IEc

Quantifying and Valuing Cancer Risks Associated with Air Toxics Exposure

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INDUSTRIAL ECONOMICS, INCORPORATED

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California Air Resources Board

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LIST OF ACRONYMS

AEO	Annual Energy Outlook
ASAF	Age-specific adjustment factor
ATCM	Airborne Toxic Control Measure
ATS	AirToxScreen
ATSDR	Agency for Toxic Substances and Disease Registry
BAU	Business-as-usual
BCA	Benefit-cost analysis
BenMAP	Environmental Benefits Mapping and Analysis Program
CAPCOA	California Air Pollution Control Officers Association
CARB	California Air Resources Board
CCR	California Cancer Registry
CDC WONDER	Centers for Disease Control and Prevention Wide-ranging ONline Data for Epidemiologic Research
CDOF	California Department of Finance
CES	CalEnviroScreen
CI	Confidence interval
CMAF	Cancer mortality adjustment factor
CPF	Cancer potency factor
CSF	Cancer slope factor
DPM	Diesel particulate matter
EPA SAB	U.S. Environmental Protection Agency Science Advisory Board
FV	Future value
GDP	Gross domestic product
HAPEM8	Hazardous Air Pollutant Exposure Model, Version 8
IARC	International Agency for Research on Cancer
IEc	Industrial Economics, Incorporated

IPCS	International Programme on Chemical Safety
IUR	Inhalation unit risk
LCFS	Low Carbon Fuel Standard
MHAF	Mortality Hazard Adjustment Factor
MLE	Maximum likelihood estimate
NCI	National Cancer Institute
OECD	Organisation for Economic Co-operation and Development
OEHHA	California Office of Environmental Health Hazard Assessment
OPPT	Office of Pollution, Prevention, and Toxics
$PM_{2.5}$	Fine Particulate Matter (smaller than 2.5 microns in diameter)
PV	Present value
RAGS	Risk Assessment Guidance for Superfund
RR	Relative risk
SEER	Surveillance, Epidemiology, and End Results Program
SRIA	Standardized Regulatory Impact Assessment
TAC	Toxic air contaminant
TAF	Threshold adjustment factor
TRU	Transport Refrigeration units
TSCA	Toxic Substances Control Act
U.S. EPA	United States Environmental Protection Agency
VMR	Value of a fatal risk reduction of one-in-one million
VNR	Value of a non-fatal risk reduction of one-in-one million
VSL	Value per statistical life
WTP	Willingness to pay

AI Use Statement

Claude 4.x was used on a closed, internal system to review a draft version of this report for style, consistency, and grammar issues. Claude was also used for advice on Python code design and troubleshooting.

Chapter 1| Introduction

1.1 Project Overview

Industrial Economics, Incorporated (IEc) is supporting the California Air Resources Board (CARB) in its efforts to advance approaches used to quantify and value the cancer risks associated with exposure to toxic air contaminants (TACs, also known as air toxics or hazardous air pollutants).[1] In previous regulatory analyses, CARB has expressed benefits related to TAC control in terms of reductions in cancer risks (per million persons exposed) or as changes in populations exposed to particular risk levels. CARB is currently limited in its ability to express these benefits in monetary terms and is interested in developing approaches to TAC risk characterization that are appropriate for and amenable to economic valuation.

The objective of this project is to develop a peer-reviewed methodology that can support CARB quantitative economic health risk assessments at the state, regional, and local levels. To accomplish this objective, IEc is recommending methods that can help adapt CARB's standard cancer risk assessment methods to produce values more compatible with the framework of regulatory benefit-cost analysis. IEc is also synthesizing and adapting economic literature on the value of avoided cancer cases to monetize these benefits. Where possible, IEc is leveraging local data and building upon existing methods published in peer reviewed journals or conducted by established regulatory agencies and scientific bodies. These methods will supplement existing risk assessments conducted by CARB and California Office of Environmental Health Hazard Assessment (OEHHA).

1.2 Background: Risk Assessment

Risk assessment methods can vary depending on the risk management purpose they serve. For example, TAC risk assessment methods employed by CARB to evaluate risks from stationary sources will generate estimates of changes in a hypothetical individual's cancer risk over a lifetime so that these values can be compared against regulatory thresholds developed by CARB and the California Air Pollution Control Officers Association (CAPCOA) that have similarly been established on an individual lifetime basis (2015). Other risk assessments attempt to estimate changes in risk across a population, estimating the changes in the numbers of cases of death or illness in a population that may be expected to occur given a change in pollutant exposure (U.S. EPA 2024b). These latter types of risk assessment are useful when conducting a socioeconomic analysis, which "characterize[es] the economic value of changes in human health and environmental outcomes that result from different policy alternatives" (OECD 2017). Often in these analyses, the monetized value of the benefits of a pollution reduction strategy is compared against the implementation costs of that strategy, in part because there is a larger body of economic literature to help value avoided deaths, illnesses, hospitalizations, and other adverse health events. These analyses also tend to be more data intensive, requiring information about the composition and health of populations exposed as well as risk information derived from human studies.

Risk assessments of exposures to TACs have historically been conducted using the first risk assessment method. In the late 1990s and 2000s, U.S. EPA extensively explored ways in which to adapt TAC risk assessments to

[1] Within this document, we define air toxics according to the following quote from CARB's 2015 guidance, The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments: "The substances included on the Hot Spots Program list of substances are defined in the statute as those substances found on lists developed by the following sources:
- International Agency for Research on Cancer (IARC);
- U.S. Environmental Protection Agency (U.S. EPA);
- U.S. National Toxicology Program (NTP);
- ARB Toxic Air Contaminant Identification Program List;
- Hazard Evaluation System and Information Service (HESIS) (State of California);
- Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986) list of carcinogens and reproductive toxicants (State of California);
- Any additional substance recognized by the State Board as presenting a chronic or acute threat to public health when present in the ambient air."

better evaluate the economic impacts of TAC-control as part of its series of Section 812 benefit-cost analyses of the entire Clean Air Act and its amendments (U.S. EPA 2011). While this effort did result in advancements, TAC risk assessments, including those conducted by CARB, still tend toward the former approach, in part because of data limitations for many TACs. However, researchers and analysts continue to advance risk assessment methods that could be used to bridge the gap between the individual-risk type assessments and the generation of scientifically and economically supportable valuation estimates. We highlight two key developments below:

Organization for Economic Co-operation and Development (OECD) working paper on risk assessment methods. While an analyst could simply multiply the exposed population by the changes in maximum individual risks estimated using the current TAC risk assessment approach in order to generate a crude estimate of population impacts, this approach would ignore differences in these methods that affect suitability for conducting an economic valuation. As highlighted by IEc Expert Advisor Dr. Weihsueh Chiu in his 2017 OECD paper, adapting traditional chemical or air toxic risk assessments for use in socioeconomic analysis requires additional steps that help to address these methodological differences by:

- Providing reasonable estimates of central tendency for exposure and cancer response to pollutants, rather than focusing on highly exposed or sensitive individuals;
- Hazard identification focused on well-supported and non-overlapping adverse health impacts that can be valued using current economic methods;
- Incorporating probabilistic methods to separate and characterize uncertainty and interindividual variability in exposure estimates and concentration-response relationships, to ensure that variability in the population is reflected in results and to accurately express what is currently known about the inputs to the risk assessment; and
- Expressing risk as a time series of annual changes in the frequency and intensity of health effects.

Valuation applicable to individual risks. In addition to modifying risk assessment methods to estimate population risk, Abt Global and U.S. EPA have in recent years developed an approach that produces unit values for small changes in cancer risks (Abt Global 2022, LaPenta et al. 2024). This approach folds some of the changes recommended in the OECD paper into the derivation of these unit values, particularly with respect to modeling impacts as a time series of risk reductions and mortality impacts. This method has been used by U.S. EPA to develop monetized estimates of risk reductions under the Toxic Substances Control Act (TSCA) for pollutants such as methylene chloride (U.S. EPA 2023).

The methodology described in this document includes the following features that build on these advances and other relevant risk assessment and economic research:

- **Central estimate of exposure and potency.** It emphasizes that the primary estimates of risk for socioeconomic analysis should be based on characterization the expected or typical values in a population for both exposure and cancer potency, instead of values for those more highly exposed or sensitive. These can characterized using mean or median values of the distributions of risk assessment input parameters.
- **Incorporation of variability and uncertainty.** This methodology also recognizes the need to represent the range of possible exposures and susceptibility in the population plus uncertainty in cancer potency when developing valuation estimates. It facilitates the inclusion of quantitative characterizations of these elements using probabilistic modeling of the distributions of exposure and cancer potency in the population, as well as statistical uncertainty in economic values for cancer risk reductions. Results of the methodology should include both the central point estimate and the estimates reflecting variability and uncertainty.

- **Distribution of benefits over time.** Aggregated lifetime measures of risk are less amenable to valuation, because little information is provided about when over one's lifetime that risk change occurs and because the concept of lifetime can vary depending on one's age when an exposure change occurs. To better align these results with valuation estimates, this methodology employs peer-reviewed approaches used by U.S. EPA to distribute how the calculated reduction accrues over time for individuals of different ages. This process reflects inherent delays in the way population risks change following a change in exposure, due to factors such as the lag between exposure to a pollutant and diagnosis of cancer, or, where cases are fatal, between diagnosis of cancer and death. This methodology uses historical incidence data of cancers by age to model the lag effect. It then uses historical data on survival rates to estimate both the fraction of cancers that are fatal and the timing between diagnosis and death. These adjustments are critical for properly valuing the benefits of air regulations using the economic concept of discounting; while we understand that CARB prefers to present undiscounted estimates in their analyses, this approach will preserve the option for CARB to include discounted estimates if desired (e.g., as sensitivity analyses).
- **Use of valuation methods consistent with criteria air pollutant analyses.** This approach uses valuation methods consistent with those applied for analyses of pollutants such as fine particulate matter, with values based on the latest peer-reviewed economic literature.

In addition, the framework for this methodology:

- **Provides flexibility.** The methodology includes options for CARB that promote efficient and cost-effective analyses, rather than an approach that emphasizes high degrees of quantification in all cases.
- **Incorporates California-specific data,** including baseline cancer incidence rates stratified by county, race, and ethnicity; and state-specific inflation, income growth, and population projections.
- **Supports generating data for environmental justice analysis.** The methodology described in this document can generate stratified data for individual population subgroups, particularly when lag and survival timing is considered.

We emphasize that the risk and valuation calculations recommended in support of socioeconomic analysis serve a separate purpose than risk analyses currently conducted by CARB; thus, calculations recommended in this methodology should be considered supplemental to CARB's current practice.

1.3 Background: Health Valuation

While risk assessments for TACs exposure typically aim to quantify human health risks (e.g., changes in cancer risks), CARB analyses may also require some measure of the economic value of these changes in human health risks. Regulatory benefit-cost analysis seeks to provide decision-makers with an understanding of whether, and to what extent, the anticipated benefits of a proposed policy exceed the costs of implementing the policy. In this context, values for health-related benefits are ideally based on estimates of individuals' willingness to pay (WTP) for the risk changes they are likely to experience (Boardman et al. 2018, U.S. EPA 2024c, Robinson and Hammitt 2015). For measuring the benefits of reduced TACs exposure, WTP would reflect the maximum amount an individual would be willing to pay to reduce their own risk of illness or death.

Because risk reductions are not directly bought and sold in the marketplace, nonmarket methods must be used for valuation. WTP estimates may be derived using stated preference or revealed preference techniques. In stated preference studies, researchers present survey respondents with hypothetical choices (e.g., purchasing decisions or proposed policies) that vary in their cost and risk level, among other attributes. These responses are used to infer individual WTP for decreasing the risk of illness or death. In contrast, revealed preference studies leverage data from market behaviors to understand how individuals value changes in these risks. For example, wage-risk studies evaluate whether individuals are compensated more for participating in occupations with

higher risks of injury or death, controlling for other factors influencing compensation (e.g., age, education, years of experience).

The methodology proposed in this report includes the following elements:

- **Synthesis of WTP estimates for cancer risk reductions.** Utilizing a recent and comprehensive literature review by LaPenta et al. (2024), this framework provides a set of WTP estimates for a variety of non-fatal cancers. The report further discusses recommendations for valuing mortality risks using the value per statistical life (VSL) adopted by U.S. EPA and utilized by both CARB and LaPenta et al. (2024).
- **Adjustments for income growth and inflation.** The methodology will also cover adjustments to selected unit values for factors such as income growth and inflation. In the case of income growth, we will cover appropriate adjustments to WTP-based estimates, including recommendation of data series characterizing growth in real income and appropriate assumptions on how elastic WTP estimates are to income levels. We will also discuss adjustments for inflation, ensuring that recommendations are consistent with CARB practices in benefit-cost analysis.
- **Discounting.** An important element in the analysis of cancer risks is the lag between exposure and diagnosis, or the so-called "cessation lag." Given the potential for decades-long delays between exposures and material health benefits, this temporal dimension requires careful consideration when monetizing benefits to ensure the values reflect the present value of incident cases.
- **Central estimate and uncertainty.** This methodology provides WTP estimates for risk reductions associated with several cancer sites, as well as a general cancer estimate for unspecified or unknown sites. Our estimates are accompanied by probability distributions allowing for the quantitative characterization of uncertainty.

1.4 Report Organization

In the remainder of this report, we present our recommended approach for CARB to quantify and value cancer risks associated with TACs exposure. The following bullets provide an overview of the contents of each chapter:

- **Chapter 2 (Risk Assessment in the Context of Regulatory Analysis)** describes how risk assessment is used by regulatory agencies to inform analysis of proposed policy measures. We include a summary of CARB's current standard practices and compare these with alternative approaches for analyzing the benefits of air toxics controls.
- **Chapter 3 (Valuing Cancer Risk Reductions)** presents our proposed approach for valuing changes in fatal and non-fatal cancer risks. This chapter summarizes recent work published by LaPenta et al. (2024) and proposed adaptations by IEc to leverage California-specific data, provide central estimates for decision-making purposes, characterize uncertainty, and better match the risk assessment approaches recommended in subsequent chapters.
- **Chapter 4 (Methodology Overview)** includes a roadmap for IEc's proposed risk assessment approaches, including a decision tree for selecting from one of three approaches ranging in complexity and applicability to various TACs, based on elements such as the spatial extent of exposures and the design and findings of epidemiological and/or toxicological research.
- **Chapters 5 through 7 (Approaches I – III)** detail our proposed methodology for quantifying cancer risks attributable to TACs exposure. Each chapter provides CARB with guidance on when the methodological approach is most appropriate to apply and instructions on how to implement the approach.
- **Chapter 8 (Case Studies)** will include case studies illustrating the methods presented in this report in subsequent drafts.

In addition, we include three appendices. **Appendix A** contains further information on data sources and data aggregation; **Appendix B** contains detailed information on the process of incorporating uncertainty and variability into population risk; **Appendix C** contains a complete explanation of our methods for calculating weighted average one-in-one million cancer risk; and **Appendix D** contains further details on the process of deriving a pooled distribution of non-fatal WTP estimates. **Appendix E** provides details regarding the exposure modeling conducted for the case studies.

CHAPTER 2 | Risk Assessment in the Context of Regulatory Analyses

2.1 Key Considerations for TACs Risk Assessment

The previous chapter introduced key considerations for adjusting exposure assessment and risk characterization methods when generating risk estimates for use in socioeconomic analysis. In this chapter, we expand on these recommendations in the context of CARB TAC cancer risk analyses. Our recommended adjustments focus what Chiu (2017) refers to as "bridging" approaches: or a means to modify risk assessment methods to better reflect the types of risk assessments conducted in support of socioeconomic analyses. It allows for modifications that, short of reworking the entire analysis, help bridge the gap between the two analyses and generate values appropriate for the socioeconomic context. We discuss bridging approaches for each step of the risk assessment process below.

Hazard Identification. In the hazard identification step, CARB identifies the cancer-related hazard(s) associated with exposure to a given TAC. Chiu emphasizes identifying all health effects associated with exposure to the pollutant where the relationship has either been demonstrated as causal or where the likelihood of causality has been or can be quantified for purposes of a probabilistic analysis. Processes such as expert elicitation have been used to estimate these values (Roman et al. 2008), and the development of causal epidemiological methods is encouraging more quantitative characterizations. In practice, however, quantitative probabilities are unlikely to be available for most TACs. We recommend that CARB consult with OEHHA when selecting carcinogens for performing socioeconomic analysis.

Another element of hazard identification that should be modified in a socioeconomic analysis setting involves prioritizing health impacts that can be readily valued. In practice this means avoiding impacts that are too far removed from clinically diagnosable effects, such as changes in biomarkers or gene expression, which may be challenging for the public to value (Chiu 2017). The final consideration is to select health impacts carefully to avoid combining overlapping impacts (e.g., a risk estimate for all leukemias and a separate estimate for a specific leukemia subtype), which could result in double-counting (i.e., overstating) potential benefits. This seems unlikely to be an issue for most cancers associated with TACs, but if in doubt, a consultation with OEHHA staff is recommended.

Key Considerations – Hazard Identification

- Identify health effects that can be valued using peer-reviewed literature and quality data sources
- Focus on known or probable carcinogens with a good foundation of studies supporting causal impact; carcinogens with suggestive evidence could be considered for use in separate sensitivity analysis.
- Avoid cancer impacts with overlapping definitions that may lead to double-counting of benefits

Exposure Assessment. Generally, CARB uses spatially-gridded emissions changes and modeling of TAC fate and transport to produce estimated TAC concentrations and descriptions of the frequency and intake of the contaminant by maximally exposed individuals to estimate a lifetime average measure of dose. Bridging approaches that allow risk assessments to better support socioeconomic analysis may be more relevant for estimates of lifetime dose. Chiu emphasizes that socioeconomic analysis focuses on mean or expected values for the primary estimates that are reported, often combined with a quantitative characterization of variability in exposure in the population using bounding estimates or probabilistic distributions. Quantitative estimates of uncertainty are also recommended where possible. CARB will likely need to generate supplemental risk estimates based on adjusted central tendency exposure estimates and population variability when generating

values suitable for valuation. For population-scale exposure, we are recommending CARB apply U.S. EPA's Hazardous Air Pollutant Exposure Model, Version 8 (HAPEM8) model for this purpose, as described in Chapter 6 and Appendix A.1.

Key Considerations – Exposure Assessment

- Establish primary estimates for key exposed groups based on central tendency exposures, not conservative or high-end estimates.
- Focus on exposure factors and activity patterns to establish interindividual variability for exposed populations.
- Represent interindividual variability in exposure and uncertainty in exposure estimates quantitatively where possible to reflect full range of population experiences.

Dose-Response Assessment. CARB quantitatively characterizes the carcinogenic potency of exposures to a given TAC in the population. Higher potency corresponds with a higher likelihood that exposures result in carcinogenesis in exposed individuals and, consequently, result in greater benefits of reducing these exposures. In CARB's analyses, these potency values are the cancer slope factors and unit risks recommended by OEHHA for the critical cancer effects associated with a TAC. In a socioeconomic analysis, the primary focus emphasized by Chiu involves first characterizing potency for a typical individual in the population exposed, incorporating both uncertainty in that potency estimate and interindividual variability in cancer susceptibility. This typical individual's dose-response estimate would ideally be represented by a measure of central tendency, such as a mean or median value of the population.

Key Considerations – Dose-Response Assessment

- Review basis for cancer potency estimates and associated uncertainty
- Establish primary estimates based on central tendency response.
- Represent interindividual variability in response and uncertainty in potency estimates quantitatively where possible to reflect full range of population responses.

Risk Characterization. In the risk characterization the product of the above steps is used to convey the impact of exposures on the risk of identified hazards. In simple terms, CARB's risk characterization involves estimating some measure of risk (e.g., lifetime cancer risk) associated with individual or population exposures to a given TAC. For socioeconomic analysis, Chiu emphasizes the need to identify the change in risk between a status quo baseline scenario and a policy scenario, where the results convey changes in both the frequency and severity (i.e., fatal vs. non-fatal cases) of the adverse health outcome as a function of time. For CARB to develop supplemental risk estimates that can be readily valued, this would involve: 1) estimating changes in lifetime risk; 2) assessing the likelihood that a potential case of cancer would be fatal or non-fatal, since the valuations would differ; and 3) quantitatively modeling of the realization of risk reductions in the exposed population over time.[2] The latter step is needed to support the economic concept of discounting, discussed in Chapter 3, and it also meets California's Standardized Regulatory Impact Assessment (SRIA) requirements for describing the time horizons of the impacts of major regulations. Finally, applying methods that convert estimates of individual lifetime risk changes to estimates of avoided (statistical) cases in the exposed population is a required element when modeling benefits accrual over discrete timeframes.

[2] Ultimate health outcomes for a given case of a disease are informed by many factors, including access to healthcare, socioeconomic status.

Key Considerations – Risk Characterization

- Ensure risk estimate characterize difference between baseline/status quo conditions and policy scenario.
- Ensure risk estimates convey not only total change in cancer risk, but estimates of severity (i.e., fatal vs. non-fatal cases)
- Model benefits accrual over time to support valuation adjustments for income growth and discounting.

2.2 Examples of the Need for Bridging Steps

In this section, we use the guidance above to critically evaluate a recent CARB risk analysis for its compatibility with socioeconomic analysis and identify potential aspects that would benefit from bridging adjustments to modify risk assessment methods so that they better inform socioeconomic-style risk assessment and valuation.

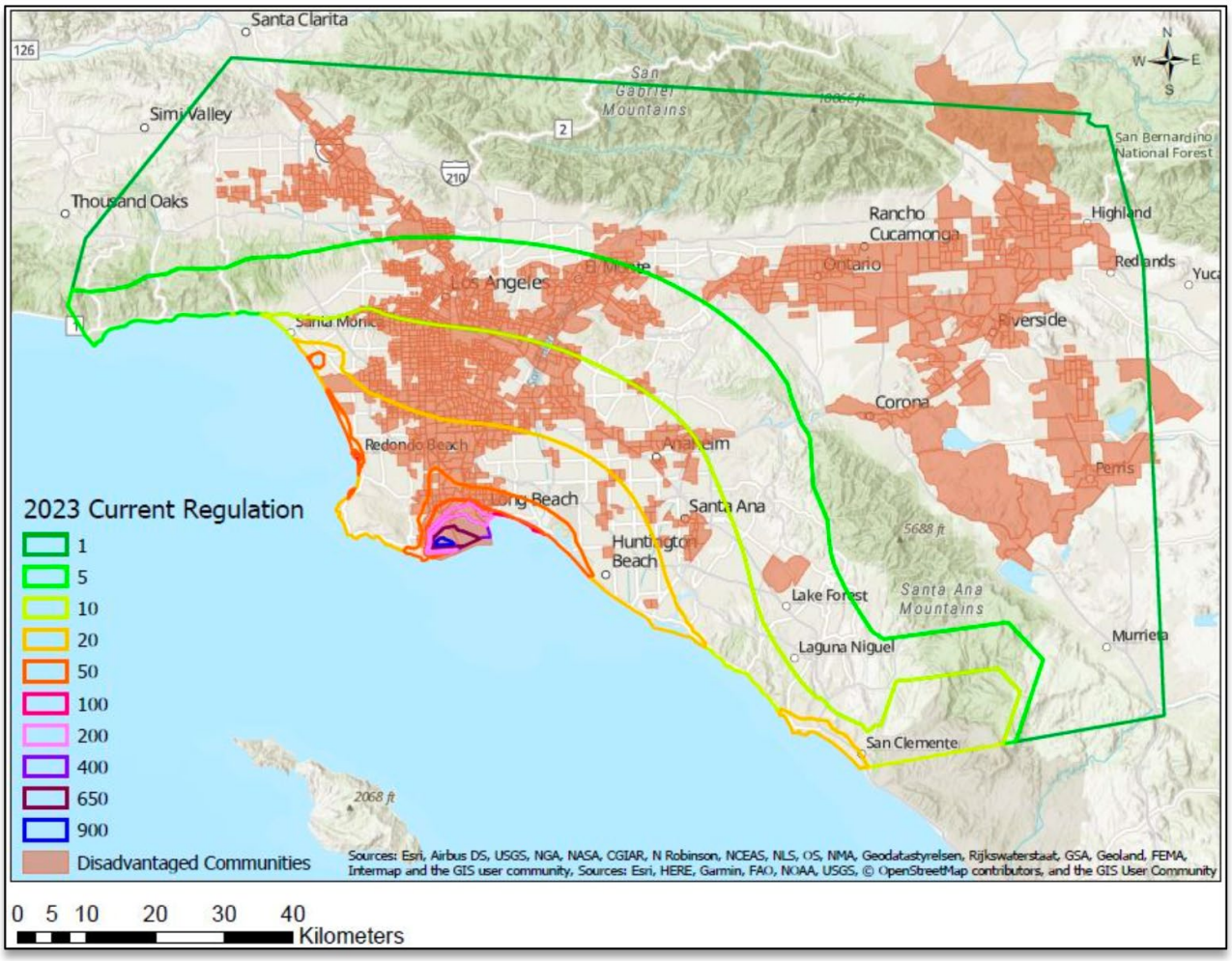
In 2021, CARB conducted a risk assessment of potential benefits of a proposed rule for commercial harbor craft limiting diesel particulate matter (DPM) emissions. This analysis quantifies lung cancer risk reductions related to decreased exposures to DPM, as well as reductions in premature mortality and morbidity related to decreases in $PM_{2.5}$ more generally. This risk assessment includes several examples of opportunities to apply bridging methods to support economic valuation for socioeconomic assessment.

- **Hazard Identification.** DPM represents a subset of $PM_{2.5}$, and thus the potential for double-counting of benefits should be carefully considered when health impacts of both exposures are quantified. For example, both DPM and $PM_{2.5}$ have been associated with the development of lung cancer (e.g., Krewski et al., 2009; Garshick et al., 2008). CARB's approach appears to address this concern by focusing on cardiopulmonary deaths exclusively in its $PM_{2.5}$ analysis; nonetheless, it would be useful to confirm that the International Classification of Disease codes associated with the cardiopulmonary mortality estimate exclude lung cancer, and that lung cancer impacts are excluded from the morbidity estimates as well.
- **Exposure Assessment.** CARB notes that it follows existing guidance from CARB/CAPCOA for characterizing inhalation exposures for cancer risks by using the 95th percentile breathing rates for age bins less than two years old and the 80th percentile breathing rates for age bins greater than or equal to two years old. While this may be appropriate for comparing to established risk thresholds, these high-end exposure assumptions are not consistent with the population central tendency estimates used to characterize primary results of socioeconomic analyses. Similarly, CARB applies a 70-year exposure duration when generating population estimates, also per existing guidance (CARB and CAPCOA 2015, OEHHA 2015). This focus on lifetime exposure would not be consistent with the approach to socioeconomic analysis, which attempts to reflect differing exposures experienced by individuals using a distribution of exposure durations across the exposed population.
- **Dose-Response Assessment.** CARB acknowledges uncertainty in the DPM lung cancer potency factor created by OEHHA but stops short of quantifying that uncertainty, noting the many challenges of doing so. IEc agrees that uncertainty characterization can be challenging but recommends quantifying uncertainty to the extent possible given available data, even if that characterization may be incomplete. Partial quantification of uncertainty coupled with qualitative discussions acknowledging additional sources of uncertainty can provide a fuller picture of the state-of-knowledge in these cases.
- **Risk Characterization.** CARB characterizes results for DPM related lung cancer risk using both maps and tables. The maps include isopleths denoting boundaries of areas where individual risks may reach a certain value, according to CARB's individual risk estimate calculations (see Figure 1). However, because these calculations used fixed values for exposure parameters and dose-response,

the maps tend to mask the variation in risk across the population living within the various risk bands. As a result, these values would be suboptimal for generating population risk estimates to support a socioeconomic assessment which aims at characterizing a central estimate and variability in the risk experienced by the exposed population. A summary table of population risk estimates does provide a more stratified presentation of the potential numbers of persons in different risk ranges, but modifying CARB's approach for a socioeconomic analysis affords an opportunity to expand characterization of variability and uncertainty in these values.

Figure 1. Example Figure from CARB 2021 DPM Cancer Risk Assessment

Figure G-9 2023 Impacts from CHC Under the Current Regulation South Coast Air Basin Potential Cancer Risk Isopleths (chances per million)[25]



CHAPTER 3 | Valuing Cancer Risk Reductions

Regulatory interventions addressing the release of TACs often impose costs on emitters, such as when regulated industries are required to purchase, install, and maintain emissions control technologies. While TAC controls are also expected to produce societal benefits, such as improvements in human health, regulatory agencies often perform benefit-cost analyses to understand, in quantitative terms, whether these benefits are likely to equal or exceed the implementation costs associated with a given emissions control strategy. Within this context, health benefits are typically valued, or expressed in monetary terms, to facilitate comparison with costs.

Our recommended methodology values change in the risk of adverse health outcomes, consistent with socioeconomic analyses conducted by CARB and other environmental and public health agencies (e.g., South Coast Air Quality Management District, U.S. EPA, Health and Human Services). As described in Chapter 2, in this context, health-related benefits are estimated in a probabilistic manner in which (a) every member of the exposed population is assumed to benefit—to varying degrees—from reduced exposures through reductions in their annual or lifetime risk of illness or death, and (b) individual risk reductions are typically aggregated across the exposed population and expressed as the total number of avoided statistical cases of illnesses and/or premature deaths resulting from a policy change.[3] Within this context, WTP is the preferred economic valuation measure and reflects the maximum amount an individual would be willing to spend to reduce their risk of illness and/or death. For the purposes of valuation, these units—individual risk reductions and avoided cases of illness or death—are interchangeable. WTP values are estimated for specified risk reductions (e.g., 1 in 10,000 risk of death) and may alternatively be expressed as values per statistical case when divided by the associated risk change. For example, if an individual's WTP for a reduction in his or her own fatal risk of 1 in 10,000 in the current year is $900, then that individual's VSL—the preferred measure for valuing reductions in mortality risks—is calculated as:

$$\$900 \div (1/10{,}000) = \$9{,}000{,}000$$

In practice, benefit-cost analysis practitioners identify and apply population average WTP estimates produced by peer-reviewed economic studies, and risk changes are valued consistently across all individuals and population subgroups.[4] Because regulations represent complex processes that are implemented over years or decades, the timing of benefits (and costs) is an important consideration in benefit-cost analyses. Development of the time series of benefits is an important nexus between risk analysis and valuation (see 'Accounting for Timing in the Valuation of Cancer Risk Reductions' text box).

In the remainder of this chapter, we propose methods for CARB's valuation of human health benefits in the form of reductions in cancer-related risks and cases resulting from TAC emissions reductions. We first address valuation of fatal cancer risks, supporting CARB's VSL approach and expanding the methods to better characterize uncertainty in the estimates. We then outline recent efforts to value changes in non-fatal cancer risks as identified in a recent literature review by LaPenta et al. (2024) and our proposed adaptations to these methods to better suit CARB's needs.

[3] Throughout this document, references to avoided cases of health impacts represent statistical cases avoided. That is, these cases represent an aggregation of individual risk reductions in a population and do not represent a diagnosis or death associated with a particular person.

[4] In some contexts, economic analyses may value health benefits using non-WTP measures, such as cost of illness, that can vary across geographies based on location-specific medical costs and/or wages.

Accounting for Timing in the Valuation of Cancer Risk Reductions

One challenge in valuing cancer risk reductions is accounting for the timing of pollutant exposure, diagnosis of resulting cancers, and, in some cases, subsequent premature deaths. There is a lag between exposure change and the realization of benefits; in part this reflects a delay between exposure and diagnosis—referred to as the "latency" period of a given cancer— but other factors may affect this lag as well. The total lag can be years to decades long in some cases, and individuals diagnosed with cancer may die from the disease years after diagnosis. Two adjustments to economic values necessitate that analysts model the timing of these occurrences: (1) income growth and (2) discounting.

Income growth adjustments refer to the practice of scaling WTP values to account for the influence of income on individual WTP. In the context of air pollution benefits analysis, this adjustment typically involves (1) assessing differences in real per capita income between the year of the underlying economic study and the year in which benefits occur, and (2) applying an income elasticity that reflects the sensitivity of WTP to real income growth. For example, scaling a WTP value estimated in Year 1 to value risk reductions accruing in Year 2 would take the following form:

$$WTP_2 = WTP_1 * \left(\frac{Income_2}{Income_1}\right)^{\varepsilon}$$

where *Income* reflects real (i.e., inflation-adjusted) per capita income and ε is the income elasticity.

Discounting is the practice of expressing future benefits and costs in present value terms. For socioeconomic analyses conducted by regulatory agencies, discount rates generally reflect the rate at which society is willing to trade current consumption for future consumption, a value typically referred to as the social rate of time preference. In simple terms, benefits accruing today are preferred to comparable benefits accruing in future years. The selection of discount rate can meaningfully impact the results of regulatory analyses, particularly when benefits and/or costs are observed decades after government intervention. Discounting is conducted as follows:

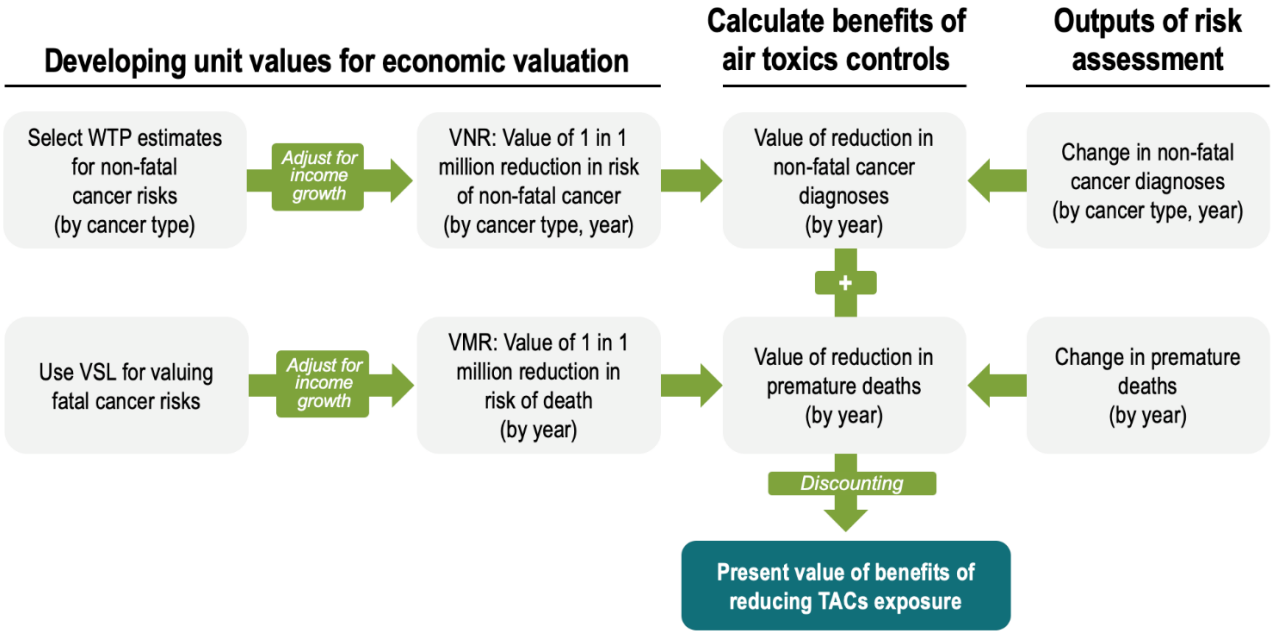
$$PV = \frac{FV}{(1+r)^t}$$

where *PV* is the present value (e.g., of the benefits of future risk reductions), *FV* is the future value, *r* is the specified discount rate, and *t* is the time (in years) between the discounting base year (oftentimes the start year of a regulation) and the realization of the future benefit and/or cost.

3.1 Valuation Approach Overview

This chapter provides CARB with an approach to valuing cancer risk changes. The approach distinguishes between non-fatal and fatal cancer risks, accounting for the timing of exposures, diagnoses, and, in some cases, deaths. For valuing mortality risk changes, IEc endorses CARB's use of the U.S. EPA VSL, adjusted for inflation and income growth. We then adapt non-fatal WTP measures identified in a literature review by LaPenta et al. (2024) to value changes in non-fatal cancer risks. In both cases, IEc provides methods for characterizing statistical uncertainty in the underlying WTP estimates. Figure 2 describes the key steps in this process, including how we integrate economic unit values with outputs from risk assessment.

Figure 2. Overview of Valuation Approach



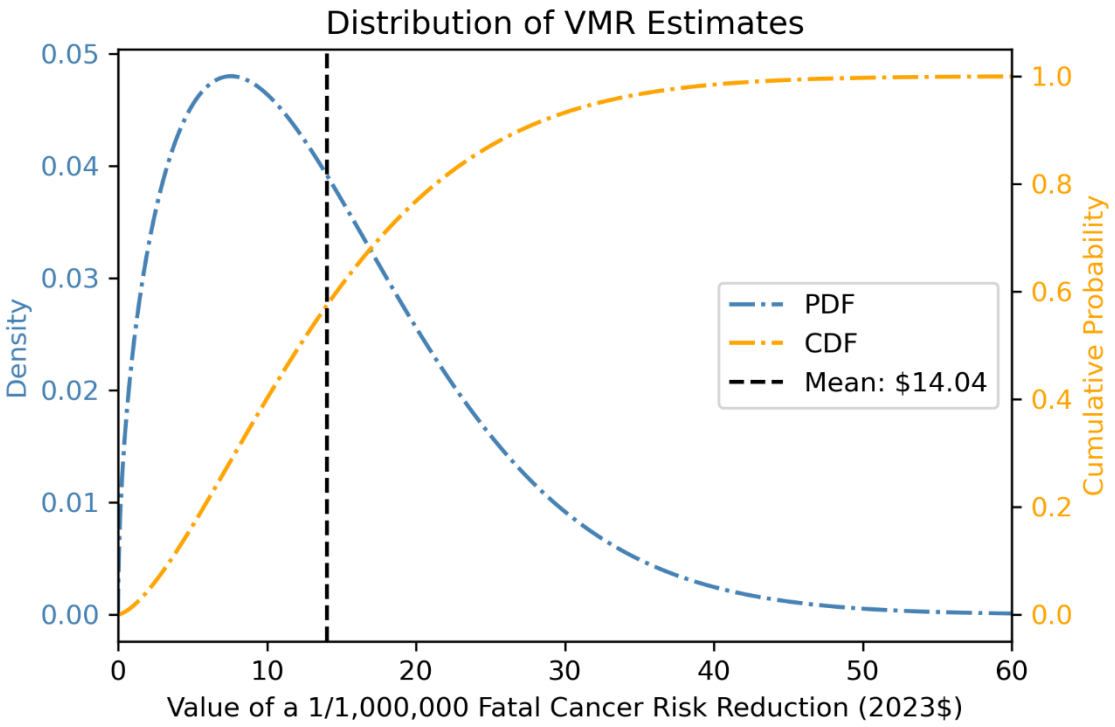
3.1.1 Valuing Fatal Cancer Risks

Consistent with current CARB practices, we recommend applying U.S. EPA's standard VSL for valuing reductions in fatal cancer risks, accounting for both inflation and growth in real (i.e., inflation-adjusted) per capita income.[5] U.S. EPA's preferred VSL estimate of $7,400,000 in 2006 dollars is equivalent to $14,030,101 in 2023 dollars when adjusted for income growth and California-specific inflation. We note that other recent reviews of WTP literature for fatal illnesses often result in comparable VSL estimates (for example, see reviews conducted by Robinson et al. (2016a) for the South Coast Air Quality Management District and by Robinson et al. (2016b) for the U.S. Department of Health and Human Services).

The U.S. EPA VSL is the mean estimate of a Weibull distribution fitted to 26 VSL studies reviewed by U.S. EPA (1997). This distribution of values is used in U.S. EPA's Environmental Benefit Mapping and Analysis Program (BenMAP) to value reductions in fatal cancer risk (BenMAP 2025). As in BenMAP, we incorporate statistical uncertainty into the unit value consistent with EPA's approach by including a confidence interval around the mean VSL estimate. **In this report, consistent with LaPenta et al. (2024), we express mortality risk values as a 1 in 1 million reduction in fatal risk, or value of mortality risk reduction (VMR).** Figure 3 shows the Weibull distribution EPA uses to characterize VSL, converted here to VMR.

