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Changing risk factors that contribute to premature mortality from ambient air pollution between 2000 and 2015

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Abstract

Exposure to ambient air pollution is a major global health risk factor. Using recently updated hazard ratio functions, we estimate that the global premature mortality burden attributable to ambient $PM_{2.5}$ and O_3 exposure increased by about 30% and 17%, respectively, from 2000 to 2015. We analyzed these trends in terms of tradeoffs between changes in baseline mortality, population size, age distribution and exposure. Global population growth alone increased the mortality burden by about 29% and 42% among adults and children, respectively. On the other hand, decreasing baseline mortality during the same period lowered the burden by about 18% and 55% among adults and children, respectively. In high-income countries, aging of the population contributed to increasing premature mortality, while in low-income countries, in Central and West Africa for example, the growing youth population reduced premature mortality. Our results show that improved air quality generally does not keep up with the increasing air pollution-associated disease burden in middle- and high-income countries. We conclude that improvements in health care that reduce baseline mortality along with air pollution mitigation strategies are needed to maximize benefits, and counter the growth and aging of the global population.

1. Introduction

Long-term exposure to ambient air pollution is associated with child mortality from lower respiratory infections (LRI), and adult mortality from LRI and non-communicable diseases (NCDs) that include but are not limited to chronic obstructive pulmonary diseases (COPD), ischemic heart diseases (IHD), stroke, lung cancer and diabetes (Dockery *et al* 1993, Jerrett *et al* 2009, Hansell *et al* 2016, Yin *et al* 2017, Stanaway *et al* 2018, Li *et al* 2018, Burnett *et al* 2018, Lelieveld *et al* 2019). The Global Burden of Disease (GBD) has adopted a framework to estimate the premature mortality burden attributable to ambient air pollution on a comparative scale with other major health risk factors (Stanaway *et al* 2018, Balakrishnan *et al* 2019, Cohen *et al* 2017, Anenberg *et al* 2010) with the help of disease-specific integrated exposure-response (IER) functions that were developed by cumulating information about the relative risk from ambient air pollution, household air pollution, active and second hand smoking (Burnett *et al* 2014). Recently, the Global Exposure Mortality Model (GEMM) was developed by using hazard ratio functions only from studies involving ambient air pollution exposure (Burnett *et al* 2018) yielding a global premature mortality burden from NCDs and LRI attributable to ambient $PM_{2.5}$ exposure of 8.9 (95% confidence interval 7.5–10.3) million (Burnett *et al* 2018), which is significantly larger than those reported in earlier studies that used the IER exposure response functions (Lelieveld *et al* 2015, Murray and Collaborators 2016).

Over the last decade, there have been important developments towards estimating $PM_{2.5}$ exposure by fusing data from satellite, chemical transport

modelling and ground based measurements (van Donkelaar *et al* 2010, 2014, Brauer *et al* 2012, Dey *et al* 2012, Saraswat *et al* 2013, Just *et al* 2015, Di *et al* 2016, Chowdhury *et al* 2019, Lelieveld *et al* 2019), resulting in the Data Integration Model for Air Quality (DIMAQ) for modelling ambient $PM_{2.5}$ exposure at high resolution ($0.1^\circ \times 0.1^\circ$) across the globe (Shaddick *et al* 2018). The exposure to ozone (O_3) was estimated using an atmospheric chemistry model (Jöckel *et al* 2010, 2016, Righi *et al* 2015, Yan *et al* 2018).

While the estimates of premature mortality are regularly updated with the evolution of risk functions and exposure assessment techniques, it is important to understand the relative importance of the underlying factors modulating these estimates of premature mortality. Here, we distinguish the contributions by changes in air pollution, demography and patterns of baseline mortality rates in changing the global premature mortality burden attributable to ambient air pollution from 2000 to 2015. A few recent studies have discussed the contributions of these factors but only in a few countries and regions (Cohen *et al* 2017, Butt *et al* 2017). The current study advances this work in several ways. Firstly, we used the new GEMM that estimates age-specific risks for the NCD and LRI for adults and LRI among children. Secondly, we introduced correction factors to methodically disassociate the impacts of changing air pollution exposure on the changes in baseline mortality and from the baseline mortality changes that incorporate the exposure effect. Further, we provide statistics for all countries and major regions worldwide.

2. Methods

Annual population-weighted $PM_{2.5}$ exposure data at $0.1^\circ \times 0.1^\circ$ spatial resolution for the years 2000 and 2015 were taken from the DIMAQ model (Shaddick *et al* 2018) which combines estimates from about 9690 ground-based monitoring locations with output from chemical transport models and satellite retrievals of aerosol optical depth to arrive at the annual ambient $PM_{2.5}$ exposures at $0.1^\circ \times 0.1^\circ$ spatial resolution over the globe. In addition, the ECHAM/MESSy Atmospheric Chemistry (EMAC) model (Jöckel *et al* 2010) was used to estimate the global surface O_3 concentrations for 2000 and 2015. The model was applied to provide hourly O_3 data at the horizontal resolution of 2.8° longitude by 2.8° latitude, with 90 vertical levels (topmost level of 0.01 hPa). The RC1SD-base10a simulation with prescribed seasonally resolved anthropogenic emissions from MACCity (Monitoring Atmospheric Composition and Climate/City) was used (Lamarque *et al* 2010, Granier *et al* 2011). Additional emissions for biomass burning, biogenic VOCs (volatile organic carbon), lightning NOx and volcanic SO_2 were also prescribed to the model. The model was nudged against the meteorological data from ERA-Interim (European Reanalysis-Interim) to represent actual weather conditions rather than climatological values. The model simulated columnar O_3 was extensively evaluated against observations in an earlier study (Jöckel *et al* 2016). The RC1SD-base10a simulation is described in detail by Jöckel *et al* 2016 and references therein. We generated the annual average daily maximum 8-hourly O_3 concentration metric (ADM8h) for each grid by estimating 24 eight-hourly running mean O_3 concentrations for each day, and then the maximum of the 24 eight-hour O_3 concentrations for each day was averaged over the year to obtain the ADM8h metric for a grid.

