Air pollution deaths attributable to fossil fuels: observational and modelling study

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ABSTRACT

OBJECTIVES

To estimate all cause and cause specific deaths that are attributable to fossil fuel related air pollution and to assess potential health benefits from policies that replace fossil fuels with clean, renewable energy sources.

DESIGN

Observational and modelling study.

METHODS

An updated atmospheric composition model, a newly developed relative risk model, and satellite based data were used to determine exposure to ambient air pollution, estimate all cause and disease specific mortality, and attribute them to emission categories.

DATA SOURCES

Data from the global burden of disease 2019 study, observational fine particulate matter and population data from National Aeronautics and Space Administration (NASA) satellites, and atmospheric chemistry, aerosol, and relative risk modelling for 2019.

RESULTS

Globally, all cause excess deaths due to fine particulate and ozone air pollution are estimated at 8.34 million (95% confidence interval 5.63 to 11.19) deaths per year. Most (52%) of the mortality burden is related to cardiometabolic conditions, particularly ischaemic heart disease (30%). Stroke and chronic obstructive pulmonary disease both account for 16% of mortality burden. About 20% of all cause mortality is undefined, with arterial hypertension and neurodegenerative diseases possibly implicated. An estimated 5.13 million (3.63 to 6.32) excess deaths per year globally are attributable to ambient air pollution from fossil fuel use and therefore could potentially be avoided by phasing out fossil fuels. This figure corresponds to 82% of the maximum number of air pollution deaths that could be averted by controlling all anthropogenic emissions. Smaller reductions, rather than a complete phase-out, indicate that the responses are not strongly non-linear. Reductions in emission related to fossil fuels at all levels of air pollution can decrease the number of attributable deaths substantially. Estimates of avoidable excess deaths are markedly higher in this study than most previous studies for these reasons: the new relative risk model has implications for high income (largely fossil fuel intensive) countries and for low and middle income countries where the use of fossil fuels is increasing; this study accounts for all cause mortality in addition to disease specific mortality; and the large reduction in air pollution from a fossil fuel phase-out can greatly reduce exposure.

CONCLUSIONS

Phasing out fossil fuels is deemed to be an effective intervention to improve health and save lives as part the United Nations' goal of climate neutrality by 2050. Ambient air pollution would no longer be a leading, environmental health risk factor if the use of fossil fuels were superseded by equitable access to clean sources of renewable energy.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Ambient air pollution is the leading environmental health risk factor for morbidity and mortality

Estimates of the attributable mortality burden differ substantially between studies, primarily due to differences in the exposure-response associations and the causes of death included

Few global studies attributed mortality to specific air pollution sources; their results differ widely

WHAT THIS STUDY ADDS

A new relative risk model optimises the exposure-response association throughout the global range of ambient exposure levels

Estimates of cause specific and all cause mortality from long term exposure to particulate matter with a diameter of <2.5 µm and ozone are attributed to pollution sources

Major reductions in air pollution emissions, notably through a phase-out of fossil fuels, could have large, positive health outcomes

Results show that the mortality burden attributable to air pollution from fossil fuel use is higher than most previous estimates

Introduction

Global air quality guidelines from World Health Organization (WHO) call attention to the huge toll of air pollution on human health, leading to millions of deaths yearly, comparable to tobacco smoking.[1] The 2019 global burden of disease (GBD) study estimated that all forms of air pollution account for about 11.3% of total deaths worldwide for women and 12.2% for men.[2] Improvements to air quality contribute to many of the United Nations' sustainable development goals for 2030, and air pollution is directly mentioned in two targets to achieve these goals.[3 4] Previous studies have suggested that transitioning from fossil fuels to clean, renewable energy sources in the coming decades will help save many lives from air pollution and limit the global mean temperature rise caused by greenhouse gases to below 2°C, thereby meeting the Paris Climate Agreement.[5-7] However, mortality estimates attributable to air pollution and the causes of death vary widely, with few studies estimating the mortality burden from all causes.[8] We assess the consequences

of a fossil fuel phase-out for disease specific and all cause mortality through the concomitant effects of air pollution. The pace and scale of achievement of such a phase-out will depend on the speed of the global transition towards renewable energy sources. Although technically and economically a fossil fuel phase-out, or at least carbon neutrality, is considered feasible by 2050, the process is hampered by a scarcity of global cooperation on policies involving carbon pricing, government regulations, and investments.[9] We did not consider international policies and socioeconomic pathways in this study, however, we aimed to incentivise large, rapid reductions of greenhouse gas emissions by increasing awareness of the public health benefits of concurrent air pollution reductions.

Global health burden studies have attributed four to ten million deaths per year to outdoor (ambient) particulate pollution attributed to $PM_{2.5}$ (particulate matter with a diameter smaller than 2.5 µm), and attributed 0.3-1.3 million deaths per year to ozone (O_3).[8] These wide ranges reflect the number of health outcomes accounted for and the relative risk functions used to perform the calculations. Using the 2018 relative risk model that Burnett and colleagues developed for all cause mortality from global $PM_{2.5}$ exposure,[10] along with that from O_3, we previously estimated 8.79 million (95% confidence interval 7.11 to 10.41) deaths per year.[6] Weichenthal and colleagues refined the exposure-response association and found that the health impacts at low $PM_{2.5}$ concentrations had generally been underestimated.[11] However, Burnett and colleagues' research, published in 2022, found that attributable mortality for heavily polluted air had been overestimated and provided a model optimised for the entire $PM_{2.5}$ concentration distribution.[12] Countries with either low or high $PM_{2.5}$ concentrations typically have different emission characteristics, therefore, re-evaluation of the contributions of fossil fuel use and other sources of air pollution is needed.[13 14] In this article, we apply Burnett and colleagues' new relative risk FUSION model,[12] optimised for health benefits analyses, and we update exposure estimates to $PM_{2.5}$ and O_3. Although climate change due to fossil fuel use may also contribute to mortality, for example, by increasing weather extremes, the present work solely investigates deaths related to air pollution.