[5] See, for example, CARB's Standardized Regulatory Impact Assessment (SIRA) of the Low Carbon Fuel Standard (LCFS) 2023 Amendments (CARB 2023). CARB employs the U.S. EPA central VSL estimate ($7.4 million, 2006$) adjusted for California-specific inflation ($14 million, 2023$) to value reductions in morality risk (State of California Department of Industrial Relations). Following U.S. EPA, CARB adjusts WTP estimates to account for per capita income growth, assuming an income elasticity of 0.4 for adjusting the VSL and 0.45 for adjusting non-fatal WTP values. CARB employs California-specific inflation adjustments using the California CPI-U index when valuing non-fatal risk (State of California Department of Industrial Relations 2025).

Figure 3. Distribution of Fatal Cancer Risk Valuation



In Chapter 5, we discuss generating VSL estimates adjusted using California-specific inflation data.

3.1.2 Valuing Non-Fatal Cancer Risks

Our recommended approach to valuing non-fatal cancer risks involves two stages: identifying relevant WTP literature and, as needed, pooling studies to better characterize underlying uncertainties. These items are described below. Appendix D provides additional detail on how we derive 95 percent confidence intervals for WTP estimates presented in our recommended studies.

3.1.2.1Identifying WTP Estimates for Non-Fatal Cancer Risks

The basis for our proposed methods to valuing changes in cancer risks is economic literature estimating individual WTP for comparable risk reductions. We leverage a recent literature review synthesizing this research conducted by Abt Global for U.S. EPA's Office of Pollution, Prevention and Toxics (OPPT) and later published in the *Journal of Benefit Cost Analysis* (Abt Global 2022, LaPenta et al. 2024). Using comparable search criteria, we conducted a supplementary literature review to consider any U.S.-based research published between February 2020 and October 2024; we identified no relevant WTP literature published in this time period. The authors of the literature review developed WTP estimates for one-in-one million risk reductions for both fatal cancers (VMR) and non-fatal cancers (VNR, value of non-fatal risk). LaPenta et al. focused their literature review on identifying studies estimating WTP for avoiding non-fatal cancer risks, with the goal of

compiling WTP values for 108 of the most common cancer groups and sites.[6,7] The authors identified five relevant articles: three focused on cancers (Bosworth et al. 2009, Gerking et al. 2014, Magat et al. 1996), and two focused on health outcomes that were deemed reasonable substitutes for non-fatal cancer due to similarities in symptoms, treatments, and latency periods (Sloan 1998, Viscusi et al. 1991).

Due to the limited WTP literature for non-fatal cancers, the LaPenta et al. literature review identified two studies that value non-cancer health outcomes. First, the estimates for chronic bronchitis risks (Viscusi et al. 1991) have historically been used by U.S. EPA to value reductions in select non-fatal cancer risks (U.S. EPA SAB 2001). Specifically, the Viscusi et al. and Magat et al. estimates have been employed by U.S. EPA to value avoided risks for non-fatal cancers such as bladder, nasopharyngeal, kidney, and liver in support of 2013, 2016, 2017, and 2024 rulemakings (U.S. EPA 2013, 2016, 2017, 2024). LaPenta et al. (2024) employ estimates from Viscusi et al. (1991) to value cancers of the respiratory system aside from lung and bronchus including nose, nasal cavity, and middle ear, larynx, pleura, trachea, mediastinum, and other respiratory organs. In addition, LaPenta et al. include estimates from Sloan (1998) for non-fatal multiple sclerosis (MS). In the absence of WTP research tailored to cancer risks, IEc currently supports use of the Viscusi et al. chronic bronchitis estimates. As described below, we do not include Sloan's MS risk estimates in our valuation approach for non-fatal cancer risks.

Considering Sloan (1998) WTP Estimates for Multiple Sclerosis

LaPenta et al. (2024) apply WTP estimates for multiple sclerosis (MS) from Sloan (1998) to value risks associated with non-fatal brain cancer and other cancers of the nervous system. We do **not** recommend using the Sloan (1998) estimates for valuing cancer risks due to the following concerns:

- The experiences of individuals living with MS may vary considerably relative to individuals diagnosed with non-fatal brain cancers.
- Sloan (1998) employs a standard gamble survey approach in which respondents are asked to assume they have MS and then make a tradeoff between (a) living with MS and (b) a surgery involving a probability of instant death and the complementary probability of instant cure. Whereas the risk-risk approach used in Viscusi (1991) and Magat (1996) presents non-fatal outcomes as a risk (i.e., with an associated, small probability of occurrence), Sloan's standard gamble approach presents the non-fatal outcome as a certain, baseline condition. In this context, the initial probability of death (15%) presented to survey respondents by Sloan (1998) is orders of magnitude higher than (a) the probability of death in most WTP studies and (b) the size of risk reductions produced by most regulatory interventions for air pollution.

In the absence of site-specific WTP values for non-fatal brain cancer and other nervous system cancers, IEc recommends applying the low- and high-end estimates used for cancers without a site-specific value ($0.46 and $7.99, respectively).

[6] Abt Global (2022) searched for literature in four databases (EconLit, Environment Complete, JSTOR, and Science Direct) using search terms that included one valuation and one health endpoint term (e.g., "contingent valuation" and "lung cancer"). Search results were initially reviewed by title and abstract only, followed by a full text review of articles of interest. Articles were excluded if they valued WTP for treatment of fatal health outcomes, estimated the value of quality- or disability-adjusted life years, or measured cost-of-illness values. Studies conducted outside of the United States were also excluded to avoid the need to account for differences in cultural context, healthcare, and income across countries. Publishing date was not restricted. Abt Global included results for non-cancer health outcomes that may be comparable to non-fatal cancers; however, health outcomes such as migraines or mental illness were deemed as unsuitable substitutes and were excluded.

[7] The Abt Global (2022) literature review was conducted in January and February of 2020. IEc conducted an updated literature review to search for articles published after February 2020, but no studies meeting the criteria above were identified.

In total, LaPenta et al. assign the limited set of WTP values to 108 cancer groups and sites; 101 of these were identified in SEER's Site Recode ICD-O-3/WHO 2008 Definition, while the remaining seven were of specific interest to U.S. EPA's Office of Pollution, Prevention, and Toxics.[8] We use the WTP values identified for the 101 groups and sites listed in the SEER Site Recode.

3.1.2.2Challenges in Valuing Non-Fatal Cancer Risks

Prior to presenting our recommended methods for valuing changes in non-fatal cancer risks, we highlight several limitations associated with existing literature and the LaPenta et al. (2024) valuation approach:

- **Limited literature.** As described above, few studies estimate WTP for non-fatal cancer risks. As a result, regulatory analysts must rely on a thinner evidence base, with fewer recent, high-quality, and relevant estimates available. The values presented by La Penta et al. (2024) and in this report include some studies that use older data (e.g., Viscusi et al. 1991), evaluate non-cancer outcomes (e.g., chronic bronchitis), and rely on smaller, sub-national samples (e.g., Gerking et al. 2014 survey of 1,303 adults in Florida and Mississippi.)
- **Limited cancer sites.** Due to the limited number of studies assessing non-fatal cancer risks, WTP estimates are available for a small number of cancer sites (described in the following section). Willingness to pay to avoid cancer risks may vary meaningfully based on the type of cancer. To address this research gap, LaPenta et al. map the few non-fatal cancer WTP estimates to over 100 cancer sites.
- **Measures of central tendency.** The synthesis of non-fatal cancer WTP estimates identified in the literature review by LaPenta et al. includes, in many cases, a range of potential values using a low- and high-end estimate from the underlying literature. For regulatory analyses requiring one "best" or "central" estimate, the LaPenta et al. estimates do not include measures of central tendency, such as mean or median WTP values.
- **Addressing lack of statistical significance.** Two of the non-fatal WTP estimates recommended for use by the LaPenta et al. literature review are not statistically significant. Specifically, Bosworth et al. (2008) estimate non-fatal WTP for several cancers; the general cancer and colon/bladder cancer estimates are not statistically significant at the 95 percent level.

While our approach is unable to fill major research gaps in the underlying literature estimating WTP for non-fatal cancers, we adapt the LaPenta et al. approach to better characterize statistical uncertainty and to provide decision-makers with central estimates. These methods are described below.

[8] We exclude the seven cancer sites not listed in SEER. These cancers are not associated with the priority toxic air contaminants identified by CARB and listed in Table 4 of this report. Further, the seven cancer sites are more specific versions of cancer sites already listed in SEER's Site Recode ICD-O-3/WHO 2008 Definition (e.g. Brain (glioma malignant) rather than Brain).

3.1.2.3Applying WTP Estimates for Non-Fatal Cancer Risks

Application of WTP estimates to non-fatal cancer risks requires consideration of whether one or more WTP estimates are available for the cancer site being evaluated. **If a suitable WTP estimate is available for the cancer site being evaluated, we recommend applying the central WTP value and accompanying statistical uncertainty to generate estimates of monetized benefits.** For example, Bosworth et al. (2009) estimate WTP for reducing non-fatal lung cancer; regulatory analyses of air toxics found to cause lung cancer should utilize this estimate. All monetized results should be accompanied by a qualitative discussion of uncertainties and limitations, including the lack of a wider research base, in this case for lung cancer valuation.

For generic cancer risks (i.e., unspecified sites), we recommend applying a pooled WTP estimate across non-fatal cancer risks. This approach considers evidence across several studies and cancer sites, allowing for both for the characterization of statistical uncertainty and variability between cancers. We do not recommend relying solely on the Bosworth et al. (2009) estimate for generic cancer because the value is not statistically significant. To develop the pooled distribution, we calculate a 90 percent confidence interval around the mean WTP value from each study in Table 1. More details on how these calculations were performed can be found in Appendix D. We applied the same approach as LaPenta et al. to adjust WTP values from relevant studies for inflation and income growth to present results in 2023 dollars.[9] Resulting values for reductions in non-fatal cancer risk are described in Table 1 and presented as one-in-one million reductions in annual non-fatal cancer risk. Mean WTP values are weighted in the distribution by the proportion of total non-fatal cancer cases represented by each type of cancer.[10] Non-fatal case counts are calculated using the California Cancer Registry's cancer incidence data and CDC WONDER's California-specific cancer mortality data. The resulting distribution heavily weights the Bosworth et al. (2009) generic cancer estimates, as most cancer sites are not evaluated by the limited economic research into non-fatal cancer risks.[11]

Table 1. Non-Fatal Cancer Risk WTP Values

Source	Health Endpoint in Original Study	Percent of Non-Fatal Cases	Mean VNR (2023$)
Bosworth et al. (2009)	Lung cancer	3.8%	$1.58
	General Leukemia	2.6%*	$1.44
	Colon/bladder cancer	12.1%	$0.75
	Generic cancer	71.2%	$0.46
Gerking et al. (2014)	General Leukemia	2.6%*	$0.15
Magat et al. (1996)	Curable Lymphoma	5.5%	$4.59

[9] Values were adjusted by LaPenta et al. for inflation using the Consumer Price Index and for income growth using real GDP per capita and EPA's recommended income elasticity of 0.45 for severe illness.

[10] A preferable approach may include developing a distribution weighted by the proportion of TAC-induced non-fatal cancers; however, this would require a full accounting of the relative frequency of cancer sites resulting from specific TACs. We recommend additional consideration of such an approach in future research..

[11] Estimates for the value of a non-fatal case of leukemia come from both Bosworth et al. (2009) and Gerking et al. (2014). For purposes of valuation, these two estimates are pooled together into a single estimate using variance-derived weights following the approach in U.S. EPA's BenMAP tool. Random effects weights are used to combine these estimates into a single distribution with a central estimate of 0.717 and a variance of 0.638. See Appendix K to the User Manual for the desktop BenMAP-CE application, available here, for a description of the approach for developing weights for pooling incidence and valuation estimates for use in health impact analysis. The approach is based on methods originally proposed by DerSimonian and Laird, 1986. For this pooling exercise, we calculated random effects weights for these two studies after the fixed-effect hypothesis was rejected.

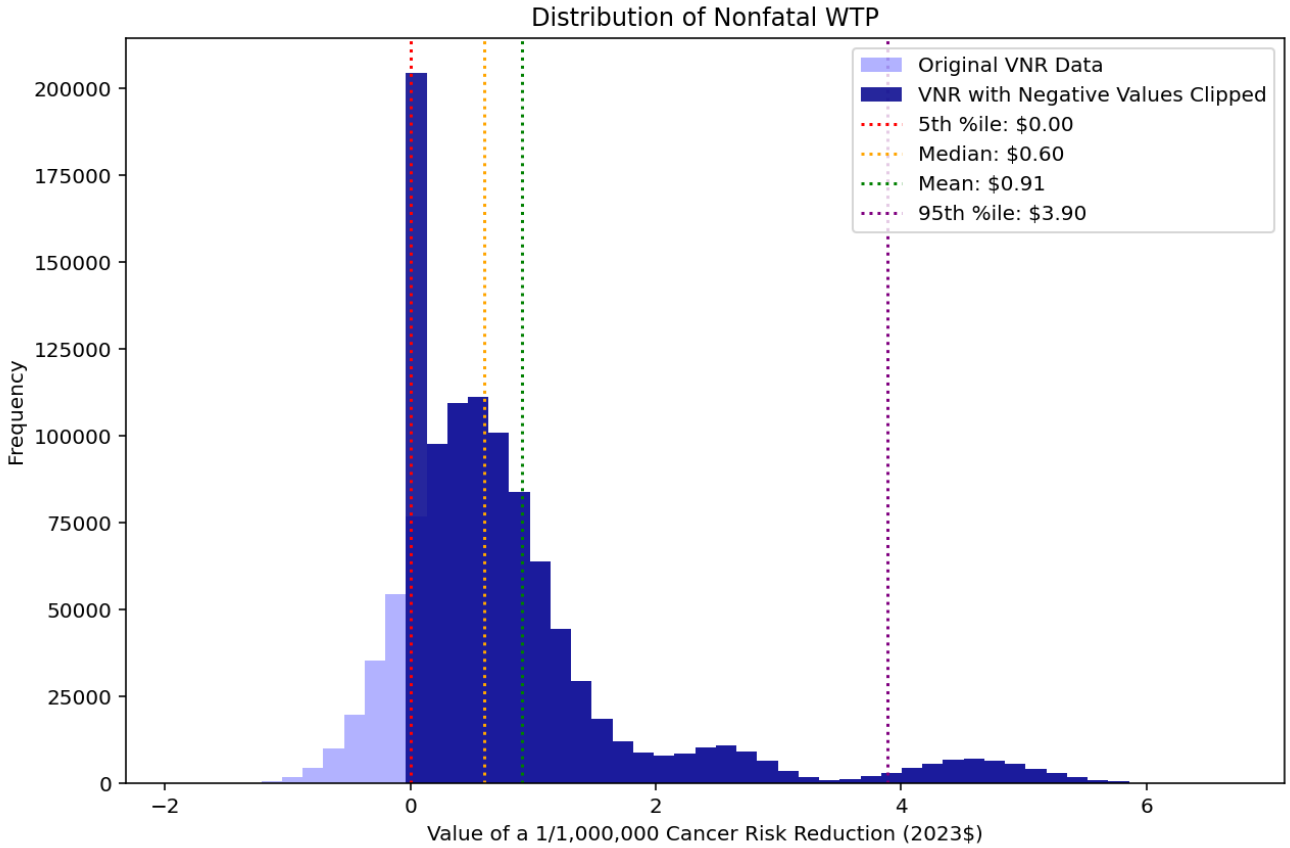
Source	Health Endpoint in Original Study	Percent of Non-Fatal Cases	Mean VNR (2023$)
Viscusi et al. (1991)	Chronic bronchitis	4.8%**	$1.34

*See Footnote 11 for more detail on pooling the two WTP estimates for non-fatal leukemia.

**Represents prevalence of all other respiratory cancers aside from lung (nose, nasal cavity, and middle ear; larynx; pleura; trachea, mediastinum, and other respiratory organs)

Figure 4 presents the pooled distribution for non-fatal WTP estimates for cancer risks. We estimate a mean VNR of $0.87, with a 90 percent confidence interval of -$0.30 to $3.805. When estimating reductions in risk for cancers without a site-specific value, we sample from this distribution to represent statistical uncertainty in the underlying studies. As described in the text box below, we restrict the WTP distribution to remove negative values.

Figure 4. Distribution of Values for a 1/1,000,000 Reduction in Non-fatal Cancer Risk (VNR)



Restricting WTP to Non-Negative Values

Figure 4 presents the VNR distribution resulting from pooling seven WTP estimates for non-fatal illnesses published in four articles. Each of the estimates is accompanied by statistical uncertainty, influenced by factors such as study sample size, variation in respondent preferences, and model specifications. For normally distributed errors around these WTP estimates, some weight is necessarily assigned to the negative domain (i.e., below zero). Most notably, two of four WTP estimates for non-fatal cancer risks (generic cancer, colon/bladder cancer) in Bosworth et al. (2009) are not found to be statistically significant (90 percent confidence level). Without adjustments, the resulting pooled distribution would include meaningful weight (14 percent) below zero.

While individuals exhibit different risk preferences—some are willing to pay more for reductions in risk—WTP for cancer risk reductions should be strictly positive. We recommend CARB truncate the distribution after calculating mean WTP values:

- Economic research finds that individuals are broadly willing to pay a premium to avoid non-fatal illnesses. The mean VNR value is positive ($0.87) and represents our best characterization of WTP for unspecified non-fatal cancers.
- Alongside mean VNR estimates, regulatory analysts should present the associated confidence interval to communicate uncertainty in the population VNR. After calculating mean VNR and subsequently setting negative values to zero, a 90 or 95 percent confidence interval would include 0.
- While regulatory analysts typically prioritize individual WTP estimates when high-quality studies are available, cost of illness (COI) research provides an alternative measure demonstrating societal costs of illness. COI studies typically consider medical expenditures associated with treating an illness and measures of lost productivity, such as missed work for patients and their caregivers. In many instances, COI estimates are lower than WTP estimates for the same outcome. CARB may wish to conduct supplementary research to quantify COI measures for non-fatal cancer valuation or, at a minimum, to demonstrate that there is some quantifiable (and monetizable) benefit to reducing non-fatal cancer risks.

3.1.3 Income Growth Adjustments

We recommend adjusting both fatal and non-fatal WTP values for growth in real income in future years. Consistent with EPA procedures for income growth adjustments, we employ the income growth dataset provided in the BenMAP tool. These calculations are based on GDP projections presented in the 2020 Annual Energy Outlook (AEO), adjusted for inflation using the GDP Chain-type Price Index reported by AEO and divided by AEO's population projections to estimate per capita GDP. BenMAP provides income-based WTP adjustments for 1990 through 2050 (BenMAP 2023). This model utilizes U.S. EPA's recommended income elasticity estimates of 0.45 for severe illness and 0.4 for premature mortality, applied to non-fatal and fatal WTP measures, respectively (U.S. EPA 2014). Income growth adjustments take the following form:

Equation 1. Adjustment to WTP Values for Real Income Growth and Elasticity

$$WTP_y = WTP_{2023} * \left(\frac{I_y}{I_{2023}}\right)^{\varepsilon}$$

Where:

- WTP_y: the value of a cancer risk reduction in the year of interest
- WTP_{2023}: the value of a cancer risk reduction in 2023$
- I_y: Real income in the year of interest
- I_{2023}: Real income in 2023
- ε: Income elasticity

For analyses with benefits realized after 2050, we recommend applying the AEO / BenMAP income growth rate from 2049 to 2050 (1.2 percent) to all future years.

3.1.4 Discounting

For policies addressing the release of TACs, the benefits of reduced TACs exposure are likely to occur years, or decades, after compliance costs are incurred (see Chapters 4-7 for a discussion of these lags). Discounting, the practice of expressing future benefits and costs in present value terms, is recommended when evaluating benefits and costs that do not take place in the same period (OMB 2003, U.S. EPA 2024c, HHS 2016). For socioeconomic analyses conducted by regulatory agencies, discount rates generally reflect the rate at which society is willing to trade current consumption for future consumption, a value typically referred to as the social rate of time preference.

In this report, we provide discussion and illustrative calculations that are undiscounted (consistent with current CARB practices) and discounted at a 2 percent rate. We recommend discounting future benefits and costs using this 2 percent rate, which reflects recommendations by the U.S. Office of Management and Budget's updated Circular A-4 (issued in 2023 and rescinded in 2025). The 2 percent value is based on the "real (inflation-adjusted) rate of return on long-term U.S. government debt [which] provides a fair approximation of the social rate of time preference" (OMB 2023). As described earlier in this chapter, present value (*PV*) of future benefits or costs is calculated as:

$$PV = \frac{FV}{(1+r)^t}$$

where *FV* is the future value, *r* is the specified discount rate, and *t* is the time (in years) between the discounting base year (oftentimes the start year of a regulation) and the realization of the future benefit and/or cost.

3.1.5 Applying Unit Values to Generate Benefits Estimates

In the following chapters, we describe how risk changes should be quantified and valued. The precise valuation methodology differs across the three risk analysis approaches, but the general structure remains the same. For any given cancer site, age, and sex of diagnosis, we recommend valuing risk changes by accounting for both non-fatal and fatal cases, adjusting estimates as needed for inflation and income growth, and discounting future benefits to a common base year.

3.2 Limitations and Uncertainties

While our recommended approach for valuing changes in cancer risks is intended to align with best practices for health benefits analyses, the approach contains limitations and uncertainties that should be considered when interpreting results:

- **Limited WTP research for non-fatal cancers:** While LaPenta et al. (2024) is a recent, comprehensive, and peer-reviewed literature review of U.S.-based valuation estimates for non-fatal cancers, limited research on this topic is available. We utilize WTP estimates from just four studies examining a limited set of cancer sites. Two of these studies are decades old (e.g., Viscusi et al. 1991, Magat et al. 1996). Additional research is needed to better characterize WTP for non-fatal cancers across a range of cancer sites.
- **Benefits transfer to all cancer sites:** Due to the limited set of WTP estimates for non-fatal cancer risks, we generate a pooled distribution of seven non-fatal WTP estimates for use with generic cancers and for cancer sites not present in the underlying economic research. Risk preferences are likely to vary across cancer sites due to differences in quality of life and medical treatments.

CHAPTER 4 | Methodology Overview

TACs vary in terms of the data available to characterize exposure and cancer risk. Some have been extensively studied for their cancer-causing potential, including in epidemiological studies that associate human exposures with changes in cancer incidence; others may rely on cancer studies based on tumor incidence in animals. Risk analyses of TACs can also vary in their geographic scope. This can be a function of the nature of the TAC itself (e.g., it may have more localized impacts because its physical and chemical properties limit its potential for transport) or a function of the characteristics (e.g., stack height) and numbers of emitting sources affected by a specific rule. We have designed three different approaches for valuing TAC cancer risk reductions that will accommodate this variability and ensure that the data requirements and level of effort to conduct this supplemental analysis are "fit-for-purpose" and reflect the data available.

We begin this chapter with an overview of the methodology and then present a decision tree to help CARB analysts identify which approach, or approaches, may be suitable for a given TAC and risk assessment scope. We also illustrate how this process would be conducted for the eleven priority TACs identified earlier in the project in collaboration with CARB. Subsequent chapters present more detailed information about each approach.

4.1 Overview and Approach Selection

Table 2 presents a summary of the three approaches we have defined for developing valuation estimates, including the objective, key data needs, scope, types of calculations required, and interpretation of output. Some approaches will require more data and computational analysis; however, all approaches share the following context:

- Each method is intended to be applied as a supplement to, not in place of, CARB's risk analysis conducted in accordance with the 2015 Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments (OEHHA 2015) and Risk Management Guidance for Stationary Sources of Air Toxics (CARB and CAPCOA 2015).
- Because this represents a supplemental analysis, the methodology assumes that certain information will be available from CARB's main risk assessment, most notably the development of baseline and regulatory scenarios, emissions estimation, and air quality modeling. IEc does not anticipate CARB will need to revise these steps to conduct the valuation analysis.
- Each methodology may entail modifications to the assumptions and data used to conduct the exposure assessment, dose-response, or risk characterization steps of the risk assessment. These changes are recommended as the sort of bridging steps discussed in OECD (2017) to align risk assessment methods with those used to inform socioeconomic analysis. The resulting modifications will enable CARB to develop a population average risk estimate and additional estimates that illustrate the spread of possible values resulting from population variability and uncertainty in our understanding of exposure and dose-response.
- Decisions regarding how to best implement certain steps in the methodology may require CARB to engage in discussions with relevant stakeholders; for example, identifying an appropriate epidemiological-based relative risk value for use with an Approach III lifetable analysis may require engagement with OEHHA experts.

Table 2. Summary of Approaches for TAC Risk Assessment and Valuation

Approach	Objective	Data Needs	Scope of impact	Types of calculations	How to interpret
Approach I	Develop monetized values for quantified changes in individual cancer risks from proposed TAC policy. Results can be reported individually or used to inform break-even-type economic analyses. Characterize central tendency and uncertainty as data allow. Minimizes additional analysis required.	CARB cancer risk analysis inputs (e.g., air quality modeling, exposure parameters) and results. Data on uncertainty and variability in exposure parameters. Data on uncertainty and variability in cancer potency; data could be based on animal studies, human studies, or both. $ per one-in-a-million risk reduction for specific cancers and populations from Abt Global (2024).	Appropriate for localized risk impacts.	No modification to CARB risk assessment approach, except where adjustments are necessary to estimate central tendency individual and to characterize uncertainty. Pooling of WTP values for non-fatal cancer risks. Probabilistic sampling from fatal and non-fatal WTP distributions.	Dollar estimate represents the value to society of the estimated cancer risk reduction per-person exposed. If used in a break-even context, analyst can estimate the number of individuals who would need to achieve this benefit for the overall social benefits to equal the cost of implementing the rule.
Approach II	Use available data to quantify and monetize changes in population risk resulting from proposed TAC pollution control policy. Results can be used to inform benefit-cost analysis, or other socioeconomic analyses. Characterize central tendency and uncertainty using supplemental data. Moderate additional analysis required.	CARB cancer risk analysis inputs (e.g., air quality modeling, ambient concentration data, exposure parameters). Data on central tendency exposure and uncertainty in exposure parameters; U.S. EPA's HAPEM8 model recommended Data on central tendency cancer potency and uncertainty; risk estimates based on animal studies. Historical age-specific cancer incidence data and survival data to convert lifetime cancer risk reductions into streams of benefits over time that reflect anticipated lags in the realization of benefits. Uses VSL and VNR values to monetize benefits (See Section 3.1.2)	Appropriate for TACs with regional or statewide exposures.	Adjustments to estimate central tendency of exposure and cancer potency and generate a probability distribution to reflect variability and uncertainty. Probabilistic analysis of the product of individual risk reductions and size of exposed population. Application of lag model to reflect the anticipated realization of reduced cancer rates over time. Estimation of the fraction of fatal and non-fatal cancer cases Pooling of WTP values for non-fatal cancer risks. Probabilistic sampling from fatal and non-fatal WTP distributions.	Dollar estimate represents the value to society of the estimated TAC-related cancer risk reduction on a population basis (number of statistical cases of fatal and non-fatal cancers avoided). Time stream of monetized benefits can be used to estimate a discounted Net Present Value of total benefits if desired, in addition to CARB's standard non-discounted estimate. Value could be used in a benefit-cost analysis (BCA).

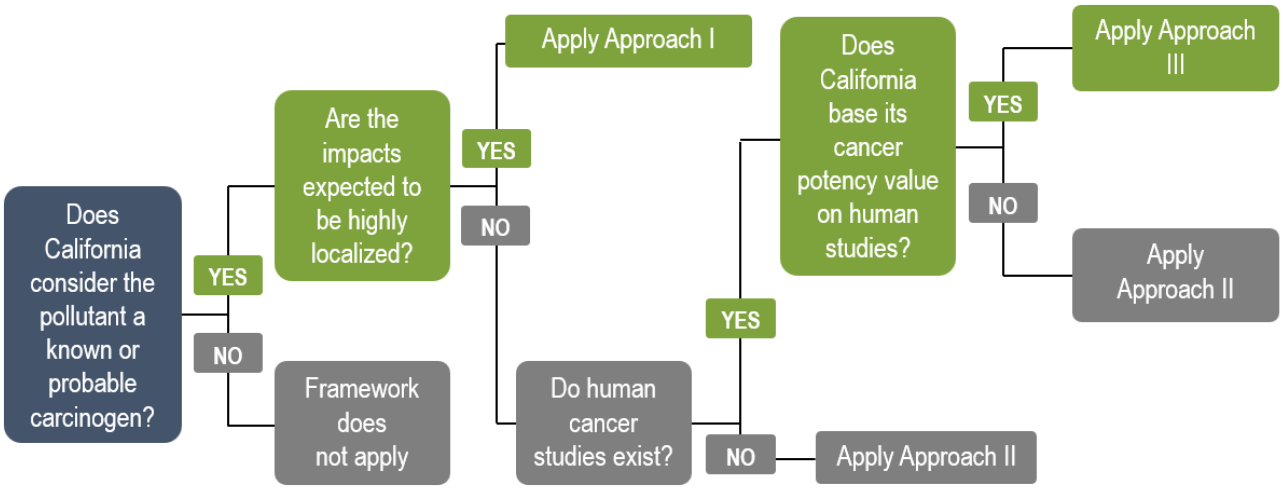
Approach	Objective	Data Needs	Scope of impact	Types of calculations	How to interpret
Approach III	Use available data to quantify and monetize changes in population risk resulting from proposed TAC pollution control policy using a life table risk model. Quantify the change in cancer incidence over time for specific geographies and subpopulations. Results can be used to inform benefit-cost analysis, other socioeconomic analyses, and environmental justice analyses. Characterize central tendency and uncertainty using supplemental data where necessary. Requires cancer potency data from human studies. Entails the most additional analysis but is the most comprehensive approach and generates the most granular data.	CARB cancer risk analysis inputs (e.g., air quality modeling, exposure parameters); Data on central tendency exposure and uncertainty in exposure parameters; U.S. EPA's HAPEM8 model recommended. Data on central tendency plus uncertainty and variability in cancer potency, measured using relative risk estimates (RRs) from one or more studies of human populations (i.e., epidemiological studies). Proportion of cancer cases that are fatal for individual cancer types by age, stratified by age, sex, and race/ethnicity. State mortality rate data by age and sex. Historical age-specific cancer survival data to convert lifetime cancer risk reductions into streams of benefits over time that reflect anticipated lags in the realization of benefits. VSL estimates for fatal cancers. Value of morbidity risk for non-fatal cancers (See Section 3.1.2)	Appropriate for TACs with regional or statewide exposures.	Adjustments to estimate central tendency of exposure and cancer potency and the probability distribution around those estimates to reflect variability and uncertainty. Probabilistic analysis of the product of individual risk reductions and size of exposed population expected to receive that benefit. Application of lag model to reflect the anticipated realization of reduced cancer rates over time. Estimation of the fraction of cancer cases expected to be fatal and non-fatal. Probabilistic analysis of the product of the population risk and population for avoided fatal and non-fatal cancers, respectively. Pooling of WTP values for non-fatal cancer risks. Probabilistic sampling from fatal and non-fatal WTP distributions.	Dollar estimate represents the value to society of the estimated TAC-related cancer risk reduction on a population basis (number of statistical cases of fatal and non-fatal cancers avoided). Time stream of monetized benefits can be used to estimate a discounted Net Present Value of total benefits if desired, in addition to CARB's standard non-discounted estimate. Value could be used in a BCA to compare health benefits against the costs of implementing the rule. Estimates of impacts for subpopulations could be used to inform environmental justice related analyses.

4.1.1 Decision Tree for Approach Selection

Figure 5 presents a decision tree that CARB analysts can use to determine which analytic approach may be most appropriate for a given TAC and risk assessment scope. We have endeavored to minimize the complexity of this decision process by focusing on a few key decision points that should in most cases identify the appropriate approach.

Following the decision tree will identify the most comprehensive cancer risk assessment approach that can be implemented for a given TAC and analysis type. CARB may opt to use a lower approach in some instances if that best serves available resources and risk management objectives.

Figure 5. Decision Tree for Approach Selection



Carcinogenicity. When considering a particular TAC, the first question is whether California classifies the TAC as carcinogenic to humans; OEHHA classifies TACs using the International Agency for Research on Cancer's (IARC) four-group carcinogenicity classifications. This methodology does not apply to TACs that are associated primarily with non-cancer risks nor to TACs where less is known about the carcinogenic potential of exposure. Restricting the methodology to TACs in the highest two categories for carcinogens emphasizes TACs with a strong scientific database and less uncertainty related to risk; this is consistent with U.S. EPA's recommendations in risk analyses of $PM_{2.5}$ to focus on health effects where U.S. EPA's Integrated Science Assessment concludes there is a causal or likely causal relationship between exposure and health effect. Furthermore, as noted in OECD (2017), the health endpoints used in risk assessments for socioeconomic analysis should be those that can be reliably assigned an economic value. TACs that exhibit evidence of mutagenicity or genotoxicity may ultimately someday prove to be human carcinogens, but in the absence of data on specific tumor formation, valuation will be difficult because little research exists on willingness to pay to reduce exposure to genotoxins or mutagens.

Geographic scope of impacts. The next question to answer is whether impacts of this TAC, as measured in the proposed risk assessment, are likely to be geographically localized. This could reflect a point source-related hot spot scenario with a small, more highly exposed populations nearby, or a scenario featuring a set of area sources with impacts in close proximity, where the size of the exposed population may be uncertain. In these cases, we recommend applying Approach I, which focuses on valuation of individual cancer risk reductions estimated for persons in proximity to TAC emitting sources; the valuation of individual risks can be useful for certain types of economic analysis where the exposed population is uncertain and these results can also be useful for equity-focused analyses. Some metals analyses (e.g., hexavalent chromium) would be an example of a TAC exhibiting

localized impacts. For TACs for which exposure is more widespread in the state, we recommend continuing along the tree to evaluate the use of Approach II and Approach III.

Animal versus Human toxicity data. This topic includes multiple questions, the first of which asks whether studies of carcinogenicity in humans resulting from inhalation exposures to the TAC have been conducted and if so whether any of those studies form the basis of OEHHA's recommended cancer potency estimate. If human studies do not exist or do not directly inform OEHHA's cancer potency value, CARB should conduct an Approach II analysis, which does not require the use of data from epidemiological studies. If a relative risk value is available from one or more human studies that inform OEHHA's dose-response assessment, CARB should conduct an Approach III analysis.

4.1.2 Suggested Approaches of Analysis for Priority TACs

IEc analyzed available carcinogenic data for each of the eleven priority TACs identified in an earlier phase of this project. IEc reviewed OEHHA's Technical Support Document for Cancer Potency Factors (OEHHA 2011) to understand the toxicological data for each TAC identified by CARB. For determination of the likelihood of localized effects, IEc reviewed data on chemical properties, sources of emissions, and extent of exposure from OEHHA's Technical Support Documents, supported by research into Toxicological Profiles prepared by the Agency for Toxic Substances and Disease Registry (ATSDR).

The results suggested approaches for each of the priority TACs based on IEc's initial review of the materials available are summarized in Table 3. Where multiple approaches may be relevant, we have listed a primary and secondary category.

Table 3. Suggested Approaches for Priority TACs

Priority TAC	Primary Category	Rationale
Diesel Particulate Matter	Approach III	Widespread exposure; human epi studies supporting cancer toxicity data in OEHHA Technical Support Document (TSD) for Cancer Potency Factors.
Benzene	Approach III	Widespread exposure; human epi studies supporting cancer toxicity data in OEHHA Technical Support Document (TSD) for Cancer Potency Factors
1,3 Butadiene	Approach II	Widespread exposure; OEHHA inhalation potency values based on animal studies; database also informed by human studies; OEHHA bases cancer potency on animal studies.
Hexavalent Chromium	Approach I	Limited areal extent of exposure is primary driver.
Nickel	Approach I	Limited areal extent of exposure is primary driver.
Cadmium	Approach I	Limited areal extent of exposure is primary driver.
Lead	Approach I	Lead exposure can be widespread; decision should be based on scope of risk analysis being considered for valuation; if focus is localized impacts, use Approach I; if broader, regional analysis is conducted, use Approach II (reflecting animal studies as basis of OEHHA cancer potency values).
Arsenic	Approach I	Arsenic exposure can be widespread; decision should be based on scope of risk analysis being considered for valuation; if focus is localized impacts, use Approach I; if broader,

Priority TAC	Primary Category	Rationale
		regional analysis is conducted, use Approach III (reflecting human studies as basis of OEHHA cancer potency values).
Formaldehyde	Approach II	Widespread exposure; OEHHA relies on animal data for IUR.
PCBTF	Approach II	Extent of exposure is uncertain; OEHHA relies on animal data for IUR. Selection of Approach depends on use case and whether exposure is localized.
Ethylene Oxide	Approach II	Widespread exposure; OEHHA uses animal data to derive an IUR in its current risk assessment. IEc understands that OEHHA is currently drafting a new ethylene oxide cancer toxicity assessment using human data. If OEHHA updates this value, ethylene oxide will become an Approach III pollutant.

4.2 Data Sources

4.2.1 Minimum Lag

For fatal and non-fatal cancer health outcomes, there is an expected minimum time lag between changes in pollutant exposure in a given year and the realization of health effect benefits from pollution in the form of avoided cancer, commonly referred to as the "cessation lag." Due to the mechanisms of cell mutation and carcinogenicity, the time between exposure and diagnosis can be on the order of years and varies across cancer sites. We establish a minimum for this value based on the minimum lag periods between exposure and diagnosis taken from the Center for Disease Control's (CDC) World Trade Center Health Program, a source initially identified by LaPenta et al. (Howard 2015). The values identified in Howard (2015) are presented in Table 4 below. The actual lag modeled for a given risk reduction in Approach II is also a function of the age at exposure and the historical cancer incidence by age; thus, the total lag applied may be larger than this value but cannot be less than it.

Table 4. Minimum Lag by Cancer Site

Cancer Site	Minimum Lag (years)
Childhood cancers	1
Lymphoproliferative and hematopoietic cancers	0
Thyroid cancer	3
All other cancer sites	4
Mesothelioma	11

4.2.2 Incidence Rates

Incidence data are used in two ways in this methodology:

1. In Approaches I and II, historical cancer incidence rate data by age are used following the total population risk calculation to allocate those expected population risk impacts over time, using the method applied in LaPenta et al. (2024). For example, the risk reduction experienced by a 70-year old

following TAC emissions reductions is first decreased to account for some "avoided" lifetime risk due to reaching age 70 without cancer. The remaining lifetime risk is then allocated over that individual's remaining lifetime based on the relative probability of a relevant cancer diagnosis at various ages.

2. In Approach III, where the risk values employed in the risk assessment are relative risks that are estimated as proportional to existing baseline incidence rates, these data are incorporated directly to estimate the risk to a given age group at any particular point in the analysis, based on their exposure history relative to baseline.

To distribute lifetime incidence through the years of a person's life and adjust risk for age at the time of exposure, we create cancer-specific incidence profiles that we use to adjust and distribute lifetime risk for Approaches I and II. We calculate the proportion of cases that occur at each age from 0 to 100 for each relevant population group, building an age profile of case incidence based on observed cancer incidence data. For Approach III, baseline incidence rate data by age are built directly into the risk model and adjusted based on relative risks of exposed versus baseline groups.

Our model employs incidence rates that are a combination of California-specific and national data. The incidence data we use reflects the rate of diagnoses for a specific cancer in a given population each year. We obtain cancer incidence data for all cancer sites listed in the SEER Site Recode from the California Cancer Registry (CCR) for the years 2016-2021 (excluding 2020 due to confounding effects of COVID). These data are stratified by sex, age, race/ethnicity, and county of residence (CCR 2025).[12] For situations in which data were suppressed by CCR due to low incidence rates and/or small county population, CCR incidence rates were supplemented with nationwide incidence rates from CDC WONDER for years 1999-2021 (CDC). More details on this process are described in Appendix A.2.