We used the GEMM to calculate the age-dependent hazard ratios for NCD + LRI in adults (>25 years) and LRI in children below 5 years (Lelieveld *et al* 2018). GEMM was built by incorporating 41 epidemiological studies on ambient air pollution performed across 16 countries, including a study involving Chinese men with long-term ambient $PM_{2.5}$ exposure up to about 84 $\mu g\ (m^3)^{-1}$ (Burnett *et al* 2018). Hazard ratio (HR) for exposure to $PM_{2.5}$ exposure is elaborated as equation (1):

$$HR(e) = \exp\left(\log\left(1+\frac{e}{\alpha}\right) \times \frac{\theta}{1+\exp\left(\frac{-e+\mu}{\nu}\right)}\right),$$
$$e = \max\left(0, PM_{2.5} - 2.4\,\mu g m^{-3}\right), \quad (1)$$

Where, 2.4 $\mu g\ (m^3)^{-1}$ is the counterfactual concentration, below which no risk of premature mortality from $PM_{2.5}$ exposure was considered. θ, α, β and δ are parameters of fit for the GEMM model. These parameters vary with population-age, the values of which are provided in Burnett *et al* 2018. The GEMM model can be utilized to estimate HR for NCD + LRI at 5 year-interval age classes, enabling our analysis of changing age distributions, which was not possible in this way in previous studies (Cohen *et al* 2017, Butt *et al* 2017) as the IERs are limited to estimate relative risk for ischemic heart disease and stroke only.

For exposure to O_3, we estimated the HRs of premature mortality due to respiratory illness (ICD-10 codes: J00-J99) using the coefficients provided by (Turner *et al* 2016) using equation (2)

$$HR = \exp^{\beta \Delta x}, \quad \Delta x = \max\left[0, (O3 - LCC)\right]. \quad (2)$$

(Turner *et al* 2016) recently updated the American Cancer Society- Cancer Prevention Study II, with a longer population follow-up and larger study-population than (Jerrett *et al* 2009). For the current study, we estimated β by taking *ln*-HR of 1.12(95% CI: 1.08, 1.16) for a 10 ppb increase in O_3 from a model (Turner *et al* 2016), adjusted for near-source $PM_{2.5}$, regional $PM_{2.5}$ and NO_2. The β obtained was then used to obtain the *HR* for exposure to O_3 using two low-concentration cut-offs (LCCs: 26.7 ppb and 31.1 ppb).

Table 1. Formulation of the scenarios. AF is the attributable fraction, which may be written as $AF = \frac{HR-1}{HR}$. AF_{2000} and AF_{2015} are the attributable fractions for 2000 and 2015. P_{2000} and P_{2015} are the exposed population for 2000 and 2015 respectively. $P_{size2015}$ is the population size for 2015 adjusted for the age structure of 2000. $P_{age2015}$ is the population size for 2000 adjusted for the age-structure of 2015. BM_{2000} and BM_{2015} are the baseline mortality for 2000 and 2015 respectively. $BM_{2000}{}^{**} = kb \times BM_{2000}$, $BM_{2015}{}^{**} = ka \times BM_{2015}$. ka and ka are described in details in the SI section S1.

Scenario Name	Exposure to $PM_{2.5}$	Exposure to O_3	% change in premature mortality
BMOR	yes	yes	$\left\{\frac{[AF_{2000}\times BM_{2015}{}^{**}\times P_{2000}]-[AF_{2000}\times BM_{2000}\times P_{2000}]}{[AF_{2000}\times BM_{2000}\times P_{2000}]}\right\}\times 100$
POPS	yes		$\left\{\frac{[AF_{2000}\times BM_{2000}\times P_{size2015}]-[AF_{2000}\times BM_{2000}\times P_{2000}]}{[AF_{2000}\times BM_{2000}\times P_{2000}]}\right\}\times 100$
POPA	yes		$\left\{\frac{[AF_{2000}\times BM_{2000}\times P_{age2015}]-[AF_{2000}\times BM_{2000}\times P_{2000}]}{[AF_{2000}\times BM_{2000}\times P_{2000}]}\right\}\times 100$
POPS + POPA		yes	$\left\{\frac{[AF_{2000}\times BM_{2000}\times P_{2015}]-[AF_{2000}\times BM_{2000}\times P_{2000}]}{[AF_{2000}\times BM_{2000}\times P_{2000}]}\right\}\times 100$
EXPO	yes	yes	$\left\{\frac{[AF_{2015}\times BM_{2000}{}^{**}\times P_{2000}]-[AF_{2000}\times BM_{2000}\times P_{2000}]}{[AF_{2000}\times BM_{2000}\times P_{2000}]}\right\}\times 100$

Premature mortality (*Mort*) for exposure to ambient $PM_{2.5}$ and O_3 for the years 2000 and 2015 was then estimated following our earlier work (Lelieveld *et al* 2015, 2018, 2019, Chowdhury and Dey 2016, Chowdhury *et al* 2018, Balakrishnan *et al* 2019) as follows:

$$Mort = \left(\frac{HR-1}{HR} \times p \times BM\right), \quad (3)$$

Where p is the exposed population. For estimating the age-specific premature mortality due to ambient $PM_{2.5}$, we used the exposed adult population in a country, above 25 years at 5 year intervals and child (0–4 years) population from the United Nations Department of Economic and Social Affairs (UNDESA)—Population Division. For estimating premature mortality due to exposure to O_3 the UNDESA population above 25 years was used. Age-specific baseline mortality rates (BM) for NCD, LRI (for exposure to $PM_{2.5}$) and respiratory illness (for exposure to O_3) for each country was obtained from GBD (https://vizhub.healthdata.org/gbd-compare/).