Methods

Study design and data sources

We developed a data constrained, global, atmospheric modelling method to compute gaseous and particulate air pollutants and attributed them to source categories.[6] We present new model results for 2019. This year was selected to be consistent with recent disease burden studies (also before covid-19 lockdowns).[15] The exposure to $PM_{2.5}$ for 2019 was calculated using the observational satellite and air quality station (hybrid) data product of Van Donkelaar and colleagues at 10 km global resolution.[16] The atmospheric model was used to compute the fractional changes in $PM_{2.5}$ related to emission sectors based on simulations in which source categories have been sequentially switched off. The fractional changes calculated by the model were then applied to the high resolution observational $PM_{2.5}$ data to calculate exposure reductions according to four scenarios. The first scenario assumes that all fossil fuel related emission sources are phased out. The second and third scenarios assume that 25% (quarter way) and 50% (half way) of the exposure reduction towards the fossil phase-out are realised. The fourth scenario removes all anthropogenic sources for reference, thus, only accounting for natural sources such as aeolian dust, marine and terrestrial biosphere emissions, and natural wildfires. Evaluation of our model has shown that the $PM_{2.5}$ results are in close agreement with the observational data product of Van Donkelaar and colleagues and with measurements in ground based networks.[17 18] The exposure to ozone in 2019 was calculated based on atmospheric model results, as GBD data are available for 2017.[2 19] Since the calculated exposure to ozone depends on model results only, which tend to underestimate local (eg, urban level) ozone trends due to the limited grid resolution, uncertainty is expected to exceed the 95% confidence interval. Nevertheless, deaths attributable to the ozone account for less than 5% of those attributed to $PM_{2.5}$, so the effect of this uncertainty on our mortality

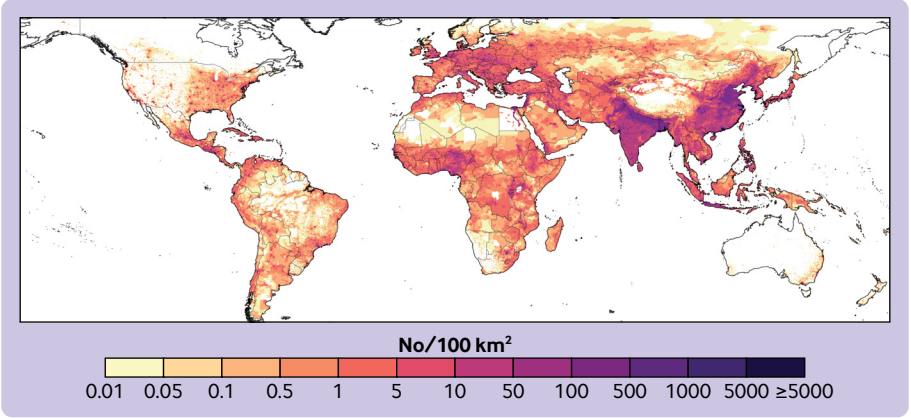

Fig 1 | Annual all cause deaths attributable to fine particulate matter ($PM_{2.5}$) and ozone (O_3) in 2019. Units are numbers per area of 10 km×10 km

assessment is likely to be inconsequential (further discussion later).

Atmospheric model description

Our model for atmospheric composition and climate (known as EMAC) accounts for the global atmospheric circulations. It includes comprehensive routines for atmospheric chemistry and aerosol processes and has been evaluated against observations.[18 20] We used the recent setup described by Kohl and colleagues to represent the meteorology of the year 2019.[21] Newtonian assimilation ("nudging") towards meteorological reanalysis data from the European centre for medium-range weather forecasts was applied.[20 22] Emissions of gaseous and particulate pollutants for 2019 were adopted from the community emission data system (supplementary figure S1).[15] Sources include fossil fuel combustion in sectors such as energy production, industry, land transport, shipping, aviation, residential energy from solid biofuels, waste incineration, agriculture, and solvent production and use. The household combustion of solid biofuels is also a substantial contributor to ambient air pollution, particularly in parts of Asia and Africa. Biomass burning emissions for 2019 were based on satellite data and emission factors,[23] desert dust sources, and natural emissions from vegetation, volcanoes, and marine phytoplankton were calculated online.[24 25] Scenario calculations presented here refer to the same time period, population, and health data, etc. We considered only emission changes while other conceivable future developments, including socioeconomic, healthcare, and demographic pathways, were not taken into account.

All cause and cause specific mortality

We used the FUSION relative risk model of Burnett and colleagues that accounts for six causes of death

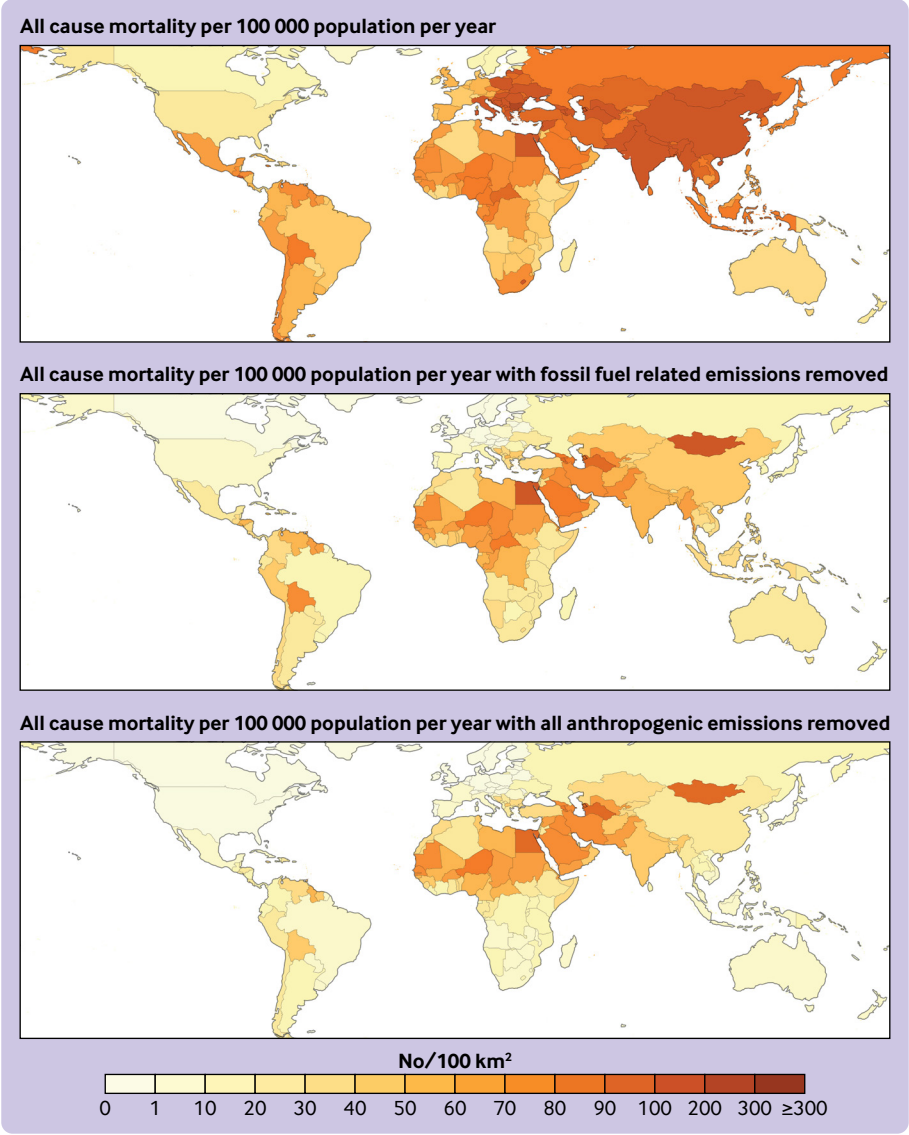

Fig 2 | Country average, all cause deaths per 100 000 population per year attributable to fine particulate matter ($PM_{2.5}$) and ozone (O_3), and with fossil fuel related and all anthropogenic emissions removed

