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Ambient $PM_{2.5}$ Reduces Global and Regional Life Expectancy

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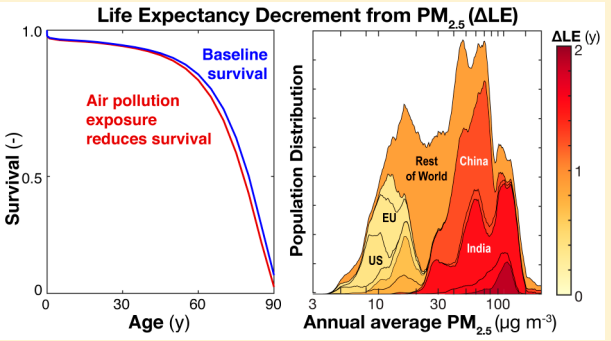
Supporting Information

ABSTRACT: Exposure to ambient fine particulate matter ($PM_{2.5}$) air pollution is a major risk for premature death. Here, we systematically quantify the global impact of $PM_{2.5}$ on life expectancy. Using data from the Global Burden of Disease project and actuarial standard life table methods, we estimate global and national decrements in life expectancy that can be attributed to ambient $PM_{2.5}$ for 185 countries. In 2016, $PM_{2.5}$ exposure reduced average global life expectancy at birth by ∼1 year with reductions of ∼1.2−1.9 years in polluted countries of Asia and Africa. If $PM_{2.5}$ in all countries met the World Health Organization Air Quality Guideline (10 $\mu g\ m^{-3}$), we estimate life expectancy could increase by a population-weighted median of 0.6 year (interquartile range of 0.2−1.0 year), a benefit of a magnitude similar to that of eradicating lung and breast cancer. Because background disease rates modulate the effect of air pollution on life expectancy, high age-specific rates of cardiovascular disease in many polluted low- and middle-income countries amplify the impact of $PM_{2.5}$ on survival. Our analysis adds to prior research by illustrating how mortality from air pollution substantially reduces human longevity.



1. INTRODUCTION

Exposure to ambient fine particulate matter ($PM_{2.5}$) air pollution causes important adverse health outcomes that result in premature death, including ischemic heart disease, strokes, lung cancer, chronic obstructive pulmonary disease, and respiratory infections. Despite the well-documented global burden of disease from $PM_{2.5}$[1−3] (∼4.1 million deaths in 2016),[4] prior research has not systematically explored how global variations in $PM_{2.5}$ exposure affect life expectancy. Here, we use an actuarial modeling approach and data from the Global Burden of Disease (GBD) 2016 study to address the question: "How much does $PM_{2.5}$ air pollution shorten human life expectancy around the world?"

How air pollution affects human longevity has been a topic of continued interest for analysts at the science−policy interface of air pollution over at least the past five decades.[5−13] For the lay public and policymakers alike, health risks that substantially reduce survival time are more compelling than those that merely hasten death by a few days. In the 1980s and 1990s, much research investigated the so-called "harvesting" hypothesis that air pollution might most strongly influence the mortality of those who were already at risk of imminent death.[8−10] By the mid-2000s, the weight of evidence from several large, carefully designed long-term cohort studies suggested a substantial decrement in survival associated with air pollution mortality, because the risks of long-term $PM_{2.5}$ exposure were approximately an order of magnitude greater than risks from day-to-day air quality variation.[9,14] Baccarelli and colleagues demonstrated that the U.S. communities with the most exceptional aging (e.g., populations of >85 or >100) had low ambient air pollution in addition to low rates of smoking, poverty, and obesity, providing suggestive evidence of the benefits of clean air for longevity.[15] Several groups have estimated the relationship between changes in air pollution and changes in life expectancy.[11−13,16,17] For example, Correia et al.[12] used a differences-in-differences approach to model the relationship between $PM_{2.5}$ and life expectancy for 545 U.S. counties and determined that decadal-scale improvements in regional air quality resulted in ∼0.35 year of increased life expectancy at birth per 10 $\mu g\ m^{-3}$ change in $PM_{2.5}$. Similarly, Ebenstein et al. used a regression discontinuity approach to

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evaluate spatial contrasts in PM_{10} across China to estimate ~0.64 year of increased life expectancy per 10 $\mu g\ m^{-3}$ increment in PM_{10}.[12,13]

The advent of globally consistent age-resolved estimates of mortality from $PM_{2.5}$ from the Global Burden of Disease (GBD) collaboration[3,4] now enables systematic assessment of the global variation in the decrement from air pollution. Here, we build upon the actuarial approach of Brunekreef, Dockery, and Pope, who combined data on baseline survival curves with illustrative examples of excess mortality risk from $PM_{2.5}$ to arrive at approximate estimates of life expectancy decrements for simplified exposure scenarios.[10,18]