These data were used to create age profiles of incidence detailing the proportion of cases that occur in each year. For each age of a person's life, we multiply the total risk by the proportion of diagnoses that occur at that age. For instance, if the lifetime risk for an individual is 10 per million, and 5 percent of lung and bronchus cancer diagnoses for that individual's demographic group occur at age 65, the risk to that person in their 65th year is 5 percent of 10 per million, or 0.5 per million. By nature, this method lowers the overall risk experienced by an individual commensurate with their age. For instance, if, for a given demographic group and cancer, 60 percent of the cases occur after the age of 65, the sum of each age-specific incidence proportion after age 65 will equal 60 percent, effectively reducing the total risk borne by 40 percent. This age-based adjustment addresses the need to augment lifetime incidence to reflect future lifetime risk.

4.2.3 Survival Rates

Survival data are used to determine the probability of death in each year after diagnosis for a given cancer. LaPenta et al. use 20-year age and sex-specific relative survival rates in their analysis obtained from SEER*Stat using NCI's CanSurv software (LaPenta et al. 2024). In addition to their methods, LaPenta et al. published the survival data used in their analysis. We use this same data with permission from the authors.

4.2.4 Age-Specific Adjustment Factors

We use OEHHA's age-specific adjustment factors to adjust the inhalation cancer risks resulting from exposure during childhood. OEHHA's age-specific adjustment factors, shown in Table 5, are defined in its 2009 *Technical Support Document for Cancer Potency Factors* (OEHHA 2009).

[12] Cancer site naming conventions differ by data source and some sources provide more specific cancer sites than others. For the purposes of this model, we categorize cancer sites using more generic cancer groupings (e.g. leukemia rather than leukemia, acute myeloid leukemia, and chronic myeloid leukemia).

Table 5. OEHHA Age-Specific Adjustment Factors

Age Range	Age-Specific Adjustment Factor
0–1 Years	10
2–16 Years	3
17+ Years	1

If, for instance, the risk to 14-year-olds in a given county is calculated to be six per million, we apply the age-specific adjustment factor, yielding a final risk of eighteen per million for that group of 14-year-olds.

4.2.5 Sex-Specific Incidence and Mortality Data

Throughout our methodology, we sometimes stratify incidence rates by race and county, and we sometimes do not; however, we always use sex-stratified incidence and survival rates. Unlike county- and race-specific incidence data, where differences in incidence rates are largely a product of environmental exposure, sex-specific incidence and mortality rate disparities have a nontrivial biological component. The current scientific literature has not identified the exact degree to which biological factors drive observed differences in male and female carcinogenicity, but the current literature suggests that biological factors are the predominant driver of these differences (Jackson et al. 2022). Due to these factors and the ongoing state of research into sex-specific cancer incidence, we maintain sex-stratified incidence and survival rates throughout our analysis.

CHAPTER 5 | Approach I: Valuing Individual Risk Changes

5.1 When to Use Approach I

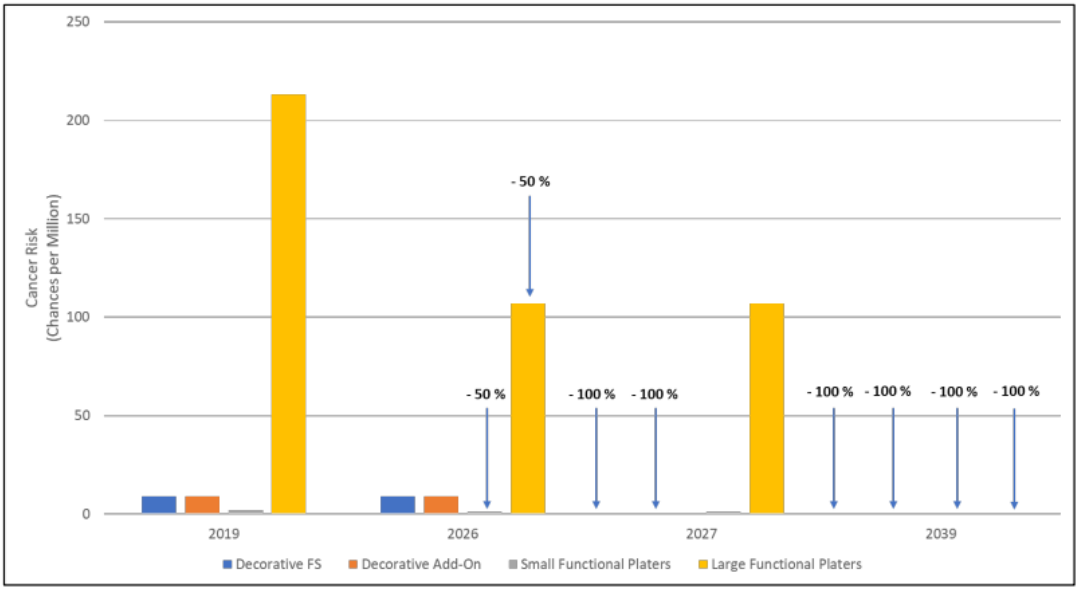
Our first method for analysis, Approach I, provides a mechanism for valuing TAC-related cancer incidence changes that occur on highly localized scales. This can occur due to several factors. For example, a TAC may be released from a smaller number of individual facilities across the state and/or its physical and chemical properties or emissions characteristics (e.g., stack height) may limit its dispersal. Hexavalent chromium is one example of a recent TAC risk assessment with localized impacts (CARB, 2022b). These chemicals contrast with widespread, dispersed pollutants like benzene, which are emitted from a widely-distributed network of stationary and mobile sources, and which are more evenly dispersed throughout the state. The emissions pattern of these localized pollutants, combined with the low atmospheric dispersion exhibited by these chemicals, means that pollution from these chemicals often contribute to a series of hotspots.

In these cases, population-scale analysis is hampered by the fact that the relevant impacted area, which may be on the scale of several hundred meters, can be smaller than the smallest demographic data available, increasing uncertainty in population estimates. Due to the heterogeneity of specific developments within census tracts or census block groups, assuming even population density throughout a census subdivision is inappropriate. Furthermore, the cancer-related impacts of TAC exposures may prove difficult to observe in smaller populations, even when the risk change to individuals living in close proximity to these facilities may be substantial. In these cases, we believe it is most appropriate to value changes in individual lifetime cancer risks for exposed populations, rather than undertake a population risk analysis. Recent methodological advances in the economic valuation of cancer risks discussed in Chapter 3 now make it possible to incorporate the "bridging" adjustments that allow a risk assessment to better inform a socioeconomic valuation process into the economic values themselves (LaPenta et al. 2024). These advances provide a stronger economic foundation for direct valuation of individual cancer risk changes because they account for issues of timing of risk changes that are not typically addressed in CARB's TAC risk analyses. For Approach I, we provide weighted average economic values corresponding to a one-in-one million individual lifetime cancer risk change, enabling CARB to value the individual risk change estimates they calculate for exposed individuals living or working near an emitting facility. These individual values could be used, for example, to inform break-even analyses which would aim to answer the question, "How many individuals would need to experience reduced TAC exposure for the benefits of the policy to justify its costs?"

5.1.1CARB Approach for Exposure Assessment and Dose-Response Assessment

We derive our understanding of CARB's localized risk assessment methodology from CARB's 2023 *Proposed Amendments to the Airborne Toxic Control Measure for Chromium Electroplating and Chromic Acid Anodizing Operations: Appendix F*, which contains an analysis of the risks posed by chrome plating facilities of different sizes. For this risk assessment, CARB combines data on wind patterns, stack height, annual emissions, and cancer potency to develop risk data for individuals living near small, large, decorative and functional chrome plating facilities. The impact of the proposed regulation is presented as changes to the baseline cancer risk faced by someone living or working in the immediate vicinity of the facility. **Error! Reference source not found.** shows the impact of the proposed hexavalent chromium emissions regulation on individual resident cancer risk from the baseline year of 2019 to the final year of 2039. Each color of column represents a different type of chrome plating facility: blue being decorative platers with fume suppressants, red being decorative add-on platers, grey being small functional platers, and yellow being large functional platers.

Figure 6. Individual Resident Cancer Risk, from ATCM: Appendix F



Individual resident cancer risk estimates are based on a 30-year exposure duration using the Risk Management Policy (RMP) derived method (95th percentile/80th percentile daily breathing rates (DBR)). FAH equals 1 for age bins <16 years and 0.73 for age bin 16-30 years. All numbers are rounded.

These risk reductions are estimated in the year in which the regulation affects the polluting facilities, and CARB indicates that estimates are based on 30 years of exposure. It is not clear to IEc, however, what pattern of exposure is assumed over those 30 years. For example, individuals living near large functional platers experience exposure reductions that are phased in over time. Does the 30-year lifetime risk estimate for 2019 above assume constant exposure at 2019 levels (i.e., is it a baseline estimate), or does it incorporate the expected changes to exposure in 2026 and 2039? Similarly, does the 2026 estimate include the expected additional reduction in 2039? If not, the expected lifetime risk reduction as of 2026 could be even larger compared to a constant baseline. Clarifications of these risk assumptions is key for understanding what is being valued.

5.2 Approach to Risk Assessment and Valuation

Approach I generally features no changes to individual changes in lifetime cancer risk estimated in standard CARB risk assessments; changes focus on the allocation of risk changes over time and the valuation of those risks. Below we describe the valuation-related changes, and then indicate possible additional bridging adjustments to risk that CARB could consider.

5.2.1Valuation

The bulk of the bridging adjustments under an Approach I analysis occurs in the development of the valuation estimates. As noted in Chapter 3, timing plays a key role in the valuation process, and accounting for the timing of risk reductions is critical to valuing risk changes properly. Furthermore, CARB needs to estimate the proportion of the risk that represents a fatal cancer versus a non-fatal one, so that the appropriate valuation estimates can be applied. The method described below applies the VMR/VNR method described in Chapter 3, to incorporate these considerations directly into the generation of an economic value of a one-in-one-million cancer risk change, so that the risk estimates themselves do not need to be adjusted. Furthermore, IEc has adapted this method to enable CARB to develop California specific values for use with Approach I and to incorporate income growth in these values over time.

To create estimates of the impact of a one-in-one-million risk change to a person in a given age group, we take a weighted average of the impact of a one-in-one-million risk change to the age group CARB is investigating. To calculate these values, we apply the methodology employed in research conducted by Abt Global for U.S. EPA's Office of Pollution, Prevention and Toxics and later published in the *Journal for Benefit Cost Analysis* (Abt Global 2022, LaPenta et al. 2024):

- Distribute one-in-one-million risk proportionally by age and sex;
- For each age/sex combination, distribute proportional one-in-one-million risk from the first possible age of diagnosis to the end-of-life age;
- For each diagnosis year, distribute the statistical risk of dying from that cancer;
- Value the non-fatal and fatal risks for each year by **a)** discounting the values of a one-in-one-million non-fatal cancer risk reduction and mortality reduction, respectively, back to the year of exposure, and **b)** adjusting these values to account for income growth and elasticity;
- Calculate and sum these values for each age of exposure, and for each year of diagnosis possible for each age of exposure; and
- Divide this sum by the sum of the proportional risk in Step 2 and the distributed statistical risk value in Step 3 across all ages of exposure and diagnosis, creating a weighted average value of statistical risk change in that year

We follow this approach to create weighted average economic values of cancer risk for different cancer types, using California-specific incidence profiles and population distributions. This approach yields cancer- and population group-specific weighted average values of a one-in-one million cancer risk reduction. The following sections present and explain how we create these weighted average values.

5.2.1.1 Valuation of One-in-one-million Risk Change

The proposed Approach I valuation is fundamentally straightforward. The value is calculated as the product of the individual lifetime risk reduction for a particular cancer, expressed as chances per one million, multiplied by the corresponding value for a one-in-one million lifetime risk reduction for that cancer. The correct value to use depends on the type of cancer; the sex and age of the individuals experiencing the exposure change; and the year in which the risk reduction occurs. Details of how that calculation in performed are described in Appendix C.

Example Valuation Table

Table 6 presents an example table of year-specific values of one-in-one million changes in lifetime lung and bronchus cancer risk resulting from exposure reductions in the specified year for selected age groups. These values differ primarily due to differences in survival rates between age groups and the proportion of lifetime risk experienced by different age groups. The calculation of these valuation estimates involves the same VSL and non-fatal WTP values for both age groups.

Table 6. Example Valuation of a One-in-One Million Lifetime Change in Lung and Bronchus Cancer Risk (2023$)†

Year of Exposure	0–17-year-olds (Male and Female Combined)		50–75-year-olds (Male and Female Combined)	
	Undiscounted	2% discount rate*	Undiscounted	2% discount rate*
2025	$16.82	$4.68	$13.64	$9.38
2026	$16.90	$4.70	$13.70	$9.43
2027	$16.98	$4.72	$13.77	$9.48

Year of Exposure	0–17-year-olds (Male and Female Combined)		50–75-year-olds (Male and Female Combined)	
	Undiscounted	2% discount rate*	Undiscounted	2% discount rate*
2028	$17.07	$4.74	$13.84	$9.53
2029	$17.15	$4.77	$13.91	$9.57
2030	$17.23	$4.79	$13.98	$9.62
2031	$17.31	$4.81	$14.05	$9.67
2032	$17.39	$4.84	$14.12	$9.72
2033	$17.48	$4.86	$14.19	$9.76
2034	$17.56	$4.88	$14.26	$9.81
2035	$17.65	$4.91	$14.33	$9.86
2036	$17.73	$4.93	$14.40	$9.91
2037	$17.82	$4.95	$14.47	$9.96
2038	$17.90	$4.98	$14.54	$10.01
2039	$17.99	$5.00	$14.61	$10.06

* Columns are discounted back to the year of exposure in the left-hand column.
† Income growth adjustments included in all estimates.

It is important to note that these data are discounted back to the year of exposure in the left-hand column, not back to any given reference year. If CARB were to discount these values back to a base year, they would perform that discounting on these values, according to the form:

$$\text{Value}_{\text{Base Year}} = \frac{\text{Value}_{\text{Exposure Year}}}{(1 + \text{Discount Rate})^{\text{Exposure Year} - \text{Base Year}}}$$

The higher *undiscounted* valuation for 0–17-year-olds is due to their greater remaining life-expectancy. The 50–75-year age group experiences slightly decreased lifetime risks due to their shorter expected remaining life-years. The older age group has a higher *discounted* valuation because the risk that they face is likely to occur sooner, given the higher incidence of lung cancer among this age group than among 0-17 year olds. Finally, the annual values increase over time due to the income growth adjustments that IEc has incorporated (see Chapter 3). Assuming per-capita income continues to grow over time, these adjustments will act in the opposite direction of the discounting effect.

See Appendix C for a complete explanation of how we perform these weighted average valuation calculations.

5.2.2 Approach I Outputs Generated

Outputs of the Approach I process include:

- A table of year- and age-specific economic valuation estimates for the target cancer type;
- Summary tables of the dollar values corresponding to risk reduction benefits per individual living in proximity to a TAC emitting source. Tables will provide values for each type of exposed group (e.g.,

resident, worker) under each regulatory scenario, where benefits represent the difference in individual lifetime risk between the baseline and regulatory scenario.

5.2.3 Additional Modifications Possible for Approach I

For Approach I analyses, characterization of population risk is not the goal; as a result, there is no need to develop full population distributions of exposure and dose-response as we do for Approaches II and III. There may be situations, however, where CARB may want to consider developing additional individual risk estimates that modify exposure and dose-response to better reflect a central tendency individual.

- Following CARB guidance, these inhalation exposures are modeled using a combination of OEHHA's cancer potency factor and individual inhalation rates; use of a risk estimate based on a comparison of exposure concentrations to an IUR may better reflect central tendency exposure than a risk estimate based on the CSF. This is supported by U.S. EPA's Risk Assessment Guidance for Superfund, Part F, which notes that "It is generally not appropriate to make adjustments based on ventilation rate and body weight using the intake equation [with cancer slope factor values], because the amount of the chemical that reaches the target site of the chemical through the inhalation pathway is not a simple function of the inhalation rate and body weight."
- Although a 70-year exposure duration is recommended in CARB guidance for calculating population risk, it may make more sense to include in the Approach I context so as to illustrate an upper bound value on the per-person benefit of individual risk reduction, in addition to calculations using 30 years.
- Developing low and high estimates based on variation in dose response (see Approach II methodology described in Appendix B) would provide added understanding of the potential range of benefit values that could accrue to individuals.

The resulting additional risk estimates, while not strictly necessary, could allow for a more nuanced discussion of a break-even analysis. Rather than estimating only a single break-even estimate for the individuals who meet the criteria in CARB's risk guidance, CARB could also present values for individuals with different characteristics and reflect a range of possible values that might better help decision makers evaluate the likelihood of a break-even scenario. If a break-even analysis is not conducted, it can help better present the potential range of values of the individual risk reductions that may result from the regulation.

An alternative approach for valuation purposes would involve estimating the incremental lifetime cancer risk associated with a single year's emissions reduction, rather than 30 years. This lifetime risk reduction for a one-year change in exposure, expressed as chances per million risk could then be multiplied by the value of a one-in-a-million cancer risk reduction to estimate an annual benefit to the individual experiencing that change. That value would accrue annually for as long as that exposure reduction remained in place. If the exposure decreases again in 2039, the annual risk-reduction benefit would increase accordingly, starting in that year. This would mirror the approach followed by U.S. EPA in its TSCA risk assessment for proposed reductions in exposure to methylene chloride (2023). These results could then be used to generate a stream of annual benefits that could be reported as is or used in break-even analysis.

5.3 Limitations and Uncertainties

Because it relies mostly on adjustments made in the valuation step, key limitations mirror those cited in Chapter 3 regarding the methods and data underlying the estimates of VNR and VMR, as well as the estimates of cancer mortality used to weight the VNR and VMR. Other limitations include:

- **Uncertainty in timing of cancer and mortality risk changes.** The assumptions about timing of cancer risk changes over one's remaining lifetime and the likelihood of mortality post-diagnosis inform the valuation figures; they are informed by data on cancer incidence by age and survival rates that are

representative of a specific period of time. Approach I valuations assume that risk reductions in the future will follow similar patterns; if this is not the case, e.g., if medical advances reduce cancer mortality rates in the future, the values could differ from the predicted values.

- **Limited characterization of uncertainty and variability.** The Approach I methodology does not incorporate more extensive probabilistic characterizations of exposure and dose-response variability and may give a more limited characterization of the full range of potential benefit values experienced by exposed individuals. This would limit its usefulness in the context of efficiency-focused analyses such as benefit-cost analysis. However, this characterization may be sufficient for evaluating impacts from an equity perspective.
- **20-year survival rates.** As discussed in Section 4.2.3, we utilize 20-year survival rates to allocate cancer risks between fatal and non-fatal cases. While most cancer deaths occur within this window, a relatively trivial portion of deaths occur after the first two decade for some cancers. Due to the higher unit value (VMR) for fatal risks, the approach may understate the benefits of TAC controls by a modest margin.

CHAPTER 6 | Approach II: Valuing Population-Level Risk Changes Based on Animal Toxicity Data

6.1 When to use Approach II

The Approach II methodology presents a methodology for valuing population-level changes in cancer cases that result from TACs with more widespread exposures throughout California. Approach II analysis quantifies and values an estimated number of avoided fatal and non-fatal cases of a target cancer due to changes in ambient pollutant levels of a TAC. It is appropriate for known or likely carcinogens whose cancer potency is based exclusively on animal studies.

Exposures to Approach II TACs are likely to affect a broader range of the population, with varying life histories, activity patterns, and susceptibility. Consequently, in order to adequately portray the risk to the population for a socioeconomic analysis, Approach II focuses on bridging methods that describe more expansively the range of exposure and cancer susceptibility within that population.

Pollutants appropriate for Approach II analysis are widespread, ambient pollutants that cause population-scale epidemiological effects. While it is undoubtedly true that nearly every pollutant release affects multiple people, a population-level exposure is one that transcends a block, or a neighborhood, and affects the population at a macroscopic scale.

Where human epidemiological data exist and support OEHHA's cancer potency values, we strongly recommend applying Approach III, described in Chapter 7, since it represents the most rigorous method that produces the most detailed sets of results. There may be situations, however, where CARB may wish to use Approach II analysis with unit risk data, instead of Approach III. Examples include:

- CARB considers the human epidemiological data for a specific carcinogen-cancer site relationship to be outdated or inadequate for the purpose of providing a relative risk value
- Human toxicity data exist for one human cancer site (e.g., lung and bronchus cancer) but not for another relevant site (e.g., leukemia), and a high degree of concordance exists between that animal cancer site and the associated human cancer site

The following chapter presents the structure and decision points necessary for conducting Approach II analysis, clarifying how we unite various data sources and statistical techniques to quantify and value human cancer changes.

6.2 Quantifying Risk Changes

6.2.1 Hazard Identification

As with other approaches in this methodology, the hazard identification step of risk assessment should be reviewed to ensure that the health effect for which risk is being calculated is one that is amenable to valuation. For example, evidence of genotoxicity or mutagenicity alone, or evidence of modification to some upstream cancer biomarker is unlikely to have associated WTP values.

TACs that involve a mixture of contaminants (e.g., DPM) should focus on health endpoints that are based on studies of that mixture that are similar to exposures likely to be experienced by the population being analyzed. Where mixtures may differ from studied exposures this difference should be acknowledged as a limitation, and

quantification of this uncertainty, e.g., using risk assessment of speciated components, is encouraged where possible.[13]

6.2.2 Exposure Characterization

This section describes bridging methods recommended to enable the risk assessment to better support socioeconomic analysis under Approach II, These bridging steps include air quality surface development and incorporation of variability and uncertainty.

6.2.2.1 Air Quality Surfaces

As previously discussed, Approach II analyses involve widespread, ambient pollutant changes. To conduct an Approach II risk analysis for a certain regulatory action, CARB will require air quality surfaces for the pollutant in question under the baseline and regulatory scenarios being evaluated. We expect that the modeling of ambient TAC concentrations conducted for a standard CARB risk assessment, following OEHHA guidelines for air quality modeling, will be suitable for use in the Approach II analysis (OEHHA 2015). Since the Approach II methodology should not alter the baseline and regulatory scenarios, no modifications to this modeling are expected.

To perform an Approach II analysis of the impact of a regulatory action, CARB can either:

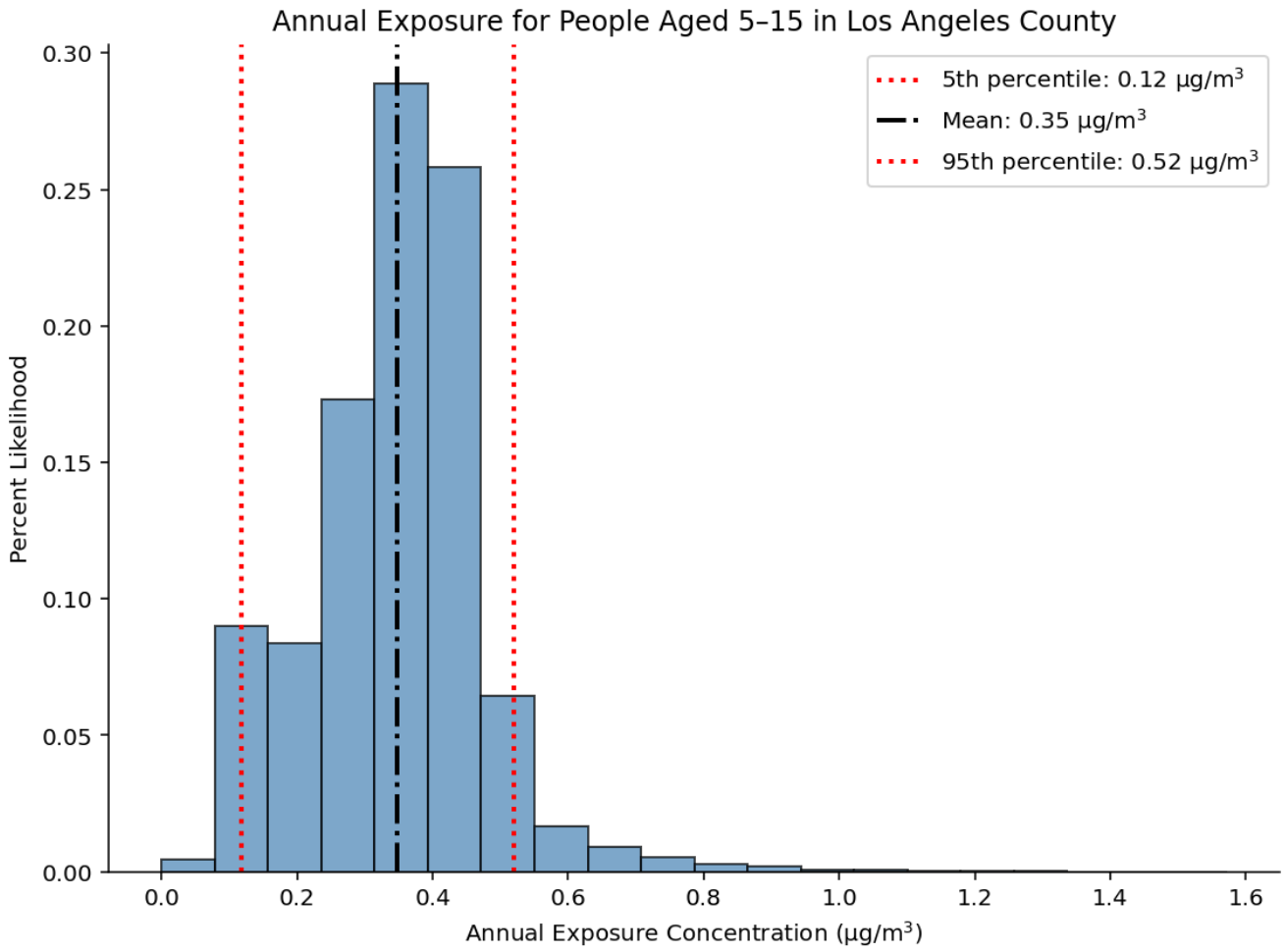
a) Run a counterfactual "business-as-usual" scenario and a regulatory scenario through our Approach II analysis, taking the difference in total health costs between the two scenarios as the benefit for that regulatory action; or
b) Run a direct "benefits analysis" by estimating avoided pollution between the two scenarios and running these changes in pollution through our Approach II analysis.

6.2.2.2 Incorporating Variability and Uncertainty in Exposure

Once CARB has modeled the quality surface for an Approach II pollutant, we recommend that CARB uses U.S. EPA's HAPEM8 software to transform ambient pollutant concentrations into estimates of annual exposure concentrations to exposed groups that account for variability in time-activity patterns. HAPEM8 will produce county- and age-specific exposure distributions resembling the example in Figure 7.

[13] https://ww2.arb.ca.gov/speciation-profiles-used-carb-modeling

Figure 7. Example HAPEM8 Output for Benzene Exposure for 5-15-Year-Olds LA County



More information about U.S. EPA's HAPEM8 model can be found in Appendix A. Using HAPEM8 will help to bridge the central estimate of exposure to be more consistent with the average individual and will provide a distribution of potential exposures within a county based on a range of tract-specific exposures, interindividual variability in exposure levels, and uncertainty in population composition.

6.2.3 Dose-Response Assessment

6.2.3.1 Cancer Site Selection

For chemicals that we recommend CARB conduct an Approach II analysis, dose-response relationships are based on animal toxicity studies. Assessing animal and human tumor site concordance is an area of ongoing research, so, for Approach II, we recommend CARB assess default "all cancer" valuation unless otherwise indicated with OEHHA. CARB should consult OEHHA prior to selection of any single cancer site for analysis in Approach II. Even where animal toxicity studies assess carcinogenicity in a single tumor, mechanisms of carcinogenicity in laboratory animals, such as mice and rats, may differ from those of humans, meaning that the types of cancer in humans may differ. Furthermore, some test animal cancer sites, such as the preputial gland,

are not found in humans (See OEHHA 2009 Appendix B: Benzene), further complicating assignment of a cancer unit risk value to a specific tumor site.[14]

6.2.3.2 Uncertainty and Variability

As previously discussed, our Approach II methodology values population-scale changes in incremental cancer risk expected to result from TAC emissions regulations. We provide more detail below on how we bridge dose-response data from CARB's current approach to risk values appropriate for population-scale socioeconomic analyses.

First, IEc recommends that CARB use IUR values published by OEHHA, expressed in $(\mu g/m^3)^{-1}$, when calculating risk changes under Approach II to support valuation and socioeconomic analysis. We recommend that CARB use unit risk values rather than cancer slope factors for the following reasons:

1. Per U.S. EPA's Risk Assessment Guidance for Superfund, Part F (RAGS): "It is generally not appropriate to make adjustments based on ventilation rate and body weight using the intake equation [with cancer slope factor values], because the amount of the chemical that reaches the target site of the chemical through the inhalation pathway is not a simple function of the inhalation rate and body weight." (U.S. EPA 2009). This suggests that use of an IUR to characterize central tendency and variation in exposure may be a better approach than by scaling the CSF by body weight and inhalation rate.
2. Coupling the IUR with HAPEM8 will incorporate variability across population activities to help compensate for any loss of flexibility resulting from use of the IUR.

IURs provide an easily understandable, health-protective toxicity value that is useful for many kinds of risk screenings and health analyses. For socioeconomic population-scale analyses, incorporating population variability in potency is key for characterizing the interpersonal variability in cancer susceptibility and risk in a given population. This assertion rests on the notion that two people may have very different health outcomes despite being exposed to the same exact pollutant dose. Thus, we recommend constructing a probabilistic distribution based on OEHHA's unit risk value that represents the range and distribution of cancer risk profiles throughout a population. We conducted a literature review of risk assessment methods development, with a focus on characterizing uncertainty; that review identified methods of varying complexity. In the interest of developing a scientifically sound method that is applicable to the broadest number of TACs, we are recommending a method that incorporates guidance on animal-human extrapolation provided by the National Research Council in their book *Science and Decisions: Advancing Risk Assessment* (2009) and by the WHO/IPCS (2018).

6.2.3.3 Creating a Population Distribution for the IUR

Following guidance from the National Research Council and WHO/IPCS, we recommend that CARB model variability in population cancer susceptibility lognormally and treat the animal data-derived unit risk as reflective of the population median value of a lognormal distribution of potency.[15] Both the National Research

[14] For an overview of the current state of animal-human site concordance research, we recommend consulting Krewski et al. (2019), a meta-analysis of cancer site concordance in animal and human cancer studies conducted by the IARC, an agency of the World Health Organization (WHO). The paper reviews the 119 *Monograph* volumes produced by the IARC Monographs Programme and identifies the concordance of animal and human carcinogenicity for 112 Group I agents found to meet the criteria for classification as "carcinogenic to humans." The paper reviews both animal and human studies for each carcinogen-tumor site combination and identifies whether there was evidence of carcinogenicity at that site in humans only, animals only, or both humans and animals.

[15] See recommendations for treatment interindividual variability in dose-response on pages 168 – 169 of *Science and Decisions: Advancing Risk Assessment* (2009).

Council and WHO/IPCS recommend modeling the interindividual cancer susceptibility lognormally. A lognormal distribution helps to capture the "long tail" of highly susceptible individuals who experience significantly elevated carcinogenicity than an individual of more "typical" susceptibility at equal doses. Animal studies are performed on highly homogeneous animal populations that lack the genetic and immunological variability of human populations. As such, the National Research Council recommends treating animal-derived human toxicology estimates as median estimates for human populations. The shape of the distribution, containing this median, however, is uncertain. A range of shapes of this distribution is provided by WHO/IPCS, who provide a distribution of 95th-to-50th percentile cancer susceptibility ratios in humans. By applying these varied 95th-to-50th percentile ratios to the existing 50th percentile (median) estimate, CARB can create a range of population susceptibility distributions, which can be combined with the exposure distribution to estimate population mean cancer risk (See Appendix B).

6.2.3.4 Monte Carlo Analysis

To perform the Monte Carlo analysis, we first recommend sampling from the distribution of 95th-to-50th ratios, selecting a value to parameterize the population distribution of risk. Then, we recommend sampling from that population distribution (for a range of individual susceptibilities) and the HAPEM8 exposure distribution (for a range of individual exposure estimates), multiplying each sampled value to create a composite distribution of cancer risk estimates reflecting the difference between baseline and regulatory exposures. This step is designed to account for population variability in susceptibility and exposure which change the shape of the final risk distribution. The resulting (unitless) distribution of risk values is calculated by multiplying exposure (in $\mu g/m^3$) by unit risk (in $[\mu g/m^3]^{-1}$). We repeat these steps for different 95th-to-50th percentile ratios to account for uncertainty in that parameter, and extract a mean population risk from each trial, resulting in a final distribution for the mean population risk. See Appendix B for a complete explanation of this process.

6.3 Risk Characterization

6.3.1 Lifetime Risk Profiles

After performing the Monte Carlo analysis described in step 6.2.3, CARB will possess a mean estimate, 5th percentile estimate, and 95th percentile estimate, for the lifetime cancer risk change for each demographic and age group in our analysis. This lifetime risk does not all occur in the same year; therefore, we must distribute this risk from the day on which each statistical case may begin to the end of that person's life. We accomplish this by applying the incidence profiles we derive from California-specific and national data, in the same manner used by LaPenta et al. (2024) and by U.S. EPA in its TSCA risk assessments (e.g., the 2023 methylene chloride assessment). For each age of exposure assessed, we multiply the risk by the proportion of diagnoses that happen in each eligible year of diagnosis to distribute lifetime risk throughout the remaining life profile. (See Section 4.2.2 and Appendix A.2 for more detail.) We will multiply these proportions by the size of the population, the calculated population risk, and the relevant Age-Specific Adjustment Factor to calculate the number of diagnoses at each possible age of diagnosis for exposure to a given age group. (See Section 6.4.3.)

6.3.2 Calculating Cancer-Specific Mortality

For each diagnosis age, we distribute mortality risk across a 20-year mortality window by using cancer site-, sex-, and age of diagnosis-specific survival rates from SEER. (See Section 4.2.3 for more.)

6.3.3 Combined Approach II Equation

The following equation unites all of the aforementioned elements of our Approach II methodology, combined with data on the size of the affected population, to produce the value of a specified risk change in a given county, ϕ, as distributed across all of the different ages living in that county.

Equation 2. Value of Sex- and County-Specific Exposure Changes

$$\text{Benefit}_{i,s} = \sum_{\alpha=0}^{100-\text{lag}_\alpha} \sum_{\beta=\alpha+\text{lag}_\alpha}^{100} \phi_{\alpha,i,s} \times \text{ASAF}_\alpha \times \text{Pop}_{\alpha,i,s} \times d_{\beta,i,s} \times \left(S_{\beta,s} \times \frac{\text{VNR} \times \left(\frac{I_{y+\beta-\alpha}}{I_y}\right)^{\varepsilon_{NF}}}{(1+\rho)^{\beta-\alpha}} + \sum_{k=1}^{20} m_{\beta,k,s} \frac{\text{VMR} \times \left(\frac{I_{y+\beta+k-\alpha}}{I_y}\right)^{\varepsilon_F}}{(1+\rho)^{\beta+k-\alpha}} \right)$$

Where:

- α = Age of exposure
- β = Age of diagnosis
- y = 2023, the base year for income growth adjustments for VNR and VMR estimates from LaPenta et al. (2024)
- i = County
- s = Sex
- k = Years since diagnosis
- ρ = Discount rate
- lag_α = Minimum lag between exposure and diagnosis for exposure at age α
- $\phi_{\alpha,i,s}$ = Risk borne by people at age α of sex s in county i
- $\text{Pop}_{\alpha,i,j,s}$ = The number of people at age α, race j, and sex s in county i
- ASAF_α = The age-specific lifetime risk adjustment factor for exposure at age α
- $d_{\beta,i,j,s}$ = The proportion of lifetime diagnoses that take place at a given diagnosis age, dependent on the county, race, and sex of the group in question
- $\varepsilon_{NF}, \varepsilon_F$ = Income elasticity for non-fatal (*NF*) and fatal (*F*) cancer risk, respectively
- $S_{\beta,s}$ = The probability of surviving a given cancer given sex and age at diagnosis
- $m_{\beta,k,s}$ = The probability of dying from a given cancer case k years after diagnosis at age β for sex s
- $I_{y+\beta-\alpha}/I_y$ is the income growth factor for the year of diagnosis, i.e., $2023 + \beta - \alpha$. For mortality, the year is instead given as $2023 + \beta + k - \alpha$
- VNR = The value of a 1/1,000,000 reduction in risk of contracting a non-fatal case of cancer
- VMR = The value of a 1/1,000,000 reduction in the risk of dying

6.3.4 Approach II Equation Explanation

Here, we deconstruct the Approach II equation, explaining how its component pieces combine to value risk changes to different groups in California.

First, the valuation term is identical in structure to that of the Approach I equation, and takes the following form:

$$\left(S_{\beta,s} \times \frac{\text{VNR} \times \left(\frac{I_{y+\beta-\alpha}}{I_y}\right)^{\varepsilon_{NF}}}{(1+\rho)^{\beta-\alpha}} + \sum_{k=1}^{20} m_{\beta,k,s} \frac{\text{VMR} \times \left(\frac{I_{y+\beta+k-\alpha}}{I_y}\right)^{\varepsilon_F}}{(1+\rho)^{\beta+k-\alpha}} \right)$$

When simplified (See Appendix C.1 for an explanation of this simplification in our Approach I calculations), this expression can be thought of as:

$$\left(S_{\beta,s} \times \text{NF\$}_{\alpha,\beta} + \sum_{k=1}^{20} m_{\beta,k,s} \times \text{F\$}_{\alpha,\beta,k}\right)$$

Where $\text{NF\$}_{\alpha,\beta}$ is the value of a non-fatal cancer case for a person exposed at age α and diagnosed at age β, and $\text{F\$}_{\alpha,\beta,k}$ is the value of a fatal cancer case occurring k years after diagnosis at age β due to exposure at age α. This term gives us the total value of a risk change for a given age of diagnosis, distributed between the value of cancer-related deaths and non-fatal cancer cases.

This value is multiplied by the following expression:

$$\phi_{\alpha,i} \times \text{ASAF}_{\alpha} \times \text{Pop}_{\alpha,i,s} \times d_{\beta,i,s}$$

Taken together, this expression represents the actual population-adjusted risk for a given age group at a given age of diagnosis. The first three terms are constant for each age group. The $\phi_{\alpha,i}$ term represents the calculated risk (whether the mean, 5th percentile, or 95th percentile estimate) faced by people in county i at age α. This risk is multiplied by OEHHA's Age-Specific Adjustment Factor (ASAF_{α}) that scales risk for a given age (See Section 4.2.4 for more). Finally, this is multiplied by the size of the population for the group being analyzed, as determined by age of exposure and sex. These first three terms together give the magnitude of risk faced by a given age- and sex-determined group.

Consider a hypothetical risk analysis in Monterey County, CA involving 100,000 13-year-old boys in Monterey County, and that these boys are subject to a lifetime individual risk change of 0.00004, or 40 in a million. These boys, as a population cohort, have a population risk of $\phi_{\alpha,i} \times \text{ASAF}_{\alpha} \times \text{Pop}_{\alpha,i,s} = 0.00004 \times 3 \times 100{,}000 = 12$ statistical cases, since their age-specific adjustment factor is 3.

These 12 statistical cases must now be distributed throughout the lifetimes of the age cohort. We iterate through all possible ages of exposure and multiply this risk by $d_{\beta,i,s}$, the proportion of lifetime diagnoses that occur in each age group. We value these diagnoses using the valuation expression above.

Then, we iterate through every age of exposure for that sex in that county, evaluating each age of exposure for its associated diagnosis ages. For this iteration, we begin our calculation of benefits at age 0, as we account for exposures in the first year of life. We end our calculation of benefits at age $100 - \text{lag}_{\alpha}$, since we stop counting diagnoses at age 100, and age $100 - \text{lag}_{\alpha}$ is the latest age at which someone can be diagnosed in our model.

Combining these individual components yields the following conceptual representation of our Approach II equation:

Equation 3. Conceptual Simplification of Approach II Equation

$$\text{Benefit}_{i,s} = \sum_{\alpha=0}^{\substack{\text{Oldest exposure year} \\ \text{that can be diagnosed}}} \sum_{\substack{\beta=\text{first} \\ \text{diagnosis year}}}^{100} \text{Risk change at a given diagnosis age} \times \text{Value of risk change at a given diagnosis age}$$

Running this analysis for both sexes in a given county will give the total benefit to that county for the specified pollutant change in a given year.

