RESEARCH ARTICLE

Potential health benefits of eliminating traffic emissions in urban areas

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Abstract

Traffic is one of the major contributors to $PM_{2.5}$ in cities worldwide. Quantifying the role of traffic is an important step towards understanding the impact of transport policies on the possibilities to achieve cleaner air and accompanying health benefits. With the aim of estimating potential health benefits of eliminating traffic emissions, we carried out a meta-analysis using the World Health Organisation (WHO) database of source apportionment studies of $PM_{2.5}$ concentrations. Specifically, we used a Bayesian meta-regression approach, modelling both overall and traffic-related (tailpipe and non-tailpipe) concentrations simultaneously. We obtained the distributions of expected $PM_{2.5}$ concentrations (posterior densities) of different types for 117 cities worldwide. Using the non-linear Integrated Exposure Response (IER) function of $PM_{2.5}$, we estimated percent reduction in different disease endpoints for a scenario with complete removal of traffic emissions. We found that eliminating traffic emissions results in achieving the WHO-recommended concentration of $PM_{2.5}$ only for a handful of cities that already have low concentrations of pollution. The percentage reduction in premature mortality due to cardiovascular and respiratory diseases increases up to a point (30–40 ug/m^3), and above this concentration, it flattens off. For diabetes-related mortality, the percentage reduction in mortality decreases with increasing concentrations—a trend that is opposite to other outcomes. For cities with high concentrations of pollution, the results highlight the need for multi-sectoral strategies to reduce pollution. The IER functions of $PM_{2.5}$ result in diminishing returns of health benefits at high concentrations, and in case of diabetes, there are even negative returns. The results show the significant effect of the shape of IER functions on health benefits. Overall, despite the diminishing results, a significant burden of deaths can be prevented by policies that aim to reduce traffic emissions even at high concentrations of pollution.

Introduction and background

Motorised traffic is growing rapidly in many low- and middle-income countries (LMICs) resulting from increasing ownership of vehicles and rapid urbanisation. In these settings, emissions are still increasing compared to Europe and the United States where emissions have

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stabilised or are decreasing [1]. As expected, fine particulate matter ($PM_{2.5}$) concentrations in East Asia, South Asia, and parts of Sub-Saharan Africa have increased markedly over the past decades and are currently the highest in the world [2, 3]. These concentrations of pollution result in disproportionate share of premature mortality due to cardiovascular and respiratory diseases in LMICs [3]. New evidence on the impact of $PM_{2.5}$ pollution on infant mortality [4] and diabetes [5], now included in the Global Burden of Disease estimates [6], has added to the previously known health burden of $PM_{2.5}$. Air pollution is one of the key pathways through which transport impacts public health in the cities [7]. Health impact studies that focus on reducing the use of motorised travel through a shift to active modes of travel have highlighted the health benefits from reduction in traffic emissions [8, 9].

Several previous studies investigated different aspects of traffic-related air pollution and its health impacts [10–15]. For example, Pan et al. [10] estimated potential impacts of electric vehicles on air quality and health endpoints in Houston (USA) in 2040. Tong et al. [13] examined health effects of $PM_{2.5}$ emissions from on-road vehicles during weekdays and weekends in Beijing, China. Teixeira et al. [14] estimated the impact of PM emissions from heavy-duty trucks on human health. A review by Health Effects Institute [16] found suggestive evidence of a causal relationship between exposure to traffic-related air pollution and onset of childhood asthma, non-asthma respiratory symptoms, impaired lung function, total and cardiovascular mortality, and cardiovascular morbidity. These studies have all pointed towards the potential of achieving health benefits from reduction in traffic-related air pollution in urban settings.

The potential of gaining health benefits in a city through reduction in traffic emissions depends on the proportion of $PM_{2.5}$ concentrations that is contributed by this sector as well as the total $PM_{2.5}$ burden. Since the dose-response functions are non-linear, with a curve that is steep at low concentrations and flattens towards higher concentrations, there are diminishing returns of reduction in pollution levels at higher concentrations [2]. The two factors (proportion of traffic and overall $PM_{2.5}$ concentrations) vary greatly across the world [17–19]. For example, Heydari et al. [19] showed that traffic contribution estimates as well as uncertainties around these estimates vary largely across various cities and regions worldwide. Many LMIC cities have high concentrations of pollution because of contributions from multiple sectors, of which transport is only one of them [18, 20]. Many of the high-income countries have achieved cleaner air due to the reduction of emissions across multiple sectors. Based on the WHO source apportionment database [21], employing a population-weighted approach, Karagulian et al. [17] conducted a systematic review of local source contributions of PM in cities across the world. Using the same database and based on a Bayesian meta-regression approach, Heydari et al. [19] estimated the expected percentage contribution of traffic to $PM_{2.5}$ and PM_{10}, and their respective uncertainties, in various cities and regions worldwide.

