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

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Bay Area Air Quality Management District

Project Lead

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

Reviewed by

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

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

Advisors and Contributors

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

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

Judith Cutino, DO, Health Officer
Policy and Equity Office

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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
CEQA	California Environmental Quality Act
HRA	Health risk assessment
MEI	Maximally exposed individual
NAAQS	National Ambient Air Quality Standards
OEHHA	Office of Environmental Health Hazard Assessment
$PM_{2.5}$	Particulate matter less than 2.5 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

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

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

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

Modeling of responses to exposure. To re-express the modeled incremental average exposure intensities in the form of health risks, we leverage response functions from epidemiologic studies of the health effects of $PM_{2.5}$. In this version of the methodology, we leverage response

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

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

functions for (a) premature adult mortality and (b) pediatric asthma onset, applying these to residential, off-site worker, school, and daycare receptors.

The response functions that we rely on are used to calculate relative risks. We convert these to risk differences using information about baseline rates.[3] To illustrate: suppose we take the *relative* risk of asthma onset, per μg/m^3, to be 1.04 for five-year-old children. Suppose that we further take the baseline annual incidence rate to be 10 per 1,000; that is, on average we expect 1% of asthma-free five-year-olds to develop asthma before turning six, given a baseline level of exposure.[4] For a scenario in which the annual average exposure intensity at a corresponding receptor is increased by 1 μg/m^3, we take that baseline rate and multiply it by 1.04. Subtracting the baseline rate from this scaled result yields an estimate of the excess risk of developing asthma between the ages of five and six, compared to the baseline scenario. In this case, that difference is $0.01 \times 1.04 - 0.01 = 4\times10^{-4}$.

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

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

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

The effect size, or the change in y associated with a unit change in x, is represented in these equations by the term β. Typically, β will be based on an epidemiologic study in which ambient outdoor $PM_{2.5}$, measured or estimated at some locations, was the independent variable. Generally, epidemiologic studies estimate β by adjusting for other measured factors in such a way that β will (ideally) approximate the causal effect of x alone. Most such studies report an estimated risk ratio, such as a relative risk (RR), for a given increment of $PM_{2.5}$. In the equations above, β is the natural logarithm of that risk ratio.[5]

When assessing population impacts, these response functions include population size as a multiplier. In omitting that multiplier—or, equivalently, dividing the total expected impact by the size of the population—we are obtaining a result for a "statistically average individual." Such an entity does not represent any actual individual, but the result corresponds to the result we would expect if we modeled a large sample of a representative population and then took

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

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

the average of those results. The marginal effect size that β then represents will reflect the distribution of factors that lay on the causal pathways between ambient $PM_{2.5}$ and the outcome of interest in the population that was studied. For example, the breathing rates of the studied population will be implicit in the population-average estimate of β. In Section 4, to account for at-risk populations, we incorporate adjustments to some of these factors.

Multi-year exposures. To extend the exposure duration to more than one year, we follow the principles behind existing guidance developed for HRAs (OEHHA 2015; BAAQMD 2021). For residential receptors, current guidelines assume a window of exposure that is up to 30 years (OEHHA 2015; see also OEHHA 2012 chap. 11). Consistent with a focus on maximal risk, in cancer-risk HRAs this is taken to be the first 30 years of life.[6] 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 our baseline incidence rates (Table **1**), approximately half the population should survive to age 85. Taking this into account, when assessing the risk of mortality for a residential receptor we define the exposure window to be age 55–84. For off-site workers, current guidelines specify an exposure duration of no more than 25 years. Here we define the exposure window for a worker receptor to be age 40–64, consistent with an assumed retirement at 65.

For pediatric asthma onset, BenMAP-CE (US EPA 2022a) calculates impacts for the population aged 0–17, so we take this to be the corresponding exposure window when calculating the relevant risk increments for residential receptors. To represent children at a school, we follow existing HRA guidance, which for screening calculations specifies an age range of 5–13 (BAAQMD 2021). For a daycare receptor, we calculate risks for age 0–5.

By applying relative risks in a sequential fashion to each year within a defined window of exposure, and by comparing a less-exposed scenario to a more-exposed scenario, we arrive at overall results that summarize excess risk on an additive scale. Figure **1** illustrates this approach. The following two sections provide a series of worked examples, culminating in the results reported in Table **12**.

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

3. Example Calculations

This section illustrates the application of the concepts and methods described above. Example calculations are provided in stages. For simplicity, we refer to a hypothetical concentration increase of 0.1 µg/m^3 at all stages, but later provide a lookup table for larger and smaller increments.[7] After illustrating the fundamentals, in the next section ("Sensitive Individuals") we complete the method by accounting for children and adults who are more at risk.

In this section, we first calculate the risk of premature mortality for a residential receptor that is maximally exposed but has otherwise "statistically average" characteristics—breathing rate, health status, and so on—from age 55 to 84. Next, we shorten and shift the exposure window to match that of an adult of working age, and adjust the incremental average exposure intensity to reflect default assumptions about the co-presence of the worker and the source. Third, we introduce another health endpoint (pediatric asthma onset) and calculate relevant risks for residential, school, and daycare receptors.

