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 (RIVAS 2021) ALLOWING REMOTE MEETINGS. THIS MEETING WILL BE ACCESSIBLE VIA WEBCAST, TELECONFERENCE, AND ZOOM. A ZOOM PANELIST LINK WILL BE SENT SEPARATELY TO COUNCIL MEMBERS

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

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/82765473261

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

WEBINAR ID: 827 6547 3261

- **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, SEPTEMBER 12, 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.*

CONSENT CALENDAR

3. Approval of the Advisory Council Meeting Minutes of July 11, 2022

The Council will consider approving the attached draft minutes of the meeting of July 11, 2022.

PRESENTATION(S)

4. Fine Particulate Matter Local Risk Methodology Update

This is an informational item only and will be presented by David Holstius, PhD, Senior Projects Advisor.

5. Source Prioritization Framework

This is an informational item only and will be presented by Elizabeht Yura, Director of Rules and Strategic Policy.

OTHER BUSINESS

6. Report of the Executive Officer/APCO

7. 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.

8. 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.

9. Time and Place of Next Meeting

Monday, November 14, 2022, at 8:30 a.m., via webcast, teleconference, or Zoom, pursuant to procedures in accordance with Assembly Bill 361 (Rivas 2021).

10. 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

SEPTEMBER 2022

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Community Advisory Council Mtg.	Thursday	8	6:00 p.m.	Webcast only pursuant to Assembly Bill 361
Advisory Council Meeting	Monday	12	8:30 a.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Legislative Committee - CANCELLED	Monday	12	1:00 p.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Community Equity, Health and Justice Committee - CANCELLED	Thursday	15	9:30 a.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Stationary Source and Climate Impacts Committee	Monday	19	9:00 a.m.	Webcast only pursuant to Assembly Bill 361
Path to Clean Air Community Emissions Reduction Plan Steering Committee	Monday	19	5:30 p.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Meeting	Wednesday	21	9:00 a.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Administration Committee	Wednesday	21	1:00 p.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Mobile Source and Climate Impacts Committee - CANCELLED	Thursday	22	9:30 a.m.	Webcast only pursuant to Assembly Bill 361
Board of Directors Budget and Finance Committee - CANCELLED	Wednesday	28	9:30 a.m.	Webcast only pursuant to Assembly Bill 361

OCTOBER 2022

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Board of Directors Legislative Committee	**Monday**	**3**	**1:00 p.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Meeting	**Wednesday**	**5**	**9:00 a.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Community Equity, Health and Justice Committee	**Thursday**	**6**	**9:30 a.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Legislative Committee- CANCELLED AND RESCHEDULED TO MONDAY, OCTOBER 3, 2022 AT 1:00 P.M.	**Monday**	**10**	**1:00 p.m.**	**Webcast only pursuant to Assembly Bill 361**
Technology Implementation Office (TIO) Steering Committee	**Friday**	**14**	**1:00 p.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Stationary Source and Climate Impacts Committee	**Monday**	**17**	**9:00 a.m.**	**Webcast only pursuant to Assembly Bill 361**
Path to Clean Air Community Emissions Reduction Plan Steering Committee	**Monday**	**17**	**5:30 p.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Meeting	**Wednesday**	**19**	**9:00 a.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Administration Committee	**Wednesday**	**19**	**1:00 p.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Budget and Finance Committee	**Wednesday**	**26**	**9:30 a.m.**	**Webcast only pursuant to Assembly Bill 361**
Board of Directors Mobile Source and Climate Impacts Committee	**Thursday**	**27**	**9:30 a.m.**	**Webcast only pursuant to Assembly Bill 361**

HL 9/8/22 – 10:20 a.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: Sharon L. Landers
Interim Executive Officer/APCO

Date: September 12, 2022

Re: Approval of the Advisory Council Meeting Minutes of July 11, 2022

RECOMMENDED ACTION

Approve the attached draft minutes of the Advisory Council meeting of July 11, 2022.

BACKGROUND

None.

DISCUSSION

Attached for your review and approval are the draft minutes of the Advisory Council meeting of July 11, 2022.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Sharon L. Landers
Interim Executive Officer/APCO

Prepared by: Marcy Hiratzka
Reviewed by: Vanessa Johnson

ATTACHMENTS:

1. Draft Advisory Council Meeting Minutes of July 11, 2022

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

Advisory Council Meeting
Monday, July 11, 2022

DRAFT MINUTES

Note: Audio recordings of the meeting are available on the website of the Bay Area Air Quality Management District at www.baaqmd.gov/bodagendas

This meeting was conducted under procedures in accordance with Assembly Bill 361. Members of the Advisory Council participated by teleconference.

CALL TO ORDER

1. **Opening Comments:** Advisory Council (Council) Co-Chairperson, Dr. Linda Rudolph, called the meeting to order at 8:30 a.m.

   Roll Call:

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

   Absent: Member Dr. Pallavi Phartiyal.

2. **PUBLIC MEETING PROCEDURE**

3. **APPROVAL OF THE MINUTES OF APRIL 11, 2022**

Public Comments

No requests submitted.

Council Comments

None.

Council Action

Vice Chair Kleinman made a motion, seconded by Board Liaison Haubert, to **approve** Minutes of April 11, 2022; and the motion **carried** by the following vote of the Council:

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

4. PARTICULATE MATTER (PM) MODELING: CONTEXT, PRODUCTS AND PROGRESS

Greg Nudd, Deputy Air Pollution Control Officer of Policy, gave the staff presentation *PM Modeling: Context, Products, and Progress,* including: outline; the larger PM context; four recent Advisory Council presentations; the takeaway; PM modeling efforts in summary; modeling products and timeline; combustion analysis progress – building appliances, all source assessment: application of InMAP; and next steps.

Public Comments

Public comments were given by Jed Holtzman, San Francisco resident.

Council Comments

The Council and staff discussed the difference between the InMAP and BenMAP-CE modeling programs; whether modeling programs accurately capture peaks during upset conditions and disruptions in normal activity; how wildfire smoke and vehicular emissions are incorporated into cumulative impacts; whether regulated facilities are required to notify the Air District when they begin flaring; types of fuels of combustion and which have the greatest health impacts; why the Air District allows woodburning in homes within impacted neighborhoods; whether modeling methods that differentiate between vehicular brake and tire wear exist; whether the Air District has implemented the Council's recommendation from 2019 to establish more a more stringent, health-protective PM target (for the Bay Area region) than the current federal standards; and the request for a list of wildfire smoke preparedness tips.

Council Action

None; receive and file.

5. FINE PARTICULATE MATTER LOCAL RISK METHODOLOGY: UPDATE AND KEY QUESTIONS

Mr. Nudd introduced Dr. David Holstius, Senior Advanced Projects Advisor, who gave the staff presentation *Fine PM Local Risk Methodology: Update and Key Questions* including: overview; key questions; recap: average annual impact; advancements; revised approach; illustration; exposure window; points of reference; safety/uncertainty considerations; relevant papers; receptors to consider; and key questions. Dr. Holstius illustrated revisions to the methodology since the last meeting: a multi-

year exposure window; a focus on maximum impacts; and the assessment of asthma risks for children. The key question posed to the Council was whether available evidence supported a factor of at least 3 to account for sensitive individuals. For consideration, Dr. Holstius presented evidence from recent large-scale scientific studies of the health effects of fine particulate matter ($PM_{2.5}$) at or below levels corresponding to the current National Ambient Air Quality Standards (NAAQS).

Public Comments

Public comments were given by Jed Holtzman, San Francisco resident.

Council Comments

As clarification, Dr. Michael Kleinman requested confirmation of the statistical significance of the numbers presented, via the provision of error bars. Dr. Holstius affirmed that error bars could be provided and would not include "no effect." Dr. Kleinman expressed thanks for the inclusion of asthma in the revised methodology, which was later seconded by Dr. Linda Rudolph. Dr. Kleinman remarked that scientific data indicate that the dose-response curve could be super-linear, so that the effect per microgram may be more at levels below the current NAAQS. Dr. Gina Solomon inquired whether the proposed model included a threshold. Dr. Holstius clarified that it did not, that there is no evidence of a threshold at a population level, and that the method is concerned with the effects of small changes within a policy-relevant range around baseline levels, not effects close to zero.

