases of AML (fatal and non-fatal) between 1990 and 2020 due to CAAA-related changes in benzene exposure. Ten-year survival rates for AML are approximately 20 percent. These data can be used as an approximation for how many cases are expected to be fatal and non-fatal. Therefore, we would expect that of the three avoided cases of AML, approximately two would be fatal and one would be non-fatal.

Alternate Exposure Matrix

Exposure assessment for the Pliofilm Cohort has been investigated by three separate research groups, Rinsky et al. (1981 & 1987), Crump and Allen (1984), and Paustenbach et al. (1992), yielding a variety of results. The different exposure assessment results of these three analyses can be attributed to various assumptions made by the investigators in relation to exposure of the workers, such as exposure concentrations experienced before sufficient monitoring data was available. Paustenbach et al. estimates are the highest, followed by Crump and Allen, and then Rinsky et al. Accordingly, the Paustenbach et al. estimates yield lower relative risks than the other two exposure estimates.[71] These dose-response slope factors assumed the same health endpoint (all leukemia) and lag (weighted) as the primary estimate. We found much lower health benefits using the Paustenbach exposure estimates, with only two cases of leukemia avoided between 1990 and 2020.

Alternate Lag

Our primary estimate relied on a "weighting" scheme to calculate a cumulative exposure value, with the peak weight being applied 5.3 years prior to the current year as an estimate of the latency period for leukemia. We also ran the model using alternative risk models that assumed a different lag structure. Because the lag structure is an integral part of how the risk coefficient is estimated in the benzene epidemiological analyses, different lag structures also imply different risk coefficients. We applied two models from Crump (1994), one derived assuming that all previous exposures were weighted equally (with no lag) and the other derived assuming all previous exposure were weighted equally with the exception of the most recent five years, which were weighted with zero. In addition to applying the alternative dose-response slope factors from these risk models, we also applied the corresponding exposure weights from each model to the exposure values from

[71] The estimates by Paustenbach et al. (1992) have been criticized for being based upon worst-case assumptions for the exposure scenarios that existed during the early years of the cohort (Utterback and Rinsky, 1995). In fact, critics have noted that prolonged exposure to the high levels of benzene estimated by Paustenbach et al. would have resulted in much higher prevalence of benzene poisoning than was actually seen in the cohort. Nevertheless, we performed a sensitivity analysis using dose-response slope factors from the Crump (1994) analysis derived using the Paustenbach exposure matrix to test the model's sensitivity to this input.

HAPEM6.[72] The dose-response slope factors associated with the zero- and five-year lags are lower than the dose-response slope factor used for the primary estimate (0.017 versus 0.84), in part because the weighted exposure values for these lag models are considerably higher than for our main model. The effect of the lower coefficient counteracts the effect of the shorter lags, and apparently has a greater impact; the results we found for these alternate lags were lower than the primary estimate. The zero-year lag model run yielded an estimate of two avoided cases between 1990 and 2020 and the five-year lag yielded an estimate of one case.

Discount Rate

We also estimated total monetary benefits using alternative discount rates of 0, 3, and 7 percent, as described in Section 2.5.2. The results of this sensitivity analysis are presented in Table 9 and range from $4.9 – 7.1 million for the high discount rate to $19 – 27 million when no discount rate is applied.

TABLE 9: TOTAL BENEFITS DUE TO CAA-RELATED CHANGES IN BENZENE OCCURING WITHIN THE STUDY PERIOD, CALCULATED WITH ALTERNATIVE DISCOUNT RATES

DISCOUNT PERCENTAGE	TOTAL BENEFITS (1990 NPV, MILLIONS OF2006$)		
	BENEFITS FROM FATAL CASES OF LEUKEMIA	BENEFITS FROM NON-FATAL CASES OF LEUKEMIA	TOTAL BENEFITS
Primary Estimate (5%)	**$6.7**	**$0.3 - 3.3**	**$7.0 - 10**
No Discounting	$18	$0.9 – 9.0	$19 – 27
Low Discount Rate (3%)	$9.8	$0.5 – 4.9	$10 – 15
High Discount Rate (7%)	$4.7	$0.2 – 2.3	$4.9 – 7.1

VSL

We selected a VSL of $7.4 million in 1990 (2006$) for our primary estimate, from a 2003 meta-analysis of wage-risk studies by Viscusi and Aldy (Model 5 from Table 8). To explore the sensitivity of the results to this assumption, we calculated the economic benefits using the following alternative VSL estimates:

- An alternative estimate from Viscusi and Aldy (2003) (Model 2 from Table 8) that assumes a log-normal distribution with a mean of $5.8 million (in 2000$);
- The estimate used in the recent PM NAAQS RIA assuming a normal distribution with a mean of $5.5 million (in 2000$); and
- An estimate used by EPA in past benefits analysis assuming a Weibull distribution based on 26 studies, with a mean of $4.8 million (in 1990$).

