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Morbidity changes induced by future air quality and demographic structure changes

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ABSTRACT

Climate change mitigation policies can enhance health by improving air quality. Previous studies have evaluated historical years lived with disability (YLDs) for cardiovascular and respiratory diseases attributable to particulate matter ($PM_{2.5}$). However, the impact of dementia, which can significantly affect YLDs, has not been thoroughly examined in the context of climate change mitigation. In this study, we estimated global YLDs attributable to $PM_{2.5}$ using health impact assessment models and $PM_{2.5}$ concentrations simulated by a global chemical transport model under two scenarios: with and without climate change mitigation. To address appropriately the issue of dementia, we explicitly considered future demographic patterns, particularly the aging population. YLDs are projected to increase globally by 2100 in both scenarios due to global aging, increasing from 7.1 million years in 2015 to 18 million years without mitigation and to 12.5 million years with it. Mitigation measures could reduce global YLDs by 5.33 million years, by 2100, limiting the increase from 2.5 times to 1.8 times. Although mitigation measures can reduce the health impacts attributable to $PM_{2.5}$, the role of population aging remains critical for the future.

1. Introduction

The Agreement aims to limit the rise in global average temperature to well below 2 $^{\circ}\text{C}$ above pre-industrial levels, and to pursue efforts to keep it within 1.5 $^{\circ}\text{C}$. The Agreement sets a long-term target to hold the increase in the global average temperature to well below 2 $^{\circ}\text{C}$ above pre-industrial levels or to pursue efforts to limit the temperature increase to 1.5 $^{\circ}\text{C}$ above pre-industrial levels. Countries that have signed the Paris Agreement should set and submit GHG emissions reduction targets.

The IPCC Sixth Assessment Report (AR6) highlighted that GHG reduction as part of climate change mitigation can bring multiple cobenefits, such as improved air quality and reduced morbidity and premature mortality. (Cissé et al., 2022; Markandya et al., 2018; Sampedro et al., 2020; Vandyck et al., 2018; Xie et al., 2018). GHGs and other air pollutants are largely emitted from the same sources, so climate change mitigation measures can result in significant health benefits (Gao et al.,

2018).

Several studies have evaluated the health benefits of climate change mitigation using integrated assessment models (IAMs), atmospheric chemical transport models (CTMs), and health impact assessment models (Rafaj et al., 2021; Rauner et al., 2020; Shindell et al., 2018; Tang et al., 2022). For example, reducing GHG emissions to achieve the 2 °C target could avoid 0.7–1.5 million premature deaths worldwide annually by 2050 (Vandyck et al., 2018) and reduce morbidity by 7 % in Asia by 2050 (Xie et al., 2018). Additionally, achieving the 1.5 °C target could prevent approximately 153 \pm 43 million premature deaths worldwide from 2020 to 2100 (Shindell et al., 2018).

Air pollution itself remains a serious environmental issue and was the fourth leading risk factor for all-cause death in 2019 (Health Effects Institute, 2020). In particular, fine particulate matter (PM $_{2.5}$) is considered a major threat to health (Azimi and Rahman, 2024; Feng et al., 2016; Sampedro et al., 2020; Yang et al., 2020). PM $_{2.5}$ is defined as

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particles with an aerodynamic diameter of less than $2.5~\mu m$ (Thangavel et al., 2022). Long-term exposure to $PM_{2.5}$ increases the risk of major chronic diseases such as stroke, lung cancer (LC), ischemic heart disease (IHD), chronic obstructive pulmonary disease (COPD), and lower respiratory infections (LRIs). These are hereafter referred to as the five main causes of disease (5-COD). In 2019, 99 % of the world's population was living in places where the WHO air quality guidelines were not met (WHO, 2024). In addition, the Global Burden of Disease study (GBD) reported 4,140,970 deaths due to ambient $PM_{2.5}$ worldwide in 2019 (GBD, 2019b).

The impacts of air pollution on health are worse in older adults (Cissé et al., 2022; Rafaj et al., 2021; Tang et al., 2022); thus, the rate of pollution-related morbidity is increasing as the population ages. Furthermore, this increase may offset the benefits of GHG reduction measures, and without additional controls (i.e., under a business-as-usual scenario), morbidity will continue to rise. With increasing life expectancy due to medical advances and other factors, the global number of people living with chronic disease-related disabilities is expected to rise. In this context, the progression of global population aging is a significant factor in health impact assessments (Turnock et al., 2023). As mortality attributable to PM_{2.5} among older adults is expected to increase in the future (Chen et al., 2023; Rafaj et al., 2021; Tang et al., 2022; Turnock et al., 2023), the impacts of such morbidities are also projected to rise. Years lived with disability (YLDs) is widely used as a health metric to assess morbidity (GBD, 2019a; GBD, 2019b). Thus, as the rate of morbidity increases, this metric will also increase, making it a useful tool for assessing the impacts of air pollution on health (GBD, 2019a).