Regarding the key question posed to the Council, Dr. Rudolph recommended the largest factor that the Air District can support, for the following reasons: there is no threshold; the evidence presented was well selected, but still limited by not looking at a full range of endpoints such as reproductive and cognitive; that $PM_{2.5}$ has a very wide range of impacts; that baseline exposures do not take into account large excursions from wildfire smoke; and that people are exposed at all life stages, while the modeled exposure windows are limited to 30 years. Dr. Solomon agreed with Dr. Rudolph and stated that the large number of outcomes that are not yet quantified justifies a larger factor. Dr. Solomon remarked that there is often a standard three-fold (3x) default factor for data-based deficiencies that was not mentioned during the presentation, that it would be supportable based on the material presented, and that it should be included. Dr. Solomon further remarked that it also would be no problem to support a factor of 3x for vulnerable subpopulations, based on the data presented, but that even more could be supported, and that if both factors were included, a minimum of 10x would be required.

Council Action

None; receive and file.

6. REPORT OF THE EXECUTIVE OFFICER/AIR POLLUTION CONTROL OFFICER

Veronica Eady, Senior Deputy Executive Officer of Policy & Equity, made the following announcements:

- Sharon Landers, Interim Executive Officer/Air Pollution Control Officer, is taking time off due to medical reasons.
- The Air District's Community Advisory Council has met three times, and a joint meeting of the Council and Community Advisory Council is anticipated.

Dr. Ranyee Chiang, Director of Meteorology and Measurement, was asked by Ms. Eady to provide a summary on recent air quality.

7. PUBLIC COMMENTS ON NON-AGENDA MATTERS

No requests received.

8. COUNCIL MEMBER COMMENTS/OTHER BUSINESS

None.

9. TIME AND PLACE OF NEXT MEETING

At the end of the meeting, the next Advisory Council meeting was to be scheduled at the call of the Co-Chairs. After the meeting adjourned, the next meeting was scheduled for Monday, September 12, 2022, at 8:30 a.m., via webcast, teleconference, or Zoom, pursuant to procedures in accordance with Assembly Bill 361 (Rivas 2021).

10. ADJOURNMENT

The meeting adjourned at 10:35 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: Sharon L. Landers
Interim Executive Officer/APCO

Date: September 12, 2022

Re: Fine Particulate Matter Local Risk Methodology Update

RECOMMENDED ACTION

None; receive and file.

BACKGROUND

A regional regulatory framework has been successful in reducing PM2.5 exposures for the Bay Area population overall, but an expanded toolset is warranted to accelerate exposure reductions for the Bay Area's most impacted populations. Responding to the Advisory Council's 2020 *Particulate Matter Reduction Strategy Report PM Reduction Strategy Report,* staff have assembled a draft methodology for use in managing health risks posed by specific sources of PM2.5 at a local level.

At the Advisory Council Meeting on July 11, 2022, Agenda Item 5 ("Fine Particulate Matter Local Risk Methodology: Update and Key Questions") presented relevant epidemiological evidence and posed key questions to the Advisory Council concerning safety/uncertainty factors. The updates presented in this item are responsive to the comments and recommendations offered by the Council at that time.

DISCUSSION

Staff will present updates to the methodology that are responsive to Advisory Council feedback concerning factors that would be protective of at-risk populations. Staff will present an updated approach that uses multiplicative factors to adjust population-average (a) breathing rates and (b) effect sizes. For breathing rates, we propose using 95^{th} percentile age- and activity-specific rates, in line with existing guidance on health risk assessments. For effect sizes, we recommend a factor of three, based on empirical studies of sensitive populations that report variations in health outcomes. We link the corresponding adjustments to (1) the concentration-exposure-dose-response framework, and (2) the key equation supporting calculations of impacts based on relative risks.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Sharon L. Landers
Interim Executive Officer/APCO

Prepared by:	David Holstius
Reviewed by:	Phil Martien and Greg Nudd

ATTACHMENTS:

1. PM25-local-risk-method-v0.6.1_090622

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

August 2022

Bay Area Air Quality Management District

Project Lead

David Holstius, PhD, Senior Advanced Projects Advisor
Assessment, Inventory and Modeling Division

Reviewed by

Greg Nudd, Deputy Air Pollution Control Officer
Policy and Equity Office

Phil Martien, PhD, Director
Assessment, Inventory and Modeling Division

Advisors and Contributors

Gina Solomon, MD, MPH, Co-Chair
Linda Rudolph, MD, MPH, Co-Chair
Michael Kleinman, PhD
Adrienne Hollis, PhD, JD
Pallavi Phartiyal, PhD
Danny Cullenward, PhD, JD
Garima Raheja
Bay Area Air Quality Management District Advisory Council

Yuanyuan Fang, PhD, Statistician
Air Quality Modeling and Analysis Section

Judith Cutino, DO, Health Officer
Policy and Equity Office

The Bay Area Air Quality Management District (Air District) also wishes to acknowledge the thoughtful feedback offered by staff at the Office of Environmental Health Hazard Assessment (OEHHA), the California Air Resources Board (CARB), and the United States Environmental Protection Agency (US EPA): Lauren Zeise; Vincent Cogliano; John Faust; Rupa Basu; Keita Ebisu; Xiangmei Wu; Heather Bolstad; Bonnie Holmes-Gen; Hye-Youn Park; Jinhyok Heo; Arash Mohegh; Ken Davidson; and Neal Fann. Special thanks also go to Amy Kyle for her feedback on earlier drafts. Finally, our thanks to all the stakeholders who have participated throughout the public process and strengthened this methodology through their critiques and suggestions.

Abbreviations

BAAQMD	Bay Area Air Quality Management District
BenMAP-CE	Benefits Mapping and Analysis Program, Community Edition
CARB	California Air Resources Board
CDC	Centers for Disease Control and Prevention
HRA	Health risk assessment
MEI	Maximally exposed individual
NAAQS	National Ambient Air Quality Standards
OEHHA	Office of Environmental Health Hazard Assessment
$PM_{2.5}$	Particulate matter less than 2.5µm in aerodynamic diameter
RR	Relative risk
US EPA	United States Environmental Protection Agency
WAF	Worker adjustment factor

Contents

1. Introduction and Background

This document updates and extends a draft white paper (BAAQMD 2022) on modeling risk from local sources of fine particulate matter ($PM_{2.5}$) developed by the Bay Area Air Quality Management District (Air District), presented to the Air District's Advisory Council (Advisory Council), and distributed to staff at the United States Health Protection Agency (US EPA), the California Air Resources Board (CARB), and California's Office of Environmental Health Hazard Assessment (OEHHA). As summarized in presentations to the Advisory Council, the draft white paper's methodology assessed only the risk of premature mortality for a statistically average adult. The Advisory Council enjoined staff to augment the methodology with factors to protect sensitive groups, and to consider assessing chronic health impacts as well. Staff at OEHHA also requested that the Air District be mindful of vulnerabilities, and members of the public urged the Air District to include asthma. This updated methodology is responsive to those requests.

The purpose of this document is to propose and demonstrate a general methodology that can support the assessment and regulation of health risks from fine particulate matter ($PM_{2.5}$) at a local level. National- and regional-scale assessments for $PM_{2.5}$ have been conducted for many years (e.g., Fann et al. 2011; Tanrikulu et al. 2011, 2019; see also Hubbell et al. 2009), 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 current NAAQS in many regions. However, it has become increasingly clear that gaps left by the NAAQS-centered approach must be addressed.

A gap that this work seeks to help close is the persistent exposure of some populations to locally elevated concentrations of $PM_{2.5}$. Although a large fraction of $PM_{2.5}$ is regionally contributed (Blanchard 2004), elevated concentrations of $PM_{2.5}$ exist near sources of emissions (Ito et al. 2004; Wilson et al. 2005; Karner et al. 2010; Gu et al. 2018; Wang et al. 2020; Chambliss et al. 2021), have persisted in the same patterns over decades (Colmer et al. 2020), and have been linked to structural and institutional discrimination (Houston et al. 2004, 2008; Fisher et al. 2006; Morello-Frosch and Lopez 2006; Banzhaf et al. 2019; Colmer et al. 2020).

