BAY AREA AIR QUALITY MANAGEMENT DISTRICT

BOARD OF DIRECTORS
ADVISORY COUNCIL

COMMITTEE MEMBERS

Dr. Linda Rudolph (Co-Chair), MD, Center for Climate Change and Health
Dr. Gina Solomon (Co-Chair), MD, University of California San Francisco
Dr. Danny Cullenward, PhD, JD, CarbonPlan
Dr. Adrienne L. Hollis, PhD, JD, Hollis Environmental Consulting, LLC
Dr. Michael Kleinman, PhD, University of California Irvine
Dr. Pallavi Phartiyal, PhD, Rainforest Action Network
Garima Raheja, PhD candidate, Columbia University
David Haubert, Air District Board of Directors Liasion

**THIS MEETING WILL BE CONDUCTED UNDER PROCEDURES AUTHORIZED BY ASSEMBLY BILL 361**

- **THE PUBLIC MAY OBSERVE THIS MEETING THROUGH THE WEBCAST BY CLICKING THE LINK AVAILABLE ON THE AIR DISTRICT'S AGENDA WEBPAGE AT**

**https://www.baaqmd.gov/about-the-air-district/advisory-council/agendasreports**

- **THE PUBLIC MAY PARTICIPATE REMOTELY VIA ZOOM AT THE FOLLOWING LINK OR BY PHONE**

**https://bayareametro.zoom.us/j/82716207050**

**(669) 900-6833 or (408) 638-0968**

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- **THOSE PARTICIPATING BY PHONE WHO WOULD LIKE TO MAKE A COMMENT CAN USE THE "RAISE HAND" FEATURE BY DIALING "*9". IN ORDER TO RECEIVE THE FULL ZOOM EXPERIENCE, PLEASE MAKE SURE YOUR APPLICATION IS UP TO DATE**

# ADVISORY COUNCIL MEETING
# AGENDA

**MONDAY, FEBRUARY 14, 2022**
**8:30 AM**

1. **Call to Order - Roll Call**

2. **Public Meeting Procedure**

*The Council Chair shall call the meeting to order and the Clerk of the Boards shall take roll of the Council members.*

***Public Comment on Agenda Items:*** *The public may comment on each item on the agenda as the item is taken up. Members of the public who wish to speak on matters on the agenda for the meeting, will have three minutes each to address the Council. No speaker who has already spoken on that item will be entitled to speak to that item again.*

**REGULAR AGENDA (Items 3 - 6)**

3. Approval of the Minutes of December 13, 2021

*The Council will consider approving the draft minutes of the Advisory Council meeting of December 13, 2021.*

4. Building Appliance Rules: Benefits to Outdoor Air Quality and Health

*The Advisory Council will receive an overview of the results of a modeling-based evaluation of outdoor air quality and health benefits of proposed amendments to rules on natural gas-fired space heaters, water heaters, and boilers.*

5. Regulatory Toolbox and PM Health Impacts Methodology

*The Advisory Council will receive a presentation on the Air District's regulatory tools and how they relate to the development of a PM2.5 local risk methodology.*

6. 2022 Advisory Council Work Plan Discussion

*The Council will review and discuss the 2022 Advisory Council work plan.*

**OTHER BUSINESS**

7. Report of the Executive Officer/APCO

8. Public Comment on Non-Agenda Matters

*Pursuant to Government Code Section 54954.3*
*Members of the public who wish to speak on matters not on the agenda for the meeting, will have three minutes each to address the Council.*

9. Council Member Comments / Other Business

*Council members may make a brief announcement, provide a reference to staff about factual information, or ask questions about subsequent meetings.*

10. Time and Place of Next Meeting

*At the Call of the Chair.*

11. Adjournment

*The Council meeting shall be adjourned by the Chair.*

**CONTACT:**
**MANAGER, EXECUTIVE OPERATIONS** **(415) 749-4941**
**375 BEALE STREET, SAN FRANCISCO, CA 94105** **FAX: (415) 928-8560**
**vjohnson@baaqmd.gov** **BAAQMD homepage:**
**www.baaqmd.gov**

- Any writing relating to an open session item on this Agenda that is distributed to all, or a majority of all, members of the body to which this Agenda relates shall be made available at the Air District's offices at 375 Beale Street, Suite 600, San Francisco, CA 94105, at the time such writing is made available to all, or a majority of all, members of that body.

**Accessibility and Non-Discrimination Policy**

The Bay Area Air Quality Management District (Air District) does not discriminate on the basis of race, national origin, ethnic group identification, ancestry, religion, age, sex, sexual orientation, gender identity, gender expression, color, genetic information, medical condition, or mental or physical disability, or any other attribute or belief protected by law.

It is the Air District's policy to provide fair and equal access to the benefits of a program or activity administered by Air District. The Air District will not tolerate discrimination against any person(s) seeking to participate in, or receive the benefits of, any program or activity offered or conducted by the Air District. Members of the public who believe they or others were unlawfully denied full and equal access to an Air District program or activity may file a discrimination complaint under this policy. This non-discrimination policy also applies to other people or entities affiliated with Air District, including contractors or grantees that the Air District utilizes to provide benefits and services to members of the public.

Auxiliary aids and services including, for example, qualified interpreters and/or listening devices, to individuals who are deaf or hard of hearing, and to other individuals as necessary to ensure effective communication or an equal opportunity to participate fully in the benefits, activities, programs and services will be provided by the Air District in a timely manner and in such a way as to protect the privacy and independence of the individual. Please contact the Non-Discrimination Coordinator identified below at least three days in advance of a meeting so that arrangements can be made accordingly.

If you believe discrimination has occurred with respect to an Air District program or activity, you may contact the Non-Discrimination Coordinator identified below or visit our website at www.baaqmd.gov/accessibility to learn how and where to file a complaint of discrimination.

Questions regarding this Policy should be directed to the Air District's Non-Discrimination Coordinator, Suma Peesapati, at (415) 749-4967 or by email at speesapati@baaqmd.gov.

# BAY AREA AIR QUALITY MANAGEMENT DISTRICT
## 375 BEALE STREET, SAN FRANCISCO, CA 94105
## FOR QUESTIONS PLEASE CALL (415) 749-4941

## EXECUTIVE OFFICE:
## MONTHLY CALENDAR OF AIR DISTRICT MEETINGS

# FEBRUARY 2022

| TYPE OF MEETING | DAY | DATE | TIME | ROOM |
|---|---|---|---|---|
| Advisory Council Meeting | Monday | 14 | 8:30 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Legislative Committee | Monday | 14 | 1:00 p.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Meeting | Wednesday | 16 | 9:00 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Administration Committee | Wednesday | 16 | 1:00 p.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Legislative Committee - Cancelled and rescheduled to Monday, February 14, 2022 at 1:00 p.m. | Wednesday | 16 | 1:00 p.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Stationary Source and Climate Impacts Committee – Cancelled and rescheduled to Monday, February 28, 2022 at 9:00 a.m. | Monday | 21 | 9:00 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Budget and Finance Committee | Wednesday | 23 | 9:30 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Mobile Source and Climate Impacts Committee - Cancelled | Thursday | 24 | 9:30 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Stationary Source and Climate Impacts Committee | Monday | 28 | 9:00 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Path to Clean Air Community Emissions Reduction Plan Steering Committee | Monday | 28 | 5:30 p.m. | Webcast only pursuant to Assembly Bill 361 |

# MARCH 2022

| TYPE OF MEETING | DAY | DATE | TIME | ROOM |
|---|---|---|---|---|
| Board of Directors Meeting | Wednesday | 2 | 9:00 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Community Equity, Health and Justice Committee | Thursday | 3 | 9:30 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Legislative Committee | Monday | 14 | 1:00 p.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Special Meeting as the Sole Member of the Bay Area Clean Air Foundation | Wednesday | 16 | 8:30 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Meeting | Wednesday | 16 | 9:00 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Administration Committee | Wednesday | 16 | 1:00 p.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Stationary Source and Climate Impacts Committee | Monday | 21 | 9:00 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Path to Clean Air Community Emissions Reduction Plan Steering Committee | Monday | 21 | 6:00 p.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Budget and Finance Committee | Wednesday | 23 | 9:30 a.m. | Webcast only pursuant to Assembly Bill 361 |
| Board of Directors Mobile Source and Climate Impacts Committee | Thursday | 24 | 9:30 a.m. | Webcast only pursuant to Assembly Bill 361 |

HL 2/4/2022 – 2:50 P.M. G/Board/Executive Office/Moncal

**BAY AREA AIR QUALITY MANAGEMENT DISTRICT**
Memorandum

To: Chairpersons Linda Rudolph and Gina Solomon, and Members of the Advisory Council

From: Jack P. Broadbent
Executive Officer/APCO

Date: February 14, 2022

Re: Approval of the Minutes of December 13, 2021

RECOMMENDED ACTION

Approve the attached draft minutes of the Advisory Council meeting of December 13, 2021.

BACKGROUND

None.

DISCUSSION

Attached for your review and approval are the draft minutes of the Advisory Council meeting of December 13, 2021.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Jack P. Broadbent
Executive Officer/APCO

Prepared by: Marcy Hiratzka
Reviewed by: Vanessa Johnson

ATTACHMENTS:

1. Draft Minutes of the Advisory Council Meeting of December 13, 2021

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Bay Area Air Quality Management District
375 Beale Street, Suite 600
San Francisco, CA 94105
(415) 749-5073

**DRAFT MINUTES**

Advisory Council Regular Meeting
Monday, December 13, 2021

*An audio recording of the meeting is available on the website of the Bay Area Air Quality Management District at*
*http://www.baaqmd.gov/about-the-air-district/advisory-council/agendasreports*

**This meeting was conducted under procedures authorized by Assembly Bill 361. Members of the Council participated by teleconference.**

1. **CALL TO ORDER**

Advisory Council (Council) Co-Chairperson, Dr. Linda Rudolph, called the meeting to order at 8:31 a.m.

**Roll Call:**

Present: Advisory Council (Council) Co-Chairpersons Drs. Linda Rudolph and Gina Solomon; Vice Chairperson Prof. Michael Kleinman; and Members Dr. Danny Cullenward, Dr. Adrienne Hollis, Garima Raheja; and Board Liaison David Haubert.

Absent: Member Dr. Pallavi Phartiyal.

2. **APPROVAL OF THE MINUTES OF OCTOBER 25, 2021, AND NOVEMBER 8, 2021**

Public Comments

No requests received.

Council Comments

None.

Council Action on Minutes of October 25, 2021

Dr. Hollis made a motion, seconded by Dr. Cullenward, to **approve** the Minutes of October 25, 2021; and the motion **carried** by the following vote of the Board:

| | |
|---|---|
| AYES: | Cullenward, Hollis, Kleinman, Raheja, Rudolph, Solomon. |
| NOES: | None. |
| ABSTAIN: | None. |
| ABSENT: | Haubert, Phartiyal. |

Council Action on Minutes of November 8, 2021

Vice Chair Kleinman made a motion, seconded by Dr. Hollis, to **approve** the Minutes of November 8, 2021; and the motion **carried** by the following vote of the Board:

| | |
|---|---|
| AYES: | Cullenward, Hollis, Kleinman, Raheja, Rudolph, Solomon. |
| NOES: | None. |
| ABSTAIN: | None. |
| ABSENT: | Haubert, Phartiyal. |

## 3. OVERVIEW OF PARTICULAR MATTER

NOTED PRESENT: Board Liaison Haubert was noted present at 8:40 a.m.

Greg Nudd, Deputy Air Pollution Control Officer of Policy, Dr. David Holstius, Senior Advanced Projects Advisor, and Dr. Ranyee Chiang, Director of Meteorology and Measurement, gave the staff presentation *Overview of Particulate Matter (PM),* including: highlights from Council recommendations (December 2020); permitting regulatory needs; modeling local-scale $PM_{2.5}$ impacts for risk management; overview/framing; update: state of the science; policy-relevant range; emissions, concentrations, exposures; risk: $PM_{2.5}$ and mortality; from modeling to evaluation; complexity: multiple metrics and population dependence; selected references; setting an air quality target; highlights from Council recommendations (December 2020); objectives and considerations; options for implementing recommendations; tools to identify issues and demonstrate progress; regional approach; local disparities approach; other open questions on metrics; and particulate matter key issues.

Public Comments

Public comments were given by Charles Reed, Emerald New Deal; Dr. Stephen Rosenblum, Palo resident; and Tara Cahn, Tara Cahn Architecture.

Council Comments

The Council and staff discussed the types of air pollution issues that the Air District studies and subsequent processes, and whether the Bay Area Air District is the only California air district pursuing the issues that it pursues; how Air District Board members can utilize the scientific information it receives from the Council; whether the Air District should consider morbidity rates; background levels of PM exposure and how wildfire smoke could be incorporated into standard PM regulation; whether studies regarding exposure of disproportionate burden on race (and gender or income), versus an average population, are available; whether the Air District should prioritize the protection of vulnerable/overburdened populations, versus average populations, in studies and in policy interventions, and how the Air District would respond if a member of the public inquired about that; why the Air District believes that current federal standards are not health protective, and whether the Air District believes that the federal

standards will become more stringent over time; the cumulative effects of multiple sources of air pollution, and whether it is more beneficial to analyze regional, versus local, air pollution; and the need for a health protective standard for regional PM.

Council Action

None; receive and file.

## 4. DISCUSSION OF ADVISORY COUNCIL MEETINGS AND 2022 TOPICS

Mr. Nudd gave the staff presentation *Discussion of Advisory Council Meetings and 2022 Topics*, including: research and discussion questions: climate, equity and community health, and PM.

Public Comments

Public comments were given by Dr. Stephen Rosenblum, Palo Alto resident; Jan Warren, Interfaith Climate Action Network of Contra Costa County; Christine Wolfe, California Council for Environmental and Economic Balance; and Tara Cahn, Tara Cahn Architecture.

Council Comments

The Council and staff discussed the Air District's impact on green economy jobs; the suggestion of looking at co-benefits regarding climate change, and prioritizing things that the Air District can do to reduce Toxic Air Contaminants, PM, and climate gases; the suggestion of establishing a budget to address health risks in various communities; the suggestion of initiating a series of case studies with a modeling approach that looks at various populations, health metrics, and time periods; how to prioritize interventions regarding climate pollution, and the difficulties of doing so when regulation has legal limitations; tradeoff decisions that the Air District faces, regarding the high impacts but short atmospheric lifetimes of non-carbon dioxide pollutants; which average $PM_{2.5}$ standards (24-hour or annual) are most utilized by the United States Environmental Protection Agency's (EPA) Clean Air Scientific Advisory Committee and California's Office of Environmental Health Hazard Assessment; how PM emissions from wildfires fit into California's assessment; the Air District's partnerships with the US EPA and California Air Resources Board to address community climate resilience clean air centers, particularly at schools for children during high-exposure (wildfire) situations; and whether the Air District has a standard method for presenting scientific information to its Board of Directors.

Council Action

None; receive and file.

## 5. REPORT OF THE EXECUTIVE OFFICER/APCO

Mr. Nudd reported that Jack P. Broadbent, Executive Officer/APCO, chose to waive his report.

## 6. PUBLIC COMMENT ON NON-AGENDA MATTERS

No requests received.

## 7. COUNCIL MEMBER COMMENTS/ OTHER BUSINESS

Co-Chair Solomon asked whether it can be proven that woodsmoke air pollution is greater than motor vehicle and refinery pollution during the winter in the Bay Area, and whether the Air District models wildfire smoke PM impacts by neighborhood.

Co-Chair Rudolph asked how much air pollution is generated by commercial operations and expressed the desire to hold such facilities accountable for their emissions.

Vice Chair Kleinman suggested that the Air District invite Dr. Timothy Larson of the University of Washington to speak about his findings regarding the health effects woodsmoke.

## 8. TIME AND PLACE OF NEXT MEETING

The time and place of the next Council meeting was originally at the Call of the Chair. After the meeting adjourned, the time and place of the next meeting was set for Monday, February 14, 2022, at 8:30 a.m., via webcast, conducted under procedures authorized by Assembly Bill 361.

## 9. ADJOURNMENT

The meeting adjourned at 11:03 a.m.

Marcy Hiratzka
Clerk of the Boards

**BAY AREA AIR QUALITY MANAGEMENT DISTRICT**
Memorandum

To: Chairpersons Linda Rudolph and Gina Solomon, and Members of the Advisory Council

From: Jack P. Broadbent
Executive Officer/APCO

Date: February 14, 2022

Re: Building Appliance Rules: Benefits to Outdoor Air Quality and Health

RECOMMENDED ACTION

None; receive and file.

BACKGROUND

The Air District's 2017 Clean Air Plan identifies the importance of reducing nitrogen oxide ($NO_x$) emissions from residential appliances; these sources are responsible for a significant portion of total $NO_x$ emissions in the Bay Area. To reduce these emissions, Air District staff recently crafted draft amendments to Regulation 9, Rule 4: Nitrogen Oxides from Fan Type Residential Central Furnaces ("Rule 9-4") and Regulation 9, Rule 6: Nitrogen Oxides Emissions from Natural Gas-Fired Boilers and Water Heaters ("Rule 9-6"). In the near term, these draft rule amendments include low-NOx requirements. In the longer-term (initial compliance dates from 2027 to 2031), they introduce a zero-$NO_x$ requirement. In practice, a zero-$NO_x$ standard would be expected to eliminate combustion emissions from new equipment and encourage adoption and development of alternative technologies for building appliances in the Bay Area. Air District staff plan to bring the proposal for adoption of the draft amendments to the Board of Directors in the summer of 2022.

As supplemental information to support the development of Rules 9-4 and 9-6, Air District staff have conducted a modeling-based evaluation of the impacts of natural gas combustion from residential and commercial space heaters, water heaters, and boilers. This evaluation quantifies benefits to outdoor air quality and health from the rules. It includes an estimate of the health benefits of reductions in secondary fine particulate matter ($PM_{2.5}$) that would result from reducing $NO_x$. It also includes an estimate of the health benefits of reductions in total $PM_{2.5}$ (directly emitted and secondary) from eliminating all natural gas combustion emissions from these building appliances. This item presents progress to date on the modeling-based evaluation.

DISCUSSION

Air District staff applied its regional air quality modeling system to estimate air pollution levels in a baseline emissions scenario and a control emissions scenario, with reductions in the control scenario matching emission estimates from natural gas-fired building appliances covered under Rules 9-4 and 9-6. Differences between baseline and control scenarios provided an estimate of the building appliance contributions to outdoor air pollution. Differences in $PM_{2.5}$ were used as inputs to the US Environmental Protection Agency's (EPA) Benefits Mapping and Analysis Program (BenMAP) to estimate health benefits from the proposed rules and monetary valuations associated with those benefits. Methods applied for this Bay Area study were similar to those applied in prior studies of the benefits of eliminating natural gas combustion in building appliances in the U.S. and in California. This presentation includes a summary-level comparison to those prior studies, in terms of methods and findings.

Modeled benefits of eliminating primary and secondary $PM_{2.5}$ generated by natural gas-fired combustion from the building appliances targeted in amendments to Rules 9-4 and 9-6 included the prevention of 39 to 89 premature deaths per year, with lower and upper estimates corresponding to the set of functions used by the US EPA to link $PM_{2.5}$ concentrations to health outcomes. Modeled benefits also included reductions in many non-fatal adverse health outcomes, such as heart attacks, strokes, and asthma onset and symptoms. The total valuation of all modeled health benefits was estimated to be between 410 and 930 million dollars per year. About 60% of the estimated benefits were attributed to reductions in secondary $PM_{2.5}$.

