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$PM_{2.5}$-specific Reductions in Life Expectancy Across Two Time Periods and Identification of Race-ethnicity and Vulnerability Disparities

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Disclaimer

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Abbreviations

AOD – Aerosol Optical Depth

Aggr2 – Aggregation by 2 adjacent age groups (to form a 10-age-group dataset)

Aggr4 – Aggregation by 4 adjacent age groups (to form a 5-age-group dataset)

CARB – California Air Resources Board

CDPH – California Department of Public Health

CHF – Congestive Heart Failure

CIscoreP – CalEnviroScreen Percentile Score

COPD – Chronic Obstructive Pulmonary Disease

COVID-19 – Coronavirus Disease 2019

CTRL – Control Area (outside GMC and NGMC)

CT – Census Tract

DEM – Digital Elevation Model

DigitizeIt – Software used for extracting numeric values from graphs

D/S/A – Deletion/Substitution/Addition machine learning algorithm

EPA – Environmental Protection Agency

GMC – Goods Movement Corridor

GRADE – Grading of Recommendations Assessment, Development and Evaluation

GridMet – Gridded Meteorological Data

HIPAA – Health Insurance Portability and Accountability Act

HR – Hazard Ratio

IQR – Interquartile Range

LUR – Land Use Regression

MAIAC – Multi-Angle Implementation of Atmospheric Correction

MI – Myocardial Infarction

MOD13Q1.006 – MODIS Vegetation Indices Product

NAAQS – National Ambient Air Quality Standard

NASA – National Aeronautics and Space Administration

NGMC – Non-Goods Movement Corridor

NO_2 – Nitrogen Dioxide

NOS – Newcastle-Ottawa Scale

NLCD – National Land Cover Database

O_3 – Ozone

OMI – Ozone Monitoring Instrument

OR – Odds Ratio

PAF – Population Attributable Fraction

PeMS – Performance Measurement System (traffic data)

Period 1 – Years 2000-2010

Period 2 – Years 2011-2021

$PM_{2.5}$ – Particulate Matter ≤2.5 micrometers

PRISMA – Preferred Reporting Items for Systematic Reviews and Meta-Analyses

R – Statistical programming language

RAMP – Regional Asthma Management and Prevention

ROBINS-E – Risk of Bias in Non-Randomized Studies of Exposures

RS – Road Segment

RR – Relative Risk

SRDC – UC Berkeley Secure Research Data Center

UCB – University of California, Berkeley

USGS – United States Geological Survey

WHO – World Health Organization

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Project Summary/Abstract

While air quality has substantially improved over the past two decades, fine particulate matter ($PM_{2.5}$) remains a critical environmental health concern in California. This project provides one of the most comprehensive evaluations to date of how long-term $PM_{2.5}$ exposure affects mortality risk and life expectancy in California. Using two decades of statewide mortality data linked to high-resolution exposure surfaces, the research team quantify $PM_{2.5}$-attributable mortality and life-expectancy impacts for the periods 2000–2010 (Period 1) and 2011–2021 (Period 2). The study addresses a gap in the scientific literature: although many studies estimate mortality risks from $PM_{2.5}$, relatively few provide life-expectancy estimates, and almost none examine how both mortality risk and life-expectancy impacts change across two distinct decades. There is also limited evidence on how these impacts differ across detailed age groups and race-ethnicity groups. To our knowledge, no prior study has examined how the distribution of $PM_{2.5}$ impacts changes over time.

The research team apply a dual population-weighted and death-weighted framework to characterize the distribution of per-person $PM_{2.5}$ impacts across age groups. Population-weighted impacts reflect effects on the full population, while death-weighted impacts emphasize the age groups that contribute most to mortality. Together, these metrics reveal whether $PM_{2.5}$ impacts are concentrated among older adults, younger adults, or broadly distributed across ages. Statewide, both mortality risks and life-expectancy impacts from $PM_{2.5}$ decreased from Period 1 to Period 2. Long-term $PM_{2.5}$ exposure remained a significant determinant of mortality in both decades, but with a weaker association in Period 2, consistent with California's air quality improvements. The death-weighted life-expectancy impact statewide declined from 0.61 to 0.37 years, demonstrating that reductions in ambient $PM_{2.5}$ translated into meaningful reductions in mortality burden. Stratified analyses reveal differences across race-ethnicity groups. In Period 1, older adults generally bore the higher $PM_{2.5}$ impacts for most race-ethnicity groups. In Period 2, younger and middle-aged groups contributed more heavily relative to earlier years. Asians remained an exception, with population- and death-weighted impacts nearly identical in Period 1, reflecting broad age distribution of impacts. Black populations showed consistently low impacts among younger adults and concentrated impacts at older ages, while Hispanic populations exhibited large benefits from $PM_{2.5}$ reductions across a wide age span in Period 1 and continued to exhibit measurable $PM_{2.5}$-attributable mortality and life-expectancy impacts across multiple age groups in Period 2, despite overall declines in exposure and risk. ~~retained notable sensitivity~~. When comparing decades, Black and Hispanic populations experienced the largest reductions in death-weighted life-expectancy losses, approximately 0.59 and 0.57 years, respectively, reflecting substantial life-expectancy gains associated with improvements in ambient $PM_{2.5}$ concentrations from Period 1 to Period 2 due to sustained state and federal air-quality regulations ~~policy gains~~. By Period 2, death-weighted impacts were lowest for Black and White populations, whereas Asian, Hispanic, and Other groups continued to experience relatively higher $PM_{2.5}$-attributable life-expectancy losses compared with other groups.

The research team translated the age- and race-ethnicity–specific $PM_{2.5}$ impact estimates to the census-tract (CT) level. This was done by applying the modeled age-group effects for each race-ethnicity group to the corresponding demographic composition of each tract. The resulting spatial patterns confirm that statewide progress reduced $PM_{2.5}$-related death burdens but also show persistent local variation. Some tracts experienced large improvements, while others exhibited only modest gains, reflecting differences in composition and historical exposure patterns. In general, tracts with larger improvements tended to be those that had higher $PM_{2.5}$-attributable life-expectancy losses in Period 1 and therefore experienced larger absolute reductions as statewide $PM_{2.5}$ concentrations declined in Period 2, whereas tracts with smaller improvements often had lower baseline $PM_{2.5}$ impacts and correspondingly less room for improvement.

Together, these findings provide a clearer understanding of how $PM_{2.5}$ mortality impacts have evolved in California, where policy progress achieved the largest gains, and where residual disparities remain. The results highlight both the success of air quality regulations and the need for continued and targeted interventions to address remaining $PM_{2.5}$-related mortality risks.

Lay Person Summary

Air pollution from fine particles, known as $PM_{2.5}$, is one of the most harmful environmental risks to health. Although California's air has improved substantially over the last twenty years, the research team still lack a clear understanding of how much this pollution affects people's chances of dying and how much life could be extended if there were no $PM_{2.5}$ exposure. This project looked at all recorded deaths in California from 2000 to 2021 and combined that information with highly detailed long-term air pollution estimates. The research team studied two separate time periods, 2000-2010 and 2011-2021, to see how the health impacts of $PM_{2.5}$ changed as air quality improved. The research team also looked at differences across racial and ethnic groups and across age groups to see if $PM_{2.5}$ affected all communities in the same way.

The research team found that $PM_{2.5}$ continued to increase the risk of dying early in both time periods, but the risk was smaller in the more recent period, consistent with declining pollution levels across California. The research team also estimated how much longer people might live if people were not exposed to $PM_{2.5}$. This measure, referred to as $PM_{2.5}$-attributable life-expectancy loss, declined from about 0.61 years in the first decade to 0.37 years in the second, showing that cleaner air has produced meaningful health benefits.

The study also identified important differences across age groups and communities. During the first period, older adults were generally the most affected across most racial and ethnic groups. In the second decade, younger and middle-aged adults accounted for a larger share of the remaining $PM_{2.5}$-related health impacts. For race-ethnicity, Black and Hispanic populations experienced the largest improvements over time, with $PM_{2.5}$-related life-expectancy losses decreasing by about half a year between the two periods. By the second period, Black and White populations had the lowest benefits from $PM_{2.5}$ removal, while Asian, Hispanic, and Other groups still experienced notable life-expectancy games after removing air pollutant $PM_{2.5}$. These differences should be interpreted in the context of long-standing demographic patterns in California, where Asian and Hispanic populations generally have higher baseline life expectancy and Black populations have lower baseline life expectancy. Thus, a smaller remaining $PM_{2.5}$ impact for Black populations in Period 2 does not imply lower overall vulnerability but rather reflects reductions in $PM_{2.5}$-attributable burden relative to their own earlier levels.

The research team also mapped these life expectancy impacts at the community (i.e., census tract) level across California and found that life expectancy improved from period 1 to period 2. However, some neighborhoods were found to have benefited much more than others. Certain areas still have $PM_{2.5}$-related health burdens, reflecting differences in community characteristics and long-standing exposure patterns. Overall, the study shows that California's efforts to reduce air pollution have worked and have improved public health across the state. Yet important differences remain between communities. Continued action, especially targeted efforts in places with higher remaining impacts, is needed to make sure everyone benefits equally from cleaner air.

Executive Summary

a. Background

Fine particulate matter ($PM_{2.5}$) continues to pose a significant public-health burden in California, even as concentrations have declined substantially over the past two decades because of regulatory and technological progress. Long-term exposure to $PM_{2.5}$ is well established as a contributor to premature mortality, but relatively few studies quantify life-expectancy impacts, and even fewer examine how both mortality risks and life-expectancy losses evolve over multiple decades. Almost no prior research has evaluated how these impacts differ simultaneously by age group and race-ethnicity or how the distribution of $PM_{2.5}$ effects, whether concentrated in younger or older adults, changes over time. This study fills these gaps by assessing changes in mortality risks, life-expectancy impacts, age-distribution patterns, race-ethnicity disparities, and CT-level (census track level) outcomes across two distinct decades in California.

b. Objectives

The objectives of this study were to quantify the mortality effects of long-term $PM_{2.5}$ exposure in California, to translate those effects into life-expectancy impacts for two distinct time periods, and to evaluate how these impacts changed over time. Specifically, they include:

- Quantifying the mortality effects of long-term $PM_{2.5}$ exposure in California using individual-level death records from the California Department of Public Health (CDPH) and high-resolution exposure estimates, producing integrated and period-specific effect estimates for 2000-2010 and 2011-2021.
- Translating $PM_{2.5}$-related mortality risks into integrated and period-specific life-expectancy impacts by generating all-cause and counterfactual ($PM_{2.5}$-removed) life tables and taking their difference as the estimated $PM_{2.5}$ impact.
- Examining heterogeneity across race-ethnicity and age groups, using matched conditional logistic regression models stratified by age groups and by race-ethnicity categories, to assess subgroup-specific burden and how these patterns changed over time.
- Identifying whether younger or older age groups bore the greater $PM_{2.5}$-related burden by applying the study's new dual population-weighted and death-weighted $PM_{2.5}$ impact framework, which detects whether impacts were concentrated in younger, middle-aged, or older adults in each period.
- Assessing and visualizing $PM_{2.5}$ exposure, $PM_{2.5}$-attributable mortality, and life-expectancy impacts at the CT level across California by producing statewide, change-over-time, and region-specific maps, including stratification by age groups, race-ethnicity, and vulnerability, overlays with policy-relevant boundaries, and regional atlases.

b. Methods

The analysis linked individual-level mortality records from the California Department of Public Health (CDPH) to high-resolution daily $PM_{2.5}$ exposure estimates (100-meter resolution) developed by Dr. Su. For each decedent, long-term $PM_{2.5}$ exposure was defined as the one-year rolling average preceding the date of death. All-cause and $PM_{2.5}$-removed mortality rates were derived by first estimating age-specific mortality risks using conditional logistic regression models, where each model quantified the association between long-term $PM_{2.5}$ exposure and mortality within each age group (and race-ethnicity group, when stratified). The observed all-cause mortality life expectancy was constructed directly from CDPH individual-level death records and corresponding population denominators. The counterfactual $PM_{2.5}$-removed mortality schedule was generated by applying the regression coefficients to predict mortality risks under a scenario in which $PM_{2.5}$ exposure was set to zero, and the resulting observed vs. counterfactual mortality rates were then used to build life tables whose differences yielded life-expectancy impacts. To characterize how $PM_{2.5}$ impacts were distributed across age groups, the study applied a dual population-weighted and death-weighted framework, enabling clear identification of whether younger, middle-aged, or older adults contributed more to $PM_{2.5}$-related losses.

CT-level life-expectancy impacts were generated by applying race-ethnicity and age-group specific $PM_{2.5}$ effects to each tract's demographic composition, producing spatially resolved estimates for both Period 1 and Period 2. These results were visualized through a comprehensive set of maps, including CT-level life-expectancy impact layers for five- and ten-age-group frameworks, tract-level annual average $PM_{2.5}$ concentrations for 2010 and 2020 to illustrate long-term regulatory progress, and statewide maps of $PM_{2.5}$-attributable mortality and life-expectancy loss stratified by race-ethnicity and vulnerability. Additional change maps depict differences in $PM_{2.5}$ exposure and $PM_{2.5}$-related life-expectancy loss between the two decades, while overlays with policy-relevant boundaries such as region, air districts, and goods-movement corridors provide regulatory context.

c. Results

Across the full population, $PM_{2.5}$ remained a statistically significant determinant of mortality in both decades, with risk estimates weaker in Period 2 (2011–2021) than in Period 1 (2000–2010), consistent with statewide improvements in air quality. The statewide death-weighted life-expectancy impact declined from 0.61 years in Period 1 to 0.37 years in Period 2, demonstrating that reductions in ambient $PM_{2.5}$ translated into substantial reductions in $PM_{2.5}$-attributable mortality burden. Age- and race-ethnicity-specific analyses revealed important disparities. In Period 1, older adults generally bore larger $PM_{2.5}$ impacts across most race-ethnicity groups. In Period 2, younger and middle-aged adults contributed relatively more to the $PM_{2.5}$ impacts, reflecting a shift in age distribution as overall pollution levels decreased. Asians were an exception: their population-weighted and death-weighted impacts were nearly identical in Period 1 (0.94 vs. 0.91), indicating broad age distribution of impacts rather than concentration in younger or older groups. Black populations exhibited consistently low impacts among younger

adults in both periods and concentrated impacts among older adults. Hispanic populations showed substantial $PM_{2.5}$ impacts in Period 1 and continued to exhibit notable impacts in Period 2. Using death-weighted changes to quantify policy gains, Black and Hispanic populations experienced the largest reductions between decades, approximately 0.59 and 0.57 years, indicating that regulatory progress yielded the greatest mortality improvements for these groups. By Period 2, Black populations exhibited comparatively low $PM_{2.5}$ life-expectancy impacts (0.08 years), whereas Asian (0.74), Hispanic (0.36), and Other (0.38) groups continued to show meaningful remaining impacts.

Mapping analyses at community (CT) level confirmed statewide improvements but also revealed persistent geographic variability. Tract-level death-weighted life-expectancy impacts declined from 0.82 years to 0.61 years between decades, reflecting broad policy success. Gains varied by neighborhood disadvantage: in the most disadvantaged tracts, mean impacts fell from 0.82 to 0.66 years, whereas in the most advantaged tracts, impacts declined from 0.81 to 0.57 years, yielding larger average policy benefits (0.25 years vs. 0.16 years).However, improvement was uneven: some tracts experienced substantial life-expectancy gains from reduced $PM_{2.5}$, while others saw only modest progress, shaped by demographic composition and spatial exposure patterns. Spatial mapping highlighted hotspots in the San Joaquin Valley and Inland Empire, where some tracts continued to experience PM2.5-related life expectancy losses near 0.8 years, underscoring the persistence of environmental inequities despite overall improvements.

d. Conclusion

This study provides one of the most detailed examinations to date of how $PM_{2.5}$ affects mortality and life expectancy across age groups, race-ethnicity groups, and communities in California, and how these impacts have changed over two decades. The findings demonstrate that regulatory actions produced large reductions in $PM_{2.5}$-related mortality, with especially substantial gains for Black and Hispanic populations. At the same time, meaningful residual impacts persist among Asian, Hispanic, and Other race-ethnicity groups and in specific census tracts. The results show that $PM_{2.5}$ control continues to yield tangible public-health benefits, but achieving equitable protection requires maintaining statewide emission reductions while also targeting local areas and demographic groups with remaining high impacts. Together, these findings underscore both the success of California’s air-quality regulations and the ongoing need for focused policies to further reduce $PM_{2.5}$-attributable mortality and address remaining disparities.

Introduction

Long-term exposure to ambient air pollution is a well-established driver of premature mortality, and the existing scientific literature is substantial, contributing to our understanding of the mortality risks associated with particulate matter ≤2.5 μm ($PM_{2.5}$) (see Task 1 Literature Review for detail). Among the 39 studies identified in our systematic review, 32 examined $PM_{2.5}$ exposure and nearly all reported positive associations with all-cause mortality. These studies provide important evidence demonstrating that even low levels of $PM_{2.5}$ are associated with elevated mortality risk. However, despite the large and growing mortality literature, few studies have quantified the impact of air pollution on life expectancy, a metric with greater interpretability for policymakers and the public, and one that directly reflects the cumulative survival consequences of environmental exposures across the lifespan.

Only three of the 39 studies the research team reviewed estimated changes in life expectancy due to long-term $PM_{2.5}$ exposure.[1–3] Through these limited studies, life expectancy losses ranged from approximately 0.3 to 0.89 years, illustrating reductions in expected lifespan associated with sustained pollution exposure. Nonetheless, life expectancy methods remain underutilized in environmental epidemiology despite their relevance for public health burden assessment. Moreover, existing life expectancy studies are few in number, rely on mostly ecologic designs, and provide limited insight into how air pollution-related life expectancy loss varies across demographic subgroups. As a result, policymakers have had limited information on how long-term air pollution exposure affects remaining life expectancy across age groups, race-ethnicity populations, socioeconomic strata, and geographic settings.

Our literature review (see Task 1) also identified substantial evidence of disparities in air pollution-related mortality. Older adults, particularly those aged 65-74, consistently showed higher mortality risks from $PM_{2.5}$ and nitrogen dioxide (NO_2) exposure; Black populations in the United States experienced greater mortality impacts per unit increase in $PM_{2.5}$ than White populations; and individuals with lower educational attainment or living in high-poverty neighborhoods were consistently more vulnerable to pollution-related mortality. Urban residents, individuals with chronic cardiometabolic or respiratory conditions, and Medicare beneficiaries with Medicaid dual eligibility were repeatedly identified as higher-risk subgroups. However, no identified study evaluated whether these disparities translate into differences in pollution-attributable life expectancy, nor did any report life expectancy impacts stratify by race-ethnicity, age group, comorbidity burden, or urbanicity. This represents a major gap in literature: although mortality disparities by air pollution exposure are well documented, their implications for life expectancy and therefore for cumulative survival over time have not been characterized.

An additional gap is the near absence of research examining how changes in life expectancy attributable to air pollution evolve over time. While several studies have quantified temporal trends in $PM_{2.5}$ concentrations and associated mortality burdens, none of the studies in our review compared pollution-attributable life expectancy impacts across different historical

periods. Such analyses are essential for understanding the public health benefits of regulatory actions, reductions in emissions, technological improvements, and changes in the built environment. Without evaluating changes over time, it is not possible to quantify how improvements in air quality have translated into gains in life expectancy or whether disparities in these gains have widened or narrowed.

In this project, the research team address a series of methodological and equity-related gaps not examined in prior literature. First, unlike most existing mortality-only studies, our project uses life-table-based life expectancy modeling to quantify age-specific and population-wide survival impacts from long-term (i.e., 365 days before death) $PM_{2.5}$ exposure, providing a more intuitive and policy-relevant measure of population health burden. Second, the research team incorporate fine-grained demographic stratification by age and race-ethnicity to assess disparities in $PM_{2.5}$-attributable life expectancy loss. This extends beyond previous studies, which largely confined subgroup analyses to mortality risk and did not evaluate cumulative survival outcomes. Third, our analysis examines two separate multiyear periods, enabling assessment of whether life expectancy improvements associated with declining $PM_{2.5}$ levels have been uniform across California or experienced disproportionately by specific communities. Fourth, the research team leverage population-weighted and death-weighted estimators, along with age-specific life-table metrics, to distinguish between population burden, mortality burden, and survivor impacts, an approach rarely used in environmental health studies. Fifth, the research team applied modeled race-ethnicity-specific age-group impacts to each census tract's (CT) race-ethnicity-specific age-group composition, allowing us to estimate CT-level life expectancy impacts attributable to $PM_{2.5}$ for both study periods. This approach enabled us to quantify changes in tract-level life expectancy from Period 1 to Period 2 and to evaluate whether improvements in air quality translated into equitable gains in longevity across California's diverse communities. Together, these methodological advancements provide a more comprehensive, equity-centered, and policy-relevant assessment of the long-term health benefits associated with air quality improvements in California.

Task 1. Literature Review

Background

Ambient (outdoor) air pollution poses a major threat to human health, contributing to approximately four million premature deaths worldwide each year.[4] While air pollution affects entire populations, there is a growing recognition that certain groups based on sociodemographic characteristics may face disproportionately greater health risks.[5–8] Studies have reported elevated rates of air pollution-related hospitalizations, respiratory infections, and cardiopulmonary mortality among older adults, women, and individuals of lower socioeconomic status.[9,10] In the United States, higher rates of respiratory morbidity and hospital admissions have been observed specifically in African American communities and in areas with high poverty levels.[10–12]

Individuals with chronic conditions such as chronic obstructive lung disease (COPD) and diabetes have also been documented with more frequent hospitalizations and exacerbations following exposure to air pollutants compared to their counterparts.[10,13,14] Residents of urban areas have been shown to experience elevated rates of air pollution-related morbidity and mortality.[15–17] These previous studies highlight the importance of elucidating both broad population-level health impacts of air pollution and the disproportionate risks borne by specific vulnerable subpopulations.

Particulate matter $PM_{2.5}$, NO_2, and ozone have garnered significant attention given their well-established associations with adverse health outcomes.[4,5,18–20] Both short- and long-term exposure to $PM_{2.5}$ - fine inhalable particles emitted primarily from combustion processes such as vehicle exhaust, industrial activities, and biomass burning - are consistently linked with increased cardiopulmonary morbidity and mortality.[18,19] NO_2, predominantly derived from traffic emissions and fossil fuel combustion, is known to cause airway irritation and exacerbate respiratory conditions.[18,20] Ozone, a principal component of photochemical smog, is also associated with increased respiratory morbidity.[21] In recognition of the health risks posed by these pollutants, the World Health Organization (WHO) revised its global air quality guidelines in 2021, recommending more stringent annual mean exposure limits of 5 $\mu g/m^3$ for $PM_{2.5}$, 10 $\mu g/m^3$ for NO_2, and a peak-season limit of 60 $\mu g/m^3$ for ozone.[5] The U.S. Environmental Protection Agency (EPA) also finalized a revised National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ that lowers the primary (health-based) annual $PM_{2.5}$ standard from 12.0 $\mu g/m^3$) to 9.0 $\mu g/m^3$. [22] However, air pollutant concentrations in many regions across the globe continue to exceed recommended thresholds.[23,24] For example, 98.8 percent of people were exposed to pollution levels above the WHO guideline.[25]

While prior meta-analyses have assessed the relationship between long-term exposure to $PM_{2.5}$, NO_2, and ozone and all-cause mortality, few have systematically evaluated whether these associations differ across the vulnerable subpopulations.[26–28] This represents a critical knowledge gap, as understanding differential mortality risks across diverse sociodemographic populations is essential for informing equitable public health interventions and air quality standards.

In 2023, the California Air Resources Board commissioned a project entitled “Impacts of Air Pollution on Life Expectancy across Multiple Generations: Race, Ethnicity and Vulnerability Perspectives.” As a part of this project, our objective was to conduct a systematic review to evaluate associations between long-term exposure to ambient air pollutants ($PM_{2.5}$, NO_2, and ozone) and all-cause mortality, with a focus on assessing differential risks among potentially more vulnerable subpopulations, specifically older adults, women, individuals with pre-existing health conditions, socioeconomically disadvantaged communities, urban populations, and minoritized racial and/or ethnic groups.

Methods

Eligibility Criteria and Search Strategy

Population

This review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (A.1 of the Appendix).[29,30] The research team included studies examining general human populations with long-term exposure to ambient air pollution, specifically to particulate matter $PM_{2.5}$, ozone, or nitrogen oxides (e.g. nitrogen dioxide). The research team excluded studies focusing on newborns, and instead included only studies that focused on persons older than 1 year of age. Studies focusing on highly specific disease cohorts (e.g. patients with COVID-19, respiratory disorders, cardiovascular disease) were excluded to ensure generalizability to the broader population. Additionally, the research team excluded studies on populations affected by time-limited acute pollution events such as wildfires or dust storms. The research team did not place any restrictions on other demographic variables such as sex, race, ethnicity, occupation, or residential location of study populations.

Exposure

Eligible studies assessed long-term exposure defined as 1+ years to $PM_{2.5}$, ozone, or nitrogen oxide/dioxide (NO_x/NO_2). Studies evaluating pollutants other than these three (e.g. PM10, sulfur dioxide, black carbon, etc.) were excluded. Studies focusing exclusively on occupational exposure, or indoor air pollution were excluded. The review reflects the broader criteria pollutant exposure relevant to health but does not explicitly stratify wildfire-specific impacts. Any studies that assessed those criteria pollutant exposures, including those incorporating wildfire contributions, would have been captured if they met the general inclusion criteria for long-term ambient air pollution exposure.

Comparator

The research team only included studies that assessed air pollution exposure as a continuous variable. Reference exposure levels varied by study but generally included a lower exposure group within the same cohort for comparison or matched with another cohort for comparison.

Outcomes

Eligible studies reported all-cause mortality excluding accidents as an outcome. Studies assessing cause-specific mortality only (e.g. cardiovascular mortality, respiratory mortality, or lung cancer mortality) or morbidity outcomes (e.g. hospitalization rate, lung cancer rate) were excluded. The research team also excluded studies reporting outcomes exclusively related to newborns or birth-related outcomes such as maternal mortality.

Study

The research team included peer-reviewed original research studies using retrospective cohort, prospective cohort, case-control, and observational study designs. Systematic reviews and meta-analyses were not included in the quantitative synthesis but were reviewed for

additional references. Conference abstracts were excluded. The research team included non-English studies when a translation was available.

A systematic search of PubMed, MEDLINE, and Web of Science was conducted to identify peer-reviewed articles published between January 1, 2000 and October 19, 2023. The search strategy, detailed in the Appendix, included terms related to air pollutants ($PM_{2.5}$, Ozone, NO_x/NO_2), mortality outcomes, and socially vulnerable populations.

Study Selection and Data Collection

Two trained reviewers independently screened all records by title and abstract using the Rayyan web application,[31] which supports blinded dual screening, enables tagging and categorization of studies, and automatically detects conflicts between reviewers' decisions. Any disagreements were resolved through discussion or consultation with the full study team. After reviewing the full texts of studies deemed potentially eligible by title and abstract screening, the two reviewers jointly made final decisions to include or exclude.

Two reviewers independently performed data extraction using a standardized data collection form. Extracted data included study characteristics such as geographical location, population demographics, sample size, and study design. The research team also recorded exposure details including pollutant type, exposure window, calculation methods, and data sources. Length of follow-up and mortality outcomes were documented alongside the data sources and statistical methods used to derive effect estimates. When studies reported multiple statistical models, the research team extracted effect estimates from the fully adjusted model. Additionally, the research team extracted a list of confounders each study controlled for and recorded any subgroup analyses conducted by age, sex, race, ethnicity, socioeconomic status, region, and urbanicity. Any discrepancies in data extraction were resolved through joint review and consensus.

Risk of Bias Assessment

The risk of bias (ROB) for each included study was assessed using the Risk of Bias in Non-Randomized Studies of Exposures (ROBINS-E) tool.[32] The research team initially considered evaluating studies using the Newcastle-Ottawa Scale (NOS), but elected to use ROBINS-E as it provided a more comprehensive and structured assessment of bias domains critical to air pollution studies, including exposure misclassification, time-varying confounding, and selection bias. Two independent reviewers conducted the ROBINS-E assessments, and disagreements were resolved through discussion. Studies were evaluated across the seven ROBINS-E domains including risk of bias due to confounding, exposure measurement, selection of participants, post-exposure interventions, missing data, outcome measurement, and selection of reported result. The overall ROB based on these domains was determined as low, some concerns, high risk, or very high risk. A summary table of completed ROBINS-E evaluations is available in the A.3. of the Appendix.

Data Synthesis and Narrative Review

A narrative synthesis was conducted to summarize findings across studies, with a focus on effect estimates such as hazard ratios and risk ratios for all-cause mortality. The research team examined variations in risk across demographic, socioeconomic, and geographic subgroups. In studies that only presented a graphical subgroup analysis, numeric data were extracted from scanned images of published graphs by using the DigitizeIt software, version 2.5.9, [Germany].[33,34] The research team note that this digitization process can introduce some measurement uncertainty, particularly for smaller demographic subgroups where effect sizes or confidence intervals are more difficult to extract precisely. Due to heterogeneity in study designs, exposure assessment methods, and statistical models, a meta-analysis was not performed. Instead, the research team explored sources of heterogeneity qualitatively, considering differences in study populations and subgroup analyses. Specifically, the research team examined how the relationship between air pollution and all-cause mortality differed in vulnerable subpopulations, including older adults, women, historically minoritized racial or ethnic groups, urban residents, individuals of low socioeconomic status, and those with chronic health conditions. No formal sensitivity analyses were conducted, as findings were synthesized narratively.

Certainty of Evidence

No formal assessment of certainty or confidence in the body of evidence (e.g. using GRADE) was performed, as the included studies were heterogeneous in design, exposure assessment, population characteristics, and analytic approaches, precluding standardized grading across outcomes.

Results

Article Selection and Description

A total of 3,764 unique records were identified through our systematic electronic search from January 1, 2020 to October 19, 2023 (Figure S 1). Two reviewers screened all 3,764 records by title and abstract; of these, reviewers selected 192 records for full-text review. After full-text evaluation, 39 peer-reviewed original research articles met the inclusion criteria and were included in the final analysis. Representative examples of studies excluded after full-text screening are available in A.4. of the Appendix.

Study periods spanned from the 1970s to the 2010s, but most studies were published within the past decade (Figure S 2). Geographically, the majority of these studies originated from North America (n=22) and Europe (n=9), with an increasing number of contributions from Asia, Australia, South America, and Africa in recent years. Many of the included mortality studies rely on national cohorts, such as Medicare populations, whose demographic composition, including age structure and race-ethnicity, differs from that of California. Therefore, caution is warranted when generalizing these findings to California’s population.

Among the included studies, sample sizes ranged from 2,734 participants[35] to 73.4 million[36] (Table S 1). Three studies examined the association between air pollution and all-cause mortality in children under five,[37–39] while the remaining studies focused on adults across a broad age spectrum. Three studies focused specifically on middle-aged populations (40s-50s).[40–42] Eleven studies focused on individuals aged >=65, representing the Medicare population in the United States.[36,43–52]

Other sample characteristics varied widely across the included studies. Some studies examined specific populations only based on race (e.g. Caucasian adults only[53–55]), sex (women only[40,56] or men only[42]), residential urbanicity (urban residents only[57–61]), or occupation (e.g. teachers only[56]). In contrast, some studies utilized broader, nationally representative samples, including those from the United States,[62,63] Spain,[64] Taiwan,[65] China,[66,67] and Brazil.[68] Overall, 32 studies evaluated ambient $PM_{2.5}$ as the primary air pollutant exposure in relation to all-cause mortality; 11 studies examined nitrogen dioxides; and 9 studies included ozone.

The majority of studies controlled for individual-level confounders, including age, sex, smoking status, chronic health conditions, and at least one socioeconomic indicator most commonly education or income level. In population-based ecologic studies lacking individual-level data, area-level metrics (e.g. county-level or neighborhood level data) were used to adjust for potential confounding variables.

Exposure periods varied across studies, ranging from one year[35,45,51,61,69] to 31 years.[54] Follow-up durations also differed considerably, from one year preceding mortality[51] to lifelong follow-up from age 11 to death.[35]

Risk of bias was assessed using the ROBINS-E tool. The majority of studies were rated as low risk of bias across most domains. “Some concerns” were frequently identified in domains related to confounding (D1) and classification of exposures (D3). Only a small number of studies were rated as having “some concerns” overall, and no studies were rated as high or very high risk of bias.

$PM_{2.5}$ and all-cause mortality risk

A total of 31 studies quantified all-cause mortality risk in association with $PM_{2.5}$ exposure (Table S 2). The mean/median $PM_{2.5}$ exposure levels varied widely across studies, ranging from 3.6 μg/m³ in Queensland, Australia[70] to 47.1 μg/m³ across mainland China.[66] The association between $PM_{2.5}$ and all-cause mortality was positive in nearly all included studies, with higher $PM_{2.5}$ levels associated with higher subsequent mortality risk (HRs and RRs ranging from 1.01 to 1.19 per 10 μg/m³ increase in $PM_{2.5}$).

Three studies estimated changes in life expectancy from long-term $PM_{2.5}$ exposure[49,62,65] and reported life expectancy loss by 0.3 years per 10 μg/m³ increase in $PM_{2.5}$[65] to 0.89 years per 4.5 μg/m³ increase.[49]

Effect sizes varied based on study-specific characteristics such as cohort size, exposure level, and covariates. Larger-scale studies based on the U.S. Medicare population data or nationwide ecologic data consistently reported statistically significant associations between $PM_{2.5}$ exposure

and all-cause mortality. In contrast, three studies,[56,59,71] all with relatively smaller sample sizes, found non-significant associations between $PM_{2.5}$ and all-cause mortality. ~~There were no clear trends over time~~.

Ozone and all-cause mortality risk

Nine studies assessed the association between ambient ozone exposure and all-cause mortality (Table S 2). Mean/median ozone concentrations ranged from 21.9 ppb in South Korea[57] to 57.7 ppb in U.S. metropolitan areas.[72] Compared to $PM_{2.5}$, the ozone-mortality relationship was more variable and generally weaker, with risk estimates (HRs and RRs) ranging from 0.92 (95% CI: 0.89-0.96)[41] to 1.18 (95% CI: 1.07-1.29)[57] per 10 ppb increase in ozone exposure. Five of the nine studies reported a significant positive association between ozone and all-cause mortality, whereas the remaining four found non-significant results. Notably, studies with larger cohorts[46,64] consistently observed positive associations, suggesting that the true effect of ozone on mortality may be marginal and require large samples to detect.

Nitrogen oxides and all-cause mortality risk

Eleven studies evaluated all-cause mortality risk in relation to nitrogen oxide (NO_x/NO_2) exposure (Table S 2). Reported mean or median nitrogen oxide concentrations varied substantially across studies, ranging from 9.48 μg/m³ in Spain[64] to 50 μg/m³ in China.[58] The association between nitrogen oxides and all-cause mortality was generally positive with hazard ratios (HRs) and relative risks (RRs) ranging from 0.96 (95% CI: 0.93-0.98)[63] per 10 ppb increase in NO_2 exposure to 1.22 (95% CI: 1.10-1.35)[67] per 10 μg/m³ increase in NO_2 exposure. The strongest associations were observed in Wang 2023[67] (HR 1.22, 95% CI: 1.10-1.35) and Heinrich 2012[40] (HR 1.18, 95% CI: 1.07-1.30), both conducted in regions with relatively high NO_2 levels in China and Germany. Large-scale cohort studies consistently found small but significant increases in all-cause mortality risk (HR 1.05, 95% CI: 1.04-1.05[52]; HR 1.01, 95% CI: 1.00-1.03[73]).