The primary goal of this research is to estimate the health benefits that can be gained by reduction in traffic emissions. This is achieved by carrying out a rigorous meta-analysis exercise, with the aim of pooling strength over several previous studies on the concentrations of $PM_{2.5}$ in multiple cities. To this end, in this work we introduce and discuss an analytical framework that can draw valuable inferences regarding the overall (due to all sources) and traffic-related (exhaust and non-exhaust emissions) concentrations of air pollutant concentrations in various locations worldwide from a collection of previous studies. Our specific objectives are summarised as follows:

1. Develop a meta-regression model that simultaneously analyses overall and traffic-related $PM_{2.5}$ concentrations in urban areas based on the previous studies collected in the WHO source apportionment database. Doing so, (i) we can explain variability in the reported concentrations by previous studies; (ii) estimate the magnitude of dependence between overall

and traffic-related $PM_{2.5}$ concentrations; and (iii) estimate expected concentrations of $PM_{2.5}$ of different types (traffic-related, non-traffic-related, and overall due to all sources) with their associated uncertainties in multiple cities worldwide.

2. Use the above estimates (specifically, the estimates obtained in 1.iii) to investigate the potential of achieving cleaner air and preventing premature mortality from multiple disease outcomes through reductions in traffic-related $PM_{2.5}$.

Materials and methods

Review framework and study selection

We present a meta-analysis of traffic-related $PM_{2.5}$ and overall (due to all sources) $PM_{2.5}$ concentrations reported in the latest available World Health Organization (WHO) database (at the time of writing) on source apportionment studies [21]. The WHO database reports overall $PM_{2.5}$ concentrations and the contributions of different source categories (i.e., traffic, industry, domestic fuel burning, natural sources, and unspecified sources of human origins) to particulate matter for various locations. Given the aim of our research, we excluded studies that did not report the share of traffic. Also, we only included studies that were reported from urban areas, excluding other site typologies such as industrial, rural, etc., to obtain a homogenous sample, reducing non-comparability between studies. This improves the quality of our meta-analysis.

For each study, we obtained traffic-related $PM_{2.5}$ concentrations by multiplying overall $PM_{2.5}$ concentrations by the reported percentage traffic contributions to $PM_{2.5}$. To carry out our quantitative synthesis of previous research, we considered a series of explanatory variables available in the WHO database. These included publication year, study location (city, country, region, and continent), population, geographic coordinates, and estimation method. Another potentially relevant information was whether a study reported sea salt contribution to $PM_{2.5}$. To better capture variability in the data, cities were assigned to 12 different regions mostly according to geographic proximity, and classifications reported by [17] and [19]. These are North America, Central Europe, East Asia, East/West Africa, Middle East, North-western Europe, Oceania/Japan, South/Central America, South-eastern Asia, Southern Asia, Southwestern Europe, and Western Europe. List of countries in each region are reported in Table B of the S1 File.

Characteristics of the final data

Our final dataset includes 182 observations of source contributions of $PM_{2.5}$ concentration in urban areas. These observations are measurements reported by 118 studies (Table A of the S1 File), corresponding to 117 cities worldwide, from 1987 to 2014. A summary of the final sample used in our study is reported in Tables 1 and 2. Around 36% of the reported measurements

Table 1. Descriptive statistics of the data.

Variables	Mean	Std. Dev.	Min	Max
Ln(city population)	-0.13	1.68	-6.97	2.64
Latitude	30.97	23.61	-41.27	64.83
Sea salt contribution reported	0.50	0.50	0.00	1.00
Study published after 2005	0.74	0.44	0.00	1.00
Study was conducted in North America or Oceania	0.36	0.48	0.00	1.00
Study was conducted in North Western or Western Europe	0.11	0.31	0.00	1.00
Study was conducted in the rest of Europe	0.23	0.42	0.00	1.00

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Table 2. Distribution of observations in each region.

Regions	Frequency	Percent
Africa	1	0.55
Central and Eastern Europe	3	1.65
East Asia	18	9.89
Middle East	5	2.75
North America	57	31.32
Northwestern Europe	12	6.59
Oceania/Japan	9	4.95
South/Central America	12	6.59
Southeastern Asia	9	4.95
Southern Asia	9	4.95
Southwestern Europe	39	21.43
Western Europe	8	4.40

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were estimated based on studies conducted in North America or Oceania. Around 10% were from North Western or Western Europe while 23% of the observations were from studies conducted in the rest of Europe. Two-thirds (74.2%) of the measurements were reported after year 2005, and 50% of the observations in the data reported percentage contribution of sea salt to $PM_{2.5}$. In our final data, the reported overall $PM_{2.5}$ concentrations varied largely across cities: from around 12 ug/m^3 to 97 ug/m^3, with a mean (and standard deviation) of 35.11 ug/m^3 (36.97 ug/m^3) at a global level. Similarly, traffic-related $PM_{2.5}$ varied from 1.10 ug/m^3 to 64.02 ug/m^3, with a mean (and standard deviation) of 9.13 ug/m^3 (12.41 ug/m^3).

