BAY AREA AIR QUALITY MANAGEMENT DISTRICT

BOARD OF DIRECTORS
ADVISORY COUNCIL

COUNCIL 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. Michael Kleinman, PhD, University of California Irvine
Garima Raheja, PhD candidate, Columbia University
Vacant
Vacant
Vacant
David Haubert, Air District Board of Directors Liasion

MEETING LOCATION(S) FOR IN-PERSON ATTENDANCE BY COUNCIL MEMBERS AND MEMBERS OF THE PUBLIC

Bay Area Metro Center
1st Floor Board Room
375 Beale Street
San Francisco, CA 94105

THE FOLLOWING STREAMING OPTIONS WILL ALSO BE PROVIDED

These streaming options are provided for convenience only. In the event that streaming connections malfunction for any reason, the Advisory Council reserves the right to conduct the meeting without remote webcast and/or Zoom access.

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.

Members of the public may participate remotely via Zoom at https://bayareametro.zoom.us/j/89014507527, or may join Zoom by phone by dialing (669) 900-6833 or (408) 638-0968. The Webinar ID for this meeting is: 890 1450 7527

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 a matter on the agenda will have two minutes each to address the Council on that agenda item, unless a different time limit is established by the Co-Chairs. No speaker who has already spoken on an item will be entitled to speak to that item again.

The Council welcomes comments, including criticism, about the policies, procedures, programs, or services of the District, or of the acts or omissions of the Council. Speakers shall not use threatening, profane, or abusive language which disrupts, disturbs, or otherwise impedes the orderly conduct of a Council meeting. The District is committed to maintaining a workplace free of unlawful harassment and is mindful that District staff regularly attend Council meetings. Discriminatory statements or conduct that would potentially violate the Fair Employment and Housing Act – i.e., statements or conduct that is hostile, intimidating, oppressive, or abusive – is *per se* disruptive to a meeting and will not be tolerated.

ADVISORY COUNCIL MEETING AGENDA

MONDAY, SEPTEMBER 11, 2023
9:30 AM

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

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

CONSENT CALENDAR (Items 2 - 3)

2. Approval of the Draft Minutes of the Advisory Council Meeting of January 30, 2023

   The Council will consider approving the Draft Minutes of the Advisory Council meeting of January 30, 2023.

3. Approval of the Draft Minutes of the Advisory Council Meeting of June 12, 2023

   The Council will consider approving the Draft Minutes of the Advisory Council meeting of June 12, 2023.

INFORMATIONAL ITEM(S)

4. Fine Particulate Local Risk Methodology Update

   The Advisory Council will receive and discuss a presentation from staff regarding the proposed methodology for modeling health risks from local sources of fine particulate matter ($PM_{2.5}$).

ACTION ITEM(S)

5. Vote to Submit Letter of Support to Air District Board of Directors

   The Advisory Council will review and consider submitting a letter of support to the Board of Directors for the research and methodology in the white paper, Modeling Health Risks from Local Sources of Fine Particulate Matter $PM_{2.5}$, version 2.0 (August 2023).

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 will be given an opportunity to address the Advisory Council. Members of the public will have two minutes each to address the Council, unless a different time limit is established by the Chair. The Council welcomes comments, including criticism, about the policies, procedures, programs, or services of the District, or of the acts or omissions of the Council. Speakers shall not use threatening, profane, or abusive language which disrupts, disturbs, or otherwise impedes the orderly conduct of a Council meeting. The District is committed to maintaining a workplace free of unlawful harassment and is mindful that District staff regularly attend Council meetings. Discriminatory statements or conduct that would potentially violate the Fair Employment and Housing Act – i.e., statements or conduct that is hostile, intimidating, oppressive, or abusive – is per se disruptive to a meeting and will not be tolerated.

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

At the Call of the Chair.

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 2023

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Advisory Council Meeting	Monday	11	9:30 a.m.	1st Floor Board Room
Board of Directors Stationary Source and Climate Impacts Committee	Wednesday	13	10:00 a.m.	1st Floor, Yerba Buena Room
Board of Directors Mobile Source and Climate Impacts Committee - CANCELLED	Wednesday	13	1:00 p.m.	1st Floor, Yerba Buena Room
Community Advisory Council Retreat (2-day event)	Thurs/Fri	14/15	11:00 a.m. / 8:00 a.m.	Sheraton Sonoma Wine Country Petaluma Hotel Ballroom 745 Baywood Drive Petaluma, CA 94954
Board of Directors Meeting	Wednesday	20	9:00 a.m.	1st Floor Board Room
Board of Directors Community Equity, Health and Justice Committee	Wednesday	20	1:00 p.m.	1st Floor Board Room

OCTOBER 2023

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Board of Directors Meeting	Wednesday	4	9:00 a.m.	1st Floor Board Room
Board of Directors Legislative Committee	Wednesday	4	11:30 a.m.	1st Floor Board Room
Board of Directors Finance and Administration Committee	Wednesday	4	1:00 p.m.	1st Floor Board Room
Board of Directors Stationary Source and Climate Impacts Committee	Wednesday	11	10:00 a.m.	1st Floor, Yerba Buena Room
Board of Directors Mobile Source and Climate Impacts Committee	Wednesday	11	1:00 p.m.	1st Floor, Yerba Buena Room
Board of Directors Meeting	Wednesday	18	9:00 a.m.	1st Floor Board Room
Board of Directors Finance and Administration Committee	Wednesday	18	11:30 a.m.	1st Floor Board Room

OCTOBER 2023

TYPE OF MEETING	DAY	DATE	TIME	ROOM
Board of Directors Community Equity, Health and Justice Committee	**Wednesday**	**18**	**1:00 p.m.**	**1st Floor Board Room**

MB 9/06/2023 – 5:10 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: Philip M. Fine
Executive Officer/APCO

Date: September 11, 2023

Re: Approval of the Draft Minutes of the Advisory Council Meeting of January 30, 2023

RECOMMENDED ACTION

The Council will consider approving the draft minutes of the Advisory Council meeting of January 30, 2023.

BACKGROUND

None.

DISCUSSION

Attached for your review and approval are the Draft Minutes of the Advisory Council meeting of January 30, 2023.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Philip M. Fine
Executive Officer/APCO

Prepared by: Marcy Hiratzka
Reviewed by: Vanessa Johnson

ATTACHMENTS:

1. Draft Minutes of the Advisory Council Meeting of January 30, 2023

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

Advisory Council Meeting
Monday, January 30, 2023

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 authorized by Assembly Bill 361 (Rivas 2021), allowing remote meetings. Members of the Advisory Council participated by teleconference.

CALL TO ORDER

1. **Opening Comments:** Advisory Council (Council) Co-Chairperson, Dr. Gina Solomon, called the meeting to order at 8:33 a.m.

   Roll Call:

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

   Absent: Dr. Pallavi Phartiyal.

2. **PUBLIC MEETING PROCEDURE**

   At this point in the meeting, the Council wished to take a formal vote to transfer this meeting's facilitation from that of Co-Chair Rudolph to Vice Chair Kleinman, at Dr. Solomon's request.

Council Action

Board Liaison Haubert made a motion, seconded by Dr. Cullenward, to **designate** Vice Chair Kleinman as the facilitator for the Advisory Council meeting of January 30, 2023; 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.

3. APPROVAL OF THE ADVISORY COUNCIL MEETING MINUTES OF SEPTEMBER 12, 2022

Public Comments

No requests received.

Council Comments

None.

Council Action

Board Liaison Haubert made a motion, seconded by Dr. Cullenward, to **approve** the Minutes of the Advisory Council meeting of September 12, 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. UPDATE ON THE PROPOSED METHODOLOGY FOR DETERMINING LOCAL HEALTH RISKS FROM FINE PARTICULATE MATTER ($PM_{2.5}$)

Greg Nudd, Deputy Air Pollution Control Officer of Policy, introduced Dr. David Holstius, Senior Advanced Projects Advisor, who gave the staff presentation *Local Risk Methodology Update*, including: overview; recap of progress during 2022; infiltration; recommendation; revised net adjustments; methodological considerations; public comments – overview and methodology; and effect size.

Public Comments

No requests received.

Council Comments

The Council and staff discussed the potential application of $PM_{2.5}$ infiltration factors to the proposed methodology. Councilmember Raheja inquired whether a recent study of infiltration factors during a wildfire would be helpful, and/or a plot of concentration versus infiltration factor; staff explained that the factors need to correspond to the conditions in the studies from which effect size estimates were derived. Co-Chair Solomon inquired whether staff had identified studies that more closely parallel the Bay Area's housing conditions. Staff clarified that they had, and that the numerator would be the term to reflect those, while the denominator should correspond to the conditions of the epidemiological studies; since the numerator is intended to reflect a 95^{th} percentile or similar, its value should be at least 0.9, but that 1 is recommended, in order to align with the cancer-risk framework, where the receptor is effectively unsheltered; this also addresses a previous Council comment about whether unsheltered groups were being accounted for. Co-Chair Solomon agreed that this was a sensible approach.

Councilmember Cullenward thanked staff for the provision of confidence intervals for published relative risks, and for the explanation of how they were standardized to unit concentrations. The Council expressed support for an administratively streamlined approach to selecting effect-size estimates, that settling on a representative choice while retaining supporting evidence is reasonable, and that continuing to follow the progress of the US EPA in determining effect sizes is warranted.

Director Haubert brought up that the Bay Area has unique approaches to particulate matter (PM) regulation that do not exist elsewhere, and asked whether a Bay Area specific study would be possible or had been considered. Staff responded that the specific conditions of the Bay Area will be carefully considered when policy applications are considered, that a conversation with Board members on that topic is needed, and that the intent of this methodology is to provide a number, likely based on national studies with the required amount of data to form reliable estimates of risk. Co-Chair Solomon added that PM is unusual in how well it has been studied in many areas, and that trying to replicate that level of effort in the Bay Area could take several decades. Co-Chair Solomon then remarked that the approach taken to arrive at an effect-size should be described as a "convergence," rather than an "approximation," since there are multiple studies, including meta-analyses, that are converging on the same number; that this is showing consistency of the epidemiologic evidence, and the approach taken by staff is correct, looking at multiple lines of evidence, without attempting a new meta-analysis. Acting Co-Chair Kleinman concluded by commending the report, noting that it has helped to clarify much of the way to think about this topic, and that further work is needed to understand the implications, but that the methodology appears consistent with the thinking of the US EPA in their ongoing reconsideration of national standards.

Council Action

None; receive and file.

5. COMMENTS ON THE PROPOSED METHODOLOGY FOR DETERMINING LOCAL HEALTH RISKS FROM FINE PARTICULATE MATTER ($PM_{2.5}$)

The Council received presentations from three organizations that provided public comment on the Air District's draft white paper, *Modeling Local Sources of Fine Particulate Matter ($PM_{2.5}$) for Risk Management.*

Firstly, Christine Wolfe of the California Council for Environmental and Economic Balance (CCEEB), gave the presentation *Risk Management and Regulatory Context.* Ms. Wolfe gave recommendations for guiding principles, including: best available science; input and lessons learned from other agencies; speciation and source apportionment; regional vs local impacts and control strategies; economic evaluation; prioritization via near-term cost-effectiveness; avoiding duplication or conflict with other regulations; achievable and easily understood pathways to compliance; and proportionality. Ms. Wolfe noted that, while several comments would be policy-focused—for example, that the approach may be in conflict with the Health and Safety Code, which identifies the Office of Environmental Health Hazard Assessment (OEHHA) as the entity that identifies and establishes health values for Toxic Air Contaminants in consultation with the California Air Resources Board (CARB)—it was important to address the method itself in the context of its application. Ms. Wolfe expressed concern that simplifying assumptions in the method would result neither in expeditious implementation nor ease of

understanding, and that best available data should be used, even where it adds complexity, but that this would also depend on the application context, to balance accuracy versus consistency, if it is intended to support multiple regulatory programs. Regarding the proposed adjustment factors for sensitivity, Ms. Wolfe stated that the proposed methodology is extraordinarily conservative, and asked in which contexts a maximum risk framework would be most appropriate. As an example, Ms. Wolfe asked whether it would be appropriate for every California Environmental Quality Act (CEQA) project review to trigger the highest level of review; or whether the methodology's application would change the number or type of permit applications that go through a Health Risk Assessment (HRA). Ms. Wolfe expressed interest in hearing the Advisory Council discuss how the Air District should communicate and contextualize the screening-level risk scores, noting that the numbers are much higher than those typically discussed in the context of cancer risk. The following recommendations were presented: that the potential applications be prioritized, and the methodology be revised in line with those; that existing regional, statewide, and federal regulations be summarized, to assess potential alignments and/or conflicts with applications of the proposed methodology; that an independent third party publicly conduct a validation of all equations and calculations, via a test case; that staff clarify how the methodology would be updated in the future, including updates for efficacy and accuracy as the Air District's $PM_{2.5}$ inventory changes over time; and that a full regulatory analysis be conducted prior to any application.

Secondly, Ken Szutu, a member of the Air District's Community Advisory Council, gave a presentation on recent activities of the Citizen Air Monitoring Network in Vallejo, of which he is also a member. Mr. Szutu welcomed the attention to local sources and risks, and stated: that modeling needs to reflect the experience of the community; that $PM_{2.5}$ emissions from incidents, and from startup and shutdown, should be included; and that $PM_{2.5}$ speciation is necessary for risk considerations. An aerial photo was shown of a plume from a fire at the NuStar facility on October 15, 2019, as an illustration of an incident affecting the community; a map of the affected area's priority population designations (from California Climate Investments) was also shown; then a chart showing elevated asthma rates in Vallejo. Mr. Szutu concluded by enjoining staff to ensure that the model reflects reality, and covers the experience of the community, specifically including $PM_{2.5}$ releases from industrial incidents, as well as startup and shutdown emissions, and speciation of $PM_{2.5}$.

Finally, Kevin Buchan from the Western States Petroleum Association (WSPA) introduced Dr. Julie Goodman of Gradient, who gave the presentation *Modeling Local Sources of Fine Particulate Matter ($PM_{2.5}$) for Risk Management.* Dr. Goodman argued that the model highly overestimates risks associated with $PM_{2.5}$ increments of 0.001–0.3 ug/m^3, and that the observed associations of $PM_{2.5}$ with mortality in the scientific literature do not necessarily reveal a causal relationship, but are explainable instead by: exposure measurement error (ambient vs personal, indoor vs outdoor); bias (due to the conflation of historically higher levels with estimates used in studies); confounding (by unmeasured confounders, or by imprecise measures); chance; or the wrong model (threshold vs no-threshold, arguing that model and measurement error linearize the exposure-response curve). Dr. Goodman also proposed that there is a level of $PM_{2.5}$ below which the human body will not be adversely affected, and that a threshold-based approach is taken with all currently assessed non-cancer endpoints. Dr. Goodman further stated that the risk estimates are too small to be significant, due to the possibility of bias or confounding, and are so small as to be negligible in comparison with hourly and daily variability in the levels of $PM_{2.5}$ in the Bay Area. With regard to premature mortality, Dr. Goodman stated that the range of mortality risk estimates supported by the literature is much wider than that modeled using the US EPA's BenMAP platform, and that with regard to childhood asthma onset, this has not been fully examined, but is in her opinion not likely accurate or reliable, noting that the risk estimate is based on

a single study. Dr. Goodman then presented charts of daily variability in $PM_{2.5}$ levels across the Bay Area, stating that the ranges of daily or hourly data are much larger than the (annual average) increments considered by the methodology, such that the latter appear negligible. Dr. Goodman concluded by recommending that the model consider using a threshold and look at much larger increments of $PM_{2.5}$.

