# Supporting Material for Mortality from Ship Emissions: A Global Assessment

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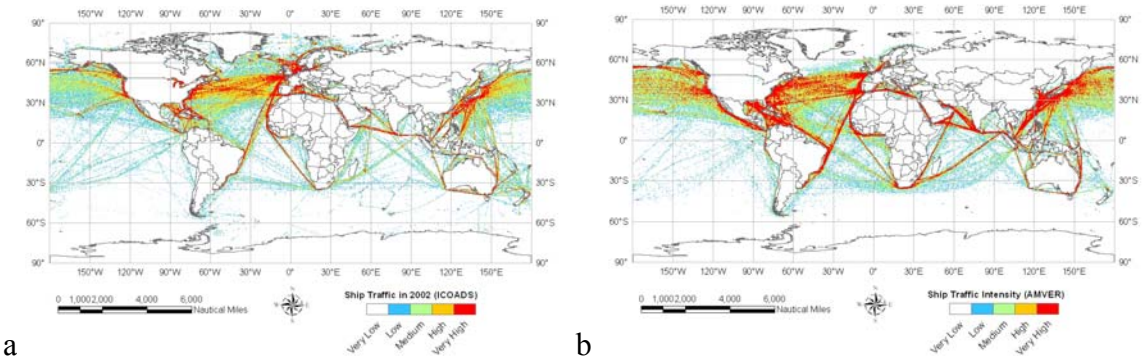
We present supporting material describing the atmospheric aerosol model parameters, calculations for cardiopulmonary mortality estimates, discussion of uncertainty in our analysis, and additional discussion of our results.

## Global Gridded Ship Emissions

Figure S1 illustrates the underlying ship emissions used for this study. Major trade lanes are clearly identifiable.




a b

**Figure S1. Ship Emissions represented by a) ICOADS and b) AMVER.**

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## Additional Detail on Atmospheric Aerosol Models

GEOS–Chem model is a global 3-D model of atmospheric composition driven by assimilated meteorological observations from the Goddard Earth Observing System (GEOS) of the NASA Global Modeling and Assimilation Office. In the present application, GEOS-Chem was exercised in an aerosol-only mode using the ICOADS ship-emission inventory and archived oxidant fields (OH, $NO_3$, and $O_3$), total inorganic nitrate fields, and $H_2O_2$ production rates and photolysis frequencies, from a coupled oxidant-aerosol simulation [1]. The aerosol simulation includes ammonium, sulfate, nitrate, primary black and organic carbon, secondary organics, dust (4 size bins), and sea-salt (2 size bins) [1-5]. The model has a horizontal resolution of $2^o$ latitude x $2.5^o$ longitude, and is driven with archived meteorological fields from the GEOS-4 data assimilation system [6]. $PM_{2.5}$ mass concentrations are estimated as the sum of all aerosol components excluding dust and sea-salt that are in coarse mode size bins, and including a correction factor of 1.4 to account for noncarbon mass attached to organic carbon aerosol [5].

ECHAM5/MESSy1-MADE (referred to as E5/M1-MADE) is an aerosol microphysics module [7] coupled to the global general circulation model ECHAM5 [8] within the framework of the Modular Earth Submodel System MESSy [9].. Aerosols are described by three log-normally distributed modes, the Aitken, the accumulation and the coarse mode. Aerosol components considered are sulfate (SO4), nitrate (NO3), ammonium (NH4), aerosol liquid water, mineral dust, sea salt, black carbon and particulate organic matter. All simulations were conducted for year 2000 meteorological conditions in T42 horizontal resolution (about for a 2.8°×2.8° longitude by latitude of the corresponding quadratic Gaussian grid cell) with 19 vertical, non-equidistant layers from the surface up to 10 hPa (~30 km). Emissions of trace gases from non-ship sources are taken from the Emission Database for Global Atmospheric Research

EDGAR 3.2 FT2000 [10], and emissions of $SO_2$ and PM from Dentener et al. [11]. In order to obtain significant differences from model simulation with and without taking into account shipping with a reasonable number of model years, model dynamics have been nudged using operational analysis data from the European Centre for Medium-Range Weather Forecasts (ECMWF) from 1999 to 2004.

## Estimating Cardiopulmonary and Lung Cancer Mortalities

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 US [12]. We apply these US-derived C-R functions to our entire spatial dataset, recognizing that transferring US-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 [13, 14]. However, epidemiological studies have found a relatively consistent association between short-term PM exposure and mortality across several countries and continents—from South America to Western Europe [15-17]; therefore we assume that long-term PM exposures will be similarly consistent. We employ a log-linear exposure function using Pope [12] to estimate long-term mortality effects of $PM_{2.5}$, as recommended and described in Ostro [17][2].

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[2]Ostro (2004) calls this the *log-linear* formulation because it relates risk to a logarithmic function of concentration, as shown below in the equation for relative risk (RR) and its alternative:

$$RR = \frac{e^{[\alpha+\beta\ln(X_1+1)]}}{e^{[\alpha+\beta\ln(X_0+1)]}} = \left[\frac{(X_1+1)}{(X_0+1)}\right]^{\beta} \quad (3)$$

This is different to what Ostro (2004) has called a "linear" formulation , where RR and its simplification is given as follows:

$$RR = \frac{e^{(\alpha+\beta X_1)}}{e^{(\alpha+\beta X_0)}} = e^{\beta(X_1-X_0)} \quad (4)$$

For our base case estimates, Relative risk (RR) is calculated considering the C-R function and change in $PM_{2.5}$ concentration, and is given by:

$$RR = \left[\frac{(X_1 + 1)}{(X_0 + 1)}\right]^{\beta} \quad (1)$$

where $\beta = 0.1551$ (95% CI = 0.05624, 0.2541) for cardiopulmonary mortality; $\beta = 0.232179$ (95% CI = 0.08563, 0.37873) for lung cancer related mortality; $X_1$ is the pollutant concentration for the case under study in $\mu g/m^3$; and $X_0$ is the pollutant background concentration in $\mu g/m^3$ [17].