6.4 Description of Approach II Outputs

In the end, Approach II will generate county-stratified, annual benefits of regulatory pollution controls. Figure 8 and Figure 9 present an example Approach II output, showing county-stratified benefits.[16]

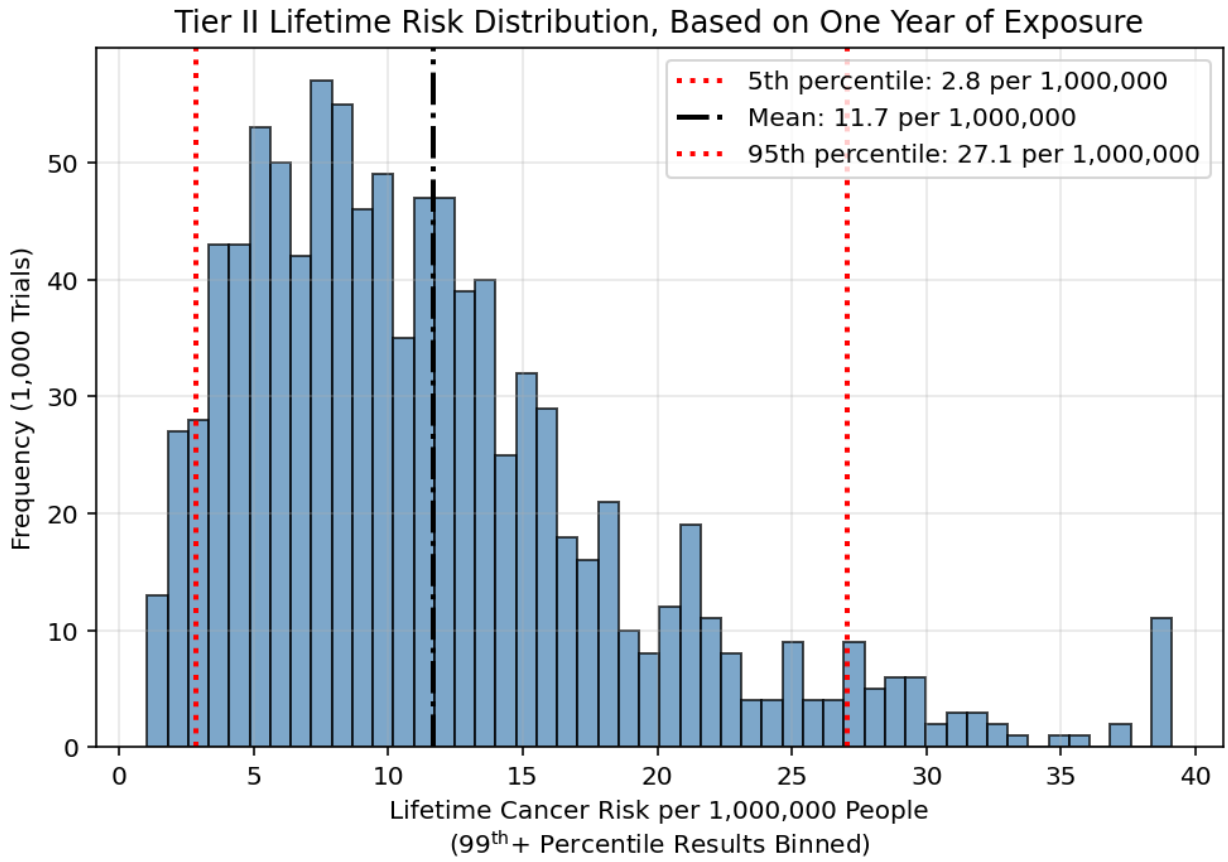
Figure 8. Example Approach II Output (Exact Numbers Illustrative)

County	Year						
	2026	2027	2028	2029	2030	2031	2032
All California	$ 2,732,908	$ 2,679,321	$ 2,626,785	$ 2,575,280	$ 2,524,784	$ 2,475,279	$ 2,426,744
Alameda County	$ 7,854	$ 7,700	$ 7,549	$ 7,401	$ 7,256	$ 7,114	$ 6,974
Alpine County	$ 62,144	$ 60,925	$ 59,731	$ 58,559	$ 57,411	$ 56,285	$ 55,182
Amador County	$ 97,574	$ 95,661	$ 93,785	$ 91,946	$ 90,143	$ 88,376	$ 86,643
Butte County	$ 2,000	$ 1,960	$ 1,922	$ 1,884	$ 1,847	$ 1,811	$ 1,776
Calaveras County	$ 78,176	$ 76,644	$ 75,141	$ 73,667	$ 72,223	$ 70,807	$ 69,418
Colusa County	$ 74,927	$ 73,458	$ 72,018	$ 70,605	$ 69,221	$ 67,864	$ 66,533
Contra Costa County	$ 95,715	$ 93,838	$ 91,998	$ 90,195	$ 88,426	$ 86,692	$ 84,992
Del Norte County	$ 20,419	$ 20,019	$ 19,626	$ 19,242	$ 18,864	$ 18,494	$ 18,132
El Dorado County	$ 13,813	$ 13,542	$ 13,276	$ 13,016	$ 12,761	$ 12,510	$ 12,265
Fresno County	$ 79,952	$ 78,384	$ 76,847	$ 75,340	$ 73,863	$ 72,415	$ 70,995
Glenn County	$ 84,016	$ 82,368	$ 80,753	$ 79,170	$ 77,617	$ 76,096	$ 74,603
Humboldt County	$ 16,624	$ 16,298	$ 15,979	$ 15,665	$ 15,358	$ 15,057	$ 14,762
Imperial County	$ 18,087	$ 17,732	$ 17,385	$ 17,044	$ 16,710	$ 16,382	$ 16,061
Inyo County	$ 7,095	$ 6,956	$ 6,819	$ 6,685	$ 6,554	$ 6,426	$ 6,300
Kern County	$ 38,917	$ 38,154	$ 37,406	$ 36,673	$ 35,954	$ 35,249	$ 34,558
Kings County	$ 47,281	$ 46,354	$ 45,445	$ 44,554	$ 43,681	$ 42,824	$ 41,984
Lake County	$ 98,856	$ 96,918	$ 95,018	$ 93,154	$ 91,328	$ 89,537	$ 87,782
Lassen County	$ 713	$ 699	$ 685	$ 672	$ 659	$ 646	$ 633
Los Angeles County	$ 89,205	$ 87,456	$ 85,741	$ 84,060	$ 82,411	$ 80,795	$ 79,211
Madera County	$ 55,043	$ 53,964	$ 52,906	$ 51,869	$ 50,852	$ 49,855	$ 48,877
Marin County	$ 67,428	$ 66,106	$ 64,810	$ 63,539	$ 62,293	$ 61,072	$ 59,874
Mariposa County	$ 35,238	$ 34,547	$ 33,870	$ 33,206	$ 32,555	$ 31,916	$ 31,290
Mendocino County	$ 3,504	$ 3,436	$ 3,368	$ 3,302	$ 3,237	$ 3,174	$ 3,112
Merced County	$ 29,306	$ 28,732	$ 28,168	$ 27,616	$ 27,074	$ 26,543	$ 26,023
Modoc County	$ 8,890	$ 8,715	$ 8,544	$ 8,377	$ 8,213	$ 8,052	$ 7,894
Mono County	$ 22,035	$ 21,603	$ 21,179	$ 20,764	$ 20,357	$ 19,958	$ 19,566

These valuation estimates differ from existing CARB risk assessment methods, such as isopleth analysis, in that they reflect population profiles of risk, such as the example shown in Figure 9 below. These estimates are drawn from data that reflect the range of biological responses to carcinogen exposure as well as the range of activity patterns in a given population. For more information on how we incorporate these factors, see Appendix B.

[16] The numbers in the figure have been randomly generated for 2026 and discounted for each subsequent year. Our final calculations may reflect more interannual variability.

Figure 9. Example Baseline Distribution of Lifetime Risk in a California County



6.5 Limitations and Uncertainties

The methods outlined in this Chapter are accompanied by several limitations and uncertainties. Below, we highlight some key factors to consider when conducting risk assessments using the Approach II methodology:

- **Dose-response uncertainty.** In our literature search, we reviewed several methods for addressing uncertainty in animal-derived human dose-response estimates. The most promising method was from Jang et al. (2023), which provides uncertainty analysis for re-estimating dose-response curves from animal toxicology data for many different carcinogenic chemicals. The authors comprehensively applied probabilistic methods to a database of cancer toxicity data to develop predictions for population cancer incidence and individual cancer risk across an array of chemicals. Despite its innovative approach to estimating low-dose carcinogenicity, the paper's applicability to this project was severely limited by the fact that it only incorporated oral exposures, not inhaled exposures, into its assessments. Due to the mechanistic differences in exposure between inhaled and orally ingested contaminants, we opted not to adopt this approach to uncertainty characterization. Future adaptation of this method to inhaled contaminants would represent a significant enhancement of CARB's ability to characterize uncertainty in dose-response for Approach II analyses.
- **20-year survival rates.** As discussed in Section 4.2.3, we utilize 20-year survival rates to allocate cancer risks between fatal and non-fatal cases. While most cancer deaths occur within this window, a relatively trivial portion of deaths occurs after the first decade for some cancers. Due to the higher unit value (VMR) for fatal risks, the approach may understate the benefits of TAC controls by a modest margin.

- **Exposure assessment.** Exposure distributions are subject to uncertainties described in documentation for the HAPEM8 model.
- **Lack of consideration of baseline cancer rates.** Because this approach relies on animal-based data, which uses a risk model independent of baseline cancer incidence, this latter information is not incorporated into the risk characterization. Risk models based on human data, which typically use proportional hazard models, would better capture differences in risk experienced by populations whose likelihood of cancer varies.

CHAPTER 7 | Approach III: Valuing Population-Level Risk Changes Using Life Tables

7.1 When to Use Approach III

Approach III provides CARB with the opportunity to assess TAC-related cancer risks based on data from studies in human populations, which often constitute the strongest evidence of carcinogenicity. It also allows CARB to incorporate local baseline data on cancer incidence rates directly into the risk calculations and stratify results by subpopulations, both of which can provide more detailed output on the distribution of cancer related benefits spatially and among different subpopulations. These added features do require a more data-intensive approach to developing TAC risk values for economic valuation, however. It is reserved for TACs whose cancer potency is characterized by a strong base of research that includes studies of cancer impacts in human populations (epidemiological studies). Ideally, the OEHHA potency values for these TACs are either based on or significantly influenced by epidemiological studies. Approach III is also best suited for analyses of TACs with regional or state-wide effects.

Approach III is similar to Approach II in that both generate estimates of population cancer risk; however, the way in which they model changes in risk differs. Approach II results are based on estimates of incremental cancer risk; this means that for populations experiencing the same change in exposure, the size of the risk reduction would be identical for both, even if one population has a higher baseline rate of the cancer being evaluated. Approach III employs risk estimates from human studies that are typically estimated using models where the impact is proportional to a baseline hazard rate (e.g., Cox proportional hazard model). This difference enables CARB to better estimate spatial variability in risk and variability in subpopulations by incorporating county- and group-specific baseline cancer incidence rates. Life table analyses also incorporate California specific mortality rates to model changes in the population at risk over time.

7.2 Life Table Background

A life table is a statistical tool used to model mortality and survivorship at various ages in a population over time (CDC 2025). Miller and Hurley (2002) proposed using standard life table calculation methods to develop a risk assessment framework for pollutants whose impacts affect mortality over long time periods. The use of a life table structure allows risk analysts to better model how risk reductions interact with, and affect, a dynamically changing population over time to better understand both the total impact and the time course of these effects. Life tables allow analysts to compare two population cohorts over time, one where pollution and pollution-attributable mortality rates are at baseline levels (the baseline group), and the other where pollution-attributable mortality rates decline over time following reductions in pollutant concentrations (the regulatory group). The risk model modifies the mortality rates in the regulatory scenario and flows those changes through the population cohort over time to develop the alternative population structure. The differences between these two cohorts represent the impacts of the regulation being modeled.

Life tables have been used to analyze impacts of many different inhaled contaminants over time, including the effects of $PM_{2.5}$ exposure on mortality (Apte et al. 2018), tobacco smoke exposure on leukemia (Fiebelkorn and Meredith 2018), and ethylene oxide and cancer (Valdez-Flores et al. 2011). In 2001 and 2004, the U.S. EPA SAB supported agency efforts to pursue a life table approach in a proof-of-concept case study estimating the effects of ambient benzene reductions on leukemia rates in Houston, TX. This recommendation was aimed at improving risk assessment methods for air toxics pollutant so that they could be incorporated into socioeconomic analyses similar to those conducted for criteria pollutants. The life table case study was implemented as part of the second prospective Benefit Cost Analysis of the Clean Air Act, 1990 – 2020 (U.S. EPA 2011). Details of the life table risk model approach can be found here.

7.3 Approach III Overview

This section describes the proposed workflow of an Approach III analysis with respect to the key "bridging" steps needed to allow risk assessments to inform a socioeconomic analysis and provides an overview of the structure and calculations associated with a life table model.

7.3.1Workflow

Approach III will follow the standard methodology applied in socioeconomic 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

As previously discussed, we assume that the regulatory scenarios will remain unchanged from the main CARB risk assessment, and that the emissions estimation and air quality modeling steps will remain the same as well. The main elements that require bridging from the main CARB risk assessment will be adjustments to the exposure and health effect modeling steps and the addition of the valuation step. While Approach III is the most computationally intensive approach with the largest data requirements, much of the data will be embedded in the life table. Data collection will focus on preparing appropriate exposure and dose-response inputs for the life table.

Figure 10 illustrates the workflow for an Approach III analysis, focusing on Steps 3 through 5 above. As with other approaches, we recommend the analysis focus on exposures to the general population (e.g., residents) and characterize the exposure to an average individual in that population along with a probabilistic characterization of variability and uncertainty in risk. Note that Figures 10 through 13 assume that the modeling is conducted based on risk estimates for cancer mortality, which would be most relevant for analyzing the priority TACs identified in this project.[17] We propose as the first step in the Approach III workflow that CARB use U.S. EPA's HAPEM8 model to characterize exposure variability, using initial air quality concentration inputs based on the modeled ambient TAC concentrations from the corresponding main CARB analysis.[18]

The next step in the workflow – the life table model run – is informed by updating the hazard identification and dose-response as necessary to comport with the life table approach. Consistent with the other approaches, the hazard identification from the main analysis should be reviewed to ensure that (a) the cancer effect being modeled represents a health effect that can be valued (i.e., TAC exposure should be associated with either a new cancer diagnosis or a cancer fatality) and (b) an identification of all cancers for which the underlying database is strong enough to support toxicity data estimation based on studies of human exposure. As for dose-response, the life table approach requires that cancer potency be expressed using a RR value from one or more epidemiology studies in order to adjust baseline cancer incidence rates.[19] We recommend beginning this process by consulting

[17] The model could be modified for use with relative risks for total cancer incidence, by replacing baseline mortality data with baseline cancer incidence data, so that the model tracks cases of cancer rather than deaths and subsequently revising the step apportioning fatal and non-fatal cases to estimate cancer deaths based on survival data.

[18] Note that time-weighted average exposures generated by the HAPEM8 tool may need to be adjusted prior to use in the life table model to ensure that the exposure concentration metric (e.g., daily 24-hour average exposure) matches the metric associated with the RR selected for use in the model.

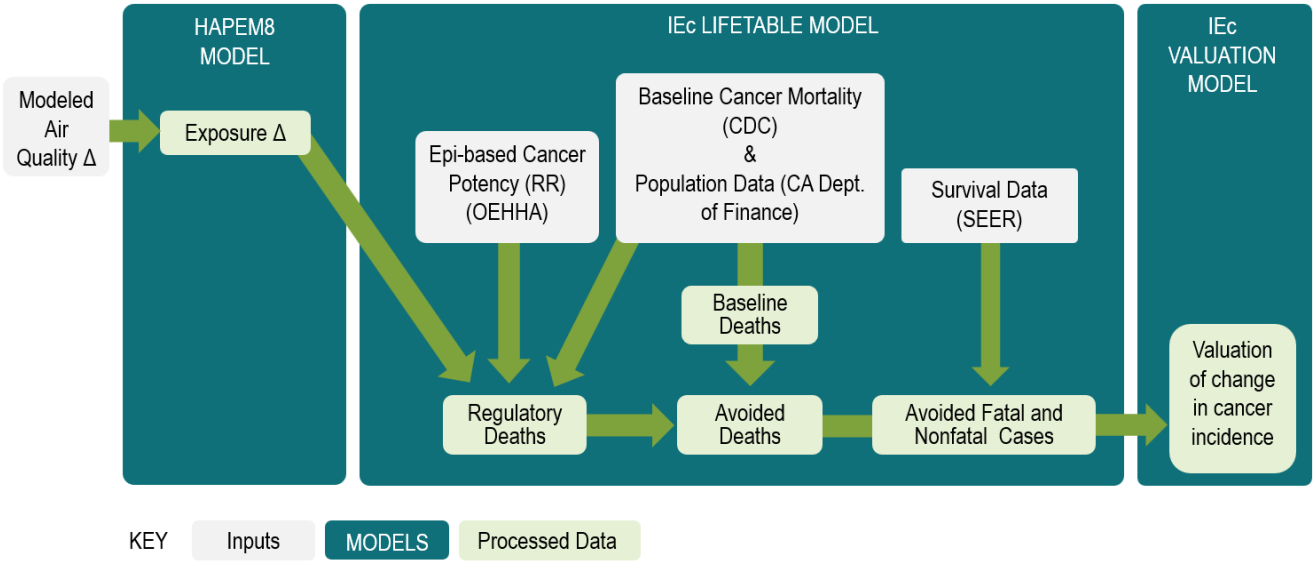
[19] Relative risk estimates the magnitude of an association between exposure and a health effect such as mortality or the incidence of cancer; it is a ratio that indicates the likelihood of developing the health effect in the exposed population relative to the unexposed (or less exposed) group. (Hennekens and Buring, 1987).

OEHHA's documentation of cancer potency factor (CPF) development for the TAC to understand how human data inform the process and which RR was used to develop the CPF/IUR. If necessary, consult with OEHHA staff to understand the role of epidemiology and whether a suitable RR from the review process exists for use in Approach III – considerations include study sample size, population studied, exposure characterization, control for confounding factors, relevance to non-occupational exposures and other factors affecting the quality and generalizability of the study. In cases where human studies exist but OEHHA bases its CPF/IUR on animal data (e.g., 1,3-butadiene), Approach II is recommended.

If multiple RRs exist for the same cancer and discussions with OEHHA suggest both to be of value, developing a single pooled RR estimate that integrates both studies using meta-analysis methods such as that described by Der Simonian and Laird (1986) can be a viable solution. Where separate RRs exist for different cancers (e.g., U.S. EPA's 2025 risk assessment for arsenic) CARB may either run the life table model separately for each cancer and then sum results (assuming simple additivity) or if a multi-cancer RR exists, run the model once using that value. (The latter may require post-processing based on evidence-informed assumptions about the relative fraction of each cancer type.)

Once the exposure analysis has been conducted and the RR identified, CARB may apply a life table model to generate spatially gridded differences in fatal and non-fatal cancers between the baseline and regulatory TAC scenarios. The added valuation step at the end of the process takes as inputs the revised population cancer risk results from the life table model and applies appropriate values for avoided statistical cases of fatal and non-fatal cancers, as discussed in Chapter 3.

Figure 10. Approach III Model Workflow



7.3.2 Life Table Structure

IEc recommends that for Approach III, CARB employ a life table risk model similar to that used in the second prospective Section 812 Benefit-Cost Analysis of the Clean Air Act (IEc 2009). [20] That model is directly relevant, as it was used to demonstrate benefit cost analysis of air toxics controls under Tier III of the Act by quantifying the change in incidence of cancer cases and cancer deaths over time. Figure 11 through Figure 13

[20] Appendix D of the *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* provides a detailed description of the model used in a case study of benzene exposures and leukemia cases in Houston.

provide an overview of the proposed structure of the tool. We propose to use a period life table, which is typically used to estimate life expectancy for people assuming current mortality patterns remain fixed over the rest of their lifetime (CDC 2024). Because we are using a period life table, we also assume fixed baseline cancer mortality rates as well.

The purpose of the risk model is to calculate the expected number of fatal and non-fatal cases of a specific cancer avoided as a result of implementing of a regulation reducing exposure to a TAC associated with that cancer. The approach consists of a traditional life table analysis that calculates the difference in cancer fatalities between two scenarios: a baseline, or "business as usual scenario" where the probability death in a given year, from all causes is unchanged from current conditions, and a regulatory scenario where the probability of death changes over time due to reduced TAC exposures resulting a lower probability of dying from cancer for certain age cohorts in a given time period.

Figure 11 illustrates the baseline scenario calculations; it represents population survival rates and mortality rates under the status quo (i.e. without implementation of the regulation under study). It includes information on population stratified by age, county, sex, and race/ethnicity that informs baseline mortality and survival rates. Beginning with the starting year specified by the analyst, the life table selects a California population cohort to follow over time. For each year of the analysis, the life table calculates population changes each year due to deaths and births. The mortality rates in these calculations represent the probability of death from all causes, as well as the probability of death from specific cancer of interest, for each age category in a given year conditional on surviving to that period. The analyst should identify either historic or projected mortality rates that are closest to the starting year of the analysis.[21] Ideally, rates should be stratified by age, sex, race, and ethnicity at the state or county level, obtained from CDC WONDER. The life table records the year's population values on survivorship, general mortality, and cancer-specific mortality and passes the data forward to update cohort tables for the next year before repeating the process, continuing until the end year specified by the user.

Figure 12 illustrates the regulatory scenario calculations. The overall calculation is similar to the baseline but modifies the hazard data describing the probability of death in a given year, based on the TAC exposure difference in that year and the mortality RR estimate derived from the selected epidemiological study. Nonfatal cases are then inferred from changes in mortality rates, based on the overall fatality rate and the 20-year mortality curve for the cancer type in question. The regulatory scenario components affected by this adjustment are indicated in a darker blue. For each county, the life table uses the HAPEM8 exposure estimate and the specified RR of the target cancer to determine changes in each age group's risk of dying from the target cancer type. The size of this effect depends on the size of the change in TAC exposure; the magnitude of the RR; and the size of the baseline mortality rate for the target cancer. This modified cancer death rate is then used to estimate cancer deaths in that year and to reduce the overall death rate from all causes, which gets applied to the population in each age group in the current year to determine the surviving population for the following year. The module then repeats this process for the entire analysis period. Once annual calculations are completed (Figure 13), the life table model subtracts the cancer deaths in each year of the regulatory scenario from the corresponding year in the baseline scenario to estimate reductions in avoided cancer deaths in each county in each study year. It also estimates total non-fatal cancer cases in each year by adjusting the number of cancer fatalities by the 20-year survival rate, obtained from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) database.

The life table can also integrate an expected lag period into our model, to reflect issues such as delays between exposures and diagnosis, consistent with the lags described in LaPenta et al., 2024.

[21] The current lifetable model uses all-cause and multiple-cause mortality estimates CDC WONDER's 2018-2024 data. Generally, recent historical mortality data are preferred to projected mortality data given uncertainties in future mortality.

Figure 11. Baseline Life Table Annual Calculation

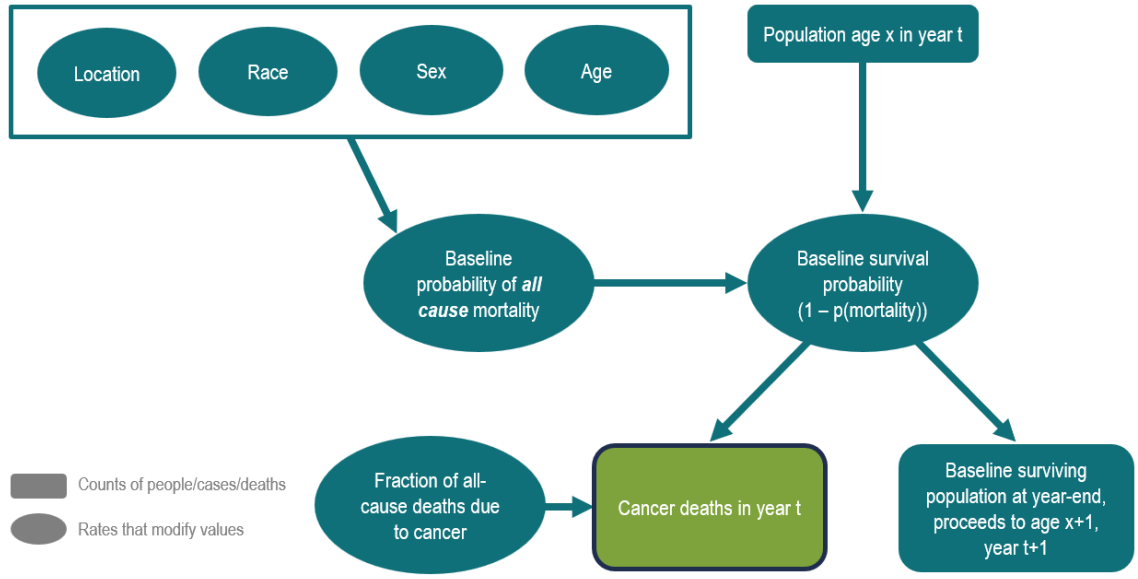


Figure 12. Regulatory Life Table Annual Calculation

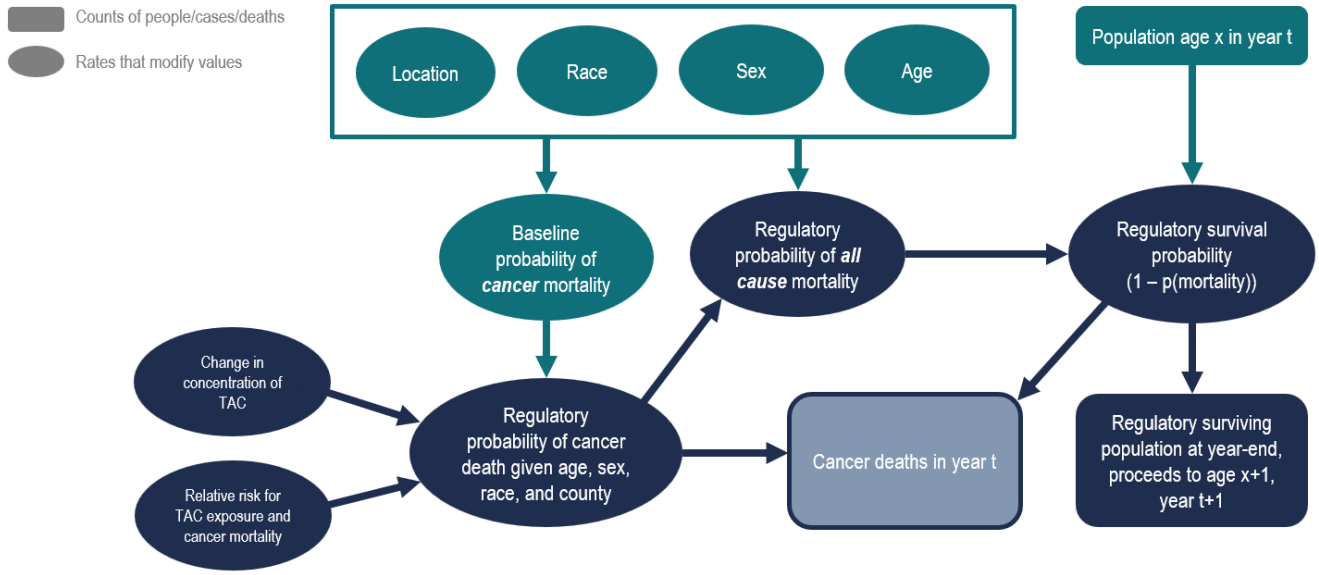
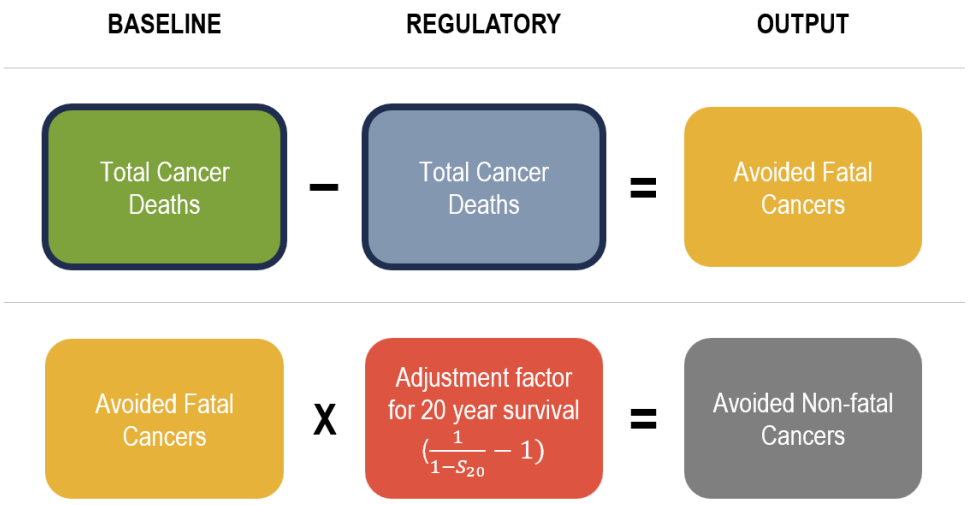


Figure 13. Calculation of Avoided Cases



7.3.3 Model Inputs

This section describes data sources for use with the life table model, each of which is described in greater detail in Section 4.2: Data Sources.

- Population. Historical population and future population projections stratified by county, age, gender, race, ethnicity, and year are available from the California Department of Finance (CDOF). These data describe the size and structure of the population in terms of age, sex, race, and location.
- Baseline cancer incidence rates. County rates by cancer type, stratified by age, gender, and race/ethnicity averaged over the years 2016-2021 (excluding 2020 due to COVID) are available from CCR. These data could potentially be used in the life table in place of mortality rates.
- Mortality rates. Statewide survival and mortality rates from 2018-2024 by sex and single year of age are available from the Centers for Disease Control's National Center for Health Statistics via CDC WONDER.
- Cancer survival rates. Cancer rates by cancer type, age, and year since diagnosis are available from the SEER 8 Survival Statistics database.

7.4 Exposure and Risk in Approach III

7.4.1 Exposure

As in Approach II, we recommend characterizing variability in exposure by running modeled ambient TAC concentration estimates through U.S. EPA's HAPEM8 to generate a distribution of exposure values. Additional information on HAPEM8 can be found in Section 6.2.1.2 and in Appendix A.1. When conducting the exposure modeling, ensure the concentration metric is consistent with the metric associated with the RR value being used for dose-response.

7.4.2 Calculating Lifetime Cumulative Exposure

To calculate lifetime cumulative exposure (ΔC in the above model), we use the following equation:

$$\Delta C_{t,j,l} = \sum_{y=t_0}^{t} \left(Exp_{y,regulatory} - Exp_{y,baseline}\right) \times w_y \times ASAF_{age\ in\ year\ y}$$

Where:

- t = current year of analysis
- t_0 = start year of analysis
- j = age group
- l = county or other geographic unit
- Exp_y =average exposure in a given year, in units of concentration
- w_y = weight assigned to exposure change in that year (1 if y is < t – latency, 0 otherwise)
- $ASAF$ = age specific adjustment factor based on age in year y; used to reflect susceptibility of certain age groups (10 for ages 0 and 1; 3 for ages 2 through 15; 1 for all other ages).

To accommodate dynamic year-to-year variation in TAC concentrations and for consistency with underlying epidemiological functions, the cancer mortality adjustment factor (CMAF) for each calendar year is calculated based on the change in cumulative exposure, where the exposure is calculated as a weighted sum of changes exposures in past year, with weights determined by the lag structure chosen by the user. The expected latency, or delay between changes in TAC exposure and the clinical diagnosis of cancers due to previous exposure, is modeled by applying these weights to previous years' exposure changes. We recommend a simple lag structure that applies weights of either one or zero, with zero weight applied to concentration changes in the x most recent years, where x is determined by the minimum latency in years of the target cancer type reported by Howard (2015) and has an integer value of one or greater. All years prior to the latency period are assigned a weight of one. This is an optional element but can be used to incorporate lags consistent with LaPenta et al., (2024).

Another modifier, the Age-Specific Adjustment Factor (ASAF), allows analysts to accommodate age-based differences in susceptibility to the TAC if recommended by OEHHA. We recommend using the same age-adjustment factors applied by CARB in the main risk assessment.

The life table performs the calculation above for a random sample selected from the exposure distribution output from the HAPEM8 model; this sample is carried through the risk characterization step.

7.4.3 Cancer Risk Calculations

The mortality risk or hazard estimation component of the model would apply user-specified parameters to calculate a CMAF that would scale the baseline cancer mortality rate in a given year based on cumulative impacts across all previous years in the study period. It would use an equation similar to the following, adapted from Roman et al. (2022):

Equation 4. Approach III Cancer Mortality Adjustment Factor Calculation

$$CMAF_{j,k,l,t} = e^{\Delta C_{j,l,t} \times \beta}$$

Where:

- j = age group

- $l =$ county
- $t =$ year of analysis
- $k =$ race (optional)
- $CMAF_{j,k,l,t} =$ Cancer Mortality Adjustment Factor for age group j, county l, race k, in year t;
- $\beta =$ the beta value derived from the RR from the selected epidemiologic study, which is assumed to be constant throughout the range of exposures; If ΔP is the concentration change reported in the study, $\beta = \ln(RR) / \Delta P$;
- $\Delta C_{j,l,t} =$ the change in cumulative TAC exposure concentration for age group j and county l in year t;

Once CMAFs have been calculated, cancer mortality rates in the regulatory scenario are calculated by modifying the baseline incidence rate of cancer, $P_{baseline}(cancer)$ as follows:

$$P_{regulatory}(cancer\ mortality)_{j,k,l,t} = P_{baseline}(cancer\ mortality)_{j,k,l,t} * CMAF_{j,k,l,t}$$

The life table incorporates uncertainty and variability into these calculations by sampling from distributions for the RR (from epidemiological studies) and individual TAC exposure (from HAPEM8) and using those values in the CMAF calculations above. Distribution of the beta value can be derived by converting the reported RR and its lower and upper confidence bounds to beta values, calculating the resulting average standard error for beta, and assuming errors around the central beta are normally distributed. The distribution for changes in TAC concentrations is generated by the HAPEM8 model.

7.4.4 Estimating TAC-Attributable Cancer Mortality

TAC-attributable cancer mortality is calculated as shown in Figure 13 as the difference in total surviving population each year between the regulatory and baseline periods. We estimate the number of non-fatal cases of cancer in a post processing step, where the fatal cases in each year are multiplied by a factor based on 20-year survival rates for the target cancer from the SEER database. We assume individuals surviving 21 or more years following cancer diagnosis represent non-fatal cases.

If the RR for a given TAC's cancer potency value is based on cancer incidence rather than cancer mortality, the life table can be adapted to substitute cancer incidence rates for mortality rates. This would be most appropriate for situations where the survival rates for the target cancer are high.

7.4.5 Approach to Valuation

Once fatal and non-fatal cancer risk changes are by age/sex and calendar year, non-fatal cancer risk changes are multiplied by the corresponding VNR, and fatal cancer risk changes are multiplied by the corresponding VMR, with each value corresponding to the age/sex and calendar year of interest. The valuation of both fatal and non-fatal cancer risk changes are then summed across years of analysis. A description of how VNR and VMR are calculated can be found in Chapter 3. Description of Approach III Outputs

The lifetable model output will consist of summary data containing the numbers of fatal and non-fatal cancer cases avoided, which represents the health impact of the regulatory scenario. Results may be geocoded to facilitate mapping and can be reported by age, county, race and/or year. The life table provides an option to run the model using population specific to race/ethnicity subgroups, which will produce results stratified by population subgroup and county; these results can be exported for subsequent use in environmental justice analyses. In addition, the life table model output will include summary tables of the monetized benefits (in 2023 dollars) associated with the avoided cancer mortality and morbidity.

7.5 Limitations and Uncertainties

The methods outlined in this chapter are accompanied by several limitations and uncertainties. Below, we highlight some key factors to consider when conducting risk assessments using the Approach III methodology:

- **Dose-response uncertainty.** In the Approach III methodology, we characterize uncertainty in the dose-response function using the reported uncertainty bounds on the RR estimate used in the life table, as reported in the underlying epidemiology study. This methodology captures some of the uncertainty in the risk estimate due to sample size and other study design factors, but it is only a partial representation of uncertainty in this input. Other uncertainties associated with epidemiological studies in general (e.g. potential confounding), and occupational studies in particular (e.g., the healthy worker effect[22]) may also apply. Unlike the Approach II methodology, it does not include a probabilistic assessment of interindividual variability in sensitivity.
- **20-year survival rates.** As discussed in Section 4.2.3, we utilize 20-year survival rates to allocate cancer risks between fatal and non-fatal cases. While most cancer deaths occur within this window, a relatively trivial portion of deaths occurs after the first decade for some cancers. Due to the higher unit value (VMR) for fatal risks, the approach may understate the benefits of TAC controls by a modest margin.
- **Use of fixed mortality and cancer rates.** The life table approach we propose assumes constant mortality rates over time in the baseline, based on data from 2018-2024 for all-cause mortality in California. Survival data for cancer are similarly fixed. Analyses of future years do not account for potential changes in these rates due to medical advancements or other factors.
- **Exposure assessment.** Exposure distributions are subject to uncertainties described in documentation for the HAPEM8 model.
- **Births.** Births are assumed to remain constant throughout the study period, which may introduce some minor uncertainty into the results.
- **Migration.** The life table model currently assumes no net migration into or out of California over the period of analysis.

[22] See for example Chowdhury, R., Shah, D. and Payal, A.R., 2017. Healthy worker effect phenomenon: revisited with emphasis on statistical methods–a review. Indian journal of occupational and environmental medicine, 21(1), pp.2-8.

CHAPTER 8 | Case Studies

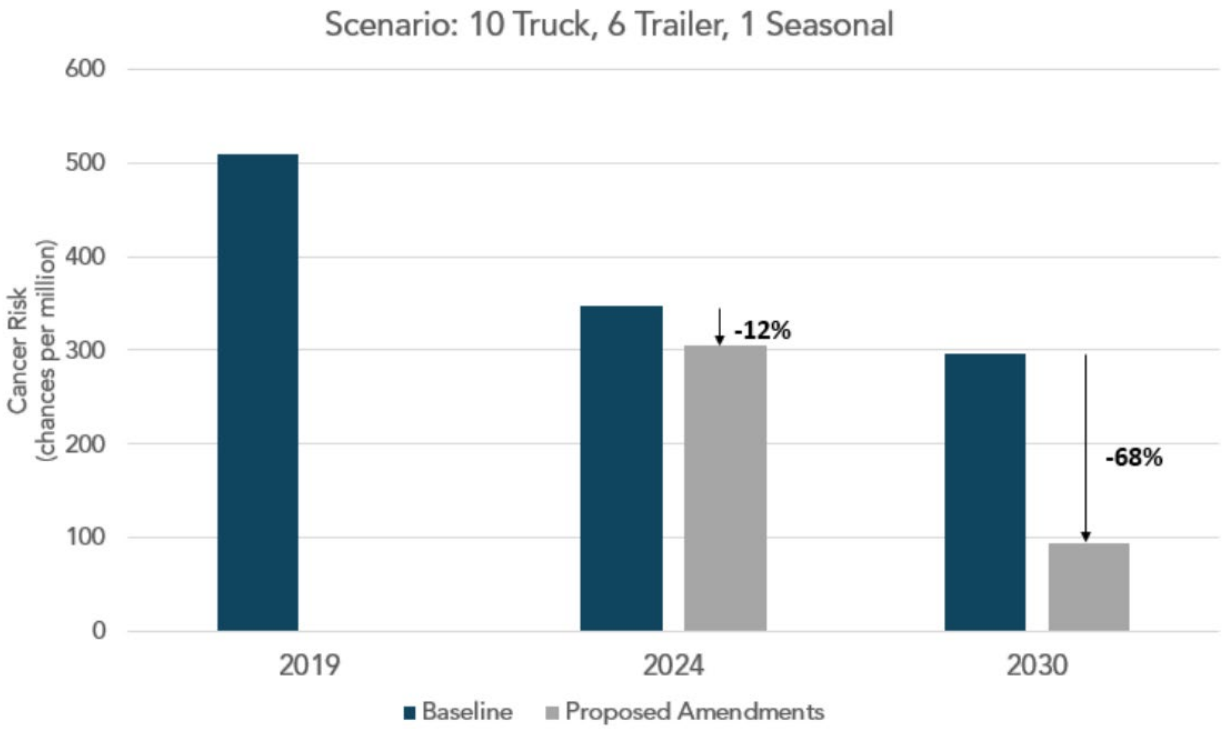
This chapter presents overviews of three proof-of-concept case studies illustrating how each of the three approaches described in this document could be applied in practice to complement existing CARB risk assessments with monetized estimates of avoided cancer health impacts. Each of the case studies is designed to yield results that could be incorporated into socioeconomic analyses, however because the nature of the information generated by each approach differs, the specific type(s) of socioeconomic analysis supported will also differ.