Public Comments

Public comments were given by Dr. Stephen Rosenblum, Palo Alto, who noted concern with the presentation by Dr. Goodman, specifically in terms of its being sponsored by an industry stakeholder group, and parallels with arguments made decades ago in the context of regulating risks from nuclear radiation, while the precautionary principle justifies action now, rather than waiting for decades for harms to manifest, particularly given the Advisory Council's position on the risks from levels of $PM_{2.5}$ below the current NAAQS; by Janelle Payne, who noted a recent New York Times article reporting that Dr. Goodman provided testimony on behalf of industry stakeholders in a case in Oregon involving exposures to pollution from gas stoves, without acknowledging their sponsorship; and by Bob Brown (on behalf of WSPA), who stated that it was clear that Dr. Goodman's services had been retained by WSPA in the present context.

Council Comments

Co-Chair Solomon expressed that many of the topics related to Dr. Goodman's points had been grappled with very seriously during the Advisory Council's writing of its report on particulate matter. Regarding exposure measurement error in particular, no evidence of differential misclassification was noted, and non-differential misclassification tends to lead to under-estimates of true effects; the effect of the difference between indoor and outdoor levels is an example of the latter, but Dr. Goodman seemed to imply the opposite, which was not understood. Dr. Goodman encouraged the Council to consider the biological plausibility of a no-threshold model; Co-Chair Solomon responded that while this is among the scenarios generally discussed in the National Academy of Science, Engineering, and Medicine's *Science and Decisions* (2009) report, the relevant scenario in the present case is one where one can observe a linear dose-response when people are already above a threshold, so that there may be in theory some biological threshold, but there are already people who are sensitive and also exposed. Co-Chair Solomon added that she had not seen any evidence for a threshold in the literature.

Councilmember Cullenward addressed Dr. Goodman, citing a number of studies and conference presentations authored by Dr. Goodman, and confirming that they had been funded by industry stakeholder groups including the Western States Petroleum Association, the Electric Power Research Institute, the American Petroleum Institute, the American Gas Association, the American Plastics Council, and Philip Morris. Dr. Goodman replied that she has worked extensively on behalf of a number of clients and strives to disclose her funding sources, but that the most important thing is that her methods are as transparent as possible, and encouraged attention to her methods. Dr. Cullenward inquired whether Dr. Goodman would represent her methods as being internally consistent and in line with applicable scientific consensus. Dr. Goodman replied that there is not always consensus, and science progresses by everyone doing their best to be objective. Dr. Cullenward offered to present a court decision in which a Massachusetts judge determined the testimony from another consultant at Gradient, in support of which Dr. Goodman had provided statistical analysis, to be "inconsistent with and contrary to the consensus of the scientific community."

Co-Chair Solomon then discussed the possibility of temporal misclassification that Dr. Goodman had raised, noting that studies with a wide range of time windows, including very rapid changes in $PM_{2.5}$, had found evidence of effects, and that the intent was to act promptly to prevent further harms. Dr. Goodman agreed that there was no benefit to increasing pollution, but stated that decrements of 0.001–0.3 ug/m3 were "in the noise," to which Co-Chair Solomon replied that bringing down the mean decreases the entire distribution, including the "noise," which is important, and this activity is aimed at that. Acting Co-Chair Kleinman stated that looking at hourly and daily variation exaggerates the degree of variation, when we are concerned with longer-term changes, and annual averages have much smaller variation; even so, some of the earliest Harvard studies that looked at very different cities found that, on the whole, rates of disease and death were associated with contrasts in average pollution; moreover, that long-term follow-up showed that as pollution levels dropped, the mortality and respiratory disease rates dropped; further, that happened in the cities that had very low levels, as well as the cities that had high levels; while there may be a threshold at a very low level, we have not come anywhere near that level, in part because there is such a wide range of susceptibility in the human population, and our mission is to protect people at the most sensitive levels; a margin of safety is needed to cover them; therefore, there is good reason to use a conservative approach in assessing risks.

Director Haubert expressed hope, as a non-scientist, that the Advisory Council will be able to offer to the Board of Directors its assessment of the science, and a preference that the discussion, while allowing for different viewpoints, stay focused on the science. Director Haubert expressed that the Board will have to take into account policy matters including the economic impacts of regulations, and alternatives.

Co-Chair Solomon responded to Dr. Goodman's earlier mention of the meta-review by Burns et al (2017), noting that while it reviewed the effectiveness of interventions around the world to reduce $PM_{2.5}$, it reported that in most cases the data was insufficient to draw conclusions, but when the data was adequate, it did find effects, and in no cases did it find any evidence to the contrary, which was reassuring. Co-Chair Solomon asked whether the Council or staff had any thoughts on the topic of demonstrating efficacy.

With apologies to Director Haubert, Councilmember Cullenward expressed a desire to make a comment about the overall situation, coming from a long career in climate science and policy. Councilmember Cullenward shared that in the early 2000s, when colleagues began linking "climate denial" patterns to tobacco litigation, he found them uncredible, alarmist, and muddying of the already-difficult policy waters; that context was similar to the one faced here in a local environmental regulation context; since then, faced with growing evidence, he has changed his mind; he opined that the Council had just heard a textbook example of strategies used to delay, create uncertainty and doubt and fear in tobacco litigation, being applied to environmental science; and that the connections between individuals and firms need to be made and talked about publicly, along with the track record, so that discussion can proceed more appropriately.

Co-Chair Solomon recalled that the first commenter (Ms. Wolfe, on behalf of CCEEB) requested a case study, and found that proposal interesting and reasonable. Co-Chair Solomon requested clarification on future direction, including the potential for regular updates mentioned by Ms. Wolfe: would it take the form of rulemaking, or guidance? Staff agreed that a case study would be a good idea, especially in the context of risk communication, to help ground the discussion in the context of, for example, setting significance thresholds for CEQA guidance; similarly, in permitting, there would be a full regulatory context, while staff recommend that this methodology itself be kept outside that context. Co-Chair

Solomon concurred with the idea, and with the value of being able to incorporate newer science and make adjustments in practice.

Co-Chair Solomon recalled that the second commenter (Mr. Szutu, of the Air District's Community Advisory Council and the Citizen Air Monitoring Network in Vallejo) had urged consideration of PM speciation; agreeing that speciation is important to inform control strategies and efforts, Dr. Solomon noted that it becomes tricky in a risk-assessment context, where there are important data gaps, and advised that wrapping it into this process might grind things to a halt. On the issue of upset and startup/shutdown conditions, Co-Chair Solomon expressed that this is an important area of focus, and that we want to avoid such events impacting communities, without knowing whether that is best accommodated through this methodology. Acting Co-Chair Kleinman agreed that when starting with a focus on risk, it is much more difficult to take speciation into account.

Dr. Kleinman recalled a comment by Ms. Wolfe about "unrealistic assumptions," and invited staff to comment on the use of some of the more conservative aspects of the methodology, like higher-than-average breathing rates. Staff clarified that there are two aspects in which the proposed methodology departs from written OEHHA guidance (for the Hot Spots program): first, it assumes that seniors are at home 100% of the time, instead of 73%; second, it prescribes a factor of 3 to offer protection for variation along the dimensions of socio-economic status and race/ethnicity. Apart from this, staff clarified, every parameter and every value for a parameter is adapted directly from written OEHHA guidance (for the Hot Spots program).

Co-Chair Solomon, returning to the issue of biological mechanisms, described the wide range of defense mechanisms that people have against PM; therefore, while one can make a theoretical argument that a threshold does exist, in the real world, it is very challenging to imagine what that is, or what it would look like; when people have such a wide range of background exposures and sensitivities, the argument falls apart, and we are left with essentially the same problem we have with carcinogens, and the assumption that there is no safe number; this is absolutely consistent with the science and with our approach to other pollutants where we cannot discern a safe level; it is important to clarify that we are saying that there are already people tipped over into disease, and we cannot identify a safe additional exposure for those people. Acting Co-Chair Kleinman added that perhaps individuals may have their own thresholds, but that has largely to do with how we are able to make measurements, and we should remain cognizant that what results in a small effect for one person may result in larger effects for someone else with, for example, a smaller airway, or other respiratory impairments; the policies that are developed should protect this diverse group of people; therefore, a threshold is not the real point of providing public protection; rather, we see important changes in some populations even when we look at levels below the current standard.

Council Action

None; receive and file.

6. REVISION OF THE $PM_{2.5}$ NATIONAL AMBIENT AIR QUALITY STANDARD: THE ROLE OF AIR MONITORING DATA

Mr. Nudd introduced Dr. Kate Hoag, Meteorology and Measurement Assistant Manager, who gave the staff presentation *Revision of the $PM_{2.5}$ National Ambient Air Quality Standard: The Role of Air Monitoring Data*, including: outcome; outline; information only; National Ambient Air Quality Standards (NAAQS); PM NAAQS (primary); Revised Annual $PM_{2.5}$ NAAQS Proposal; Commenting on the PM NAAQS Proposal; What Happens After EPA Revises a NAAQS; Goals for Air Monitoring; How Should We Compare a Highly Variable Dataset (Air Monitoring Data) To One Number (NAAQS); Design Value (DV): A Statistic to Summarize Air Monitoring Data to Compare to NAAQS; Example: DV for a Monitoring Site; Annual $PM_{2.5}$ Design Value Trends; $PM_{2.5}$ Trends: Wildfire Impacts; NAAQS Designations & Implementation; Finalizing the NAAQS; Initial Area Designations; Developing a State Implementation Plan (SIP); and next steps.

Public Comments

Public comments were given by Dr. Stephen Rosenblum, Palo Alto resident.

Council Comments

The Council and staff discussed whether the frequency of wildfires, which are currently considered "exceptional events" needs to be considered when revising the NAAQS; and whether currently identified Air District priorities would need to shift to accommodate the required development of the State Implementation Plan, due 2026.

Council Action

None; receive and file.

OTHER BUSINESS

7. REPORT OF THE INTERIM EXECUTIVE OFFICER/AIR POLLUTION CONTROL OFFICER (APCO)

Sharon L. Landers, Interim Executive Officer/APCO, reported the following:

— Air District staff has released proposed amendments to Rules 9-4 and 9-6 to reduce emissions of nitrogen oxides from residential and commercial furnaces and water heaters in buildings in the Bay Area. These rules govern point of sale emission standards for small, typically residential and commercial, water and space heating systems. Emissions of nitrogen oxides impact local and regional air quality and contribute to the formation of ozone and secondary particulate matter. The Air District Board of Directors will conduct a public hearing to consider adoption of the proposed amendments and certification of the Environmental Impact Report (EIR) on March 15, 2023, at 9:00 AM. Staff plans to convene a formalized ongoing Implementation Working Group (IWG) to support the proposed rule amendments after potential adoption. The IWG is intended to consist of a variety of stakeholders with different areas of expertise in reference to the implementation of the rule amendments. This may include community-based organizations, environmental justice groups, advocacy, and subject matter expert organizations,

building technology experts, affordable and market rate housing developers and managers, local and state government staff, funding and financing agencies, equipment manufacturers and distributors, tenant representation organizations and labor organizations.

- On February 28, 2022, the Governor's State of Emergency will expire, requiring a return to in-person meetings of local legislative bodies, under the Ralph M. Brown Act and Assembly Bill (AB) 2449 (Rubio). The Air District is developing new procedures for in-person meetings that will enable limited remote attendance in publicly accessible remote locations.
- The Governor's 2023-24 Budget was released on January 10, 2023. A budget deficit of $22.5 billion is anticipated. Program cuts are anticipated to affect programs of interest to the Air District. Budget hearings will be held, leading up to the 2022-23 May Revision to the Governor's Budget.
- The 2023 Legislative Session has begun, and members will soon be introducing bills; 2,500 are anticipated in February. Air District Legislative staff tracks air quality-related bills and participates in committee hearings and advocacy activities, per the Board's Legislative Committee.
- On December 21, 2022, the Board of Directors confirmed the appointment of Dr. Philip M. Fine as the Air District's new Executive Officer/APCO, effective February 21, 2023.
- Recruitment for all seven Advisory Council positions is open until February 24, 2023, as the current Councilmembers' terms end in October 2023. Current Councilmembers are encouraged to reapply.

Council Comments

Members of the Council thanked Ms. Landers for her service as Interim Executive Officer/APCO.

8. PUBLIC COMMENT ON NON-AGENDA MATTERS

No requests received.

9. BOARD MEMBER COMMENTS

None.

10. TIME AND PLACE OF NEXT MEETING

At the end of the meeting, the next Advisory Council meeting was to be held at the Call of the Chair. After the meeting adjourned, the next meeting was scheduled for Monday, June 12, 2023, at 8:30 a.m., at 375 Beale Street, San Francisco, California, 94105. The meeting will be in-person for members of the public will be able to either join in person or via webcast.

11. ADJOURNMENT

The meeting adjourned at 11:48 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: Philip M. Fine
Executive Officer/APCO

Date: September 11, 2023

Re: Approval of the Draft Minutes of the Advisory Council Meeting of June 12, 2023

RECOMMENDED ACTION

Approve the Draft Minutes of the Advisory Council meeting of June 12, 2023.

BACKGROUND

None.

DISCUSSION

Attached for your review and approval are the Draft Minutes of the Advisory Council meeting of June 12, 2023.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Philip M. Fine
Executive Officer/APCO

Prepared by: Marcy Hiratzka
Reviewed by: Vanessa Johnson

ATTACHMENTS:

1. Draft Minutes of the Advisory Council Meeting of June 12, 2023

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

Advisory Council Meeting
Monday, June 12, 2023

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

CALL TO ORDER

1. **Opening Comments:** Advisory Council (Council) Co-Chairperson, Dr. Gina Solomon, called the meeting to order at 8:34a.m.

   Roll Call:

   Present: Co-Chairperson Dr. Gina Solomon; and Vice Chairperson Professor Michael Kleinman.

   Absent: Co-Chairperson Dr. Linda Rudolph; and Members Dr. Danny Cullenward, Dr. Adrienne Hollis, Dr. Pallavi Phartiyal, Garima Raheja; and Board Liaison David Haubert.

