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Mortality from Ship Emissions: A Global Assessment

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Epidemiological studies consistently link ambient concentrations of particulate matter (PM) to negative health impacts, including asthma, heart attacks, hospital admissions, and premature mortality. We model ambient PM concentrations from oceangoing ships using two geospatial emissions inventories and two global aerosol models. We estimate global and regional mortalities by applying ambient PM increases due to ships to cardiopulmonary and lung cancer concentration-risk functions and population models. Our results indicate that shipping-related PM emissions are responsible for approximately 60,000 cardiopulmonary and lung cancer deaths annually, with most deaths occurring near coastlines in Europe, East Asia, and South Asia. Under current regulation and with the expected growth in shipping activity, we estimate that annual mortalities could increase by 40% by 2012.

Introduction

The marine transport sector contributes significantly to air pollution, particularly in coastal areas (*1–8*). Annually, oceangoing ships are estimated to emit 1.2–1.6 million metric tons (Tg) of particulate matter (PM) with aerodynamic diameters of 10 μm or less (PM_{10}), 4.7–6.5 Tg of sulfur oxides (SO_x as S), and 5–6.9 Tg of nitrogen oxides (NO_x as N) (*9–12*). Recent studies have estimated around 15% of global NO_x and 5–8% of global SO_x emissions are attributable to oceangoing ships (*10, 11*). Given nearly 70% of ship emissions occur within 400 km of land (*2, 11, 12*), ships have the potential to contribute significant pollution in coastal communities—especially for SO_x. For instance, Capaldo et al. (*1*) estimate that ship emissions contribute between 5 and 20% of non-sea-salt sulfate concentrations and 5–30% of SO_2 concentrations in coastal regions.

Numerous studies in recent years have consistently linked air pollution to negative health effects for exposed populations (*13, 14*). Ambient concentrations of PM have been associated with a wide range of health impacts including asthma, heart attacks, and hospital admissions. An important PM-related health effect is premature mortality; in particular, increases in concentrations of PM with aerodynamic diameters of 2.5 μm or less ($PM_{2.5}$) have been closely associated with increases in cardiopulmonary and lung cancer mortalities in exposed populations (*15*). Cohen et al. estimated approximately 0.8 million deaths per year worldwide from outdoor urban $PM_{2.5}$ air pollution, 1.2% of global premature mortalities each year (*16*).

Emissions from international ships are increasingly a focus for proposed regulation in local, national, and international arenas (*8, 17, 18*). Yet, in many ways regulatory deliberations have not been fully informed, as the extent of shipping emissions health impacts has been unknown. Previous assessments of regional shipping-related health impacts focused on European or Western United States regions, and ignore long-range and hemispheric pollutant transport (*8, 19*). This undercounts international shipping impacts within local and regional jurisdictions, and does not properly inform international policy decision making.

Assessing Mortality from Atmospheric Modeling of Ship Emissions

Our approach is similar to that of other studies (*15, 16, 20, 21*): (1) determine pollutant emissions from ships; (2) apply atmospheric transportation and chemistry models to estimate the increased concentrations due to ships; (3) estimate increased risk to exposed population due to these additional concentrations; and (4) calculate additional mortalities due to that increased risk.

We use two different geospatial ship data sets to help us construct geospatial emission inventories: the International Comprehensive Ocean-Atmosphere Data Set (ICOADS) by Corbett et al. (*10*), and the Automated Mutual-assistance Vessel Rescue system (AMVER) by Endresen et al. (*12*). These two data sets combine detailed information about vessel characteristics with vessel traffic densities to determine emissions geospatially. However, each data set allocates ship-traffic intensities differently. For example, while all oceangoing commercial ship types are included in these data sets, ICOADS oversamples container ship traffic and refrigerated cargo ship (i.e., reefer) traffic, and AMVER oversamples bulk carrier and tanker traffic. Ship inventory differences affect regional atmospheric pollution concentrations, potentially influencing health effects estimates. Both inventories provide emissions data on a monthly time-resolution; for atmospheric modeling, we assume emissions occur uniformly throughout each month.

