Spatial and sector-specific contributions of emissions to ambient air pollution and mortality in European cities: a health impact assessment

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See **Comment** page e480

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Summary

Background Ambient air pollution is a major risk to health and wellbeing in European cities. We aimed to estimate spatial and sector-specific contributions of emissions to ambient air pollution and evaluate the effects of source-specific reductions in pollutants on mortality in European cities to support targeted source-specific actions to address air pollution and promote population health.

Methods We conducted a health impact assessment of data from 2015 for 857 European cities to estimate source contributions to annual $PM_{2·5}$ and NO_2 concentrations using the Screening for High Emission Reduction Potentials for Air quality tool. We evaluated contributions from transport, industry, energy, residential, agriculture, shipping, and aviation, other, natural, and external sources. For each city and sector, three spatial levels were considered: contributions from the same city, the rest of the country, and transboundary. Mortality effects were estimated for adult populations (ie, ≥20 years) following standard comparative risk assessment methods to calculate the annual mortality preventable on spatial and sector-specific reductions in $PM_{2·5}$ and NO_2.

Findings We observed strong variability in spatial and sectoral contributions among European cities. For $PM_{2·5}$, the main contributors to mortality were the residential (mean contribution of 22·7% [SD 10·2]) and agricultural (18·0% [7·7]) sectors, followed by industry (13·8% [6·0]), transport (13·5% [5·8]), energy (10·0% [6·4]), and shipping (5·5% [5·7]). For NO_2, the main contributor to mortality was transport (48·5% [SD 15·2]), with additional contributions from industry (15·0% [10·8]), energy (14·7% [12·9]), residential (10·3% [5·0]), and shipping (9·7% [12·7]). The mean city contribution to its own air pollution mortality was 13·5% (SD 9·9) for $PM_{2·5}$ and 34·4% (19·6) for NO_2, and contribution increased among cities of largest area (22·3% [12·2] for $PM_{2·5}$ and 52·2% [19·4] for NO_2) and among European capitals (29·9% [12·5] for $PM_{2·5}$ and 62·7% [14·7] for NO_2).

Interpretation We estimated source-specific air pollution health effects at the city level. Our results show strong variability, emphasising the need for local policies and coordinated actions that consider city-level specificities in source contributions.

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Introduction

Ambient air pollution is a main global environmental risk to health and wellbeing. Ambient air pollution has been associated with various adverse health effects, including the development and aggravation of cardiovascular and respiratory disease, cancer, cognitive decline, mental health disorders, adverse birth outcomes, and premature mortality.[1–3] Previous studies have estimated a substantial health burden related to ambient air pollution. Globally, 4–9 million annual premature deaths were associated with exposure to particulate matter with diameter of ≤2·5 μm ($PM_{2·5}$) in 2015.[4,5] In Europe, the health burden related to exposure to ambient air pollution has decreased in the past 30 years, but remained high at an estimated 307 000 annual premature deaths for $PM_{2·5}$ and 40 400 for nitrogen dioxide (NO_2) in 2019,[6] emphasising the urgent need to take actions to further reduce air pollution and protect health in Europe.

To establish policy actions that achieve effective reductions in air pollution, it is important to identify the origins of air pollutants, both spatially (ie, from local or non-local sources) and by polluting sectors. Source apportionment studies can help to identify origins and can be achieved by use of distinct approaches, such as incremental methods, mass-transfer methods, and emission reduction impact (ERI) methods.[7] Among these approaches, ERI methods have been widely used in previous global and European assessments[8–12] and

Research in context

Evidence before this study

We searched the PubMed and Google Scholar databases, without language restrictions, from database inception until March 1, 2023, for studies on source apportionment of ambient air pollution and source-specific health effects. Our search terms were "air pollution" OR "$PM_{2·5}$" OR "NO_2" OR "particulate matter" OR "nitrogen dioxide" AND "source" OR "contribution" OR "source contribution" OR "source apportionment" AND "health impact" OR "impact assessment" OR "mortality" OR "premature mortality" OR "health burden" AND "city" OR "cities" OR "Europe". We included studies based on emission reduction impact methods that presented estimates on source apportionment of ambient air pollution or health effects related to each source. Only studies based on emission reduction impact methods (including chemical transport model [CTM] simulations and other approaches, such as reduced-complexity CTM or adjoint sensitivity methods) were included because these were considered most informative for policy making due to their ability to adequately reflect the effects of changes in emissions on pollutant concentrations. We considered only health impact assessment or burden of disease studies and excluded any epidemiological studies (ie, cohort, case-control, and cross-sectional studies) and studies that did not cover the European region. We identified three relevant studies that assessed source contributions to ambient $PM_{2·5}$ and NO_2 and their health burden on a global scale. These studies were based on CTM simulations at a horizontal resolution of approximately 50×50 km^2. However, this scale of analysis provided little evidence for cities and local air quality plans. We additionally identified three city-level studies that estimated $PM_{2·5}$ source contributions for cities in the Danube and Western Balkans region, for 150 European cities, and for 96 global cities. These studies were based on reduced-complexity CTM methods and were at finer resolutions starting at approximately 3×4 km^2 and up to 250×250 km^2 for the 96 cities study and at 10×10 km^2 for the other two studies. Nevertheless, none of these studies included NO_2 nor estimated the health effects associated with source-specific pollution levels.

Added value of this study

This is the first study to estimate source contributions to ambient $PM_{2·5}$ and NO_2 concentrations and evaluate the effects of source-specific pollutant reductions on mortality for over 800 European cities. We present detailed estimates for each city and provide policy-relevant and novel insights into actions that might be most effective to target air pollution and promote population health. The main strengths of this study include the detailed city-level analyses, the large number of analysed cities, the robust source apportionment methodology at an improved resolution of 6×6 km^2, the estimation of the mortality burden associated to each pollutant source, and the overall harmonised assessment and direct applicability for policy making.

Implications of all the available evidence

The results presented in this study have direct implications for policy. We found partial agreement with previous global studies on the importance of residential and agricultural contributions to $PM_{2·5}$ mortality and transport contributions to NO_2 mortality. Adding up to the previous evidence, our results provide spatial estimates and emphasise local differences in source contributions, providing novel insights into what targeted actions could be taken specifically in each local context. Further research is needed to evaluate which particulate matter components might have a more detrimental effect on health and provide more accurate evidence on what sectors should be targeted to prioritise health benefits.

provide valuable insights for policy making.[7] ERI methods are generally based on chemical transport model (CTM) simulations and estimate spatial and sectoral contributions to air pollution on the basis of concentration differences resulting from modelling considering all emissions versus reduced emissions over specific regions or source sectors.[7] Additionally, ERI methods can be based on simplified approaches that reduce computational time, such as reduced-complexity CTM[11–14] or adjoint sensitivity methods.[15] Overall, the ERI approach has the advantage of showing the effects of changes in emissions on pollutant concentrations; being specific, meaning that concentration changes are related to only one source or one group of sources; and showing an additive response in source contributions, meaning that the sum of contributions estimated for each source equals the total estimated for all sources together, for annual concentrations under specific emission reduction ranges.[7] Accordingly, this approach is informative for air quality planning and can be used to evaluate the effects of distinct policy actions to target air pollution.[7]

Previous studies based on ERI methods have assessed source contributions to ambient $PM_{2·5}$ and NO_2 and their health burden on global and regional scales.[8–10] Although these studies have provided useful insights into the sectors with the highest associated health burden and regional policy actions, the scale of the analyses impairs their implementation into local air quality plans. To address this limitation, studies have evaluated source contributions at the city level.[11–15] City-level analyses allow the study of local contributions to ambient air pollution and show greater detail than regional analyses, allowing for more targeted actions.[13] Currently, city-level source contributions to ambient $PM_{2·5}$ are available for specific regions, capitals, and major urban areas in Europe and globally.[11–14] Nevertheless, none of these studies have included NO_2 or estimated the health effects of source-specific pollution concentrations for European cities, emphasising the need for city-level studies to provide

local evidence on what strategies for reducing air pollution might be the most effective to protect population health.