[72] For example, for the five-year lag, we applied a weight of 0 to the most recent five years of exposure and a weight of 1 to all other past exposures within the study period.

The total benefits estimated using these alternative VSL estimates, converted to 2006 dollars, are displayed in Table 10 below.

TABLE 10: TOTAL BENEFITS DUE TO CAA-RELATED CHANGES IN BENZENE OCCURING WITHIN THE STUDY PERIOD, CALCULATED WITH ALTERNATIVE VSL ESTIMATES

VSL	TOTAL BENEFITS (1990 NPV, MILLIONS OF2006$)		
	BENEFITS FROM FATAL CASES OF LEUKEMIA	BENEFITS FROM NON-FATAL CASES OF LEUKEMIA	TOTAL BENEFITS
Primary Estimate (Viscusi and Aldy, 2003, Model 5)	**$6.7**	**$0.3 - 3.3**	**$7.0 - 10**
Viscusi and Aldy, 2003, Model 2	$6.2	$0.3 – 3.1	$6.5 – 9.3
Normal Distribution	$5.9	$0.3 – 2.9	$6.2 – 8.8
Weibull Distribution	$6.7	$0.3 – 3.3	$7.0 – 10

Fatality Rate

In our primary estimate, we assumed that the difference between running the model with incidence data and mortality data constituted the number of leukemia cases that would be non-fatal. We found that of the four avoided cases of leukemia that would occur between 1990 and 2020, two would be fatal and two would be non-fatal (i.e., a 50 percent fatality rate). Although ten-year survival data for 1988-2004 presented on the SEER website supports this (the data indicate a 60 percent fatality rate within ten years), it is possible that those that survive ten years could come out of remission and eventually die of leukemia. In order to test the sensitivity of the results to this assumption, we calculated an alternate estimate of the monetary benefits assuming that all cases were fatal. We found that the total monetary benefits would increase to $13 million (in 2006$) using a five percent discount rate.

Summary

Table 11 displays annual avoided cases (fatal and non-fatal) of leukemia by study year and total cumulative cases occurring within the study period for the primary estimate as well as estimates for the sensitivity analyses. Total avoided cases between 1990 and 2020 for the primary estimate is four and the sensitivity analyses range between one and seven. Figure 8 presents the annual avoided cases of leukemia between 1990 and 2020 for the primary case as well as five of the sensitivity analyses in graphical form.

We also assessed the economic benefits associated with the avoided cases of leukemia for the sensitivity analyses. Table 12 below presents the total monetary benefits (for both fatal and non-fatal cases of leukemia) for the primary case as well as the sensitivity analyses.