We calculate the total effect (E) of changes in $PM_{2.5}$ concentrations in each grid cell as:

$$E = AF \times B \times P \quad (2)$$

where *AF* is the attributable fraction of deaths to the shipping-related $PM_{2.5}$ pollution, and is given by:

$$AF = \frac{RR - 1}{RR} \quad (5)$$

*B* represents the incidence of the given health effect (e.g., cardiopulmonary deaths/person/year), and *P* represents the relevant exposed population. The results of our analysis are discussed in the next section.

## Detailed Uncertainty Discussion

We identify seven major uncertainties falling into three groupings (ship inventory and PM constituent uncertainties, health effects uncertainties, and atmospheric modeling uncertainties).

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It should be noted that others have referred to Ostro's "linear" formulation as "log-linear", for example as discussed in supporting documentation for the EPA's Benefits Mapping (BenMAP) software which uses this form, but calls it "log-linear" (see Section F of citation 7).

We quantified health effects uncertainties in our assessments, as recommended in the literature [17-19]. Ostro [17] identifies major areas of uncertainty in a $PM_{2.5}$-related mortality assessment as: i) the shape of the C-R function; ii) the inclusion of a subset of health impacts in the assessment; iii) the choice of epidemiological study for C-R coefficients; iv) baseline incidence rates; v) $PM_{2.5}$ exposure estimates; and, vi) background $PM_{2.5}$ concentrations for comparison. We acknowledge population age demographics as an additional uncertainty. In terms of this study, the major sources of uncertainty are discussed in order of importance:

1) *Global Ship Emissions:* Uncertainty in ship inventories remains significant, despite a convergence in recent inventories within published uncertainty bounds [20]. Corbett modeled cargo ship emissions and Eyring modeled total fleet emissions, a difference of about 30%. Adjusting for emissions totals, Case 1c and 2b inventory totals (and similarly Case 1a, Case 1b and Case 2a differences) are within 15%.
2) *Global Ship Traffic:* Global distribution of these emissions differs between ICOADS and AMVER. Spatial differences in ship emissions patterns between ICOADS and AMVER can regionally be greater than a factor of two, even when similar emissions totals are used.
3) *Modeling of Ship $PM_{2.5}$ Composition:* Cases 1c and 2b with all PM included demonstrate that additional $PM_{2.5}$ constituents typical of marine fuel combustion produce additional mortality effects where shipping occurs (Case 1c versus Case 1b or Case 2b versus Case 2a). Even using common inventories and traffic distributions, the two models compared here produced disparate results, slightly greater than a twofold difference in estimated health impacts (Case 1a versus Case 1b).

4) *Shape of the C-R function*: Our base case analyses assumed a log-linear C-R function shape. As recommended in Ostro [17], we conducted alternate analyses using the linear function from Pope [12]. Using the linear functional form, we calculate global cardiopulmonary and lung cancer mortality from ship $PM_{2.5}$ emissions to be: 13,980; 40,130; 64,000; 41,580; 62,900; and, 19,850 for Cases 1a, 1b, 1c, 2a, 2b, and 3, respectively. These numbers represent changes in mortality estimates from our non-linear cases ranging from -28% to +3%. Health function uncertainties (including items 4 and 5, below) would be independent from and additive to uncertainty items 1 and 2; although new information on PM health effects would likely adjust them in concert.

5) *Choice of epidemiological study for C-R functions:* Our base case analyses used C-R functions from Pope [12], which combined exposure coefficients from the years 1979-1983 and 1999-2000. We also conduct analysis using Pope 1979-1983 C-R coefficients alone to quantify any uncertainty of using the 2002 combined coefficients. Using the older C-R coefficients, we calculate mortality to be: 14,140; 29,050; 47,970; 30,040; 46,230; and 20,190 for Cases 1a, 1b, 1c, 2a, 2b, and 3, respectively. Older C-R functions estimate about 25% fewer mortality incidents than our base cases, although more recent work appears to confirm the higher C-R relationships [21].

6) *Baseline incidence rates and population age demographics:* Our base case analysis assumed uniform incidence rates by WHO region, and homogenous portions of the population in the 30+ age bracket by continent. These numbers may not be accurate at the grid cell level. For example, cells with disproportionately older populations would expect to see higher $PM_{2.5}$ related mortality than cells with disproportionately younger populations. Though we do not directly quantify this uncertainty, we recognize that any

percentage change in population values will affect mortality in proportional fashion (see Equation 2).

7) *Non-ship $PM_{2.5}$ Concentration/Background Concentration:* Our base case analysis uses $PM_{2.5}$ inventory data and atmospheric modeling to estimate $PM_{2.5}$ concentrations. Each of these introduces uncertainty into our results. Given that the contributions of ship $PM_{2.5}$ emissions are only a small percentage of total $PM_{2.5}$ concentrations, the effect of non-ship inventories on background incident estimates in the ship-mortality C-R functions may be small. However, uncertainty in this approach affects all similar estimates of mortality attributed to an added source or increase in PM. In addition, the relationship between concentrations and mortality also behaves proportionally (at least within reasonable percentage uncertainty bounds).