Table 1 | Deaths (95% confidence interval) attributable to long term exposure to $PM_{2.5}$ and ozone (O_3) in thousands per year

Regions	Population (million)	All cause	LRI	IHD	ST	DM	LC	COPD ($PM_{2.5}$)	COPD (O_3)	Sum of disease specific
Central-eastern Europe, central Asia	418 (363 to 473)	477 (303 to 684)	18 (11 to 26)	238 (151 to 334)	77 (42 to 116)	16 (10 to 22)	22 (12 to 33)	14 (9 to 21)	8 (3 to 14)	393 (237 to 566)
Latin America, caribbean	583 (512 to 652)	310 (196 to 448)	32 (18 to 46)	78 (49 to 109)	28 (15 to 43)	42 (25 to 59)	10 (5 to 15)	19 (12 to 28)	7 (2 to 14)	215 (127 to 314)
North Africa and Middle East	608 (526 to 690)	549 (379 to 719)	33 (21 to 43)	279 (202 to 350)	72 (45 to 98)	38 (25 to 49)	17 (11 to 24)	24 (16 to 31)	13 (6 to 22)	476 (325 to 615)
South Asia	1805 (1613 to 2013)	2761 (1936 to 3548)	214 (142 to 272)	928 (708 to 1109)	362 (236 to 465)	179 (122 to 220)	45 (29 to 58)	429 (308 to 528)	187 (83 to 295)	2345 (1629 to 2948)
Sub-Saharan Africa	1078 (931 to 1225)	643 (437 to 850)	195 (124 to 259)	119 (85 to 152)	85 (52 to 117)	54 (35 to 70)	11 (6 to 15)	25 (17 to 33)	6 (2 to 12)	496 (322 to 657)
Southeast and East Asia, Oceania	2158 (1887 to 2418)	3082 (2108 to 4080)	118 (74 to 158)	798 (568 to 1018)	668 (409 to 915)	140 (91 to 183)	243 (149 to 329)	361 (247 to 464)	130 (52 to 225)	2257 (1589 to 3291)
High income countries	1084 (971 to 1195)	542 (286 to 892)	44 (22 to 71)	105 (53 to 172)	41 (19 to 70)	25 (13 to 41)	40 (18 to 69)	31 (16 to 52)	32 (11 to 61)	317 (151 to 536)
Worldwide	7709 (6782 to 8642)	8342 (5632 to 11192)	652 (410 to 872)	2542 (1815 to 3240)	1332 (817 to 1820)	491 (319 to 640)	385 (229 to 539)	903 (624 to 1154)	382 (158 to 642)	6686 (4372 to 8908)

COPD=chronic obstructive pulmonary disease; DM=diabetes mellitus type 2; IHD=ischaemic heart disease; LC=lung cancer; LRI=lower respiratory infections (including children <5 years); ST=stroke.

from long term exposure to $PM_{2.5}$:[12] lower respiratory infections, stroke, ischaemic heart disease, type 2 diabetes, lung cancer, and chronic obstructive pulmonary disease. Estimates of the relative risk of mortality from long term exposure to air pollution were derived from epidemiological cohort studies that account for multiple risk factors, including smoking and unhealthy diets. Cohort members were monitored longitudinally, accounting for causes of death (ie, during multiple years up to decades). The impact of long term exposure to air pollution on cause of death is derived by statistical association with local air quality data. To estimate the global number of deaths attributable to air pollution, both recently and projected according to emission scenarios, the FUSION model describes the shape and magnitude of the association between exposure to $PM_{2.5}$ and all cause and disease specific mortality. Attributable excess mortality is defined by the number of deaths that would not have occurred that year without exposure. Relative risk functions have been derived from a meta-analysis of cohort studies that accounts for the global ambient pollution concentration range.[12]

A common method to estimate the relative risk is by modelling the logarithm of the mortality incidence as a linear function of the concentration of $PM_{2.5}$. FUSION applies an improvement by "fusing" the log-linear function at low concentrations with functions of which the derivatives decline with increasing levels of $PM_{2.5}$, advancing the approach described by Vodonos and colleagues[26] This limits the magnitude of the relative risk at very high concentrations, that is, above those observed in cohort studies. A detailed mathematical description of the method is given by Burnett and colleagues.[12] Attributable deaths were calculated for the population for people older than 25 years and for lower respiratory infections in children younger than five years. FUSION was applied to derive all cause mortality, reported as non-communicable diseases plus lower respiratory infections. We selected FUSION rather than GBD relative risk functions because they allow for calculations of all cause mortality. By contrast, GBD individually accounts for the aforementioned disease categories only. For comparison with our results, we also calculated the attributable mortality using the GBD method.

At both the high and low ends of the ambient $PM_{2.5}$ concentration range, the FUSION model yields larger health benefits from reductions in air pollution compared with the integrated exposure-response

Table 2 | Deaths attributable to long term exposure to $PM_{2.5}$ and ozone (O_3) from fossil fuel use in thousands per year. In parentheses are the percentages of attributable deaths that could be avoided by phasing out fossil fuels relative to the maximum number that could be averted by controlling all anthropogenic emissions

Regions	Population (million)	All cause	LRI	IHD	ST	DM	LC	COPD ($PM_{2.5}$)	COPD (O_3)	Sum of disease specific
Central-Eastern Europe, Central Asia	418	361 (92.4)	12 (92.3)	175 (92.1)	57 (91.7)	10 (92.8)	18 (93.6)	11 (92.6)	7 (92.0)	290 (92.2)
Latin America, Caribbean	582	151 (64.4)	14 (62.4)	36 (62.6)	13 (63.8)	20 (63.0)	5 (63.2)	10 (64.0)	6 (92.0)	104 (64.2)
North Africa and Middle East	608	157 (86.2)	8 (85.9)	70 (85.7)	19 (85.9)	10 (86.5)	6 (88.2)	8 (87.4)	11 (84.4)	132 (85.9)
South Asia	1805	1655 (85.3)	110 (83.4)	538 (86.0)	227 (87.1)	97 (84.5)	27 (86.5)	268 (87.5)	115 (71.8)	1381 (84.7)
Sub-Saharan Africa	1078	163 (51.5)	38 (47.8)	29 (49.9)	23 (51.0)	15 (52.5)	3 (53.1)	7 (52.0)	4 (70.3)	120 (50.4)
Southeast and East Asia, Oceania	2158	2192 (80.7)	70 (70.5)	558 (80.0)	482 (81.5)	86 (72.4)	176 (82.4)	263 (82.7)	111 (87.9)	1746 (80.7)
High income countries	1084	462 (90.3)	36 (86.0)	89 (90.5)	34 (89.1)	20 (89.3)	34 (90.3)	27 (90.7)	31 (96.8)	271 (90.3)
Worldwide	7709	5127 (81.7)	287 (72.4)	1494 (82.6)	853 (82.3)	257 (76.2)	267 (83.6)	592 (84.3)	284 (81.0)	4034 (81.5)

COPD=chronic obstructive pulmonary disease; DM=diabetes mellitus type 2; IHD=ischaemic heart disease; LC=lung cancer; LRI=lower respiratory infections (including children <5 years); ST=stroke.