Under this item, staff will seek Advisory Council guidance on ideas for refining health impact evaluations, for enhancing presentation materials, and for identifying productive next steps. Because this study may serve as a prototype for future assessments, such guidance may find broader application in the Air District’s future work. One next step, currently underway, is an equity assessment to evaluate exposures of residential populations, by race and ethnicity, to identify who is most and least exposed to the $PM_{2.5}$ generated by the building appliances covered under the proposed rule amendments. Staff will specifically invite Advisory Council suggestions and ideas on well-designed and informative equity assessment methods, examples, and clear visual representations and framings of findings.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Jack P. Broadbent
Executive Officer/APCO

Prepared by: Phil Martien
Reviewed by: Greg Nudd

ATTACHMENTS:

None.

**BAY AREA AIR QUALITY MANAGEMENT DISTRICT**
Memorandum

To: Chairpersons Linda Rudolph and Gina Solomon, and Members of the Advisory Council

From: Jack P. Broadbent
Executive Officer/APCO

Date: February 14, 2022

Re: Regulatory Toolbox and PM Health Impacts Methodology

RECOMMENDED ACTION

None; receive and file.

BACKGROUND

The 2020 Advisory Council researched and provided a report to the Air District Board of Directors on particulate matter at a joint meeting of the Advisory Council and Board of Directors in December 2020. In 2021, the Advisory Council received a presentation on particulate matter health impacts.

This report highlighted the significant health impacts of localized exposure to fine particulate matter (PM2.5). Unfortunately, the current tools for regulating air pollution do not adequately address these impacts. The Air District has been working with the Office of Environmental Health Impacts Assessment, the California Air Resources Board, and the U.S. Environmental Protection Agency on a new methodology for quantifying the localized health impacts of PM2.5. A detailed white paper on the draft methodology is attached.

DISCUSSION

In order to guide the development of the PM2.5 health impacts methodology, it's important to understand the regulatory context in which that methodology is likely to be used.

The Air District's regulatory authority over stationary sources of air pollution can be described by three complimentary approaches:

- **New Source Review Permitting**—New Source Review applies to new and modified sources; any significant modifications at a regulated stationary source trigger a new source review. Smaller sources may be exempt from permitting because they are individually not significant contributors to regional air pollution.

- **Regulations for Existing Sources**—Regulations for Existing Sources require feasible emissions reductions at existing sources and usually require retrofits. Regulations of Existing Sources may include emission limits or health impact limits.

- **Point-of-Sale Rules—**Point-of-Sale Rules set emissions performance limits for air pollution-emitting products sold and used within the Air District's jurisdiction. Examples of this include volatile organic compound limits on architectural coatings or nitrogen oxides limits on residential space and water heaters.

The Air District's regulatory authority is limited to stationary sources. Mobile sources are regulated under the jurisdiction of the California Air Resources Board (CARB) and/or the U.S. Environmental Protection Agency. In addition, there must be technically feasible mechanisms for reducing emissions and the source or source category must significantly contribute to emissions and/or health risk.

The Air District's work on the development of a PM2.5 local risk methodology will fill gaps in the Air District's regulatory tools and strengthen the Air District's ability to reduce emissions. As the Air District develops this methodology, it must consider key questions including the following:

- Focus on mortality or include other health endpoints?
- How to incorporate baseline incidence rates?

Air District staff will provide a detailed presentation and request the Advisory Council's input and feedback.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Jack P. Broadbent
Executive Officer/APCO

Prepared by: Sonam Shah-Paul
Reviewed by: Greg Nudd

ATTACHMENTS:

1. BAAQMD Modeling Local Sources of Fine Particulate Matter (PM2.5) for Risk Management Methodology, Implementation, and Case Studies

.

# Modeling Local Sources of Fine Particulate Matter ($PM_{2.5}$) for Risk Management

Methodology, Implementation, and Case Studies

Bay Area Air Quality Management District (BAAQMD)

DRAFT | January 6, 2022

# Contents

# Authors and Contributors

This report has benefited from extensive collaboration, and we thank all those who have contributed. This work builds on earlier work at the Air District integrating BenMAP with regional air quality modeling assessments.

Yuanyuan Fang was the primary author of the first draft, and the primary author of an early second draft. Yuanyuan ran the U.S. EPA's Environmental Benefits Mapping and Analysis Program-Community Edition (BenMAP-CE) and produced data, code, and draft figures for the case studies.

David Holstius served as the primary author of this draft. David was an early consultant to this work and took a lead role in documenting and exploring the methodology, including the development of supplemental material.

Judith Cutino helped to develop contacts, especially within the Office of Environmental Health Hazard Assessment (OEHHA), and drafted a letter requesting OEHHA participation.

Phil Martien served as a consultant, reviewer, and project coordinator.

Neal Fann and Ken Davidson at the US Environmental Protection Agency (US EPA) provided technical training, support, and consultation on BenMAP-CE software, which they and their team developed. Lauren Zeise, Rupa Basu, and their team at the Office of Environmental Health Hazard Assessment (OEHHA) lent their expertise concerning health-impact functions. Both US EPA and OEHHA staff reviewed and provided extensive comments on an early draft of this document that helped strengthen this report and methodology.

Amy Kyle, Consulting Scientist in Health and Environment, reviewed a previous

version and provided insightful comments and suggestions.

The Bay Area Air Quality Management District's Advisory Council, through their deliberations and recommendations, have spurred additional action in protecting Bay Area residents from the adverse health impacts of fine particulate matter.

Finally, many members of the Bay Area public and representatives of community and environmental organizations have long advocated for additional stringency in regulating sources of air pollution that impact community health. This proposed methodology has benefited from their input and advocacy, and is intended to advance the protection of public health through the quantification and management of risks posed by local sources of fine particulate matter.

# Executive Summary

The Bay Area Air Quality Management District (BAAQMD) has assembled a draft methodology for use in managing health risks posed by specific sources of fine particulate matter ($PM_{2.5}$) at the community level. Its primary purpose is to inform $PM_{2.5}$ air quality assessments and policies designed and implemented by local air quality agencies, including technical assessments, regulations, and plans to reduce local $PM_{2.5}$ emissions and exposures.

In this whitepaper, we focus on adult mortality from long-term exposures, guided primarily by evidence and methods reviewed, summarized, and applied by the US EPA (US EPA 2019, 2021b, 2021a). We also focus on modeling a certain class of facilities: those with inventoried $PM_{2.5}$ emission rates that are a relatively small fraction of regional totals, but still large enough to be a significant local concern. Compared to the largest permitted stationary sources, such facilities typically have larger exposure factors (impact per ton of emissions). They are often located in closer proximity to residential populations and may not be equipped with the tall, hot stacks associated with the largest sources.

As a proof-of-concept, BAAQMD conducted two case studies, drawing upon recent air quality modeling conducted in support of the recent West Oakland Community Action Plan (BAAQMD and WOEIP 2019). We caution that our purpose in this report is not to attribute impacts or risks to any actual facility. For our case studies, we assumed baseline conditions and population characteristics uniformly equivalent to a Bay Area average, and a relative risk consistent with important cohort studies on which US EPA evaluations are based. With these assumptions, we estimated that $PM_{2.5}$ emissions from such a facility could be increasing mortality rates by approx-

imately +3/M (i.e., deaths per million persons per year) per facility, on average, across residential neighborhoods in a community similar to West Oakland in size and composition. The impact at short distances could be on the order of +100/M. These estimates are for a statistically representative resident of the Bay Area, without any added margin of safety.

Within a larger risk-management context, we recommend that known modifiers of risk be considered. At the time of this writing, for the endpoint we are considering, the US EPA has determined these to include lifestage and race/ethnicity (US EPA 2019, 2021b). We provide information and sensitivity analyses for race/ethnicity alongside the results from our case studies. In the Discussion, we articulate some relevant limitations and tradeoffs in calculations for at-risk groups.

We modeled several metrics of impact: concentrations; relative risks; risk differences; exposures; and burdens. The ideal metric or set of metrics to model and evaluate depends on the functional form and intent of the larger risk-management process. For example: whether it is to be based on a maximum impact or an impact across a local area; whether additional endpoints are to be considered; tolerance for uncertainties and errors of different kinds; and the relative weight placed on other aspects of the process, such as implementation requirements, transparency, and robustness. Some combinations of these argue for a metric based on relative risk; others, for a risk difference; still others, a population-dependent metric such as exposure or burden. Next steps for this work include the assessment, in consultation with risk assessors and managers, of the relative feasibility and fitness-for-purpose of each.

In the text that follows, we explain the components of the framework and how we arrived at the statements above. The explanation is presented in three parts: (1) a description of the general framework and concepts; (2) details of our implementation, including datasets and parameters; and (3) the results of our case studies. We close by discussing the strengths, limitations, and implications of the work, including issues that we are actively working to resolve.

# 1 Introduction

Our purpose in this report is to propose a general methodology, demonstrate its application, and discuss its strengths, limitations, and implications for the practice of regulating fine particulate matter ($PM_{2.5}$) at the community level. National- and regional-scale health impact assessments (HIAs) for $PM_{2.5}$ have been conducted for many years (Fann et al. 2011; Howard et al. 2019; Tanrikulu, Tran, and Beaver 2011; Tanrikulu et al. 2019), corresponding to the needs of current regulatory frameworks that focus on reducing regional $PM_{2.5}$ levels to meet the National Ambient Air Quality Standards (NAAQS). Continuous observation of ambient $PM_{2.5}$ levels, through agencies' official measurement networks, has also been successful in monitoring and verifying the success of policies to reduce average ambient $PM_{2.5}$ and meet the NAAQS in many regions, including the Bay Area. However, it has become increasingly clear that gaps left by the NAAQS-centered approach must be addressed.

A gap that this work contributes to closing is the persistent exposure of some communities and populations to locally elevated concentrations of $PM_{2.5}$. Although a large fraction of $PM_{2.5}$ is regionally contributed (Blanchard 2004; Robinson et al. 2007), variations in exposure exist within communities (Colmer et al. 2020; Chambliss et al. 2021; Wilson et al. 2005; Blanchard 2004; Eeftens et al. 2012) and have been linked to legacies of structural and institutional discrimination (Morello-Frosch and Lopez 2006; Fisher, Kelly, and Romm 2006; Houston et al. 2004; Houston, Krudysz, and Winer 2008; Quiros et al. 2013; Jacobson, Hengartner, and Louis 2005).

One way to accelerate the closure of this gap may be to introduce a complementary approach to local air quality regulation, one that relies on estimates of health impacts from specific sources at small spatial scales. Such an approach has been taken for

many years to regulate the impacts from toxic air contaminants (TACs) emitted by individually permitted sources, but not the impacts from $PM_{2.5}$.[1]



[1]With the exception of diesel exhaust particulate matter (DPM), which is treated as a TAC. DPM is typically a small fraction of total ambient $PM_{2.5}$ by mass.

# 2 Concepts and Methods

In this section, we describe a methodology for the estimation of impacts from long-term exposure to $PM_{2.5}$ on adult mortality. We introduce the relevant concepts and methods in two parts: first, the general framework; and second, an example implementation of that framework. Its application is illustrated through case studies sited in West Oakland, the results of which are reported in the following section. Additional details and considerations may be found in the Discussion.

## 2.1 General Framework

The general framework proposed here is similar in some ways to a framework that is widely employed in health risk assessments (HRAs) of toxic air contaminants (TACs). For the reader who is already familiar with such HRAs, understanding the similarities and differences may be helpful.[1]

Table 2.1 lists ten elements common to both (a) the proposed framework ("$PM_{2.5} \rightarrow$ Mortality"), and (b) the TAC framework ("TAC $\rightarrow$ Cancer").[2] Below, we explain each of these elements, comparing and contrasting them with respect to (a) and (b).

[1] In comparing the two frameworks, we are not advancing any arguments for any particular legal justification for the regulation of $PM_{2.5}$. Neither are we endorsing the TAC framework, or its use in practice, without reservation. Rather, we are simply illustrating by way of analogy.

[2] For a complete description of the "TAC $\rightarrow$ Cancer" framework, see OEHHA (2015) and ARB/CAPCOA (2015). See also Appendix B.

Table 2.1: Elements of established and proposed frameworks.

| | TAC → Cancer | $PM_{2.5}$ → Mortality |
|---|---|---|
| Emissions | As inventoried | As inventoried |
| Concentrations | Modeled ambient (annual average) | Modeled ambient (annual average) |
| Health Endpoint | Cancer | Mortality |
| Exposure Duration | Long-term (30 years) | Long-term (chronic) |
| Population | All ages | Adults |
| Response Function | Linear | Log-linear, though approximately linear |
| Intermediate Factors | Linear decomposition into components of exposure and dose (age-dependent) | Not applicable; population responses are estimated directly from ambient concentrations |
| Effect Size | From toxicological and/or epidemiological studies | From epidemiological studies |
| Baseline Conditions | Not applicable | Baseline mortality rate |
| Margin of Safety | Included in slope and unit-risk factors | To be determined |

## Emissions and Concentrations

Both frameworks shown in Table 2.1 assume that contributions to near-field ambient concentrations can be adequately estimated using dispersion models. These models rely on user input of pollutant emission rates, release parameters, site conditions, and meteorological conditions to predict annual average concentrations on a user-defined grid of coordinates ("receptor locations").

In the TAC → Cancer framework, modeled concentrations are weighted by "toxicity factors" that are effectively determined by both the particular TAC and by the scope of the assessment (e.g., inhalation-only *vs* multipathway). This step puts all designated carcinogens on a common scale. In this $PM_{2.5}$ → Mortality framework, the intensity of $PM_{2.5}$ concentrations is assumed to be adequately captured by a mass-concentration metric (US EPA 2019).

Because of the short distances (and hence, short timescales) involved, we consider here only emissions and transport of primary $PM_{2.5}$, holding aside the complex chemistry involved in the formation of secondary $PM_{2.5}$. While we acknowledge that there is evidence for varying toxicity among $PM_{2.5}$ subspecies, we do not yet have enough information to conduct assessments based on subspecies (US EPA 2019, 2021b).

## Health Endpoint

In principle, the mortality endpoint can be evaluated for all age groups, but for reasons explained in Section 2.2, we restricted it to adults $\geq 30$ years of age.

**Mortality rate.** Dividing mortality (deaths) by population size and time (over which both the deaths and the persons are counted) yields a metric known as the *mortality rate*. Conventionally, annual mortality rates are typically reported as "deaths per 100,000 persons [per year]." For ease of comparison with the TAC → Cancer framework, hereafter we will express them in terms of "deaths per million persons [per

year]."

**Mortality risk.** As a corollary, "*annual* mortality risk" is analogous to what "*annual* cancer risk" would be in the TAC → Cancer framework (which actually evaluates contributions to a *lifetime* risk of cancer.) Risk in a technical sense is the probability of an outcome in a given period of time; the length of time is important, since the lifetime risk of mortality is always 100%.

Although risks to human health are technically only present where exposures are not zero, in practice many stakeholders use the work "risk" to mean risk *conditional* on a "statistically representative person" being exposed. In this whitepaper, we follow that convention.

**Mortality.** Finally, "mortality" (count of deaths [per year]) is analogous to "cancer *burden*" (count of cases). The equations in the next section estimate mortality *rates* ($y$) — or changes therein ($\Delta y$) — rather than mortality counts. Simply multiplying $y$ by the size of the population at risk yields mortality instead; likewise, multiplying $\Delta y$ by the population size yields an estimated change.

## Response Function

A response function—also known as a health impact function, an exposure-response function, or a concentration-response function—expresses a relationship between $x$ (an independent variable; often but not always "exposure") and $y$ (the outcome). In our context, $x$ is a "unit increment" of exposure to $PM_{2.5}$ (e.g., 10 µg/$m^3$), while $y$ is a mortality rate.

Generally, a response function can be written in the form of a mathematical equation, like the ones that follow. In the TAC → Cancer framework, the response function $y = f(x)$ is linear in $x$. This means that an additive change in $x$ induces, and can only induce, an additive change in $y$. In this proposed PM25 -> Mortality framework,

the response function, Equation (2.1), is nonlinear.[3] (See Section 4.4 and Appendix C for discussion.)

$$\begin{aligned} \ln(y) &= \beta x + C \\ y &= \exp(\beta x + C) \end{aligned} \tag{2.1}$$

The term $e^{\beta\,\Delta x}$ in Equations (2.2) and (2.3) expresses relative risk (RR), or the *multiplicative* change in $y$ that is associated with a linear change in $x$. However, suppose we are interested in modeling excess risk on an additive scale, as in the TAC $\rightarrow$ Cancer framework. Let $\Delta x = x - x_0$ and $\Delta y = y - y_0$, where $x_0$ and $y_0$ represent the baseline (i.e., existing) $PM_{2.5}$ concentration and mortality rate, respectively. Taking $\Delta x > 0$ to mean an increase in $PM_{2.5}$, and $\Delta y > 0$ a corresponding increase in $y$, we have:

$$y/y_0 = e^{\beta\,\Delta x} \tag{2.2}$$

$$\Delta y = y - y_0 = y_0\left(e^{\beta\,\Delta x} - 1\right) \tag{2.3}$$

In the TAC $\rightarrow$ Cancer framework, because risk is assumed to increase linearly with exposure, "risk" always means "risk difference" (additive scale), never "relative risk" (multiplicative scale). As such, an estimate of baseline risk ($y_0$) is never needed. (See Section 4.5.3 and Appendix A for discussion.)

A delta-response function can offer a more convenient way of evaluating a *change* in impacts, starting with a *change* in $PM_{2.5}$. This can be evaluated either on a multiplicative scale, as in Eq (2.2), or on an additive scale, as in Eq (2.3). In either case, when a source does not yet exist, we can set $\Delta x$ in Eq (2.2) or (2.3) proportional to the potential $PM_{2.5}$ concentrations attributable to that source, or to a proposed

[3]Note: $C$ is a constant offset, not an ambient concentration.

increase. When a source already exists, we may wish to estimate the benefit from a potential reduction in emissions. The same equations can be used; the signs on $\Delta x$ and $\Delta y$ should be positive, corresponding to reductions in emissions and damages respectively.[4]

## Effect Size, Exposure, and Dose

The *effect size*, or the change in $y$ associated with a unit change in $x$, is represented in Equations (2.2) and (2.3) by the term $\beta$. Typically, $\beta$ will be based on an epidemiological study in which ambient $PM_{2.5}$, measured or estimated at some locations, was the independent variable. This means that $\beta$ will encompass all of the factors (indoor/outdoor ratios, breathing rates, fractions of time at home, etc.) that lay on the causal pathways between ambient $PM_{2.5}$ and mortality for the population that was studied. Generally, epidemiological studies estimate $\beta$ by adjusting for other measured factors in such a way that $\beta$ will (ideally) approximate the causal effect of $x$ alone. Most such studies report an estimated risk ratio, such as a relative risk (RR), hazard ratio (HR), or odds ratio (OR), for a given increment of $PM_{2.5}$ (such as +10 $\mu g/m^3$). In the equations above, $\beta$ is effectively the natural logarithm of that risk ratio.

## Baseline Conditions

In Equation (2.3), the parameter $y_0$ stands for the baseline mortality rate. It is not the mortality rate that would exist in the absence of any $PM_{2.5}$, but rather the mortality rate in the world as-is. Issues related to obtaining or estimating $y_0$ do not arise in the TAC $\rightarrow$ Cancer framework, which is independent of baseline conditions.

[4] In some settings, a positive $\Delta x > 0$ is instead taken to mean a reduction, rather than an increase. Similarly, a positive $\Delta y > 0$ is instead interpreted as beneficial, rather than harmful. For such an interpretation, the appropriate form of the equation is $\Delta y = y_0 \left(1 - e^{-\beta \Delta x}\right)$. See Appendix C for details.

These are discussed in Section 4.5.3.

Estimates of baseline rates can be allowed to vary by subgroup. This may sometimes, but not always, be protective of at-risk populations; Section 3.3 illustrates this paradox using example data.

## Conceptual Diagram

Figure 2.1 is a conceptual diagram of the relationships between the concepts and metrics described above. Generally, these increase in complexity as one moves from top to bottom. Figure 2.1 also serves as a guide to the next full section (Results and Case Studies).