## Elaboration of results

Table 1 compares our estimates of cardiopulmonary mortalities due to *all sources* of $PM_{2.5}$ to previous estimates of cardiopulmonary mortalities due to outdoor pollution [19]. The table describes also the normalization approach used to address the differences in the studies' approaches, which include: i) $PM_{2.5}$ composition; assumed baseline $PM_{2.5}$ level for comparison; ii) examined population; and, iii) year.

Figure S2 depicts our *North America, Europe/Mediterranean, East Asia, South Asia and East South America Regions*, for which we estimated mortalities separately.

Table S2 compares: 1) our estimates of U.S. mortalities to estimates using EPA's environmental Benefits Mapping and Analysis Program with our $PM_{2.5}$ concentration grid; and, 2) our California mortality estimates to California Air Resources Board (ARB) estimates of mortalities attributable to shipping emissions in the state. The table describes the assumptions

and functions used in each study, and the assumptions we employed to normalize our estimates for comparison purposes.

**Table S1: Comparison to Cohen (2004): Methodological Differences, Normalization, and Results**

| Study Inputs | Cohen et al. (2004) | This Study | Normalization Approach | Cohen Estimate/ Case 1a Estimate (% difference) | Cohen Estimate/ Case 2b Estimate (% difference) |
|---|---|---|---|---|---|
| **Counterfactual/ baseline $PM_{2.5}$ for comparison**[a] | 7.5 µg/m$^3$ | $PM_{2.5}$ concentration without shipping (grid cell-specific) | Compared *2002 With Shipping* concentrations to 7.5 µg/m$^3$ baseline. | 2,355,500 Before urban population adjustment | 2,815,300 Before urban population adjustment |
| **$PM_{2.5}$ Composition**[b] | Monitoring and modeling, includes nitrates | Modeling, nitrates not included | Increased $PM_{2.5}$ to assume 13% nitrates in $PM_{2.5}$ | | Nitrates and ammonium included |
| **Examined Population**[c] | Capital cities, cities with >100,000 Year 2000 (2.02 Billion) | Entire global population, Year 2005 (6.45 Billion) | Multiplied our estimate by 31.3% | **712,000/ 737,000 (+3.5%)** | **712,000/ 881,000 (+24%)** |

[a]For most regions, Cohen et al. (2004) compared actual $PM_{2.5}$ concentrations to a 7.5 µg/m$^3$ background level assumption in order to analyze mortality effects above this background level. To compare our work with Cohen et al's, we do the same calculation, but use concentration results from our *2002 With Shipping* scenario.
[b]Cohen et al. (2004) include all components of $PM_{2.5}$ including nitrates, which we do not include in Case 1a. To compare our work with Cohen et al's, we increased the concentration of $PM_{2.5}$ in each cell by 13%--the average percentage of nitrates in $PM_{2.5}$ based on EPA estimates [22] and in close agreement with our own model results.
[c]Cohen et al. (2004) looked at populations of capital cities and cities with more than 100,000 people in 2000 (2.02 billion). This is 31.3% of our examined population—the entire global population in the year 2005 (6.45 billion). To compare our work with Cohen et al's, we multiply our estimates by 31.3%.