2. **APPROVAL OF THE ADVISORY COUNCIL MEETING MINUTES OF JANUARY 30, 2023**

Public Comments

No requests received.

Due to a lack of quorum, this item was continued until the next meeting.

INFORMATIONAL ITEMS

3. **COMMENTS ON THE PROPOSED METHODOLOGY FOR MODELING HEALTH RISKS FROM LOCAL SOURCES OF FINE PARTICULATE MATTER ($PM_{2.5}$)**

Greg Nudd, Deputy Executive Officer of Science and Policy, introduced staff from the California Office of Environmental Health Hazard Assessment (OEHHA), who gave the presentation *Comments on "Modeling Local Sources of Fine Particulate Matter (PM2.5) for Risk Management"*.

Dr. Lauren Zeise, the Director of OEHHA, opened the presentation by remarking that OEHHA fully supports the proposed overall approach to assess health risks from local levels of PM2.5, finding the approach scientifically rigorous; that OEHHA agrees with the underlying assumption that the health outcomes are linear and without a threshold within the range of current exposure; that OEHHA has carefully considered the model and the underlying assumptions; and that OEHHA has a number of recommendations for fine-tuning the proposed approach.

Dr. Zeise elaborated that there are background exposures producing health effects in the population; that incremental changes in the exposure either increase or decrease linearly within the range of observations; and that this is supported by a number of epidemiological observations, most recently captured by US EPA in its Integrated Science Assessment (ISA) Supplement as well as its previous ISA. Dr. Zeise further remarked that the understanding of linearity with dose for PM has been accepted for some time, and pointed to a 2009 National Academy of Sciences report, *Science and Decisions*, that reflects that basic assumption.

Dr. Zeise then stated that there are three key features for consideration: first, that the proposed approach is different from the classic RfD [reference dose] approach, such as under the Hot Spots program, where the concern is the difference between some reference level and the level in the population, which is very different from the reference level, outside of a linear area, where variability might be driving thresholds; second, there is a good deal of epidemiological information to inform the assessment; and third, that there is a most-susceptible population as a target, therefore adjustments are being made to address the risk to that population, so one would not use the approach to obtain an estimate for the complete population around the facility—what this is focused on is looking at the most exposed susceptible group.

Dr. Zeise introduced Dr. Keita Ebisu, who stated that OEHHA agrees with the model selection and structure represented by the key equation in the proposed methodology. Next, Dr. Ebisu reviewed the five combinations of receptor type and health outcome proposed in the methodology, and stated that OEHHA agrees with the model application to these health outcomes.

Dr. Rupa Basu then reviewed evidence that the proposed methodology relies on to establish effect sizes for premature mortality and pediatric asthma onset, which are based on epidemiological studies. Dr. Basu stated that OEHHA agrees with BAAQMD's selection of values for effect sizes ("betas"). Dr. Basu introduced a recent study (Alexeeff 2023) that examined PM2.5 exposures in a Northern California cohort, inclusive of the Bay Area, noting that while it focused specifically on cardiovascular mortality, much of the total PM2.5-attributable total mortality is cardiovascular. Dr. Basu remarked that it could also be a source of information about sensitive subgroups by, for example, socioeconomic status across all age groups, and recommended considering the study for those reasons. Finally, Dr. Basu stated that OEHHA's recommendation was that receptors be characterized as maximally exposed susceptible groups, rather than maximally exposed individuals.

Dr. Vincent Cogliano then spoke about the approach to adjusting Δx, which is the term in the key equation used to represent a change in PM2.5, and recommended that changes in breathing rate be addressed via an inter-individual variability factor. Dr. Cogliano stated that OEHHA agreed that unsheltered populations do not receive protections from things like air conditioning and filtration, that lower the levels of particulates indoors, and that an adjustment factor of 1.5 would be reasonable to account for this. Dr. Cogliano explained that OEHHA agrees with the method for adjusting for overlap

between the schedules of the source and the receptor, and recommends 100% FAH ("fraction of time at home") for children at a daycare, school, or residence. Dr. Cogliano then remarked that in the white paper, there is an adjustment applied in the key equation, a factor of 3 for groups with no empirical basis; that this is acceptable and in accordance with standard practices, and the assumption that there is some variability in physiology among humans, and how they may respond to a particular pollutant; that a default at OEHHA is generally 30, with some agents going higher; that here a factor of three may be acceptable; and that there is still variability among a population like Medicare recipients.

Dr. Cogliano stated that OEHHA recommended a single adjustment factor of three or more to account for inter-individual variability in sensitive populations to address factors including age, socioeconomic status, race/ethnicity, comorbidities, breathing rate variability, and other susceptibilities in seniors, children, off-site workers, and unsheltered populations (apart from having increased exposure relative to a sheltered population, the unsheltered population will tend to have higher rates or degrees of other risk factors, such as comorbidities that render them more vulnerable, or inadequate healthcare). Dr. Cogliano urged that the factor should be over and above what was in the epidemiological studies, and that it should be at least three or possibly more; if this is done, then a data deficiency factor is not necessary.

Dr. Cogliano presented OEHHA's proposal to change the key equation to include a new factor *F*, a multiplicative factor applied to the product of the PM2.5 increment (Δx) and effect size (beta); this *F* would be a composite factor, comprising both adjustments for exposure and adjustments for inter-individual variability.

Dr. Cogliano closed by remarking that OEHHA finds the overall approach for assessing health risks from localized PM2.5 to be warranted and scientifically sound; that OEHHA's input and recommendations are to improve the proposed approach by fine-tuning; that OEHHA suggests looking at the recent study by Alexeeff et al (2023) to inform estimates of the sensitivities of various segments of the population; and that an inter-individual factor of 3 or more could be applied to protect sensitive populations.

Clarifying Questions from Council Members

Regarding the unsheltered population, Vice-Chair Michael Kleinman remarked that worker environments are often dusty, and their exposures greater than what one would calculate from regional levels; Dr. Kleinman asked whether this fact is adequately included, such that a data deficiency factor is unnecessary. Dr. Cogliano responded that it might be good to think about how a very dusty environment for off-site workers compares to available epidemiology studies; that the focus would be on the increase relative to a dusty baseline; that different subgroups can be assessed; that he would not consider it part of a data deficiency factor; and that if it could be covered in the exposure part of the model, that would be best. Dr. Kleinman clarified that it was not a critique, and that the proposal as it stood was solid.

Co-Chair Gina Solomon remarked that she appreciated the recommendation to eliminate any data deficiency factor; and that in previous discussions, other endpoints had come up, such as neurodevelopmental, neurodegenerative, and reproductive endpoints, that were not included. Dr. Solomon inquired whether OEHHA could explain their thinking on those, and whether it could be seen as a data deficiency, or in some other category. Dr. Cogliano responded that he would apply a data-based deficiency factor when there are no studies of endpoints for which one has a reasonable

expectation would be outcomes of the exposure: if there are no studies of neurodevelopmental effects, that would be an example, or if asthma exacerbations had not been studied; it is appropriate when there are hypotheses and mechanisms by which PM can affect a a health endpoint of concern, but there are as yet no actual studies of it. In follow-up, Dr. Solomon stated that the methodology includes effect sizes for asthma onset and mortality, but not for the other endpoints being discussed, and asked whether there is a recommendation to develop effect sizes for additional endpoints individually, or whether those could be adequately protected against via the effect size for all-cause mortality: that is, if we protect against all-cause mortality, will we be sufficiently protecting against, for example, neurodegenerative disease or reproductive outcomes. Dr. Cogliano agreed that it was a difficult question, to compare mortality to other diseases that are not necessarily fatal, such as when the [increased] incidence of some non-fatal disease might be higher than the [increased] incidence of mortality, and stated that there is likely no simple answer. Dr. Kleinman added that his feeling was that while we always focus on mortality, we do not value morbidity as much, because we put a very high dollar value, in cost-benefit analyses, on a human life; and that our ability to put a dollar value on morbidity is much more limited; but that a closer look might reveal that non-mortality endpoints might in the aggregate equal or exceed the cost to society of premature mortality, with cascading effects throughout society; and that improved methods to express this are needed. Dr. Basu responded that mortality indeed does not capture all endpoints, and that her understanding was that this methodology could be used for other endpoints, such as adverse birth outcomes, with studies of those endpoints specifically used for their effect sizes, rather than extrapolating from mortality studies to other endpoints.

Public Comments

Christine Wolfe from the California Council for Environmental and Economic Balance (CCEEB) expressed appreciation for the opportunity to present in January. Ms. Wolfe inquired whether the same formula is to be used in more than one situation, with one set of parameters, or whether the thought is to adjust one or more parameters in other situations depending on the application—where the facility is located, for example, and the receptors around the facility.

Comments were also provided by Ken Szutu, of the Vallejo Citizen Air Monitoring Network (CAMN) and the Air District's Community Advisory Council. Mr. Szutu expressed that he would like to hear OEHHA's position on the speciation of PM, if local health impacts are the subject. Mr. Szutu also asked about indoor and outdoor exposure, and stated that when indoor monitoring has been conducted in his community, after a short while the indoor will match up with the outdoor; moreover, that they have found that indoor air can sometimes be more polluted than outdoor air; he asked for OEHHA's position on this.

Council Comments

Co-Chair Solomon asked whether OEHHA could first address the questions from the commenter about speciation and indoor air. Dr. Ebisu replied that, in regard to speciation, the starting point for this whitepaper is undifferentiated or total PM2.5; although a next step could be to estimate species, a species-specific effect size (beta) is not generally available, although in the next few years some may become available; that exposure to wildfire smoke is currently a very active area of epidemiological research; and that activity patterns during wildfires, such as whether windows are open or not, could be very different.

Council Action

None; receive and file.

4. UPDATE ON THE PROPOSED METHODOLOGY FOR MODELING HEALTH RISKS FROM LOCAL SOURCES OF FINE PARTICULATE MATTER ($PM_{2.5}$)

Mr. Nudd introduced Dr. David Holstius, Senior Advanced Projects Advisor, who gave the staff presentation *Update on Modeling Health Risks from Local Sources of $PM_{2.5}$*. Dr. Holstius opened with a slide presented at the last Advisory Council meeting, which grouped prior feedback into three main sets of topics: one about the methodology proper; a second about policy and implementation—those being more of a matter for the next phase of the project; and third, some requests for clarification or technical details, some of which will be obviated by revisions, but which the Air District will still respond to in writing. Dr. Holstius then remarked on the comments about the methodology proper: that as far as the strength of the science goes, this was thoroughly addressed by the Council at its last meeting, as well as by its recent Symposium, and the report that emerged from that Symposium, as well as by the US EPA's CASAC [Clean Air Scientific Advisory Committee], and finally by OEHHA in the presentation that was just heard.

As far as completeness goes, Dr. Holstius acknowledged that it is known that we cannot model everything, for example that there are important indoor exposures from indoor sources, whereas this methodology is about the contributions from sources that are generally outdoors, that are regulated by the Air District; likewise, that wildfires are happening and are consequential, with exposure intensities sometimes orders of magnitude larger, although shorter in duration, compared to the evidence base that this methodology is drawing from in terms of mortality and asthma. Dr. Holstius stated that it is also known that PM2.5 has very broad effects, and that the evidence base is still growing; from a regulatory perspective, premature adult mortality and childhood asthma onset provide at least some risk score for all segments of the population, for a wide range of receptor types and from the very young to the very old. Dr. Holstius gave assurances that the Discussion would be revised to feature more prominently some of the endpoints for which scores are not currently being calculated, which may address some of the concerns about data deficiencies; and that staff would work to keep this issue front of mind for participants during the next phases, during discussions of policymaking, along with other considerations that come up during risk management that will not be directly addressed here, in the risk assessment methodology development phase. Dr. Holstius pointed out that right now there are two scores—mortality and asthma—and deciding what to do about that is a policy matter; the Hot Spots framework may offer a pattern to follow, where there already are three scores being considered: chronic hazard, acute hazard, and cancer risk, each with a corresponding threshold for policy or action; and that there is as yet no prescribed threshold for risk of mortality or asthma onset, so that does offer a degree of freedom that will determine protectiveness in practice, making it not simply about the selection of endpoints, and that is a matter for the Board to consider. Dr. Holstius suggested that it might be best to resolve this with two endpoints before adding more.

As far as the risk magnitude and adjustments for vulnerable groups go, Dr. Holstius remarked, at the last meeting the Council heard arguments, on the one hand, from Dr. Julie Goodman about the values being too small to be real or significant, while on the other hand, concerns were raised by CCEEB about unrealistic assumptions and values perhaps being alarmingly large. Dr. Holstius stated that it is neither of those; it is meant to be protective of those who are more susceptible based on our scientific understanding. Dr. Holstius recalled Dr. Kleinman's remark that we should remain mindful that what

results in a small effect for one person may result in a larger effect for someone else, with for example a smaller airway, or other respiratory impairments, and the approach we take should protect this diverse group of people. Dr. Holstius stated that a good communication plan is needed, with supporting materials that may involve case studies and other kinds of contextualization suggested and supported by the Co-Chairs at the last meeting. In terms of the magnitudes of adjustments for vulnerable groups, Dr. Holstius stated that the Air District continues to be advised by OEHHA on what is supportable by the science, and warranted; OEHHA feedback and the Air District's revisions will make some technical misunderstandings obsolete, and that will resolve remaining technical issues.

Dr. Holstius indicated that the feedback just provided by OEHHA was overall supportive of the draft methodology, and of its scientific foundations; specific recommendations for improvements would be incorporated into a version 2.0 of the white paper. Next steps, Dr. Holstius stated, include that update to version 2.0, along with responses being posted to written comments; then there will be a transition to more policy-oriented issues and applications, likely leaning on case studies to illustrate some of the finer points and demonstrate correctness without getting lost in details.

Public Comments

No requests received.

Council Comments

Co-Chair Gina Solomon remarked that the paper by Alexeeff et al (2023) was notable for three reasons: first, that Director Haubert had at the last meeting [January] asked staff if there were any studies that had been done in the Bay Area that were relevant to the present purpose, and that this study is a high quality and very relevant example of that, with nearly 3.8 million people and substantial geographic overlap with the 9-county Bay Area. Dr. Solomon noted that the outcomes are somewhat different—ischemic heart disease mortality and cardiovascular mortality, and acute myocardial infection not resulting in death—but the magnitude of the effect was quite similar, which gives confidence. Dr. Solomon remarked that another issue raised by commenters at the last meeting was that some of the referenced studies were on the older side, whereas the Alexeeff et al (2023) study is quite recent. Dr. Solomon clarified that it may not be necessary to change the effect size or basis for the analysis, but that it is important to take a careful look at the study and incorporate it, as this study used high-quality and precise information on individual-level residential addresses and confounders, with the potential caveat that the members of the Alexeeff cohort (Kaiser insurance) tend to be healthier than the general population. Dr. Holstius responded that yes, it would be considered carefully and taken as strong support.