We present the changes in premature mortality (ΔM)[1] due to air pollution exposure in 2015 relative to the baseline year 2000 and attribute the changes in each country to the transition in the four major factors *viz.* baseline mortality rates, population size, population age structure and $PM_{2.5}$ and O_3 exposure. To evaluate the relative importance of the individual factors (table 1), we estimated the burden for 2015 with that factor for 2015 and all others for 2000, so that the difference between premature mortality in 2015 and 2000 represents the impact of the transitional factor on the total premature mortality in 2015. The first case, 'BMOR' was developed to derive the impact of changing baseline mortality from 2000 to 2015. 'POPS' and 'POPA' were formulated to estimate the impacts of population size and population age structure, respectively. Finally, 'EXPO' was developed to estimate the effects of change in $PM_{2.5}$ and O_3 exposure. Since the exposure-response relationship for O_3 is not age-dependent, we calculated the combined impact of demographical change ('POPS + POPA') to premature mortality associated with exposure to O_3. We emphasize that the baseline mortality rate inevitably includes the impact of air pollution exposure. Thus, to obtain meaningful numbers for 'BMOR', we developed the correction factor '*ka*' which distinguishes the impact of air pollution exposure from baseline mortality change. Similarly, for accurately estimating the numbers for 'EXPO', we developed the correction factor '*kb*', which aims to correct the estimate for changing exposure estimates alone, by negating the impact of exposure on baseline mortality. The formulation of the correction factors is presented in the SI section S1 (stacks.iop.org/ERL/15/074010/mmedia). The calculations were carried out for 186 countries and presented in the SI data, while we concentrate the discussion on 19 major continental sub-regions (see figure S1 and section S2 in SI).

All estimates of premature mortality in this study are accompanied by uncertainty ranges, indicated as the 95% Confidence Intervals (CI). For exposure to $PM_{2.5}$, the error in the coefficients of the GEMM function was used to estimate the 95% CI values, being distributed log-normally, and 1000 random draws of the hazard ratios were selected for an exposure estimate in each country. For exposure to O_3, the hazard ratio for an exposure was distributed lognormally, and then 1000 random draws of hazard ratio were selected. The LCC considered for this study was 26.7. Premature mortality estimates associated with O_3 with LCC of 31.1 was found to be ~15% lower (please see SI, section S4 for further details). Similarly, the baseline mortality estimates (central value with 95% CI) obtained from the Global Burden of Diseases initiative (https://vizhub.healthdata.org/gbd-compare/) were distributed log-normally and 1000 random draws were selected for each country. Therefore, from 1000 × 1000 (1000 000) estimates of premature mortality obtained for an air pollutant exposure

[1] $\Delta M = \frac{Premature\ mortality_{2015} - Premature\ mortality_{2000}}{Premature\ mortality_{2000}}$

Table 2. Statistics of premature mortality for 2000 and 2015, % change in premature mortality and the % contribution of the transition factors 'BMOR', 'POPS', 'POPA' and 'EXPO' for exposure to ambient $PM_{2.5}$ in the 15 countries with highest premature mortality in 2015 among the adults, children and the total exposed population.

Country	Region	Exposed population	Total Premature mortality in 2000. (95th CI)	Total Premature mortality in 2015 (95th CI)	% change in total premature mortality in 2015	% change in total premature mortality for 'BMOR'	% change in total premature mortality for 'POPS'	% change in total premature mortality for 'POPA'	% change in total premature mortality for 'EXPO'
China	East Asia	Total	1819 000(1518 000–2101 000)	2406 000(2012 000–2775 000)	32.3	−23.2	24.7	26.2	8.1
		Adult	1758 000(1478 000–2021 000)	2392 000(2003 000–2757 000)	36.1	−21.2	24.6	27.6	8.1
		Child	61 130(39 530–80 280)	13 480(8690–17 940)	−78	−78.8	24.8	−15.8	8.2
India	South Asia	Total	1434 000(1166 000–1685 000)	2121 000(1780 000–2448 000)	47.9	−18.5	33.6	14.5	17.4
		Adult	1254 000(1054 000–1449 000)	2030 000(1720 000–2328 000)	61.9	−14.2	33.4	20.7	18
		Child	179 700(111 620–236 160)	91 000(59 990–119 500)	−49.4	−48.3	34.8	−28.3	13.2
Pakistan	South Asia	Total	223 000(180 000–265 000)	312 000(246 000–385 000)	40.4	−10.4	47.4	0.7	6.5
		Adult	197 000(164 000–229 000)	293 000(236 000–357 000)	49.2	−6.7	47.5	3.1	6.6
		Child	25 750(15 940–35 780)	18 940(10 900–28 290)	−26.5	−38.8	46.5	−17.3	6.1
Bangladesh	South Asia	Total	165 000(133 000–198 000)	234 000(194 000–276 000)	41.9	−33.6	35.9	10.6	33.8
		Adult	131 000(110 000–153 000)	223 000(187 000–260 000)	70.4	−24.9	36	22.1	37.9
		Child	34 230(22 810–45 340)	11 250(7050–15 770)	−67.1	−66.8	35.2	−33.2	18
United States of America	North America	Total	201 000(166 000–236 000)	206 000(169 000–242 000)	2.3	−13.7	15.9	15.3	−12.2
		Adult	201 000(165 000–236 000)	206 000(169 000–242 000)	2.3	−13.7	15.9	15.3	−12.2
		Child	230(140–320)	150(90–200)	−38.5	−28.6	16.8	−12.6	−13.9