**Figure S2: Regions for which detailed data are provided in Table 2 of main article.**

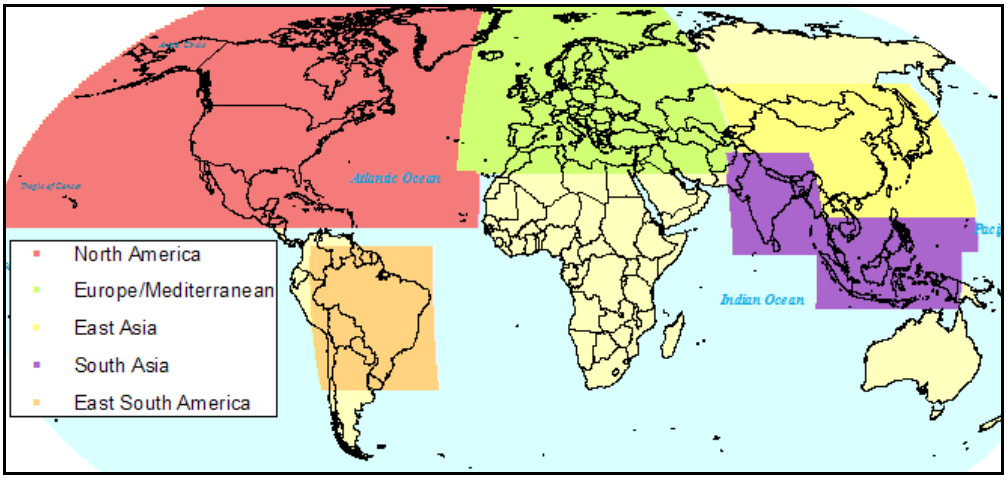


**Table S2: Comparison of Results for National and Local Level**

| Health Impact | Case | Results (% change) |
|---|---|---|
| ***National Scale Comparison*** | | |
| Comparison Study and Assumptions | Case 1a BenMAP Analysis, U.S. All-Cause[3] Mortality U.S. Census-level incidence, linear[4] C-R | **800** |
| Our Study and Assumptions | Case 1a U.S. All-Cause Mortality U.S. average incidence (.0146) linear C-R | 850 (+6%) |
| | Case 1a U.S. All-Cause Mortality WHO Incidence (.0128), linear C-R | 750 (-6%) |
| ***Local Scale Comparison*** | | |
| Comparison Study and Assumptions | ARB study, California All-Cause Mortality California incidence, log-linear C-R (excludes sulfates, includes nitrates) | **190** |
| Our Study and Assumptions | Case 1a, California Grid Cells, All-Cause Mortality WHO incidence, log-linear C-R (includes sulfates, excludes nitrates) | 350 (+86%) |
| | Case 2b, California Grid Cells, All-Cause Mortality WHO incidence, log-linear C-R (includes all shipping PM) | 460 (+142%) |

Figures S2 through S5 illustrate our case-specific estimates of $PM_{2.5}$ concentrations and mortalities. Maps depicting estimates are presented for each of our six cases, to demonstrate differences in estimates due to inventory and model dissimilarities. Cases 1 and 2 , which estimate $PM_{2.5}$ concentrations attributable to shipping in the year 2002, include five sub-cases:

- *Case 1a* uses GEOS-Chem to model ICOADS-derived ship emissions, including black carbon (BC), organic particles (POM), and sulfates only;

[3] Note that for both comparisons, we used the C-R function for "all-cause" mortalities in our estimations, as this was the C-R function available in the BenMAP program, and employed in the ARB study. As discussed in the article, examining "all-cause" mortality on a global scale is inappropriate due to differences in incidence rates by region, and due to lack of reliable data related to many causes of death.

[4] The BenMAP program uses a linear C-R function; thus, we used the linear function in our comparison.

- *Case 1b* uses E5/M1-MADE to model ICOADS-derived ship emissions of BC, POM, and sulfates;
- *Case 1c* uses E5/M1-MADE to model ICOADS-derived ship emissions of all PM constituents.
- *Case 2a* uses E5/M1-MADE to model AMVER-derived ship emissions of BC, POM, and sulfates; and
- *Case* 2*b* uses E5/M1-MADE to model AMVER-derived ship emissions of all PM constituents.

Case 3 estimates PM2.5 concentrations attributable to shipping in the year 2012, using ICOADS-derived ship emissions and forecasting. Case 3 examines BC, POM, and sulfates only, using the GEOS-Chem model.

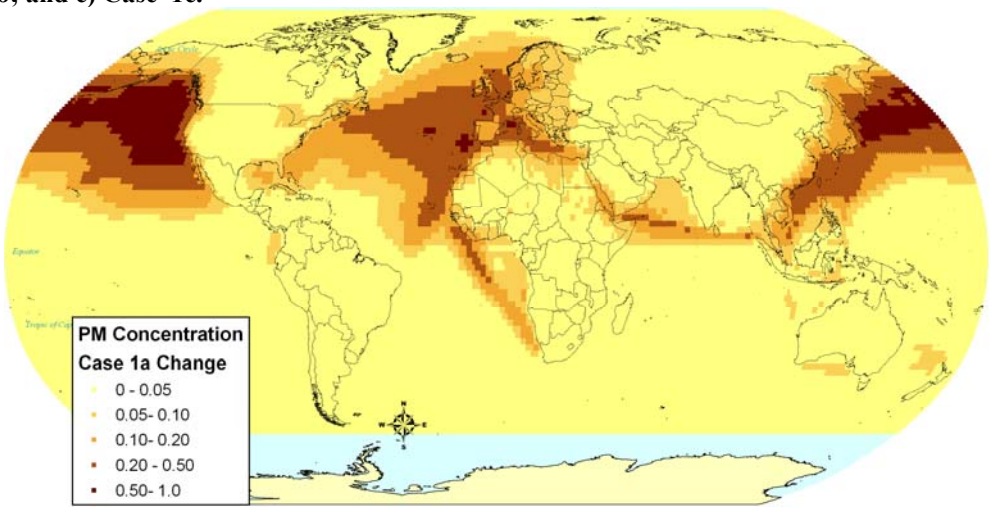
We present results in two sets: *Set 1* depicts the annual average contribution of shipping to $PM_{2.5}$ concentrations; *Set 2* maps depict case-specific cardiopulmonary mortality estimates.

We also present a population-weighted mortality map representing the mortality per million persons per grid cell. This represents one depiction of mortality risk due to ship PM pollution. We point out that this illustration averaged in each grid cell the mortality estimates for Cases 1c and 2b; in other words, the population risk due to shipping in some cells would be estimated higher if Cases 1c and 2b were not averaged, and the locations of greatest risk would vary individually among the scenarios.

