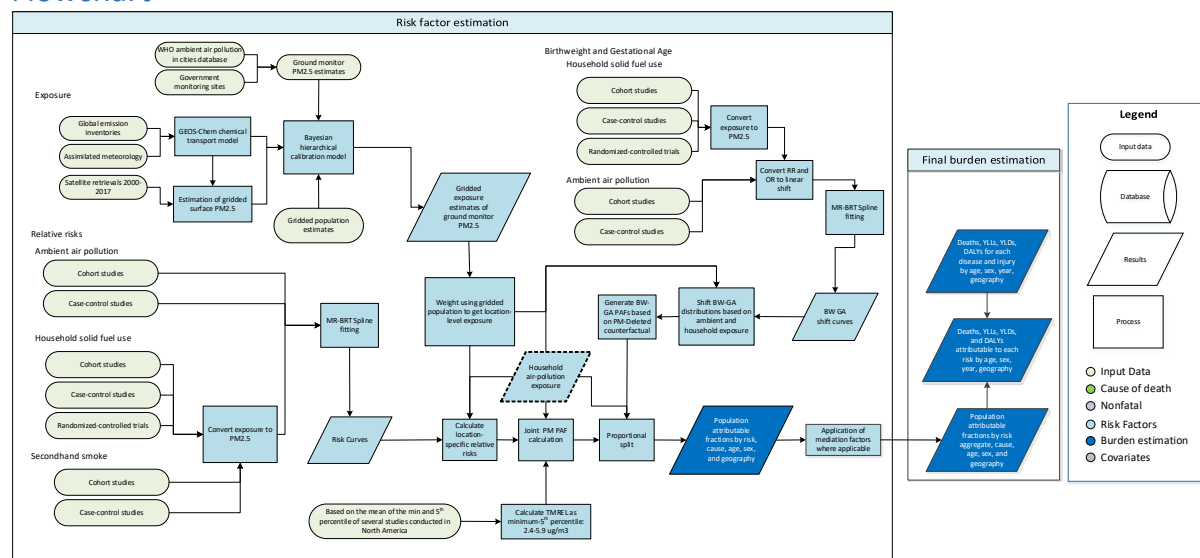


Ambient particulate matter pollution

Flowchart



Input data and methodological summary

Exposure

Definition

Exposure to ambient particulate matter pollution is defined as the population-weighted annual average mass concentration of particles with an aerodynamic diameter less than 2.5 micrometers ($PM_{2.5}$) in a cubic meter of air. This measurement is reported in $\mu g/m^3$.

Input data

Ambient air pollution exposure estimates use input data from multiple sources. These include satellite observations of aerosols in the atmosphere, ground monitor measurements, chemical transport model simulations, population estimates, and land-use data.

Table 1: Data inputs for exposure for ambient particulate matter pollution

Input data	Exposure
Site-years (total)	5442
Number of countries with data	204
Number of GBD regions with data (out of 21 regions)	21
Number of GBD super-regions with data (out of 7 super-regions)	7

Details for updates in exposure methodology and input data for the Global Burden of Disease (GBD) Study 2020 are as follows.

$PM_{2.5}$ ground measurement database

For GBD 2020, ground monitor measurements were updated to include more recent measurements from sites included in GBD 2019 and additional measurements from new monitors. New data were added to the database from several sources, including the European Environment Agency, United

States Environment Protection Agency, and the OpenAQ database. The complete, updated dataset included measurements of PM₁₀ and PM_{2.5} concentrations between 2018 and 2020 from 18,406 ground monitors from 120 countries, primarily from the USA, China, European countries, and USA embassies and consulates. Annual averages were excluded if they were based on less than 75% coverage within a year. If information on coverage was not available, data were included unless there was already sufficient data within the country of interest (monitor density greater than 0.1).

For sites with PM₁₀ measurements only, these observations were converted from PM₁₀ to PM_{2.5} measurements using a hierarchy of conversion factors (PM_{2.5}/PM₁₀ ratios): (i) where possible, a “local” conversion factor was used, constructed as the ratio of the average measurements (of PM_{2.5} and PM₁₀) from within 50 km of the location of the PM₁₀ measurement, and within the same country, if such measurements were available; (ii) where local information was not sufficient to construct a conversion factor, a country-wide conversion factor was used; and (iii) where appropriate information within a country did not exist, a region-level factor was used. In each case, to avoid the possible effects of outliers in the measured PM_{2.5} and PM₁₀ data, extreme values of the ratios were excluded. These extreme values were defined as those greater/lesser than the 95th and 5th quantiles of the empirical distributions of conversion factors. As with the GBD 2013, 2015, 2016, 2017, and 2019 databases, in addition to values of PM_{2.5} and whether they were direct measurements or conversions from PM₁₀, the GBD 2020 database also included additional information (where available) concerning the ground measurements, such as monitor geo-coordinates and monitor site type.

Satellite-based estimates

Global satellite-derived estimates (V4.GL.03.NoGWR) used as inputs to DIMAQ2 for 1998–2019 and for January to August 2020 are used at 0.1° x 0.1° resolution (~11 x 11 km resolution at the equator) and follow the methodology described in Hammer et al., 2020.¹ The algorithm uses aerosol optical depth (AOD) from several updated satellite products (MAIAC, MODIS, and MISR). Ground-based observations from a global sunphotometer network (AERONET version 3) are used to combine different AOD information sources. The GEOS-Chem chemical transport model was used for geophysical relationships between surface PM_{2.5} and AOD. For GBD 2020, an additional update to biomass burning emissions from 2015 to 2020 was made. This update allows for time-varying biomass burning emissions in the simulation for those years, where they had previously been unavailable after 2014. Given lags in releases of available meteorological information used in the GEOS Chem simulations, for September to December 2020, the estimates incorporate satellite retrievals from 2020, but GEOS-Chem simulated values for 2019 as well as biomass burning emissions from 2019. Further, satellite retrievals for all of 2020 were limited to MODIS DT, DB, and MAIAC. We included MISR inputs for January to June 2020 only, as this product was not available past June when the satellite-based estimates were generated.

Chemical transport model simulations

Estimates of the sum of particulate sulfate, nitrate, ammonium, and organic carbon and the compositional concentrations of mineral dust simulated using the GEOS-Chem chemical transport model, and a measure combining elevation and the distance to the nearest urban land surface (as described in van Donkelaar et al. 2016² and Hammer et al. 2020)¹ were available for 2000–2020 for each 0.1° x 0.1° grid cell.

Population data

We obtained a comprehensive, high-resolution gridded population dataset from the Gridded Population of the World (GPW) database. Estimates for 2000, 2005, 2010, 2015, and 2020 were available from the GPW version 4, with estimates for 1990 and 1995 obtained from the GPW version 3. These data are provided on a $0.0083^\circ \times 0.0083^\circ$ resolution. Aggregation to each $0.1^\circ \times 0.1^\circ$ grid cell was accomplished by summing the central 12×12 population cells. Populations estimates for 2001–2004, 2006–2009, 2011–2014 and 2016–2019 were obtained by interpolation using natural splines with knots placed at 2000, 2005, 2010, 2015, and 2020. This was performed for each grid cell.

Modelling strategy

The following is a summary of the modelling approach, known as the Data Integration Model for Air Quality (DIMAQ) used in GBD 2015, 2016, 2017, 2019, and 2020.^{3,4}

Before the implementation of DIMAQ in GBD 2010 and 2013, exposure estimates were obtained using a single global function to calibrate available ground measurements to a “fused” estimate of $PM_{2.5}$: the mean of satellite-based estimates and those from the TM5 chemical transport model, calculated for each $0.1^\circ \times 0.1^\circ$ grid cell. This approach was recognised to represent a trade-off between accuracy and computational efficiency when utilising all the available data sources. In particular, the GBD 2013 exposure estimates were known to underestimate ground measurements in specific locations (see discussion in Brauer et al., 2015).⁵ This underestimation was largely due to the use of a single, global calibration function, whereas in reality, the relationship between ground measurements and other variables varies spatially.

In GBD 2015 and 2016, coefficients in the calibration model were estimated for each country through DIMAQ. Where data were insufficient within a country, information was “borrowed” from a region-level aggregation, and where information was still insufficient, from the super-region-level aggregation. Individual country-level estimates were therefore based on a combination of information from the country and its region and super-region. This was implemented within a Bayesian hierarchical modelling (BHM) framework. BHMs provide an extremely useful and flexible framework in which to model complex relationships and dependencies in data. Uncertainty can also be propagated through the model, allowing uncertainty arising from different components (both data sources and models) to be incorporated within estimates of uncertainty associated with the final estimates. The results of the modelling comprise a posterior distribution for each grid cell, rather than just a single point estimate, allowing a variety of summaries to be calculated. The primary outputs for this process are the median and 95% uncertainty intervals for each grid cell. Based on the availability of ground measurement data, modelling and evaluation were focused on the year 2016.

The model used from GBD 2017 onward (GBD 2017, 2019, and now 2020) also included within country calibration variation.⁶ This model, henceforth referred to as DIMAQ2, provides a number of substantial improvements over the initial formulation of DIMAQ. In DIMAQ, ground measurements from different years were all assumed to have been made in the primary year of interest and then regressed against values from other inputs (satellites, etc.) made in that year. In the presence of changes over time, therefore, and particularly in areas where no recent measurements were available, there was the possibility of mismatches between the ground measurements and other variables. In DIMAQ2, ground measurements are matched with other inputs (over time), and the (global-level) coefficients are allowed to vary over time, subject to smoothing that is induced by a first-order random walk process. In addition, the manner in which spatial variation can be incorporated within the model has developed: where there are sufficient data, the calibration equations can now vary (smoothly) both within and between countries, achieved by allowing the

coefficients to follow (smooth) Gaussian processes. Where there are insufficient data within a country, to produce accurate equations, information is borrowed as before from lower down the hierarchy and is supplemented with information from the wider region.

DIMAQ2 as described above was used for all regions except for the north Africa/Middle East and sub-Saharan Africa super-regions, where there are insufficient data across years to allow the extra complexities of the new model to be implemented. In these super-regions, a simplified version of DIMAQ2 is used in which the temporal component is dropped.

Inference and prediction

Continuous explanatory variables:

- (SAT) Estimate of $PM_{2.5}$ (in $\mu g/m^3$) from satellite remote sensing on the log-scale.
- (POP) Estimate of population for the same year as SAT on the log-scale.
- (SANOC) Estimate of the sum of sulfate, nitrate, ammonium, and organic carbon simulated using the GEOS-Chem chemical transport model.
- (DST) Estimate of compositional concentrations of mineral dust simulated using the GEOS-Chem chemical transport model.
- (EDxDU) The log of the elevation difference between the elevation at the ground measurement location and the mean elevation within the GEOS-Chem simulation grid cell multiplied by the inverse distance to the nearest urban land surface.

Discrete explanatory variables:

- (LOC) Binary variable indicating whether exact location of ground measurement is known.
- (TYPE) Binary variable indicating whether exact type of ground monitor is known.
- (CONV) Binary variable indicating whether ground measurement is $PM_{2.5}$ or converted from PM_{10} .

Interactions:

- Interactions between the binary variables and the effects of SAT.

Random effects:

- Regional temporal (random walk) hierarchical random-effects on the intercept
- Regional hierarchical random-effects for the coefficient associated with SAT
- Regional hierarchical random-effects for the coefficient associated with POP
- Smoothed, spatially varying, random-effects for the intercept
- Smoothed, spatially varying, random-effects for the coefficient associated with SAT

Due to both the complexity of the models and the size of the data, notably the number of spatial predictions that are required, recently developed techniques that perform “approximate” Bayesian inference based on integrated nested Laplace approximations (INLA) were used.⁷ Computation was performed using the R interface to the INLA computational engine (R-INLA). For GBD 2019 and GBD 2020, the model also implements an innovative way to use samples from the (Bayesian) model to represent distributions of estimated concentrations in each grid cell. Estimates, and distributions representing uncertainty, of concentrations for each grid cell are obtained by taking repeated (joint) samples from the posterior distributions of the parameters and calculating estimates based on a linear combination of those samples and the input variables.⁸

DIMAQ2 was used to produce grid-cell ($0.1^\circ \times 0.1^\circ$) level estimates of ambient $PM_{2.5}$ for 1990, 1995, and 2010–2020 by matching the gridded estimates with the corresponding coefficients from the

calibration. For the year 2020, additional analysis was conducted to incorporate updated ground monitor (1777 observations for 2020) and satellite-based data (as described above) to examine potential impacts of the COVID-19 pandemic on ambient particulate matter pollution.

Model evaluation

Model development and comparison was performed using within- and out-of-sample assessment. For evaluation, cross-validation was performed using 25 combinations of training (80%) and validation (20%) datasets. Validation sets were obtained by taking a stratified random sample, using sampling probabilities based on the cross-tabulation of PM_{2.5} categories (0–24.9, 25–49.9, 50–74.9, 75–99.9, 100+ $\mu\text{g}/\text{m}^3$) and super-regions, resulting in sets with the same distribution of PM_{2.5} concentrations and super-regions as the overall set of sites. The following metrics were calculated for each training/validation set combination: for model fit— R^2 ; for predictive accuracy—root mean squared error (RMSE) and population-weighted root mean squared error (PwRMSE).

Evaluation of model results for GBD 2020 were comparable to those from GBD 2013 and GBD 2017 (the most recent model evaluation prior to GBD 2020). For GBD 2020, DIMAQ2 predictions of ground measurements in all super-regions produced a mean out of sample population-weighted RMSE of 8.50 (95% UI 6.17–12.77) $\mu\text{g}/\text{m}^3$ and an R^2 of 0.909 (0.886–0.926). The high-income super-region produced the most accurate predictions, with a mean population-weighted RMSE of 2.16 (2.09–2.23) $\mu\text{g}/\text{m}^3$, while south Asia produced the largest population-weighted mean RMSE, 31.56 (18.95–51.88) $\mu\text{g}/\text{m}^3$. Trends in relative magnitude of PwRMSE are consistent with previous DIMAQ evaluations in GBD 2017 and 2019.

Figure 1: Summary measure of predictive ability, globally and by super-region. Points denote median values of out-of-sample population-weighted root mean square error ($\mu\text{g}/\text{m}^3$) from 25 validation sets. Vertical lines denote 95% uncertainty interval bounds.

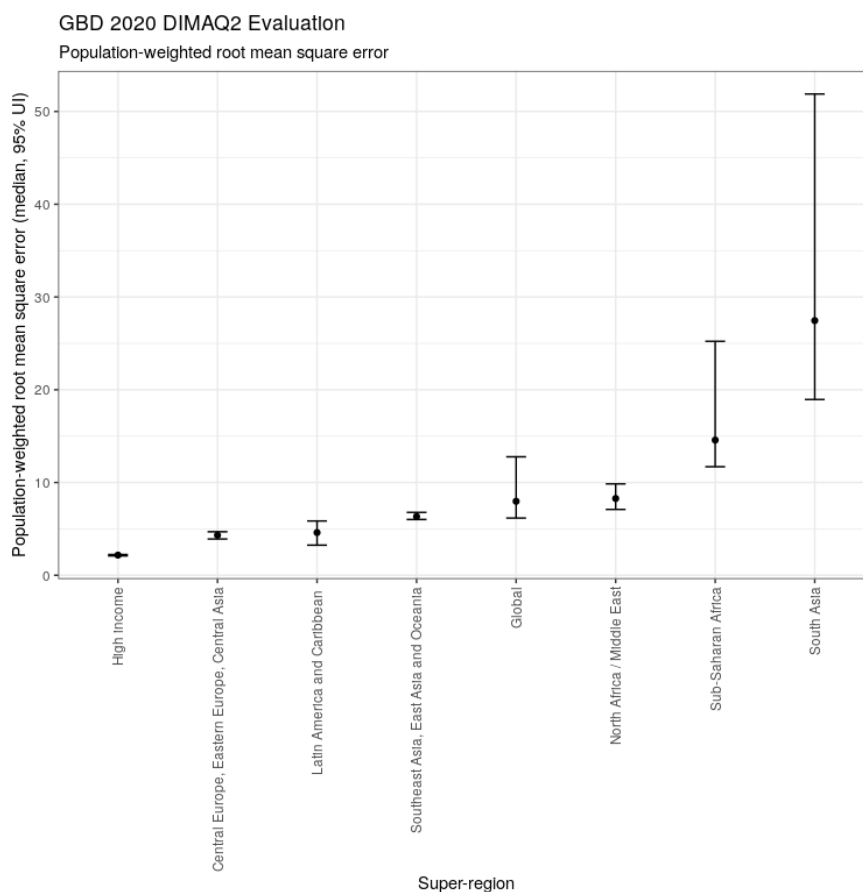


Table 2: Summary measure of predictive ability, globally and by super-region. Values denote median, lower, and upper 95% uncertainty interval bounds of out-of-sample population-weighted relative error (root mean square error/mean PM_{2.5} prediction reported in µg/m³) from 25 validation sets.

Location	Median	Lower	Upper
Global	0.115	0.105	0.133
Central Europe, eastern Europe, central Asia	0.189	0.180	0.199
High income	0.151	0.147	0.155
Latin America and Caribbean	0.234	0.179	0.313
North Africa and Middle East	0.243	0.217	0.263
South Asia	0.452	0.349	0.616
Southeast Asia, east Asia, and Oceania	0.174	0.169	0.184
Sub-Saharan Africa	0.322	0.256	0.409

Theoretical minimum-risk exposure level

The theoretical minimum-risk exposure level (TMREL) was assigned a uniform distribution with lower/upper bounds given by the average of the minimum and 5th percentiles of outdoor air pollution cohort studies exposure distributions conducted in North America, with the assumption that current evidence was insufficient to precisely characterise the shape of the concentration-response function below the 5th percentile of the exposure distributions. The TMREL was defined as a uniform distribution rather than a fixed value in order to represent the uncertainty regarding the level at which the scientific evidence was consistent with adverse effects of exposure. The specific outdoor air pollution cohort studies selected for this averaging were based on the criteria that their 5th percentiles were less than that of the American Cancer Society Cancer Prevention II (CPSII) cohort's 5th percentile of 8.2 based on Turner et al. (2016).⁹ This criterion was selected because GBD 2010 used the minimum, 5.8, and 5th percentile solely from the CPS II cohort. The resulting lower/upper bounds of the distribution for GBD 2020 were 2.4 and 5.9. This has not changed since GBD 2015.

Relative risks and population attributable fractions

Input data

For GBD 2020, as in previous GBD cycles, we created one set of cause-specific risk curves for both household air pollution and ambient particulate matter pollution as two different sources of PM_{2.5}. In GBD 2017, we estimated the particulate matter-attributable burden of disease based on the relation of long-term exposure to PM_{2.5} with ischaemic heart disease, stroke (ischaemic and haemorrhagic), COPD, lung cancer, acute lower respiratory infection, and type 2 diabetes. In GBD 2019, we added adverse birth outcomes including low birthweight and short gestation as contributors to PM_{2.5}-attributable burden. Because these are risk factors (not outcomes) included in the GBD study, we performed a mediation analysis, in which a proportion of the burden attributable to low birthweight and short gestation is attributed to PM_{2.5} pollution.