**Concentrations and Relative Risks.** We begin with a spatially resolved map of predicted *concentrations*, which itself depends on emissions and meteorology (not shown). With an estimated effect size ($\beta$) that is spatially invariant and population-average, we can apply Eq (2.2) to generate a map of *relative risk* that looks essentially the same.[5] This relative risk can itself be visualized as a smooth surface or converted to contour lines (see Figure 3.1 for an example).

**Risk Differences.** We can then incorporate a constant baseline ($y_0$) to arrive at a *risk difference* map. Since our case studies do not cross county lines, we can readily adopt either a county-specific baseline, or a regional average; for our case studies, we opt for the former.

It is also possible to estimate a "local" baseline rate by combining stratified[6] county-level rates with similarly stratified block-level population counts. This option requires the determination of a "local" boundary (i.e., which blocks to include in an assessment). We do not take that path here, but we report such an estimate for the purposes

[5]The units will be different, of course, and there will be some nonlinear warping of the surface, but the apparent difference is small and the shapes of contours do not change.

[6]Commonly-used strata include age, sex, and race/ethnicity. Any combination is possible.

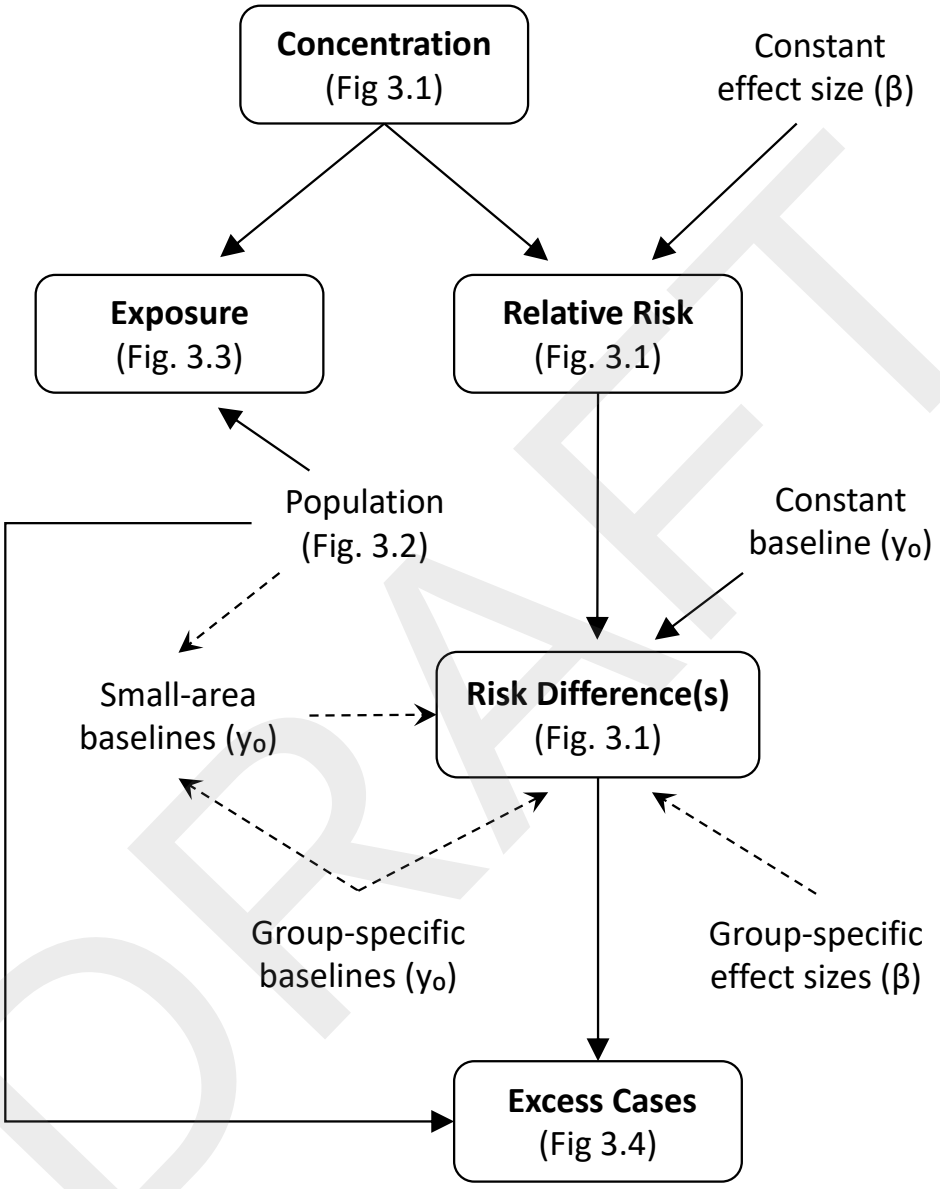



Figure 2.1: Pathways to the construction of spatially-resolved predictions of: ambient concentrations, population exposures, excess risk, and excess cases of, e.g., mortality. Dashed lines indicate certain options that can increase the specificity of predictions, potentially at some cost to reliability or feasibility. Tradeoffs and recommendations, as well as enhancements not depicted here, are taken up in the Discussion.

of sensitivity analysis.

**Constant *vs* Group-Specific Parameters.** To construct maps of population-average relative risks or risk differences, we do not require data on the spatial distribution, composition, and/or risk factors of the local population. We can think of these as estimates of the excess risk *conditional on* a "statistically average person" being exposed.

It is also possible to incorporate group-specific effect sizes and baselines into risk-difference maps, which in effect generates maps of risks for different hypothetical populations. We do not take that path here, for reasons articulated in the Discussion, but we supply Table 2.4 and accompanying results in order to demonstrate that incorporating some baseline variation alone, without considering effect modification, is not necessarily protective of at-risk populations.

**Exposure and Excess Mortality.** Excess mortality (deaths) can be calculated by combining a risk-difference estimate with population-density data. This is conceptually equivalent to combining estimates of relative risk and exposure.

The Discussion covers additional considerations relevant to risk management, decision-making, and the selection and/or combination of appropriate metrics. The next section outlines the specifics of our implementation, including parameters and datasets that we used to construct our case studies.

## 2.2 Implementation and Case Studies

Our implementation has three kinds of elements: first, the input data; second, our additional assumptions; and third, the methods and tools used to calculate results. From a procedural perspective, we can also think of these as comprising:

- Estimates of $PM_{2.5}$ emissions from the BAAQMD inventory;

- The dispersion model AERMOD;
- US EPA methodology, consistent with its BenMAP implementation (US EPA 2021a, 2021c);
- Estimate(s) of effect sizes; and
- Estimate(s) of baseline mortality rates (for risk differences) and/or population (for exposure and burden).

To illustrate the application of this methodology, we conducted two case studies. Each simulated a single facility having multiple sources of $PM_{2.5}$ emissions. We caution that these simulations are intended to illustrate and explain our implementation, rather than to quantify the actual impacts of any actual facility. For a discussion of the representativeness of these case studies, see Section 4.10.

## West Oakland

Our case studies focus on West Oakland, a community in the San Francisco Bay Area (Figure 2.2). West Oakland has previously been the subject of relatively extensive air quality measurement studies and modeling efforts — see, for example, the West Oakland Community Action Plan (BAAQMD and WOEIP 2019) and its supporting projects, including a regional-scale photochemical model developed by BAAQMD (Tanrikulu et al. 2019).

There is one BAAQMD-run air quality monitoring site in West Oakland, and another nearby at Laney College (Figure 2.2). In recent years (2016-2018), three-year average $PM_{2.5}$ concentrations at these two sites have ranged from approximately 9 to 12 $\mu g/m^3$ (US EPA 2020).[7] For comparison, in a recent modeling assessment conducted at 1×1km scale, simulated annual average $PM_{2.5}$ concentrations across West Oakland varied from approximately 7 to 9 $\mu g/m^3$ (Tanrikulu et al. 2019).

[7]The upper end of this range has been significantly influenced by wildfires. The main point is that, in terms of magnitude, average ambient $PM_{2.5}$ concentrations in West Oakland are much closer to 10 $\mu g/m^3$ than 100 $\mu g/m^3$.

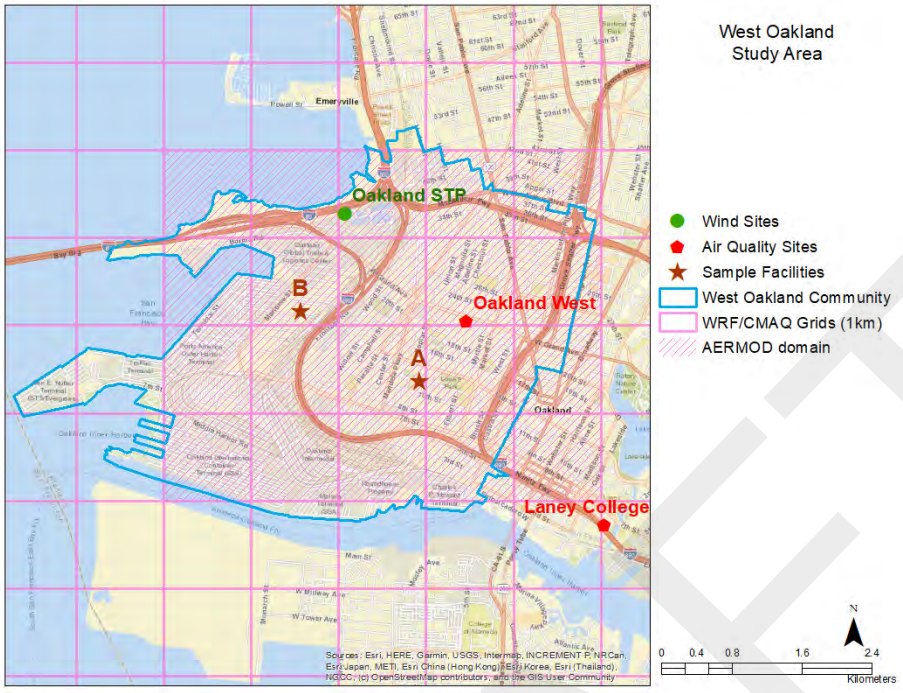



Figure 2.2: Map of the West Oakland study area

## Emissions

Estimated $PM_{2.5}$ emission rates (Table 2.2) were taken from BAAQMD's emission inventory. These were derived by applying source-specific or category-specific speciation factors ($PM_{2.5}$ / $PM_{10}$) to inventoried estimates of $PM_{10}$. We note that they have not been evaluated against direct measurements of $PM_{2.5}$ from these specific sources. For further discussion, see Sections 4.10 and 4.12.

## Modeled $PM_{2.5}$

To generate estimates of the primary $PM_{2.5}$ attributable to modeled sources, we used the AERMOD model (US EPA 2018) to predict directly attributable ambient $PM_{2.5}$ concentrations ($\Delta PM_{2.5}$) on a 20×20m scale for each source at each facility. AERMOD is a steady-state plume model that incorporates air dispersion based

Table 2.2: Emissions from modeled sources, as inventoried.

| Facility | Source Type | Emissions (ton/yr) |
|---|---|---|
| Facility A | Stack | 0.002 |
| | Stack | 0.010 |
| | Stack | 0.000 |
| | Fugitive | 0.564 |
| Facility B | Fugitive | 0.714 |
| | Fugitive | 0.769 |

on planetary boundary layer turbulence structure and scaling concepts, including treatment of both surface and elevated sources. Details regarding our AERMOD configuration (local meteorology, release heights, flow rates, etc.) can be found in the technical appendices to the West Oakland Action Plan (BAAQMD and WOEIP 2019).

After generating modeled 20×20m annual average $PM_{2.5}$ concentrations with AERMOD, we also computed the corresponding area-weighted arithmetic means for all West Oakland Census blocks, for use in calculating block-level risks and exposures.

## Population

Estimates of the size of the West Oakland population vary, depending on how the boundary is defined and which populations are counted (e.g. residents vs daytime). We adopted the boundary shown in Figure 2.2, which is consistent with the West Oakland Action Plan (BAAQMD and WOEIP 2019). Using the PopGrid tool included with the BenMAP-CE platform, we extracted block-level residential adult (age $\geq$ 30) population counts from from 2010 US Census data, stratified by age, sex, race, and ethnicity,Using the PopGrid tool included with the BenMAP-CE platform, we extracted block-level residential adult (age $\geq$ 30) population counts from from

Table 2.3: Selected estimates of the long-term effect of $PM_{2.5}$ on adult mortality.

| | Basis | | |
|---|---|---|---|
| Publication | Cohort | Exposure | RR per 10 $\mu g/m^3$ |
| Jerrett (2013) | ACS CSP-II[a] | LUR-DSA[1] | 1.065 (1.035, 1.096) |
| Jerrett (2013) | ACS CSP-II[ab] | LUR-DSA[1] | 1.060 (1.003, 1.120) |
| Pope (2015) | ACS CSP-II[a] | LUR-BME[2] | 1.07 (1.06, 1.09) |
| Turner (2016) | ACS CSP-II[a] | Hybrid[34] | 1.06 (1.04, 1.08) |
| Di (2017) | Medicare[c] | Hybrid[3] | 1.073 (1.071, 1.075) |
| Di (2017) | Medicare[c] | Monitor[5] | 1.061 (1.059, 1.063) |

[1] Land Use Regression with Deletion-Substitution-Addition
[2] Land Use Regression with Bayesian Max Entropy kriging of residuals
[3] Ground-level monitoring combined with photochemical model predictions
[4] Hierarchical Bayesian space-time model (HBM)
[5] Nearest ground-level monitor within 50 km
[a] Adults age $\geq 30$
[b] California subset of national cohort
[c] Adults age $\geq 65$

2010 US Census data for blocks contained within or intersected by that boundary. We then used the Woods and Poole (2015) forecasting method, as implemented by BenMAP-CE, to predict block-level population counts for 2018.

## Effect Sizes and At-Risk Populations

Table 2.3 lists different estimates of the effect of a +10 µg/m$^3$ increase in long-term $PM_{2.5}$ exposure on an adult population. For our case studies, we provisionally adopted a relative risk estimate of 1.07. As Table 2.3 shows, despite variation among the exposure-assignment methods used, and the cohorts studied, there is enough agreement to consider this a serviceable estimate.[8]

Although $PM_{2.5}$ has varying effects on different kinds of mortality (cardiopulmonary,

[8]For the purposes of this whitepaper, we can set aside more complex techniques like pooling or meta-analysis. The point is to illustrate and examine the framework itself, for which one digit of precision is enough.

all-cause, etc.), these studies examined all-cause adult mortality. Therefore, we also used an all-cause adult mortality baseline when calculating risk differences, and restricted our estimates of excess exposure and mortality to an adult population as well (age $\geq$ 30).

Table 2.4: Hazard ratios for a +10 µg/m$^3$ increase in annual mean $PM_{2.5}$ exposure. Reproduced from the Supplement to Di et al (2017); these data also appear in the current version (v1.5.8) of BenMAP-CE.

| Race / Ethnicity | Relative Risk |
|---|---|
| White | 1.063 (1.060, 1.065) |
| Black | 1.208 (1.199, 1.217) |
| Asian | 1.096 (1.075, 1.117) |
| Hispanic | 1.116 (1.100, 1.133) |
| Native American | 1.100 (1.060, 1.140) |
| (All) | 1.073 (1.071, 1.075) |

As described in Section 2.1, it is possible to incorporate group-specific effect-size estimates. Table 2.4, reproduced from the Supplement to Di et al (2017), lists estimates obtained from a large cohort study of $PM_{2.5}$ and mortality in the United States.

According to these data, for the same fixed increment of $PM_{2.5}$, the relative risks for non-white populations are larger. For example, the excess relative risk for Black cohort members is estimated to be 2.8 times as large as the average.[9] In the Results (Section 3.3), we use these data to illustrate the effects of incorporating group-specific variation.

[9] A ratio of excess relative risks is calculated as $(RR_1 - 1)/(RR_0 - 1)$.

## Baseline Mortality Rate

To obtain an estimate of the risk difference (RD), equation (2.3) requires an estimate of the baseline mortality rate ($y_0$) as input. For our primary analysis, we used the crude (not age-adjusted) 2007-2016 rate for the nine counties in the San Francisco Bay Area. For a sensitivity analysis, we obtained two additional rates from CDC-WONDER[10]: an age-adjusted regional rate, and a county-level crude rate. We also derived a (provisional) community-specific estimate for West Oakland by downscaling age- and race/ethnicity- stratified adult mortality rates for Alameda County, using similarly stratified BenMAP-exported population projections for 2018. Specifically, we calculated a single $y_0$ for West Oakland as:

$$y_0 = \frac{\sum \left( P_{ij} \times R_j \right)}{\sum P_{ij}} \tag{2.4}$$

… where:

- $i$ is the geographic unit[11];
- $j$ is the population stratum[12];
- $P_{ij}$ is the population size for block $i$, stratum $j$; and
- $R_j$ is the county-level mortality rate (deaths/person) for stratum $j$.

Geographic units $i$ consisted of Census blocks in West Oakland. Strata for the mortality rates $R_j$ were limited by BenMAP to the crossing of `Race` (`WHITE` / `Non-WHITE`) with `Age` (7 brackets). Strata for $P_{ij}$ were consolidated to match, with ages restricted to 30 years and up.

[10]Wide-ranging Online Data for Epidemiologic Research (WONDER) database, operated by the U.S. Centers for Disease Control and Prevention (CDC).

[11]Frequently-encountered geographic units include Census blocks, Census blockgroups, Census tracts, and ZIP code tabulation areas (ZCTAs). In our case studies, we used Census blocks.

[12]The population strata may be defined by a combination of variables including Age, Race, Ethnicity, and/or Sex.

See Appendix A for tables of stratified mortality rates ($R_j$), as well as examples and discussion of issues related to small counts, age-adjustment, and race/ethnicity.

## Increments *vs* Ambient Concentrations

Of interest in this proposed framework are not total ambient concentrations *per se*, but rather *incremental* concentrations (that is, contributions or changes to those totals).[13] These increments are generally on the order of $\pm 1$ µg/m$^3$ or less, within a policy-relevant range centered on roughly 10 µg/m$^3$. Figure 2.3 illustrates that there is ample evidence, based on contrasts within that policy-relevant range, supporting the two major U.S. cohort studies listed in Table 2.3.[14] Estimates of the impacts of such increments, within that range, will therefore be well supported. For extended discussion, please see US EPA (2021a), Section 4.4, and the Appendices.



[13]To maintain the distinction, we have attempted to prefix incremental concentrations and their corresponding impacts with a plus symbol ("+") throughout this report.

[14]US EPA (2021a) characterizes the range for cohort studies evaluated for the 2019 ISA at 5.9 to 16.5 µg/m$^3$, and the range for the most recent studies as 5.9 to 11.65 µg/m$^3$.