Meta-regression

We adopted a joint meta-regression approach to identify factors that can explain variations in reported overall and traffic-related $PM_{2.5}$ concentrations in the WHO database. This allowed us to model both outcomes simultaneously through a system-equation approach rather than modelling each outcome separately, improving the reliability of our statistical inferences. We assumed the log-transformed concentrations follow a multivariate normal density. Let $\boldsymbol{y}_{ki}$ denote the vector of log-transformed concentrations of k different types reported by previous studies i ($i = 1, 2, \ldots, N$). Here $k = 2$; therefore, $\boldsymbol{y} = (y_{1i}, y_{2i})$, where y_{1i}, y_{2i} denote $PM_{2.5}$ due to traffic and overall $PM_{2.5}$, respectively. Let $\boldsymbol{X}_k = (X_{k1}, X_{k2}, \ldots, X_{km})$ be the vector of m explanatory variables (e.g., population) associated with the outcomes of interest (y_{1i}, y_{2i}) with their respective regression coefficients $\boldsymbol{\gamma} = (\gamma_{k1}, \gamma_{k2}, \ldots, \gamma_{km})$. Let $\boldsymbol{\eta} = (\eta_1, \eta_2)$ denote the vector of intercepts corresponding to y_{1i} and y_{2i}, respectively. Let R and K denote the scale matrix and the degrees of freedom, respectively, in a Wishart distribution. We can then write

$$\begin{aligned} \boldsymbol{y} &\sim MVN(\boldsymbol{\mu}, \textstyle\sum) \\ \mu_{ik} &= \eta_k + \gamma_k \mathbf{X}_{ik} \\ \boldsymbol{\Sigma} &= \begin{bmatrix} \sigma_{11} & \cdots & \sigma_{1k} \\ \vdots & \ddots & \vdots \\ \sigma_{k1} & \cdots & \sigma_{kk} \end{bmatrix} \\ \boldsymbol{\Sigma}^{-1} &\sim Wishart(R, K) \end{aligned} \tag{1}$$

As it can be seen in the above model, the dependency across outcomes is captured through the covariance matrix Σ. This specification allowed us to investigate the magnitude of correlation between overall $PM_{2.5}$ and traffic-related $PM_{2.5}$ concentrations.

Prior specification and model computation

Normally distributed non-informative priors, normal (0,100), were used for the regression coefficients. In the joint modelling of correlated outcomes, it is a common practice to specify a Wishart distribution for the inverse of covariance matrix Σ^{-1} [22], with $K = 2$ (for two correlated outcomes) and a 2x2 scale matrix R ($R[1,1] = R[2,2] = 0.01$ and $R[1,2] = R[2,1] = 0$), which leads to a non-informative prior specification. For model computation, we employed WinBUGS [23] to draw posterior densities for our Markov chain Monte Carlo simulations running two chains each containing 15,000 iterations. The posterior densities are based on 20,000 samples as the first 5,000 iterations were discarded for convergence requirements. Based on the Gelman-Rubin statistic [24], history plots, and Monte Carlo errors, this number of iterations was sufficient.

Computing probabilities of exceeding the WHO-recommended concentration of $PM_{2.5}$

We estimated the potential of a city to attain the WHO-recommended concentration of $PM_{2.5}$ concentrations (5 ug/m^3) [18] if all traffic emissions were removed. For this, we estimated the posterior densities of non-traffic-related $PM_{2.5}$ concentrations for each city based on our meta-regression approach. We then estimated the probability that the latter posterior mean exceeds 5 ug/m^3. The higher this probability, the lower the ability of a city to reach the WHO-recommended concentration of $PM_{2.5}$ even from the complete removal of traffic emissions. To compute these probabilities, we created an *Mx1* matrix of indicator variables [I_c] for each city c, where M is the total number of cities under investigation. At each iteration of our MCMC simulations and for each city, we compared the expected non-traffic-related $PM_{2.5}$ concentration with the WHO-recommended value of 5 ug/m^3 as shown in (2). Finally, we averaged the indicator variable value over all iterations to obtain the probabilities of exceeding for each city.

$$I_c = \begin{cases} 1 & if\ \eta_i \geq 5 \\ 0 & if\ \eta_i < 5 \end{cases} \tag{2}$$