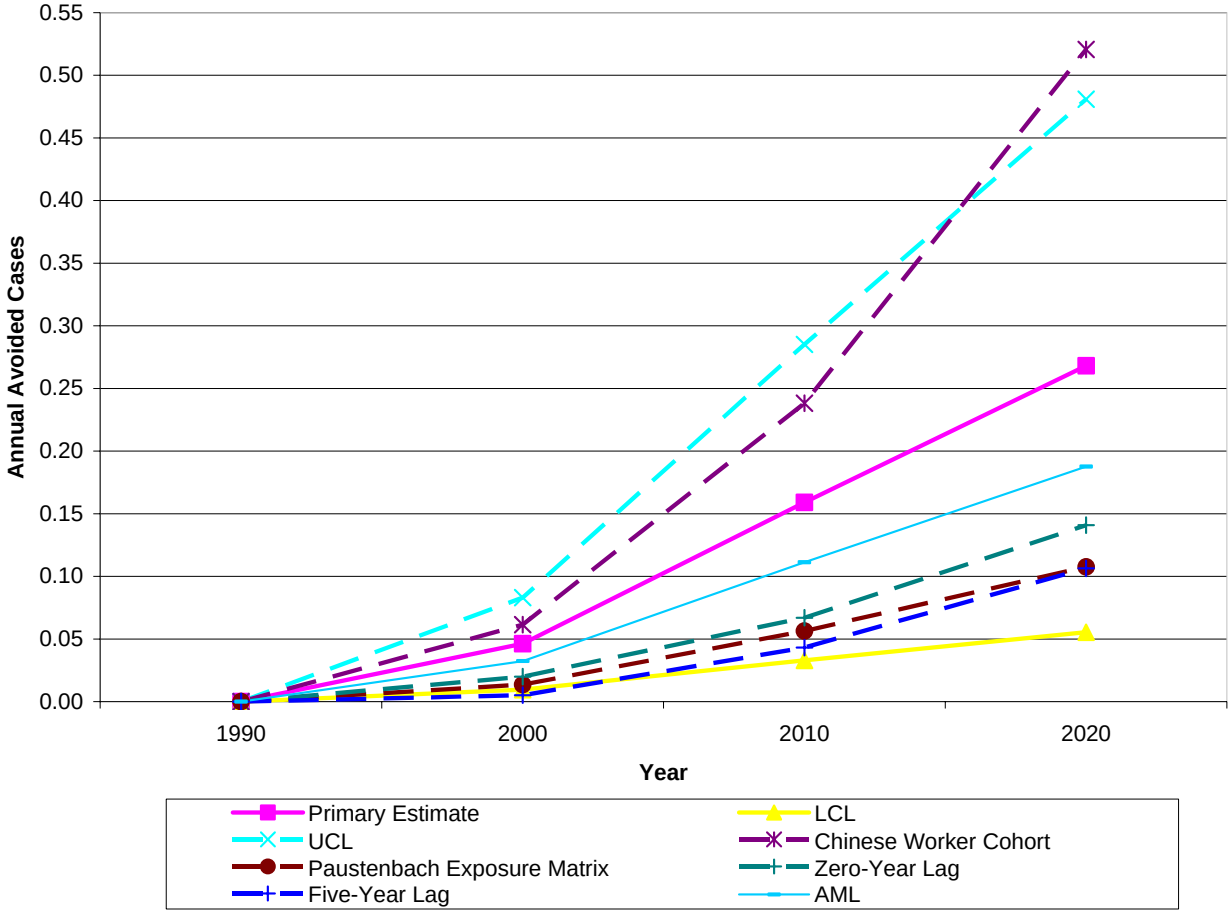
TABLE 11: TOTAL AVOIDED CASES OF LEUKEMIA DUE TO CAAA-RELATED REDUCTIONS IN BENZENE IN THE HOUSTON AREA – PRIMARY ESTIMATE AND SENSITIVITY ANALYSES RESULTS

YEAR	PRIMARY ESTIMATE	LCL	UCL	CHINESE WORKER COHORT	PAUSTENBACH EXPOSURE MATRIX	ZERO-YEAR LAG	FIVE-YEAR LAG	AML
1990	**0**	0	0	0	0	0	0	0
2000	**0.05**	0.01	0.08	0.06	0.01	0.02	0.01	0.03
2010	**0.2**	0.03	0.3	0.2	0.06	0.07	0.04	0.1
2020	**0.3**	0.06	0.5	0.5	0.1	0.1	0.1	0.2
Total Cumulative Cases	**4**	0.8	7	7	2	2	1	3

TABLE 12: TOTAL MONETARY BENEFITS OF CAAA-RELATED REDUCTIONS IN BENZENE IN THE HOUSTON AREA – PRIMARY ESTIMATE AND SENSITIVITY ANALYSES RESULTS (IN MILLIONS OF 2006$)

PRIMARY ESTIMATE	LCL	UCL	CHINESE WORKER COHORT	PAUSTENBACH EXPOSURE MATRIX	ZERO-YEAR LAG	FIVE-YEAR LAG	ALL FATAL
$7.0 - 10	$1.5 – 2.0	$13 - 18	$11 – 16	$2.5 – 3.6	$3.2 – 4.6	$2.0 – 2.8	$13

FIGURE 8: ANNUAL AVOIDED CASES OF LEUKEMIA DUE TO CAAA-RELATED REDUCTIONS IN BENZENE IN THE HOUSTON AREA – PRIMARY ESTIMATE AND SENSITIVITY ANALYSES RESULTS



Note: We have linearly interpolated between the avoided leukemia estimates for each target year; however, the true shape of the curve between each of these points is uncertain.

CHAPTER 4 | DISCUSSION AND CONCLUSIONS

This chapter discusses the key findings of this case study and the uncertainties associated with its results. It also presents an assessment of the strengths and limitations of the modeling approach used in this analysis and its implications for potential future assessment of the benefits of HAP controls.