In this Article, we aimed to estimate spatial and sectoral contributions of emissions to ambient concentrations of $PM_{2·5}$ and NO_2 and evaluate the effects of source-specific reductions in these pollutants on mortality for European cities for the year 2015, the latest year for which all data were available for all cities included in the study. We used the Screening for High Emission Reduction Potentials for Air Quality (SHERPA) tool, based on the ERI approach and developed by the Joint Research Centre.[16] By focusing on cities, we aimed to emphasise local differences in spatial and sectoral contributions to air pollution and mortality, thus providing an indication of what targeted actions could be taken specifically in each local context.

Methods

Study design and city definition

We did a health impact assessment of data from 2015 for European cities and greater cities defined in the Urban Audit 2018 dataset, which provides a harmonised definition of all European cities on the basis of population density and local administrative boundaries.[17,18] Overall, this dataset contains 980 cities and 49 greater cities in 31 European countries (ie, the 27 EU countries, the UK, Norway, Switzerland, and Iceland). The 49 greater cities cover 161 cities either by representing a city of larger area or by constituting a combination of several smaller-size cities. We excluded cities located outside of Europe, such as Saint Denis (Reunion) and Fort-de-France (Martinique); nine cities and two greater cities located in Madeira (Portugal), Azores (Portugal), and the Canary Islands (Spain) due to lack of modelled air pollution estimates; and Reykjavik (Iceland) due to lack of coverage by the SHERPA model. To avoid double-counting, we excluded the smaller cities within the greater city areas and conducted the analysis for the remaining 857 cities (appendix 1).

Procedures

We estimated spatial and sectoral contributions to annual mean $PM_{2·5}$ and NO_2 concentrations in European cities by use of the SHERPA tool, based on the ERI approach.[16] SHERPA is a simplified version of a CTM based on source receptor relationships (SRRs), which relate gridded emission changes to concentration changes and account for the same processes modelled in a CTM, such as meteorology or chemical transformations.[13] In this way, SHERPA mimics the dynamic responses of a CTM but with reduced computational time, allowing the simultaneous evaluation of source contributions to air pollution for many cities.[13] We focused on $PM_{2·5}$ and NO_2 due to the predominance of these pollutants in urban areas, larger effect of local policies on their concentrations than on other pollutant concentrations,[19] and strong associations between annual exposures and health effects. Although O_3 also has notable health effects,[20] this pollutant was not considered in our analysis because it shows strong seasonal variability and SHERPA is currently not able to capture processes over time periods shorter than 1 year due to the large non-linearities that occur over time.

In the implementation used in this Article, the SRRs for $PM_{2·5}$ and NO_2 were built at a resolution of 0·1×0·05° (approximately 6×6 km²) on the basis of the European Monitoring and Evaluation Programme for Transboundary Long-Range Transported Air Pollutants Meteorological Synthesizing Centre-West (EMEP MSC-W) model (version 4.34) for the meteorological year 2015 (appendix 2 pp 4–5).[21,22] Baseline emissions for 2015 were available from the Copernicus Atmosphere Monitoring Service regional inventory (version 4.2) and classified into 12 sectors by use of the Gridded Nomenclature for Reporting: public power, industry, other stationary combustion, fugitives, solvents, road transport, shipping, aviation, off-road transport, waste burning, livestock agriculture, and other agriculture (appendix 2 p 6). We considered five emission precursors: nitrogen oxides, non-methane volatile organic compounds, NH_3, primary $PM_{2·5}$, and sulphur oxides. The primary $PM_{2·5}$ emissions from other stationary combustion were replaced with those from the REF2 bottom-up inventory, which consistently includes the condensable particulate matter fraction from residential wood combustion.[23–26]

To evaluate local versus non-local source contributions to $PM_{2·5}$ and NO_2, we defined three spatial levels of source emission reductions: the city, the rest of the country, and transboundary (ie, contributions from all other European countries in the model domain, except the country of interest; appendix 2 pp 8–10). Receptor points at which changes in pollutant concentrations due to changes in source emissions are evaluated were defined for each city on the basis of the emissions and the Global Human Settlement Layer (GHSL) grid cells, which contain population data at 250 m resolution (appendix 2 pp 11–12).[27] We applied SHERPA to each receptor and spatial level for all Gridded Nomenclature for Reporting sectors and precursors at a time and calculated the relative source contributions grouped into: transport, industry, energy, residential, agriculture, shipping, aviation, and other sources (appendix 2 p 6). For $PM_{2·5}$, we estimated contributions from natural sources by use of the salt and dust components from the EMEP MSC-W model, and for NO_2, the natural component was assumed negligible. An additional category of external contributions was created for $PM_{2·5}$ when the sum of all source contributions was below 100%, representing contributions from emissions out of the EMEP geographical area or not accounted for by the considered emission sources (eg, biogenic emissions or volcanic emissions in Sicily).[13]

Given that the SRRs resolution was 0·1×0·05°, the local city contribution estimates for small-size cities of less than