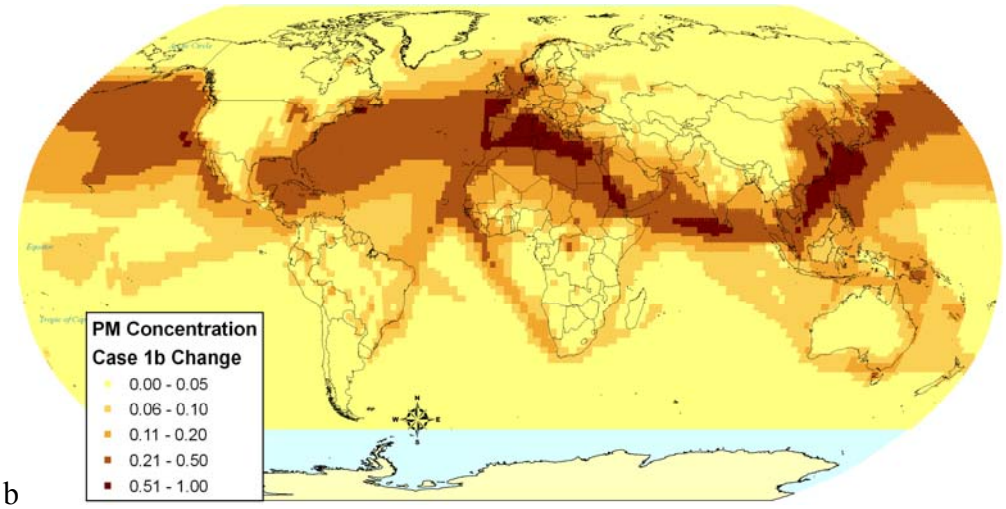
*Set 1: Shipping contribution to Global $PM_{2.5}$ Concentrations*

**Figure S3: Annual average contribution of shipping to $PM_{2.5}$ concentrations (µg/m3) for a) Case 1a; b) Case 1b; and c) Case 1c.**

a




b




c

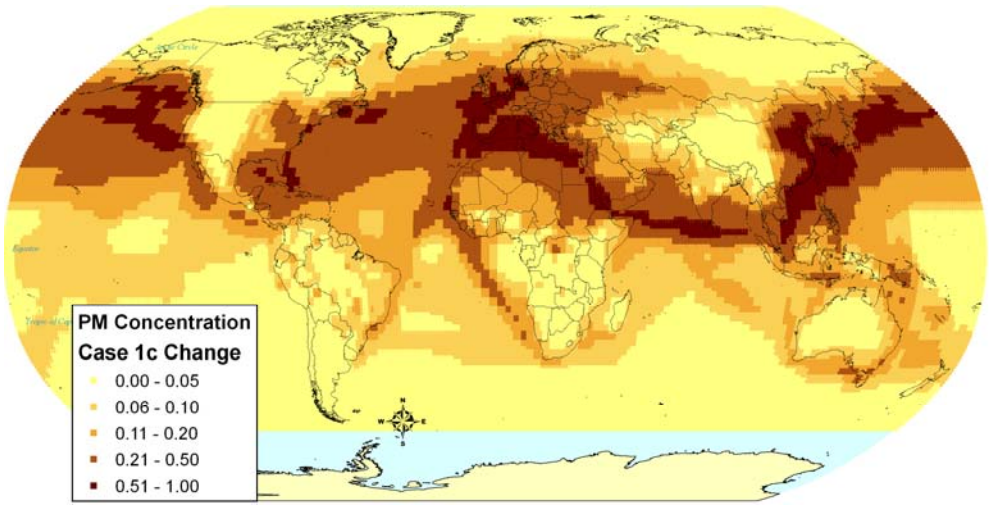
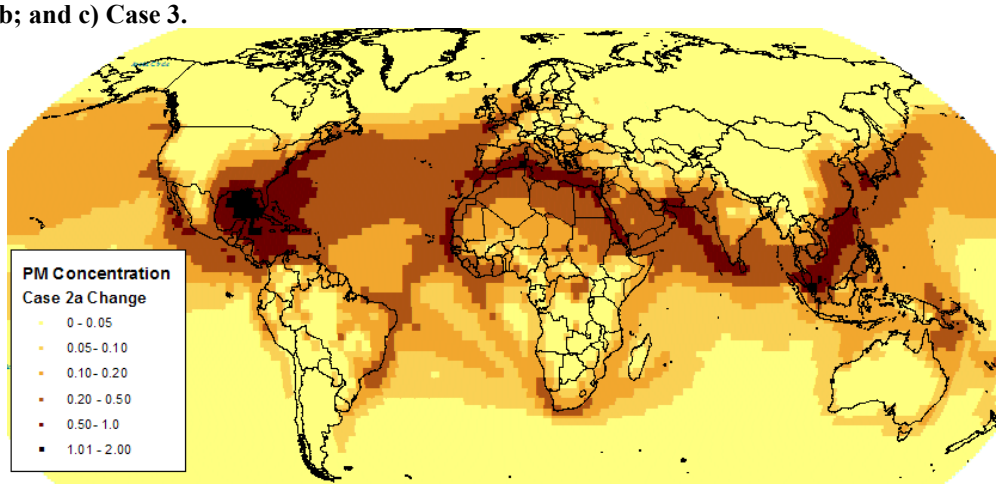
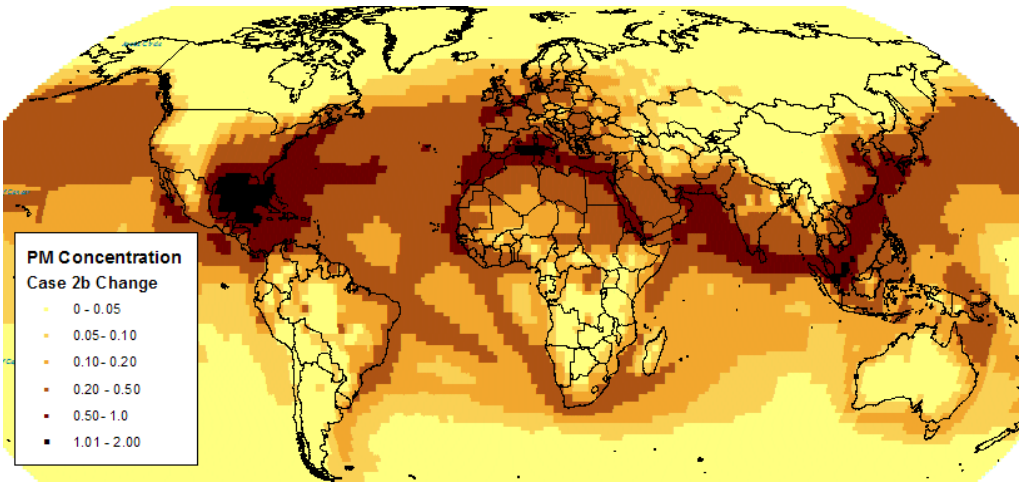