4.1 KEY FINDINGS

This case study demonstrates that the 1990 CAAA controls on benzene emissions are expected to result in reductions in the incidence of leukemia in the greater Houston area over the period 1990 to 2020. Key findings include:

- CAAA programs are expected to reduce benzene emissions across all source categories in the study area by thousands of tons per year, with the largest reductions in the point and non-point source category, followed by on-road and non-road sources;
- The largest reductions in benzene exposures are expected to occur in downtown Houston and the surrounding area, and in two areas with significant point sources: the Texas City area of Galveston County and southeastern Brazoria county;
- Reductions in benzene levels are expected to continue, and hence benefits are expected to increase in the latter decades of the study period, as engine and other capital stock turns over and the impact of CAAA controls on on-road and non-road mobile sources in the area increases;
- Primary benefit estimates indicate four fewer cases of leukemia would occur in the three-county area in the study period, two of which we expect would have been fatal. We also expect benefits from the benzene changes that occur between 1990 and 2020 will continue accruing through at least 2030, potentially avoiding another two leukemia cases between 2020 and 2030. We estimate the net present value (NPV) in 1990 of the two fatal and two non-fatal leukemia cases avoided to be between $7.0 – 10 million in 2006 dollars, using a five percent discount rate.
- 1990 CAAA controls on benzene are expected to significantly reduce individual leukemia risk levels for those living in census tracts with the highest estimated benzene levels by one to two orders of magnitude. For example, median risks in Brazoria County drop from an increased lifetime leukemia risk of 2 in ten thousand (i.e., 2×10^{-4}) to 3 in a million (3×10^{-6}). In four of the six census tracts with the highest risks, individual lifetime leukemia risks are reduced by at least 80 percent.

- Additional health benefits may accrue to individuals living in homes with attached garages. Back-of-the-envelope estimates of the benefits of CAAA-related benzene reductions in the garages of these homes suggest these benefits may be similar in magnitude to our primary estimate. Therefore, these results suggest that adding attached garage-related benefits to our primary estimate could result in an approximate doubling of our primary estimate.

Although the actual benefit results appear modest, we note that leukemia is a rare disease with a low baseline rate among the population - for people under 50, the baseline risk in the study area was generally less than 5 in 100,000. Therefore, even significant percentage reductions in the baseline leukemia mortality rate may translate to relatively small numbers of avoided cases. We also note that the cases avoided are associated with only three U.S. counties containing just over one percent of the total U.S. population. We would expect significantly higher numbers of leukemia cases avoided when looking nationally at benzene reductions.

4.2 UNCERTAINTIES AND DATA GAPS

The results of this case study reflect limitations in available data and resources for conducting this analysis, as well as in the models and assumptions inherent in our analysis. Where feasible, we have conducted quantitative analysis to estimate potential impacts of these uncertainties; in other cases, we discuss qualitatively the source of uncertainty and our best estimate of the direction and size of its potential impact. We believe that overall, the uncertainties in our analysis are likely to cause our results to be underestimated.

We reach this conclusion for several reasons. First, the apparent systematic underestimation of benzene levels throughout the study area, due to upstream uncertainties in emissions and air quality modeling, constitutes one of the most major biases affecting our results. Further down in the analytical chain, additional factors contributing to a downward bias include the exclusion of a number of potential benzene-related health endpoints that we were unable to quantify for this case study; the exclusion of a ME for attached garages in the exposure modeling step, and uncertainties in the appropriate model for the C-R relationship between benzene exposure and leukemia. We describe the potential uncertainties of the study in greater detail below.

4.2.1 ESTIMATION OF BENZENE LEVELS

We believe that modeled benzene levels in this case study on average underpredict true ambient levels. Comparison of modeled ambient benzene levels from the *With-CAAA* AERMOD run for the year 2000 with observed monitor results in Appendix B shows a significant fraction of results are less than half of the observed values. These low results may be due to uncertainty or bias in emissions estimates, in air quality modeling, or a combination of the two. We discuss below the uncertainties we believe are likely to have a more significant impact on results. For more detail on uncertainties in emissions and air quality and exposure modeling, please consult Appendices A and B, respectively.

On the emissions side, air quality studies in Southeast Texas (TexAQS I and II) have led researchers to conclude that there is a high level of uncertainty in the HG area point source VOC and air toxic contaminant emission estimates, especially for petrochemical facilities.[73] As discussed in Appendix A, TexAQS II in 2006 confirmed that inventories based on standard EPA emission factors significantly underestimated VOC emissions from petrochemical facilities; it found that ethane emissions from petrochemical facilities were underestimated in the 2004 TCEQ point source database by one or two orders of magnitude.[74] Airborne measurements of VOCs, including benzene, from TexAQS II further support this hypothesis – measurements as high as 50 ppb benzene were detected in the Houston Ship Channel area where many petrochemical facilities are located, concentrations which are not consistent with emissions reported in the area's point source inventories. Given the significant contributions of point source sector regulations to the overall benzene reductions observed in the case study, this has the potential to be a major source of bias. However, the true impact of this downward bias depends on how well the missing emissions would be controlled by CAAA-related regulations, such as the highly reactive volatile organic compound (HRVOC) rules initiated in the study area since 2000. If these rules are effective in reducing benzene emissions from fugitive emission sources, we would underestimate the benefits of the CAAA. In its review of this case study, the SAB (USEPA, 2008b) strongly emphasized the need for additional investigation into missing or underestimated HAP emissions categories for point sources in Houston or other major metropolitan areas with significant point source HAP emissions to help reduce this bias in future assessments.