8.1 Approach I

Our proof-of-concept case study to illustrate Approach I focuses on cancer risk reductions associated with reductions in emissions of diesel particulate matter (DPM). We apply the value of one-in-one million cancer risk changes to CARB's calculated estimates of risk from Transport Refrigeration Units (TRUs). We summarize below the details of our scenario, risk assessment and valuation inputs, and the bridging adjustments made for consistency with regulatory analysis practices.

- **Scenario.** This case study develops valuation estimates for cancer risk reductions associated with a historical CARB regulation that reduced emissions of DPM from mobile refrigeration trucks outside grocery stores. The baseline scenario assumes business-as-usual (BAU) emission reduction from TRUs, while the regulatory scenario assumed a reduction of 12 percent from baseline emissions by 2024 and a reduction of 68 percent from baseline emissions in 2030. Figure 14, adapted from Figure II.F.4 in Appendix I of CARB's final TRU regulation, displays this emissions reduction trajectory (CARB 2021).

Figure 14. Potential Individual Resident Cancer Risk Reduction from TRU Regulation



The TRU regulation is assumed to be implemented by 2024 and begin generating benefits in that year. The regulation is expected to reduce concentrations of DPM by 12 percent by year 2024 compared to the baseline, decreasing to a 68 percent reduction by 2030.

- **Geographic scope and population affected.** Impacts of the emissions of TAC are expected to be localized rather than widespread. As a result, the impacts evaluated in this risk assessment focus on exposures in proximity to emission sources. The exposed population may be large in aggregate given the number of potential sources throughout California, but information on the size of the exposed populations surrounding each source is uncertain, as is the total size of the affected population.

- **Type of cancer risk.** Given the uncertainty in the size of the exposed population, the risk characterization focuses on changes in incremental lifetime cancer risk to individuals either living nearby or working within a given distance from TRUs instead of population-level estimates of avoided cancer cases. Exposure to DPM has been associated with lung and bronchus cancer in humans, and OEHHA has published a unit risk estimate of 3.0 E-4 $(\mu g/m^3)^{-1}$ for this TAC.

8.1.1 Methods

As described in the earlier chapters of this document, the basic risk assessment steps (hazard identification, exposure assessment, dose-response analysis, and risk characterization) for these case studies remained the same for this case study. We also added the valuation step that assigned a monetary estimate to the quantified health benefits associated with the regulation.

Approach I relies to the greatest extent on the risk estimates generated by CARB in its typical risk assessments. Thus, we applied no adjustments to the CARB exposure and potency assumptions and inputs. We list below any bridging modifications to CARB's risk assessment for the purpose of implementing Approach I.

- **Hazard Identification.** DPM is a well-studied TAC emitted by diesel fueled engines. A substantial base of epidemiological studies of railroad workers and long-haul truck drivers identifies lung cancer as an outcome of chronic inhalation exposure to DPM. In 1998, OEHHA conducted a health risk assessment of DPM and found sufficient evidence to establish a unit risk of 3.0 E-4 $(\mu g/m^3)^{-1}$ for lung cancer associated with DPM inhalation exposure. Additional details regarding the evidence base supporting this determination and unit risk can be found in Appendix B of OEHHA, 2009. Because valuation estimates exist for the associated health effects (i.e., mortality and non-fatal lung cancer), we proceeded with the case study with no adjustments applied to this hazard identification step.

- **Exposure Assessment.** The approach for exposure assessment was not modified; it is consistent with the approach taken by CARB in past risk assessments for TACs with localized health impacts. The impacts evaluated in this risk assessment focused on exposures in proximity to emission sources. The exposed population may be large in aggregate given the number of potential sources throughout the state, but information on the size of the exposed populations surrounding each source was uncertain. Air quality modeling was not performed for each specific emission source, but estimates of the concentrations expected at a given distance from a generic source were generated for different source sizes. The resulting modeling allowed CARB to estimate a spatial gradient of concentration near a generic source under baseline and regulatory conditions.

- **Dose-Response Assessment.** The risk assessment applied the unit risk value noted above for DPM inhalation exposure and lung cancer incidence.

- **Risk Characterization.** No changes were made to risk characterization. Results remained expressed as individual risk reductions for purposes of valuation.

- **Valuation.** The valuation step was added and conducted using the methods described in Chapter 5 and Appendices C and D.

8.1.2 Results

In Table 7, we present the monetized benefits of a one-in-one-million reduction in lifetime lung cancer risk for individuals in two illustrative age groups (0 to 17 year olds, 50 to 75 year olds) in Los Angeles County. Each value represents the value of an exposure reduction in the specified year that corresponds to a risk reduction of one-in-one-million to a person within the specified age group. These values are calculated using the equations and data described in Chapter 6. As described in that chapter the values differ by age due to differences in survival rates between age groups and the proportion of lifetime risk already experienced by different age groups at the time the exposure occurs. The two bolded values ($9.43, $2.69) correspond with exposure changes in 2025; discounting results in a significantly lower present value of benefits due to the significant lag between exposure, cancer onset, and—in some cases—premature death. We utilize these bolded values in the tables that follow.

Table 7. Monetized Per-person Benefits of a One-in-One-Million Risk Reduction for LA County (2023$)

Year of Exposure	0–17-year-olds		50–75-year-olds	
	Undiscounted	2% discount rate*	Undiscounted	2% discount rate*
2025	**$9.43**	**$2.69**	$8.42	$5.44
2026	$9.47	$2.65	$8.46	$5.36
2027	$9.52	$2.61	$8.50	$5.28
2028	$9.56	$2.57	$8.54	$5.20
2029	$9.61	$2.54	$8.59	$5.13
2030	$9.66	$2.50	$8.63	$5.05
2031	$9.70	$2.46	$8.67	$4.98
2032	$9.75	$2.42	$8.72	$4.90
2033	$9.80	$2.39	$8.76	$4.83
2034	$9.84	$2.35	$8.80	$4.76
2035	$9.89	$2.32	$8.85	$4.69
2036	$9.94	$2.28	$8.89	$4.62
2037	$9.99	$2.25	$8.93	$4.55
2038	$10.04	$2.22	$8.98	$4.49

Tables 8a and 8b show the results of applying the unit values from Table 7 to the total individual risk reductions that result from each scenario and downwind distance from grocery store fence lines for an example age group. The tables present per-person values for youth (ages 0 to 17) exposure to DPM as both discounted (Table 8a) and undiscounted (Table 8b) individual benefits.

Table 8a. Resident Age 0-17 Total Lifetime Cancer Risk Reduction (chances per million) Per Person and Associated Discounted Monetized Benefits Per Person in Year 2025(2023$)

Scenario	Total Hours of TRU Engine Operation		Value	Downwind Distance (m) from Grocery Store Fence Line															
	Per Week	Per Year		0	10	25	50	75	100	150	200	250	300	350	400	500	600	700	800
1 Daily Truck 1 Daily Trailer 1 Seasonal Trailer	202	3,940	Individual risk reduction	150	110	74	45	31	23	14	10	7	6	5	4	3	2	2	2
			Value of a one-in-one million risk reduction	$2.69															
			Monetized benefits (discounted, 2% DR)	$404	$296	$199	$122	$83	$62	$38	$27	$19	$16	$13	$11	$8	$5	$5	$5
7 Daily Trucks 2 Daily Trailers 1 Seasonal Trailer	274	7,717	Individual risk reduction	270	200	130	81	56	41	26	18	14	11	9	7	6	5	4	3
			Value of a one-in-one million risk reduction	$2.69															
			Monetized benefits (discounted, 2% DR)	$726	$538	$350	$218	$151	$110	$70	$48	$38	$30	$24	$19	$16	$13	$11	$8
10 Daily Trucks 6 Daily Trailers 1 Seasonal Trailer	402	14,334	Individual risk reduction	510	370	250	150	100	77	48	33	25	20	16	14	10	8	7	6
			Value of a one-in-one million risk reduction	$2.69															
			Monetized benefits (discounted, 2% DR)	$1,372	$995	$673	$404	$269	$207	$129	$89	$67	$54	$43	$38	$37	$22	$19	$16

Table 8b. Resident Age 0-17 Total Lifetime Cancer Risk Reduction (chances per million) Per Person and Associated Undiscounted Monetized Benefits Per Person in Year 2025(2023$)

Scenario	Total Hours of TRU Engine Operation		Value	Downwind Distance (m) from Grocery Store Fence Line															
	Per Week	Per Year		0	10	25	50	75	100	150	200	250	300	350	400	500	600	700	800
1 Daily Truck 1 Daily Trailer 1 Seasonal Trailer	202	3,940	Individual risk reduction	150	110	74	45	31	23	14	10	7	6	5	4	3	2	2	2
			Value of a one-in-one million risk reduction	$9.43															
			Monetized benefits (undiscounted)	$1400	$1000	$700	$420	$290	$220	$130	$94	$66	$57	$47	$38	$28	$19	$19	$19
7 Daily Trucks 2 Daily Trailers 1 Seasonal Trailer	274	7,717	Individual risk reduction	270	200	130	81	56	41	26	18	14	11	9	7	6	5	4	3
			Value of a one-in-one million risk reduction	$9.43															
			Monetized benefits (undiscounted)	$2500	$1900	$1200	$760	$530	$390	$250	$170	$130	$100	$85	$66	$57	$47	$38	$28
10 Daily Trucks 6 Daily Trailers 1 Seasonal Trailer	402	14,334	Individual risk reduction	510	370	250	150	100	77	48	33	25	20	16	14	10	8	7	6
			Value of a one-in-one million risk reduction	$9.43															
			Monetized benefits (undiscounted)	$4800	$3500	$2400	$1400	$940	$730	$450	$310	$240	$190	$150	$130	$94	$75	$66	$57

8.1.3 Limitations

Limitations of these Approach I results are consistent with those described in Chapter 5. No additional limitations were introduced in conducting the proof-of-concept case study.

8.2 Approach II

Our case study for Approach II focused on formaldehyde. Formaldehyde was identified as a toxic air contaminant by OEHHA in 1998, and prolonged exposure via inhalation has been shown to cause cancer in animal toxicological studies and human occupational epidemiology studies, though OEHHA's IUR and CPF values are based on the results of animal studies. We summarize below the details of our scenario, risk assessment and valuation inputs, and the bridging adjustments made for consistency with regulatory analysis practices:

- **Scenario.** This case study develops valuation estimates for cancer risk reductions associated with a hypothetical regulation that reduces emissions of formaldehyde from sources throughout the state such as industrial emissions and fuel combustion. The baseline scenario assumes ambient formaldehyde concentrations remain at 2025 levels into the future, while the regulatory scenario assumes a statewide reduction of ambient concentrations that grows linearly from 4 percent in 2025 to 40 percent in 2035, as shown in Figure 15, with benefits calculated for changes in exposures through 2050.

- **Geographic scope and population affected.** Approach II focuses on two different geographic scopes: county-level analysis for all of California, and tract-level analysis for Alameda County, CA. Within these geographies, we assessed cancer risk to all residents. We used California Department of Finance population projections to characterize population in future years.

- **Type of cancer risk.** Since Approach II chemicals are derived from animal toxicological studies, and identifying concordance between animal tumor sites and human tumor sites remains a developing area of study, Approach II analyses focus on generic "All Sites" cancer risk (see Section 6.2.2.1 Air Quality Surfaces).

8.2.1 Methods

The case study applied the basic steps of risk assessment (hazard identification, exposure assessment, dose-response analysis, and risk characterization), but changes to the assumptions or inputs within those steps were made as "bridges" to a socioeconomic-style benefits analysis. We also added the valuation step that assigned a monetary estimate to the quantified health outcomes associated with the regulation.

We list below any bridging modifications to CARB's risk assessment for the purpose of implementing Approach II.

- **Hazard Identification.** The unit risk for exposures to formaldehyde is based on extrapolation from animal toxicological studies. Those studies showed an association between inhalation exposures to formaldehyde and the development of squamous cell carcinomas in the nasal passages of rats and mice (OEHHA, 2009). Because of the uncertainties associated with mapping particular cancers observed in animal studies to humans and the limited human studies available, we followed the Approach II recommendation of associating exposures with a non-specific cancer. This facilitated valuation of these impacts using VNR values for generic cancer plus application of VSL values based on generalized survival data for all cancer types.
- **Exposure assessment.** The bridging approach for exposure sought to: 1) develop a central estimate of exposure to formaldehyde of the average individual in the population affected; and 2) develop a

distribution of values around that central estimate that reflects uncertainty and variability due to time-activity patterns (including daily commuting patterns) and other factors that may vary from person to person. As noted in Chapter 6, we recommend that CARB retain the same baseline and regulatory scenarios used in its standard TAC risk assessment and that it use the results of air quality modeling performed, so long as that modeling does not represent atypical conditions. Though this case study was not based on a historical CARB risk assessment, we did identify air toxics modeling results available that represented typical conditions. We started with modeled estimates of ambient statewide formaldehyde concentrations throughout California at the census tract-scale, obtained from the 2020 version of U.S. EPA's ATS model. As recommended in Section 7.4, to facilitate the bridging step, we then fed these data into U.S. EPA's HAPEM8 model, version 8.0, which combines demographic information, time-activity patterns, and modeling on indoor/outdoor air quality to translate ambient chemical concentrations into lived exposure values. HAPEM8 results are stratified by age groups, allowing for fine-resolution analysis. Additional details regarding this process can be found in Appendix E. Since HAPEM8 processes pollution data linearly (i.e. a 50 percent reduction in input concentrations equals a 50 percent reduction in output concentrations), we applied percent changes in formaldehyde emissions directly to our HAPEM8 results. We then conducted Monte Carlo sampling to combine individual exposures and susceptibilities as part of exposure assessment and risk characterization, as described in Section 6.2.2.

- **Dose response assessment.** To assess dose-response for formaldehyde, we began with OEHHA's unit risk of 6.0 E-6 $(\mu g/m^3)^{-1}$, which is based on a 95 percent upper confidence limit on the central estimate dose-response. We then applied the approach described in Section 6.3.2 of this document to build distributions of interindividual variability around this unit risk value and account for uncertainty in the shape of these distributions.

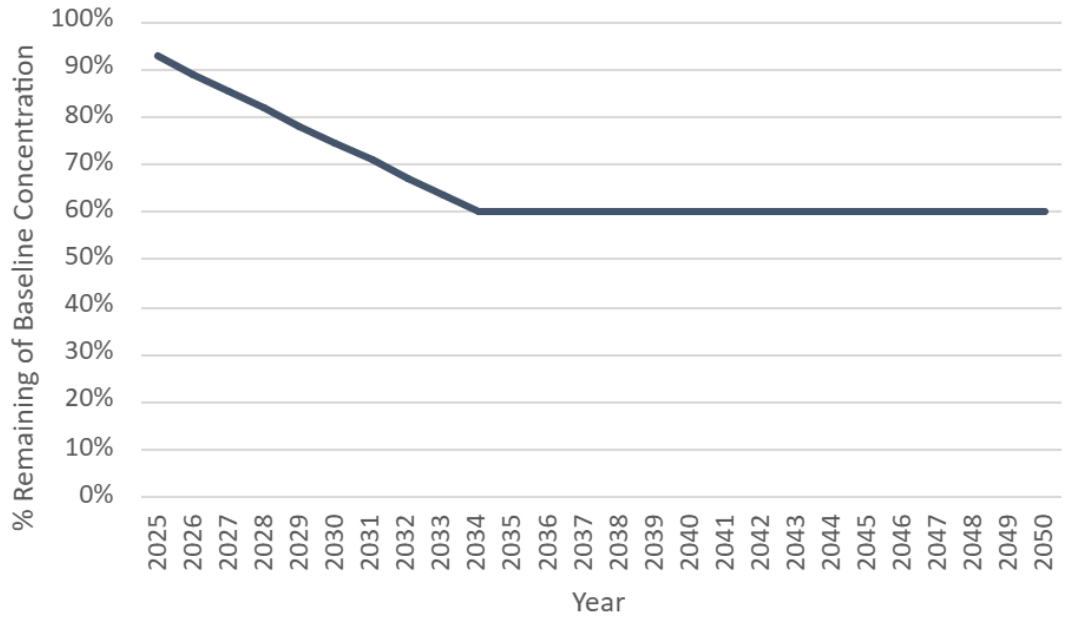
- **Risk characterization.** In Approach II, we used OEHHA's unit risk values to calculate a change in lifetime cancer risk resulting from a one-year change in exposure. We incorporate uncertainty in interindividual susceptibility to cancer and variability in both susceptibility of cancer and exposure to ambient carcinogens. To calculate population risks, we multiplied individual risk changes by population estimates from the California Department of Finance. Carcinogenesis, however, can exhibit lags between exposure and initial diagnosis as discussed in Section 4.2.2. Therefore, as described in that section, we set minimum lag values based on data from Howard (2015) and combine this with California incidence data profiles from the California Cancer Registry (CCR) to further model the lag and distribute these changes in avoided lifetime risk over the course of an exposed person's remaining life (See Section 6.3.1). This allowed us to distribute the change in avoided lifetime risk for exposure in a given year as changes in likelihood of diagnosis in each subsequent year, which we then treated as avoided statistical non-fatal cancer cases. From each year of diagnosis, we then applied cancer survival data to similarly distribute avoided statistical deaths over the subsequent 20-year mortality window using mortality data from CDC WONDER. This resulted in a distribution of timing of fatal and non-fatal cancer cases for a given sex and age group in a given year.

- **Valuation.** We used VNR and VMR values to value avoided fatal and non-fatal cancer cases, respectively. We valued avoided fatal cases in the year in which the death occurred, and we valued avoided non-fatal cases in the year in which the cancer diagnosis occurred.

8.2.1.1Hypothetical Regulatory Scenario

Our policy scenario assumes a uniform decrease of 40 percent formaldehyde concentrations between 2025 and 2035, with benefits calculated to 2050.

Figure 15. Scenario Reduction in Formaldehyde Concentration



While this emissions reduction is likely to be more homogeneous than any true assessment of emission changes, a uniform emissions reduction provides a useful what-if scenario for understanding the functioning of Approach II and its implications on valuation of cancer risk.

8.2.2 Results

8.2.2.1Risk Characterization

Figures 16 and 17 depict the spatial distribution of baseline formaldehyde concentrations and total cancer cases avoided over the study period. Figure 16 provides tract-level results for Alameda County; Figure 17 provides county-level results across California. In both figures, we provide additional context on the population affected by mapping the cumulative burden percentile—an index measure of pollution exposure and population vulnerability—from CalEnviroScreen (CES) 4.0. Higher percentiles (depicted in dark green) represent environmentally disadvantaged communities.

Figure 16. Map of Cancer Cases Averted through Hypothetical Formaldehyde Regulation, Alameda County 2025-2050

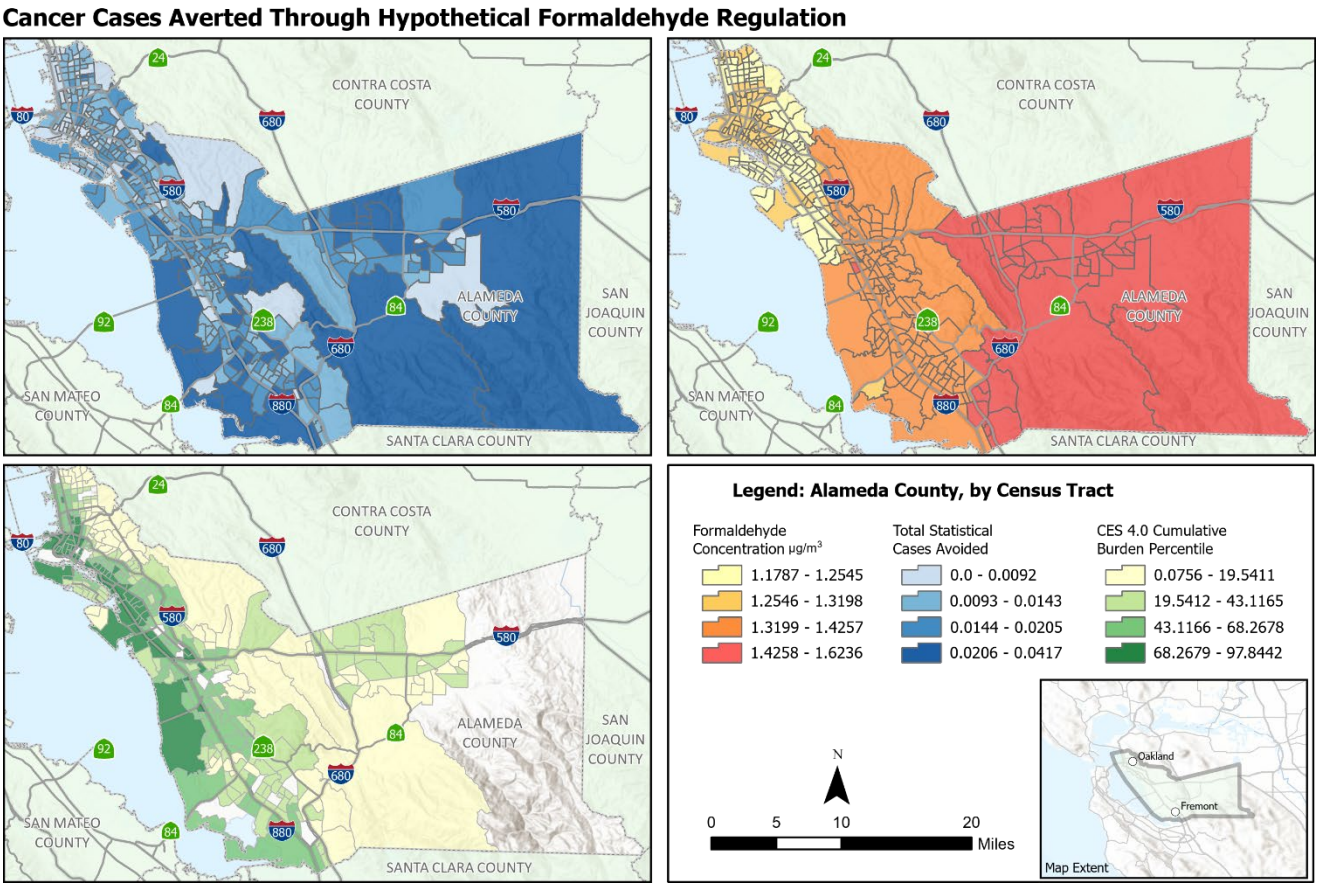


Figure 17. Map of Cancer Cases Averted through Hypothetical Formaldehyde Regulation, Statewide

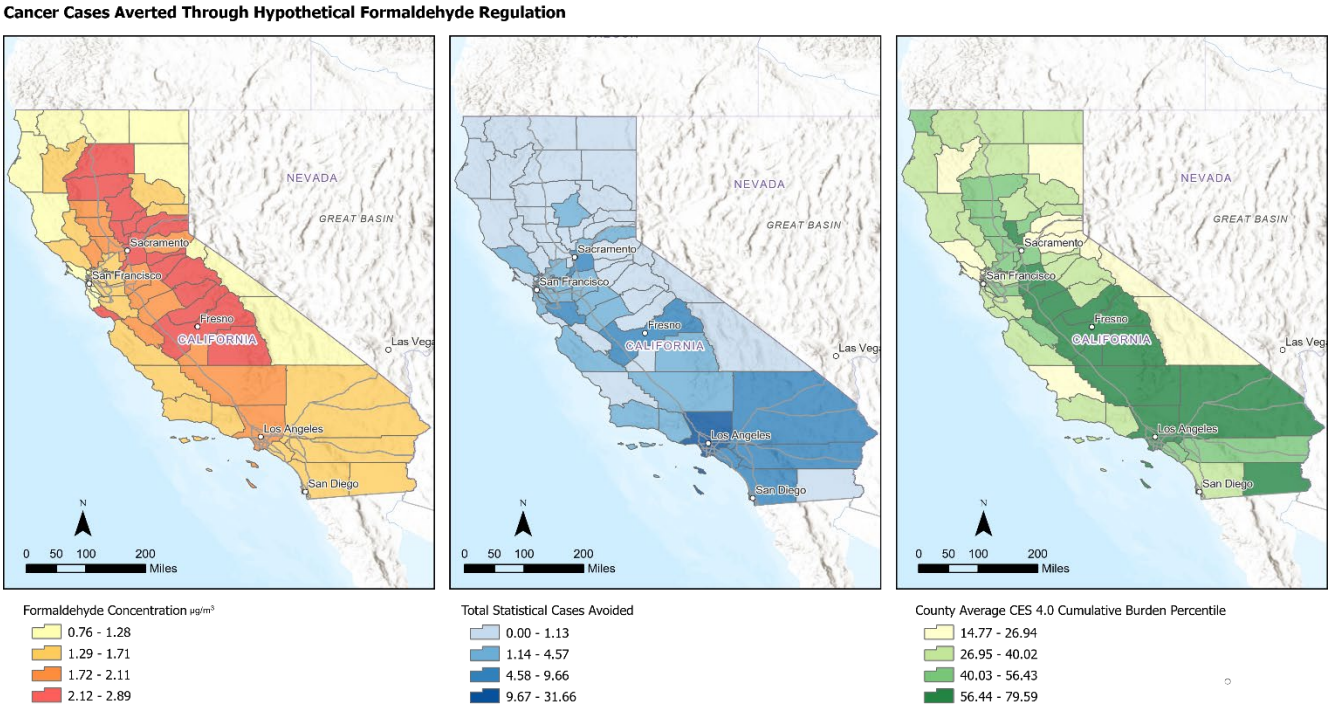


Table 9 displays tabular results detailing non-fatal and fatal statistical cases avoided by county in our hypothetical formaldehyde regulatory scenario.

Table 9. Non-fatal and Fatal Statistical Cases Avoided, Hypothetical Formaldehyde Regulation

County	Non-fatal Statistical Cases Avoided			Fatal Statistical Cases Avoided		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Alameda	0.88	2.4	3.3	0.81	2.2	3.1
Alpine	0.0004	0.0009	0.0014	0.0004	0.0009	0.0014
Amador	0.038	0.088	0.14	0.037	0.085	0.14
Butte	0.28	0.69	1.1	0.26	0.65	0.99
Calaveras	0.034	0.082	0.13	0.033	0.080	0.13
Colusa	0.018	0.045	0.070	0.017	0.042	0.065
Contra Costa	0.77	1.8	2.9	0.71	1.65	2.7
Del Norte	0.011	0.025	0.041	0.010	0.023	0.038
El Dorado	0.15	0.35	0.59	0.15	0.33	0.55
Fresno	1.2	2.8	4.4	1.1	2.6	4.0
Glenn	0.027	0.061	0.10	0.025	0.057	0.096

County	Non-fatal Statistical Cases Avoided			Fatal Statistical Cases Avoided		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Humboldt	0.048	0.12	0.18	0.045	0.11	0.17
Imperial	0.13	0.32	0.50	0.12	0.29	0.46
Inyo	0.0050	0.013	0.019	0.0047	0.012	0.018
Kern	0.86	2.1	3.3	0.76	1.8	2.9
Kings	0.15	0.35	0.55	0.13	0.31	0.49
Lake	0.048	0.11	0.18	0.046	0.11	0.17
Lassen	0.015	0.036	0.057	0.014	0.034	0.053
Los Angeles	7.1	17	27	6.5	15	25
Madera	0.19	0.43	0.71	0.17	0.40	0.65
Marin	0.10	0.25	0.39	0.098	0.24	0.37
Mariposa	0.013	0.031	0.049	0.013	0.030	0.048
Mendocino	0.047	0.11	0.18	0.045	0.11	0.17
Merced	0.33	0.80	1.3	0.29	0.72	1.1
Modoc	0.0022	0.0055	0.0083	0.0021	0.0053	0.0080
Mono	0.0045	0.011	0.017	0.0042	0.011	0.016
Monterey	0.34	0.81	1.3	0.31	0.74	1.2
Napa	0.094	0.23	0.36	0.090	0.22	0.34
Nevada	0.073	0.18	0.28	0.071	0.18	0.27
Orange	2.1	4.8	7.9	1.9	4.4	7.3
Placer	0.49	1.2	1.9	0.47	1.1	1.8
Plumas	0.0093	0.023	0.035	0.0089	0.022	0.034
Riverside	1.8	4.4	7.0	1.7	4.1	6.4
Sacramento	1.8	4.4	6.9	1.6	4.0	6.3
San Benito	0.055	0.13	0.21	0.050	0.12	0.19
San Bernardino	1.7	4.0	6.3	1.48	3.6	5.6
San Diego	2.1	5.0	8.1	2.0	4.6	7.5
San Francisco	0.33	0.75	1.2	0.30	0.69	1.2

County	Non-fatal Statistical Cases Avoided			Fatal Statistical Cases Avoided		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
San Joaquin	0.81	2.0	3.1	0.73	1.8	2.8
San Luis Obispo	0.18	0.43	0.68	0.17	0.41	0.65
San Mateo	0.31	0.70	1.1	0.29	0.65	1.1
Santa Barbara	0.30	0.73	1.2	0.28	0.68	1.1
Santa Clara	1.2	3.0	4.7	1.1	2.7	4.3
Santa Cruz	0.25	0.59	0.97	0.23	0.54	0.89
Shasta	0.18	0.41	0.68	0.17	0.39	0.64
Sierra	0.0014	0.0032	0.0051	0.0012	0.0030	0.0047
Siskiyou	0.018	0.084	0.069	0.018	0.080	0.067
Solano	0.33	0.79	1.3	0.31	0.73	1.2
Sonoma	0.28	0.64	1.1	0.27	0.61	1.0
Stanislaus	0.53	1.2	2.0	0.48	1.1	1.8
Sutter	0.12	0.30	0.47	0.11	0.28	0.43
Tehama	0.071	0.17	0.27	0.066	0.16	0.25
Trinity	0.0089	0.021	0.033	0.0086	0.020	0.033
Tulare	0.55	1.4	2.1	0.50	1.3	1.9
Tuolumne	0.046	0.11	0.17	0.045	0.11	0.17
Ventura	0.50	1.2	1.9	0.47	1.1	1.8
Yolo	0.22	0.50	0.83	0.20	0.45	0.76
Yuba	0.11	0.25	0.41	0.098	0.23	0.37
California Total	**29**	**70**	**110**	**27**	**64**	**102**

Valuation

Table 10 shows the mean, 5th, and 95th percentile total valuation for statistical cases avoided due to hypothetical formaldehyde emissions reductions. Cases are valued using Monte Carlo sampling from EPA's VSL distribution and the full "All Sites" valuation distribution developed in Appendix D and combined to calculate the total valuation estimate. We applied the same VSL and non-fatal WTP distribution across all races, counties, sexes, and age groups.

Table 10. Example Valuation of Statistical Cases Avoided (2023$) for Formaldehyde Scenario 2025-2050

County	Total Statistical Cases Avoided			Total Valuation (2023$)		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Alameda	1.7	4.6	6.4	$1,00,000	$12,000,000	$25,000,000
Alpine	0.0007	0.0019	0.0027	$400	$5,000	$11,000
Amador	0.074	0.17	0.28	$44,000	$480,000	$1,100,000
Butte	0.54	1.3	2.0	$310,000	$3,500,000	$7,700,000
Calaveras	0.067	0.16	0.26	$40,000	$450,000	$980,000
Colusa	0.035	0.088	0.13	$20,000	$230,000	$500,000
Contra Costa	1.5	3.4	5.6	$850,000	$9,400,000	$21,000,000
Del Norte	0.021	0.049	0.079	$12,000	$130,000	$300,000
El Dorado	0.30	0.68	1.1	$180,000	$1,900,000	$4,400,000
Fresno	2.2	5.4	8.4	$1,200,000	$14,000,000	$31,000,000
Glenn	0.052	0.12	0.20	$30,000	$320,000	$730,000
Humboldt	0.093	0.22	0.35	$55,000	$620,000	$1,400,000
Imperial	0.25	0.61	0.95	$140,000	$1,600,000	$3,600,000
Inyo	0.0097	0.025	0.037	$6,000	$71,000	$150,000
Kern	1.6	3.9	6.2	$900,000	$10,000,000	$23,000,000
Kings	0.27	0.65	1.0	$160,000	$1,700,000	$3,900,000
Lake	0.093	0.22	0.35	$53,000	$590,000	$1,300,000
Lassen	0.029	0.070	0.11	$17,000	$190,000	$420,000
Los Angeles	13.62	32	52	$8,200,000	$90,000,000	$200,000,000
Madera	0.36	0.83	1.4	$200,000	$2,200,000	$4,900,000
Marin	0.20	0.48	0.76	$120,000	$1,300,000	$3,000,000
Mariposa	0.025	0.062	0.097	$15,000	$170,000	$370,000
Mendocino	0.091	0.22	0.35	$52,000	$580,000	$1,300,000
Merced	0.62	1.5	2.4	$340,000	$3,900,000	$8,600,000
Modoc	0.0043	0.011	0.016	$3,000	$32,000	$67,000
Mono	0.0087	0.022	0.033	$5,000	$60,000	$130,000

County	Total Statistical Cases Avoided			Total Valuation (2023$)		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Monterey	0.66	1.6	2.5	$370,000	$4,100,000	$9,200,000
Napa	0.18	0.45	0.70	$110,000	$1,200,000	$2,700,000
Nevada	0.14	0.35	0.55	$89,000	$990,000	$2,200,000
Orange	4.0	9.2	15	$2,400,000	$26,000,000	$59,000,000
Placer	0.95	2.3	3.6	$560,000	$6,200,000	$14,000,000
Plumas	0.018	0.045	0.069	$11,000	$130,000	$270,000
Riverside	3.5	8.5	13	$2,000,000	$23,000,000	$50,000,000
Sacramento	3.4	8.3	13	$2,000,000	$23,000,000	$50,000,000
San Benito	0.10	0.25	0.40	$60,000	$670,000	$1,500,000
San Bernardino	3.2	7.6	12	$1,800,000	$21,000,000	$45,000,000
San Diego	4.1	9.7	16	$2,400,000	$27,000,000	$60,000,000
San Francisco	0.63	1.4	2.4	$400,000	$4,400,000	$9,900,000
San Joaquin	1.5	3.9	5.9	$860,000	$9,900,000	$21,000,000
San Luis Obispo	0.35	0.85	1.3	$210,000	$2,300,000	$5,100,000
San Mateo	0.59	1.4	2.2	$360,000	$3,900,000	$8,900,000
Santa Barbara	0.58	1.4	2.2	$330,000	$3,700,000	$8,100,000
Santa Clara	2.4	5.6	9.0	$1,400,000	$16,000,000	$35,000,000
Santa Cruz	0.49	1.1	1.9	$290,000	$3,300,000	$7,200,000
Shasta	0.35	0.80	1.3	$200,000	$2,200,000	$5,000,000
Sierra	0.0026	0.0062	0.0098	$2,000	$1,700	$38,000
Siskiyou	0.036	0.16	0.14	$21,000	$310,000	$520,000
Solano	0.64	1.5	2.4	$370,000	$4,200,000	$9,300,000
Sonoma	0.55	1.3	2.1	$320,000	$3,500,000	$8,000,000
Stanislaus	1.0	2.3	3.9	$560,000	$6,200,000	$14,000,000
Sutter	0.24	0.58	0.90	$130,000	$1,500,000	$3,200,000
Tehama	0.14	0.33	0.52	$76,000	$850,000	$1,900,000
Trinity	0.018	0.042	0.067	$11,000	$120,000	$260,000

County	Total Statistical Cases Avoided			Total Valuation (2023$)		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Tulare	1.05	2.7	4.0	$580,000	$7,000,000	$15,000,000
Tuolumne	0.091	0.22	0.35	$54,000	$600,000	$1,300,000
Ventura	0.97	2.3	3.7	$570,000	$6,200,000	$14,000,000
Yolo	0.42	0.95	1.6	$240,000	$2,600,000	$5,900,000
Yuba	0.21	0.48	0.79	$120,000	$1,300,000	$2,900,000
California Total	**57**	**134**	**214**	**$33,000,000**	**$370,000,000**	**$820,000,000**

8.2.3 Limitations

Limitations of these Approach II results are consistent with those described in Chapter 6 . No additional limitations were introduced in conducting the proof-of-concept case study. Although the case study is based on a simplified hypothetical regulation, it illustrates the process of implementing bridging methods and applying valuation to the results. Application to real-world regulations may introduce additional uncertainties.

8.3 Approach III

We describe in this section the proof-of-concept case study used to demonstrate application of Approach III for quantification and valuation of fatal and non-fatal risks of TAC exposure. Approach III is the most granular of the three approaches to quantifying and valuing TAC cancer risks.

- **Scenario.** This case study develops valuation estimates for cancer risk reductions associated with a hypothetical regulation that reduces emissions of DPM from sources statewide. The baseline scenario assumes a BAU emissions trajectory for DPM emissions. The regulatory scenario is assumed to be implemented by 2025 and begin generating benefits in that year. The regulation is expected to reduce concentrations of DPM by 40 percent by 2035 compared to the baseline, and that change is modeled linearly from 2025 to 2035. Additional benefits are calculated for exposures that occur through 2050.
- **Geographic scope and population affected.** Impacts of the emissions of DPM are expected to be widespread throughout the state, given the widespread nature of diesel fuel-burning trucks, trains, and boats in California. DPM can be found throughout the entire state, and is especially concentrated near major roadways, ports, and transit lines. While DPM can be modeled as a localized pollutant, as was done in the case study for Approach I, here we focus on larger-scale modeling of DPM concentrations. The broad-based regulation envisioned in this hypothetical case study supports the assessment of changes in population-level risks, where benefits are expressed as changes in statistical cases of fatal and non-fatal cancers relative to baseline conditions. The term "statistical cases" refers to the fact that we are estimating changes in the incidence of disease in the population, not identifying specific cases of cancer for specific individuals. The results represent the reduction in numbers of cancer cases expected in the exposed population over the study period.

8.3.1 Methods

The case study applied the basic steps of risk assessment (hazard identification, exposure assessment, dose-response analysis, and risk characterization), but changes to the assumptions or inputs within those steps were

made as "bridges" to a socioeconomic-style benefits analysis. We also added the valuation step that assigns a monetary estimate to the quantified health benefits associated with the regulation.

Approach III is designed to complement risk analyses of widespread, ambient pollutants for which cancer toxicology data are derived from human epidemiological studies. This requires spatially modeled data that show either a baseline level of ambient pollutant concentrations (from which a percent reduction would be calculated) or a baseline level and regulatory level of ambient pollution concentrations (which would be compared against each other).

- **Hazard Identification.** DPM is a well-studied TAC emitted by diesel fueled engines. A substantial base of epidemiological studies of railroad workers and long-haul truck drivers identifies lung cancer as an outcome of chronic inhalation exposure to DPM. In 1998, OEHHA conducted a health risk assessment of DPM and found sufficient evidence to establish a unit risk of 3.0 E-4 $(\mu g/m^3)^{-1}$ for lung cancer associated with DPM inhalation exposure. Additional detail regarding the evidence base supporting this determination and unit risk can be found in Appendix B of OEHHA, 2009. Because this health effect, a fatal cancer or a diagnosis of a non-fatal cancer, is one for which valuation estimates exist, we proceeded with the case study with no adjustments to the particular hazard.