**Figure S4: Annual average contribution of shipping to $PM_{2.5}$ concentrations (µg/m3) for a) Case 2a; b) Case 2b; and c) Case 3.**

a




b

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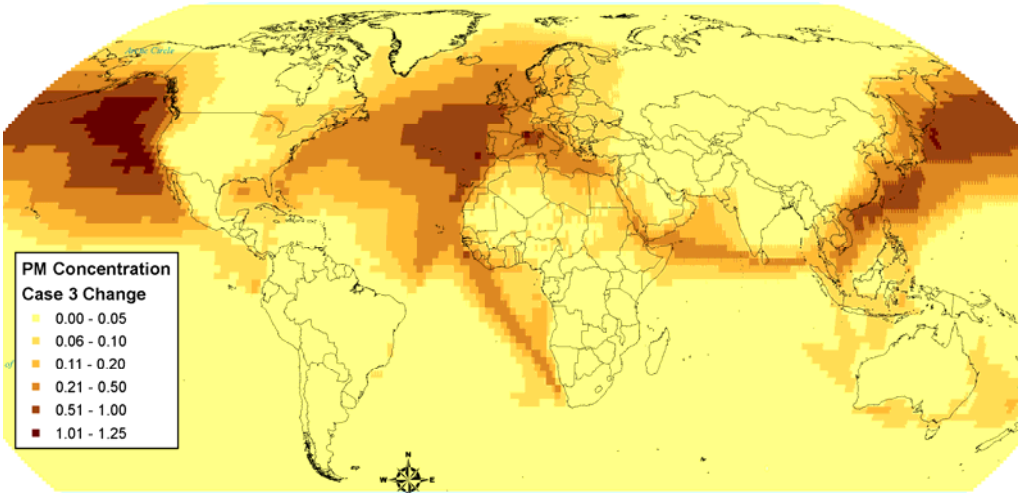
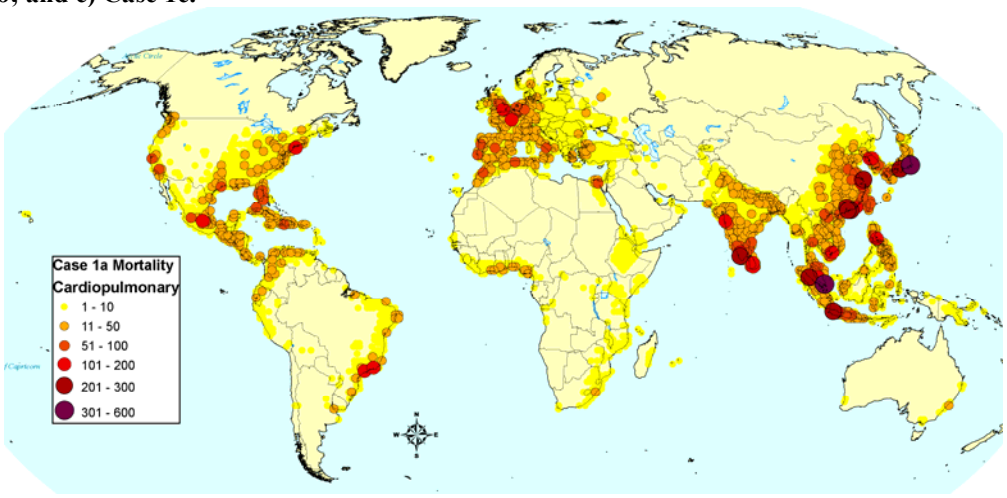


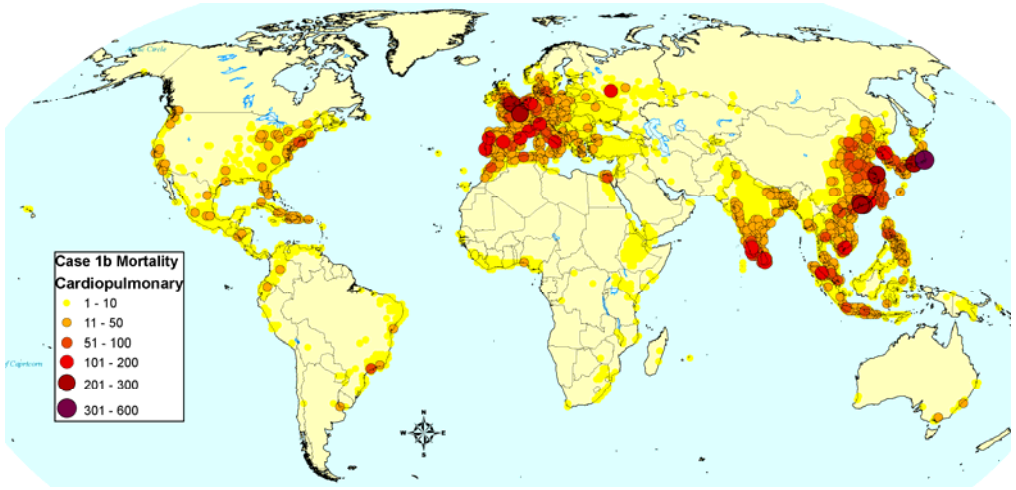
*Set 2: Global Cardiopulmonary Mortality Case Estimates*