Among the issues related to aging, dementia is a significant concern, particularly within the context of air pollution. The effects of $PM_{2.5}$ on dementia were not previously considered in the GBD study (Livingston et al., 2020). The Lancet Commissions report on dementia identified air pollution as one of the risk factors with newer and convincing evidence (Livingston et al., 2020). Several studies have investigated the link between ambient $PM_{2.5}$ exposure and dementia, finding that high levels of exposure significantly increase the risk of developing it (Fu et al., 2019; Peters et al., 2019; Shi et al., 2021; Sullivan et al., 2021). Ru et al. (2021) estimated that in 2015, dementia accounted for 30 % of the total YLDs from $PM_{2.5}$ -related diseases. Therefore, in the future, with a high population of aging individuals, it is important to assess the impact of

dementia. Previous studies have estimated YLDs attributable to PM2.5 only for the past or present. Although some have projected future DALYs (the sum of YLDs and years of life lost, YLLs), these were derived indirectly from DALY-to-mortality ratios. Few studies have directly projected future YLDs for both 5-COD and dementia. (Brauer et al., 2024; Rauner et al., 2020; Tiwari et al., 2023).

Therefore, we investigated the impact of climate change mitigation policy and global population aging on future YLD projections by focusing on 5-COD and dementia. We compared the impact of $PM_{2.5}$ on morbidity under two different emissions pathways, including baseline and mitigation scenarios, by coupling emissions inventory outcomes from the Asia-Pacific Integrated Assessment Model (AIM-Hub) with a CTM and health impact assessment models.

2. Methodology

2.1. Overview

Fig. 1 shows the research framework. We estimated the global grid-based annual average $PM_{2.5}$ concentrations using the Goddard Earth Observing System Chemistry (GEOS-Chem) model, based on gridded emissions flux under scenarios with and without GHG emissions reduction from AIM-Hub (Fujimori et al., 2012, 2018).

Using these $PM_{2.5}$ concentration estimates together with the health impact assessment framework described below, we calculated the relative risk (RR) for five major causes of disease (5-COD) and dementia attributable to $PM_{2.5}$ exposure. The health impact assessment framework combines exposure–response functions, population structure, disease prevalence, and disability weights (DW) to estimate years lived with disability (YLDs). We then derived $PM_{2.5}$ -attributable YLDs and quantified the potential reductions associated with climate change mitigation policies.

We focused on target population individuals aged 65 and over because, due to limitations with the health impact assessment model, and selected years 2015, 2030, 2050, and 2100 to evaluate the nearterm, medium-term, and long-term effects.

2.2. $PM_{2.5}$ concentration estimation using GEOS-Chem model

To simulate the atmospheric distribution of PM_{2.5} concentrations, we

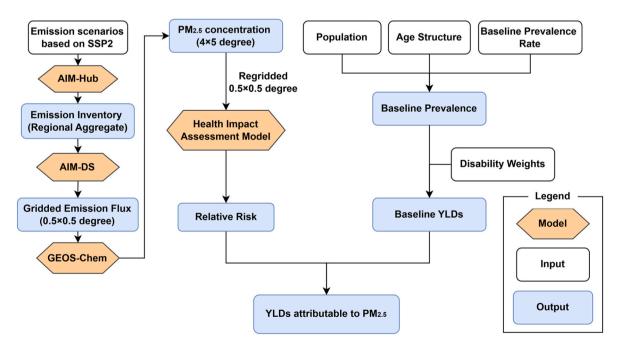


Fig. 1. The research framework in this study.

used the GEOS-Chem (12.9.3) model, a global, three-dimensional CTM designed to simulate the movement and transformation of chemical species in the atmosphere. It is driven by reanalysis meteorological data from NASA Global Modeling Assimilation Office MERRA2, which offers aggregated data at coarse resolution (Bey et al., 2001).

Surface $PM_{2.5}$ concentrations were simulated using the GEOS-Chem chemical transport model at a horizontal resolution of $4.0^{\circ} \times 5.0^{\circ}$ with 72 vertical layers. The choice of this resolution was based on the overall framework and objectives of this study. As illustrated in Fig. 1, our YLD estimation relies on emission projections from AIM-Hub and their spatial distributions provided by AIM-DS. The AIM-Hub projections, which have also participated in multi-model intercomparison projects such as CMIP6, exhibit substantial inter-model variability, indicating large uncertainties in future emission levels. Moreover, AIM-DS down-scales national emissions to a grid level based on current emission source locations and future distributions of population and GDP. While this method follows established approaches, the future spatial allocation of emissions remains highly uncertain.

Jansakoo et al. (2024a, 2024b) compared GEOS-Chem simulations at resolutions of 0.5° \times 0.625° and 4° \times 5° using a modeling framework similar to ours. Their results showed that although small regional differences exist, the overall performance and regional averages were largely consistent with observations. Since the present study focuses on region-based assessments of future YLD projections driven by climate change mitigation policies and population aging—rather than on local exposure variations—the influence of spatial resolution on the results is expected to be minimal. Considering these factors and computational efficiency, a 4.0° \times 5.0° resolution was adopted to ensure robustness and consistency within the modeling framework.

The chemical mechanism implemented in GEOS-Chem incorporates a detailed Ox-NOx-hydrocarbon-aerosol-bromine system (Mao et al., 2013; Parrella et al., 2012). PM_{2.5} components include natural mineral dust (Duncan Fairlie et al., 2007), sea salt (Jaeglé et al., 2011), BC aerosols (Wang et al., 2014), primary and secondary organic aerosols (Pai et al., 2020), and secondary inorganic aerosols (sulfate, nitrate, and ammonium). For the thermodynamics of secondary inorganic aerosols, the ISORROPIA II model was applied (Fountoukis and Nenes, 2007; Pye et al., 2010).