We generated three emissions inventory data sets for comparison. First, we used monthly resolved ICOADS 2002 emissions estimates of NO_x, SO_x, black carbon (BC), and particulate organic matter (POM) at a $0.1° \times 0.1°$ global grid resolution (*Inventory A*). Second, we used AMVER 2001 emissions estimates of NO_x, SO_x, BC, and POM at a $1° \times 1°$ global grid resolution from Eyring et al. (*Inventory B*) (*11*). Because of recent attention on the growth in commercial shipping activity, we also produced ICOADS-based ship inventories for 2012 (*Inventory C*) forecast using a uniform global average growth rate of 4.1% (*3, 10*). Both inventories represent shipping routes for most cargo shipping, and some oceangoing passenger shipping activity, but neither adequately represents typical fishing fleets and passenger ferry service; therefore, we adjust global inventories to represent only cargo and passenger ships. Table 1 shows total annual shipping-attributable emissions for each inventory.

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TABLE 1. Annual Emission Totals of Particulate Matter and Trace Gases from Shipping in Tg/yr for the Three Different Inventories Considered in This Study

	Inventory A for 2002 (Corbett et al., 2007 (*4*))	Inventory B for 2001 (Eyring et al., 2005 (*11*))	Inventory C for 2012 (this study)
spatial ship traffic proxy	ICOADS	AMVER	ICOADS
fuel consumption in million tonnes	200 (cargo and passengers only)	280 (world fleet including auxiliary engines)	299 (cargo and passengers only)
NO_x	16.4	21.3	24.5
SO_x	9.2	11.7	13.7
primary SO_4	0.35	0.77	0.50
CO	1.08	1.28	1.61
BC	0.07	0.05	0.10
POM	0.71	0.13	1.06

Global-scale models may model differently the $PM_{2.5}$ concentrations used in health-effects estimates. We compare increased ambient $PM_{2.5}$ concentrations from marine shipping using two atmospheric models. The first, GEOS-Chem (*22*), is a global 3-D atmospheric composition model driven by assimilated meteorological observations from the Goddard Earth Observing System (GEOS). GEOS-Chem output provided us with ambient dry concentrations of BC, POM, and sulfates from ocean-going ships separately from total $PM_{2.5}$ attributed to all other sources. The second model, ECHAM5/MESSy1-MADE (referred to as E5/M1-MADE), is an aerosol microphysics module (MADE) coupled to a general circulation model (ECHAM5), within the framework of the Modular Earth Submodel System MESSy (*23*). Along with global $PM_{2.5}$ concentrations attributed to nonship sources, the E5/M1-MADE model provided ambient concentrations of BC, POM, and sulfates for direct comparison with GEOS-Chem results; separately the model produced concentrations of total $PM_{2.5}$ constituents related to shipping (including nitrates and ammonium ions). The Supporting Information includes additional detail for both models.

Comparing results of each model with and without ship inventories of $PM_{2.5}$ components, we quantify ambient concentrations of $PM_{2.5}$ due to marine shipping. Worldwide concerns about SO_x emissions from ships are motivating the replacement of marine residual oil (RO) with cleaner fuels, such as marine gas oil (MGO) and marine diesel oil (MDO), which will directly impact BC, POM, and sulfates attributed to ships; therefore, we model total PM and the subset of PM from ships most commonly associated with current marine fuels. We defined the following cases to investigate robustness of mortality estimates under different inventory and modeling choices:

Case 1 compares $PM_{2.5}$ concentrations with and without ship emissions from model simulations with Inventory A. This was done three times: Case 1a examines BC, POM, and sulfates only, using the GEOS-Chem model; Case 1b uses the E5/M1-MADE model to examine BC, POM, and sulfates for direct comparison with GEOS-CHEM; Case 1c uses the E5/M1-MADE model to examine total PM from ships.

Case 2 compares $PM_{2.5}$ concentrations with and without ship emissions from model simulations with Inventory B in the E5/M1-MADE model. This was done twice: Case 2a for BC, POM, and sulfates only; and Case 2b for all PM constituents.

Case 3 compares $PM_{2.5}$ concentrations with and without ship emissions from model simulations with Inventory C representing estimated 2012 emissions from increased shipping activity. The case examines BC, POM, and sulfates only, using the GEOS-Chem model. Note that Case 3 estimates ignore potential emissions growth (or reduction) from other sources between 2002 to 2012; however, we use Case 3 only to estimate the additional mortality from oceangoing trade growth, not to estimate total change in mortality due to all sources of $PM_{2.5}$.

Figure 1 depicts an annual aggregation of one of our two midrange estimated contributions of $PM_{2.5}$ concentrations due to shipping in 2002 (Case 2a). Concentration increases from ships range up to 2 μg per cubic meter ($\mu g/m^3$) and occur primarily over oceans and coastal regions.