**Figure S5: Cardiopulmonary mortality attributable to ship PM2.5 emissions worldwide: a) Case 1a; b) Case 1b; and c) Case 1c.**

a




b




c

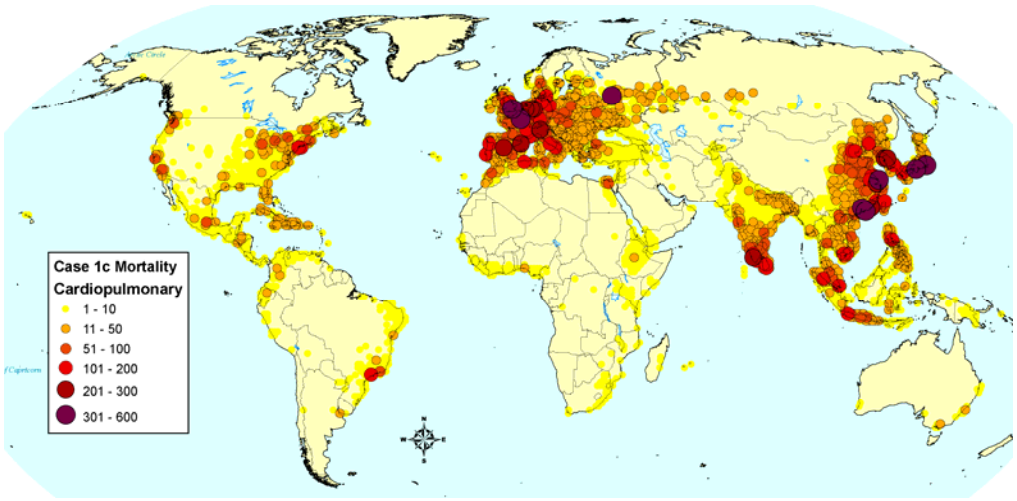
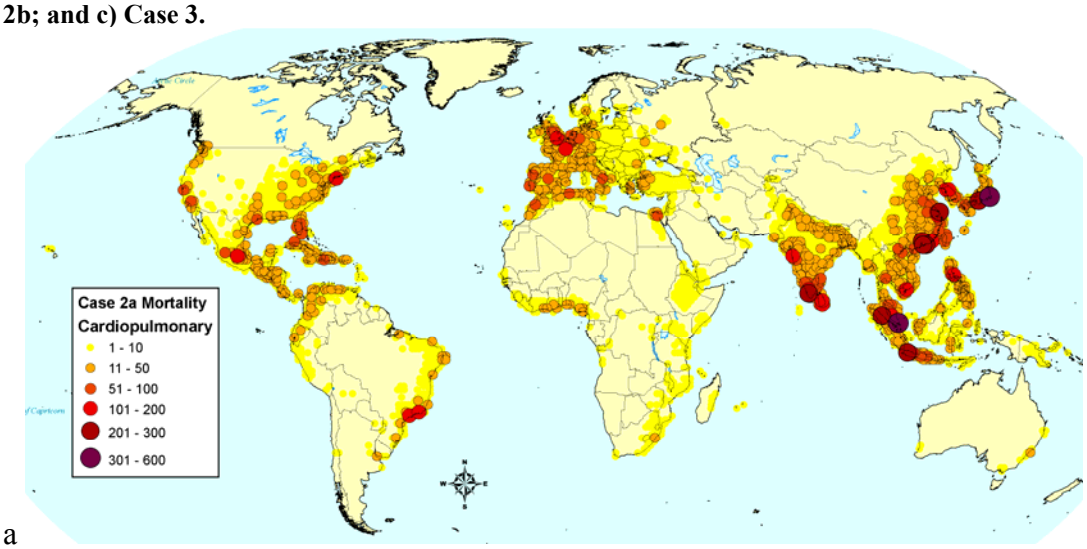
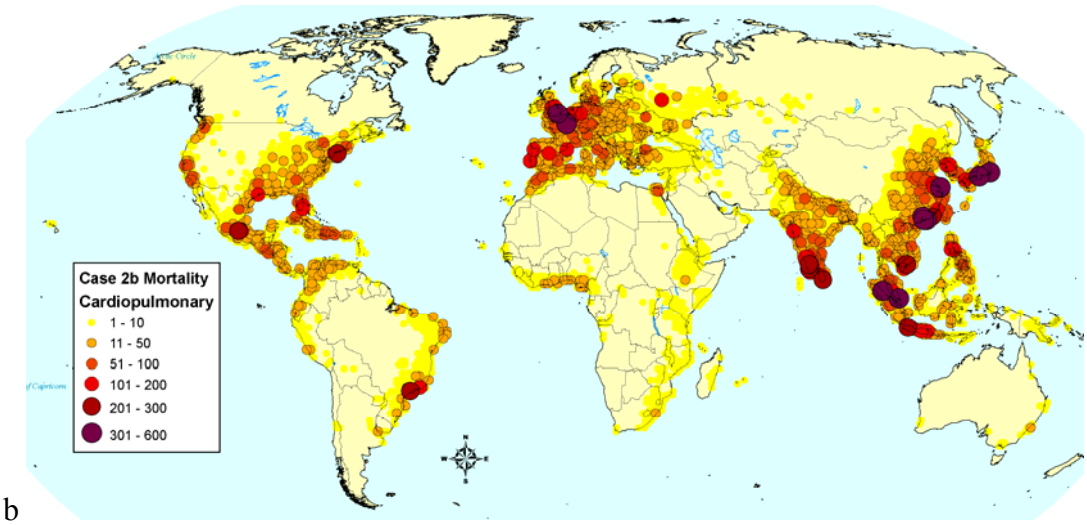