2.3. Health impact assessment

2.3.1. Overview of the health impact assessment framework

In this study, we quantified the health burden attributable to ambient $PM_{2.5}$ exposure using an integrated Health Impact Assessment (HIA) framework. The assessment combines exposure–response relationships for individual diseases, population structure, disease prevalence, and disability weights (DW) to estimate years lived with disability (YLDs) attributable to $PM_{2.5}$.

The overall analytical workflow consists of several sequential steps. First, the relative risk (RR) of PM $_{2.5}$ exposure for each disease and age group is estimated. The RR quantifies the nonlinear relationship between exposure concentration and disease risk using the Global Exposure Mortality Model (GEMM; Burnett et al., 2018) for five major causes of death (5-COD) and the model of Ru et al. (2021) for dementia. Next, baseline prevalence is calculated by age, sex, disease, and grid cell to establish a reference level of disease burden in the population. This baseline prevalence is then combined with the corresponding DW to obtain baseline YLDs.

Subsequently, the contribution of $PM_{2.5}$ exposure to YLDs is derived using the population attributable fraction (PAF), yielding $PM_{2.5}$ -attributable YLDs. Finally, to analyze the drivers of future changes in disease burden, factor decomposition is performed by separating the effects of population size, age structure, baseline prevalence, DW, and $PM_{2.5}$ concentration.

2.3.2. Relative risk

We estimated the RR of 5-COD attributable to $PM_{2.5}$ exposure using the Global Exposure Mortality Model (GEMM), a nonlinear model developed by Burnett et al. (2018). GEMM covers not only low-concentration areas but also high-concentration areas for outdoor $PM_{2.5}$, and we applied it globally. The equation follows.

$$RR(z) = \exp(\theta \log(1 + (z - z_{cf})/\alpha)/(1 + \exp((\mu - (z - z_{cf}))/\nu)))$$
(1)

where RR is relative risk, z is the exposure concentration of PM_{2.5}, z_{cf} is the concentration threshold, and α , μ , and ν are disease- and age-specific parameters. Disease-specific parameters were obtained from Burnett et al. (2018) and the threshold was set to 2.4 μ g m⁻³. For IHD and stroke, which have age-dependent RR, the parameters were calculated for different 5-year age groups. GEMM parameters were assumed to be constant over time and space and were applied uniformly to all years and grid cells.

For dementia, we used a function developed by Ru et al. (2021) (Eq. (2)). This is a function based on GEMM, and the target age is 65 years or older. It follows:

$$RR(x) = \exp(\theta \log(1 + (x - x_{cf}))/(1 + \exp(-((x - x_{cf}) - \mu)/\tau r)))$$
(2)

where RR is relative risk of dementia, x is $PM_{2.5}$ exposure concentration ($\mu g m^{-3}$), x_{cf} is the concentration threshold, with a uniform distribution between 2.7 $\mu g m^{-3}$ and 7.6 $\mu g m^{-3}$, r is exposure range, and θ , μ , and τ are parameters (equal to 0.12, 1.3, and 0.1, respectively, taken from Ru et al., 2021). The cited study addresses ambient air pollution (AAP) and secondhand smoke (SHS). Thus, we also used AAP and AAP + SHS + active smoking (AS) models in sensitivity analyses.

As the GBD2019 study found no significant differences between RR estimates for morbidity and mortality (GBD, 2019b), the same RR curve was applied to both. Accordingly, we evaluated the impact of $PM_{2.5}$ on morbidity under the assumption that the health impact function used for mortality is equally applicable to morbidity.

As GBD2019 found no significant difference between RR estimates for incidence and mortality (GBD, 2019b), the same RR curve was applied to both. Accordingly, we evaluated morbidity impacts under the assumption that the health impact function is equally applicable to morbidity.

2.3.3. Baseline prevalence

We calculated baseline prevalence for each age group a, disease d, grid g, sex s, and year y using Eq. (3). Baseline prevalence refers to the prevalence of all risk factors, including air pollution. The baseline prevalence represents the total prevalence of disease in the population before accounting for the contribution of individual risk factors. In other words, it provides the reference level of disease burden under existing exposure conditions, serving as a foundation for estimating the fraction of YLDs attributable specifically to air pollution through the relative risk approach described in the following sections.

$$BP_{a,d,g,s,y} = P_{g,y} \times A_{a,s,y} \times BPR_{a,d,s,y}$$
(3)