See Online for appendix 2

For the **emissions data** see https://eccad.aeris-data.fr

See Online for appendix 1

	Contribution to premature mortality, %		Premature deaths, point estimate (95% CI)	Deaths per 100 000 population, n	
	Mean (SD)	Median (IQR; range)		Mean (SD)	Median (IQR; range)
Overall effects					
Transport	13·5% (5·8)	13·1% (8·6–18·2; 1·5–28·5)	26 679 (24 387–28 969)	14·7 (7·7)	14·0 (9·5–18·3; 1·7–60·0)
Industry	13·8% (6·0)	12·6% (10·1–15·7; 3·1–53·5)	29 977 (27 463–32 493)	15·5 (8·6)	13·5 (9·7–18·6; 2·8–61·4)
Energy	10·0% (6·4)	8·4% (5·0–13·9; 0·1–41·3)	19 424 (17 863–20 995)	12·3 (11·1)	8·0 (4·4–16·2; 0·0–62·7)
Residential	22·7% (10·2)	21·3% (13·9–30·6; 3·7–63·7)	48 433 (44 402–52 470)	26·9 (18·9)	20·1 (13·5–34·8; 3·3–102·0)
Agriculture	18·0% (7·7)	18·8% (12·0–24·4; 0·0–34·7)	33 780 (30 958–36 600)	20·3 (11·0)	19·7 (12·0–28·4; 0·0–57·1)
Shipping	5·5% (5·7)	3·5% (1·2–8·2; 0·0–41·5)	10 116 (9239–10 999)	5·5 (6·3)	3·2 (1·4–7·8; 0·0–50·4)
Aviation	0·4% (0·8)	0·2% (0·1–0·4; 0·0–15·6)	1000 (914–1086)	0·4 (0·8)	0·2 (0·1–0·4; 0·0–15·5)
Natural	8·8% (9·3)	5·4% (3·5–9·7; 1·3–69·8)	15 339 (14 016–16 667)	9·0 (9·5)	5·1 (3·9–10·2; 0·6–80·1)
Other	3·4% (2·6)	2·8% (1·8–4·3; 0·1–30·8)	9429 (8620–10 236)	3·8 (3·1)	3·1 (1·9–4·6; 0·1–28·9)
City effects					
City	13·5% (9·9)	10·2% (6·3–18·4; 0·4–59·1)	44 251 (40 558–47 936)	15·2 (13·3)	11·2 (6·5–19·5; 0·4–119·0)
Transport	2·3% (1·9)	1·7% (1·1–2·8; 0·1–15·1)	7807 (7133–8476)	2·4 (1·9)	1·9 (1·2–3·0; 0·1–21·0)
Industry	3·2% (4·1)	1·7% (0·7–4·0; 0·0–43·1)	10 110 (9272–10 945)	3·6 (5·5)	1·9 (0·8–4·1; 0·0–48·0)
Energy	1·4% (2·8)	0·4% (0·1–1·2; 0·0–28·5)	4502 (4147–4862)	1·7 (4·0)	0·4 (0·1–1·4; 0·0–44·6)
Residential	4·5% (4·6)	2·7% (1·4–5·9; 0·2–31·9)	13 526 (12 409–14 642)	5·1 (5·8)	2·9 (1·4–7·1; 0·2–49·6)
Agriculture	0·8% (0·6)	0·6% (0·4–1·0; 0·0–5·1)	1795 (1642–1947)	0·8 (0·7)	0·6 (0·4–1·1; 0·0–4·9)
Shipping	0·4% (1·3)	0·0% (0·0–0·1; 0·0–13·9)	1407 (1287–1528)	0·4 (1·3)	0·0 (0·0–0·1; 0·0–16·2)
Aviation	0·1% (0·7)	0·0% (0·0–0·0; 0·0–15·3)	470 (431–510)	0·1 (0·6)	0·0 (0·0–0·0; 0·0–15·2)
Other	0·9% (1·2)	0·6% (0·3–1·0; 0·0–19·1)	4634 (4237–5029)	1·0 (1·3)	0·6 (0·3–1·1; 0·0–17·9)
Country effects					
Country	46·8% (16·5)	46·9% (34·7–60·2; 4·5–83·6)	87 658 (80 264–95 055)	52·4 (27·7)	47·1 (35·2–63·7; 1·5–194·0)
Transport	8·0% (4·6)	7·4% (3·9–11·4; 0·1–20·9)	13 695 (12 512–14 879)	8·6 (5·8)	7·9 (4·5–11·2; 0·0–43·2)
Industry	7·2% (4·9)	6·2% (4·2–9·1; 0·2–43·2)	14 060 (12 869–15 254)	7·9 (5·6)	6·5 (4·0–10·1; 0·2–46·3)
Energy	4·8% (4·6)	3·3% (1·1–7·1; 0·0–30·8)	8377 (7694–9060)	5·5 (6·3)	3·1 (1·2–7·8; 0·0–45·8)
Residential	12·8% (6·8)	10·7% (7·8–17·7; 0·3–32·2)	25 108 (23 004–27 213)	15·1 (11·6)	11·3 (7·0–19·3; 0·4–75·2)
Agriculture	11·2% (5·6)	11·1% (6·6–15·2; 0·0–24·8)	21 211 (19 428–22 992)	12·4 (7·2)	11·4 (7·3–16·4; 0·0–42·3)
Shipping	0·7% (1·3)	0·3% (0·1–0·7; 0·0–15·5)	1166 (1067–1266)	0·7 (1·4)	0·3 (0·1–0·7; 0·0–20·4)
Aviation	0·2% (0·5)	0·1% (0·0–0·2; 0·0–5·0)	433 (395–471)	0·2 (0·4)	0·1 (0·0–0·2; 0·0–4·1)
Other	1·9% (2·1)	1·1% (0·6–2·6; 0·0–18·1)	3607 (3294–3920)	2·0 (2·4)	1·3 (0·7–2·5; 0·0–20·3)
Transboundary effects					
Transboundary	27·0% (13·6)	23·6% (16·1–35·4; 4·8–74·5)	46 929 (43 023–50 858)	31·7 (23·3)	26·4 (16·0–39·2; 2·1–183·0)
Transport	3·2% (2·5)	2·5% (1·4–4·2; 0·3–17·7)	5177 (4741–5615)	3·7 (3·2)	2·8 (1·4–4·9; 0·2–19·6)
Industry	3·5% (2·2)	2·8% (2·0–4·5; 0·4–14·9)	5807 (5322–6295)	4·0 (3·2)	3·0 (1·9–4·9; 0·3–24·6)
Energy	3·8% (4·2)	2·2% (1·1–4·8; 0·1–24·6)	6545 (6021–7073)	5·0 (7·0)	2·4 (1·0–5·8; 0·0–51·8)
Residential	5·3% (4·2)	4·1% (2·3–6·9; 0·4–30·8)	9799 (8989–10 615)	6·7 (7·2)	4·4 (2·0–8·3; 0·3–61·8)
Agriculture	6·0% (4·3)	4·9% (2·7–9·0; 0·0–23·0)	10 774 (9888–11 661)	7·1 (6·2)	5·3 (2·6–10·1; 0·0–41·5)
Shipping	4·4% (4·7)	2·8% (1·0–6·2; 0·0–35·2)	7543 (6885–8205)	4·4 (5·2)	2·4 (1·1–5·9; 0·0–42·8)
Aviation	0·1% (0·1)	0·0% (0·0–0·1; 0·0–1·2)	97 (89–106)	0·1 (0·1)	0·0 (0·0–0·1; 0·0–1·0)
Other	0·6% (0·5)	0·5% (0·3–0·8; 0·0–3·1)	1188 (1088–1288)	0·7 (0·6)	0·5 (0·3–1·1; 0·0–4·7)

Table 1: **$PM_{2·5}$ effects on premature mortality by sector and spatial level**

60 km² (covered by less than one 0·1×0·05° emission grid cell) could be underestimated. To explore the influence of city size on the city contribution estimates, we used Pearson's test to calculate the correlation between both variables and the mean estimates by city-size group (ie, small [<60 km², n=191), medium [60–300 km², n=498], and large [>300 km², n=168]).

We retrieved population data from the GHSL and mortality data from Eurostat for European cities for the year 2015.[27–30] For each city, we estimated the natural-cause mortality rates (excluding the external causes of death) by 5-year age group (appendix 2 p 13). We followed the comparative risk assessment approach[31–35] to estimate the effects of spatial and sector-specific reductions in annual mean $PM_{2·5}$ and NO_2 concentrations on natural-cause mortality for adult residents (ie, ≥20 years) of European cities, assuming equal toxicity for all sources. We used the risk estimates of 1·08 (95% CI 1·06–1·09)

per 10 µg/m³ for $PM_{2·5}$ and 1·02 (1·01–1·04) per 10 µg/m³ for NO_2 increase in pollutant concentrations as exposure response functions (ERFs), available from meta-analyses.[36,37] For each GHSL population grid cell, we used the modelled estimates from our previous work for European cities as baseline $PM_{2·5}$ and NO_2 exposure values (appendix 2 p 13)[32] and assigned the relative source contributions from the corresponding receptor points. We multiplied the baseline exposures by the relative contributions and used the resulting values as the exposure difference due to spatial and sector-specific reductions in $PM_{2·5}$ and NO_2. We used the ERFs to calculate the relative risk associated with the exposure difference and estimated the population attributable fractions. Finally, we calculated the number of natural-cause deaths by age group for each GHSL grid cell and multiplied it by the population attributable fractions. For each city, we aggregated the results for all GHSL grid cells and age groups to calculate the number of premature deaths that could be prevented on each spatial and sector-specific reduction in $PM_{2·5}$ and NO_2. Note that premature mortality refers to the excess number of annual deaths that could be avoided if pollutant concentrations related to each emission source were eliminated. For a comparison of the relative importance of each source in contributing to premature mortality, we calculated the percent contribution of each source as the ratio between the premature deaths associated with each source and the sum of premature deaths due to all sources. Point estimates and 95% CIs were estimated by propagating the uncertainties in the ERFs, baseline $PM_{2·5}$ and NO_2 concentrations, and the mean age of death using Monte Carlo simulations.[34–37]