Dr. Solomon then turned to the topic of other endpoints, stating that if she resided next to a significant emitter of PM2.5, she might worry about dying, but she would worry about dementia, and also be concerned about reproductive outcomes, neurodevelopmental outcomes. Dr. Solomon stated that these are important and need to be acknowledged, and that there are data on these outcomes, so they can't be ignored. The reason for including a data-deficiency factor was, Dr. Solomon said, to simplify and not have to develop an effect-size parameter for every potential outcome, but it is a little problematic not knowing how they all relate to one another; on the other hand, asking staff to go back and develop models and effect sizes for every outcome for which that could be done, could result in significant delays, and in the end just with one, whichever is the most relevant to the population at hand that is the most protective. Dr. Solomon stated that we need to make sure that we are thinking about the science,

and what is strongly supported, and how to move forward with something that's workable, and not a lot of different slope factors to have to deal with in any individual decision process; that she understood OEHHA to be recommending a factor of at least, but not necessarily just, three-fold; that this would include age, socio-economic status, co-morbidities (very important), variability across the population, and the vulnerability of the unhoused—that this seemed like more than 3-fold, when justice will not really be done to the risk of neurological or reproductive outcomes at this phase in the process. Dr. Solomon then strongly encouraged staff to consider a factor of 10-fold, that would span and clearly itemize all issues that are not fully addressed in the current equation, and stated that in her opinion, a factor of 10-fold would be entirely reasonable at this point.

Dr. Kleinman agreed that an additional level of protection is needed, because even at the proposed new levels for the [national] standards, it is very hard to argue that there is an adequate margin of safety; we are seeing effects at levels considerably below the annual PM2.5 standard in epidemiological studies, and there are strong corroborations with many endpoints where the evidence may not yet be sufficient to call them "causally related" but much of that is due to deficiencies in the number of studies; therefore, we need to think about what we are going to incorporate into the notion of "data deficiency"; many of these endpoints will have cascading effects that last through decades; therefore our assessment should take into account that there is very strong evidence that other endpoints are related to PM2.5 exposure, but lack of funding and other resources mean that, in his opinion, these remain "known unknowns."

Dr. Zeise stated that, listening to the discussion, there was clearly a concern that not all endpoints are being fully captured, and that making sure that the most sensitive are considered, as well as a number of effects—OEHHA would need to think more about the factor, but one of the considerations is that data deficiency factors are typically used when working in the RfD [reference dose] framework; that said, there is a large body of data pointing to other endpoints, and that it might be possible to develop a narrative, as well as to consider the effect sizes estimated by studies of other endpoints, to develop an understanding of the extent to which risks may not be adequately covered by considering estimates for susceptible populations for mortality and asthma; the question is also to the Air District to the extent to which there has to be a full accounting, quantitatively, of all endpoints, in making a decision, or can there be a narrative that also enables weighing of those other outcomes as they move toward making a decision on a permit or similar.

Mr. Nudd responded that right now, the way that the Air District's permitting regulations are set up, it does not consider the localized impacts of PM at all; therefore, considering mortality would be a huge improvement; therefore, while recognizing that other endpoints could be discovered to be driving the principal risk concern, what he would advocate is that we move forward with something that addresses mortality in the policy contexts currently being considered.

Mr. Nudd stated that one potential policy context would be CEQA (California Environmental Quality Act) thresholds and local land-use decision-making, where the Air District provides guidance to local decision-makers on how to conduct air quality analyses, including significance thresholds; currently the significance thresholds for PM are based on an old methodology driven more by compliance with the NAAQS (National Ambient Air Quality Standards), which has a number of problems; for that, the intent would be to take an approach similar to air toxics, and take a look at those localized impacts; this would involve "what is truly significant?" and would have to look at the body of decisions that local land-use decision makers are making, because having everything count as significant would be undesirable, insofar as people would no longer pay attention.

Mr. Nudd stated that a second policy context could be pre-construction permits; in that case, the Board would set a general policy; currently the Board has set a 10 per million threshold for cancer risk across the Bay Area, but a 6 per million threshold for overburdened communities, defined as communities in the 75th percentile in CalEnviroScreen; a permit would be denied if the modeling shower a higher cancer risk score; that was based on a consideration, again, of the body of the decisions that the Board is making with respect to permitting, and what the impacts are of drawing a particular line.

Mr. Nudd stated that a third policy option could be to set facility-level risk limits for PM, and create a structure that allows staff to go to the Board and have them set limits for overall risk from PM exposure from individual facilities, and the facilities would have to find ways to get below those thresholds; in that case, the Board would be considering the cost implications, as well as the health implications.

Mr. Nudd explained that while this methodology may not provide a number for every known endpoint, that other policy considerations will probably drive the selection of a threshold, more than the exact number that comes out of a methodology like the one proposed; therefore, whether a factor is 3 or 10 may not make a large difference in the policy outcome; therefore, he would advocate moving forward with a mortality-based analysis across these policy use cases, while continuing to evaluate other endpoints.

Mr. Nudd stated that as work moves forward with the Advisory Council, the focus will likely broaden to look at cumulative impacts, especially in permitting, that go beyond looking at CalEnviroScreen and look at multiple chemical exposures and similar questions; therefore, for present purposes we should not let the perfect be the enemy of the good, and move forward with assessments of mortality risks while making the process more robust and inclusive, including of endpoints associated with exposures to other chemicals, and within the context of the socioeconomic vulnerability to be most protective in those communities that are most vulnerable and, to Dr. Kleinman's point, least able to afford the health impacts of the insults.

Co-Chair Solomon thanked Mr. Nudd for the helpful context, including longer-term ideas about incorporating CalEnviroScreen and cumulative impacts, which could go a long way toward addressing issues and concerns raised by community members in this forum.

Co-Chair Solomon offered a recap of the developments so far in the process, remarking that she was quite happy with where the white paper currently stands: looking at PM2.5 at a local level is relatively new approach but the proposed approach is methodologically sound; this has been discussed since the 2019 meeting, by two iterations of the Advisory Council; it has now been reviewed by OEHHA and found to be scientifically appropriate and sound, which is important; looking at PM2.5 using a linear, non-threshold model has been an extremely important topic of discussion for several years, and is also now supported by a scientific consensus, that there is no discernable threshold in the population, especially when looking at mortality, as we are here; therefore we are on good scientific ground; we have a model that makes sense and is time-tested; we have an appropriate focus on the highly exposed group in a local area; and we have had a lot of discussion about the fact that this is somewhat different than a cancer-risk assessment, including the issue of data deficiencies for other endpoints, being that here there are multiple endpoints, resulting in potentially an array of linear dose-response relationships, which is challenging.

Co-Chair Solomon remarked that it is appropriate to consider exposures and adjustment factors in particular for unhoused populations, or people who may have very leaky homes, which is especially

pertinent to the Bay Area, and reinforced by her experience with field studies in the Bay Area, where a significant number of homes are for example cooled by swamp coolers, resulting in no filtration and pulling in large quantities of outdoor air, including PM2.5 from outdoors; therefore the adjustment factor for the unhoused and for other populations that may essentially have no barrier or protection from housing is appropriate. Dr. Solomon remarked that the issue of adjustment factors has involved a good deal of discussion, and is always challenging; she stated that she would feel more comfortable with an adjustment factor greater than 3, and more comfortable with a factor of 10, for the reasons that she stated previously, but that the most important thing at this point is to make progress on the issue, and actually apply the methodology. Dr. Solomon wondered if it might help to look at effect sizes for other endpoints, especially neurological and reproductive outcomes, to have a sense of whether the all-cause mortality and asthma analyses would be reasonably protective against those other outcomes. Dr. Solomon concluded by remarking that there has been a great deal of progress, and that the work is being handed back to staff to incorporate everything discussed.

Vice-Chair Kleinman offered a final remark on the topic of wildfires, which was that going forward we are likely to see this trend continuing, possibly worsening over the years; this is outside the control of the Air District, but asked whether there should be re-evaluations built into the model or policy, possibly on a periodic basis, to take account of worsening baseline conditions, and possibly lead to increased protections during times when PM2.5 levels are higher, akin to Spare the Air Days. Mr. Nudd responded that rather than building that into this risk-assessment methodology, that might be better incorporated into policy implementation; staff are aware of some approaches in other jurisdictions that, for example, require cessation of dust-generating activities during high PM days; those kinds of examples can be addressed as staff go through the process of identifying and modifying regulatory requirements for PM sources, based on this methodology; that is something to consider insofar as it is practical; for some large facilities this may not be a practical approach, so it would likely require looking at individual source types and how they might actually mitigate their emissions temporarily during high-PM events.

So-Chair Solomon concluded by expressing interest on behalf of herself, Co-Chair Rudolph (absent), and Dr. Kleinman, in continuing to help in any way with any aspect of this report, mindful of Brown Act limitations.

Council Action

None; receive and file.

OTHER BUSINESS

5. REPORT OF THE EXECUTIVE OFFICER/AIR POLLUTION CONTROL OFFICER (APCO)

On behalf of Dr. Philip M. Fine, Executive Officer/Air Pollution Control Officer, Mr. Nudd reported that because the current Councilmembers' terms expire in July 2023, recruitment will open soon. Air District staff hopes to have one final meeting of the current group of Councilmembers and have them take formal action to approve Version 2.0 of the Air District's Proposed Methodology for Modeling Health Risks from Local Sources of $PM_{2.5}$. The Air District hopes to implement policy changes based on endorsement of the Council and OEHHA as soon as possible. The Air District also hopes to work with the Council to shape the Air District's policy response regarding PM endpoints and cumulative impacts of air pollution on overburdened communities.

6. PUBLIC COMMENT ON NON-AGENDA MATTERS

No requests received.

7. BOARD MEMBER COMMENTS

None.

8. TIME AND PLACE OF NEXT MEETING

At the end of the meeting, the next Advisory Council meeting was to be held at the Call of the Chair. After the meeting adjourned, the next meeting was scheduled for Monday, September 11, 2023, at 9:30 a.m., at 375 Beale Street, San Francisco, California, 94105. The meeting will be in-person for members of the public will be able to either join in person or via webcast.

9. ADJOURNMENT

The meeting adjourned at 10:12 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: Philip M. Fine
Executive Officer/APCO

Date: September 11, 2023

Re: Fine Particulate Local Risk Methodology Update

RECOMMENDED ACTION

None; receive and file.

BACKGROUND

The Air District has developed a white paper, *Modeling Health Risks from Local Sources of Fine Particulate Matter*, that describes and illustrates a methodology for modeling increases in health risks attributable to local sources of fine particulate matter, or $PM_{2.5}$. It has been developed by the Air District with guidance from the Air District's Advisory Council and in consultation with staff at the United States Environmental Protection Agency, the California Air Resources Board, and California's Office of Environmental Health Hazard Assessment.

DISCUSSION

The Advisory Council will receive a presentation from staff summarizing and illustrating key aspects of the methodology, including its foundations, goals, and approach. The staff presentation will also be responsive to a request made at the last meeting of the Advisory Council, which was to review effect sizes for health endpoints other than premature adult mortality and pediatric asthma onset.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Philip M. Fine
Executive Officer/APCO

Prepared by: David Holstius and Song Bai
Reviewed by: Gregory Nudd

ATTACHMENTS:

1. Fine Particulate Matter Local Risk Methodology White Paper v2

Modeling Health Risks from Local Sources of Fine Particulate Matter ($PM_{2.5}$)

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Bay Area Air Quality Management District

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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
NAAQS	National Ambient Air Quality Standards
OEHHA	Office of Environmental Health Hazard Assessment
$PM_{2.5}$	Particulate matter less than 2.5 µm in aerodynamic diameter
RR	Relative risk
US EPA	United States Environmental Protection Agency

Contents

1 Introduction and Background

This document proposes and demonstrates a methodology for modeling health risks attributable to local sources of fine particulate matter ($PM_{2.5}$). It has been developed by the Bay Area Air Quality Management District (Air District) with guidance from the Air District's Advisory Council (Advisory Council) and in consultation with staff at the United States Environmental Protection Agency (US EPA), the California Air Resources Board (CARB), and California's Office of Environmental Health Hazard Assessment (OEHHA).

The purpose of this methodology is to 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 established that many regions of California now meet those standards. Despite this progress, some populations continue to be exposed 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 (CA HSC §§ 44300-44384, BAAQMD 2021). The Air District has also modeled source-specific contributions to local elevations of $PM_{2.5}$ (e.g., BAAQMD and WOEIP 2019; Reid et al. 2021), but to date has not conducted any corresponding health risk assessments. This methodology would enable those assessments.

2 Concepts and Methods

2.1 Modeling of source-attributable 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. It assumes 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. Receptor types pertinent to this methodology are listed in Table 1, along with default values for scheduling-related parameters, such as the number of hours per day that off-site workers are assumed to work, or students to attend school. These default values are guided by existing HRA protocols for assessing long-term exposure and risk in the context of linear, no-threshold effects (see Section **Error! Reference source not found.** for more on linearity).

This methodology deals exclusively with annual averaging times. Having identified the most-impacted receptor locations for annual average $PM_{2.5}$, and the corresponding contributions of the modeled source, it proceeds with assumptions and/or site-specific data about the time-activity patterns of a given receptor type, and the operational schedule of the source as well (Table 1; OEHHA 2015; BAAQMD 2021). Using this information, the modeled incremental annual average *concentrations* are converted 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 (e.g., during the working hours of off-site workers), 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.

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[1] "Receptor" in air quality modeling terminology can refer to (a) an entity exposed to pollution or (b) a 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.

2.2 Modeling of responses to exposure

To re-express modeled incremental average $PM_{2.5}$ exposure intensities in the form of health risks, this methodology leverages response functions from epidemiologic studies of the health effects of $PM_{2.5}$. To ensure that at least one risk score can be generated for all receptor types, two endpoints are assessed: (a) premature adult mortality and (b) pediatric asthma onset. For more on the selection of these endpoints, the relevance of other endpoints, and related policy implications, please see the Discussion (Section 4).

The response functions yield estimates of relative risks, which are then converted to differences using information about baseline rates.[3] To illustrate: suppose one takes the *relative* risk of asthma onset, per μg/m^3, to be 1.04 for five-year-old children. Suppose that one further takes the baseline annual incidence rate to be 10 per 1,000; that is, on average one expects 1% of asthma-free five-year-olds to develop asthma before turning six, given baseline conditions, including a baseline level of $PM_{2.5}$ exposure.[4] For a scenario in which the annual average exposure intensity is increased by 1 μg/m^3, that baseline rate should then be multiplied by 1.04. Subtracting the baseline rate from this scaled result yields an estimate of the attributable increase in the incidence rate among such children, compared to the baseline scenario. In this case, that difference is 0.01 × 1.04 - 0.01 = 4×10^{-4}, or 0.4 per 1,000 per year. Equivalently, this can be regarded as an increase of 400 per million in the risk, for such children, of developing asthma during that year. This can be compared to the baseline risk over that same single year, which would be 10,000 per million, corresponding to the baseline annual incidence rate of 10 per 1,000.