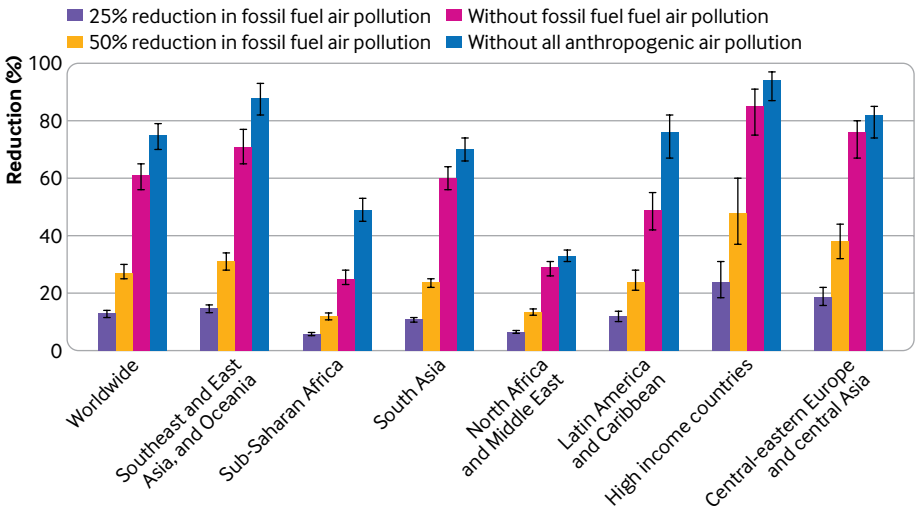

Fig 3 | Percentage reductions in annual deaths attributable to air pollution from removing fossil fuel related and all anthropogenic emissions of $PM_{2.5}$ and O_3 for seven macro-regions and the world. The yellow bars depict the "half way" scenario by assuming that 50% of the fossil fuel phase-out is achieved and the purple bars indicate the "quarter way" scenario by assuming that 25% is achieved. The error bars indicate the 95% CI

functions of the GBD studies. The GBD functions apply proxies (ie, second hand smoke, household pollution, and active smoking) to characterise highly polluted environments, whereas FUSION relies on ambient $PM_{2.5}$ data. FUSION improves a previous version of this method,[10] which was biased by a single cohort at high $PM_{2.5}$ concentrations (higher than 40 μg/m^3). Furthermore, the response at $PM_{2.5}$ concentrations below 40 μg/m^3, especially at low concentrations (<10 μg/m^3), is not as strong as the previous version, although still sensitive to changes at low concentrations. FUSION is particularly suited for global applications, including low, middle, and high income countries and a wide range of exposures. While FUSION applies a counterfactual concentration range between 0 and 5.4 μg/m^3, we aimed for consistency with the GBD and used the 2.4-5.9 μg/m^3 range. Our results show that this leads to a slightly lower global mortality burden than that estimated by Burnett and colleagues.[12]

We maintained consistency with the GBD method for mortality attributable to ozone,[2] with chronic obstructive pulmonary disease being the cause of death, by applying the highest six month average of the daily maximum eight hourly ozone concentration (in parts per billion by volume (ppbv)). The counterfactual concentration (mixing ratio) range was 29.1-35.7 ppbv. Note that these calculations account for only deaths related to chronic obstructive pulmonary disease, hence not for all cause mortality, because most cohort studies only report significance for disease specific results. Although mortality related to ozone that we present here may thus be a lower limit compared with that for all causes, it accounts for less than 5% of the mortality related to $PM_{2.5}$ and therefore is unlikely to significantly affect our results.

Patient and public involvement

No patient involvement was deemed necessary because no individual patient data were used, and the results are primarily relevant to policy makers and the public rather than to individual patients. The research was developed through communication with members of the air quality, climate, and public health communities, but not with patients. We will disseminate results to the public through a press release, social media, and the websites of our research institutions. We will also present results at public conferences and stakeholder meetings.

Results

Global mortality burden

Figure 1 presents global all cause deaths attributable to long term exposure to ambient fine particulate matter ($PM_{2.5}$) and ozone (O_3). South and East Asia have very high numbers. These values are partly related to the large number of people exposed to pollutants in these regions, about 55% of the global population, and to the high levels of air pollution; together, these account for 70% of the attributable deaths worldwide. The mortality burden is also very high in Eastern Europe, the Middle East, and West Africa. From six disease categories, 52% of the mortality is associated with cardiometabolic conditions (ie, diabetes mellitus type 2, stroke, and ischaemic heart disease), with heart disease contributing 30% to the attributable mortality. The sum of cause specific deaths, 6.68 million (95% confidence interval 4.37 to 8.91) deaths per year is lower than the global all cause total of 8.34 million (5.63 to 11.19) deaths per year by about 20%, reflecting increased risks for a range of other health outcomes that have not been fully characterised.

Figure 2 (top panel) presents attributable mortality rates expressed per 100 000 population. The data suggest that South and East Asia, the Middle East, and eastern Europe are regions with high attributable deaths on a per capita basis. The highest total attributable mortality occurs in China, with 2.44 million (95% confidence interval 1.68 to 3.20) deaths

per year, followed by India with 2.18 million (1.53 to 2.81) deaths per year. Results for all countries and regions and the different causes of death and pollutants can be found in the supplementary data; global maps for six disease categories are presented in supplementary figure S3.

Table 1 distinguishes the prevailing causes of death in macro-regions defined by the GBD studies (supplementary figure S2).[2] For example, mortality related to air pollution from ischaemic heart disease in high income countries and Sub-Saharan Africa is nearly a factor of two lower than the global average, whereas, in central and eastern Europe, North Africa, and the Middle East, the effect is nearly a factor of two higher. The reason for relatively low number of deaths from ischaemic heart disease related to air pollution in low income countries may be that infectious diseases still lead to substantial proportions of deaths and the population is relatively young. The burden of non-communicable diseases is rising in much of Sub-Saharan Africa from population growth and ageing as countries undergo demographic transitions. In high income countries, access to clean water, sanitation, and effective health care, for example, reduces mortality from communicable diseases and ischaemic heart disease. Mortality from lower respiratory infections attributable to air pollution is very high in low income countries in Asia and Africa, especially among children who have a substantially lower life expectancy than elsewhere. Another notable difference is that attributable mortality from chronic obstructive pulmonary disease is a three to four times higher in South and East Asia compared with high income countries.

