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Article

Inequality in historical transboundary anthropogenic $PM_{2.5}$ health impacts

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ABSTRACT

Atmospheric transport of fine particulate matter ($PM_{2.5}$), the leading environmental risk factor for public health, is estimated to exert substantial transboundary effects at present. During the past several decades, human-produced pollutant emissions have undergone drastic and regionally distinctive changes, yet it remains unclear about the resulting global transboundary health impacts. Here we show that between 1950 and 2014, global anthropogenic $PM_{2.5}$ has led to 185.7 million premature deaths cumulatively, including about 14% from transboundary pollution. Among four country groups at different affluence levels, on a basis of per capita contribution to transboundary mortality, a richer region tends to exert severer cumulative health externality, with the poorest bearing the worst net externality after contrasting import and export of pollution mortality. The temporal changes in transboundary mortality and cross-regional inequality are substantial. Effort to reduce $PM_{2.5}$-related transboundary mortality should seek international collaborative strategies that account for historical responsibility and inequality.

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1. Introduction

Fine particulate matter ($PM_{2.5}$) in the air is estimated to cause millions of premature deaths each year at present [1–3], and might be a major atmospheric carrier for transmission of deadly viruses like SARS-CoV-2 [4,5]. To date, actions to mitigate $PM_{2.5}$ pollution have mainly focused on reducing local emissions [6–8]. However, it is being increasingly recognized that ambient $PM_{2.5}$ can be transported various distances to exert worldwide impacts [3,9–11]. Thus the success of local air quality protection is affected by how adequately transboundary pollution is accounted for. Since the start of the Third Industrial Revolution decades ago, the world has seen profound changes in the magnitude and distribution of emissions driven by growth and geographical restructuring of the global economy and coincident movement of emission source regions [12–14]. Such emission changes may have been companied by considerable variation in transboundary health impacts associated with $PM_{2.5}$, which are complicated by emission-dependent chemistry to form $PM_{2.5}$ [15], location-dependent transboundary transport pathway and efficiency [9,16,17], spatial matching between population and transboundary pollution [18–20], and nonlinear health response to pollution exposure [1].

The historical evolution of transboundary impacts has important implications for cross-regional health inequality, as each region may have caused and been exposed to different and temporally varying transboundary pollution. Health inequality is associated with unnecessary, avoidable, unfair and unjust differences in health status between population groups [21,22]; and a strategy to foster health equality is crucial to the sustainability of economic growth [23]. Indeed, the transboundary $PM_{2.5}$ pollution is closely tied to the 3rd (Good Health and Well-being) and 10th (Reduced Inequalities) Sustainable Development Goals (SDGs) of the United Nations [24]. As successful $PM_{2.5}$ mitigation is vital for achievement of the SDGs, a historical understanding of transboundary $PM_{2.5}$ health impacts helps uncover regional responsibilities and cross-regional inequality lessons, which together serve as a basis to establish future concerted mitigation strategy.

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However, little is known about the quantitative historical evolution of health impacts and embedded equality issues associated with transboundary $PM_{2.5}$ during the past several decades, despite that many studies have addressed the global health impacts of $PM_{2.5}$ in recent years [1–3,10,16,17,19,25–34]. The global $PM_{2.5}$-related health impact estimate from the Global Burden of Disease studies are since 1990 [30–34], and a recent model-based work extended the analysis back to 1960 [29]. These work estimated millions of annual global premature mortality associated with total $PM_{2.5}$ exposure, with no investigation of the contribution of transboundary $PM_{2.5}$. Studies of transboundary health impacts were conducted based on model simulations of pollution chemistry and transport, typically for specific, recent years (e.g., 2007 in Zhang et al. [3], 2001 in HTAP1 [17], 2010 in HTAP2 [16], 2000 in Liu et al. [10], and 2014 in Du et al. [18]), or for limited times and regions (e.g., 2005, 2011 and 2018 within the United States in Dedoussi et al. [19], and 2017 within China in Fang et al. [20]). These studies showed considerable contributions of transboundary $PM_{2.5}$ to present-day global/regional premature mortality; yet they did not address the long-term changes in global transboundary health impacts and associated cross-regional inequality.