For the six non-mediated outcomes, we used results from cohort and case-control studies of ambient PM_{2.5} pollution and cohort studies, case-control studies, and randomised-controlled trials of household use of solid fuel for cooking. For GBD 2020, we excluded secondhand smoke cohort and case-control studies from risk curve input data.

We conducted a literature review for studies of PM_{2.5} (ambient and household air pollution) and risk of lower respiratory infection using the search string below. We searched the PubMed database for studies published between January 1, 2017, and July 22, 2020 (date of search). 32 initial results were obtained from the database, 31 of which were excluded during title-abstract and full-text screening. The remaining study was later excluded due to insufficient information reported on the study-specific exposure distribution.

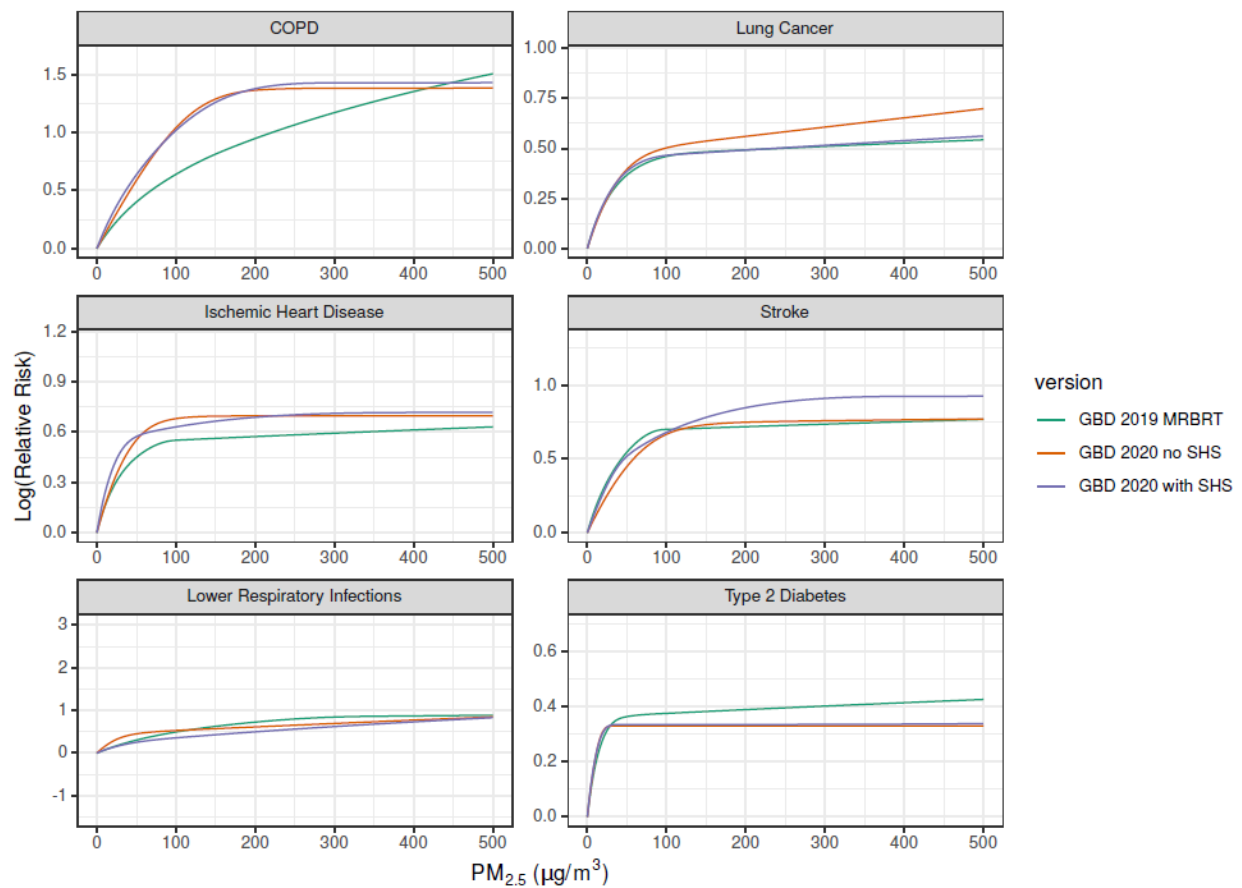
Search string: (((("Air Pollution"[Mesh] OR "Particulate Matter"[Mesh] OR "air pollution"[Title/Abstract] OR "urban air pollution"[Title/Abstract] OR "ambient air pollution"[Title/Abstract] OR "airborne particulate matter"[Title/Abstract]) OR ("Air Pollution, Indoor"[Mesh] OR "Household air"[Title/Abstract] OR "Indoor air pollution"[Title/Abstract] OR "Indoor fine particulate matter"[Title/Abstract] OR "Indoor particulate matter"[Title/Abstract] OR "Indoor air quality"[Title/Abstract])) AND ("lower respiratory infection"[Title/Abstract] OR "LRI"[Title/Abstract]))

Table 3: Data inputs for relative risks for ambient particulate matter pollution

Input data	Relative risk
Site-years (total)	160
Number of countries with data	37
Number of GBD regions with data (out of 21 regions)	15
Number of GBD super-regions with data (out of 7 super-regions)	7

For GBD 2020, as in GBD 2019, the meta-regression—Bayesian, regularised, trimmed (MR-BRT) meta-regression tool was used to create relative risk estimates, with three key updates to input data. In GBD 2017, we used relative estimates for active smoking and secondhand smoke (converting cigarettes per day to PM_{2.5} exposure) to estimate relative risk predictions for PM_{2.5} exposure at the highest end of the exposure–response curve. These data were included because the majority of the air pollution epidemiological studies have been performed in high-income countries which have lower levels of ambient PM_{2.5} pollution. This posed a barrier to extrapolating relative risk estimates from the steep relationship at the beginning of the exposure range to locations with high exposures but no relative risk estimates, such as India and China. In GBD 2019, we incorporated estimates at high PM_{2.5} levels by adding recently published ambient PM_{2.5} studies conducted in China and other higher-exposure settings and additional HAP studies.^{10,11,12,13,14} Additionally, the switch to MR-BRT splines in GBD 2019 (instead of the integrated exposure–response function employed in GBD 2017) presented a more flexible approach that allowed the curve to fit ambient and household data and removed the need for active smoking data to anchor the curve at higher exposures. The inclusion of active smoking and secondhand smoking data in previous GBD cycles required conversion from cigarettes per day to PM_{2.5} exposure and introduced other differences, including differences in dose rates and those between voluntary (active smoking) and involuntary (ambient PM_{2.5}, household air pollution, secondhand smoke) exposures. Due to these factors, in GBD 2019, we removed active smoking data from the relative risk model’s input data. In GBD 2020, we also removed secondhand smoking data, completing the transition to only using PM_{2.5} and HAP relative

risk input data. This removes important sources of uncertainty in our earlier estimates.^{15,16} The following plot displays PM_{2.5} risk curves from GBD 2019 and from GBD 2020, with and without secondhand smoking RR input data:



For GBD 2019, we implemented age-specific risk curves for cardiovascular diseases (ischaemic heart disease and stroke) due to evidence suggesting relative risk decreases with age for these outcomes.¹⁷ These risk curves were created for five-year age groups from 25–29 to 95+. For GBD 2020, we dropped the use of age-specific risk curves for cardiovascular disease outcomes. Linear regressions on cardiovascular disease input data predicting log(RR) by mean cohort age, with and without random effects on study ID, were fit to ischaemic heart disease and stroke input data separately. None of these regressions showed evidence for a significant association between the two variables. Additionally, we used the MR-BRT automated covariate selection tool (detailed below) to test mean cohort age for significance as a bias covariate and found no significant results. We therefore generated a single risk curve for each of the cardiovascular outcomes and applied it across all age groups.

For all PM_{2.5} outcomes, the standard error of observations from studies with multiple observations for a single cohort that reported an unstratified sample size were multiplied by the square root of n , where n is the total number of observations for a given cohort. This adjustment was made to prevent a single cohort or study from unduly weighting the final risk curve.

As in previous GBD cycles, we considered the published relative risk over a range of exposure data when fitting the risk curves. For OAP studies, the relative risk informs the curve from the 5th to the 95th percentile of observed exposure. When this is not available in the published study, we estimate the distribution from the provided information (mean and standard deviation, mean and IQR, etc.).

We scale the RR to this range. For HAP studies, we allow each study to inform the curve from the Exp_{OAP} to the $Exp_{OAP+Exp_{HAP}}$, where Exp_{OAP} is the GBD 2019 estimate of the ambient exposure level in the study location and year, and Exp_{HAP} is the GBD 2020 estimate of the excess exposure for those who use solid fuel for cooking in the study location and year.

MR-BRT risk splines

To estimate relative risk curves for each of the $PM_{2.5}$ outcomes, we used the MR-BRT meta-regression tool to fit splines on the input datasets of OAP and HAP studies. We used the following functional form, where X and X_{CF} represent the range of exposure characterised by the effect size:

$$\log\left(\frac{MRBRT(X)}{MRBRT(X_{CF})}\right) \sim \log(Published\ Effect\ Size)$$

Several key updates were made to the model fitting methods. For each risk-outcome pair, model settings and priors were tested when fitting the MR-BRT splines. The final models used third order splines with three interior knots and a constraint on the right-most segment forcing the fit to be linear rather than cubic. Splines were also constrained to be concave and monotonically increasing, the most biologically plausible shape for the $PM_{2.5}$ risk curve. We used an ensemble approach to generate final spline predictions, in which 50 different models were run with randomly placed knots, then weighted and combined based on a measure of fit that penalises excessive changes in the maximum derivative of the curve. Knots were free to be placed across the entire domain of the input exposure data. To prevent over-fitting, on the non-linear segments, we implemented a Gaussian prior on the third derivative of mean 0 and variance $1e-4$. On the linear segment, a stronger prior of mean 0 and variance $1e-6$ was used to ensure that the risk curves do not continue to increase beyond the range of the data. 10% of all observations were trimmed during model fitting, in accordance with GBD protocol across risk factor teams.

To select significant covariates from those extracted (see table below) to quantify between-study heterogeneity, we performed covariate selection. The MR-BRT automated covariate selection tool implements a two-step process. First, a series of loosening Lasso penalty parameters are applied to a log-linear meta-regression on all input effect size observations. Then, covariates with a non-zero coefficient are tested for significance using a Gaussian prior (significance threshold = 0.05). A Gaussian prior was used on each covariate's beta during spline fitting with a mean 0 and variance of 0.1 multiplied by the standard deviation of the beta from the log-linear meta-regression. Type 2 diabetes was the only outcome for which a significant covariate was identified. Its selected covariate was cv_hap , a binary indicator for whether or not an observation was from a household air pollution study.

Covariate name	Covariate description
$cv_subpopulation$	Study represents the general population; study represents a subgroup (eg, high-risk group)
$cv_exposure_population$	Study measures individual-level exposure (≤ 1 km radius); study measures population-level exposure
$cv_exposure_self_report$	Exposure is self-reported; exposure is measured externally
$cv_exposure_study$	Exposure is measured multiple times; exposure is measured only at baseline
$cv_outcome_self_report$	Outcome is self-reported; outcomes is based on death certificate or medical record
$cv_outcome_unblinded$	Study implements unblinded assessment; assessment of outcome is blind to exposure (and vice versa)

cv_reverse_causation	Study presents no risk of reverse causation; risk of reverse causation
cv_confounding_nonrandom	Non-randomised study; randomised study
cv_confounding_uncontrolled	Study is randomised/outcome controlled for age, sex, education, income, and all critical determinants of outcome; study is controlled for age, sex, and other critical determinants of outcome; study is controlled for only age and sex
cv_selection_bias	Study reports >95% follow-up; study reports 85-95% follow-up; study reports <85% follow-up
cv_hap	Studies household air pollution; studies ambient air pollution

1000 predictions of the effect size were generated across the exposure distribution for use in calculating burden estimates. These predictions were created by incorporating predictions of between-study heterogeneity to characterise the model's uncertainty. We implemented the Fisher Scoring correction to the heterogeneity parameter, which corrects for data-sparse situations. In such cases, the between-study heterogeneity parameter estimate may be 0, simply from lack of data. The Fisher Scoring correction uses a quantile of gamma, which is sensitive to the number of studies, study design, and reported uncertainty.

Evidence scoring

Evidence scores provide an empirical measure of the strength of evidence for risk-outcome pairs across risk factors in the GBD and are therefore useful for standardised comparison. Evidence scores evaluate the area between the lower bound of the 95% uncertainty interval and the x-axis for harmful risk factors, including PM_{2.5} pollution.

Prior to generating an evidence score, we conducted an additional post-analysis step to detect and flag publication bias in the input data. This approach is based on the classic Egger's Regression strategy, which is applied to the residuals in our model. In the current implementation, we do not correct for publication bias, but flag the risk-outcome pairs where the risk for publication bias is significant. Of the PM_{2.5} outcomes, three were flagged for publication bias: birthweight, ischaemic heart disease, and type 2 diabetes.

Outcome	Egger p-value	Egger mean	Egger SD	Publication bias
Birthweight	0.0208	-0.322	0.158	X
Gestational age	0.249	-0.130	0.192	
Ischaemic heart disease	0.0164	0.322	0.151	X
Stroke	0.0717	0.186	0.127	
LRI	0.178	0.102	0.110	
Lung cancer	0.191	0.108	0.123	
COPD	0.423	0.0359	0.186	
Type 2 diabetes	0.0419	0.408	0.236	X

A modified Trim-and-Fill approach was implemented in order to adjust for publication bias. Using this method, 5, 4, and 7 additional points were filled for birthweight, ischaemic heart disease, and type 2 diabetes, respectively, before refitting the model with the adjusted dataset. This adjusted

model was used only to generate an adjusted evidence score, not to calculate population attributable fractions.

To calculate the evidence score, we generated an uncertainty interval from 1000 draws of the adjusted summary effect size (retaining uncertainty information from between-study heterogeneity predictions and the Fisher information correction). We then evaluated the evidence score between the 15th and 85th percentiles of the input data exposure distribution. Evidence scores and star ratings are below. Evidence scores are not reported for birthweight and gestational age because these are mediated outcomes.

Outcome	Evidence score	Star rating
Ischaemic heart disease	0.259	3
Stroke	0.167	3
LRI	0.126	2
Lung cancer	0.342	3
COPD	0.441	4
Type 2 diabetes	0.188	3

The following table includes all ambient and household sources used to generate GBD 2020 risk curves.

Source	Reference
1	Abusalah A, Gavana M, Haidich AB, Smyrnakis E, Papadakis N, Papanikolaou A, Benos A. Low birth weight and prenatal exposure to indoor pollution from tobacco smoke and wood fuel smoke: a matched case-control study in Gaza Strip. <i>Matern Child Health J.</i> 2012; 16(8): 1718-27.
2	Akhtar T, Ullah Z, Khan MH, Nazli R. Chronic bronchitis in women using solid biomass fuel in rural Peshawar, Pakistan. <i>Chest.</i> 2007; 132(5): 1472-5.
3	Al-Sonboli N, Hart CA, Al-Aghbari N, Al-Ansi A, Ashoor O, Cuevas LE. Human metapneumovirus and respiratory syncytial virus disease in children, Yemen. <i>Emerg Infect Dis.</i> 2006; 12(9): 1437-9.
4	Alam DS, Chowdhury MAH, Siddiquee AT, Ahmed S, Hossain MD, Pervin S, Streatfield K, Cravioto A, Niessen LW. Adult Cardiopulmonary Mortality and Indoor Air Pollution: A 10-Year Retrospective Cohort Study in a Low-Income Rural Setting. <i>Glob Heart.</i> 2012; 7(3): 215-21.
5	Alexander DA, Northcross A, Karrison T, Morhasson-Bello O, Wilson N, Atalabi OM, Dutta A, Adu D, Ibigbami T, Olamijulo J, Adepoju D, Ojengbede O, Olopade CO. Pregnancy outcomes and ethanol cook stove intervention: A randomized-controlled trial in Ibadan, Nigeria. <i>Environ Int.</i> 2018; 111: 152-163.
6	Atkinson RW, Carey IM, Kent AJ, van Staa TP, Anderson HR, Cook DG. Long-term exposure to outdoor air pollution and the incidence of chronic obstructive pulmonary disease in a national English cohort. <i>Occup Environ Med.</i> 2015; 72(1): 42-8.
7	Azizi BH, Zulkifli HI, Kasim MS. Protective and risk factors for acute respiratory infections in hospitalized urban Malaysian children: a case control study. <i>Southeast Asian J Trop Med Public Health.</i> 1995; 26(2): 280-5.