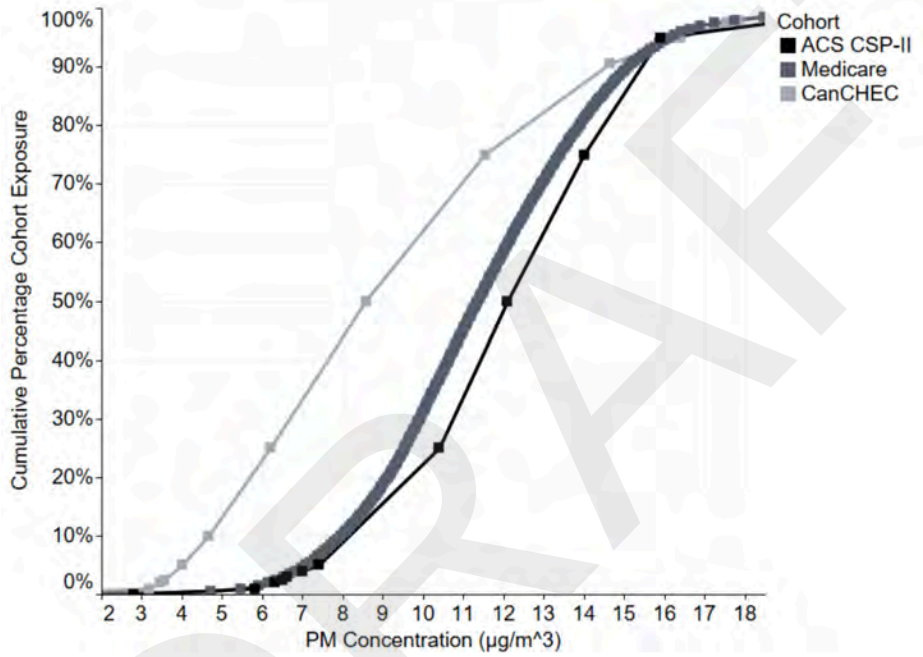



Figure 2.3: Cumulative percentile of $PM_{2.5}$ cohort exposure from the ACS CSP-II, Medicare, and CanCHEC cohorts. Reproduced from p. 98 of EPA-HQ-OAR-2020-0272 (EPA 2021 TSD).

# 3 Results and Case Studies

## 3.1 Overview

Five types of calculated impacts are presented in this section: $PM_{2.5}$ concentration; relative risk; risk difference; exposure, and excess mortality. For a conceptual diagram, see Figure 2.1 in the previous section.

## 3.2 $PM_{2.5}$

Figure 3.1 shows the incremental contributions to ambient concentrations ($\Delta PM_{2.5}$) modeled using AERMOD. For each facility, a contour line is drawn corresponding to an increment of $\Delta PM_{2.5} = +0.1$ µg/m$^3$. (This is approximately 1% of total annual average ambient $PM_{2.5}$ in the Bay Area.) Both facilities are modeled using the same meteorological data and model parameters (deposition, etc.), so the results differ only due to differences in release parameters and emission rates. A slightly smaller +0.1 µg/m$^3$ contour line is associated with Figure A, which has lower emissions (Table 2.2). However, the two contour lines are similar in shape.

Across the modeled domain, the population-weighted average $\Delta PM_{2.5}$ contributed by Facility A is 0.049 µg/m$^3$; for Facility B, it is 0.042 µg/m$^3$. For Facility A, the maximum[1] modeled $\Delta PM_{2.5}$ for any receptor within a residential block is +17.5 µg/m$^3$. For Facility B, it is +0.8 µg/m$^3$.

[1]Maxima for gridded $\Delta PM_{2.5}$ can be sensitive to aspects of the grid that was used, including its orientation, offset, and resolution. In typical applications of the TAC → Cancer framework, screening for maxima is limited to identifiable residential parcels, rather than all locations within a residential block.

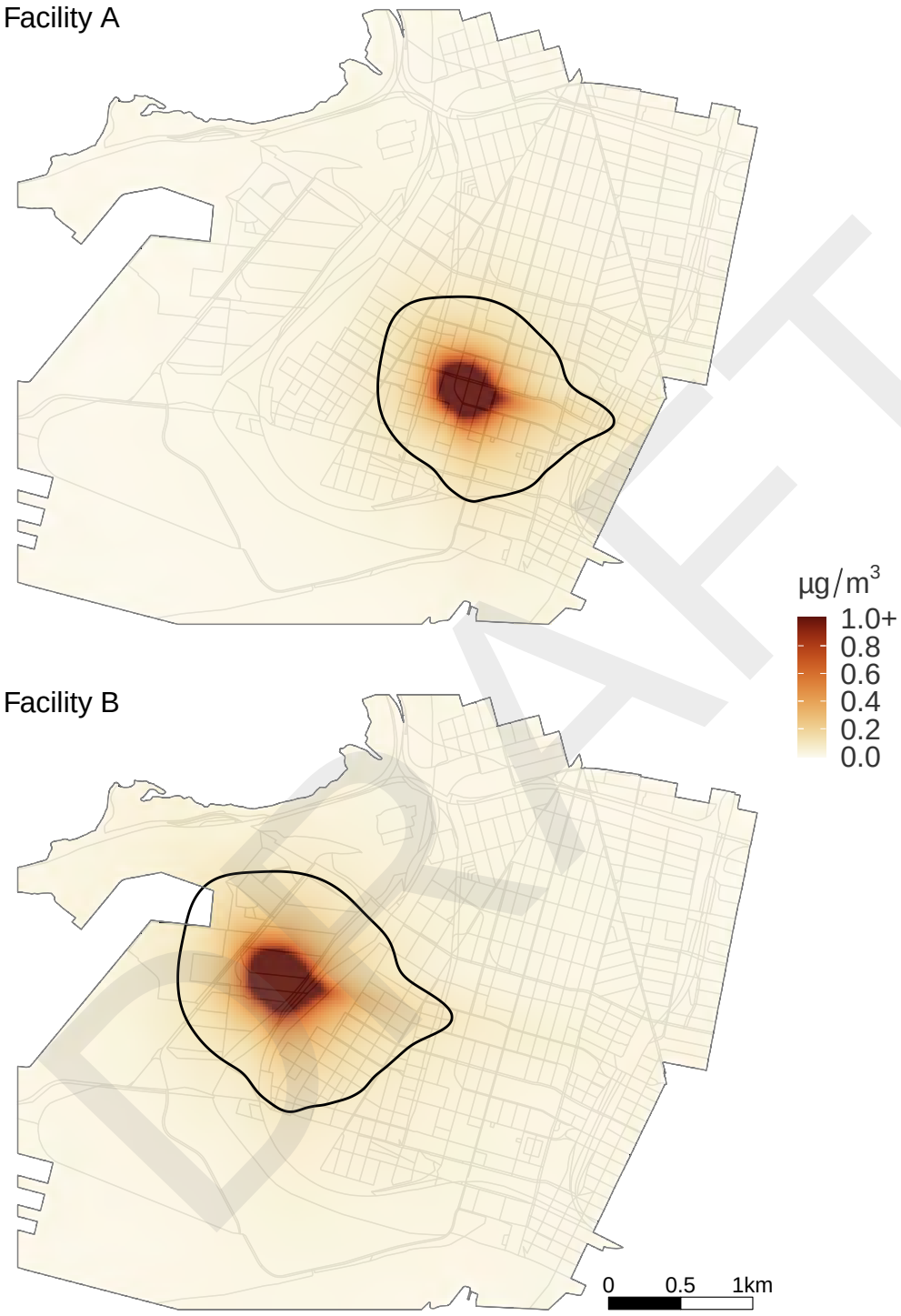



Figure 3.1: Modeled contributions to $PM_{2.5}$ from selected facilities. For each facility, a contour line is drawn at an increment of +0.1 µg/m$^3$. With assumptions (see text), these same contour lines also correspond to an excess relative risk of +0.07%, or a risk difference of +6/M.

## 3.3 Risk

As discussed in Section 2.1, excess risk can first be calculated on a multiplicative scale. This is *relative risk* (RR), which does not depend on baseline conditions. Using an estimate of baseline risk, that relative risk can be converted to risk on an additive scale. This is known as a *risk difference* (RD).

### Relative Risk (Multiplicative)

With the assumptions described in Section 2.2 (i.e., a relative risk of 1.07 per +10 $\mu g/m^3$ $PM_{2.5}$), an increment of +0.1 $\mu g/m^3$ $PM_{2.5}$ corresponds to an increase in the annual risk of mortality, for our hypothetical population, of approximately +0.07%. The same +0.1 $\mu g/m^3$ $PM_{2.5}$ contour line in Figure 3.1 thus also corresponds to that measure of excess risk (+0.07%).

### Risk Difference (Additive)

To convert a relative risk into a risk difference, an estimate of the baseline ($y_0$) is required. This baseline can be taken as constant across the study area, or it can be allowed to vary geographically. In our case studies, we employ the former approach; see Section 4.5.3 for our rationale.

For the Bay Area as a whole, the 2007-2016 crude regional all-cause adult mortality rate obtained from CDC-WONDER is 8,733/M (Table 3.1). In other words, approximately 0.87% of the adult population died each year during that ten-year span. From detailed historical and/or auxiliary data, it can be possible to forecast more accurate present-day or future rates, but we do not attempt that here. (See Section 4.5.3 for discussion.)

Taking that estimate of 8,733/M as serviceable, the +0.1 $\mu g/m^3$ contour line in Figure

Table 3.1: 2007-2016 all-cause adult mortality rates (age $\geq$ 30). Basis: CDC-WONDER.

| Geography | Method | Rate | Analysis | Difference* |
|---|---|---|---|---|
| Bay Area | Crude | 8,896/M | Primary | — |
| Bay Area | Age-adjusted | 8,733/M | Sensitivity | -2% |
| Alameda County | Crude | 8,608/M | Sensitivity | -3% |
| Alameda County | Adjusted | 9,102/M | Sensitivity | +2% |
| West Oakland | Downscaled | 8,982/M | Sensitivity | +1% |

* For risk-difference and mortality metrics, compared to primary analysis.

3.1 corresponds not only to a relative risk increment of +0.07%, but also to a risk difference of +6/M.

Tables 3.3 and 3.4 contain summaries of modeled block-level risk differences, as well as summaries of other metrics of impact, which the reader is encouraged to consider. Using a different baseline mortality rate would affect the overall result in a linear way. See Table 3.1 for examples, and Section 4.7 for discussion.

## Group-Specific Parameters

In Equation (2.3), we can allow the baseline ($y_0$) to vary by subgroup. This results in different calculated risk differences for different subpopulations. Crucially, this is not the same as allowing the effect size ($\beta$) to vary. It can actually have an opposite effect, which we illustrate by example below.

It is well-established that, despite a number of higher-than-average risk factors, Hispanic/Latino populations in the United States consistently exhibit a lower-than-average mortality rate (Ruiz, Steffen, and Smith 2013). This has been termed the "Hispanic mortality advantage," and it holds true within the Bay Area. Taking Alameda County as an example, the 2007-2016 age-adjusted all-cause adult mortality rate for Hispanic individuals was 7,558/M, -17% lower than the county average. Regionally, the rate was -16% lower. (See also Appendix A.)

If we do not allow $\beta$ to vary in Equation (2.3), a lower mortality rate ($y_0$) always yields a lower estimated risk difference. However, recall the data in Table 2.4: for a fixed increment of $PM_{2.5}$, the incremental *relative risk* borne by Hispanic populations is estimated to be larger than average (1.6 times as large, to be exact).

Table 3.2 shows three different results obtained by using this data, allowing (a) the effect size, and/or (b) the baseline rate, to vary while we calculate results for the Hispanic population. For the sake of illustration, this example uses age-adjusted rates for Alameda County.[2]

Table 3.2: Three example calculations performed for a Hispanic population exposed to a $PM_{2.5}$ increment of $+0.1\ \mu g/m^3$. Second row: when using a group-specific baseline, the calculated risk difference is decreased. Third row: when a group-specific effect size is also incorporated, the result is increased, even beyond the original result.

| Effect Size | | Baseline Mortality | | |
|---|---|---|---|---|
| Basis[1] | Relative Risk | Basis[2] | Value | Risk Difference |
| (All) | +7.3% | (All) | 9,102/M | +6/M |
| (All) | +7.3% | Hispanic | 7,558/M | +5/M |
| Hispanic | +11.6% | Hispanic | 7,558/M | +8/M |

[1] Di et al (2017) and the BenMAP-CE User Manual (EPA 2021)

[2] Alameda County, 2007-2016 (age-adjusted all-cause adult, CDC-WONDER)

In the first row of Table 3.2, we rely on a population-wide mortality rate, and on a population-wide estimate of the effect size. We are simply not taking any group-specific information into account. In the second row, we use a baseline mortality rate that is specific to the Hispanic population (7,558/M). Because it is lower than average, this step lowers the calculated risk difference. In the third row, we additionally employ

[2] Age-stratified calculations, such as those that would be performed by BenMAP if this particular set of response functions were selected, would be more difficult to follow. If regional data are used, instead of county-specific data, the results are identical to one significant digit.

an effect-size estimate (RR = 1.116) that is also group-specific. This increases the risk difference, even compared to the first row.

Thus, if we did not take a group-specific effect size into account, but only a group-specific baseline, we would obtain an estimate for impact on Hispanic individuals that was smaller than average. If we took both into account, the estimate would be larger. We note that this does not apply to all groups—only those for which there is an effect size that is both larger and in the opposite direction from the baseline, relative to the average.

## 3.4 Population

Figure 3.2 shows the modeled spatial distribution of the 2018 residential population within the West Oakland area. The definition (i.e., boundary) of the area is taken from the West Oakland Action Plan (BAAQMD and WOEIP 2019). Figure 3.2 also shows, for reference, the same contour lines depicted in Figure 3.1 and discussed in Section 3.2.

Within this area, we calculated the total residential population (all ages) to be $n =$ 32,697 persons. Of these, 61% ($n =$ 20,040) were adults (age $\geq$ 30). For additional breakdowns by age and race/ethnicity, see Appendix A.

## 3.5 Exposure

Figure 3.3 combines the adult population-density data (Figure 3.2) with the modeled $PM_{2.5}$ data (Figure 3.1). The result is a map of population-weighted impacts, which are larger where (a) the concentration of $PM_{2.5}$ is greater, or (b) the concentration of adult residents is greater.

Despite having similar contour lines, the impact of Facility A—as measured by expo-

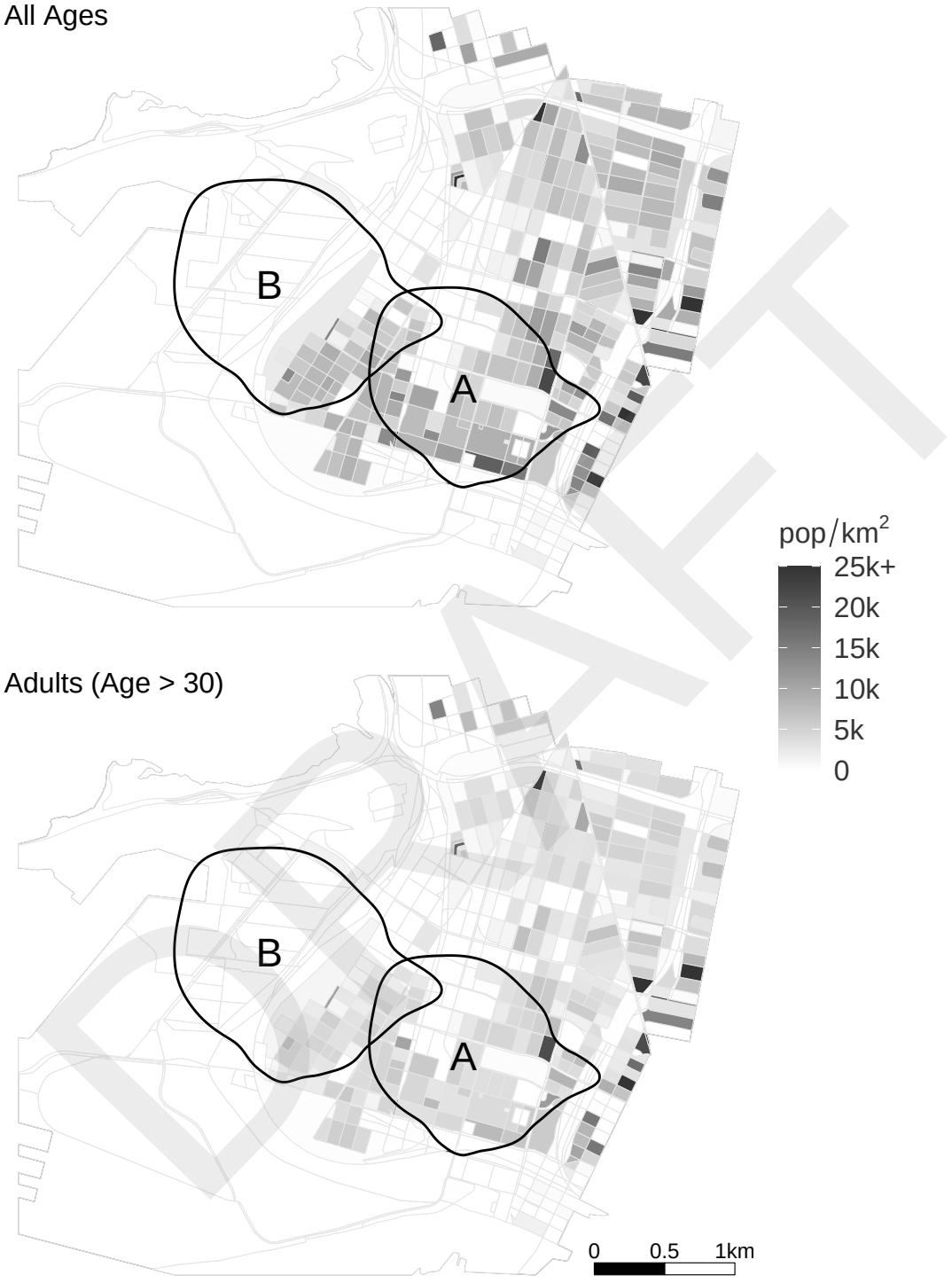



Figure 3.2: Block-level residential population density. For ease of reference, the contour lines from Figure 3.1 are reproduced here. In later calculations of exposure and mortality (Figures 3.3 and 3.4), results are restricted to the adult population (age $\geq$ 30). The all-ages population is shown here for reference.

Table 3.3: Summary of modeled impacts for residential blocks in West Oakland.

| Metric | Facility A | Facility B | Unit |
|---|---|---|---|
| Maximum relative risk at any block | +1.79% | +0.23% | (unitless) |
| Maximum risk difference at any block | +156/M | +20/M | death/person-yr |
| Average risk difference across all blocks | +2.9/M | +2.5/M | death/person-yr |
| Total population exposure across all blocks | +983 | +843 | person-μg/m$^3$ |
| Total excess mortality across all blocks | +0.058 | +0.050 | death/yr |

*Note:*
All metrics reported in this table are restricted to the adult population (age ≥ 30).

Table 3.4: Counts of impacted residents and blocks, split by facility and level of impact. Impact levels are divided into four tiers.

| | Excess Risk | | Facility A | | Facility B | |
|---|---|---|---|---|---|---|
| $PM_{2.5}$ | Relative[1] | Difference[12] | Blocks | Population | Blocks | Population |
| 0 to 0.1 | 0 to 0.07% | 0 to 6/M | 337 | 18,401 | 324 | 18,294 |
| 0.1 to 0.6 | 0.07 to 0.4% | 6 to 40/M | 31 | 1,515 | 48 | 1,746 |
| 0.6 to 1 | 0.4 to 0.7% | 40 to 60/M | 3 | 70 | – | – |
| >1 | >0.7% | >60/M | 1 | 54 | – | – |

*Note:*
All metrics reported in this table are restricted to the adult population (age ≥ 30).
[1] Assuming effect size of 1.07 per +10 μg/m$^3$ $PM_{2.5}$.
[2] Assuming baseline mortality rate of 8,733/M.

sure, so defined—is larger, as it is located in closer proximity to more adult residents.

## 3.6 Excess Mortality

Figure 3.4 shows the block-by-block variation in impacts attributed to each facility, given a baseline mortality rate of 8,733/M. Since we used a spatially invariant baseline mortality rate, the block-by-block variation is solely driven by (a) variation in the modeled $PM_{2.5}$ contributions, and (b) variation in population density, just as in Figure 3.3. As with our risk-difference results, adopting a sub-regional mortality rate would change these results by a small percentage (Table 3.1).