Here we assess the health impacts of ambient anthropogenic $PM_{2.5}$ pollution from 1950 through 2014, effects of transboundary pollution, and embedded cross-regional health inequality. By integrating historical emission data, atmospheric chemical transport model (CTM) simulations, satellite-based surface $PM_{2.5}$ data and pollution exposure–response modeling, we calculate worldwide $PM_{2.5}$-related premature deaths attributable to anthropogenic emissions in four individual country groups categorized by affluence level (i.e., per capita gross national income). These groups include high-income group (i.e., developed countries), upper middle income group (e.g., China and Russia), lower middle income group (e.g., India and Indonesia) and low-income group (in Africa and parts of Asia) (Fig. S1 online), following the definitions of the United Nations [35]. We then analyze the driving factors of the evolution of anthropogenic $PM_{2.5}$-related mortality, and reveal the transboundary mortality impacts and the embedded inequality problem.

2. Materials and methods

Supplementary materials provide our methodological details and uncertainty discussion. Fig. S2 (online) shows the flowchart of our study. First, we combine anthropogenic emissions datasets from the Community Emissions Data System (CEDS) [12] and the Multi-resolution Emission Inventory for China (MEIC) [36–39] to drive the GEOS-Chem CTM (version 11-01) to simulate near-surface $PM_{2.5}$ concentrations at a horizontal resolution of 2.5° longitude × 2.0° latitude in the years of 1951, 1960, 1970, 1980, 1990, 2000 and 2014. Simulated aerosols include secondary inorganic aerosols (SIOA, sulfate + nitrate + ammonium), black carbon, primary organic aerosols (POA), secondary organic aerosols (SOA), dust and sea salt. For each year, a set of six simulations are conducted, with a base case including all (global anthropogenic and natural) emissions and five additional simulations excluding anthropogenic emissions of the globe or one country group; everything else is kept unchanged. In all simulations, meteorological conditions are fixed at 2014 levels to better quantify the anthropogenic contributions; a sensitivity test for 1990 shows that using the meteorological data in 1990 or 2014 in the model leads to similar numbers of $PM_{2.5}$-related deaths and similar patterns of transboundary health impacts (Fig. S3 online). See detailed information of GEOS-Chem simulations in Supplementary materials: S1.

Second, we calculate chemical efficiencies (CEs) (i.e., annual mean concentration per unit of emission) for SIOA, black carbon and POA in these simulation years, linearly interpolate these CEs to the years over 1950–2013 with no explicit GEOS-Chem simulations, and then convert anthropogenic emissions to concentrations in those years. The use of CEs follows our previous work [15,18,40]; this is a reasonable choice to save computational resources given the relatively linear relationships between emissions and concentrations for these pollutants [40]. We linearly interpolate anthropogenic SOA concentrations to other years; a sensitivity test suggests that using either interpolation or CE has a small effect on the calculated global population-weighted SOA (with NRMSD less than 7%, Fig. S4 online), thus a minor effect on the total $PM_{2.5}$ concentrations and resulting mortality. Natural $PM_{2.5}$ concentrations represent the 2014 situation to be consistent with the meteorology. Supplementary materials: S2 provides detailed information about this step.

Third, for the year of 2014 we use a satellite-derived surface $PM_{2.5}$ concentration dataset (V4.GL.02) [41] at a resolution of 0.1°×0.1° to further establish a conversion map from the CTM results for total (anthropogenic + natural) $PM_{2.5}$ concentrations at 2.5°×2.0° to the fine-resolution grid and correct the model bias. We then apply the same conversion map to other years. Comparison of such CTM-based, corrected results against the satellite-based $PM_{2.5}$ data from 2001 to 2013, for which years there are no explicit GEOS-Chem simulations, shows high consistency for 12 individual regions across the globe with R^2 ranging from 0.91 to 0.99 and the NRMSD lower than 1%. See more information in Supplementary materials: S3. This supports our method in the 2nd and 3rd steps.

Finally, we apply the corrected yearly total $PM_{2.5}$ concentrations to GEMM NCD + LRI [1] to calculate $PM_{2.5}$-related premature deaths worldwide, as detailed in Supplementary materials: S4. We then calculate annual mortality associated with anthropogenic $PM_{2.5}$ originating from individual country groups, by multiplying the total $PM_{2.5}$-related mortality by the ratio of CTM simulated anthropogenic concentrations caused by each group to total $PM_{2.5}$ concentrations; this direct proportion approach has been widely used [3,25,40]. Year-, country-, disease- and age-specific baseline mortality data since 1990 are taken from the GBD 2017 dataset [42]. The baseline mortality rates in 1990 are applied to all prior years with no data available; a sensitivity test linearly extrapolating the rates to earlier years, following a previous work [29], shows similar mortality results (Fig. S5 online).