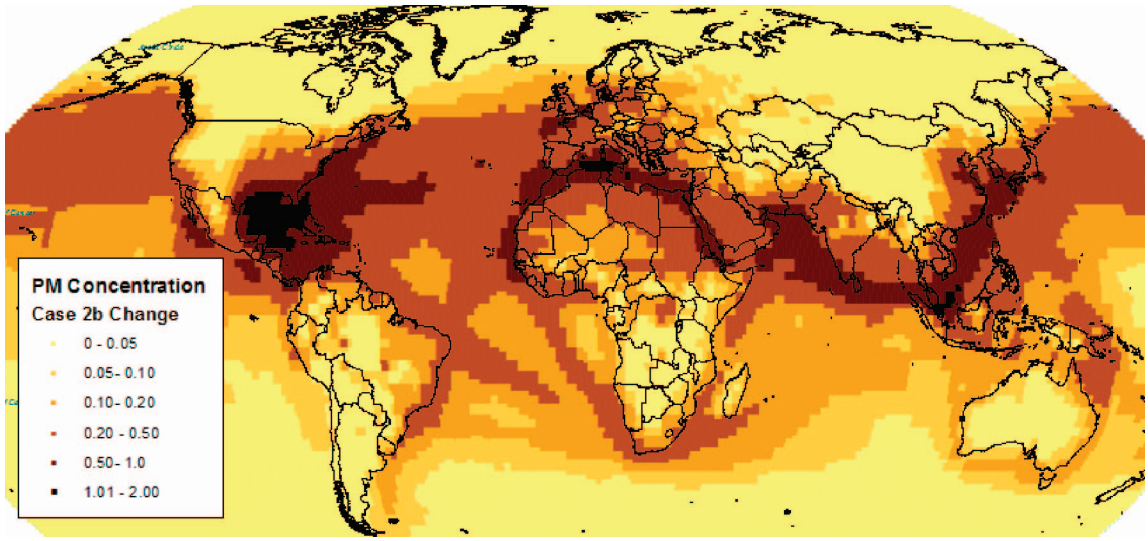

FIGURE 1. Annual average contribution of shipping to $PM_{2.5}$ concentrations for Case 2b (in $\mu g/m^3$)

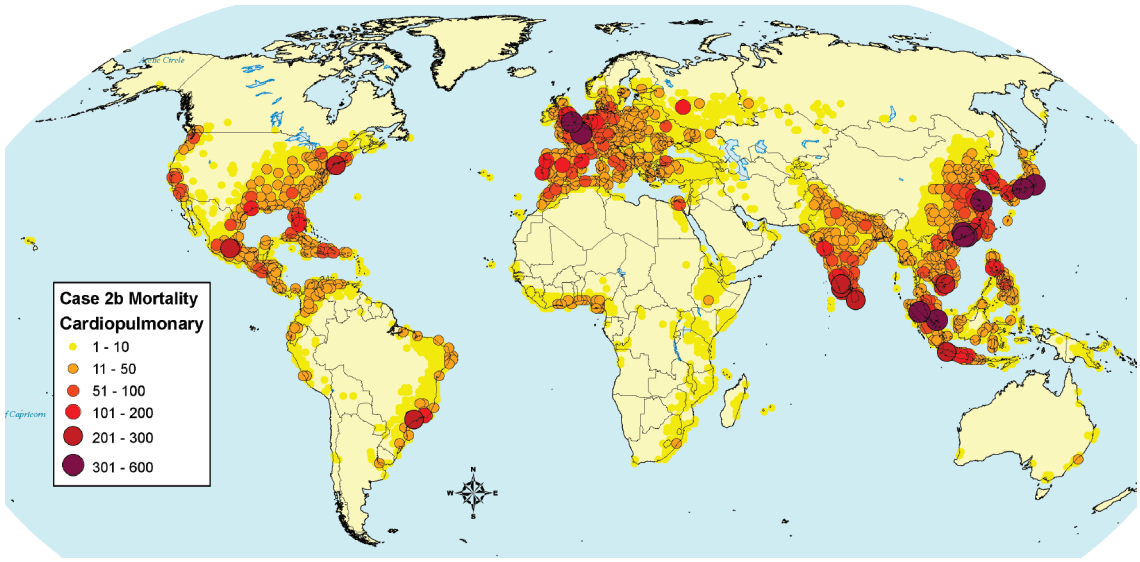

FIGURE 2. Cardiopulmonary mortality attributable to ship $PM_{2.5}$ emissions worldwide, Case 2b.