Mortality related to fossil fuels

Table 2 shows mortality attributable to emissions related to fossil fuels, which typically stem from industry (eg, iron, steel, and aluminium production), the chemical and transformation sectors (eg, coal and coke production and combustion, petroleum refining), and transportation and power generation (coal, oil, and gas). Coal combustion contributes more than half of these emissions.[14] The global mortality attributable to fossil fuel-related emissions is 5.13 (95% confidence interval 3.63 to 6.32) deaths per year. The proportion of the total (avoidable plus unavoidable) excess mortality burden of fossil fuels is 61%, and nearly 82% of that due to anthropogenic emissions (table 2). This 82% is the fossil fuel-related proportion of the theoretical maximum avoidable number of excess deaths from all human induced (anthropogenic) sources of ambient air pollution. A substantial mortality reduction is shown for cardiometabolic diseases (eg, ischaemic heart disease, stroke, type 2 diabetes) of 2.61 million (1.91 to 3.09) per year. Figure 3 summarises mortality decreases that could potentially be achieved in seven macro-regions. Relatively small reductions can be realised by phasing out fossil fuels in Sub-Saharan Africa (25%), where residential energy use and natural emissions are predominant, and in North Africa and the Middle East (29%), where desert dust is a primary contributor to $PM_{2.5}$. The largest mortality reduction from phasing out fossil fuels can be achieved in high income countries (85%) that are largely dependent on fossil energy. Removing all anthropogenic air pollution would even reduce attributable deaths by about 94% in high income countries; thus, the natural component is small.

If or when a phase-out of fossil fuels will be achieved is unclear, therefore, we have also considered a "quarter way" and a "half way" scenario. Regional and national scale results are presented in the supplementary data files (ie, for all cause and disease specific mortality in all countries). Figure 3 illustrates that the half way (and quarter way) scenario (ie, a 50% (or 25%) decrease in exposure from fossil fuel-related emissions in highly polluted regions such as in South and East Asia) reduces mortality relatively less effectively compared with a complete phase-out (supra-linear response). In this case, a drastic pollution emission reduction has a disproportionally positive health outcome compared with a relatively minor reduction. In less polluted regions, such as in high income countries, drastic emission reductions are proportionally more efficient. These responses are a consequence of the shape of the relative risk functions at different levels of exposure. Overall, however, the responses are not strongly non-linear and emission reductions at all levels of air pollution can be expected to decrease the number of attributable deaths substantially.

Figure 2 (middle panel) shows attributable death rates per 100 000 population in a world without fossil fuels. For example, lower respiratory infections would diminish particularly in South Asia, ischaemic heart disease in North Africa and the Middle East, and cardiovascular diseases (ischaemic heart disease and stroke) in East, Southeast, and South Asia. The lower panel of figure 2 illustrates the theoretical effect of stopping all ambient anthropogenic air pollution emissions, including those from solid fuel burning in households. The remaining natural sources include wind-blown desert dust and volcanic and biomass burning emissions from wildfires, contributing about 25% to global attributable deaths, which we assume to be unavoidable. Additional studies will be needed to attribute the anthropogenic component of wind-blown dust and wildfire emissions as possible consequences of climate and land-use change. Note that the fraction related to climate change (eg, wildfire emissions from increasing heat extremes and droughts), should be mainly attributed to fossil fuels. Atmospheric desert dust is a largely natural phenomenon that is primarily predominant in North Africa, the Middle East, and South and central Asia, an extended area of the globe referred to as the dust belt. About 25% of dust sources globally are estimated to be anthropogenic, varying from 8% in North Africa to 75% in Australia.[28] Supplementary figure S4 present the cause specific results of a world without fossil fuels.

Discussion

General findings

Applying the new FUSION risk model and updated data for exposure to ambient fine particulate matter ($PM_{2.5}$) and ozone (O_3), we estimate that global all cause attributable mortality is 8.34 million (95% confidence interval 5.63 to 11.19) deaths per year. This figure is higher than that reported by the GBD 2019 study,[2] which was 4.45 million (3.60 to 5.29) deaths per year, although that accounted for six specific diseases. Our all cause mortality related to $PM_{2.5}$ exposure (ie, without ozone) is 7.96 million (5.47 to 10.55) deaths per year, slightly lower than the estimate by Burnett and colleagues of 8.16 million (5.92 to 9.95).[12] Differences with the GBD results are primarily related to the FUSION relative risk calculations that optimise the shape of the exposure-response relationship throughout the range of ambient $PM_{2.5}$. We adjusted the counterfactual concentration range in FUSION, following the method used in the GBD study, which explains our slightly lower global total compared with Burnett and colleagues. A primary reason for the relatively low number of deaths reported from the GBD study is that the study applies integrated exposure-response functions that use cohort studies of second-hand smoke and household air pollution to represent high $PM_{2.5}$ concentrations. These functions rely on assumptions about the dosage and toxicity of $PM_{2.5}$ (eg, between air pollution and tobacco smoke), whereas FUSION only considers studies involving ambient air pollution. Furthermore, the GBD estimates account for cause specific but not all cause mortality, which explains a global difference of about 1.66 million per year in our calculations. Finally, the GBD estimate for mortality related to ozone is slightly lower (for 2017), which accounts for a small difference of less than 0.1 million deaths per year.

Our results underscore that the attributable mortality is particularly high in South and East Asia, related to high pollution levels and population densities. Yet, we noted that the mortality rate per 100 000 is equally high in eastern Europe and part of the Middle East. South, East, and, Southeast Asia are home to 55% of the world's population, accounting for 70% of the mortality related to air pollution. Even so, figure 1 illustrates that air pollution is a major health risk factor worldwide. Although air quality has improved in some regions, such as North America, Europe, and East Asia, the ageing populations are more susceptible to risk from long term exposure.[29] More than half (52%) of the global mortality burden from ambient air pollution is accounted for by cardiometabolic diseases (ischaemic heart disease (30%), stroke (16%), and diabetes (6%)). Chronic obstructive pulmonary disease is also an important health outcome, contributing to nearly 16% of mortality from air pollution, of which $PM_{2.5}$ accounts for about 11% and ozone about 5%. Thus, mortality caused by long term exposure to ambient air pollution is approximately 95% attributed to $PM_{2.5}$. Note that we did not account for exposure to NO_2, which is also associated with enhanced mortality risk, possibly independent from $PM_{2.5}$ and ozone.[30] We derived a large contribution (about 20%) from other, unspecified diseases. Likely, this contribution is at least partly related to arterial hypertension and neurodegenerative disorders such as Alzheimer's and Parkinson's disease, for which evidence for an association with air pollution is growing, but the number of cohort studies is still too small to derive risk functions.[31-35] Continued health assessments of impacts on specific disease outcomes from air pollution in cohort studies are expected to reduce the number of uncategorised deaths in future. Lower respiratory infections contribute about 8% to mortality globally. They are more predominant in low income countries (eg, those in Sub-Saharan Africa (30%)), substantially lowering children's life expectancy.