Estimating health benefits of reducing traffic emissions

We estimated the percentage reduction in health burden resulting from a complete removal of traffic emissions. To estimate changes in health burden we used Integrated Exposure Response (IER) functions for six disease endpoints that were used in Global Burden of Disease (GBD) 2017 [6]. These are ischemic heart disease (IHD), stroke, chronic obstructive pulmonary disease (COPD), lung cancer, lower respiratory infections (LRI), and type-II diabetes (diabetes). For IHD and Stroke, IER is age-specific and, for these, we present calculations for 55–60 years age group for illustration of the method. The relative risk using IER function is calculated as:

$$\begin{aligned} RR(z) &= 1 + \vartheta(1 - \exp(-\omega z^{\delta})) \\ z &= \max(0,\ PM_{2.5} - x) \end{aligned} \tag{3}$$

where ϑ, ω, δ are parameters specific to each disease end-point and x is a counterfactual value below which the assumption is that there are no increased mortality. The value of x is obtained from a uniform distribution, representing its uncertainty, with lower and upper bounds of 2.4 ug/m^3 and 5.9 ug/m^3, respectively [25]. The dose-response function for the six disease end-points are presented in Fig 1. This graph presents the average value of relative risks at each concentration value, calculated for 1000 iterations of the four IER parameters ϑ, ω, δ, x reported by Burnett [26].

We used the comparative risk assessment approach to estimate the population attributable fraction (PAF) for each city and expressed it as percentage (see Eq 4). We defined counterfactual $PM_{2.5cf}$ as the concentrations achieved after the complete removal of traffic emissions.

$$PAF = 1 - \frac{RR_{cf}}{RR_b} \tag{4}$$

where RR_{cf} and RR_b are, respectively, the relative risk values (see Eq 3) for the concentrations of $PM_{2.5cf}$ and $PM_{2.5b}$; the latter stands for overall $PM_{2.5}$ concentrations at the baseline. For each city, we calculated $PM_{2.5b}$ as the mean value of the posterior densities of overall $PM_{2.5}$ concentrations, obtained from our meta-regression analysis. We calculated $PM_{2.5cf}$ as the difference between the mean values of the posterior densities of overall and traffic-related $PM_{2.5}$. Lastly, using the pair of these concentrations, for each city, we calculated PAF for each of the 1000 iterations of the parameters in the IER functions, and we present the mean PAF of those iterations.

Results

The results relating to the estimated posterior densities of $PM_{2.5}$ for the cities included in our study are reported in the S1 File (Tables C-E in S1 File). Note that to investigate robustness of

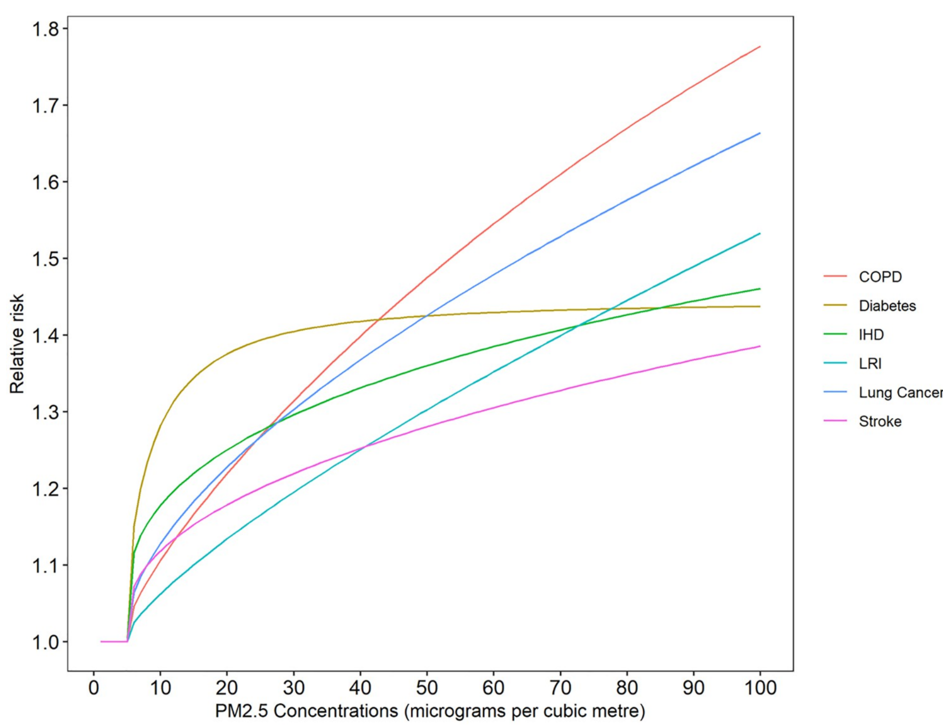

Fig 1. Integrated exposure response function (age group 55–60 years for IHD and stroke).

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the multivariate normal density assumption in (1), we used a scale mixing approach that can address skewness in the data; this confirmed the suitability of our assumption.