Compared to the NAAQS, the US EPA's air toxics program "places comparatively greater emphasis on reducing risks among highly exposed individuals." (Fann et al. 2016) Thus, to regulate carcinogens, for several decades the Air District has conducted local-scale modeling and set corresponding source-specific or project-specific thresholds for maximum contributions to a lifetime risk of cancer. The Air District has also modeled source-specific contributions to local elevations of $PM_{2.5}$, but to date has not conducted any corresponding health risk assessments (HRAs). This methodology would enable those assessments.

2. Concepts and Methods

Modeling of exposure. The general framework proposed here is similar to a framework that is widely employed in health risk assessments (HRAs) of toxic air contaminants. It is source-specific and based on modeling. We assume that a given source's contributions to near-field ambient concentrations can be adequately estimated using a steady-state dispersion model, which relies on user-supplied data to describe site conditions and meteorological conditions. When data are also supplied to describe the emissions of some pollutant from a source, including the way those emissions are released (at what elevation, velocity, and so on), such a model can be used to predict that source's direct contribution to the total concentration of the given pollutant at any nearby coordinate ("receptor location"). Detailed explanations and discussions are available in other publications (OEHHA 2012, 2015; BAAQMD 2021).

For a given source and pollutant, it is conventional to model impacts on different types of receptors[1] in the vicinity, each with its own characteristics. These include residents, off-site workers, students, and so forth. For each combination of receptor type, averaging time, and pollutant[2], dispersion-modeling results are used to identify a location corresponding to the most-impacted receptor of that type. These are termed "maximally exposed individual" (MEI) receptors. For a given source, averaging time, and pollutant, there will be at most one residential MEI, one off-site worker MEI, and so on.

In this version of the methodology, we work exclusively with annual averaging times. Having identified the MEI receptor locations for annual average $PM_{2.5}$, and the corresponding contributions of the source, we proceed with assumptions and/or site-specific data about the time-activity patterns of a given receptor type, and potentially the operational schedule of the source as well. (OEHHA 2015; BAAQMD 2021). Using this information, we convert from incremental average *concentrations* to incremental average *exposure intensities*. The latter take the co-presence of the source's emissions, and the envisioned receptor, into account. If 100% of a source's emissions are assumed to occur when a modeled receptor is present at the given receptor location (e.g., during the working hours of an off-site worker), then the incremental average exposure intensity will be equal to the incremental average concentration. If they never coincide, then it will be zero. Although the receptor may be exposed to other sources, this methodology is concerned with contributions from the modeled source.

Modeling of responses to exposure. To re-express the modeled incremental average exposure intensities in the form of health risks, we leverage response functions from epidemiological

[1] "Receptor" as a term of art in air quality modeling can refer either to (a) an entity exposed to pollution, or (b) the location at which that exposure is assumed to occur.

[2] Impacts from multiple pollutants may be aggregated, so long as they can be expressed in terms of the same impact metric.

studies of the health effects of $PM_{2.5}$. In this version of the methodology, we leverage response functions for (a) premature adult mortality and (b) pediatric asthma onset, applying these to residential, off-site worker, school, and daycare receptors.

The response functions that we rely on are used to calculate relative risks. We convert these to incremental absolute risks using information about baseline rates. To illustrate: suppose we take the *relative* risk of asthma onset, per μg/m^3, to be 1.04 for five-year-old children. For a scenario in which the annual average exposure intensity at a corresponding receptor is increased by 1 μg/m^3, we take the baseline annual incidence[3] rate of asthma and multiply it by 1.04. Subtracting the baseline from this scaled result yields an estimate of the excess probability (risk) of developing asthma before turning six, compared to the baseline scenario.

The following equations express this in mathematical terms.[4] Let $\Delta x = x - x_0$ and $\Delta y = y - y_0$, where x_0 and y_0 represent the baseline $PM_{2.5}$ concentration and the baseline incidence rate of some health endpoint. Taking $\Delta x > 0$ to mean an increase in $PM_{2.5}$, and $\Delta y > 0$ a corresponding increase in risk, we have:

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

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

The effect size, or the change in y associated with a unit change in x, is represented in these equations by the term β. Typically, β will be based on an epidemiological study in which ambient outdoor $PM_{2.5}$, measured or estimated at some locations, was the independent variable. Generally, epidemiological studies estimate β by adjusting for other measured factors in such a way that β 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 $PM_{2.5}$. In the equations above, β is essentially the natural logarithm of the risk ratio. The average marginal effect size that β is intended to represent will reflect the distribution of factors that lay on the causal pathways between ambient $PM_{2.5}$ and the outcome of interest in the population that was studied. For example, the breathing rates of the studied population will be implicit in the resulting population-average estimate of β. In Section 4, to account for at-risk populations, we incorporate adjustments to some of these factors.

Multi-year exposures. To extend the exposure duration to more than one year, we follow the principles behind existing guidance developed for HRAs (OEHHA 2015; BAAQMD 2021). For residential receptors, current guidelines assume a window of exposure that is up to 30 years. Consistent with a focus on maximal risk, in cancer-risk HRAs this is taken to be the first 30 years

[3] The baseline rate here is in terms of *incidence* (new cases per unit time), rather than *prevalence* (existing cases at a point in time).

[4] For additional discussion, see Fann et al (2011) and US EPA (2010, 2022).

of life.[5] For premature mortality, on the other hand, the most vulnerable window is during the later years of life. For pediatric asthma onset, by definition, the window is within the first 18 years of life.

By applying relative risks in a sequential fashion to each year within a defined window of exposure, and by comparing a less-exposed scenario to a more-exposed scenario, we can arrive at an overall result that summarizes the multi-year risk on an additive scale. Figure **1** illustrates this approach. The following two sections provide a series of worked examples, culminating in the results reported in Table **11**.

[5] It also includes the third trimester of pregnancy.

3. Example Calculations

This section illustrates the application of the concepts and methods described above.[6] Example calculations are provided in stages. For simplicity, we refer to a hypothetical concentration increase of 0.1 μg/m^3 at all stages, but later provide a lookup table for larger and smaller increments. After illustrating the fundamentals, in the next section ("Sensitive Individuals") we complete the method by accounting for children and adults who are more at risk.

In this section, we first calculate the risk of premature mortality for a residential receptor that is maximally exposed but has otherwise "statistically average" characteristics—breathing rate, health status, and so on.[7] Such a receptor does not represent any actual person, but the result corresponds to the result we would expect if we modeled a representative sample of a very large number of people and then took the average of the results.[8] Second, we model premature mortality for a statistically average adult of working age, shortening and shifting the exposure window so that it ends with retirement. Third, we introduce another health endpoint (pediatric asthma onset) and calculate relevant risks for residential, school, and daycare receptors.

Senior resident. As explained in Section 2, the relevant exposure window when assessing premature mortality should be later in life. Currently, life expectancy in the Bay Area is just under 80 years, and given our baseline rates, approximately half the population should survive to age 85. Taking this into account, when assessing the risk of mortality for a residential receptor we define the exposure window to be ages 55–84.

To calculate an incremental average exposure intensity, we multiply our example concentration increment (0.1 μg/m^3) by factors that describe the overlap between the schedules of the source and receptor. Following guidance from OEHHA (2015), for an adult residential receptor, we assume that 73% of the time is spent at home, 350 days per year, yielding an overall conversion factor of 0.70. The resulting incremental average exposure intensity is then 0.7×0.1 μg/m^3 = 0.07 μg/m^3. Consistent with the ranges reported in the Air District's recent evaluations of health impacts on regional populations (Fang et al. 2021a, 2021b), we take the relative risk of premature mortality to be 1.01 per 1 μg/m^3 $PM_{2.5}$. (For a justification, see Appendix B.) The relative risk of mortality corresponding to this increment, using the equations from Section 2, is then $e^{\beta \cdot \Delta x} = e^{\ln(1.01) \cdot 0.07} \approx 1.0007$.

Next we set up a comparison of baseline rates versus rates for baseline plus this increment. In Table **2**, columns labeled "A" represent the baseline, while columns labeled "B" represent

[6] An interactive spreadsheet is also available on request.