2. MATERIALS AND METHODS

2.1. Estimation Approach. We used a standard life table method[19] to estimate the baseline life expectancy at birth e_0 for each of 185 countries. For each country, we estimated abridged (i.e., multiyear interval) life tables using all-cause death rates for 23 age strata in the Global Burden of Disease 2016 data set. The Supporting Information describes procedures for computing life tables and baseline life expectancy from age-specific death rates. The standard life expectancy at birth (e_0) can be interpreted as the expected lifespan for an individual born into a population where current age-specific death rates are held constant over time. As such, the life expectancy metric is an actuarial construct (in reality, the structure of mortality for most populations is dynamic) but one that usefully summarizes the comparative survival of different populations in time and space. Our analyses emphasize one common life expectancy metric, life expectancy at birth (e_0). We refer to the general concept of life expectancy with the acronym LE and to decrements in life expectancy at birth that arise from pollution exposure as ΔLE.

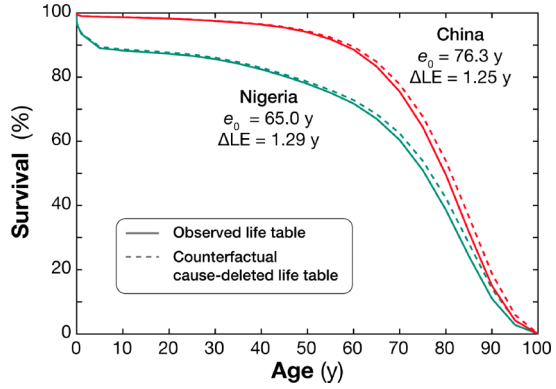

Figure 1. Example survival curves for observed life tables (solid lines) and simulated cause-deleted life tables (dashed lines) where ambient $PM_{2.5}$ exposure is eliminated as a mortality risk factor. Life expectancy e_0 can be visualized as the integral of the survival curve over the age spectrum. Life expectancy for the counterfactual case is increased after removing $PM_{2.5}$ as a mortality risk. For a given country, the reduction in life expectancy attributed to $PM_{2.5}$ (ΔLE) relative to a counterfactual scenario with no excess mortality risk from $PM_{2.5}$ can be visualized as the area between the solid and dashed curves.

We use a cause-deleted life table approach to simulate the life expectancy decrement that is attributable to $PM_{2.5}$ risk factors.[10,18−20] This approach involves four steps: (i) estimating the age-specific death rate attributable to ambient $PM_{2.5}$ for each location, (ii) assuming that in the absence of this risk factor, age-specific death rates would be proportionally lower, (iii) recomputing a counterfactual "cause-deleted" or "cause-eliminated" life table that would exist in the absence of this risk factor (see the Supporting Information),[19,20] and (iv) estimating the counterfactual life expectancy at birth (e_0'). The life table approach requires an assumption that the baseline health status of those who die prematurely from air pollution is similar to that of the general population.[10,18−20] Finally, we attribute the difference between the baseline life expectancy and the cause-deleted counterfactual life expectancy to the life expectancy decrement caused by $PM_{2.5}$: $\Delta LE = e_0' - e_0$. Figure 1 illustrates baseline and cause-deleted survival curves for two countries.

2.2. Attributable Mortality. We used data from the Global Burden of Disease 2016 study to obtain age-specific attributable death rates for each country for ambient $PM_{2.5}$. Briefly, the GBD approach[3,21] involves estimating age-specific attributable mortality for each analysis region as the product of (i) age-resolved populations, (ii) age-specific background disease rates for five key causes, and (iii) regionally population-weighted age-specific population attributable fractions for $PM_{2.5}$ mortality for each of the five causes, computed on the basis of nonlinear integrated-exposure-response (IER) functions[22,23] and a 0.1° × 0.1° gridded $PM_{2.5}$ exposure surface.[24−26] The five GBD causes of death for which $PM_{2.5}$ is a risk factor are ischemic heart disease, cerebrovascular disease (stroke), chronic obstructive pulmonary disease, lung cancer, and lower respiratory infections. As described by Cohen et al.,[3] relative risks for the IER functions are estimated relative to a distribution of the theoretical minimum risk exposure level (TMREL) that ranges from 2.4 to 5.9 μg of $PM_{2.5}$ m^{-3}, consistent with the lowest concentrations observed in long-term epidemiological studies. We input age-specific mortality into our life table analysis as the sum of the five cause-specific death rates. Uncertainties in the GBD approach include (i) the fact that underlying cause-specific mortality data are modeled and therefore uncertain for some countries,[27] (ii) contributions to attributable mortality from diseases other than the five major causes considered here, and (iii) the assumption that the IER functions reasonably describe mortality risk from $PM_{2.5}$ over the full ambient concentration spectrum.[21,28] In addition to using the published GBD attributable death rates for ambient $PM_{2.5}$, we obtained[4,29] similar age-specific mortality data sets for other risk factors (e.g., tobacco smoking) and other major causes of death (e.g., cancers) to provide comparison and context.