Age-stratified all-cause mortality risk associated with air pollution

Studies that compared age groups <65 years and >65 years tended to report higher all-cause mortality risk estimates for $PM_{2.5}$ exposure in the older age groups (>65), although not consistent across all studies (Table S 3). However, among populations >65 years, studies consistently found strongest associations in the 65-74 age group, with attenuated effects observed in those >75 or >85 years.[43,46,47] Significant age differences were observed only in large cohort studies, whereas studies with relatively smaller sample sizes generally found no variation in risk estimates across age groups.[35,60,71]

For ozone exposure, one study[46] conducted age-stratified analyses of all-cause mortality and reported no significant differences across age groups.

Three studies examined age-stratified all-cause mortality risk due to nitrogen oxides and reported a higher risk with increasing age[42,67] with a decline in risk above 80 years of age.[52]

Sex-stratified all-cause mortality risk from air pollution

Twelve studies conducted sex-stratified analyses of all-cause mortality from $PM_{2.5}$ exposure and yielded mixed findings (Table S 4). Some reported higher mortality risk[41,46,60] and greater reductions in life expectancy[49] in men, while others found higher risks in women.[62,71] However, most studies found no significant difference between the sexes. For ozone, two studies conducted sex-stratified analyses and found no significant differences in all-cause mortality risk between men and women. Two studies assessed sex differences in mortality risk associated with nitrogen oxide/dioxide, with conflicting results.[42,63,67]

Race-stratified all-cause mortality risk from air pollution

Six U.S.-based studies examined racial differences in all-cause mortality risk from ambient air pollution (Table S 5). For $PM_{2.5}$, three studies reported significantly higher effect estimates for Black individuals compared to White individuals,[46,47,50] while two also found elevated risk among Hispanic, Asian, and Native American populations.[46,47] For ozone, one study found no significant variation in mortality risk across racial groups;[46] and for nitrogen oxides, one study reported higher mortality risk for Black individuals compared to White individuals.[52]

SES-stratified all-cause mortality risk from air pollution

Twelve studies examined socioeconomic status (SES) as an effect modifier in the association between air pollution and all-cause mortality risk (Table S 6-Table S 8). SES was assessed using individual-level indicators (e.g., education level, employment, income, Medicaid eligibility) and area-level measures (e.g., neighborhood income level, percentage below the poverty level, median household income).

Studies consistently found higher all-cause mortality risk from $PM_{2.5}$ and nitrogen oxide exposure in individuals with lower educational attainment (Table S 6). In contrast, findings for neighborhood- and area-level SES metrics were more variable (Table S 7); while some studies reported stronger associations in lower-income or high-poverty areas, suggesting greater vulnerability in economically marginalized communities[42,44,47]; others found no clear pattern.[43,52]

Among Medicare beneficiaries, studies used Medicaid-Medicare dual eligibility as a proxy for low SES (Table S 8) and reported varied results, with only two studies reporting significantly higher mortality risk among Medicaid-eligible individuals compared to non-eligible counterparts.[46,50]

Comorbidity-stratified all-cause mortality risk from air pollution.

Five studies examined whether pre-existing chronic health conditions modified the association between air pollution and all-cause mortality risk (Table S 9). One study found a significantly higher mortality risk from $PM_{2.5}$ among individuals with prior hospitalizations for chronic heart failure (CHF), myocardial infarction (MI), chronic obstructive pulmonary disease (COPD), or diabetes[34] compared to healthy individuals. Individuals with such cardiovascular, respiratory, and diabetic diseases also had increased susceptibility to death by any cause from nitrogen oxide exposure.[42,67] However, with few studies available, the limited evidence base

prevents definitive conclusions. No included study examined effect modification by pre-existing health conditions on the association between all-cause mortality and ozone exposure.

Urbanicity-stratified all-cause mortality risk from air pollution.

Fourteen studies examined the association between air pollution and all-cause mortality risk stratified by urbanicity (Table S 10). Most studies reported higher mortality risk and life expectancy loss from $PM_{2.5}$ exposure in urban areas compared to rural areas.[45,47,62,68,70] For ozone and nitrogen oxide exposure, only a small number of studies analyze urban-rural differences and reported conflicting results.

$PM_{2.5}$ exposure and life expectancy

Correia et al.[1] estimated that sustained exposure to elevated $PM_{2.5}$ levels was associated with a 0.35-year reduction in life expectancy across U.S. counties ($p = 0.033$). Schwartz et al.[2] reported a larger effect, finding 0.89 years of life expectancy lost (95% CI: 0.88-0.91) associated with long-term $PM_{2.5}$ exposure using updated exposure reconstruction and extended follow-up. Chen et al.[3] analyzing a national cohort with additional control for socioeconomic and behavioral factors, found a 0.3-year loss in life expectancy (95% CI: 0.1-0.6) linked to $PM_{2.5}$. Together, these limited studies show that across different analytic approaches and cohorts, $PM_{2.5}$ exposure is consistently associated with measurable reductions in population life expectancy, generally in the range of 0.3 to 0.9 years.

Summary

Our systematic review of the relationship between air pollution ($PM_{2.5}$, NO_2 and O_3) and all-cause mortality found consistent evidence that long-term exposure to $PM_{2.5}$ is associated with increased mortality risk, with suggestive but less consistent associations for NO_2 and O_3. Subgroups such as older adults, individuals with lower socioeconomic status and Black populations in the US appear to face disproportionate health risks. These findings underscore the need for targeted public health interventions to better understand and mitigate air pollution-related health disparities.

Task 2. Develop $PM_{2.5}$ Surfaces for the Study Population

The research team developed daily $PM_{2.5}$ surfaces for the purpose of assigning rolling annual average air pollution exposure for the date of death of any individual between 2000 and 2021. The $PM_{2.5}$ surfaces were thus developed for 1999-2021 to make sure each subject has an annual average exposure before death. In developing daily land use regression (LUR) models for $PM_{2.5}$, The research team ~~UCB~~ first identified factors (i.e., source or sink) that might impact $PM_{2.5}$ concentrations and use them as potential predictors. The research team also identified the optimal distance of impact for a potential predictor and the models should be able to deal with multicollinearity among predictors and can reduce model overfit. Further, the research team aimed to avoid excessive number of predictors in the final selected model and will allow a

maximum of 15 predictors (in addition to four Seasons) in a LUR model. Due to those considerations, the research team applies the Deletion/Substitution/Addition (D/S/A) algorithm for developing a daily prediction model.[74–76] The modeling process is described in detail below.

Development of Comprehensive Data Sources

The research team developed comprehensive data sources that have potential impact on the concentrations measured at California Environmental Protection Agency (CalEPA) monitoring sites. The data sources include daily traffic data, daily remote sensing data, daily weather data, every two-week vegetation index, one time land use and land cover data, and other potential impact factors. The research team hypothesizes that greater daily traffic is associated with higher $PM_{2.5}$ concentrations. Remote sensing Aerosol Optical Depth (AOD) data is an indirect measure of $PM_{2.5}$ concentrations with greater AOD values being directly associated with higher $PM_{2.5}$ concentrations. Different land use types have different impacts on $PM_{2.5}$ concentrations with, for example, higher industrial and commercial land use being associated with greater concentrations. Similarly, different land cover types can have other impacts on $PM_{2.5}$ concentrations with, for example, high intensity urban developed land cover being associated with greater concentrations but greater vegetation cover (as a sink) being associated with lower concentrations. Further, greener vegetation has a much better air pollutant absorption effect than less green vegetation and thus the former helps reduce concentrations. For weather data, greater wind speed is associated with lower concentrations while lower visibility is associated with higher concentrations. The research team also collected daily $PM_{2.5}$ concentrations data at the CARB regulatory monitoring sites (i.e., CalEPA sites) for the years 1998-2021 and used them as a response variable in generating daily $PM_{2.5}$ concentration models. The potential predictors are listed in Table 1.

Table 1. The potential LUR predictors for the daily LUR model development.

Category	Variable for Prediction	Resolution	Description
Buffer (50m-5km)	Daily Traffic	vector	California Department of Transportation (CalTrans)
	Land Use	vector	Agricultural, residential, commercial, industrial, government and institutions, open land, parks, and recreational facilities (Parcel data)
	Land Cover	vector	Forest, herbaceous/grassland, shrubland, developed, agriculture, wetlands, water and other (USGS NLCD)
Non-Buffer Remote Sensing Data	Daily GridMET	4 km	Maximum temperature, minimum temperature, precipitation accumulation, downward surface shortwave radiation, wind-velocity, humidity (maximum and minimum relative humidity and specific humidity)
	Two-week Interval Vegetation Index	250 m	Normalized difference vegetation index (NDVI) (NASA MOD13Q1.006 Terra)
	Daily Aerosol Optical Depth (AOD)	1 km	NASA Multiangle Implementation of Atmospheric Correction (MAIAC) algorithm
	Daily Ozone from Ozone Monitoring Instrument (OMI)	27 km	Global for both NO_2 and O_3 measurements for 2004 - current (NASA)
	Annual $PM_{2.5}$	1 km	North America for 1989-2016 (Univ. Washington Randall Martin)
	Annual NO_2	1 km	Global for 1990-2020 (NASA reanalysis)
Other Non-buffer Variables	Digital Elevation Model (DEM)	30 m	U.S. Geological Survey (USGS)
	Distance to Coast	30 m	U.S. Geological Survey (USGS)
	Distance to Roadways	30 m	Environmental Systems Research Institute (ESRI)
	Distance to Ports	30 m	U.S. Geological Survey (USGS)
	Location category	vector	California Department of Transportation (CalTrans)/ESRI

Daily traffic data: For daily traffic data, the research team used the data collected by the California Department of Transportation (CalTrans) Performance Measurement System (PeMS) (https://dot.ca.gov/programs/traffic-operations/mpr/pems-source). PeMS data are collected in real-time from nearly 40,000 individual detectors spanning the freeway system across all major metropolitan areas of the State of California and provide an archived data user service that provides over fifteen years of data for historical analysis. PeMS integrates a wide variety of information from Caltrans and other local agency systems including traffic flow, speed, occupancy, incident, toll charge, and other information. The research team used PeMS five-minute road link/segment traffic flow data in the analysis. In PeMS, traffic flow (volume) is a quantity representing the number of vehicles that passed over each detector on the roadway in a given time period (i.e. five-minute flow, hourly flow, etc.). The detector measured traffic flow that covered 12.52 percent highway segments and the research team summed hourly traffic to daily traffic for all the stations across California. The following interconnected stages were used to derive daily traffic for all the California highways for the study period:

1) For a road segment with station traffic measure for a day, use all the station traffic measures on that road segment to generate a daily mean traffic for that road segment for that day.

2) For those road segments without traffic measures for a day, assign them using the assigned segments from step 1 by matching route, county, district, route type and day, and find the one with the smallest distance if having multiple matches. California has 58 counties which are included in one of the 12 CalTrans air districts (1 - Eureka, 2 - Redding, 3 - Marysville / Sacramento, 4 - Bay Area / Oakland, 5 - San Luis Obispo / Santa Barbara, 6 - Fresno / Bakersfield, 7 - Los Angeles, 8 - San Bernardino / Riverside, 9 - Bishop, 10 - Stockton, 11 - San Diego, 12 - Orange County). Highways in California are split into at least four different types of systems: Interstate Highways, U.S. Highways, state highways, and county highways.

3) For those road segments without traffic being assigned from steps 1 & 2, assign them using the assigned segments from steps 1 & 2 by matching route, district, route type and day, and find the one with the smallest distance if having multiple matches. In this step county was not used as a restricting factor in daily traffic assignment.

4) For those road segments without traffic being assigned from the above steps, assign them using the above assigned segments by matching route, county, district and route type, plus at most one day difference in data availability and find the one with the smallest distance if having multiple matches.

5) Identify those not assigned and assign them using the assigned segments from above steps by matching county, district, route type and day and find the one with the smallest distance if having multiple matches. Here the restricting factor of route number is removed.

6) Identify those not assigned and assign them using the assigned segments from the above steps by matching district, route type and day and find the one with the smallest distance if having multiple matches. Here the restricting factors of route number and county are removed.

   7a) Identify those not assigned and assign them using the assigned state highway segments from the above steps by matching district and day. Here the restricting factors of route number, route type and county are removed.

   7b) Identify those not assigned and assign them using the assigned U.S. highway segments from the above steps by matching district and day. Here the restricting factors of route number, route type and county are removed.

   7c) Identify those not assigned and assign them using the assigned interstate highway segments from the above steps by matching district and day. Here the restricting factors of route number, route type and county are removed.

8) Identify those not assigned and assign them using the assigned segments from steps 1-4 by matching district and season to find the one with the smallest distance if having multiple matches. Here route number, county and route type are not required to match.

Table 2 shows the daily traffic assignment statistics for the 12 California districts for the study period. Overall, 12.52 percent California highways had daily traffic measurements for the study period, with ranges being from 0 percent (district 9) to 38.24 percent (district 12). The research team found that the districts with great population (i.e., metropolitan areas) had more roadways and more traffic measures. Those districts thus had smaller proportions of roadways being assigned traffic from greatly relaxed conditions (e.g., by gradually relaxing matching criteria on route, county, district, route type or day). The roadways in the vastly rural districts were the ones with much less proportion of traffic measures. Greater proportion of roadways were thus assigned through greatly relaxed conditions for those rural districts. The CalTrans PeMS traffic data started in 2001. A trend analysis from years 2001-2020 was used to extend the daily traffic data back to the years 1999 and 2000.

$PM_{2.5}$ remote sensing data: The research team obtained Aerosol Optical Depth (AOD) data from the Moderate Resolution Imaging Spectroradiometer instruments onboard the National Aeronautics and Space Administration Terra and Aqua satellites. The Multiangle Implementation of Atmospheric Correction algorithm was used to derive 1 km resolution AOD surfaces.[77] Due to extensive missing data presented at the 1 km resolution AOD surfaces, the research team aggregated the daily AOD surfaces into monthly means.

Table 2. Traffic data assignment statistics based on the stages of assignment.

	District #1				District #2				District #3			
Stage	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)
1	34,197	2.93	34,197	2.93	64,284	4.94	64,284	4.94	75,002	4.41	75,002	4.41
2	774	0.07	34,971	3.00	0	0.00	64,284	4.94	142,554	8.38	217,556	12.79
3	686,788	58.91	721,759	61.91	943,806	72.58	1,008,090	77.53	68,950	4.05	286,506	16.85
4	431,122	36.98	1,152,881	98.89	292,200	22.47	1,300,290	100.00	1,548	0.09	288,054	16.94
5	0	0.00	1,152,881	98.89					704,938	41.45	992,992	58.39
6	0	0.00	1,152,881	98.89					503,072	29.58	1,496,064	87.97
7.1	12,997	1.11	1,165,878	100.00					204,540	12.03	1,700,604	100.00
	District #4				District #5				District #6			
Stage	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)
1	360,864	17.08	360,864	17.08	19,666	1.44	19,666	1.44	53,408	3.51	53,408	3.51
2	371,428	17.58	732,292	34.66	83,650	6.14	103,316	7.59	269,068	17.67	322,476	21.18
3	257,311	12.18	989,603	46.84	133,864	9.83	237,180	17.42	107,284	7.05	429,760	28.23
4	2,560	0.12	992,163	46.96	430	0.03	237,610	17.45	552	0.04	430,312	28.27
5	903,900	42.79	1,896,063	89.75	229,642	16.86	467,252	34.32	922,574	60.60	1,352,886	88.87
6	28,870	1.37	1,924,933	91.12	887,904	65.21	1,355,156	99.52	70,128	4.61	1,423,014	93.47
7.1	162,368	7.69	2,087,301	98.8	4,144	0.30	1,359,300	99.83	99,348	6.53	1,522,362	100.00
7.2	0	0.00	2,087,301	98.8	2,352	0.17	1,361,652	100.00				
7.3	0	0.00	2,087,301	98.8								
8	25,305	1.20	2,112,606	100								
	District #7				District #8				District #9			
Stage	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)
1	288,852	25.03	288,852	25.03	68,864	5.82	68,864	5.82	0	0.00	0	0.00
2	315,340	27.32	604,192	52.35	94,562	7.99	163,426	13.81	0	0.00	0	0.00
3	23,360	2.02	627,552	54.37	87,600	7.40	251,026	21.21	198,696	45.95	198,696	45.95

4	466	0.04	628,018	54.41	194	0.02	251,220	21.23	0	0.00	198,696	45.95
5	526,172	45.59	1,154,190	100	867,906	73.34	1,119,126	94.57	0	0.00	198,696	45.95
6					0	0.00	1,119,126	94.57	0	0.00	198,696	45.95
7.1					64,284	5.43	1,183,410	100.00	233,760	54.05	432,456	100.00

	District #10				District #11				District #12			
Stage	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)	RS (#)	RS (%)	Cum RS (#)	Cum RS (%)
1	146,644	9.80	146,644	9.80	241,134	23.85	241,134	23.85	160,898	38.24	160,898	38.24
2	438,638	29.32	585,282	39.12	400,820	39.65	641,954	63.50	139,650	33.19	300,548	71.43
3	352,216	23.54	937,498	62.66	105,120	10.40	747,074	73.89	0	0.00	300,548	71.43
4	2,288	0.15	939,786	62.82	990	0.10	748,064	73.99	290	0.07	300,838	71.50
5	544,392	36.39	1,484,178	99.21	262,948	26.01	1,011,012	100.00	119,930	28.50	420,768	100.00
6	11,886	0.79	1,496,064	100.00								

Note: RS= road segment; Cum RS=cumulative road segments; District 1, 2 and 9 had no traffic station measures and were treated the same as respectively neighboring districts in 4, 3 and 8.

Parcel-level land use data: The research team acquired statewide parcel data from CARB for 2019 for all the counties in California. The parcel data provides land use information at parcel level, such as agricultural, residential, commercial, industrial, government and institutions, open land, parks, and recreational facilities. For residential land use, the parcel data is further classed into single-family homes, town houses, condominiums, and high-rise apartment buildings. The parcel data also includes building characteristics, including building age, type and existence of fireplace, gas ranges, and other information that can be used to calculate building-specific factors to characterize the indoor infiltration of pollutants.

Land cover data: The research team acquired the land cover data for years 2001, 2004, 2006, 2008, 2011, 2013, 2016, and 2019 from the National Land Cover Database (NLCD). The NLCD provides a synoptic nationwide classification of land cover into 16 classes at a spatial resolution of 30 m. The 16 land cover classes were aggregated into eight major land cover types including forest, herbaceous/grassland, shrubland, developed, agriculture, wetlands, water and other, which includes ice/snow, barren areas. The research team also acquired tree canopy and percent impervious surfaces those years having land cover classification. For LUR development, a land cover data closest to the daily $PM_{2.5}$ measures was used for analysis.

Two-week interval vegetation index: The research team has acquired 16-day interval (23 surfaces for a year) vegetation index surfaces (MOD13Q1.006 Terra Vegetation Indices) for California at a spatial resolution of 250 m for years 2012 to 2019 for the study. This dataset was traced back to 1999 through a trend analysis.

GridMET meteorological data: The research team acquired daily high-spatial resolution (~4 km, 1/24th degree) surface meteorological data covering the contiguous U.S. for years 1999-2021. Primary climate variables collected include maximum temperature, minimum temperature, precipitation accumulation, downward surface shortwave radiation, wind-velocity, humidity (maximum and minimum relative humidity and specific humidity).

Digital elevation model (DEM) - in meters: The research team acquired the national elevation dataset for California from the U.S. Geological Survey (USGS) (http://nationalmap.gov and http://seamless.usgs.gov) for 2011. The data included 45 1/3 arc-second (approx. 10 meters) raster DEM and were mosaicked into a single DEM raster for the entire State. Higher elevation is normally associated with lower $PM_{2.5}$ concentrations.

Distance to coast - in meters: The California shoreline was derived from The National Assessment of Shoreline Change: GIS Compilation of Vector Cliff Edges and Associated Cliff Erosion Data for the California Coast (http://pubs.usgs.gov/of/2007/1112). These data are integrated into the GIS mapping tool to produce a geographic view of topographical changes in California's coastline over time. The most recent view was created using data collected between 1998-2002. Greater distance is typically associated with greater $PM_{2.5}$ concentrations.

Distance to roadways - in meters: The research team used Business Analysts 2018 Street Carto map layer provided by the Environmental Systems Research Institute (ESRI in Redlands, CA) to derive distance to nearest highway (defined as feature class classification (FCC) A1 and A2), to nearest major roadway (FCC A3) and to nearest local roadway (FCC A4). Greater distance from roadways is typically associated with lower roadway traffic air pollution.

Location category - unitless: The research team classified the State of California into three exclusive location categories: Goods movement corridor (GMC) - areas within 500 m of truck-permitted freeways and ports, non-goods movement corridor (NGMC) - areas within 500 m of truck-prohibited freeways or 300 m of a connecting roadway, and control areas (CTRL) - locations out of GMC and NGMC. Typically, GMCs have the highest $PM_{2.5}$ concentrations while CTRLs have the lowest $PM_{2.5}$ concentrations. From 2012 to 2019, the number of $PM_{2.5}$ monitoring stations for GMC, NGMC and CTRL was, respectively, 51, 67 and 28. The total number of daily measurements for GMC, NGMC and CTRL for the years 2012-2019 was, respectively, 95113, 147513, and 74107. The $PM_{2.5}$ monitors were successfully deployed to significantly measure its near source impacts (those sites in GMC and NGMC) and also had a fairly number of sites located in the control areas to form a spatial representation of coverage. These statistics will be updated in this new research to include all the days with $PM_{2.5}$ regulatory monitoring.

$PM_{2.5}$ data from CalEPA monitoring: CalEPA started monitoring $PM_{2.5}$ concentrations in 1998. The number of air quality monitors increased substantially from 1998 to the current, with the largest number reaching 120 in 2021 (Figure 1). The minimum values below detection limit, the mean values close to ten microgram per cubic meter (ug/m^3) and the maximum values over 500 ug/m^3. Though Google Air also measured $PM_{2.5}$ concentrations, they were measured by five binned particle counts, not mass. The Google Air $PM_{2.5}$ measurements will thus not be used in this study.

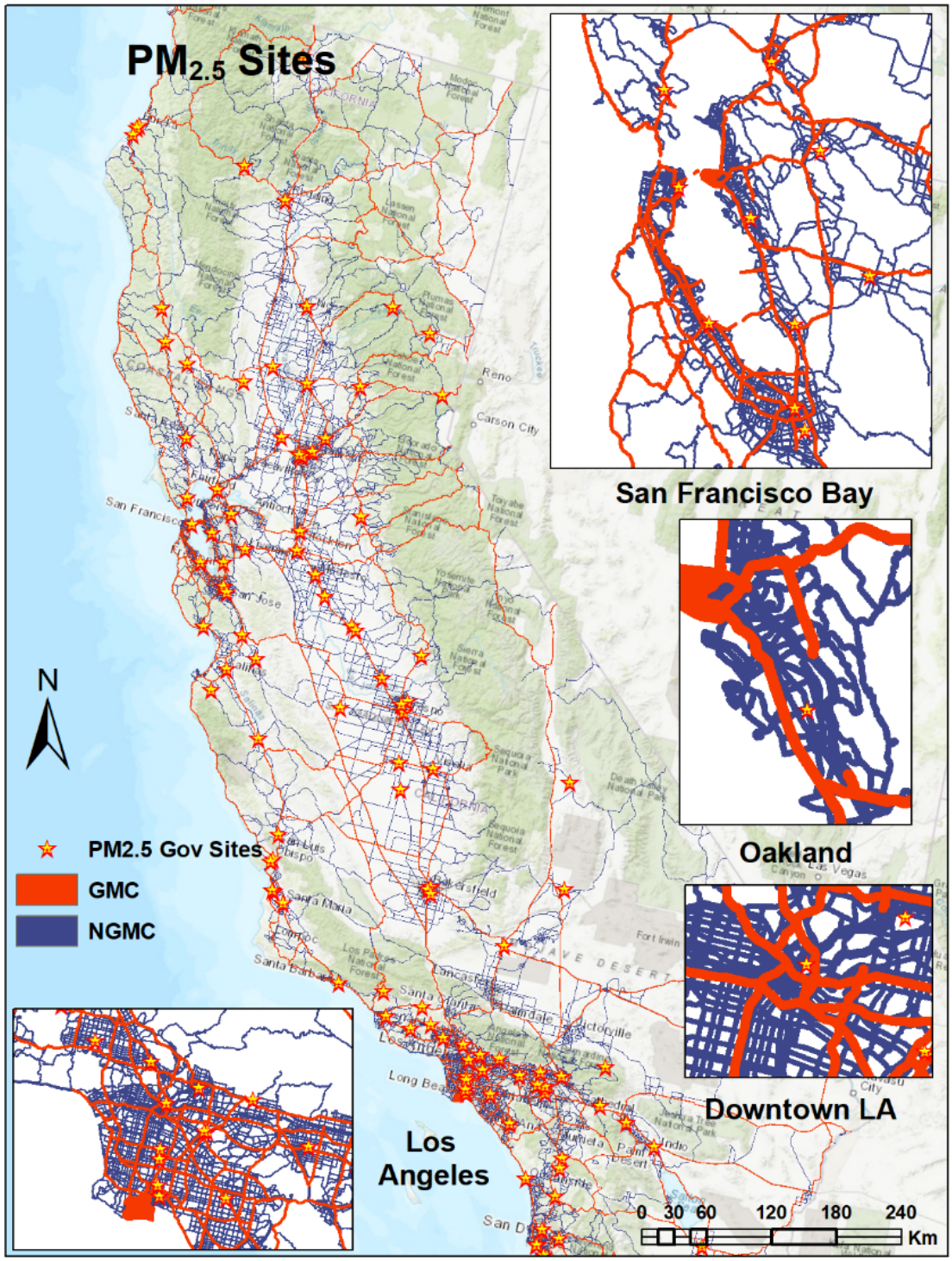


Figure 1. The spatial distribution of the CalEPA $PM_{2.5}$ air quality monitoring stations across California.

Development of daily $PM_{2.5}$ concentration models

All the data sources of vector shape (e.g., traffic data and parcel level land use data) are converted into rasters with a spatial resolution of 30 m. The following describes a series of interconnected steps to develop a daily $PM_{2.5}$ model through the D/S/A modeling framework:

Generate buffer statistics on 30 m spatial resolution potential predictors: A series of buffer statistics of 50-5000 m at an interval of 50 m are created for the potential spatial predictors with a spatial resolution of 30 m. They include daily traffic data, parcel-level land use data, NLCD land cover data, and NLCD percent impervious and tree-canopy data. For each variable, e.g., industrial land use, a total of 100 buffered statistics (i.e., covariates) are generated. For all the potential predictors, with the inclusion of buffered and non-buffered variables, about 2,500 covariates are identified for the prediction of daily pollutant concentrations. This increases the chance of identifying the optimal distance impact of a predictor and helps improve model performance. However, this also creates high-dimension covariates that are highly correlated. To solve this issue, the research team applies a data reduction strategy to reduce the number of covariates used in predicting pollutant concentration.

Apply data reduction strategy to reduce the number of predictors: To reduce the number of covariates and avoid high correlations between them for LUR modeling, The research team first creates a correlation coefficient matrix between a pollutant and all the covariates. A covariate of the highest absolute correlation coefficient with the pollutant is maintained. The maintained covariate is then used to calculate correlation coefficients with all the remaining covariates and those with an absolute correlation coefficient greater than 0.9 are removed from inclusion. A second covariate from the remaining covariates with the second highest absolute correlation coefficient with the pollutant is then maintained. Similarly, the second maintained covariate is used to calculate correlation coefficients with all the remaining covariates and those with an absolute correlation coefficient greater than 0.9 are removed from inclusion. This process continues until all the significant covariates are chosen and no two chosen covariates have an absolute correlation coefficient greater than 0.9. After applying the data reduction strategy, the number of predictors maintained in a LUR model is typically less than 100. This process is implemented before a D/S/A is run and it is carried out once for the sole purpose of reducing the number of predictors that might be collinear.

Develop daily LUR models and surfaces for $PM_{2.5}$: LUR modeling is a statistical technique used to estimate the spatial distribution of air pollution concentrations based on land-use characteristics and other variables. It analyzes measurements of air pollution levels taken at specific locations, and then identifies the key factors that influence those levels. To develop daily LUR models, The research team ran the model at 30 m spatial resolution through the D/S/A algorithm.[74,75] The D/S/A algorithm can deal with both linear and non-linear associations. However, for simplicity of model development and for the clear interpretation of the predictors selected for a model, The research team limited the predictors to be only on linear terms (the

maximum sum of powers in each variable to be 1) and disallowed any interaction except corridor by year. The D/S/A algorithm is an aggressive model search algorithm, which iteratively generates polynomial generalized linear models based on the existing terms in the current 'best' model and the following three steps: (1) a deletion step, which removes a term from the model, (2) a substitution step, which replaces one term with another, and (3) an addition step, which adds a term to the model. The search for the 'best' estimator starts with the base model specified with 'formula': typically, the intercept model except when the user requires number of terms to be forced in the final model. Before searching through the statistical model space of polynomial functions, the original sample is randomly partitioned into V equal size subsamples. Of the V subsamples, a subsample is retained as the validation data for testing the model, and the remaining V-1 subsamples are used as training data. The cross-validation process is then repeated V times, with each of the V subsamples used exactly once as the validation data. The advantage of this method over the leave-one-out cross-validation technique is that the prediction errors are less impacted by single outliers, and compared to repeated random sub-sampling, all observations in the V-folds are used for both training and validation, and each observation is used for validation once. With each iteration, an independent validation dataset is used to assess the performance of a model built using a training dataset. This technique, therefore, minimizes over-fitting to the data to maximize the probability that the models will predict well at locations that have not been sampled.

During the D/S/A modeling process, The research team classified the entire dataset into 10-folds. With each iteration, an independent validation dataset in one of the 10-folds was used to assess the performance of the model built using data from the other 9-folds. This process continued for 10 times until every fold of data is used for validation. The mean prediction errors from the validation datasets were averaged across 10 iterations and compared between a series of built models. The model with the minimum average prediction error was chosen as the final model. During the modeling process, the air quality monitoring data (and associated predictors) for a specific year were equally and randomly distributed into those 10 folds. Because one air quality monitor typically has more than one observation (i.e., multiple days of measurements), a random effect of air quality monitor (in R language this is given by 1|station_ID) was included in the modeling process however, only the fixed effects (i.e., remove the random effect) were used to construct $PM_{2.5}$ surfaces due to the requirement of deriving $PM_{2.5}$ concentrations beyond monitoring stations. The adjusted R^2 for the fixed effects was used as a measure of model performance from the LUR modeling result.

The study (Table 3) identified a positive correlation between higher aerosol optical depth (AOD) values and elevated $PM_{2.5}$ concentrations, suggesting that increased aerosol presence in the atmosphere is associated with higher particulate matter levels. Increased traffic density emerged as a contributing factor to higher $PM_{2.5}$ concentrations, emphasizing the impact of vehicular emissions on air quality. Weather factors such as higher relative humidity, wind speed, and temperature were associated with lower $PM_{2.5}$ concentrations. Developed open spaces were

linked to reduced $PM_{2.5}$ concentrations, and so were areas characterized by a higher vegetation index, shrub cover, barren land, and water bodies, emphasizing the role of natural features in mitigating air pollution. Barren land refers to areas that have little to no vegetation cover and is often characterized by exposed soil or rock.[78] Industrial land use, however, was associated with higher $PM_{2.5}$ concentrations, pointing to the impact of industrial activities on particulate matter emissions. Greater residential areas were linked to higher $PM_{2.5}$ concentrations, potentially attributed to background concentrations. In densely populated regions, the increased density of housing, traffic, and other activities can lead to elevated $PM_{2.5}$ background concentrations. Additionally, the urban heat island effect and limited air circulation in residential areas can hinder the dispersion of pollutants, allowing background $PM_{2.5}$ levels to rise. Additionally, locations farther from the coast were associated with higher $PM_{2.5}$ concentrations, indicating a spatial relationship between proximity to the coast and particulate matter levels.

The final $PM_{2.5}$ model had a predictive performance of 0.65. The predictive performance value of 0.65 was obtained through cross-validation comparing model-predicted $PM_{2.5}$ concentrations with observed monitoring data. This metric reflects the model's ability to reproduce measured concentrations while prioritizing fine-scale spatial variability across California rather than maximizing overall variance explained. In a large and heterogeneous study domain with diverse emission sources, meteorology, and land-use characteristics, models designed to capture small-area contrasts typically yield lower R^2 values than regionally smoothed models. Importantly, a predictive performance of 0.65 corresponds to an overall correlation exceeding 0.80 between predicted and observed concentrations, indicating strong agreement and reliable exposure estimation. Although inclusion of additional regional predictors could have increased R^2, doing so would have reduced spatial resolution and limited the model's suitability for epidemiologic exposure assignment. Thus, the reported predictive performance represents an appropriate balance between accuracy and spatial specificity for statewide health analyses.

Table 3. Daily $PM_{2.5}$ model covering available observational periods.

Coefficient	*Estimates*	*std. Error*	*Statistic*	*P-Value*
Year	-0.139709	0.003115	-44.847889	**<0.001**
Season [Fall]	360.125186	6.244769	57.668292	**<0.001**
Season [Spring]	356.974440	6.245309	57.158809	**<0.001**
Season [Summer]	358.493294	6.246059	57.395114	**<0.001**
Season [Winter]	360.534093	6.244547	57.735832	**<0.001**
AOD (albedo)	0.044977	0.000221	203.299221	**<0.001**
Vehicle Kilometers Traveled (VKT) (350m)	0.000012	0.000001	16.793841	**<0.001**

Wind Velocity (m/s)	-1.239394	0.006771	-183.031784	**<0.001**
Minimum Temperature (K)	-0.239641	0.002586	-92.662860	**<0.001**
Minimum Relative Humidity (%)	-0.059829	0.000649	-92.242887	**<0.001**
Roadway Area (ha) (5000m)	0.000024	0.000002	13.114503	**<0.001**
Industrial (ha) (1850m)	0.000513	0.000024	21.714939	**<0.001**
Residential (ha) (850m)	0.001185	0.000029	41.076124	**<0.001**
Unknown Land Use (ha) (450m)	-0.002008	0.000150	-13.387931	**<0.001**
Agricultural (ha) (50m)	-0.311401	0.014300	-21.776931	**<0.001**
NDVI	-0.000394	0.000010	-39.979943	**<0.001**
Barren Land (ha) (3000m)	-0.001291	0.000013	-99.546262	**<0.001**
Barren Land (ha) (50m)	-0.982108	0.074570	-13.170308	**<0.001**
Shrub Land (ha) (200m)	-0.029789	0.000822	-36.232176	**<0.001**
Developed Open Space (ha) (4950m)	-0.000037	0.000002	-16.515144	**<0.001**
Waterbody (ha) (1750m)	-0.000578	0.000020	-29.560264	**<0.001**
Distance to Highway (m)	-0.000029	0.000003	-8.723557	**<0.001**
Distance to Coast (m)	0.000017	0.000000	88.728793	**<0.001**
Elevation (m)	-0.002428	0.000053	-46.003552	**<0.001**
Observations	633277			
R^2 / R^2 adjusted	0.652 / 0.652			

Due to the requirement of more than three gigabytes of storage space for a single statewide raster surface of spatial resolution of 30 m, The research team opted to generate daily surfaces of $PM_{2.5}$ concentrations using a spatial resolution of 100 m. The 100 m spatial resolution surfaces maintain the ability to identify small area variations of pollutant concentrations, especially those heightened exposures endured by vulnerable communities.

Task 3. Obtain CDPH Vital Statistics Data Including Mortality and Covariates

In this task the research team obtained and processed the California population data, including mortality records and individual-level covariates, for the Period 2000 through 2021. This step was foundational because it allowed us to link health outcomes across a very large and diverse population to the detailed $PM_{2.5}$ exposure surfaces developed in Task 2. The availability of continuous enrollment information, together with death records and address histories, enabled us to study the impacts of air pollution on life expectancy over two time periods: 2000-2010 and 2011-2021.