Table 2. continued

Country	Region	Exposed population	Total Premature mortality in 2000. (95th CI)	Total Premature mortality in 2015 (95th CI)	% change in total premature mortality in 2015	% change in total premature mortality for 'BMOR'	% change in total premature mortality for 'POPS'	% change in total premature mortality for 'POPA'	% change in total premature mortality for 'EXPO'
Egypt	North Africa	Total	118 000(98 000–138 000)	205 000(174 000–234 000)	73.5	−15.8	52.8	−4.5	40.8
		Adult	106 000(90 000–122 000)	197 000(169 000–224 000)	85.8	−10.5	52.8	−4.7	42.9
		Child	11 700(7390–15 930)	7270(4480–10 270)	−37.9	−63.6	52.7	−2.4	22.3
Russian Federation	East Europe	Total	231 000(192 000–271 000)	204 000(169 000–239 000)	−11.8	−26.8	10.5	8	1
		Adult	230 000(191 000–270 000)	204 000(169 000–239 000)	−11.6	−26.6	10.5	7.9	0.9
		Child	900(560–1220)	380(240–500)	−58.7	−71.3	11.2	31.9	2.9
Nigeria	West Africa	Total	175 000(116 000–243 000)	197 000(130 000–279 000)	12.6	−38.1	49.7	−2.3	27.2
		Adult	89 000(62 000–125 000)	127 000(86 000–183 000)	42.9	−26.3	49.5	−3.2	35.6
		Child	85 600(54 370–117 870)	69 700(44 470–96 700)	−18.6	−50.2	50	−1.3	18.5
Indonesia	South East Asia	Total	127 000(103 000–152 000)	163 000(134 000–194 000)	28.2	−11.5	33.3	6.2	0.7
		Adult	115 000(95 000–135 000)	159 000(131 000–188 000)	38.2	−5.4	33.3	8.3	0.5
		Child	12 270(7460–16 700)	4390(2820–5980)	−64.2	−68.4	33.6	−13.3	2.7
Japan	East Asia	Total	86 000(72 000–101 000)	115 000(96 000–134 000)	33.6	−20.5	6.3	49.1	4.6
		Adult	86 000(72 000–100 000)	115 000(96 000–134 000)	33.6	−20.4	6.3	49.1	4.6
		Child	80(50–110)	50(30–60)	−48	−43.9	5.5	−15.3	5.4
Brazil	South America	Total	90 000(73 000–105 000)	106 000(87 000–124 000)	18.3	−20.5	32.6	31.1	−17
		Adult	84 000(70 000–98 000)	105 000(86 000–123 000)	24.2	−17.7	32.7	35.3	−17
		Child	5280(3210–7120)	1280(810–1750)	−75.8	−65.2	32.2	−35.9	−16.2

Table 2. continued

Country	Region	Exposed population	Total Premature mortality in 2000. (95th CI)	Total Premature mortality in 2015 (95th CI)	% change in total premature mortality in 2015	% change in total premature mortality for 'BMOR'	% change in total premature mortality for 'POPS'	% change in total premature mortality for 'POPA'	% change in total premature mortality for 'EXPO'
Ukraine	East Europe	Total	95 000(79 000–111 000)	96 000(80 000–111 000)	0.7	−5.5	0.6	10	−2.8
		Adult	95 000(79 000–110 000)	95 000(79 000–111 000)	0.7	−5.5	0.6	10	−2.8
		Child	150(100–200)	90(60–120)	−43.5	−46.7	−0.9	10.2	−2.2
Germany	West Europe	Total	81 000(67 000–95 000)	90 000(75 000–106 000)	10.9	−19.4	3.5	35.4	−3.3
		Adult	81 000(67 000–95 000)	90 000(75 000–106 000)	11	−19.4	3.5	35.4	−3.3
		Child	30(20–30)	20(10–20)	−29.9	−16.9	4.2	−13.1	−1.8
Vietnam	South East Asia	Total	67 000(54 000–81 000)	86 000(68 000–105 000)	27.8	−13.2	39	7.5	−1.5
		Adult	65 000(53 000–78 000)	85 000(68 000–103 000)	30.1	−12	39	8.4	−1.6
		Child	1970(1220–2750)	990(580–1450)	−49.6	−53.1	39.1	−23.4	2.2
Philippines	South East Asia	Total	48 000(38 000–57 000)	84 000(68 000–101 000)	76.5	11	40.1	19.1	−5.3
		Adult	41 000(34 000–48 000)	79 000(66 000–94 000)	94.7	18.1	40	26.5	−6
		Child	6850(4390–9300)	4710(2870–6820)	−31.2	−30.7	40.2	−24.9	−1.3
World	World	Total	**6874 000(5571 300–8157 600)**	**8890 000(7292 300–10494 000)**	**29.3**	**−21.7**	30.3	**16.5**	**8.9**
		Adult	**6213 000(5158 600–7259 400)**	**8517 800(7026 000–9975 400)**	**37.1**	**−18.2**	29.1	**20.1**	**8.7**
		Child	**661 000(412 700–898 160)**	**371 200(232 060–518 220)**	**−43.8**	**−54.7**	**42.1**	**−16.9**	**11.4**

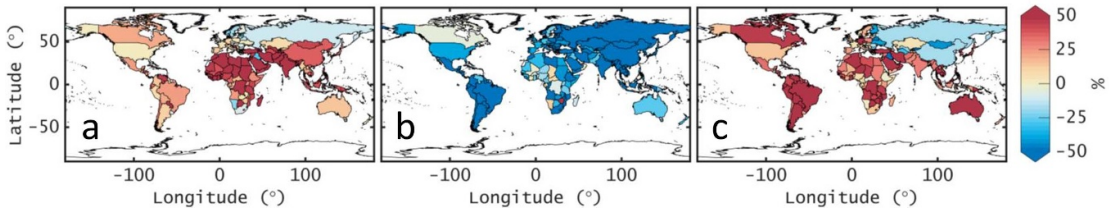

Figure 1. Percentage changes from 2000 to 2015 in (a) adult premature mortality due to ambient $PM_{2.5}$ exposure, (b) child premature mortality due to ambient $PM_{2.5}$ exposure, and (c) adult premature mortality due to O_3 exposure.

for a country, the medians (95% CI) are presented here. A sensitivity study was carried out by distributing the baseline mortality rates normally and with 1000 random draws for estimating premature mortality. The difference between this and selecting 1000 draws by distributing baseline mortality rate log-normally (as originally used for this work) appeared to be small (<1% globally). Multiple levels of uncertainty can arise while estimating premature mortality. The estimates provided in this study have the following limitations. The integral $PM_{2.5}$ exposure was considered for the analysis, although there are indications of differences in toxicological impact of particles originating from different source categories. For example, diesel exhaust particles may be more toxic than aeolian dust; which was not considered in this study as there is no quantitative evidence for a possible differentiation that could be applied to our results. The results presented in this study are solely based on changes between 2000 and 2015. Year-to-year variability in air pollution exposure, baseline mortality and demography are not addressed.

3. Results

3.1. Premature mortality changes attributable to $PM_{2.5}$ and O_3 exposure

The global premature mortality attributed to ambient air pollution exposure increased by 30% from 6.87 (95% CI 5.57–8.15) million in 2000 to 8.89 (7.29–11.04) million in 2015, with strong regional heterogeneity (table S1, figure S2). Large increases are found in South (46.1%), East (32.4%) and West (42.1%) Asia, most of Africa (24%–63%) except Southern Africa (decrease of 9.1%). On the contrary, the premature mortality burden decreased in East and North Europe (7.2% and 8.2%, respectively) and increased slightly over North America (3.5%). We reiterate that our estimates of premature mortality are significantly higher than the GBD estimates (Stanaway *et al* 2018) because of the use of GEMM (for more details see section S3 in SI).