Following Apte et al.,[21] we performed a mortality analysis for ambient $PM_{2.5}$ wherein we simulate the disease burden for alternative hypothetical exposure distributions where global $PM_{2.5}$ is limited to specific target concentration(s). To do so, we updated the gridded $PM_{2.5}$ mortality model of Apte et al.[21] with year-2016 data to reproduce the central-tendency GBD 2016 results for ambient $PM_{2.5}$ to within ±1−2% for each country. We then re-estimated attributable mortality for $PM_{2.5}$ under hypothetical scenarios in which the ambient exposure concentration distribution for each grid cell was assigned to an alternative concentration, such as the World Health Organization (WHO) annual-average air quality guideline $PM_{2.5}$ concentration of 10 $\mu g\ m^{-3}$.

3. RESULTS AND DISCUSSION

In 2016, global the population-weighted median life expectancy at birth was 72.6 years [interquartile range (IQR) of 68.2−76.3 years]. For high-income countries, the average life

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Table 1. Global and Regional Life Expectancy and Life Expectancy Decrements for Selected Risk Factors and Causes of Death

	global	East Asia	South Asia	North Africa and Middle East	sub-Saharan Africa	Latin America	high income
baseline LE (years)	72.5	76.3	68.7	73.1	62.8	75.8	80.9
all air pollution	1.65	1.90	2.54	1.54	1.97	0.73	0.40
ambient $PM_{2.5}$	1.03	1.24	1.56	1.29	0.94	0.54	0.37
ambient ozone	0.05	0.07	0.10	0.03	0.01	0.02	0.03
household air pollution	0.72	0.71	1.22	0.30	1.32	0.20	0.01
tobacco	1.82	2.39	1.51	1.60	0.73	1.23	1.82
water sanitation	0.57	0.02	1.02	0.19	1.53	0.13	0.01
dietary risks	2.67	3.10	2.58	3.13	1.54	1.82	1.91
unsafe sex	0.37	0.08	0.16	0.04	2.03	0.27	0.07
all cancer	2.37	3.03	1.26	1.70	1.52	2.31	3.53
lung cancer	0.41	0.67	0.12	0.26	0.09	0.26	0.72
breast cancer	0.14	0.09	0.10	0.14	0.12	0.16	0.23