- **Exposure assessment.** The bridging approach for exposure sought to both develop a central estimate of exposure to DPM of the average individual in the population affected; and characterize a distribution of values around that central estimate that reflects uncertainty and variability due to time-activity patterns (including daily commuting patterns) and other factors that may vary from person to person. We obtained modeled estimates of ambient DPM concentrations throughout California at the census tract-scale, obtained from the 2020 version of U.S. EPA's ATS model. These results represent typical ambient concentrations to which California residents may be exposed. As recommended in Section 7.4, we then fed these data into U.S. EPA's HAPEM8 model, version 8.0 to develop the distribution of activity-adjusted personal exposure concentrations by census tract based on HAPEM8 population sampling and time-activity data. HAPEM8 results are stratified by age groups, allowing for fine-resolution analysis. Additional details regarding this process can be found in Appendix E. Since HAPEM8 processes pollution data linearly (i.e. a 50 percent reduction in input concentrations equals a 50 percent reduction in output concentrations), we applied percent changes in DPM emissions directly to our HAPEM8 results. Our life table conducts Monte Carlo sampling from these distributions as part of the exposure assessment and risk characterization.

- **Dose response assessment** The life table risk assessment in Approach III was supported by epidemiological studies of occupational exposures to DPM, where the cancer outcome of concern is cancer of the lung or bronchus. The specific relative risk value used to calculate changes in lung cancer incidence was developed in consultation with OEHHA and is based on OEHHA's 1998 document, *Part B: Health Risk Assessment for Diesel Exhaust*. We selected as our central estimate of lung cancer potency the maximum likelihood estimate (MLE) RR value from the meta-analysis of 12 studies conducted by OEHHA as part of that assessment and described Appendix C of the 1998 document. The 95UCL value on the mean RR from that meta-analysis is consistent with the value used by OEHHA to derive its current unit risk value for DPM.[23] However, in keeping with this methodology's stated objective to model a central population estimate along with a probability distribution to characterize uncertainties, we opted to center our estimate on the MLE RR of 1.43, while including the full distribution of uncertainty around the RR value based on the reported 90 percent confidence interval.

[23] See OEHHA (2009) for the derivation of the DPM unit risk value of 3.0 E-4 $(\mu g/m^3)^{-1}$ associated with risk of lung cancer mortality.

This distribution was used in the Monte Carlo analyses conducted as part of the life table model. We used this RR combined with OEHHA's estimate of DPM exposure for the meta-analysis from OEHHA (1998, 2009) to develop our MHAF value, as described in Chapter 7:

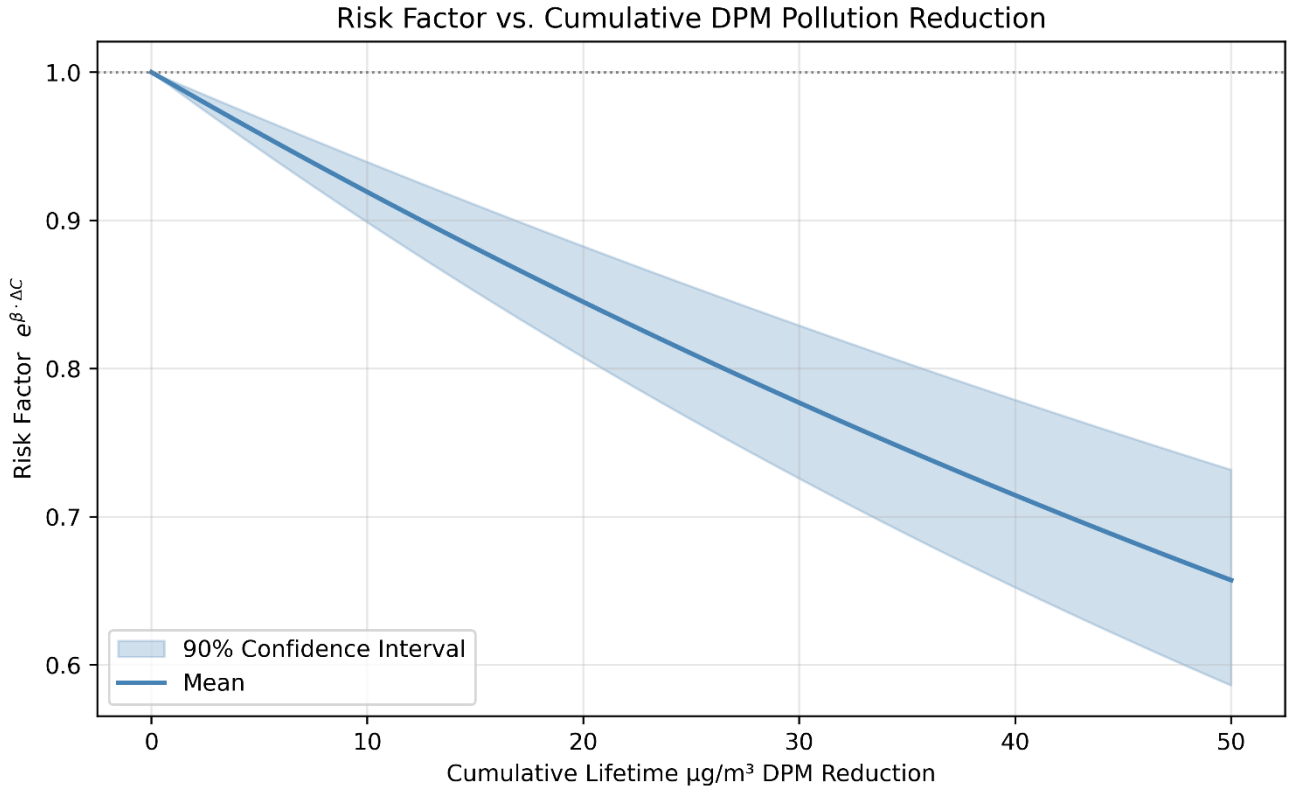
$$\text{MHAF}_{\text{year}} = e^{\Delta\text{Exposure}_{year-lag} \times \frac{\ln(RR)}{\text{OEHHA meta-analysis exposure}}}$$

Where:

- $\text{MHAF}_{\text{year}}$ = the cancer mortality hazard adjustment factor for a given year
- $\ln(RR)$ = the natural log of the relative risk value for cancer mortality where RR is sampled from a distribution centered on the MLE estimate of 1.43 [90% CI: 1.31, 1.57]
- OEHHA Meta-analysis Exposure, adjusted for differences in exposure between occupational cohorts and the general population $= 200 \frac{\mu g}{m^3} \times \frac{10m^3}{\frac{20m^3}{day}} \times \frac{5\ days}{7\frac{days}{week}} \times \frac{48\ wks}{52\frac{wks}{year}} \times \frac{45\ yrs}{70\frac{yrs}{lifetime}}$
- $\Delta\text{Exposure}_{\text{year-lag}}$ = the change in cumulative lifetime exposure under the regulatory scenario in the current year – the lag between exposure and diagnosis (e.g. 2027, if the year is 2032 and the lag is 5 years)

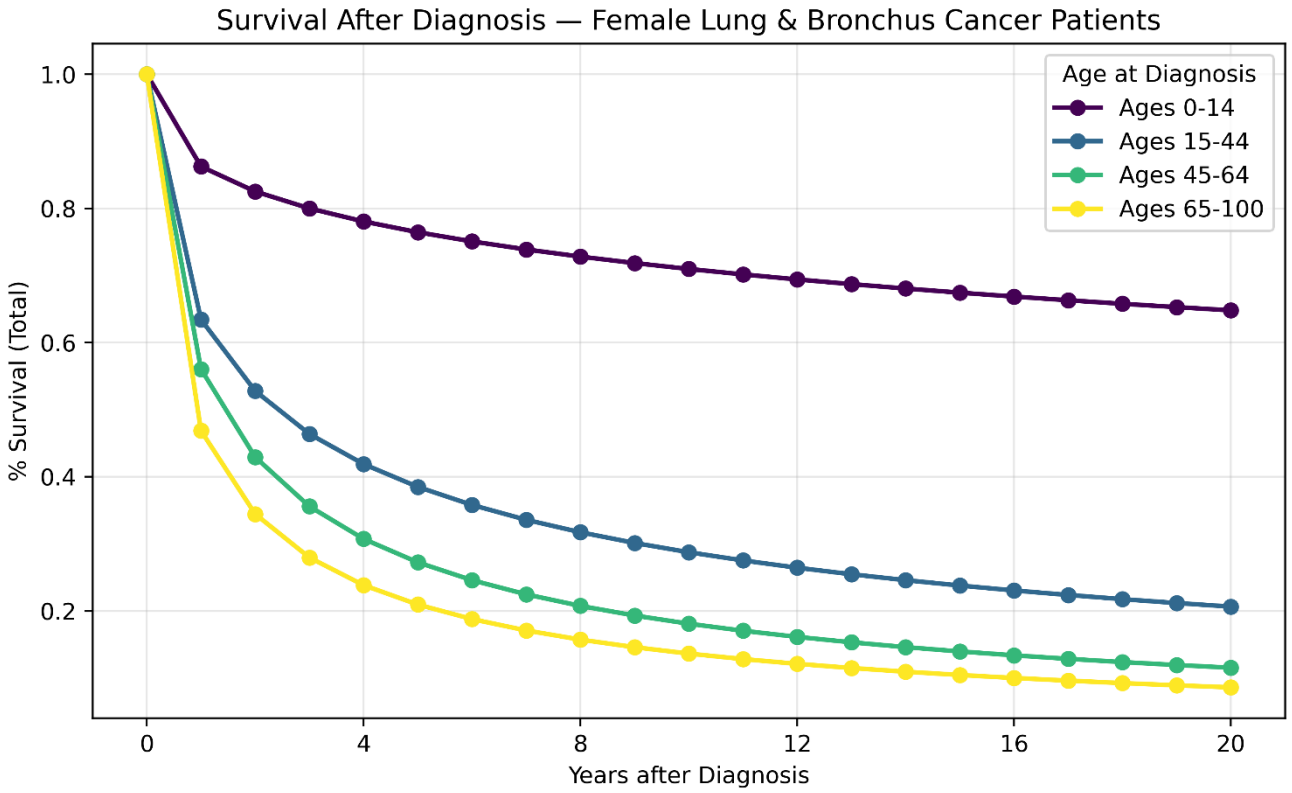
Figure 18 represents the Risk Adjustment Factor for lung cancer and DPM as a function of cumulative exposure reduction in DPM, where the x-axis represents cumulative exposure reduction and the y-axis represents risk factor. These values are less than one because they represent reductions on existing cancer risk as a result of decreases in cumulative lifetime exposure. The relative risk value is still centered around 1.43 [90% CI: 1.31, 1.57], but $\Delta\text{Exposure}$ is negative, so the exponent on e becomes negative.

Figure 18. Risk Factor (MHAF) as a Function of Cumulative DPM Pollution Exposure Reduction



- **Risk Characterization.** We estimated annual risk changes for a California statewide cohort based on 2025 population, for DPM exposure changes experienced by the cohort from 2025 through 2050. For completeness, we continue to track cancer risk changes to the 2050 population over their remaining lifetimes, though we assume no further exposure changes. After calculating cancer fatalities using life table methods, we used age- and sex-stratified lung cancer survival data to estimate the number of non-fatal cases that would have accompanied that cohort of fatal cases. We multiplied the number of fatalities by $\frac{1-\text{Survival\%}}{\text{Survival\%}}$ to obtain the number of expected non-fatal cases that accompany that cohort of deaths, where Survival% is based on age- and sex-stratified 20-year lung cancer mortality estimates. Figure 19 shows 20-year survival after diagnosis of lung and bronchus cancer in women at different age groups in California.

Figure 19. 20-Year Survival After Diagnosis for Female Lung Cancer Patients



Given these survival data, we back-calculated likely years of diagnosis. We took the proportion of fatalities that occur *n* years after diagnosis to assign a proportion of diagnoses associated with deaths in year *y*-*n*, as illustrated in the following example:

Given a calculation of 60 deaths in the year 2040, and a 20-year total mortality rate of 60 percent from lung cancer, we assigned 40 non-fatal cases to be distributed in the years before 2040 according to the observed survival probabilities. Referring to the survival curves in Figure 19, we observe that most deaths (as a percent of total deaths) occur in the year after diagnosis. Thus, we assumed that most deaths in 2040 came from diagnoses in the year prior, i.e., 2039. We continued to apply this curve to distribute total non-fatal cases across the preceding period (e.g., 50 percent of deaths in the year after diagnosis, a further 25 percent two years after diagnosis, and the remainder distributed from 2037 and earlier years). This distribution of non-fatal cases stops twenty years before death, or in the first eligible year of analysis, whichever comes first. The first eligible year

of analysis was determined by adding the minimum lag between exposure and diagnosis for that cancer type (4 years for lung cancer, see Howard 2015). Thus, for an analysis beginning with the first pollution reduction in 2025, the first non-fatal case would occur in 2029.

For deaths, we assumed a 1-year minimum lag, yielding a total lag between exposure and death of lag+1, or, in the case of lung cancer, 5 years. This mirrors OEHHA's approach in Part B of the Health Risk Assessment for Diesel Exhaust (OEHHA 1998), where exposures in the five years preceding death are assigned zero weight.

- **Valuation:** After calculating fatal and non-fatal lung cancer cases by year, we valued these cases using EPA's VSL distribution and the lung cancer valuation parameters from Bosworth et al. (2008). We applied the same VSL and non-fatal WTP distribution across all races, counties, sexes, and age groups in a given analysis.

8.3.2 Results

We present below the risk and exposure results estimated for this hypothetical DPM reduction using Approach III.

8.3.2.1Risk characterization

Figures Figure 20 and Figure 21 depict the spatial distribution of baseline DPM concentrations and total cancer cases avoided over the study period. Figure 20 provides tract-level results for Kern County; Figure 21 provides county-level results across California. In both figures, we contextualize the results by depicting the poverty percentile from CalEnviroScreen (CES) 4.0. Higher percentiles (depicted in dark green) represent impoverished communities. We note that tract results are associated with minor reductions, in relative terms, in cancer risks. When aggregated to the county level (Figure 21Figure 17), these reductions depict meaningful improvements in human health.

Figure 20. Map of Cancer Cases Averted through Hypothetical DPM Regulation, Kern County 2025-2050

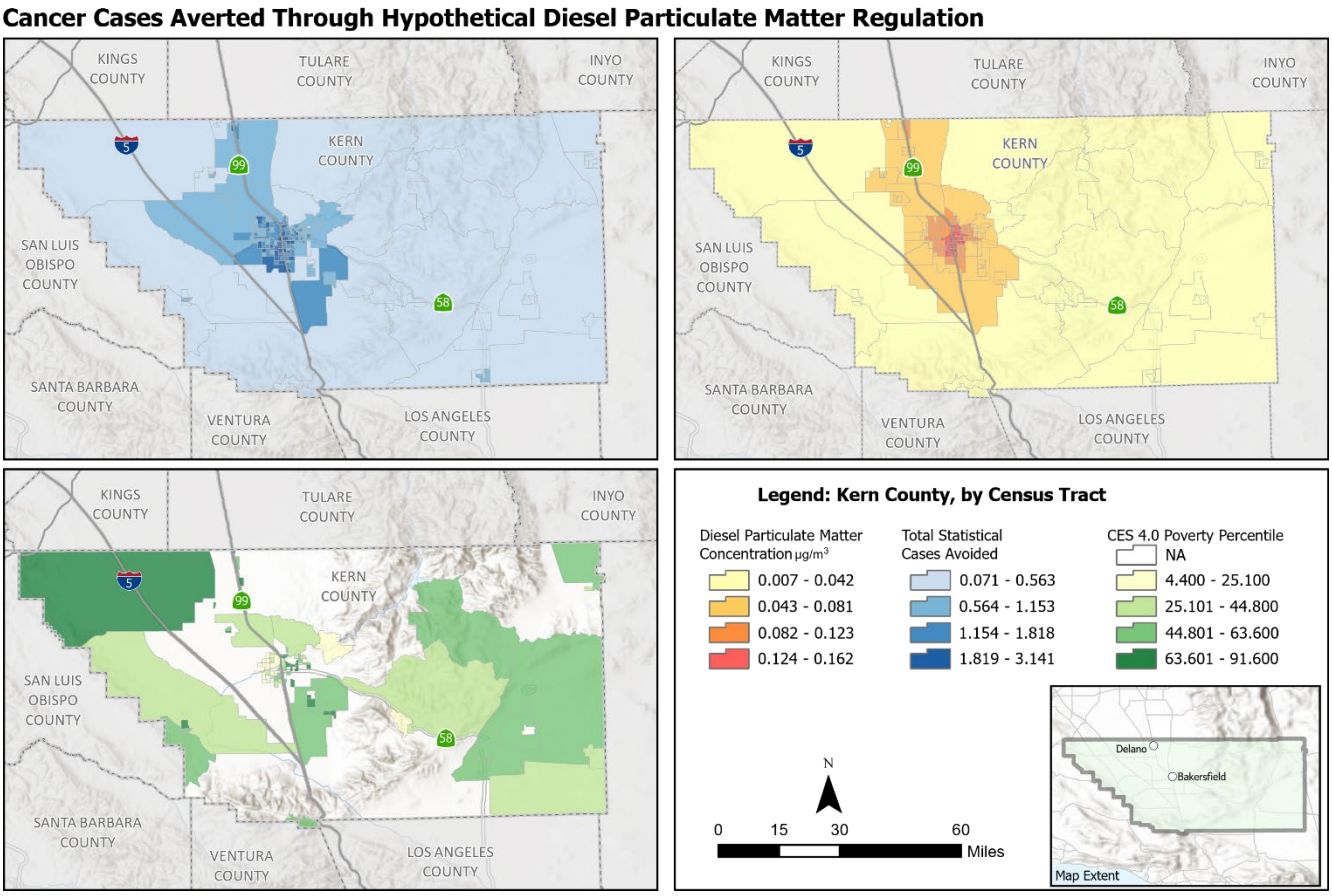
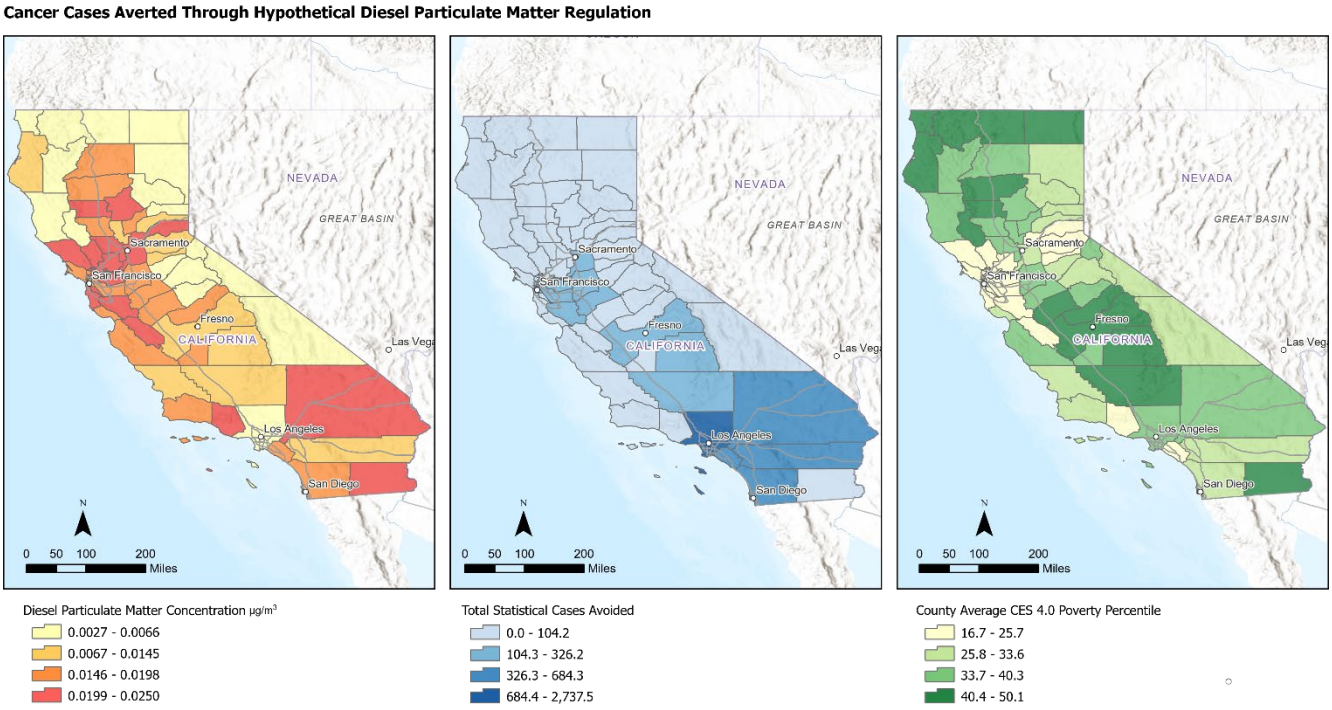


Figure 21. Map of Cancer Cases Averted through Hypothetical DPM Regulation, Statewide



The results depicted above are aggregate measures across all racial and ethnic groups. In Figure 22, we present the change in cancer cases across three racial/ethnic groups: Asian American and Pacific Islander; Black; and Hispanic/Latino. These results largely reflect the relative sizes and locations of these groups – the model outputs support computing additional metrics, such as per capita deaths or percent changes in baseline deaths.

Figure 22. Comparison of Averted Cancer Cases by Race/Ethnicity, Statewide 2025-2050

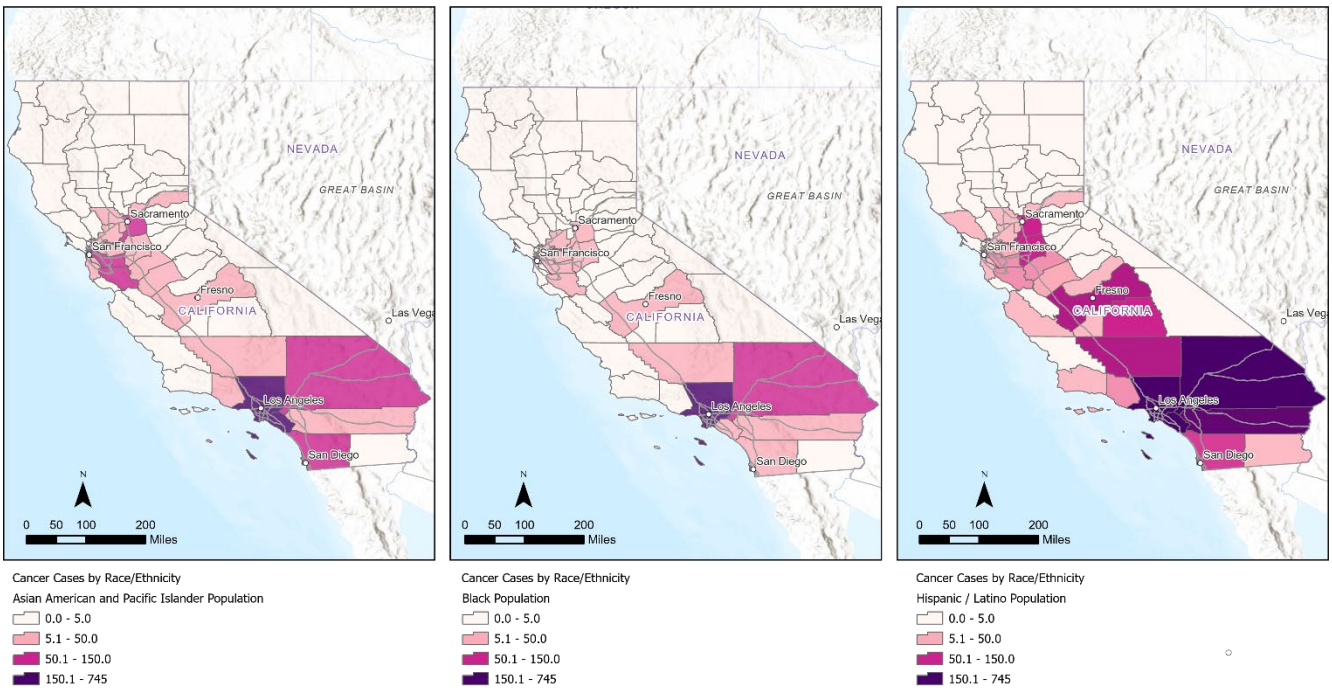


Figure 23 displays the proportion of cancer deaths by race/ethnicity in California, which is influenced by baseline cancer mortality rates and population demographics.

Figure 23. Proportion of Cancer Deaths by Race/Ethnicity, DPM Scenario/Statewide Analysis

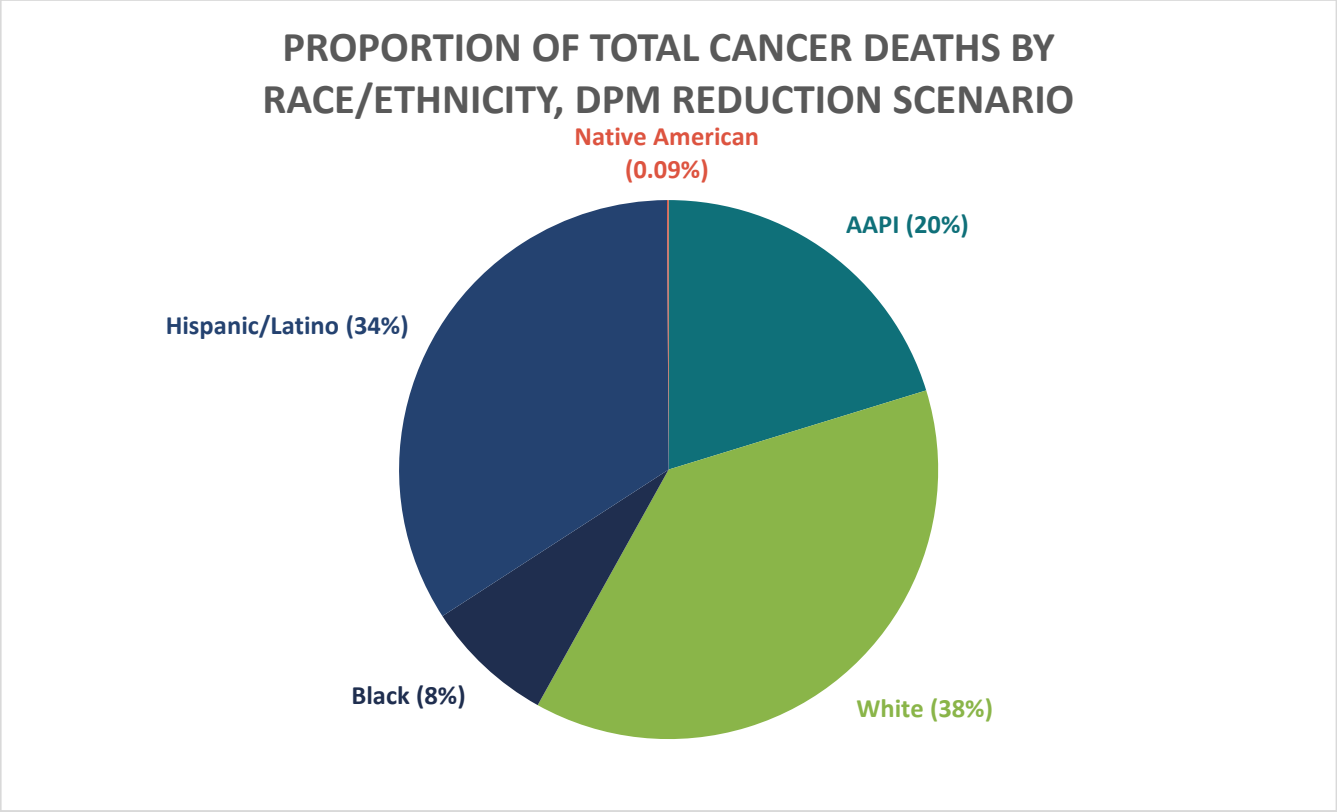


Table 11 shows example results for our 40 percent DPM reduction from 2025-2035, held constant until 2050.

Table 11. Example Results for Hypothetical DPM Regulation

County	Non-fatal Statistical Cases Avoided			Fatal Statistical Cases Avoided		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Alameda	14	20	24	190	260	320
Alpine	0	0	0	0	0	0
Amador	0.09	0.13	0.16	1.2	1.7	2.1
Butte	1.1	1.6	1.9	15	21	25
Calaveras	0.09	0.13	0.16	1.2	1.7	2.1
Colusa	0.08	0.11	0.14	1.1	1.5	1.8
Contra Costa	7.5	10	13	98	140	170
Del Norte	0.03	0.04	0.05	0.4	0.5	0.6

County	Non-fatal Statistical Cases Avoided			Fatal Statistical Cases Avoided		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
El Dorado	0.48	0.67	0.82	6.3	8.7	11
Fresno	17	23	29	220	300	370
Glenn	0.13	0.19	0.23	1.7	2.4	2.9
Humboldt	0.27	0.38	0.46	3.5	4.9	6
Imperial	1.4	2	2.4	18	25	31
Inyo	0.02	0.02	0.03	0.2	0.3	0.4
Kern	14	19	23	180	240	300
Kings	1.6	2.2	2.7	21	29	36
Lake	0.09	0.12	0.15	1.1	1.6	1.9
Lassen	0.02	0.03	0.04	0.3	0.4	0.5
Los Angeles	140	200	240	1,800	2,500	3,100
Madera	2.2	3	3.7	28	39	48
Marin	0.75	1.1	1.3	9.8	14	17
Mariposa	0.02	0.03	0.04	0.3	0.4	0.5
Mendocino	0.12	0.17	0.21	1.6	2.2	2.7
Merced	4.1	5.7	6.9	53	73	89
Modoc	0	0.01	0.01	0	0.1	0.1
Mono	0.01	0.01	0.01	0.1	0.2	0.2
Monterey	1.8	2.5	3.1	24	33	40
Napa	0.5	0.69	0.84	6.4	8.9	11
Nevada	0.19	0.26	0.32	2.5	3.4	4.2
Orange	35	49	60	460	640	780
Placer	3.3	4.5	5.5	42	59	71
Plumas	0.02	0.02	0.03	0.2	0.3	0.4
Riverside	28	38	47	360	500	610
Sacramento	15	21	25	190	270	320
San Benito	0.29	0.4	0.49	3.8	5.2	6.4

County	Non-fatal Statistical Cases Avoided			Fatal Statistical Cases Avoided		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
San Bernardino	32	44	54	410	570	700
San Diego	29	40	49	380	530	640
San Francisco	7.7	11	13	100	140	170
San Joaquin	9.6	13	16	120	170	210
San Luis Obispo	0.71	0.99	1.2	9.2	13	16
San Mateo	3.6	5	6.1	47	65	79
Santa Barbara	2.4	3.3	4	31	42	52
Santa Clara	14	20	24	180	260	310
Santa Cruz	0.8	1.1	1.4	10	14	18
Shasta	0.75	1	1.3	9.7	14	17
Sierra	0	0	0	0	0	0
Siskiyou	0.05	0.07	0.09	0.7	0.9	1.1
Solano	2.7	3.7	4.6	35	48	59
Sonoma	1.9	2.6	3.2	24	34	41
Stanislaus	6.4	8.9	11	83	120	140
Sutter	0.65	0.9	1.1	8.4	12	14
Tehama	0.34	0.47	0.57	4.4	6.1	7.5
Trinity	0.01	0.01	0.01	0.1	0.2	0.2
Tulare	6.5	8.9	11	84	120	140
Tuolumne	0.08	0.11	0.14	1.1	1.5	1.8
Ventura	5.4	7.5	9.1	70	97	120
Yolo	1.7	2.4	2.9	22	31	38
Yuba	0.57	0.78	0.96	7.3	10	12
California Total	**420**	**580**	**710**	**5,400**	**7,500**	**9,200**

Valuation

Table 12 shows the mean, 5th, and 95th percentile total valuation for statistical cases avoided due to emissions reductions. Cases are valued using EPA's VSL distribution and the lung cancer valuation parameters from Bosworth et al. (2008) and combined to calculate the total valuation estimate. We applied the same VSL and non-fatal WTP distribution across all races, counties, sexes, and age groups. Results are separated by county, totaling to a mean estimated benefit for all of California valued at $ 50 billion (2023$).

Table 12. Example Valuation of Statistical Cases Avoided (2023$) for DPM Scenario

County	Total Statistical Cases Avoided			Total Valuation (2023$)		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Alameda	200	280	340	$290,000,000	$1,800,000,000	$4,400,000,000
Alpine	0.007	0.009	0.011	$9,900	$61,000	$150,000
Amador	1.3	1.8	2.2	$1,900,000	$12,000,000	$29,000,000
Butte	16	22	27	$22,000,000	$130,000,000	$330,000,000
Calaveras	1.3	1.8	2.2	$1,900,000	$12,000,000	$29,000,000
Colusa	1.2	1.6	1.9	$1,500,000	$9,500,000	$23,000,000
Contra Costa	110	150	180	$140,000,000	$890,000,000	$2,200,000,000
Del Norte	0.39	0.54	0.66	$520,000	$3,200,000	$7,800,000
El Dorado	6.7	9.4	11	$9,700,000	$60,000,000	$150,000,000
Fresno	240	330	400	$310,000,000	$1,900,000,000	$4,700,000,000
Glenn	1.9	2.6	3.2	$2,500,000	$15,000,000	$37,000,000
Humboldt	3.8	5.3	6.4	$5,400,000	$33,000,000	$82,000,000
Imperial	20	27	33	$26,000,000	$160,000,000	$390,000,000
Inyo	0.24	0.34	0.41	$340,000	$2,100,000	$5,200,000
Kern	190	260	320	$250,000,000	$1,500,000,000	$3,700,000,000
Kings	23	31	38	$30,000,000	$180,000,000	$450,000,000
Lake	1.2	1.7	2.1	$1,600,000	$10,000,000	$25,000,000
Lassen	0.32	0.44	0.54	$450,000	$2,700,000	$6,800,000
Los Angeles	2,000	2,700	3,300	$2,800,000,000	$17,000,000,000	$42,000,000,000
Madera	30	42	51	$39,000,000	$240,000,000	$590,000,000
Marin	11	15	18	$15,000,000	$94,000,000	$230,000,000
Mariposa	0.31	0.43	0.53	$470,000	$2,900,000	$7,100,000

County	Total Statistical Cases Avoided			Total Valuation (2023$)		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Mendocino	1.7	2.4	2.9	$2,300,000	$14,000,000	$35,000,000
Merced	57	79	96	$73,000,000	$440,000,000	$1,100,000,000
Modoc	0.053	0.073	0.09	$78,000	$480,000	$1,200,000
Mono	0.12	0.16	0.2	$170,000	$1,000,000	$2,600,000
Monterey	26	35	43	$34,000,000	$210,000,000	$510,000,000
Napa	6.9	9.6	12	$9,400,000	$58,000,000	$140,000,000
Nevada	2.7	3.7	4.5	$3,900,000	$24,000,000	$58,000,000
Orange	490	680	830	$700,000,000	$4,300,000,000	$11,000,000,000
Placer	45	63	77	$61,000,000	$380,000,000	$930,000,000
Plumas	0.24	0.33	0.41	$340,000	$2,100,000	$5,100,000
Riverside	390	540	660	$520,000,000	$3,200,000,000	$7,900,000,000
Sacramento	210	290	350	$280,000,000	$1,700,000,000	$4,300,000,000
San Benito	4.1	5.6	6.9	$5,600,000	$34,000,000	$84,000,000
San Bernardino	450	620	750	$600,000,000	$3,700,000,000	$9,100,000,000
San Diego	410	570	690	$570,000,000	$3,500,000,000	$8,700,000,000
San Francisco	110	150	180	$160,000,000	$1,000,000,000	$2,500,000,000
San Joaquin	130	180	230	$170,000,000	$1,100,000,000	$2,600,000,000
San Luis Obispo	9.9	14	17	$13,000,000	$83,000,000	$200,000,000
San Mateo	50	70	86	$73,000,000	$450,000,000	$1,100,000,000
Santa Barbara	33	46	56	$43,000,000	$260,000,000	$650,000,000
Santa Clara	200	270	340	$280,000,000	$1,700,000,000	$4,300,000,000
Santa Cruz	11	16	19	$16,000,000	$96,000,000	$240,000,000
Shasta	10	15	18	$14,000,000	$87,000,000	$210,000,000
Sierra	0.026	0.036	0.044	$37,000	$220,000	$550,000
Siskiyou	0.72	1	1.2	$1,000,000	$6,100,000	$15,000,000
Solano	38	52	64	$51,000,000	$310,000,000	$770,000,000
Sonoma	26	36	44	$36,000,000	$220,000,000	$540,000,000
Stanislaus	90	120	150	$120,000,000	$720,000,000	$1,800,000,000

County	Total Statistical Cases Avoided			Total Valuation (2023$)		
	5th Percentile	Mean	95th Percentile	5th Percentile	Mean	95th Percentile
Sutter	9.1	13	15	$12,000,000	$73,000,000	$180,000,000
Tehama	4.7	6.6	8	$6,200,000	$38,000,000	$94,000,000
Trinity	0.12	0.17	0.21	$180,000	$1,100,000	$2,700,000
Tulare	90	120	150	$120,000,000	$720,000,000	$1,800,000,000
Tuolumne	1.2	1.6	2	$1,600,000	$9,900,000	$24,000,000
Ventura	75	100	130	$100,000,000	$630,000,000	$1,600,000,000
Yolo	24	33	40	$32,000,000	$200,000,000	$490,000,000
Yuba	7.9	11	13	$10,000,000	$63,000,000	$150,000,000
California Total	**5,800**	**8,100**	**9,900**	**$8,100,000,000**	**$50,000,000,000**	**$120,000,000,000**

8.3.3 Limitations

Limitations of these Approach III results are consistent with those described in Chapter 7 . An additional limitation encountered in implementing this case study was the need to use state-level race- and gender-stratified mortality rate data in place of finer scale estimates as originally intended, in order to address data suppression issues. As a result, results for finer scale geographies may not fully reflect local variation in cancer mortality. Future applications may wish to further explore means to impute masked data or to re-aggregate rates across other dimensions (e.g., sex) to address these data gaps. Although the case study is based on a simplified hypothetical regulation, it illustrates the process of implementing bridging methods to allow risk assessments to better support socioeconomic analyses and applying valuation to the results. Application to real-world regulations may introduce additional uncertainties.

8.4 Recommendations for Future Work

The three proof-of-concept case studies successfully demonstrate the application of the three proposed approaches for quantifying and valuing TAC-related cancer risk changes; however, we recommend that CARB considers undertaking additional efforts prior to implementation. These efforts may include:

- **Conduct additional case studies.** The existing studies are limited in number (one per approach) and two of three are for hypothetical policies. Undertaking case studies contributes to the refining of methods to account for variability across TACs, cancer types, and policy scenarios; and code developed for these case studies would benefit from additional testing to ensure robustness across varying scenarios and data. Additional case studies will allow for exploration and testing of other refinements suggested by reviewers, including;
    - how CARB might augment Approach III to incorporate elements of interpersonal variability in potency, which is currently addressed in Approach II, along with the uncertainty in RR values;
    - how CARB might best adapt the Approach III life table model to address RR values focused on cancer incidence instead of cancer mortality;
    - how CARB might use Tier I results from an equity perspective in addition to efficiency-focused analyses such as break-even analysis; and
    - exploring ways to better incorporate finer-scale EJ-relevant data into Approach III.

Possible TACs to consider for future case studies include analysis of a metallic TAC, which can exhibit localized impacts where the exposed population is uncertain and thus would support Approach I; acrolein, for which CARB recently released a draft cancer IUR in May 2026 based on studies in mice and rats, as a candidate for Approach II; and ethylene oxide or benzene for Approach III, noting that CARB released a draft cancer IUR in May 2026 based on human studies for the former.