For the adult population only, premature mortality from ambient $PM_{2.5}$ exposure increased by 37.1% from 6.21 (5.15–7.25) million in 2000 to 8.51 (7.02–9.97) million in 2015 (figure 1(a) and table S2). Regionally, the adult premature mortality burden increased by 60.8%, 49.6%, 36.8% and 36% in South, West, South East and East Asia, respectively, while it decreased in North and East Europe and Southern Africa by 8.1%, 7% and 6.2%, respectively. In the rest of Africa it was estimated to increase greatly by ~50% (table S2). Table 2 lists the top 15 countries (see SI for statistics of all countries) by premature adult mortality from exposure to ambient $PM_{2.5}$ in 2015. China was estimated to have the highest burden with 1.75 (1.47–2.02) million deaths in 2015, an increase by 36.1% from 2000 followed by India with 1.25 (1.78–2.44) million in 2015, an increase by 61.9% from 2000. Egypt and the Philippines experienced very high relative increases (85.8% and 94.7%, respectively) in this period. Overall, we estimate that the mortality burden (figure S3) increased significantly for the age groups above 50 years in all major regions except in Europe.

On the other hand, premature child mortality from ambient $PM_{2.5}$ exposure decreased globally by 43.8% from 0.66 (0.41–0.89) million in 2000 to 0.37 (0.23–0.51) in 2015 (table S2 and figure 1(b)). A large decrease was found in South (50%), East (77.1%), South-east (59.6%) and West (59.3%) Asia, respectively. In most of Africa, the improvement was not as prominent as in West Africa (18.2%) and Central Africa (18%). Though the child mortality burden decreased by 50% in India, it was still estimated to have the highest number of child premature mortality with 0.09 (0.06–0.11) million in 2015. Nigeria was estimated to have the second largest child premature mortality burden with 0.07 (0.04–0.1) million in 2015, a decrease of 18% since 2000. In high-income countries like Germany, the USA and Japan, estimated child mortality from exposure is negligible compared to the premature adult mortality (table 2). On the other hand, the child mortality burden increased in a few African countries like Zimbabwe (by 75%), Namibia (9.6%), Central African Republic (9%) and Chad (6.5%), see supplementary data.

The mortality burden from O_3 exposure increased globally by 17% from 1.11 (0.79–1.42) million in 2000 to 1.3 (0.93–1.68) million in 2015. It should be noted that we estimate a larger premature mortality burden from exposure to O_3 compared to earlier studies which used an older exposure response function (Jerrett *et al* 2009, Anenberg *et al* 2010, Cohen *et al*

2017) but our numbers for respiratory illness mortality burden from O_3 exposure are comparable with a recent study (Malley *et al* 2017). A detailed discussion on choice of risk function for estimating premature mortality associated with exposure to O_3 is provided in SI section S4. India was estimated to have the highest premature mortality burden due to O_3 exposure in 2015 at 0.54 (0.40–0.67) million, an increase of 28% from 2000, followed by China with 0.23 (0.16–0.3) in 2015, a decrease of 18% from 2000. Statistics for all countries are provided in the supplementary data. Table S3 lists the premature mortality due to exposure to O_3 from respiratory illness for 2000, 2015 and the ΔM for the major regions. The mortality burden from O_3 exposure increased considerably over the entire continent of Africa, with 15%, 38.4%, 35.2%, 19% and 24.9% increases in East, Central, North, Southern and West Africa, respectively (figure 1(c)). However, in contrast to the trend in ambient $PM_{2.5}$ exposure, East and Central Asia experienced a decrease in premature mortality due to O_3 exposure in this period by 11.7% and 39.5%, respectively. The burden increased in South (71.4%), Central (33.2%) and North (14.9%) America and in most of Europe except in East Europe (17.1% decrease).

3.2. Relative importance of the transitional factors

Knowledge about the relative contributions of the major transitional factors to premature mortality burden changes is critical for air quality mitigation policies. Epidemiologically, the risk of dying from a disease is expected to decrease with improved health care over time (which can include nutritional status and medical care), whereas demographic change in many regions is associated with a shift towards older age groups, enhancing the risk of dying from NCDs. It is a global tendency that morbidity and mortality is shifting from infectious diseases to NCDs, although in Africa the former still dominate. It should be noted that the fractional contributions by the transitional factors are not additive and should be carefully interpreted. The transitional factors are discussed below.

3.2.1. Attribution to changes in baseline mortality rate

The global changes in baseline mortality rates from 2000 to 2015 (case 'BMOR') are associated with a reduction of premature mortality burden due to ambient $PM_{2.5}$ exposure among adults by 18.2% from 6.2 (5.1–7.2) million in 2000 to 5.1 (3.9–6.3) million in 2015, and among children by 54.7% from 0.66 (0.41–0.89) million in 2000 to 0.3 (0.14–0.51) million in 2015 (figure 2). Among adults, the largest benefits were in Southern Africa (−35%), West Asia (−29%) and East Africa (−26.2%), while the Caribbean (3.6%) and Central America (0.9%) benefitted least. In the densely populated South and East Asia, the decreases for adult mortality burden are 14.1% and 21.5%, respectively. However, the impact of improving baseline mortality on reducing premature mortality from 2000 to 2015 is much larger among children than for adults across the globe (figures 2(a) and (b), table S2). The contributions of baseline mortality changes to the changes in burden vary among age groups (figure S4(a)), and closely match the percentage changes in baseline mortality by NCD among adults (figure S4(b)), LRI among adults and children (figure S4(c)). The regional analysis (figures 3(a) and (b)) reveals that in China the changes in baseline mortality reduced the adult and child premature mortality burden by 21.2% from 1.75 (1.47–2.02) million in 2000 to 1.38 (1.08–1.69) million in 2015 and by 78.8% from 0.06 (0.04–0.08) million in 2000 to 1.38 (1.08–1.69) million in 2015, respectively. An increase in baseline mortality among adults increased the premature mortality by 18% in Philippines. The corresponding numbers for the top 15 countries are listed in table 2. The numbers for all the countries are provided in supplementary data.