Climate neutrality and health benefits

Our results suggest that a global phase-out of fossil fuels will have large health benefits, much larger than indicated by most previous studies. These data support increasing the share of clean, renewable energy, advocated by the UN through the sustainable development goals for 2030 and the ambition of climate neutrality for 2050. We find that mortality from chronic obstructive pulmonary disease (especially by ozone) and lung cancer will be reduced proportionately more than other non-communicable diseases. At the same time, lower respiratory infections, mainly in low income countries with less abundant use of fossil fuels currently, will decrease less. The number of global studies that we can compare our results with is small.[6 14] In previous work, we calculated similar global mortality from anthropogenic sources as the present results but attributed a smaller fraction (65%) to sources related to fossil fuels (here about 82%).[6] Several reasons account for this difference. Firstly, in the past decade, fossil fuel use has increased substantially in low and middle income countries, notably by coal-fired power plants in Asian economies. In many low income settings, fossil fuels replace traditional energy sources, such as solid biofuels, for residential heating and cooking. Secondly, the FUSION model applies optimised exposure-response associations, adjusting the sensitivity at low and high $PM_{2.5}$ concentrations. For high income countries, we calculate lower mortality attributable to $PM_{2.5}$ than previously but attribute a larger fraction to fossil fuel use because of the increased sensitivity between low to moderately high concentrations (about 10-40 µg/m^3), a range relatively strongly affected by emissions related to fossil fuels.

Another reason for the relatively substantial effects of a fossil fuel phase-out compared with small emission reductions is the supra-linearity of risk models at low exposure. This is most striking for the integrated exposure-response functions applied by McDuffie and colleagues,[14] following the GBD approach.[2] For a given air pollution decrease (eg, 10 µg/m^3), reductions in attributable deaths along the linear part of the log-linear relative risk functions are smaller than those in

the supra-linear part at low exposures. McDuffie and colleagues calculated the health benefits of eliminating specific types of fuels, which yields different results than eliminating all or a major part of emissions related to fossil fuels.[14] A study of the implications of coal-fired power plants in Europe, also using integrated exposure-response functions, illustrates these limited health benefits.[36] A modest reduction of attributable deaths by about 2.5% could be achieved by phasing out the emissions of coal power plants only, even though they contribute substantially to air pollution, especially in eastern Europe. When simultaneously and drastically reducing multiple pollution sources, the mean reduction in attributable deaths due to phasing out coal-fired power plants was estimated at 14% and up to 20% in eastern Europe. The muted response of the integrated-response functions to relatively small emission reductions is a result of their low slope at high levels of exposure (ie, relatively flat risk curves), which has been improved with the FUSION model.

Limitations

The FUSION relative risk model relies on numerous epidemiological cohort studies in various parts of the world. Many were undertaken in North American, European, and Asian countries, while other continents are still under-represented. An analysis published in 2020 of more than 25 years of cohort studies concluded that many initial controversies and concerns about mortality risk calculations have at least been partially resolved.[37] This refers, for example, to reanalyses and replication of studies, and the evaluation of causality, implying that the full account of confounding factors associated with exposure to air pollution has been challenging. Although cohort studies often adjust for multiple individual and area level covariates, residual confounding cannot be ruled out. Dozens of cohort studies across continents in the past few decades have reported all cause and disease specific mortality attributable to $PM_{2.5}$, adopted into the meta-analytic framework of the FUSION model. However, in some parts of the world, especially countries in Africa, such studies have not yet been performed. Additional health surveillance data and dedicated cohort studies at high exposure levels are needed, especially in low income countries.

The assessment of long term health impacts from air pollution, based on numerous epidemiological cohort studies, has become part of the regular disease burden updates of the GBD. In the past decades, since about 1990, disease burden assessments have increasingly acknowledged air pollution as a major global health risk factor. However, the diverse methods of such assessments translate into substantial heterogeneity between published results because a common standard has yet to be formulated.[38] Differences between assessments are primarily associated with exposure data, relative risk functions, counterfactual concentration levels, and the health outcomes associated with air pollution (eg, all cause versus disease specific mortality).[8 39] We used updated methods and data sources, but differences in published results about the health benefits of controlling air pollution remain challenging to communicate. Therefore, mortality burden results were recommended to be complemented with the number of deaths that could be prevented by reducing air pollution to promote the understanding of implications.[39] Our results contribute in this respect, also because the fractional change in attributable mortality does not depend on the total number of deaths. Nevertheless, assumptions about emission reductions are also associated with uncertainty. Future projections involve socioeconomic, technological, demographic, and policy developments that are hard to foresee.

Although epidemiological studies have not identified substantial differences in health outcomes of long-term exposure to different $PM_{2.5}$ source categories, toxicological analyses show that the oxidative potential can differ markedly.[40] Particle bound reactive oxygen species, when leading to an excess in human tissue (eg, the lung), are health hazardous and depend on various compounds, such as trace metals and organics, which can be specific for pollution sources. FUSION and other risk models assume that all $PM_{2.5}$ particles are equally toxic, an assumption that needs to be studied more intensely. Epidemiological studies could account for differential toxicity when air quality monitoring stations report particle size and composition. Evidence suggests that ultrafine particles (smaller than 0.1 µm) can bypass respiratory defences, transmigrate into the bloodstream, and affect other organs.[41] Ultrafine particles are not assessed in this study, indicating that our results may be conservative. Exposure modelling is associated with uncertainties, which are difficult to quantify because they depend on a range of emission, atmospheric chemistry, transport, and wet and dry deposition processes. Model results are continually tested against observations (eg, reference [18]), but uncertainty remains. Often, grid resolution is mentioned as a source of uncertainty. We addressed this concern in previous work and found that the overall uncertainty in attributable burden calculations, expressed by the 95% confidence intervals (of order 35%), is much more strongly determined by the relative risk calculations than by the resolution of the analysis.[42] Finally, air pollution was shown to contribute to deaths from covid-19,[43 44] estimated at a proportion of about 15% globally,[45] but this additional health burden does not apply to the estimates for 2019.

Conclusions

Air pollution continues to be a leading public health risk, and the annual number of attributable deaths exceeds the cumulative number of covid-19 deaths to date, according to WHO estimates.[46] A major proportion is potentially avoidable, caused by anthropogenic emissions, of which globally 82% is attributable to using fossil fuels in industry, power generation, and transportation. Given the Paris Climate Agreement's goal of climate neutrality by 2050, the replacement of

fossil fuels by clean, renewable energy sources would have tremendous public health and climate co-benefits. The forthcoming UN's 28th climate summit (COP28) in the United Arab Emirates offers an opportunity to make substantial progress towards phasing out fossil fuels. Of which, the health benefits should be high on the agenda.