As detailed in Supplementary materials: S5, the premature deaths estimate is affected by errors in anthropogenic emissions, GEOS-Chem simulations, use of chemical efficiency and linear interpolation, and use of GEMM NCD + LRI modeling. Together, these factors introduce an about 38% error (two standard deviations) to our best estimates of premature deaths discussed in the main text. Details of our methods, data and uncertainty discussions are presented in Supplementary materials.

3. Results

3.1. Enormous cumulative loss of lives

Accumulated from 1950 to 2014, there are 185.7 million premature deaths caused by global anthropogenic $PM_{2.5}$ pollution, with an average of 2.9 million in each year (Fig. 1a). The number of annual deaths is higher than the cumulative reported deaths (2.6 million) as of 2021-03-09 caused by COVID-19 (https://coronavirus.jhu.edu/map.html, last access: 2021-03-10). About 53.8 million cumulative global deaths over 1950–2014 are contributed by emissions in the high-income group, 82.3 million by the upper middle group, 47.4 million by the lower middle group, and 2.2 million by the low-income group.

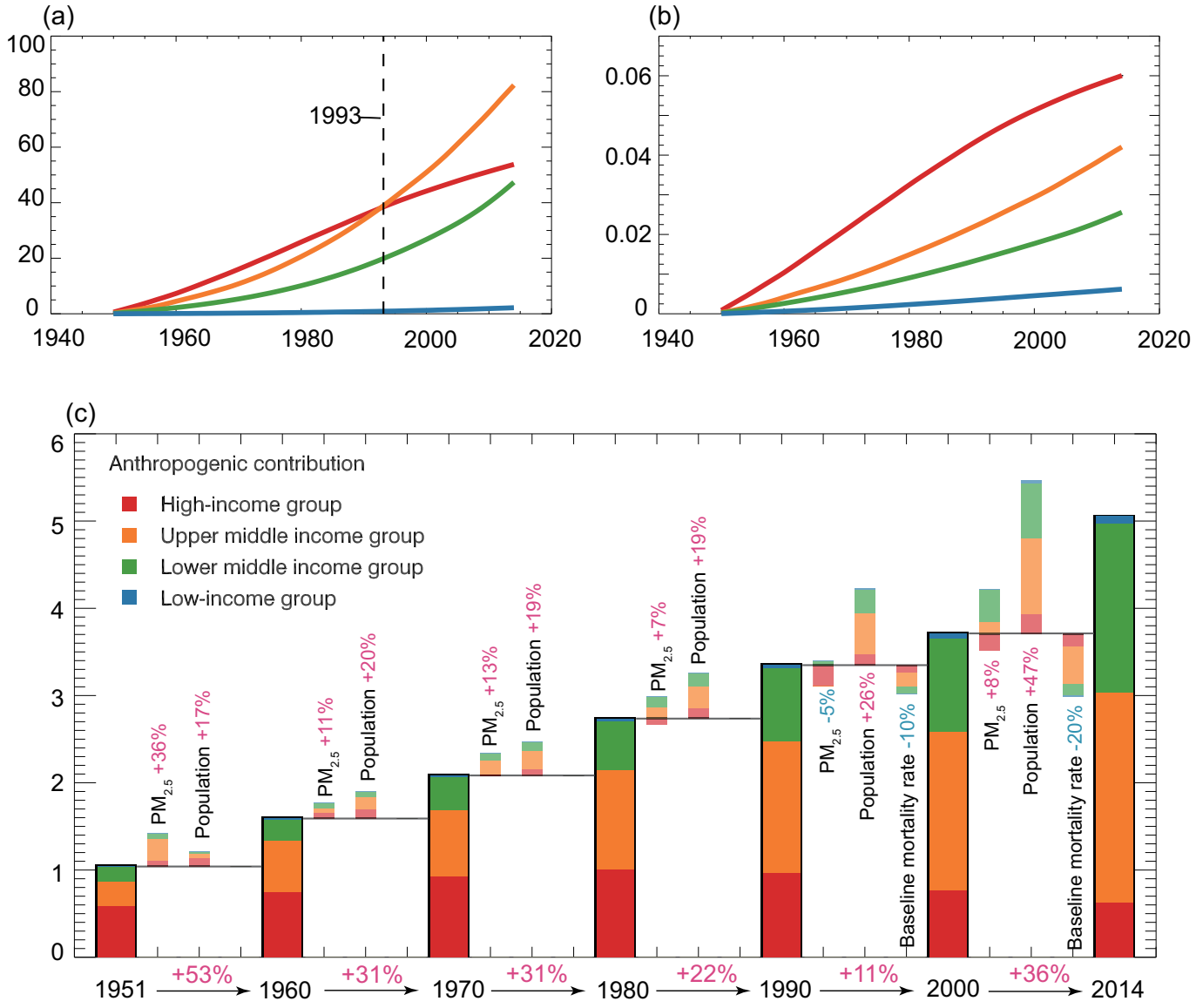

Fig. 1. Historical changes in anthropogenic $PM_{2.5}$-related premature deaths. The cumulative premature mortality (a, in Million), cumulative per capita contribution to premature mortality (b), and annual premature mortality in all simulation years (c, the wider bars, in Million) worldwide attributable to anthropogenic emissions in the four individual groups. The narrower bars in (c, in Million) with lighter colors represent the individual contributions of $PM_{2.5}$ pollution, population and baseline mortality rates to the change in mortality. The changes in baseline mortality rates before 1990 is not taken into account due to data limitation.

Fig. 1a shows that among the four groups, the high-income group has caused the largest number of cumulative deaths until the year of 1993, although some countries in this group started to control anthropogenic emissions decades before 1990 — for example, through implementing the Clean Air Act in the United States in 1970 [43] and the Clean Air Act in 1956 in the United Kingdom [44]. This result is partly because the group-based emissions of high-income countries start to decline much later when taken as a whole (e.g., around 1990 for NO_x and around 1980 for CO, as shown in Fig. S6 online). Another factor is population growth and aging (Fig. 1c). The cumulative deaths caused by the upper middle income group have been growing in acceleration over the past decades and has become the highest among the four groups since 1993. The cumulative deaths caused by the lower middle group have also grown at an increasing rate throughout the years. The 1950–2014 cumulative premature deaths at individual locations worldwide caused by each income group are presented in Fig. S7 (online), which further highlights the historical, global effect of the $PM_{2.5}$ pollution.