In the case of O_3 exposure (figure 2(c)), the mortality burden globally decreased by 19.5% from 1.11 (0.79–1.42) million in 2000 to 0.89 (0.56–1.27) million in 2015 due to improvement in baseline mortality. Large improvements were estimated (figure 3(c), table S3) in East (33.2%), Central (39.5%) and South Asia (17.9%) and all of Africa (by 8–40.7%) while the burden increased in West Europe (7%), South Europe (16.3%), North America (10.8%), South America (8.6), Central America (5.6%) and Oceania (10.5%) due to an increase in baseline mortality in these regions.

3.2.2. Attribution to changes in population size and age structure

Globally, the growing population size in this period (case 'POPS') increased the premature mortality among adults by 29.1% from 6.21 (5.15–7.25) million in 2000 to 8.02 (6.64–9.38) million in 2015 and among children by 42.1% from 0.66 (0.41–0.89) million in 2000 to 0.93 (0.59–1.27) million in 2015. Regionally, the impact of change in population size on ΔM among adults and children in most of Africa is large with 58% and 60.6%, 53.7% and 53.6%, 51.5% and 51.5%, and 50.4% and 50.5% increase in Central Africa, West Africa, East Africa and North Africa, respectively (figure 2(a) and table S2). The same is the case for low- and middle-income countries like Egypt (52.8%) Nigeria (49.5%), Pakistan (47.5%) and India (33.4%) (table 2 and figures 4(a) and (b)). In East and South Asia, growing population increased the burden by 23.6% and 36.1% among adults and 24.8% and 37.1% among children, respectively. In contrast, the growing population only increased the adult mortality burden by 10.9%, 9%, 7.4% and 7.3% and the child mortality burden by 10.6%, 7.5%, 6.4% and 8.3% in North, South, East and West Europe, respectively (table S2, figures 2(a) and (b)). In high-income

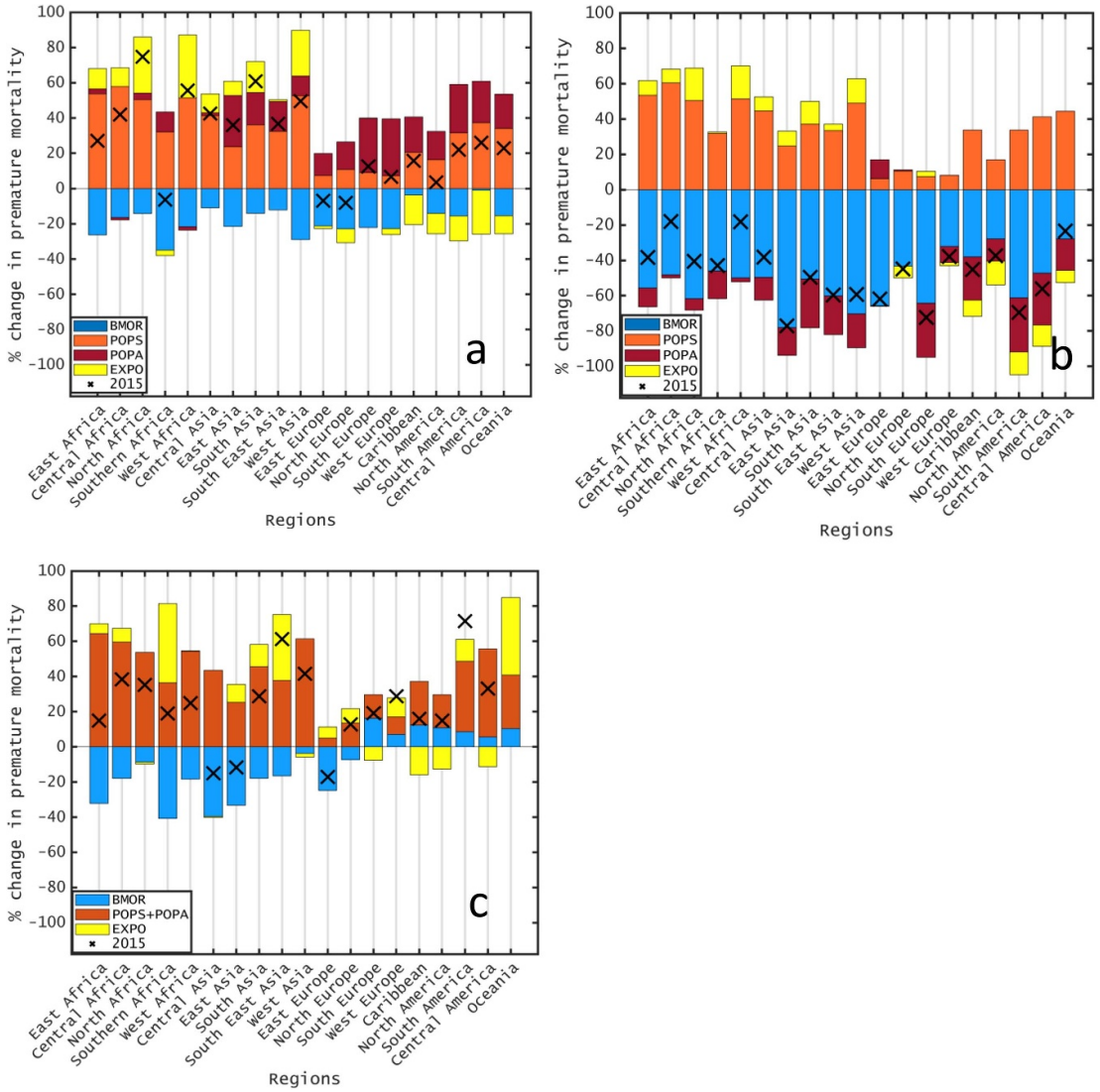

Figure 2. Attribution of changes in premature mortality burden from 2000 to 2015 to transitional factors in all regions due to (a) ambient $PM_{2.5}$ exposure among adults and (b) children, and (c) to ambient O_3 exposure. 'X' imbedded in the bars indicate the percentage change in total premature mortality in 2015 compared to 2000.