Where *BP* is the baseline prevalence, *P* is the gridded population, *A* is the percentage of the population by age group, and *BPR* is the baseline prevalence rate. In terms of the shared socioeconomic pathways (SSPs) considered, we used SSP2, which is a "middle of the road" scenario. To estimate the gridded population, we first calculated the ratio by dividing the country-level population in the updated version of the SSP2 (Samir et al., 2024) by that in the previous version (Jones and O'Neill, 2016). Then we multiplied this ratio, calculated for each country, by the gridded population (Jones and O'Neill, 2016) for each grid within the corresponding country. Notably, the spatial distribution of the gridded population remained the same as in Jones and O'Neill (2016). We obtained the percentage of population by age group using data from Samir

et al. (2024). The total global and regional populations and age structure of each population under SSP2 for 2015, 2030, 2050, and 2100 are shown in Figs. S2 and S4. The baseline prevalence rates in 2015 were obtained from IHME (2024), using country-, disease-, and age group-specific values. For future years, these rates were projected following the method of Hughes et al. (2011). For 5-COD, we applied the same change rates for mortality to morbidity. Mortality rates for 2019 to 2100 were used as input data, and the baseline prevalence rates for 2030, 2050, and 2100 were projected starting from 2019 using Eq. (4):

$$BPR_{y} = BPR_{2019} \times \prod_{k=2020}^{y} (1 + MCR_{k} \times \alpha)$$
 (4)

where BPR_y is the baseline prevalence rate in year y, BPR_{2019} is that in 2019, MCR_k is the change in the rate of mortality for the previous year, α is a disease-specific parameter from Hughes et al. (2011). Baseline prevalence rates in 2019 were obtained from IHME (2024), and mortality rates from 2019 to 2100 were obtained from the Institute for International Futures (2023). For dementia, the baseline prevalence rate for 2015 was applied to future years as it was assumed to remain constant. The baseline prevalence rates for both 5-COD and dementia were assumed to be uniform across all grids within each country.

2.3.4. Baseline YLDs

Following the method of the GBD 2019 study (GBD, 2019a), we calculated baseline YLDs for each age group, disease, grid, sex, and year, multiplying the prevalence by the disability weight (DW) (Eq. (5)).

$$BYLDs_{a,d,g,s,y} = BP_{a,d,g,s,y} \times DW_{a,d,g,s,y}$$
(5)

where *BYLDs* are the baseline YLDs, and *DW* is the disability weight, which takes a value between 0 and 1. Using data from IHME (2024), *DW* was calculated by dividing baseline YLDs by baseline prevalence according to Eq. (5). For future years, it was assumed that *DW* in 2015 remained constant and was the same for all grids in each country.

2.3.5. YLDs attributable to PM_{2.5}

In accordance with the methodology from GBD 2019; GBD, 2019b), we calculated YLDs attributable to PM_{2.5} by multiplying the baseline YLDs by the population attributable fraction (PAF) of disease *d*, scenario *sc*, and year *y* (Eq. (6)). PAF is calculated as 1 minus the inverse of the *RR* where *YLDs* are those attributable to PM_{2.5} and *BYLDs* and *RR* are defined above. The calculated YLDs are aggregated into the 17 countries/regions shown in Table S1. These 17 regions were used to calculate regional emissions in the emissions scenarios described below. Then these were further aggregated into the five regions listed in Table S2.

$$YLDs_{d,sc,y} = \sum_{a,g,s} BYLDs_{a,d,g,s,y} \times (RR_{a,d,g,sc,y} - 1) / RR_{a,d,g,sc,y}$$
 (6)

2.3.6. Factor decomposition of YLDs attributable to PM_{2.5}

We conducted factor decomposition to analyze changes in estimated YLDs for future years relative to the baseline year, 2015. The five contributing factors considered were population, population age structure, baseline prevalence rate, DW, and PM2.5 concentration. Since DW remains constant over future years and the baseline prevalence rate of dementia is also assumed to be constant, their contribution rates are zero. In this study, we adopted the method developed by Gupta (1993).

2.4. Emission scenarios

We used GHG and air pollutant emissions data from Fujimori et al. (2020) for two emissions scenarios: the baseline scenario, which assumes no emissions restrictions, and the mitigation scenario, which assumes emissions reductions in line with limiting global temperature increase to within 1.5 °C. These data are obtained from AIM-Hub and based on the SSP2 scenario. Details about AIM-Hub are provided in Fujimori et al. (2012), and SSPs are outlined by O'Neill et al. (2017). The

pollutants considered include carbon dioxide (CO_2) , methane (CH_4) , nitrous oxide (N_2O) , fluorinated gases (Fs), black carbon (BC), carbon monoxide (CO), ammonia (NH_3) , non-methane volatile organic compounds (NMVOCs), nitrogen oxides (NOx), organic carbon (OC), and sulfur oxides (SOx). These emissions significantly influence atmospheric processes, contributing to the formation of $PM_{2.5}$. In these scenarios, changes in temperature and precipitation due to climate change were not considered, that is, meteorological conditions were held constant among the two scenarios and years. Furthermore, we did not consider the socioeconomic impacts of climate change.

The annual trajectories of anthropogenic emissions of CO_2 and other air pollutants in each scenario are shown in Fig. S1. Generally, emissions decrease under mitigation, although those of BC and OC increase. This is primarily due to emissions from the land use sector and is attributable to an increase in forest fires following an increase in global afforestation. On the other hand, in the energy sector, these emissions decrease.