Posterior estimates of the meta-regression coefficients

As mentioned previously, we considered variables available in the WHO database to develop our models. Through an exploratory data analysis, we noticed a relatively significant difference between measurements reported by studies published before 2005 and those published after 2005 with respect to traffic-related $PM_{2.5}$ concentrations. This is in accordance with [19]. We therefore created a categorical variable for publication year with the aim of capturing the above difference in the model. Also, we created several categorical variables to include the location of studies in the models, considering different possibilities and combinations of regions according to geographic proximity, income, and classifications reported by [17] and [19]. The final location variable had four categories: (1) North America, Oceania or Japan; (2) North Western or Western Europe; (3) the rest of Europe; and (4) the rest of the world. Japan being a high-income country and having $PM_{2.5}$ concentrations similar to North America and Oceania was included in the first category mentioned above. Table 3 reports the posterior summary of the estimated regression coefficients.

Correlation between overall $PM_{2.5}$ and traffic-related $PM_{2.5}$

Our modelling approach allowed us to estimate the magnitude of the correlation between overall $PM_{2.5}$ and traffic-related $PM_{2.5}$ across a sample of 117 cities worldwide. The mean (standard deviation) of the correlation is 0.63 (0.05), with a 95% credible interval varying from 0.54 to 0.72. This indicates that the correlation between overall and traffic-related $PM_{2.5}$ is statistically important and that traffic-related $PM_{2.5}$ and overall $PM_{2.5}$ are highly correlated.

Table 3. Meta-regression estimation results.

Variable	Mean	Std. Dev.	95% Credible interval	
Traffic-related $PM_{2.5}$ concentrations				
Ln(city population)	0.21	0.03	0.14	0.27
Latitude (divided by 10)	0.06	0.03	0.01	0.11
Sea salt contribution reported[1]	-0.29	0.11	-0.51	-0.07
Study published after 2005[2]	-0.36	0.11	-0.57	-0.14
Study was conducted in North America, Oceania or Japan[3]	-1.25	0.13	-1.50	-0.99
Study was conducted in North Western or Western Europe[3]	-1.69	0.20	-2.08	-1.30
Constant	2.54	0.16	2.23	2.86
Overall $PM_{2.5}$ concentrations				
Ln(city population)	0.08	0.03	0.02	0.13
Latitude (divided by 10)	0.13	0.02	0.08	0.17
Sea salt contribution reported[1]	-0.33	0.08	-0.50	-0.17
Study was conducted in North America, Oceania or Japan[3]	-1.11	0.11	-1.33	-0.89
Study was conducted in North Western or Western Europe[3]	-1.49	0.16	-1.81	-1.17
Study was conducted in the rest of Europe[3]	-0.41	0.11	-0.63	-0.19
Constant	3.63	0.09	3.46	3.81

[1] Group of studies that do not report sea salt contribution is the reference group.

[2] Group of studies conducted on or before 2005 is the reference group.

[3] Rest of the world is the reference group.

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Probability of non-traffic emissions exceeding the WHO threshold

Fig 2 presents the probabilities of $PM_{2.5}$ concentrations exceeding the WHO-recommended concentration of $PM_{2.5}$ (i.e., 5 ug/m^3) for the cities under investigation if all traffic emissions are removed. This probability accounts for $PM_{2.5}$ concentrations as well as their associated uncertainties, both of which are represented by posterior distribution of concentrations, estimated based on our analysis. Fig 2 shows that, when the overall concentration is beyond 15 ug/m^3, the probability of exceeding the WHO-recommended concentration of $PM_{2.5}$ remains greater than 80 percent.

Health benefit potential of reducing traffic emissions

Fig 3 presents percent reduction in the premature mortality due to four disease end-points (COPD, IHD, lung cancer and diabetes) for a counterfactual scenario of complete removal of traffic-related $PM_{2.5}$ concentrations. Note that stroke and LRI have similar shaped curves as IHD and Lung Cancer, respectively, and their results are not shown here. According to these graphs, the benefits of prevented mortality increase up to a point (30–40 ug/m^3) for COPD, IHD and lung cancer, at which point there are large variations, and then flatten off. The flattening is most prominent for IHD and less so for COPD and lung cancer. The diabetes-related