8	Balakrishnan K, Ghosh S, Thangavel G, Sambandam S, Mukhopadhyay K, Puttaswamy N, Sadasivam A, Ramaswamy P, Johnson P, Kuppuswamy R, Natesan D, Maheshwari U, Natarajan A, Rajendran G, Ramasami R, Madhav S, Manivannan S, Nargunanadan S, Natarajan S, Saidam S, Chakraborty M, Balakrishnan L, Thanasekaraan V. Exposures to fine particulate matter (PM2.5) and birthweight in a rural-urban, mother-child cohort in Tamil Nadu, India. <i>Environ Res.</i> 2018; 161: 524–31.
9	Basu R, Harris M, Sie L, Malig B, Broadwin R, Green R. Effects of fine particulate matter and its constituents on low birth weight among full-term infants in California. <i>Environ Res.</i> 2014; 128: 42–51.
10	Basu R, Pearson D, Ebisu K, Malig B. Association between PM2.5 and PM2.5 Constituents and Preterm Delivery in California, 2000-2006. <i>Paediatr Perinat Epidemiol.</i> 2017; 31(5): 424-434.
11	Beelen R, Hoek G, van den Brandt PA, Goldbohm RA, Fischer P, Schouten LJ, Jerrett M, Hughes E, Armstrong B, Brunekreef B. Long-Term Effects of Traffic-Related Air Pollution on Mortality in a Dutch Cohort (NLCS-AIR Study) [Unpublished data]. <i>Environ Health Perspect.</i> 2008; 116(2): 196–202.
12	Beelen R, Hoek G, van den Brandt PA, Goldbohm RA, Fischer P, Schouten LJ, Jerrett M, Hughes E, Armstrong B, Brunekreef B. Long-Term Effects of Traffic-Related Air Pollution on Mortality in a Dutch Cohort (NLCS-AIR Study). <i>Environ Health Perspect.</i> 2008; 116(2): 196–202.
13	Beelen R, Stafoggia M, Raaschou-Nielsen O, Andersen ZJ, Xun WW, Katsouyanni K, Dimakopoulou K, Brunekreef B, Weinmayr G, Hoffmann B, Wolf K, Samoli E, Houthuijs D, Nieuwenhuijsen M, Oudin A, Forsberg B, Olsson D, Salomaa V, Lanki T, Yli-Tuomi T, Oftedal B, Aamodt G, Nafstad P, De Faire U, Pedersen NL, Östenson CG, Fratiglioni L, Penell J, Korek M, Pyko A, Eriksen KT, Tjønneland A, Becker T, Eeftens M, Bots M, Meliefste K, Wang M, Bueno-de-Mesquita B, Sugiri D, Krämer U, Heinrich J, de Hoogh K, Key T, Peters A, Cyrus J, Concini H, Nagel G, Ineichen A, Schaffner E, Probst-Hensch N, Dratva J, Ducret-Stich R, Vilier A, Clavel-Chapelon F, Stempfelet M, Grioni S, Krogh V, Tsai MY, Marcon A, Ricceri F, Sacerdote C, Galassi C, Migliore E, Ranzi A, Cesaroni G, Badaloni C, Forastiere F, Tamayo I, Amiano P, Dorronsoro M, Katsoulis M, Trichopoulou A, Vineis P, Hoek G. Long-term exposure to air pollution and cardiovascular mortality: an analysis of 22 European cohorts. <i>Epidemiology.</i> 2014; 25(3): 368–378.
14	Bell ML, Belanger K, Ebisu K, Gent JF, Lee HJ, Koutrakis P, Leaderer BP. Prenatal Exposure to Fine Particulate Matter and Birth Weight: Variations by Particulate Constituents and Sources. <i>Epidemiology.</i> 2010; 21(6): 884–91.
15	Bell ML, Ebisu K, Belanger K. Ambient Air Pollution and Low Birth Weight in Connecticut and Massachusetts. <i>Environ Health Perspect.</i> 2007; 115(7): 1118–24.
16	Benmarhnia T, Huang J, Basu R, Wu J, Bruckner TA. Decomposition Analysis of Black-White Disparities in Birth Outcomes: The Relative Contribution of Air Pollution and Social Factors in California. <i>Environ Health Perspect.</i> 2017; 125(10): 107003.
17	Bowe B, Xie Y, Li T, Yan Y, Xian H, Al-Aly Z. The 2016 global and national burden of diabetes mellitus attributable to PM2.5 air pollution. <i>Lancet Planet Health.</i> 2018; 2(7): e301–12.

18	Boy E, Bruce N, Delgado H. Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. <i>Environ Health Perspect.</i> 2002; 110(1): 109-14.
19	Brauer M, Lencar C, Tamburic L, Koehoorn M, Demers P, Karr C. A cohort study of traffic-related air pollution impacts on birth outcomes. <i>Environ Health Perspect.</i> 2008; 116(5): 680-6.
20	Broor S, Pandey RM, Ghosh M, Maitreyi RS, Lodha R, Singhal T, Kabra SK. Risk factors for severe acute lower respiratory tract infection in under-five children. <i>Indian Pediatr.</i> 2001; 1361-9.
21	Burnett RT. Cox Proportional Survival Model Hazard Ratios from Census Year to 2011 for Adults Aged 25 to 89 in CanCHEC Cohort.
22	Cai J, Zhao Y, Kan J, Chen R, Martin R, van Donkelaar A, Ao J, Zhang J, Kan H, Hua J. Prenatal Exposure to Specific PM _{2.5} Chemical Constituents and Preterm Birth in China: A Nationwide Cohort Study. <i>Environ Sci Technol.</i> 2020; 54(22): 14494-14501.
23	Cakmak S, Hebbern C, Pinault L, Lavigne E, Vanos J, Crouse DL, Tjepkema M. Associations between long-term PM _{2.5} and ozone exposure and mortality in the Canadian Census Health and Environment Cohort (CANCHEC), by spatial synoptic classification zone. <i>Environ Int.</i> 2018; 111: 200-211.
24	Carey IM, Atkinson RW, Kent AJ, van Staa T, Cook DG, Anderson HR. Mortality associations with long-term exposure to outdoor air pollution in a national English cohort. <i>Am J Respir Crit Care Med.</i> 2013; 187(11): 1226-33.
25	Cassidy-Bushrow AE, Burmeister C, Lamerato L, Lemke LD, Mathieu M, O'Leary BF, Sperone FG, Straughen JK, Reiners JJ Jr. Prenatal airshed pollutants and preterm birth in an observational birth cohort study in Detroit, Michigan, USA. <i>Environ Res.</i> 2020; 189: 109845.
26	Cesaroni G, Badaloni C, Gariazzo C, Stafoggia M, Sozzi R, Davoli M, Forastiere F. Long-term exposure to urban air pollution and mortality in a cohort of more than a million adults in Rome. <i>Environ Health Perspect.</i> 2013; 121(3): 324–31.
27	Chang HH, Reich BJ, Miranda ML. A spatial time-to-event approach for estimating associations between air pollution and preterm birth. <i>J R Stat Soc Ser C Appl Stat.</i> 2013; 62(2).
28	Chen H, Burnett RT, Kwong JC, Villeneuve PJ, Goldberg MS, Brook RD, van Donkelaar A, Jerrett M, Martin RV, Brook JR, Copes R. Risk of incident diabetes in relation to long-term exposure to fine particulate matter in Ontario, Canada. <i>Environ Health Perspect.</i> 2013; 121(7): 804–10.
29	Chen LH, Knutsen SF, Shavlik D, Beeson WL, Petersen F, Ghamsary M, Abbey D. The association between fatal coronary heart disease and ambient particulate air pollution: Are females at greater risk? <i>Environ Health Perspect.</i> 2005; 113(12): 1723-9.
30	Chen G, Guo Y, Abramson MJ, Williams G, Li S. Exposure to low concentrations of air pollutants and adverse birth outcomes in Brisbane, Australia, 2003-2013. <i>Sci Total Environ.</i> 2018; 622-623: 721-726.
31	Chen J, Fang J, Zhang Y, Xu Z, Byun HM, Li PH, Deng F, Guo X, Guo L, Wu S. Associations of adverse pregnancy outcomes with high ambient air pollution exposure: Results from the Project ELEFANT. <i>Sci Total Environ.</i> 2021; 761: 143218.

32	Clark C, Sbihi H, Tamburic L, Brauer M, Frank LD, Davies HW. Association of Long-Term Exposure to Transportation Noise and Traffic-Related Air Pollution with the Incidence of Diabetes: A Prospective Cohort Study. <i>Environ Health Perspect.</i> 2017; 125(8): 087025.
33	Clemens T, Turner S, Dibben C. Maternal exposure to ambient air pollution and fetal growth in North-East Scotland: A population-based study using routine ultrasound scans. <i>Environ Int.</i> 2017; 107: 216–26.
34	Coker E, Ghosh J, Jerrett M, Gomez-Rubio V, Beckerman B, Cockburn M, Liverani S, Su J, Li A, Kile ML, Ritz B, Molitor J. Modeling spatial effects of PM(2.5) on term low birth weight in Los Angeles County. <i>Environ Res.</i> 2015; 142: 354-64.
35	Collings DA, Sithole SD, Martin KS. Indoor woodsmoke pollution causing lower respiratory disease in children. <i>Trop Doct.</i> 1990; 20(4): 151–5.
36	Coogan PF, White LF, Yu J, Burnett RT, Seto E, Brook RD, Palmer JR, Rosenberg L, Jerrett M. PM2.5 and Diabetes and Hypertension Incidence in the Black Women’s Health Study. <i>Epidemiology.</i> 2016; 27(2): 202–10.
37	Cramer J, Jørgensen JT, Hoffmann B, et al. Long-Term Exposure to Air Pollution and Incidence of Myocardial Infarction: A Danish Nurse Cohort Study. <i>Environ Health Perspect.</i> 2020;128(5):57003. doi:10.1289/EHP5818
38	Dadvand P, Ostro B, Figueras F, Foraster M, Basagaña X, Valentín A, Martínez D, Beelen R, Cirach M, Hoek G, Jerrett M, Brunekreef B, Nieuwenhuijsen MJ. Residential proximity to major roads and term low birth weight: the roles of air pollution, heat, noise, and road-adjacent trees. <i>Epidemiology.</i> 2014; 25(4): 518-25.
39	Darrow LA, Klein M, Strickland MJ, Mulholland JA, Tolbert PE. Ambient Air Pollution and Birth Weight in Full-Term Infants in Atlanta, 1994–2004. <i>Environ Health Perspect.</i> 2011; 119(5): 731–7.
40	Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. Woodsmoke exposure and risk for obstructive airways disease among women. <i>Chest.</i> 1996; 109(1): 115–9.
41	Dherani M, Pope D, Mascarenhas M, Smith KR, Weber M, Bruce N. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. <i>Bull World Health Organ.</i> 2008; 86(5): 390-398C and Kossove D. and Jeena PM, Ayannusi OE, Annamalai K, Naidoo P, Coovadia HM, Guldner P. Risk factors for admission and the role of respiratory syncytial virus-specific cytotoxic T-lymphocyte responses in children with acute bronchiolitis. <i>S Afr Med J.</i> 2003; 93(4): 291–4.
42	Ebisu K, Bell ML. Airborne PM2.5 chemical components and low birth weight in the northeastern and mid-Atlantic regions of the United States. <i>Environ Health Perspect.</i> 2012; 120(12): 1746-52.
43	Ebisu K, Berman JD, Bell ML. Exposure to coarse particulate matter during gestation and birth weight in the U.S. <i>Environ Int.</i> 2016; 94: 519–24.
44	Ebisu K, Belanger K, Bell ML. The Association between Airborne PM2.5 Chemical Constituents and Birth Weight-Implication of Buffer Exposure Assignment. <i>Environ Res Lett.</i> 2014; 9(8).
45	Erickson AC, Ostry A, Chan LH, Arbour L. The reduction of birth weight by fine particulate matter and its modification by maternal and neighbourhood-level factors: a multilevel analysis in British Columbia, Canada. <i>Environ Health.</i> 2016; 15: 51.

46	Fleischer NL, Meriardi M, van Donkelaar A, Vadillo-Ortega F, Martin RV, Betran AP, Souza JP. Outdoor air pollution, preterm birth, and low birth weight: analysis of the world health organization global survey on maternal and perinatal health. <i>Environ Health Perspect.</i> 2014; 122(4): 425-30.
47	Fong KC, Kosheleva A, Kloog I, Koutrakis P, Laden F, Coull BA, Schwartz JD. Fine Particulate Air Pollution and Birthweight: Differences in Associations Along the Birthweight Distribution. <i>Epidemiology.</i> 2019; 30(5): 617-623.
48	Fonseca W, Kirkwood BR, Victora CG, Fuchs SR, Flores JA, Misago C. Risk factors for childhood pneumonia among the urban poor in Fortaleza, Brazil: a case-control study. <i>Bull World Health Organ.</i> 1996; 74(2): 199–208.
49	Galeone C, Pelucchi C, La Vecchia C, Negri E, Bosetti C, Hu J. Indoor air pollution from solid fuel use, chronic lung diseases and lung cancer in Harbin, Northeast China. <i>Eur J Cancer Prev.</i> 2008; 17(5): 473–8.
50	Gan WQ, FitzGerald JM, Carlsten C, Sadatsafavi M, Brauer M. Associations of ambient air pollution with chronic obstructive pulmonary disease hospitalization and mortality. <i>Am J Respir Crit Care Med.</i> 2013; 187(7): 721–7.
51	Gan WQ, Koehoorn M, Davies HW, Demers PA, Tamburic L, Brauer M. Long-Term Exposure to Traffic-Related Air Pollution and the Risk of Coronary Heart Disease Hospitalization and Mortality. <i>Environ Health Perspect.</i> 2011; 119(4): 501–7.
52	Garcia CA, Yap PS, Park HY, Weller BL. 2016. Association of long-term PM2.5 exposure with mortality using different air pollution exposure models: impacts in rural and urban California. <i>International Journal of Environmental Health Research</i> , 26(2), 145-15.
53	Geer LA, Weedon J, Bell ML. Ambient air pollution and term birth weight in Texas from 1998 to 2004. <i>J Air Waste Manag Assoc.</i> 2012; 62(11): 1285–95.
54	Gehring U, Tamburic L, Sbihi H, Davies HW, Brauer M. Impact of Noise and Air Pollution on Pregnancy Outcomes. <i>Epidemiology.</i> 2014; 25(3): 351–8.
55	Gehring U, Wijga AH, Fischer P, de Jongste JC, Kerkhof M, Koppelman GH, Smit HA, Brunekreef B. Traffic-related air pollution, preterm birth and term birth weight in the PIAMA birth cohort study. <i>Environ Res.</i> 2011; 111(1): 125–35.
56	Ger LP, Hsu WL, Chen KT, Chen CJ. Risk Factors of Lung Cancer by Histological Category in Taiwan. <i>Anticancer Res.</i> 1993; 13(5A): 1491–500.
57	Giorgis-Allemand L, Pedersen M, Bernard C, Aguilera I, Beelen RM, Chatzi L, Cirach M, Danileviciute A, Dedele A, van Eijsden M, Estarlich M, Fernández-Somoano A, Fernández MF, Forastiere F, Gehring U, Grazuleviciene R, Gruzieva O, Heude B, Hoek G, de Hoogh K, van den Hooven EH, Håberg SE, Iñiguez C, Jaddoe VW, Korek M, Lertxundi A, Lepeule J, Nafstad P, Nystad W, Patelarou E, Porta D, Postma D, Raaschou-Nielsen O, Rudnai P, Siroux V, Sunyer J, Stephanou E, Sørensen M, Eriksen KT, Tuffnell D, Varró MJ, Vrijkotte TG, Wijga A, Wright J, Nieuwenhuijsen MJ, Pershagen G, Brunekreef B, Kogevinas M, Slama R. The Influence of Meteorological Factors and Atmospheric Pollutants on the Risk of Preterm Birth. <i>Am J Epidemiol.</i> 2017; 185(4): 247-258.
58	Gray SC, Edwards SE, Schultz BD, Miranda ML. Assessing the impact of race, social factors and air pollution on birth outcomes: a population-based study. <i>Environ Health.</i> 2014; 13(1): 4.
59	Gray SC, Gelfand AE, Miranda ML. Hierarchical spatial modeling of uncertainty in air pollution and birth weight study. <i>Stat Med.</i> 2011; 30(17): 2187-98.

60	Guo T, Wang Y, Zhang H, Zhang Y, Zhao J, Wang Q, Shen H, Wang Y, Xie X, Wang L, Xu Z, Zhang Y, Yan D, He Y, Yang Y, Xu J, Peng Z, Ma X. The association between ambient PM2.5 exposure and the risk of preterm birth in China: A retrospective cohort study. <i>Sci Total Environ.</i> 2018; 633: 1453-1459.
61	Gupta D, Boffetta P, Gaborieau V, Jindal SK. Risk factors of lung cancer in Chandigarh, India. <i>Indian J Med Res.</i> 2001; 113: 142–50.
62	Ha S, Hu H, Roussos-Ross D, Haidong K, Roth J, Xu X. The effects of air pollution on adverse birth outcomes. <i>Environ Res.</i> 2014; 134: 198-204.
63	Ha S, Zhu Y, Liu D, Sherman S, Mendola P. Ambient temperature and air quality in relation to small for gestational age and term low birthweight. <i>Environ Res.</i> 2017; 155: 394-400.
64	Han Y, Ji Y, Kang S, Dong T, Zhou Z, Zhang Y, Chen M, Wu W, Tang Q, Chen T, Wang Y, Xia Y. Effects of particulate matter exposure during pregnancy on birth weight: A retrospective cohort study in Suzhou, China. <i>Sci Total Environ.</i> 2018; 615: 369-374.
65	Hansen AB, Ravnskjaer L, Loft S, Andersen KK, Brauner EV, Bastrup R, Yao C, Ketzel M, Becker T, Brandt J, Hertel O, Andersen ZJ. Long-term exposure to fine particulate matter and incidence of diabetes in the Danish Nurse Cohort. <i>Environ Int.</i> 2016; 91: 243–50.
66	Hao H, Chang HH, Holmes HA, Mulholland JA, Klein M, Darrow LA, Strickland MJ. Air Pollution and Preterm Birth in the U.S. State of Georgia (2002-2006): Associations with Concentrations of 11 Ambient Air Pollutants Estimated by Combining Community Multiscale Air Quality Model (CMAQ) Simulations with Stationary Monitor Measurements. <i>Environ Health Perspect.</i> 2016; 124(6): 875-80.
67	Hao Y, Strosnider H, Balluz L, Qualters JR. Geographic Variation in the Association between Ambient Fine Particulate Matter (PM2.5) and Term Low Birth Weight in the United States. <i>Environ Health Perspect.</i> 2016; 124(2): 250-5.
68	Harris G, Thompson WD, Fitzgerald E, Wartenberg D. The association of PM(2.5) with full term low birth weight at different spatial scales. <i>Environ Res.</i> 2014; 134: 427-34.
69	Hart J, Garshick E, Dockery D, Smith T, Ryan L, Laden F. Long-Term Ambient Multipollutant Exposures and Mortality. <i>Am J Respir Crit Care Med.</i> 2011; 183: 75–8.
70	Hart JE, Puett RC, Rexrode KM, Albert CM, Laden F. Effect Modification of Long-Term Air Pollution Exposures and the Risk of Incident Cardiovascular Disease in US Women. <i>J Am Heart Assoc.</i> 2015; 4(12).
71	Heft-Neal S, Burney J, Bendavid E, Burke M. Robust relationship between air quality and infant mortality in Africa. <i>Nature.</i> 2018; 559(7713): 2548.
72	Hertz-Picciotto I, Baker RJ, Yap P-S, Dostál M, Joad JP, Lipsett M, Greenfield T, Herr CEW, Benes I, Shumway RH, Pinkerton KE, Srám R. Early childhood lower respiratory illness and air pollution. <i>Environ Health Perspect.</i> 2007; 115(10): 1510-8.
73	Honda T, Pun VC, Manjourides J, et al. Associations between long-term exposure to air