The following equations express this in mathematical terms, taking $\Delta x > 0$ to mean an increase in $PM_{2.5}$ above baseline levels, $\Delta y > 0$ a corresponding increase in the risk or rate of some endpoint, and y_o the baseline risk or rate for that endpoint (given a baseline level of $PM_{2.5}$):

$$y/y_0 = \exp(\beta \cdot \Delta x) \quad (1)$$

$$\Delta y = y_0 \cdot [\exp(\beta \cdot \Delta x) - 1] \quad (2)$$

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 epidemiologic study or studies in which ambient $PM_{2.5}$, estimated or measured at some outdoor locations, was the independent variable. Generally, epidemiologic studies estimate β by adjusting for other measured factors, with the goal of approximating the causal effect of x alone. Most such studies report an estimated risk ratio, such as a relative risk (RR), for a given increment of $PM_{2.5}$. In the equations

[3] Both "relative risk" and "risk difference" compare the probability of an outcome in a more-exposed group or scenario to the probability of that outcome in a less-exposed group or scenario. A relative risk is calculated by dividing, while a risk difference is calculated by subtracting.

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

above, β is the natural logarithm of that risk ratio.[5] Detailed explanations and discussions are available in Fann et al (2011) and US EPA (2010, 2022a).

Table 2 lists the effect sizes (β) adopted for use in this methodology, along with key studies that informed their selection. For premature adult mortality, the value selected to represent the effect size ($\beta = 1.0\times10^{-2}$) is consistent with the ranges reported in the District's recent evaluations of impacts on large 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 μg/m^3 ($\beta = 0.70\times10^{-2}$), and 1.136 per 10 μg/m^3 ($\beta = 1.28\times10^{-2}$) for exposures less than 12 μg/m^3. Di et al (2017) is the core study on which the US EPA currently relies for estimates of attributable mortality among seniors (US EPA 2022a). Wu et al (2020) report results consistent with Di et al (2017), including higher effect sizes at lower baseline levels: 1.23 to 1.37 per 10 μg/m^3 for exposures always less than 12 μg/m^3, vs 1.06 to 1.08 per 10 μg/m^3, respectively ($\beta = 2.07\times10^{-2}$ to 3.15×10^{-2}, vs 0.58×10^{-2} to 0.77×10^{-2}). Summarizing other recent studies via a random-effects model, Di et al (2017 fig. S6) arrive at a pooled result of 1.11 per 10 μg/m^3 ($\beta = 1.04\times10^{-2}$). Vodonos et al (2018), summarizing a wide range of studies across all ages via meta-regression, and controlling for the baseline level of $PM_{2.5}$, arrive at a relative risk of 1.0129 per 1 μg/m^3 ($\beta = 1.28\times10^{-2}$) for a baseline centered on 10 μg/m^3. In the Bay Area, about 98% of the population resides where a modeled annual average $PM_{2.5}$ concentration is less than 12 μg/m^3, and 75% where it is less than 10 μg/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.

For pediatric asthma calculations, this methodology adopts the same effect size used by the US EPA's BenMAP-CE platform (US EPA 2022a) to calculate population-level impacts within the United States. Derived from a large cohort study of children in Québec (Tétreault et al. 2016), this effect size is RR = 1.33 per 6.53 μg/m^3 ($\beta = 4.37\times10^{-2}$). The mean $PM_{2.5}$ concentration in the supporting study was approximately 10 μg/m^3.

2.3 Adjustments for higher-than-average risk

Consistent with existing HRA frameworks for local sources of air pollution (BAAQMD 2021; OEHHA 2015), this methodology focuses on characterizing risk for scenarios with higher-than-average risk. To accomplish this, it augments the key equations with an adjustment factor F, as recommended by OEHHA[6] and as shown below:

$$y/y_0 = \exp(\beta \cdot \Delta x \cdot F) \quad (3)$$

$$\Delta y = y_0 \cdot [\exp(\beta \cdot \Delta x \cdot F) - 1] \quad (4)$$

[5] For a relative risk expressed per u μg/m^3, $\beta = (\ln RR)/u$.

[6] Remarks presented by OEHHA at the June 12, 2023 meeting of the BAAQMD Advisory Council.

Table **3** matches each combination of receptor type and endpoint with a corresponding value for F. To inform the values presented for F in

Table **3**, three components were considered: increased sensitivity for certain groups ("sensitive groups"); situations involving higher incremental exposure, given the same incremental concentration ("exposure modification"); and situations involving higher intake, given the same increase in exposure ("intake modification"). Keeping in mind both that (a) the simultaneous realization of extrema on all three dimensions will tend to be rare, but also that (b) it is possible for a more typical value on one dimension to be counterbalanced by more extreme variation on another dimension, the overall values presented for F are based generally on the product of the estimates for these three components (

Table **3**).

There are differences in the nature of the components, and considering all three at the same time requires a melding of epidemiological and mechanistic understandings of $PM_{2.5}$. The first component ("sensitive groups") is directly informed by epidemiological evidence, as well as by a precautionary stance when sub-group analyses for a particular endpoint are as yet unavailable. The second and third components (exposure and intake modification, respectively) rely on mechanistic considerations. The foundation for these considerations is a conceptual model of $PM_{2.5}$ in which there are approximately linear relationships between changes in concentration, exposure, intake, and effect at a group level. Adjustments made to reflect exposure modification and intake modification are then consistent with the way that exposure (for all receptor types) and intake (for non-residential receptor types) are treated in existing HRA guidance. See Section **Error! Reference source not found.** ("**Error! Reference source not found.**") for additional discussion of linearity and adjustments.

The three-part, bottom-up approach presented here is not necessarily the only way that values for F could be suitably determined. The intent of this white paper is to present a viable option for policymakers to consider. Other approaches to arriving at values for F, or components of F, may ultimately be preferred. Allowing for the possibility of such modifications, the following sections provide explanations and examples to illustrate the general approach described above.

Sensitive groups (inter-individual variation). The overall adjustment factor F should address reasonable degrees of inter-individual variation in factors such as socio-economic status, race/ethnicity, comorbidities, and other important dimensions that are not already reflected in age-specific variation in the baseline rate y_0. To characterize variation in the relative risks of premature mortality along these other dimensions, the relevant literature offers some informative findings. 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 times the average or more (e.g., Di et al. 2017; Yazdi et al. 2021). A recent and high-powered study of cardiovascular mortality[7] specific to northern California reported finding no effect modification by race/ethnicity, but a similar degree of effect modification by neighborhood socioeconomic status (Alexeeff 2023).

Overall, for this component, a factor of 3 has been determined to be adequate. Contemporary analyses of effect modification for $PM_{2.5}$ and mortality are not wholly consistent in the sets of subgroups that they analyze, nor in their definitions of those subgroups, nor the findings they report (Hickens 2023). Analyses of other subgroups by future studies could therefore potentially support larger or smaller estimates for this type of effect-size modification. Comparable studies of vulnerable subgroups do not yet exist for pediatric asthma onset; as a precautionary approach, and following the expert guidance of the Air District's Advisory Council and staff at OEHHA, it has been determined that a factor of 3 is also warranted for this component when considering receptor/endpoint combinations involving pediatric asthma onset.

Exposure modification (lack of shelter). This adjustment is also applicable to all receptor types, but corresponds to a situational factor, rather than a context-independent attribute of individuals. It requires that long-term incremental effects for mortality and asthma onset are approximately linear in terms of exposure, and the recognition that built environments are a key modifier of the relationship between outdoor concentrations and actual exposures.

Most of the time, most populations similar to those in the Bay Area are residing, working, or otherwise spending time in an indoor environment. This affords them a degree of protection from $PM_{2.5}$ of outdoor origin. Some fraction of that $PM_{2.5}$ will be removed by a building's envelope, before it can pass indoors. Of the fraction that becomes entrained indoors, an additional fraction will be removed on short timescales by mechanisms such as filtration (intake, recirculating, or spot) or deposition onto surfaces. The overall result is that the steady-state indoor concentration of $PM_{2.5}$ of outdoor origin will be less than or equal to its steady-state concentration outdoors.[8] The ratio is termed the infiltration factor, F_{inf}.

There are important exceptions. Situations where residents inhabit especially leaky buildings, when students attend classes with open windows, when nearby workers labor outdoors, or in general when any group is not sheltered from the modeled source, are equivalent to having F_{inf} = 1. This value can be used to construct the numerator of an adjustment ratio, under the assumption that these scenarios represent plausible and appropriate scenarios for modeling

[7] Cardiovascular mortality represents a substantial fraction of air pollution related mortality.

[8] When there are indoor sources of $PM_{2.5}$, the steady-state concentration of $PM_{2.5}$ indoors can be higher than it is outdoors. However, this methodology is concerned only with the $PM_{2.5}$ that originates with the modeled source. Assuming that they are independent of the impacts from the modeled source, the impacts of indoor sources (like those of all other sources) are effectively held aside.

potential risk.[9] The denominator should represent an average infiltration factor across the typical time-activity contexts of the corresponding epidemiological studies. Those contexts (i.e., being indoors, having typical levels of protection) will have dominated the person-time in those studies and hence the basis for the effect-size estimates β. A review of the literature suggests that a reasonable assumption for $PM_{2.5}$ in environments like the ones that were studied is F_{inf} ≈ 2/3 (Diapouli 2013). This is consistent with empirically validated predictions made by US EPA's population exposure model for particulate matter (SHEDS-PM), which predicts a median ratio of ambient $PM_{2.5}$ exposure to ambient $PM_{2.5}$ concentration of approximately 2/3 as well (Burke et al. 2002). The corresponding adjustment ratio should then be (1) ÷ (2/3) = 1.5x.

In addition to being protective of groups that are unsheltered while they are in proximity to the modeled source, this is consistent with the conceptual treatment of exposure in existing HRA guidance for cancer-risk assessments. In that guidance, no discount is given for any protection afforded by an indoor environment. Dose calculations proceed directly from the modeled increment in the ambient (outdoor) concentration.

Intake modification (higher breathing rate). A third consideration is that certain situations systematically involve higher intake rates, given the same level of exposure, even after adjusting for a potential lack of shelter. This methodology assumes that, all else being equal, a higher breathing rate results in a higher intake of $PM_{2.5}$, given the same exposure increment (Δx). Further, it assumes that this makes a difference to incremental group-level effects, at least for the endpoints considered (premature adult mortality and pediatric asthma onset). If these effects do increase with long-term intake, then places and situations where people are breathing more intensely—while they are exposed to the modeled local source—should correspond to higher incremental risks, given the same exposure intensities and durations. It is less preferable to permit a new localized source of $PM_{2.5}$ in proximity to such situations, all else being equal; this methodology aims to inform consideration of that relative difference in risk.

The corresponding intake-modification adjustment aims for consistency with current HRA guidance (BAAQMD 2015), which uses higher-than-average breathing rates for student and off-site worker receptors. It also extends the same treatment to daycare receptors. The following paragraphs illustrate the adjustment, using the student receptor type and the asthma endpoint as a motivating example. As with the exposure-modification adjustment, what was observed in the epidemiological study forms the basis for the denominator of an adjustment ratio. The overall effect size (RR = 1.045 per μg/m^3) reported by Tetréault et al (2016) reflects what was observed in that study, which was a large cohort of children engaged in activities and contexts spanning the full range of daily life.

During school hours—that is, while exposed to the modeled source, such as a permitted facility next to the school—existing HRA guidance states that "breathing rates that reflect playground

[9] The quality of shelter for residents, students, daycare attendees, or workers is only relevant while they may be influenced by emissions from (i.e., in proximity to) the modeled source. It is not necessary that they be unhoused at other times, e.g. while away from the source or while the source is not operating.

activities and classroom activities are appropriate"; "moderate activity" levels are considered to be an appropriate characterization (OEHHA 2012). For age 2–15, the mean 8-hour moderate activity level rate is 380 L per kg body weight per 8 hours, or 47.5 L/kg-h (OEHHA 2012, Table 3.3a).[10] For "sedentary and passive activities," the mean 8-hour rate is 80 L per kg body weight per 8 hours, or 10.0 L/kg-h. Students who are breathing at a rate of 47.5 L/kg-h while attending school for 10 hours per day, and 10.0 L/kg-h during the other 14 hours of the day, will have an average rate of [(47.5 × 10) + (10.0 × 14)] ÷ 24 = 25.6 L/kg-h. Based on this, their breathing rate while at school will be higher than their average rate by a factor of approximately (47.5 / 25.6) = 1.9. Therefore, given the same average exposure intensity and duration of exposure, compared to a residential receptor of the same age, the $PM_{2.5}$ intake for such a student receptor will be 1.9 times as high. Adjustment factors based on ratios of breathing rates can be constructed in this manner for daycare and off-site workers as well, using analogous (age-specific) values from OEHHA (2012). These ratios are 1.8 and 2.2, respectively. Overall, this supports an adjustment factor of 2 for intake modification for student, daycare, and off-site worker receptor types (Table 3).

The intake-modification adjustment is unlike the exposure-modification adjustment in that it is not applied to residential receptors, either for the mortality endpoint or the asthma endpoint. The reason is that the residential receptor type corresponds to a 100% fraction of time at home (FAH); therefore, it must correspond to a typical distribution of breathing rates, conditional on age. The underlying studies for premature mortality and asthma onset (Table 2) are also assumed to correspond to typical distributions of breathing rates (conditional on age) because, like the residential receptor type, those studies involved a full range of daily activities. Therefore, the adjustment ratios would be 1; that is, no adjustment in either case.

2.4 Cumulative risk over time

When assessing impacts on morbidity or mortality among a large population, the focus is often on annual incidence (counts) or incidence rates (per person). Consistent with existing HRA methodology, this methodology instead assesses cumulative risk over a longer period of time. 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, it arrives at overall results that summarize excess risk on an additive scale.

Assessing cumulative risk over time begins with the definition of a multi-year exposure window for each combination of receptor type and endpoint. The length of the exposure window (i.e., the exposure duration, ED) is specified to match existing guidelines. For residential receptors, current cancer-risk HRA guidelines prescribe a window of exposure that is up to 30 years

[10] Table 3.3a of OEHHA (2012) is the basis for all breathing-rate values used. Note that the mean 8-hour rate is used, rather than the 95th percentile 8-hour rate. The adjustment is intended to capture the shift across an entire group's distribution, given a new level of activity—not the fact that some individuals may generally breathe more than others, per kg body weight ("inter-individual variability").

(BAAQMD 2021; OEHHA 2015). Consistent with a focus on maximal risk, in cancer-risk HRAs this is taken to be the first 30 years of life.[11]

For premature mortality, the most vulnerable window is during the later years of life. Currently, the average life expectancy in the Bay Area is just under 80 years, and given the relevant baseline incidence rates (Table 4), approximately half the population should survive to age 85. Taking this into account, when assessing the risk of mortality for the residential receptor type, this methodology defines the exposure window to be age 55–84. For the off-site worker receptor type, current guidelines specify an exposure duration of no more than 25 years. When assessing mortality risk, this methodology defines the exposure window for the worker receptor type to be age 40–64.