The first component of this task was securing regulatory approvals and data access. The research team submitted applications to both the UC Berkeley Institutional Review Board (for reliance on State CPHS) and the California Health and Human Services Committee for the Protection of Human Subjects, both of which reviewed and approved our research protocol. Following approval, the research team worked with the California Department of Public Health to acquire the mortality data under a strict data-use agreement to protect confidentiality. The data was stored on secure UC Berkeley Secure Research Data Center (SRDC) servers in compliance with HIPAA and state requirements.

For Period 1 (2000-2010), an annual average number of 219,795 deaths were recorded across all age groups and race-ethnicity categories in the dataset provided by the California Department of Public Health (CDPH) (Table 4). Deaths were not evenly distributed, with the vast majority occurring in older age groups and among the White population. By race and ethnicity, Whites accounted for the largest share of deaths, with 153,157 deaths, representing about 70% of the total. Hispanics were the second largest group with 31,404 deaths (14%), followed by Blacks with 16,576 deaths (8%), Asians with 13,721 deaths (6%), and Other with 4,937 deaths (2%). In the study period analyzed, deaths among Native American populations constituted a very small proportion of total deaths statewide; even after aggregation into the “Other” category, this group represented only approximately 2% of all deaths. Analyzing Native American populations as a standalone group would have resulted in unstable estimates, wide confidence intervals, and increased risk of disclosure, particularly at finer geographic or age stratifications. These figures reflect both population size differences and disparities in mortality patterns across groups. By age group, deaths rose steeply with age. Only 769 deaths (0.3%) occurred among children ages 0-11, while more than a quarter of all deaths (55,585 deaths, 25%) occurred among adults aged 87 and over. The next highest concentrations were in the 81-86 age group with 41,445 deaths (19%) and the 75-80 group with 32,887 deaths (15%). In contrast, young and middle-aged adults (ages 12-45 combined) accounted for only about 7% of total deaths. Patterns also varied by race within age categories. White deaths increased steadily with age, peaking in the 87+ group, while Black deaths showed relatively higher representation in the 36-65 range compared to other groups. Asian and Hispanic populations recorded fewer deaths overall, but their age distribution

followed a similar pattern, with the majority of deaths concentrated in older age groups. Overall, the data highlight how mortality between 2000 and 2010 was heavily age-dependent, with nearly 60% of deaths occurring among those older than 75. At the same time, the figures point to racial disparities, particularly in the middle-aged groups, where Black mortality was disproportionately higher compared with other race-ethnicity groups.

For Period 2 (2011-2021), the dataset recorded a total of 258,345 deaths across all age groups and race-ethnicity categories, reflecting population growth, demographic aging, and changes in mortality patterns compared to the previous decade (Table 4). Deaths occurring in 2020–2021 include those recorded in the CDPH Vital Statistics mortality files, which reflect all registered deaths by underlying cause as coded on death certificates. COVID-19 specific deaths were not separately identified or excluded in this analysis because COVID-19 cause-of-death information was not available in a consistent, finalized form across all demographic and geographic strata at the time of data preparation, and was not harmonized with earlier study years. As a result, mortality during 2020–2021 was treated consistently with prior years as all-cause mortality. This approach is consistent with the study's focus on long-term, population-level $PM_{2.5}$ impacts rather than short-term mortality shocks. While the COVID-19 pandemic likely contributed to elevated mortality in the later years of Period 2, particularly among older adults and vulnerable populations, this effect is not expected to materially bias relative $PM_{2.5}$-related life expectancy estimates, which are driven primarily by long-term exposure contrasts rather than year-specific causes of death. Results for Period 2 should therefore be interpreted as reflecting overall mortality patterns during 2011–2021, inclusive of the pandemic period.

Deaths remained heavily concentrated in older age groups, but notable shifts occurred in the youngest and middle-aged categories. For instance, the 0-11 age group saw 2,613 deaths, more than triple the number observed in Period 1 (769 deaths), highlighting either data reporting differences, population growth, or other epidemiologic factors affecting child mortality. Young adults (ages 12-25) accounted for 3,420 deaths, while middle-aged adults aged 26-55 contributed 29,702 deaths (ages 26-35: 5,151; 36-45: 7,562; 46-55: 16,989). The majority of deaths continued to occur among older adults: ages 56-65 totaled 33,998, 66-74 had 41,097, 75-80 had 32,528, 81-86 had 40,505, and 87 and over had 74,482 deaths. Overall, more than two-thirds of deaths occurred among adults aged 66 and older, consistent with the expected age gradient in mortality.

By race and ethnicity, Whites remained the largest group with 157,069 deaths (61%), followed by Hispanics with 50,439 deaths (20%), Asian populations with 24,866 deaths (10%), Blacks with 19,631 deaths (8%), and Other populations with 6,340 deaths (2%). Compared to Period 1, the share of deaths among minority populations increased, particularly for Hispanics and Asians, reflecting demographic growth and changing age structures within these groups. The age-specific racial patterns also shifted: while White deaths remained concentrated among the oldest age groups, Hispanics and Asians experienced substantial increases in both middle-aged and older categories. Black mortality remained disproportionately elevated in middle-aged

groups (36-65), though total counts increased only modestly compared with other groups. These patterns illustrate not only the continuing predominance of age as a determinant of mortality but also the evolving race-ethnicity composition of California's population, which has implications for public health planning and interventions aimed at reducing disparities.

For Table 4, deaths count differs between Period 1 and Period 2 in part because the underlying population size and demographic composition of California changed substantially over time. However, year-by-year population counts by age and race/ethnicity were not consistently available for the full study period, particularly at the level of detail required for this analysis. As a result, population growth and demographic shifts could not be displayed alongside annual death counts in Table 4. Instead, baseline population distributions were applied separately within each period to support internally consistent life expectancy calculations. Consequently, increases in the number of deaths among Asians and Hispanics between periods should be interpreted as reflecting a combination of population growth, aging, and changes in mortality patterns, rather than increases in per-capita mortality risk.

Table 4. Annual average death statistics across California for the two study periods

Period	Age Group	White	Black	Asian	Hispanics	Other[ξ]	Total
2000-2010	0-11	221	83	55	373	36	769
	12-25	1,217	495	206	1,608	152	3,678
	26-35	1,520	556	224	1,507	153	3,960
	36-45	4,053	1,028	431	2,294	290	8,095
	46-55	9,751	2,147	974	3,580	554	17,006
	56-65	15,613	2,682	1,521	4,157	747	24,720
	66-74	21,025	2,831	2,117	4,813	863	31,649
	75-80	23,589	2,165	2,219	4,200	714	32,887
	81-86	31,742	2,135	2,676	4,219	673	41,445
	87 & Over	44,426	2,454	3,299	4,652	754	55,585
	Total	153,157	16,576	13,721	31,404	4,937	219,795
2011-2021	0-11	588	288	216	1,268	254	2,613
	12-25	998	405	219	1,577	221	3,420
	26-35	1,893	610	352	2,018	278	5,151
	36-45	2,913	866	625	2,797	360	7,562
	46-55	7,897	1,933	1,382	5,119	658	16,989
	56-65	18,382	3,812	2,776	7,882	1,146	33,998
	66-74	24,673	3,730	3,696	7,845	1,153	41,097
	75-80	20,405	2,417	3,277	5,707	722	32,528
	81-86	26,561	2,375	4,366	6,508	695	40,505
	87 & Over	52,759	3,195	7,957	9,718	853	74,482
	Total	157,069	19,631	24,866	50,439	6,340	258,345

[ξ] The "Other" category grouped together non-Hispanic American Indian/Alaska Native, non-Hispanic Hawaiian/Pacific Islander, non-Hispanic Other, those reporting multiple races, and those with missing or unknown race.

Residential address histories were a particularly valuable component of the data, which enabled us to geocode each individual's location at high spatial resolution. The research team then linked these geocoded addresses to the daily 100 m $PM_{2.5}$ surfaces produced in Task 2. For each decedent, the research team assigned the one-year rolling average $PM_{2.5}$ exposure leading up to the date of death. For matched controls, the research team assigned exposures for the same span of time, thereby allowing for precise contrasts between those who died and those who survived. To structure the analysis across generations, the research team divided the population into two periods: the years 2000-2010 defined the first generation, while 2011-2021 defined the second generation. This division allowed us to examine how declining $PM_{2.5}$ concentrations influenced life expectancy across time and to assess whether improvements were equitably distributed.

Task 4. Calculate $PM_{2.5}$-Specific Reductions in Life Expectancy Across Two Time Periods and Identify Race-Ethnicity Disparities

The analytic foundation of Task 4 involved estimating the causal effect of $PM_{2.5}$ on mortality and life expectancy across two distinct time periods and two generations of the California population, and then examining disparities by race, ethnicity, and vulnerability. To estimate the mortality and life-expectancy impacts attributable to long-term $PM_{2.5}$ exposure, the research team first quantified age-specific associations between $PM_{2.5}$ and all-cause mortality using conditional logistic regression models. Models were fit separately within predefined age strata and time periods, with long-term $PM_{2.5}$ exposure defined as the one-year rolling average preceding the date of death. These age-specific effect estimates represent the relative change in mortality risk associated with incremental changes in $PM_{2.5}$ exposure and form the basis for all subsequent counterfactual and life-expectancy calculations. Life expectancy was calculated under two scenarios: an observed (all-cause) scenario reflecting existing $PM_{2.5}$ exposure levels, and a counterfactual scenario in which $PM_{2.5}$ exposure was hypothetically removed. For each age group, the estimated $PM_{2.5}$–mortality association was used to adjust age-specific mortality hazards under the counterfactual scenario. Life tables were then constructed using standard demographic techniques to generate age-specific survival probabilities and expected remaining life years under both scenarios. The $PM_{2.5}$-attributable life-expectancy impact was defined as the difference between life expectancy under observed conditions and life expectancy under the $PM_{2.5}$-removed counterfactual. The proportion of deaths attributable to $PM_{2.5}$ exposure was quantified using a population attributable fraction (PAF) framework. For each age group and stratum, the PAF represents the fraction of deaths that would not have occurred under the counterfactual scenario of no $PM_{2.5}$ exposure, given the estimated exposure-response relationship. Because $PM_{2.5}$ exposure was modeled as a continuous variable rather than

dichotomized, the PAF implicitly reflects the full exposure distribution rather than a binary exposed versus unexposed comparison. In this framework, all individuals are considered exposed to some degree, and the PAF captures the proportional reduction in mortality that would result from reducing exposure to the counterfactual level.

To summarize impacts across age groups, the research team implemented both population-weighted and death-weighted aggregation approaches. Population-weighted impacts were calculated by weighting age-specific life-expectancy changes by the size of the corresponding population, reflecting the average per-person impact across the full population. Death-weighted impacts were calculated by weighting age-specific life-expectancy changes by the number of deaths occurring in each age group, thereby emphasizing the contribution of age groups that account for the largest share of mortality. These two metrics capture complementary aspects of $PM_{2.5}$ burden and allow assessment of whether impacts are concentrated among younger, middle-aged, or older populations.

Policy benefits were defined as the change in $PM_{2.5}$-attributable life-expectancy impact between Period 1 and Period 2, with a focus on death-weighted estimates. This difference quantifies the reduction in mortality burden attributable to lower $PM_{2.5}$ exposure and changing exposure-response dynamics over time. Positive policy benefits indicate that regulatory actions and associated emission reductions translated into meaningful decreases in $PM_{2.5}$-related life-expectancy loss. Importantly, this metric does not attribute benefits to any single regulation but instead reflects the cumulative effect of regulatory, technological, and behavioral changes that occurred between the two periods.

For race-ethnicity and age-specific analyses, the same modeling and counterfactual framework was applied within each subgroup. Age-specific $PM_{2.5}$ effect estimates were combined with subgroup-specific population and mortality distributions to compute both population-weighted and death-weighted life-expectancy impacts. These stratified estimates were subsequently used to evaluate heterogeneity in vulnerability and to assess how the distribution of $PM_{2.5}$ impacts shifted across demographic groups and time periods.

The analysis was divided into two major periods: 2000-2010 (Period 1) and 2011-2021 (Period 2). By splitting the data in this way, the research team were able to test directly how declines in $PM_{2.5}$ concentrations over time translated into reductions in mortality risk and gains in life expectancy. Within each period, logistic regression models were run under three primary stratification schemes to balance epidemiologic detail with statistical stability. First, models were estimated using all twenty original age groups without race-ethnicity stratification. Second, models were estimated after aggregating the twenty age groups into ten broader age groups (Aggr2) with race-ethnicity stratification applied. This aggregation combined every two adjacent age groups and was used to increase sample size within each race-ethnicity stratum while retaining meaningful age differentiation. Third, models were estimated using five aggregated age groups (Aggr4), formed by combining every four adjacent age groups, and with race-ethnicity

stratification. This further aggregation was implemented to ensure sufficient numbers of deaths and matched controls within smaller race-ethnicity groups and older age strata, where sparse data could otherwise lead to unstable estimates. The abbreviations “Aggr2” and “Aggr4” are used consistently to denote the ten-age-group and five-age-group aggregation schemes, respectively, hereafter.

The research team conducted both the five-group and ten-group age analyses to balance statistical stability with the ability to detect meaningful differences within age ranges. The five age-group approach reduces small-sample problems, especially for smaller race-ethnicity populations, by aggregating individuals into broader categories, ensuring reliable estimates. However, these broader groups can mask important variations that occur within the same age bracket. The ten age-group analysis allows us to uncover those within-group differences, although this finer stratification can introduce small-sample limitations for some populations. These concerns are minimal for Hispanic and White populations, which have sufficiently large sample sizes to support the ten-group structure. Using both approaches lets us verify whether patterns are consistent across grouping strategies and provides greater confidence that our findings are robust and not driven by grouping artifacts. Together, these complementary approaches provided both granularity and robustness, allowing us to see how different ways of grouping ages and populations influenced the estimated mortality effects of $PM_{2.5}$.

The original twenty age groups were defined as 0-5, 6-11, 12-17, 18-25, 26-30, 31-35, 36-40, 41-45, 46-50, 51-55, 56-60, 61-65, 66-70, 71-74, 75-77, 78-80, 81-83, 84-86, 87-89, 90-95, and 96 years and over (similar to the American Community Survey categories: https://www.census.gov/programs-surveys/acs.html). These fine-grained strata allowed us to observe age-specific patterns of susceptibility with high resolution. The Aggr2 grouping reduced these categories into ten broader ranges—0-11, 12-25, 26-35, 36-45, 46-55, 56-65, 66-74, 75-80, 81-86, and 87 years and over—while the Aggr4 grouping condensed them further into five ranges: 0-25, 26-45, 46-65, 66-80, and 81 years and over. Each set of groupings was carefully tested to balance statistical power with interpretability.

Study Design

Identification of impact of $PM_{2.5}$ exposure on mortality

The process of preparing the data for logistic regression was highly detailed and required careful handling of age, race-ethnicity, and other covariates. Each death was matched with a maximum of 2 controls based on birth year and month, race-ethnicity and sex. Allowing 0–2 matched controls per death maximized use of the available mortality data while maintaining strict matching criteria and avoiding forced or inappropriate matches. The raw mortality and matched control data sets were compiled across the years within each period. For Period 1, death data spanned 2000-2010, while control data extended slightly further to 2012 to allow proper matching. Mortality records beyond 2010 were used only to identify control individuals who were alive at the time of Period 1 case death and therefore eligible to serve as controls. For all

controls matched to Period 1 deaths, $PM_{2.5}$ exposure was defined using the same one-year rolling average prior to the matched case death date, and no exposure information beyond 2010 for controls was used in the Period 1 analysis. These annual files were consolidated into master analytic data tables that included both case and control individuals, their assigned one-year average $PM_{2.5}$ exposures, and demographic covariates. Each individual was then assigned to the appropriate age group according to the grouping scheme being used. This matched case–control design inherently accounts for secular trends in all-cause mortality over time. By selecting controls who were alive at the time of each death and matched on birth year and month, sex, and race-ethnicity, cases and controls were drawn from the same underlying population and mortality risk context within each period. As a result, long-term improvements in healthcare, prevention, and baseline mortality risk operate similarly on cases and controls and do not confound the estimated $PM_{2.5}$ effects. Calendar year indicators were not included because the analysis did not estimate year-specific models; instead, observations were pooled within each period, and $PM_{2.5}$ exposure was defined as a rolling 365-day average prior to death, making explicit year effects neither identifiable nor necessary under this modeling framework.

Race and ethnicity were reclassified into the following categories: non-Hispanic White, non-Hispanic Black, non-Hispanic Asian, Hispanic, and Other. This recoding ensured adequate sample sizes for robust estimation while preserving the major race-ethnicity contrasts central to the analysis. Marital status and education were also cleaned and reclassified, with missing education values imputed to the lowest category (assumed less than high school diploma) and missing marital status values assigned to "Unknown." In the CDPH mortality data, missing education values primarily occur for individuals with less than a high school diploma, a pattern that reflects known limitations in death certificate reporting rather than random missingness. Education is recorded categorically, and missingness is concentrated among individuals with very low educational attainment. Assigning missing education to the lowest category (less than high school) is therefore a conservative and commonly used approach that avoids overstating socioeconomic advantage. For marital status, missing values occur predominantly among decedents under age 18, for whom marital status is typically not reported on death certificates. Because marital status is not meaningfully defined for these individuals, missing values were not imputed to a specific category but instead coded as "Unknown," allowing these records to be retained in the analysis without introducing misclassification. Both education and marital status were included only as adjustment covariates and were not primary variables of interest. The matched case-control design, with matching on age, sex, and race-ethnicity, limits sensitivity of the $PM_{2.5}$ effect estimates to assumptions about these variables. Given the small proportion of missing values and their role as control variables, this handling of missing education and marital status is unlikely to materially affect the estimated $PM_{2.5}$-related mortality risks or life-expectancy impacts.

Before fitting the logistic regression models, the research team applied filters to reduce exposure misclassification. Specifically, individuals with less than one year of residence in their county

were excluded, on the grounds that their assigned exposures may not have accurately reflected their true environmental context. This step reduced the analytic sample size slightly but significantly improved exposure validity.

The core modeling step involved fitting logistic regressions within each age group and race-ethnicity stratum. The dependent variable was mortality status (death vs. survival), and the independent variable of interest was $PM_{2.5}$ exposure, expressed as the one-year rolling average in micrograms per cubic meter. We selected a one-year rolling average $PM_{2.5}$ exposure because the analysis is based on individual deaths with precisely dated events and daily exposure surfaces, rather than cohort-based long-term averages. Many chronic exposure studies rely on multi-year averages because exposure data are only available as long-term spatial surfaces and because outcomes are assessed at fixed follow-up intervals rather than at the time of death. In contrast, our study links each death to a continuous daily $PM_{2.5}$ time series and assigns exposure as the average over the 365 days preceding death, which captures cumulative exposure immediately relevant to mortality risk while preserving temporal alignment between exposure and outcome. Using a one-year rolling window also avoids introducing exposure misclassification that would arise from averaging over years well before the death event, particularly during a period of rapid air quality improvement in California. The one-year window therefore represents a pragmatic and epidemiologically appropriate definition of long-term exposure for a mortality-based, case-control design with daily exposure data.

Covariates included sex, age, education, race-ethnicity, and marital status, with the exact set of included variables adjusted according to the availability of variation within each stratum. We conducted conditional logistic regression to account for the matched case-control design. Deaths were matched to up to two controls based on birth year and month, sex, and race-ethnicity. Although age, sex, and race-ethnicity defined the matching criteria, these variables were also included as covariates in selected models. This was done because matching was not exact for all cases (i.e., some cases had zero, one, or two matched controls), and inclusion of these variables as covariates helps control for residual confounding arising from incomplete or unbalanced matching while preserving adjustment across all observations. When models were stratified by race-ethnicity or age group, race-ethnicity was not included as a covariate due to lack of variation within strata, while age was still included as a continuous covariate to account for residual age differences within the same age-group category. This approach ensures that the primary association between $PM_{2.5}$ exposure and mortality is estimated within matched strata while maintaining appropriate adjustment for key demographic factors across the full analytic sample. In some instances, for example, marital status was dropped from models of younger age groups where all individuals were coded as “never married.” Coefficients, confidence intervals, and significance values for $PM_{2.5}$ were extracted from each model, and results were organized by age group and race-ethnicity. This modeling framework was implemented in both Period 1 and Period 2, yielding two full sets of coefficients across all stratification schemes. Furthermore, the

research team merged the two periods of data into a consolidated dataset to identify the overall impact of $PM_{2.5}$ on mortality across major age groups.

Identification of $PM_{2.5}$ exposure impact on life expectancy

Building on the logistic regression models described above, the second major stage of Task 4 involved the calculation of life expectancy by race, ethnicity, and age group, and the quantification of gains achieved between Period 1 and Period 2. This work required the integration of population counts, death distributions, and $PM_{2.5}$ effect coefficients into formal life table methods. The research team implemented this process separately for three stratification schemes in a way corresponding to their logistics modeling framework: (1) models with all twenty detailed age groups without race-ethnicity stratification; (2) models with ten aggregated age groups (Aggr2) across major race-ethnicity categories; and (3) models with five aggregated age groups (Aggr4) across major race-ethnicity categories. This parallel structure ensured that the life expectancy estimates were robust and comparable under different levels of aggregation.

Population counts by age group and race-ethnicity were drawn from the Business Analysts data acquired by the research team, adjusted to reflect annual populations by dividing decadal totals into annualized estimates. These population files were restructured so that the age categories matched the aggregation scheme being used. For example, in the Aggr2 framework, the raw Business Analyst categories (such as 0-5 and 6-11) were collapsed into a single 0-11 group. Parallel collapsing was applied to death distributions, which were estimated from the mortality data provided by CDPH and expressed as annualized death counts by age group and race-ethnicity. After reclassification, deaths were joined with population data, yielding a mortality profile that aligned with the exposure-effect coefficients derived from logistic regression.

The next step was to incorporate the estimated $PM_{2.5}$ coefficients. For each age and race-ethnicity group, the research team extracted the beta coefficient for $PM_{2.5}$ from the logistic regression models. These coefficients quantified the log-odds increase in mortality per unit $PM_{2.5}$ exposure. Because not all age-by-race strata had stable coefficient estimates, particularly in smaller population groups, the research team implemented an interpolation procedure. Missing or unstable beta values were replaced with interpolated values derived from the nearest available age groups within the same race category. A beta coefficient was classified as unstable if the conditional logistic regression failed to converge, produced an infinite or undefined estimate, or yielded a standard error larger than the absolute value of the coefficient itself, indicating insufficient information for reliable estimation. In some strata, no coefficient was estimated at all due to the absence of informative matched case-control sets or complete separation. For strata with missing or unstable coefficients, replacement values were derived from the closest valid neighbors, defined as the immediately adjacent age groups within the same race-ethnicity category for which the model converged and produced finite coefficients and standard errors. When both adjacent age groups were available, the replacement value was calculated as the

average of those coefficients. When only one adjacent valid age group existed, its coefficient was carried forward or backward as appropriate. This procedure primarily affected the youngest age groups and effectively assigned coefficients from slightly older age groups when direct estimation was not possible, while preserving within-race smoothness across age.

Once the data set contained complete age-specific population counts, deaths, and $PM_{2.5}$ effect coefficients, the research team proceeded to the life expectancy calculations. For each race and ethnicity, the research team began by calculating baseline mortality rates within each age group as the ratio of deaths to population. These mortality rates were then converted into probabilities of death within the age interval, accounting for the width of each age group (denoted N_i). The hazard ratio for $PM_{2.5}$ exposure was calculated as the exponential of the beta coefficient, and from this the research team derived the population attributable fraction (PAF), which represents the proportion of deaths within each age group attributable to $PM_{2.5}$ exposure. The counterfactual probability of death with $PM_{2.5}$ removed was then calculated by dividing the observed hazard by the hazard ratio, ensuring that the adjusted probability reflected the absence of pollution-related risk. These calculations were conducted separately for Period 1 and Period, and the research team merged the two periods of data into a consolidated dataset to identify the overall impact of $PM_{2.5}$ on life expectancy across major age groups. The research team calculated life expectancy impacts attributable to $PM_{2.5}$ by combining mortality, population, and exposure-response estimates (β_1) derived from logistic regression results. The approach translates individual-level $PM_{2.5}$ exposure effects into life expectancy impacts. Let $D_i$and $N_i$denote deaths and population in age interval i, and let $\beta_1$be the exposure-response coefficient (log hazard ratio) for $PM_{2.5}$. The observed mortality rate in age interval i is:

$$M_i = \frac{D_i}{\text{Population}_i}$$

The baseline probability of death in the interval is:

$$q_i = 1 - \exp(-M_i \cdot n_i)$$

where $n_i$is the width of the age interval (number of years). The relative risk associated with $PM_{2.5}$ is computed on the hazard scale:

$$RR_i = \exp(\beta_1)$$

$\beta_1$is estimated as a log hazard ratio (HR) in the life table context. In our analysis, $\beta_1$had been derived from a logistic regression, exponentiating it would yield an odds ratio (OR), which approximates the HR because outcome death is rare compared to population size. Because the ORs/HRs in our study are small, this approximation is reasonable. From this, the PAF in age interval $i$is:

$$\text{PAF}_i = \frac{RR_i - 1}{RR_i}$$

The $PM_{2.5}$-attributable probability of death is:

$$q_{\mathrm{PM},i} = q_i \cdot \mathrm{PAF}_i$$

The counterfactual probability of death if $PM_{2.5}$ were removed is estimated using the hazard-scale adjustment:

$$q_i' = 1 - \exp\left(-\frac{M_i}{RR_i} \cdot n_i\right)$$

This ensures that the removal of $PM_{2.5}$ is modeled consistently on the hazard scale rather than simply adjusting probabilities linearly. In life table construction, let $l_x$denote the number of survivors at the beginning of each age interval:

$$l_0 = L_0, l_{x+1} = l_x \cdot (1 - q_x)$$

The counterfactual survival with $PM_{2.5}$ removed is:

$$l_x' = l_x \cdot (1 - q_x')$$

The person-years lived in the interval:

$$L_x = \frac{l_x + l_{x+1}}{2} \cdot n_i, L_x' = \frac{l_x' + l_{x+1}'}{2} \cdot n_i$$

Cumulative person-years above age x (total years remaining):

$$T_x = \sum_{j=x}^{\text{max age}} L_j\,, T_x' = \sum_{j=x}^{\text{max age}} L_j'$$

Life expectancy at age x:

$$e_x = \frac{T_x}{l_x}, e_x' = \frac{T_x'}{l_x'}$$

The impact of $PM_{2.5}$ removal on life expectancy:

$$\Delta e_x = e_x' - e_x$$

The research team constructed life tables using both the observed and the counterfactual death probabilities. For each race-ethnicity and age group, the research team calculated the number of survivors entering the interval (l_x), the number surviving with $PM_{2.5}$ removed (l_x'), and the person-years lived within each age interval (L_x and L_x'). The cumulative total person-years lived above each age (T_x and T_x') was then computed, and life expectancy at each age (e_x and e_x') was obtained by dividing T_x or T_x' by the number of survivors at the beginning of the interval. The difference between the observed and counterfactual life expectancies yielded the life years lost

due to $PM_{2.5}$ exposure. To ground the estimates in reality, the research team anchored the life tables to known baseline life expectancy at birth for each race-ethnicity group, based on period-specific state and national statistics. For Period 1, these values were 83.0 years for Asians, 80.5 years for Hispanics, 77.8 years for non-Hispanic Whites, 72.1 years for non-Hispanic Blacks, and 78.2 years for Other groups (https://www.ppic.org/wp-content/uploads/content/pubs/cacounts/CC_504HJCC.pdf). For Period 2, these values were 86.3 years for Asians, 83.2 years for Hispanics, 79.8 years for non-Hispanic Whites, 75.1 years for non-Hispanic Blacks, and 80.2 years for Other groups (https://www.chcf.org/wp-content/uploads/2019/10/DisparitiesAlmanacRaceEthnicity2019.pdf). By aligning the modeled estimates with these known values, the research team corrected for discrepancies introduced by limited sample sizes or interpolation errors.

An additional hazard-scale adjustment method was used to ensure that the attributable fractions and counterfactual probabilities of death were consistent with the underlying log-linear structure of the models. This method prevented the emergence of biologically implausible results, such as negative life years gained, and scaled impacts appropriately when mismatches occurred between expected and observed directions of effect. In cases where the raw calculations produced impacts exceeding plausible bounds, the results were scaled relative to the maximum observed within-group effect, ensuring comparability across race and age groups.

Aggregate life-expectancy impact

The research team estimated the overall impact of $PM_{2.5}$ on life expectancy across all age groups using aggregate weighting approaches using population-weighted and death-weighted estimates. Population-weighted estimates reflect the age distribution of the population, while death-weighted estimates reflect the distribution of deaths across age groups.

The population-weighted $PM_{2.5}$ impact on life expectancy was estimated by combining the modeled age-specific life-expectancy impacts with the corresponding age-specific population distribution. For each age group, the research team multiplied the estimated $PM_{2.5}$-attributable life-expectancy impact by the number of individuals in that age group. These weighted contributions were then summed across all age groups and divided by the total population represented in the dataset. This approach ensures that age groups comprising larger portions of the population have greater influence on the overall estimate and produces a single population-weighted metric that reflects the demographic structure of the population.

The death-weighted $PM_{2.5}$ impact on life expectancy was estimated by weighting the modeled age-specific life-expectancy impacts using the observed number of deaths in each age group. For each age group, the research team multiplied the estimated $PM_{2.5}$-attributable life-expectancy impact by the number of deaths occurring in that group. These values were summed across all age groups and divided by the total number of deaths in the dataset to obtain a single death-weighted estimate. This method places greater emphasis on age groups with higher mortality, recognizing that life-expectancy impacts are driven primarily by the groups in which

deaths occur. As a result, the death-weighted metric reflects the distribution of actual mortality burden across age groups.

In this project, the research team focus on the death-weighted $PM_{2.5}$ impact because our modeling is designed to capture the effects of $PM_{2.5}$ on mortality. The research team also use the population-weighted $PM_{2.5}$ impact, alongside the death-weighted $PM_{2.5}$ impact, to determine whether $PM_{2.5}$ life-expectancy impact fall disproportionately on younger populations or on older age groups.

When the population-weighted impact is relatively high but the death-weighted impact is relatively low, it indicates that younger or middle-aged groups, who make up most of the population, experience moderate-to-high per-person $PM_{2.5}$ impacts, while the elderly experience smaller per-person impacts and therefore contribute less to the death-weighted average. When the population-weighted impact is relatively low but the death-weighted impact is relatively high, the pattern is reversed: the numerically dominant younger or middle-aged groups have relatively low per-person $PM_{2.5}$ impacts, while elderly groups have moderate-to-high per-person impacts, causing deaths to carry most of the overall burden.

When both population-weighted and death-weighted impacts are high, all major age groups, including young, middle-aged, and elderly, exhibit moderate-to-high per-person $PM_{2.5}$ impacts, producing large totals regardless of whether the impacts are weighted by population or by deaths. When both population-weighted and death-weighted impacts are low, per-person $PM_{2.5}$ impacts are uniformly small across all age groups, resulting in low overall values under either weighting approach.

Study Results

Integrated analysis for 20 age groups across the entire study period (2000-2021)

To improve model stability and interpretability for younger populations, the research team aggregated the four youngest age groups (0-5, 6-11, 12-17, and 18-25) into a single 0-25 age category. This decision was motivated by the relatively low mortality counts and similar exposure-response patterns across these age bands, which could otherwise lead to unstable estimates and wide confidence intervals when modeled separately. The resulting 0-25 group thus provides a more reliable and interpretable estimate of $PM_{2.5}$-related mortality effects in early life stages, while maintaining consistency across the full age spectrum.

The conditional logistic regression results (Figure 2) revealed a clear age-dependent pattern in the association between $PM_{2.5}$ exposure and mortality risk. The estimated $PM_{2.5}$ coefficients were positive and statistically significant across all age groups for the integrated study period, indicating that higher $PM_{2.5}$ exposure consistently increased mortality risk throughout the lifespan. The effect generally increased from younger to middle-aged groups, peaking between ages 66-70 and again around 84-86, followed by a gradual decline among the oldest populations (90 years and above). This pattern suggests that sensitivity to $PM_{2.5}$ rises steadily through

adulthood and early older age, possibly reflecting the accumulation of chronic disease burden and diminished physiological resilience, before declining at extreme ages where competing mortality risks or survivor bias may attenuate the observed associations.

The subsequent analysis of $PM_{2.5}$ impacts on life expectancy across age groups revealed a similar and distinct age-dependent pattern, with the magnitude of life expectancy gains from $PM_{2.5}$ removal varying systematically by age (Figure 3). The positive life expectancy impact of $PM_{2.5}$ removal was observed across all age groups, indicating that reducing air pollution consistently extended expected lifespan throughout the population. Among younger age groups (0-25 years), the effect was modest, with an estimated improvement of about 0.16 years, reflecting the lower baseline mortality risk and shorter immediate exposure windows typical of early life. The impact then increased gradually through adulthood, peaking during the mid- to late-older age groups (ages 84-86) with an average improvement of 0.95 years, suggesting that chronic exposure over the lifespan culminates in greater health benefits from pollution reduction later in life. Beyond this peak, the effect began to decline slightly in the very oldest age groups (90 years and above), where the estimated gains ranged between 0.46 and 0.68 years.

For the aggregated impact, when using a population-weighted approach, long-term $PM_{2.5}$ exposure was associated with an average loss of 0.35 years. Using a death-weighted approach, the estimated loss increased to 0.61 years. The 0.61 years indicate a moderate life expectancy loss from air pollution $PM_{2.5}$. The population-weighted impact is relatively low but the death-weighted impact is relatively high, and this clearly indicates that the numerically dominant younger or middle-aged groups have relatively low $PM_{2.5}$ impacts, while elderly groups have moderate-to-high per-person impacts, causing deaths to carry most of the overall burden.

In summary, this pattern underscores how cumulative exposure and age-related vulnerability shape the life expectancy benefits of cleaner air. The steady increase in $PM_{2.5}$-related life expectancy gains from young adulthood to late old age likely reflects both longer exposure histories and the compounding effects of pollution on chronic diseases such as cardiovascular and respiratory conditions. The slight decline at the extreme ages may be due to survivor bias, where only the healthiest individuals reach those ages and the limited room for further life expectancy extension in very old populations. Further, increased exposure misclassification might occur among older adults who spend more time indoors. Despite this, exposure studies[79] have documented strong positive correlations between ambient and indoor $PM_{2.5}$ concentrations, indicating that outdoor $PM_{2.5}$ is a dominant contributor to personal exposure (e.g., Pearson $r \geq 0.90$ in community residential settings and correlation approaches 1.0 in urban areas under typical conditions) and supports the use of ambient concentrations as a surrogate in long-term mortality modeling.