[7] Conditional on age, which is linked to the exposure window.

[8] For attributes generally regarded as categorical, such as Medicare eligibility or sex, this "statistically average individual" becomes perhaps more obviously the construct that it is.

baseline plus an increment of 0.1 μg/m^3 $PM_{2.5}$. As described in Section 2, comparing A and B allows us to assess the attributable risk. For baseline rates of mortality (Table **1**; Table **2**, second column), we rely on data for the nine-county Bay Area obtained from the Centers for Disease Control and Prevention (CDC 2021). During any given year, the expected rate or risk for the more exposed scenario (B) should be 1.0007 times that for A. Given this ratio, and the age-specific annual mortality rates for A, we calculate the age-specific annual mortality rates for B (Table **2**, under "Incidence Rate").

The probability of surviving any given year is equal to one minus the risk of mortality during that year. The columns labeled "Survival" in Table **2** contain the cumulative products of these annual probabilities; they represent the overall probabilities of survival from age 55 until the end of any given year. Given our assumptions, we calculate the difference (A - B) at the end of the 30-year exposure window to be 54.3654% - 54.3419% = 0.0235% = 2.3×10^{-4}.

Off-site worker. For a worker receptor, the Air District's cancer-risk HRA methodology (OEHHA 2015; BAAQMD 2021) specifies a 25-year exposure duration. Work is assumed to end with retirement at age 65, so the exposure window for seniors is unsuitable for workers. However, the same principle applies: older workers are generally expected to experience a higher risk of mortality for the same level of $PM_{2.5}$. Thus, for worker receptors, we adopt a 25-year window of exposure that begins at age 40 and ends with age 64.

Basic assumptions for an off-site worker receptor include a schedule of 8 hours per day, 5 days per week, 250 days per year. Consistent with existing HRA guidance (OEHHA 2015; BAAQMD 2021), we also apply a default "worker adjustment factor" (WAF) of $\frac{24}{8} \times \frac{7}{5}$ = 4.20 to the average exposure intensity, to account for a scenario in which the source's operations and the receptor's schedule overlap to a large degree.[9] For our reference increment of +0.1 μg/m^3 in the modeled annual average concentration, this results in a mortality-risk score of 90.5208% - 90.5122% = 0.0086% = 8.6×10^{-5}. Calculations are shown in Table **3**.

Pediatric asthma onset. We calculate the risk of pediatric asthma onset in the same way. In this case, "survival" translates to remaining asthma-free. The relevant schedule at a daycare or K-8 school is assumed to be 10 hr/day, 5 day/wk, 180 day/yr, and the relevant exposure windows are ages 0–5 and 5–13, respectively. and To account for potential overlap with the source's schedule, we apply a default adjustment factor of $\frac{24}{10} \times \frac{7}{5}$ = 3.36. The overall ratio of incremental exposure intensity to incremental modeled concentration is therefore $\frac{180}{365} \times \frac{10}{24} \times 3.36$ = 0.69. For a daycare receptor, we calculate the increased risk corresponding to our reference increment of +0.1 μg/m^3 to be 87.8488% - 87.8141% = 3.5×10^{-4} (Table **4**). For a receptor at a K-8 school, it is 2.4×10^{-4} (Table **5**).

[9] The WAF is a good example of a parameter that may be refined using site-specific information. In this document, we focus on screening-level calculations.

In these screening-level calculations of the risk of pediatric asthma onset for a residential receptor, the fraction of time at home (FAH) is assumed to be 100% for ages 0–15, consistent with (BAAQMD 2021).[10] We calculate the corresponding risk to be 80.0128% - 79.9381% = 7.5×10^{-4} (Table **6**).

Lookup table. Table **7** summarizes the results that we obtain, following the steps above, for $PM_{2.5}$ increments spanning several orders of magnitude. Values from this table can be linearly interpolated to yield good approximations of exact calculations for intermediate values.

Some adults and children will be more at risk. The next section completes the methodology by accounting for variation in sensitivity among individuals.

[10] Air District guidance for cancer-risk assessment allows relaxation of this assumption if no schools are identified within the corresponding 1.0×10^{-6} isopleth (BAAQMD 2021).

4. Sensitive Individuals

Up to this point, calculations have assumed a maximal annual average exposure, but apart from the selection of an exposure window, no consideration has yet been given to other factors relevant to a maximal risk. Other factors include:

1. Factors on the pathway from concentration to dose (e.g., indoor/outdoor ratios; breathing rates; etc.); and
2. Factors that mediate dose-response relationships (e.g., co-stressors, pre-existing conditions, other predispositions, etc.)

The focus of this methodology is on maximal risks. As such, potential variation in the factors above must be considered. During the development of this methodology, the Air District's Advisory Council determined that available evidence supported factors of at least three to account for known and unknown variation.

Taking the above into consideration, we can complete the picture by accounting for variation in two ways. First, we can adjust the exposure intensities for different receptors to reflect variation in factors on the pathway from concentration to exposure or dose. Second, we can adjust the estimates of relative risk to compensate for individuals who exhibit a larger or more severe exposure-response or dose-response relationship. We can also do this to account for data deficiencies. Table **8** summarizes these factors, and the calculations to which we apply them. Instead of re-working the calculations of the preceding section step-by-step, we conclude by providing a final lookup table that reflects these considerations (Table **11**).

Breathing rates. Variation in breathing rates is accounted for in current HRA guidance concerning the risk of cancer. It is well established that children breathe more air than adults per kg of body mass. For our pediatric asthma onset calculations, this fact has generally been captured, as the relevant study excluded adults (Tétreault et al. 2016). However, among different children, as well as adults, there is also individual variation: conditional on age, 95th percentiles of average daily breathing rates are approximately 60% higher than means, and 8-hour moderate activity rates can be four times as high as daily rates (OEHHA 2012 chap. 3; 2015).

Table **9** shows the breathing rate data we use to adjust results for all receptors and endpoints. For daycare, school, and off-site worker receptors, we select 95th percentile moderate-activity 8-hour rates; for residential receptors, we select 95th percentile daily rates. We then divide those rates by the mean daily rates for the corresponding ages, and use the resulting ratios (Table **10**) to scale the average exposure intensities (Δx) in our multi-year calculations.

Sensitive groups. To characterize variation in the relative risks of premature mortality among seniors, we have an empirical basis: important studies of $PM_{2.5}$ report effect sizes for sensitive groups—including seniors of color, seniors eligible for Medicaid, and seniors residing in low-income ZIP codes—that are two to three times the average (e.g., Di et al. 2017; Yazdi et al.

2021). On reviewing the relevant evidence presented at the Advisory Council meeting in July 2022, public commenters recommended "factor(s) higher than 3x" to "safeguard the most vulnerable." The Advisory Council Co-Chairs stated that "we need the largest safety or uncertainty factor that [the Air District] can possibly support," seeing no reasons not to have a "robust" factor and, conversely, "many reasons to go in that direction." The Advisory Council further advised that it would be "no problem to support a three-fold [factor] for vulnerable subpopulations based on the data that [the Air District] presented, but that even more could be supported." Taking this into account, we scale the population-average relative risk of premature mortality (RR = 1.01) by a factor of 3, resulting in a relative risk of 1.03 per 1 $\mu g/m^3$.[11]

Data deficiencies. There are gaps in the data concerning other endpoints and groups, where variations in impacts are not yet adequately quantified. To compensate for this, the Advisory Council remarked that a factor of three is conventionally used by default, and that this factor should be included "at a minimum." Taking this into account, we adopt a factor of three for data deficiencies concerning pediatric asthma onset and premature mortality among working-age adults. The adjusted relative risks for those receptors and endpoints are then 1.99 per 6.53 $\mu g/m^3$ and 1.03 per 1 $\mu g/m^3$, respectively.

Lookup table. Table **11** summarizes the corresponding results for $PM_{2.5}$ increments spanning several orders of magnitude. The next section discusses Table **11** in more detail.

[11] To adjust by a multiplicative factor a, the formula is $RR_{adj} = 1 + [a \cdot (RR - 1)]$.