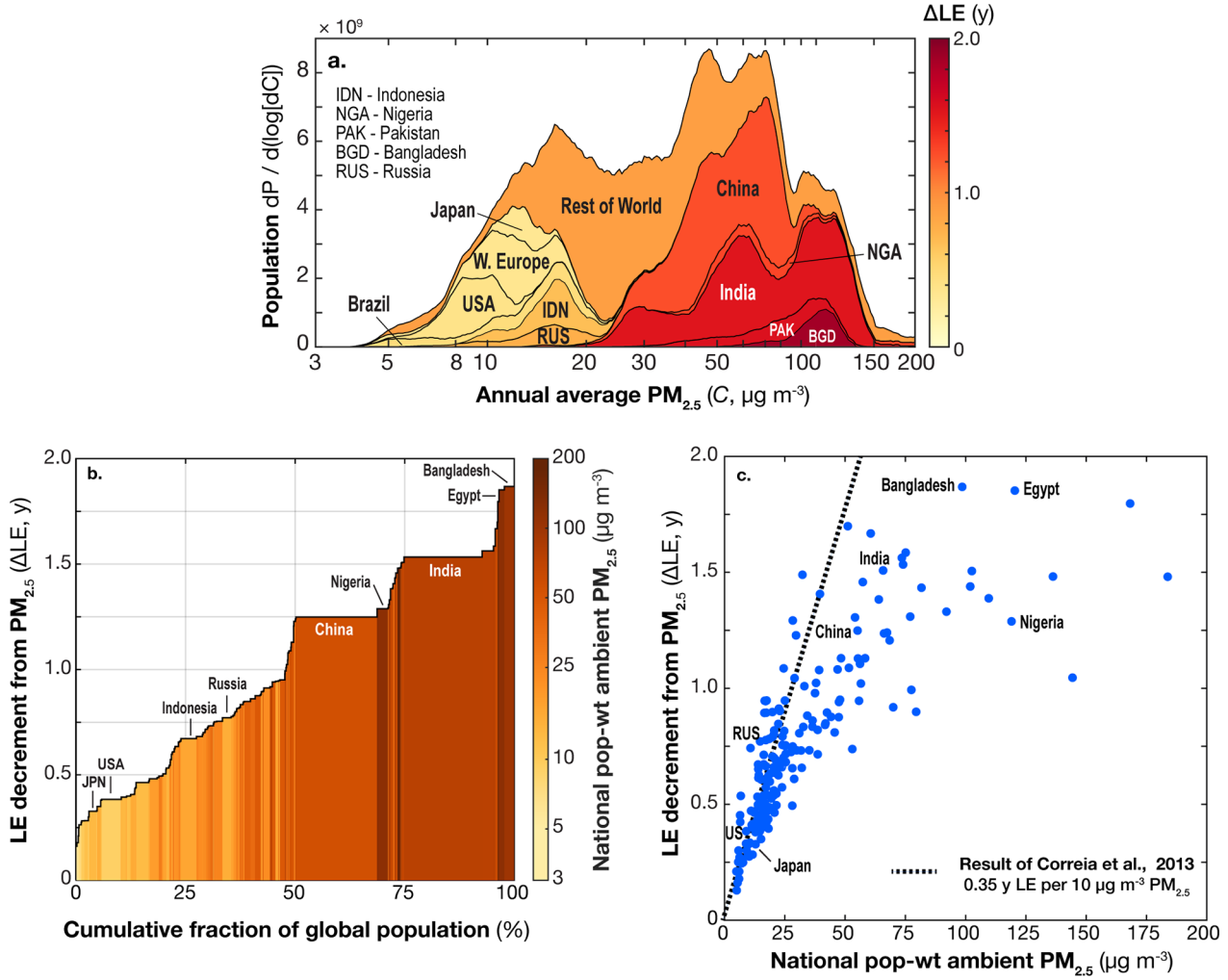

Figure 2. Relationship among the global distribution of ΔLE, the life expectancy decrement from $PM_{2.5}$, and global $PM_{2.5}$ concentrations *C*. ΔLE is generally higher in countries with higher $PM_{2.5}$ levels. (a) Global distribution of population with respect to annual-average $PM_{2.5}$ for year 2016. Plotted data reflect local smoothing of bin-width-normalized distributions computed over 400 logarithmically spaced bins: equal-sized plotted areas reflect equal populations. Each country is colored proportionally to the ΔLE from $PM_{2.5}$ exposure. (b) Cumulative distribution of ΔLE over the global population. The global population-weighted median value for ΔLE is 1.22 years, corresponding to conditions in China. Shading for each country shows the national population-weighted mean $PM_{2.5}$, illustrating how ΔLE has a strong but imperfect association with $PM_{2.5}$. (c) National decrements in ΔLE vs $PM_{2.5}$. Owing to the supralinear concentration−response relationship of mortality with $PM_{2.5}$, the slope of this distribution is higher for countries with lower average $PM_{2.5}$ concentrations.

expectancy was 80.9 years (Table 1). The lowest life expectancies are generally in sub-Saharan Africa (average of ~62.8 years). Global population exposures to ambient $PM_{2.5}$ are unequally distributed (Figure 2a). For the 2016 population,[26] 95% of the global population lived in regions where $PM_{2.5}$ concentrations exceed the WHO guideline concentration of 10 $\mu g\ m^{-3}$. Ambient $PM_{2.5}$ concentrations for the 10th and 90th percentiles of the global concentration−