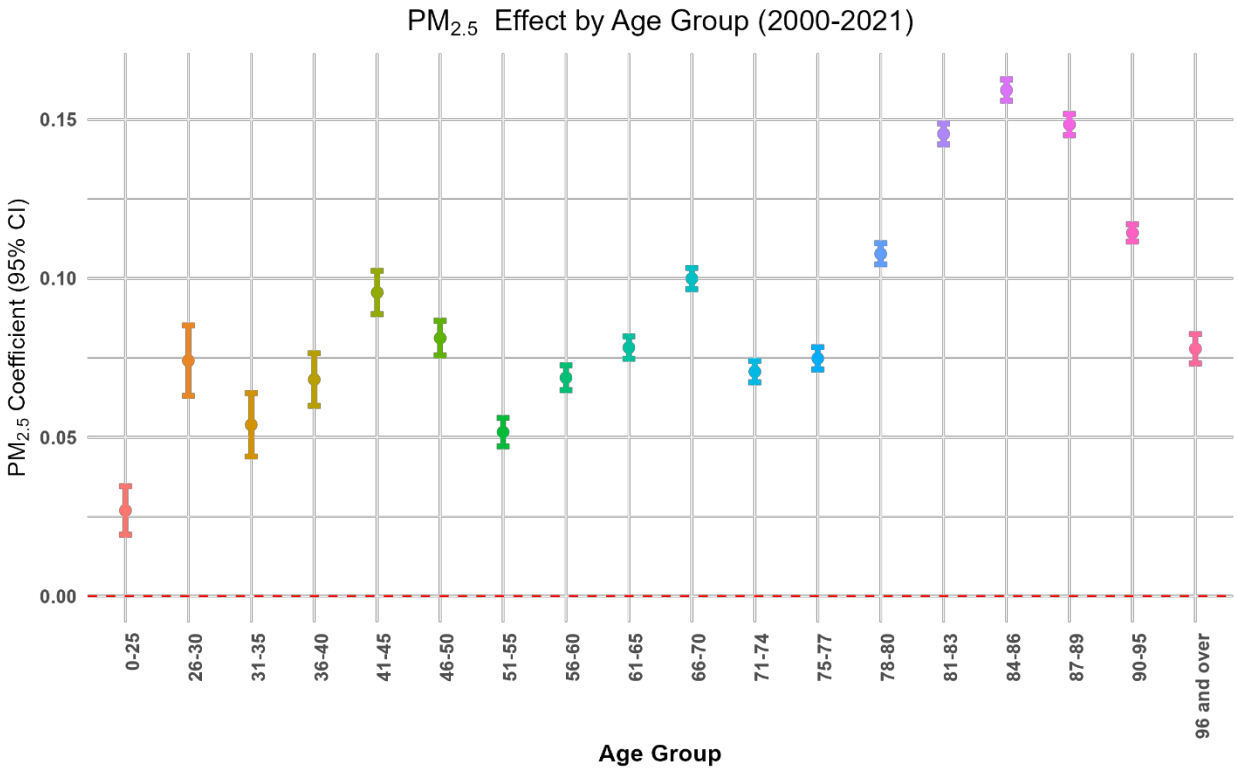


Figure 2. The impact of $PM_{2.5}$ on mortality across the major age groups for the entire study period (2000-2021).

Notes: The y-axis displays regression coefficients (β_1) from age-specific mortality models, expressed per interquartile range (IQR) increase in $PM_{2.5}$. These coefficients quantify log-scale mortality risk effects and may be exponentiated to obtain odds ratios. Coefficients are shown to maintain consistency with subsequent life-table and population impact calculations.

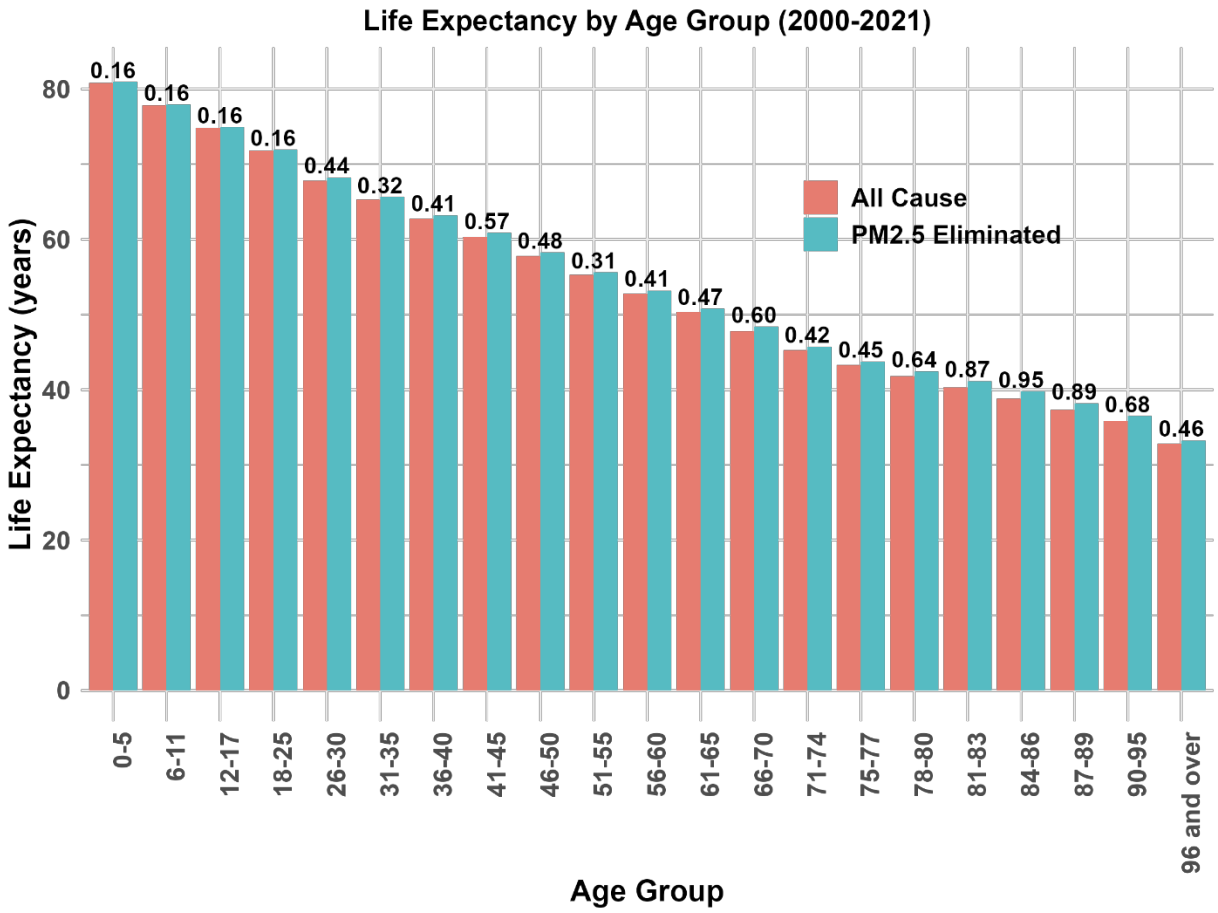


Figure 3. The impact of $PM_{2.5}$ on life expectancy across the major age groups for the entire study period (2000-2021).

Notes: The y-axis shows cumulative period life expectancy from birth up to each age-group entry, as derived from life-table calculations. It is not the expected remaining years of life conditional on survival to that age. For example, the 90–95 age group has a y-axis value of ~40 because this represents the cumulative life expectancy measure at the entry to that age interval, not the remaining years a 90-year-old individual is expected to live. Bars labeled "$PM_{2.5}$ Eliminated" represent counterfactual life expectancy under a scenario in which $PM_{2.5}$-attributable mortality risk is removed. The difference between observed and $PM_{2.5}$-eliminated bars indicates the estimated life expectancy loss attributable to $PM_{2.5}$.

Period-specific impact for twenty age groups without race-ethnicity stratification

The logistic regression modeling results for Period 1 (2000-2010) (Figure 4, top) demonstrate that $PM_{2.5}$ exposure is consistently and significantly associated with increased mortality risk across age groups, with effects strengthening as age advances. Among younger adults (18-35), coefficients ranged from 0.056 to 0.064, corresponding to odds ratios of about 1.06-1.07 per inter-quartile range (IQR) increase in $PM_{2.5}$ exposure, while middle-aged adults (36-55) showed stronger effects, particularly at ages 41-45 (OR ≈ 1.11) and 46-50 (OR ≈ 1.09). Early seniors (61-74) exhibited even higher risks, with odds ratios between 1.10 and 1.12, and the effect peaked in the mid-80s, where coefficients reached 0.182 (OR ≈ 1.20), indicating nearly 20 percent higher mortality odds per unit increase in $PM_{2.5}$. At the very oldest ages (96 and over), the effect

declined slightly to an OR of about 1.12, likely reflecting survivor bias or smaller sample sizes. All confidence intervals were narrow and excluded zero, and p-values were < 0.001~~-effectively~~, providing strong evidence that $PM_{2.5}$ exposure is a robust predictor of mortality. Overall, the results reveal a clear age-related gradient of vulnerability, with the elderly facing the greatest risks, underscoring the critical public health importance of mitigating air pollution exposure in California.

The logistic regression results for Period 2 (2011-2021) (Figure 4, bottom) show that $PM_{2.5}$ exposure remained a significant predictor of mortality across most age groups, though the magnitude of effects was generally lower and more variable compared to the earlier decade. In younger adults, associations were modest but significant, with coefficients ranging from 0.035 to 0.082 (OR ≈ 1.04-1.09), peaking in the 26-30 age group. Among middle-aged adults, effects weakened considerably, with coefficients around 0.012-0.030 (OR ≈ 1.01-1.03), indicating much smaller impacts than observed in Period 1. For seniors, the associations reemerged, with ages 66-70 showing one of the strongest effects (coef. ≈ 0.067, OR ≈ 1.07), while later elderly groups exhibited modest but statistically significant risks, such as 81-86 with coefficients of 0.025-0.027 (OR ≈ 1.03) and 87-89 at 0.043 (OR ≈ 1.04). At the oldest ages (96+), the coefficient remained significant at 0.039 (OR ≈ 1.04). All estimates had narrow confidence intervals and very small p-values, confirming robust significance despite attenuated magnitudes compared to 2000-2010. Taken together, the results suggest that while $PM_{2.5}$ exposure continued to adversely impact mortality in California, the effect sizes in the more recent decade were smaller and less steeply age-graded than in Period 1, possibly reflecting improved air quality, stronger health protections, or shifting population dynamics. Notably, the age distribution of deaths shifted in Period 2 toward relatively younger adults compared with Period 1, increasing the relative contribution of younger mortality to the overall $PM_{2.5}$-mortality relationship.

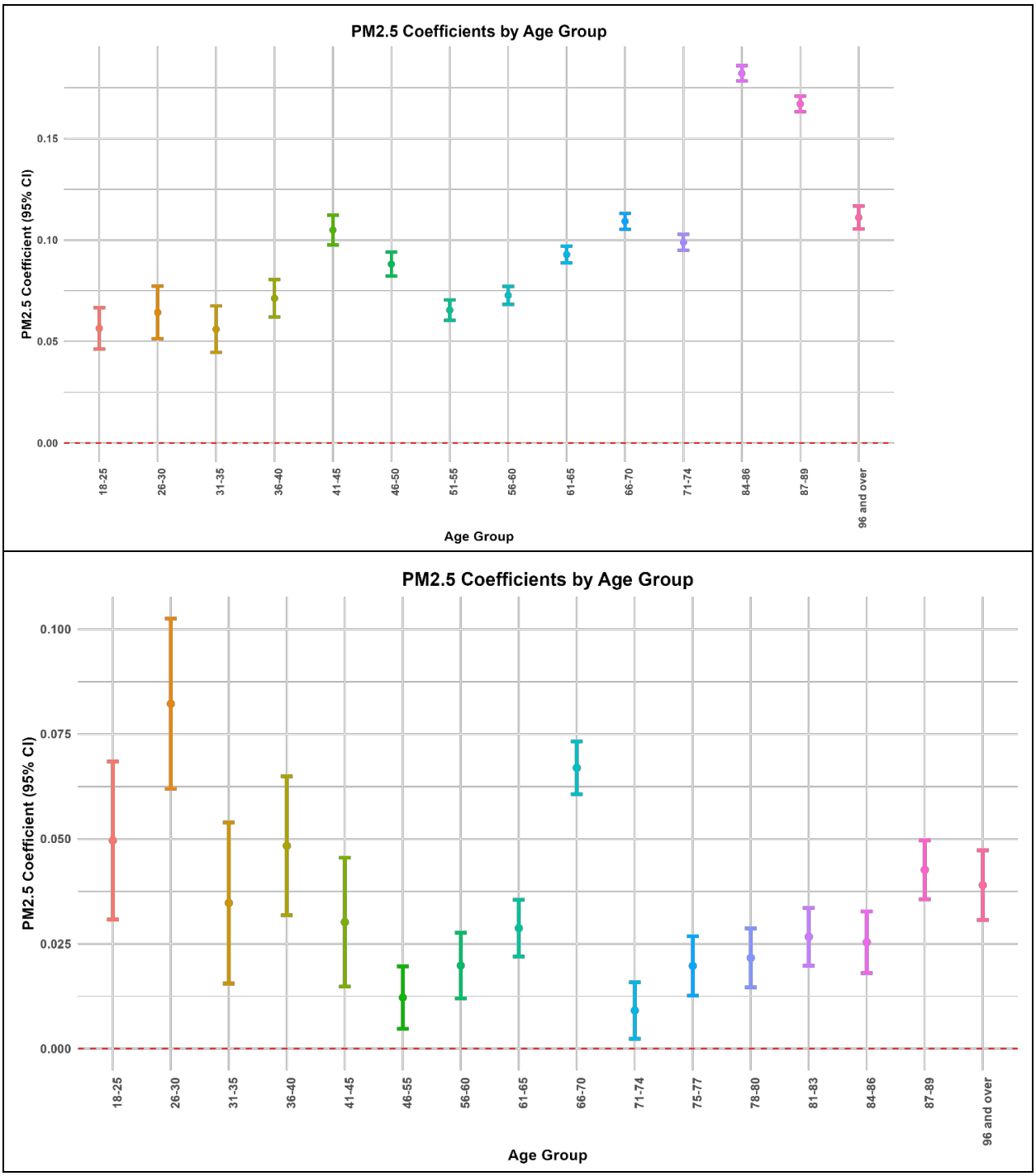


Figure 4. The age group stratified logistic regression modeling results on the overall impact of $PM_{2.5}$ on mortality for Period 1 (top) and Period 2 (bottom).

Notes: Separate y-axis scales are used to enhance visual resolution; Period 2 coefficients are substantially smaller than Period 1. Confidence intervals (95% CI) are proportional to the coefficient range provided; although Period 2 CIs appear wider relative to the coefficient, the absolute uncertainty is comparable to Period 1.

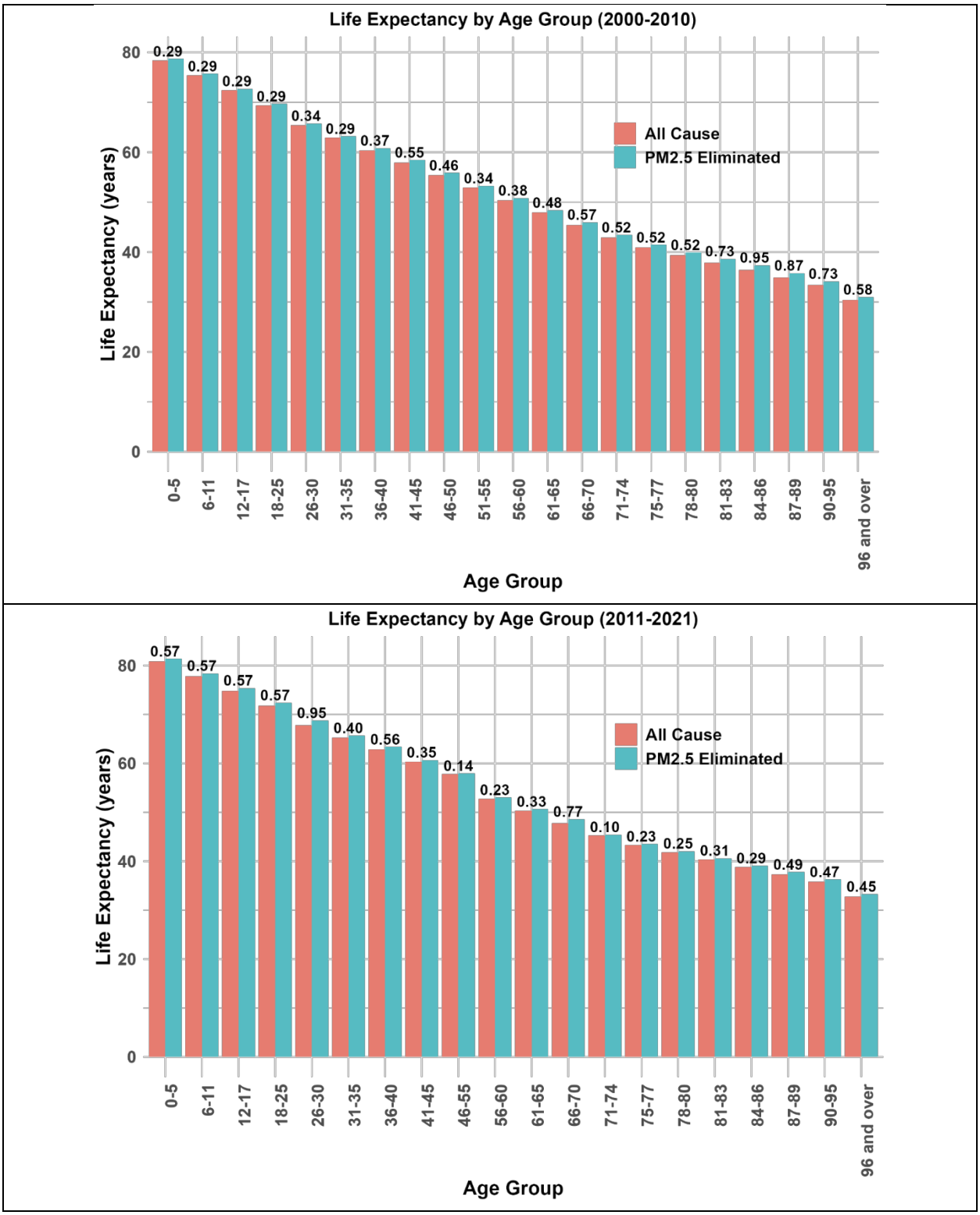


Figure 5. All cause and $PM_{2.5}$-eliminated life expectancy by age groups for Period 1 (top) and Period 2 (bottom).

Notes: The y-axis shows cumulative period life expectancy from birth up to each age-group entry, as derived from life-table calculations. Bars labeled "$PM_{2.5}$ Eliminated" represent counterfactual life expectancy under a scenario in

which $PM_{2.5}$-attributable mortality risk is removed. The difference between observed and PM2.5-eliminated bars indicates the estimated life expectancy loss attributable to $PM_{2.5}$.

The life expectancy estimates for Period 1 (2000-2010) (Figure 5 top) show that eliminating $PM_{2.5}$ exposure would have produced consistent gains across all age groups, with the most pronounced benefits at older ages. At birth, life expectancy was 78.40 years, which increased to 78.78 years once $PM_{2.5}$ was removed, reflecting an overall gain of 0.38 years based on population-weighted impact. The corresponding death-weighted impact was 0.61 years, indicating a moderate effect for Period 1. The population-weighted impact is relatively low but the death-weighted impact is relatively high, indicating that the numerically dominant younger or middle-aged groups have relatively low per-person $PM_{2.5}$ impacts, while elderly groups have moderate-to-high per-person impacts, causing deaths to carry most of the overall burden. Across childhood and adolescence, the benefit remained modest at about 0.29 years, but by adulthood the impacts became more visible. For example, individuals aged 26-30 would have gained 0.34 years, while those aged 41-45 gained 0.55 years. The largest effects appeared among older adults, with the 66-70 group experiencing an improvement of 0.57 years, and the 84-86 group gaining nearly one full year of life expectancy (0.95 years). Even at ages 96 and over, $PM_{2.5}$ removal was associated with an additional 0.58 years of life. These results highlight how air pollution shortened life expectancy at every age, with its heaviest toll among seniors.

In Period 2 (2011-2021) (Figure 5 bottom), life expectancy rose overall, and the gains from removing $PM_{2.5}$ exposure also remained significant. At birth, life expectancy was 80.80 years, increasing to 81.26 years without $PM_{2.5}$, an overall gain of 0.46 years based on population-weighted impact. The corresponding death-weighted impact was 0.37 years, a significant reduction in effect compared to that in Period 1. Here the overall population-weighted impact is slightly higher than the death-weighted impact. It indicates that younger or middle-aged groups, who make up most of the population, experience slightly higher per-person $PM_{2.5}$ impacts, while the elderly experience slightly smaller per-person impacts and therefore contribute less to the death-weighted average. Children and young adults showed consistent improvements after removing $PM_{2.5}$ impact, with the 26-30 age group experiencing the largest single benefit of 0.95 years. Middle-aged adults showed smaller impacts than in the previous decade, with increases of only 0.14 to 0.35 years, suggesting possible attenuation of pollution effects or stronger resilience. Among older adults, however, the benefits reappeared, with the 66-70 group gaining 0.77 years and those aged 87-89 and 90-95 gaining about 0.49 and 0.47 years, respectively. The moderate values of both death-weighted and population-weighted impact indicate the $PM_{2.5}$ impact became moderate.

From Period 1 to Period 2, the death-weighted $PM_{2.5}$ impact decreased substantially, from 0.61 years to 0.37 years, indicating that improvements in emissions control, cleaner vehicle fleets, and strengthened regulatory actions collectively reduced the mortality consequences of $PM_{2.5}$ exposure. This shift, coupled with the observation that population-weighted impacts became slightly higher than death-weighted impacts in Period 2, reflects a redistribution of

$PM_{2.5}$-related burden from primarily older adults in the early decade toward somewhat younger age groups in the later decade. The pattern suggests that although seniors still experienced meaningful life-expectancy improvements when $PM_{2.5}$ was removed, the relative contribution of younger and middle-aged populations to the overall impact became more pronounced in Period 2. Taking together, these results show that while $PM_{2.5}$ continued to reduce life expectancy in both decades, its magnitude diminished and its age distribution shifted in ways consistent with cleaner air and evolving population structures. The increasing proportional impact among younger age groups in Period 2 highlights the importance for policymakers and public health agencies to pay closer attention to early-life and mid-life exposures, including opportunities for prevention and continued air-quality improvements that protect future generations.

Period-specific impact for five age groups with race-ethnicity stratification

Mortality risks

The five-age group stratification for Period 1 (Figure 6, top) provides a summary of how the effects of $PM_{2.5}$ on mortality vary across race and ethnicity. For Non-Hispanic Whites, the associations were positive and highly significant at every stage of life, beginning with a modest coefficient of 0.03 for ages 0-25 and rising sharply to 0.12 in both the 26-45 and 66-80 ranges. The effect was strongest for the oldest adults, with a coefficient of 0.19 for ages 81 and over, reflecting nearly a 21 percent increase in the odds of mortality per unit increase in $PM_{2.5}$. This pattern suggests both early susceptibility and steadily mounting vulnerability with age, culminating in particularly elevated risks for the elderly.

Non-Hispanic Blacks showed a very different profile. In younger and midlife groups, the associations were negative, with coefficients of -0.078 for ages 0-25 and -0.068 for ages 26-45, both highly significant. These counterintuitive findings may reflect unmeasured confounding, data limitations, or differences in exposure patterns. Beginning at midlife, however, the direction shifted, with coefficients becoming positive and significant at 0.031 for ages 46-65 and strengthening further to 0.070 and 0.075 for the 66-80 and 81+ groups, respectively. Thus, while early life associations diverged from expectations, later life showed the more familiar pattern of higher mortality risk with $PM_{2.5}$ exposure.

Among Non-Hispanic Asians, associations were positive across all age groups, with the strongest effect observed in the 66-80 cohort, where the coefficient was 0.145, one of the largest across any group. While early life coefficients were smaller and sometimes less precise, such as 0.053 for ages 0-25, they nonetheless suggested heightened susceptibility. For those aged 81 and over, the coefficient of 0.059 confirmed a persistent, though smaller, association in the very old. The overall picture for Asians is one of consistent sensitivity to $PM_{2.5}$, with the clearest elevation in later adulthood.

Hispanic populations demonstrated a subtler but still significant set of associations. Early life effects were small and nonsignificant, with a coefficient of 0.008 for ages 0-25. Beginning in midlife, however, associations strengthened, rising to 0.028 for ages 26-45, 0.044 for ages 46-65,

and 0.071 for ages 66-80. The strongest effect appeared in the 81+ category, where the coefficient was 0.117, indicating nearly a 12 percent increase in mortality odds with $PM_{2.5}$ exposure. These results suggest that while early life risks were muted or difficult to detect, older Hispanics experienced some of the steepest increases in risk of any group.

Finally, results for the "Other" category were inconsistent, with smaller sample sizes and heterogeneity likely contributing to variability. While younger groups such as 26-45 showed significant positive associations (0.041), the oldest cohorts exhibited negative associations, including a coefficient of -0.030 for those aged 81 and over. Those results are small in values and the research team suggest caution in generalizing patterns for this diverse category.

In sum, the five-age group results in Period 1 demonstrate that Non-Hispanic Whites, Asians, and Hispanics show clear positive associations between $PM_{2.5}$ and mortality that strengthen with age, peaking among the elderly. Non-Hispanic Blacks display a distinct age pattern, with negative associations in youth and early adulthood but convergence toward positive and significant effects in later life. The "Other" category presents the least consistent evidence, highlighting limitations in statistical power or subgroup heterogeneity. Together, these findings reinforce the conclusion that $PM_{2.5}$ exposure disproportionately affects older populations across nearly all race and ethnicity groups, though the trajectory of risk across the life course differs meaningfully between them.

The five-age group stratification for Period 2 (2011-2021) (Figure 6, bottom) highlights both continuity and important shifts in how $PM_{2.5}$ exposure related to mortality risk across race and ethnicity. Among Non-Hispanic Whites, the associations were weaker than in Period 1 but still largely positive and significant. The effect was strongest in the youngest group, ages 0-25, with a coefficient of 0.048, while middle-aged adults (26-45) showed no significant effect. For older adults, risks reemerged, with coefficients of 0.023 for ages 46-65 and 0.033 for ages 66-80, though the very old (81 and over) displayed a smaller effect at 0.015. Non-Hispanic Blacks exhibited a more complex pattern: effects were null in youth, strongly positive in early adulthood (0.107 for ages 26-45), and significantly negative in midlife (-0.084 for ages 46-65), before turning positive again in older age, with a very large effect of 0.162 among those 81 and over. This suggests shifting vulnerabilities by life stage, with late-life Black populations facing particularly elevated risks.

For Non-Hispanic Asians, associations were consistently positive and highly significant across all ages, with some of the largest coefficients observed in any group. Children and young adults had a coefficient of 0.109, rising slightly in early adulthood to 0.108, and continuing upward through midlife at 0.078. The strongest effects appeared among older adults, with coefficients of 0.163 for ages 66-80 and 0.126 for 81 and over, underscoring pronounced sensitivity among Asian populations at later ages. Hispanics displayed moderate to strong positive effects in younger and middle-aged groups, including coefficients of 0.078 for ages 0-25 and 0.071 for ages 26-45. However, the association diminished sharply in midlife, with a very

small positive coefficient of 0.015 at ages 46-65, and even reversed direction for those 66-80, where the effect was negative at -0.014. Among the oldest Hispanics, however, risks became strongly positive again, with a coefficient of 0.077. The "Other" category remained inconsistent, with positive effects in early adulthood (0.163 for ages 26-45) and late life (0.107 for 81+), but null or negative coefficients in other groups, including a significant -0.040 in the 66-80 range.

Overall, the Period 2 results reveal smaller and more variable effect sizes compared with Period 1, particularly among Whites and Hispanics in midlife, while late-life vulnerability remained a consistent theme across nearly all race-ethnicity groups. Non-Hispanic Asians showed the most stable and uniformly strong associations, while Non-Hispanic Blacks and Hispanics displayed more fluctuation across the life course, with both groups experiencing their highest risks in the oldest age category. These results suggest that although improvements in air quality or population health may have attenuated $PM_{2.5}$ impacts for some groups, the elderly across nearly all race-ethnicity backgrounds continue to face substantial mortality risks tied to pollution exposure.

Life expectancy – age specific impact

For life expectancy, the impact of $PM_{2.5}$ varies by race and age in Period 1 (Figure 7). Among Asians, the gains are very significant in younger and middle-aged adults, with life expectancy increasing by nearly 0.95 years for ages 0-65, indicating a strong benefit from cleaner air. Older Asians (66-80 and 81+) see slightly smaller but still meaningful improvements, with gains of 0.93 and 0.87 years, respectively. Black populations show a more mixed pattern: children and young adults (0-25) actually experience a slight reduction in life expectancy, but adults aged 26-65 gain substantially (0.83-0.95 years), and older adults continue to benefit moderately (0.84-0.93 years). Hispanics consistently achieve very significant life expectancy gains across all ages, from 0.92 to 0.95 years, reflecting a uniform benefit of $PM_{2.5}$ removal. Among Whites, the most substantial improvements occur in younger and middle-aged adults (0.74-0.95 years), while older adults experience only minor gains (0.29-0.30 years), indicating that $PM_{2.5}$ reduction has limited impact on life expectancy at advanced ages for this group. The Other race category benefits strongly in children and middle-aged adults (0.95 years), but older adults show minimal or even slightly negative changes, suggesting that the effect of $PM_{2.5}$ removal diminishes with age or may interact with other risk factors in these populations.

In Period 2, the life expectancy gains from $PM_{2.5}$ removal show a mixed pattern across race-ethnicity groups and age (Figure 8). Among Asians, the gains remain substantial but vary by age: younger and middle-aged adults (0-65) see moderate improvements ranging from 0.45 to 0.64 years, slightly lower than the very significant gains near 0.95 years observed in Period 1. However, older adults benefit more than before, with the 66-80 group reaching the maximum gain of 0.95 years and the 81+ group gaining 0.73 years, suggesting improved benefits in late life. Black populations experience smaller or even negative impacts in some age groups. Children (0-25) now see minimal improvement (0.047 years), while adults aged 26-45 gain 0.63 years, similar to Period 1. Notably, the 46-65 group shows a negative impact, a reversal from the

positive gains in Period 1, though the oldest adults (81+) again reach a maximum gain of 0.95 years, reflecting concentrated benefits in late life.

For Hispanics, Period 2 shows very significant gains in young adults (0-25: 0.95 years) and strong improvements in ages 26-65 (0.85-0.91 years), slightly smaller than Period 1 for middle-aged adults, while the 66-80 age group now experiences a negative impact, marking a notable reversal from prior gains. Among Whites, younger adults maintain high gains (0-25: 0.95 years), but adults 26-45 see minimal improvement (0.15 years), while middle-aged and older adults gain moderately (0.46-0.65 years), representing a small improvement over Period 1 in some ages but still far below the maximum gain. The Other race category shows very strong improvements in children and adults 26-65 (0.92-0.95 years).

Life expectancy – aggregate impact

Taken together in our aggregated analysis (bottom right of Figure 7 and Figure 8, and Table 5 for five age groups), Period 1 for Asians showed nearly identical population-weighted (0.94) and death-weighted (0.91) impacts, indicating that $PM_{2.5}$ effects were broadly distributed across all age groups rather than concentrated in either younger or older adults. In Period 2, both metrics decreased (0.62 population-weighted; 0.74 death-weighted), consistent with an overall reduction in $PM_{2.5}$ impact. The slight rise of the death-weighted value relative to the population-weighted value suggests a modest shift toward a greater proportional impact among older Asian adults in the later decade.

For Whites, Period 1 showed moderately high population-weighted impact (0.82) but a lower death-weighted value (0.40), indicating that $PM_{2.5}$ impacts were more pronounced among younger and middle-aged Whites than among older adults. In Period 2, both metrics declined (0.56 population-weighted; 0.43 death-weighted), and the gap between them narrowed. This convergence reflects a more even age distribution of $PM_{2.5}$ impact among Whites in the later decade, with neither young nor old groups disproportionately driving the overall effect.

For Hispanics, the population-weighted and death-weighted values were almost identical in Period 1 (0.95 vs. 0.94), indicating that the $PM_{2.5}$ burden was distributed across all ages. In Period 2, the population-weighted impact remained moderately high (0.75), but the death-weighted impact decreased substantially (0.36), showing a shift toward higher proportional impact among younger and middle-aged Hispanic populations and a reduced proportional contribution from older Hispanic adults. The overall policy effect of 0.57 years indicates Hispanics benefited significantly from regulatory actions from Period 1 to Period 2.

For Blacks, Period 1 showed an unusual pattern: a negative population-weighted value (-0.24) alongside a positive death-weighted value (0.68). This combination indicates that younger and middle-aged Black populations experienced very small or slightly inverse $PM_{2.5}$ associations, while older Black adults experienced substantial per-person impacts, indicating that older adults disproportionately drove the $PM_{2.5}$-related life-expectancy losses in this group. In Period 2, both metrics decreased sharply (0.00 population-weighted; 0.08 death-weighted),

reflecting an overall reduction in $PM_{2.5}$ impact across ages and a particularly large reduction in older-adult impact (policy effect = 0.59), consistent with improved conditions for older Black populations.

For the Other category, Period 1 showed moderately high population-weighted impact (0.80) but very low death-weighted impact (0.06), indicating disproportionately higher impacts among younger adults and almost negligible impacts among older adults. In Period 2, both metrics increased slightly (0.78 population-weighted; 0.38 death-weighted), with the death-weighted value rising more sharply. This shift indicates that while younger adults continued to show elevated impacts, older adults in this group experienced a greater increase in proportional share of $PM_{2.5}$ impact in the later period, though population fluctuations and heterogeneity within this broad category likely influence these patterns.

Overall, the combined population-weighted and death-weighted analysis reveals that Period 2 showed smaller overall $PM_{2.5}$ impacts across all racial-ethnicity groups, consistent with the effects of cleaner air. However, the age distribution shifted, with several groups (Whites, Hispanics, Others) showing a greater proportional impact among younger and middle-aged populations in Period 2 compared to Period 1, while others (Asians, Blacks) showed either balanced or declining older-adult impacts. These patterns underscore that improvements in air quality benefited all racial-ethnicity groups, but the demographic profile of risk, whether predominantly in younger or older age groups, differs across groups and changed over time.

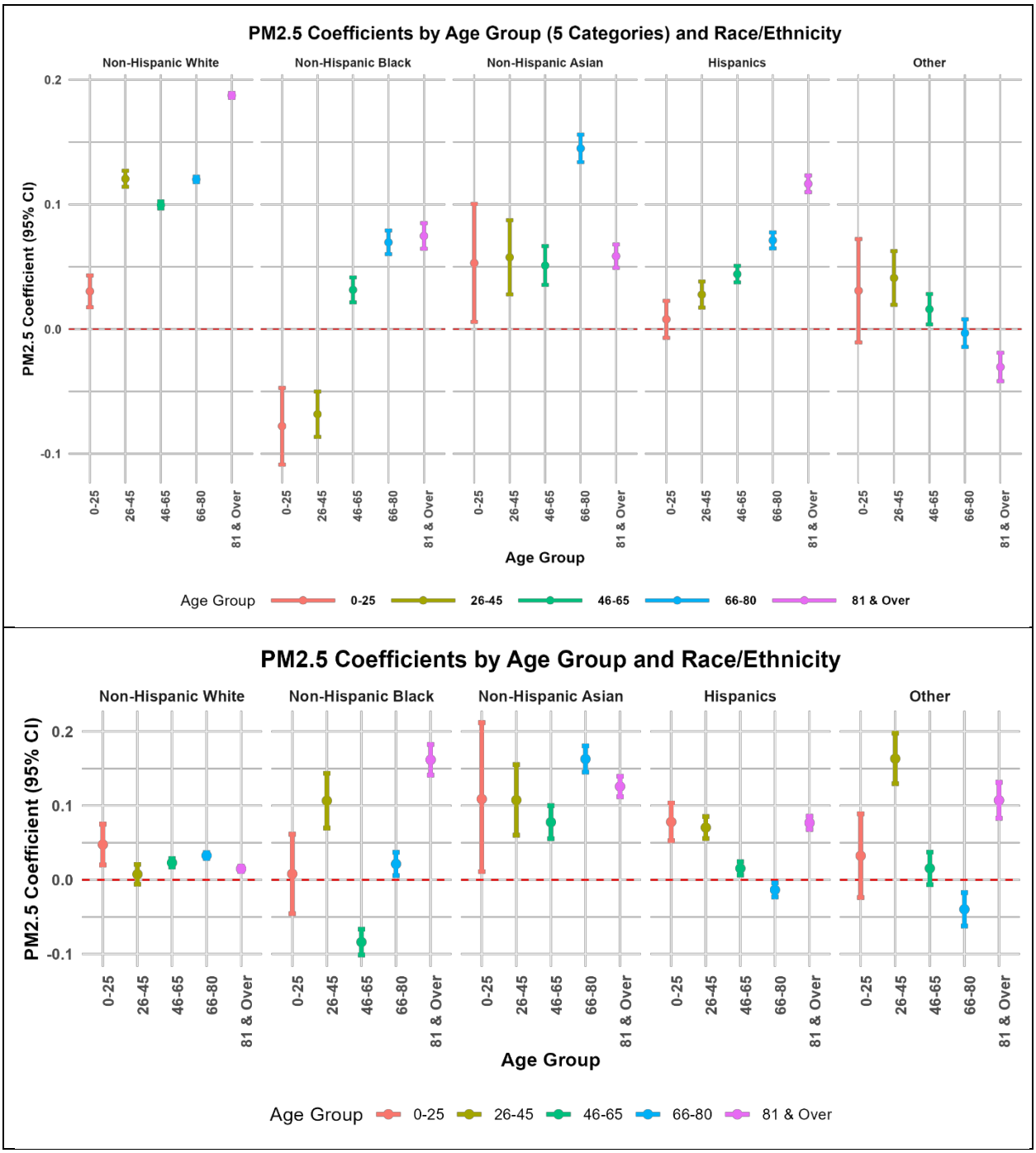


Figure 6. The race-ethnicity stratified logistic regression modeling results over five age-groups on the impact of $PM_{2.5}$ on mortality for Period 1 (top) and Period 2 (bottom).