Senior resident. As explained in Section 2, we define the exposure window for a senior resident to be age 55–84. To calculate an incremental average exposure intensity, we multiply our example concentration increment (0.1 µg/m^3) by factors that describe the overlap between the schedules of the source and receptor. Consistent with existing guidance (OEHHA 2015; BAAQMD 2021), for younger seniors (age 55–64), we assume that the fraction of time spent at home is 73%, 350 day/yr. For older seniors (age 65–84), we assume it is 100%, 365 day/yr. The overall conversion factor for younger seniors is then 0.70, and the resulting incremental average exposure intensities are 0.70 × 0.1 µg/m^3 = 0.07 µg/m^3 and 1.00 × 0.1 µg/m^3 = 0.10 µg/m^3 (Table **2**, column "Δx").

Consistent with the ranges reported in the Air District's recent evaluations of health impacts on regional populations (Fang et al. 2021a, 2021b), we take the relative risk of premature mortality to be 1.01 per 1 µg/m^3 $PM_{2.5}$. (For details and justification, see Section 7.) Applying the equations from Section 2, the relative risks of mortality corresponding to the incremental average exposure intensities calculated above are then $e^{\beta \cdot \Delta x} = e^{\ln(1.01) \cdot 0.070} \approx 1.00070$ and $e^{\ln(1.01) \cdot 0.100} \approx 1.00100$, respectively (Table **2**, column "Ratio").

Next we set up a comparison of two scenarios: one for baseline concentrations and rates, and another for baseline plus an increment of 0.1 µg/m^3 $PM_{2.5}$.[8] As described in Section 2, comparing the two scenarios allows us to assess the attributable risk. For baseline rates of mortality, we rely on data for the nine-county Bay Area obtained from the Centers for Disease Control and Prevention (Table **1**, CDC 2021). To obtain the age-specific annual mortality rates

[7] The Supplemental Material contains an interactive spreadsheet that implements these calculations.

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

under the baseline-plus-increment scenario, we simply multiply those baseline rates by the factors calculated in the preceding paragraph: 1.00070 for age 55–64, and 1.00100 for age 65–84. Table **2** shows the results (column "Increased", under "Incidence Rate").

The probability of surviving any given year is equal to one minus the risk of mortality during that year. The columns labeled "Survival" in Table **2** contain the cumulative products of these annual probabilities; they represent the overall probabilities of survival from age 55 until the end of the specified age. Given our assumptions, we calculate the difference at the end of the 30-year exposure window to be 54.3654% - 54.3329% = 0.0325% = 3.2×10^{-4}.

Off-site worker. As explained in Section 2, we define the exposure window for an off-site worker to be age 40–64. Consistent with existing HRA guidance (OEHHA 2015; BAAQMD 2021), default assumptions for an off-site worker receptor include a schedule of 8 hr/day, 5 day/wk, 250 day/yr. Also consistent with guidance, for screening-level calculations we assume that the source operates on the same daily and weekly schedule (8 hr/day, 5 day/wk), but for the entire year, rather than 250 day/yr. The overall conversion factor, from concentration to exposure intensity, is then 0.96. For our reference increment of +0.1 μg/m^3 in the modeled annual average concentration, this results in a mortality-risk score of 90.5208% - 90.5122% = 0.0086% = 8.6×10^{-5}. Calculations are shown in Table **3**.

Pediatric asthma onset. We calculate the risk of pediatric asthma onset in the same way. In this case, "survival" translates to remaining asthma-free. For baseline incidence rates, we rely on 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).

Consistent with existing HRA guidance (BAAQMD 2021), the relevant schedule at a K-8 school is assumed to be 10 hr/day, 5 day/wk, 180 day/yr, and the relevant exposure window is age 5–13. For a daycare receptor, we assume the same schedule, and set the exposure window to age 0–5. As with workers, for screening-level calculations we assume that the source has the same daily and weekly schedule as the receptor (in this case, 10 hr/day, 5 day/wk) but operates for the entire year, rather than 180 day/yr. The overall conversion factor, from concentration to exposure intensity, is then 0.69. For a daycare receptor, we calculate the increased risk corresponding to our reference increment of +0.1 μg/m^3 to be 87.8488% - 87.8141% = 3.5×10^{-4} (Table **5**). For a receptor at a K-8 school, it is 2.4×10^{-4} (Table **6**).

For a residential receptor, the fraction of time at home is assumed to be 100% for age 0–15 and 73% for age 16–17, consistent with existing guidance (BAAQMD 2021).[9] We calculate the corresponding risk of asthma onset to be 80.0128% - 79.9381% = 7.5×10^{-4} (Table **7**).

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

Lookup table. Table **8** summarizes the results that we obtain, following the steps above, for $PM_{2.5}$ increments spanning several orders of magnitude. Values from this table can be linearly interpolated to yield good approximations of exact calculations for intermediate values.

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

4. Sensitive Individuals

Up to this point, calculations have assumed a maximal annual average exposure, but apart from the selection of an exposure window, no consideration has yet been given to other factors relevant to a maximal risk. These other factors can be divided into two groups:

1. Factors on the pathway from exposure to dose (e.g., breathing rates); and
2. Factors that mediate dose-response relationships.