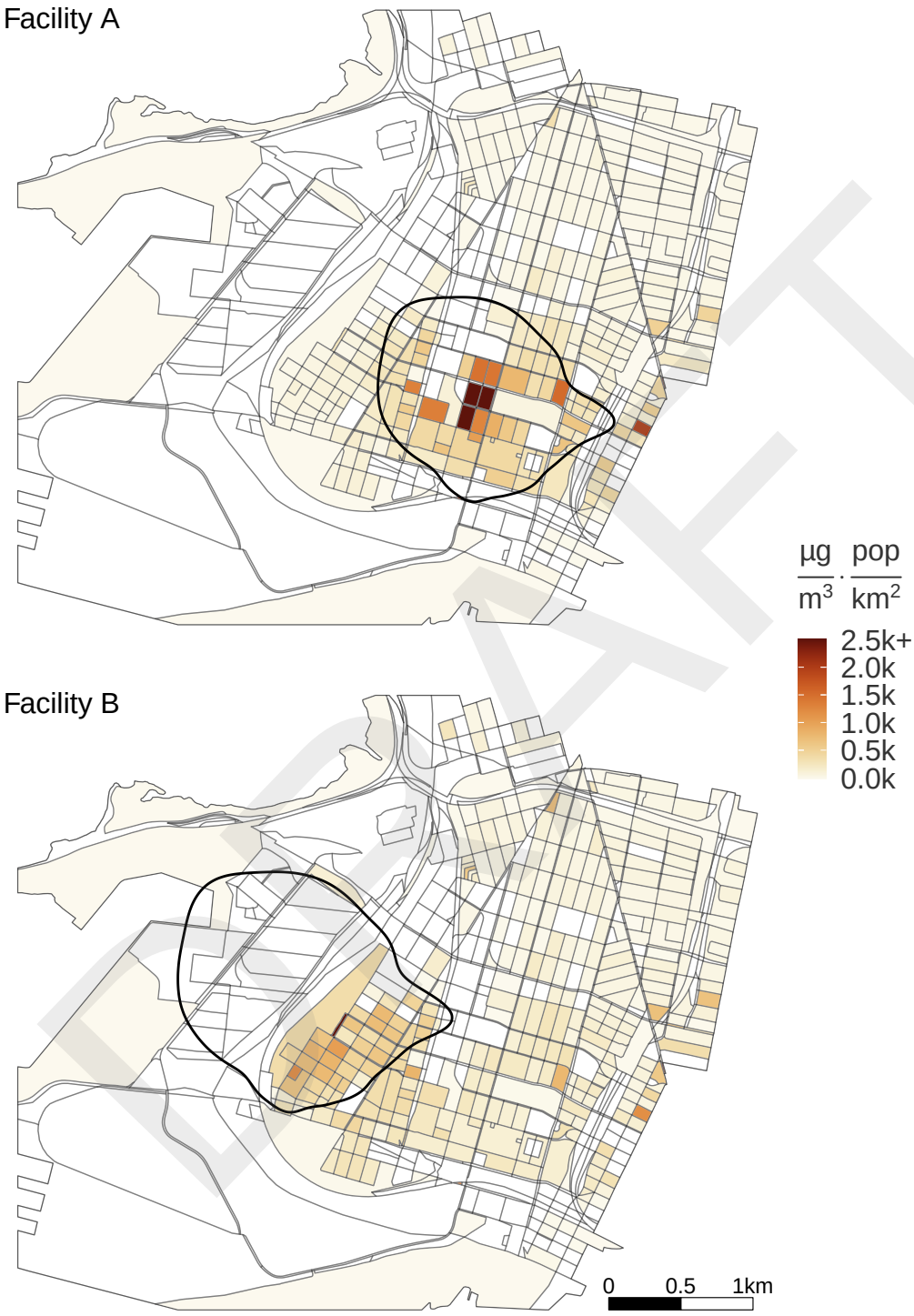



Figure 3.3: Adult exposure, calculated as the product of adult residential population density (pop/km$^2$) and $PM_{2.5}$ concentration (µg/m$^3$).

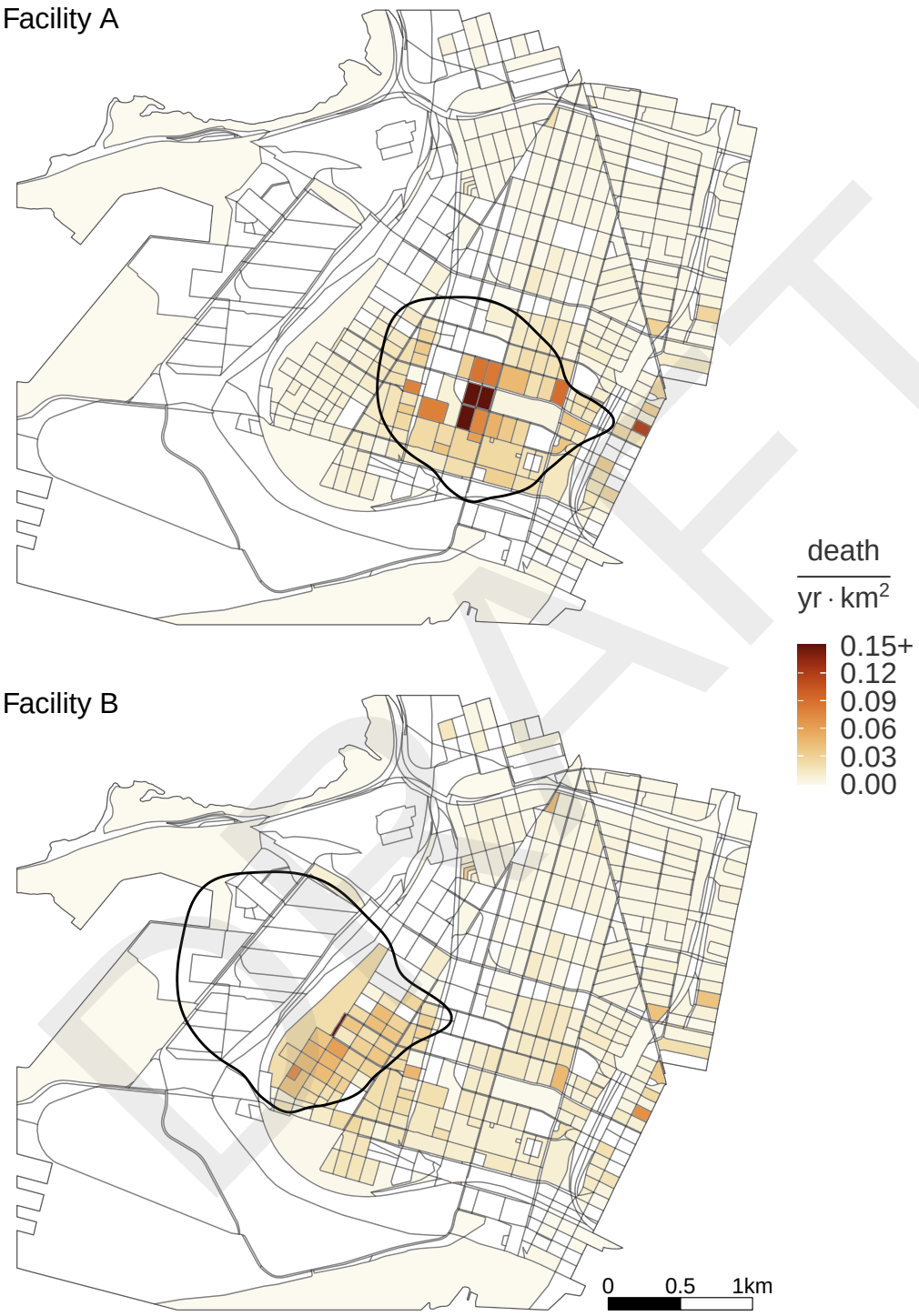



Figure 3.4: Excess mortality, calculated using a constant baseline mortality rate $y_0$ = 8,733/M and an effect size $RR$ = 1.07 per +10 µg/m$^3$ $PM_{2.5}$.

# 4 Discussion

In this section, we discuss the strengths, limitations, and implications of the proposed methodology for the assessment and regulation of impacts from $PM_{2.5}$ emissions at community scale. We also discuss issues related to (a) nonlinearity, (b) baseline mortality rates, and (c) subgroup analyses. We use examples from the results of our case studies to illustrate these issues. Supporting material is provided in the Appendices.

## 4.1 Health Endpoint and Exposure Duration

The EPA's most recent Integrated Science Assessment (US EPA 2019) has linked $PM_{2.5}$ exposure to a broad set of human health outcomes, including respiratory effects, cardiovascular effects, nervous system effects, cancer, and mortality (Table 4.1). Studies consistently find that long-term exposure to $PM_{2.5}$ is associated with increased risk of lung cancer, cardiovascular, respiratory and all-cause mortalities (Pope et al. 2002, 2020; Pope and Dockery 2006; Krewski et al. 2009; Lipsett et al. 2011; Jerrett et al. 2013; Ostro et al. 2010, 2015; Thurston et al. 2016).

Table 4.1: Causality determinations for $PM_{2.5}$, adapted from Table 1-1 of EPA (2019). A "causal" determination reflects the highest degree to which the evidence reduces chance, confounding, and other biases in the exposure-health effect.

| Health Effect Category | Short-Term | Long-Term |
|---|---|---|
| Mortality | Causal | Causal |
| Cardiovascular | Causal | Causal |
| Nervous System | – | Likely to be causal |
| Cancer | – | Likely to be causal |

Mortality is a critical endpoint and, as such, a useful motivation for this methodology. In spite of decades of progress, current $PM_{2.5}$ pollution has been estimated to be responsible for over 30,000 deaths each year in the United States (Bennett et al. 2019). There are also known populations at risk for increased impacts (US EPA 2019, 2021b, 2021a).

The risks of premature mortality induced by long-term exposures and short-term exposures may overlap. The position taken by the US EPA in its 2021 Technical Support Document (TSD) for the Final Revised Cross-State Air Pollution Rule Update for the 2008 Ozone Season NAAQS is as follows:

> "We assume that effects found in studies of long-term exposures may include some effects of short-term exposures. Therefore, only mortality impacts from long-term $PM_{2.5}$ exposure will be quantified, so as not to overestimate impacts. This may potentially bias [...] estimates toward the null in the main benefit estimate." (US EPA 2021a)

We note that our focus on long-term exposures in this whitepaper in no way precludes the consideration of of short-term exposures in future work (Section 4.12).

Apart from mortality, other health endpoints are significant and clearly merit attention. And, the set of impacts considered could affect evaluations or decisions. As an example: cases of impaired lung development will be driven by the presence of younger populations, whose spatial distribution differs from that of the adult population. See Section 4.9 for discussion of multiple metrics and their evaluation.

## 4.2 Comparing Frameworks

Health risk assessments (HRAs) conducted to estimate impacts of toxic air contaminants (TACs) are a mainstay of regulatory activity. Our proposed framework has much in common with the framework for these (Table 2.1), including some limitations.

Examples of limitations common to both frameworks include the accuracy and completeness of: estimates of emissions; modeled transport of emissions; and the pathways, functions, and parameters used to estimate health effects.

Because these shared limitations are widely discussed elsewhere, we focus on other issues here. However, efforts to address some of them are discussed in Future Work.

## 4.3 Pathway Composition

HRAs for TACs are conducted using a framework that breaks apart the *ambient concentration* → *response* pathway into a set of factors (Appendix B) which are then multiplied together. This "bottom-up" approach provides a way to plug in estimates of different factors for specific parts of the pathway, such as the fraction of time at home (FAH). This facilitates the integration of information accumulated from multiple studies, with different scopes and methodologies, over time (US EPA 2005).

$$ambient\ concentration \rightarrow exposure \rightarrow dose \rightarrow response$$

The proposed framework takes a different approach. In the epidemiological studies that it relies on, the total effect size $\beta$ has been estimated directly, using ambient concentrations as the independent variable, and mortality as the dependent variable.[1] Thus, although databases and models of human time-activity patterns (US EPA 2017) might be used in an attempt to decompose $\beta$ into more specific factors, those factors have not been left out. Factors in the TAC → Cancer pathway do include margins of safety, which are not covered by epidemiologically-derived response functions. We discuss margins of safety in Section 4.11.

[1] By way of analogy, $\beta$ can thought of as a composite of all the intermediate factors along the entire pathway, from ambient concentration to response.

## 4.4 Linearity

Conventional response functions for $PM_{2.5} \rightarrow$ Mortality (Eqs (2.2) and (2.3)) are non-linear. This is not unique to mortality; any statistical approach used by epidemiologists to estimate a risk *ratio* for any endpoint will effectively be fitting an equation of the same log-linear form as Eq (2.1).

In Eq (2.3), the term driving the nonlinearity is $\beta\,\Delta x$. The smaller this term is, the more closely Eqs (2.2) and (2.3) will approximate a linear function. In Section 2.2, we characterized the average annual ambient $PM_{2.5}$ in West Oakland as being on the order of ~10 $\mu g/m^3$ (as opposed to 1 $\mu g/m^3$ or 100 $\mu g/m^3$). In our case studies, and in our envisioned applications, a modeled $\Delta x$ will rarely exceed 10 $\mu g/m^3$, even at the most impacted residential blocks (see Table 3.4). Therefore, for any relative effect size that is close to the one used in our case studies, the shape of Eqs (2.2) and (2.3) will be approximately linear over the range with which we are concerned. See Appendix A for figures depicting the magnitude of the non-linearity, given our assumptions, across a policy-relevant range.

The question of whether the true effect of $PM_{2.5}$ on mortality is *exactly* linear, within the range of $PM_{2.5}$ concentrations we are considering, cannot be answered directly. This problem is not unique to $PM_{2.5}$ (May and Bigelow 2005). However, the scientific evidence is consistent with a linear concentration-response relationship within the range, centered on typical ambient concentrations, that we have here characterized as "policy-relevant" (Section 2.2; (US EPA 2019, 2021b, 2021a).)

Adopting a linear approximation of (2.2) and (2.3) would sidestep a number of the issues described below. It would also be more consistent with the TAC $\rightarrow$ Cancer framework, which does assume linearity. However, exactly how to linearize—and how to linearize group-specific effect sizes, which have larger estimated exponents for at-risk populations—would be an important question. Another important question

would be how to reconcile differences with established tools and frameworks (e.g., BenMAP) that rely on log-linear functions for calculating mortality.

## 4.5 Dependence on Population Characteristics

Some of the metrics we have calculated require data on population characteristics, while others do not. We divide these characteristics into two kinds, according to their relevant properties for the selection of appropriate metric(s) by the risk assessor: first, the spatial distribution, extent, and composition of the local population; and second, its baseline incidence rate(s).[2]

Contributions to ambient $PM_{2.5}$ concentrations, as we have modeled them, are independent of the local population. The population-average excess *relative risk* can also be calculated independently of the local population density, assuming a constant effect size, as in the results presented in Section 3.3.[3] In contrast, exposure and burden are dependent (by definition) on population counts and their spatial distributions.

### 4.5.1 Risk Differences *vs* Relative Risks

If this approach were to be embedded in a decision tool, a key metric—alone, or one of several—could be based on thresholds in relative risk(s), rather than risk differences(s). As an example: rather than drawing a line at a a *risk difference* for premature mortality of +6/M, a line could instead be drawn at a *relative risk* of +0.07%. As demonstrated in the Results, with our assumptions, those two contours are essentially the same (to one digit of precision).

[2]By composition we primarily mean demographics (age, sex, race/ethnicity, etc.). In this whitepaper, we are concerned only with the baseline incidence of mortality, but future work (Section 4.12) could consider the extent to which our considerations apply to baseline rates for additional endpoints.

[3]All else being equal, we can expect that a relative-risk estimate will be more accurate insofar as the composition of the population matches the cohort(s) that formed the basis of effect-size estimates.

An advantage of setting thresholds in terms of relative risk is that this would remove the dependence on baseline conditions. Relative risk contours for specific at-risk populations could be evaluated independently and, if desired, the most health-protective could be evaluated on its own. This would be functionally equivalent to adding a margin of safety to the population average (see Section 4.11). However, to combine group-specific estimates, and arrive at an estimate for the population overall, one must still weight intermediate results by population. At that point, one is effectively calculating a risk difference.[4]

In this $PM_{2.5} \rightarrow$ Mortality framework, maps of risk differences can be compromised to the degree that they depend on outdated or otherwise inaccurate data on population characteristics. Some potential disadvantages of relying on impact metrics that depend on such data are listed and discussed below.

First, reliance on population data brings up the same "when-to-update" challenge that any reliance on baseline conditions does (Section 4.5.3). Extrapolations from Decennial base years may miss the net effects of migration, as well as urban or rural development. For example, the construction of a new housing project will not be captured in assessments of exposure or burden based on such an extrapolation. Predictive errors such as these are discussed further in Section 4.5.2.

Second, at smaller spatial scales, residential surveys become less reliable predictors of out-of-sample and post-survey populations. This issue is exacerbated when the sample size is smaller relative to the target population (as with the American Community Survey, which is a primary source of population estimates for inter-decennial years, and especially for years just preceding the Decennial Census.)

Third, in a large-scale survey like the Census, hard-to-reach populations may be undercounted to an extent that compromises local risk assessment.

Fourth, while the basis for most conventional population-density estimates (and the

[4]Technically, one is perfoming all of the calculations needed to generate burden or exposure estimates as well.

basis for most health-impact functions derived from epidemiological studies) is the Census, it is worth noting that a modeled spatial distribution based on residential locations can be quite different than the actual population distribution during the day (consider, for example: schools, workplaces, and commercial districts).

Finally, Census data are intentionally imperfect measures of residential density: some noise is deliberately added to protect individuals from identification.

### 4.5.2 Prospective Risk Management *vs* Retrospective Impact Assessment

The objective of risk management is to intervene on a potential future, rather than to describe an actual past. Accountability studies conducted using BenMAP may be similar in appearance to the case studies we have conducted, but they are generally concerned with describing actual pasts. Such retrospective assessments can include statistical uncertainties, but they do not include predictive uncertainties.

Like sampling error, predictive error is exacerbated at small spatial scales. It can be mitigated by more-frequent updates, but in practice, the frequency of updating may be limited by the factors described in the preceding section. We recommend that risk assessors establish or consider an appropriate tolerance for predictive error when selecting between metrics and statistics for use within a prospective risk-management process (see sections 4.6 and 4.7).

### 4.5.3 Baseline Rates

In developing this whitepaper, we also considered limitations of baseline mortality-rate estimates. Many of these are shared with the limitations of population estimates listed above, so we do not repeat them here. However, we note that the magnitudes of these limitations are greater, since mortality rates involve smaller counts. In ad-

dition, rates are ratios, meaning that they also propagate the uncertainties of the denominator (i.e., population counts).

Dependence on baseline rates challenges a fundamental assumption common to many risk-assessment frameworks, including the TAC → Cancer framework. This assumption is that the same modeled concentrations would have the same impact on any population.[5] In our proposed framework ($PM_{2.5}$ → mortality), the risk difference *depends on*, and is a multiple of, the existing mortality rate. The form of this model dictates that the estimated risk difference for a population with higher baseline mortality rates will be greater than for an average population. Besides age, many factors are known to be associated with higher mortality rates (sex, poverty, racism, education, healthcare access, etc.); some of these factors can vary considerably over small spatial scales and over time.

As with population-count data, the question also arises of how often—or under what circumstances—to update estimates. This can pose logistical challenges if the need for updates exceeds the capacity of the risk assessor(s) and manager(s) to generate and process risk-assessment products. Since risk assessments are part of a larger social process that unfolds over time, updates can also create challenges in comparing, standardizing, and settling agreement among products.

When risk-driving factors are under the control or jurisdiction of permit-issuing agencies, it has typically been the practice that risk assessments are only updated when those factors change (e.g. in response to a change in emissions); such changes are foreseeable, and can be managed accordingly. Generally speaking, mortality rates in developed countries are declining over time, but they are not under the direct control of any regulatory agency. The downward trend has short-term fluctuations, which are reasonably dampened and/or foreseeable at regional, state, and national scales. County and city rates may be affected on shorter timescales by demographic trends

[5] Age-specific sensitivity factors have been added to OEHHA's guidelines for cancer risk assessment. However, if we think of the resulting risk as being estimated for a statistical person, that person always has the same exposure window.