To obtain gridded emissions at the $0.5^{\circ} \times 0.5^{\circ}$ resolution required for the GEOS-Chem model, we used the AIM downscaling (AIM-DS) model and downscaled the emissions to the grid base from a 17-region inventory outcome from AIM-Hub. The downscaling approach varies by sector and emissions source and categorizes emissions into three groups. Group 1 emissions from energy, industry, island transport, buildings, solvents, and waste sectors are mainly driven by GDP and population. In this context, energy-related emissions are assumed to be strongly linked to these socioeconomic factors. Group 2 emissions from agriculture, forestry, and land use are established in proportion to the base year (2015). Group 3 emissions from aviation are downscaled in proportion to global emissions from geographic distribution in the base year (Hoesly et al., 2018). Further methodological details are available in Fujimori et al. (2017, 2018).

3. Results

3.1. Annual average PM_{2.5} concentrations

Fig. 2 illustrates the global distribution of the annual average PM_{2.5} concentrations at baseline estimated using GEOS-Chem. In 2015, there were regions with concentrations of 20 μ g m⁻³ or more over a wide area from Asia to Europe. In particular, in China and Africa, levels exceeded 100 μ g m⁻³.

At baseline, $PM_{2.5}$ concentrations show a declining trend globally from 2015 to 2100. Those for China, India, the Middle East, and Africa remain high in 2030, 2050, and 2100, significantly exceeding WHO airquality guidelines. This is particularly true in Africa, where the concentrations consistently remain above 100 $\mu g\ m^{-3}$, highlighting the severity of air pollution in this region.

In the mitigation scenario, $PM_{2.5}$ concentrations decrease at all three time points (Fig. 2), with the greatest reductions in developing countries such as China and India.

Fig. S6 shows population-weighted $PM_{2.5}$ concentration by region. In 2015, it is highest in China, followed by the rest of Asia and India. However, in 2100, at baseline, it is highest in Africa, followed by the Middle East and Asia; under mitigation, it is highest in Africa, followed by the Middle East.

3.2. YLD estimates

We compared YLD estimates for 5-COD in 2015 to those for the same year estimated in the GBD2019 study. Fig. 3 shows the results by region (Table S1). Our estimates are higher than those in previous studies, particularly for high-concentration areas such as China and India.

The figure also shows the global and regional YLD projections for each scenario. Under mitigation, YLDs decrease due to a lower $PM_{2.5}$ concentration compared to baseline. At the global level, YLDs for both baseline and mitigation scenario increase, from 7.1 million years in 2015 to 18 million years (a 2.5-fold increase) and to 12.5 million years

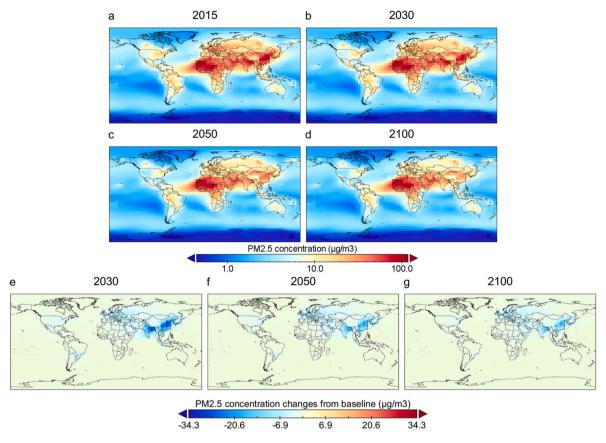


Fig. 2. Distribution of global $PM_{2.5}$ concentrations at baseline in 2015 (a), 2030 (b), 2050 (c) and 2100 (d), and changes in $PM_{2.5}$ concentration (mitigation – baseline) in 2030 (e), 2050 (f) and 2100 (g).

(a 1.8-fold increase) in 2100, respectively. At the regional level, they increase in all regions at baseline. In the mitigation scenario, they tend to decrease (about 14 %) in OECD90+EU countries but increase in other regions.

The figure also shows YLD projections separately for 5-COD and dementia in each scenario. At the global level, for both 5-COD and dementia, YLDs are lower under mitigation compared to baseline due to reduced $PM_{2.5}$ concentrations. However, both increase over time, from 5.5 million years in 2015 to 9.7 million years (for baseline) and 7.1 million years (for mitigation) and from 1.6 million years in 2015 to 8.1 million years (for baseline) and 5.4 million years (for mitigation) in 2100, respectively. The rate of increase is higher for dementia than for 5-COD

At the regional level, YLDs for 5-COD and dementia are largest at baseline in Asia, followed by the Middle East and Africa and then OECD90+EU countries in 2100. The same pattern is seen under mitigation but only for 5-COD. For dementia, they are largest in the Middle East and Africa, followed by Asia and then OECD90+EU. Yearly changes vary by region. In OECD90+EU and Asian countries, the changes in 5-COD YLDs differ between baseline and mitigation, where the Middle East and Africa show a high rate of increase for both disease groups under both scenarios. In all five regions, the rate of increase in YLD estimates is larger for dementia, for which the associated YLDs in 2100 exceed those for 5-COD in all regions except Asia under both scenarios.