Another source of downward bias in emissions is the omission from the *With*-CAAA scenario of industrial leak detection and repair reductions that are part of the Texas SIP for ozone. Because these programs have been adopted in order to reduce fugitive VOC emissions that have not been captured in the VOC emission inventory for the study area, their emission benefit is difficult to model. Emissions for source categories affected by LDAR rules are likely underestimated. Additional research into the potential magnitude of these emissions reductions would benefit future studies. Of the other categories of benzene emissions controls that were not included in our analysis, the most significant is likely the set of controls associated with the MSAT program, which was established after the *With-CAAA* scenario was fixed.[75]

Reduction of benzene emissions in the mobile source category constitutes another significant contribution to CAAA benefits. Comparison of the base on-road inventory we used (the 2002 NEI on-road inventory) with the on-road emissions from the 1999 NEI showed significantly lower emissions in our inventory (about 760 tpy) than those from

[73] See, for example, Ryerson et al., 2003, Kleinman et al., 2002, and Allen and Durrenberger, 2003.

[74] For more information on TexAQS II, see http://www.tceq.state.tx.us/nav/eq/texaqsII.html.

[75] Other control categories not addressed by this case study include portable fuel containers, which may contribute to attached garage-related exposures, and new evaporative emissions categories such as tank and hose permeation included in the most recent NONROAD model (NONROAD2005). We also note that cold temperature start emissions for Tier 1 and later vehicles are underestimated by MOBILE6 (USEPA, 2007a); however this is not likely to be a major factor in the warmer Houston climate.

the 1999 NEI (about 1,940 tpy). Further investigation of this discrepancy identified three major contributors: use of local input data for the vehicle registration distribution for the 2002 inventory, revised 2002 summer fuel benzene levels, and reductions from control programs between 1999 and 2002 (Cook, 2007). This comparison illustrates that our results are highly sensitive to fleet distribution and fuel benzene content assumptions. While it is possible this may also contribute to the downward bias, we believe the selection of the 2002 inventory to generate on-road emissions was reasonable given its use of local, rather than national, registration data and its use of more up-to-date data and assumptions.

On the air quality modeling side, model-to-monitor comparisons suggest our AERMOD runs may have underestimated ambient benzene concentrations in 2000, as more than a quarter of the estimates are less than half the corresponding monitor values. If the air quality modeling systematically underestimated concentrations for both scenarios, it is possible that the difference between the two scenarios may also be underestimated, biasing our benefits estimates downward. If the size of the modeling error is approximately constant, the error would be subtracted out when we calculate the difference between the two scenarios and would not affect our results. If however, the error is proportional to the magnitude of the concentration modeled, then the error could result in an underestimate the difference between the scenarios.[76]

As noted by the SAB during its review of this case study (USEPA, 2008b), the lack of modeling of benzene concentrations during calm periods ("calms"), when high exposures are expected to occur, is likely a contributor to the observed downward bias. AERMOD by design is unable to estimate concentrations during calms (i.e., zero wind speed), and there is some uncertainty related to how well AERMOD performs when one substitutes a very low wind speed (e.g., less than 1m/s) for a calm. EPA is continuing to investigate approaches to address this issue. EPA is also considering for future analyses means of integrating multiple years of meteorological data into the air quality modeling step; this will help address potential uncertainties associated with using a single year's meteorological data to model conditions across multiple target years.

The modeling of non-point/area sources may also play a role. When compared against the 1990 base year AERMOD run, the average benzene concentration attributed to non-point/area sources in the 2000, 2010, and 2020 *Without-CAAA* runs appears to decrease, despite greater non-point/area source emissions in each of those years (see Appendix B). These results appear to reflect the sensitivity of the air quality modeling to differences between the surrogate data used in the 1990 model run to allocate non-point/area source emissions and the surrogate data from 2000 used in all the future year model runs. This is a potentially significant source of uncertainty; if the 2000 allocation surrogate data *Without-CAAA* allocate area source emissions in such a way that the dispersion model systematically underestimate concentrations from area sources in the *Without-CAAA*

[76] This would occur because the benzene concentrations in the *Without-CAAA* scenario are typically higher than those in the *With-CAAA* scenario. If the downward bias is proportional to the concentration, the *Without-CAAA* value would be more significantly underestimated than the *With-CAAA* value, resulting in a smaller than expected difference between the two values.

scenario, our benefits estimates could be underestimated. Because the 2000 allocation is based on more recent data, we believe it is likely more accurate than the 1990 allocation. However, we note that the 2000 allocation surrogate has not yet been validated. Use of a consistent set of validated surrogates is recommended for future assessments.