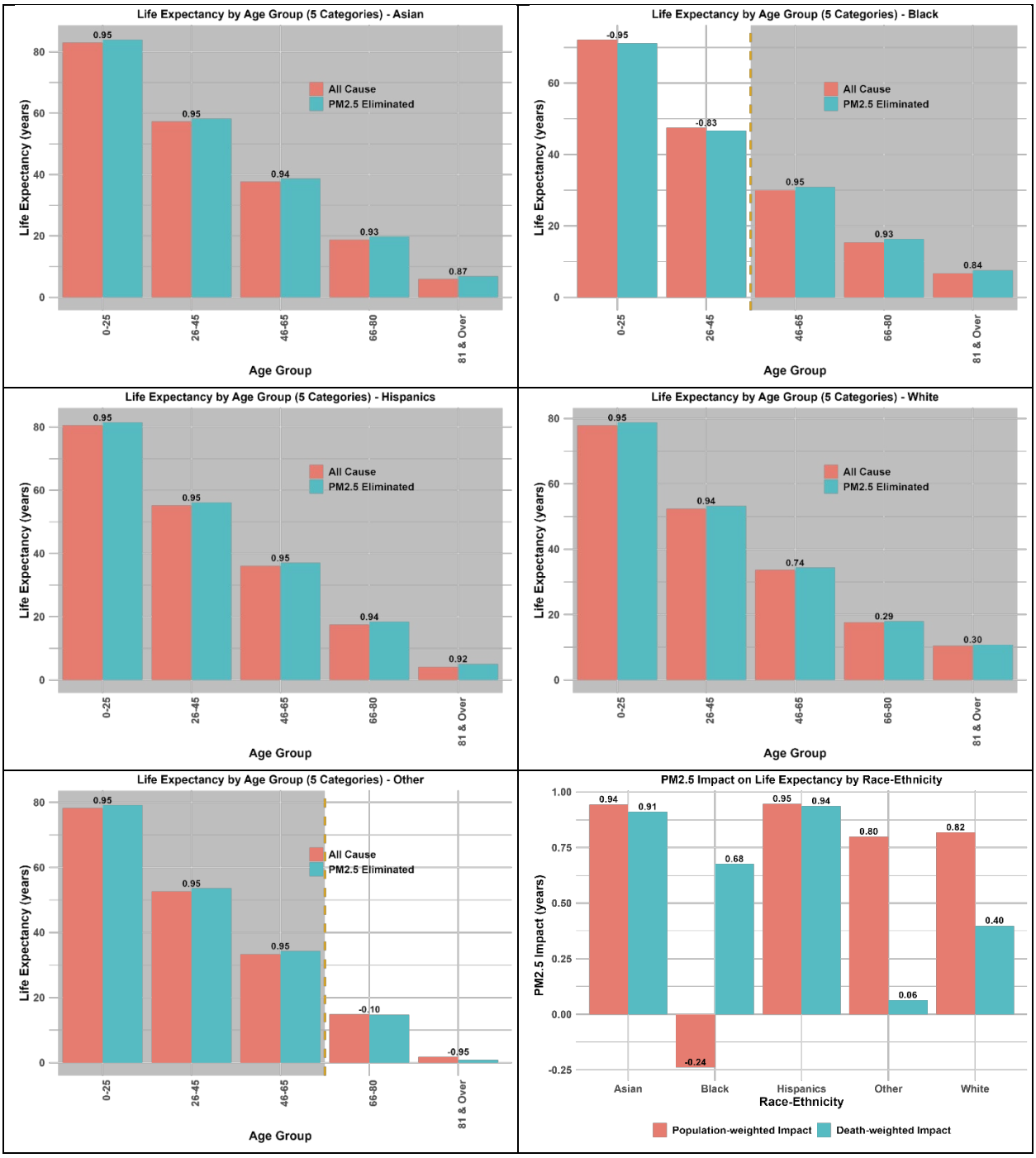


Figure 7. All cause and $PM_{2.5}$-eliminated life expectancy by race-ethnicity for Period 1 (2000-2010) across five age groups.

Note: The dashed vertical line indicates the point at which the relative magnitude of life expectancy shifts between the all-cause and $PM_{2.5}$-eliminated estimates (i.e., from all-cause > $PM_{2.5}$-eliminated to all-cause < $PM_{2.5}$-eliminated, or vice versa). The shaded grey area denotes age groups for which the estimated life expectancy under $PM_{2.5}$ removal exceeds the observed all-cause life expectancy (expected).

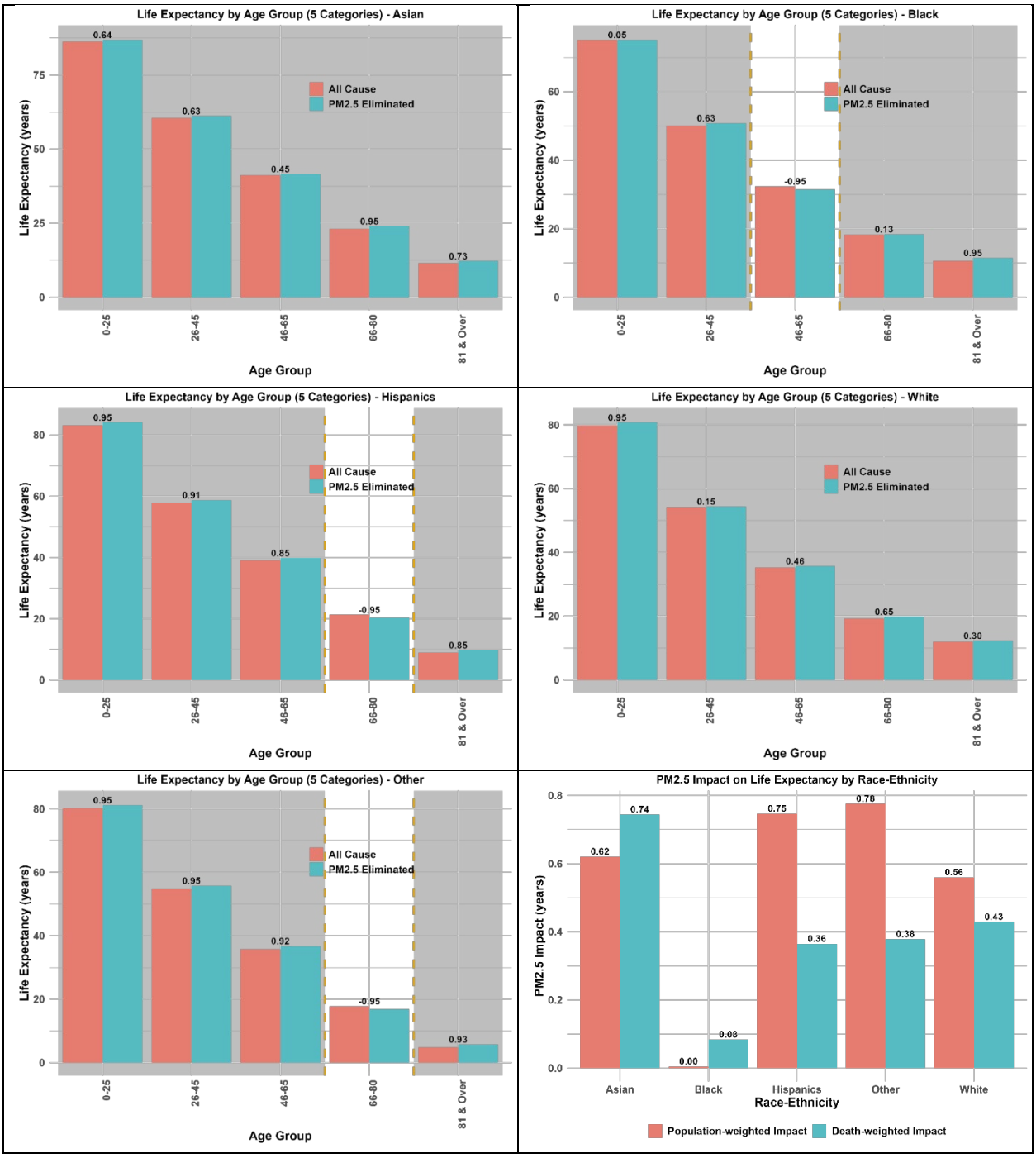


Figure 8. All cause and $PM_{2.5}$-eliminated life expectancy by race-ethnicity for Period 2 (2011-2021) across five age groups.

Note: The dashed vertical line indicates the point at which the relative magnitude of life expectancy shifts between the all-cause and $PM_{2.5}$-eliminated estimates (i.e., from all-cause > $PM_{2.5}$-eliminated to all-cause < $PM_{2.5}$-eliminated, or vice versa). The shaded grey area denotes age groups for which the estimated life expectancy under $PM_{2.5}$ removal exceeds the observed all-cause life expectancy (expected).

Period-specific impact for ten age groups with race-ethnicity stratification

Mortality risks

The 10 age groups provide much finer detail on how $PM_{2.5}$ exposure relates to health outcomes across lifespan (Figure 9). In Period 1 (Figure 9, top) for Non-Hispanic Whites, the association between $PM_{2.5}$ and adverse health outcomes strengthens with age until late adulthood. In early childhood (ages 0-11), the $PM_{2.5}$ coefficient is negative and significant, suggesting a small protective or unstable effect likely due to small sample size or competing risk factors. From adolescence onward, the coefficients turn positive and remain highly significant. The effect size grows steadily from 0.04 at ages 12-25 to 0.14 at 36-45 and remains elevated through midlife (0.09-0.10 for ages 46-65). The highest sensitivity appears among those aged 81-86, with a coefficient of 0.22, indicating a strong and consistent relationship between higher $SPM_{2.5}$ and adverse outcomes. This pattern reflects the cumulative damage hypothesis: long-term exposure over the life course compounds into larger late-life health effects, particularly for cardiorespiratory mortality among older Whites.

Non-Hispanic Blacks exhibit a distinct pattern characterized by negative associations in early and mid-adulthood and positive associations emerging only after middle age. For ages 0-45, most coefficients are negative and significant, implying that higher $PM_{2.5}$ levels are not strongly associated—or even inversely associated—with adverse outcomes in youth. However, starting at age 56-65, the relationship reverses, showing strong positive coefficients (0.09-0.12) with high statistical significance into the oldest groups. The largest effect (0.12) occurs for those aged 87 and over. This transition supports the interpretation that early-life mortality among Black populations is more strongly influenced by social and structural determinants (e.g., socioeconomic disadvantage, access to care, and neighborhood conditions), while air pollution effects accumulate and become more biologically evident in later life through the exacerbation of chronic diseases such as hypertension, diabetes, and cardiovascular disorders.

For Non-Hispanic Asians, the association between $PM_{2.5}$ and health outcomes is weak or inconsistent at younger ages but becomes substantial in older adulthood. The youngest age groups (0-35) show small, nonsignificant coefficients, but significant positive effects emerge from ages 36-65. The relationship becomes very strong among those aged 66-74, with a coefficient of 0.22—the largest among Asian age groups—followed by a moderate decline thereafter. This pattern suggests that early-life exposures may be less immediately impactful for this population, possibly due to protective social or health factors, while later-life sensitivity increases sharply as cumulative exposure interacts with aging-related vulnerabilities and cardiopulmonary risk. The results for Asians align with epidemiologic evidence showing greater late-life pollution sensitivity linked to urban living and longer lifespans that allow chronic exposure effects to manifest.

Hispanics display a steady and consistent positive association between $PM_{2.5}$ and adverse outcomes across nearly all ages, with increasing effect sizes in older adults. The coefficients

progress from modest positive values in childhood and adolescence (around 0.02-0.04) to stronger associations in adulthood (0.05-0.09), and finally peak in late life, with a large coefficient of 0.17 among those aged 87 and over. Every age group shows statistically significant effects. This stable, monotonic increase suggests that Hispanics are broadly sensitive to $PM_{2.5}$ across the life course, possibly reflecting higher baseline exposure levels in communities located near traffic corridors or industrial zones. The accumulation of exposure over decades likely compounds existing metabolic and cardiovascular risk factors, making pollution reduction especially beneficial for this group at all ages.

For the "Other" group, results are more variable and less consistent, likely due to smaller sample sizes and population heterogeneity. Younger and middle-aged adults generally show positive but modest associations, with significance emerging mainly between ages 26 and 55. However, from age 75 onward, the coefficients turn negative and statistically significant, suggesting lower or even inverse relationships between $PM_{2.5}$ and adverse outcomes in late life. These apparent reversals may arise from survivor bias (i.e., healthier individuals reaching older ages) or unstable estimates in small subpopulations. Nevertheless, the early- and mid-adulthood findings align with general expectations that $PM_{2.5}$ exposure contributes to elevated health risks before mortality selection effects dominate in advanced age.

Across all race-ethnicity groups, $PM_{2.5}$ effects strengthen with age, though the timing and intensity differ. Whites and Hispanics show steady positive associations throughout adulthood, while Blacks transition from negative to strongly positive coefficients later in life. Asians experience delayed but steep increases in sensitivity at older ages, and the "Other" group exhibits mixed effects due to smaller representation. Collectively, these findings reinforce that $PM_{2.5}$ exposure contributes most strongly to adverse health outcomes in middle and late adulthood, consistent with cumulative biological damage and long-term cardiopulmonary stress from chronic exposure.

From Period 1 to Period 2 (Figure 9, bottom), $PM_{2.5}$-related health effects declined across most race-ethnicity groups, reflecting the success of California's air quality regulations in reducing exposure. Despite this overall improvement, distinct age- and race-specific sensitivity patterns persisted, highlighting differences in biological vulnerability, exposure histories, and underlying health conditions. For Non-Hispanic Whites, $PM_{2.5}$ impacts were strongest in midlife (ages 36-65) during Period 1, with positive and highly significant coefficients, suggesting vulnerability linked to occupational exposure and chronic disease burden. In Period 2, these effects weakened and shifted toward older ages (56-74), consistent with reduced exposure levels and improved public health and healthcare access that delayed pollution-related health risks.

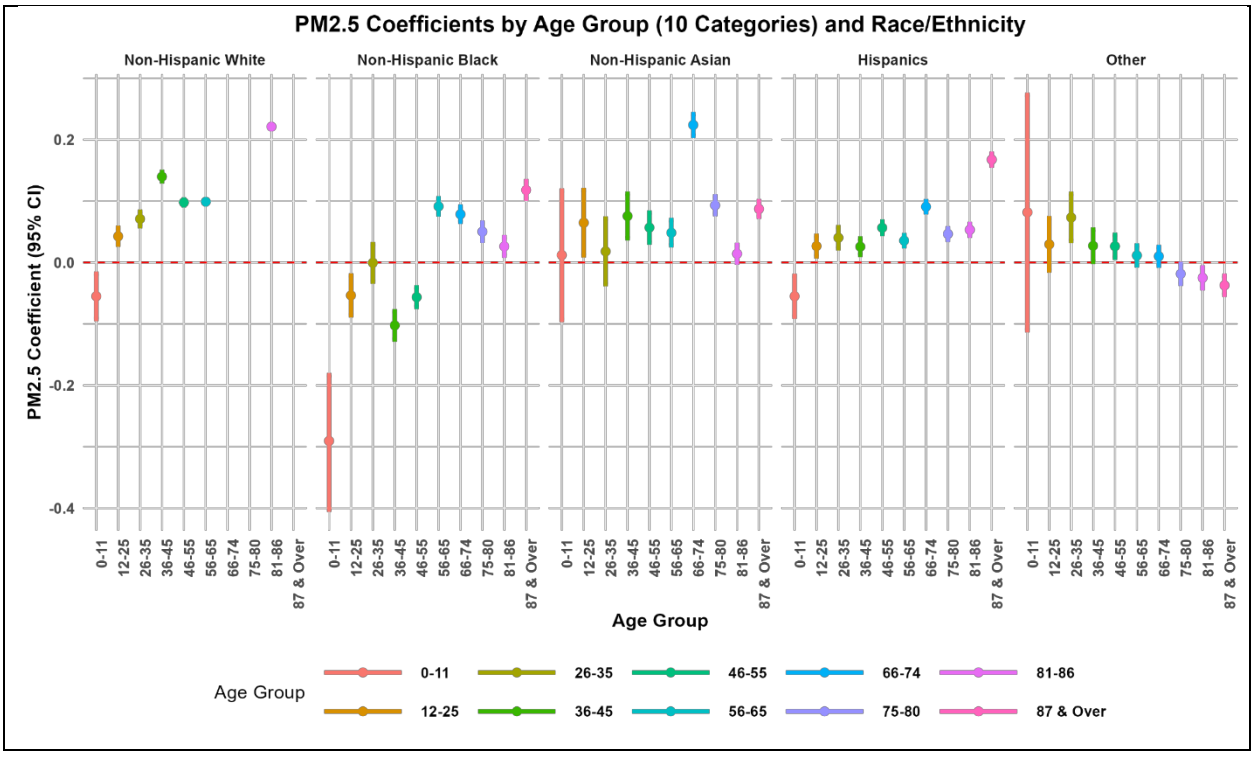
Among Non-Hispanic Blacks, Period 1 showed minimal or negative associations at younger ages and strong positive effects in older adulthood, indicating that cumulative exposure and chronic stress factors may manifest later in life. In Period 2, results became more variable, with some midlife sensitivity emerging but generally weaker effects. The persistence of later-life

vulnerability likely reflects the combined impacts of chronic conditions and environmental disadvantage accumulated over time.

For Non-Hispanic Asians, $PM_{2.5}$ effects were modest but consistent in both periods. In Period 1, sensitivity appeared mainly after age 45, while in Period 2, significant impacts concentrated in older ages (46-86). This pattern suggests cumulative exposure and age-related cardiopulmonary vulnerability, potentially intensified by indoor pollution or differences in preventive healthcare access.

Among Hispanics, $PM_{2.5}$ consistently had positive and significant effects across nearly all ages in both periods. In Period 1, associations strengthened with age, while in Period 2, effects slightly decreased in midlife but remained high among older adults. The persistent sensitivity across the lifespan likely reflects higher community-level exposure and occupational risks, despite generally favorable baseline health.

For Other race-ethnicity groups, results were heterogeneous due to smaller sample sizes. Period 1 showed mixed directions of effect, while Period 2 revealed stronger positive associations in select age bands (especially 26-45 and 81-86), likely reflecting demographic diversity and regional exposure differences within this category.



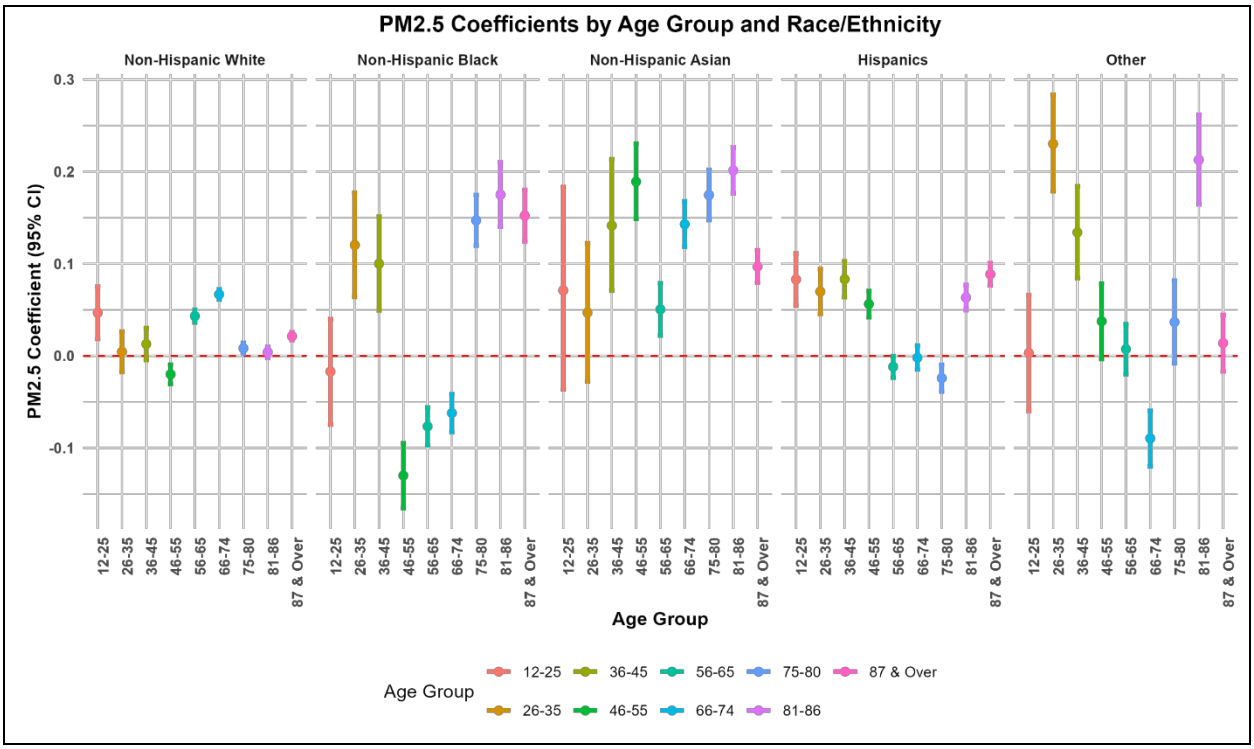


Figure 9. The race-ethnicity stratified logistic regression modeling results over ten age-groups on the impact of $PM_{2.5}$ on mortality for Period 1 (top) and Period 2 (bottom).

Life expectancy – age specific impact

Life expectancy in Period 1 improved across all race and ethnicity groups after the removal of $PM_{2.5}$, though the degree of benefit varied by age and group (Figure 10). Overall, most groups showed gains approaching the upper limit of 0.95 years, particularly during mid- and older adulthood when cumulative exposure to $PM_{2.5}$ becomes more consequential for mortality risk. This pattern is consistent with the underlying age- and race-ethnicity stratified conditional logistic regression models used to estimate $PM_{2.5}$-related mortality risk.

Among Asian populations, $PM_{2.5}$ removal consistently extended life expectancy across nearly all ages. Gains were strongest from early childhood through midlife, remaining close to 0.95 years through about age 55 before gradually declining at older ages. This pattern suggests that cleaner air particularly benefits younger and middle-aged Asians, likely reflecting high sensitivity of cardiometabolic and respiratory development to pollution during earlier life stages and relatively lower susceptibility once baseline health declines later in life.

For Black populations, the pattern differed markedly. Life expectancy gains from $PM_{2.5}$ removal were minimal or slightly negative during childhood through middle age, but benefits grew sharply from the late 50s onward, reaching near the 0.9 level by older adulthood. This delayed improvement likely reflects the accumulation of lifetime exposure burdens and chronic disease conditions that heighten pollution vulnerability later in life. It also indicates that earlier

exposures may already have produced lasting physiological effects, reducing reversible benefit in youth.

Hispanic populations demonstrated stable, positive gains across almost all ages, with $PM_{2.5}$ removal improving life expectancy consistently and near the upper range of 0.94-0.95 years. This uniform response suggests that Hispanics experience sensitivity to pollution throughout the life course, possibly due to a combination of occupational exposure, high-density living conditions, and intergenerational environmental influences. The sustained benefit at both younger and older ages indicates broad responsiveness to air quality improvements.

Among White populations, life expectancy gains were moderate but widespread. $PM_{2.5}$ removal improved life expectancy across most ages, with small increases in younger and middle ages and larger benefits, up to 0.95 years, among the oldest age groups. These patterns suggest a generally uniform exposure reduction, with stronger benefits manifesting later in life as aging amplifies pollution-related health risks.

For Other race-ethnicity groups, improvements followed a similar trend through middle adulthood, with gains near 0.94-0.95 years, but declined at advanced ages. The reduced benefit among the oldest adults may be due to smaller population size or the greater influence of non-pollution-related mortality factors at those ages.

In summary, removal of $PM_{2.5}$ during Period 1 increased life expectancy across all populations, though the timing and magnitude of these benefits differed. Asians and Hispanics exhibited strong and broad sensitivity across the life span, Whites showed steady and late-life responsiveness, and Blacks displayed delayed but substantial gains later in life, reflecting cumulative exposure and health inequities.

Life expectancy in Period 2 continued to show measurable gains from the removal of $PM_{2.5}$, though the magnitude of improvement was generally smaller than in Period 1 (Figure 11). This overall reduction in sensitivity is consistent with California's progressively lower ambient $PM_{2.5}$ concentrations following statewide emission controls and cleaner energy transitions. With less pollution in the environment, the relative benefit of removing $PM_{2.5}$ naturally diminished, though age- and group-specific patterns remained evident.

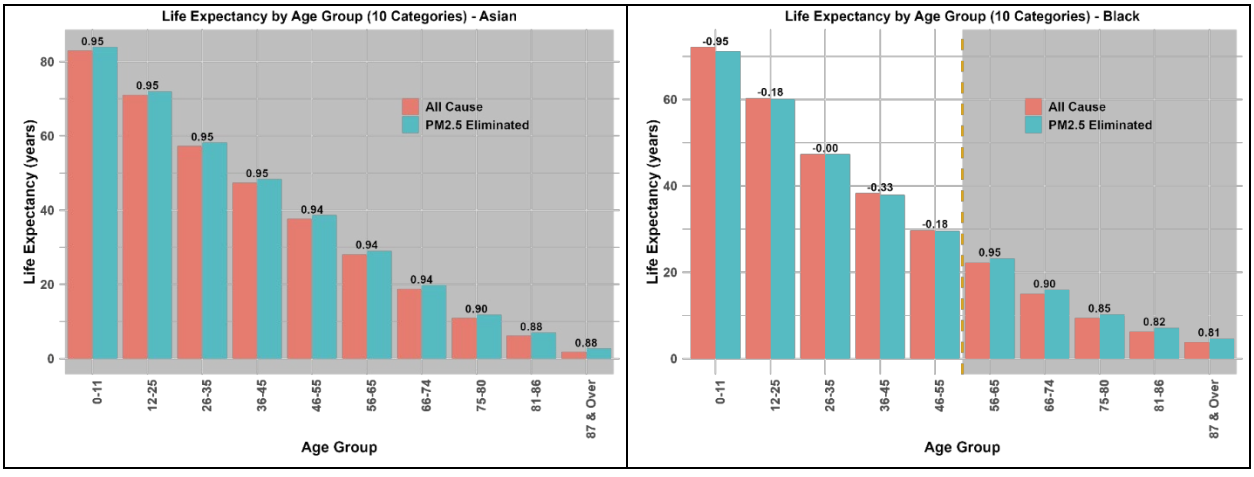
Among Asians, life expectancy gains from $PM_{2.5}$ removal were smaller in early and middle ages compared with Period 1, stabilizing around modest positive levels. The largest improvements appeared during later adulthood, particularly from ages 46-80, where the benefits again approached the upper limit. This shift indicates that as baseline air quality improved, younger Asians—who had already experienced cleaner air for much of their lives—had less exposure to reverse, while older adults still carried the cumulative effects of historical pollution. The persistence of late-life sensitivity suggests that cardiovascular and metabolic pathways remain vulnerable even under lower exposure conditions.

For Blacks, the life expectancy response again contrasted with other groups. Early and middle ages continued to show negligible or negative gains, and the strongest improvements appeared only at the oldest ages. This late-life concentration of benefits points to the enduring effects of cumulative exposure and social determinants such as residential proximity to traffic or industrial areas. In cleaner air conditions, the reversible component of $PM_{2.5}$-related risk becomes smaller in youth, but chronic conditions accumulated over decades still respond to air-quality improvements later in life.

Hispanics retained relatively broad benefits across age groups, although the magnitude declined in middle and older ages compared with Period 1. Life expectancy improvements remained high through about age 55 but turned slightly negative around ages 56-80 before recovering at the oldest ages. This pattern may reflect a transition in exposure profiles—earlier cohorts benefitting from cleaner air policies while older adults retained legacy exposure burdens. Occupational exposure and neighborhood factors likely continued to sustain sensitivity across the life course, but with diminishing returns as overall $PM_{2.5}$ levels fell.

Among Whites, $PM_{2.5}$ removal continued to yield modest but consistent gains across much of the age spectrum, though overall benefits were smaller than in Period 1. The improvements were moderate in younger ages and became most evident in older adults, especially those over 65. The pattern suggests that the remaining gains largely reflect pollution-related cardiovascular and respiratory fragility that persists in later life, while earlier cohorts have already benefited from long-term exposure reduction under California's cleaner-air era.

For Other race-ethnicity groups, life expectancy improvements followed a pattern similar to Period 1, with substantial gains through most ages but some decline in later years. The slight reduction in benefit after about age 70 likely reflects both smaller population sizes and reduced $PM_{2.5}$ exposure intensity statewide, leaving less pollution-related mortality to offset. Nonetheless, their response remained strong overall, suggesting continued vulnerability among populations living in higher-exposure microenvironments.



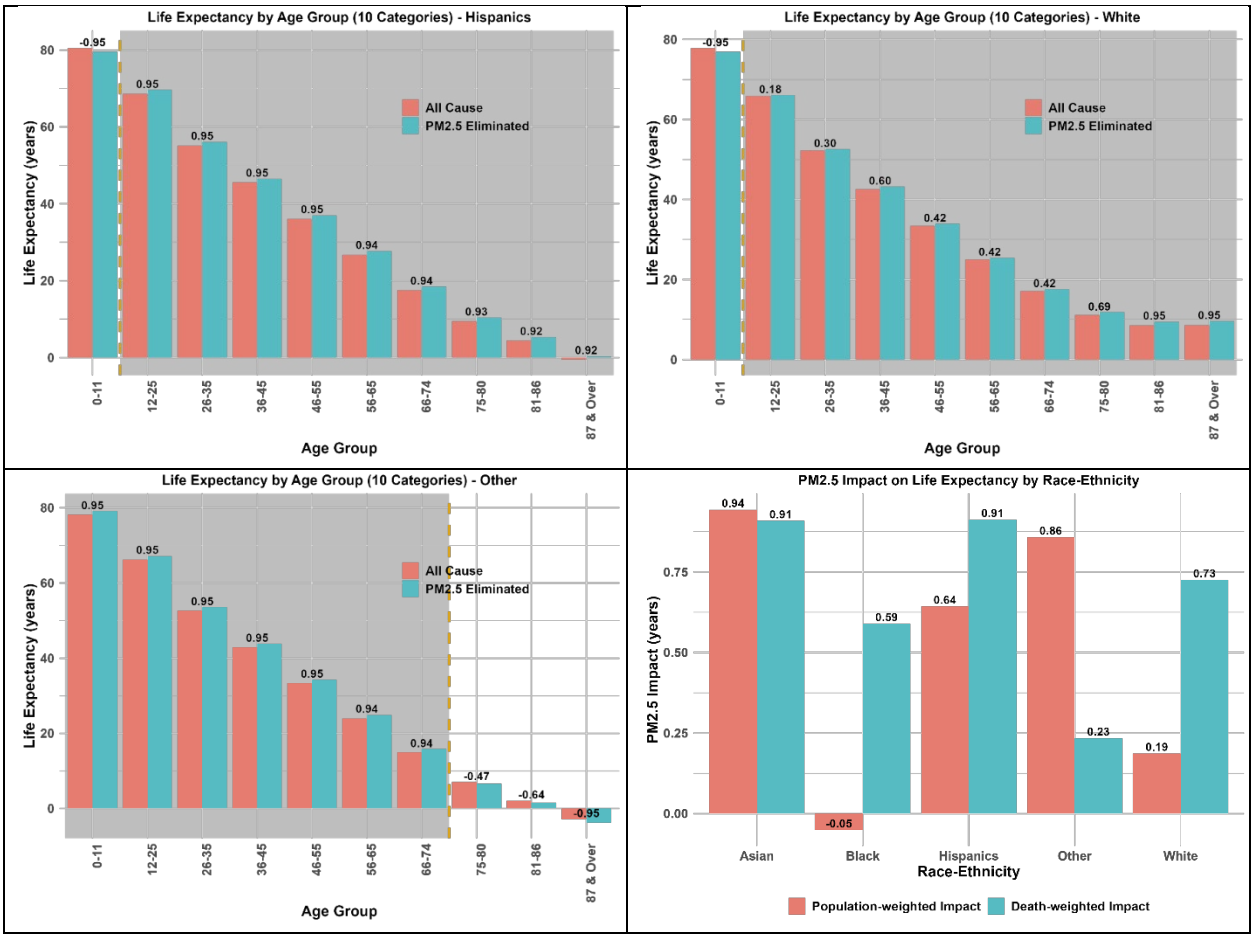


Figure 10. All cause and $PM_{2.5}$-eliminated life expectancy by race-ethnicity for Period 1 (2000-2010) across ten age groups.

Note: The dashed vertical line indicates the point at which the relative magnitude of life expectancy shifts between the all-cause and $PM_{2.5}$-eliminated estimates (i.e., from all-cause > $PM_{2.5}$-eliminated to all-cause < $PM_{2.5}$-eliminated, or vice versa). The shaded grey area denotes age groups for which the estimated life expectancy under $PM_{2.5}$ removal exceeds the observed all-cause life expectancy (expected).