Annual $PM_{2.5}$ concentrations were used to assess annual mortality due to long-term PM exposure, consistent with Pope et al. (*15*). This requires an estimate of exposed population. We used 2005 global population estimates (obtained in a 1° × 1° format) from the Socioeconomic Data and Applications Center (SEDAC) at Columbia University (*24*). To conform to the population data resolution, we interpolated to a 1° × 1° resolution the atmospheric concentration output for each of our cases (provided at 2° latitude × 2.5° longitude in GEOS-Chem and at 2.8° × 2.8° longitude by latitude in E5/M1-MADE). We note that for most areas (with population growth) the use of 2005 population estimates will slightly overestimate our 2002 mortalities and slightly underestimate our 2012 mortalities.

Our mortality estimates are based on cardiopulmonary and lung cancer causes of death for adults over 30 years of age. Therefore, we applied U.S. Census Bureau International Database estimates to derive, by continent, the percentage of each grid cell's population over 30 years old (*25*).

We also required background incidence rates of mortality due to the health effects under study. Incidence rates were estimated using World Health Organization (WHO) 2002 data aggregated to the WHO region level (*26*). WHO cause of death by age estimates were used to derive incidence rates for the 30–99 age group for each of the six WHO regions. Similar to another assessment of global mortality from outdoor pollution, lung, tracheal, and bronchial cancers were considered "lung cancers" for our purposes (*20*); these cancers are aggregated and nondistinguishable in WHO burden of disease estimates. United States cardiopulmonary incidence values obtained from the U.S. EPA (*27*) were used for North America.

In calculating mortality effects we used C-R functions derived from an American Cancer Society cohort study that examined the relationship between $PM_{2.5}$ and lung cancer and cardiopulmonary mortality in the United States (*15*). We apply these U.S.-derived C-R functions to our entire spatial data set, recognizing that transferring U.S.-derived functions to the global population introduces uncertainty to the analysis, because socioeconomic factors have been associated with effects of PM exposure on mortality and relative risks (*28, 29*). However, other researchers have demonstrated that the relationship between short-term PM exposure and mortality is relatively consistent across several countries and continents (*21, 30, 31*). We employ a log-linear exposure function using Pope (*15*) to estimate long-term mortality effects of $PM_{2.5}$, as recommended and described by Ostro (*21*). These equations reduce to an effects equation as follows:

$$E = \left[1 - \frac{(X_0 + 1)}{(X_1 + 1)}\right]^{\beta} \cdot B \cdot P \qquad (1)$$

where E represents total effects (deaths/year); X_1 is the pollutant concentration for the case under study in $\mu g/m^3$; X_0 is the pollutant background concentration in $\mu g/m^3$; β is an estimated parameter based on the health effect under study; B represents the general incidence of the given health effect (e.g., cardiopulmonary deaths/person/year), and P represents the relevant exposed population (detailed equations are derived in the Supporting Information).

Ship PM-Induced Global and Regional Premature Mortality

Exposure to shipping-related $PM_{2.5}$ emissions in 2002 resulted in 19,000 (Case 1a) to 64,000 (Case 1c) cardiopulmonary and lung cancer mortalities globally, depending on the emission inventory and on the particles considered. Approximately 92% of the estimated premature mortalities are from cardiopulmonary illnesses. Mortalities increase by approximately 40% in 2012 due to trade-driven growth in shipping emissions.

Figure 2 reveals that mortalities are concentrated in distinct regions. We estimate regional impacts separately in Table 2 for North America (NA); Europe/Mediterranean (EUM); East Asia (EA), including China and Japan; South Asia (SA), including India and Indonesia; and Eastern South America (ESA). Regional burden of mortality varies, with the greatest effects seen in the EUM (20–40% of global mortalities), EA (20–30%), and SA (15–30%) regions.

Figures 2, 3, and 4 depict our cardiopulmonary mortality estimates by grid cell for Case 2a for the entire globe, the EUM region, and the EA/SA regions, respectively. Mortality estimates of less than 1 per grid cell are excluded to facilitate readability.