- **Refine guidance and develop materials for non-technical audiences.** The existing report describes methods that are highly technical, and external peer reviewers of a previous draft noted that in places, it could be difficult to follow. We have made some revisions in this current document to help address these issues. However, following any further case study development, we recommend refreshing the report with an eye towards additional transparency and clarity. We also recommend generating public-facing documents as needed for non-technical audiences, such as fact sheets or user guides. One option to advance these documentation efforts is to develop a manuscript for submission to a peer-reviewed journal, which could also provide the opportunity for wider review.
- **Methodological enhancements.** Several methodological enhancements could be considered, following recent feedback from CARB and peer reviewers. These efforts could include review of recent life table analyses to identify potential improvements to Approach III; further examining the possibility of mapping cancers from animal studies to human cancer sites for Approach II, as supported by available data; and/or fine-scale modeling of population and/or health incidence and construction of "synthetic datasets" to address data limitations (i.e., suppression of small counts for fine spatial scales).
- **Recurrent literature review.** IEc leveraged recent data and literature wherever possible. In some cases—notably for non-fatal WTP values—literature was sparse. We recommend that CARB continues to monitor publications and data releases to inform these methods.

Finally, following any additional testing and refinement, we recommend that CARB plan to implement this methodology for TAC socioeconomic analysis first as a sensitivity analysis alongside an upcoming traditional CARB TAC risk assessment. This can help introduce the approach and highlight the key differences in objectives and methods, while also soliciting public comments for possible further refinement before finalizing the methodology as standalone supplement to the traditional risk assessment.

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Appendix A. Data Sources and Software

A.1 HAPEM8

In the real world, the concentrations of ambient pollutants people inhale differ from annual average outdoor pollutant concentrations like those CARB models. The quantities of pollutants people inhale on a given day depend on their activity levels, the microenvironments in which they spend their time, their breathing rates, their distance from major sources like roadways and power plants, employment level, age, and more. The substantial heterogeneity of many of these factors, combined with the uncertainty of the everyday routines across members of a large population, pose a significant challenge for estimating population-level pollutant exposures.

U. S. EPA's HAPEM8, is an exposure model designed to assess long-term, population-level exposures. As a screening-level model, its inputs are simple enough for CARB to run while still providing the information needed for the Approach II and III methods. It has been reviewed by U. S. EPA's Science Advisory Board, and it is used at the federal level for exposure screening analyses.

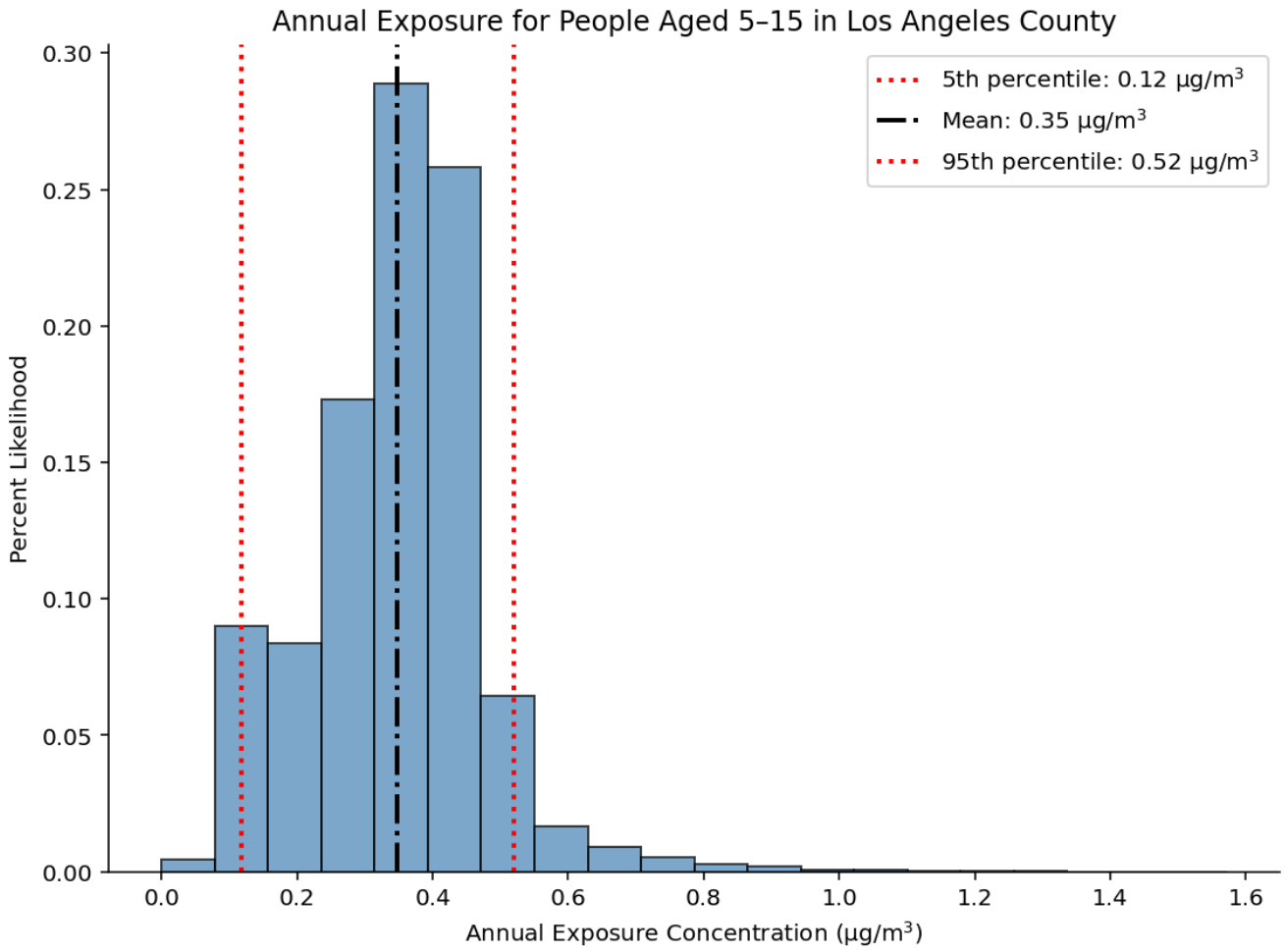
HAPEM8 uses annual average air quality data. HAPEM8's default configuration accepts census tract-level data, although pollution may be assessed at the county, state, or national level. These pollution data may be apportioned to specific source types, including point, nonpoint, onroad, and nonroad sources, but they also may be in the form of general ambient concentrations.

HAPEM8 combines inputted pollution concentration data with default data on population (from the 2020 Census), estimated concentrations in different microenvironments – i.e., indoors residence, indoors other, outdoors near-roadway, outdoor other, and in-vehicle (from HAPEM8), time-activity data (from U. S. EPA's Consolidated Human Activity Database, or CHAD). The software incorporates stochasticity into these measurements, drawing a predetermined number of random time-activity and population combinations for each demographic group in each census tract. The default demographic groups for HAPEM8 consist of the following six age groups:

- Ages 0–1
- Ages 2–4
- Ages 5–15
- Ages 16–17
- Ages 18–64
- Ages ≥ 65

When the population-weighted exposure data for each census tract and demographic group are combined, we end up with a distribution of exposures in a given county for a given demographic group. Figure A1 shows an example distribution for benzene in Los Angeles County.

Figure A1. Example Annual Benzene Exposure Distribution for 5-15-Year-Olds in LA County



For more information on HAPEM8, see EPA website and the HAPEM user guide.

A.2 Creation of California-Specific Incidence Rates

Our model employs California-specific cancer incidence rates that are supplemented by national data when necessary. The incidence data we use reflects the rate of diagnoses for a specific cancer in a given population or sub-population (e.g., 58-year-old non-Hispanic Black women in Santa Clara County) each year. We obtain cancer incidence data for all cancer sites listed in the SEER Site Recode from the California Cancer Registry (CCR) for the years 2016-2021 (excluding 2020 due to confounding effects of COVID). These data are stratified by sex, age, race/ethnicity, and county of residence (CCR 2025). CCR incidence rates are reported in terms of cases per 100,000 people and are suppressed if a county's population is below 20,001 or the number of cases reported is below eleven.

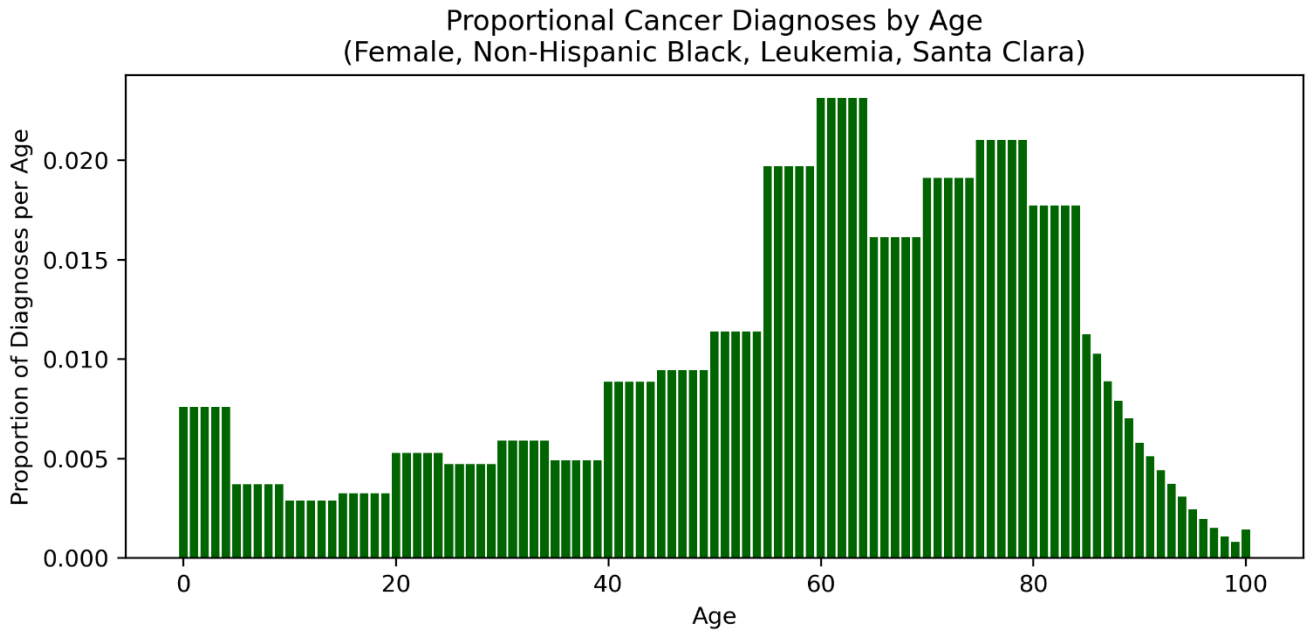
If county-level incidence rates for a given cancer site, sex, and race/ethnicity are unavailable, we first used state-level incidence rates from CCR for the same site, sex, and race/ethnicity to supplement the suppressed data. If state-level incidence rates are also unavailable, rates for the same sex and racial/ethnic group are used from national-level CDC WONDER data using the same years of reporting as CCR (2016-2021, excluding 2020) (CDC).

If national-level data are unavailable for a given cancer site, sex, and race/ethnicity, national-level incidence rates from all races/ethnicities are used for the cancer site and sex of interest. In all other cases where data are still unavailable an incidence rate of zero is used. This is most often the case for cancers with low incidence rates at the general population level, suggesting that there would be very low, if any incidence for smaller population groups (e.g. American Indian males above 65).

After incidence rates are assigned to each sex and race/ethnicity for a given cancer site, we normalize case counts for each demographic group by the proportional population at each age. The population for each age in our distribution is multiplied by the incidence rates identified through the methodology above and renormalized so that the distribution of lifetime incidence sums to one. For every age group from 0-84, we treat the population in each age bin as evenly distributed. For the 85–100-year-old age group, however, we normalize incidence proportional with the number of people at each age from 85–100 years, as determined from sex-specific U.S. Census data. We perform this adjustment to compensate for both **a)** the larger size of the age bin (16 vs 10 years), and **b)** to compensate for the high mortality rates among 85+ year olds that skew the proportion of overall new diagnoses each year to the low end of this age range.

Our final, resultant incidence distributions resemble that shown in Figure A2.

Figure A2. Proportional Lifetime Leukemia Incidence Rates for Non-Hispanic Black Women in Santa Clara County



Appendix B. Incorporation of Uncertainty and Variability into Approach II Risk Estimates

In Appendix B, we provide specific recommendations of how to incorporate uncertainty and variability into distributions of risk potency for our proposed Approach II calculations. These methods are primarily based on recommendations from *Science and Decisions* and from WHO/IPCS regarding interindividual human variability and uncertainty in estimates derived from animal studies. Our recommended methodology constructs a distribution around a given cancer unit risk, reflecting interindividual variability in human responsiveness to carcinogen exposure. We incorporate uncertainty in the parameters defining this distribution, which in turn affects the conversion of unit risk to **population** unit risk (to be used in risk assessments).

In the lognormal population distribution of cancer risk developed in 2009's *Science and Decisions*, the National Research Council proposes that a reasonable assumption of the **ratio** of the **95th percentile individual risk** to the **median individual risk** in a given population is a factor of 25 (i.e. the 95th percentile individual has 25 times greater risk than the 50th percentile individual, given the same exposure). These values are a general rule-of-thumb and were suggested as realistic bounds on the 95th-to-50th percentile ratio. WHO/IPCS's 2017 *Guidance Document on Evaluating and Expressing Uncertainty in Hazard Characterization* analyzes toxicity data to produce quantitative, probabilistic estimates of this 95th-to-50th percentile ratio. Their work improves upon the theoretical framework of *Science and Decisions*, clarifying the 90% confidence interval of the 95th-to-50th percentile ratio as (1.77, 14.02) and the median ratio as 3.41.[24]

We follow the WHO/IPCS methodology to recreate, and sample from, this distribution. Our entire treatment of uncertainty and variability in risk and exposure is explained below.

B.1 Parameterizing the 95th Percentile-to-Median Ratio Distribution

In the following steps, we explain how we model the **uncertainty** in the **95th percentile-to-median ratio** of the **population risk distribution** consistent with the recommendations clarified in the WHO/IPCS paper. This lognormal curve is represented by X, where each draw from X represents a $\frac{P_{95}}{P_{50}}$ ratio.

B.1.1 Relating X to the WHO/IPCS Methodology

The WHO/IPCS methodology defines a variable, GSD_H, that represents the geometric standard deviation of the "interindividual variability in the human equipotent dose distribution." The methodology defines $\log(GSD_H)$ as lognormally distributed. They also define the relationship between GSD_H and the 95th-to-50th ratio of the population distribution (our distribution X) as follows:

$$X = GSD_H^{z_{95}}$$

Putting everything together yields the following formula for finding our distribution X:

$$X = 10^{z_{95} \times lognormal(\mu,\sigma)}$$

[24] These values represent ratios for an effect with population incidence of 5%. Table 4.5 of the WHO/IPCS document shows a wider range of ratios as the incidence rate declines. Thus for rate health effects or types of cancers, this range may underestimate interindividual variability.

B.1.2 Quantifying Our Uncertainty Distribution $\log(GSD_H)$

In a lognormal equation,

$$Y_{50} = e^{\mu} \quad \text{and} \quad Y_N = e^{\mu + \sigma z_N},$$

where Y_{50} represents the median of the lognormal distribution, Y_N represents the Nth percentile of the distribution, z_N represents the z-score of the Nth percentile value in a normal distribution, and μ and σ represent the mean and standard deviation of the underlying normal distribution, respectively.

The WHO/IPCS methodology specifies that the lognormal distribution that parameterizes GSD_H has a median value of 0.324 and a 95th-to-50th percentile ratio of 2.152. To fit a lognormal curve in this instance, we must find a μ-value and a σ-value from our known values Y_{50}, Y_{95}/Y_{50}, and z_{95}.

We can rewrite the first equation above to isolate μ, giving us the equation:

$$\mu = \ln(Y_{50})$$

Substituting this into the second equation shown above, we find:

$$Y_N = e^{\ln(Y_{50}) + \sigma z_N}$$

Taking the natural logarithm of both sides and rewriting some terms yields the following equation, where σ is a function of the ratio of the Nth percentile to median value.

$$\sigma = \frac{\log\left(\frac{Y_N}{Y_{50}}\right)}{z_N}$$

Substituting the 95th percentile in for N yields the following:

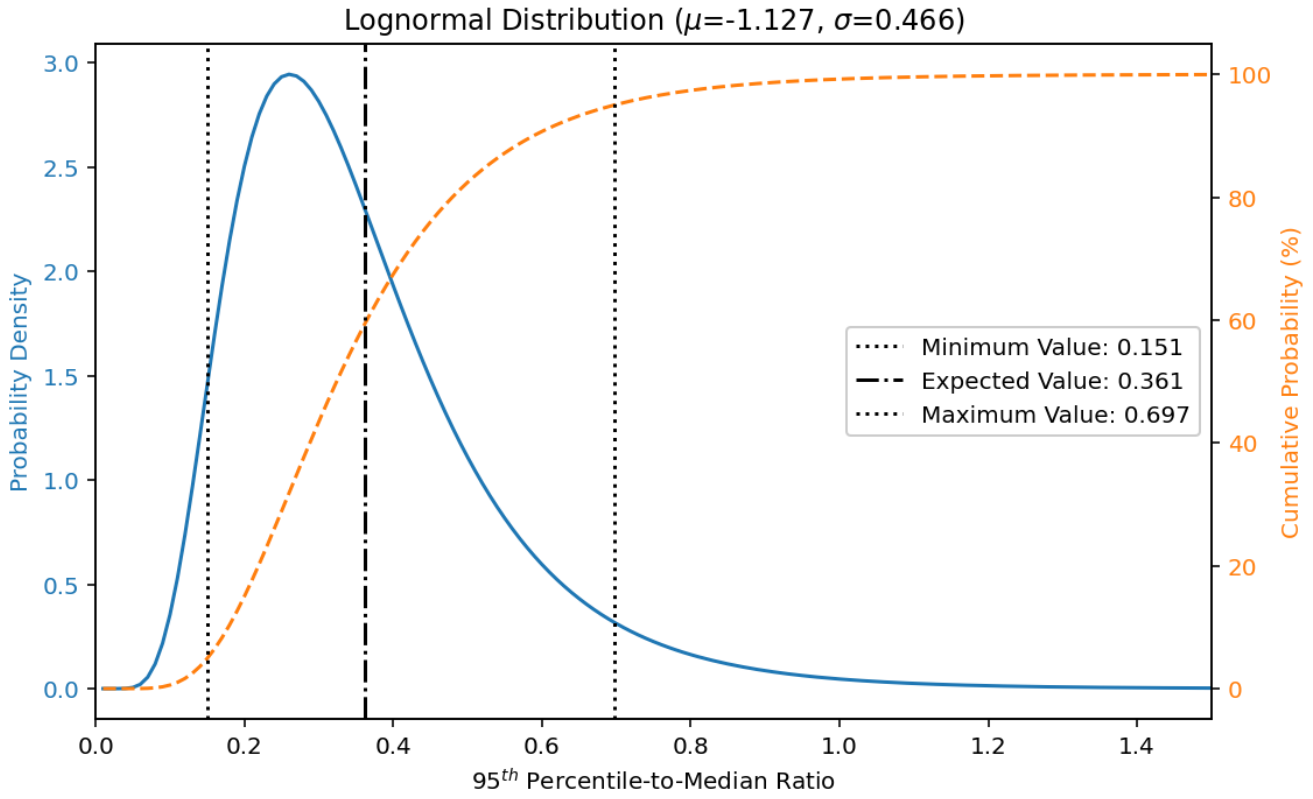
$$\sigma = \frac{\log\left(\frac{Y_{95}}{Y_{50}}\right)}{z_{95}}$$

Now, we can then substitute in the following values. The first two values are taken from the WHO/IPCS distribution, and the last represents the z-score for the 95th percentile of a normal distribution.

- $Y_{50} = 0.324$
- $\frac{Y_{95}}{Y_{50}} = 2.152$
- $z_{95} = 1.645$

We thus solve the value of σ, which is 0.466. We can also solve for the underlying μ-value, which is -1.127. The final distribution is plotted below in Figure B1.

Figure B1. Distribution of Log Uncertainty in Interindividual Variability



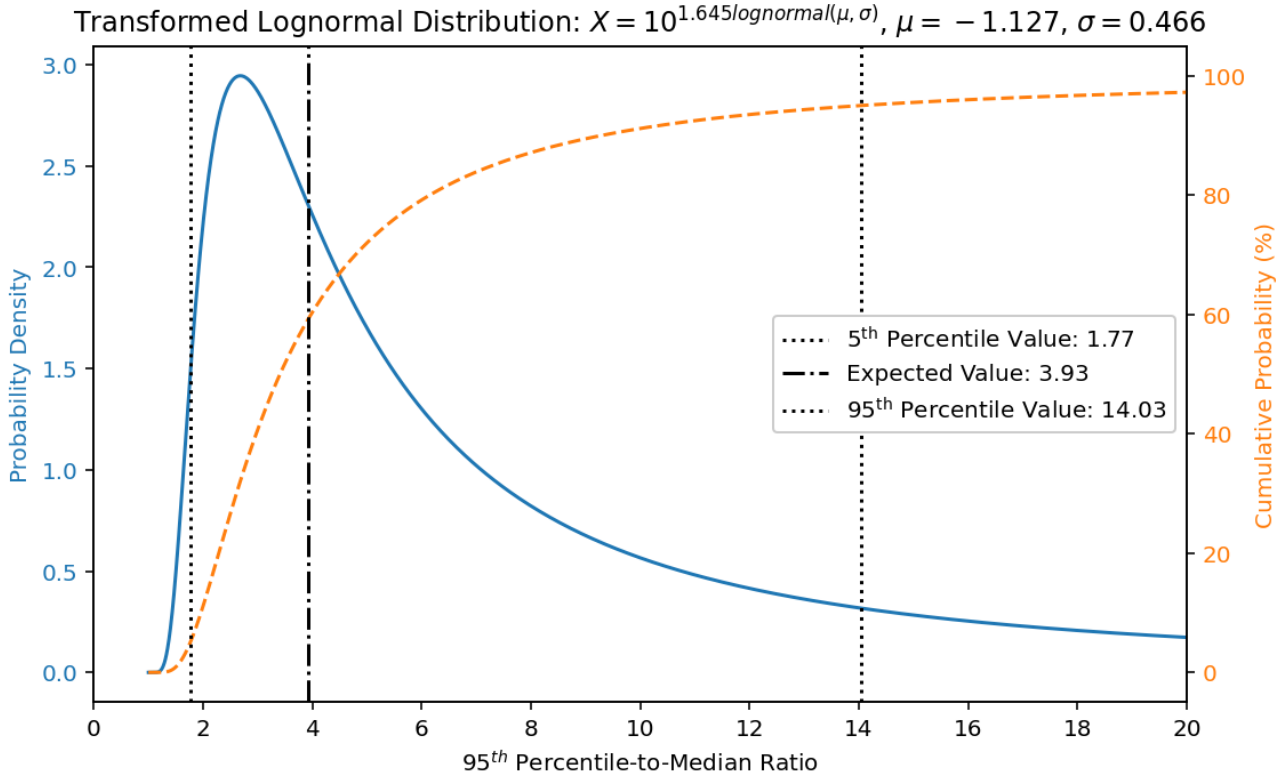
B.1.3 Building a Distribution for X

As explained above, we then take the values from the distribution above and perform the following adjustment:

$$X = 10^{z_{95} lognormal(-1.127, 0.466)} = 10^{1.645 \times lognormal(-1.127, 0.466)}$$

This adjustment gives us the final distribution of X, which models the probabilistic distribution of 95th percentile-to-50th percentile intrapersonal variability of cancer risk in the general population (Figure B2). Note that the results are bounded from $(0, \infty)$, as the lower limit of the distribution is at $10^{1.645 \times 0} = 1$.

Figure B2. Distribution of 95th Percentile-to-Median Ratios



In this distribution, the expected value (or mean) is 3.93, the 5th percentile is 1.77, and the 95th percentile is 14.03.

B.2 Find Standard Deviation of Population Distribution

Once we sample the above distribution to find a probabilistically generated 95th percentile-to-median ratio, we must plug each sampled value into the lognormal distribution representing population risk variability. **At this point, we are dealing with a new lognormal equation.** The prior equation represented *uncertainty* in our choice of 95th percentile-to-median ratio, while this new equation represents the *population variability* distribution we will actually use to sample population unit risk. This means that we now must define new μ-values and σ-values for this distribution based on our choice of ratio in Step 1. While the values of our uncertainty distribution were written as X, the values of this population distribution will be identified using P.

As shown before, in a lognormal distribution:

$$P_{95} = e^{\mu + \sigma z_{95}}$$

And:

$$P_{50} = e^{\mu}$$

Therefore:

$$\ln(P_{95}) = \ln(P_{50}) + \sigma z_{95}$$

Rewriting this equation to isolate σ yields the following:

$$\sigma = \frac{\ln\left(\frac{P_{95}}{P_{50}}\right)}{z_{95}}$$

Now, the standard deviation of our *population* distribution is a function of the 95th percentile-to-median ratio pulled from our *uncertainty* distribution. We can now use this new standard deviation, along with OEHHA's inhalation unit risk as the distribution's median, to define the population distribution of cancer risk.

B.3 Adjust Population Distribution Using Standard Deviation

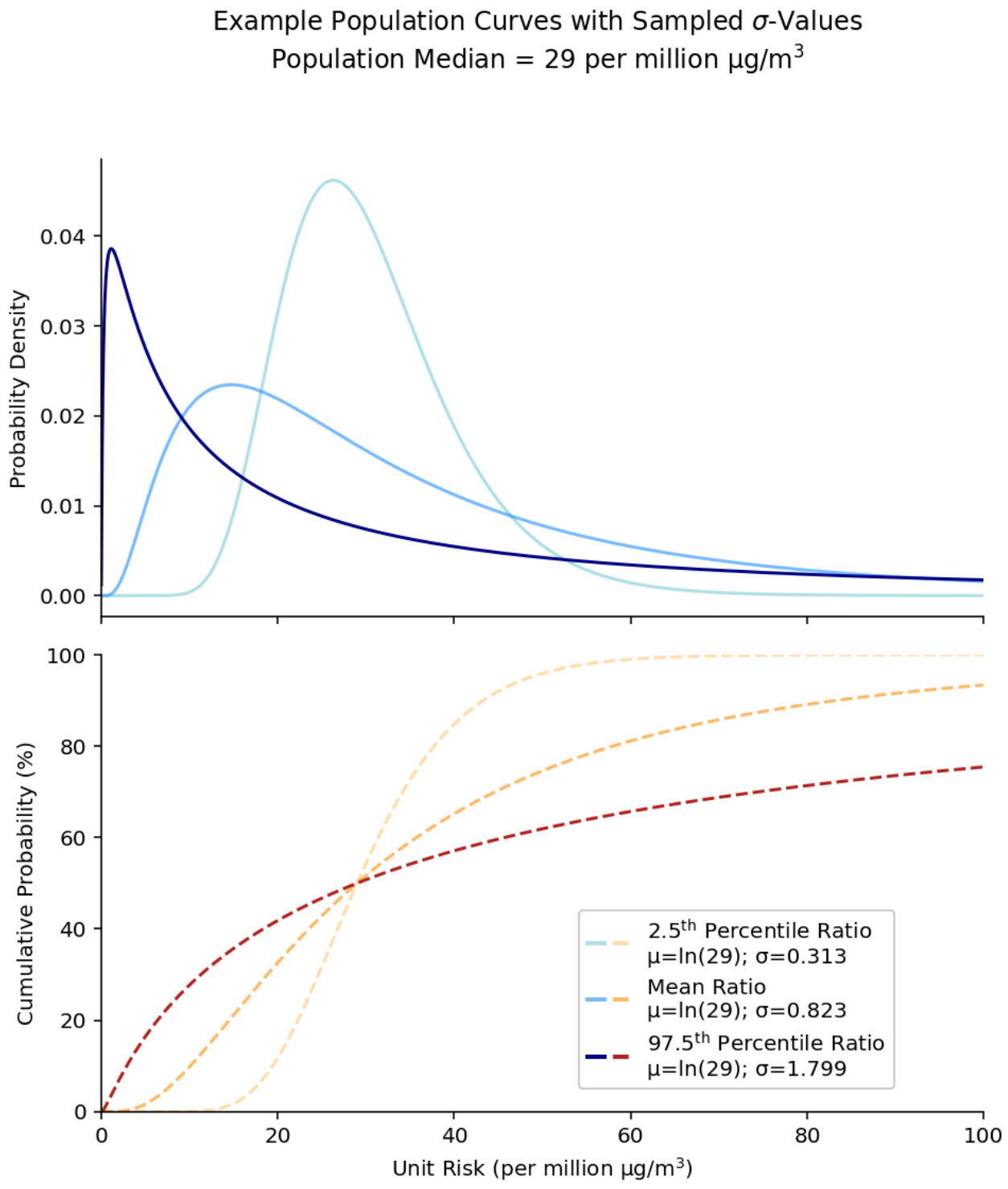
Now, we use OEHHA's IUR, denoted here as IUR, and our previously calculated standard deviation, to define a new lognormal curve representing population variability. Since, in a lognormal distribution, $P_{50} = e^{\mu}$, $\mu = \ln(P_{50}) = \ln(IUR)$. Therefore:

$$\text{Population Unit Risk Distribution} = lognorm\left(\ln(\text{IUR}), \frac{\ln\left(\frac{P_{95}}{P_{50}}\right)}{z_{95}}\right)$$

The following curves demonstrate the effect of modeling population unit risk distributions with different standard deviations. Since we are trying to use OEHHA's IUR estimates as constants, we parameterize three population distribution curves with the same median (and therefore underlying mean, or μ).

As an example, the following graph (Figure B3) shows the results of three different samples from our uncertainty distribution X, the results of which are $\frac{P_{95}}{P_{50}}$ ratios. In this example plot below, we sample from our uncertainty distribution 1,000 times, taking the mean, the 2.5th percentile, and the 95th percentile ratios. (In this specific draw, these values are 5.043, 1.613, and 17.628, respectively.) Using these three different $\frac{P_{95}}{P_{50}}$ draws, we parameterize three different population curves according to the equation above. These results are shown below:

Figure B3. Example Population Cancer Susceptibility Curves using 2.5th Percentile, Mean, and 97.5th Percentile Values for the Population 95th Percentile-to-Median Ratio



In this example, the darkest lines correspond to the curve developed from the 97.5th percentile $\frac{P_{95}}{P_{50}}$ ratio, the lightest lines correspond to the 2.5th percentile $\frac{P_{95}}{P_{50}}$ ratio, and the intermediate darkness lines correspond to the

mean $\frac{P_{95}}{P_{50}}$ ratio. The three CDFs converge at their 50th percentile, which is our chosen OEHHA unit risk value of 2.9 E-5 $(\mu g/m^3)^{-1}$, or 29 per million $\mu g/m^3$.

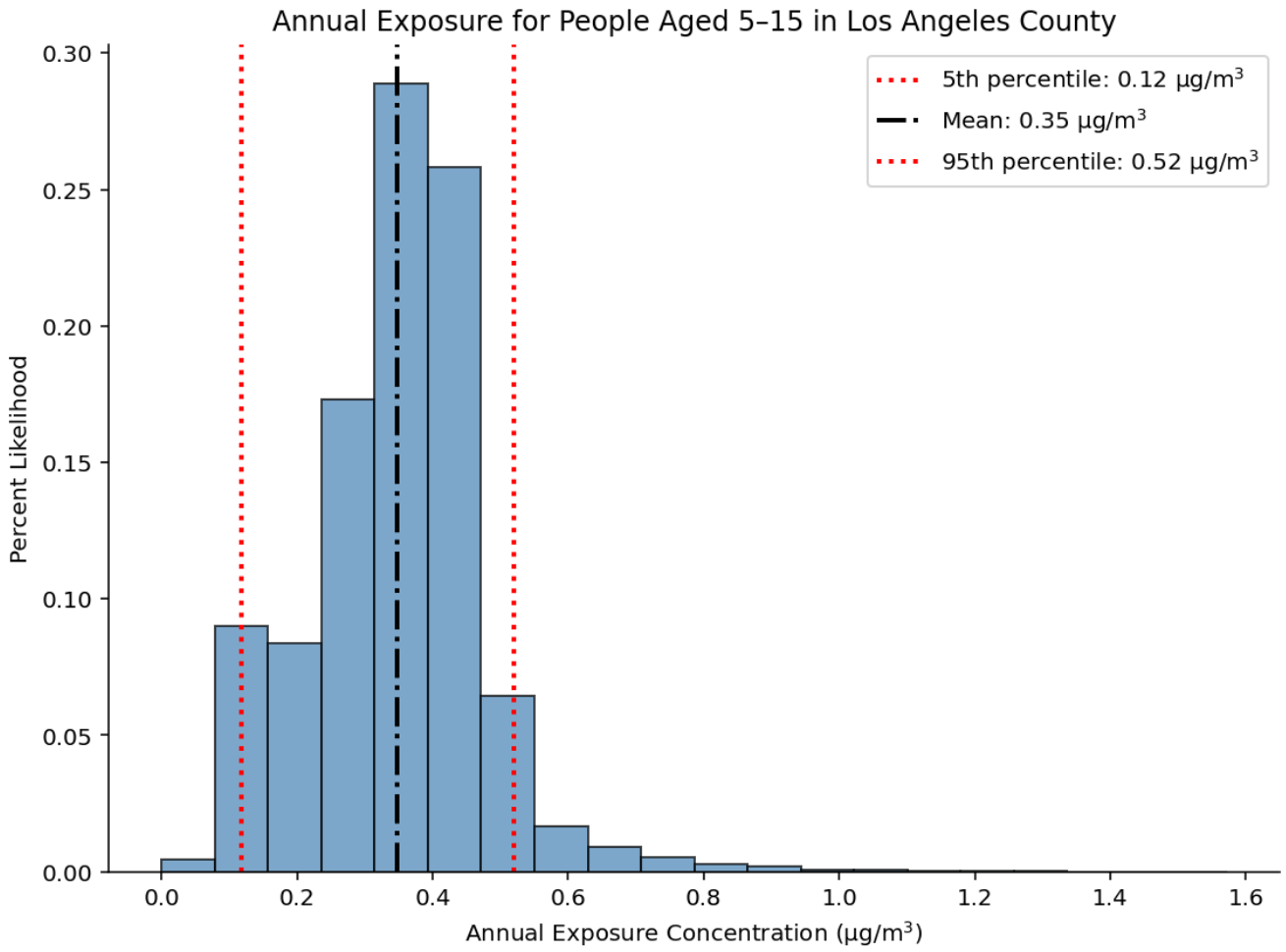
It is important to note that this example does not incorporate any underlying uncertainty in OEHHA's unit risk estimate; that is something we can incorporate later, if CARB expresses their comfort with adding uncertainty to OEHHA's values.

B.4 Final Monte Carlo Example

Now, we combine our distributions of risk uncertainty and variability with our distribution of exposure in a Monte Carlo sampling run (Figure B4). First, we take a county- and age-specific exposure distribution output from HAPEM8. The distribution below reflects uncertainty and variability in exposure for people in a certain age group within a certain county based on ambient pollutant concentrations. These exposures represent example interactions between people and ambient pollution based on daily activity patterns and source locations, for a certain age group in each census tract in a county. The range of exposures approximates the distribution of activity patterns for a given group; however, we do not know if the distribution of exposures we have represents the *actual* distribution of exposure for the group in question. Therefore, we sample from these exposures to reflect our lack of knowledge of specific distribution of activity patterns of people within different age groups in each county in California as well as the general stochasticity in activity levels among people in these groups. We repeat this sampling process to reflect the many and varied activity profiles for a given group in a given year. Thus, sampling from this distribution reflects a somewhat combined measure of uncertainty and variability in our exposure estimates.[25]

[25] This uncertainty is entirely contained within HAPEM8's modeling, which contains a probabilistic element reflective of uncertainty in activity estimations. It does not reflect uncertainty in the air quality surface that is fed into HAPEM8. CARB models air quality changes based on proposed regulatory actions, and CARB will be responsible for considering any uncertainty in those air quality estimates.

Figure B4. Example Benzene Exposure Distribution for 5-15-Year-Olds in LA County



We then combine our three distributions in a final Monte Carlo analysis.

First, we draw 1,000 samples from our distribution of 95th percentile-to-median ratios, characterizing **uncertainty** in the **shape** of the **population distribution.** For each of these 1,000 samples, we follow these steps:

1. We convert each 95th percentile-to-median ratio to a standard deviation value according to the equation $\sigma = \frac{\ln\left(\frac{P_{95}}{P_{50}}\right)}{z_{95}}$.
2. We use this new σ-value to parameterize a population risk distribution with the above standard deviation, and an underlying mean of the following:

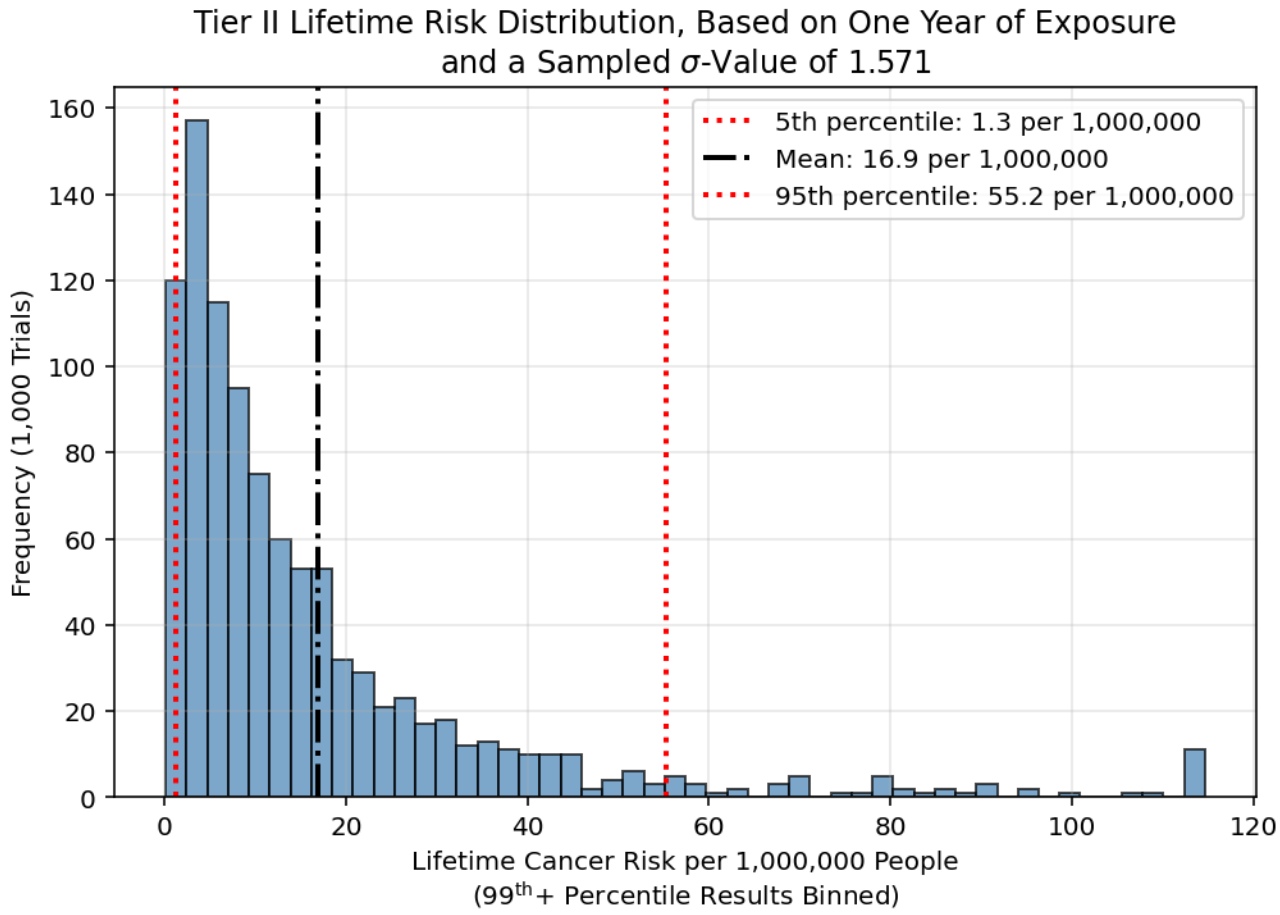
$$\mu = \ln(\text{OEHHA IUR}) = \ln(2.9 \times 10^{-5}) = -10.448$$

3. We draw 1,000 samples from this population distribution. These samples represent interindividual variability in cancer dose-response for a particular value of σ from Step 1.
4. We draw 1,000 samples, with replacement, from our distribution of annual exposure concentrations for the county and demographic group in question. This distribution represents variability within the population as well as uncertainty in the actual activity patterns of residents in each county.