On the exposure assessment side, there are a limited number of microenvironments included in the HAPEM6 model; as a result, we were unable to estimate benefits expected to occur in certain high-exposure microenvironments such as service stations and homes with attached garages. As a result our benefits may underestimate benefits that occur in these microenvironments. In a supplemental back-of-the-envelope calculation of the magnitude of benefits to those living in homes with attached garages, we estimated benefits of similar magnitude to our primary estimate in 2020. Future analyses would benefit from collecting improved data on the benzene exposures due to attached garages, and from exploring the proportion of benzene exposure risk attributable to indoor sources to provide an overall public health context.

4.2.2 HEALTH BENEFITS MODELING AND VALUATION

Uncertainties related to health benefits modeling and valuation include the following:

- We only quantified health benefits due to avoided cases of leukemia. Other health endpoints associated with benzene exposure that are biologically plausible but lacked sufficient data to quantify a dose-response relationship include other cancers, such as Hodgkin's Lymphoma, and non-Hodgkin's Lymphoma, multiple myeloma, and myeodysplastic syndrome as well as potential non-cancer effects. Therefore, our results do not provide a comprehensive estimate of health benefits from benzene reductions in the Houston area. The SAB (USEPA, 2008b) specifically recommended examining recent studies linking benzene and non-Hodgkin's lymphoma for future benzene benefits analyses.
- We obtained the widest range of benefits from our model (between 0.8 and 7 avoided cases of leukemia) by applying the model to the bounds of the 95 percent confidence interval around our primary dose-response coefficient. Our model is also sensitive to alternative assumptions about dose-response and cessation lag models for benzene-induced leukemia. Sensitivity analyses show that our results can vary by plus 66 percent to minus 81 percent, depending on the choice of cohort study (Pliofilm vs. Chinese Worker), exposure matrix (Crump and Allen versus Paustenbach), health endpoint (total leukemias vs. Acute Myelogenous Leukemia (AML)), or risk/lag model.
- The leukemia cohort studies are based on occupational exposure levels. Extrapolation of the dose-response function to ambient environmental levels requires an assumption of the shape of the function in the observable range. While we have assumed a linear function, as described in Chapter 2 and Appendix C, there is some evidence to suggest the function may be supra-linear; if so, we will have underestimated the benefits of CAAA benzene reductions. Additional research, both epidemiological and toxicological, can help further our

understanding of the mode of action of benzene and will help analysts better ascribe probabilities to the alternative functional forms.

- We have applied the relative risk model derived from Crump 1994 to all age groups; however the risk estimates were derived from an occupationally exposed cohort of adults. We may under- or overestimate risk to age groups not included in the cohort if their true relative risk is higher or lower, respectively, than that of the age groups in the worker cohort.[77]
- Application of risk estimates derived from an occupational epidemiological study to the general population typically underestimates risks to that population because the population studied was on average healthier than the general population (i.e., the "healthy worker" effect; Hennekens and Buring, 1987). Because we apply the leukemia risk estimate without adjustment for this effect, the healthy worker effect will tend to bias our results downwards.
- We assumed our linear dose-response model exhibited no threshold (i.e., no exposure level below which no effect would be observed). As discussed in Appendix C, there exists some limited evidence suggesting that a threshold may exist; if the true model exhibits a threshold, our results would be biased upward. The degree of bias would depend on the location of that threshold.
- Our approach for quantifying non-cancer health effects resulting from benzene exposure relied on the RfC reported in IRIS. More recent studies have reported decreased lymphocyte count at benzene concentrations lower than the RfC. Therefore, it is possible that CAAA controls may have resulted in reductions in non-cancer effects in the study population that are not quantified in our analysis.
- Our primary monetized benefit results are highly sensitive to the discount rate applied, because the cessation lag effect delays the full realization of health benefits.[78]
- The VSL value we applied ($7.4 million in 2006$) is a central estimate from a distribution of values obtained from the benefits valuation literature. Use of alternative values from this distribution would scale our monetized benefits accordingly. However, this VSL distribution does not reflect any additional willingness-to-pay to avoid the additional pain and suffering associated with a cancer-related death, and is not included in the pre-mortality morbidity estimate we add to the VSL. To the extent individuals would pay more to avoid cancer-related pain and suffering prior to death, we are underestimating the value of the avoided leukemia cases (i.e., our results do not incorporate a "cancer premium").