Supplementary materials: S4 provides a detailed comparison of our yearly mortality results with previous estimates based on various approaches. Results based on GEMM NCD + LRI, as done here, are compared in Fig. S8b (online). The results in 2014 of this study are consistent with our previous studies [18,40]. Comparing our results to those of Burnett et al. [1] and Chowdhury et al. [27] shows slight differences (around 20%) in global mortality. This is because we employ an updated version of baseline mortality data upon their studies, and estimate the premature deaths based on $PM_{2.5}$ exposure at individual grid cells rather than using the national/regional average population weighted $PM_{2.5}$ concentrations.

Fig. 1b shows the cumulative premature deaths attributed to each person of these individual groups. This calculation removes the effect of population size in each group (916 million for the high-income group, 1810 million for the upper middle group, 1661 million for the lower middle group, and 319 million for the lower middle group, averaged over 1950–2014). We first calculate yearly deaths attributable to each source region divided by the

population of that source region in each year, and then sum over such a death quantity from 1950 to individual end years. A similar approach is adopted in many previous studies to calculate the cumulative per capita carbon dioxide emission [45–47]. The per capita cumulative mortality contribution by the high-income group has grown linearly until around 1990, with a subsequent slowdown in growth. From 1950 to 2014, the high-income group has always had the highest per capita cumulative mortality impact among the four groups. Statistically, the number of cumulative deaths between 1950 and 2014 attributed to every thousand residents in the high-income group reaches 60, followed by 42 attributed to the upper middle group, 26 to the lower middle group, and 6 to the low-income group.

Fig. 1c further attributes the temporal changes in annual deaths to three driving factors (see detailed information in Supplementary materials: S6). The factors include $PM_{2.5}$ concentrations, population (including size and age structure) and baseline mortality rates (i.e., the death rates caused by individual diseases, for each age range). The baseline mortality rates have been declining throughout the years reflecting improvement in medical and living conditions, and thus have had a negative effect on the number of annual global premature deaths. This effect, however, is more than offset by the growth in $PM_{2.5}$ concentrations and the change in population, which together have driven the continuous growth of global annual premature mortality. The population change includes growing size and aging (see changes in age structure in Fig. S9 online), with elder populations being less resilient against $PM_{2.5}$ pollution exposure [1]. The contribution to mortality by $PM_{2.5}$ concentrations from each of the four groups has changed substantially over the years, reflecting the changes in their anthropogenic emissions (Fig. S6 online). The $PM_{2.5}$ pollution originating from the high-income group switches from growing to declining in 1980 as a result of stringent domestic emission control. The pollution from other three groups has grown continuously throughout the years.