5. The 1,000 risk samples are multiplied by the 1,000 exposure samples, resulting in 1,000 lifetime cancer risk estimates for a given 95th percentile-to-median ratio, county, and demographic group.

Since this process involves 1,000 samples, and we repeat this process 1,000 times, with a different population distribution standard deviation each time, we are left with 1,000 different distributions for lifetime risk for the given county, demographic group, and year of exposure in question. From each distribution, we draw the mean risk. An example distribution is shown in Figure B5:

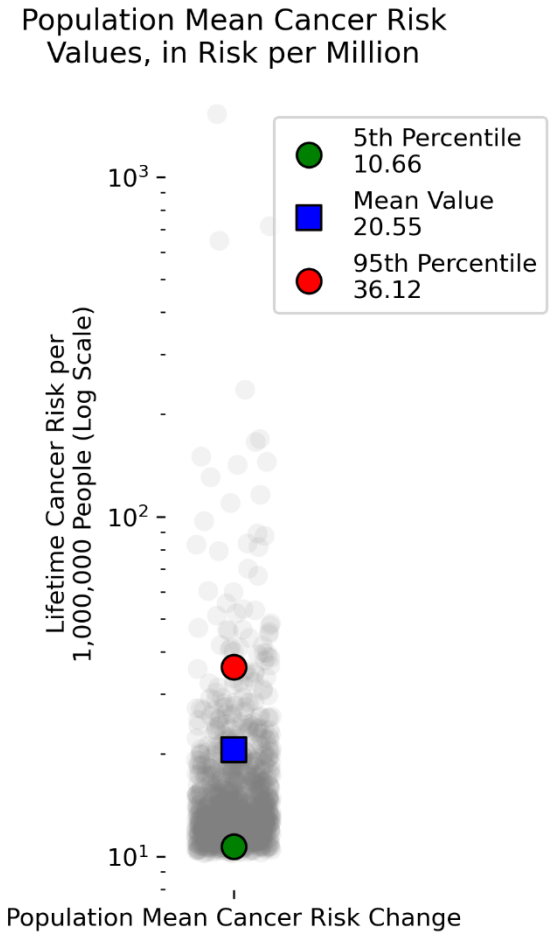
Figure B5. Example Approach II Lifetime Risk Distribution for a Sampled σ-Value of 1.571



We take 1,000 such draws, so that we are left with 1,000 estimates for the population mean risk change in each location.

The following graph (Figure B6) shows our final estimate of mean population risk change, with the 90 percent confidence interval highlighted:

Figure B6. Distribution of Estimates of 5th Percentile, Mean, and 95th Percentile Population Risk Values



Appendix C. Approach I One-in-One Million Weighted Average Cancer Risk Valuation Equation

C.1 Equation for Valuing a One-in-One Million Weighted Average Risk Change

To calculate the weighted average valuation of a one-in-one million cancer risk for a given cancer site, we employ the following expression:

Equation 5. Complete Approach I Equation Valuing a One-in-One Million Weighted Average Risk Change

$$\frac{\sum_{\alpha=a_0}^{a_f}\sum_{\beta=\alpha+lag_\alpha}^{100} p_{\alpha,s}\times d_{\beta,s}\times\left(S_{\beta,s}\frac{VNR\left(\frac{I_{y+\beta-\alpha}}{I_y}\right)^{\varepsilon_{NF}}}{(1+r)^{\beta-\alpha}}+\sum_{k=1}^{20} m_{\beta,s,k}\frac{VMR\left(\frac{I_{y+\beta+k-\alpha}}{I_y}\right)^{\varepsilon_F}}{(1+r)^{\beta+k-\alpha}}\right)}{\sum_{\alpha=a_0}^{a_f}\sum_{\beta=\alpha+lag_\alpha}^{100} p_{\alpha,s}\times d_{\beta,s}}$$

In this equation:

- α is the age of exposure
- a_0 and a_f are the youngest and oldest age considered, respectively
- β is the age of diagnosis
- lag_α is the lag given exposure at age α
- s is the sex of the population
- $y = 2023$, the base year for income growth adjustments for VNR and VMR estimates from LaPenta et al. (2024)
- $p_{\alpha,s}$ is the proportion of risk borne by age group α and sex s
- $d_{\beta,s}$ is the share of lifetime diagnoses that happen in that given diagnosis year β for sex s (this incorporates the lowering factor intrinsically by not normalizing by the total amount of exposure available)
- S_β is the probability of 20-year survival based on diagnosis at age β and sex s
- k is the year since diagnosis
- $m_{\beta,s,k}$ is the probability of dying based on diagnosis at age β, sex s, and number of years since diagnosis k (this incorporates the probability of dying overall by not normalizing by the total probability of death)
- VNR is the value of a one-in-one million non-fatal cancer risk reduction
- VMR is the value of a one-in-one million mortality risk reduction
- $I_{y+\beta-\alpha+2023}/I_y$ is the income growth factor for the specified year, either the year of diagnosis ($y+\beta-\alpha$) or the year of death ($y+\beta+k-\alpha$)
- ε is the income elasticity term, either for non-fatal risks (ε_{NF}) or fatal risks (ε_F)
- r is the discount rate

Considering this equation in terms of its component parts helps to explain the process. The equation contains two valuation expressions, which take the general form:

$$\frac{VMR\left(\frac{I_{y+\beta+k-\alpha}}{I_y}\right)^{\varepsilon_F}}{(1+r)^{\beta+k-\alpha}}$$

This expression represents the following concept, as explained in Sections 3.1.4 and 3.1.5.

$$\frac{VMR(\text{Income Growth})^{\varepsilon}}{(\text{Discounting})}$$

This term, taken holistically, can be thought of as the unit value of a 1/1,000,000 risk change for either mortality or morbidity. The outcome of this term is a dollar value, like $12.00 or $6.50. As we rebuild the equation, we will substitute a dollar sign ($) for this section.

The mortality term, which values and calculates the likelihood of death in the 20-year window following diagnosis, is given in the following form, after substituting a "Fatal$" in for the valuation piece:

$$\sum_{k=1}^{20} m_{\beta,s,k} \times \text{Fatal\$}_{\alpha,\beta,k}$$

This term iterates through each 20-year mortality window, multiplying the probability of dying in that given year since diagnosis (m) by the value of dying in that given year, calculated using the age of exposure (α), the age of diagnosis (β), and the number of years elapsed between diagnosis and death (k).

The morbidity term, which values and calculates the likelihood of a cancer case being non-fatal, takes the following form, after substituting a "Non-fatal$" in for the valuation piece:

$$S_{\beta,s} \times \text{Non} - \text{fatal\$}_{\alpha,\beta}$$

This term multiplies the probability of survival for the entire 20-year mortality window by the value of a non-fatal case.

These two terms are added together, then multiplied by the population proportion and incidence weighting factors, given as follows:

$$p_{\alpha,s} \times d_{\beta,s} \times \left(S_{\beta,s} \times \text{Non} - \text{fatal\$}_{\alpha,\beta} + \sum_{k=1}^{20} m_{\beta,s,k} \times \text{Fatal\$}_{\alpha,\beta,k}\right)$$

This term represents the adjustment of the value (both of mortality and morbidity) of a 1/1,000,000 cancer risk change by **a)** the proportion of the studied population found in that age group (given by p), and **b)** the proportion of diagnoses for that population group that fall at that age (given by d).

To illustrate this point, consider a study group of 20–29-year-olds of both genders. Let us assume the population is evenly distributed across these ten ages and across both genders, so 25-year-old women are $1/20^{th}$, or 5 percent, of the studied population. Let's say that for women, 10 percent of diagnoses happen at age 60. In this instance, each of the twenty age-sex combinations in the study group will have 5 percent of the risk apportioned to them as given by $p_{\alpha,s}$. Then, as we iterate through all of the possible ages of diagnosis for someone aged 25, we consider the proportion of diagnoses that happen in each age. Let's say the likelihood of a cancer diagnosis

for a woman occurring at age 60 is 10 percent, or 1 in 10 cases for that particular cancer in women are diagnosed at age 60. We combine these two terms to weight the valuation of a 1/1,000,000 risk change for an individual who is a 25-year-old woman. Thus, the value of a 1/1,000,000 cancer risk at age 60 for women exposed at age 25 is multiplied by a factor of $5\% \times 10\% = 0.5\%$, or 0.005, as this age of diagnosis, age of exposure, and sex combination contains 0.5 percent of the total 1/1,000,000 risk to the group of 20–29-year-olds studied.

Finally, we calculate this term for every age of exposure, α, and age of diagnosis, β, iterating through both:

$$\sum_{\alpha=a_0}^{a_f} \sum_{\beta=\alpha+lag_\alpha}^{100} p_{\alpha,s} \times d_{\beta,s} \left(S_{\beta,s} \times \text{Non} - \text{fatal}\$_{\alpha,\beta} + \sum_{k=1}^{20} \left(m_{\beta,s,k} \times \text{Fatal}\$_{\alpha,\beta,k} \right) \right)$$

We divide these values by the sum of just the weight of diagnosis year and incidence ($p_{\alpha,s} \times d_{\beta,s}$) calculated for each age of exposure and diagnosis to turn our numerator's sum into a weighted average value.

Thus, our final equation aggregates the diagnosis- and population-weighted value of a 1/1,000,000 risk reduction (portioned into fatal and non-fatal valuation) and divides by the diagnosis and population weights to create a weighted average value for a 1/1,000,000 risk change to a population from age a_0 to age a_f.

Appendix D. Development of Pooled Distribution for Non-Fatal WTP Estimates

To provide CARB with a single non-fatal WTP estimate for cancer sites without supporting economic research, IEc developed a pooled distribution using a selection of the non-fatal WTP estimates from the LaPenta et al. (2024) literature review discussed in Section 3.1.3.

The sections below describe the derivation of a confidence interval around the mean value for each study. Methods vary due to differences in study design. Values were adjusted for inflation (to 2023$) and real income growth.[26] All values are represented as one-in-one million reductions in cancer risk.

D.1 Bosworth et al. (2009)

Bosworth et al. provide 5th, 50th, and 95th percentiles of WTP for reductions in various non-fatal cancer risks. The WTP values for each health outcome are ratios of maximum likelihood estimates. Because maximum likelihood estimators are asymptotically normal, the WTP values represent a ratio of two normally distributed variables. While the exact distribution of this ratio is not normal, the asymptotic distribution is normal, if the denominator of the ratio does not go to zero. Given that the denominator is marginal utility of income, we assume that the denominator is bounded away from zero and that the sampling distributions of the estimated WTP values are approximately normal in large samples.

Assuming a normal distribution, we calculate the standard error using the following formula:

$$SE = \frac{(P_{95} - P_5)}{2 * 1.645}$$

Where:

- SE = standard error
- P_{95} = 95th percentile
- P_5 = 5th percentile

After adjusting for inflation and income growth and converting the estimate to a one-in-one million reduction in risk, the result is a 90 percent confidence interval surround the mean WTP value for each health effect.

Table D1. Confidence Intervals Derived for Bosworth et al. (2008)

Health effect	Standard Error	90% Confidence Interval (2023$)
General Cancer	0.49	$0.46 (-0.03, 0.95)
Leukemia	0.62	$1.44 (0.82, 2.06)
Colon/Bladder	0.59	$0.75 (0.16, 1.34)
Lung	0.62	$1.58 (0.96, 2.20)

[26] Values were adjusted by LaPenta et al. for inflation using the Consumer Price Index and then adjusted for income growth using real GDP per capita and EPA's recommended income elasticity of 0.45 for severe illness.

D.2 Gerking et al. (2014)

Gerking et al. (2014) estimates the mean value for a one-in-ten thousand risk reduction in non-fatal leukemia as $9.62 (2008$). The standard error around this mean value is provided (0.57), allowing for the calculation of a 90 percent confidence interval around the mean using the following formula:

$$CI = \mu \pm 1.645 * SE$$

Where:

- CI = confidence interval
- μ = mean
- SE = standard error

Applying the values from Gerking et al.:

$$CI = 9.62 \pm 1.645 * 0.57$$

$$CI = 9.62 \pm 0.938$$

After adjusting for inflation and income growth and converting the estimate to a one-in-one million reduction in risk, the result is a 90 percent confidence interval surrounding a mean value of $0.15 (0.14, 0.17) (2023$).

D.3 Magat et al. (1996)

Magat et al. (1996) reported a median-based estimate of the risk-risk tradeoff for non-fatal lymphoma of 0.583 but did not report an estimated mean or variance. This section derives an estimate of the mean (0.327) and variance (0.987) of the risk-risk tradeoff for non-fatal lymphoma. Based on a sample size of 716, the 95 percent confidence interval for the mean is (0.255, 0.400).

Equation (7) in Magat et al., rewritten here as Equation 6, shows the linear relationship among the risk-risk tradeoffs for non-fatal, curable, and fatal lymphoma:

Equation 6

$$R_{NF} = \left(\frac{1}{1-z}\right) R_c + \left(\frac{-z}{1-z}\right) R_F$$

Where:

- R_{NF} = Risk-risk tradeoff for non-fatal lymphoma
- R_c = Risk-risk tradeoff for curable lymphoma
- R_F = Risk-risk tradeoff for fata lymphoma
- z = Assumed probability that lymphoma is fatal

Z is the assumed probability that lymphoma is fatal and set equal to 0.1. Equation 6 implies that the mean and variance of the risk-risk tradeoff for non-fatal lymphoma are:

Equation 7

$$E[R_{NF}] = \left(\frac{1}{1-z}\right) E[R_C] + \left(\frac{-z}{1-z}\right) E[R_F]$$

Equation 8

$$Var(R_{NF}) = \left(\frac{1}{1-z}\right)^2 Var(R_C) + \left(\frac{z}{1-z}\right)^2 Var(R_F) - 2\left(\frac{1}{1-z}\right)\left(\frac{z}{1-z}\right) Cov(R_C, R_F)$$

Where:

- $E[\cdot]$ = Expected value or population mean
- $Var(\cdot) =$ Population variance
- $Cov(\cdot) =$ Population covariance

Magat et al. computed the risk-risk tradeoffs R_C and R_F from the inverses of the tradeoffs that were elicited from survey respondents. The corresponding risk-risk tradeoffs were computed as:

Equation 9

$$R_C = \frac{1}{X_C}, \qquad X_C > 0, \quad R_F = \frac{1}{X_F}, \qquad X_F > 0$$

Where:

- $X_C =$ Inverse tradeoffs for curable lymphoma
- $X_F =$ Inverse tradeoffs for fatal lymphoma

Magat et al. also computed the ratio of the risk-risk tradeoffs, $\frac{R_C}{R_F}$ as:

Equation 10

$$\frac{R_C}{R_F} = \frac{X_F}{X_C}, \qquad X_C > 0$$

The mean and variance of the risk-risk tradeoff for non-fatal lymphoma in Equations 7 and 8 depend on means, variances, and covariances of risk-risk tradeoffs for curable and fatal lymphoma. Although Magat et al. did not report estimates of means, variances or covariances of risk-risk tradeoffs, we derive estimates from statistics reported for inverse tradeoffs (Magat et al. Table 1). The derivations repeatedly use the continuous mapping theorem and delta method.

The continuous mapping theorem (e.g., Hansen, *Econometrics* (2022), pp. 161-162) implies that a continuous function of population parameters can be estimated consistently by evaluating the function at consistent estimators of the parameters. The functions considered here are continuous, and sample means, variances and covariances are consistent estimators of corresponding population parameters. The delta method approximates the variance of a nonlinear function of random variables; see Greene *Econometric Analysis* (2018), Appendix D, or Hansen, *Econometrics* (2022), pp. 161-162. It is based on a first-order Taylor's series expansion around the means of the random variables.

The mean risk-risk tradeoff for non-fatal lymphoma is estimated in two steps.

M1. Estimate mean risk-risk tradeoffs for curable and non-fatal lymphoma based on sample means of the inverse tradeoffs presented by Magat et al.

M2. Estimate the mean risk-risk tradeoff for non-fatal lymphoma using Equation 7 and the means estimated in of step M1.

M1. Estimate mean risk-risk tradeoffs for curable and fatal lymphoma

The continuous mapping theorem implies that the population means of R_C and R_F can be estimated consistently by evaluating Equation 9 at the sample means of X_C and X_F . Using sample means reported by Magat et al. (Table 1) of 2.775 forX_C and 1.523 for X_F, the estimated population means are $\frac{1}{2.775} = 0.360$ for R_C and $\frac{1}{1.523} = 0.657$ for R_F.

For comparison, Magat et al. (Table 1) report sample median values of 1.600 for $X_C$and 1.000 for X_F, implying median risk-risk tradeoffs of $\frac{1}{1.600} = 0.625$ for R_C and 1.000 for R_F.

M2. Estimate the mean risk-risk tradeoff for non-fatal lymphoma

With consistent estimators of the population means of R_C and R_F from step M1, the population mean of the risk-risk tradeoff for non-fatal lymphoma is estimated using Equation 7 as:

$$\hat{E}[R_{NF}] = \left(\frac{1}{0.9}\right)0.360 + \left(\frac{-0.1}{0.9}\right)0.657 = 0.327$$

Where:

- $\hat{E}[\cdot]$ = estimated mean

For comparison, Magat et al. applied Equation 6 using sample median values of R_C and R_F to obtain a median-based estimate of the risk-risk tradeoff for non-fatal lymphoma of

$$\left(\frac{1}{0.9}\right)0.625 - \left(\frac{0.1}{0.9}\right)1.000 = 0.583$$

The variance of risk-risk tradeoffs for non-fatal lymphoma is estimated in five steps.

V1. Compute sample variances of the inverse tradeoffs based on the standard errors of the means reported by Magat et al.

V2. Estimate the variances of the risk-risk tradeoffs for curable and non-fatal lymphoma by applying the delta method to Equation 9 and using the results of step V1.

V3. Compute the sample variance of the ratio of risk-risk tradeoffs $\frac{R_C}{R_F}$ based on the standard error of the mean reported by Magat et al.

V4. Estimate the covariance between R_C and R_F based on the continuous mapping theorem and delta method applied to Equation 10 and the variance of the ratio computed in step V4.

V5. Estimate the variance of the risk-risk tradeoff for non-fatal lymphoma based on Equation 8 using the variances and covariances of R_C and R_F estimated in steps V2 and V4.

V1. Compute the variances of the inverse risk-risk tradeoffs

Using standard errors of mean inverse tradeoffs reported in Table 1 by Magat et al., sample variances are:

$$\widehat{Var}(X_C) = N \times [semean(X_C)]^2 = 719 \times (0.214)^2 = 32.927,$$

$$\widehat{Var}(X_F) = N \times [semean(X_F)]^2 = 724 \times (0.129)^2 = 12.048$$

Where:

- $\widehat{Var}(\cdot)$ = estimated variance
- N = sample size
- $semean(\cdot)$ = standard error of the mean

V2. Estimate the variances of the risk-risk tradeoffs for curable and fatal lymphoma

Applying the delta method to Equation 9, the approximate variances of R_C and R_F are:

Equation 11

$$Var(R_C) \cong \left(\frac{1}{E[X_C]}\right)^4 Var(X_C), \qquad Var(R_F) \cong \left(\frac{1}{E[X_F]}\right)^4 Var(X_F)$$

Applying the continuous mapping theorem and using the sample means (Magat et al. Table 1) and sample variances (step V1) of X_C and X_F, the estimated variances of the risk-risk tradeoffs are:

Equation 12

$$\widehat{Var}(R_C) = \left(\frac{1}{2.775}\right)^4 \times 32.927 = 0.555, \widehat{Var}(R_F) = \left(\frac{1}{1.523}\right)^4 \times 12.048 = 2.239$$

V3. Compute the sample variance of the ratio of risk-risk tradeoffs

Using the standard error of the mean of the ratio $\frac{R_C}{R_F}$ reported in Table 1 by Magat et al., the sample variance of the ratio is:

Equation 13

$$\widehat{Var}\left(\frac{R_C}{R_F}\right) = N \times \left[seman\left(\frac{R_C}{R_F}\right)\right]^2 = 716 \times (0.089)^2 = 5.671$$

V4. Estimate the covariance between risk-risk tradeoffs for curable and fatal lymphoma

The covariance between R_C and R_F can be computed using the variance of the ratio $\frac{R_C}{R_F}$. Applying the delta method to the ratio, its variance is approximately equal to

Equation 14

$$Var\left(\frac{R_C}{R_F}\right) \cong \frac{1}{E[R_F]^2} Var(R_C) + \frac{E[R_C]^2}{E[R_F]^4} Var(R_F) - 2\frac{E[R_C]}{E[R_F]^3} Cov(R_C, R_F)$$

Solving Equation 14 for $Cov(R_C, R_F)$ gives:

Equation 15

$$Cov(R_C, R_F) = \frac{1}{2}\left(\frac{E[R_F]}{E[R_C]}Var(R_C) + \frac{E[R_C]}{E[R_F]}Var(R_F) - \frac{E[R_F]^3}{E[R_C]}Var\left(\frac{R_C}{R_F}\right)\right)$$

Using continuous mapping and consistent estimators of the means and variances on the right-hand side of Equation 15 the estimated covariance between R_C and R_F equals:

Equation 16

$$\widehat{Cov}(R_C, R_F) = \frac{1}{2}\left(\frac{0.657}{0.360}(0.555) + \frac{0.360}{0.657}(2.239) - \frac{0.657^3}{0.360}(5.671)\right) = -1.107$$

Where:

- $\widehat{Cov}(\cdot)$ = estimated covariance

V5. Estimate the variance of the risk-risk tradeoff for non-fatal lymphoma

Plugging consistent estimators of the variances and covariance in the right-hand side of Equation 8 yields a consistent estimator of the population variance of the risk-risk tradeoff for non-fatal lymphoma:

Equation 17

$$\widehat{Var}(R_{NF}) = \left(\frac{1}{1-z}\right)^2 \widehat{Var}(R_C) + \left(\frac{z}{1-z}\right)^2 \widehat{Var}(R_F) - 2\left(\frac{1}{1-z}\right)\left(\frac{z}{1-z}\right)\widehat{Cov}(R_C, R_F)$$

Using the estimates obtained in Equations 12 and 15, the estimated variance equals

$$\widehat{Var}(R_{NF}) = \left(\frac{1}{0.9}\right)^2 0.555 + \left(\frac{0.1}{0.9}\right)^2 2.239 + 2\left(\frac{1}{0.9}\right)\left(\frac{0.1}{0.9}\right)1.107 = 0.987$$

The estimated variance of R_{NF} lies between the estimated variances of R_C and R_F. It is about 1.8 times the estimated variance of R_C and about 0.4 times the estimated variance of R_F.

Using a sample size of 716, the standard error of the mean of the risk-risk tradeoff for non-fatal lymphoma is $\sqrt{\frac{0.987}{716}} = 0.037$ with a mean value of 0.327.

Then, we multiply by CA VSL to get final one-in-one million risk reduction where $0.327 \times \$14{,}030{,}101 = \$4.59$. The result is a 90 percent confidence interval surrounding a mean value of \$4.59 (\$4.06, \$5.12) (\$2023).

D.4 Viscusi et al. (1991)

Viscusi et al. (1991) estimate the mean value of a statistical case of chronic bronchitis as \$883,000 (1990\$). The standard error around this mean value is provided (114,000), allowing for the calculation of a 90 percent confidence interval around the mean using the following formula:

$$CI = \mu \pm 1.645 * SE$$

Where:

- CI = confidence interval
- μ = mean
- SE = standard error

Applying the values from Viscusi et al.:

$$CI = 883{,}000 \pm 1.645 * 114{,}000$$

$$CI = 883{,}000 \pm 187{,}530$$

After adjusting for inflation and income growth and converting the estimate to a one-in-one million reduction in risk, the result is a 90 percent confidence interval surrounding a mean value of $2.58 (1.93, 3.24) (2023$).

D.5 Leukemia Distribution

To estimate a distribution for the VNR for non-fatal case of leukemia, we have two separate estimates available, one from Bosworth et al. (2009) and the other from Gerking et al. (2014). To simplify the valuation process, we opted to pool these two estimates into a single non-fatal leukemia valuation distribution, using study-specific weights. We generate weights following the approach described in Appendix K to the User Manual for the desktop BenMAP-CE application. As noted in that Appendix, weighting methods based on those originally proposed by DerSimonian and Laird (1986) can be applied to study results informing both incidence and valuation calculations for regulatory impact analyses. The weights are based on within-study variance under a fixed-effects model, which assumes both studies are estimating the same true value, or based on both within- and between-study variance under a random-effects model that relaxes this assumption. We first calculate a test statistic, Q_w, to test the fixed-effect hypothesis. Our Q_w value exceeded the critical value for a 5 percent one-tailed test, thus we rejected the fixed-effect hypothesis and calculated the following random-effects weights: $w_b = 0.44$ for the Bosworth study and $w_g = 0.56$ for the Gerking study. The result is a weighted distribution with a 90 percent confidence interval surrounding a central estimate VNR of $0.72 (-$0.60, $2.03).

Appendix E. HAPEM8 Exposure Modeling for Approaches II and III

MEMORANDUM

TO: Eric Ruder, IEc, Inc.
FROM: Steve Fudge, SC&A Inc.
DATE: January 28, 2026
SUBJECT: Case Study Modeling Support

This memorandum documents the steps taken to model California Formaldehyde and Diesel Particulate Matter (DPM) air concentrations in the Hazardous Air Pollutant Exposure Model version 8 (HAPEM8) model. This work was done in support of Task 3 (Case Study Modeling Support) of the Air Toxic Pollutants Associated Cancer Health Monetization Research Project (Modification 1).

For this task, SC&A ran HAPEM8 for two different scenarios: 1) Formaldehyde concentrations in California, and 2) Diesel Particulate Matter concentrations in California. The air quality data used in both modeling scenarios came from the U.S. EPA's 2020 AirToxScreen (ATS) assessment. The DPM air quality data was provided by the U.S. EPA for the entire nation in a format ready for input into HAPEM8, i.e. data by Census tract, grouped by point, nonpoint, onroad, and nonroad sources, and temporally allocated to 8 daily 3-hour time periods. SC&A simply extracted the Census tracts in California from this national file for input to HAPEM8.

Formaldehyde air concentrations from the 2020 ATS were not available in a HAPEM8-ready format. What was available were pollutant specific files containing annual air concentrations broken out by emission sources and at the Census block resolution. The processing of this data to prepare it for input into HAPEM8 is described below.

Formaldehyde Data Processing

Formaldehyde air concentrations used as air quality input to HAPEM8 were derived from the 2020 ATS assessment. The annual, block-level air concentrations can be downloaded from https://gaftp.epa.gov/rtrmodeling_public/AirToxScreen/2020/Pollutant%20Summaries/Region1/ by selecting the FORMALDEHYDE_AMBCONC.zip file. The HAPEM8 air quality input file must be at the Census tract resolution, data grouped by point, nonpoint, onroad, and nonroad, and the air concentrations temporally allocated to 8 3-hour time periods. The following steps were performed to format the annual ATS data for use in HAPEM8.

1. Group data by point, nonpoint, onroad, and nonroad.
    a. Point is made up of just the point source
    b. Nonpoint consists of:
        i. NP-industrial
        ii. NP-CommercialCooking
        iii. NP-OilGas
        iv. NP-SolventsCoatings
        v. NP-StorageTransfer_BulkTerminals_GasStage1
        vi. NP-MiscellaneousNonindustrial
        vii. NP-FuelCombustion_not_RWC
        viii. NP-ResidentialWoodCombustionRWC
        ix. NP-WasteDisposal
        x. NP-AgricultureLivestock (Waste)
        xi. NP-AgricultureLivestock (Silage)
    c. Onroad consists of:
        i. OR-LightDuty-OffNetwork-Gas
        ii. OR-LightDuty-OffNetwork-Diesel
        iii. OR-HeavyDuty-OffNetwork-Gas
        iv. OR-HeavyDuty-OffNetwork-Diesel
        v. OR-LightDuty-OnNetwork-Gas
        vi. OR-LightDuty-OnNetwork-Diesel
        vii. OR-HeavyDuty-OnNetwork-Gas
        viii. OR-HeavyDuty-OnNetwork-Diesel
        ix. OR-Refueling
        x. OR-HeavyDuty-Hoteling
    d. Nonroad consists of:
        i. NR-Recreational-inc-PleasureCraft
        ii. NR-Construction
        iii. NR-CommercialLawnGarden
        iv. NR-ResidentialLawnGarden
        v. NR-Agriculture
        vi. NR-CommercialEquipment
        vii. NR-All Other
        viii. NR-CMV_C1C2_ports
        ix. NR-CMV_C3_ports
        x. NR-CMV_C1C2C3_underway
        xi. NR-Locomotives
        xii. NR-Point-Airports
        xiii. NR- Point-Railyards

2. Allocate block-level concentrations to tract-level by population weighted averaging. See Appendix A for an example calculation.
3. Temporally allocate annual concentrations to 8 daily 3-hour time blocks using EPA's temporal factors. See Appendix B for information about these factors and how they were applied.

HAPEM8 Data Sources

HAPEM8 uses four primary sources of information: population data from the U.S. Census Bureau (census), population activity data, air quality data, and Microenvironment (ME) data. The population activity data is made up of: activity-pattern data from the U.S. Environmental Protection Agency (EPA) Consolidated Human Activity Database (CHAD), commuting-flow data, commuting-time data, and commuting-fraction data. Specifics of these datasets are listed below.

1. Tract-level population data from the 2020 Census,
2. Air quality data from the 2020 ATS,
3. Population activity data from the U.S. EPA Consolidated Human Activity Database (CHAD) (https://www.epa.gov/fera/consolidated-human-activity-database-chad),
4. Commuting-flow data derived from the 2012–2016 U.S. Census Bureau American Community Survey (ACS) (https://transportation.org/census-transportation-solutions/datasets/previous-datasets/2012-2016-ctpp/),
5. Default commuting-time file derived from the 2016–2020 ACS (File is part of the HAPEM8 install package. See HAPEM8 User's Guide pgs. A-11 – A-12),
6. Default commuting-fraction file derived from the 2016-2020 ACS (File is part of the HAPEM8 install package. See HAPEM8 User's Guide pgs. A-14 – A-15).

HAPEM8 Settings

Default HAPEM8 settings were used for both the DPM and Formaldehyde model runs. HAPEM8 uses two parameter input files that direct how the five executable modules that comprise the HAPEM8 model are to be run. The contents of the two parameter files are shown below for the DPM model run. The only difference between the DPM and Formaldehyde model runs were the air quality, factors, and mobiles input files. The Formaldehyde run used a factors file named "factors_gas_HAPEM8.txt" and a mobiles file named "factors_OnroadMobile_Benzene_HAPEM8.txt".

DPM Parameter #1 file

```
INPUT FILES:
  activity file    = input/activity pattern/durhw_HAPEM8.txt
  cluster file      = input/activity pattern/cluster_HAPEM8.txt
  population file  = input/population/population_HAPEM8.txt
  commuting file   = input/commute/commute_flow_HAPEM8.txt
  CommutTime file  = input/others/commute_time_HAPEM8.txt
  CommutFrac file  = input/others/Commute_fraction_HAPEM8.txt
  DistToRoad file  = input/others/proximity_road_HAPEM8.txt
  statefip file    = input/FIPS_StateCrosswalk_HAPEM8.dat

OUTPUT FILES:
  log file         = output/carb_dpm_log_file.txt
  counter file     = output/carb_dpm_counter.dat
  mistract file    = output/carb_dpm_mistract.dat

PARAMETER SETTINGS:
  keep       = YES
  region1    = 5
  region2    = 5
  nmicro   = 18  ! Number of microenvironments
  nblock   = 24  ! Number of time blocks/day in CHAD file
  hblock   = 8       ! Number of time blocks/day in
  ntype    = 3       ! Number of day types
  ngroup   = 6         ! Number of demographic groups
```

DPM Parameter #2 file

```
INPUT FILES:
  activity file    = input/activity pattern/durhw_HAPEM8.txt
  ClusTrans file   = input/Activity Pattern/clustrans_HAPEM8.txt
  population file  = input/population/population_HAPEM8.txt
  commuting file   = input/commute/commute_flow_HAPEM8.txt
  air quality file= input/airqual/carb_HAPEM8_airqual_ats2020_dpm.txt
  factors file = input/factor/factors_particulate_HAPEM8.txt
  mobiles file = input/factor/factors_OnroadMobile_Diesel_HAPEM8.txt
  CommutTime file  = input/others/commute_time_HAPEM8.txt
  DistToRoad file  = input/others/proximity_road_HAPEM8.txt
  statefip file    = input/FIPS_StateCrosswalk_HAPEM8.dat

  product file Pathname   = input/Add/
  AutoPduct file  = input/Add/AutoGarage.txt

Demographic Groups:
```

```
  B_00  =  Ages 0-1
  B_02  =  Ages 2-4
  B_05  =  Ages 5-15
  B_16  =  Ages 16-17
  B_18  =  Ages 18-64
  B_65  =  Ages >= 65

OUTPUT FILES:
  log file         = output/carb_dpm_log_file.txt
  counter file   = output/carb_dpm_counter.dat
  mistract file  = output/carb_dpm_mistract.dat
  afile             = output/
  bfile             = output/
  Keep intermediate files = YES

PARAMETER SETTINGS:

  pollutant  = DPM
  CAS        = 99999
  units      = ug/m3
  year       = 2020
  region1    = 5
  region2    = 5
  EPA Region = 9
  sarod      = 99999

  Rseed1 = -10 ! Random Seed (Negative) for Selecting Activity
Pattern Data
  Rseed2 = -1  ! Random Seed (Negative) for Selecting Micro Factors
  Rseed3 = -1  ! Random Seed (Negative) for Selecting Air Quality
Dataset

    backg    = 0.0
    nmicro   = 18        ! Number of microenvironments
    nblock   = 24        ! Number of time blocks/day in CHAD file
    hblock   = 8     ! Number of time blocks/day in airqual file
    ntype    = 3     ! Number of day types
    ngroup   = 6     ! Number of demographic groups
    nsource  = 4     ! Number of source categories
    nmobiles = 3         ! Sequence # of source categories which are
on-road mobile sources
    nbmicro  = 1     ! beginning # of micros which are indoor
environments
    nemicro  = 10        ! ending # of micros which are indoor
environments
    nvehicles= 7 12      ! Sequence # of micros which are cars/trucks
used in commuting
```

```
    npublict = 8 10 11 13    ! Sequence # of micros which are public
transit (bus, train, and metro)
    nreplic  = 30        ! Number of replicates/demo group in output
file
```

HAPEM8 Limitations

HAPEM8 calculates long-term average exposure concentrations in order to address exposures to HAPs with carcinogenic and other long-term effects. Thus, HAPEM8 does not preserve the time-sequence of exposure events when sampling from the time-activity databases. The result is that information used to evaluate possible correlations in exposures to different HAPs due to activities that are related in time is not preserved. HAPEM8 only estimates exposures experienced through inhalation. For certain HAPs, inhalation might not be the major route of exposure, and, therefore, HAPEM8 may underestimate exposures in these instances. Also, although HAPEM8 is an inhalation-exposure model, it does not include any measures of the ventilation rate associated with an activity, so there is no ability to calculate the potential dose received when engaging in various activities.

Uncertainty in the prediction distributions is not addressed. Some of the uncertainties are as follows.

- The activity-pattern data are limited. Only three of the 23 studies in the version of CHAD used for the current HAPEM8 were national in scope (with several other studies covering multiple metropolitan areas or state-scale); therefore, the combined dataset does not constitute a representative sample, at least with respect to geographic region.
- Commuting-pattern data address only home-to-work travel. The population not employed outside the home is assumed to always remain in the residential census tract.
- Further, although several of the HAPEM8 MEs account for time spent in travel, the travel is assumed to always occur either in the home or work tract. No provision is made for the possibility of passing through other tracts during travel.
- The ME penetration factor distributions incorporated into the current HAPEM8 were derived from reported measurement studies. The data available were quite limited. As a result, most factors were not derived from a representative sample of measurements, and many were inferred from measurements of different HAPs and/or MEs that would be expected to be similar. In addition, the derivation of the penetration factors assumed that measured indoor:outdoor ratios of 1.0 or less indicate the absence of indoor emission sources. Because this assumption is unlikely

to be uniformly valid, penetration factors are likely to overestimate penetration by some unknown amount.

- The ME proximity factor distributions incorporated into the current HAPEM8 for the onroad-vehicle source category were derived from modeling studies for Portland, Oregon. They are subject to the standard uncertainties of air-dispersion modeling. They also are subject to the uncertainties of extrapolating from the traffic patterns of Portland to other locations.
- Air-quality data from modeling studies are uncertain, due to simplifications incorporated into modeling algorithms and limitations of input data (e.g., emissions, meteorology). Air-quality measurements also are uncertain due to limitations of measurement technology (e.g., minimum detection limits) and unknown representativeness of monitoring locations.

Appendix A

Averaging Census Block Concentrations to Tract

The following formula is used to convert Census block level concentrations to Census tract level concentrations using population weighting.

$$C_T = \frac{\sum_1^n (C_i * P_i)}{\sum_1^n P_i}$$

Where :
C_T= The population-weighted average concentration for the Tract
C_i= The air concentration in block i
P_i = The population in block i
n = Total number of blocks within the tract

Example
In this example, California Census tract 06001403701 contains 7 populated blocks as reported in the 2020 Census. The 2020 ATS point source Formaldehyde air concentrations and 2020 Census population in those blocks are :

Block	Air Concentration (ug/m3)	Population
060014037011000	0.00796381	791
060014037011003	0.00821559	53
060014037011005	0.00760816	435
060014037012000	0.00769452	564
060014037012001	0.00783564	301
060014037012002	0.00749612	336
060014037012003	0.00735149	306
	Tract population	2,786

Using the block to tract formula and the data in the table, tract 06001403701 has a population weighted average air concentration of 0.007721048 ug/m3.

Appendix B

Temporal Allocation of Tract Concentrations

HAPEM8 typically uses annual average, diurnally distributed ambient air concentrations as input data. SC&A used USEPA temporal factors to temporally allocate the annual 2020 ATS Formaldehyde air concentrations to 8 3-hour diurnal time blocks (e.g., 12-2 AM, 3-5 AM, 6-8 AM, etc.). The USEPA derived these temporal factors using factors developed from sector specific diurnal emission profiles (e.g. point, onroad, nonroad, and nonpoint).

Table 1 provides the temporal factors for point, nonpoint, onroad, and nonroad sources that the USEPA used in the preparation of the 2020 ATS. SC&A used these factors to allocate the annual tract-level air concentrations into 8 3-hour time blocks. This was done by taking the sector specific (e.g. point) 2020 ATS Formaldhyde concentrations and for each Census tract in the data multiplying the annual air concentration by each of the 8 temporal factors from the appropriate sector specific table. The result is tract-level, diurnally distributed air concentrations ready for input into HAPEM8.

Table 1. Diurnal Factors

Category	Factor Basis	Factors for First Time Period	Factors for Second Time Period	Factors for Third Time Period	Factors for Fourth Time Period	Factors for Fifth Time Period	Factors for Sixth Time Period	Factors for Seventh Time Period	Factors for Eighth Time Period
Point	Domain Average	1.391879	1.388972	0.935441	0.448263	0.368923	0.809512	1.313447	1.343562
Nonpoint	Domain Average	0.73184704	0.84424737	0.891063038	0.590145051	0.450037061	1.343070606	2.022987773	1.126602068
Onroad	Domain Average	0.411883	0.658735	1.685817	0.96586	0.589002	1.40834	1.504065	0.776297
Nonroad	Domain Average	0.885817614	0.997886981	1.058653918	0.648037549	0.523703993	1.230596884	1.534267066	1.121035995