3.3. Co-benefits from climate change mitigation policies

The differences in the YLD estimates between baseline and mitigation represent the co-benefits of climate change mitigation measures. According to our projections, 2.13 million years (1.71 million years for 5-COD and 0.42 for dementia) of YLDs would be avoided globally by

2030; by 2050 and 2100, the values are 3.73 million years (2.55 for 5-COD and 1.18 for dementia) and 5.33 million years (2.68 for 5-COD and 2.65 for dementia), respectively (Fig. 4). Such co-benefits are greatest in China, followed by OECD90+EU countries and India. By disease, the co-benefits in relation to 5-COD are greatest in China across all years; this is followed by India and OECD90+EU countries in 2030 and 2050 and by India and other Asian regions in 2100. With regard to dementia, the co-benefits are greatest for OECD90+EU countries, followed by China and other Asian regions in 2030, but greatest in China, followed by OECD90+EU countries and other Asian countries in 2050 and 2100.

As shown in Figs. 3 and 4, changes in population age structure contribute most to the increase in YLDs observed by 2100 under both scenarios and reduced $PM_{2.5}$ concentrations play the greatest role in suppressing increasing YLDs.

4. Discussion

4.1. Trends in YLD changes

We compared the effects of ambient PM_{2.5} on morbidity, as measured via YLDs, under two different emissions pathways: baseline and mitigation scenarios based on SSP2 product. Globally, YLDs tended to increase for both scenarios (Fig. 3). By disease group, YLDs from dementia increased more rapidly than those from 5-COD, mainly due to global population aging, consistent with previous studies (Chen et al., 2023; Nichols et al., 2022). Although mitigation reduces PM2.5 concentrations, YLDs still increase overall due to population aging.

The changes in YLDs varied by region. OECD90+EU countries showed an overall decrease in YLDs due to decreases in PM2.5 levels. On the other hand, in the Middle East and Africa, mitigation had no such

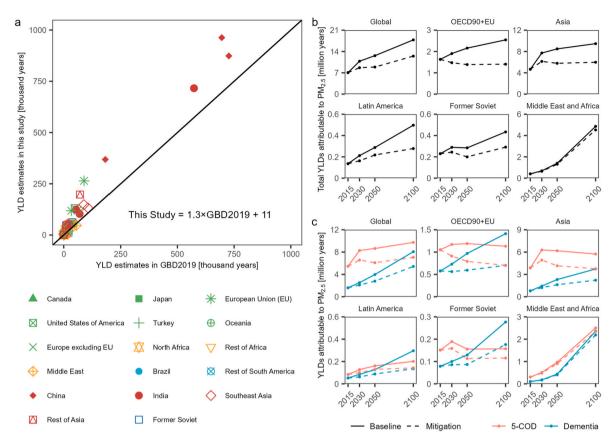


Fig. 3. YLD estimates from the GBD2019 study and the present study for 2015 by region (a). The solid line represents the 1:1 ratio between the two studies and the dotted line is the regression line. Global and regional total YLD estimates for each scenario (b) and YLD estimates separately for 5-COD and dementia (c).

effect, i.e., mitigation scenario showed a high rate of increase from 2015 to 2100 compared to other regions. The small difference between the baseline and mitigation scenarios in the Middle East and Africa can be attributed to the high PM2.5 concentrations originating from sources such as wildfires and dust. Under such conditions, the co-benefits of climate mitigation measures, such as reduced fossil fuel use, are likely to have a limited impact.

In the Middle East and Africa, unlike in other regions, a rapid increase in YLDs continued under both scenarios until 2100. This is because, under the SSP2 scenario, these regions are projected to experience particularly rapid population growth and population aging. In fact, the rate of increase in older people worldwide is expected be the highest in North Africa, West Asia, and Sub-Saharan Africa over the next 30 years (United Nations, 2023). In these regions, population growth and aging are expected to offset the mobility reductions achieved through decreased emissions under mitigation scenarios. This finding underscores the need for more comprehensive air pollution control measures beyond the co-benefits of climate mitigation.

These findings suggest that climate mitigation alone may not be sufficient to reduce the health burden associated with air pollution in rapidly aging and growing regions such as Africa and the Middle East. To address these persistent disparities, aging-sensitive policies that integrate air quality management with public health and social protection measures will be essential. Moreover, regionally targeted mitigation and adaptation strategies, such as improving household energy systems, strengthening healthcare infrastructure, and implementing local airquality monitoring, could help to more effectively reduce the disease burden associated with $\mathrm{PM}_{2.5}$ exposure.

4.2. YLDs reduced by climate change mitigation policies

Regions with the largest reductions in PM_{2.5} also showed the greatest

decreases in YLDs. This suggests that $PM_{2.5}$ concentrations have a significant impact on morbidity, in particular that related to 5-COD and dementia. In addition, our data predicts that overall YLDs will decrease in all regions and countries from 2030 to 2100 if mitigation measures are implemented. This highlights the importance of continuously promoting such measures.

4.3. Sensitivity analysis

Our main results assume the SSP2 pathway of global development, which represents the "middle of the road" scenario. For sensitivity analyses, we used SSP1 ("sustainability") and SSP3 ("regional rivalry"). SSP1 assumes inclusive, environmentally conscious development, whereas SSP3 reflects fragmented cooperation and poor environmental management. We also consider two concentration-response models for dementia: one based only on AAP and another based on AAP, SHS, and AS. For an overview of each concentration response model, see Ru et al. (2021).