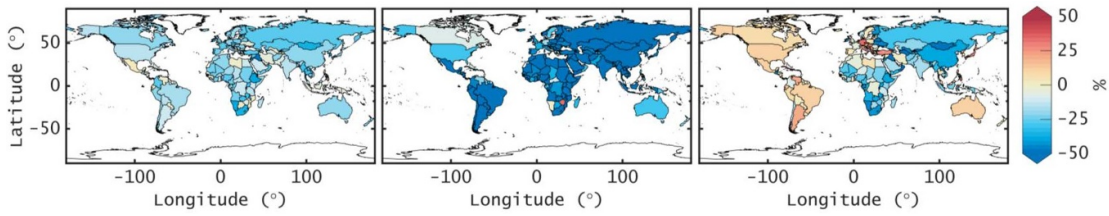

Figure 3. Impacts of changes in baseline mortality rate ('BMOR' case) on premature mortality burden change from 2000 to 2015 attributable to (a) ambient $PM_{2.5}$ exposure among adults and (b) children, and to (c) O_3 exposure.

countries like Japan, Germany and the USA, with small population growth rates, the contribution of population size is relatively minor (table 2).

Due to the strong age dependency of the NCDs and LRI risk functions, any substantial change in the population age structure is expected to impact premature mortality burden due to $PM_{2.5}$ exposure. Unlike the impact of growing population, the shift in age structure does not have a unidirectional influence. For example, in Europe and North America, a considerable shift in age distribution towards an older population resulted in a decrease in the lower (<45 years) and an increase in older age (>45 years) groups (figure S5(a)). Globally, the changing age distribution (case 'POPA') increased the mortality burden due to

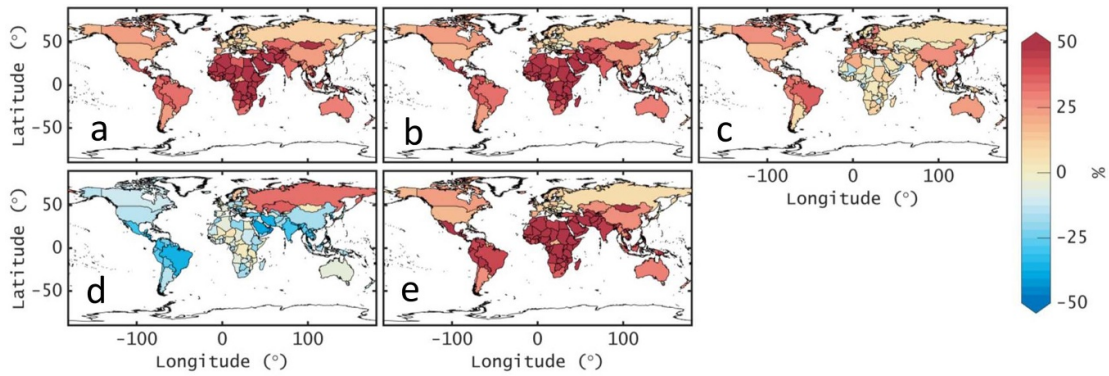

Figure 4. Changes (in %) in mortality burden by ambient $PM_{2.5}$ exposure from 2000 to 2015 due to the changing population size ('POPS') among (a) adults and (b) children, due to the changing age structure ('POPA') among (c) adults and children and (e) by O_3 exposure due to the changing population age and size ('POPS' + 'POPA') from 2000 to 2015.

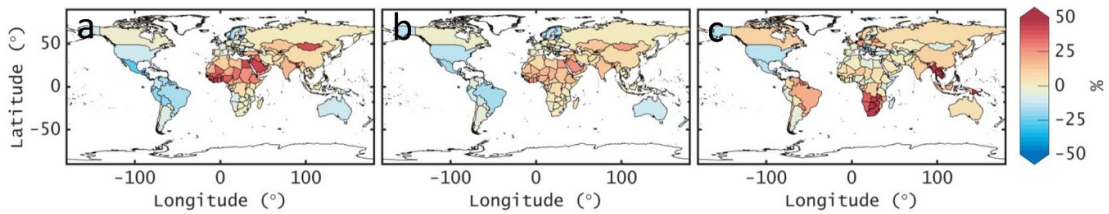

Figure 5. Impact of changing ambient $PM_{2.5}$ exposure on the changes in mortality burden among (a) adults and (b) children and for (c) changing O_3 exposure on the changes in mortality burden among adults from 2000 to 2015.

ambient $PM_{2.5}$ exposure by 20.1% among adults from 6.21 (5.15–7.25) million in 2000 to 7.45 (6.2–8.75) million in 2015, and decreased by 16.9% among children from 0.66 (0.41–0.89) million in 2000 to 0.54 (0.34–0.74) million in 2015. Positive changes among adults are found in South Europe (30.9%), West Europe (32.2%), North Europe (15.5%), North America (16.1%) and South America (27.5%) (table S2, figure 2(a)), which is primarily driven by the aging population older than 45 years old. On the other hand, in most of Africa, a significant increase in the younger population reduced the mortality burden across age groups (table S2, figure S5(b), figure 2(a)). The changes in population age structure increase premature mortality in all regions for the population >45 years and decrease it in the population <45 years of age (figure S5(b)). The change in population age structure leads to reduced premature mortality in 2015 among children except in East Europe, as it acts to increase premature mortality in children by 10.6% (table S2, figures S5(b) and 2(a)) across the globe, except in East Europe, where the age structure shift increased the burden by 10.6%. In China and India (figures 4(c) and (d)), the shifting age structure increased the burden by 27.6% and 20.7% among adults, and decreased it by 15.8% and 28.3% among children, respectively. In countries with rapidly aging populations like Germany and Japan, the changing age structure increased premature mortality in adults significantly by 35.4% and 49.1% respectively (table 2). Statistics for all the countries are provided in supplementary data.