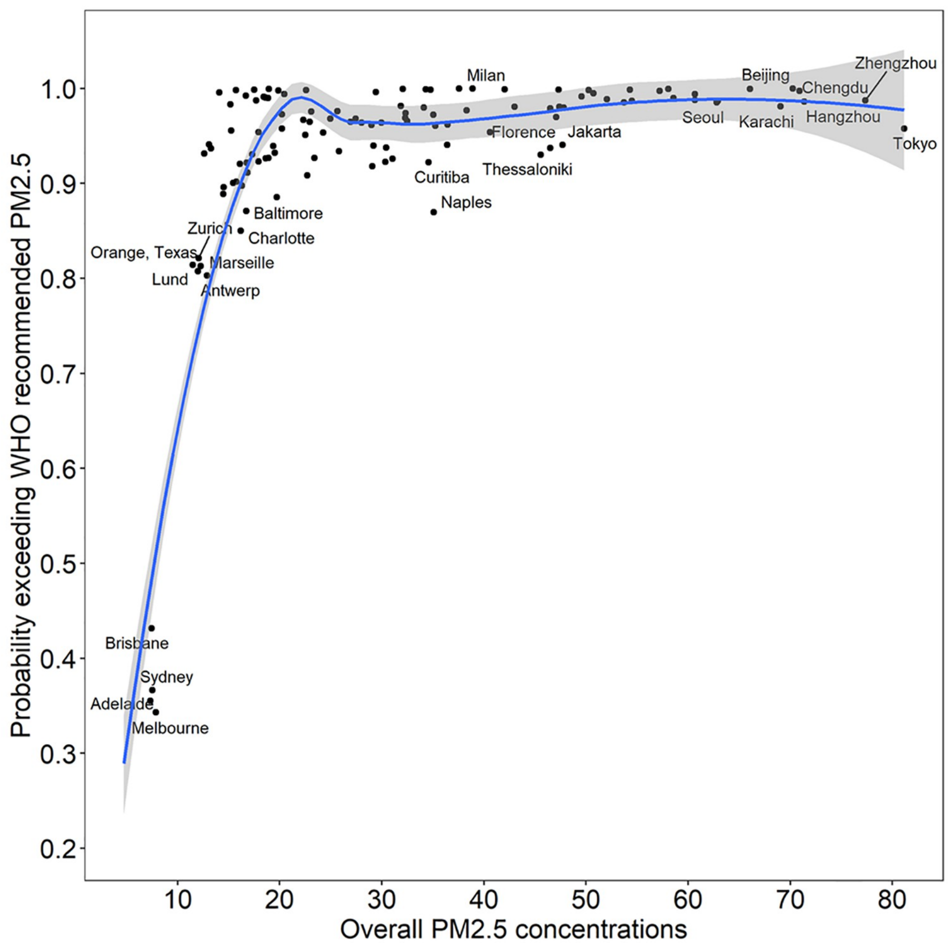

Fig 2. Probability of exceeding WHO-recommended $PM_{2.5}$ if traffic emissions are removed.

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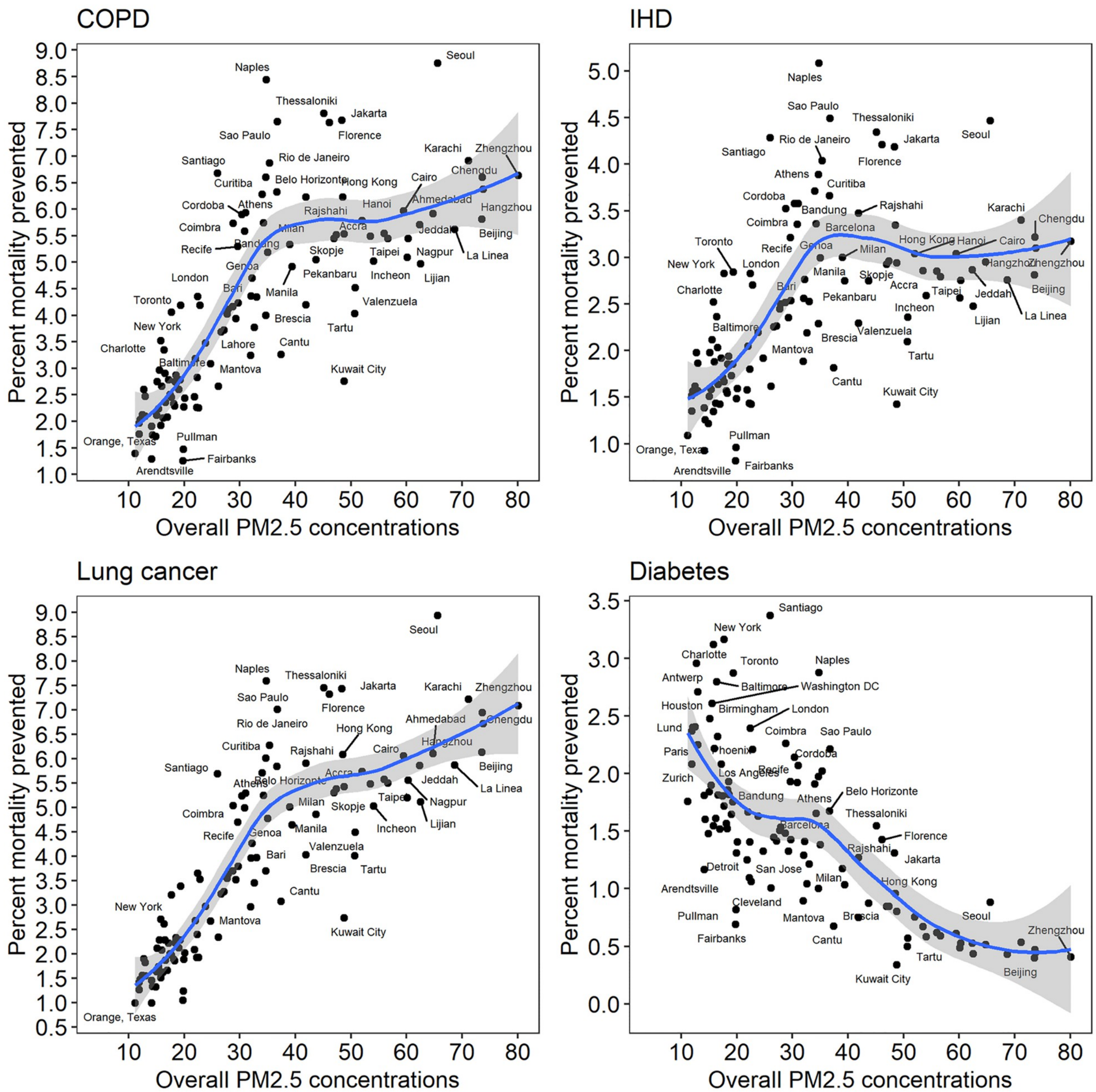