As the focus of this methodology is on maximal risks, potential variation in the factors above must be considered. We can complete the picture by accounting for variation in two ways. First, we can adjust the term Δx to reflect variations in factors on the pathway from exposure to dose. Within the range indicated by Table **8** (0.001–0.3 μg/m^3), we assume that incremental concentrations, exposures, and doses are linearly related. Therefore, any multiplicative factor intended to adjust any of these can simply be applied to Δx. Second, we can adjust the estimates of relative risk (as represented by the term β) to compensate for individuals who exhibit a larger or more severe dose-response relationship. We can also do this to account for data deficiencies.

Details are provided below, along with tables summarizing the specific adjustments that we apply. (See also the Technical Notes.) Instead of re-working the calculations of the preceding section step-by-step, we conclude by providing a final lookup table that reflects these considerations (Table **12**).

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

Table **10** shows the breathing rate data we use to adjust results for all receptors and endpoints. For daycare, school, and off-site worker receptors, we select point estimates of 95th percentile moderate-activity 8-hour rates; for residential receptors, we select 95th percentile daily rates. We then divide those rates by the mean daily rates for the corresponding ages, and use the resulting ratios (Table **11**) to scale the average exposure intensities (Δx) in our multi-year calculations.

Sensitive groups. To characterize variation in the relative risks of premature mortality among seniors, we have an empirical basis: important studies of $PM_{2.5}$ report effect sizes for sensitive groups—including seniors of color, seniors eligible for Medicaid, and seniors residing in low-income ZIP codes—that are two to three times the average (e.g., Di et al. 2017; Yazdi et al. 2021). Taking this into account, based on their expert judgment, the Advisory Council has recommended a factor of at least three to account for vulnerable seniors. For senior residents,

we therefore scale the population-average relative risk of premature mortality (1.01 per 1 μg/m^3) by a factor of 3, resulting in a relative risk of 1.03 per 1 μg/m^3.

Data deficiencies. There are gaps in the data concerning other endpoints and groups, where variations in impacts are not yet adequately quantified. To compensate for this, the Advisory Council has recommended a default factor of three to account for data deficiencies.[10] We therefore adopt this factor of three for data deficiencies concerning (a) pediatric asthma onset and (b) premature mortality among working-age adults, and use it to scale the population-average relative risks. The adjusted relative risks for those receptors and endpoints are then 1.134 and 1.03 per 1 μg/m^3, respectively.

Lookup table. Table **12** summarizes the corresponding results for $PM_{2.5}$ increments spanning several orders of magnitude. The next section discusses Table **12** in more detail.

[10] Multiples of 3 or 10 are conventional in risk assessment. See, e.g., NRC (2009), Table 6-2.

5. Discussion and Conclusion

The response functions that we leverage are derived from population-based studies in which a cohort of individuals is followed over a long period of time, and small contrasts in modeled or measured $PM_{2.5}$ concentrations are observed. Within a policy-relevant range of baseline $PM_{2.5}$ concentrations, from potentially 5 μg/m^3 to 15 μg/m^3 or higher, estimates of the average marginal impacts of the increments in Tables **8** and **12** will therefore be well supported. To account for situations where sensitive individuals may be more at risk, we make adjustments. Specifically, where a bottom-up methodology would select higher-than-average point estimates for key parameters to use directly in calculations, we either employ those higher values directly (as in the case of exposure duration), or adjust implicit components based on ratios of those higher values to typical values (as in the case of breathing rates). For the values themselves, we turn to existing HRA guidance where possible, and otherwise follow the underlying principles of that guidance (e.g., in the timing of the exposure window). Table **13** summarizes the components that we adjust.

As mentioned in the Introduction, while NAAQS-related programs are focused on regional attainment, the US EPA's air toxics program tends to focus on individuals who may be more at risk. Specifically, it "seeks to protect the greatest number of individuals from a lifetime cancer risk greater than 1×10^{-6} and in all cases limit risk to the individual most exposed to no greater than 1×10^{-4}" (Fann et al. 2016). Given an increment of 0.1 μg/m^3 $PM_{2.5}$, we calculate a maximal excess risk of mortality to be 1.5×10^{-3} for a residential receptor (Table **12**). For worker receptors, although breathing rates are higher (Table **11**), lower baseline mortality rates (Table **1**) mean that the net result is slightly lower (9.6×10^{-4}). In terms of pediatric asthma onset, we calculate an excess risk of 3.6×10^{-3} for a residential receptor. For a daycare receptor, the exposure window is shortened to ages 0–5, but higher breathing rates and baseline rates yield a larger net result (4.3×10^{-3}). In all cases, the values reported in Table **12** can be interpolated to yield screening-level estimates for other increments of $PM_{2.5}$ within the given range (see Technical Notes). We report values to two significant digits to support that interpolation.

In the case of larger sources, estimating impacts on a local population (Hubbell et al. 2009) can be a valuable complement to this methodology. Such an approach has been recommended by OEHHA as a complement to MEI-focused risk assessments (e.g., OEHHA 2012 chap. 11). Presently, the Air District models annual health and welfare impacts for the regional population using BenMAP-CE (US EPA 2022a; e.g., Tanrikulu et al. 2011, 2022), and has done so for sub-populations as small as 1 million residents (e.g., Fang et al. 2021a, 2021b). For a given increase in $PM_{2.5}$, the annual per-capita impact derived from such an assessment will be substantially smaller than the risk estimates that this methodology provides, both because it is calculated for a single year and because this methodology is oriented toward "worst case" potential risks.