	pollution, glycosylated hemoglobin and diabetes. <i>Int J Hyg Environ Health</i> . 2017; 220 (7): 1124-1132.
74	Huang C, Zhang X, Qiao Z, Guan L, Peng S, Liu J, Xie R, Zheng L. A case-control study of dietary factors in patients with lung cancer. <i>Biomed Environ Sci</i> . 1992; 5(3): 257–65.
75	Huang H, Woodruff TJ, Baer RJ, Bangia K, August LM, Jellife-Palowski LL, Padula AM, Sirota M. Investigation of association between environmental and socioeconomic factors and preterm birth in California. <i>Environ Int</i> . 2018; 121(Pt 2): 1066-1078.
76	Huang K, Liang F, Yang X, Liu F, Li J, Xiao Q, Chen J, Liu X, Cao J, Shen C, Yu L, Lu F, Wu X, Zhao L, Wu X, Li Y, Hu D, Huang J, Liu Y, Lu X, Gu D. Long term exposure to ambient fine particulate matter and incidence of stroke: prospective cohort study from the China-PAR project. <i>BMJ</i> . 2019; 367: l6720.
77	Huynh M, Woodruff TJ, Parker JD, Schoendorf KC. Relationships between air pollution and preterm birth in California. <i>Paediatr Perinat Epidemiol</i> . 2006; 20(6): 454-61.
78	Hyder A, Lee HJ, Ebisu K, Koutrakis P, Belanger K, Bell ML. PM2.5 Exposure and Birth Outcomes: Use of Satellite- and Monitor-Based Data. <i>Epidemiology</i> . 2014; 25(1): 58–67.
79	Hystad P, Demers PA, Johnson KC, Carpiano RM, Brauer M. Long-term residential exposure to air pollution and lung cancer risk. <i>Epidemiology</i> . 2013; 24(5): 762-72.
80	Hystad P, Duong M, Brauer M, Larkin A, Arku R, Kurmi OP, Fan WQ, Avezum A, Azam I, Chifamba J, Dans A, du Plessis JL, Gupta R, Kumar R, Lanas F, Liu Z, Lu Y, Lopez-Jaramillo P, Mony P, Mohan V, Mohan D, Nair S, Puoane T, Rahman O, Lap AT, Wang Y, Wei L, Yeates K, Rangarajan S, Teo K, Yusuf S, on behalf of Prospective Urban and Rural Epidemiological (PURE) Study investigators. Health Effects of Household Solid Fuel Use: Findings from 11 Countries within the Prospective Urban and Rural Epidemiology Study [Unpublished]. <i>Environ Health Perspect</i> . 2019; 127(5): 57003.
81	Hystad P, Duong M, Brauer M, Larkin A, Arku R, Kurmi OP, Fan WQ, Avezum A, Azam I, Chifamba J, Dans A, du Plessis JL, Gupta R, Kumar R, Lanas F, Liu Z, Lu Y, Lopez-Jaramillo P, Mony P, Mohan V, Mohan D, Nair S, Puoane T, Rahman O, Lap AT, Wang Y, Wei L, Yeates K, Rangarajan S, Teo K, Yusuf S, on behalf of Prospective Urban and Rural Epidemiological (PURE) Study investigators. Health Effects of Household Solid Fuel Use: Findings from 11 Countries within the Prospective Urban and Rural Epidemiology Study. <i>Environ Health Perspect</i> . 2019; 127(5): 57003.
82	Hystad P, Larkin A, Rangarajan S, PURE country investigators, Yusuf S, Brauer M. Outdoor fine particulate matter air pollution and cardiovascular disease: Results from 747 communities across 21 countries in the PURE Study [Unpublished].
83	Jedrychowski W, Perera F, Mrozek-Budzyn D, Mroz E, Flak E, Spengler JD, Edwards S, Jacek R, Kaim I, Skolicki Z. Gender differences in fetal growth of newborns exposed prenatally to airborne fine particulate matter. <i>Environ Res</i> . 2009; 109(4): 447-56.
84	Jerrett M, Burnett RT, Beckerman BS, et al. 2013. Spatial analysis of air pollution and

	mortality in California. <i>American Journal of Respiratory and Critical Care Medicine</i> , 188(5), 593-599.
85	Jin C, Rossignol AM. Effects of passive smoking on respiratory illness from birth to age eighteen months, in Shanghai, People's Republic of China. <i>J Pediatr.</i> 1993; 123(4): 553–8.
86	Johnson AW, Adererele WI. The association of household pollutants and socio-economic risk factors with the short-term outcome of acute lower respiratory infections in hospitalized pre-school Nigerian children. <i>Ann Trop Paediatr.</i> 1992; 12(4): 421–32.
87	Karr C, Lumley T, Schreuder A, Davis R, Larson T, Ritz B, Kaufman J. Effects of subchronic and chronic exposure to ambient air pollutants on infant bronchiolitis. <i>Am J Epidemiol.</i> 2007; 165(5): 553-60.
88	Karr CJ, Rudra CB, Miller KA, Gould TR, Larson T, Sathyanarayana S, Koenig JQ. Infant exposure to fine particulate matter and traffic and risk of hospitalization for RSV bronchiolitis in a region with lower ambient air pollution. <i>Environ Res.</i> 2009; 109(3): 321-7.
89	Katanoda K, Sobue T, Satoh H, Tajima K, Suzuki T, Nakatsuka H, Takezaki T, Nakayama T, Nitta H, Tanabe K, Tominaga S. An association between long-term exposure to ambient air pollution and mortality from lung cancer and respiratory diseases in Japan. <i>J Epidemiol.</i> 2011; 21(2): 132-43.
90	Kim C, Seow WJ, Shu X-O, Bassig BA, Rothman N, Chen BE, Xiang Y-B, Hosgood HD, Ji B-T, Hu W, Wen C, Chow W-H, Cai Q, Yang G, Gao Y-T, Zheng W, Lan Q. Cooking Coal Use and All-Cause and Cause-Specific Mortality in a Prospective Cohort Study of Women in Shanghai, China. <i>Environ Health Perspect.</i> 2016; 124(9): 1384–9.
91	Kingsley SL, Eliot MN, Glazer K, Awad YA, Schwartz JD, Savitz DA, Kelsey KT, Marsit CJ, Wellenius GA. Maternal ambient air pollution, preterm birth and markers of fetal growth in Rhode Island: results of a hospital-based linkage study. <i>J Epidemiol Community Health.</i> 2017; 71(12): 1131-1136.
92	Kirwa K, McConnell-Rios R, Manjourides J, Cordero J, Alshawabekeh A, Suh HH. Low birth weight and PM2.5 in Puerto Rico. <i>Environ Epidemiol.</i> 2019; 3(4).
93	Kleinerman RA, Wang Z, Wang L, Metayer C, Zhang S, Brenner AV, Zhang S, Xia Y, Shang B, Lubin JH. Lung cancer and indoor exposure to coal and biomass in rural China. <i>J Occup Environ Med.</i> 2002; 44(4): 338–44.
94	Kloog I, Melly SJ, Ridgway WL, Coull BA, Schwartz J. Using new satellite based exposure methods to study the association between pregnancy pm2.5 exposure, premature birth and birth weight in Massachusetts. <i>Environ Health.</i> 2012; 11(1).
95	Ko YC, Lee CH, Chen MJ, Huang CC, Chang WY, Lin HJ, Wang HZ, Chang PY. Risk factors for primary lung cancer among non-smoking women in Taiwan. <i>Int J Epidemiol.</i> 1997; 26(1): 24-31.
96	Kumar N. Uncertainty in the relationship between criteria pollutants and low birth weight in Chicago. <i>Atmos Environ.</i> 2012; 49: 171–9.
97	Kumar S, Awasthi S, Jain A, Srivastava RC. Blood zinc levels in children hospitalized with severe pneumonia: a case control study. <i>Indian Pediatr.</i> 2004; 41(5): 486–91.
98	Lamichhane DK, Lee SY, Ahn K, Kim KW, Shin YH, Suh DI, Hong SJ, Kim HC. Quantile

	regression analysis of the socioeconomic inequalities in air pollution and birth weight. <i>Environ Int.</i> 2020; 142: 105875.
99	Lan Q, He X, Shen M, Tian L, Liu LZ, Lai H, Chen W, Berndt SI, Hosgood HD, Lee K-M, Zheng T, Blair A, Chapman RS. Variation in lung cancer risk by smoky coal subtype in Xuanwei, China. <i>Int J Cancer.</i> 2008; 123(9): 2164–9.
100	Laurent O, Hu J, Li L, Cockburn M, Escobedo L, Kleeman MJ, Wu J. Sources and contents of air pollution affecting term low birth weight in Los Angeles County, California, 2001-2008. <i>Environ Res.</i> 2014; 134: 488-95.
101	Laurent O, Hu J, Li L, Kleeman MJ, Bartell SM, Cockburn M, Escobedo L, Wu J. A Statewide Nested Case-Control Study of Preterm Birth and Air Pollution by Source and Composition: California, 2001-2008. <i>Environ Health Perspect.</i> 2016; 124(9): 1479-86.
102	Laurent O, Hu J, Li L, Kleeman MJ, Bartell SM, Cockburn M, Escobedo L, Wu J. Low birth weight and air pollution in California: Which sources and components drive the risk? <i>Environ Int.</i> 2016; 92-93: 471-7.
103	Laurent O, Wu J, Li L, Chung J, Bartell S. Investigating the association between birth weight and complementary air pollution metrics: a cohort study. <i>Environ Health.</i> 2013; 12(1).
104	Lavigne E, Yasseen AS 3rd, Stieb DM, Hystad P, van Donkelaar A, Martin RV, Brook JR, Crouse DL, Burnett RT, Chen H, Weichenthal S, Johnson M, Villeneuve PJ, Walker M. Ambient air pollution and adverse birth outcomes: Differences by maternal comorbidities. <i>Environ Res.</i> 2016; 148: 457-466.
105	Lavigne É, Burnett RT, Stieb DM, Evans GJ, Godri Pollitt KJ, Chen H, van Rijswijk D, Weichenthal S. Fine Particulate Air Pollution and Adverse Birth Outcomes: Effect Modification by Regional Nonvolatile Oxidative Potential. <i>Environ Health Perspect</i> . 2018; 126(7): 077012.
106	Le CH, Ko YC, Cheng LS, Lin YC, Lin HJ, Huang MS, Huang JJ, Kao EL, Wang HZ. The heterogeneity in risk factors of lung cancer and the difference of histologic distribution between genders in Taiwan. <i>Cancer Causes Control.</i> 2001; 12(4): 289–300.
107	Lepeule J, Laden F, Dockery D, Schwartz J. Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009 - Unpublished data. <i>Environ Health Perspect.</i> 2012; 120(7): 965-70.
108	Lepeule J, Laden F, Dockery D, Schwartz J. Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009. <i>Environ Health Perspect.</i> 2012; 120(7): 965-70.
109	Li Q, Wang YY, Guo Y, Zhou H, Wang X, Wang Q, Shen H, Zhang Y, Yan D, Zhang Y, Zhang H, Li S, Chen G, Lin L, Zhao J, He Y, Yang Y, Xu J, Wang Y, Peng Z, Wang HJ, Ma X. Effect of airborne particulate matter of 2.5m or less on preterm birth: A national birth cohort study in China. <i>Environ Int.</i> 2018; 121(Pt 2): 1128-1136.
110	Li Q, Wang YY, Guo Y, Zhou H, Wang X, Wang QM, Shen HP, Zhang YP, Yan DH, Li S , Chen G, Lin L, He Y, Yang Y, Peng ZQ, Wang HJ, Ma X. Folic Acid Supplementation and the Association between Maternal Airborne Particulate Matter Exposure and Preterm Delivery: A National Birth Cohort Study in China. <i>Environ Health Perspect.</i> 2020; 128(12): 127010.
111	Li Z, Yuan X, Fu J, Zhang L, Hong L, Hu L, Liu L. Association of ambient air pollutants and birth weight in Ningbo, 2015-2017. <i>Environ Pollut.</i> 2019; 249: 629-637.

112	Lim CC, Hayes RB, Ahn J, Shao Y, Silverman DT, Jones RR, Garcia C, Thurston GD. Association between long-term exposure to ambient air pollution and diabetes mortality in the US. <i>Environ Res.</i> 2018; 165: 330-36
113	Lin L, Li Q, Yang J, Han N, Jin C, Xu X, Liu Z, Liu J, Luo S, Raat H, Wang H. The associations of particulate matters with fetal growth in utero and birth weight: A birth cohort study in Beijing, China. <i>Sci Total Environ.</i> 2020; 709: 136246.
114	Lipsett MJ, Ostro BD, Reynolds P, Goldberg D, Hertz A, Jerrett M, Smith DF, Garcia C, Chang ET, Bernstein L. Long-term exposure to air pollution and cardiorespiratory disease in the California teachers study cohort [Unpublished data]. <i>Am J Respir Crit Care Med.</i> 2011; 184(7): 828-35.
115	Lipsett MJ, Ostro BD, Reynolds P, Goldberg D, Hertz A, Jerrett M, Smith DF, Garcia C, Chang ET, Bernstein L. Long-term exposure to air pollution and cardiorespiratory disease in the California teachers study cohort. <i>Am J Respir Crit Care Med.</i> 2011; 184(7): 828-35.
116	Lissowska J, Bardin-Mikolajczak A, Fletcher T, Zaridze D, Szeszenia-Dabrowska N, Rudnai P, Fabianova E, Cassidy A, Mates D, Holcatova I, Vitova V, Janout V, Mannerje A, Brennan P, Boffetta P. Lung cancer and indoor pollution from heating and cooking with solid fuels: the IARC international multicentre case-control study in Eastern/Central Europe and the United Kingdom. <i>Am J Epidemiol.</i> 2005; 162(4): 326–33.
117	Luo RX, Wu B, Yi YN, Huang ZW, Lin RT. Indoor burning coal air pollution and lung cancer--a case-control study in Fuzhou, China. <i>Lung Cancer.</i> 1996; 14 Suppl 1: S113-119.
118	MacIntyre EA, Gehring U, Mölter A, Fuertes E, Klümper C, Krämer U, Quass U, Hoffmann B, Gascon M, Brunekreef B, Koppelman GH, Beelen R, Hoek G, Birk M, de Jongste JC, Smit HA, Cyrus J, Gruziova O, Korek M, Bergström A, Agius RM, de Vocht F, Simpson A, Porta D, Forastiere F, Badaloni C, Cesaroni G, Esplugues A, Fernández-Somoano A, Lerxundi A, Sunyer J, Cirach M, Nieuwenhuijsen MJ, Pershagen G, Heinrich J. Air Pollution and Respiratory Infections during Early Childhood: An Analysis of 10 European Birth Cohorts within the ESCAPE Project. <i>Environ Health Perspect.</i> 2014; 122(1): 107–13.
119	Mahalanabis D, Gupta S, Paul D, Gupta A, Lahiri M, Khaled MA. Risk factors for pneumonia in infants and young children and the role of solid fuel for cooking: a case-control study. <i>Epidemiol Infect.</i> 2002; 129(1): 65–71.
120	Melody S, Wills K, Knibbs LD, Ford J, Venn A, Johnston F. Adverse birth outcomes in Victoria, Australia in association with maternal exposure to low levels of ambient air pollution. <i>Environ Res.</i> 2020; 188: 109784.
121	Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD. Long-term exposure to air pollution and incidence of cardiovascular events in women. <i>N Engl J Med.</i> 2007; 356(5): 447-58.
122	Morello-Frosch R, Jesdale BM, Sadd JL, Pastor M. Ambient air pollution exposure and full-term birth weight in California. <i>Environ Health.</i> 2010; 9(1).
123	Naess Ø, Nafstad P, Aamodt G, Clausen B, Rosland P. Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhoods in Oslo, Norway. <i>Am J Epidemiol.</i> 2007; 165(4): 435-43.

124	Ng C, Malig B, Hasheminassab S, Sioutas C, Basu R, Ebisu K. Source apportionment of fine particulate matter and risk of term low birth weight in California: Exploring modification by region and maternal characteristics. <i>Sci Total Environ.</i> 2017; 605-606: 647-654.
125	Ostro B, Hu J, Goldberg D, et al. 2015. Associations of mortality with long-term exposures to fine and ultrafine particles, species and sources: results from the California Teachers Study Cohort. <i>Environmental Health Perspectives</i> , 123(6), 549-556.
126	Ottone M, Broccoli S, Parmagnani F, Giannini S, Scotto F, Bonvicini L, Luberto F, Bacco D, Trentini A, Poluzzi V, Angelini P, Colacci A, Giorgi Rossi P, Ranzi A. Source-related components of fine particulate matter and risk of adverse birth outcomes in Northern Italy. <i>Environ Res.</i> 2020; 186: 109564.
127	Park SK, Adar SD, O'Neill MS, Auchincloss AH, Szpiro A, Bertoni AG, Navas-Acien A, Kaufman JD, Diez-Roux AV. Long-term exposure to air pollution and type 2 diabetes mellitus in a multiethnic cohort. <i>Am J Epidemiol.</i> 2015; 181(5): 327–36.
128	Parker JD, Woodruff TJ, Basu R, Schoendorf KC. Air Pollution and Birth Weight Among Term Infants in California. <i>Pediatrics.</i> 2005; 115(1): 121–8.
129	Parker JD, Woodruff TJ. Influences of study design and location on the relationship between particulate matter air pollution and birthweight. <i>Paediatr Perinat Epidemiol.</i> 2008; 22(3): 214–27.
130	Parker JD, Kravets N, Vaidyanathan A. 2018. Particulate matter air pollution exposure and heart disease mortality risks by race and ethnicity in the United States: 1997 to 2009 National Health Interview Survey with mortality follow-up through 2011. <i>Circulation</i> , 137(16), 1688-1697.
131	Pedersen M, Giorgis-Allemand L, Bernard C, Aguilera I, Andersen AM, Ballester F, Beelen RM, Chatzi L, Cirach M, Danileviciute A, Dedele A, Eijdsden Mv, Estarlich M, Fernández-Somoano A, Fernández MF, Forastiere F, Gehring U, Grazuleviciene R, Gruzieva O, Heude B, Hoek G, de Hoogh K, van den Hooven EH, Håberg SE, Jaddoe VW, Klümper C, Korek M, Krämer U, Lerchundi A, Lepeule J, Nafstad P, Nystad W, Patelarou E, Porta D, Postma D, Raaschou-Nielsen O, Rudnai P, Sunyer J, Stephanou E, Sørensen M, Thiering E, Tuffnell D, Varró MJ, Vrijkotte TG, Wijga A, Wilhelm M, Wright J, Nieuwenhuijsen MJ, Pershagen G, Brunekreef B, Kogevinas M, Slama R. Ambient air pollution and low birthweight: a European cohort study (ESCAPE). <i>Lancet Respir Med.</i> 2013; 1(9): 695-704.
132	Pereira G, Belanger K, Ebisu K, Bell ML. Fine particulate matter and risk of preterm birth in Connecticut in 2000-2006: a longitudinal study. <i>Am J Epidemiol.</i> 2014; 179(1): 67-74.
133	Pereira G, Bell ML, Belanger K, de Klerk N. Fine particulate matter and risk of preterm birth and pre-labor rupture of membranes in Perth, Western Australia 1997-2007: a longitudinal study. <i>Environ Int.</i> 2014; 73: 143-9.
134	Pinault L, Tjepkema M, Crouse DL, Weichenthal S, van Donkelaar A, Martin RV, Brauer M, Chen H, Burnett RT. Risk estimates of mortality attributed to low