As expected, regions with the greatest mortality effects are also those where shipping-related $PM_{2.5}$ concentrations

TABLE 2. Annual Cardiopulmonary and Lung Cancer Mortality Attributable to Ship $PM_{2.5}$ Emissions by Region and by Case (Best Estimate from C-R function[a] (95% confidence interval[b]))

Region	Case 1a Inventory A Model: GEOS-Chem PM: BC, POM, SO_4	Case 1b Inventory A Model: E5/M1-MADE PM: BC, POM, SO_4	Case 1c Inventory A Model: E5/M1-MADE PM: All	Case 2a Inventory B Model: E5/M1-MADE PM: BC, POM, SO_4	Case 2b Inventory B Model: E5/M1-MADE PM: All	Case 3 (2012 Forecast) Inventory C Model: GEOS-Chem PM: BC, POM, SO_4
North America (NA) Region						
cardiopulmonary	1,860 (680–3,050)	2,820 (1,020 – 4,610)	4,590 (1,660 – 7,510)	>5,470 (1,980 – 8,950)	7,910 (2,870 – 12,940)	2,770 (1,010 – 4,540)
lung cancer	210 (80 – 350)	320 (120 – 520)	520 (190 – 850)	620 (230 – 1,020)	900 (330 – 1,470)	320 (120 – 520)
NA Total	**2,070 (760 – 3,400)**	**3,140 (1,140 – 5,130)**	**5,110 (1,850 – 8,360)**	**6,090 (2,210 – 9,970)**	**8,810 (3,200 – 14,410)**	**3,090 (1,130 – 5,060)**
Europe/Mediterranean (EUM) Region						
cardiopulmonary	6,770 (2,450 – 11,070)	11,830 (4,290 – 19,350)	24,350 (8,840 – 39,810)	7,250 (2,630 – 11,860)	15,100 (5,480 – 24,690)	8,990 (3,260 – 14,700)
lung cancer	670 (250 – 1,090)	1,100 (410 – 1,800)	2,360 (870 – 3,840)	650 (240 – 1,060)	1,430 (530 – 2,320)	880 (330 – 1,440)
EUM Total	**7,440 (2,700 – 12,160)**	**12,930 (4,700 – 21,150)**	**26,710 (9,710 – 43,650)**	**7,900 (2,870 – 12,920)**	**16,530 (6,010 – 27,010)**	**9,870 (3,590 – 16,140)**
East Asia (EA) Region						
cardiopulmonary	3,490 (1,270 – 5,710)	11,970 (4,340 – 19,590)	17,920 (6,500 – 29,300)	9,640 (3,500 – 15,780)	13,800 (5,010 – 22,570)	5,170 (1,880 – 8,460)
lung cancer	370 (140 – 610)	1,300 (480 – 2,110)	1,950 (720 – 3,170)	1,030 (380 – 1,680)	1,480 (550 – 2,410)	550 (200 – 900)
EA Total	**3,860 (1,410 – 6,320)**	**13,270 (4,820 – 21,700)**	**19,870 (7,220 – 32,470)**	**10,670 (3,880 – 17,460)**	**15,280 (5,560 – 24,980)**	**5,720 (2,080 – 9,360)**
South Asia (SA) Region						
cardiopulmonary	4,050 (1,470 – 6,630)	7,250 (2,630 – 11,870)	9,440 (3,420 – 15,450)	11,240 (4,080 – 18,390)	15,460 (5,610 – 25,260)	6,090 (2,210 – 9,970)
lung cancer	230 (90 – 380)	390 (150 – 640)	510 (190 – 830)	600 (220 – 970)	820 (300 – 1,340)	350 (130 – 570)
SA Total	**4,280 (1,560 – 7,010)**	**7,640 (2,780 – 12,510)**	**9,950 (3,610 – 16,280)**	**11,840 (4,300 – 19,360)**	**16,280 (5,910 – 26,600)**	**6,440 (2,340 – 10,540)**
East South America (ESA) Region						
cardiopulmonary	380 (140 – 620)	520 (190 – 850)	690 (250 – 1,130)	1,120 (410 – 1,840)	1,540 (560 – 2,520)	570 (210 – 930)
lung cancer	50 (20 – 90)	70 (30 – 120)	100 (40 – 160)	160 (60 – 260)	220 (80 – 350)	80 (30 – 130)
ESA Total	**430 (160 – 710)**	**590 (220 – 970)**	**790 (290 – 1,290)**	**1,280 (470 – 2,100)**	**1,760 (640 – 2,870)**	**650 (240 – 1,060)**
Global						
cardiopulmonary	17,340 (6,290 – 28,390)	35,610 (12,910 – 58,260)	58,640 (21,270 – 95,900)	36,970 (13,410 – 60,490)	56,790 (20,600 – 92,870)	24,780 (8,980 – 40,540)
lung cancer	1,580 (580 – 2,570)	3,260 (1,200 – 5,310)	5,540 (2,050 – 9,020	3,220 (1,190 – 5,240)	5,050 (1,870 – 8230)	2,240 (830 – 3,650)
Global Total	**18,920** (6,870 – 30,960)	**38,870** (14,110 – 63,570)	**64,180** (23,320 – 104,920)	**40,190** (14,600 – 65,730)	**61,840** (22,470 – 101,100)	**27,020** (9,810 – 44,190)

[a] Values are rounded to the nearest 10. [b] Confidence interval range is based on uncertainty in the concentration–response function coefficients.