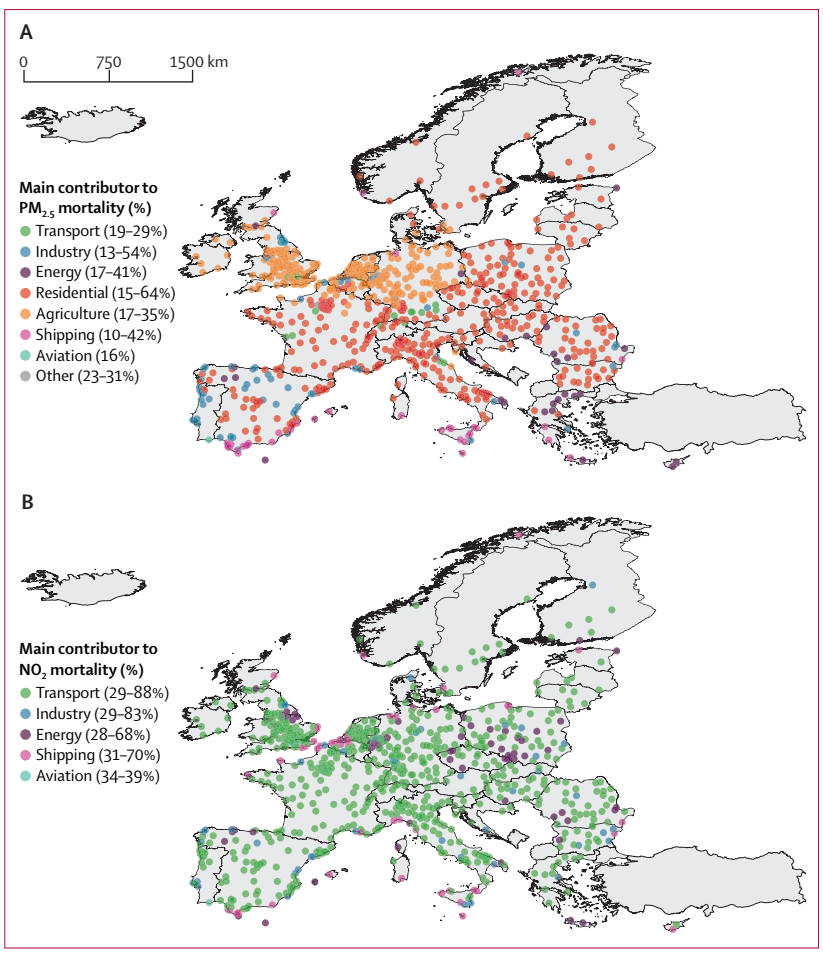

Figure 1: **Main anthropogenic sectors contributing to premature mortality**
Sectors contributing to mortality for $PM_{2·5}$ (A) and NO_2 (B).

Sensitivity analyses

We conducted sensitivity analyses to evaluate the effects of alternative SHERPA model assumptions. First, we evaluated the effect of using the previous SHERPA version at 0·1×0·1° resolution (approximately 10×10 km²), which did not account for condensable particulate matter.[11] Second, we evaluated the effect of the city boundary definition and an alternative location of the receptor points. For the first analysis, we considered small-size cities of less than 60 km² (n=191). We created buffers around each city boundary from 1–5 km in 1 km increments and repeated the analysis for each buffer area. For the second analysis, we defined a unique receptor point per city located at the centroid of all GHSL grid cells, representing a central and populated area of each city, as the alternative location (appendix 2 p 12). Additionally, we conducted sensitivity analyses to evaluate the effect of alternative source apportionment models. We retrieved sectoral contributions for $PM_{2·5}$ and NO_2 from two global CTMs: the Earth System Atmospheric Chemistry Model and the Global Burden of Disease Major Air Pollution Sources Model.[8–10] These models were chosen because they constitute two main global models based on ERI methods for which data could be accessed. For comparability, the sectors evaluated in both models were grouped to match the SHERPA sector definitions (appendix 2 p 7). Statistical analyses were conducted in Python (version 3.9.6) and R (version 4.0.3).

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

A total of 168 024 201 adults aged 20 years or older resided in the 857 included European cities in 2015, representing 32% of the total population in the analysed countries. The adult population by city had a median of 93 946 inhabitants (IQR 60 298–163 647; range 6314–6 250 746). Overall, 1 895 599 deaths from natural causes were observed in 2015 among all cities (appendix 1). Median annual exposure to $PM_{2·5}$ was 12·5 µg/m³ (IQR 10·1–14·1; range 0·7–30·8) among all

	Contribution to premature mortality, %		Premature deaths, point estimate (95% CI)	Deaths per 100 000 population, n	
	Mean (SD)	Median (IQR; range)		Mean (SD)	Median (IQR; range)
Overall effects					
Transport	48·5% (15·2)	49·3% (38·4–59·9; 3·4–88·0)	43 540 (28 512–58 551)	22·6 (7·5)	22·6 (17·5–27·3; 1·8–49·1)
Industry	15·0% (10·8)	11·8% (8·4–17·3; 1·7–82·8)	13 073 (8626–17 534)	7·3 (5·8)	5·5 (3·5–9·0; 0·5–41·6)
Energy	14·7% (12·9)	10·4% (4·8–22·2; 0·0–68·3)	14 111 (9468–18 744)	7·5 (7·3)	4·9 (2·1–10·9; 0·0–42·0)
Residential	10·3% (5·0)	9·5% (7·0–12·9; 0·5–31·9)	11 105 (7268–14 958)	4·9 (2·8)	4·4 (3·0–6·4; 0·2–22·1)
Agriculture	0·0% (0·1)	0·0% (0·0–0·0; 0·0–1·6)	18 (12–23)	0·0 (0·1)	0·0 (0·0–0·0; 0·0–0·8)
Shipping	9·7% (12·7)	4·3% (1·4–12·9; 0·0–70·0)	9020 (5906–12 140)	4·7 (6·6)	1·9 (0·7–6·2; 0·0–39·4)
Aviation	1·4% (3·0)	0·5% (0·2–1·2; 0·0–38·8)	1991 (1304–2682)	0·7 (1·4)	0·2 (0·1–0·6; 0·0–13·9)
Other	0·4% (0·6)	0·3% (0·2–0·5; 0·0–8·1)	396 (260–533)	0·2 (0·4)	0·1 (0·1–0·2; 0·0–6·6)
City effects					
City	34·4% (19·6)	31·3% (18·3–48·5; 2·7–90·4)	44 034 (28 962–59 078)	15·8 (9·5)	13·8 (8·5–20·9; 1·2–52·6)
Transport	17·0% (12·1)	13·7% (7·8–24·0; 0·4–70·6)	20 988 (13 730–28 228)	7·5 (4·9)	6·5 (3·8–10·3; 0·2–35·4)
Industry	5·7% (8·5)	3·0% (1·3–5·9; 0·0–68·9)	5678 (3746–7612)	2·7 (4·2)	1·3 (0·6–2·7; 0·0–30·6)
Energy	5·0% (8·1)	1·7% (0·2–5·4; 0·0–54·5)	6448 (4337–8550)	2·4 (4·2)	0·8 (0·1–2·8; 0·0–32·0)
Residential	4·2% (3·3)	3·2% (1·9–5·5; 0·1–27·0)	6838 (4472–9213)	2·0 (1·7)	1·5 (0·9–2·5; 0·0–13·5)
Agriculture	0·0% (0·0)	0·0% (0·0–0·0; 0·0–0·3)	1 (1–2)	0·0 (0·0)	0·0 (0·0–0·0; 0·0–0·1)
Shipping	1·9% (5·1)	0·0% (0·0–0·7; 0·0–40·0)	2772 (1818–3715)	0·9 (2·6)	0·0 (0·0–0·3; 0·0–24·6)
Aviation	0·4% (2·1)	0·0% (0·0–0·0; 0·0–35·7)	1117 (732–1503)	0·2 (0·9)	0·0 (0·0–0·0; 0·0–12·6)
Other	0·2% (0·4)	0·1% (0·0–0·2; 0·0–4·1)	192 (126–259)	0·1 (0·2)	0·0 (0·0–0·1; 0·0–2·9)
Country effects					
Country	48·9% (20·1)	49·2% (34·3–64·2; 0·6–94·3)	37 262 (24 504–50 034)	24·0 (12·9)	22·3 (14·2–31·7; 0·2–72·3)
Transport	26·0% (12·0)	25·5% (17·0–34·2; 0·3–66·3)	18 763 (12 273–25 247)	12·4 (6·3)	11·8 (7·8–16·4; 0·1–35·4)
Industry	7·4% (6·1)	5·8% (3·6–9·4; 0·0–52·6)	6184 (4073–8302)	3·7 (3·5)	2·7 (1·6–4·7; 0·0–29·4)
Energy	7·5% (8·8)	3·9% (1·5–10·2; 0·0–55·8)	6269 (4195–8342)	3·9 (5·1)	1·9 (0·6–5·1; 0·0–30·8)
Residential	5·1% (3·8)	4·4% (2·8–6·3; 0·0–28·6)	3553 (2325–4787)	2·5 (2·2)	2·1 (1·2–3·1; 0·0–20·1)
Agriculture	0·0% (0·1)	0·0% (0·0–0·0; 0·0–0·6)	8 (5–11)	0·0 (0·0)	0·0 (0·0–0·0; 0·0–0·3)
Shipping	1·8% (3·8)	0·5% (0·2–1·7; 0·0–44·9)	1531 (1008–2059)	1·0 (2·2)	0·2 (0·1–0·8; 0·0–23·6)
Aviation	0·9% (1·9)	0·3% (0·1–0·7; 0·0–18·1)	790 (518–1066)	0·4 (0·9)	0·1 (0·0–0·4; 0·0–9·5)
Other	0·2% (0·3)	0·1% (0·1–0·2; 0·0–3·1)	164 (107–221)	0·1 (0·2)	0·1 (0·0–0·1; 0·0–1·8)
Transboundary effects					
Transboundary	16·7% (13·3)	12·9% (6·4–23·4; 0·7–72·1)	11 959 (7890–16 050)	8·2 (7·2)	6·2 (2·9–11·4; 0·3–54·1)
Transport	5·4% (5·0)	3·6% (1·9–7·5; 0·2–39·9)	3789 (2509–5075)	2·7 (2·7)	1·8 (0·8–3·9; 0·1–19·0)
Industry	1·8% (2·2)	1·2% (0·6–2·2; 0·1–38·2)	1212 (807–1621)	0·9 (1·2)	0·5 (0·3–1·2; 0·0–16·0)
Energy	2·3% (3·7)	1·0% (0·4–2·6; 0·0–38·9)	1394 (937–1853)	1·2 (2·0)	0·5 (0·2–1·3; 0·0–19·6)
Residential	1·0% (1·0)	0·6% (0·3–1·3;0·0–8·6)	714 (472–958)	0·5 (0·5)	0·3 (0·1–0·7; 0·0–4·3)
Agriculture	0·0% (0·1)	0·0% (0·0–0·0; 0·0–1·6)	8 (6–11)	0·0 (0·0)	0·0 (0·0–0·0; 0·0–0·7)
Shipping	6·0% (8·1)	2·9% (1·0–7·2; 0·0–48·9)	4718 (3079–6367)	2·8 (4·0)	1·3 (0·5–3·6; 0·0–30·3)
Aviation	0·1% (0·4)	0·0% (0·0–0·1; 0·0–7·5)	83 (54–113)	0·1 (0·2)	0·0 (0·0–0·1; 0·0–3·0)
Other	0·1% (0·1)	0·0% (0·0–0·1; 0·0–2·4)	40 (27–53)	0·0 (0·1)	0·0 (0·0–0·0; 0·0–1·9)