Fig 3. Percentage reduction in mortality resulting from total removal of traffic emissions (for COPD and IHD, age group is 50–55 years).

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mortality reduction show that the largest benefits are limited to concentrations below 25 ug/ m^3, at which point there is large variation across the cities. Above this concentration, the benefits in terms of reduced mortality become progressively smaller.

Discussion

Statement of principal findings

We used the WHO database of source apportionment studies to estimate overall, traffic-related, and non-traffic-related $PM_{2.5}$ concentrations using a Bayesian meta-regression approach. For the posterior distributions of the expected concentration of non-traffic-related $PM_{2.5}$, we estimated the probability that concentrations remained higher than the WHO-recommended annual concentration of $PM_{2.5}$ if traffic-related emissions were removed completely. We found that this probability rises steeply up to 20 ug/m^3, and then remains greater than 90 percent. In other words, for the cities with overall concentrations greater than 20 ug/m^3, the removal of traffic-related emissions are highly unlikely to reduce concentration levels down to the WHO-recommended $PM_{2.5}$ annual guideline.

We estimated the percentage reduction in premature mortality due to four disease end-points (COPD, IHD, lung cancer and diabetes) if all traffic-related emissions are removed. We used non-linear IER functions along with the comparative risk assessment approach to estimate population attributable fraction corresponding to this reduction in concentrations. We found that for COPD, IHD and lung cancer, the percent reduction in mortality increases up to 30–40 ug/m^3, and at higher concentrations, it flattens off, showing diminishing returns. The flattening is far more prominent in IHD than in COPD and lung cancer. This is expected from the shape of their IER functions (Fig 1). IER for COPD and lung cancer have steeper functions of relative risk than IHD. In case of Diabetes, the pattern is opposite to that of the other three outcomes. With increasing concentrations, there are negative returns in the reduction of premature mortality. This is expected given that diabetes is the only disease outcome that has a prominently flat IER function after a steep jump up to 20 ug/m^3. Mathematically, a flat function implies that the ratio of relative risks in Eq 3 approaches unity at higher concentrations.

Strengths and weaknesses of the study

We employed a multivariate meta-regression approach to estimate the expected concentrations of $PM_{2.5}$ of different types (in terms of source) for 117 cities in various regions worldwide. Note that compared to relaying on information from one study only, conclusions from a meta-analytic approach are more reliable for evidence-based policy making. Further, we modelled traffic-related and overall $PM_{2.5}$ concentrations jointly; therefore, our estimates have superior statistical properties. This is due to the fact that data points borrow strength from other related data points [27]. Also, a joint analysis allowed us to investigate the magnitude of correlation between traffic related $PM_{2.5}$ and overall $PM_{2.5}$ based on a systematic approach, providing further insight into the relationship between the two types of concentrations. Understanding this correlation is quite interesting as it allows estimating the range of one type of concentration from the other one when both pieces of information are not available for a given location. Note that our meta-regression, being developed under the Bayesian paradigm, accounted for uncertainties in both regression parameter estimates and predicted values fully.

One of the limitations of our study is that we have only estimated reduction in mortality due to different diseases as percentage of baseline health burden. Depending on the magnitude of baseline health burden, a given percentage reduction will translate to highly divergent values across the cities. This is because, in certain countries that have much greater proportion of older adults, baseline incidence rates of mortality from the cardiovascular, respiratory, and metabolic diseases are much greater than countries with much younger population. Also, LMICs have much greater incidence rates of mortality than HICs. Secondly, many city estimates used in our analysis are more than a decade old or even older. As a result, our study does not represent the latest situation of most of the cities. However, our study provides an analytical approach that can be readily updated as new data (other studies) become available.