(e.g. migration and aging). However, unusual events may cause even state-level or national rates to rise temporarily.

We currently recommend against constructing and using "hyper-local" baseline rate estimates (as small as Census blocks) to generate mortality-risk estimates at the same hyper-local scale. This is mathematically possible, but it introduces considerable statistical and predictive uncertainty, to a degree that we consider inadvisable for prospective local risk management.

Further discussion, examples, and illustrations concerning mortality rates may be found in Appendix A.

## 4.6 Selection of Statistic(s)

Maxima exhibit higher variance than sums or averages, which means that all of the above sources of uncertainty (including predictive error) will be exacerbated if the risk-assessment method overall focuses on a predicted maximum impact, rather than an area-wide impact.

Area-wide statistics—such as the burden, exposure, and average risk differences reported in Table 3.3—require that the extent of the area be defined. In our case studies, we adopted an area that had been previously defined. If this option were not available, the risk assessor could construct an area defined in terms of the impact itself—for example, the set of Census blocks for which the predicted $PM_{2.5}$ increment exceeded some threshold. The risk assessor could also construct an area, starting from the location of the source, that proceeded outward until some other limit were reached—for example, until the number of residents in the area reached some predetermined count, or until the total population exposure reached some amount.

Our present position is that the modeled metric that is most acceptable depends on whether a larger risk-assessment framework is designed to operate on the basis of

(a) maxima and/or (b) area-wide summaries (for some definition of the local area), as well as the tolerance (of the risk assessors) of different types and magnitudes of predictive error, which are grounded in the nature of population-dependent estimates (counts and rates). In our future work (Section 4.12), we aim to continue to resolve these tradeoffs, and to work with risk assessors and additional case studies to ensure feasibility and fitness-for-purpose.

## 4.7 Selection of Metric(s)

If population-dependence is acceptable, and if risk differences are a preferred metric, then for risk-management purposes attuned to individual facilities, we currently recommend the use of a baseline rate at a spatial scale that covers at least $1 \times 10^5$ people. This is larger than the scale of our case studies, and considerably mitigates the compromises enumerated above.

Above, we recommended against using hyper-local (e.g., block-level) estimates of baseline risk to generate and report results—whether single numbers, tables, or maps—at a hyper-local scale. In particular, we are not persuaded that mortality-rate estimates at a hyper-local scale are stable or reliable enough to adequately support the decisions that we would expect to follow. However, if hyper-local calculations were to be re-aggregated to a larger extent before reporting (as with the community-wide total excess mortality reported in Table 3.3), the uncertainty would be considerably mitigated. A risk-management protocol based on such an area-wide measure could be adequately robust, and would meet calls by scientists to integrate the consideration of exposure into modern risk-management protocols. It is, however, reasonable to anticipate calls for "the underlying data," and the subsequent use of those fine-scale intermediate calculations in ways that we expect would lead to inadvisable inferences and/or decisions. See Section 4.7 for additional discussion of area-wide statistics versus local maxima, and Section 4.12 concerning integration and uptake by communities

of practice.

For the purposes of the entity conducting risk assessments, and the other stakeholders in those assessments, it may be most appropriate to use a community-specific rate, a county-specific rate, or a regional rate. We note that, compared to other parameters in the implementation, sensitivity to this choice (i.e., regional *vs* county *vs* community-level) appears relatively low: the values in Table 3.1 are all within ±3% of each other. For calculating risk differences and mortality burdens, we have used a regional rate. An advantage of using a regional rate is that no discontinuities will be generated at county lines. (See Figure A.1 in Appendix A for a comparison of county-specific rates.)

In calculating exposures and burdens, we have assumed that block-level variation in residential population is a satisfactory predictor of variations in exposure. As discussed in the preceding sections, this approach has limitations and known deficiencies. There may be options available to mitigate some of these deficiencies, which we regard as potential future work (Section 4.12). However, an exposure or burden metric does reveal a meaningful difference between the two facilities in our case study: although their $PM_{2.5}$ contours are quite similar, the different siting of the facilities (relative to the residential population) creates a significant difference in the exposures and burdens attributed to each (Table 3.3; Figure 3.3; Figure 3.4).

## 4.8 Commensurability

To put "+10/M" (as in the TAC -> Cancer framework) and "+10/M" (as in this PM25 -> Mortality framework) on the same scale, at least four adjustments could conceivably be attempted. Making such adjustments is outside the scope of the current work, but we list them here for the sake of discussion.

- **Margin of Safety.** A "+10/M" cancer risk value is typically derived from

published slope factors or unit risk factors that incorporate margins of safety. For some pollutants, these margins of safety may represent factors of 3, 10, or more. See Section 4.11 for further discussion.

- **Exposure window and time at risk.** We estimated changes in *annual* mortality rates for adults 30+ years of age. In the TAC → Cancer framework, the exposure window is the third trimester through 30 years of age, and the time at risk is 70 years (Table 2.1), leading to an estimate of "lifetime" cancer risk. Future epidemiological studies might support estimates of impacts on mortality rates for younger adults and children.[6]

- **Maximum point of impact vs weighted average.** In the HRA process that the District follows, which the TAC -> Cancer framework supports, the cancer-risk metric is calculated for a "maximum exposed individual" (MEI) receptor, typically the closest residential location outside the facility boundary. We generated results averaged across Census blocks, which can be aggregated at any level to produce area-weighted or population-weighted estimates of impact. We did not attempt to identify a maximally exposed individual (MEI) in terms of a single receptor location. It is practically certain, however, that the estimated MEI impact within the 16 blocks subject to "over +10/M" impacts from Facility A would be greater than +10/M.[7]

- **Valuation.** Using conventional metrics of loss, cost, and/or preference, mortality is typically weighted more heavily than cancer. BenMAP-CE provides a library of conventional valuation functions for mortality. Such valuations can be problematic, however. See Section 4.9 for a brief discussion.

[6]There are studies available to support infant-mortality impact estimates, which we did not leverage. In the United States, typically, the population baseline mortality and hence most of the calculated risk difference accrues at ages 30 and up.

[7]The likelihood that 665 residents all live on the downwind side (of their block centroids) is, intuitively, very small.

## 4.9 Managing Multi-Dimensional Impacts

It is worth noting that TAC impacts are assessed and regulated not only in terms of cancer risk, but on the basis of two other metrics as well. These are *chronic hazard* and *acute hazard*, both of which are evaluated using a different metric: the hazard index, or HI (Strum, Eyth, and Vukovich 2017).

Thus, for several decades, there have actually been three metrics in use for regulation of impacts from TAC-emitting sources at local scale. A particular source triggers concern and/or action when any one of these metrics reaches its respective threshold. The methodology we propose here for assessing impacts from $PM_{2.5}$ could similarly support mechanisms that would trigger concern and action.

The logical-composition method described above ($Y_1 > A \mid Y_2 > \mathrm{B} \mid Y_3 > C$) is also applied in other contexts, such as NAAQS attainment, or the identification of over-burdened communities. Other approaches to dimensionality reduction are possible. For example, it is possible to normalize scores and then combine them: this is the approach taken to assemble the Hazard Index itself, which is actually a sum of ratios. The Healthy Places Index (Maizlish et al. 2019) is also a sum, but of z-scored transformed data. The CalEnviroScreen tool uses both sums and products of rank-transformed data. Valuation on a currency-based scale is another approach. US EPA typically provides such valuations, although the appropriateness of combining or comparing willingness-to-pay (WTP) and cost-based valuations is debated, and willingness-to-pay is subjective in ways that may differ for at-risk populations.

Many other variations are possible, and some methods may be more desirable in a particular context. To the extent that the simultaneous reduction of multiple health endpoints—rather than just mortality—is a goal, the methods above offer some possibilities.

Another possibility is to manage risk on the basis of modeled exposure, without

attempting to model health endpoints or pathways directly. Exposure requires information on population density, but it does not require an estimate of effect size, nor an estimate of baseline conditions, nor the selection of a set of endpoints. Exposure can also be calculated for all age groups, whereas we have had to restrict mortality estimates to the population aged $\geq 30$. (Approximately 39% of the modeled population of West Oakland is younger than age 30.) Modeled exposure could be thought of as offering more general coverage of impacts—compared to a metric based on a single health endpoint or a finite set of endpoints—but in a manner that is not as precisely articulated, nor weighted toward any particular endpoint or sub-population (although exposures can be assessed for specific groups).

Here we have considered only a metric of long-term exposure. If it were deemed appropriate to consider a shorter-term exposure metric as well, it could be combined with the long-term exposure metric via logical composition, as above.

## 4.10 Representativeness

The class of facilities we are interested in characterizing (via these case studies) is comprised of those that are both (a) sited generally upwind of, and close to, residential populations, and (b) emitting $PM_{2.5}$ at rates from one to three orders of magnitude below that of the largest emitters (Table 4.2). The facilities we simulated are a convenience sample, in that they were selected from modeling that had already been conducted (BAAQMD and WOEIP 2019). We believe they are reasonable examples of the *emission rates* and *exposure factors* (Bennett et al. 2002; Roumasset and Smith 1990) encountered among this class of facilities.

**Emission rates.** As modeled, the emission rates of the facilities in our case studies (Table 2.2) fall within the first two rows of Table 4.2. We can quantify the sensitivity of our results to uncertainties in $PM_{2.5}$ emissions; it is very close to linear (Appendix C). So, if the true emissions were actually three times larger—holding the siting and

meteorology constant—then the impacts would be approximately three times larger as well. Likewise, if the emissions were three times smaller, so would the impacts. A three-fold adjustment in either direction would still place these facilities within the class that we are focused on here (Table 4.2).

Table 4.2: Count of Bay Area facilities by magnitude of inventoried $PM_{2.5}$ emissions, circa 2016. Fewer than ten facilities in BAAQMD's jurisdiction emit more than 100 ton/yr $PM_{2.5}$. Several hundred facilities emit $PM_{2.5}$ at rates that are one to three orders of magnitude lower (0.1 to 10 ton/yr).

| $PM_{2.5}$ Emission Rate | Facilities ($n$) |
|---:|---:|
| 10 to 100 ton/yr | 43 |
| 1 to 10 ton/yr | 146 |
| 0.1 to 1 ton/yr | 241 |

**Exposure factors.** In combination with local meteorology, siting arrangements drive the *exposure factors*, or the impact per ton of emissions from a given source (Bennett et al. 2002; Roumasset and Smith 1990). Siting and meteorology involve many parameters that are difficult to simulate convincingly in the abstract. Until more data are available to characterize this part of the exposure pathway for a broad class of facilities, convenience sampling from available results is our best approximation strategy.

## 4.11 Margins of Safety

Incorporating margins of safety within risk assessments is a well-established principle and practice (NRC 2009; US EPA 2005). However, health-protective margins of safety are not built in to the approach described thus far.

Published estimates of the effect ($\beta$) of $PM_{2.5}$ on mortality express some uncertainty in the form of statistical confidence intervals (CIs). This uncertainty can be carried

forward into risk evaluations, but it is a subset of total uncertainty. The effects of exposure-assignment error, model selection error, and predictive error may also be of interest, especially when the intent is to estimate an upper bound or quantile. For the sake of illustration within this document, we focus on the case where the central estimate of ($\beta$) alone is evaluated, but a margin of safety may be desirable in risk management contexts.

In cancer-risk assessments, margins of safety are found within *slope* or *unit risk* factors (NRC 2009; US EPA 2005). Margins of safety address many different dimensions of uncertainty and vulnerability.[8] Where effect sizes have been estimated from toxicological studies, for example, safety factors may be introduced to account for animal-to-human extrapolations. In assessments based on human studies, safety factors may account for (a) other uncertainties (e.g. in exposure-dose and dose-response relationships), and for (b) vulnerabilities among the exposed population (stemming from genetics, predisposing exposures, physiology, lifestage, and/or other factors). An excerpt from *Science and Decisions* (NRC 2009) is illustrative:

> Consideration of the most exposed receptors (individuals) is accomplished by estimating chronic exposures at the Census block level … [while] consideration of sensitive subpopulations is considered in so far as it is explicitly built into the dose-response metrics that EPA uses to estimate risk (i.e., where data supporting such distinctions are available). Unit risk estimates typically incorporate protective low-dose extrapolation assumptions and are based on statistical upper confidence limits. (NRC 2009)

The effect size ($\beta$) we have discussed was estimated directly for humans (as opposed to animal species), but it is a population-average effect. Characterizing effect modification for subgroups can require epidemiological studies of very large size, which

[8] Some "upstream" uncertainties are typically excluded from margins of safety: for example, uncertainties in emission rates.

then necessitates tradeoffs in terms of specificity—geographic, demographic, or otherwise. Di et al. (2017) relied on a Medicare population of over 60 million people, larger than the current total population of the state of California, to estimate the effect sizes for subgroups listed in Table 2.4. For the purposes of health-protective decisionmaking, adopting an appropriate margin of safety could help to protect overburdened and/or vulnerable groups without requiring infeasible or impossible estimates of group-specific effects.

Typically, an overall margin of safety is composed of more than one factor. Usually these factors are not precisely estimable; multiples of 3 or 10 are common. The different dimensions of uncertainty and/or vulnerability captured by these factors may be independent, synergistic, or associated in positive or negative ways, but the ultimate goal is for the relevant set of safety factors to be adequately protective when multiplied together.

A margin of safety could also potentially account for uncertainty in the composition, and therefore the toxicity, of modeled $PM_{2.5}$ emissions.

## 4.12 Future Work

Future work will generally have two aims: (1) improving the inputs and methods; and (2) expanding the scope of the work.

### Improving Inputs

**$PM_{2.5}$ Emissions.** For facilities that may impose higher health risks in a local-scale $PM_{2.5}$ risk assessment, BAAQMD will work to reduce the uncertainty in the emissions estimates. Larger sources tend to have the benefit of direct testing, which can help to improve the precision and accuracy of $PM_{2.5}$ emission estimates. Fugitive sources of $PM_{2.5}$ are more difficult to quantify with comparable certainty. If this

methodology were to lead to additional facility-level requirements, then it is possible that requirements for demonstrating compliance, such as stack testing, could be used in a systematic way to constrain some of the relevant uncertainties.[9] A general discussion of uncertainties in inventoried $PM_{2.5}$ emission estimates is outside the scope of this report.[10]

**Emissions → Concentrations.** Non-steady-state models, such as CALPUFF and SCICHEM, are being explored by BAAQMD staff. Comparing results from different models may help to better understand the uncertainty associated with simulated ambient $PM_{2.5}$ concentrations.

**Baseline mortality.** Collaboration between BAAQMD and OEHHA may improve estimates of baseline mortality rates ($\Delta y_0$). We may also evaluate newly available tools for producing smoothed and/or age-adjusted small-area estimates (Quick et al. 2019). As illustrated in Appendix A, overall regional and county-specific estimates will probably not vary by more than $\pm 50\%$.[11] This may be smaller in magnitude than the uncertainty in our modeled estimates of $\Delta PM_{2.5}$, at least for the class of facilities considered in our case studies.

## Expanding Scope

**Health endpoints.** This whitepaper has focused on all-cause adult mortality. Other outcomes are associated with both long- and short-term exposures to $PM_{2.5}$, and some of these outcomes have been studied in younger populations (US EPA 2019). There are ongoing efforts between the Air District and OEHHA to develop estimates of effect sizes for other outcomes that might be estimated for communities similar to the one in our case studies.

[9] See https://www.epa.gov/sites/production/files/2013-09/documents/cmspolicy.pdf.

[10] For an overview, see NRC (2009), p. 114.

[11] Within a given county, baseline mortality rates for different race/ethnicities may vary by a factor of two or more.

**Case studies.** We limited our case studies to stationary sources in West Oakland, but there is no technical reason why the methodology could not also be applied to mobile sources, nor to sources in other communities. For the West Oakland Action Plan (BAAQMD and WOEIP 2019), AERMOD was also applied to generate 20×20m estimates of $\Delta PM_{2.5}$ from mobile sources in and around West Oakland. The resulting data were archived and are readily available. Results for stationary and non-stationary sources in another community, Richmond-North Richmond-San Pablo, are also being developed in parallel with this whitepaper, and could potentially be used to improve and expand our set of case studies.

**Cumulative risks.** In principle, the methodology described and illustrated here can be applied to any number of sources. As mentioned above, for the West Oakland Action Plan (BAAQMD and WOEIP 2019), AERMOD was also applied to generate 20×20m estimates of $\Delta PM_{2.5}$ for many non-stationary sources. We plan to conduct future case studies that will incorporate more of this data at the same time.

**Uncertainties.** In future work, we intend to further characterize the magnitude and form of the predictive and statistical uncertainties described above. We plan to use a combination of case studies based on historical data (e.g., 2010 vs 2020 populations), simulations, and (if possible) consultation with demographers and/or statisticians with relevant expertise. This will support additional future work aimed at improving integration with risk management practices.

**Integration with risk assessment and management.** Our work to date has been scoped to the development of model-based products that offer spatially-resolved predictions. These products are intended to be nested within a risk-management protocol, which itself is nested within larger social processes. The refinement of such a protocol will be supported by consultations with risk assessors and risk managers.

# A Baseline Mortality

This appendix illustrates and explains several issues related to the selection, use, and reporting of baseline mortality rates in the Bay Area, with an emphasis on estimates for specific racial/ethnic populations.

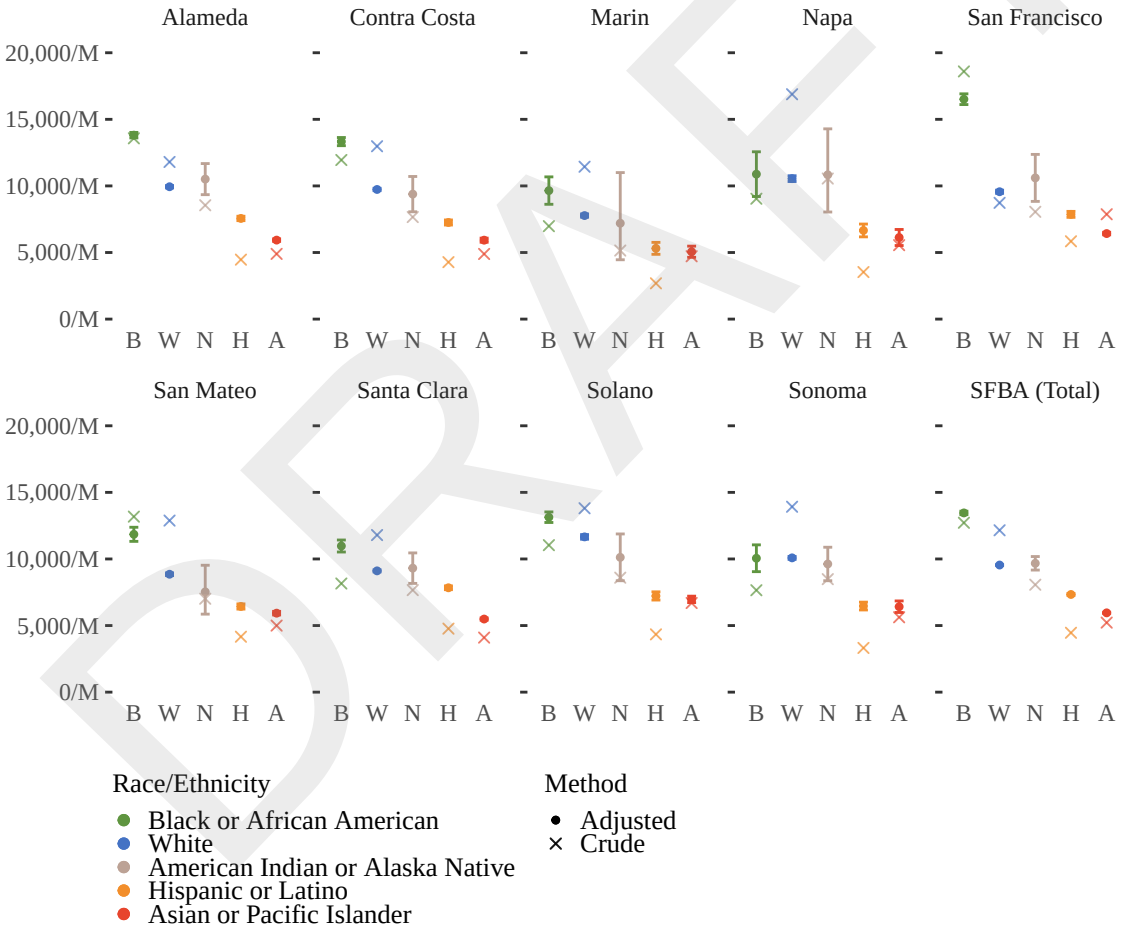



Figure A.1: Adult (age 25+) all-cause mortality, 2007-2016. Error bars are 95% confidence intervals. When the crude rate (×) is higher than the age-adjusted rate (•), the population is older. Adjusting for age brings other factors, like race/ethnicity, into sharper focus.