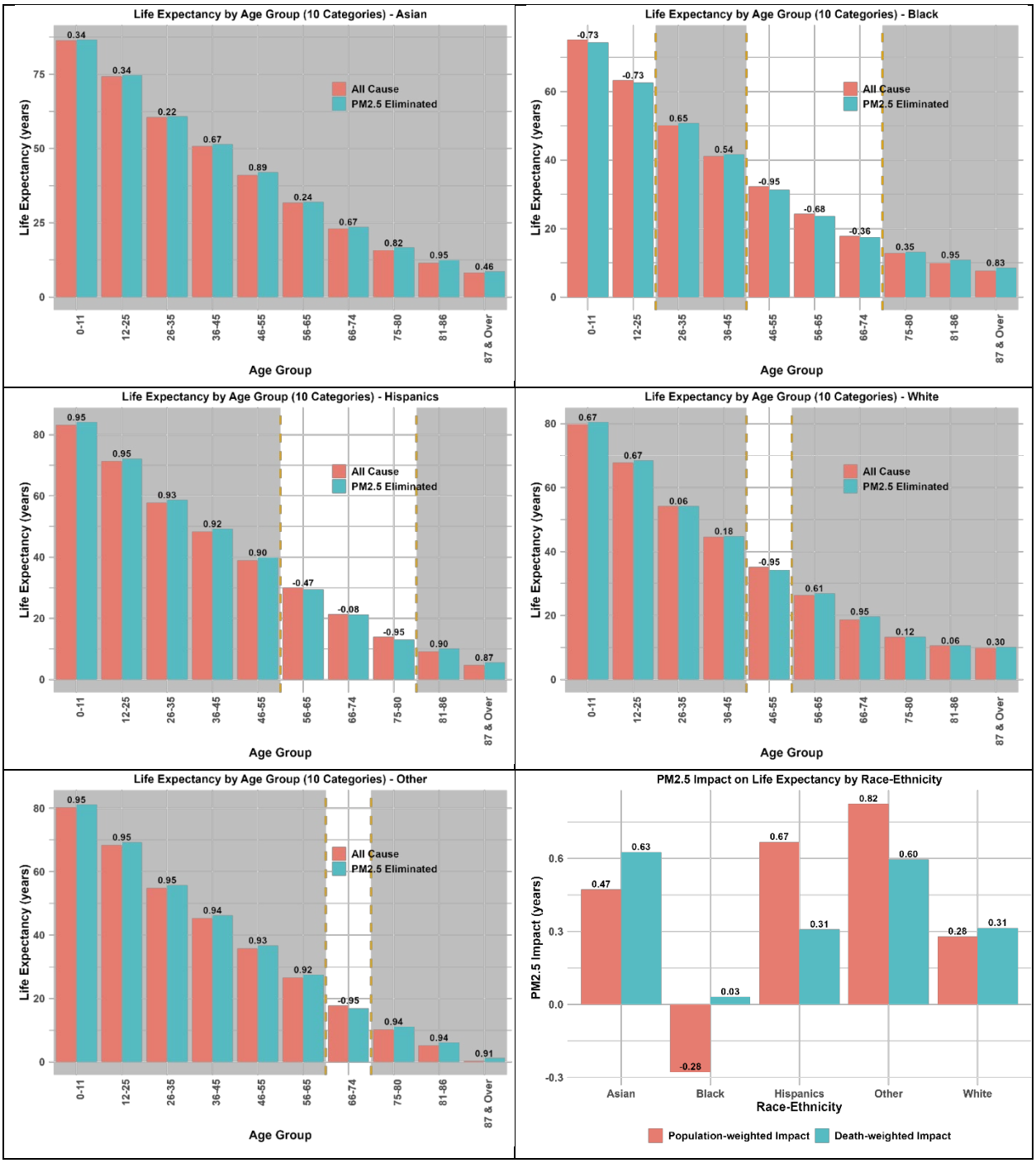


Figure 11. All cause and $PM_{2.5}$-eliminated life expectancy by race-ethnicity for Period 2 (2011-2021) across ten age groups.

Note: The dashed vertical line indicates the point at which the relative magnitude of life expectancy shifts between the all-cause and $PM_{2.5}$-eliminated estimates (i.e., from all-cause > $PM_{2.5}$-eliminated to all-cause < $PM_{2.5}$-eliminated, or vice versa). The shaded grey area denotes age groups for which the estimated life expectancy under $PM_{2.5}$ removal exceeds the observed all-cause life expectancy (expected).

Life expectancy – aggregate impact

When the research team expand the analysis from five to ten age groups, the overall race-ethnicity patterns remain broadly consistent with the coarser five-group results, but several important nuances emerge (Table 5). These differences point to meaningful variation within the broader age categories, variation that becomes visible only when age is modeled more finely.

For Asians, the ten age-group results in Period 1 closely mirror those of the five-group analysis. Population-weighted and death-weighted impacts remain nearly identical (0.94 vs 0.91), reinforcing the earlier conclusion that $PM_{2.5}$ effects are broadly distributed across young, middle-aged, and older adults. In Period 2, both metrics decreased (0.47 population-weighted; 0.63 death-weighted), consistent with an overall reduction in $PM_{2.5}$ impact. The slight rise of the death-weighted value relative to the population-weighted value suggests a modest shift toward a greater proportional impact among older Asian adults in the later decade, a phenomenon also seen in the five age-group results.

For Blacks, the ten age-group results uncover similar effect to the five-group analysis. In Period 1, population-weighted impact (-0.05) is near zero, while death-weighted impact (0.59) is substantially elevated. This again matches the direction of the five-group conclusion, namely that younger Black individuals experience very small per-person $PM_{2.5}$ effects while older adults experience much larger impacts. Period 2 shows a similar pattern to the five-groups, with low population-weighted (-0.28) and death-weighted impact (0.03), again reinforcing the effect of regulatory actions on improving life expectancy (0.56 years in the ten age-group vs 0.59 years in the five age-group analysis).

For Hispanics, the ten-group results reveal an internal age gradient that was obscured in the five-group analysis. Under the five-group structure for Period 1, Hispanics showed relatively uniform benefits across all ages. But with ten groups, Period 1 displays a mismatch: population-weighted impact is moderately high (0.64), but death-weighted impact is very high (0.91). This indicates that while younger and middle-aged Hispanics experience moderate per-person $PM_{2.5}$ effects, older Hispanic adults experience especially elevated impacts that pull the death-weighted average upward. In Period 2, both metrics fall and show a pattern very close to the five age-group analysis: a shift toward higher proportional impact among younger and middle-aged Hispanic populations and a reduced proportional contribution from older Hispanic adults. The overall policy effect of 0.60 years represents the reduction in $PM_{2.5}$-attributable life-expectancy loss between the two periods and is almost identical to the five age-group estimate (0.57 years).

For the Other race-ethnicity category, ten-group results have a similar effect to the five-group results. Period 1 shows high population-weighted impact (0.86) but very low death-weighted impact (0.23), a phenomenon very close to the five-group results. This implies that younger or mid-life adults consistently contribute much more to the observed $PM_{2.5}$ burden than older adults. Period 2 shows both measures increasing (population-weighted 0.82; death-weighted 0.60), indicating that while younger adults continued to show elevated impacts, older adults in

this group experienced a greater increase in their proportional share of $PM_{2.5}$ impact in the later period, a phenomenon similar to that seen in the five age-group analysis.

For Whites, the shift from five to ten age groups produces a dramatic internal re-interpretation. In Period 1, the population-weighted impact drops substantially to 0.19, while the death-weighted impact increases to 0.73. The ten-group results indicate that younger White individuals contribute relatively little, while older Whites experience substantially larger $PM_{2.5}$ impacts. In the five-group version, Whites showed larger impacts at younger ages with diminished effects at older ages. This inconsistency signals that the broad age brackets in the five-group design inadvertently obscured differences within the middle-aged and older-aged ranges, where certain subgroups appear to experience much higher burden than others. Because the White population in California (~35%) is significantly larger than other race-ethnicity groups, the ten age-group stratification did not suffer from small-sample issues (Figure 9, bottom). The ten-group results are therefore the more accurate representation of true age-specific patterns for Whites. In Period 2, the death-weighted impact significantly decreased (from 0.73 to 0.31), suggesting that improvements in air quality reduced late-life $PM_{2.5}$ impacts. Both population-weighted (0.28) and death-weighted impacts are low in Period 2, indicating that per-person $PM_{2.5}$ impacts were uniformly small across all age groups.

Overall, most race-ethnicity groups showed that older adults experienced substantially larger $PM_{2.5}$ impacts in Period 1, while in Period 2 younger and middle-aged individuals contributed relatively more, except for Asians, whose impacts remained broadly distributed, and Blacks, who showed consistently low impacts among younger adults in both periods. Black and Hispanic populations benefited most from regulatory actions, with policy gains of about 0.56–0.59 and 0.57–0.60 years, respectively. By Period 2, $PM_{2.5}$ impacts among Black and White populations had fallen to very low levels, while Asian, Hispanic and Other category populations still exhibited room for further improvement.

Table 5. Summary of life expectancy and policy effects, 2000-2010 vs. 2011-2021

Race Ethnicity	2000-2010				2011-2021				Policy Effect
	All Cause	Remove $PM_{2.5}$	$PM_{2.5}$ Impact (PopWt)	$PM_{2.5}$ Impact (DthWt)	All Cause	Remove $PM_{2.5}$	$PM_{2.5}$ Impact (PopWt)	$PM_{2.5}$ Impact (DthWt)	(DthWt)
	All 20 Age Groups				19 Age Groups				
All	78.40	78.78	0.38	0.61	80.80	81.26	0.46	0.37	0.24
	Five Age Groups								
Asian	83.00	83.94	0.94	0.91	86.30	86.92	0.62	0.74	0.17
Black	72.10	71.86	-0.24	0.68	75.10	75.10	0.00	0.08	0.59
Hispanics	80.50	81.45	0.95	0.94	83.20	83.95	0.75	0.36	0.57
Other	78.20	79.00	0.80	0.06	80.20	80.98	0.78	0.38	-0.31
White	77.80	78.62	0.82	0.40	79.80	80.36	0.56	0.43	-0.03
	Ten Age Groups								
Asian	83.00	83.94	0.94	0.91	86.30	86.77	0.47	0.63	0.28
Black	72.10	72.05	-0.05	0.59	75.10	74.82	-0.28	0.03	0.56
Hispanics	80.50	81.14	0.64	0.91	83.20	83.87	0.67	0.31	0.60
Other	78.20	79.06	0.86	0.23	80.20	81.02	0.82	0.60	-0.36
White	77.80	77.99	0.19	0.73	79.80	80.08	0.28	0.31	0.41

Note: PopWt = population-weighted $PM_{2.5}$ impact on life expectancy; DthWt = death-weighted $PM_{2.5}$ impact on life expectancy.

Task 5. Create GIS Maps for the Study Results

The research team estimated CT-level life expectancy impacts attributable to $PM_{2.5}$ exposure across California for two time periods (2000-2010 and 2011-2021). For each tract and period, the research team first derived population distributions by race-ethnicity and age group. Using modeled life expectancy impacts associated with $PM_{2.5}$ for each age group of a race-ethnicity category, the research team applied age-specific population weights to estimate race-ethnicity level $PM_{2.5}$ effects within a CT. These were then combined using race-ethnicity population weights to generate a single $PM_{2.5}$-related life expectancy impact value for each CT. A general formula is:

$$\mathrm{LE}_{\mathrm{tract}} = \sum_{r}\sum_{a}(\mathrm{Pop}_{r,a} \cdot \mathrm{Effect}_{r,a}) / \sum_{r}\sum_{a}\mathrm{Pop}_{r,a}$$

where r indexes race-ethnicity groups (e.g., Black, Asian, Hispanic, White, Other); a indexes age groups (e.g., 0–11, 12–17, …); $\mathrm{Pop}_{r,a}$ is the population count of age group $a$and race/ethnicity $r$in the tract and $\mathrm{Effect}_{r,a}$ is the estimated PM2.5 impact on life expectancy for that age–race/ethnicity group.

Two aggregation schemes were used: one with five broad age groups and another with ten finer age groups. Because detailed race-ethnicity by age-group mortality data were not available at the CT-level for either period, PM2.5 impacts could not be estimated using death-weighted estimates. Instead, population counts by race-ethnicity and age group are available for all tracts, so they served as the basis for all CT-level $PM_{2.5}$ impact calculations. Here, the CT-level results reflect population-weighted $PM_{2.5}$ impacts. A key assumption is that the estimated effects for a specific age–race/ethnicity group (e.g., Blacks aged 0–11) apply uniformly to all individuals of that age–race/ethnicity group within each tract. This allows the tract-level estimates to reflect local demographic structure, which shapes the spatial patterns of estimated impacts.

Table 6. $PM_{2.5}$ impact and life expectancy statistics over California census tracts.

Impact Assessment (years)	Min	5th Pcnt	10th Pcnt	25th Pcnt	Mean	95th Pcnt	Max	Std
	Five Age Groups							
PM_{25} Impact (2010)	0.05	0.68	0.74	0.79	0.82	0.90	0.95	0.09
PM_{25} Impact (2020)	-0.02	0.50	0.53	0.56	0.61	0.74	0.95	0.08
All Cause LE (2010)	72.90	77.92	78.07	78.31	78.75	80.11	82.33	0.79
All Cause LE (2020)	75.95	80.22	80.38	80.72	81.29	83.09	85.64	0.91
Policy Benefits (P1 -> P2)	-0.36	0.07	0.12	0.18	0.21	0.29	0.52	0.07
	Ten Age Groups							
PM_{25} Impact (2010)	-0.09	0.31	0.34	0.39	0.46	0.66	0.88	0.11
PM_{25} Impact (2020)	-0.19	0.33	0.35	0.38	0.46	0.65	0.81	0.11
All Cause LE (2010)	72.90	77.92	78.07	78.31	78.75	80.11	82.33	0.79
All Cause LE (2020)	75.95	80.22	80.38	80.72	81.29	83.09	85.64	0.91
Policy Benefits (P1 -> P2)	-0.72	-0.16	-0.13	-0.07	0.00	0.22	0.57	0.11

Overall Effect

Under the five age groups scenario (Table 6, top), $PM_{2.5}$ removal in 2010 was associated with an average life expectancy gain of approximately 0.82 years across tracts, ranging from 0.05 to 0.95 years. By 2020, the average tract-level impact declined to about 0.61 years, consistent with overall reductions in $PM_{2.5}$ concentrations statewide. The research team defines policy benefit as the estimated reduction in $PM_{2.5}$-attributable life expectancy loss due to the change in $PM_{2.5}$ concentrations between two time points (here, 2010 and 2020). In other words, it quantifies how much life expectancy is "gained" in years if $PM_{2.5}$ levels are reduced according to observed or modeled improvements, holding all other factors constant. The average policy benefit (i.e., improvement between the two periods) was approximately 0.21 years, suggesting that ongoing air quality regulations and emission controls contributed to measurable public health gains.

Results from the ten age groups scenario (Table 6, bottom) showed smaller average $PM_{2.5}$ impacts in both time periods (around 0.46 years in 2010 and 0.46 years in 2020), and the mean

policy benefit was close to zero. The smaller estimated impacts under the ten age groups framework may partly reflect reduced statistical stability due to smaller subgroup sample sizes, particularly within tracts that have lower population counts or higher proportions of minority residents. This finer age stratification increases model granularity but can also amplify uncertainty in $PM_{2.5}$ effect estimates, especially for subpopulations with limited data. Therefore, while the ten age groups results provide additional detail, the five age groups estimates are likely more robust for summarizing overall $PM_{2.5}$-related life expectancy impacts at the tract level. Although aggregating to five age groups improves statistical stability relative to the ten–age group scenario, CT-level estimates may still be less precise in sparsely populated rural tracts. Accordingly, some spatial variability in the maps, particularly in low-population areas, should be interpreted with caution, as estimates in these tracts remain subject to greater uncertainty due to limited underlying population counts.

Disadvantage Status Stratification

Across California, $PM_{2.5}$-related life expectancy impacts changed between Period 1 and Period 2 in ways that vary by neighborhood disadvantage, as measured by CalEnviroScreen (CES). CalEnviroScreen is a composite index developed by the California Office of Environmental Health Hazard Assessment (OEHHA) that ranks census tracts statewide based on pollution burden (e.g., air pollution, traffic, drinking water contaminants) and population vulnerability (e.g., poverty, education, linguistic isolation, race-ethnicity, and health outcomes). The CES percentile score (here denoted as CIscoreP) ranges from 0 (most advantaged) to 100 (most disadvantaged). For this analysis, tracts were grouped into four disadvantage status categories: most disadvantaged with CES percentile [75, 100], moderately disadvantaged: [50, 75), less disadvantaged: (25, 50) and most advantaged: (0, 25]. The research team discuss the impact of five age groups here (Table 7).

In the most disadvantaged tracts the mean $PM_{2.5}$ impact fell from 0.82 years in Period 1 to 0.66 years in Period 2, and mean all-cause life expectancy rose from about 78.81 to 81.35 years; these tracts also show the greatest variability in $PM_{2.5}$ impacts (SD ≈ 0.12 in Period 1 and 0.09 in Period 2), indicating substantial heterogeneity in exposure and potential benefit within the most disadvantaged communities. The mean policy benefit (Period1 → Period2 difference in $PM_{2.5}$ impact) in this group is 0.16 years.

Moderately disadvantaged tracts follow a similar trajectory: mean $PM_{2.5}$ impact declined from 0.82 to 0.62 years while all-cause life expectancy increased from about 78.82 to 81.39 years. The variability here is notable but slightly smaller than in the most disadvantaged category (SD ≈ 0.10 then 0.09), and the average policy benefit is modestly larger than in the most disadvantaged group (mean ≈ 0.20 years), reflecting somewhat more consistent but still spatially variable gains.

In less disadvantaged tracts the mean $PM_{2.5}$ impact also declined (about 0.82 → 0.58 years) with life expectancy increasing from roughly 78.78 to 81.33 years. These tracts display narrower

dispersion in impacts (SD ≈ 0.06 for both periods) compared with the two more disadvantaged groups, and the mean policy benefit is larger (≈ 0.24 years), indicating more uniform gains from cleaner air across these neighborhoods.

Finally, the most advantaged tracts experienced a mean decline in $PM_{2.5}$ impact from about 0.81 to 0.57 years and an increase in life expectancy from ~78.64 to 81.11 years. This group has the highest average policy benefit (mean ≈ 0.25 years) and the smallest spread in impacts (SD ≈ 0.05 in Period 1 and 0.04 in Period 2), reflecting more homogeneous exposure conditions and more consistent marginal gains from $PM_{2.5}$ reductions.

In sum, $PM_{2.5}$ impacts declined in all strata between periods and overall life expectancy rose everywhere, but the magnitude and consistency of gains vary systematically: the most disadvantaged tracts show the largest heterogeneity (widest spread) and smaller mean policy benefit, while the most advantaged tracts have the most consistent exposures and the largest average policy benefit.

Table 7. $PM_{2.5}$ impact and life expectancy statistics over California census tracts across disadvantage status using five age groups.

Impact Assessment (years)	Min	5th Pcnt	10th Pcnt	25th Pcnt	Mean	95th Pcnt	Max	Std
				Most Disadvantaged				
PM_{25} Impact (2010)	0.05	0.58	0.69	0.79	0.82	0.91	0.93	0.12
PM_{25} Impact (2020)	0.12	0.51	0.57	0.63	0.66	0.77	0.84	0.09
All Cause LE (2010)	73.93	77.41	78.02	78.57	78.81	79.62	82.14	0.78
All Cause LE (2020)	76.98	80.19	80.62	81.10	81.35	82.33	85.64	0.74
Policy Benefits (P1 -> P2)	-0.25	0.01	0.06	0.12	0.16	0.26	0.38	0.07
				Moderately Disadvantaged				
PM_{25} Impact (2010)	0.07	0.67	0.73	0.80	0.82	0.90	0.93	0.10
PM_{25} Impact (2020)	-0.02	0.50	0.53	0.58	0.62	0.73	0.82	0.09
All Cause LE (2010)	72.90	77.89	78.11	78.46	78.82	80.12	82.31	0.85
All Cause LE (2020)	75.95	80.36	80.55	80.95	81.39	82.99	85.60	0.91
Policy Benefits (P1 -> P2)	-0.12	0.09	0.12	0.17	0.20	0.28	0.43	0.06
				Less Disadvantaged				
PM_{25} Impact (2010)	0.30	0.72	0.76	0.80	0.82	0.89	0.93	0.06
PM_{25} Impact (2020)	0.06	0.50	0.52	0.55	0.58	0.69	0.89	0.06
All Cause LE (2010)	74.02	78.00	78.09	78.30	78.78	80.46	82.33	0.78
All Cause LE (2020)	76.97	80.28	80.42	80.70	81.33	83.56	85.57	0.98
Policy Benefits (P1 -> P2)	-0.15	0.14	0.17	0.22	0.24	0.30	0.43	0.05
				Most Advantaged				
PM_{25} Impact (2010)	0.40	0.74	0.76	0.79	0.81	0.88	0.92	0.05
PM_{25} Impact (2020)	0.34	0.50	0.52	0.55	0.57	0.63	0.87	0.04
All Cause LE (2010)	76.57	78.02	78.07	78.20	78.64	80.17	81.99	0.69
All Cause LE (2020)	79.24	80.19	80.27	80.48	81.11	83.29	85.32	0.96
Policy Benefits (P1 -> P2)	-0.17	0.18	0.20	0.23	0.25	0.31	0.48	0.05

The goal of Task 5 was to translate the extensive modeling and analytic results from Tasks 2 through 4 into a geographic format that would be accessible to policymakers, community stakeholders, and researchers. This task required the creation of high-resolution GIS maps that displayed both exposure distributions and the estimated life expectancy impacts attributable to $PM_{2.5}$, stratified by generation, race and ethnicity, and neighborhood vulnerability status. These maps allow identification of areas where life expectancy is most affected, which often correspond to regions with known persistent emissions, including goods-movement corridors, wildfire-prone areas, and other locations with local air pollution sources. By connecting modeled health impacts to geographic patterns and underlying emission sources, the mapping component

provides a critical interpretive tool for understanding the spatial dimensions of $PM_{2.5}$-related health disparities and can guide targeted interventions and community-level decision-making.

The first stage of this work involved preparing the exposure surfaces generated in Task 2 for mapping. The research team aggregated the statewide daily $PM_{2.5}$ surfaces at 100-meter resolution into annual averages at the CT-level, thereby balancing resolution with usability. By summarizing to tracts, the research team were able to align the exposure surfaces directly with demographic and vulnerability data from the Census and CalEnviroScreen. The research team produced tract-level annual averages for 1990, 2000, 2010, and 2020, allowing comparisons across three decades of regulatory progress. These maps revealed dramatic declines in $PM_{2.5}$ concentrations across much of California, particularly in major urban centers such as Los Angeles and the Bay Area. However, they also showed persistent hotspots in the San Joaquin Valley and Inland Empire, regions that remain subject to meteorological inversions and high levels of goods movement-related emissions.

The second stage involved mapping mortality impacts and life expectancy loss attributable to $PM_{2.5}$. Using the coefficients and life table estimates from Task 4, the research team generated tract-level estimates of years of life lost due to $PM_{2.5}$ exposure for each decade. These impacts were mapped under both the twenty-group and aggregated age group frameworks, and stratified by race-ethnicity and vulnerability. For example, in the Aggr2 framework, the research team calculated $PM_{2.5}$-attributable life expectancy loss for Hispanics, non-Hispanic Blacks, non-Hispanic Whites, non-Hispanic Asians, and Others in each CT. These estimates were then visualized in maps that showed spatial clustering of disparities. The San Joaquin Valley consistently displayed the largest life expectancy losses, particularly among Hispanic residents, while non-Hispanic Black populations in Los Angeles tracts were disproportionately affected despite overall improvements in exposure levels.

The research team also developed change maps that illustrated the difference in $PM_{2.5}$ exposure and $PM_{2.5}$-attributable life expectancy loss between the first and second generations. These maps revealed both the magnitude of progress and the persistence of inequities. Statewide, the average $PM_{2.5}$ concentration fell substantially between 1990-2005 and 2006-2020, leading to measurable gains in life expectancy. Yet tracts in the top quartile of vulnerability, as identified by CalEnviroScreen, showed smaller gains and in some cases persistent losses. For example, in many disadvantaged tracts of the Central Valley, the $PM_{2.5}$-related life expectancy loss in the second generation remained as high as the statewide average in the first generation, indicating that improvements have lagged in precisely the communities most burdened by environmental and social vulnerabilities.

The mapping process also allowed us to overlay $PM_{2.5}$ exposures and impacts with policy-relevant boundaries, such as air basins, air districts, and goods movement corridors. This was particularly important for connecting results back to regulatory frameworks. For example, maps stratified by air basin highlighted how improvements in the South Coast and San Joaquin Valley

districts differed in magnitude, and how continued non-attainment challenges in these regions translated into health disparities. Maps of goods movement corridors revealed elevated $PM_{2.5}$ burdens along major freight routes and near ports, illustrating the intersection between economic activity, emissions, and health.

Beyond statewide maps, the research team prepared regional and community-level atlases designed for dissemination in stakeholder meetings. For each region, including Bay Area, Los Angeles, San Joaquin Valley, Inland Empire, and Sacramento, the research team created a series of maps showing baseline exposures, life expectancy loss in the first generation, life expectancy loss in the second generation, and changes over time. These regional atlases were accompanied by demographic overlays, showing the distribution of race-ethnicity and vulnerability. This design allowed community members and local policymakers to see how air pollution health burdens intersect with population characteristics in their own areas.

The final stage of this task involved dissemination formats. All maps were prepared in ArcGIS Pro and exported in formats suitable for integration into ArcGIS Online, making them accessible as interactive layers. Users can zoom into tracts, filter by race or vulnerability, and view time trends. The research team also provided CARB with map packages in CARB-compatible formats for internal use. To further enhance accessibility, maps were incorporated into presentations with plain-language captions, fact sheets, and infographics that explained what the colors and patterns represented in terms of real years of life lost or gained.

The outputs of Task 5 clearly demonstrated that GIS mapping can make the complex statistical findings of Tasks 2-4 both interpretable and actionable. They revealed the tangible successes of air quality policy in reducing $PM_{2.5}$-related mortality, while also underscoring the stubborn persistence of environmental justice disparities across California. For example, Figure 12 illustrates that the San Joaquin Valley experienced some of the highest $PM_{2.5}$-related reductions in life expectancy in 2010, reflecting historically elevated exposure levels from a combination of agricultural emissions, transportation corridors, and meteorological conditions that trap pollution. By 2020, the overall $PM_{2.5}$ impact in the Valley has decreased slightly, consistent with statewide improvements in air quality, yet it remains substantial relative to other regions. The policy benefits map shows the estimated gains in life expectancy attributable to reductions in $PM_{2.5}$ between 2010 and 2020, highlighting that the Valley continues to see meaningful, but comparatively smaller, improvements than less-impacted areas. These patterns underscore the persistent vulnerability of the region to air pollution and suggest that targeted mitigation in high-exposure corridors could yield additional health benefits. The deliverables from this task included a statewide GIS atlas, regional atlases for key air basins, and a full suite of map files suitable for CARB's use in both internal policy analysis and external communication.

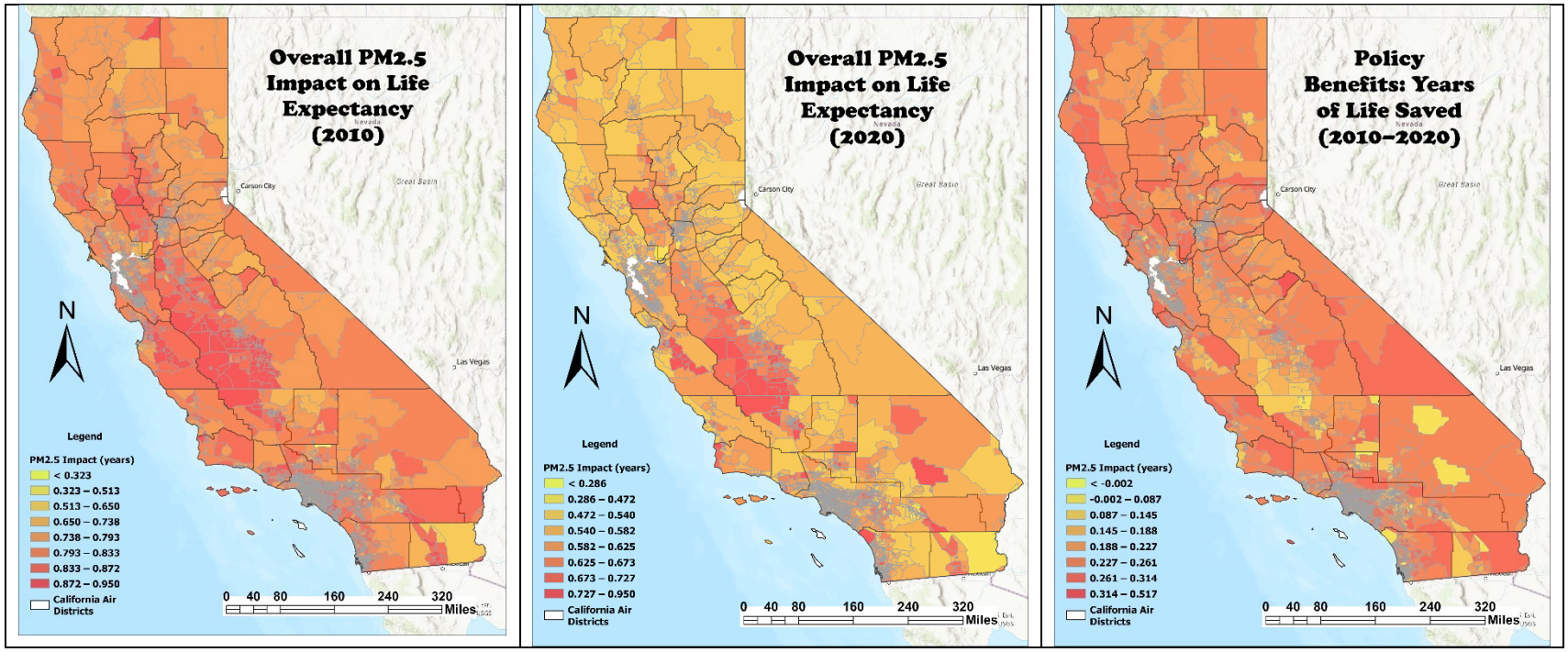


Figure 12. Overall $PM_{2.5}$ impact on life expectancy for 2010 (left), 2020 (middle), and the policy benefits representing years of life saved from 2010 to 2020 (right).

Task 6. Address Impacts in Communities

As part of the project's community outreach efforts, the Regional Asthma Management and Prevention program (RAMP) played a central role in communicating the study's goals, design, and early findings to diverse audiences across California. RAMP is widely recognized for its long-standing leadership in asthma prevention, environmental health, and health equity, as well as for its strong collaborations with community organizations, health professionals, environmental justice advocates, and policy networks. Their established relationships and community trust made them an ideal partner to ensure that the project's communication reached stakeholders who are both directly affected by air pollution and actively engaged in improving community health.

In March 2024, RAMP hosted two community webinars titled "Understanding the Impacts of Air Pollution on Life Expectancy Across Communities – Study Design and Partner Feedback." The sessions, held on March 26 and March 28 at different times to accommodate varied schedules, presented identical material and were organized to reach community members, medical providers, environmental justice and tenant-rights advocates, and other stakeholders in asthma, healthcare, and health equity. RAMP broadly advertised the webinars through its RAMP Digest e-newsletter (which reaches over 1,200 recipients), the Community Action to Fight Asthma Network, and the California Alliance for Children's Environmental Health. In addition to these channels, RAMP provided a preview of the webinar content during a meeting of the San Diego–Tijuana Air Quality Task Force after a RAMP partner shared the webinar announcement with the Task Force facilitator; this preview functioned as an additional outreach touchpoint prior to the webinars themselves. To support accessibility, the presentation slides were translated into Spanish and attendees were offered gift cards. More than 100 participants attended the March 26 session and more than 40 attended the March 28 session. RAMP shared the webinar slides with registrants after the events. Webinar participants represented a diverse cross-section of California communities, including both urban and rural areas, regions with high $PM_{2.5}$ exposure, and neighborhoods with elevated asthma prevalence and other environmental health burdens. Attendance included stakeholders from environmental justice communities, public health organizations, healthcare providers, and advocacy groups, reflecting the geographic and demographic diversity of populations disproportionately impacted by air pollution. This breadth of participation helped ensure that outreach efforts captured perspectives from communities most affected by the study's findings.

Each webinar opened with introductions and an overview, included contextual background from RAMP about the links among air pollution, asthma, and health including mortality, featured the study presentation by Dr. Jason Su, and concluded with a question-and-answer and discussion session. Participants' feedback, collected during and after the webinars, affirmed the relevance of the research to a range of practical activities. Respondents noted the project's applicability to asthma care and indoor-air-quality work, the usefulness of satellite-based PM

monitoring for research contexts, and the importance of identifying interventions that reduce pollutant exposure for people with respiratory conditions. Several participants urged more investment in public transportation as a means to reduce emissions. Others emphasized dissemination methods that would bring the findings to community members directly, proposing flyers, posters, emails, recorded videos, and webinars as channels to broaden reach. Suggestions to increase public awareness and to tailor communications to community needs, for example, through translated materials and visually accessible flyers, were raised repeatedly. The project team plans to incorporate several participant-recommended dissemination approaches in future efforts. These include producing translated materials, visually accessible flyers and infographics, short recorded videos, targeted emails, and continued webinars to broaden reach. By operationalizing these suggestions, the team aims to enhance the accessibility and impact of research findings for both technical and non-technical audiences, including families, community organizations, and policymakers.

Participants also described how the research could inform policy and systems change, noting that clearer and more accessible presentation of results could support advocacy and education. Several respondents said dissemination via community-facing materials such as flyers or posters, targeted emails, recorded videos on platforms like YouTube, and inclusion in newsletters would help reach non-technical audiences. Attendees recommended continuing monitoring and outreach and suggested that policy makers be presented with concise, actionable information. A number of respondents commented that the research should be disseminated through both academic outlets and public-facing channels to serve distinct audiences: some recommended publication in journals while others emphasized community education and practical tools for affected families. Finally, participants proposed additional research directions, including investigation of whether and how policy makers incorporate air-pollution evidence into project decisions, indicating interest in future work that connects scientific findings to policy uptake. Insights from participants highlight the importance of connecting scientific findings on $PM_{2.5}$ and life expectancy to actionable guidance. For example, emphasizing tract-level disparities and clearly illustrating health benefits from $PM_{2.5}$ reductions can help policymakers prioritize interventions in high-exposure areas. Recommendations on concise, visually clear, and community-tailored communications will guide future outreach strategies, supporting evidence-informed decisions in public health planning, environmental justice initiatives, and local policy development.

Overall, community feedback from RAMP's webinars directly informed the study's approach to communication and dissemination. Suggestions from participants helped shape the framing of results, emphasizing accessible presentation of life-expectancy impacts and clear contextualization of $PM_{2.5}$ exposure. Input on visual materials, translated slides, and community-friendly formats guided refinements to outreach products, while participant questions and discussion highlighted topics for further clarification and explanation. This engagement ensured that stakeholder perspectives were considered in translating complex modeling outcomes into practical, actionable information for diverse audiences.

Discussion

This study provides a unique comprehensive evaluation to date of how $PM_{2.5}$ affects mortality risk and life expectancy in California, combining two decades of population-wide mortality data with high-resolution exposure modeling and multiple layers of stratification. A central contribution of this work is addressing a critical gap in literature: while a large body of research evaluates $PM_{2.5}$-related mortality risks, only a small number of studies provide direct life-expectancy estimates. Among those that do, most present single-period estimates, rarely examine how impacts evolve over time, and almost none evaluates both mortality risks and life-expectancy impacts across two distinct decades. Even fewer studies incorporate race-ethnicity and detailed age-group stratification, and to our knowledge, no prior work has examined how the distributional structure of $PM_{2.5}$ impacts, across population versus deaths, changes from one period to another. This analysis therefore adds substantially to the scientific evidence base by providing (1) $PM_{2.5}$ impacts on mortality risks and their changes from one period to another, (2) decade-specific overall life-expectancy impacts and changes between periods, (3) multi-age and multi-race analyses within a period and changes across periods, and (4) the first application of a dual population-weighted and death-weighted $PM_{2.5}$ impact framework to detect whether younger or older age groups were impacted more.