5. Discussion and Conclusion

This update on modeling risk from local sources of fine particulate matter ($PM_{2.5}$) makes several important advances beyond the previous draft (BAAQMD 2022). The updated methodology:

- Accounts for variations in sensitivity;
- Expands the set of health endpoints and populations considered, by including pediatric asthma onset;
- Improves consistency with existing HRA methods, by calculating risk differences for multi-year exposure windows; and
- Provides a screening table, spanning several orders of magnitude, that can be used when $PM_{2.5}$ concentrations have already been modeled.

The response functions that we leverage are derived from population-based studies in which a cohort of individuals is followed over a long period of time, and small contrasts in modeled or measured $PM_{2.5}$ concentrations are observed. Within a policy-relevant range of baseline $PM_{2.5}$ concentrations, from potentially 5 $\mu g/m^3$ to 15 $\mu g/m^3$ or higher, estimates of the average marginal impacts of the increments we have considered in Tables **7** and **11** will therefore be well supported.

The US EPA's air toxics program "seeks to protect the greatest number of individuals from a lifetime cancer risk greater than 1×10^{-6} and in all cases limit risk to the individual most exposed to no greater than 1×10^{-4}" (Fann et al. 2016). Given an increment of 0.1 $\mu g/m^3$ $PM_{2.5}$, we calculate a maximal excess risk of premature mortality to be 1.1×10^{-3} for a residential receptor (Table **11**). For worker receptors, although breathing rates are higher (Table **10**), lower baseline mortality rates (Table **1**) mean that the net result is slightly lower (9.6×10^{-4}). In terms of pediatric asthma onset, we calculate an excess risk of 3.0×10^{-3} for a residential receptor. In the context of a daycare, the exposure window is shortened to ages 0–5, but higher breathing rates and higher baseline rates result in a larger net result (3.6×10^{-3}). In all cases, the values reported in Table **11** can be linearly interpolated to yield screening-level estimates for larger or smaller increments of $PM_{2.5}$ (see Technical Notes). We report values to two significant digits to support that interpolation.

In the case of larger sources, estimating impacts on a local population (Hubbell et al. 2009) can be a valuable complement to this methodology. Such an approach has been recommended by OEHHA (2012) as a complement to MEI-focused risk assessments. Presently, the Air District models annual health and welfare impacts for the regional population using BenMAP-CE (US EPA 2022; e.g., Tanrikulu et al. 2011, 2022), and has done so for sub-populations as small as 1 million residents (e.g., Fang et al. 2021a, 2021b).

Finally, while the methodology we have developed here can calculate risk, it cannot determine acceptable levels of risk. Work remains to establish appropriate thresholds for risk management.

6. Figures and Tables

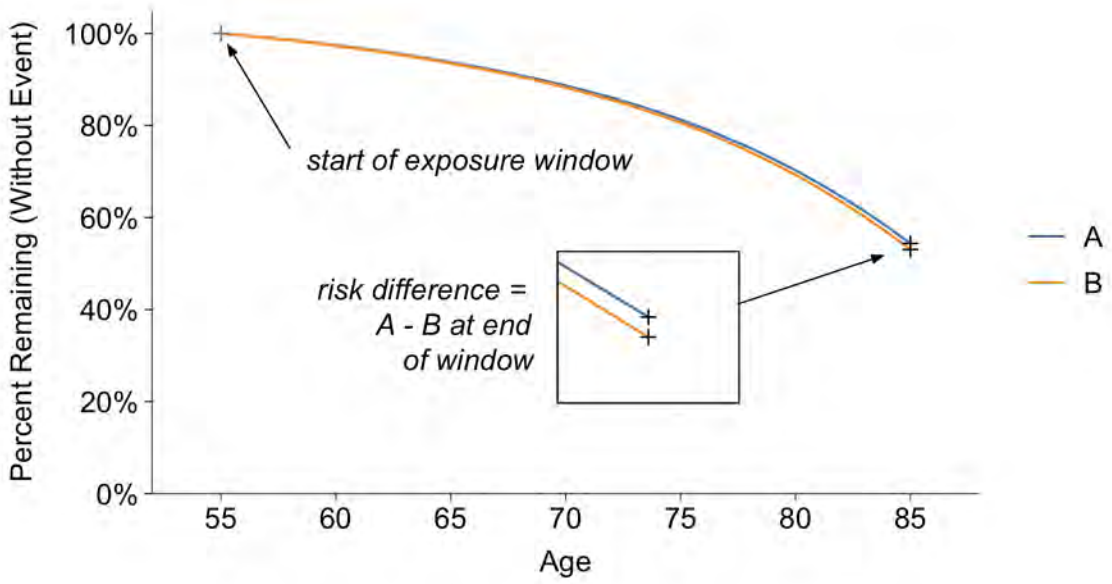

Figure 1: Illustration of the method applied to a multi-year exposure window. B is consistently exposed to more $PM_{2.5}$ than A.

Table 1: Mortality data for the nine-county Bay Area, 2007-2016 (CDC-WONDER).

Age	Person-Years	Deaths	Rate per 100k
40	1,076,380	1,142	106.1
41	1,070,670	1,311	122.4
42	1,075,763	1,446	134.4
43	1,083,573	1,620	149.5
44	1,087,421	1,744	160.4
45	1,089,624	1,852	170.0
46	1,086,099	2,138	196.9
47	1,087,746	2,349	216.0
48	1,075,963	2,552	237.2
49	1,078,475	2,845	263.8
50	1,075,008	3,137	291.8
51	1,072,370	3,342	311.6
52	1,062,414	3,583	337.3
53	1,044,307	3,950	378.2
54	1,028,359	4,199	408.3
55	1,005,568	4,566	454.1
56	982,292	4,743	482.8
57	961,176	4,806	500.0
58	935,149	5,241	560.4
59	908,344	5,546	610.6
60	883,480	5,784	654.7
61	849,086	6,077	715.7
62	810,649	6,133	756.6
63	762,413	6,340	831.6
64	728,685	6,428	882.1
65	691,688	6,576	950.7
66	648,937	6,463	995.9
67	605,206	6,711	1,108.9
68	564,743	6,666	1,180.4
69	527,713	6,879	1,303.5
70	483,456	6,980	1,443.8
71	458,660	6,977	1,521.2
72	432,137	7,431	1,719.6
73	406,466	7,650	1,882.1
74	381,014	7,907	2,075.3
75	357,940	8,313	2,322.5
76	341,736	8,821	2,581.2
77	327,610	9,111	2,781.1
78	311,751	9,767	3,132.9
79	295,780	10,242	3,462.7
80	279,343	11,109	3,976.8
81	266,362	11,775	4,420.7
82	253,935	12,264	4,829.6
83	239,396	13,302	5,556.5
84	224,786	14,031	6,241.9

Table 2: Mortality rates and cumulative probabilities of survival for an average senior, age 55–84. Columns labeled "A" represent exposure to a baseline concentration of $PM_{2.5}$. Columns labeled "B" represent baseline plus an increment of 0.1 μg/m^3.

	Incidence Rate (per 100,000)		Survival (Cumulative)	
Age	**A**	**B**	**A**	**B**
55	454.07	454.39	99.54593%	99.54561%
56	482.85	483.19	99.06527%	99.06462%
57	500.01	500.36	98.56993%	98.56894%
58	560.45	560.84	98.01750%	98.01613%
59	610.56	610.99	97.41904%	97.41726%
60	654.68	655.14	96.78126%	96.77905%
61	715.71	716.21	96.08858%	96.08590%
62	756.55	757.08	95.36162%	95.35846%
63	831.57	832.15	94.56862%	94.56493%
64	882.14	882.75	93.73440%	93.73016%
65	950.72	951.38	92.84325%	92.83843%
66	995.94	996.63	91.91859%	91.91317%
67	1,108.88	1,109.65	90.89932%	90.89326%
68	1,180.36	1,181.18	89.82638%	89.81964%
69	1,303.55	1,304.46	88.65545%	88.64798%
70	1,443.77	1,444.78	87.37547%	87.36722%
71	1,521.17	1,522.23	86.04634%	86.03729%
72	1,719.59	1,720.79	84.56669%	84.55676%
73	1,882.08	1,883.39	82.97508%	82.96423%
74	2,075.25	2,076.70	81.25314%	81.24131%
75	2,322.46	2,324.07	79.36607%	79.35321%
76	2,581.23	2,583.03	77.31745%	77.30349%
77	2,781.05	2,782.99	75.16721%	75.15214%
78	3,132.95	3,135.13	72.81226%	72.79602%
79	3,462.71	3,465.12	70.29099%	70.27355%
80	3,976.83	3,979.60	67.49563%	67.47694%
81	4,420.68	4,423.76	64.51187%	64.49193%
82	4,829.58	4,832.95	61.39622%	61.37507%
83	5,556.48	5,560.36	57.98474%	57.96240%
84	6,241.94	6,246.29	**54.36537%**	**54.34190%**

Table 3: Mortality rates and cumulative probabilities of survival for an average off-site worker receptor, age 40–64. Columns labeled "A" represent exposure to a baseline concentration of $PM_{2.5}$. Columns labeled "B" represent baseline plus an increment of 0.1 μg/m^3.