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

6. Figures and Tables

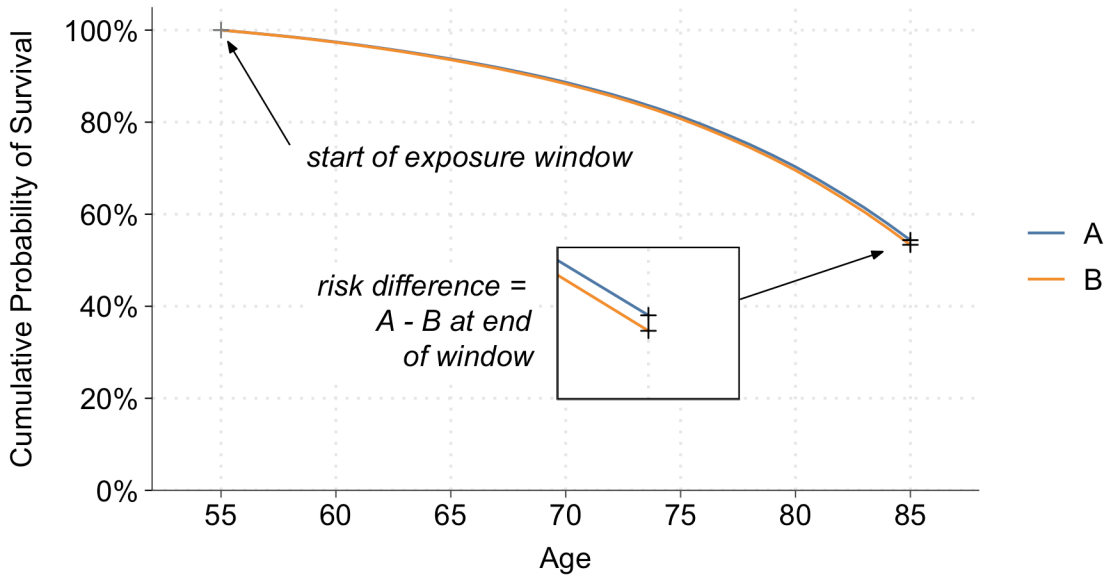

Figure 1: Illustration of the method applied to a multi-year exposure window. B is consistently exposed to more $PM_{2.5}$ than A. At the beginning of the exposure window, the receptor has not yet experienced the adverse event (e.g., mortality or asthma onset).

Table 1: Baseline mortality rates (per 100,000) for the nine-county Bay Area, 2007-2016 (CDC 2021).

Age	Rate
40	106.1
41	122.4
42	134.4
43	149.5
44	160.4
45	170.0
46	196.9
47	216.0
48	237.2
49	263.8
50	291.8
51	311.6
52	337.3
53	378.2
54	408.3
55	454.1
56	482.8
57	500.0
58	560.4
59	610.6
60	654.7
61	715.7
62	756.6
63	831.6
64	882.1
65	950.7
66	995.9
67	1,108.9
68	1,180.4
69	1,303.5
70	1,443.8
71	1,521.2
72	1,719.6
73	1,882.1
74	2,075.3
75	2,322.5
76	2,581.2
77	2,781.1
78	3,132.9
79	3,462.7
80	3,976.8
81	4,420.7
82	4,829.6
83	5,556.5
84	6,241.9

Table 2: Mortality rates and cumulative probabilities of survival for a statistically average residential receptor, age 55–84, given a relative risk of 1.01 per 1 μg/m^3 and an annual average incremental concentration of +0.1 μg/m^3.

		Incidence Rate (per 100,000)			Survival (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
55	0.070	454.07	1.00070	454.39	99.5459%	99.5456%
56	0.070	482.85	1.00070	483.19	99.0653%	99.0646%
57	0.070	500.01	1.00070	500.36	98.5699%	98.5689%
58	0.070	560.45	1.00070	560.84	98.0175%	98.0161%
59	0.070	610.56	1.00070	610.99	97.4190%	97.4173%
60	0.070	654.68	1.00070	655.14	96.7813%	96.7790%
61	0.070	715.71	1.00070	716.21	96.0886%	96.0859%
62	0.070	756.55	1.00070	757.08	95.3616%	95.3585%
63	0.070	831.57	1.00070	832.15	94.5686%	94.5649%
64	0.070	882.14	1.00070	882.75	93.7344%	93.7302%
65	0.100	950.72	1.00100	951.66	92.8432%	92.8382%
66	0.100	995.94	1.00100	996.93	91.9186%	91.9126%
67	0.100	1,108.88	1.00100	1,109.98	90.8993%	90.8924%
68	0.100	1,180.36	1.00100	1,181.54	89.8264%	89.8185%
69	0.100	1,303.55	1.00100	1,304.85	88.6555%	88.6465%
70	0.100	1,443.77	1.00100	1,445.21	87.3755%	87.3654%
71	0.100	1,521.17	1.00100	1,522.68	86.0463%	86.0351%
72	0.100	1,719.59	1.00100	1,721.31	84.5667%	84.5541%
73	0.100	1,882.08	1.00100	1,883.95	82.9751%	82.9612%
74	0.100	2,075.25	1.00100	2,077.32	81.2531%	81.2378%
75	0.100	2,322.46	1.00100	2,324.77	79.3661%	79.3492%
76	0.100	2,581.23	1.00100	2,583.80	77.3175%	77.2990%
77	0.100	2,781.05	1.00100	2,783.82	75.1672%	75.1471%
78	0.100	3,132.95	1.00100	3,136.07	72.8123%	72.7905%
79	0.100	3,462.71	1.00100	3,466.16	70.2910%	70.2674%
80	0.100	3,976.83	1.00100	3,980.79	67.4956%	67.4702%
81	0.100	4,420.68	1.00100	4,425.08	64.5119%	64.4846%
82	0.100	4,829.58	1.00100	4,834.39	61.3962%	61.3672%
83	0.100	5,556.48	1.00100	5,562.02	57.9847%	57.9539%
84	0.100	6,241.94	1.00100	6,248.15	**54.3654%**	**54.3329%**