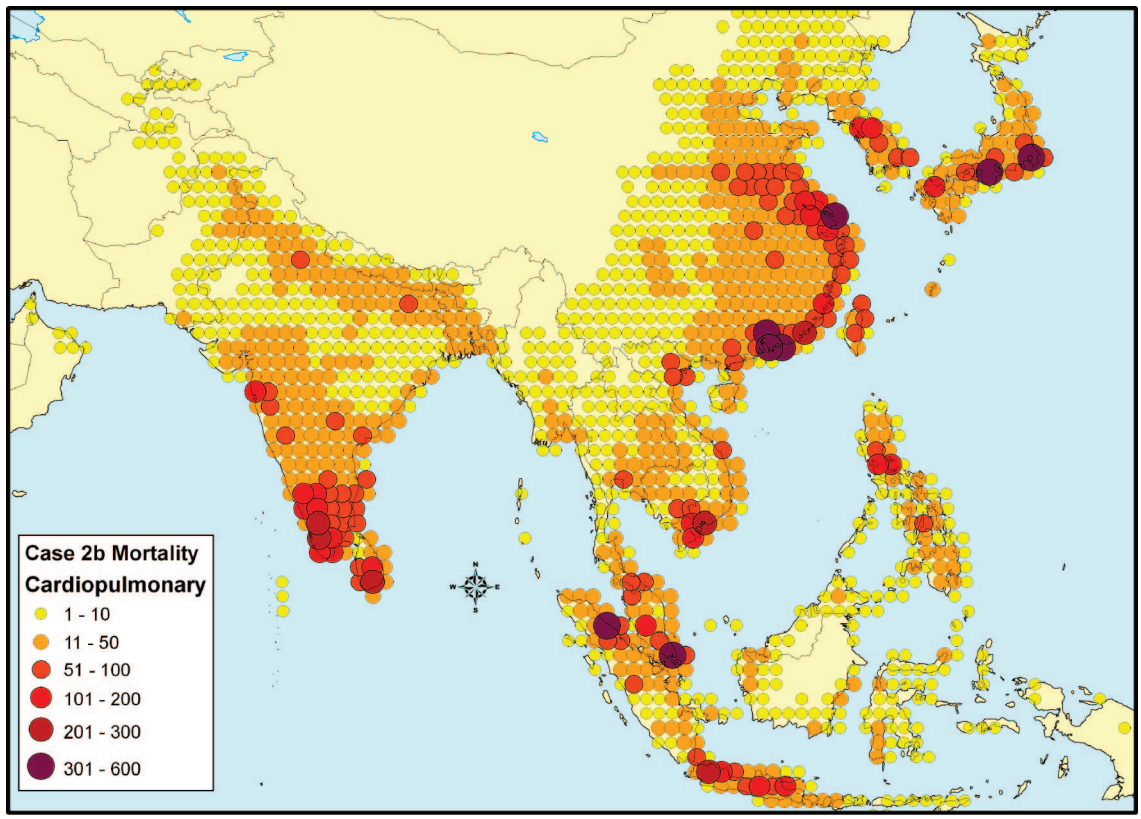

FIGURE 3. Case 2b annual cardiopulmonary mortality attributable to ship $PM_{2.5}$ emissions for Asia.