[77] We used the same relative risk estimates for all groups in this analysis. Because benzene's MOA has not been established at this time, we did not apply the age dependent adjustment factors (ADAFs) recommended in the Supplemental Guidance for Assessing Susceptibility Early-Life Exposures to Carcinogens (USEPA, 2005b) for chemicals with a mutagenic MOA. Early-life adjustments could be explored in a future case study.

[78] Alternative risk models with shorter lags are less sensitive to choice of discount rate, because benefits of exposure reductions will be realized sooner.

Additional studies addressing this issue would significantly benefit HAP analyses, since many of these compounds exhibit carcinogenic effects.

- As noted in Chapter 2, valuation estimates for non-fatal cancers are quite limited. While the approach we employed does build on precedent from past regulatory analyses to generate a willingness-to-pay (WTP) estimate, this estimate consists of two data points, only one of which represents WTP to avoid a case of cancer and neither or which specifically addresses leukemia. Additional research is needed to develop WTP estimates for leukemias and other non-fatal cancers.

4.3 IMPLICATIONS FOR FUTURE ANALYSIS

This case study has demonstrated a benefits methodology that can be used to assess the health impacts of changes in benzene concentrations in an urban area. As EPA moves forward in its development of benefit analysis tools for HAPs, it should consider the potential role of this methodology in more broadly documenting the effects of HAP regulation on health.

In 2001, Agency staff and members of the EPA SAB held a joint workshop to explore the issue of how to best estimate and value the benefits of HAP reductions. The workshop, which included experts in economics, health science, and risk assessment, engendered extensive discussion, but yielded no consensus as to the best methodology. Participants were divided over the use of traditional damage-function approach, such as the one applied in this case study. The SAB workshop report cites a number of obstacles to this approach, including limited, often contradictory, health data; difficulty assessing the effects of multiple exposures; uncertainties in extrapolating from animals to humans and from high doses to low doses; and limited resources to evaluate a large number of chemicals (USEPA SAB, 2002b). The workshop concluded with recommendations for two research directions: one pursuing the demonstration of the damage-function approach for a well-studied HAP and the other pursuing alternative approaches suggested at the meeting, such as assessing the value of HAP regulation as an insurance policy or assessing the value of shifts in the curve of a population's onset of disease (USEPA SAB, 2002b).

This study provides insights into the strengths and limitations of a damage-function approach. Specific strengths of the methodology applied in this case study include:

- It provides a comprehensive assessment of the impact of benzene controls from multiple CAAA Titles on cancer incidence in an urban population;
- It uses a combination of national and local data to develop emissions inventories cost-effectively, which include improved resolution link-level mobile source emissions estimates;
- It assesses exposure using EPA's HAPEM model, which combines air quality modeling output from AERMOD with local activity pattern (e.g., commuting) data to generate both more realistic, age-specific estimates of exposures at the census tract level and probabilistic distributions of exposure that reflect interpersonal variability in exposure;

- It generates health benefit estimates based on central, rather than upper-bound, estimates of cancer potency, which is more appropriate for regulatory analysis;
- It applies a life-table model which allows for the assessment of the CAAA benzene controls on the population over time, using the age-specific HAPEM exposure estimates and local, age-specific baseline incidence rates to generate estimates of local health impacts by census tract;
- It simplifies the consideration of cessation lag by integrating it directly into the life-table model, which uses a damage-function based on weighted exposures; and
- It generates monetized estimates of avoided cancer cases, both fatal and non-fatal, using current EPA guidance on VSL estimates for cancer.
- It uses a modular approach to the analysis, which provides opportunities for scaling the level of complexity of the analysis in accordance with needs and resources.

Specific limitations of the methodology and drawbacks to wider application include:

- The damage-function approach requires both significant resources and extensive data sets to perform local-scale modeling;
- The number of HAPs with a sufficient toxicological database in terms of number and quality of studies and weight of evidence to support this type of health benefits modeling remains limited;
- Use of the model with HAPs other than benzene may require additional effort to estimate a central-estimate dose-response function from available data, as many published toxicological values for other HAPs represent upper bound estimates of potency or reference values that do not allow for quantitative risk assessment;
- The model has not yet been demonstrated for a non-cancer dose-response analysis.
- The critical effects associated with published non-cancer toxicological benchmarks for many HAPs may be difficult to value economically, because while they may serve as an indicator of an adverse biological process, the effects themselves may not necessarily be clinically significant (e.g. increased kidney weights); and
- Air toxics monitoring is more limited than criteria pollutant modeling, making it more difficult to conduct quality control model-to-monitor comparisons in some locations or for certain HAPs.