Figure A.1 depicts 2007-2016 (ten-year average) adult[1] mortality rate data from the Wide-ranging Online Data for Epidemiologic Research (WONDER) database operated by the U.S. Centers for Disease Control and Prevention (CDC). Tables A.1 and A.2 show the same data. The CDC's WONDER database is a primary source of mortality data for many applications, analyses, and tools, including BenMAP-CE.

## Small Counts

In Figure A.1, the error bars vary in size.[2] When the error bars for two groups overlap, as they do for Black adults and White Adults in Sonoma County, a general rule is to avoid drawing the conclusion that the two rates are different, and rather to conclude that there is simply not enough evidence to tell either way. The error bars are largest for racial/ethnic subgroups that are smaller relative to others in the Bay Area (like Native American adults), especially in counties with fewer people in total (like Marin County).[3] They are smallest for the Bay Area as a whole (bottom right panel). At a regional scale, there are simply many more people, and many more events (i.e., deaths) to observe. CDC-WONDER does not provide estimates for geographic areas smaller than counties, but any source or calculation method that yields such estimates would—or should—naturally frame them with larger error bars.

A second thing to note is that these are ten-year averages, based on the most recent data available. We could obtain five-year averages for 2012-2016 instead, three-year averages for 2014-2016, or even one-year averages for 2016. Since mortality rates change over time, these would perhaps be better estimates of current rates. However, they would also be based on increasingly smaller counts of observed deaths, and thus

[1] "Adult" here means the `25 to 34` age bracket and above.

[2] For the sake of legibility, error bars are shown only for age-adjusted rates.

[3] Among Native American adults in Marin County, from 2007 to 2016 there were a total of 28 deaths, or an average of approximately 3 per year. The average size of the adult population in any given year was 544, according to same data source (CDC-WONDER). The corresponding crude rate estimate, over that ten-year period, would then be $28 \div 5{,}440 \approx 5{,}147$/M with a 95% confidence interval of (3,420/M–7,439/M).

the error bars would grow larger.

## Coding Race/Ethnicity

CDC-WONDER data, like Census data, can be downloaded with race and ethnicity split in several ways. "Hispanic or Latino Origin" is coded as true or false. "Race" is independently coded. Often, "Race" and "Hispanic or Latino Origin" (hereafter, "Hispanic") are consolidated into a single variable, with one category for "Hispanic" (of every race combined), and several more categories for non-Hispanic persons, segmented by race. (Labels for these categories do not always include an explicit "non-Hispanic" qualifier, as it is usually assumed/implied by the existence of a "Hispanic" category). This is the approach taken in Figure A.1 and the accompanying tables.

We cannot here report data separately for populations that self-identified in the Census as multi-racial. The CDC applies a technique called *race bridging* (Ingram 2007) to WONDER data, which essentially re-distributes multi-racial populations into single-race categories. This is "to make multiple-race and single-race data collection systems sufficiently comparable to permit estimation and analysis of race-specific statistics."[4]

BenMAP-CE, although it relies on the same underlying CDC-WONDER data, will only export data grouped into two categories: `WHITE` and `Non-WHITE`. These are coded by BenMAP strictly according to race, and not ethnicity, meaning that `WHITE` includes white Hispanic adults (which, in California, constitute over 90% of the Hispanic adult population). In this Appendix, we use the term `WHITE` (all caps) when referring to BenMAP's categorization, to help avoid confusion with "White" as understood in many other contexts.

[4]https://www.cdc.gov/nchs/nvss/bridged_race.htm

Table A.1: Age-adjusted adult all-cause mortality rates in the Bay Area (2007-2016), by county and race/ethnicity.

| County | Black | White | NatAmer | Hispanic | Asian |
|---|---|---|---|---|---|
| Alameda | 13,812/M | 9,938/M | 10,511/M | 7,558/M | 5,931/M |
| Contra Costa | 13,328/M | 9,733/M | 9,384/M | 7,254/M | 5,927/M |
| Marin | 9,649/M | 7,774/M | 7,197/M | 5,311/M | 5,058/M |
| Napa | 10,884/M | 10,547/M | 10,835/M | 6,656/M | 6,128/M |
| San Francisco | 16,511/M | 9,563/M | 10,602/M | 7,865/M | 6,423/M |
| San Mateo | 11,851/M | 8,853/M | 7,528/M | 6,432/M | 5,930/M |
| Santa Clara | 10,973/M | 9,107/M | 9,310/M | 7,836/M | 5,488/M |
| Solano | 13,135/M | 11,658/M | 10,122/M | 7,223/M | 6,966/M |
| Sonoma | 10,053/M | 10,083/M | 9,623/M | 6,464/M | 6,414/M |

# Age-Adjustment

Two kinds of estimates are shown in Figure A.1: *crude* rates and *age-adjusted* rates. Crude rates are simply the actual, unadjusted data: deaths divided by population. Age-adjustment answers the question "if this population had the same age distribution as a reference population, what would its mortality rate look like?"[5]

Crude rates are less often reported by public-health agencies. This does not mean that the crude rates are wrong or in need of correction. Age is not something that can be changed, so in many epidemiological contexts, age is not a factor of primary interest. However, it is a dominant predictor of mortality risk. The effects of other factors, which are of interest, may appear to be washed out (or artificially enhanced) when they are correlated with age. So, adjusting for age usually helps to bring them into focus.[6] This is a critical tool in public health practice, both for highlighting disparities and for identifying factors on which we can intervene. For estimating actual mortalities, however, crude rates can be a more appropriate tool.

[5]CDC WONDER's default reference population is the 2000 U.S. Census.

[6]Old age is associated with declining health; it is also generally associated with lower income, which we are not adjusting for here. So, even age-adjustment does not always tell the whole story.

Table A.2: Crude adult all-cause mortality rates in the Bay Area (2007-2016), by county and race/ethnicity.

| County | Black | White | NatAmer | Hispanic | Asian |
|---|---|---|---|---|---|
| Alameda | 13,574/M | 11,800/M | 8,548/M | 4,464/M | 4,896/M |
| Contra Costa | 11,945/M | 12,979/M | 7,662/M | 4,277/M | 4,890/M |
| Marin | 6,978/M | 11,441/M | 5,147/M | 2,687/M | 4,717/M |
| Napa | 9,040/M | 16,883/M | 10,553/M | 3,531/M | 5,551/M |
| San Francisco | 18,596/M | 8,729/M | 8,057/M | 5,842/M | 7,872/M |
| San Mateo | 13,180/M | 12,883/M | 7,015/M | 4,162/M | 4,998/M |
| Santa Clara | 8,157/M | 11,795/M | 7,663/M | 4,769/M | 4,096/M |
| Solano | 11,036/M | 13,808/M | 8,596/M | 4,337/M | 6,693/M |
| Sonoma | 7,652/M | 13,921/M | 8,484/M | 3,319/M | 5,621/M |

Age-adjustment can cause apparent relationships to reverse. For example, in Contra Costa County (Figure A.1, Table A.2), the crude rate (x) among Black adults is lower than among White adults. However, adjusting for age reverses the relationship: the age-adjusted rate ( • ) among Black adults is higher than among White adults. The same thing happens with Marin, Napa, Santa Clara, Solano, and Sonoma. In these counties, the rate of mortality for Black adults is higher than that of White adults *of the same age*. However, the Black adult populations in these counties are younger than the corresponding White adult populations, and younger adults have much lower mortality rates.

## Small Counts Revisited

In Section 2.2, we explained how we could "downscale" county-level mortality rates to the level of West Oakland. First we converted rates (death/person/yr) to mortalities (death/yr), using age-specific population data, and then converted back to mortality rates using total population data. This is not possible with age-adjusted data, but it is possible with age-stratified data.

Table A.3 shows crude rates, stratified by age, for Alameda County. When they are

Table A.3: Crude adult all-cause mortality rates in Alameda County (2007-2016), by age bracket and race/ethnicity.

| Age | Black | White | NatAmer | Hispanic | Asian |
|---|---|---|---|---|---|
| 25 to 34 | 1,985/M | 627/M | — | 603/M | 358/M |
| 35 to 44 | 2,981/M | 1,245/M | 2,294/M | 990/M | 642/M |
| 45 to 54 | 6,431/M | 3,037/M | 5,710/M | 2,428/M | 1,566/M |
| 55 to 64 | 14,415/M | 6,912/M | 8,355/M | 5,591/M | 3,603/M |
| 65 to 74 | 25,597/M | 15,574/M | 15,786/M | 12,448/M | 8,953/M |
| 75 to 84 | 53,909/M | 44,491/M | 39,482/M | 32,959/M | 25,461/M |
| 85 and up | 125,414/M | 135,618/M | 128,333/M | 98,615/M | 91,831/M |

stratified by age, the available data (deaths) are again apportioned among many table cells. This leads to the same small-counts problems identified above, except that we are now *also* trying to spend the "data budget" on slicing by age bracket, in addition to slicing by time, geography, and race/ethnicity. And, in Table A.3, we hit a wall: the death count for Native Americans ages `25 to 34` in Alameda County is too low, and has been suppressed. For reasons of confidentiality, CDC-WONDER will not provide data when the number of deaths is less than 10.[7]

## Downscaling to West Oakland

In Section 2.2, we described how we combined county-level mortality-rate estimates with (b) block-level population estimates to obtain a "downscaled" estimate of adult all-cause mortality covering the extent of West Oakland.

For illustration's sake only, Table A.4 shows, in addition to the rates themselves, the numerator (deaths) and denominator (persons) within each computed cell. Following the guideline that any rate with a numerator smaller than 10 should not be reported or relied upon, we can see that half of the cells in this table are, on their own, unreliable

[7]This also guards against drawing inferences based on data that are statistically unreliable. To work around this, BenMAP *imputes* ("fills in") suppressed county-level rates by borrowing estimates from larger geographies (e.g. state averages).

Table A.4: Mortality rates for West Oakland, derived from block-level estimates. Units = deaths per million persons.

| Age | WHITE | Non-WHITE | (all) |
|---|---|---|---|
| 30 to 34 | 646/$M$ (1÷1,489) | 752/$M$ (1÷1,549) | 700/$M$ (2÷3,039) |
| 35 to 44 | 1,190/$M$ (2÷2,099) | 1,193/$M$ (3÷2,787) | 1,192/$M$ (6÷4,886) |
| 45 to 54 | 2,936/$M$ (4÷1,205) | 2,982/$M$ (8÷2,751) | 2,968/$M$ (12÷3,955) |
| 55 to 64 | 6,769/$M$ (6÷915) | 7,000/$M$ (20÷2,861) | 6,944/$M$ (26÷3,776) |
| 65 to 74 | 15,245/$M$ (7÷474) | 14,027/$M$ (31÷2,189) | 14,244/$M$ (38÷2,663) |
| 75 to 84 | 42,846/$M$ (8÷181) | 33,339/$M$ (36÷1,074) | 34,711/$M$ (44÷1,255) |
| 85 and up | 131,625/$M$ (8÷57) | 102,106/$M$ (42÷409) | 105,740/$M$ (49÷466) |
| (all) | 5,565/$M$ (36÷6,421) | 10,349/$M$ (141÷13,619) | 8,817/$M$ (177÷20,040) |

and should be suppressed. We can see, however, that when aggregated to the level of West Oakland (bottom right), the calculated result has a numerator larger than 10.

In the bottom row of Table A.4, the (crude) rate among the `Non-WHITE` population is fully twice the rate among the `WHITE` population. Recalling that the rates for `Non-WHITE` are actually slightly smaller than those for `WHITE` in the older age brackets, and that older populations drive the overall mortality rate, we can see that this is not because `WHITE` adults in West Oakland are longer-lived, but because `Non-WHITE` adults in West Oakland are considerably older, whereas `WHITE` adults are younger.

Superficially, this yields an intuitive comparison of crude rates by race/ethnicity—things seem to be in the right direction, with `WHITE` adult rates being lower. But, the effect is too large, and the reason for it is not what we are expecting.

Consider what would happen if we applied the same calculation to the populations of Contra Costa, Marin, Napa, Santa Clara, Solano, and Sonoma counties. In those

Table A.5: Projected 2018 population estimates for West Oakland, exported from BenMAP and then aggregated to match strata for BenMAP-exported mortality rates. Estimates are displayed to the nearest whole number. Total = 20,040.

| Age | WHITE | Non-WHITE |
|---|---|---|
| 30 to 34 | 1,489 | 1,549 |
| 35 to 39 | 1,235 | 1,527 |
| 40 to 44 | 864 | 1,260 |
| 45 to 49 | 682 | 1,360 |
| 50 to 54 | 523 | 1,391 |
| 55 to 59 | 521 | 1,501 |
| 60 to 64 | 395 | 1,360 |
| 65 to 69 | 263 | 1,212 |
| 70 to 74 | 211 | 977 |
| 75 to 79 | 124 | 682 |
| 80 to 84 | 57 | 392 |
| 85 and up | 57 | 409 |

counties, the crude mortality rates are highest among `White` adults, but again, this is because they are older. The same `WHITE`/`Non-WHITE` comparison in those counties would lead to the opposite result—`WHITE` being higher—even though we have seen that `WHITE` adults in those counties do not have the highest mortality rates, once age is taken into account.

Table A.4 is based in part on BenMAP-exported mortality rates for Alameda County, consistent with CDC-WONDER data (Table A.3). The remainder is based on the population estimates shown in Table A.5, which consolidates BenMAP-exported estimates for adult (age $\geq 30$) populations in West Oakland (projected to 2018) into the same strata as Table A.3. The estimates are originally stratified into 19 brackets for `Age`, 4 categories for `Race`, 2 categories for `Ethnicity`, and 2 categories for `Gender` (sex)[8].

Table A.4 shows a combination of the population counts from Table A.5 and the

[8] `Gender`, rather than `Sex`, is the term used by BenMAP and CDC-WONDER. It is coded as `M` or `F`.

mortality rates from Table A.3. In a smaller font are shown both the corresponding population, and the calculated deaths (mortality rate × population), in the format `(deaths ÷ population)`. In combining these, we have made a few small but noteworthy assumptions:

- The estimated baseline mortality rate ($y$) for the `25 to 34` bracket is an unbiased estimate of $y$ for the `30 to 34` bracket[9];
- Estimates of $y$ provided for the 10-year brackets `35 to 44`, `45 to 54`, et cetera are unbiased estimates of $y$ for the corresponding 5-year brackets `(35 to 39, 40 to 44)`, `(45 to 49, 50 to 54)`, etc.; and
- Performing these calculations without also stratifying by `Sex` is acceptable.

Aggregating the population and death counts across both rows and columns, and then dividing the total deaths by the total population, should[10] yield the same result reported in Section 3.3 ($y_0 = 8,982/M$, bottom right cell). Aggregating across rows, and then dividing, yields crude (unadjusted) estimates for the `WHITE` and `Non-WHITE` subgroups (bottom row).



[9] It will probably be biased; ages 25-29 typically have lower mortality rates than ages 30-34.

[10] The result is not exactly the same. This could be because BenMAP is internally performing calculations using more and/or finer-grained strata than it will export. We are reaching out to BenMAP experts about this issue.

## Non-Stratified Rates

For reference, Table A.6 shows adult all-cause mortality rates, obtained from CDC-WONDER, for all race/ethnicities combined (2007-2016). The corresponding crude regional rate (for all nine counties combined) is 8,896/M, and the age-adjusted regional rate is 8,733/M.

Table A.6: Crude and age-adjusted adult all-cause mortality rates, 2007-2016.

| County | Age-Adjusted | Crude |
|---|---|---|
| Alameda | 9,102/M | 8,608/M |
| Contra Costa | 9,228/M | 9,753/M |
| Marin | 7,570/M | 9,801/M |
| Napa | 9,808/M | 12,414/M |
| San Francisco | 8,621/M | 8,664/M |
| San Mateo | 7,984/M | 8,757/M |
| Santa Clara | 7,944/M | 7,429/M |
| Solano | 10,501/M | 10,243/M |
| Sonoma | 9,636/M | 11,363/M |

# B TAC Framework

Risk assessments conducted by the Bay Area Air Quality Management District (BAAQMD) for toxic air contaminants (TACs) follow guidelines from Cal/EPA's Office of Environmental Health Hazard Assessment (OEHHA) and the risk management guidance for stationary sources adopted by the California Air Resources Board (CARB) and the California Air Pollution Control Officers Association (CAPCOA). (OEHHA 2015; ARB/CAPCOA 2015) In this framework, some TACs (here, "pollutants") are specific chemicals; others may be classes of compounds (e.g. PAHs or DPM). Two types of endpoints are covered by the OEHHA/CARB/CAPCOA guidance: (a) cancer outcomes; and (b) non-cancer outcomes (both chronic and acute). For brevity's sake, we focus here on cancer.

## Cancer-Risk Calculations

Cancer risks are calculated by multiplying annual average pollutant concentrations, estimated using an air dispersion model, by the pollutant intakes and the pollutant-specific potency factors (CPFs). Pollutant concentrations are modeled utilizing site-specific release parameters, from the point of release to the point of exposure at downwind locations. The pollutant intake or dose describing the frequency and duration of the exposure is estimated using receptor's breathing rates, exposure duration, and exposure frequency. In accordance with OEHHA's revised health risk assessment guidelines, California Air Districts have adopted more stringent intake methodology that addresses children's greater sensitivity and health impacts from early exposure to carcinogenic compounds. The updated calculation procedures include the use of

age-specific weighting factors, breathing rates, fraction of time at home, and reduced exposure durations.

The cancer risk is equal to the dose multiplied by the pollutant-specific CPF.
CPF is specific to the pathway whereby individuals are exposed to the pollution via inhalation, ingestion, or dermal contact. To account for exposure through all pathways, multi-pathway CPFs are available from OEHHA. Contributions from all significant sources including stationary and mobile sources are aggregated to determine the cumulative risks. Risks are not estimated for pollutants lacking OEHHA approved toxicity values.

The pollutant intake or dose describes the frequency and duration of the exposure, estimated using the breathing rates, exposure durations, and exposure frequencies. In accordance with OEHHA's revised health risk assessment guidelines (OEHHA 2015; ARB/CAPCOA 2015), the intake methodology was updated to address children's greater sensitivity and health impacts from early exposure to carcinogenic compounds.