Table 2: **NO_2 effects on premature mortality by sector and spatial level**

GHSL grid cells, and median annual exposure to NO_2 was 20·3 μg/m³ (15·2–25·8; 0·7–84·5).

For $PM_{2·5}$, the main sectoral contributors to premature mortality were the residential and agricultural sectors, followed by industry, transport, energy, and shipping (table 1; figure 1). For NO_2, the main sectoral contributor to premature mortality was transport, with substantial additional contributions from industry, energy, residential, and shipping sectors (table 2; figure 1).

We observed considerable variability in sectoral and spatial contributions to mortality among the analysed cities (figures 2–3). Overall, for $PM_{2·5}$, contributions to premature mortality from the residential sector were highest in eastern and northern Europe (appendix 2 p 14). The contributions from agriculture were highest over Germany, the Netherlands, Belgium, and the UK (appendix 2 p 14). Contributions from industry were widespread with highest estimates for cities in the UK, Spain, Portugal, Germany, France, and Romania

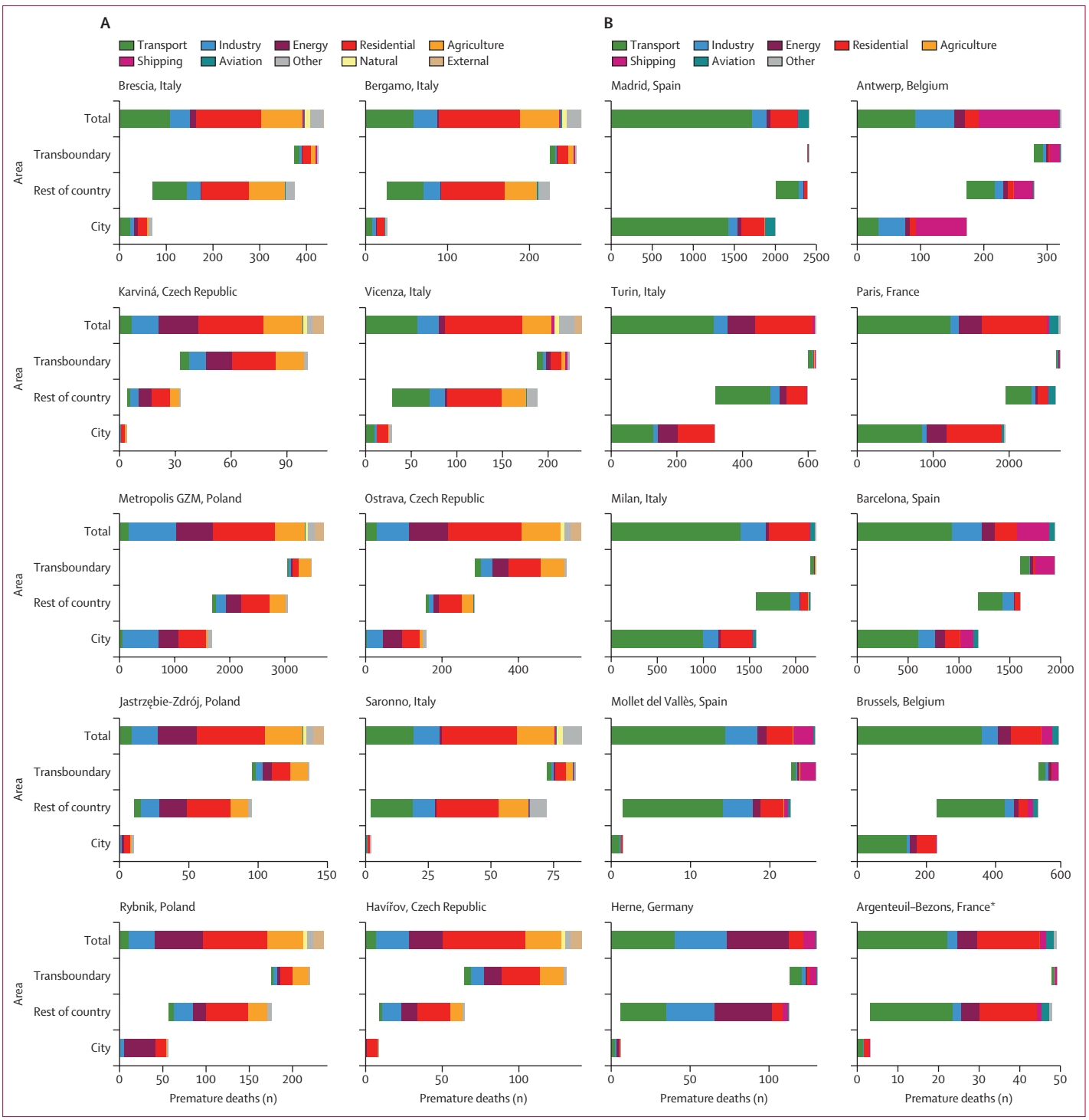

Figure 2: **Sectoral and spatial contributions to mortality in cities**
Contributions to $PM_{2·5}$ mortality for cities with the highest relative $PM_{2·5}$ health effects (A) and to NO_2 mortality for cities with the highest relative NO_2 health effects (B), as reported by Khomenko and colleagues.[33] In each panel, cities are listed in the order of highest effects to lowest effects on natural-cause (or non-accidental) mortality associated with air pollution, from left to right and top to bottom, as previously reported.[32] *Argenteuil and Bezons were defined as one city in our dataset.