Contributors: JL, AH and AP conceptualised the study. JL, AP, and RB developed the method. AP and KK performed the model calculations and produced the original figures. JL drafted the manuscript. All authors contributed to the data analysis and contributed important intellectual content during manuscript drafting and revision. All authors approved the final version of the manuscript. JL is the study guarantor. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

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Ethical approval: This work was based on open access data and model calculations therefore ethical approval was not required.

Data sharing: The results from this study, including the supplementary data files are available from the EDMOND research data repository of the Max Planck Society: (https://edmond.mpg.de/dataset.xhtml?persistentId=doi:10.17617/3.T7XAXH). All health related information, including age-dependent baseline mortality rates, needed to compute the mortality burden for different disease categories was adopted from the Global Burden of Disease (https://ghdx.healthdata.org/gbd-2019). Population data were provided by the NASA Socioeconomic Data and Applications Center (SEDAC), hosted by Columbia University (http://www.ciesin.columbia.edu/data.html). The climate model (ECHAM) is available to the scientific community under a software license agreement (https://code.mpimet.mpg.de/projects/mpi-esm-license). The Modular Earth Submodel System (MESSy) describes the atmospheric chemistry and aerosol processes, in the combined ECHAM/MESSy Atmospheric Chemistry (EMAC) model. Access to the source code is licensed to all affiliates of institutions that are members of the consortium. Institutions can become a member by endorsing a memorandum of understanding. More information can be found at https://www.messy-interface.org. The global emission dataset for 2019 was updated according to McDuffie and colleagues,[15] and is publicly available (https://github.com/JGCRI/CEDS/).

The lead author (JL) affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned and registered have been explained.

Dissemination to participants and related and public communities: We will disseminate our results to stakeholders and the general public through a press release, social media, and the websites of the research institutions that carried out this work. We will also present the results at scientific conferences and meetings with stakeholders involved in public health, air quality and climate change impacts.

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1 World Health Organization. *WHO global air quality guidelines.* WHO, 2021.
2 Murray CJL, Aravkin AY, Zheng P, et al, GBD 2019 Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet* 2020;396:1223-49. doi:10.1016/S0140-6736(20)30752-2
3 Rafaj P, Kiesewetter G, Gül T, et al. Outlook for clean air in the context of sustainable development goals. *Glob Environ Change* 2018;53:1-11. doi:10.1016/j.gloenvcha.2018.08.008.
4 Amann M, Kiesewetter G, Schöpp W, et al. Reducing global air pollution: the scope for further policy interventions. *Philos Trans A Math Phys Eng Sci* 2020;378:20190331. doi:10.1098/rsta.2019.0331
5 Shindell DT, Lee Y, Faluvegi G. Climate and health impacts of US emissions reductions consistent with 2°C. *Nat Clim Change* 2016;6:503-71. doi:0.1038/nclimate2935.
6 Lelieveld J, Klingmüller K, Pozzer A, Burnett RT, Haines A, Ramanathan V. Effects of fossil fuel and total anthropogenic emission removal on public health and climate. *Proc Natl Acad Sci U S A* 2019;116:7192-7. doi:10.1073/pnas.1819989116
7 Intergovernmental Panel On Climate Change. Climate Change 2021 – The Physical Science Basis: Working Group I Contribution to the Sixth Assessment Report of the Intergovernmental Panel on Climate Change. 1st edn. Cambridge University Press2023. doi:10.1017/9781009157896
8 Pozzer A, Anenberg SC, Dey S, Haines A, Lelieveld J, Chowdhury S. Mortality attributable to ambient air pollution: a review of global estimates. *Geohealth* 2023;7:GH000711. doi:10.1029/2022GH000711.
9 Hafner M, Tagliapietra S, eds. *The Geopolitics of the Global Energy Transition.* Springer International Publishing, 2020. doi:10.1007/978-3-030-39066-2.
10 Burnett R, Chen H, Szyszkowicz M, et al. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. *Proc Natl Acad Sci U S A* 2018;115:9592-7. doi:10.1073/pnas.1803222115
11 Weichenthal S, Pinault L, Christidis T, et al. How low can you go? Air pollution affects mortality at very low levels. *Sci Adv* 2022;8:eabo3381. doi:10.1126/sciadv.abo3381
12 Burnett RT, Spadaro JV, Garcia GR, Pope CA. Designing health impact functions to assess marginal changes in outdoor fine particulate matter. *Environ Res* 2022;204:112245. doi:10.1016/j.envres.2021.112245
13 Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature* 2015;525:367-71. doi:10.1038/nature15371
14 McDuffie EE, Martin RV, Spadaro JV, et al. Source sector and fuel contributions to ambient $PM_{2.5}$ and attributable mortality across multiple spatial scales. *Nat Commun* 2021;12:3594. doi:10.1038/s41467-021-23853-y
15 McDuffie EE, Smith SJ, O'Rourke P, et al. A global anthropogenic emission inventory of atmospheric pollutants from sector- and fuel-specific sources (1970–2017): an application of the Community Emissions Data System (CEDS). *Earth Syst Sci Data* 2020;12:3413-42. doi:10.5194/essd-12-3413-2020.
16 van Donkelaar A, Hammer MS, Bindle L, et al. Monthly global estimates of fine particulate matter and their uncertainty. *Environ Sci Technol* 2021;55:15287-300. doi:10.1021/acs.est.1c05309
17 Chowdhury S, Pozzer A, Haines A, et al. Global health burden of ambient $PM_{2.5}$ and the contribution of anthropogenic black carbon and organic aerosols. *Environ Int* 2022;159:107020. doi:10.1016/j.envint.2021.107020
18 Pozzer A, Reifenberg SF, Kumar V, et al. Simulation of organics in the atmosphere: evaluation of EMACv2.54 with the Mainz Organic Mechanism (MOM) coupled to the ORACLE (v1.0) submodel. *Geosci Model Dev* 2022;15:2673-710. doi:10.5194/gmd-15-2673-2022.
19 DeLang MN, Becker JS, Chang K-L, et al. Mapping yearly fine resolution global surface ozone through the odellin maximum entropy data fusion of observations and model output for 1990-2017. *Environ Sci Technol* 2021;55:4389-98. doi:10.1021/acs.est.0c07742
20 Jöckel P, Tost H, Pozzer A, et al. Earth System Chemistry integrated Modelling (ESCiMo) with the Modular Earth Submodel System (MESSy) version 2.51. *Geosci Model Dev* 2016;9:1153-200. doi:10.5194/gmd-9-1153-2016.
21 Kohl M, Lelieveld J, Chowdhury S, et al. Numerical simulation and evaluation of global ultrafine particle concentrations at the Earth's surface. *Atmos Chem Phys* 2023;23:13191-215. doi:10.5194/acp-23-13191-2023.