	concentrations of ambient fine particulate matter in the Canadian community health survey cohort [Unpublished]. <i>Environ Health</i> . 2016; 15: 18.
135	Pinault L, Tjepkema M, Crouse DL, Weichenthal S, van Donkelaar A, Martin RV, Brauer M, Chen H, Burnett RT. Risk estimates of mortality attributed to low concentrations of ambient fine particulate matter in the Canadian community health survey cohort. <i>Environ Health</i> . 2016; 15(1): 18.
136	Pinault L, Brauer M, Crouse DL, et al. 2018. Diabetes status and susceptibility to the effects of PM2.5 exposure on cardiovascular mortality in a National Canadian Cohort. <i>Epidemiology</i> , 29(6), 784-794.
137	Pope CA, Burnett R, Thun M, Calle E, Krewski D, Ito K, Thurston G. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. <i>JAMA</i> . 2002; 287(9): 1132-41.
138	Pope CA, Lefler JS, Ezzati M, et al. 2019. Mortality Risk and Fine Particulate Air Pollution in a Large, Representative Cohort of US Adults. <i>Environmental Health Perspectives</i> , 127(7), 077007.
139	Puett RC, Hart JE, Suh H, Mittleman M, Laden F. Particulate matter exposures, mortality, and cardiovascular disease in the health professionals follow-up study. <i>Environ Health Perspect</i> . 2011; 119(8): 1130-5.
140	Puett RC, Hart JE, Yanosky JD, Paciorek C, Schwartz J, Suh H, Speizer FE, Laden F. Chronic fine and coarse particulate exposure, mortality, and coronary heart disease in the Nurses' Health Study. <i>Environ Health Perspect</i> . 2009; 117(11): 1697-701.
141	Qian Z, Liang S, Yang S, Trevathan E, Huang Z, Yang R, Wang J, Hu K, Zhang Y, Vaughn M, Shen L, Liu W, Li P, Ward P, Yang L, Zhang W, Chen W, Dong G, Zheng T, Xu S, Zhang B. Ambient air pollution and preterm birth: A prospective birth cohort study in Wuhan, China. <i>Int J Hyg Environ Health</i> . 2016; 219(2): 195-203.
142	Qiu H, Schooling CM, Sun S, Tsang H, Yang Y, Lee RS, Wong CM, Tian L. Long-term exposure to fine particulate matter air pollution and type 2 diabetes mellitus in elderly: A cohort study in Hong Kong. <i>Environ Int</i> . 2018; 113: 350-56.
143	Qiu H, Sun S, Tsang H, Wong CM, Lee RS, Schooling CM, Tian L. Fine particulate matter exposure and incidence of stroke: A cohort study in Hong Kong. <i>Neurology</i> . 2017; 88(18): 1709-1717.
144	Raaschou-Nielsen O, Andersen ZJ, Beelen R, Samoli E, Stafoggia M, Weinmayr G, Hoffmann B, Fischer P, Nieuwenhuijsen MJ, Brunekreef B, Xun WW, Katsouyanni K, Dimakopoulou K, Sommar J, Forsberg B, Modig L, Oudin A, Oftedal B, Schwarze PE, Nafstad P, De Faire U, Pedersen NL, Ostenson C-G, Fratiglioni L, Penell J, Korek M, Pershagen G, Eriksen KT, Sørensen M, Tjønneland A, Ellermann T, Eeftens M, Peeters PH, Meliefste K, Wang M, Bueno-de-Mesquita B, Key TJ, de Hoogh K, Concin H, Nagel G, Vilier A, Grioni S, Krogh V, Tsai M-Y, Ricceri F, Sacerdote C, Galassi C, Migliore E, Ranzi A, Cesaroni G, Badaloni C, Forastiere F, Tamayo I, Amiano P, Dorronsoro M, Trichopoulou A, Bamia C, Vineis P, Hoek G. Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE). <i>Lancet Oncol</i> . 2013; 14(9): 813-22.
145	Renzi M, Cerza F, Gariazzo C, et al. Air pollution and occurrence of type 2 diabetes in a large cohort study. <i>Environ Int</i> . 2018, 112: 68-76.

146	Robin LF, Less PS, Winget M, Steinhoff M, Moulton LH, Santosham M, Correa A. Wood-burning stoves and lower respiratory illnesses in Navajo children. <i>Pediatr Infect Dis J</i> . 1996; 15(10): 859–65.
147	Sapkota A, Gajalakshmi V, Jetly DH, Roychowdhury S, Dikshit RP, Brennan P, Hashibe M, Boffetta P. Indoor air pollution from solid fuels and risk of hypopharyngeal/laryngeal and lung cancers: a multicentric case-control study from India. <i>Int J Epidemiol</i> . 2008; 37(2): 321–8.
148	Sasco AJ, Merrill RM, Dari I, Benhaïm-Luzon V, Carriot F, Cann CI, Bartal M. A case-control study of lung cancer in Casablanca, Morocco. <i>Cancer Causes Control</i> . 2002; 13(7): 609–16.
149	Savitha MR, Nandeeshwara SB, Pradeep Kumar MJ, ul-Haque F, Raju CK. Modifiable risk factors for acute lower respiratory tract infections. <i>Indian J Pediatr</i> . 2007; 74(5): 477–82.
150	Savitz DA, Bobb JF, Carr JL, Clougherty JE, Dominici F, Elston B, Ito K, Ross Z, Yee M, Matte TD. Ambient Fine Particulate Matter, Nitrogen Dioxide, and Term Birth Weight in New York, New York. <i>Am J Epidemiol</i> . 2014; 179(4): 457–66.
151	Schembari A, de Hoogh K, Pedersen M, Dadvand P, Martinez D, Hoek G, Petherick ES, Wright J, Nieuwenhuijsen MJ. Ambient Air Pollution and Newborn Size and Adiposity at Birth: Differences by Maternal Ethnicity (the Born in Bradford Study Cohort). <i>Environ Health Perspect</i> . 2015; 123(11): 1208-15.
152	Sezer H, Akkurt I, Guler N, Marako?lu K, Berk S. A case-control study on the effect of exposure to different substances on the development of COPD. <i>Ann Epidemiol</i> . 2006; 16(1): 59–62.
153	Shah N, Ramankutty V, Premila PG, Sathy N. Risk factors for severe pneumonia in children in south Kerala: a hospital-based case-control study. <i>J Trop Pediatr</i> . 1994; 40(4): 201–6.
154	Shang L, Huang L, Yang L, Leng L, Qi C, Xie G, Wang R, Guo L, Yang W, Chung MC. Impact of air pollution exposure during various periods of pregnancy on term birth weight: a large-sample, retrospective population-based cohort study. <i>Environ Sci Pollut Res Int</i> . 2021; 28(3): 3296-3306.
155	Shen M, Chapman RS, Vermeulen R, Tian L, Zheng T, Chen BE, Engels EA, He X, Blair A, Lan Q. Coal use, stove improvement, and adult pneumonia mortality in Xuanwei, China: a retrospective cohort study. <i>Environ Health Perspect</i> . 2009; 117(2): 261–6.
156	Sheridan P, Ilango S, Bruckner TA, Wang Q, Basu R, Benmarhnia T. Ambient Fine Particulate Matter and Preterm Birth in California: Identification of Critical Exposure Windows. <i>Am J Epidemiol</i> . 2019; 188(9): 1608-1615.
157	Siddiqui AR, Gold EB, Yang X, Lee K, Brown KH, Bhutta ZA. Prenatal exposure to wood fuel smoke and low birth weight. <i>Environ Health Perspect</i> . 2008; 116(4): 543-9.
158	Smith KR, McCracken JP, Weber MW, Hubbard A, Jenny A, Thompson LM, Balmes J, Díaz A, Arana B, Bruce N. Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. <i>Lancet</i> . 2011; 378(9804): 1717-26.
159	Smith RB, Fecht D, Gulliver J, Beevers SD, Dajnak D, Blangiardo M, Ghosh RE, Hansell AL, Kelly FJ, Anderson HR, Toledano MB. Impact of London’s road traffic

	air and noise pollution on birth weight: retrospective population based cohort study. <i>BMJ</i> . 2017; 359: j5299.
160	Stafoggia M, Cesaroni G, Peters A, Andersen ZJ, Badaloni C, Beelen R, Caracciolo B, Cyrus J, de Faire U, de Hoogh K, Eriksen KT, Fratiglioni L, Galassi C, Gigante B, Havulinna AS, Hennig F, Hilding A, Hoek G, Hoffmann B, Houthuijs D, Korek M, Lanki T, Leander K, Magnusson PK, Meisinger C, Migliore E, Overvad K, Ostenson C-G, Pedersen NL, Pekkanen J, Penell J, Pershagen G, Pundt N, Pyko A, Raaschou-Nielsen O, Ranzi A, Ricceri F, Sacerdote C, Swart WJR, Turunen AW, Vineis P, Weimar C, Weinmayr G, Wolf K, Brunekreef B, Forastiere F. Long-term exposure to ambient air pollution and incidence of cerebrovascular events: results from 11 European cohorts within the ESCAPE project. <i>Environ Health Perspect</i> . 2014; 122(9): 919–25.
161	Starling AP, Moore BF, Thomas DSK, Peel JL, Zhang W, Adgate JL, Magzamen S, Martenies SE, Allshouse WB, Dabelea D. Prenatal exposure to traffic and ambient air pollution and infant weight and adiposity: The Healthy Start study. <i>Environ Res</i> . 2020; 182: 109130.
162	Stieb DM, Chen L, Beckerman BS, Jerrett M, Crouse DL, Omariba DW, Peters PA, van Donkelaar A, Martin RV, Burnett RT, Gilbert NL, Tjepkema M, Liu S, Dugandzic RM. Associations of Pregnancy Outcomes and PM2.5 in a National Canadian Study. <i>Environ Health Perspect</i> . 2016; 124(2): 243-9.
163	Strickland MJ, Lin Y, Darrow LA, Warren JL, Mulholland JA, Chang HH. Associations Between Ambient Air Pollutant Concentrations and Birth Weight: A Quantile Regression Analysis. <i>Epidemiology</i> . 2019; 30(5): 624-632.
164	Sun Z, Yang L, Bai X, Du W, Shen G, Fei J, Wang Y, Chen A, Chen Y, Zhao M. Maternal ambient air pollution exposure with spatial-temporal variations and preterm birth risk assessment during 2013-2017 in Zhejiang Province, China. <i>Environ Int</i> . 2019; 133(Pt B): 105242.
165	Tapia VL, Vasquez BV, Vu B, Liu Y, Steenland K, Gonzales GF. Association between maternal exposure to particulate matter (PM2.5) and adverse pregnancy outcomes in Lima, Peru. <i>J Expo Sci Environ Epidemiol</i> . 2020; 30(4): 689-697.
166	Thompson LM, Bruce N, Eskenazi B, Diaz A, Pope D, Smith KR. Impact of reduced maternal exposures to wood smoke from an introduced chimney stove on newborn birth weight in rural Guatemala. <i>Environ Health Perspect</i> . 2011; 119(10): 1489-94.
167	Thurston GD, Ahn J, Cromar KR, Shao Y, Reynolds HR, Jerrett M, Lim CC, Shanley R, Park Y, Hayes RB. Ambient Particulate Matter Air Pollution Exposure and Mortality in the NIH-AARP Diet and Health Cohort [Unpublished]. <i>Environ Health Perspect</i> . 2016; 124(4): 484-90.
168	Tielsch JM, Katz J, Thulasiraj RD, Coles CL, Sheeladevi S, Yanik EL, Rahmathullah L. Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in south India. <i>Int J Epidemiol</i> . 2009; 38(5): 1351-63.
169	To T, Zhu J, Villeneuve PJ, Simatovic J, Feldman L, Gao C, Williams D, Chen H, Weichenthal S, Wall C, Miller AB. Chronic disease prevalence in women and air pollution--A 30-year longitudinal cohort study. <i>Environ Int</i> . 2015; 80: 26–32.

170	Tseng E, Ho W-C, Lin M-H, Cheng T-J, Chen P-C, Lin H-H. Chronic exposure to particulate matter and risk of cardiovascular mortality: cohort study from Taiwan. <i>BMC Public Health</i> . 2015; 15: 936.
171	Turner MC, Jerrett M, Pope CA 3rd, Krewski D, Gapstur SM, Diver WR, Beckerman BS, Marshall JD, Su J, Crouse DL, Burnett RT. Long-term ozone exposure and mortality in a large prospective study. <i>Am J Respir Crit Care Med</i> . 2016; 193(10): 1134-42.
172	Turner MC, Krewski D, Pope CA, et al. 2011. Long-term ambient fine particulate matter air pollution and lung cancer in a large cohort of never-smokers. <i>American Journal of Respiratory and Critical Care Medicine</i> , 184(12), 1374-1381.
173	Victoria CG, Fuchs SC, Flores JA, Fonseca W, Kirkwood B. Risk factors for pneumonia among children in a Brazilian metropolitan area. <i>Pediatrics</i> . 1994; 977-85.
174	Villeneuve PJ, Weichenthal SA, Crouse D, Miller AB, To T, Martin RV, van Donkelaar A, Wall C, Burnett RT. Long-term exposure to fine particulate matter air pollution and mortality among Canadian women. <i>Epidemiology</i> . 2015; 26(4): 536-45.
175	Wang Q, Benmarhnia T, Zhang H, Knibbs LD, Sheridan P, Li C, Bao J, Ren M, Wang S, He Y, Zhang Y, Zhao Q, Huang C. Identifying windows of susceptibility for maternal exposure to ambient air pollution and preterm birth. <i>Environ Int</i> . 2018; 121(Pt 1): 317-324.
176	Wayse V, Yousafzai A, Mogale K, Filteau S. Association of subclinical vitamin D deficiency with severe acute lower respiratory infection in Indian children under 5 y. <i>Eur J Clin Nutr</i> . 2004; 58(4): 563-7.
177	Weichenthal S, Villeneuve PJ, Burnett RT, van Donkelaar A, Martin RV, Jones RR, DellaValle CT, Sandler DP, Ward MH, Hoppin JA. Long-term exposure to fine particulate matter: association with nonaccidental and cardiovascular mortality in the agricultural health study cohort. <i>Environ Health Perspect</i> . 2014; 122(6): 609-15.
178	Weinmayr G, Hennig F, Fuks K, Nonnemacher M, Jakobs H, Möhlenkamp S, Erbel R, Jöckel K-H, Hoffmann B, Moebus S, Heinz Nixdorf Recall Investigator Group. Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution. <i>Environ Health</i> . 2015; 14: 53.
179	Wesley AG, Loening WE. Assessment and 2-year follow-up of some factors associated with severity of respiratory infections in early childhood. <i>S Afr Med J</i> . 1996; 86(4): 365-8.
180	Wilhelm M, Ghosh JK, Su J, Cockburn M, Jerrett M, Ritz B. Traffic-related air toxics and preterm birth: a population-based case-control study in Los Angeles County, California. <i>Environ Health</i> . 2011; 10: 89.
181	Wong CM, Lai HK, Tsang H, Thach TQ, Thomas GN, Lam KBH, Chan KP, Yang L, Lau AKH, Ayres JG, Lee SY, Man Chan W, Hedley AJ, Lam TH. Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents. <i>Environ Health Perspect</i> . 2015; 123(11): 1167-72.
182	Wu AH, Henderson BE, Pike MC, Yu MC. Smoking and other risk factors for lung cancer in women. <i>J Natl Cancer Inst</i> . 1985; 74(4): 747-51.