(appendix 2 p 15). Transport contributions were highest in western and central Europe and northern Italy (appendix 2 p 15). Energy contributions were predominant in eastern Europe (appendix 2 p 16). Contributions from shipping were highest in coastal cities and, lastly, contributions from natural sources

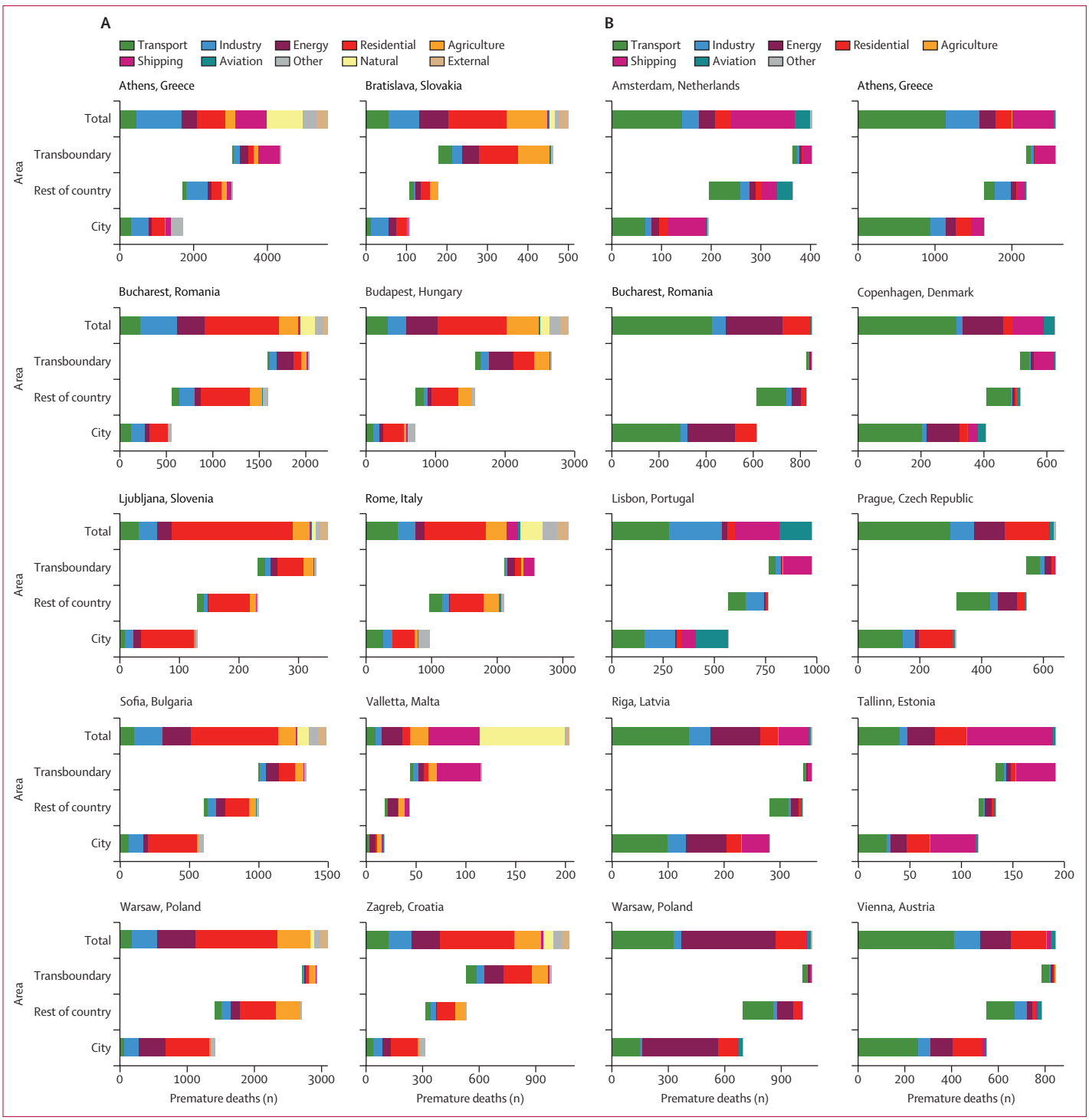

Figure 3: **Sectoral and spatial contributions to mortality in European capitals**
Contributions to $PM_{2.5}$ mortality (A) and NO_2 mortality (B) for selected European capitals.

were most substantial in southern European cities (appendix 2 pp 16–17). For NO_2, contributions to premature mortality from the transport sector were highest in western and southern Europe (appendix 2 p 18). Industry contributions showed a widespread pattern with highest contributions for cities in Romania, Greece, Spain, Poland, Slovakia, France, and Italy (appendix 2 p 18). Contributions from the energy