The results for the different pathways are shown in Fig. 5, S2, S3, and S5. We find that YLDs overall, across all conditions, increase over time. In addition, in all cases, mitigation suppresses the increase in YLD (compared to baseline). YLDs rise faster under SSP1 and slower under SSP3 than under SSP2. For example, for SSP1, YLDs increase from 11 million years in 2015 to 47 million years in 2100 (a 4.3-fold increase) at baseline and to 34 million years in 2100 (a 3.1-fold increase) in the mitigation scenario. For SSP3, the values are 11 million years—21 million years (a 1.9-fold increase) and to 16 million years (a 1.45-fold increase), respectively. These results are in line with our main findings and suggest that mitigation has a positive impact on health under each scenario. At the same time, however, mitigation itself does little to slow the progression on YLDs, as demonstrated by the much higher rates of YLDs under the SSP1 pathway. Hence, population aging primarily drives these

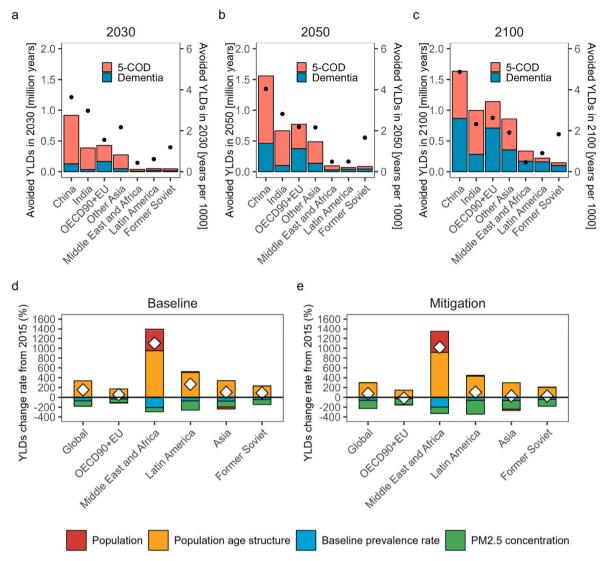


Fig. 4. Regional YLDs avoided, representing co-benefits of mitigation, in 2030 (a), 2050 (b) and 2100 (c) (dots represent per 1000), and factor decomposition of global and regional total YLD estimates in 2100 under the baseline (d) and mitigation (e) scenarios.

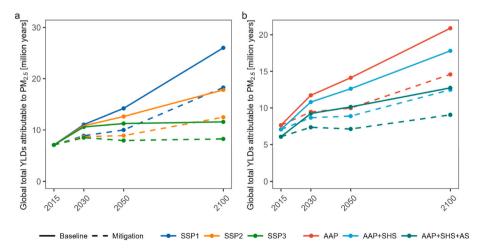


Fig. 5. Sensitivity analyses of total YLD estimates using data from SSP1, SSP2, and SSP3 (a) and using three concentration-response models (b).

patterns. As shown in Fig. S7, YLDs increase due to global population aging across all SSP pathways. Indeed, it is conceivable that the higher rate for SSP1 is related to improvements in healthcare and longevity,

resulting in more people living longer with chronic conditions. This could explain the much higher rates of YLDs.

Next, we estimated global YLDs using three concentration-response

models, as shown in Fig. 5. All models show an increasing trend in YLDs, but they are lowest for AAP + SHS + AS. In that model, global YLDs increase from 9.4 million years in 2015 to 23 million years in 2100 (a 2.4-fold increase) at baseline and to 17 million years (a 1.8-fold increase) under the mitigation scenario. Fig. S8 shows the impact of each different factor (aging, air pollution, secondhand smoke, and active smoking) on the YLDs associated with dementia in 2100. Dementia-related YLDs increase primarily due to global population aging, highlighting that aging itself is a key driver, regardless of other factors.

4.4. Comparison of YLD estimates of 5-COD with GBD2019

In 2015, our YLD estimates for 5-COD exceeded those of GBD2019, likely due to differences in risk curves, exposure thresholds, and PM_{2.5} concentration estimates. For example, to calculate RR, we used the GEMM model while GBD2019 used the Meta-regression Bayesian, Regularized, Trimmed (MR-BRT) model, and the risk curves differ between the two models (GBD, 2019b). GEMM predicts higher relative risks than MR-BRT, especially at high PM_{2.5} levels, because it relies only on outdoor air pollution cohorts and does not include active, secondhand, or household exposures. In contrast, MR-BRT integrates multiple exposure sources, which attenuates estimated hazard ratios (Burnett et al., 2018; GBD, 2019b). In addition, we applied a PM_{2.5} exposure threshold of 2.4 μg m⁻³, whereas GBD2019 used a threshold range of 2.4 μg m⁻³ to 5.9 μg m⁻³. We used GEOS-Chem alone, while GBD2019 combined it with satellite and ground data, preventing precise comparison of PM_{2.5} estimates. Overall, we believe that these three factors likely interacted and contributed to the higher YLD estimates observed in our study. Therefore, GEMM may be more appropriate for estimating health impacts in regions with higher PM2.5 concentrations, such as parts of Asia and Africa, where exposure levels exceed those typically represented in MR-BRT.