## Dose Equation (Inhalation-Only)

The equation used to calculate the dose for the *inhalation pathway* is as follows:

$$\text{Dose}_i = \text{CF} \times \text{EF} \times \sum_j \left(\text{C}_{i,j} \times \text{DBR}_j \times \text{FAH}_j \times \text{ED}_j \times \text{ASF}_j\right) \div \text{AT} \qquad \text{(B.1)}$$

where:

- $\text{Dose}_i$ = Accumulated dose for an individual breathing carcinogen $i$ from the 3rd trimester through the $30^{\text{th}}$ year of life ($\frac{mg}{kg \cdot day}$);
- CF = Conversion factor $\left(10^{-6}\ \frac{\text{mg} \cdot m^3}{g \cdot L}\right)$
- EF = Exposure frequency ($350\ day/yr$);
- $\text{DBR}_j$ = Daily breathing rate $\left(\frac{L}{kg \cdot day}\right)$ for year $j$;

- $FAH_j$ = Fraction of time at home (unitless) for year $j$;
- $ED_j$ = Exposure duration ($yr$) for year $j$;
- $C_{i,j}$ = Annual average concentration ($\frac{g}{m^3}$)of pollutant $i$ for year $j$;
- $ASF_j$ = Age sensitivity factor (unitless) for year $j$; and
- AT = Averaging time (25,550 days, equivalent to 70 year lifespan)

# Key Factors

The updated procedures in OEHHA (2015) include the use of age-specific weighting factors, breathing rates, fraction of time at home, and reduced exposure durations.

**Age Sensitivity Factors (ASFs)** account for the heightened sensitivity of children to carcinogens during fetal development and early childhood. Consistent with OEHHA (2015), BAAQMD uses ASF values as listed in Table B.1. BAAQMD has incorporated ASFs in its air permits since 2010.

**Daily Breathing Rate (DBR)** is the age-specific daily air intake. OEHHA developed a range of rates for four age groups: last trimester to newborn, newborn to two years of age, two years to 16 years of age, and older than 16 years of age. CAPCOA and CARB recently recommended the use of 95$^{th}$ percentile breathing rates for the most sensitive age group (less than two years of age) and 80$^{th}$ percentile for all other age groups (ARB/CAPCOA 2015).

**Fraction of Time at Home (FAH)** refers to the estimated amount of time residents stay at home. In past HRAs, BAAQMD assumed that residents are home 24 hours per day, 7 days per week. In (OEHHA 2015), OEHHA recommends less than 100% of time to be used as a FAH based on population and activity statistics. Consistent with (OEHHA 2015), this analysis incorporates a FAH of 0.73 for individuals $\geq$ 16 years old and 1.0 for individuals $<$ 16 years old to address exposures at local schools in close proximity to emitting facilities (Table B.1).

Table B.1: Factors used to calculate dose. (OEHHA)

| Factor | Description | Units | 3rd Trimester | 0-2 yrs | 2-16 yrs | 16-30 yrs |
|---|---|---|---|---|---|---|
| DBR | Daily breathing rate | L/kg-day | 361 | 1090 | 572 | 261 |
| ASF | Age sensitivity factor | — | 10 | 10 | 3 | 1 |
| FAH | Fraction of time at home | — | 1 | 1 | 1 | .73 |
| ED | Exposure duration | years | .25 | 2 | 14 | 14 |

**Exposure Duration (ED)** is the length of time an individual is continuous exposed to air toxics. Previously, BAAQMD used a 70-year lifetime exposure duration for residents over a 70-year lifespan. Based on updated demographic data, BAAQMD now follows the OEHHA recommendation of a 30-year exposure duration, consistent with US EPA, for residents.

The values of these factors are summarized in Table B.1.

# C Response Function

## Background

In the literature, we find two distinct functions that yield estimates of the change in mortality rate, given some change in exposure. Both are nonlinear.

One such delta-response function looks like this:

$$\Delta y = y_0 \left(e^{\beta \Delta x} - 1\right) \tag{C.1}$$

The other looks like this:

$$\Delta y = y_0 \left(1 - e^{-\beta \Delta x}\right) \tag{C.2}$$

## Explanation

From a mathematical perspective, the two equations are easy to reconcile. If we supply Eq (C.1) with a change in exposure, *putting a negative sign on that change*, we will obtain a change in mortality that is also negative. The magnitude of that result will be exactly the same as the magnitude of the result that we get if we plug in the same change in exposure — but *without* a negative sign — into Eq (C.2). So, the two equations do express the same relationship between $x$ and $y$. The difference is simply due to a flipping of of sign on $\Delta x$ and $\Delta y$ in Eq (C.2).

From an applied perspective, the key difference is that Eq (C.1) yields a (**negative**)

Table C.1: Which equation to use depends on the meaning of the sign on $\Delta x$.

| Input | Using | Interpretation | Comment |
|---|---|---|---|
| $\Delta x > 0$ meaning "increase" | $y_0\left(e^{\beta\,\Delta x} - 1\right)$ | $\Delta y > 0$ is "harmful" | As in TAC HRAs |
| $\Delta x > 0$ meaning "reduction" | $y_0\left(1 - e^{-\beta\,\Delta x}\right)$ | $\Delta y > 0$ is "beneficial" | As in BenMAP |

Table C.2: Using the wrong equation results in an error.

| Intent | Using | Result |
|---|---|---|
| $\Delta x > 0$ meaning "increase" | $y_0\left(1 - e^{-\beta\,\Delta x}\right)$ | Error |
| $\Delta x > 0$ meaning "reduction" | $y_0\left(e^{\beta\,\Delta x} - 1\right)$ | Error |

estimated *decrease* in mortality from a (**negative**) hypothetical *decrease* in exposure, as would be obtained by abating an existing source. Thus, in the world of Eq (C.1), a negative $\Delta y$ is beneficial. This is also interpretable as the existing mortality attributable to an existing source (which would not exist if the source did not exist). If we supply Eq (C.1) instead with a (**positive**) hypothetical *increase* in exposure, we obtain an estimated *increase* in mortality. This is interpretable as the increase in mortality that would be due to the introduction of a source that does not yet exist.

For Eq (C.2), on the other hand, "reductions" have a positive sign. Eq (C.2) yields a (**positive**) estimated *reduction* in mortality from a (**positive**) hypothetical *reduction* in exposure, as would be obtained by abating an existing source. This is how we frame "benefit" in the world of Eq (C.2).

# Practical Concerns

An inexperienced or hurried user might make one of the errors listed in Table C.2.

This is not so much a problem in a linear framework, because of the ease of detection and repair. If a user of the linear equation $\Delta y = \beta\ \Delta x$ obtains but was not expecting a negative $\Delta y$, they can reasonably just "flip the sign" on $\Delta y$ and it will be exactly as if they had flipped the sign on $\Delta x$ when providing $\Delta x$ as input.

In our nonlinear framework, this does not yield a correct $\Delta y$. It is true that, for small values of $\beta$ and small values of $\Delta x$, the result of simply flipping the sign on $\Delta y$ will be *approximately* correct. However, to obtain a correct result, one must follow Table C.1 and not Table C.2.

We illustrate this, providing a rough idea of the magnitude of potential errors, with figures and exact calculations below.

# Illustration

To make things more concrete, assume $RR$ (the multiplicative risk ratio) for a "unit increment" ($\Delta x$) equal to +10 µg/m$^3$ $PM_{2.5}$ to be 1.07; then $\beta = ln(1.07) \approx 0.0677$. Assume a population at risk (Pop) of **1 million** ($1 \times 10^6$) persons, and assume a baseline mortality rate ($y_0$) of **1% per year**.

Figure C.1 illustrates the different results we obtain by employing Eq (C.1) or Eq (C.2). Two domains are shown. The 0-3 µg/m$^3$ domain (inset) represents a "plausible" domain of potential changes. The 0-30 µg/m$^3$ domain (main figure) highlights the divergence. Because this is a practical example, we are scaling the y-axis by `Pop`, thereby converting from a change in the annual *mortality rate* to a change in annual *mortality*.

Looking at the curve for Eq (C.1), when $\Delta x$ is 3 µg/m$^3$, $\Delta y$ is 205 death/yr. The correct interpretation here is that an *increase* of 3 µg/m$^3$, starting from baseline conditions, will *induce* an estimated 205 death/yr.

Looking at the curve for Eq (C.2), when $\Delta x$ is 3 µg/m$^3$, $\Delta y$ is 201 death/yr. In this case, the correct interpretation is that a *reduction* of 3 µg/m$^3$, starting from baseline conditions, will *avert* an estimated 201 death/yr.

If we expand the domain of $\Delta x$ to include negative values (Figure C.2), we can see that the two curves are symmetric. This makes it clear that they are identical if we

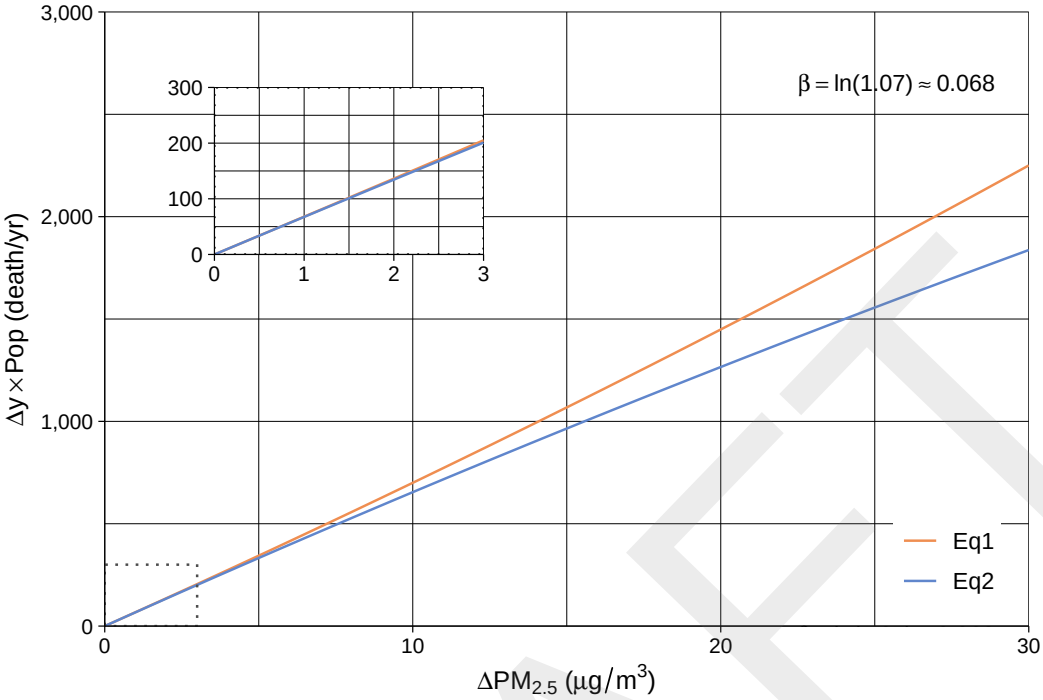



Figure C.1: Comparison of two delta-response functions.

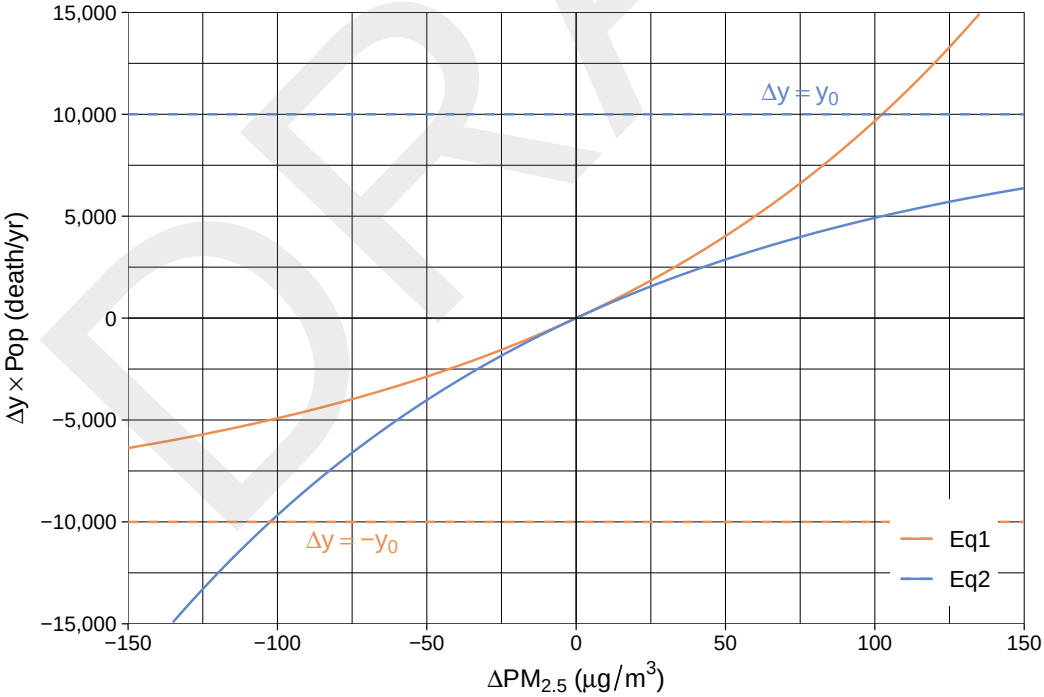



Figure C.2: Expansion to include negative values and asymptotes, demonstrating symmetry.

substitute $-\Delta x$ for $\Delta x$ and $-\Delta y$ for $\Delta y$ in one or the other.

A useful double-check is to inspect the limits as $\Delta x$ approaches infinity. We could never avert more deaths than are already occurring in the population. This should hold true — and it does — for both Eq (C.1) and Eq (C.2).

The R code we used to implement these two equations, and generate the figures above, is available at https://github.com/BAAQMD/PM25-HIA-methodology/.

## Direct Calculations ($PM_{2.5} \rightarrow$ Mortality)

This section demonstrates direct calculations with $x$ and $y$, instead of with $\Delta x$ and $\Delta y$. In this log-linear framework, the exposure $x$ is related to the mortality rate $y$ like so:

$$\ln(y) = \beta x + C$$
$$y = \exp(\beta x + C)$$

As above, let $\beta = ln(RR) = ln(1.060) \approx 0.058269$, and $y_0 = 0.01 \times 10^6$ (deaths per million persons per year).

Assume the baseline $PM_{2.5}$ is **9 µg/m³**, comparable to the West Oakland estimate provided in Section 2.2. The unit increment for $x$ is 10 µg/m$^3$, so the baseline $x$ is then 0.9. Call this $x_0$. Now we can work out $C$:

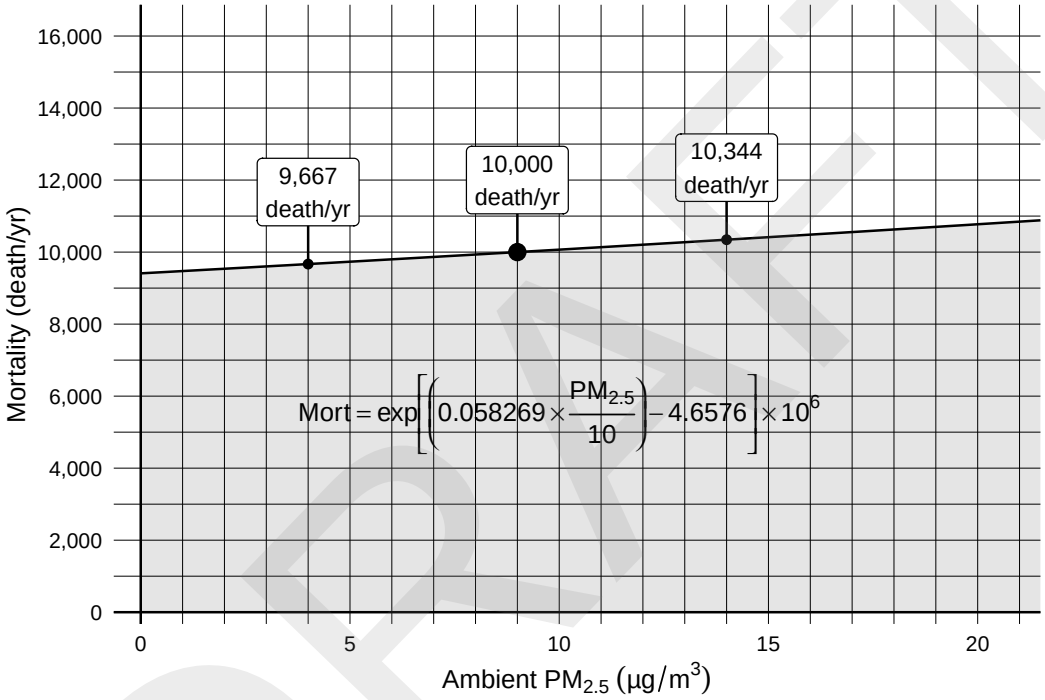
$$\ln(y_0) = \beta x_0 + C$$
$$C = \ln(y_0) - \beta x_0$$
$$C \approx \ln(0.01) - (0.058269 \times 0.9)$$
$$C \approx -4.6576$$

Substituting $x = \frac{PM_{2.5}}{10}$, $\beta \approx 0.058269$, $C \approx -4.6576$, and Pop $= 10^6$ into Mort $=$

$y \times$ Pop, we have:

$$\text{Mort} = \exp(\beta x + C) \times \text{Pop}$$
$$\text{Mort} \approx \exp\left[\left(0.058269 \times \frac{\text{PM}_{2.5}}{10}\right) - 4.6576\right] \times 10^6$$

Now we can explore both specific calculations, and the general form of the response function, in a more visual way:




As we can see from this figure:

- When the exposure is **9 µg/m³**, we have 10,000 death/yr. These are baseline conditions.
- When the exposure is **14 µg/m³**, we have 10,296 death/yr. This is an additive change of +5 µg/m$^3$ (*vs* baseline). It results in a multiplicative change in the response: 1 * 1.0296 = 102.96% as many deaths. Or, on an additive scale, **296** more deaths.
- When the exposure is **4 µg/m³**, we have 9,713 death/yr. This is an additive

change of -5 µg/m$^3$ (*vs* baseline). It results in a multiplicative change in the response: 1 / 1.0296 = 97.13% as many deaths. Or, on an additive scale, **287** fewer deaths. Note that this is less than **296**.

Because the relationship between $x$ and $y$ is supralinear, in this framework, when we start from the same baseline conditions, the increased mortality due to an increase of $PM_{2.5}$ will *always* be larger in magnitude than the decrease in mortality due to a reduction of the same magnitude.

The potential error (in using the wrong equation) would be most salient in a risk-assessment framework whose chief metric(s) were based on maximum impacts, as opposed to means or totals. Our model-based case studies suggest that maximum impacts could be on the order of +1 µg/m$^3$ or more for certain sources at short distances. The error would then be on the order of 1%—not large, but enough to be noticeable if the results were reported to two or more significant digits.

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**BAY AREA AIR QUALITY MANAGEMENT DISTRICT**
Memorandum

To: Chairpersons Linda Rudolph and Gina Solomon, and Members of the Advisory Council

From: Jack P. Broadbent
Executive Officer/APCO

Date: February 14, 2022

Re: 2022 Advisory Council Work Plan Discussion

RECOMMENDED ACTION

None; receive and file.

BACKGROUND

In 2021, the Advisory Council received presentations and information on a variety of subjects and in December 2021 the Councilmembers discussed which topics the Council might research further.

DISCUSSION

Advisory Councilmembers will receive an overview of the 2022 Advisory Council work plan for discussion.

The workplan proposed to focus on four key elements:

- Working with Air District staff and other external experts to develop a standard methodology to assess the impacts of fine particulate matter (PM2.5) exposure.
- Developing a strategy to address combustion sources culminating in a report to the Board of Directors by the end of the year.
- Addressing questions raised by the Community Advisory Council about air pollution and health.
- Reviewing and commenting on other Air District staff work developed to support key decisions by the Board of Directors.

Staff will present an initial plan based on these elements for discussion with the Advisory Council including expected agenda items for the next few meetings.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Jack P. Broadbent
Executive Officer/APCO

Prepared by: Sonam Shah-Paul
Reviewed by: Greg Nudd

ATTACHMENTS:

None.