3.2. Considerable transboundary mortality

$PM_{2.5}$ pollution formed from local emissions can be transported in the atmosphere by winds to a variety of distances outside the emission source region, which further causes nonlocal, transboundary mortality [3,10,18]. Fig. 2a shows the cumulative nonlocal $PM_{2.5}$-related premature deaths caused by emissions in each country group since 1950. By 2014, there are about 25.6 million cumulative deaths caused by transboundary $PM_{2.5}$, contributing about 14% of total cumulative premature deaths. From 1950 to 2014, the annual nonlocal contribution ranges from 12% to 15%. The percentage contribution is close to Zhang et al. [3] (about 12%) for 2007 and two HTAP studies for 2001 and 2010, after correcting for the difference in source attribution method; see more discussion in Supplementary materials: S4. Among the four groups, the upper middle group causes the highest number of cumulative nonlocal mortality since 1970 (Fig. 2a). This is in contrast to its second highest contribution to total (local + nonlocal) mortality in the years before 1993 (Fig. 1a). The cumulative nonlocal mortality caused by the lower middle income group has been getting closer to that by the high-income group. The low-income group has always had the lowest nonlocal mortality impacts throughout the years.

Each row in Fig. 2c shows the cumulative premature deaths between 1950 and 2014 in individual receptor regions caused by anthropogenic emissions in a source region. Here, a source or receptor region represents a country group. Between 1950 and 2014, the upper middle group as a source region has caused about 11.2 million cumulative deaths outside its domain, including 5.4 million in the high-income group, 5.1 million in the lower middle group, and 0.7 million in the low-income group. By 2014, the high-income, lower middle and low-income groups are responsible for 6.9 million, 6.5 million and 1.1 million cumulative nonlocal premature deaths, respectively. Overall, about 13%, 14%, 14% and 49% of cumulative deaths caused by the high-income, upper middle, lower middle and low-income groups occur outside their own territories.

Each column in Fig. 2c shows the 1950–2014 cumulative deaths in each income group as a receptor region. There are about 7.0 million, 8.5 million, 8.4 million and 1.8 million premature deaths in the high-income, upper middle, lower middle and low-income groups, respectively, caused by transboundary pollution originating from external source regions. The corresponding contributions to the total mortality in each receptor region are 13%, 11%, 17% and 62%. In general, between any two among four country groups at different affluence levels, the less affluent group bears a larger transboundary mortality impact by the more affluent group than the reciprocal effect. One exception is that the number of deaths in the high-income group caused by the upper middle group is higher than the reciprocal effect (5.4 million versus 4.3 million).

The significant amounts of transboundary mortality contributed by and exerted upon each country group highlight the necessity of mitigating transboundary pollution. The largest percentage contributions of transboundary mortality (62% as receptor and 49% as source) for the low-income country group mean that international technological and financial aids are crucial to help these least capable countries to protect against pollution and to reduce their pollution externality.

We further demonstrate the importance to reduce transboundary pollution by contrasting the estimated historical mortality change caused by the high-income group against a hypothetical scenario which assumes $PM_{2.5}$ concentrations attributable to the high-income group to have kept constant since 1980, i.e., with no effective emission control in the high-income group to reduce $PM_{2.5}$ concentrations. Fig. S10 (online) shows that the hypothetical scenario leads to much more deaths than our estimated historical pathway, with a 1980–2014 cumulative difference of about 9.9 million, including about 8.1 million more local deaths and about 1.8 million more nonlocal deaths. By comparison, during 1980–2014, the cumulative deaths in the high-income group caused by transboundary pollution from all other groups are about 4.8 million. This implies that about 59% of avoided local deaths (8.1 million) due to emission control in the high-income group over 1980–2014 might have been offset by transboundary pollution. The substantial transboundary mortality effect calls for internationally concerted action to reduce emissions for local and nonlocal health benefits.

3.3. Inequality in transboundary mortality

Contemporary history has clearly not been free of anthropogenic pollution and its associated mortality. One would argue that to achieve health equality, in the absence of other factors, every person in individual regions should cause (and bear) the same number of transboundary premature deaths associated with transboundary anthropogenic air pollution.