The decision to divide the analysis into two consecutive periods, 2000–2010 and 2011–2021, was driven by methodological, interpretive, and data-related considerations rather than an intent to maximize differences or align with any single regulatory milestone. First, our primary objective was to evaluate how $PM_{2.5}$-related mortality risks and life-expectancy impacts evolved over time as air quality improved, while maintaining sufficient continuity in population structure, exposure assessment, and mortality data quality. Using consecutive periods allows us to examine temporal change under broadly comparable demographic and epidemiologic contexts, reducing confounding from large shifts in population composition, healthcare access, or baseline mortality patterns that would be more likely with widely separated periods. A larger temporal gap could indeed produce greater contrasts in $PM_{2.5}$ concentrations, but it would also introduce additional sources of heterogeneity that would complicate interpretation of changes in life expectancy and mortality burden. Second, the selected cut point was not chosen to align with a single regulatory action or to amplify observed differences. California's air quality improvements reflect a long sequence of regulatory and technological changes implemented over several decades, rather than a discrete policy event occurring around 2010. There was no specific regulation enacted in that year that would justify treating 2010 as a causal breakpoint. Instead, the division approximately separates an earlier decade characterized by higher ambient $PM_{2.5}$ concentrations from a more recent decade in which concentrations were substantially lower and more spatially homogeneous. This allows us to assess whether health impacts persisted, attenuated, or shifted across age and race-ethnicity groups as the exposure distribution changed. Third, dividing the study period into two roughly equal-length intervals improves statistical stability and interpretability. Each period contains sufficient deaths across age and race-ethnicity strata to support stratified mortality

modeling and life-expectancy estimation. Using shorter or more fragmented time windows would reduce power and increase uncertainty, while using a single long period would obscure meaningful temporal changes in both exposure levels and health impacts. Finally, the research team emphasize that our conclusions do not rely on large absolute differences between periods. In fact, the central finding is that although $PM_{2.5}$-related mortality risks and life-expectancy impacts declined from Period 1 to Period 2, substantial impacts remained, and the distribution of those impacts across age groups and race-ethnicity groups changed. The observed trends are therefore interpreted as evidence of regulatory progress coupled with persistent and evolving vulnerability, rather than as an artifact of period selection.

In addition to filling these scientific gaps, the results align with and extend the established epidemiologic literature. Numerous long-term $PM_{2.5}$ studies worldwide report elevated all-cause mortality risks per incremental increase in $PM_{2.5}$, typically with odds ratios between 1.04 and 1.15. In our integrated analysis spanning both study periods (2000-2021), the age-specific logistic regression models produced coefficients ranging from 0.025 to 0.16. The reported coefficients correspond to the estimated change in the log-odds of mortality per IQR increase in $PM_{2.5}$. This approach allows the coefficients to reflect the effect associated with a typical population-level range of variation in $PM_{2.5}$ exposure and facilitates comparison across age groups and with other studies reporting IQR-based effect estimates. These values correspond to odds ratios between approximately 1.03 and 1.17, indicating modest but consistent increases in risk associated per IQR increase in $PM_{2.5}$ exposure. The effect estimates obtained in our study fall squarely within this expected range, which reinforces their plausibility and supports the robustness of our findings. Likewise, the death-weighted life-expectancy losses estimated here, 0.61 years in Period 1 and 0.37 years in Period 2, closely match those reported by major national life-expectancy studies, including Correia et al., Schwartz et al., and Chen et al.,[1–3] all of which identify $PM_{2.5}$-related life-expectancy reductions between 0.3 and 0.9 years. These consistencies provide strong external support for the magnitude of effects estimated in this report and for the observed reductions in $PM_{2.5}$ impacts across decades.

Beginning with the unstratified, population-wide analyses, the results confirm that $PM_{2.5}$ remained a significant determinant of mortality and life expectancy in California. When the analysis was carried out separately for Period 1 (2000–2010) and Period 2 (2011–2021), mortality risks associated with long-term $PM_{2.5}$ exposure were positive and statistically significant across all age groups, and the effect weakened in Period 2, reflecting improvements in air quality resulting from California's long-standing regulatory actions and emission-control programs. The overall death-weighted life-expectancy impact fell from Period 1 to Period 2 (0.61 to 0.37 years), which is central to the study's principal finding and reflects the combination of lower ambient $PM_{2.5}$ concentrations and the cumulative effects of California's regulatory and technological advances. Because the death-weighted metric places emphasis on age groups that account for most deaths, it is especially informative about the public-health burden that $PM_{2.5}$ imposes on the mortality structure. The reduction in the death-weighted impact demonstrates that

regulatory progress translated into meaningful reductions in $PM_{2.5}$-related mortality at the state level.

When results are disaggregated by age group and race-ethnicity, care must be taken to not overinterpret these highly disaggregated results. However, important and policy-relevant patterns emerge. Across most race-ethnicity groups, older adults tended to bear the larger $PM_{2.5}$ mortality impacts in Period 1. In Period 2, the contribution of younger and middle-aged groups grew relative to older adults. This pattern is likely driven by reductions in $PM_{2.5}$ exposure among older adults, improved baseline health in later life, and demographic shifts that increased the population and baseline mortality of working-age adults. Additionally, ongoing exposures in occupational and commuting settings, which are more relevant for younger and middle-aged adults, may have amplified the relative impact on these groups despite overall lower per-unit risk. These findings suggest that while older adults remain highly susceptible, interventions targeting ambient and workplace exposures among younger and middle-aged populations could meaningfully reduce the total $PM_{2.5}$ burden.

Asians are a notable exception: their $PM_{2.5}$ impacts remained broadly distributed across ages in both periods, with population- and death-weighted impacts nearly identical in Period 1 (0.94 vs. 0.91), indicating that impacts were not concentrated exclusively in either the young or the old. Black populations consistently exhibited low impacts among younger adults in both periods and concentrated impacts in later life. Hispanic populations showed large, broadly distributed gains from $PM_{2.5}$ removal in Period 1 and retained substantial sensitivity in Period 2. When policy gains between periods are quantified using the change in death-weighted impacts, Black and Hispanic groups stand out as having realized the largest improvements: approximately 0.59 years for Black populations and 0.57 years for Hispanic populations, reflecting sizable reductions in $PM_{2.5}$-attributable mortality that accrued to these groups between the two decades. By Period 2, PM2.5 impacts for Black and White populations had fallen to comparatively low levels on a death-weighted basis, whereas Asian, Hispanic, and Other groups still showed meaningful remaining life-expectancy losses attributable to $PM_{2.5}$. Interpretation of racial-ethnic patterns in $PM_{2.5}$-attributable life-expectancy loss requires consideration of baseline life-expectancy differences across groups. In California, Asian and Hispanic populations have the highest baseline life expectancy, while Black populations have the lowest. These demographic patterns mean that a given $PM_{2.5}$-attributable reduction represents a different proportional burden across groups. For example, Asian and Hispanic populations may show notable remaining $PM_{2.5}$ impacts in Period 2 despite having long life expectancy overall, while Black populations may show smaller remaining $PM_{2.5}$ impacts but still experience lower total life expectancy due to accumulated historical and structural factors. Clarifying these contextual differences helps ensure that the results are interpreted as changes in $PM_{2.5}$-related burden rather than as statements about absolute longevity or intrinsic population vulnerability. These race-ethnicity patterns point to the combined effects of historical exposure patterns, differential prevalence of chronic conditions, and the spatial distribution of populations, and broader structural determinants such as housing

conditions, cumulative environmental burdens, and differences in healthcare access, which may contribute to persistent disparities in $PM_{2.5}$ impacts despite overall statewide improvements.

To characterize spatial variation, the research team translated the race-ethnicity and age-group–specific model results to the CT-level by applying modeled, race-ethnicity specific age-group impacts to each tract's demographic composition. This CT-level implementation produced spatially explicit maps of both absolute life expectancy and the change in $PM_{2.5}$-impacted life expectancy from Period 1 to Period 2. CT-level outcomes reveal that ongoing air quality regulations and emission controls contributed to measurable public-health gains (impact from 0.82 to 0.61 years). However, these CT-level results also show that statewide progress does not eliminate local variation: policy successes reduced the statewide death burden, but tract composition and legacy exposure patterns continue to shape where residual impacts remain. Some tracts experienced marked reductions in $PM_{2.5}$-related life-expectancy losses between periods, whereas others showed only modest improvements.

During the study window, $PM_{2.5}$ standards were revised multiple times at both the federal and California levels. The federal annual $PM_{2.5}$ National Ambient Air Quality Standard (NAAQS) was established at 15 $\mu g/m^3$ in 1997 and later tightened to 12 $\mu g/m^3$ in 2012. California adopted an annual $PM_{2.5}$ standard of 12 $\mu g/m^3$ earlier, in 2002, along with a 24-hour standard of 35 $\mu g/m^3$, thereby advancing more stringent controls ahead of the federal revision. These regulatory milestones align temporally with the substantial decline in ambient $PM_{2.5}$ concentrations observed between Period 1 and Period 2. While our study does not attribute effects to any single regulatory action, the alignment between standard tightening and declining $PM_{2.5}$-attributable mortality burden supports the interpretation that regulatory progress played a central role in shaping the observed temporal trends. These standard revisions and CARB regulatory actions likely contributed to the observed reductions in life-expectancy impacts between periods by accelerating emission reductions from mobile, industrial, and area sources.

These findings provide policymakers with evidence that $PM_{2.5}$ control continues to deliver tangible mortality benefits. These findings also suggest that the distribution of remaining harms has shifted. Maintaining and strengthening source-specific emission controls, especially for mobile sources and other persistent $PM_{2.5}$ contributors, remains the most reliable path to further reducing impacts. Because younger and middle-aged contributions rose in Period 2 for many groups, policies and programs should broaden their focus beyond solely protecting older adults. Investments that reduce exposures for working-age populations (e.g., workplace protections, transit and freight routing policies, and community buffer measures) can now yield important mortality reductions. Continued targeted interventions for groups and tracts with remaining high impacts are essential. CT-level maps identify where tract composition and residual exposures concentrate risk, allowing CARB and local health departments to prioritize these locations for additional monitoring, community-level mitigation, exposure-reduction strategies in schools and workplaces, and health-system supports. These insights can also guide AB 617 communities in implementing focused interventions where the burden remains highest.

The particularly large policy gains for Black and Hispanic populations underscore that regulatory action can narrow disparities, and sustained attention to environmental justice, including ensuring that emission reductions reach communities of color and disadvantaged tracts, remains essential to extending these gains. Finally, complementing ambient reductions with policies that address downstream vulnerability, such as improving chronic disease management, expanding preventive and primary care, and addressing social determinants of health, will amplify life-expectancy gains from cleaner air.

Another key contribution of this research is the innovative application of the dual population-weighted and death-weighted $PM_{2.5}$ impact framework to detect whether younger or older age groups were more impacted. The research team identified that when the overall population-weighted impact is relatively high, but the death-weighted impact is relatively low, younger or middle-aged groups experience moderate-to-high per-person $PM_{2.5}$ impacts while the elderly experience smaller per-person impacts. When the population-weighted impact is relatively low, but the death-weighted impact is relatively high, the reverse pattern holds: the numerically dominant younger or middle-aged groups have relatively low per-person impacts while elderly groups have moderate-to-high per-person impacts. When both population-weighted and death-weighted impacts are high, all major age groups exhibit moderate-to-high per-person impacts. When both are low, per-person impacts are uniformly small across all age groups. Using these criteria, the research team can confidently identify the relative degree of $PM_{2.5}$ impact across age strata. It is important to note, however, that this framework is sensitive to the underlying mortality structure and demographic composition of the population. Differences in age-specific death rates or the relative size of age and race-ethnicity groups can influence the comparison between population-weighted and death-weighted impacts. Users applying this approach in other populations or settings should consider how local mortality patterns and population distributions may affect the interpretation of relative $PM_{2.5}$ impacts across age groups.

The observed heterogeneity across race-ethnicity and age groups in this study should not be interpreted as evidence of intrinsic biological susceptibility differences to $PM_{2.5}$. Our intent is not to imply race-specific biological mechanisms, but rather to describe how $PM_{2.5}$-related mortality impacts are distributed across populations that differ systematically in exposure histories, baseline health, socioeconomic conditions, and mortality risk profiles. Race-ethnicity in this study functions as a marker for these correlated structural and contextual factors, not as a causal biological construct. To mitigate confounding by secular trends and other time-varying factors, we used a matched case-control design in which each death was matched to up to two controls who were alive at the time of death and matched on birth year and month, sex, and race-ethnicity. This design ensures that cases and controls are drawn from the same underlying population and mortality risk context within each period, so that long-term improvements in healthcare, prevention, and baseline mortality operate similarly on both groups. As a result, differences in $PM_{2.5}$ associations are less likely to reflect broad secular changes or compositional shifts and more likely to capture contrasts in exposure within comparable risk sets. While residual

confounding by unmeasured individual-level factors is still possible, the matching strategy substantially reduces confounding related to age, cohort effects, sex, race-ethnicity, and calendar time within each period. We acknowledge that some age-by-race strata involve small numbers of deaths, particularly at younger ages and within smaller population groups. This limitation motivated the use of aggregated age-group schemes (10 age-group and 5 age-group models) to stabilize estimates and improve interpretability. Even with aggregation, some counter-intuitive or non-significant associations can arise due to limited statistical power rather than true protective effects. For this reason, we do not emphasize isolated age-specific coefficients for particular race-ethnicity groups. Instead, the primary focus of the analysis is on integrated patterns, population-weighted and death-weighted life-expectancy impacts within each period and changes between periods, which are far more stable and policy-relevant summaries of $PM_{2.5}$ burden.

This study has limitations that temper causal claims about any single mechanism. Although this study relied on high-resolution (100-m) daily $PM_{2.5}$ surfaces linked to individual residential locations at time of death, residual misclassification may arise from factors such as residential mobility, variability in address geocoding completeness, and differences in model performance across urban, suburban, and rural settings. These factors could, in principle, affect demographic groups differently if mobility patterns or residential contexts vary systematically by age or race-ethnicity. Several aspects of the study design mitigate this concern. First, we removed individuals with less than one year of residence in their county. Second, exposure was assigned at the individual level using fine-scale, spatially resolved surfaces rather than area-level averages, reducing spatial smoothing error relative to coarser exposure metrics. Third, the matched case-control framework ensures that cases and controls within each stratum are drawn from the same underlying geographic and demographic context, so non-differential exposure error within strata is more likely to attenuate effect estimates than to create spurious differences across groups. Importantly, any remaining exposure misclassification is expected to bias associations toward the null rather than generate artificial heterogeneity across age or race-ethnicity strata. As a result, the observed differences in population-weighted and death-weighted life-expectancy impacts are unlikely to be driven solely by exposure measurement error. Nevertheless, we recognize that subgroup-specific estimates, particularly for smaller populations and younger age groups, may carry greater uncertainty. For this reason, the study emphasizes aggregated, distributional metrics (population-weighted and death-weighted impacts and changes across periods) rather than isolated age-by-race coefficients, and interprets stratified findings in a descriptive and policy-relevant context rather than as precise estimates of biological susceptibility.

Another limitation of this analysis is that $PM_{2.5}$ exposure was treated as a single mass-based metric and was not differentiated by emission source or chemical composition. Because the toxicity and associated health impacts of equivalent $PM_{2.5}$ concentrations may vary by source, region, and time period, incorporating source- or composition-specific information could further inform the interpretation of results and support more targeted regulatory strategies. Accordingly,

these census tract–level estimates are best interpreted as a screening tool that can be used in conjunction with source-specific emissions information, chemical speciation data, or targeted monitoring campaigns to help identify priority locations where more detailed investigation and causal assessment may be warranted. However, we understand that implementing such source- and composition-specific analyses at a statewide scale over two decades would be technically and logistically challenging, and very likely infeasible due to the lack of consistent, long-term, high-resolution emissions and daily chemical speciation data. Even if such data were available, the required modeling and analytical effort would be substantial and would likely entail costs on the order of tens of millions of dollars.

The CT-level results depend on tract composition and modeled per-group impacts. They identify where benefits were realized and where residual harms persist, but they do not by themselves prove local source attribution. . Despite these caveats, the combination of population/death-weighted analysis, detailed age- and race-ethnicity stratification, and CT-level mapping provides a clearer picture of how regulatory progress translated into mortality gains and how those gains were distributed. In short, California's regulatory actions substantially reduced the burden of $PM_{2.5}$ between the two periods, Black and Hispanic populations appear to have realized the largest policy gains, while residual impacts remain most salient in Asian, Hispanic, and Other groups and in particular census tracts, evidence that argues both for continued statewide controls and for targeted local and health-system strategies to further reduce air-pollution-related mortality while also addressing remaining disparities.

Conclusion

This study provides the most comprehensive assessment to date of long-term $PM_{2.5}$ impacts on mortality and life expectancy in California and offers new insight into how these impacts changed over two decades of major air-quality improvements. By integrating individual-level mortality records with high-resolution modeled exposures and stratified hazard models, the analysis fills critical gaps in the literature, particularly the scarcity of studies examining long-term $PM_{2.5}$ mortality risks and life-expectancy impacts across two separate decades. The findings therefore provide a valuable evidence base for ongoing regulatory evaluation and future standard-setting. The analytical framework is readily transferable to other U.S. regions and to nationwide studies, provided that comparable long-term, daily high-resolution $PM_{2.5}$ exposure data and corresponding individual-level mortality records are available. While the magnitude and distribution of estimated impacts may differ across regions due to variations in age structure, baseline health conditions, regulatory history, and ambient $PM_{2.5}$ mixtures, such differences would reflect underlying population and exposure characteristics rather than limitations of the modeling approach itself. As a result, the conclusions drawn for California should be interpreted as context-specific in magnitude, but broadly informative regarding the application of this framework to other regions with differing demographic and environmental profiles.

The strongest and most robust results of this study are the core mortality-risk estimates, the decline in long-term $PM_{2.5}$-attributable mortality between the two periods, and the overall life-expectancy gains observed statewide. These estimates derive from well-established epidemiologic methods applied to population-wide mortality data, and their stability across modeling choices gives them high reliability. The substantial drop in the statewide death-weighted life-expectancy impact, from 0.61 years in 2000-2010 to 0.37 years in 2011-2021, reflects a clear, consistent signal that California's regulatory and technological actions have translated into real improvements in public health.

More granular findings, such as age-group specific shifts, race-ethnicity specific differences, and census-tract level heterogeneity, provide important insights but should be interpreted with somewhat more caution. These subgroup estimates illuminate meaningful patterns, such as the increasing contribution of younger and middle-aged populations in Period 2, or the particularly large policy gains for Black and Hispanic populations, but they can be influenced by smaller sample sizes, differential statistical power, and subgroup-specific demographic structures. Further, these findings should be viewed in the context of persistent differences in baseline life expectancy across racial-ethnic groups in California, which shape the proportional meaning of $PM_{2.5}$-related losses and help prevent misinterpretation of our results. Similarly, the tract-level mapping results effectively reveal where residual burdens remain, but they reflect demographic composition and modeled exposure surfaces rather than direct causal attribution to specific local sources. These subgroup- and tract-level estimates provide a basis for identifying areas or populations with higher $PM_{2.5}$-related impacts. They are intended for prioritization and screening purposes, not for attributing effects to specific emission sources. These results are therefore best viewed as informative and policy-relevant, but preliminary with respect to fine-scale causal inference. The ranking of the study findings from most trustworthy to more informative is provided in Table 8.

Table 8. Ranking of findings by reliability and policy relevance

Tier	Findings	Rationale for Level of Confidence
Most trustworthy / strongest evidence	Statewide association between long-term $PM_{2.5}$ exposure and all-cause mortality across both periods	Based on population-wide mortality data, large sample sizes, and well-established epidemiologic methods with stable estimates
	Decline in $PM_{2.5}$-attributable mortality risk from Period 1 (2000–2010) to Period 2 (2011–2021)	Consistent signal across models reflecting large-scale regulatory and air quality improvements
	Reduction in statewide death-weighted life-expectancy loss from 0.61 years to 0.37 years	Derived from integrated life-table calculations using stable mortality-risk estimates; robust to modeling choices

Tier	Findings	Rationale for Level of Confidence
High confidence, policy-relevant	Overall statewide life expectancy gains attributable to reductions in $PM_{2.5}$	Direct translation of robust mortality-risk estimates into life-expectancy metrics
Informative, interpret with some caution	Age-group–specific shifts in $PM_{2.5}$ impacts, including increased relative contribution of younger and middle-aged groups in Period 2	Relies on stratified models with reduced sample sizes within age groups
	Race- and ethnicity-specific differences in $PM_{2.5}$-related life-expectancy impacts and policy gains	Informative for equity and policy evaluation, but influenced by subgroup size and baseline life-expectancy differences
Exploratory / spatially informative	Census-tract level variation in $PM_{2.5}$-attributable life-expectancy impacts and changes over time	Stable in high-population tracts, but more uncertain in small tracts due to demographic composition and statistical variability
	Regional and community-level spatial patterns of remaining $PM_{2.5}$ burden	Useful for identifying broad areas of concern, not for fine-scale causal attribution

Taken together, the study provides CARB with a set of actionable insights. At a statewide level, the findings confirm that decades of emission controls have yielded substantial, measurable reductions in $PM_{2.5}$-related mortality and life-expectancy losses. This provides strong support for continuing to strengthen $PM_{2.5}$ standards, reinforce mobile-source and area-source regulations, and maintain California's current regulatory trajectory. The decade-over-decade improvements also give CARB an empirically grounded demonstration of the public-health value of its past policies: evidence that can be used in regulatory impact assessments, state implementation plans, and benefit-cost analyses. Additionally, these results can inform coordination with local health departments, air districts, and community organizations to guide targeted mitigation efforts and public-health communication strategies.

The subgroup and tract-level findings help CARB identify which populations and locations should be prioritized for the next generation of air-quality interventions. The results indicate that residual $PM_{2.5}$ impacts increasingly cluster in communities with specific demographic compositions, especially Asian, Hispanic, and Other populations, and that some census tracts still experience comparatively high $PM_{2.5}$-related life-expectancy losses, which likely reflect not only differences in $PM_{2.5}$ exposure but also interactions with broader social and structural determinants such as chronic disease burden, housing quality, and access to preventive care. These patterns can directly inform CARB's targeting of community-level mitigation efforts, planning of monitoring expansions, refinement of EJ-focused programs, and coordination with local agencies under initiatives such as AB 617. The shift toward greater impacts among younger

and middle-aged adults in Period 2 also suggests that CARB may consider strategies that reduce exposures in workplaces, transportation corridors, and freight systems, settings especially relevant for these age groups.

In sum, this study shows that California's regulatory actions substantially reduced the long-term mortality burden of $PM_{2.5}$, extended life expectancy statewide, and narrowed key race-ethnicity disparities. At the same time, it identifies emerging demographic patterns and geographic concentrations of remaining impacts. The results offer CARB both strong, robust evidence to support statewide regulatory strategies and a set of preliminary but highly informative indicators to guide targeted, equity-focused interventions. Together, these findings underscore that continued emission reductions, when paired with localized and population-specific investments, remain essential to ensure that cleaner air translates into longer, healthier lives for all Californians. Looking ahead, incorporating additional data sources, such as $PM_{2.5}$ chemical speciation, source-specific measurements, enhanced monitoring, and refined health-outcome linkages, could further improve future assessments and help prioritize regulatory and public-health interventions.

Supplementary Figures

Figure S 1**. PRISMA Study Selection Flow Diagram.** PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

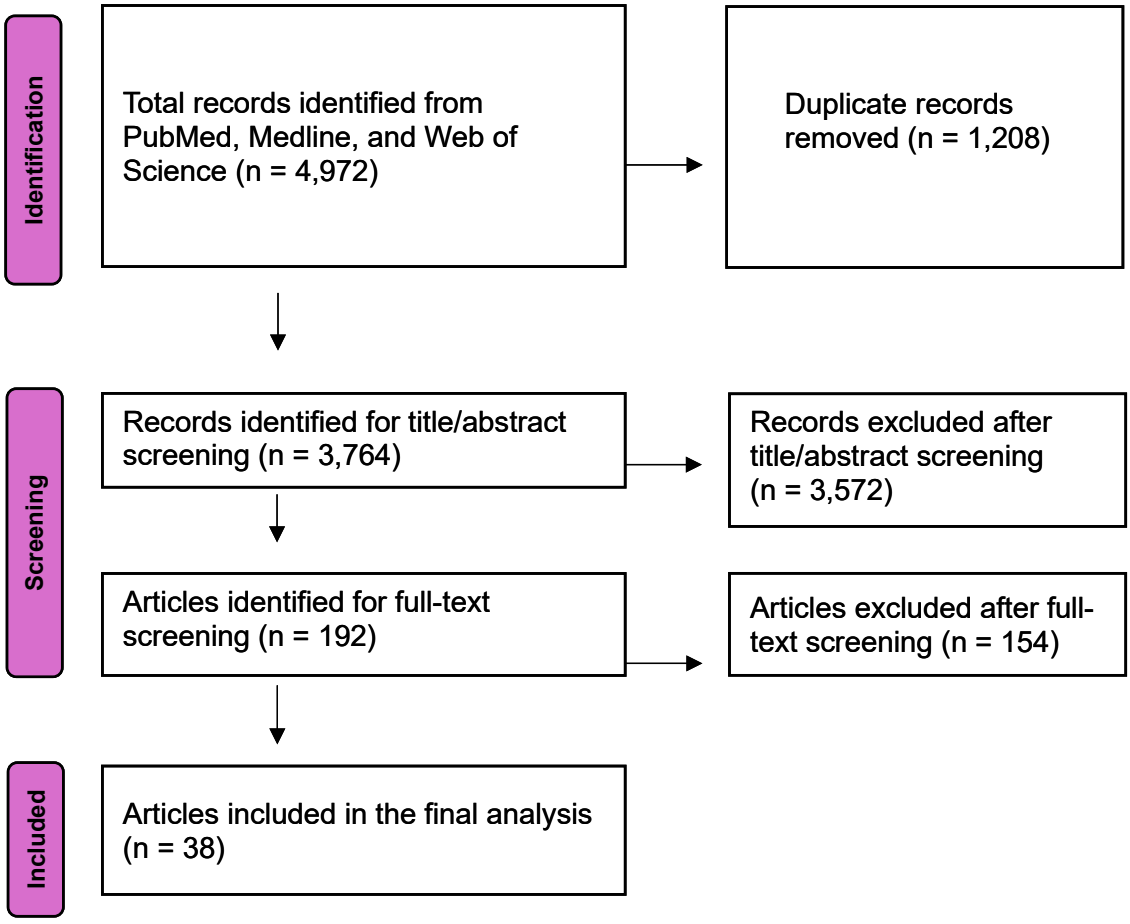
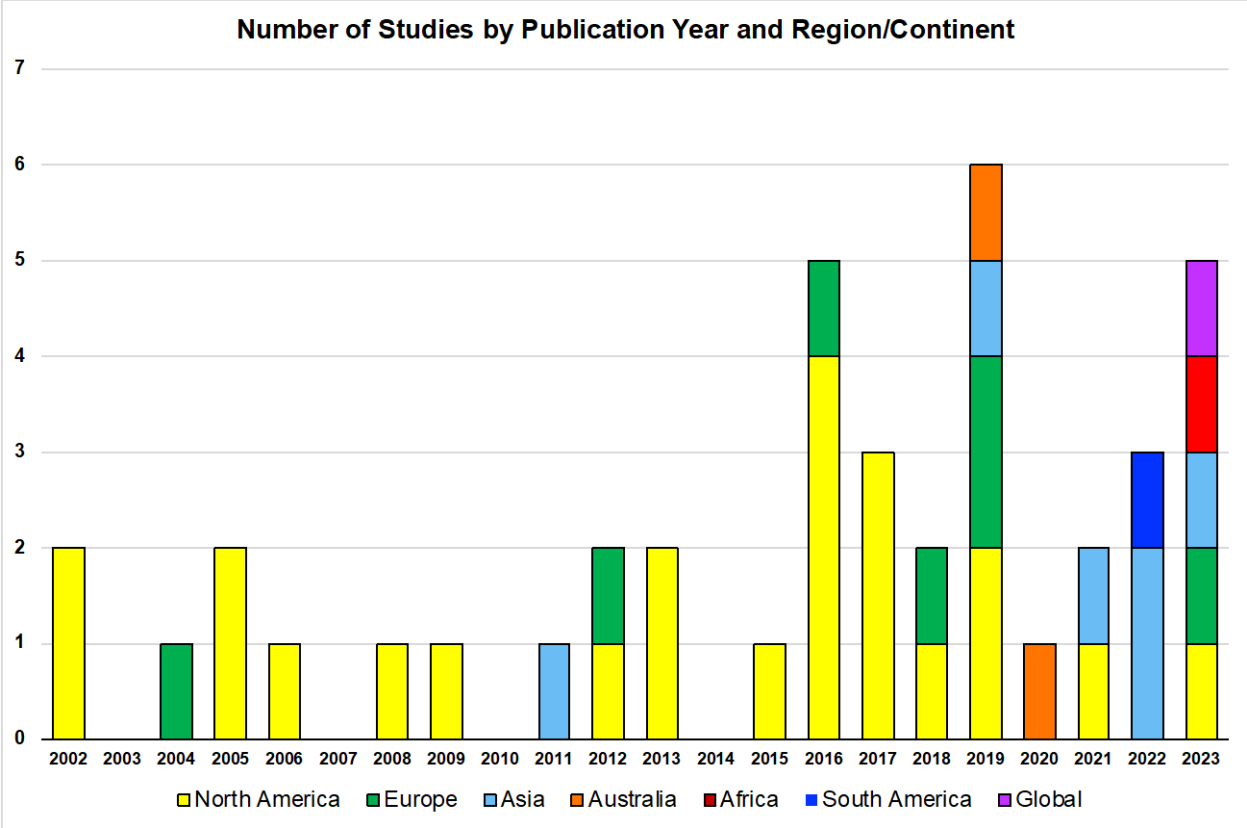


Figure S 2. Number of Studies by Publication Year and Country.



Supplementary Tables

Table S 1. Characteristics of included studies.

Author and Year	Age (Years)	Exposure	Study Period	Location	Study Size (n)
Villeneuve 2002	25-74	PM2.5	1974-1991	USA	8,111
Pope 2002	≥ 30	PM2.5	1982-1998	USA	500,000
Nafstad 2004	40-49	Nitrogen Oxides Other	1972-1998	Norway	16,209
Enstrom 2005	~65	PM2.5	1973-2002	USA	49,975
Jerrett 2005	≥ 30	PM2.5	1982-2000	USA	22,905
Laden 2006	25-74	PM2.5	1974-1998	USA	8,096
Zeger 2008	≥ 65	PM2.5	2000-2005	USA	13.2 million
Jerrett 2009	≥ 30	PM2.5 Ozone	1977-2000	USA	448,850
Cao 2011	≥ 40	Nitrogen Dioxide Other	1991-2000	China	70,947
Lepeule 2012	25-74	PM2.5	1974-2009	USA	8,096
Heinrich 2012	50-59	Nitrogen Dioxide Other	1985-2008	Germany	4,752
Jerrett 2013	≥ 30	PM2.5 Ozone Nitrogen Dioxide	1982-2000	USA	73,711
Correia 2013	N/A	PM2.5	2000-2007	USA	N/A[a]
Ostro 2015	≥30	PM2.5	2001-2007	USA	101,884
Turner 2016	≥ 30	PM2.5 Ozone Nitrogen Dioxide	1982-2004	USA	669,046
Kioumourtzoglou 2016	≥ 65	PM2.5	2000-2010	USA	35.3 million
Shi 2016	≥ 65	PM2.5	2003-2008	USA	268,050 deaths[a]
Keijzer 2016	N/A	PM2.5 Ozone Nitrogen Dioxide Other	2009-2013	Spain	44.6 million
Di 2017	≥ 65	PM2.5 Ozone	2000-2012	USA	60.9 million
Wang 2017	≥ 65	PM2.5	2000-2013	USA	13.1 million
Schwartz 2018	≥ 65	PM2.5	2000-2013	USA	17.0 million

Lefler 2019	18-84	PM2.5 Ozone Nitrogen Dioxide Other	1987-2015	USA	635,539
Hvidtfeldt 2019	50-64	PM2.5 Ozone Nitrogen Dioxide Other	1993-2015	Denmark	49,564
Dirgawati 2019	≥ 65	PM2.5 Nitrogen Dioxide Other	1996-2012	Australia	11627
Yitshak-Sade 2019	≥ 65	PM2.5	2000-2013	USA	15.4 million
Chen 2019	N/A	PM2.5	2010-2017	Taiwan	N/A[a]
Yu 2020	N/A	PM2.5	1998-2013	Australia	242,320 deaths[a]
Wang 2020	≥ 65	PM2.5	2000-2008	USA	53 million[a]
Qian 2021	≥ 65	Nitrogen Dioxide	2000-2016	USA	13.6 million
Anwar 2021	<5	PM2.5	2000-2017	Asia[b]	N/A[a]
Byun 2022	≥ 30	Ozone	2005-2015	South Korea	179,806
Liu 2022	16-110	PM2.5	2010-2017	China	30,524
Yu 2022	N/A	PM2.5	2010-2018	Brazil	N/A[a]
Baranyi 2023	~87	PM2.5	1939-2022	UK	2,734
Hao 2023	≥ 65	PM2.5 Other	2000-2017	USA	73.4 million
Xue 2023	<5	Ozone	2003-2019	55 LMICs	1.2 million
Shiferaw 2023	<5	PM2.5	2011-2016	Ethiopia	10,452
Wang 2023	≥45	Nitrogen Dioxide	2011-2018	China	15,440

[a]These were population-level ecologic studies that did not report the number of total included study participants.

[b]This study included 16 countries across Asia: Bangladesh, China, India, Indonesia, Iran, Malaysia, Mongolia, Myanmar, Nepal, Pakistan, Philippines, Russia, Sri Lanka, Thailand, Turkey, and Vietnam.

Abbreviations: LMICS =Low and Middle-Income Countries.

Table S 2. All-cause mortality and air pollution.