	Contribution to premature mortality, %		Premature deaths, point estimate (95% CI)	Deaths per 100 000 population, n	
	Mean (SD)	Median (IQR; range)		Mean (SD)	Median (IQR; range)
$PM_{2·5}$ at the city level					
Small (<60 km², n=191)	8·2% (6·9)	6·2% (3·8–10·4; 0·4–46·3)	1300 (1192–1410)	10·1 (9·1)	7·1 (3·5–14·7; 0·4–52·8)
Medium (60–300 km², n=498)	12·6% (8·0)	10·1% (6·7–16·8; 2·1–43·5)	13 436 (12 330–14 548)	15·3 (12·5)	11·1 (7·0–19·4; 1·9–78·5)
Large (>300 km², n=168)	22·3% (12·2)	21·2% (12·2–30·3; 3·6–59·1)	29 515 (27 037–31 978)	20·7 (17·0)	17·1 (9·6–24·2; 3·1–119·0)
Capitals (n=30)	29·9% (12·5)	29·9% (22·3–39·1; 5·6–50·5)	15 063 (13 825–16 319)	36·3 (20·8)	33·1 (22·6–46·8; 5·6–94·7)
$PM_{2·5}$ at the country level					
Small (<60 km², n=191)	52·1% (18·3)	52·2% (39·6–65·7; 4·5–83·6)	7726 (7062–8392)	61·6 (33·7)	54·6 (37·0–77·2; 5·3–194·0)
Medium (60–300 km², n=498)	46·8% (15·6)	47·0% (35·7–59·6; 10·4–78·0)	41 531 (38 050–45 040)	54·0 (25·8)	49·2 (38·0–65·6; 6·7–176·0)
Large (>300 km², n=168)	40·9% (15·2)	39·8% (27·9–52·5; 5·4–71·7)	38 401 (35 152–41 622)	37·2 (17·5)	37·0 (26·1–47·8; 1·5–99·8)
Capitals (n=30)	29·5% (10·3)	27·8% (23·3–37·2; 11·9–47·1)	15 820 (14 514–17 147)	36·3 (19·2)	32·5 (21·0–45·8; 11·1–86·6)
$PM_{2·5}$ at the transboundary level					
Small (<60 km², n=191)	26·9% (13·2)	23·7% (15·8–35·1; 4·8–70·6)	4026 (3682–4371)	32·6 (23·0)	26·9 (16·8–41·3; 2·5–183·0)
Medium (60–300 km², n=498)	28·8% (14·0)	25·7% (18·1–37·4; 5·4–74·5)	23 693 (21 735–25 670)	35·0 (24·2)	28·1 (19·7–43·4; 2·7–141·0)
Large (>300 km², n=168)	21·5% (11·4)	19·3% (12·5–27·0; 4·8–58·4)	19 210 (17 605–20 817)	20·9 (16·7)	16·2 (9·4–27·7; 2·1–107·0)
Capitals (n=30)	27·9% (14·4)	24·7% (17·9–35·9; 4·8–70·6)	10 240 (9412–11 085)	35·3 (23·4)	35·4 (18·0–47·1; 4·3–107·0)
NO_2 at the city level					
Small (<60 km², n=191)	21·5% (15·7)	17·7% (9·6–29·4; 2·7–78·1)	1312 (863–1763)	9·8 (7·1)	8·0 (4·5–12·5; 1·2–44·5)
Medium (60–300 km², n=498)	33·2% (16·4)	31·7% (20·7–44·6; 2·8–82·2)	14 016 (9226–18 784)	16·0 (8·5)	14·6 (9·7–20·5; 1·8–50·1)
Large (>300 km², n=168)	52·2% (19·4)	53·5% (40·3–66·9; 11·5–90·4)	28 705 (18 874–38 531)	22·1 (10·6)	20·4 (14·0–28·3; 5·5–52·6)
Capitals (n=30)	62·7% (14·7)	65·6% (56·5–71·3; 21·8–83·9)	15 810 (10 508–21 155)	35·4 (10·6)	35·0 (30·0–42·8; 9·6–52·6)
NO_2 at the country level					
Small (<60 km², n=191)	60·3% (20·0)	61·3% (46·6–76·7; 3·4–94·3)	3845 (2522–5173)	29·5 (13·5)	28·7 (19·2–38·7; 1·5–72·3)
Medium (60–300 km², n=498)	48·9% (17·9)	49·4% (37·0–62·5; 6·7–91·5)	19 054 (12 523–25 587)	24·7 (12·1)	22·9 (15·4–31·7; 1·6–68·6)
Large (>300 km², n=168)	35·9% (18·5)	32·6% (21·4–49·4; 0·6–80·9)	14 362 (9459–19 274)	15·9 (10·1)	13·6 (8·8–21·4; 0·2–43·8)
Capitals (n=30)	24·1% (10·2)	25·0% (16·6–28·1; 8·7–50·2)	5546 (3674–7436)	13·9 (7·4)	12·9 (9·2–16·8; 3·3–37·1)
NO_2 at the transboundary level					
Small (<60 km², n=191)	18·2% (14·6)	14·7% (6·2–25·9; 0·9–62·6)	1123 (742–1510)	8·8 (7·7)	6·6 (2·8–12·6; 0·3–38·9)
Medium (60–300 km², n=498)	17·8% (13·4)	14·2% (7·5–24·7; 1·2–72·1)	6323 (4182–8475)	9·0 (7·4)	7·2 (3·4–12·1; 0·3–54·1)
Large (>300 km², n=168)	11·9% (9·6)	8·7% (4·9–16·6; 0·7–51·9)	4512 (2967–6066)	5·2 (4·5)	3·5 (1·8–6·9; 0·3–24·3)
Capitals (n=30)	13·2% (10·8)	11·1% (5·4–17·2; 0·7–53·0)	1901 (1273–2536)	7·2 (5·6)	5·8 (3·2–9·9; 0·4–23·3)

Table 3: **$PM_{2·5}$ and NO_2 effect on premature mortality by spatial level and city size**

sector were predominant in eastern and northern Europe (appendix 2 p 19). Residential contributions were highest over Italy, France, and eastern Europe and, finally, shipping contributions were predominant in coastal cities (appendix 2 pp 18–20). For both pollutants, we estimated higher mean city contributions to premature mortality for cities of largest area and for European capitals (table 3). The correlations between the city size and the city contributions to mortality were r=0·521 for $PM_{2·5}$ and r=0·515 for NO_2 (appendix 2 pp 19–20). Source-specific mortality effects for European capitals are summarised in appendix 2 (pp 61–183), contributions to mortality for all individual cities for $PM_{2·5}$ are shown in appendix 3, and contributions for NO_2 for all individuals cities are shown in appendix 4.

Sensitivity analyses using the previous SHERPA version showed overall similar results, except for the city and $PM_{2·5}$ residential contributions, which were generally lower (appendix 2 pp 22–38). The inclusion of buffers for small-size cities led to increased city contributions to mortality with increased buffer size, with a higher increment for NO_2 than for $PM_{2·5}$ (appendix 2 pp 38–41). Sensitivity analyses using a unique receptor point per city resulted in similar sectoral contributions and slightly increased city contributions (appendix 2 pp 42–52). Finally, sensitivity analyses with alternative source apportionment models for $PM_{2·5}$ showed strong correlations for the residential and agricultural contributions, and weak correlations for the industrial contributions. Overall, residential and industrial contributions were lower compared with the main analysis (appendix 2 pp 52–57). For NO_2, correlations were weak for all sectors, particularly for the industrial and agricultural contributions. Overall, industrial contributions were lower than in the main analysis, and agricultural contributions were assigned to areas with null estimates (appendix 2 pp 52–57).

See Online for appendix 3

See Online for appendix 4

Discussion

We estimated the effects of source-specific reductions in ambient $PM_{2·5}$ and NO_2 concentrations on mortality for more than 800 European cities. Our results indicate that, overall, the residential and agricultural sectors are the main contributors to mortality related to $PM_{2·5}$, and transport is the main contributor to mortality related to NO_2. We estimate that the mean city contribution to its own air pollution mortality is 13·5% for $PM_{2·5}$ and 34·4% for NO_2, and is highest among the cities of largest area (22·3% for $PM_{2·5}$ and 52·2% for NO_2) and among European capitals (29·9% for $PM_{2·5}$ and 62·7% for NO_2). Most notably, our results show strong variability between cities, pointing out the need for local policies and coordinated actions that consider city-level specificities in source contributions.

Compared with previous studies based on ERI methods, our results on the relative contributions of each sector are partly in line with global assessments, which identify the residential sector as the main contributor to health burden related to $PM_{2·5}$ and the transport sector as the main contributor to NO_2.[8–10] Similarly, our sensitivity analyses using these global estimates show strong correlations for $PM_{2·5}$ residential and agricultural contributions (appendix 2 pp 52–57), supporting the robustness of our estimates and the importance of these sectors in contributing to $PM_{2·5}$ mortality. Indeed, energy consumption in the residential, commercial, and institutional sectors contributed 53% of primary $PM_{2·5}$ emissions in Europe in 2019, and the agricultural sector contributed 94% of ammonia emissions.[38]

However, despite the general agreement, $PM_{2·5}$ residential and industrial contributions estimated in the global studies were lower in comparison with our estimates, and we identified substantial differences in NO_2 sectoral contributions compared with the global estimates (appendix 2 pp 52–57). Similarly, our results had considerable differences to a previous study with 96 global cities, which estimated the highest contributions to $PM_{2·5}$ to be from the industry and energy sectors and overall lower city contributions (appendix 2 pp 58–60).[12] The higher residential contributions in our study could be explained by the substitution of residential emissions with those from the REF2 bottom-up inventory, which include the condensable particulate matter fraction from residential wood combustion.[23,26] This adjustment implies a 5-times increase in emission factors, resulting in increased emissions by a factor of 2–3.[23,26] As for the industrial and NO_2 sectoral contributions, the observed differences could be partly due to the coarser resolution used in the global studies (ie, 0·5×0·5° compared with 0·1×0·05°). Given that industrial emissions often represent a localised pollution source, areas of strong industrial emissions are best captured at a fine grid scale, whereas emissions would appear evenly distributed at a course resolution. Similarly, nitrogen oxide emissions generally have a shorter lifetime than particulates and tend to influence air quality near the emission source.[39] Therefore, NO_2 contributions and spatial patterns are better described at a finer scale than at a coarse scale, more accurately representing local emissions, atmospheric chemistry, and dispersion. Additionally, the observed differences could be due to differences in emission inventories. Our analyses were based on the Copernicus Atmosphere Monitoring Service regional inventory,[25] whereas the global assessments used the Community Emissions Data Systems inventory.[40,41] The Copernicus Atmosphere Monitoring Service regional inventory is based on nationally reported official data and redistributes emissions to a 0·1×0·05° grid using detailed local proxies.[25] Instead, the Community Emissions Data Systems inventory combines data from other emission inventories and activity drivers to estimate default emissions, which are subsequently scaled to country-level inventories and then redistributed to a 0·5×0·5° grid, mainly using gridded emissions from the Emissions Database for Global Atmospheric Research,[40,41] which potentially leads to differing emission estimates. Additional sources of variability probably include differences in city definitions,[42] which are relevant to evaluate city contributions, and differing modelling assumptions to derive changes in pollutant concentrations due to changes in emissions.