Based on such an equality argument, Fig. 2b shows the cumulative nonlocal mortality attributable to each person of each country group, by dividing the nonlocal deaths caused by that group by its population on a yearly basis and then summing up the yearly results from 1950 to individual end years. Between 1950 and any end year, the higher the affluence level of a group is, the severer the cumulative transboundary mortality caused by each person of that group is. Quantitatively, the 1950–2014 cumulative transboundary deaths caused by every thousand residents in the

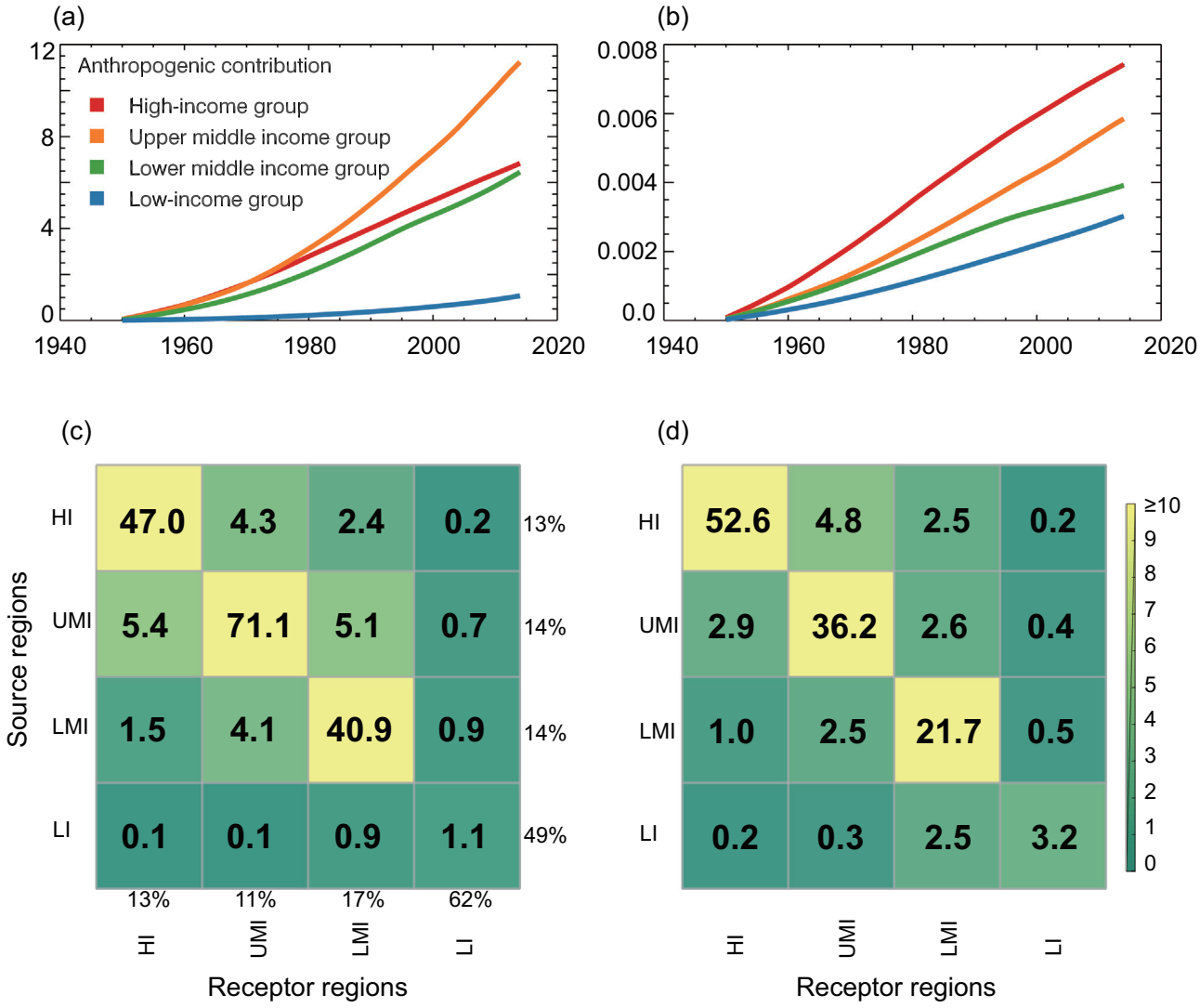

Fig. 2. Transboundary $PM_{2.5}$ health impacts. The cumulative nonlocal premature deaths (a, in Million) and cumulative per capita contribution to nonlocal premature deaths (b) caused by anthropogenic emissions in the four groups. The nonlocal premature deaths represent those deaths occurring out of but attributable to the source region. Each cell in (c) and (d) shows the cumulative deaths by 2014 in a receptor region caused by anthropogenic emissions in a source region (c, in Million) and by anthropogenic emissions per thousand people in a source region (d). In (c) and (d), HI, UMI, LMI and LI denote the high-income, upper middle, lower middle and low-income groups, respectively. Percentage values on the right of (c) represent the fraction of nonlocal deaths attributed to each source region; and percentage values at the bottom of (c) represent the fraction of deaths occurring in each receptor region due to foreign sources.