Author and Year	**Study Size (n)**	**Mean (SD) or *Median [IQR]* Exposure Level**	**All-Cause Mortality Risk (95% CI)[a]**	**Exposure Increment (increase)**	**Controlled confounders[b]**
Exposure: PM2.5					
Pope 2002	500,000	17.7 (3.7) µg/m³	RR 1.06 (1.02-1.11)	per 10 µg/m³	AGRSMH
Villeneuve 2002	8,111	11.0 (1.0)-28.5 (5.5) µg/m³	RR 1.17 (1.05-1.30)	per 10 µg/m³	AGSM
Enstrom 2005	49,975	23.4 µg/m³	RR 1.01 (0.98-1.05)	per 10 µg/m³	AGRSMH
Jerrett 2005	22,905	20 µg/m³	RR 1.17 (1.05-1.30)	per 10 µg/m³	AGRSMHUN
Laden 2006	8,096	10.2-29.0 µg/m³	RR 1.16 (1.07-1.26)	per 10 µg/m³	AGSM
Zeger 2008	13.2 million	*14.0 [3.0] µg/m³*	RR 1.07 (1.05-1.09)	per 10 µg/m³	AGRMN
Jerrett 2009	448,850	11.9 (2.5)-15.4 (3.2) µg/m³	RR 1.08 (1.05-1.11)	per 10 µg/m³	AGRSMHN
Lepeule 2012	8,096	15.9 µg/m³	RR 1.14 (1.07-1.22)	per 10 µg/m³	AGSMC
Correia 2013	N/A[c]	13.2 (3.4) µg/m³	0.35 years lost in life expectancy (p=0.033)	per 10 µg/m³	AGRMCUN[d]
Jerrett 2013	73,711	14.09 (3.52) µg/m³	RR 1.03 (1.00-1.06)	per IQR (5.30 µg/m³)	AGRSMHUN
Ostro 2015	101,884	17.9 µg/m^3	HR 1.01 (0.97-1.05)	per 10 µg/m³	ARSMHCN
Keijzer 2016	44.6 million	8.22 µg/m³	RR 1.04 (1.04-1.04)	per 2 µg/m³	N[d]
Kioumourtzoglou 2016	35.3 million	12 (1.6) µg/m	HR 1.19 (1.11-1.28)	per 10 µg/m³	AGRSCUN
Shi 2016	268,050 deaths[c]	8.12 (2.28) µg/m³	RR 1.08 (1.02-1.13)[e]	per 10 µg/m³	MN[d]
Turner 2016	669,046	12.6 (2.9) µg/m³	HR 1.04 (1.02-1.06)	per 10 µg/m³	AGRSMHN
Di 2017	60.9 million	11.0 µg/m³	HR 1.07 (1.07-1.08)	per 10 µg/m³	AGRSUN
Wang 2017	13.1 million	*10.7 [3.8] µg/m³*	HR 1.02 (1.02-1.02)	per 1 µg/m³	AGRSCUN
Schwartz 2018	17.0 million	10.3 µg/m³	0.89 years (0.88-0.91) lost in life expectancy	per 4.5 µg/m³	AGRSMCUN
Chen 2019	N/A[c]	26.37 (7.09) µg/m³	0.3 years (0.1-0.6) lost in life expectancy	per 10 µg/m³	SN[d]
Dirgawati 2019	11,627	5.1 (1.7) µg/m³	HR 1.07 (0.98-1.16)	per 5 µg/m³	ASM
Hvidtfeldt 2019	49,564	*18.0 µg/m³*	HR 1.13 (1.05-1.21)	per 5 µg/m³	AGSMHN

Hvidtfeldt 2019	49,564	*13.8 μg/m³*	HR 1.03 (1.01-1.05)	per IQR (1.36 μg/m³)	AGSMHN
Lefler 2019	635,539	10.67 (2.37) μg/m³	HR 1.05 (1.03-1.06)	per 10 μg/m³	AGRSMU
Yitshak-Sade 2019	15.4 million	6.5-14.5 μg/m³	RR 1.04 (1.03-1.05)	per IQR (3 μg/m³)	AGRSUN
Wang 2020	53 million	10.32 (3.15) μg/m³	RR 1.05 (1.04-1.06)	per 10 μg/m³	AGRSUN
Yu 2020	242,320 deaths[c]	*3.6 [2.0] μg/m³*	RR 1.02 (1.01-1.03)	per 1 μg/m³	N[d]
Anwar 2021	N/A[c]	44.18 (24.52) μg/m³	14.5% ($p<0.01$) decrease in mortality	per 1 μg/m³	UN[d]
Liu 2022	30,524	47.1 (19.5) μg/m³	HR 1.13 (1.06-1.20)	per IQR (26.7 μg/m³)	AGRSMHCU
Yu 2022	N/A[c]	7.7 μg/m³	RR 1.18 (1.15-1.21)[g]	per 10 μg/m³	UN[d]
Baranyi 2023	2,734	31.3 (32.6) μg/m³	HR 1.03 (1.01-1.04)	per 10 μg/m³	AGSU
Hao 2023 - Exposure I	73.4 million	10.03 (3.12) μg/m³	HR 1.01 (1.01-1.01)	per 1 μg/m³	AGRSMUN
Hao 2023 - Exposure II	73.4 million	9.30 (2.90) μg/m³	HR 1.01 (1.01-1.01)	per 1 μg/m³	AGRSMUN
Shiferaw 2023	10,452	20.1 (3.3) μg/m³	OR 2.29 (1.44-3.65)	per 10 μg/m³	AGS
Exposure: Ozone					
Jerrett 2009	448,850	57.7 ppb	RR 0.99 (0.98-1.00)	per 10 ppb	AGRSMHN
Jerrett 2013	73,711	50.35 (14.57) ppb	RR 1.00 (0.96-1.04)	per IQR (24.18 ppb)	AGRSMHUN
Keijzer 2016	44.6 million	80.39 μg/m³	RR 1.02 (1.02-1.02)	per 5 μg/m³	N[d]
Turner 2016	669,046	38.2 (4.0) ppb	HR 1.02 (1.01-1.04)	per 10 ppb	AGRSMHN
Di 2017	60.9 million	46.3 ppb	HR 1.01 (1.01-1.01)	per 10 ppb	AGRSUN
Hvidtfeldt 2019	49,564	*55.4 μg/m³*	HR 0.92 (0.89-0.96)	per 10 μg/m³	AGSMHN
Lefler 2019	635,539	47.45 (5.31) ppb	HR 1.00 (0.99-1.01)	per 10 ppb	AGRSMU
Byun 2022	179,806	21.9 (4.8) ppb	HR 1.18 (1.07-1.29)	per 10 ppb	AGSMHN
Xue 2023	1.2 million	51.69 (9.56) ppb	HR 1.06 (1.02-1.11)	per 10 ppb	G
Exposure: Nitrogen Dioxide/Oxide					
Nafstad 2004	16,209	*10.7 μg/m³*	RR 1.08 (1.06-1.11)	per 10 μg/m³	ASMHC
Cao 2011	70,947	50 μg/m³	HR 1.02 (1.00-1.03)	per 10 μg/m³	AGSMHC
Heinrich 2012	4,752	39 (11.85) μg/m³	HR 1.18 (1.07-1.30)	per 16 μg/m³	ASMC
Jerrett 2013	73,711	12.27 (2.92) ppb	RR 1.03 (1.01-1.06)	per IQR (4.12 ppb)	AGRSMHUN

Keijzer 2016	44.6 million	9.48 µg/m³	RR 1.00 (1.00-1.00)	per 5 µg/m³	N[d]
Turner 2016	669,046	11.6 (5.1) ppb	HR 1.01 (1.00-1.03)	per 10 µg/m³	AGRSMHN
Dirgawati 2019	11,627	13.4 (4.1) µg/m³	HR 1.06 (1.00-1.13)	per 10 µg/m³	ASM
Hvidtfeldt 2019	49,564	*25.0 µg/m³*	HR 1.07 (1.04-1.10)	per 10 µg/m³	AGSMHN
Lefler 2019	635,539	10.69 (5.73) ppb	HR 0.96 (0.93-0.98)	per 10 ppb	AGRSMU
Qian 2021	13.6 million	13.7 (5.9) ppb	HR 1.05 (1.04-1.05)	per 10 ppb	AGRSUN
Wang 2023	15,440	21.2 (6.3) µg/m³	HR 1.22 (1.10-1.35)	per 10 µg/m³	AGSMHCU

[a]All estimates of mortality risk were rounded to three significant figures.

[b]A=Age, G=Sex or Gender, R=Race or ethnicity, S=Individual socioeconomic status, M=Smoking status (individual) or smoking prevalence (area-level), H=Other health-related behaviors, C=comorbidities or chronic health conditions, U=urbanicity, N=neighborhood-level or area-level socioeconomic status.

[d]These are ecologic studies that only controlled for area-level confounders.

[c]These are population-based studies that did not report the number of total included study participants.

[e]This RR was calculated from the reported percent change in mortality.

Abbreviations: SD=Standard Deviation. IQR = Interquartile Range. CI=Confidence Interval. HR = Hazard ratio. RR = Relative risk. OR = Odds ratio.

Table S 3. All-cause mortality risk from air pollution stratified by age.

Author, Year	Youngest Age Group	All-cause Mortality Risk	Oldest Age Group	All-cause Mortality Risk	Exposure Increment (Increase)
PM2.5					
Baranyi 2023	11-55 years	HR 1.00 (0.96-1.04)	75-86 years	HR 1.03 (1.00-1.05)	per 10 µg/m³
Pope 2002[a]	30-60 years	RR 1.04 (1.00-1.09)	≥70 years	RR 1.05 (1.01-1.09)	per 10 µg/m³
Enstrom 2005	43-64 years	RR 1.03 (1.00-1.05)	65-99 years	RR 1.00 (0.98-1.02)	per 10 µg/m³
Yu 2020	<65 years	RR 1.06 (1.04-1.07)	≥65 years	RR 1.01 (1.01-1.02)	per 1 µg/m³
Zeger 2008	65-74 years	RR 1.11 (1.09-1.14)	≥85 years	RR 1.02 (1.00-1.04)	per 10 µg/m³
Kioumourtzoglou 2016	% >65 years (25^{th} pct)[b]	RR 1.11 (1.03-1.21)	% >65 years (75^{th} pct)[b]	RR 1.25 (1.16-1.35)	per 10 µg/m³
Di 2017	65-74 years	HR 1.15 (1.14-1.15)	≥85 years	HR 1.00 (0.99-1.00)	per 10 µg/m³
Wang 2017[a]	65-74 years	HR 1.04 (1.04-1.04)	≥85 years	HR 1.00 (1.00-1.00)	per 1 µg/m³
Ozone					
Di 2017	65-74 years	HR 1.01 (1.01-1.01)	≥85 years	HR 1.02 (1.01-1.02)	per 10 ppb
Nitrogen Oxides					
Nafstad 2004	40-45 years	RR 1 (Reference)	46-49 years	RR 1.65 (1.55-1.76)	per 10 µg/m³
Wang 2023	45-64 years	HR 1.01 (0.85-1.21)	≥65 years	HR 1.35 (1.19-1.53)	per 10 µg/m³
Qian 2021	65-80 years	HR 1.06 (1.03-1.08)	>80 years	HR 1.03 (1.01-1.05)	per 10 ppb

[a]Numeric data for these studies were extracted from the published figures via DigitizeIt Software.[22,23]

[b]Neighborhood level proportion of residents of >65 years at the 25^{th} percentile versus 75^{th} percentile.

Table S 4. All-cause mortality risk from air pollution stratified by sex.

Author, Year	All-cause mortality risk		Exposure increment (increase)
	Male	Female	
PM2.5			
Pope 2002	RR 1.06 (1.02-1.10)	RR 1.02 (0.98-1.06)	per 10 μg/m³
Enstrom 2005	RR 0.99 (0.97-1.02)	RR 1.03 (1.01-1.05)	per 10 μg/m³
Zeger 2008	RR 1.07 (1.05-1.09)	RR 1.09 (1.06-1.11)	per 10 μg/m³
Correia 2013	0.08 years lost in life expectancy (SE=0.20)	0.59 years lost in life expectancy (SE=0.17)	per 10 μg/m³
Wang 2017	HR 1.03 (1.02-1.03)	HR 1.02 (1.01-1.02)	per 1 μg/m³
Di 2017	HR 1.09 (1.08-1.09)	HR 1.06 (1.06-1.06)	per 10 μg/m³
Schwartz 2018	1.17 years (1.14-1.19) lost in life expectancy	0.74 years (0.72-0.77) lost in life expectancy	per 4.5 μg/m³
Hvidtfeldt 2019	HR 1.20 (0.91-1.57)	HR 1.05 (0.97-1.14)	per 5 μg/m³
Yu 2020	RR 1.01 (1.01-1.02)	RR 1.03 (1.02-1.04)	per 1 μg/m³
Yu 2022[a]	N/A	N/A	N/A
Hao 2023	HR 1.04 (1.04-1.04)	HR 1.04 (1.04-1.04)	per 3.68 μg/m³
Baranyi 2023	HR 1.04 (1.01-1.07)	HR 1.02 (0.99-1.05)	per 10 μg/m³
Ozone			
Di 2017	HR 1.01 (1.01-1.01)	HR 1.01 (1.01-1.01)	per 10 ppb
Xue 2023	No difference (p=0.502)		per 10 ppb
Nitrogen Oxides			
Hvidtfeldt 2019	HR 1.10 (1.06-1.14)	HR 1.03 (0.99-1.07)	per 10 μg/m³
Qian 2021	HR 1.01 (1.00-1.02)	HR 1.08 (1.06-1.09)	per 10 ppb
Wang 2023	HR 1.26 (1.10-1.44)	HR 1.17 (1.00-1.36)	per 10 μg/m³

[a]This study reported that avoidable loss of life expectancy from PM2.5 exposure was higher in males than in females but did not quantify risk values.

Table S 5. All-cause mortality risk from air pollution stratified by race.

Author, Year	All-cause mortality risk					Exposure increment (increase)
	White	Black	Hispanic	Asian	Native American	
PM2.5						
Kioumourtzoglou 2016	HR 1.11 (1.01-1.22)[a]	HR 1.29 (1.19-1.39)[a]	N/A	HR 1.19 (1.11-1.28)[a]	N/A	per 10 μg/m³
Wang 2017	HR 1.02 (1.02-1.02)	HR 1.03 (1.03-1.03)	Others: 1.06 (1.06-1.07)			per 1 μg/m³
Di 2017	HR 1.06 (1.06-1.07)	HR 1.21 (1.20-1.22)	HR 1.12 (1.10-1.13)	HR 1.10 (1.08-1.12)	HR 1.10 (1.06-1.14)	per 10 μg/m³
Yitshak-Sade 2019	RR 1.03 (1.03-1.04)	RR 1.10 (1.09-1.12)	N/A	N/A	N/A	per 3 μg/m³
Ozone						
Di 2017	HR 1.01(1.01-1.01)	HR 1.01 (1.01-1.01)	HR 0.98 (0.97-0.98)	HR 0.98 (0.97-0.99)	HR 0.96 (0.94-0.98)	per 10 ppb
Nitrogen Oxides						
Qian 2021	HR 1.06 (1.05-1.07)	HR 1.00 (0.98-1.02)	Other: HR 0.98 (0.94-1.02)			per 10 ppb

[a]Risk values at the 75th percentiles for the proportion of White, Black, and Asian residents, respectively.

Table S 6. All-cause mortality risk from air pollution stratified by education level.

Author, Year	All-cause mortality risk			Exposure increment (Increase)
	Low Education Level	Moderate Education Level	High Education Level	
PM2.5				
Pope 2002[a]	HR 1.09 (1.03-1.14)	HR 1.05 (1.01-1.09)	HR 1.00 (0.97-1.04)	per 10 μg/m^3
Enstrom 2005[b]	RR 1.02 (0.99-1.05)	RR 1.01 (0.97-1.04)	RR 1.01 (0.98-1.03)	per 10 μg/m^3
Kioumourtzoglou 2016[c]	HR 1.26 (1.16-1.38)	N/A	HR 1.15 (1.06-1.24)	per 10 μg/m^3
Kioumourtzoglou 2016[d]	HR 1.22 (1.13-1.31)	N/A	HR 1.14 (1.05-1.24)	per 10 μg/m^3
Wang 2017[e]	HR 1.023 (1.021-1.024)	N/A	HR 1.019 (1.018-1.020)	per 1 μg/m^3
Hvidtfeldt 2019[f]	HR 1.13 (1.04-1.23)	HR 1.14 (1.06-1.23)	HR 1.07 (0.95-1.28)	per 5 μg/m³
Nitrogen Oxides				
Nafstad 2004[g]	HR 1 (Reference)	HR 0.78 (0.73-0.84)	HR 0.66 (0.60-0.74)	per 10 ppb
Hvidtfeldt 2019[f]	HR 1.09 (1.04-1.14)	HR 1.06 (1.02-1.10)	HR 1.06 (0.99-1.13)	per 10 μg/m³
Wang 2023[h]	HR 1.27 (1.13-1.42)	HR 1.10 (0.88-1.37)	N/A	per 10 μg/m³

[a]Low, moderate, and high education levels were defined as <high school, high school, and >high school, respectively.

[b]Low, moderate, and high education levels were defined as <12 years of education, 12 years of education, and >12 years of education, respectively.

[c]These values refer to HRs at neighborhood level percentiles of residents with a college degree (20th versus 80th percentile) tabulated as low and high levels of education, respectively.

[d]These values refer to HRs at neighborhood level percentiles of residents with no high school degrees (80th versus 20th percentile) tabulated as low and high levels of education, respectively.

[e]This study reported effect modification by neighborhood level percentiles of less educated (20th versus 80th percentile) tabulated as high and low education levels respectively. The numeric HRs were extracted from published figures via DigitizeIt software.[22,23]

[f]Low, moderate, and high education levels were defined as 8-11 years of basic schooling, 11-14 upper secondary/vocational training, and 15+ years of schooling, respectively.

[g]Low, moderate, and high education levels were defined as <10, 10-12, and >12 years of education, respectively.

[h]Low and moderate education levels were defined as 0-6 years and >7 years of education, respectively.

Table S 7. All-cause mortality risk from air pollution stratified by SES metrics.

Author, Year	All-cause mortality risk		Exposure Increment (Increase)
	Low SES	High SES	
PM2.5			
Zeger 2008[a]	HR 6.9 (4.1-9.8)	HR 8.3 (5.9-10.8)	per 10 μg/m³
Kioumourtzoglou 2016[b]	HR 1.28 (1.18-1.40)	HR 1.14 (1.06-1.23)	per 10 μg/m³
Kioumourtzoglou 2016[c]	HR 1.23 (1.14-1.33)	HR 1.13 (1.04-1.23)	per 10 μg/m³
Kioumourtzoglou 2016[d]	HR 1.23 (1.14-1.33)	HR 1.13 (1.04-1.22)	per 10 μg/m³
Wang 2017[e]	HR 1.022 (1.021-1.023)[f]	HR 1.018 (1.017-1.019)[f]	per 1 μg/m³
Wang 2017[g]	HR 1.021 (1.020-1.023)[f]	HR 1.020 (1.019-1.021)[f]	per 1 μg/m³
Wang 2017[h]	HR 1.020 (1.018-1.021)[f]	HR 1.021 (1.020-1.022)[f]	per 1 μg/m³
Nitrogen Oxides			
Nafstad 2004[i]	RR 1.08 (1.00-1.17)	RR 1.00 (reference)	per 10 ppb
Qian 2021[j]	HR 1.05 (1.04-1.06)	HR 1.05 (1.04-1.06)	per 10 ppb

[a]Socioeconomic status (SES) was defined using five ZIP code-level factors (percentage with a high school diploma, percentage with a higher education degree, percentage of households above the poverty level, median household income, and percentage employed). Zip codes with higher than national median across majority of factors were categorized as high SES.

[b]25th versus 75th percentiles in median household income.

[c]25th versus 75th percentiles in percentage in poverty.

[d]25th versus 75th percentiles in percentage of city families in poverty.

[e]20th versus 80th percentile in percentage below poverty level.

[f]The numeric HRs were extracted from published figures via DigitizeIt software.[22,23]

[g]80th versus 20th percentile in median income

[h]80th versus 20th percentile in home value (80th vs. 20th percentile).

[i]Blue collar occupation was interpreted as low SES and white collar as high SES.

[j]Low SES was defined as below the median percentage below the poverty level.

Table S 8. All-cause mortality risk from air pollution stratified by Medicaid-Medicare dual eligibility as a proxy for socioeconomic status.

Author, Year	All-cause mortality risk in the Medicare Population		Exposure increment (Increase)
	Medicaid Eligible	Medicaid Non-Eligible	
PM2.5			
Di 2017	HR 1.08 (1.08-1.09)	HR 1.08 (1.07-1.08)	per 10 μg/m³
Wang 2017	HR 1.02 (1.02-1.03)	HR 1.02 (1.02-1.02)	per 1 μg/m³
Yitshak-Sade 2019	RR 1.06 (1.04-1.08)	RR 1.04 (1.03-1.04)	per 3 μg/m³
Ozone			
Di 2017	HR 1.02 (1.02-1.02)	HR 1.01 (1.00-1.01)	per 10 ppb
Nitrogen Oxides			
Qian 2021	HR 1.03 (1.01-1.05)	HR 1.05 (1.04-1.07)	per 10 ppb

Table S 9. All-cause mortality from air pollution stratified by comorbidities.

Author, Year	Comorbidity Studied	All-cause mortality risk with comorbidity	All-cause mortality risk without comorbidity	Exposure Increment (increase)
PM2.5				
Enstrom 2005	Cancer, heart disease, or stroke	0.99 (0.96-1.03)	1.01 (0.99-1.03)	per 10 µg/m³
Lepeule 2012	Hypertension COPD Diabetes	1.17 (1.03-1.32) 1.09 (0.95-1.26) 1.04 (0.85-1.27)	N/A	per 10 µg/m³
Wang 2017[a]	CHF Admission MI Admission COPD Admission Diabetes Admission	1.03 (1.03-1.03) 1.05 (1.05-1.06) 1.05 (1.05-1.05) 1.05 (1.05-1.06)	1.02 (1.02-1.02) 1.02 (1.02-1.02) 1.02 (1.02-1.02) 1.02 (1.02-1.02)	per 1 µg/m³
Nitrogen Oxides				
Nafstad 2004	CVD or Diabetes	2.69 (2.44-2.95)	1 (reference)	per 10 µg/m³
Wang 2023	CVD Respiratory Diseases	1.26 (1.09-1.46) 1.37 (1.06-1.76)	1.17 (1.01-1.34) 1.19 (1.06-1.33)	per 10 µg/m³

[a] Presence of comorbidities were defined as previous hospitalizations due to a medical condition.

Abbreviations: COPD=chronic obstructive pulmonary disease; CHF=Congestive heart failure; MI=Myocardial infarction; CVD=Cardiovascular Disease

Table S 10. All-cause mortality from air pollution stratified by urbanicity.

Author, Year	Urban	Rural	Exposure Increment (increase)
PM2.5			
Correia 2013[a]			
% urban residences[a]	0.95 years LLE (p<0.01)	-0.16 years LLE (p=0.299)	per 10 μg/m³
Population density[b]	0.72 years LLE (p<0.01)	-0.31 years LLE (p=0.165)	per 10 μg/m³
Shi 2016[c]	RR 1.13 (1.06-1.20)	RR 1.03 (0.97-1.10)	per 10 μg/m³
Keijzer 2016[d]	RR 1.02 (1.02-1.02)	RR 1.10 (1.09-1.10)	per 2 μg/m³
Kioumourtzoglou 2016	HR 1.18 (1.09-1.28)	HR 1.20 (1.11-1.29)	per 10 μg/m³
Wang 2017[e]	HR 1.021 (1.020-1.022)	HR 1.006 (1.004-1.008)	per 1 μg/m³
Di 2017[f]	HR 1.08 (1.07-1.09)	HR 1.07 (1.06-1.07)	per 10 μg/m³
Yu 2020[g]	RR 1.06 (1.04-1.07)	RR 1.02 (1.01-1.03)	per 1 μg/m³
Yu 2022[h]	1.77 years (1.51-2.03) LLE	1.43 years (1.22-1.62) LLE	Above 2.9 μg/m³
Ozone			
Keijzer 2016[c]	RR 1.02 (1.01-1.02)	RR 1.03 (1.02-1.03)	per 5 μg/m³
Di 2017[f]	HR 0.98 (0.98-0.99)	HR 1.03 (1.03-1.03)	per 10 ppb
Nitrogen Oxides			
Heinrich 2012[i]	HR 1.42 (1.12-1.79)	HR 1.00 (reference)	per 16 μg/m³
Keijzer 2016[c]	RR 1.00 (1.00-1.00)	RR 1.05 (1.04-1.05)	per 5 μg/m³
Qian 2021[j]	HR 1.06 (1.04-1.08)	HR 1.01 (0.99-1.03)	per 10 ppb
Wang 2023	HR 1.11 (0.94-1.30)	HR 1.27 (1.12-1.45)	per 10 μg/m³

[a]counties with >90% residences in urban areas defined as urban and other counties defined as rural.

[b]population density >200 people per square mile defined as urban and population density <200 people per square mile defined as rural.
[c]Urban defined as total population within zip code below median
[d]Urban defined as areas with >10,000 inhabitants
[e]Rural areas were defined as areas with population density below the first tertile of the population density (51 per square mile).
[f]Urbanicity was stratified into low, medium-low, medium-high, and high population density. Numeric cutoffs were not reported.
[g]Brisbane was defined as an urban area and the rest of the state (Queensland) as rural.
[h]Municipalities were defined by Brazilian Institute of Geography and Statistics as urban versus intermediate remote areas (categorized in this table as rural).
[i]<=50m from home to a major road was considered urban and >50m from home to a major road as rural.
[j]Urbanicity was based on quartiles of population density, with urban areas defined as those with high population density (fourth quartile) and rural defined as low population density (first quartile).
Abbreviations: LLE = lost life expectancy.

Appendix.

A.1. Search Strategy for the Systematic Review.

Topic: Air pollution & life expectancy
Search date: 10/19/2023
Limits: 2000-present; Embase limit to articles/articles in press

PubMed: Results = 1195
("air pollution" OR "Air Pollution"[Mesh] OR "air pollutants" OR "Air Pollutants"[Mesh] OR "particulate matter" OR "Particulate Matter"[Mesh] OR “ozone” OR “Ozone”[Mesh] OR “nitrogen dioxide” OR "Nitrogen Dioxide"[Mesh] OR “pollutant mixtures” OR “chemical mixtures” OR “hazardous substances” OR "Hazardous Substances"[Mesh] OR “inhalation exposure” OR “inhalation exposure” [Mesh]) AND (“life expectancy” OR "Life Expectancy"[MeSH] OR “cause of death” OR "Cause of Death"[Mesh] OR “mortality” OR “Mortality”[Mesh] OR “life table” OR “Life Tables”[Mesh]) AND (“chronic disease” OR "Chronic Disease"[Mesh] OR “asthma” OR "Asthma"[Mesh] OR “COPD” OR “pulmonary disease, chronic obstructive” [Mesh] OR “chronic obstructive pulmonary disease” OR “cardiovascular disease” OR “CVD” OR "Cardiovascular Diseases"[Mesh] OR “diabetes” OR "Diabetes Mellitus"[Mesh] OR “vulnerable populations” OR "Vulnerable Populations"[Mesh] OR “socioeconomic status” OR "Low Socioeconomic Status"[Mesh] OR “social class” OR "Social Class"[Mesh] OR “social determinants” OR "Social Determinants of Health"[Mesh] OR “Health Equity”[Mesh] or “health equity” OR “Socioeconomic Factors”[Mesh] OR “socioeconomic factors” OR “racial groups”[Mesh] OR “racial groups” OR "race factors" [Mesh] OR "race" OR “ethnicity”[Mesh] OR “ethnicity” OR “social vulnerability”[Mesh] OR “social vulnerability” OR “Demography”[Mesh] OR “demographics” OR “demographic data” OR “Age distribution”[Mesh] OR “age” OR “Sex distribution”[Mesh] OR “sex” OR “gender”) AND (“statistical models” OR "Models, Statistical"[Mesh] OR “binomial model” OR “binomial models” OR “binomial distribution” OR "Binomial Distribution"[Mesh] OR “linear model” OR “linear models” OR "Linear Models"[Mesh] OR “poisson model” OR “poisson models” OR “poisson distribution” OR "Poisson Distribution"[Mesh] OR “logistic regression” OR “logistic model” OR “logistic models” OR “Logistic Models”[Mesh] OR “machine learning” OR “Machine Learning”[Mesh] OR “random forest” OR “random forest” [Mesh])

Web of Science: 1646 results
("air pollution" OR "air pollutants" OR "particulate matter" OR “ozone” OR “nitrogen dioxide” OR “pollutant mixtures” OR “chemical mixtures” OR “hazardous substances” OR “inhalation exposure”) AND (“life expectancy” OR “cause of death” OR “mortality” OR “life tables”) AND (“chronic disease” OR "chronic diseases" OR “asthma” OR “COPD” OR “chronic obstructive

pulmonary disease” OR “chronic obstructive pulmonary diseases" OR “cardiovascular disease” OR "cardiovascular diseases" OR “CVD” OR “diabetes” OR "Diabetes Mellitus" OR “vulnerable populations” OR “socioeconomic status” OR “social class” OR “social determinants” OR "Social Determinants of Health" OR “health equity” OR “socioeconomic factors” OR “racial groups” OR "race" OR “ethnicity” OR “social vulnerability” OR “demographics” OR "demographic" OR “demographic data” OR “Age distribution” OR “age” OR “Sex distribution” OR “sex” OR “gender”) AND (“statistical models” OR "statistical model" OR “binomial models” OR "binomial model" OR “binomial distribution” OR “linear model” OR “linear models” OR “linear distribution” OR “poisson” OR “logistic regression” OR “logistic models” OR “machine learning” OR “random forest”)

EMBASE: 2131
('air pollution'/exp OR 'air pollution' OR 'air pollutants'/exp OR 'air pollutants' OR 'particulate matter'/exp OR 'particulate matter' OR 'ozone'/exp OR 'ozone' OR 'nitrogen dioxide'/exp OR 'nitrogen dioxide' OR 'pollutant mixtures' OR 'chemical mixtures' OR 'hazardous substances'/exp OR 'hazardous substances' OR 'inhalation exposure'/exp OR 'inhalation exposure') AND ('life expectancy'/exp OR 'life expectancy' OR 'cause of death'/exp OR 'cause of death' OR 'mortality'/exp OR 'mortality' OR 'life tables'/exp OR 'life tables') AND ('chronic disease'/exp OR 'chronic disease' OR 'asthma'/exp OR 'asthma' OR 'copd'/exp OR 'copd' OR 'chronic obstructive pulmonary disease'/exp OR 'chronic obstructive pulmonary disease' OR 'cardiovascular disease'/exp OR 'cardiovascular disease' OR 'cvd' OR 'diabetes'/exp OR 'diabetes' OR 'diabetes mellitus'/exp OR 'diabetes mellitus' OR 'vulnerable populations'/exp OR 'vulnerable populations' OR 'socioeconomic status'/exp OR 'socioeconomic status' OR 'social class'/exp OR 'social class' OR 'social determinants'/exp OR 'social determinants' OR 'social determinants of health'/exp OR 'social determinants of health' OR 'health equity'/exp OR 'health equity' OR 'socioeconomic factors'/exp OR 'socioeconomic factors' OR 'racial groups'/exp OR 'racial groups' OR 'ethnicity'/exp OR 'ethnicity' OR 'social vulnerability'/exp OR 'social vulnerability' OR 'demographics'/exp OR 'demographics' OR 'demographic data'/exp OR 'demographic data' OR 'age distribution'/exp OR 'age distribution' OR 'age'/exp OR 'age' OR 'sex distribution'/exp OR 'sex distribution' OR 'sex'/exp OR 'sex' OR 'gender'/exp OR 'gender') AND ('statistical models'/exp OR 'statistical models' OR 'binomial models' OR 'binomial distribution'/exp OR 'binomial distribution' OR 'linear model'/exp OR 'linear model' OR 'linear models'/exp OR 'linear models' OR 'linear distribution' OR 'poisson' OR 'logistic regression'/exp OR 'logistic regression' OR 'logistic models'/exp OR 'logistic models' OR 'machine learning'/exp OR 'machine learning' OR 'random forest'/exp OR 'random forest') AND [2000-2023]/py

A.2. PRISMA Checklist.

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	Pg. 1
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	Pg. 2
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	Pg. 3
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	Pg. 4
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	Pg. 5
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	Pg. 5-6
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	Appendix A2
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	Pg. 5-6
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently,	Pg. 5-6

Section and Topic	Item #	Checklist item	Location where item is reported
		any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	Pg. 6-7
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	Pg. 6-7
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	Pg. 6
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	Pg. 6-7
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	Pg. 6
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	Pg.6-7
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	Pg. 7
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to	Pg. 6-7

Section and Topic	Item #	Checklist item	Location where item is reported
		identify the presence and extent of statistical heterogeneity, and software package(s) used.	
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	Pg. 7
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	Pg. 7
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	Pg. 6
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	Pg. 7
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	Figure 1; Pg. 8
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	Appendix A4
Study characteristics	17	Cite each included study and present its characteristics.	Table 1
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	Appendix A3
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	Table 2-10
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among	Pg. 8-9

Section and Topic	Item #	Checklist item	Location where item is reported
		contributing studies.	
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	Pg. 8-11
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	Pg. 8-11
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	N/A
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	See 14
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	See 15
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	Pg. 12-14
	23b	Discuss any limitations of the evidence included in the review.	Pg. 14-15
	23c	Discuss any limitations of the review processes used.	Pg. 15
	23d	Discuss implications of the results for practice, policy, and future research.	Pg. 15
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	This review was not registered.
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	An internal protocol (not

Section and Topic	Item #	Checklist item	Location where item is reported
			public) was prepared.
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	N/A
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	Pg. 32
Competing interests	26	Declare any competing interests of review authors.	Pg. 32
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	Pg. 32

From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021;372:n71. doi: 10.1136/bmj.n71

A.3. Risk Of Bias In Non-randomized Studies - of Exposure (ROBINS-E) scores for included studies.

Study	D1	D2	D3	D4	D5	D6	D7	Overall
Villeneuve 2002	Some concerns	Low	Some concerns	Low	Low	Low	Low	Low
Jerrett 2005	Low	Low	Some concerns	Low	Low	Low	Low	Low
Tao Xue 2023	Low	Low	Low	Low	Low	Low	Low	Low
Zeger 2008	Some concerns	Some concerns	Low	Low	Low	Low	Low	Low
Laden 2006	Some concerns	Low	Some concerns	Low	Low	Low	Low	Low
Jie Cao 2011	Some concerns	Some concerns	Some concerns	Low	Low	Low	Low	Some concerns
Ostro 2015	Low	Some concerns	Low	Low	Low	Low	Low	Low
Lapeule 2012	Some concerns	Low	Some concerns	Low	Low	Low	Low	Low
Yan Wang 2017	Low	Low	Some concerns	Low	Low	Low	Low	Low
Nafstad 2004	Low	Low	Some concerns	Low	Low	Low	Low	Low
Dirgawati 2019	Low	Low	Some concerns	Low	Low	Low	Low	Low
Shiferaw 2023	Some concerns	Low	Low	Low	Low	Low	Low	Low
Heinrich 2012	Some concerns	Low	Some concerns	Low	Low	Low	Low	Low
Yu 2020	Some concerns	Some concerns	Low	Low	Low	Low	Low	Low
Correia 2013	Low	Low	Low	Low	Low	Low	Low	Low
Hvidtfeldt 2019	Low	Low	Low	Low	Low	Low	Low	Low
Enstrom 2005	Low	Some concerns	Low	Low	Low	Low	Low	Low
Lefler 2019	Low	Low	Low	Low	Low	Low	Low	Low

Kioumourtzoglou 2016	Low	Low	Low	Low	Low	Low	Low	Low
Hvidtfeldt 2019	Low	Low	Low	Low	Low	Low	Low	Low
Schwartz 2018	Some concerns	Low	Low	Low	Low	Low	Low	Low
Jerrett 2009	Low	Some concerns	Low	Low	Low	Low	Low	Low
Jerrett 2013	Some concerns	Some concerns	Some concerns	Low	Low	Low	Low	Some concerns
Turner 2016	Low	Some concerns	Low	Low	Low	Low	Low	Low
Arden Pope III 2002	Some concerns	Some concerns	Low	Low	Low	Low	Low	Low
Qian Di 2017	Some concerns	Low	Low	Low	Low	Low	Low	Low
de Keijzer 2016	Some concerns	Some concerns	Low	Low	Low	Low	Low	Low
Chen 2019	Some concerns	Low	Low	Low	Low	Low	Low	Low
Yitshak-Sade 2019	Low	Low	Low	Low	Low	Low	Low	Low
Anwar 2021	Some concerns	Some concerns	Some concerns	Low	Low	Low	Low	Some concerns
Qian 2021	Low	Low	Low	Low	Low	Low	Low	Low
Liu 2022	Low	Low	Low	Low	Low	Low	Low	Low
Pei Yu 2022	Some concerns	Low	Some concerns	Low	Low	Low	Low	Some concerns
Yaqi Wang 2023	Low	Low	Low	Low	Low	Some concerns	Low	Low
Byun 2022	Low	Low	Low	Low	Low	Low	Low	Low
Hao 2023	Low	Low	Low	Low	Low	Low	Low	Low
Baranyi 2023	Some concerns	Some concerns	Some concerns	Low	Low	Low	Low	Some concerns

A.4. Examples of Studies Excluded at Full-Text Screening.

This table provides representative examples of excluded studies; a full list of all studies excluded after full-text screening is available upon request.

Study (Author, Year)	Reason for Exclusion
Janes 2007	No lag time between exposure and outcome; not long-term
Kuzma 2020	Short-term (time-series) exposure study; not long-term
Liang 2019	Reported non-accidental mortality; not all-cause mortality
Crouse 2016	Reported non-accidental mortality; not all-cause mortality
Garcia 2016	Reported non-accidental mortality; not all-cause mortality
Kloog 2013	Short term (time-series) exposure study; not long-term
Sanyal 2018	Reported natural mortality; not all-cause mortality

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