	Incidence Rate (per 100,000)		Survival (Cumulative)	
Age	**A**	**B**	**A**	**B**
40	106.10	106.20	99.89390%	99.89380%
41	122.45	122.56	99.77159%	99.77137%
42	134.42	134.54	99.63748%	99.63713%
43	149.50	149.65	99.48851%	99.48803%
44	160.38	160.53	99.32896%	99.32832%
45	169.97	170.13	99.16013%	99.15933%
46	196.85	197.04	98.96493%	98.96395%
47	215.95	216.16	98.75122%	98.75003%
48	237.18	237.41	98.51700%	98.51559%
49	263.80	264.05	98.25711%	98.25546%
50	291.81	292.09	97.97038%	97.96846%
51	311.65	311.94	97.66506%	97.66286%
52	337.25	337.57	97.33569%	97.33317%
53	378.24	378.60	96.96752%	96.96467%
54	408.32	408.71	96.57158%	96.56836%
55	454.07	454.51	96.13308%	96.12946%
56	482.85	483.31	95.66890%	95.66485%
57	500.01	500.49	95.19055%	95.18606%
58	560.45	560.98	94.65706%	94.65208%
59	610.56	611.14	94.07912%	94.07362%
60	654.68	655.31	93.46319%	93.45715%
61	715.71	716.39	92.79427%	92.78763%
62	756.55	757.28	92.09223%	92.08497%
63	831.57	832.36	91.32642%	91.31849%
64	882.14	882.98	**90.52079%**	**90.51217%**

Table 4: Baseline incidence rates and cumulative probabilities of remaining asthma-free from ages 0–5, representing an average child at a daycare. Columns labeled "A" represent exposure to a baseline concentration of $PM_{2.5}$. Columns labeled "B" represent baseline plus an increment of 0.1 µg/m^3.

	Incidence Rate (per 100,000)		Asthma-Free (Cumulative)	
Age	**A**	**B**	**A**	**B**
0	2,340.00	2,347.07	97.66000%	97.65293%
1	2,340.00	2,347.07	95.37476%	95.36095%
2	2,340.00	2,347.07	93.14299%	93.12277%
3	2,340.00	2,347.07	90.96344%	90.93712%
4	2,340.00	2,347.07	88.83490%	88.80276%
5	1,110.00	1,113.35	**87.84883%**	**87.81408%**

Table 5: Baseline incidence rates and cumulative probabilities of remaining asthma-free from ages 5–13, representing an average student at a K-8 school. Columns labeled "A" represent exposure to a baseline concentration of $PM_{2.5}$. Columns labeled "B" represent baseline plus an increment of 0.1 µg/m^3.

	Incidence Rate (per 100,000)		Asthma-Free (Cumulative)	
Age	**A**	**B**	**A**	**B**
5	1,110.00	1,113.35	98.89000%	98.88665%
6	1,110.00	1,113.35	97.79232%	97.78569%
7	1,110.00	1,113.35	96.70683%	96.69699%
8	1,110.00	1,113.35	95.63338%	95.62042%
9	1,110.00	1,113.35	94.57185%	94.55582%
10	1,110.00	1,113.35	93.52210%	93.50308%
11	1,110.00	1,113.35	92.48401%	92.46207%
12	440.00	441.33	92.07708%	92.05400%
13	440.00	441.33	**91.67194%**	**91.64774%**

Table 6: Baseline incidence rates and cumulative probabilities of remaining asthma-free from ages 0–17, representing an average residential receptor. Columns labeled "A" represent exposure to a baseline concentration of $PM_{2.5}$. Columns labeled "B" represent baseline plus an increment of 0.1 µg/m^3.

	Incidence Rate (per 100,000)		Asthma-Free (Cumulative)	
Age	**A**	**B**	**A**	**B**
0	2,340.00	2,349.82	97.66000%	97.65018%
1	2,340.00	2,349.82	95.37476%	95.35558%
2	2,340.00	2,349.82	93.14299%	93.11489%
3	2,340.00	2,349.82	90.96344%	90.92686%
4	2,340.00	2,349.82	88.83490%	88.79024%
5	1,110.00	1,114.66	87.84883%	87.80054%
6	1,110.00	1,114.66	86.87371%	86.82186%
7	1,110.00	1,114.66	85.90941%	85.85409%
8	1,110.00	1,114.66	84.95581%	84.89711%
9	1,110.00	1,114.66	84.01280%	83.95080%
10	1,110.00	1,114.66	83.08026%	83.01504%
11	1,110.00	1,114.66	82.15807%	82.08970%
12	440.00	441.85	81.79658%	81.72699%
13	440.00	441.85	81.43667%	81.36588%
14	440.00	441.85	81.07835%	81.00637%
15	440.00	441.85	80.72161%	80.64845%
16	440.00	441.35	80.36643%	80.29251%
17	440.00	441.35	**80.01282%**	**79.93814%**

Table 7: Screening-level risk scores calculated without adjusting for variations in sensitivity. Exposure windows are indicated in parentheses.

	Pediatric Asthma Onset			Premature Mortality	
Annual Average Concentration Increment	**Daycare (0–5)**	**Student (5–13)**	**Resident (0–17)**	**Worker (40–64)**	**Resident (55–84)**
3×10^{-1} µg/m³	1.0×10^{-3}	7.3×10^{-4}	2.2×10^{-3}	2.6×10^{-4}	7.0×10^{-4}
1×10^{-1} µg/m³	3.5×10^{-4}	2.4×10^{-4}	7.5×10^{-4}	8.6×10^{-5}	2.3×10^{-4}
3×10^{-2} µg/m³	1.0×10^{-4}	7.3×10^{-5}	2.2×10^{-4}	2.6×10^{-5}	7.0×10^{-5}
1×10^{-2} µg/m³	3.5×10^{-5}	2.4×10^{-5}	7.5×10^{-5}	8.6×10^{-6}	2.3×10^{-5}
3×10^{-3} µg/m³	1.0×10^{-5}	7.2×10^{-6}	2.2×10^{-5}	2.6×10^{-6}	7.0×10^{-6}
1×10^{-3} µg/m³	3.5×10^{-6}	2.4×10^{-6}	7.5×10^{-6}	8.6×10^{-7}	2.3×10^{-6}

Consistent with screening-level HRA guidance from BAAQMD (2021), for a residential receptor the assumed fraction of time at home (FAH) is 100% for age 0–15 and 73% for age 16 and older, 350 days per year. Schedule parameters for an off-site worker receptor are 8 hr/day, 250 day/yr, with an adjustment factor of 4.2 applied to account for potential overlap in the schedules of the source and receptor. For a school or daycare receptor, schedule parameters are 10 hr/day, 180 day/yr, with an adjustment factor of 3.36.

The population-average relative risk for premature adult mortality is taken to be 1.01 per 1 ug/m3. For pediatric asthma onset, it is 1.33 per 6.53 ug/m3. Baseline rates for mortality are obtained from CDC-WONDER for the 9-county Bay Area, while those for asthma incidence are obtained from BenMAP.

Table 8: Factors applied to account for variations in individual response. See also Tables **9** and **10**.