Globally (figure 4(e)), premature mortality due to O_3 exposure was estimated to increase among adults by 36.7% from 1.11 (0.79–1.42) million in 2000 to 1.52 (1.08–1.95) million in 2015 due to the combined change in population size and population age structure ('POPS + POPA'). Regionally (figure 2(c), table S3), the burden from O_3 exposure due to this combined impact increased by more than 40% in most of Africa, South, Central and West Asia. Comparatively, it was estimated to have a smaller influence in Europe (5%, 10%, 13.4%, 13.5% in East, West, South and North Europe, respectively) and North America (18.8%) (table S3).

3.2.3. Attribution to changes in pollution exposure

The global population weighted ambient $PM_{2.5}$ exposure increased by 20.9% from 38.3 μg $(m^3)^{-1}$ in 2000 to 46.4 μg $(m^3)^{-1}$ in 2015 (figure S6(a)). The largest increase was found in West Africa (52.6%) and North Africa (49.5%) followed by South (23.5%) and East Asia (10.8%). In the same period, the exposure decreased by 31%, 19%, 13.6% and 10.3% over Central America, South America, North America and North Europe, respectively, but only marginally (3%–5%) over the rest of Europe. Globally, the change in $PM_{2.5}$ exposure between 2000 and 2015 (case 'EXPO') increased the mortality burden among adults by 8.7% from 6.21 (5.15–7.25) million in 2000 to

6.75 (5.24–8.37) million in 2015 and among children by 11.4% from 0.66 (0.41–0.89) million in 2000 to 0.73 (0.34–1.26) million in 2015. The large increase in $PM_{2.5}$ over North Africa, West Africa and South Asia caused an increase in the premature mortality burden by 31.7%, 35.7% and 17.5%, respectively, among adults, and 18.4%, 18.8% and 12.9% among children. In China, India and Bangladesh, the changes in $PM_{2.5}$ exposure (figures 6(a) and (b)) increased premature mortality by 8.1%, 18%, 37.9% among adults, and 8.2%, 13.2%, 18% among children, respectively. In Germany, the USA and other high-income countries in Europe, reduction in $PM_{2.5}$ decreased the burden by 5%–15%.

Population weighted exposure to O_3 increased by ~7% globally in this period. Large increases occurred over East (4%) and South Asia (5%) and most of Africa (2%–14%). On the other hand, a large decrease (11%) was observed over North America (figure S6(b)). Globally, the changes in O_3 exposure enhanced the burden among adults by 7.6% from 1.11 (0.79–1.42) million in 2000 to 1.22 (0.77–1.75) million in 2015. A notable increase of O_3 pollution in Southern Africa, South East Asia, South Asia, South America, West and North Europe increased the associated premature mortality by 45%, 37.5%, 12.6%, 12.4%, 10.8% and 8.2% respectively (figures 5(c) and 2(c)). In North America, Central America and South Europe negative changes in O_3 exposure decreased premature mortality by 12.6, 11.3% and 7.6%.

4. Discussion

Recently, there have been multiple studies that estimated the premature mortality from ambient $PM_{2.5}$ exposure and O_3 exposure (Silva *et al* 2013, Malley *et al* 2017, Burnett *et al* 2018, Stanaway *et al* 2018, Anenberg *et al* 2018, Lelieveld *et al* 2019, Balakrishnan *et al* 2019). In this study, we estimated the relative changes in premature mortality attributable to ambient $PM_{2.5}$ and O_3 exposure from 2000 to 2015, and subsequently attributed these changes to the changes in baseline mortality rate, population size and age structure, and pollution exposure in this period. The results advance earlier estimates (Cohen *et al* 2017, Butt *et al* 2017) through comprehensive population and age statistics for all countries utilizing the new GEMM hazard ratio functions to estimate risks for all NCDs and LRI due to exposure to ambient $PM_{2.5}$ and the new risk coefficients from (Turner *et al* 2016) for the mortality burden from respiratory illness associated with O_3 exposure. We find that premature mortality due to ambient $PM_{2.5}$ and O_3 exposure increased by about 30% and 17%, respectively, over this time period globally. Three key outcomes of our study are as follows. Firstly, the impacts of changing baseline mortality and population size on percentage ΔM appeared to generally act in opposing directions. Changing baseline mortality over the 15 year period mostly reduced premature mortality, whereas the increasing population size in all regions increased the ΔM. Secondly, the aging population in all regions except Africa caused an increase in premature mortality due to the susceptibility of the advanced-age population to NCD + LRI. The shifting age structure is also associated with a decrease in child mortality from LRI. Apart from the changing age distribution, the population size increased considerably in all regions and was identified as the foremost contributor to increasing premature mortality from air pollution. Thirdly, geographic changes in exposure to air pollutants influenced the premature mortality burden in two directions, enhancing it in low- and middle income countries in Asia and Africa and reducing it in high-income countries. A decrease in O_3 in South Asia resulted in a decrease in premature mortality.

Moreover, we distinguish (section S1 in SI) between the impacts of changing exposure on baseline mortality to attribute the changes in premature mortality burden to baseline mortality alone. Without the correction factor '*ka*', the contribution of the baseline mortality changes to decreasing mortality burden would have been underestimated in North America and South America by ~5% and overestimated over Asia and Africa by 5%–10%. Without the correction factor '*kb*', the impact of changes in $PM_{2.5}$ exposure to the changes in mortality burden would have been underestimated by ~10% in parts of South Asia and North Africa (figures S7(a) and (b)). It was found that improvement in air quality alone could not decrease the mortality burden due to demographic changes. For example, in South America an 18% decrease in ambient $PM_{2.5}$ exposure occurred between 2000 and 2015, but during the same period, the resulting mortality burden increased by 3.9%. The global population has clearly aged, especially in low- and middle-income countries (Kc and Lutz 2014, Chowdhury *et al* 2019), which shifts a larger fraction of the population into the most vulnerable group. Therefore, health benefit analysis for various control measures should consider changes in population size and age structure. Furthermore, rather than adopting standalone policies targeting only mitigation of $PM_{2.5}$ and O_3 exposure, it is important to also accelerate improvements in baseline mortality rates through economic development and primary health care support in order to reduce the global health burden of air pollution.

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Competing Interests

The authors declare no competing interests

Data Availability

All the data produced by this study are available in the Supplementary Data. The codes may be obtained upon reasonable request from the corresponding author.

Data and availability

All the codes and data produced by this work will be available upon request to the authors

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