Δx = Incremental annual average exposure intensity (ug/m3).

Baseline = Scenario representing baseline PM2.5 level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp[ln(RR)·Δx]. (Baseline Rate) x (Ratio) = (Increased Rate).

Table 3: Mortality rates and cumulative probabilities of survival for a statistically average off-site worker receptor, age 40–64, given a relative risk of 1.01 per 1 μg/m^3 and an annual average incremental concentration of +0.1 μg/m^3.

		Incidence Rate (per 100,000)			Survival (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
40	0.096	106.10	1.00095	106.20	99.8939%	99.8938%
41	0.096	122.45	1.00095	122.56	99.7716%	99.7714%
42	0.096	134.42	1.00095	134.54	99.6375%	99.6371%
43	0.096	149.50	1.00095	149.65	99.4885%	99.4880%
44	0.096	160.38	1.00095	160.53	99.3290%	99.3283%
45	0.096	169.97	1.00095	170.13	99.1601%	99.1593%
46	0.096	196.85	1.00095	197.04	98.9649%	98.9639%
47	0.096	215.95	1.00095	216.16	98.7512%	98.7500%
48	0.096	237.18	1.00095	237.41	98.5170%	98.5156%
49	0.096	263.80	1.00095	264.05	98.2571%	98.2555%
50	0.096	291.81	1.00095	292.09	97.9704%	97.9685%
51	0.096	311.65	1.00095	311.94	97.6651%	97.6629%
52	0.096	337.25	1.00095	337.57	97.3357%	97.3332%
53	0.096	378.24	1.00095	378.60	96.9675%	96.9647%
54	0.096	408.32	1.00095	408.71	96.5716%	96.5684%
55	0.096	454.07	1.00095	454.51	96.1331%	96.1295%
56	0.096	482.85	1.00095	483.31	95.6689%	95.6649%
57	0.096	500.01	1.00095	500.49	95.1905%	95.1861%
58	0.096	560.45	1.00095	560.98	94.6571%	94.6521%
59	0.096	610.56	1.00095	611.14	94.0791%	94.0736%
60	0.096	654.68	1.00095	655.31	93.4632%	93.4572%
61	0.096	715.71	1.00095	716.39	92.7943%	92.7876%
62	0.096	756.55	1.00095	757.28	92.0922%	92.0850%
63	0.096	831.57	1.00095	832.36	91.3264%	91.3185%
64	0.096	882.14	1.00095	882.98	**90.5208%**	**90.5122%**

Δx = Incremental annual average exposure intensity (ug/m3).

Baseline = Scenario representing baseline PM2.5 level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp[ln(RR)·Δx]. (Baseline Rate) x (Ratio) = (Increased Rate).

Table 4: 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 5: Incidence rates and cumulative probabilities of remaining asthma-free for a statistically average daycare receptor, age 0–5, given a relative risk of 1.045 per 1 µg/m^3 and an annual average incremental concentration of +0.1 µg/m^3.

		Incidence Rate (per 1,000)			Asthma-Free (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
0	0.069	23.4	1.00302	23.4707	97.6600%	97.6529%
1	0.069	23.4	1.00302	23.4707	95.3748%	95.3610%
2	0.069	23.4	1.00302	23.4707	93.1430%	93.1228%
3	0.069	23.4	1.00302	23.4707	90.9634%	90.9371%
4	0.069	23.4	1.00302	23.4707	88.8349%	88.8028%
5	0.069	11.1	1.00302	11.1335	**87.8488%**	**87.8141%**

Δx = Incremental annual average exposure intensity (ug/m3).

Baseline = Scenario representing baseline PM2.5 level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp[ln(RR)·Δx]. (Baseline Rate) x (Ratio) = (Increased Rate).

Table 6: Incidence rates and cumulative probabilities of remaining asthma-free for a statistically average student receptor, age 5–13, given a relative risk of 1.045 per 1 µg/m^3 and an annual average incremental concentration of +0.1 µg/m^3.