4.5. Reduced YLD reliability

In this study, we estimated that climate change mitigation measures would reduce YLDs by 8.81 million years globally, by 2100. We estimated PM2.5 using GEOS-Chem at $4^{\circ} \times 5^{\circ}$ resolution. Jansakoo et al. (2024b) reported that spatial resolution affects regional health estimates; differences in projected global premature deaths, however, remain within acceptable margins, so the impact on global YLDs is limited. However, Jansakoo et al. (2024b) reported that, at the regional level, such differences can influence health impact estimates.

YLDs were estimated assuming GEMM applies to morbidity. This partly explains why our 5-COD estimates exceed previous studies. Using MR-BRT would likely reduce estimates, but overall conclusions remain unchanged. Results may differ with other atmospheric or health impact models; validation using multiple models is necessary.

It is also necessary to consider the uncertainties associated with future regional emission projections from AIM-Hub and the downscaling process using AIM-DS. These uncertainties propagate along the estimation workflow and ultimately affect the estimated impacts of climate change mitigation measures on regional YLDs.

4.6. Limitations and caveats

We applied GEMM to morbidity, following Conibear et al. (2021), who estimated YLDs without discussing its appropriateness; however, the suitability of this approach remains uncertain and requires further study. Since GEMM was originally developed to estimate mortality risks, applying it to morbidity may lead to either overestimation or underestimation of the true health burden depending on disease type, age structure, and exposure patterns. The development of morbidity-specific exposure—response functions would help reduce this uncertainty.

We assumed that disability weights (DW) remained constant from 2015. Because DWs represent weighted averages across disease severity levels, future improvements in healthcare or changes in disease management could alter these values, potentially affecting long-term YLD projections. Sensitivity analyses with variable DWs could provide a more robust assessment.

Regarding future baseline prevalence, we estimated YLDs by applying the rate of change in deaths from the previous year to morbidity. Although this is currently the only feasible approach, it introduces structural uncertainty, particularly in scenarios with rapidly changing health systems or demographic profiles.

Although this study did not conduct grid-based YLD analyses, the spatial resolution of air quality simulations remains an important factor influencing exposure estimates. The relatively coarse $4^{\circ} \times 5^{\circ}$ resolution of GEOS-Chem may obscure localized pollution hotspots, particularly in urban and industrial regions where PM_{2.5} concentrations can vary sharply within small spatial scales (Jansakoo et al., 2024b). Higher-resolution modeling could enable grid-based or city-scale analyses that capture these fine-scale variations, improving the accuracy of exposure and health impact assessments. Future research integrating such high-resolution approaches would enhance understanding of spatial disparities in the health benefits of climate mitigation measures.

This study did not include estimates of indoor air pollution. Future projections of indoor air pollution could be influenced by assumptions under the SSPs and by socioeconomic indicators projected by AIM-Hub, such as household energy use, income, and urbanization rates. These factors may alter overall exposure levels, particularly in developing regions where solid fuel use remains prevalent. Therefore, excluding indoor air pollution may lead to an underestimation of the total health burden attributable to air pollution, and future studies should aim to integrate both ambient and indoor exposures within a consistent modelling framework.

5. Conclusion

We assessed future YLDs worldwide attributable to $PM_{2.5}$ for 5-COD and dementia, based on projections of global life expectancy and population aging. We also quantified YLDs that could be reduced as a cobenefit of climate change mitigation measures. For this, we estimated $PM_{2.5}$ concentrations for each scenario using GEOS-Chem, based on air pollutant emissions data with and without greenhouse gas emissions reductions. Then we estimated YLDs for each scenario globally and quantified the reduction in YLDs due to climate change mitigation measures.

The results showed that YLDs for both the baseline and mitigation scenarios increased over time, from 7.1 million years in 2015 to 18 million years in 2100 at baseline and to 12.5 million years for the mitigation scenario. Regional variation in YLDs was observed, with higher rates of increase in regions with predicted population growth and aging. In addition, climate change mitigation measures were projected to reduce YLDs as a co-benefit by 5.33 million years globally by 2100. The reduction in YLDs varied by region and was greater where larger reductions in PM_{2.5} concentrations occurred.

These results suggest that YLDs attributable to $PM_{2.5}$ will decrease due to climate change mitigation measures globally, but the impact of population aging cannot be ignored. It is crucial to implement measures based on the specific situation of each region. Future research should evaluate morbidity attributable to $PM_{2.5}$ in specific regions and countries rather than on a global scale. Achieving this will require a higher-resolution CTM and more detailed population distribution data, to identify areas where health impacts are most pronounced. Additionally, accumulating epidemiological data related to morbidity from outdoor $PM_{2.5}$ exposure and its relationship with 5-COD and dementia will enable more accurate estimation of RR.

CRediT authorship contribution statement

Hiroya Uchida: Writing - original draft, Visualization, Validation,

Supervision, Software, Resources, Methodology, Formal analysis, Data curation. Koga Yamazaki: Writing – review & editing, Supervision, Software, Methodology. Satoshi Sekizawa: Writing – review & editing, Software, Methodology. Shinichiro Fujimori: Writing – review & editing, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization. Ken Oshiro: Writing – review & editing. Thanapat Jansakoo: Writing – review & editing, Supervision, Software, Methodology.

Declaration of competing interest

The authors have no competing interests to declare that are relevant to the content of this article.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.aeaoa.2025.100396.

Data availability

Data will be made available on request.

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