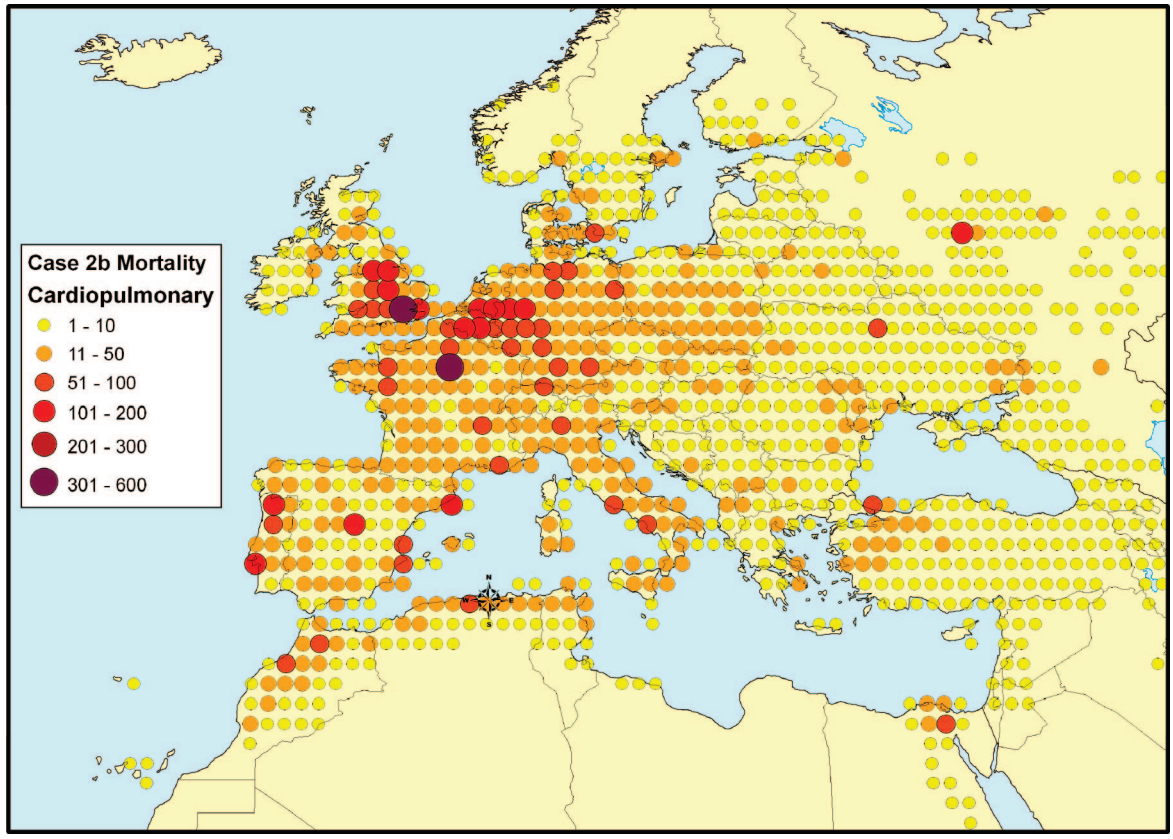

FIGURE 4. Case 2b annual cardiopulmonary mortality attributable to ship $PM_{2.5}$ emissions for Europe/Mediterranean.

are high (compare Figures 1 and 2)—near coastal regions, major waterways, and in highly populated areas. For Case 2a we estimate annual cardiopulmonary mortalities from shipping reaching densities greater than 300 per grid cell in

regions of Asia, and between 100 and 200 in the EUM region, as shown in Figures 3 and 4; coastal health-impact densities are thousands of times greater than those seen in inland regions.

Multiscale Cross-Comparisons

We compare our findings with other studies of $PM_{2.5}$ related mortality that employed alternative modeling or inventories to estimate $PM_{2.5}$ concentrations and health effects on three scales: global, national/continental, and state/regional.

Concentration–response functions are used to estimate global mortality for $PM_{2.5}$ from anthropogenic sources including shipping. These are compared to an analysis of global mortality associated with long-term exposure to $PM_{2.5}$ pollution (*16, 20*). Cohen et al. estimated that approximately 712,000 cardiopulmonary deaths are attributable to urban outdoor $PM_{2.5}$ pollution annually. With adjusting assumptions, our Case 1a estimate of 737,000 is within 4% of Cohen's (*20*) findings, and our Case 2b estimate is within 25% (see table in Supporting Information).

We evaluate potential bias of using WHO region-level incidence rates and continent-level age demographic estimates in predicting mortalities at the national scale (*24–26*)]. We compare Case 1a mortality results over the United States with mortality estimates from a similar analysis using the U.S. Environmental Protection Agency's Benefit Mapping and Analysis Program (BenMAP). BenMAP is a geographic information systems program which combines U.S. Census-level population and incidence data at county-level resolution with user-supplied air quality data to estimate heath effects. We input our $1° \times 1°$ $PM_{2.5}$ concentration data in BenMAP for the United States, and applied the C-R functions within BenMAP. We obtain Case 1a mortality estimates within 6% of BenMAP estimates, as detailed in the Supporting Information. The close agreement indicates that our population demographics and incidence rate approximations produce suitably accurate results when examining large regions, recognizing that our confidence in this statement is based on a U.S.-based analysis.