		Incidence Rate (per 1,000)			Asthma-Free (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
5	0.069	11.1	1.00302	11.1335	98.8900%	98.8866%
6	0.069	11.1	1.00302	11.1335	97.7923%	97.7857%
7	0.069	11.1	1.00302	11.1335	96.7068%	96.6970%
8	0.069	11.1	1.00302	11.1335	95.6334%	95.6204%
9	0.069	11.1	1.00302	11.1335	94.5718%	94.5558%
10	0.069	11.1	1.00302	11.1335	93.5221%	93.5031%
11	0.069	11.1	1.00302	11.1335	92.4840%	92.4621%
12	0.069	4.4	1.00302	4.4133	92.0771%	92.0540%
13	0.069	4.4	1.00302	4.4133	**91.6719%**	**91.6477%**

Δx = Incremental annual average exposure intensity (ug/m3).

Baseline = Scenario representing baseline PM2.5 level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp[ln(RR)·Δx]. (Baseline Rate) x (Ratio) = (Increased Rate).

Table 7: Incidence rates and cumulative probabilities of remaining asthma-free for a statistically average residential receptor, age 0–17, given a relative risk of 1.045 per 1 μg/m^3 and an annual average incremental concentration of +0.1 μg/m^3.

		Incidence Rate (per 1,000)			Asthma-Free (Cumulative)	
Age	**Δx**	**Baseline**	**Ratio**	**Increased**	**Baseline**	**Increased**
0	0.096	23.4	1.00420	23.4982	97.6600%	97.6502%
1	0.096	23.4	1.00420	23.4982	95.3748%	95.3556%
2	0.096	23.4	1.00420	23.4982	93.1430%	93.1149%
3	0.096	23.4	1.00420	23.4982	90.9634%	90.9269%
4	0.096	23.4	1.00420	23.4982	88.8349%	88.7902%
5	0.096	11.1	1.00420	11.1466	87.8488%	87.8005%
6	0.096	11.1	1.00420	11.1466	86.8737%	86.8219%
7	0.096	11.1	1.00420	11.1466	85.9094%	85.8541%
8	0.096	11.1	1.00420	11.1466	84.9558%	84.8971%
9	0.096	11.1	1.00420	11.1466	84.0128%	83.9508%
10	0.096	11.1	1.00420	11.1466	83.0803%	83.0150%
11	0.096	11.1	1.00420	11.1466	82.1581%	82.0897%
12	0.096	4.4	1.00420	4.4185	81.7966%	81.7270%
13	0.096	4.4	1.00420	4.4185	81.4367%	81.3659%
14	0.096	4.4	1.00420	4.4185	81.0783%	81.0064%
15	0.096	4.4	1.00420	4.4185	80.7216%	80.6484%
16	0.070	4.4	1.00306	4.4135	80.3664%	80.2925%
17	0.070	4.4	1.00306	4.4135	**80.0128%**	**79.9381%**

Δx = Incremental annual average exposure intensity (ug/m3).

Baseline = Scenario representing baseline PM2.5 level.

Increased = Scenario representing baseline + modeled increment.

Ratio = exp[ln(RR)·Δx]. (Baseline Rate) x (Ratio) = (Increased Rate).

Table 8: Screening-level risk scores for "statistically average" receptors. Exposure windows are indicated in parentheses. Baseline incidence rates vary with age, but other potential variations in sensitivity have not yet been accounted for. (See Table **12** for the final results).

	Pediatric Asthma Onset			Premature Mortality	
Annual Average Concentration Increment	**Daycare (0–5)**	**Student (5–13)**	**Resident (0–17)**	**Worker (40–64)**	**Resident (55–84)**
3×10^{-1} µg/m³	1.0×10^{-3}	7.3×10^{-4}	2.2×10^{-3}	2.6×10^{-4}	9.7×10^{-4}
1×10^{-1} µg/m³	3.5×10^{-4}	2.4×10^{-4}	7.5×10^{-4}	8.6×10^{-5}	3.2×10^{-4}
3×10^{-2} µg/m³	1.0×10^{-4}	7.3×10^{-5}	2.2×10^{-4}	2.6×10^{-5}	9.7×10^{-5}
1×10^{-2} µg/m³	3.5×10^{-5}	2.4×10^{-5}	7.5×10^{-5}	8.6×10^{-6}	3.2×10^{-5}
3×10^{-3} µg/m³	1.0×10^{-5}	7.2×10^{-6}	2.2×10^{-5}	2.6×10^{-6}	9.7×10^{-6}
1×10^{-3} µg/m³	3.5×10^{-6}	2.4×10^{-6}	7.5×10^{-6}	8.6×10^{-7}	3.2×10^{-6}

Consistent with screening-level HRA guidance from BAAQMD (2021), for a residential receptor the assumed fraction of time at home (FAH) is 100% for age 0–15, and 73% for age 16–64, 350 day/yr. For a resident aged 65–84, it is 100%, 365 day/yr. Schedule parameters for an off-site worker receptor are 8 hr/day, 250 day/yr, with an adjustment factor of 4.2 applied to account for potential overlap in the schedules of the source and receptor when modeling is used to calculate an annual average concentration increment. For a school or daycare receptor, schedule parameters are 10 hr/day, 180 day/yr, with an adjustment factor of 3.36. For details and explanation, see OEHHA (2015).

The population-average relative risk for premature adult mortality is taken to be 1.01 per 1 ug/m3. For pediatric asthma onset, it is 1.045 per 1 ug/m3.