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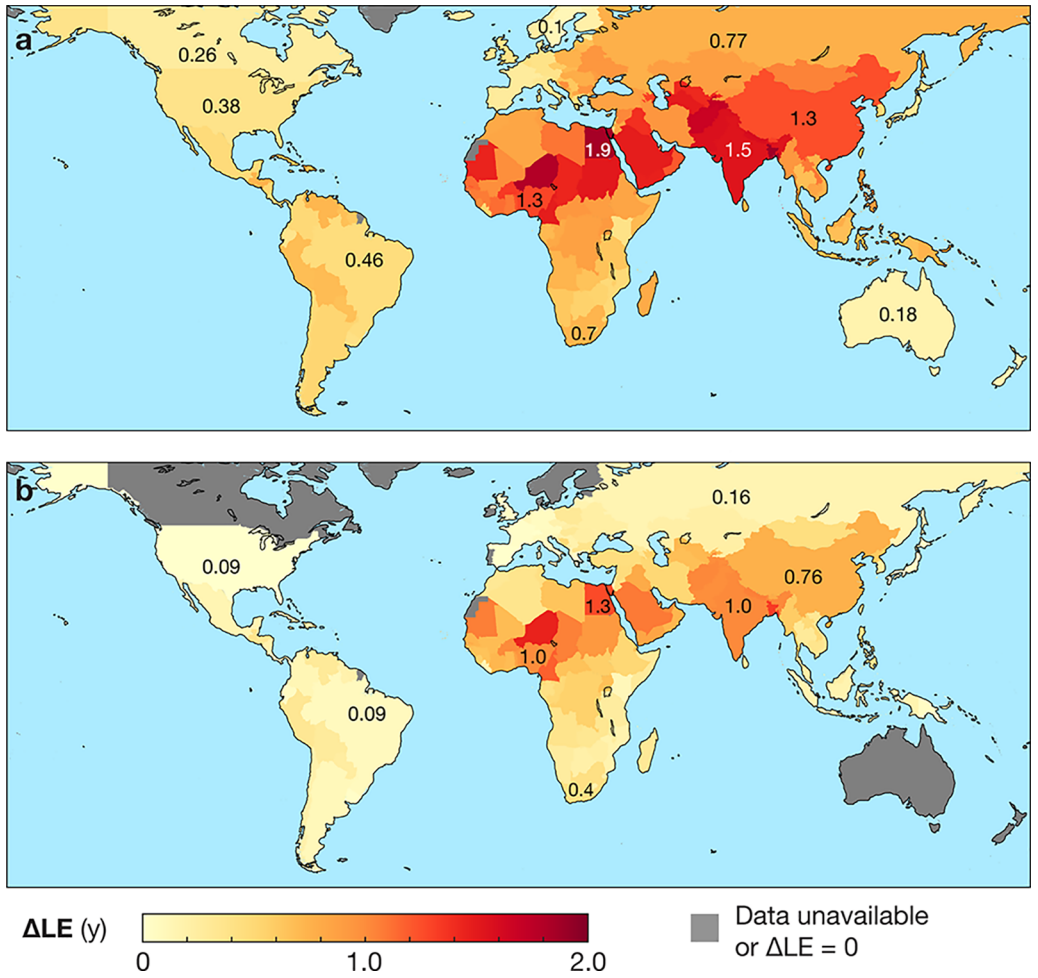

Figure 3. Global maps of the life expectancy decrement ΔLE from $PM_{2.5}$. Panel a shows baseline ΔLE for year-2016 concentrations (global population-weighted mean and median of 1.03 and 1.22 years, respectively). Panel b shows hypothetical gains in life expectancy for an alternative exposure distribution where concentrations are limited to a maximum of 10 $\mu g\ m^{-3}$, the WHO air quality guideline concentration (global-average ΔLE of ~0.59 year). See also Table S2.

population distribution span nearly an order of magnitude (11 and 97 $\mu g\ m^{-3}$, respectively).

Figures 2 and 3 show the global distribution of life expectancy impacts from $PM_{2.5}$. Globally, ambient $PM_{2.5}$ pollution was associated with a population-weighted mean decrement in global life expectancy of 1.03 years. Among 185 countries, the population-weighted median decrement in life expectancy from $PM_{2.5}$ (ΔLE) was 1.22 years [IQR of 0.67−1.51 years (Figure 2b)]. As shown in Figure 3, the life expectancy impact of ambient $PM_{2.5}$ is especially large in polluted countries of Asia, Africa, and the Middle East, including Bangladesh (1.87 years), Egypt (1.85 years), Pakistan (1.56 years), India (1.53 years), Saudi Arabia (1.48 years), Nigeria (1.28 years), and China (1.25 years).

Life expectancy decrements from $PM_{2.5}$ are positively correlated with national-average $PM_{2.5}$ concentrations [r = 0.79 (Figure 2c)]. For countries with $PM_{2.5}$ concentrations below 25 $\mu g\ m^{-3}$ (including nearly all high-income countries), ΔLE and population-weighted mean $PM_{2.5}$ track closely and approximately linearly, with a slope that is roughly consistent with the directly measured relationship between LE and $PM_{2.5}$ of Correia et al. (average of 0.35 year LE increase per 10 $\mu g\ m^{-3}$ reduction in exposure for 545 U.S. counties).[12] At higher concentrations, the nonlinear integrated exposure−response functions used to estimate mortality attributable to $PM_{2.5}$ generally lead to a decreasing marginal risk change per increment in $PM_{2.5}$. Further, national differences in the structure of underlying disease burden modulate the relationship between $PM_{2.5}$ and life expectancy, contributing to the scatter in Figure 2c. In particular, ΔLE from $PM_{2.5}$ is sensitive to age-specific death rates in each country, while death rates from $PM_{2.5}$ (but not ΔLE) are also strongly influenced by the age distribution of a country's population (see Figure S1).