The main strengths of this study include the large number of analysed cities; the robust source apportionment methodology; the estimation of the mortality burden associated to each pollutant source; the improved resolution of the SHERPA model (ie, from 0·1×0·1° to 0·1×0·05°); and the overall harmonised assessment and direct applicability for policy making.

Nonetheless, several limitations need to be discussed. This study is based on a single meteorological year, has a spatial resolution that might not be sufficient for small-size cities, and focuses on annual source contributions, whereas a higher temporal resolution could help to identify important information for pollution control. Another limitation is the use of SRRs instead of a full CTM simulation, which could lead to bias in results. Overall, the validation of SRRs indicates a generally low bias compared with the full CTM run, supporting the validity of our results.[22,43] Additionally, the Copernicus Atmosphere Monitoring Service regional inventory might not account for all local specificities in emissions, which would be better described through city-level emission inventories. Moreover, we assumed a linear dose–response relationship between pollutant concentrations and mortality. However, non-linearity is generally more prominent at high air pollution concentrations than at the exposure levels seen in Europe, and the meta-analyses used in this study from which we obtained risk estimates indicated that ERFs were either linear or supralinear for $PM_{2·5}$,[36] and provided little evidence to reject linearity for NO_2,[37] indicating that it is plausible to assume linearity,

particularly at the exposure levels observed in this study. Furthermore, we evaluated source-specific reductions in pollutants independently; thus, we never reached zero pollutant concentrations in our calculations. Nevertheless, we added the mortality burden from all sources to calculate the relative source contributions, a number that would reflect reductions to null concentrations, which might not be realistic and should be interpreted with caution. Despite this limitation, current research indicates no evidence of an exposure threshold.[44,45] Thus, our assumptions seem reasonable and in line with these previous findings. Furthermore, we used residential exposure to air pollutants, which does not reflect individual differences in exposure based on travel patterns. Despite this limitation, the ERFs used were also based on residential exposure, thus, our approach is adequate to derive mortality effects. An additional limitation is the exclusion of O_3 from the analysis, a pollutant for which previous studies have estimated significant health effects.[20] However, given that O_3 shows strong seasonal patterns and SHERPA is currently implemented only at an annual temporal resolution, the model was not considered adequate to evaluate O_3. As for the health effects, the mortality estimates presented here could be slightly overestimated due to the decrease in air pollution in Europe since 2015.[6] Our analyses were done for 2015 because it was the most recent year for which all data sources were available for all cities. However, variations in source contributions and mortality effects for years since 2015 are plausible due to differences in emissions, meteorology, population counts, and mortality. Despite this possibility, we consider the 2015 data to be representative of meteorological conditions and long-term exposure to pollutants. Moreover, for the year 2015, high-quality emission and model evaluation data were available, which is important for source attribution. All in all, it will be necessary to repeat this kind of analysis with data from more recent years to make comparisons and study temporal changes. Finally, we assumed that the health effects from the distinct particulate matter components are equal, but these could vary on the basis of their differential toxicity. Research suggests that carbonaceous particles from combustion sources could be more hazardous to health than other components due to their high oxidative potential.[10,46] However, to date, no evidence exists from meta-analyses on which source sectors might have the most detrimental effect on health. A study indicated the strongest associations with mortality for traffic and residual oil combustion components.[47] Further research is needed in this direction to provide accurate evidence on which pollution sectors should be targeted to provide the greatest benefits for health. Finally, we could not disaggregate our results by sex, gender, or ethnicity due to absence of available data and meta-analysed ERFs stratified by these factors. However, accounting for gender and socioeconomic factors is needed to provide targeted evidence for policy actions.

Our results have direct implications for policy. Given the strong variability in each city's contribution to air pollution and mortality, local city-level air quality plans are needed to account for and target these specificities. Our results suggest that city contributions to its own air pollution and mortality are more prominent for large and capital cities than for small cities (table 3). Although city contributions might not seem high, cities are not isolated entities and city emissions, particularly for $PM_{2·5}$, are likely to get dispersed from their origins, influencing neighbouring cities and regional pollutant concentrations; thus, policies to reduce city emissions are needed. National and international emissions are also a relevant source of pollutants, particularly for $PM_{2·5}$ in small and medium cities (table 3). Our findings emphasise the need for coordinated actions at all three spatial scales, considering the specificities in contributions of each local context. A larger spectrum of sources contributed to $PM_{2·5}$ than to NO_2 mortality, which had the highest contributions from transport at the local level. Local actions can reduce NO_2 concentrations and mortality and include various measures, such as the implementation of low emission zones; changes in urban design that promote pedestrian and cycling areas, urban greening, and proximity to services, employment, and recreation; incentives for public and active transport; and speed limits and overall reductions in motorised traffic.[48,49] Although these actions might also help to reduce $PM_{2·5}$ pollution and mortality, our findings emphasise the need for coordinated actions across multiple sectors and scales to effectively reduce the concentration of this pollutant. For $PM_{2·5}$, we estimated the highest mortality effects from the residential and agricultural sectors. These effects could be addressed through fuel regulations, stove replacement schemes, fuel burn bans, improved building insulation, a shift towards clean and renewable energy sources, and the implementation of cost-effective techniques for manure management and fertiliser use to improve nitrogen use efficiency in agriculture.[48] Additional measures that target the transport, industry, energy, and shipping sectors can help to reduce pollution and include emission controls, industrial materials, fuels and processes optimisation, and a complete phasing out of coal and fossil fuel burning.[48] Most notably, all of these measures can also bring in climate co-benefits, helping to achieve the net-zero goals.[49,50] Thus, although our findings support the development of local air quality plans, they also reflect the need for coordinated actions across sectors at several spatial scales. We recommend that our estimates for each city are considered as a first step in the development of local air quality plans and, subsequently, are used to explore across which sectors and scales integrated actions are needed.

Contributors

MN and SK conceptualised the study. EP, PT, and BB developed the SHERPA model. MC assisted in data collection. SK did the data analysis.

SK, EP, PT, BB, TI, EPB, HK, and MN contributed to data interpretation. HK and CT provided guidance on source apportionment methods. TI, EPB, NM, and CT provided input on health impact assessment methods. KdH and GH contributed to the development of modelled baseline air pollution estimates. SC and JL developed and provided data from the Earth System Atmospheric Chemistry Model. SK wrote the manuscript. SK, EP, PT, BB, MC, TI, and EPB accessed and verified the data. All authors reviewed the manuscript and provided feedback on the study design, data analysis, and interpretation of results. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Declaration of interests

We declare no competing interests.

Data sharing

All the data in this study are routinely collected and contain no information about specific people. The Python code to run the SHERPA model is freely available at GitHub: https://github.com/enricopisoni/SHERPA-simulation. Our data, including the modified SHERPA Python code that was used in this Article, the R code developed for the health impact assessment analysis, and all necessary datasets, are available on request to the corresponding author, subject to the agreement of the research steering group.

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