Baseline incidence rates for mortality are obtained from CDC-WONDER for the 9-county Bay Area, 2007–2016 (CDC 2021), while those for pediatric asthma onset are obtained from BenMAP (US EPA 2022; Winer et al 2012).

Table 9: Factors applied to account for variations in individual response. See also Tables **10**, **11**, and **13**.

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

Table 10: Breathing rates (L/kg-day) by level of activity, summary statistic, and age. Values obtained from Tables 5.7 and 5.8 of OEHHA (2015).

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

* Original data are for ages 16–70.

Table 11: Factors applied to account for variation in breathing rates. Values derived from Table **10**, as described in the main text (Section 4).

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

Values rounded to one decimal.

Table 12: Screening-level risk scores that incorporate potential variations in sensitivity. Exposure windows are indicated in parentheses.

	Pediatric Asthma Onset			Premature Mortality	
Annual Average Concentration Increment	**Daycare (0–5)**	**Student (5–13)**	**Resident (0–17)**	**Worker (40–64)**	**Resident (55–84)**
3×10^{-1} µg/m³	1.3×10^{-2}	7.5×10^{-3}	1.1×10^{-2}	2.9×10^{-3}	4.5×10^{-3}
1×10^{-1} µg/m³	4.3×10^{-3}	2.4×10^{-3}	3.6×10^{-3}	9.6×10^{-4}	1.5×10^{-3}
3×10^{-2} µg/m³	1.3×10^{-3}	7.2×10^{-4}	1.1×10^{-3}	2.9×10^{-4}	4.5×10^{-4}
1×10^{-2} µg/m³	4.2×10^{-4}	2.4×10^{-4}	3.5×10^{-4}	9.6×10^{-5}	1.5×10^{-4}
3×10^{-3} µg/m³	1.3×10^{-4}	7.2×10^{-5}	1.1×10^{-4}	2.9×10^{-5}	4.5×10^{-5}
1×10^{-3} µg/m³	4.2×10^{-5}	2.4×10^{-5}	3.5×10^{-5}	9.6×10^{-6}	1.5×10^{-5}

Consistent with screening-level HRA guidance from BAAQMD (2021), for a residential receptor the assumed fraction of time at home (FAH) is 100% for age 0–15, and 73% for age 16–64, 350 day/yr. For a resident aged 65–84, it is 100%, 365 day/yr. Schedule parameters for an off-site worker receptor are 8 hr/day, 250 day/yr, with an adjustment factor of 4.2 applied to account for potential overlap in the schedules of the source and receptor when modeling is used to calculate an annual average concentration increment. For a school or daycare receptor, schedule parameters are 10 hr/day, 180 day/yr, with an adjustment factor of 3.36. For details and explanation, see OEHHA (2015).

PM2.5 increments are adjusted using age-specific 95th percentile breathing rates from OEHHA (2015). Moderate-activity 8-hr rates are used for worker, student, and daycare receptors; daily rates are used for residential receptors. To account for variations in effect size, factors of 3 are applied to population-average relative risks (RR) per 1 ug/m3 for premature adult mortality and pediatric asthma onset, resulting in RR = 1.03 and 1.134 per 1 ug/m3, respectively.

Baseline incidence rates for mortality are obtained from CDC-WONDER for the 9-county Bay Area, 2007–2016 (CDC 2021), while those for pediatric asthma onset are obtained from BenMAP (US EPA 2022; Winer et al 2012).

Table 13: Protective approaches applied to key dimensions of the methodology.

Component	Protective Aspect(s)
Baseline risk	The selected exposure windows are associated with higher-than-average baseline rates.
Concentration	For each class of receptor (resident, worker, etc.), the maximally impacted potential location is selected.
Exposure intensity	For workers and children, near-100% overlaps in intra-week schedules (source vs receptor) are assumed. Seniors aged 65+ are assumed to reside at home 100% of the time.
Exposure duration	For residential receptors, the length of the exposure window (30 years) is based on the 90th percentile of residency times.
Dose	For breathing rates, 95th percentiles are used. For workers and children, moderate exertion levels are assumed.
Effect size	The starting points are central estimates of population-average effect size. These are scaled by factors of 3 to account for individual variation.

7. Frequently Asked Questions

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

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

Yes. Relatively small changes in $PM_{2.5}$ at or around baseline levels are the subject of epidemiologic studies on which this methodology is based. Sensitive individuals will be more at risk, given the same increase in exposure. 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. Those expected impacts across the general population are useful benchmarks, are statistically significant, and are supported by multiple scientific literatures (US EPA 2019, 2022b). This methodology goes a step further by taking a health-protective approach to estimating several important components of risk (Table **13**). The resulting estimates of potential risk, for situations where a receptor may be especially at risk, will be substantially higher than the per-capita impacts associated with population-wide assessments.