To place our findings in context, we used published GBD cause- and age-specific mortality data to estimate the life expectancy decrements that are attributable to other key diseases and risks (Table 1). Relative to our core global finding of a global mean ΔLE of 1.03 years for ambient $PM_{2.5}$, the full set of air pollution risk factors [including ambient O_3 and household air pollution (HAP)] decreases global life expectancy by an average of 1.65 years. In regions where both ambient $PM_{2.5}$ and HAP are major risk factors, the ΔLE for the combined set of household and ambient air pollutants is even larger (2.5 years in South Asia and 2.0 years in sub-Saharan Africa). For context, other major global risk factors for reduced life expectancy include dietary risks (2.7 years), tobacco smoking (1.8 years), unsafe water and sanitation (0.57 year), and unsafe sex (0.37 year). Globally, cancers result in ~2.4

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years of reduced life expectancy, while the most common cancer types (e.g., lung and breast) individually reduce life expectancy by ∼0.2−0.4 year. In the United States, the ΔLE for $PM_{2.5}$ (0.38 year) is substantially larger than the impact of breast cancer (0.23 year), while in South Asia, the ΔLE for $PM_{2.5}$ (1.6 years) substantially exceeds the combined impact of all cancers (1.3 years). In short, the burden of disease from air pollution results in life expectancy decrements of a magnitude similar to those of other high-priority risk factors and diseases.

Because air pollution has a disproportionate effect on the elderly, air pollution reduces life expectancy predominantly by increasing the probability of death above age 60 (Figure S2). We utilized our estimated standard life tables for each country to understand how $PM_{2.5}$ affects survival from age 60 to 85, expressed as the metric ${}_{25}q_{60}$ (Table S1). In high-income countries with a low $PM_{2.5}$, baseline survival rates for this 25-year interval are high (∼50%) and $PM_{2.5}$ exposure reduces ${}_{25}q_{60}$ by ∼3%. In contrast, for high-$PM_{2.5}$, high-mortality countries (e.g., South Asia), ${}_{25}q_{60}$ at baseline is low (∼20−30%) and the impact of $PM_{2.5}$ on elderly survival is quite large. For example, across South Asia, the probability of surviving from age 60 to 85 would have been 20% higher if $PM_{2.5}$ exposure were removed as a mortality risk factor.

To illustrate how improvements in $PM_{2.5}$ might result in increased life expectancy, we estimate ΔLE for alternative global exposure distributions (Figure 3 and Table S2). These simulations must be interpreted with care (see below), as they most properly reflect the LE for a hypothetical alternative reality where the distribution of $PM_{2.5}$ concentrations is held constant over time at a specific value. If $PM_{2.5}$ concentrations worldwide were limited to the WHO air quality guideline concentration of 10 $\mu g\ m^{-3}$, global life expectancy would be on average 0.59 year longer. The benefit of reaching this stringent target would be especially large in countries with the highest current levels of pollution, with approximately 0.8−1.4 years of additional survival in countries such as Egypt, India, Pakistan, Bangladesh, China, and Nigeria. In contrast, many high-income countries already nearly meet the WHO guideline and would have much smaller LE benefits. Because limiting the maximum $PM_{2.5}$ concentration in one area may also have air quality benefits for less polluted surroundings, our estimates may understate the possible LE benefits of reaching specific air quality guidelines. Halving $PM_{2.5}$ globally would increase e_0 globally by 0.33 year, and about 0.40−0.55 year in the most polluted countries of Asia and Africa. These benefits are large in absolute magnitude. However, because the relationship for $PM_{2.5}$ and mortality has a declining slope at higher concentrations,[21−23,28,30−33] the LE benefit of halving $PM_{2.5}$ for the highly polluted countries is only 25−30% of the total national ΔLE for $PM_{2.5}$.

Predictions about possible improvements in life expectancy must be interpreted carefully. Mortality for any risk factor will evolve over time as a result of demographic and epidemiological transitions.[34,35] LE is strongly dependent on age-specific death rates, which tend to decrease over time in countries where general population health is improving. Thus, the life expectancy benefit of $PM_{2.5}$ reductions in many polluted lower- and middle-income countries might be 20−40% lower than our core estimates if those countries also had age-specific death rates that were typical of high-income economies (Table S3). As low-income, high-mortality countries undergo epidemiological transitions, one hallmark is a trend toward "aging" populations: reducing age-specific mortality increases survival to higher ages. Because air pollution disproportionately affects the elderly, the attributable death rate for $PM_{2.5}$ is expected to increase over time in many lower- and middle-income countries where populations are just beginning to "age".[3,21] Thus, the paradoxical result is that per-capita mortality from air pollution may increase in some countries even as its life expectancy impacts fall (see Figure S1). This result can be understood in terms of diminishing returns: as populations live longer, reducing any individual risk factor will have a smaller impact on additional survival, while at the same time, competing risks for mortality will become more important. Reducing air pollution in countries at all levels of economic development could lead to substantial gains in life expectancy, gains on a par with reducing other well-recognized threats to public health.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.estlett.8b00360.

Detailed information about life table methods, supporting figures, and supporting tables (PDF)

Data file with results for 185 countries (XLSX)

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Notes

The authors declare no competing financial interest.