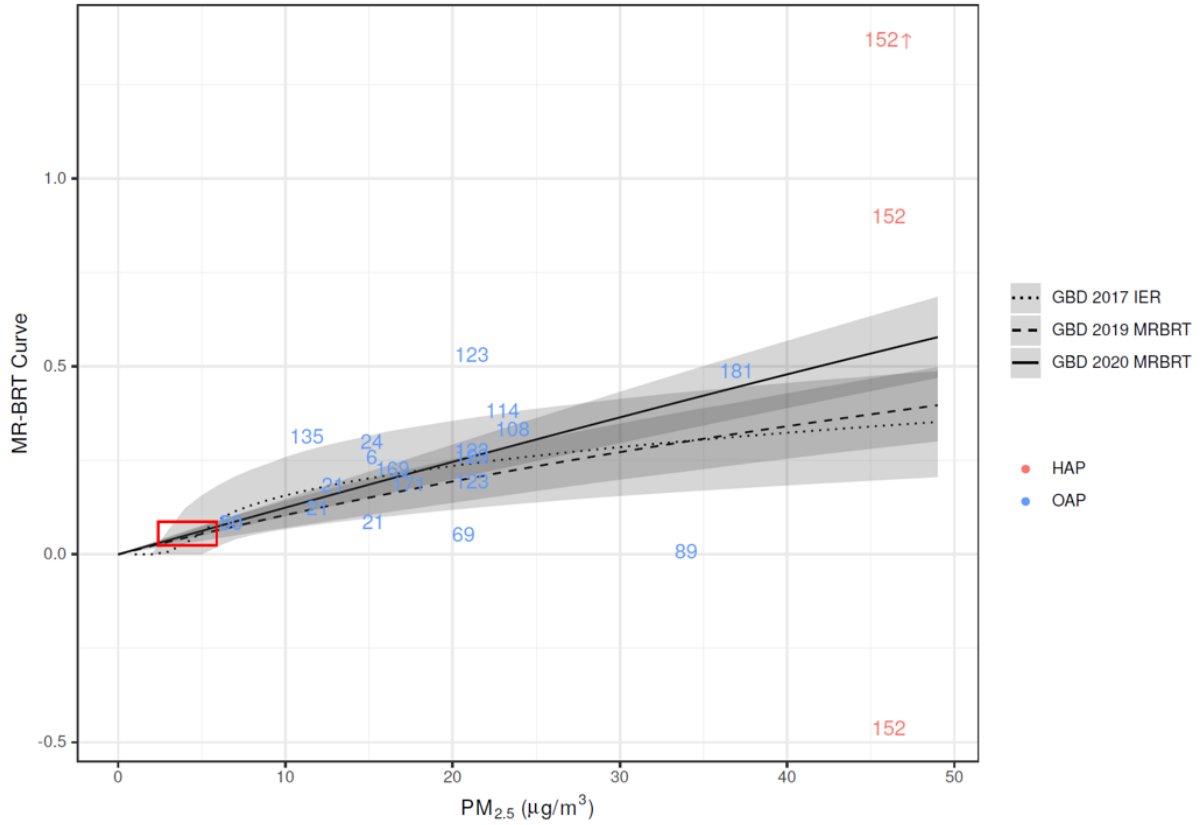
183	Wu J, Wilhelm M, Chung J, Ritz B. Comparing exposure assessment methods for traffic-related air pollution in an adverse pregnancy outcome study. <i>Environ Res.</i> 2011; 111(5): 685-92.
184	Wu H, Jiang B, Geng X, Zhu P, Liu Z, Cui L, Yang L. Exposure to fine particulate matter during pregnancy and risk of term low birth weight in Jinan, China, 2014-2016. <i>Int J Hyg Environ Health.</i> 2018; 221(2): 183-190.
185	Wylie BJ, Coull BA, Hamer DH, Singh MP, Jack D, Yeboah-Antwi K, Sabin L, Singh N, MacLeod WB. Impact of biomass fuels on pregnancy outcomes in central East India. <i>Environ Health.</i> 2014; 13(1): 1.
186	Wylie BJ, Kishashu Y, Matechi E, Zhou Z, Coull B, Abioye AI, Dionisio KL, Mugusi F, Premji Z, Fawzi W, Hauser R, Ezzati M. Maternal exposure to carbon monoxide and fine particulate matter during pregnancy in an urban Tanzanian cohort. <i>Indoor Air.</i> 2017; 27(1): 136-146.
187	Xiao Q, Chen H, Strickland MJ, Kan H, Chang HH, Klein M, Yang C, Meng X, Liu Y. Associations between birth outcomes and maternal PM2.5 exposure in Shanghai: A comparison of three exposure assessment approaches. <i>Environ Int.</i> 2018; 117: 226-236.
188	Ye L, Ji Y, Lv W, Zhu Y, Lu C, Xu B, Xia Y. Associations between maternal exposure to air pollution and birth outcomes: a retrospective cohort study in Taizhou, China. <i>Environ Sci Pollut Res Int.</i> 2018; 25(22): 21927-21936.
189	Yin P, Brauer M, Cohen A, Burnett RT, Liu J, Liu Y, Liang R, Wang W, Qi J, Wang L, Zhou M. Long-term Fine Particulate Matter Exposure and Nonaccidental and Cause-specific Mortality in a Large National Cohort of Chinese Men [Unpublished]. <i>Environ Health Perspect.</i> 2017; 125(11): 117002.
190	Yin P, Brauer M, Cohen A, Burnett RT, Liu J, Liu Y, Liang R, Wang W, Qi J, Wang L, Zhou M. Long-term Fine Particulate Matter Exposure and Nonaccidental and Cause-specific Mortality in a Large National Cohort of Chinese Men. <i>Environ Health Perspect.</i> 2017; 125(11): 117002.
191	Yu K, Qiu G, Chan K-H, Lam K-BH, Kurmi OP, Bennett DA, Yu C, Pan A, Lv J, Guo Y, Bian Z, Yang L, Chen Y, Hu FB, Chen Z, Li L, Wu T. Association of Solid Fuel Use With Risk of Cardiovascular and All-Cause Mortality in Rural China. <i>JAMA.</i> 2018; 319(13): 1351–61.
192	Yuan L, Zhang Y, Wang W, Chen R, Liu Y, Liu C, Kan H, Gao Y, Tian Y, Shanghai Birth Cohort Study. Critical windows for maternal fine particulate matter exposure and adverse birth outcomes: The Shanghai birth cohort study. <i>Chemosphere.</i> 2020; 240: 124904.
193	Yucra S, Tapia V, Steenland K, Naeher LP, Gonzales GF. Association between biofuel exposure and adverse birth outcomes at high altitudes in Peru: a matched case-control study. <i>Int J Occup Environ Health.</i> 2011; 17(4): 307-13.

The following figures display risk curves for each outcome. The dashed line depicts the GBD 2017 IER including active smoking data, the dotted line depicts the GBD 2019 MR-BRT curve without active smoking but with secondhand smoking data, and the solid line depicts the GBD 2020 MR-BRT curve without the inclusion of active smoking or secondhand smoking data. For GBD 2020, a single curve is used for cardiovascular diseases (ischaemic heart disease, stroke) for all ages, so only one plot is displayed for each of these outcomes. For the GBD 2017 and GBD 2020 curves, the curve for the age group 60–64 is plotted for the cardiovascular disease outcomes because these cycles used age-

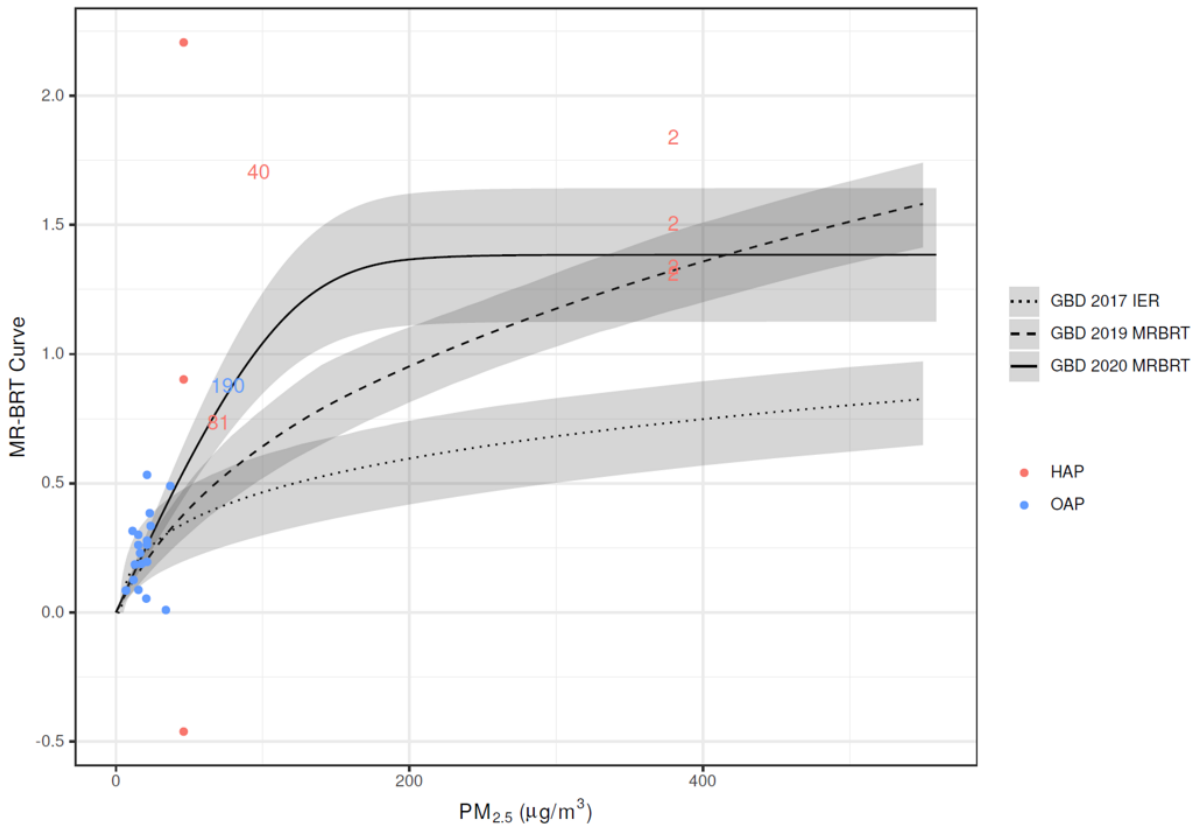
specific cardiovascular disease curves. For birthweight and gestational age, no curve is displayed for GBD 2017 because these outcomes were added to the GBD in the 2019 cycle. The grey shaded areas represent the 95% CI. The red box represents the TMREL area of the curve. On each page, the first figure depicts the typical range of outdoor exposure, whereas the second plot includes higher levels typical of household air pollution exposure.

Each point or number represents one study effect size. Each is plotted at the 95th percentile of the exposure distribution (OAP) or the expected level of exposure for individual using solid fuel (HAP). The relative risk is plotted relative to the predicted relative risk at the 5th percentile of exposure distribution (OAP) or the expected (ambient only) level of exposure for individuals not using solid fuel (HAP). For example, a study predicting a relative risk of 1.5 for an exposure range of 10 to 20 would be plotted at $(20, MRBRT(10)*1.5)$. Arrows represent studies that would have been outside the range of the plot but have been shifted to be included in the figure.