**Figure S6: Cardiopulmonary mortality attributable to ship PM2.5 emissions worldwide: a) Case 2a; b) Case 2b; and c) Case 3.**




a

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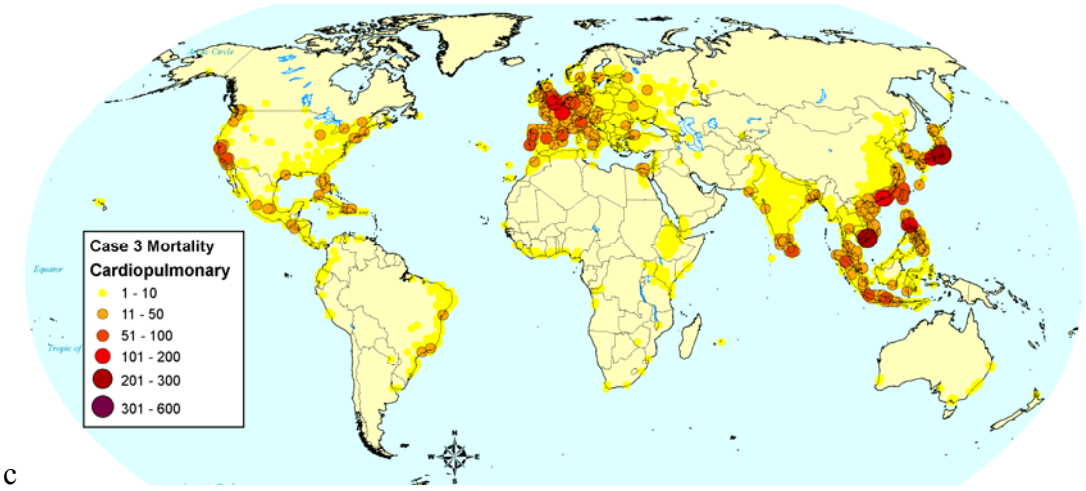
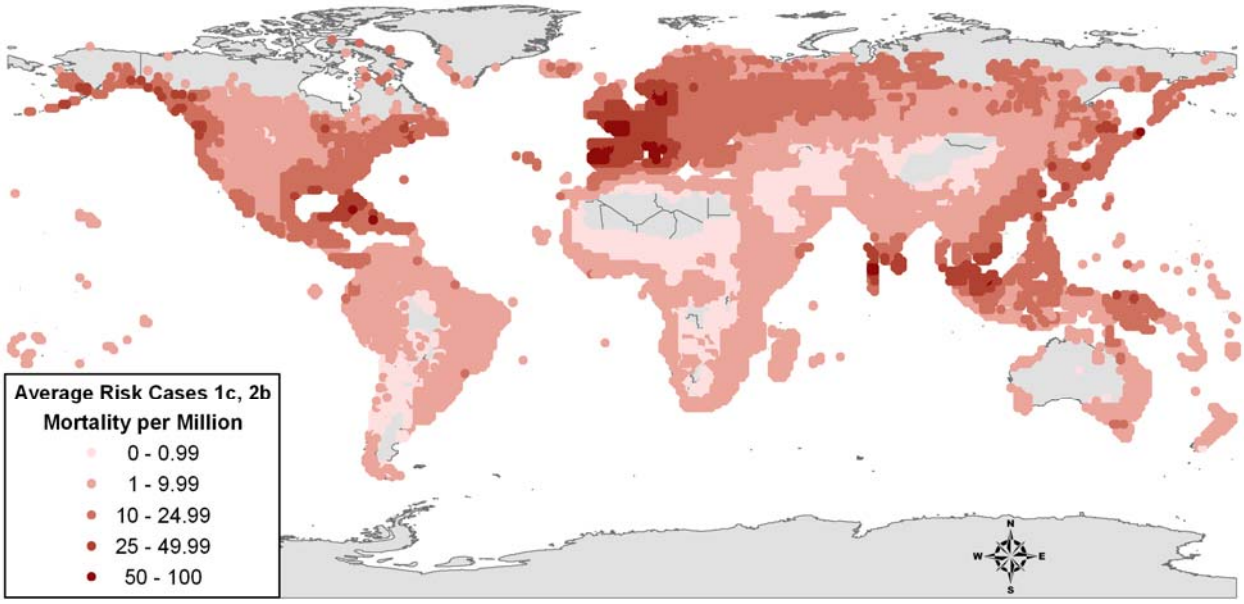



c

**Figure S7. Population-weighted mortality risk, averaging best estimates from Cases 1c and 2b.**



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