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■ REFERENCES

(1) Lim, S. S.; Vos, T.; Flaxman, A. D.; Danaei, G.; Shibuya, K.; Adair-Rohani, H.; Amann, M.; Anderson, H. R.; Andrews, K. G.; Aryee, M.; et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990−2010: A systematic analysis for the Global Burden of Disease Study 2010. *Lancet* **2012**, *380*, 2224−2260.

(2) Cohen, A. J.; Anderson, H. R.; Ostro, B.; Pandey, K. D.; Krzyzanowski, M.; Künzli, N.; Gutschmidt, K.; Pope, A.; Romieu, I.; Samet, J. M.; Smith, K. The global burden of disease due to outdoor air pollution. *J. Toxicol. Environ. Health, Part A* **2005**, *68*, 1301−1307.

(3) Cohen, A. J.; Brauer, M.; Burnett, R.; Anderson, H. R.; Frostad, J.; Estep, K.; Balakrishnan, K.; Brunekreef, B.; Dandona, L.; Dandona, R.; et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. *Lancet* **2017**, *389*, 1907−1918.

DOI: 10.1021/acs.estlett.8b00360
Environ. Sci. Technol. Lett. 2018, 5, 546−551

(4) Gakidou, E.; Afshin, A.; Abajobir, A. A.; Abate, K. H.; Abbafati, C.; Abbas, K. M.; Abd-Allah, F.; Abdulle, A. M.; Abera, S. F.; Aboyans, V.; et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990−2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* **2017**, *390*, 1345−1422.
(5) Lave, L. B.; Seskin, E. P. Does air pollution shorten lives? In *Statistical and Mathematical Aspects of Pollution Problems*; Pratt, J., Ed.; Marcel Dekker: New York, 1974; pp 223−244.
(6) Lave, L. B.; Seskin, E. P. An analysis of the association between U.S. mortality and air pollution. *J. Am. Stat. Assoc.* **1973**, *68*, 284−290.
(7) Thibodeau, L. A.; Reed, R. B.; Bishop, Y. M.; Kammerman, L. A. Air pollution and human health: a review and reanalysis. *Environ. Health Persp.* **1980**, *34*, 165−183.
(8) Schwartz, J. Harvesting and long term exposure effects in the relation between air pollution and mortality. *Am. J. Epidemiol.* **2000**, *151*, 440−448.
(9) Künzli, N.; Medina, S.; Kaiser, R.; Quenel, P.; Horak, F., Jr.; Studnicka, M. Assessment of deaths attributable to air pollution: Should we use risk estimates based on time series or on cohort studies? *Am. J. Epidemiol.* **2001**, *153*, 1050−1055.
(10) Brunekreef, B. Air pollution and life expectancy: is there a relation? *Occup. Environ. Med.* **1997**, *54*, 781−784.
(11) Pope, C. A.; Ezzati, M.; Dockery, D. W. Fine-particulate air pollution and life expectancy in the United States. *N. Engl. J. Med.* **2009**, *360*, 376−386.
(12) Correia, A. W.; Pope, C. A., III; Dockery, D. W.; Wang, Y.; Ezzati, M.; Dominici, F. Effect of air pollution control on life expectancy in the United States. *Epidemiol.* **2013**, *24*, 23−31.
(13) Ebenstein, A.; Fan, M.; Greenstone, M.; He, G.; Zhou, M. New evidence on the impact of sustained exposure to air pollution on life expectancy from China's Huai River Policy. *Proc. Natl. Acad. Sci. U. S. A.* **2017**, *114*, 10384.
(14) Pope, C. A.; Dockery, D. W. Health effects of fine particulate air pollution: Lines that connect. *J. Air Waste Manage. Assoc.* **2006**, *56*, 709−742.
(15) Baccarelli, A. A.; Hales, N.; Burnett, R. T.; Jerrett, M.; Mix, C.; Dockery, D. W.; Pope, C. A. Particulate air pollution, exceptional aging, and rates of centenarians: A nationwide analysis of the United States, 1980−2010. *Environ. Health Perspect.* **2016**, *124*, 1744−1750.
(16) Chen, Y.; Ebenstein, A.; Greenstone, M.; Li, H. Evidence on the impact of sustained exposure to air pollution on life expectancy from China's Huai River policy. *Proc. Natl. Acad. Sci. U. S. A.* **2013**, *110*, 12936−12941.
(17) Fann, N.; Kim, S.-Y.; Olives, C.; Sheppard, L. Estimated changes in life expectancy and adult mortality resulting from declining $PM_{2.5}$ exposures in the contiguous United States: 1980−2010. *Environ. Health Perspect.* **2017**, *125*, No. 097003.
(18) Pope, C. A.; Dockery, D. W. Air pollution and life expectancy in China and beyond. *Proc. Natl. Acad. Sci. U. S. A.* **2013**, *110*, 12861.