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

For premature adult mortality, the value we selected (1.01 per 1 $\mu g/m^3$) is consistent with the ranges reported in the District's recent evaluations of impacts on regional populations (Fang et al. 2021a, 2021b; Tanrikulu et al. 2022). It is also consistent with the estimates reported by Di et al (2017): 1.073 overall per 10 $\mu g/m^3$, and 1.136 per 10 $\mu g/m^3$ for exposures less than 12 $\mu g/m^3$. These are equivalent to RR = 1.0071 and 1.0128 per 1 $\mu g/m^3$, respectively.[11] Di et al (2017) is the core study on which the US EPA relies for estimates of attributable mortality among seniors (US EPA 2022a). Yazdi et al (2021) arrive at similar results using different methods, again studying baseline levels under 12 $\mu g/m^3$. Vodonos et al (2018), summarizing a wide range of studies across all ages via meta-regression, arrive at a relative risk of 1.013 per 1 $\mu g/m^3$ for a baseline centered on 10 $\mu g/m^3$. Summarizing other recent studies via a random-effects model, Di et al (2017 fig. S6) arrive at a pooled result of 1.110 per 10 $\mu g/m^3$, which is equivalent to 1.010 per 1 $\mu g/m^3$.

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

For pediatric asthma calculations, we take the relative risk supplied by the US EPA's BenMAP-CE platform (US EPA 2022a), which is 1.33 per 6.53 $\mu g/m^3$, and convert it to 1.045 per 1 $\mu g/m^3$. The

[11] Regarding the conversion formula, please see the Technical Notes.

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

mean $PM_{2.5}$ concentration in the supporting study was approximately 10 μg/m^3 (Tétreault et al. 2016, Table 2).

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

During earlier development, this methodology was restricted to premature adult mortality. In a conventional population-wide assessment, mortality typically receives over 90% of the overall valuation. However, feedback from stakeholders indicated that it was critical to assess at least one other endpoint. Respiratory effects, and asthma in particular, figure prominently in the concerns of community members and community representatives. Asthma can be measured in many ways: hospitalizations; inhaler use; progression; and new onset, to name a few. Asthma onset (newly developed or diagnosed asthma) was selected because it receives the highest valuation in the District's current population-based assessments, and because it is a necessary condition for other metrics, such as hospitalizations.

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

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

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

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

We sometimes have geographically resolved information on important predictors of the baseline rate and/or the effect. For example, studies report (varying) results for individual race/ethnicity as a predictor or modifier of the effect size. They also report comparable results for other factors, such as income and Medicaid status. The selections of variables, and the adjustments for other variables—many of which are correlated—are often inconsistent across studies. Integrating results across such studies into a single, coherent adjustment factor for the effect size (β) would be a major challenge, which we do not currently know how to solve. Acknowledging that new scientific understandings will inevitably emerge, the factors in Table **9** are intended to be adequately protective of sensitive individuals across multiple dimensions.

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

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

8. Technical Notes

The reader who is more familiar with cancer-risk calculations may note two distinct features of the delta-response equation that is central to this methodology. First, the relationship between Δy and $\beta\,\Delta x$ is nonlinear. Second, it includes a term representing baseline conditions (y_0). These features have a few practical consequences.

Additivity and symmetry. First, risk scores will not accumulate exactly in the way that they do in a linear framework. The calculated risk for an increase of 0.1 µg/m^3 will in fact be slightly more than ten times that for an increase of 0.01 µg/m^3. (This can be observed in Table **12**.) The discrepancy varies with the magnitude of the term $\beta\,\Delta x$. As our values for β and Δx are small, it will be a few percent at most. Second, calculations based on linear frameworks are symmetric, in the sense that the magnitude of Δy does not depend on the sign of Δx. Here, however, if Δx is intended to represent a reduction, then it should be assigned a negative value. Otherwise, the magnitude of Δy will be slightly too large.

Standardizing and scaling. In the literature, relative risk (RR) is sometimes expressed with respect to an increment other than 1 µg/m^3. Thus, for example, Tétreault et al (2016) report RR = 1.33 per 6.53 µg/m^3. To standardize a published relative risk RR_u from "per u µg/m^3" to a relative risk RR_1 "per 1 µg/m^3", we use the formula:

$$RR_1 = (RR_u)^{(1/u)}$$

In the main text, we have standardized to "per 1 µg/m^3" throughout. This simplifies calculations and reduces the likelihood of mistakes: in this case, $\Delta x = \frac{1}{u}\Delta\mathrm{PM}_{2.5}$ simplifies to $\Delta x = \Delta\mathrm{PM}_{2.5}$. It also means that:

$$y = y_0\, e^{\beta\,\Delta x} = y_0\, e^{\ln(RR_1)\,\Delta x} = y_0\, e^{\ln\left[(RR_1)^{\Delta x}\right]} = y_0\,(RR_1)^{\Delta x}$$

Recalling the asthma example from Section 2, when Δx was 1 µg/m^3, to obtain the new risk y we multiplied the baseline risk y_0 by RR_1 once. For Δx = 2 µg/m^3, we would do so twice.

Scaling a relative risk by a multiplicative factor k (such as $k = 3$) is also straightforward: the result is equal to $k(RR - 1) + 1$. However, the order of operations matters:

$$k\left[(RR_u)^{(1/u)} - 1\right] + 1 \neq \left[k(RR_u - 1) + 1\right]^{(1/u)} \quad \text{unless } k = 1, u = 1, \text{ or } RR_u = 1$$

This means that if additional endpoints were to be added, if relative risk estimates were to be updated, or if a larger value of k were to be adopted for some endpoint, the same approach (standardize, then scale) should be followed consistently.

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