high-income, upper middle, lower middle and low-income groups are about 7.5, 5.9, 4.0 and 3.0 respectively.

Each cell in Fig. 2d shows the 1950–2014 cumulative premature deaths occurring in a receptor region caused by every thousand residents in a source region. The mortality impact of the high-income group on the upper middle group is higher than the reciprocal impact (4.8 versus 2.9 deaths). Similar inequality exists between the high-income and lower middle groups (2.5 versus 1.0 deaths), between the upper middle and lower middle groups (2.6 versus 2.5), between the upper middle and low-income group (0.4 versus 0.2). Although the transboundary effect (on a per thousand residents' basis) of the low-income group on the lower middle group is larger than the reciprocal effect (2.5 versus 0.5; Fig. 2d), the global total transboundary deaths caused by the low-income group (3.0; Fig. 2b) is still the lowest among the four groups. Overall, the cross-regional inequality shown in Fig. 2b, d can be categorized on a per capita basis as "the richer exert severer health externality".

Another measure of cross-regional inequality can be taken by contrasting the annual nonlocal mortality caused by each person of a group (X) against the annual transboundary mortality risk exerted upon each person of that group (Y, which is calculated as the total deaths in that group caused by foreign emissions divided by the number of population in that group). A Transboundary Mortality Equality Ratio (TMER) can be further defined as the ratio of Y to X. A value of TMER lower than 1 (i.e., $Y < X$; located in light purple area) means that the group is a net source of transboundary mortality, and that the inequality works to the advantage of that group; and TMER > 1 means a net receptor. Fig. 3 tracks the year-to-year evolution of Y contrasting X since 1950. Fig. 3 shows that the high-income group has shifted from being a net source to being a net receptor since 1982. In 2014, the TMER of the high-income group reaches 1.2, that is, the transboundary mortality burden exerted upon each of its residents is 1.2 times as severe as the transboundary burden each of its residents exerts upon other income groups. The upper middle group has become a net source since 1971, and by 2014, its TMER reaches as low as 0.6. The lower middle group has remained a net receptor of transboundary mortality during the past decades, with a TMER of about 1.3 in 2014. The low-income group has also remained a net

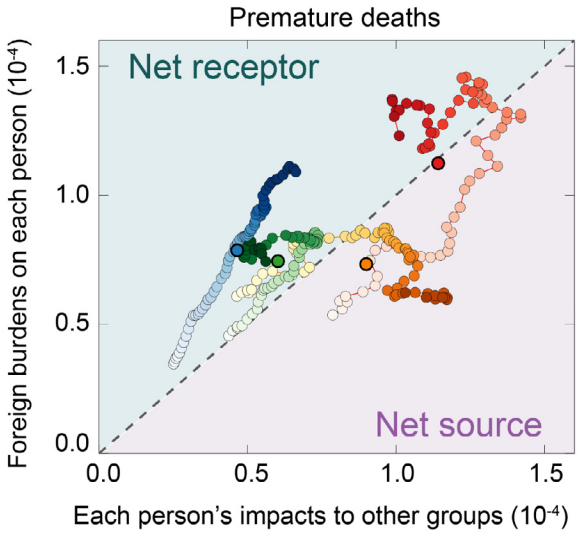

Fig. 3. Health inequality reflected in each person's roles as part of a source versus a receptor region. The *x*-axis shows the nonlocal premature deaths caused by each resident of a specific region; while the *y*-axis shows that region's premature death rate caused by its foreign sources, i.e., that region's premature deaths due to foreign sources divided by that region's population. The circles denote results for individual years, with the lightest colors representing 1950 and the darkest colors representing 2014. The colors differentiate the income groups: high-income (red), upper middle (orange), lower middle (green), and low-income (blue). The circles with thick black coats denote the average situations over 1950–2014.

receptor since 1950, and its TMER has been growing relatively constantly and is larger than that of other three groups in most years. This inequality can be categorized as "the poorest bears the worst net health externality".