Endpoint/Receptor	Factor	Description
(all)	(varies)	Age- and activity-specific breathing rates.
Mortality (senior)	3x	Consistent with epidemiological data for at-risk groups.
Mortality (worker)	3x	Default factor for data deficiencies.
Asthma onset	3x	Default factor for data deficiencies.

Table 9: Breathing rates (L/kg-day) by level of activity, summary statistic, and age. Values obtained from Tables 5.7 and 5.8 of OEHHA (2015).

Type	Statistic	0-1	2-15	> 16*
Daily	Mean	658	452	185
Daily	95th percentile	1,090	745	290
Moderate 8-hr	Mean	2,670	1,140	510
Moderate 8-hr	95th percentile	3,600	1,560	690

* Original data are for ages 16-70.

Table 10: Factors applied to account for variation in breathing rates. Values derived from Table **9**, as described in the main text (Section 4).

Receptor	Age	Factor
Resident	0–1	1.7x
Resident	2–15	1.6x
Resident	16–17	1.6x
Resident	55–84	1.6x
Worker	40–64	3.7x
Daycare	0–1	5.5x
Daycare	2–5	3.5x
Student	5–13	3.5x

Values rounded to one decimal.

Table 11: Screening-level risk scores that incorporate potential variations in sensitivity. Exposure windows are indicated in parentheses.

	Pediatric Asthma Onset			Premature Mortality	
Annual Average Concentration Increment	**Daycare (0–5)**	**Student (5–13)**	**Resident (0–17)**	**Worker (40–64)**	**Resident (55–84)**
3×10^{-1} µg/m³	1.1×10^{-2}	6.3×10^{-3}	9.1×10^{-3}	2.9×10^{-3}	3.3×10^{-3}
1×10^{-1} µg/m³	3.6×10^{-3}	2.0×10^{-3}	3.0×10^{-3}	9.6×10^{-4}	1.1×10^{-3}
3×10^{-2} µg/m³	1.1×10^{-3}	6.1×10^{-4}	8.9×10^{-4}	2.9×10^{-4}	3.3×10^{-4}
1×10^{-2} µg/m³	3.5×10^{-4}	2.0×10^{-4}	3.0×10^{-4}	9.6×10^{-5}	1.1×10^{-4}
3×10^{-3} µg/m³	1.1×10^{-4}	6.0×10^{-5}	8.9×10^{-5}	2.9×10^{-5}	3.3×10^{-5}
1×10^{-3} µg/m³	3.5×10^{-5}	2.0×10^{-5}	3.0×10^{-5}	9.6×10^{-6}	1.1×10^{-5}

Consistent with screening-level HRA guidance from BAAQMD (2021), for a residential receptor the assumed fraction of time at home (FAH) is 100% for age 0–15 and 73% for age 16 and older, 350 days per year. Schedule parameters for an off-site worker receptor are 8 hr/day, 250 day/yr, with an adjustment factor of 4.2 applied to account for potential overlap in the schedules of the source and receptor. For a school or daycare receptor, schedule parameters are 10 hr/day, 180 day/yr, with an adjustment factor of 3.36.

Average exposure intensities are adjusted using age-specific 95th percentile breathing rates from OEHHA (2015). Moderate-activity 8-hr rates are used for worker, student, and daycare receptors; daily rates are used for residential receptors.

To account for variations in effect size, population-average relative risks for premature adult mortality and pediatric asthma onset are each adjusted by a factor of three, resulting in RR = 1.03 per 1 ug/m3 and RR = 1.99 per 6.53 ug/m3, respectively. Baseline rates for mortality are obtained from CDC-WONDER for the 9-county Bay Area, while those for asthma incidence are obtained from BenMAP.

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Appendix A. Technical Notes

The reader who is more familiar with cancer-risk calculations may note two distinct features of the delta-response equation that is central to this methodology. First, it is nonlinear in the term representing $PM_{2.5}$ (Δx). Second, it includes a term representing baseline conditions (y_0). These features have a few practical consequences.

First, in modeling an increase of Δx μg/m^3, the baseline is conceptually defined as the (annual average) $PM_{2.5}$ concentration that would be obtained in the absence of the modeled source's contribution. Importantly, the baseline incidence rate y_0 is also assumed to correspond to that scenario. In modeling a reduction of Δx μg/m^3, with $\Delta x > 0$, the equation $\Delta y = y_0\left(1 - e^{-\beta\,\Delta x}\right)$ should instead be used, with $\Delta y > 0$ interpreted as a benefit. Using the wrong setup/equation will not result in a very large error; for mortality, given a plausible value for Δx, the error will be a few percent at most. This asymmetry is absent from conventional cancer-risk assessments, where the key equation is linear in Δx. It is present in population-level assessments conducted by scientists and professionals—using tools such as BenMAP-CE, for example—that use the same equations.

Second, risk scores will not accumulate exactly in the way that they do in a linear framework. The calculated risk for an increment of 0.1 μg/m^3 will in fact be slightly *more* than ten times that for an increment of 0.01 μg/m^3. (This can be observed in Table **11**.) It may help to re-conceptualize this situation in terms of ten *successive* increments of 0.01 μg/m^3. In this case it becomes clear that updating y_0 after each increment should be necessary, as the additional $PM_{2.5}$ should increase it. The importance of the potential discrepancy varies with the size of β, and with the sizes of the $PM_{2.5}$ increments. For this particular example, among the endpoints and receptors we have considered, the potential discrepancies should amount to a few percent at most.

Appendix B. Frequently Asked Questions

Questions and comments received during review of prior drafts and presentations are captured in this section.

Q. These risks seem very high. Can small amounts of $PM_{2.5}$ really be this big of a risk driver?

Yes. In the Bay Area, current levels of $PM_{2.5}$ are responsible for thousands of premature deaths each year, and even more cases of asthma. Relatively small changes in $PM_{2.5}$ at or around baseline levels are the subject of epidemiological studies on which this methodology is based. Sensitive individuals will be more at risk, given the same increase in exposure.

Q. Why did you select these particular estimates of relative risk?

For premature adult mortality, the value we selected (1.01 per 1 $\mu g/m^3$) is consistent with the ranges reported in the District's recent evaluations of impacts on regional populations (Fang et al. 2021a, 2021b; Tanrikulu et al. 2022). It is also consistent with the estimates reported by Di et al (2017): 1.073 overall per +10 $\mu g/m^3$, and 1.136 per +10 $\mu g/m^3$ for exposures less than 12 $\mu g/m^3$. Di et al (2017) is the core study on which the US EPA relies for estimates of attributable mortality among seniors (US EPA 2022). Yazdi et al (2021) arrive at similar results using different methods, again studying baseline exposures under 12 $\mu g/m^3$. Vodonos et al (2018), summarizing a wide range of studies across all ages via meta-regression, arrive at a relative risk of 1.0129 per +1 $\mu g/m^3$ for a baseline centered on 10 $\mu g/m^3$.

In the Bay Area, about 98% of the residential population lives where a modeled annual average $PM_{2.5}$ concentration[12] is less than 12 $\mu g/m^3$, and 75% where it is less than 10 $\mu g/m^3$. Recent meta-analyses indicate that marginal effects on mortality are at least as large at these baseline levels (Vodonos et al. 2018; Papadogeorgou et al. 2019), and appear to be larger, compared to the historically higher levels that were the basis of older studies. This lends additional weight to the newer studies cited above.

For pediatric asthma calculations, we use the value supplied by the US EPA's BenMAP-CE platform: 1.33 per 6.53 $\mu g/m^3$ (US EPA 2022). The mean $PM_{2.5}$ concentration in the supporting study was approximately 10 $\mu g/m^3$ (Tétreault et al. 2016).

Q. What about other health effects, like those on reproduction or cognition?

During earlier development, this methodology was restricted to premature adult mortality. In a conventional population-wide assessment, mortality typically receives over 95% of the overall valuation. However, feedback from stakeholders indicated that it was critical to assess at least one other endpoint. Respiratory effects, and asthma in particular, figure prominently in the concerns of community members and community representatives. Asthma can be measured in many ways: hospitalizations; inhaler use; progression; and new onset, to name a few. Asthma

[12] The Air District's modeling currently excludes wildfire impacts.

onset (newly developed or diagnosed asthma) was selected because it receives the highest valuation in the District's current population-based assessments, and because it is a necessary condition for other metrics, such as hospitalizations.