The drawbacks of applying this model more broadly are essentially the same as those cited in the 2001 workshop, though there have been some positive developments for HAP benefits assessment. For example, EPA's 2005 *Guidelines for Carcinogen Risk Assessment* encourages improved reporting of uncertainty in risk estimates, including central as well as high-end estimates. In addition, since 2002, EPA's IRIS database has

updated 23 toxicological summaries, 11 of which were for HAPs.[79] Unfortunately, insufficient data exist for most of these HAPs to assess their carcinogenic potency. One of the updated HAPs - 1,3-butadiene - is classified as carcinogenic to humans and does appear to have a sufficient database to support benefits analysis, including epidemiological results showing a dose-response relationship for leukemias in polymer workers in the U.S. (USEPA, 2007e). 1,3-Butadiene is one of the 12 regional cancer risk drivers identified in EPA's 1999 National Air Toxics Analysis (NATA) analysis (USEPA, 2001c), and therefore may be a good candidate for further analysis using this model.[80]

In order to apply the methodology to a non-carcinogen, additional effort would be required to develop a dose-response function for use with the health effects model. While the resulting function and estimated benefits would be uncertain, there is also significant uncertainty in the true impacts of exposures in a population simply characterized as being above the RfC. Experts have argued for a more parallel treatment of carcinogens and non-carcinogens (e.g., Clewell and Crump, 2005), and a recent paper by Woodruff et al. (2007) illustrated an approach to developing a dose-response model for acrolein, the one HAP identified as a risk driver of non-cancer effects at the national level in EPA's 1999 NATA.[81]

We believe future case studies should continue to provide both central estimates of population risk (i.e., estimates of cases of adverse health effects avoided) and estimates of individual risk reductions for highly exposed populations. The latter are particularly important, because the impacts of HAP emissions (and emission reductions) can be fairly localized, as seen in the substantial risk reductions in high exposure tracts in Brazoria and Galveston counties.

In an effort to ascertain how our benefits may compare to those estimated from a larger-scale analysis such as EPA's National Air Toxics Assessment (NATA), IEc attempted to conduct a reduced-form benefits analysis of CAAA-related benzene reductions in the three-county study area using benzene concentrations from the 1999 NATA and preliminary draft concentrations from the forthcoming 2002 NATA. However, we found the NATA results to be incompatible with our benefits model, because for many census tracts the NATA results (from both 1999 and 2002) exceeded both the with- and without-CAAA estimates from our case study. While we were unable to conduct a thorough investigation of the causes of these discrepancies, our initial efforts suggest that differences in year 2000 onroad benzene emissions are a contributing factor; additional contributors may be differences in air quality modeling (AERMOD vs. ASPEN), the

[79] See http://www.epa.gov/iris/whatsnew.htm and http://www.epa.gov/iris/whatsnewarch.htm for updated profiles. The 11 HAPs were vinylidene chloride (1,1-dichloroethylene); phenol; 1,3-butadiene; xylenes, benzene, methylisobutylketone, acrolein, toluene, hexane, phosgene, and 2,2,4-trimethylpentane.

[80] The NATA study identifies regional risk driver as carcinogens to which at least one million people are exposed at a risk level greater than 10 in one million or at least 10,000 people are exposed at a risk level greater than 100 in one million. See http://www.epa.gov/ttn/atw/nata1999/for the full list of cancer and non-cancer risk drivers.

[81] A national risk driver for non-cancer effects, as defined in the 1999 NATA, is a HAP for which at least 25 million people are exposed at levels above EPA's reference concentration. The study also identified 16 HAPs as regional drivers of non-cancer risk, defined as HAPs for which at least 10,000 people are exposed above EPA's reference concentration.

apparent gaps in our 1990 base year benzene emission inventory that contribute to underestimated benzene concentrations in 2000 and likely throughout the study (discussed earlier), and uncertainty in the benzene emissions growth factors used to generate the with- and without-CAAA scenarios for this case study. Future HAP analyses, whether at the urban-scale or using NATA would benefit from further investigations of the differences in these approaches, the associated uncertainties and data gaps and their potential impact on results.

Due to difficulties in applying the case study approach on a national scale or to extending it to other air toxics, which may have a limited epidemiological database, the SAB in its review of this case study (USEPA, 2008b) suggested that EPA also consider integrated multi-pollutant approaches to estimate the benefits of air toxics regulations. For example, OAR's Risk and Technology Review (RTR) program, which evaluates air toxics risk by source category. Another option could be the emerging 3D air quality modeling work that can include individual air toxics so that HAPs do not need to be modeled separately.

In conclusion, the methodology presented in this case study can serve as a useful tool in EPA's evolving HAP benefit assessment strategy. Determining where this approach best fits within that strategy will require additional analysis and evaluation to determine the added value of the detailed, urban-scale approach, as well as potential pool of HAPs suitable for assessment via the damage-function approach for cancer and/or non-cancer effects.

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