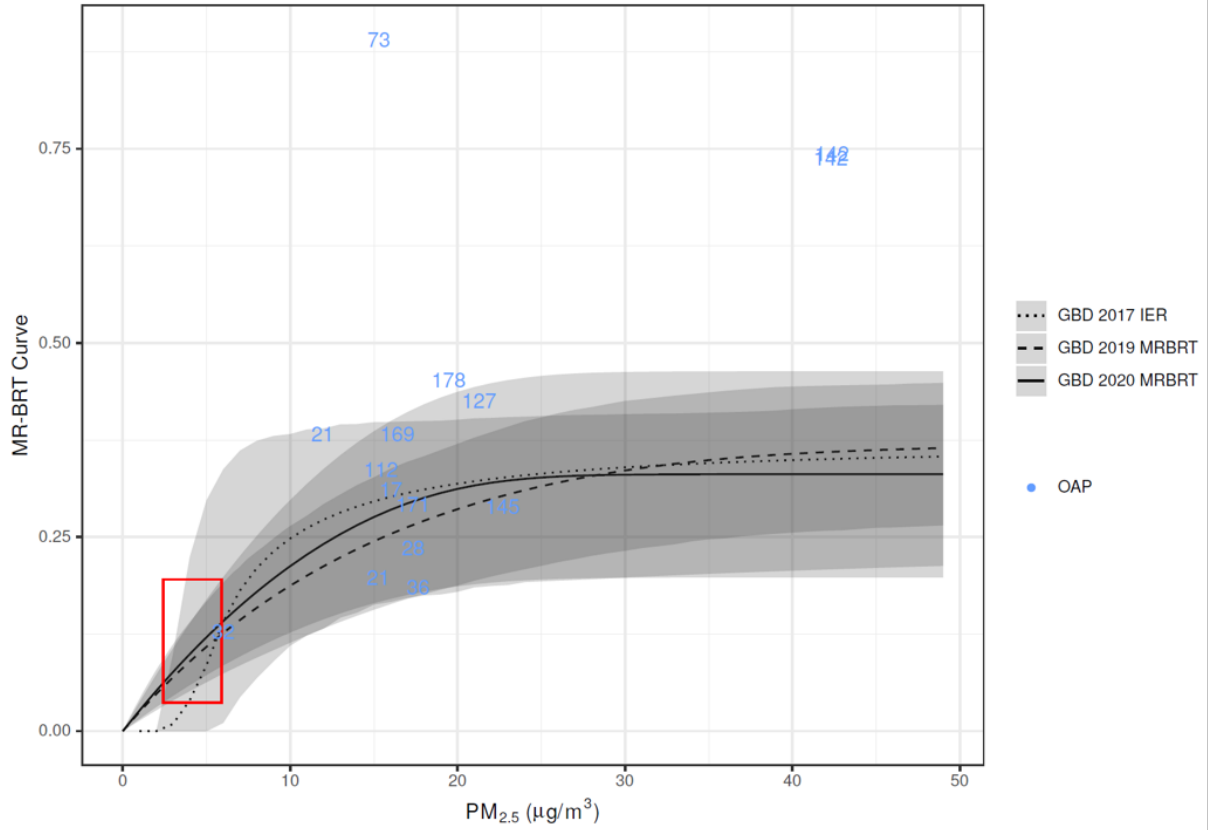
COPD, Low Exposure Range



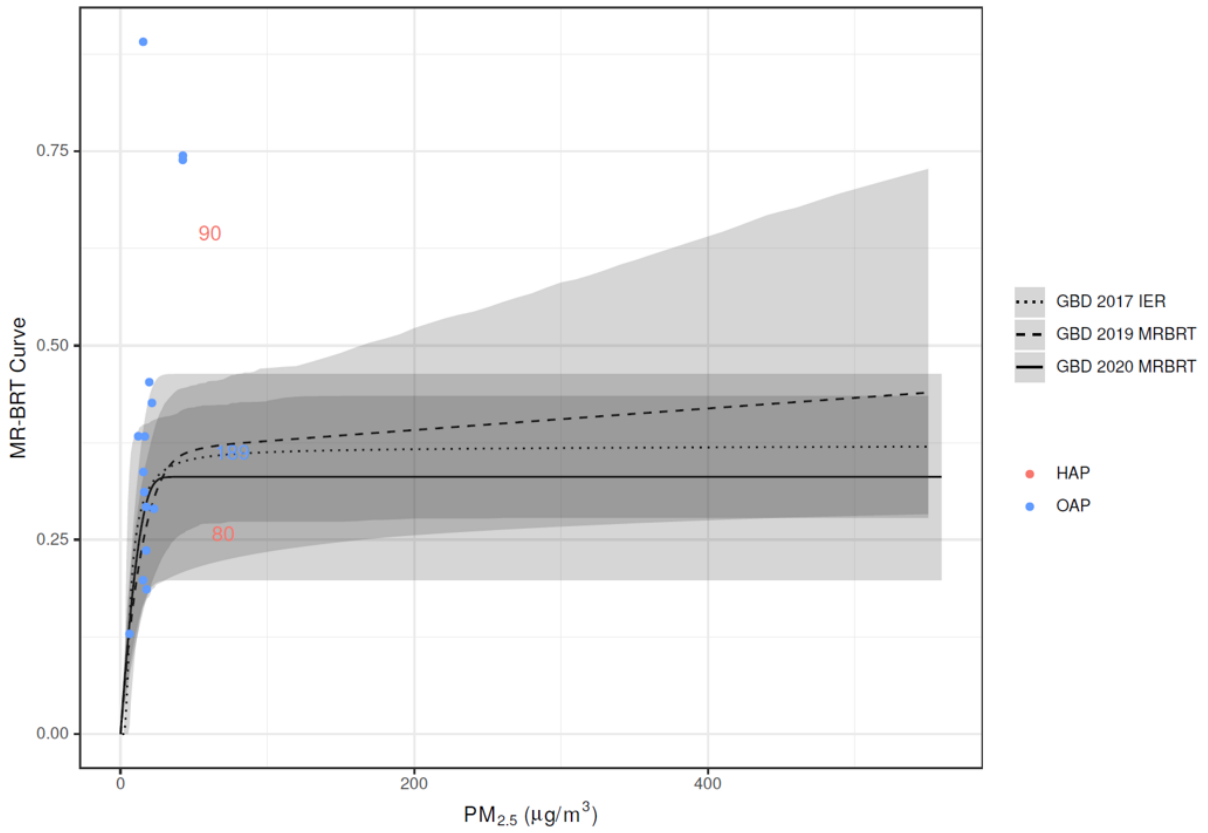
COPD, Full Exposure Range



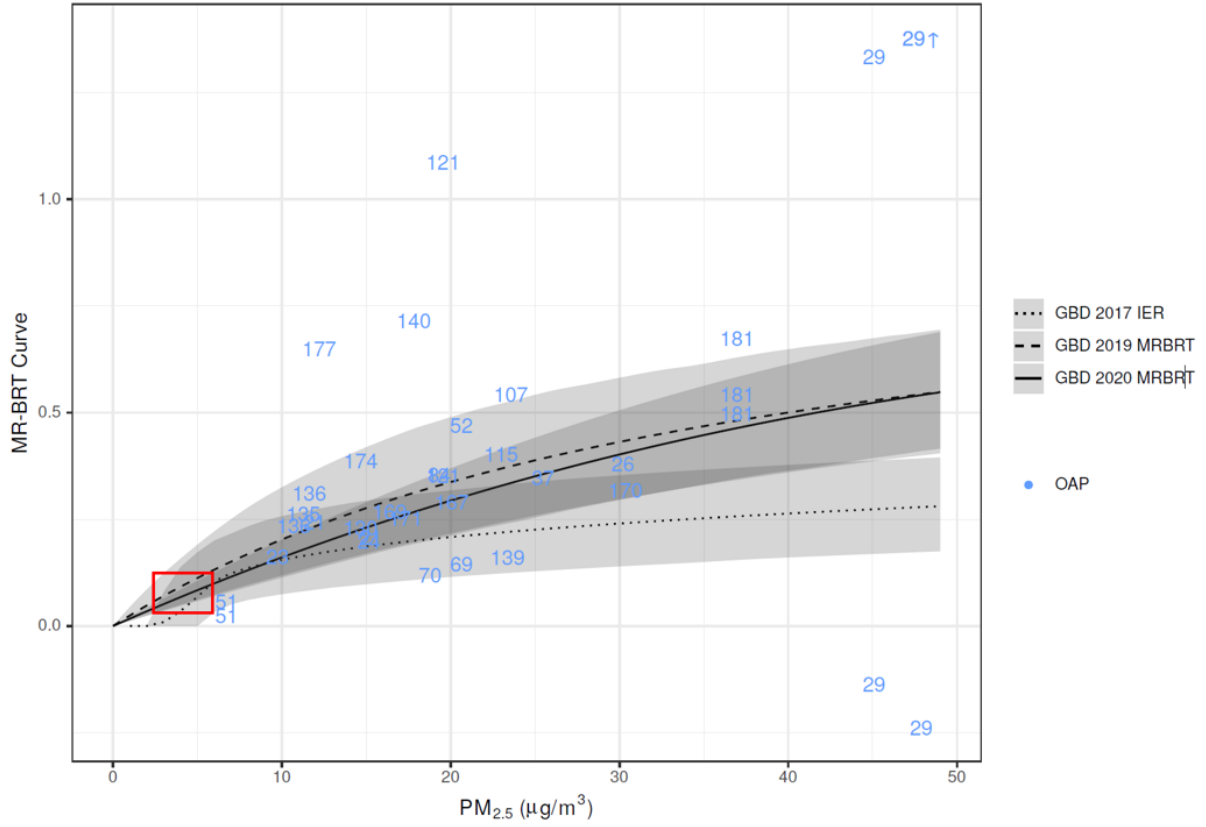
Type 2 Diabetes, Low Exposure Range



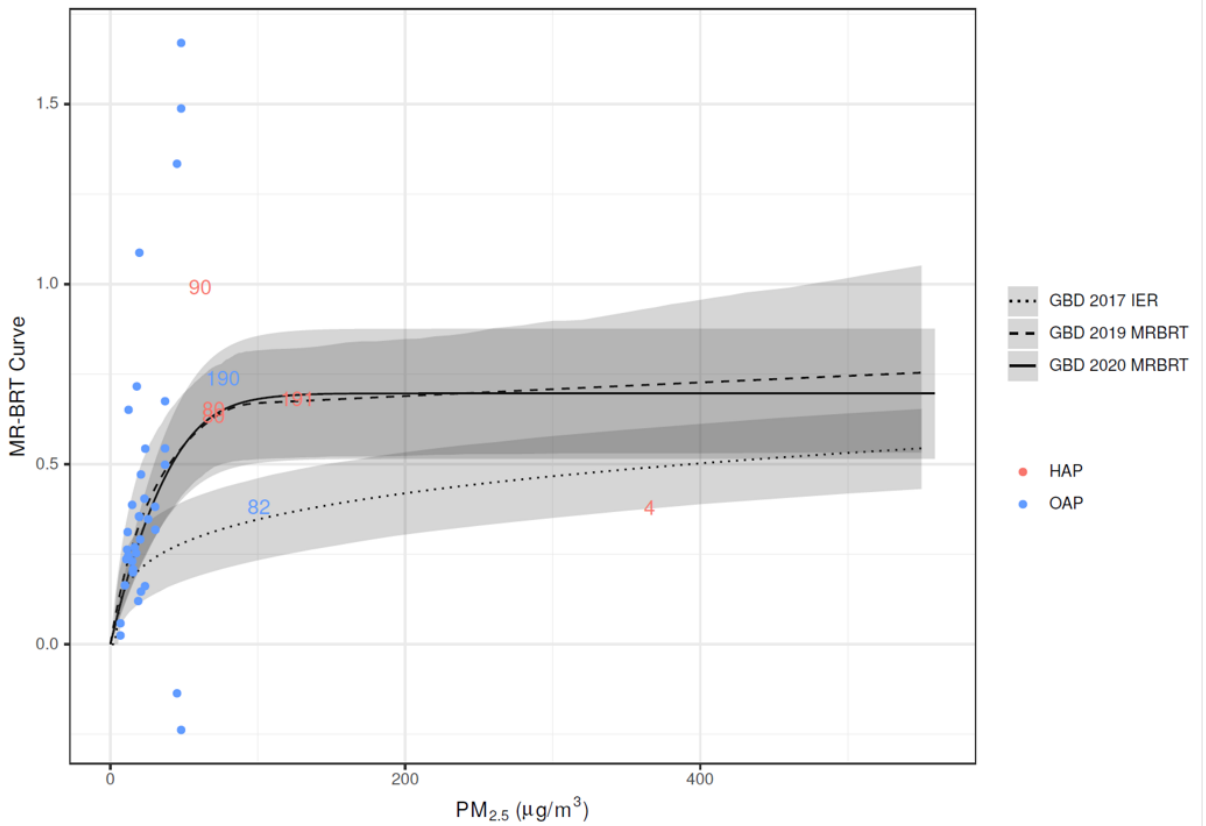
Type 2 Diabetes, Full Exposure Range



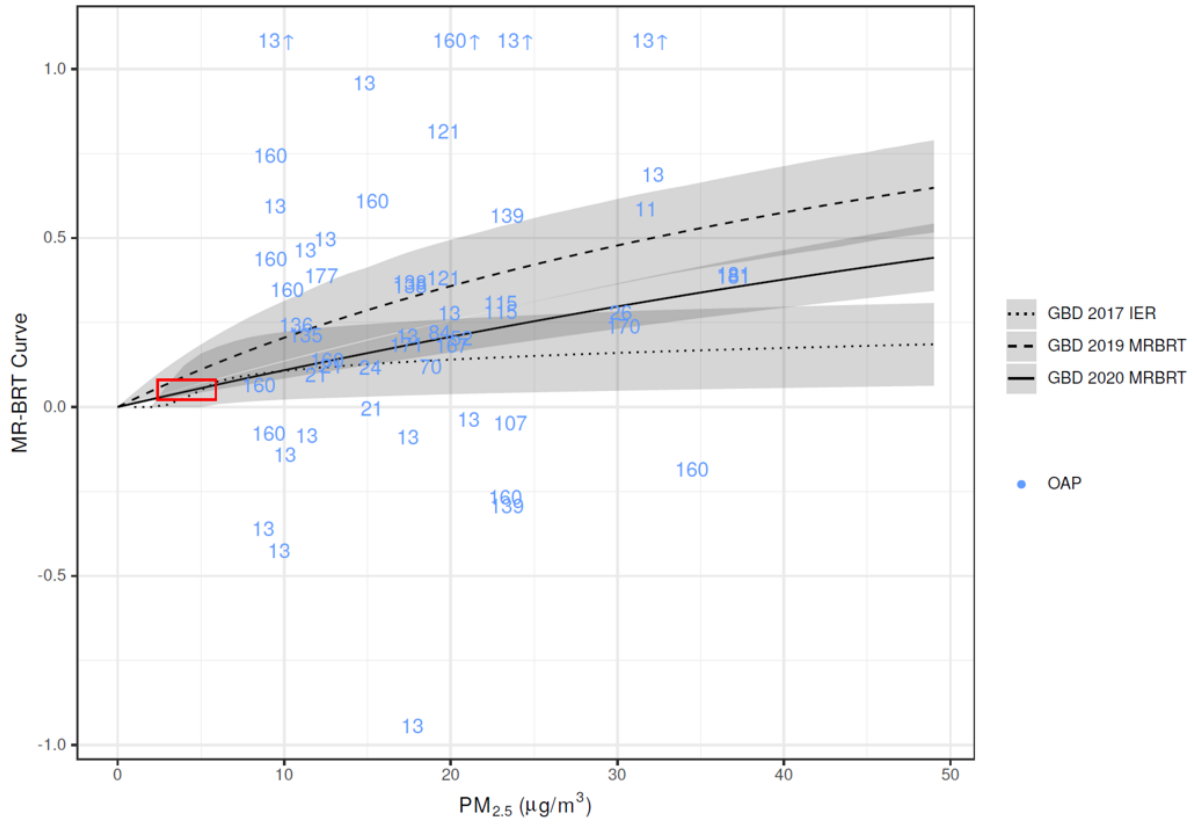
Ischemic Heart Disease, Low Exposure Range



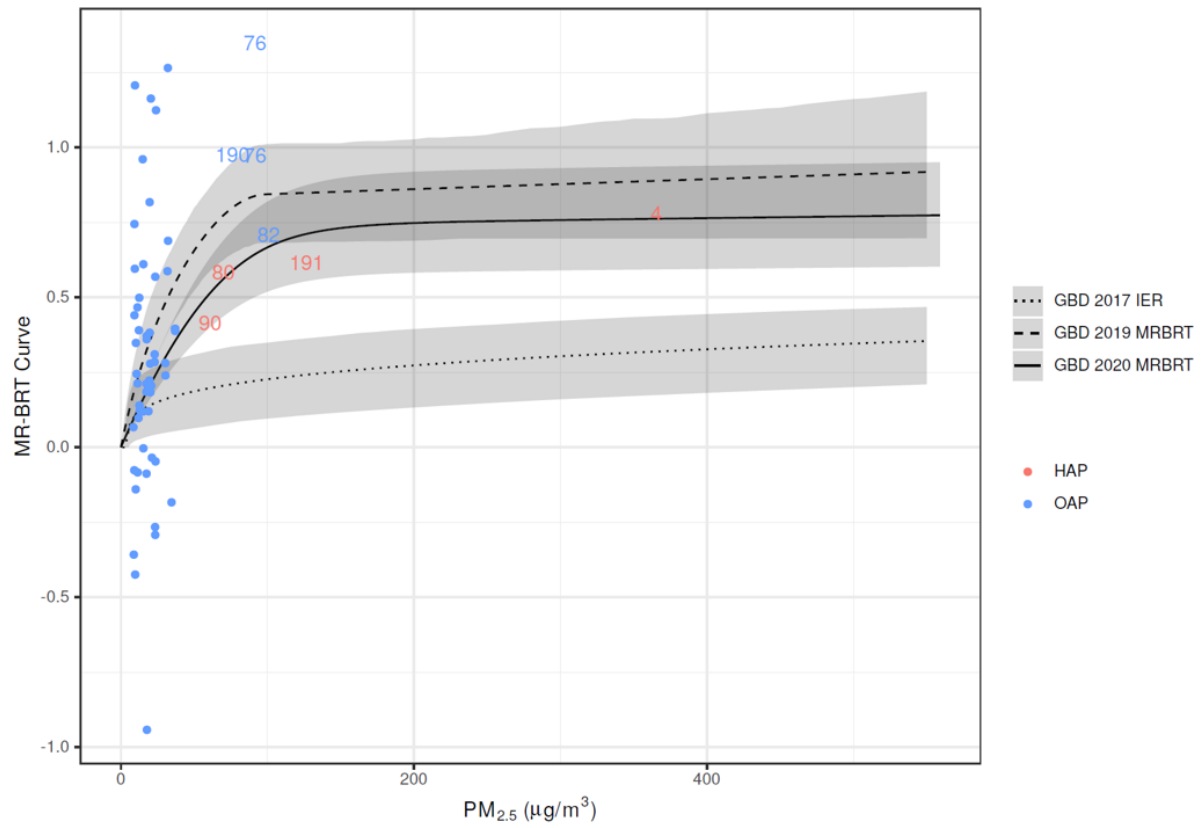
Ischemic Heart Disease, Full Exposure Range



Stroke, Low Exposure Range



Stroke, Full Exposure Range



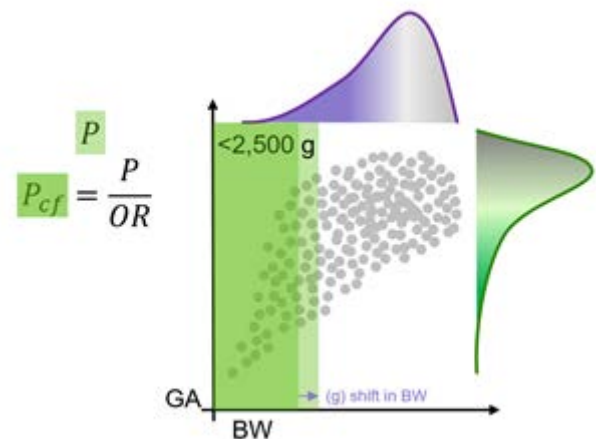
Low birthweight and short gestation mediation analysis

As in GBD 2019, in GBD 2020 low birthweight and short gestation were included as PM_{2.5} outcomes via a mediation analysis. Low birthweight and short gestation includes mortality due to diarrhoeal diseases, lower respiratory infections, upper respiratory infections, otitis media, meningitis, encephalitis, neonatal preterm birth, neonatal encephalopathy due to birth asphyxia and trauma, neonatal sepsis and other neonatal infections, haemolytic disease and other neonatal jaundice, and other neonatal disorders. Morbidity estimates were also calculated for neonatal preterm birth. These outcomes are specific to the neonatal ages: 0–6 days and 7–27 days.

The following is a summary of methods used to conduct the mediation analysis. For GBD 2019, we conducted a systematic review of all cohort, case-control, or randomised-controlled trial studies of ambient PM_{2.5} pollution or household air pollution and birthweight or gestational age outcomes for GBD 2019.¹⁸ Outcomes measured included continuous birthweight (bw), continuous gestational age (ga), low birthweight (LBW) (<2500 g), preterm birth (PTB) (<37 weeks), and very preterm birth (VPTB) (<32 weeks). We included any papers published until April 4, 2021.

Birthweight and gestational age are modelled using a continuous joint distribution for the GBD. To determine how these distributions are influenced by PM_{2.5} pollution, we used available literature to model the continuous shift in birthweight (bw, grams) and gestational age (ga, weeks) at a given PM_{2.5} exposure level. When available, we used estimates of continuous shifts in bw or ga directly from each study. When shifts were not available, we converted the published OR/RR/HR for LBW, PTB, or VPTB using the following strategy:

1. Extract the OR/RR/HR from the study.
2. Select the GBD 2017 estimated bw-ga joint distribution for the study location and year.
3. Calculate the number of grams or weeks required to shift the distribution such that the proportion of births under the specified threshold (P) is reduced by the study effect size to a counterfactual level (P_{cf}).
4. Save the resulting shift and 95% CI as the continuous effect.



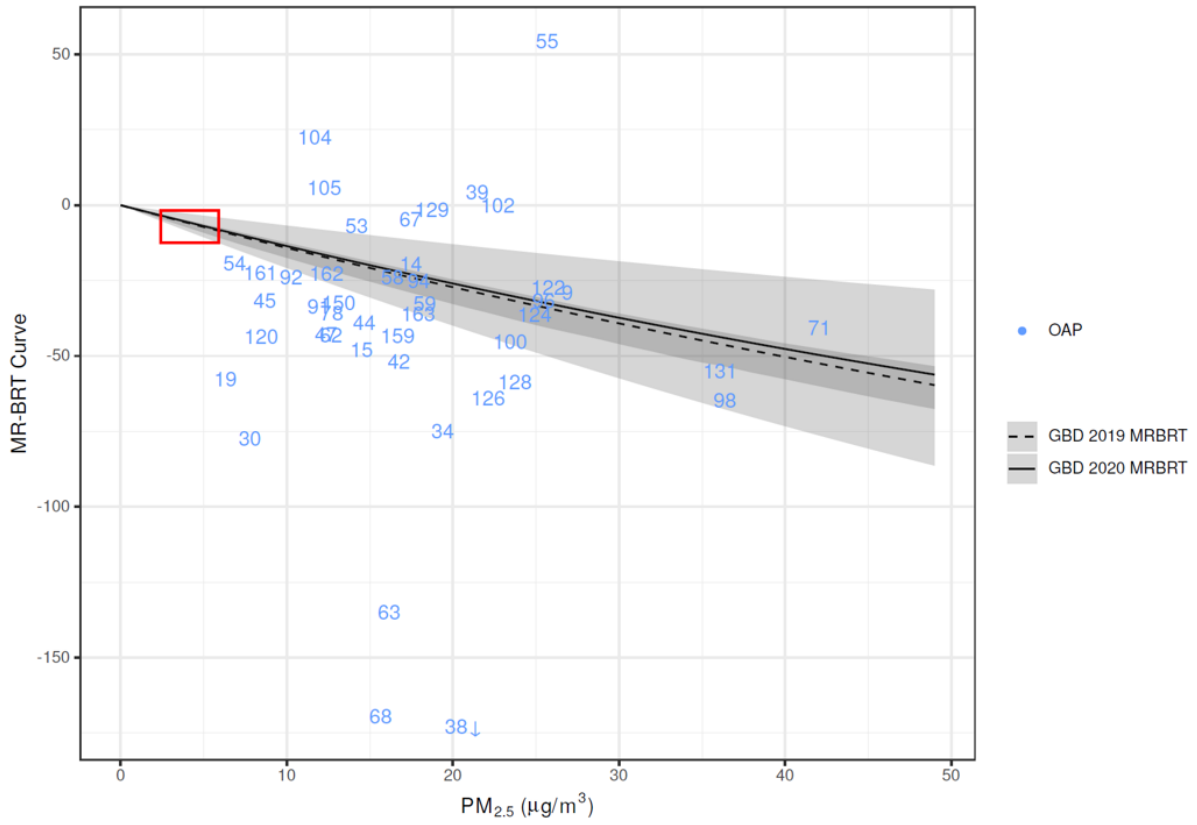
When preparing HAP data to fit splines, we used the same strategy described above for other outcomes to map HAP input data to PM_{2.5} exposure values. We then fit MR-BRT splines to the input studies, where the difference in the value of the model at the upper concentration (X) and the value of the model at the counterfactual concentration (X_{CF}) is equal to the published or calculated shift in bw or ga:

$$MRBRT(X) - MRBRT(X_{CF}) \sim Shift$$

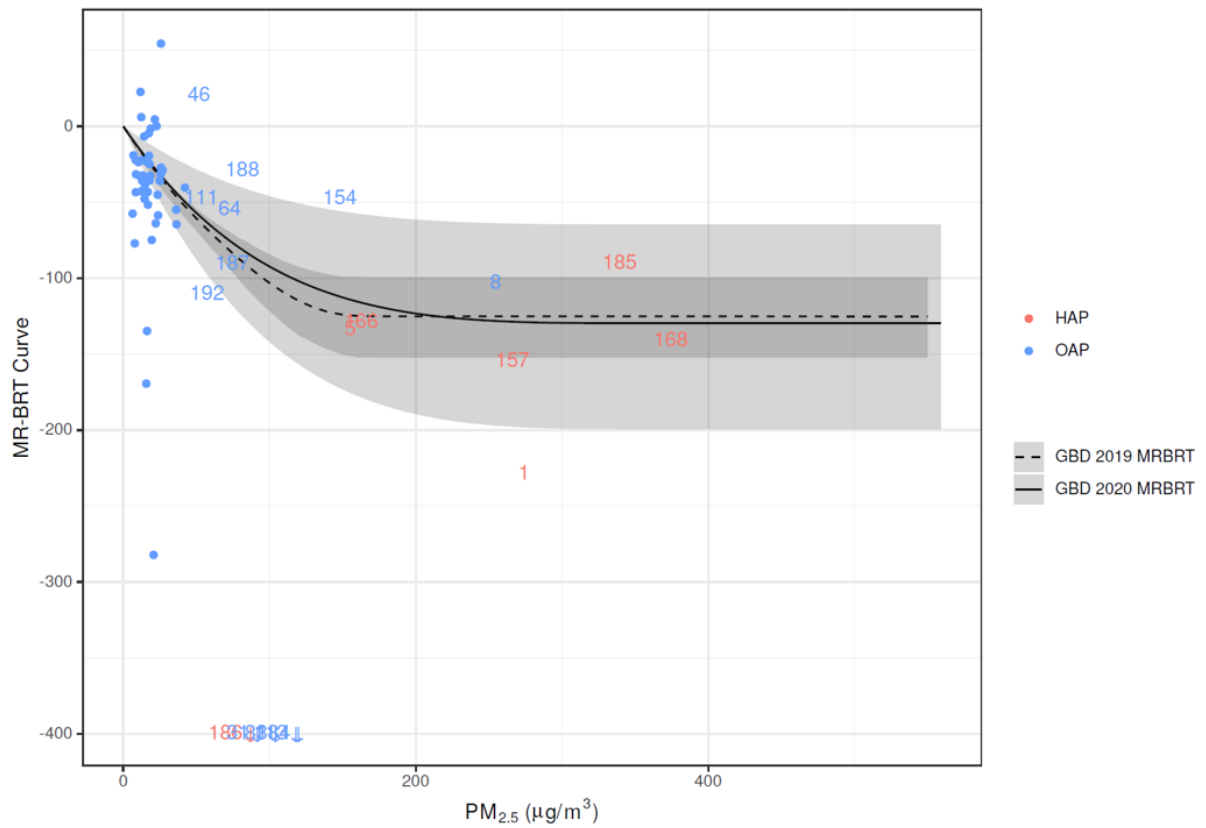
We used the same model fitting process, settings, and covariate selection process as described above for the other outcomes. The only exception is that, because the change in birthweight and gestational age was expected to be negative, the splines were constrained to be monotonically decreasing.