Importantly, this methodology does not attempt to consolidate multiple risk scores, nor does it attempt to be exhaustive. $PM_{2.5}$ has very broad effects, and evidence continues to accumulate for reproductive, neurological, and other endpoints. More endpoints could be assessed, if it became clear that this would make a practical difference to policy or risk-management outcomes. Work still remains to establish an appropriate metric, or method for combining multiple metrics, to be used in threshold-based decision-making.

Q. Some communities have higher rates of asthma and mortality. Aren't they more at risk?

Throughout the development of this methodology, this question has been a focus of discussion. People in overburdened communities are more at risk. Quantitatively accounting for this faces limitations in a HRA framework, especially when the framework is focused on modeling maximum potential risk to an individual receptor. There are ways to address the problem at a risk-management or policy level, and we recommend that approach. An example is the Air District's recently updated Regulation 2, Rules 1 and 5, which establish geographically defined "overburdened communities" based on multiple relevant factors, and then establish thresholds that vary according to whether a source is located in or near such a community.

Generally, baseline rates of disease will be higher among at-risk groups and in overburdened communities. Baseline rates can be a good indicator of susceptibility to a particular stressor, but not always. First, rates can be higher in communities that are not otherwise overburdened. This can happen, for example, with mortality in communities that are older but otherwise more well-off. Second, rates can be lower among groups that will be more impacted overall by the same increase in $PM_{2.5}$. Either of these can happen because air pollution is not the only thing that affects baseline rates. So, because the marginal impacts of air pollution are conventionally estimated relative to those rates, we can be led in the wrong direction. As an example: all-cause mortality rates are lower than average among Hispanic/Latino residents. Calculations using those baseline rates, without any additional information, would indicate that lower impacts would result from locating a source of $PM_{2.5}$ in a Hispanic/Latino community. However, additional knowledge points the other way (Di et al. 2017); differences in effect size (β) outweigh these differences in baseline rates (BAAQMD 2022).

We sometimes have geographically resolved information on important predictors of the baseline rate and/or the effect. For example, studies report (varying) results for individual race/ethnicity as a predictor or modifier of the effect size. They also report comparable results for other factors, such as income and Medicaid status. The selections of variables, and the adjustments for other variables—many of which are correlated—are often inconsistent across studies. Integrating results across such studies into a single, coherent adjustment factor for the effect size (β) would be a major challenge, which we do not currently know how to solve. Acknowledging that new scientific understandings will inevitably emerge, the semi-quantitative

factors in Section 4 are intended to be adequately protective of sensitive individuals across multiple dimensions. They can also be protective where data are currently lacking, as in the case of pediatric asthma onset.

A final practical concern is that we do not have individual-level data on potential receptors. Small-area population data can be imprecise, outdated, or inaccurate (Hubbell et al. 2009). This is especially a weakness at the spatial scales that correspond to the distances between most local sources and their MEI receptors, which in urban areas would typically be the size of a Census block or smaller. Results based on such micro-data, which often have unreported sources of error and/or uncertainty, can introduce a false sense of precision and reliability during risk communication or decision-making. This is especially true when used to evaluate maximum impacts. Statistical summaries at a community level—as provided, for example, by BenMAP-CE—are more reliable. But, this methodology is focused on risks for maximally impacted receptors, rather than impacts on the whole of a community.

For these reasons, we have elected to use age-specific but otherwise average baseline rates as a foundation, and cover potential variation in individual sensitivity by using the approach explained in Section 4. Insofar as locally elevated exposures to $PM_{2.5}$ are more frequent and more severe in overburdened communities, the regulatory application of this methodology stands to reduce those disparities in exposure. We also recommend that equity-focused extensions be implemented at a risk management or policy level. These could take the form of refinements to the screening-level parameters that we have provided, or the establishment of context-specific thresholds (for example). To implement the former, Section 4 shows how multiplicative factors can be used to adjust the average exposure intensity (as with breathing rates), or the relative risk per $\mu g/m^3$ (as with sensitive groups), as appropriate.

BAY AREA AIR QUALITY MANAGEMENT DISTRICT
Memorandum

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

From: Sharon L. Landers
Interim Executive Officer/APCO

Date: September 12, 2022

Re: Source Prioritization Framework

RECOMMENDED ACTION

None; receive and file.

BACKGROUND

Last year, Air District staff began updating the rule-making process to improve transparency and predictability. A draft source prioritization framework was developed to align rule efforts to agency priorities and improve transparency with the Board of Directors (Board), advocates, and the regulated community. There are some implications of adopting the framework, which raise questions for the Advisory Council's consideration.

DISCUSSION

Source Prioritization Framework
Staff developed a draft Source Prioritization Framework to prioritize the long list of sources and rules that need further research. The idea is to screen all sources against a set of criteria. The criteria includes commitments, health and equity impacts, legal authority, emission control or reduction potential, and other impacts. All existing commitments, born out of legal requirements or adopted community plans, would be identified first, and weighted most heavily. Commitments would then be ranked by their health and equity impacts, based on the source being controlled. Legislative authority, emission reduction potential and other economic, socio-economic, and other environmental considerations would be considered. Priority factors would also determine the appropriate policy approach. For example, if the Air District does not have regulatory authority over a source, then other strategies would be recommended.

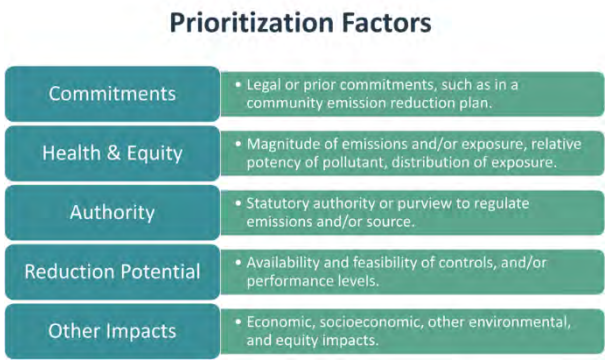

Figure 1. Priority Factors

Implications and Questions for Advisory Council

Implementing the prioritization framework has several implications, described below:

- **Only Commitments Go Forward in Coming Years:** There are many existing rules and source evaluations that the Air District has committed to, either via legal requirements or from commitments made in recently adopted plans. An example is commitments related to Assembly Bill 617. AB 617 requires that the Air District adopt Best Available Control Technology (BARCT) rules, and to adopt community emission reduction plans. The West Oakland plan has prioritized numerous rules that will have an impact on the community, and additional plans will be adopted in the coming years, including Richmond-North Richmond-San Pablo and East Oakland. AB617 has significantly contributed to the list of rulemaking to which the Air District has committed. Therefore, due to limited resources, there will be insufficient resources available in the next few years to take on any new, non-previously committed rule efforts.
- **Climate Related Rules Low Priority:** Direct local health and equity impacts from CO_2, methane, and other greenhouse gases are significantly smaller than from emissions of particulate matter and other air toxics. Since the priority framework weights health and equity highly, efforts to reduce CO_2, methane or other climate pollutants may rank lower in the prioritization, so much so that these sources would not be addressed by rule development for several years.

These implications raise several questions for the Advisory Council.

1. What is the role of rulemaking in addressing climate change? Under current state law, the Air District cannot require reductions of CO_2 from sources subject to cap-and-trade. With limited legal authority over greenhouse gasses, Air District staff have been challenged to find the right role for rulemaking.
2. Considering the implications, namely not addressing non-existing commitments and climate, are the factors the correct ones?

3. Is there more that the Air District staff should consider when quantifying local health impacts from greenhouse gas emissions in this framework, that may change the prioritization of climate-related sources?

Next Steps

Staff would like to discuss the proposed Source Prioritization Framework with the Community Advisory Committee later this year.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Sharon L. Landers
Interim Executive Officer/APCO

Prepared by: Christy Riviere
Reviewed by: Elizabeth Yura and Greg Nudd

ATTACHMENTS:

None