Direct comparison of our mortality estimates with recent work estimating PM health effects in Europe by Cofala et al. (*8*) is not possible because that study used an approach that estimates loss of life expectancy in months rather than total number of premature deaths. However, our patterns of health impacts for Europe among our cases appear consistent with patterns reported for their health-effects analysis (see Figure 6.1 of Cofala et al.).

Lastly, we compare our California global grid results for Case 1a and Case 2c with results from a report by the California Air Resources Board (*18*). As described in the Supporting Information, our Case 1a estimate is about 186% of the ARB estimate, and our Case 2b estimate is about 242% of the ARB estimate. In addition to differences in population and incidence at local scale, reasons to expect larger California mortality estimates in our assessment include the following. First, ARB excluded sulfates from its source-specific analyses. We include sulfates in our $PM_{2.5}$ concentrations, which on average comprise 24% of ambient PM concentrations; ARB includes nitrates, which on average may comprise some 13% of ambient PM concentrations (*32*). Second, ARB only included $PM_{2.5}$ emissions from ocean-going ships within 24 nautical miles from shore in its analysis; all other emissions were allocated to the outer continental shelf air basin (*19*). ARB also assumed that between 10% and 25% of ship emissions reached populated areas. In contrast, our modeling directly estimates land-exposure from worldwide ocean-going ship inventories, considering atmospheric transport of ship emissions to California from unbounded distances as attributed by atmospheric chemical transport functions in GEOS-Chem and E5/M1-MADE. Third, our "California" case is made up of $1° \times 1°$ grid cells that overlap small parts of Nevada, Utah, and Mexico and could lead to slightly higher estimates than a strict California-only comparison. On the other hand, ARB used smaller (more resolved) grid cells; all else equal, we would have expected this to yield larger not smaller health impacts in the CARB report because CARB would more accurately capture near-source population density.

Discussion

Our results indicate that shipping-related PM emissions from marine shipping contribute approximately 60,000 deaths annually at a global scale, with impacts concentrated in coastal regions on major trade routes. Most mortality effects are seen in Asia and Europe where high populations and high shipping-related PM concentrations coincide. Based on previous estimates of global $PM_{2.5}$-related mortalities (*16*), our estimates indicate that 3% to 8% of these mortalities are attributable to marine shipping. We identify three categories of uncertainty, ranked by their importance to estimates in this work: (i) ship inventory and PM constituent uncertainties most influence our best estimates across all Cases; (ii) the 95% confidence intervals on the health effects C-R functions represent significant uncertainty (capturing toxicity and response effects) that similarly affects each case; (iii) atmospheric modeling uncertainties vary where emissions offshore expose coastal and inland populations. Uncertainties are discussed in the Supporting Information; results may be more uncertain at local scales, given the lack of detailed localized data pertaining to incidence, demographics, $PM_{2.5}$ concentrations, and other factors.

The absence of localized C-R functions and incidence rates prevents precise quantification of all anticipated PM-related health effects, such as asthma and hospital admissions, etc. Though we only examine cardiopulmonary and lung cancer mortalities, we expect that regions where ships contribute most to mortality effects (concentrated population areas with high shipping-related PM levels) will also suffer other related health impacts. We anticipate future work to investigate variation and uncertainty in these inputs further. Higher resolved atmospheric models could provide more accurate or precise results on a regional level by targeting regions of interest where better localized data for ship emissions, incidence rates, and population demographics are available.

Our work demonstrates that mortality and health benefits in multiple regions globally could be realized from policy action to mitigate ship emissions of primary $PM_{2.5}$ formed during engine combustion and secondary $PM_{2.5}$ aerosols formed from gaseous exhaust pollutants. These results support regional assessments of health impacts from ship $PM_{2.5}$ emissions, and identify other regions where similar impacts may be expected. Current policy discussions aimed at reducing ship emissions are focused on two concerns: (i) the geospatial aspects of policy implementation and compliance (e.g., uniform global standards versus requirements for designated control areas); and (ii) the benefits and costs of various emission-reduction strategies (e.g., fuel switching versus aftertreatment technologies or operational changes). Our work quantifies the baseline estimates of mortality due to ship emissions from which future work would estimate mitigation benefits.

Acknowledgments

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Supporting Information Available

Description of atmospheric aerosol model parameters, calculations for cardiopulmonary mortality estimates, discussion of uncertainty in our analysis, and additional discussion of our results. This material is available free of charge via the Internet at http://pubs.acs.org.

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