With respect to potential biases in carrying out a meta-analysis [28], firstly, we ensured comparability of the studies considered in our research by focusing only on those conducted in urban settings, removing other site typologies (e.g., industrial sites) from the data. Secondly, we avoided strict inclusion/exclusion criteria in our study selection to prevent limiting generalisability. For example, we did not select urban areas of a specific type or from a specific location. Thirdly, our large sample size helps reduce bias in our estimates. Since we relied on a valid collection of previous studies made available by WHO [21], publication bias should be negligible in our meta-analysis for the period of study as this bias mostly arises when several important studies are not included in a meta-analysis. Overall, we believe such biases would not result in any important issue in our inferences. Lastly, as we adopted a Bayesian meta-regression approach, one source of bias could relate to the choice of prior distributions for the model parameters. Since we used non-informative priors (see the section of prior specification and model computation), we are confident about the reliability of our estimates.

Meaning of the study: Possible mechanisms and implications for policymakers

We found that only for a handful of cities could complete reduction in traffic emissions result in achieving the WHO-recommended guideline for annual $PM_{2.5}$ concentrations. We used the WHO guideline for the purpose of illustration which, for many settings across the world, is a highly ambitious scenario. The method presented here can be applied for more realistic targets of pollution concentrations and could include reductions across multiple sectors. Using a stochastic approach, we can make a probabilistic judgement of the impact that policies will have. Our approach, being developed under the Bayesian framework, can be used to estimate updated probabilities as more information is added or updated information is added for the same cities. The health benefits that we presented are also for a highly ambitious scenario, in which all of traffic emissions are removed. However, due to non-linearity in IER functions, we found that health benefits are not proportionally as large. While traffic as a polluting sector gains a lot of attention due to visibility of its sources (i.e., vehicles), our results imply that cities with high concentrations of pollution need a multi-sectoral framework to reduce anthropogenic emissions. This will not only help clean air much faster, it will also make investments more cost-effective as concentrations near the steeper part of the curve.

$PM_{2.5}$ emissions from vehicles are largely proportional to sulphur content in the fuel, and cleaner fuel with lower sulphur content can significantly reduce traffic emissions [29, 30]. While Europe and North America implemented use of low- and ultra-low-sulphur diesel (less than 50 ppm and less than 15 ppm sulphur content, respectively) in late 2000s [31], the progress in many of the low- and middle-income countries has been much slower [30]. A timeline of sulphur concentrations in diesel for 2013–2020 period across the world [32] shows that many countries are gradually progressing towards the use of low-sulphur fuel. However, more than half of the world's countries are still using high-sulphur fuels. These are mainly low-and middle-income countries spread across Latin America, the Caribbean, Africa, the Middle East, and Asia-Pacific [30]. It is only with low-sulphur fuels that vehicles with stricter emission standards can be effective. Thus, accelerating the desulphurisation of fuel and adoption of cleaner vehicle standards in large parts across the world has large potential to prevent health burden attributed to traffic.

Rapid adoption of electric passenger cars will add to these efforts to reduce on-road emissions. However, electric vehicles, being heavier than internal combustion engine vehicles, have greater contribution to $PM_{2.5}$ due to non-exhaust emissions such as tyre wear, break wear, road surface wear, and resuspension of road dust. As a result, they have only slightly lower PM emissions than an internal combustion engine vehicle [33]. Therefore, electric vehicles may have the greatest benefits in settings where exhaust emissions continue to be high due to lagging emission standards.

The policies to reduce transport emissions should not be restricted to cleaner fuels or vehicle technology. Instead, they should be developed within a broader framework of transport and its impact on health. Use of an electric car may result in lower emissions than conventional vehicles but does not contribute to physical activity or reduce danger on the road. Alternatively, a mode shift from passenger cars to active travel or public transport will not only reduce emissions, but also improve population health through the pathways of physical activity and reduced road injuries. This mode shift can be achieved through a transport system that prioritises widespread and safe walking, cycling, and public transport infrastructure [34].

Unanswered questions and future research

As highlighted in the limitations, the underlying dataset of cities [21] reporting source apportionment has studies that were done a decade or longer ago. Therefore, an updated review of source-apportionment studies could greatly improve our understanding of different sources of pollution in cities. The results on health impacts presented here are based on IER functions that were used in GBD 2017. The different types of risk functions for $PM_{2.5}$ have been shown to have significant impact on their respective estimates of disease burden [35]. As evidence improves on different health outcomes as well as from settings with high concentrations of pollution, we could expect changes in these functions and their respective health impact estimates.

Supporting information

S1 File.
(DOCX)

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