(19) Arias, E.; Heron, M.; Tejada-Vara, B. United States life tables eliminating certain causes of death, 1999−2001. *National Vital Statistics Reports* **2013**, *61*, 9.
(20) Tsai, S. P.; Lee, E. S.; Hardy, R. J. The effect of a reduction in leading causes of death: Potential gains in life expectancy. *Am. J. Public Health* **1978**, *68*, 966−971.
(21) Apte, J. S.; Marshall, J. D.; Brauer, M.; Cohen, A. J. Addressing global mortality from ambient $PM_{2.5}$. *Environ. Sci. Technol.* **2015**, *49*, 8057−8066.
(22) Burnett, R. T.; Pope, C. A.; Ezzati, M.; Olives, C.; Lim, S. S.; Mehta, S.; Shin, H. H.; Singh, G.; Hubbell, B.; Brauer, M.; et al. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environ. Health Perspect.* **2014**, *122*, 397−403.
(23) Nasari, M. M.; Szyszkowicz, M.; Chen, H.; Crouse, D.; Turner, M. C.; Jerrett, M.; Pope, C. A.; Hubbell, B.; Fann, N.; Cohen, A.; et al. A class of non-linear exposure-response models suitable for health impact assessment applicable to large cohort studies of ambient air pollution. *Air Qual., Atmos. Health* **2016**, *9*, 961−972.
(24) Brauer, M.; Freedman, G.; Frostad, J.; van Donkelaar, A.; Martin, R. V.; Dentener, F.; Dingenen, R. v.; Estep, K.; Amini, H.; Apte, J. S.; et al. Ambient air pollution exposure estimation for the Global Burden of Disease 2013. *Environ. Sci. Technol.* **2016**, *50*, 79−88.
(25) Shaddick, G.; Thomas, M. L.; Green, A.; Brauer, M.; van Donkelaar, A.; Burnett, R.; Chang, H. H.; Cohen, A.; van Dingenen, R.; Dora, C.; et al. Data integration model for air quality: A hierarchical approach to the global estimation of exposures to ambient air pollution. *Journal of the Royal Statistical Society. Series C, Applied statistics* **2018**, *67*, 231−253.
(26) Shaddick, G.; Thomas, M.; Amini, H.; Broday, D. M.; Cohen, A.; Frostad, J.; Green, A.; Gumy, S.; Liu, Y.; Martin, R. V.; et al. Data integration for the assessment of population exposure to ambient air pollution for global burden of disease assessment. *Environ. Sci. Technol.* **2018**, DOI: 10.1021/acs.est.8b02864.
(27) Ke, C.; Gupta, R.; Xavier, D.; Prabhakaran, D.; Mathur, P.; Kalkonde, Y. V.; Kolpak, P.; Suraweera, W.; Jha, P.; Allarakha, S.; et al. Divergent trends in ischaemic heart disease and stroke mortality in India from 2000 to 2015: a nationally representative mortality study. *Lancet Global Health* **2018**, *6*, e914−e923.
(28) Marshall, J. D.; Apte, J. S.; Coggins, J. S.; Goodkind, A. L. Blue skies bluer? *Environ. Sci. Technol.* **2015**, *49*, 13929−13936.
(29) Institute for Health Metrics and Evaluation. GBD 2016 Results Tool. http://ghdx.healthdata.org/gbd-results-tool, 2017 (accessed July 11, 2018).
(30) Pope, C. A.; Burnett, R. T.; Turner, M. C.; Cohen, A.; Krewski, D.; Jerrett, M.; Gapstur, S. M.; Thun, M. J. Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: Shape of the exposure−response relationships. *Environ. Health Perspect.* **2011**, *119*, 1616−1621.
(31) Pope, C. A.; Burnett, R. T.; Krewski, D.; Jerrett, M.; Shi, Y.; Calle, E. E.; Thun, M. J. Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: Shape of the exposure-response relationship. *Circulation* **2009**, *120*, 941−948.
(32) Smith, K. R.; Peel, J. L. Mind the gap. *Environ. Health Perspect.* **2010**, *118*, 1643−1645.
(33) Pope, C. A.; Cropper, M.; Coggins, J.; Cohen, A. Health benefits of air pollution abatement policy: Role of the shape of the concentration-response function. *J. Air Waste Manage. Assoc.* **2015**, *65*, 516−522.
(34) Smith, K. R.; Ezzati, M. How environmental health risks change with development: The epidemiologic and environmental risk transitions revisited. *Annu. Rev. Environ. Resources* **2005**, *30*, 291−333.
(35) Salomon, J. A.; Murray, C. J. L. The epidemiologic transition revisited: Compositional models for causes of death by age and sex. *Population & Development Review* **2002**, *28*, 205−228.

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