4. Discussion and conclusions

In addition to methodological uncertainties described in Supplementary materials: S5, this study is subject to a few limitations. First, the CEDS [12] + MEIC [36–39] emission inventory used here may contain larger uncertainties in the early years. To estimate such errors, we use two other widely used global anthropogenic emission inventories (EDGAR v5.0 [48] and Peking University Inventory [49–53]), combined with the chemical efficiency data and satellite-based correction, to conduct additional estimates of historical anthropogenic $PM_{2.5}$ concentrations and associated deaths. As shown in Fig. S11 (online), the mortality results based on EDGAR and Peking University Inventory are similar to those based on CEDS + MEIC. Second, the baseline mortality rate data are not available prior to 1990. Fixing baseline mortality rates before 1990 would introduce a minor underestimate of mortality over previous years (Fig. S5 online) but does not affect our conclusion. Third, although the toxicity of individual $PM_{2.5}$ components may differ, we assume the same toxicity for all components, consistent with previous studies [3,18,19,40] and World Health Organization assessments [54]. Fourth, anthropogenic emissions of each group considered here are associated with its domestic production and are physically released above its territory. They do not include the emissions released in other regions associated with production to supply that group's consumption via trade [3,11]. The transboundary impacts by each source region would be enhanced had the effect of trade been included [3,11]. Fifth, our simulation of atmospheric chemistry and transport is based on GEOS-Chem, and further multi-model studies [16,17] will help quantify the intrinsic model uncertainty related to transboundary pollution. Sixth, our analysis is focused on four aggregated income groups to provide macroscopic insight of transboundary pollution and embedded inequality. It could be complemented with further studies at national, sub-national and local scales, to reveal potential scale dependence of the significance of transboundary pollution and inequality. Seventh, the period considered in this study is from 1950 to 2014 when there are significant changes in magnitude and distribution of anthropogenic emissions worldwide. If a longer period is considered (e.g., since 1900), the dominance of high-income group's contribution to premature deaths would not be influenced, since anthropogenic emissions from high-income group are dominant for majority of pollutants before 1950 [12]. However, some turning points (such as the intersection between high-income and upper middle income groups in Fig. 1a) would be postponed.

Overall, this study shows enormous cumulative loss of lives from 1950 to 2014 as a result of transboundary $PM_{2.5}$ pollution contributed by four country groups at different affluence levels. The evolution of transboundary pollution is accompanied by a serious cross-regional health inequality problem, with the poorest country group bearing the worst net health externality after considering transboundary pollution produced by versus exerted upon each resident of each group. The upper middle group has become the leading net source of transboundary mortality since decades ago. Recently, China has substantially reduced its emissions and $PM_{2.5}$ pollution [8,14], whereas many other middle-income countries still suffer from continuous emission increases (except for the disruption by COVID-19) [13]. In the future, the geographical pattern of emissions will continue to change along with the economic globalization, with production and emissions mostly likely moved to less affluent tropical countries [55], in part accelerated by multi-lateral agreements such as the Belt and Road Initiative (BRI) [56] and the Regional Comprehensive Economic Partnership (RCEP) [57]. It is crucial that environmental cooperation be enhanced to achieve mutual benefits for regions under development and others susceptible to potential transboundary pollution from these developing regions. For example, environmental co-benefits could be made a core, mandate measure of the successfulness of multi-lateral collaborations; and technological and financial support to less affluent parties could be enforced and enhanced through the mechanism laid out in the *Paris Agreement* [58], especially given the health inequality in historical transboundary pollution to the disadvantage of these regions. To this end, our study offers important insight to historical lessens on transboundary pollution and cross-regional health equality, upon which collaborative economic-environmental strategy could be established to ensure sustainable development.

Conflict of interest

The authors declare that they have no conflict of interest.

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Author contributions

Jintai Lin and Lulu Chen conceived and designed the research. Lulu Chen performed the research. Hongjian Weng, Hao Kong, Ruijing Ni and Jun Meng contributed to model simulations. Aaron von Donkelaar and Randall Martin provided the satellite-derived $PM_{2.5}$ data. Lulu Chen and Jintai Lin led the analysis with inputs from Randall Martin and Mingxi Du. Lulu Chen and Jintai Lin wrote the paper and all authors commented on the writing.

Appendix A. Supplementary materials

Supplementary materials to this article can be found online at https://doi.org/10.1016/j.scib.2021.11.007.

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