When calculating the risk of pediatric asthma onset for the residential receptor type, an exposure window of age 0–17 is appropriate, consistent with BenMAP-CE (US EPA 2022a). For student receptors, the exposure window can be site-specific, depending on whether the modeled receptor represents exposure while at, for example, a high school or an elementary school. In this whitepaper, for the purpose of illustration, a default age range of 5–13 is used. For the daycare receptor type, again for the purpose of illustration, a default age range of 0–5 is used. These default exposure windows can potentially be modified to reflect the specifics of a particular scenario.

For each combination of receptor type and endpoint listed in Table 1, a risk increment Δy_i can be calculated for every year (age) i within the relevant exposure window. This represents the increase in the probability of experiencing the adverse event (i.e., death or asthma onset) between the start and end of that year. A baseline rate specific to each age (y_{0i}) can also be supplied. The following formula expresses the long-term increase in risk, or equivalently, the decrease in the probability of not experiencing the adverse event before the end of the window:[12]

$$\Delta\text{Risk} = \prod_i (1 - y_{0i}) - \prod_i (1 - y_{0i} - \Delta y_i) \quad (5)$$

Figure 1 illustrates this approach. The following section provides a series of worked examples, culminating in the results reported in Table 11.

[11] The 30-year duration is based on a 90th percentile of California residency times. For cancer-risk assessment, it also includes the third trimester of pregnancy. For additional details, see OEHHA (2012, chap. 11).

[12] For simplicity and consistency, this methodology defines the time at risk to be identical to the exposure window. Future work could theoretically extend the time at risk, or shift it, if enough information on lag structure became available. There are limits: while cancer risk can be framed as an increase in "lifetime risk," an increase in the lifetime risk of mortality is not a meaningful concept.

3 Example Calculations

This section illustrates the application of the concepts and methods described above. Example calculations are provided for a hypothetical source-specific contribution ΔC = +0.1 μg/m^3 to the modeled annual average $PM_{2.5}$ concentration at the receptor location.[13] Table 11 summarizes the results obtained, following the same procedure, for $PM_{2.5}$ increments spanning several orders of magnitude. Values from Table 11 can be linearly interpolated to yield good approximations of exact calculations for intermediate values. A supplemental worksheet, available on request, can be used to produce exact calculations.

3.1 Premature mortality for resident and off-site worker receptors

Parameters used in these calculations are listed in Tables Table **1**–Table **4**. When evaluating the mortality endpoint for the residential receptor type, the exposure window is defined to be age 55–84, as explained in Section 2.4. To calculate an incremental average exposure intensity, the modeled annual average outdoor concentration increment (ΔC) is multiplied by factors that describe the overlap between the schedules of the source and receptor (Table 1). For senior residents, consistent with OEHHA recommendations communicated during the development of this methodology, the fraction of time at home (FAH) is assumed to be 100% for every year in the exposure window. The overall conversion factor is then (100%) × (350/365) = 0.96 for every year, and the resulting incremental average exposure intensities (Δx) are 0.96 × 0.1 μg/m^3 = 0.096 μg/m^3.

As described in Section 2.2, the effect size (β) for premature adult mortality is 1.0×10^{-2} (Table 2). The adjustment factor listed in

Table **3**, for the residential receptor type combined with the mortality endpoint, is F = 5. Applying the equations from Section 2.3 to each year within the exposure window, the relative risks of mortality corresponding to the incremental average exposure intensities calculated above are then equal to $\exp[(1.0\times10^{-2}) \times (0.096) \times (5)] \approx 1.0048$.

Comparing two scenarios allows us to assess attributable risk: one scenario for baseline conditions, and another for baseline plus the example $PM_{2.5}$ increment. Baseline mortality rates are obtained for the nine-county Bay Area from the Centers for Disease Control and Prevention (Table 4, CDC 2021). To derive the age-specific annual mortality rates under the baseline-plus-increment scenario, those baseline rates can simply be multiplied by the relative risk calculated in the preceding paragraph, which was 1.0029. Table 5 shows the results (column “Increased”, under “Incidence Rate”).

[13] This hypothetical example increment is on the order of 1% of population-weighted annual average $PM_{2.5}$ concentrations across the Bay Area.

As described in Section 2.4, 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 5 contain the cumulative products of these annual probabilities; they represent the overall probabilities of survival from age 55 until the end of the specified age. For an increment in the modeled annual average concentration (ΔC) of +0.1 μg/m^3, and following the approach to multi-year risk expressed by Equation 5, this results in an overall difference at the end of the 30-year exposure window of 54.365% - 54.204% = 1.6×10^{-3}, or 1,600 per million. Year-by-year calculations are shown in Table 5.

When evaluating the mortality endpoint for the off-site worker receptor type, the exposure window is defined to be age 40–64, as explained in Section 2.4. Consistent with existing HRA guidance (OEHHA 2015; BAAQMD 2021), default assumptions for the off-site worker receptor type include a schedule of 8 hr/day, 5 day/wk, 250 day/yr (Table 1). Also consistent with existing guidance, a health-protective assumption is that the source operates during the same hours of the day, and on the same days of the week, that off-site workers are present.[14] The corresponding worker adjustment factor (WAF), which represents this assumption, will then be (24/8) × (7/5) ≈ 4.2. This yields an overall conversion factor, from concentration to exposure intensity, of (250/365) × (8/24) × (4.2) ≈ 0.96. The adjustment factor listed in

Table **3**, for the off-site worker receptor type combined with the mortality endpoint, is F = 10. For a reference increment ΔC = +0.1 μg/m^3 in the modeled annual average concentration, this results in an overall risk increment of 90.521% - 90.434% = 8.7×10^{-4} (870 per million). Year-by-year calculations are shown in Table 6.

3.2 Pediatric asthma onset for resident, student, and daycare receptors

The incremental risk of pediatric asthma onset is calculated in the same way as mortality. In this case, "survival" translates to remaining asthma-free. Baseline incidence rates (Table 7) are taken from nation-wide estimates derived from 2006–2008 responses to the US Behavioral Risk Factor Surveillance System (BRFSS) Asthma Call-back Survey (Winer et al. 2012). These are the same data provided and used by the US EPA, via the BenMAP-CE platform (US EPA 2022a).

For the residential receptor type, the fraction of time at home is defined to be 100% for age 0–17 (Table 1).[15] The overall conversion factor is then (100%) × (350/365) = 0.96, resulting in an incremental average exposure intensity of 0.096 μg/m^3 (Table 7, column "Δx"). For pediatric asthma onset, the effect size is β = 4.37×10^{-2} (Table 2). The adjustment factor listed in

[14] In the context of HRAs conducted pursuant to New Source Review (NSR) regulations, permit applicants are required to provide information about the operating schedule of a source for which a permit is sought. A continuously operating source will correspond to a worker adjustment factor of 1.0, instead of 4.2.
[15] Air District guidance for cancer-risk assessment allows relaxation of this assumption if no schools are identified within a 1.0×10^{-6} isopleth (BAAQMD 2021).

Table **3**, for the residential receptor type combined with the asthma endpoint, is F = 5. Again following the formula for multi-year risk shown in Equation 5, this results in an overall increase in the risk of pediatric asthma onset of 80.013% - 79.633% = 3.8×10^{-3}, or 3,800 per million. Year-by-year calculations are shown in Table 8.

For the student receptor type corresponding to a K-8 school, the relevant exposure window is age 5–13, and the relevant schedule parameters are 10 hr/day, 5 day/wk, 180 day/yr (Table 1). As with the off-site worker receptor type, a health-protective assumption for screening purposes is that the source operates during the same hours of the day, and on the same days of the week, that students attend school. Applying a schedule-adjustment factor of (24/10) × (7/5) = 3.36 then yields an overall conversion factor, from concentration to exposure intensity, of (180/365) × (10/24) × (3.36) = 0.69. For the student receptor type combined with the asthma onset endpoint, the adjustment factor listed in

Table **3** is F = 10. The increased risk corresponding to ΔC = +0.1 μg/m^3 is then 2.5×10^{-3}, or 2,500 per million (Table 9).

For a daycare receptor, the exposure window is age 0–5, with schedule parameters of 10 hr/day, 250 day/yr. Using a schedule-overlap adjustment factor of 3.36 (Table 1), the overall conversion factor, from concentration to exposure intensity, is then 0.96. For the daycare receptor type combined with the asthma onset endpoint, the adjustment factor listed in

Table **3** is F = 10. The increased risk corresponding to ΔC = +0.1 μg/m^3 is then 4.9×10^{-3}, or 4,900 per million (Table 10).

4 Discussion and Conclusion

4.1 Measures of impact

This methodology quantifies impact in terms of increased risk, with risk defined as the probability of an adverse outcome occurring during a specified interval of time. Premature mortality is one of the most well-studied consequences of $PM_{2.5}$ exposure, and in contemporary population-wide assessments, increases in mortality typically receive over 90% of an overall monetary valuation. Feedback from stakeholders, and the Air District's Advisory Council, indicated that it was critical to assess at least one other endpoint. Asthma, in particular, is a prominent concern for community members and community representatives. Asthma onset (newly diagnosed) was selected as a second endpoint because it also receives a relatively high valuation in the Air District's current population-based assessments, and because it is a necessary condition for other asthma-related outcomes, such as hospitalizations. Premature mortality and pediatric asthma onset can both be quantified in terms of risk, since they are both irreversible and binary.[16]

This methodology does not attempt to consolidate measures of risk across more than one endpoint. Nor does it attempt to be exhaustive. Long-term exposure to $PM_{2.5}$ has very broad effects, and evidence continues to accumulate for other endpoints. As one example, the evidence base for neurological effects continues to grow, with a large and recent study of the Medicare cohort (Shi et al 2020) reporting a hazard ratio of 1.13 (1.12, 1.14) per 5 $\mu g/m^3$ $PM_{2.5}$, or 1.025 per $\mu g/m^3$ ($\beta = 2.44\times10^{-2}$), for the onset of Parkinson's Disease. The same effect size was reported for Alzheimer's as well. There is also evidence for adverse birth outcomes, such as preterm birth (odds ratio = 1.164 (1.135, 1.195) per 6.96 $\mu g/m^3$; $\beta = 2.18\times10^{-2}$; Basu et al 2017). It is important to note that if the baseline annual incidence rates (y_o) for a given endpoint are relatively low, then the risk increments associated with that endpoint may not be high, even if the relative risks (β) are high.[17] More endpoints could be assessed, if it became clear that this would make a practical difference to policy or risk-management outcomes.[18] See Section 4.5 ("Conclusion") for a brief discussion of implications for risk management.

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[16] While asthma symptoms can disappear temporarily or permanently in some cases, this methodology adopts the definition "ever diagnosed," consistent with BenMAP-CE and Tetréault et al. (2016).

[17] A previous study (Kioumourtzoglou et al. 2016) had estimated larger effect sizes for these two neurological endpoints, but with less precision. Evidence should also be expected to accumulate for respiratory endpoints, and it is not unusual for later, higher-powered studies to report smaller effect sizes than earlier studies. Thus, while the incremental risks reported in Table 11 for residential receptors are currently larger for pediatric asthma onset than for respiratory mortality, this may not always be the case.

[18] For example: between the ages of 55–84, baseline annual incidence rates (y_o) for Alzheimer's are larger than those for Parkinson's, but below those for all-cause mortality. The ratio is approximately 1 to 2 orders of magnitude, depending on age. So, although the *relative* risk for Alzheimer's—relative to y_o, that is—may be more than three times as large as the relative risk for all-cause mortality, the 30-year risk increment can still be smaller on an additive scale. Using the baseline rate data supplied by the BenMAP platform (US EPA 2022), and assuming the same adjustment factor ($F = 5$), it would be smaller by approximately a factor of five.

4.2 Relationship to annual incidence

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. These annual measures of impact across the general population are useful benchmarks, are statistically significant, and are supported by multiple scientific literatures (US EPA 2019, 2022b). If they are calculated using the effect-size parameters (β) listed in Table 2, the results will be consistent with the Air District's most recent modeling of annual health and welfare impacts for the Bay Area's regional population using BenMAP-CE (US EPA 2022a; Tanrikulu et al. 2011, 2022).

For the segment of the Bay Area population aged 55–84, using BenMAP to assess the impact of a constant and uniform +0.1 μg/m^3 increase in the annual average $PM_{2.5}$ concentration for the year 2020 produces an estimated increase of approximately 1.3×10^{-5} in the rate of mortality per person per year. For the same increase in $PM_{2.5}$, the methodology described in this document yields a mortality-risk score, for the residential receptor type, of 9.7×10^{-4}. These two results may seem far apart, but they are logically and theoretically coherent. Both calculations begin with baseline mortality rate data for Bay Area residents, and both use the same core equations to calculate relative risk (Section 2.2). This risk-oriented methodology then introduces an adjustment factor to account for higher-risk situations and sensitive groups, as described in Section 2.3. The remaining difference is explained by the long-term accumulation of risk over many years, rather than just one year (Section 2.4). This last step is consistent with existing HRA principles, and uses exactly the same durations for "long term" exposure prescribed by existing HRA guidance (BAAQMD 2021; OEHHA 2012, 2015).

The two approaches share a common methodological foundation, but are aimed at characterizing different aspects of a modeled world: net annual impacts for large Bay Area populations, *vs* potential long-term impacts under certain scenarios involving higher risk.[19] As such, they are both useful tools for risk assessment and risk management. Particularly in the case of larger or more ubiquitous sources, estimating net impacts for a large population can be a valuable complement or alternative to this methodology (see, e.g., OEHHA 2012 chap. 11).

4.3 Linearity and adjustments

This methodology assumes that, within the scenarios that are its focus, there are approximately linear relationships between changes in concentration, exposure, intake, and effect at a group level. An assumption of linearity is consistent with the position expressed in the National Academy of Sciences (2009) report, in which $PM_{2.5}$ is classed as a pollutant "with noncancer endpoints for which the evidence points to a linear or other non-threshold population response at low doses" and with the US EPA's finding that extensive epidemiologic evidence provides support for a linear, no-threshold concentration-response relationship for mortality (EPA

[19] For exactly the same reasons, a calculated increase in "lifetime cancer risk" will be much higher (in terms of the raw number) than a calculated increase in the annual incidence of cancer (per capita) across the Bay Area, given the same hypothetical increase in the concentration of some carcinogen.

2022b). Impacts on mortality and respiratory endpoints are observed in populations at or below current Bay Area levels, and while the existence of a population-level threshold at very low concentrations (e.g. 5 μg/m^3 or lower) remains a theoretical possibility, no such threshold has yet been observed. This methodology is intended to apply to increments above concentrations at which a susceptible group might experience an effect.