The following figures display MR-BRT curves for linear shift in grams (bw) and weeks (ga).

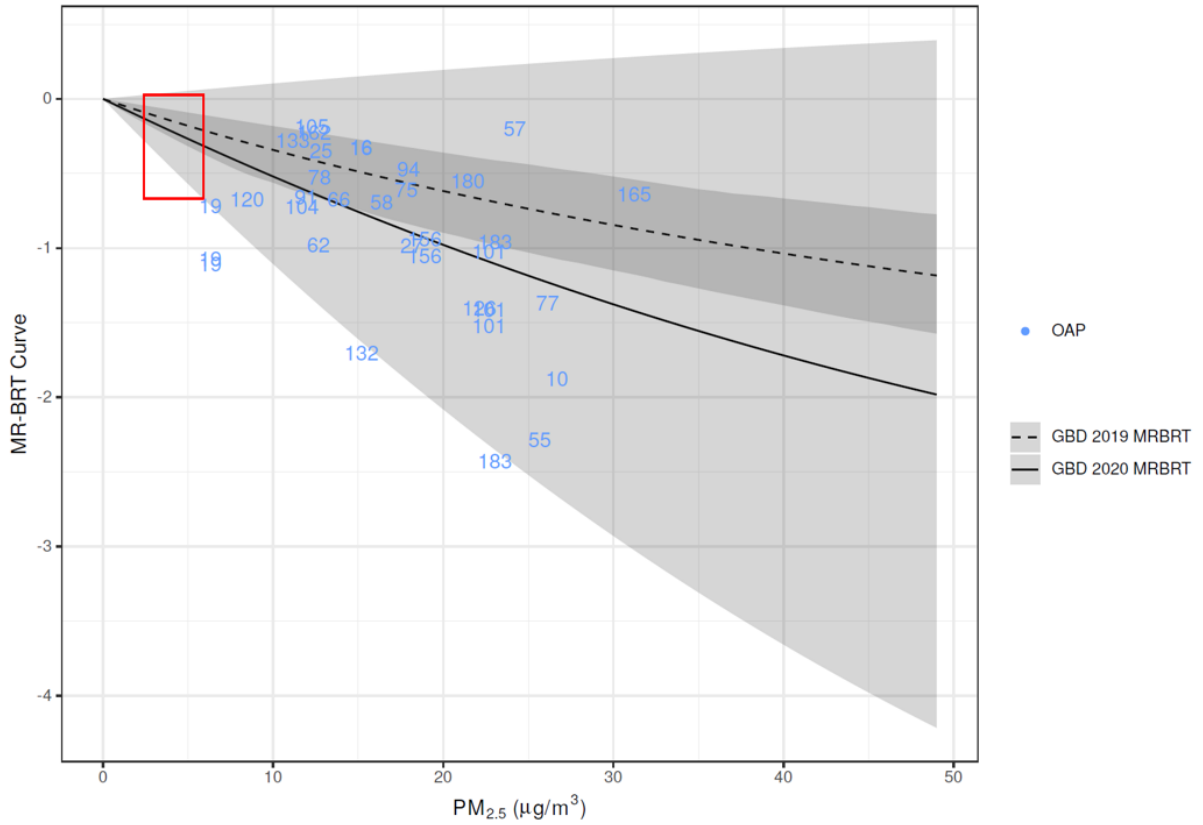
Birthweight (g), Low Exposure Range



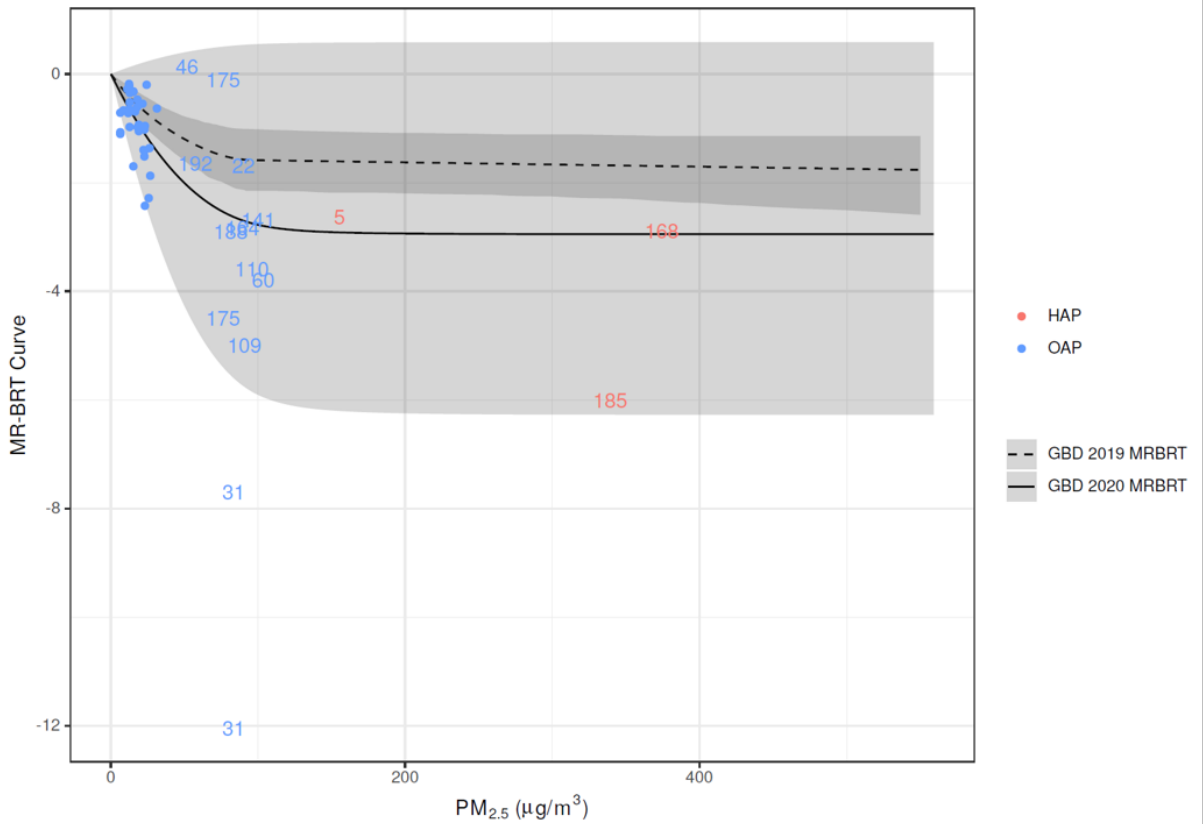
Birthweight (g), Full Exposure Range



Gestational Age (weeks), Low Exposure Range



Gestational Age (weeks), Full Exposure Range



We used the curves of estimated shifts across the exposure range to predict the shift in both birthweight and gestational age for total female particulate matter pollution exposure in each location and year. Because the epidemiological studies mutually controlled for birthweight and gestational age, we assumed these shifts are independent. We then shifted the observed distributions to reflect the expected bwga distribution in the absence of particulate matter pollution. These shifted distributions were used as the counterfactual in the PAF calculation equation to calculate the burden attributable to PM_{2.5} pollution.

To calculate PAFs, the distribution is divided into 56 bw-ga categories, each with a unique RR. Let p_i be the observed proportion of babies in category, i and p_i' be the counterfactual proportion of babies in category, i if there were no particulate matter pollution.

$$PAF_{PM} = \frac{\sum_{i \in bwga \text{ category}} RR_i p_i - \sum_{i \in bwga \text{ category}} RR_i p_i'}{\sum_{i \in bwga} RR_i p_i}$$

We proportionately split this PAF to ambient and HAP based on exposure as described below. One important assumption to note is that we assume the shift in bw and ga is linear across the bwga distribution.

For lower respiratory infections, PM_{2.5}-attributable PAFs are directly estimated in addition to estimated through bwga mediation. We expect that some of the directly estimated PAFs are mediated through bw and ga. Additionally, the directly estimated PAF is based on a summary of relative risks for all children under 5 years, so there is a possibility that the mediated PAF, which is more finely resolved, could be greater. To avoid double counting, for the two neonatal age groups (0–6 days and 0–27 days), we take the maximum of the two PAF estimates. If the directly estimated PAF is greater than the bwga-mediated PAF, we take the direct estimate, and if the mediated PAF is greater, we take the mediated estimate.

PTB incidence and mortality are both outcomes measured in the GBD. 100% of the burden for this cause is attributable to short gestation. To calculate the percentage attributable to particulate matter pollution, we estimated the percentage of babies born at less than 37 weeks (p_{ptb}) and the percentage of babies that would have been born at less than 37 weeks in the counterfactual scenario of no particulate matter pollution (p_{ptb}').

$$PAF_{ptb,pm} = 1 - \frac{p_{ptb}'}{p_{ptb}}$$

Limitations

Although for GBD 2020 we have not used active smoking or secondhand smoking data to estimate PM_{2.5} risk curves, we still use an integrated exposure–response approach because we integrate relative risk estimates across ambient and HAP sources. The use of both source types to construct a risk curve with PM_{2.5} as the exposure indicator assumes equitoxicity of particles regardless of source, despite evidence suggesting differences in health impacts by specific PM source (eg, motor vehicles, coal-fired power plant), size, and/or chemical composition. However, in the absence of sufficient estimates of source- or composition-specific exposure–response relationships and consistent and robust evidence of differential toxicity by source, integrating across all OAP and HAP studies is the approach most consistent with the current evidence, as reviewed by USA EPA and WHO.^{19,20}

Proportional PAF approach

Prior to GBD 2017, relative risks for both ambient and HAP exposures were obtained from the risk curve as a function of exposure, relative to the same TMREL. In reality, were a country to reduce only one of these risk factors, the other would remain. We did not consider the joint effects of particulate matter from outdoor exposure and burning solid fuels for cooking. For GBD 2017, we developed a new approach to use the risk curve for obtaining PAFs for both OAP and HAP, which was also implemented in GBD 2019 and 2020.

Let Exp_{OAP} be the ambient $PM_{2.5}$ exposure level and Exp_{HAP} be the excess exposure for those who use solid fuel for cooking. Let P_{HAP} be the proportion of the population using solid fuel for cooking. We calculated PAFs at each $0.1^\circ \times 0.1^\circ$ grid cell. We assumed that the distribution of those using solid fuel for cooking (HAP) was equivalent across all grid cells of the GBD location.

For the proportion of the population not exposed to HAP the relative risk was:

$$RR_{OAP} = MRBRT(z = Exp_{OAP})/MRBRT(z = TMREL),$$

And for those exposed to HAP, the relative risk was

$$RR_{HAP} = MRBRT(z = Exp_{OAP} + Exp_{HAP})/MRBRT(z = TMREL).$$

We then calculate a population-level RR and PAF for all particulate matter exposure:

$$RR_{PM} = RR_{OAP}(1 - P_{HAP}) + RR_{HAP}P_{HAP}$$
$$PAF_{PM} = \frac{RR_{PM} - 1}{RR_{PM}}$$

We population weight the grid-cell level particulate matter PAFs to get a country level PAF, and finally, we split this PAF based on the average exposure to each OAP and HAP:

$$PAF_{OAP} = \frac{Exp_{OAP}}{Exp_{OAP} + P_{HAP} * Exp_{HAP}} PAF_{PM}, \text{ and } PAF_{HAP} = \frac{P_{HAP} * Exp_{HAP}}{Exp_{OAP} + P_{HAP} * Exp_{HAP}} PAF_{PM}.$$

With this strategy, $PAF_{PM} = PAF_{HAP} + PAF_{OAP}$, and no burden is counted twice.

References

1. Hammer M. S., van Donkelaar A., Li C., Lyapustin A., Sayer A. M., Hsu C. N., Levy R. C., Garay M. J., Kalashnikova O. V., Kahn R. A., Brauer M., Apte J. S., Henze D. K., Zhang L., Zhang Q., Ford B., Pierce J. R., Martin R. V. Global Estimates and Long-Term Trends of Fine Particulate Matter Concentrations (1998–2018). *Environ. Sci. Technol.* 2020; 54(13): 7879-7890. DOI: 10.1021/acs.est.0c01764
2. van Donkelaar, A.; Martin, R. V; Brauer, M.; Hsu, N. C.; Kahn, R. A.; Levy, R. C.; Lyapustin, A.; Sayer, A. M.; Winker, D. M. Global Estimates of Fine Particulate Matter using a Combined Geophysical-Statistical Method with Information from Satellites, Models, and Monitors. *Environ. Sci. Technol.* 2016, 50 (7), 3762–3772
3. Shaddick, G., Thomas, M.L., Jobling, A., Brauer, M., van Donkelaar, A., Burnett, R., Chang, H., Cohen, A., Van Dingenen, R., Dora, C. and Gumy, S., 2016. Data Integration Model for Air

Quality: A Hierarchical Approach to the Global Estimation of Exposures to Ambient Air Pollution. *Journal of Royal Statistical Society Series C (Applied Statistics)*. 2017.
DOI: 10.1111/rssc.12227

4. Shaddick, G., Thomas, M. L., Mudu, P., Ruggeri, G. and Gumy, S. Half the world's population are exposed to increasing air pollution. Accepted by *Nature Climate and Atmospheric Science*.
5. Brauer, M.; Freedman, G.; Frostad, J.; van Donkelaar, A.; Martin, R. V; Dentener, F.; Van Dingenen, R.; Estep, K.; Amini, H.; Apte, J. S.; et al. Ambient Air Pollution Exposure Estimation for the Global Burden of Disease 2013. *Environ. Sci. Technol.* 2015, 50 (1), 79–88.
6. Shaddick G, Thomas M, Amini H, Broday DM, Cohen A, Frostad J, Green A, Gumy S, Liu Y, Martin RV, Prüss-Üstün A, Simpson D, van Donkelaar A, Brauer M. Data integration for the assessment of population exposure to ambient air pollution for global burden of disease assessment. *Environ Sci Technol.* 2018 Jun 29. doi: 10.1021/acs.est.8b02864
7. Rue, H.; Martino, S.; Chopin, N.; Approximate Bayesian inference for latent Gaussian models by using integrated nested Laplace approximations. *Journal of the royal statistical society: Series b (statistical methodology)*. 2009;71(2):319-92.
8. Thomas, M. L., Shaddick, G., Simpson, D., de Hoogh, K. and Zidek, J. V. Spatio-temporal downscaling for continental-scale estimation of air pollution concentrations. arXiv preprint arXiv:1907.00093 (also been Submitted to the *Journal of the Royal Statistical Society: Series C (Applied Statistics)*).
9. Turner MC, Jerrett M, Pope CA 3rd, Krewski D, Gapstur SM, Diver WR, Beckerman BS, Marshall JD, Su J, Crouse DL, Burnett RT. Long-term ozone exposure and mortality in a large prospective study. *Am J Respir Crit Care Med.* 2016; 193(10): 1134-42.
10. Yin P, Brauer M, Cohen A, et al. Long-term Fine Particulate Matter Exposure and Nonaccidental and Cause-specific Mortality in a Large National Cohort of Chinese Men. *Environ Health Perspect* 2017; 125: 117002.
11. Li T, Zhang Y, Wang J, et al. All-cause mortality risk associated with long-term exposure to ambient PM_{2.5} in China: a cohort study. *Lancet Public Health* 2018; 3: e470–7.
12. Yang Y, Tang R, Qiu H, et al. Long term exposure to air pollution and mortality in an elderly cohort in Hong Kong. *Environ Int* 2018; 117: 99–106.
13. Hystad P, Larkin A, Rangarajan S, AlHabib KF, Avezum A, Tumerdem Calik KB; Chifamba J, Dans A, Diaz R, du Plessis JL, Gupta R, Iqbal R, Khatib R, Kelishadi R, Lanan F, Liu Z, Lopez-Jaramillo P, Nair S, Poirier P, Rahman O, Rosengren A, Swidan H, Tse L-A, Wei L, Wielgosz A, Yeates K, Yusoff K, Zatoński T, Yusuf S, Brauer M. Outdoor fine particulate matter air pollution and cardiovascular disease: Results from 747 communities across 21 countries in the PURE Study. (Submitted to *Lancet Global Health*)
14. Joseph P, Rangarajan S, Islam S, Menten A, Hystad P, Brauer M, Raman Kutty V, Gupta R, Wielgosz A, AlHabib KF, Dans A, Lopez-Jaramillo P, Avezum A, Lanan F, Oguz A, Kruger IM, Diaz R, Yusoff K, Mony P, Chifamba J, Yeates K, Kelishadi R, Yusufali A, Khatib R, Rahman O, Zatońska K, Iqbal R, Wei L, Bo H, Rosengren A, Kaur M, Mohan V, Lear SA, Teo KK, O'Donnell M, McKee M, Dagenais G, Yusuf S. Modifiable risk factors, cardiovascular disease and mortality in 155,722 individuals from 21 high-, middle-, and low-income countries (PURE): a prospective cohort study. *The Lancet.* 2019. doi:10.1016/S0140-6736(19)32008-2
15. Burnett RT, Pope CA 3rd, Ezzati M, Olives C, Lim SS, Mehta S, Shin HH, Singh G, Hubbell B, Brauer M, Anderson HR, Smith KR, Balmes JR, Bruce NG, Kan H, Laden F, Prüss-Ustün A, Turner MC, Gapstur SM, Diver WR, Cohen A. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environ Health Perspect.* 2014; 122(4): 397-403.
16. Pope CA III, Cohen AJ, Burnett RT. Cardiovascular Disease and Fine Particulate Matter: Lessons and Limitations of an Integrated Exposure Response Approach. *Circulation Research.* 2018;122:1645-1647.

17. Lind L, Sundström J, Ärnlöv J, Lampa E. Impact of Aging on the Strength of Cardiovascular Risk Factors: A Longitudinal Study Over 40 Years. *J Am Heart Assoc.* 2018;7(1):e007061. Published 2018 Jan 6. doi:10.1161/JAHA.117.007061
18. Ghosh R, Causey K, Burkart K, Wozniak S, Cohen A, Brauer M. Ambient and household PM_{2.5} pollution and adverse perinatal outcomes: A meta-regression and analysis of attributable global burden for 204 countries and territories. *PLoS Med* 2021. Accepted; in press.
19. US Environmental Protection Agency. Integrated science assessment (ISA) for particulate matter (Final Report, Dec 2009). EPA/600/R-08/139F, 2009. Washington, DC: US Environmental Protection Agency; 2009. Available at: <http://cfpub.epa.gov/ncea/risk/recorddisplay.cfm?deid=216546>
20. World Health Organization. Review of evidence on health aspects of air pollution – REVIHAAP Project technical report. Copenhagen: WHO Regional Office for Europe; 2013. Available at: http://www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf?ua=1