22 Hersbach H, Bell B, Berrisford P, et al. The ERA5 global reanalysis. *Q J R Meteorol Soc* 2020;146:1999-2049. doi:10.1002/qj.3803.
23 Andreae MO. Emission of trace gases and aerosols from biomass burning – an updated assessment. *Atmos Chem Phys* 2019;19:8523-46. doi:10.5194/acp-19-8523-2019.
24 Klingmüller K, Metzger S, Abdelkader M, et al. Revised mineral dust emissions in the atmospheric chemistry–climate model EMAC (MESSy 2.52 DU_Astitha1 KKDU2017 patch). *Geosci Model Dev* 2018;11:989-1008. doi:10.5194/gmd-11-989-2018.
25 Guenther AB, Jiang X, Heald CL, et al. The Model of Emissions of Gases and Aerosols from Nature version 2.1 (MEGAN2.1): an extended and updated framework for odelling biogenic emissions. *Geosci Model Dev* 2012;5:1471-92. doi:10.5194/gmd-5-1471-2012.
26 Vodonos A, Awad YA, Schwartz J. The concentration-response between long-term $PM_{2.5}$ exposure and mortality; A meta-regression approach. *Environ Res* 2018;166:677-89. doi:10.1016/j.envres.2018.06.021
27 Lelieveld J, Pozzer A, Pöschl U, Fnais M, Haines A, Münzel T. Loss of life expectancy from air pollution compared to other risk factors: a worldwide perspective. *Cardiovasc Res* 2020;116:1910-7. doi:10.1093/cvr/cvaa025
28 Ginoux P, Prospero JM, Gill TE, et al. Global-scale attribution of anthropogenic and natural dust sources and their emission rates based on MODIS Deep Blue aerosol products: Anthropogenic and natural dust sources. *Rev Geophys* 2012;50. doi:10.1029/2012RG000388.
29 Chowdhury S, Pozzer A, Dey S, et al. Changing risk factors that contribute to premature mortality from ambient air pollution between 2000 and 2015. *Environ Res Lett* 2020;15:074010. doi:10.1088/1748-9326/ab8334.
30 Huang S, Li H, Wang M, et al. Long-term exposure to nitrogen dioxide and mortality: A systematic review and meta-analysis. *Sci Total Environ* 2021;776:145968. doi:10.1016/j.scitotenv.2021.145968
31 Fu P, Yung KKL. Air Pollution and Alzheimer's Disease: A Systematic Review and Meta-Analysis. *J Alzheimers Dis* 2020;77:701-14. doi:10.3233/JAD-200483
32 Weuve J, Bennett EE, Ranker L, et al. Exposure to air pollution in relation to risk of dementia and related outcomes: an updated systematic review of the epidemiological Literature. *Environ Health Perspect* 2021;129:96001. doi:10.1289/EHP8716
33 Shi L, Zhu Q, Wang Y, et al. Incident dementia and long-term exposure to constituents of fine particle air pollution: a national cohort study in the United States. *Proc Natl Acad Sci U S A* 2023;120:e2211282119. doi:10.1073/pnas.2211282119
34 Hahad O, Rajagopalan S, Lelieveld J, et al. Noise and air pollution as risk factors for hypertension: part I-epidemiology. *Hypertension* 2023;80:1375-83. doi:10.1161/HYPERTENSIONAHA.122.18732
35 Wilker EH, Osman M, Weisskopf MG. Ambient air pollution and clinical dementia: systematic review and meta-analysis. *BMJ* 2023;381:e071620. doi:10.1136/bmj-2022-071620
36 Kushta J, Paisi N, Van Der Gon HD, et al. Disease burden and excess mortality from coal-fired power plant emissions in Europe. *Environ Res Lett* 2021;16:045010. doi:10.1088/1748-9326/abecff.
37 Pope CA 3rd, Coleman N, Pond ZA, Burnett RT. Fine particulate air pollution and human mortality: 25+ years of cohort studies. *Environ Res* 2020;183:108924. doi:10.1016/j.envres.2019.108924
38 Castro A, Röösli M, de Hoogh K, et al. Methods matter: a comparative review of health risk assessments for ambient air pollution in Switzerland. *Public Health Rev* 2022;43:1604431. doi:10.3389/phrs.2022.1604431
39 Evangelopoulos D, Perez-Velasco R, Walton H, et al. The role of burden of disease assessment in tracking progress towards achieving WHO global air quality guidelines. *Int J Public Health* 2020;65:1455-65. doi:10.1007/s00038-020-01479-z
40 Bates JT, Fang T, Verma V, et al. Review of acellular assays of ambient particulate matter oxidative potential: methods and relationships with composition, sources, and health effects. *Environ Sci Technol* 2019;53:4003-19. doi:10.1021/acs.est.8b03430
41 Ohlwein S, Kappeler R, Kutlar Joss M, Künzli N, Hoffmann B. Health effects of ultrafine particles: a systematic literature review update of epidemiological evidence. *Int J Public Health* 2019;64:547-59. doi:10.1007/s00038-019-01202-7
42 Kushta J, Pozzer A, Lelieveld J. Uncertainties in estimates of mortality attributable to ambient PM 2.5 in Europe. *Environ Res Lett* 2018;13:064029. doi:10.1088/1748-9326/aabf29.
43 Ranzani O, Alari A, Olmos S, et al. Long-term exposure to air pollution and severe COVID-19 in Catalonia: a population-based cohort study. *Nat Commun* 2023;14:2916. doi:10.1038/s41467-023-38469-7
44 Zhang J, Lim Y-H, So R, et al. Long-term exposure to air pollution and risk of SARS-CoV-2 infection and COVID-19 hospitalisation or death: Danish nationwide cohort study. *Eur Respir J* 2023;62:2300280. doi:10.1183/13993003.00280-2023
45 Pozzer A, Dominici F, Haines A, Witt C, Münzel T, Lelieveld J. Regional and global contributions of air pollution to risk of death from COVID-19. *Cardiovasc Res* 2020;116:2247-53. doi:10.1093/cvr/cvaa288
46 World Health Organization. WHO Coronavirus (covid-19) dashboard. https://covid19.who.int (accessed 25 August 2023).

Web appendix: Supplementary information
Web appendix: Mortality countries, 2019, no anthropogenic emissions
Web appendix: Mortality countries, 2019, no fossil fuels
Web appendix: Mortality countries, 2019, half way fossil fuel scenario (50%)
Web appendix: Mortality countries, 2019, quarter way fossil fuel scenario (25%)
Web appendix: Mortality countries, 2019, reference
Web appendix: Mortality region, 2019, no anthropogenic emissions
Web appendix: Mortality region, 2019, no fossil fuels
Web appendix: Mortality region, 2019, half way fossil fuel scenario (50%)
Web appendix: Mortality region, 2019, quarter way fossil fuel scenario (25%)
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