The use of F to multiplicatively adjust the exponential term in the core equations reflects this linearity assumption. The equations in Section 2 are technically supra-linear. However, given (a) the range of concentration increments specified by Table 11, (b) the magnitudes of β considered here for mortality and asthma, and (c) the magnitudes of F listed in Table 2, the product $\beta \cdot \Delta x \cdot F$ remains small enough that departures from linearity will be on the order of a few percent at most.

Any future extensions of this methodology to incorporate additional endpoints, or to revise the values for β listed in this document, should carefully consider what new value(s) for F might be most appropriate in the context of new studies. A risk factor that is distributed similarly among a studied population and a corresponding target receptor type should not need to be accounted for via F. When there are substantial differences between those two distributions, the key consideration is not whether *any* individuals in the studied population(s) exhibited the risk factor,[20] but whether the estimate of β would have been substantially different had the studied person-time consisted entirely of such individuals in such contexts.

As noted in Section 2.3, the approach taken in this white paper is not necessarily the only way that values for F could be determined. The intent of this white paper is to present one viable option for policymakers to consider. A different bottom-up set of components might in the end be considered more appropriate. Or, other approaches to arriving at values for F, or components of F, may ultimately be preferred.

4.4 Baseline rates

In this methodology, the region-level baseline rates y_o are age-specific, but not place-specific, other than being specific to the Bay Area as a whole. This makes the values stable over time, and unlikely to vary by more than a few percent from rates calculated at a state level. They are not made to vary along any other dimension, such as demographic characteristics (e.g. race/ethnicity) or geography (e.g., county or census tract). There are two primary reasons.

One reason for using region-level baseline rates is that small-area population data can easily be inaccurate, imprecise, or outdated (Hubbell et al. 2009). The spatial scales that correspond to the distances between most local sources and their maximally impacted receptors are expected to be, at least in many urban locations, the size of a Census block or smaller. Such micro-data often have unreported or frequently misunderstood sources of variation, uncertainty, and/or

[20] This would be relevant if the goal were to establish a reference level for adverse effects.

error.[21] Consequently, a false sense of precision or reliability can be carried through to risk communication or decision-making. Statistical summaries at a larger scale—as provided, for example, by the BenMAP platform—are more reliable. This is especially so when the focus is on net or average expected impacts. But this methodology is focused on scenarios with higher-than-average risk.

Another reason for using regional baseline rates is that even good data can contribute to inadvisable conclusions. This can happen if the model does not reflect a complete picture. For example, it is well known that baseline rates can be differentially higher or lower for reasons unrelated to air pollution. If other adjustments are not or cannot be made, this can result in a misleading picture, given the functional form of the key equations. More concretely: the fact that Hispanic/Latino communities typically have lower baseline rates of mortality would cause calculated mortality risk scores to be lower for a new $PM_{2.5}$ source sited in a Hispanic/Latino community. In the case of this specific racial/ethnic group, there happens to be additional information that points the other way; differences in effect size (β) for Hispanic/Latino populations (e.g., Di et al. 2017) may outweigh these differences in baseline rates. Comparable information regarding effect modification for pediatric asthma onset is as yet unavailable.

Because the science on geographic and demographic predictors of susceptibility is still evolving (Hickens 2023), this methodology opts to address the potential for additional risk as a matter of uncertainty, rather than as a gap to be filled by small-area estimates of demographics or baseline rates. Acknowledging that new scientific understandings will inevitably emerge, the factor F is intended to be adequately protective of sensitive and vulnerable individuals across multiple dimensions. It is worth noting that, 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. It is also recommended that equity-focused extensions be considered at a risk management or policy level. These could take the form of refinements to the parameters provided in this white paper, or the establishment of context-specific thresholds, for example.

4.5 Conclusion

This methodology, developed through a transparent public process and vetted by experts, offers support for HRA-style assessments applied to local increments of $PM_{2.5}$. As mentioned in the Introduction, it addresses a crucial gap left by regionally-focused methods, standards, and regulatory approaches to $PM_{2.5}$. It shares many similarities with well-established HRA methodologies used for assessing risks from carcinogens: it is source-specific and based on modeling, and it focuses on quantifying the potential for higher-than-average long-term risks. Although it has a different aim, it is also logically consistent with other approaches and tools

[21] In the case of micro-scale Census data, error is introduced intentionally by the U.S. government. This is to protect vulnerable individuals from identification. The magnitude of the error generally subsides as the data are aggregated to larger scales, although it tends to persist more for smaller demographic groups.

that yield complementary measures of the impacts of $PM_{2.5}$ across large populations, such as the US EPA's BenMAP platform.

Determining how best to implement this methodology is an appropriate topic for a future public process. Health risk assessments for local sources of pollution are often associated with threshold-based policies or guidance. If a threshold-based approach is desired, further work will be necessary to establish an appropriate metric or method for combining multiple metrics to guide threshold-based decision-making. In undertaking that work, policymakers should remain aware that $PM_{2.5}$ has been linked to many other health endpoints in addition to mortality and asthma. From a practical perspective, for a given receptor type, setting a threshold in terms of risk for any one endpoint will effectively set it in terms of any other endpoints, conditional on the lengths of the exposure windows. Only the strictest threshold—that is, the one corresponding to the lowest equivalent concentration increment (ΔC)—will matter. For any threshold, this methodology supplies a way to express some of the residual risks, allowing that there may be uncertainties and unknown risks. It falls to policymakers and stakeholders to determine an appropriate means of managing these as a whole.

5 Figures and Tables

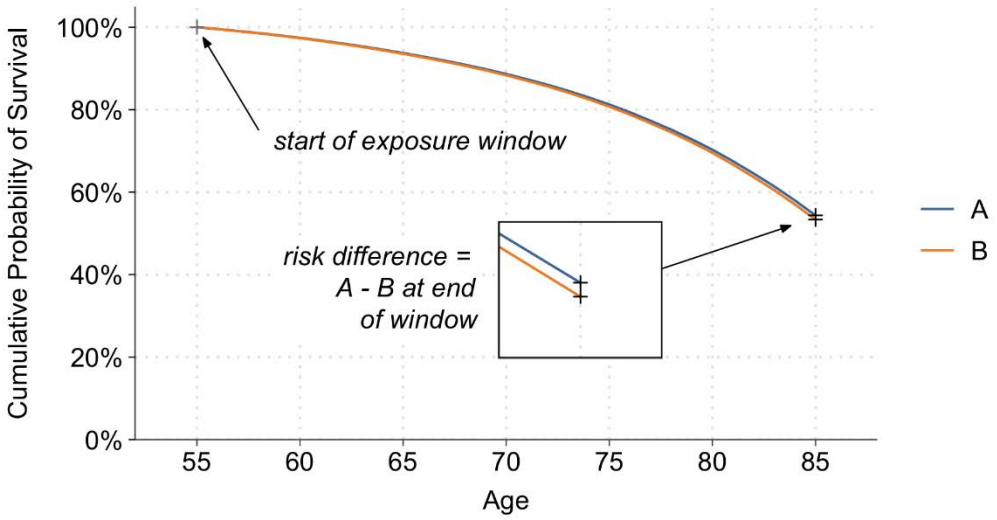

Figure 1. Illustration of the method applied to a multi-year exposure window. Scenario A is the baseline scenario, representing exposure to baseline levels of $PM_{2.5}$. Scenario B corresponds to an additional increment of $PM_{2.5}$. At the beginning of the exposure window, the receptor has not yet experienced the adverse event (e.g., mortality or asthma onset). In the case of pediatric asthma onset, "survival" would correspond to remaining asthma-free, and the exposure window would be shifted toward younger ages.

Table 1. Receptor types, exposure-related parameters, and default values. Default values can be superseded by more site-specific information in the context of a specific HRA (see table footnotes).

	Endpoint	Exposure Window (age)	Exposure Duration (yr)	Fraction of time at Home*	Exposure fraction (day/yr)	Exposure Time (hr/day)	Exposure Adjustment Factor†
Resident	Mortality	55–84	30	100%	350	—	—
	Asthma	0–17	18	100%	350	—	—
Worker	Mortality	40–64	25	—	250	8	4.2
Student	Asthma	5–13	9	—	180	10	3.36
Daycare	Asthma	0–5	6	—	250	10	3.36

* 100% FAH for residents aged 16–17 and 55+ departs from the 73% used in cancer-risk assessment, but has been explicitly recommended by OEHHA during the public process used to develop this methodology.

† Also known as Worker Adjustment Factor (WAF) in the context of the off-site worker receptor type; for school and daycare receptor types, the concept is the same. Used to adjust an annual-average modeling result, here by default assuming 100% overlap between intra-week and intra-day source and receptor schedules (i.e., discount factor DF = 1). Consistent with other HRA guidance for non-threshold effects, there will still be non-overlap of approximately 4% due to 2 weeks of "away" per year (e.g. resident = 350/365 days; worker and daycare = 250/365 days ≈ 50/52 weeks; described as "vacation" in OEHHA 2012). If the modeled source's emissions are continuous, the EAF should be 1.0. All values in other tables are calculated using the EAFs listed in this table.

Table 2. Health endpoints, effect sizes (β) adopted for this methodology, and key studies. To standardize a relative risk RR_u from "per u μg/m³" to "per 1 μg/m³", the formula is $RR_1 = (RR_u)^{(1/u)}$. β is equal to $\ln(RR_1)$ or, equivalently, $\ln(RR_u)/u$.

			Relative Risk (RR)	
Endpoint	**β**	**Key Studies**	**Reported As**	**Standardized**
Asthma onset, pediatric	4.37×10^{-2}	Tetréault et al (2016)	1.33 (1.31, 1.34) per 6.53 μg/m³	1.045 per 1 μg/m³
Premature mortality, adult	1.0×10^{-2}	Di et al (2017)	1.073 (1.071, 1.075) per 10 μg/m³	1.007 per 1 μg/m³
		Di et al (2017)[a]	1.136 (1.131, 1.141) per 10 μg/m³	1.013 per 1 μg/m³
		Di et al (2017)[b]	1.11 (1.08, 1.15) per 10 μg/m³	1.010 per 1 μg/m³
		Vodonos et al (2018)[b,c]	1.0129 (1.0109, 1.0150) per 1 μg/m³	1.0129 per 1 μg/m³
		Wu et al (2020)	1.06 (1.05, 1.08) to 1.08 (1.07, 1.09) per 10 μg/m³	1.006 to 1.008 per 1 μg/m³
		Wu et al (2020)[a]	1.23 (1.18, 1.28) to 1.37 (1.34, 1.40) per 10 μg/m³	1.021 to 1.032 per 1 μg/m³

[a] For exposures less than 12 μg/m³

[b] Systematically derived from results reported by multiple previous studies

[c] Evaluated at 10 μg/m³ baseline

Table 3. Receptor types, endpoints, and values for the overall adjustment factor F. The values shown for F are informed by the products of the values in the last three columns. For details, see Section 2.3.

Receptor	**Endpoint**	**Overall F**	**Sensitive Groups (Inter-Individual Variation)**	**Exposure Modification (Lack of Shelter)**	**Intake Modification (Increased Exertion)**
Resident	Mortality	5	3	1.5	—
Resident	Asthma	5	3	1.5	—
Worker	Mortality	10	3	1.5	2
Student	Asthma	10	3	1.5	2
Daycare	Asthma	10	3	1.5	2

Table 4. Baseline mortality rates (per 100,000) for the nine-county Bay Area, 2007–2016 (CDC 2021).

Age	Person-Years	Deaths	Rate
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 5. Mortality rates and cumulative probabilities of survival for a residential receptor, age 55–84, given a modeled increase in annual average $PM_{2.5}$ concentration ΔC = +0.1 μg/m^3, an effect size β = 1.0×10^{-2}, and an adjustment factor F = 5. The final result is 54.365% - 54.204% = 1.6×10^{-3}.

		Incidence Rate (per 100,000)			Survival (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
55	0.096	454.07	1.00481	456.25	99.546%	99.544%
56	0.096	482.85	1.00481	485.17	99.065%	99.061%
57	0.096	500.01	1.00481	502.42	98.570%	98.563%
58	0.096	560.45	1.00481	563.14	98.018%	98.008%
59	0.096	610.56	1.00481	613.50	97.419%	97.407%
60	0.096	654.68	1.00481	657.83	96.781%	96.766%
61	0.096	715.71	1.00481	719.15	96.089%	96.070%
62	0.096	756.55	1.00481	760.19	95.362%	95.340%
63	0.096	831.57	1.00481	835.57	94.569%	94.543%
64	0.096	882.14	1.00481	886.38	93.734%	93.705%
65	0.096	950.72	1.00481	955.29	92.843%	92.810%
66	0.096	995.94	1.00481	1,000.72	91.919%	91.881%
67	0.096	1,108.88	1.00481	1,114.21	90.899%	90.857%
68	0.096	1,180.36	1.00481	1,186.03	89.826%	89.780%
69	0.096	1,303.55	1.00481	1,309.81	88.655%	88.604%
70	0.096	1,443.77	1.00481	1,450.71	87.375%	87.319%
71	0.096	1,521.17	1.00481	1,528.48	86.046%	85.984%
72	0.096	1,719.59	1.00481	1,727.86	84.567%	84.498%
73	0.096	1,882.08	1.00481	1,891.12	82.975%	82.900%
74	0.096	2,075.25	1.00481	2,085.23	81.253%	81.172%
75	0.096	2,322.46	1.00481	2,333.62	79.366%	79.277%
76	0.096	2,581.23	1.00481	2,593.64	77.317%	77.221%
77	0.096	2,781.05	1.00481	2,794.42	75.167%	75.063%
78	0.096	3,132.95	1.00481	3,148.01	72.812%	72.700%
79	0.096	3,462.71	1.00481	3,479.35	70.291%	70.171%
80	0.096	3,976.83	1.00481	3,995.94	67.496%	67.367%
81	0.096	4,420.68	1.00481	4,441.92	64.512%	64.374%
82	0.096	4,829.58	1.00481	4,852.79	61.396%	61.250%
83	0.096	5,556.48	1.00481	5,583.19	57.985%	57.831%
84	0.096	6,241.94	1.00481	6,271.94	**54.365%**	**54.204%**

Δx = Incremental annual average exposure intensity (μg/m^3).

Baseline = Scenario representing baseline $PM_{2.5}$ level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp(β·Δx·F). (Baseline Rate) x (Ratio) = (Increased Rate).

Table 6. Mortality rates and cumulative probabilities of survival for an off-site worker receptor, age 40–64, given a modeled increase in annual average $PM_{2.5}$ concentration ΔC = +0.1 μg/m^3, effect size β = 1.0×10^{-2}, and adjustment factor F = 10. The final result is 90.521% - 90.434% = 8.7×10^{-4}.

		Incidence Rate (per 100,000)			Survival (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
40	0.096	106.10	1.00964	107.12	99.894%	99.893%
41	0.096	122.45	1.00964	123.63	99.772%	99.769%
42	0.096	134.42	1.00964	135.71	99.637%	99.634%
43	0.096	149.50	1.00964	150.95	99.489%	99.484%
44	0.096	160.38	1.00964	161.92	99.329%	99.323%
45	0.096	169.97	1.00964	171.60	99.160%	99.152%
46	0.096	196.85	1.00964	198.75	98.965%	98.955%
47	0.096	215.95	1.00964	218.03	98.751%	98.739%
48	0.096	237.18	1.00964	239.47	98.517%	98.503%
49	0.096	263.80	1.00964	266.34	98.257%	98.240%
50	0.096	291.81	1.00964	294.62	97.970%	97.951%
51	0.096	311.65	1.00964	314.65	97.665%	97.643%
52	0.096	337.25	1.00964	340.50	97.336%	97.310%
53	0.096	378.24	1.00964	381.89	96.968%	96.939%
54	0.096	408.32	1.00964	412.25	96.572%	96.539%
55	0.096	454.07	1.00964	458.45	96.133%	96.097%
56	0.096	482.85	1.00964	487.50	95.669%	95.628%
57	0.096	500.01	1.00964	504.83	95.191%	95.145%
58	0.096	560.45	1.00964	565.84	94.657%	94.607%
59	0.096	610.56	1.00964	616.44	94.079%	94.024%
60	0.096	654.68	1.00964	660.99	93.463%	93.402%
61	0.096	715.71	1.00964	722.61	92.794%	92.727%
62	0.096	756.55	1.00964	763.84	92.092%	92.019%
63	0.096	831.57	1.00964	839.58	91.326%	91.246%
64	0.096	882.14	1.00964	890.64	**90.521%**	**90.434%**

Δx = Incremental annual average exposure intensity (μg/m^3).

Baseline = Scenario representing baseline $PM_{2.5}$ level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp(β·Δx·F). (Baseline Rate) x (Ratio) = (Increased Rate).

Table 7. Baseline incidence rates (per 1,000) for pediatric asthma onset (US EPA 2022; Winer et al. 2012).

Age	Rate
0–4	23.4
5–11	11.1
12–17	4.4

Table 8. Incidence rates and cumulative probabilities of remaining asthma-free for a residential receptor, age 0–17, given a modeled increase in annual average $PM_{2.5}$ concentration ΔC = +0.1 μg/m^3, effect size β = 4.37×10^{-2}, and adjustment factor F = 5. The final result is 80.013% - 79.633% = 3.8×10^{-3}.

		Incidence Rate (per 1,000)			Asthma-Free (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
0	0.096	23.4	1.02116	23.895	97.660%	97.610%
1	0.096	23.4	1.02116	23.895	95.375%	95.278%
2	0.096	23.4	1.02116	23.895	93.143%	93.001%
3	0.096	23.4	1.02116	23.895	90.963%	90.779%
4	0.096	23.4	1.02116	23.895	88.835%	88.610%
5	0.096	11.1	1.02116	11.335	87.849%	87.606%
6	0.096	11.1	1.02116	11.335	86.874%	86.613%
7	0.096	11.1	1.02116	11.335	85.909%	85.631%
8	0.096	11.1	1.02116	11.335	84.956%	84.660%
9	0.096	11.1	1.02116	11.335	84.013%	83.701%
10	0.096	11.1	1.02116	11.335	83.080%	82.752%
11	0.096	11.1	1.02116	11.335	82.158%	81.814%
12	0.096	4.4	1.02116	4.493	81.797%	81.446%
13	0.096	4.4	1.02116	4.493	81.437%	81.080%
14	0.096	4.4	1.02116	4.493	81.078%	80.716%
15	0.096	4.4	1.02116	4.493	80.722%	80.353%
16	0.096	4.4	1.02116	4.493	80.366%	79.992%
17	0.096	4.4	1.02116	4.493	**80.013%**	**79.633%**

Δx = Incremental annual average exposure intensity (μg/m^3).

Baseline = Scenario representing baseline $PM_{2.5}$ level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp(β·Δx·F). (Baseline Rate) x (Ratio) = (Increased Rate).

Table 9. Incidence rates and cumulative probabilities of remaining asthma-free for a student receptor, age 5–13, given a modeled increase in annual average $PM_{2.5}$ concentration ΔC = +0.1 μg/m^3, effect size β = 4.37×10^{-2}, and adjustment factor F = 10. The final result is 91.672% - 91.427% = 2.5×10^{-3}.

		Incidence Rate (per 1,000)			Asthma-Free (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
5	0.069	11.1	1.0306	11.440	98.890%	98.856%
6	0.069	11.1	1.0306	11.440	97.792%	97.725%
7	0.069	11.1	1.0306	11.440	96.707%	96.607%
8	0.069	11.1	1.0306	11.440	95.633%	95.502%
9	0.069	11.1	1.0306	11.440	94.572%	94.409%
10	0.069	11.1	1.0306	11.440	93.522%	93.329%
11	0.069	11.1	1.0306	11.440	92.484%	92.262%
12	0.069	4.4	1.0306	4.535	92.077%	91.843%
13	0.069	4.4	1.0306	4.535	**91.672%**	**91.427%**

Δx = Incremental annual average exposure intensity (μg/m^3).

Baseline = Scenario representing baseline $PM_{2.5}$ level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp(β·Δx·F). (Baseline Rate) x (Ratio) = (Increased Rate).

Table 10. Incidence rates and cumulative probabilities of remaining asthma-free for a daycare receptor, age 0–5, given a modeled increase in annual average $PM_{2.5}$ concentration ΔC = +0.1 μg/m^3, effect size β = 4.37×10^{-2}, and adjustment factor F = 10. The final result is 87.849% - 87.358% = 4.9×10^{-3}.

		Incidence Rate (per 1,000)			Asthma-Free (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
0	0.096	23.4	1.0428	24.401	97.660%	97.560%
1	0.096	23.4	1.0428	24.401	95.375%	95.179%
2	0.096	23.4	1.0428	24.401	93.143%	92.857%
3	0.096	23.4	1.0428	24.401	90.963%	90.591%
4	0.096	23.4	1.0428	24.401	88.835%	88.381%
5	0.096	11.1	1.0428	11.575	**87.849%**	**87.358%**

Δx = Incremental annual average exposure intensity (μg/m^3).

Baseline = Scenario representing baseline $PM_{2.5}$ level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp(β·Δx·F). (Baseline Rate) x (Ratio) = (Increased Rate).

Table 11. Lookup table. Exposure windows are indicated in parentheses. All parameters, other than the concentration increment (ΔC), are as described in Sections 2 and 3.

Annual Average Concentration Increment (ΔC)	Premature Mortality		Pediatric Asthma Onset		
	Resident (55–84)	Worker (40–64)	Resident (0–17)	Student (5–13)	Daycare (0–5)
3×10^{-1} µg/m³	4.9×10^{-3}	2.6×10^{-3}	1.2×10^{-2}	7.6×10^{-3}	1.5×10^{-2}
1×10^{-1} µg/m³	1.6×10^{-3}	8.7×10^{-4}	3.8×10^{-3}	2.5×10^{-3}	4.9×10^{-3}
3×10^{-2} µg/m³	4.8×10^{-4}	2.6×10^{-4}	1.1×10^{-3}	7.3×10^{-4}	1.5×10^{-3}
1×10^{-2} µg/m³	1.6×10^{-4}	8.7×10^{-5}	3.8×10^{-4}	2.4×10^{-4}	4.8×10^{-4}
3×10^{-3} µg/m³	4.8×10^{-5}	2.6×10^{-5}	1.1×10^{-4}	7.3×10^{-5}	1.4×10^{-4}
1×10^{-3} µg/m³	1.6×10^{-5}	8.7×10^{-6}	3.8×10^{-5}	2.4×10^{-5}	4.8×10^{-5}

Table 12. Protective approaches applied to key components of the methodology.

Component	Protective Aspect(s)
Concentration	For each class of receptor (resident, worker, etc.), the maximally impacted potential location is selected.
Co-presence with local source (while emitting)	For the residential receptor type, the fraction of time at home (FAH) is assumed to be 100%. For worker, student, and daycare receptor types, a near-100% overlap in intra-week schedules (source vs receptor) may be assumed for screening purposes, as illustrated in this document.*
Exposure duration and timing	The duration of exposure is consistent with existing HRA guidance for long-term risk assessments. The timing of the exposure window aligns with higher baseline rates.
Other factors: physical, social, environmental, etc.	A multiplicative factor F accounts for scenarios with higher risk due to a combination of inter-individual variability and contextual factors, relative to the basis for β.

* It is possible for site-specific HRAs to have less than 100% overlap; see Table 1 footnote regarding the Exposure Adjustment Factor (EAF).

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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: Philip M. Fine
Executive Officer/APCO

Date: September 11, 2023

Re: Vote to Submit Letter of Support to Air District Board of Directors

RECOMMENDED ACTION

The Advisory Council will consider submitting a letter to the Air District's Board of Directors in support of the white paper, *Modeling Health Risks from Local Sources of Fine Particulate Matter ($PM_{2.5}$)*, version 2.0 (August 2023).

BACKGROUND

Over several meetings in 2022, Air District staff began discussion with the Advisory Council on the Air District's efforts to develop a $PM_{2.5}$ local risk methodology and consider key questions to help guide those efforts. The Advisory Council provided feedback for incorporation and in fall 2022, the Air District released a draft white paper, *Proposed Methodology for Determining Local Health Risks from Fine Particulate Matter* for public comment.

The Air District extended invitations to each commentor to present and share information with the Advisory Council. In early 2023, the Advisory Council received presentations from three of the organizations that provided public comment: Christine Wolfe, Policy and Communications Director, California Council for Environmental and Economic Balance, Ken Szutu, Founder and Director, Citizen Air Monitoring Network and Dr. Julie Goodman, Gradient, on behalf of Western States Petroleum Association. In June 2023, the Advisory Council received presentations from both Air District staff and staff from the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA), regarding feedback on the proposed methodology for modeling health risks from local sources of fine particulate matter ($PM_{2.5}$). Presenters from OEHHA included Dr. Lauren Zeise, Dr. Keita Ebisu, Dr. Rupa Basu and Dr. Vincent Cogliano.

DISCUSSION

Today, the Air District has presented the most recent version of the methodology to the Advisory Council. The Advisory Council will consider a letter to the Air District Board of Directors offering their support for his methodology and encouraging the Air District to continue this

important work toward reducing particulate matter in the Bay Area.

BUDGET CONSIDERATION/FINANCIAL IMPACT

None.

Respectfully submitted,

Philip M. Fine
Executive Officer/APCO

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

ATTACHMENTS:

1. Draft Advisory Council Letter to the Board of Directors

BAY AREA AIR QUALITY MANAGEMENT DISTRICT

September 11, 2023

Board of Directors
Bay Area Air Quality Management District
375 Beale St, Ste 600
San Francisco, CA 94105

RE: $PM_{2.5}$ Local Risk Methodology

Dear Chair Bauters and Members of the Board:

Over the past five years the Advisory Council has worked with Air District staff to develop a strong scientific foundation and methodology to improve protection of public health from fine particulate matter ($PM_{2.5}$). Specifically, in 2019, the Advisory Council convened a series of symposia on the health effects of $PM_{2.5}$. During the symposia we heard from subject matter experts, industry representatives, community members and others. The series culminated in the 2020 Advisory Council *Particulate Matter Reduction Strategy Report*.

Our 2020 report found that low-income communities of color are disproportionately impacted by $PM_{2.5}$. Furthermore, epidemiological research has demonstrated that people living in these communities have more serious health impacts from $PM_{2.5}$, even given the same level of exposure. The report also noted that "substantially elevated $PM_{2.5}$ exposures can occur in locations adjacent to local PM sources. Therefore, controlling emissions in these local impacted areas is of primary importance."

Over the past three years, guided by the Advisory Council, Air District staff has developed a new methodology to model increases in certain health risks resulting from local $PM_{2.5}$ exposures. The Air District has experience in modeling source-specific contributions to ambient concentrations of $PM_{2.5}$, but to date had not conducted any corresponding health risk assessments. This new methodology would enable those assessments, filling an important gap in the Air District's regulatory toolbox.

The methodology has benefited from expert review by staff at the California Office of Environmental Health Hazard Assessment (OEHHA), the California Air Resources Board (CARB), and the US Environmental Protection Agency (EPA). Key feedback was also provided by independent scientists, healthcare providers, industry, non-governmental organizations and community members through a transparent public process, in the form of public comments on the draft methodology and presentations to the Advisory Council. The methodology has been documented in detail in the white paper, *Modeling Health Risks from Local Sources of Fine Particulate Matter ($PM_{2.5}$)*, version 2.0 (August 2023).

Similar to the approach taken to regulate toxic air contaminants (TACs), this methodology is designed to protect highly exposed susceptible groups, under

higher-risk scenarios potentially involving exposures over multiple years. This methodology focuses on two health outcomes: (1) increased risk of premature adult mortality and (2) pediatric asthma onset. The Advisory Council concluded that these outcomes are strongly scientifically attributable to $PM_{2.5}$ exposures and have sufficiently well-understood dose-response relationships.

In addition to drawing on epidemiological knowledge, this methodology also draws on exposure science, taking into account the potential for Bay Area residents to be inhabiting "leaky" buildings, for students to be attending classes with open windows, for children at daycare to be playing outside, and for nearby workers to be laboring outdoors, directly exposed to $PM_{2.5}$ from the local source. Finally, it accounts for increased activity levels and breathing rates associated with certain locations such as outdoor workplaces. The adjustment factors that account for these potential situations have been carefully developed and vetted through the same public process and are consistent with the treatment of potential long-term exposure and the principle of health-protectiveness embodied in the Air District's existing guidance for health risk assessments of toxic air contaminants.

The application of this methodology would represent a substantial advance in environmental regulation, in keeping with the spirit and reputation of Bay Area policymakers as leaders in protecting and promoting public health. The Advisory Council notes that the US EPA has proposed to lower the annual average standard for $PM_{2.5}$. That revision would represent an appropriate advancement in the regulation of $PM_{2.5}$ at a regional level, but the EPA's action would still leave important gaps pertaining to source-specific local exposures, which this methodology can address.

The Advisory Council has fully reviewed the proposed $PM_{2.5}$ methodology and endorses it as scientifically sound and necessary to protect public health. Please let us know if we can be of further assistance to the Board as you consider the scientific and health aspects of the proposed methodology.

Sincerely,

Gina Solomon, MD, MPH Linda Rudolph, MD, MPH

Co-Chairs
Bay Area Air Quality Management District Advisory Council

Cc: Dr. Philip Fine, Executive Officer/Air Pollution Control Officer, Bay Area Air Quality Management District
Greg Nudd, Deputy Executive Officer, Bay Area Air Quality Management District