

## Mitigating the Health Impacts of Pollution from Oceangoing Shipping: An Assessment of Low-Sulfur Fuel Mandates

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Concerns about health effects due to emissions from ships have magnified international policy debate regarding low-sulfur fuel mandates for marine fuel. Policy discussions center on setting *sulfur content levels* and the *geographic specification* of low-sulfur fuel use. We quantify changes in premature mortality due to emissions from ships under several sulfur emissions control scenarios. We compare a 2012 *No Control* scenario (assuming 2.7% or 27 000 ppm S) with three emissions control scenarios. Two control scenarios represent cases where marine fuel is limited to 0.5% S (5000 ppm) and 0.1% S (1000 ppm) content, respectively, within 200 nautical miles of coastal areas. The third control scenario represents a global limit of 0.5% S. We apply the global climate model ECHAM5/MESSy1-MADE to geospatial emissions inventories to determine worldwide concentrations of particulate matter (PM<sub>2.5</sub>) from oceangoing vessels. Using those PM<sub>2.5</sub> concentrations in cardiopulmonary and lung cancer concentration-risk functions and population models, we estimate annual premature mortality. Without control, our central estimate is approximately 87 000 premature deaths annually in 2012. Coastal area control scenarios *reduce* premature deaths by ~33 500 for the 0.5% case and ~43 500 for the 0.1% case. Where fuel sulfur content is reduced globally to 0.5% S, premature deaths are reduced by ~41 200. These results provide important support that global health benefits are associated with low-sulfur marine fuels, and allow for relative comparison of the benefits of alternative control strategies.

### Introduction

One of the more nefarious environmental impacts of goods movement is its effect on air quality and human health (1–6).

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Specifically, particulate matter with aerodynamic diameters of 2.5  $\mu\text{m}$  or less (PM<sub>2.5</sub>) from international shipping poses special concerns. Ambient concentrations of PM<sub>2.5</sub> have been associated with a wide range of health effects including asthma, heart attacks, and hospital admissions. Increases in atmospheric PM<sub>2.5</sub> concentrations have also been closely associated with increases in premature cardiopulmonary and lung cancer mortalities in exposed populations (7, 8).

Particulate matter from ship emissions is related to the sulfur content of marine fuel. In the case of oceangoing vessels, fuel sulfur content averages around 2.7% (27 000 ppm) with upper limits as high as 4.5% S (45 000 ppm) (3). We have estimated in previous work that PM<sub>2.5</sub> due to the burning of such fuel can lead to on-land health impacts on the order of 60 000 premature mortalities in 2002 (3).

Given these health impacts and other concerns related to pollution from high-sulfur fuels, policies aimed at reducing the sulfur content of marine fuel were adopted by the International Maritime Organisation (IMO) under ANNEX VI of MARPOL 73/78 (the International Convention for the Prevention of Pollution from Ships). In particular, the global fuel sulfur cap will be reduced to 3.5% S in 2012 and 0.5% S as early as 2020, which implies a movement from residual fuel oil to distillate fuels (i.e., marine diesel oil and marine gas oil). More stringent reductions will be required for ships operating in sulfur emission control areas (SECA). These areas (of which there are currently three, but are expected to increase in the coming years) will require sulfur content of no more than 1% S (10 000 ppm) by 2010 and 0.1% S (1000 ppm) by 2015 within 200 nautical miles (nm) of coastal areas.

We quantify premature mortality due to emissions from ships operating under several sulfur emissions control scenarios. We do this by applying the global climate model ECHAM5/MESSy1-MADE to a geospatial inventory of shipping emissions to determine worldwide concentrations of PM<sub>2.5</sub> from oceangoing vessels assuming no emissions control regimes. We then use those PM<sub>2.5</sub> concentrations in cardiopulmonary and lung cancer concentration-risk functions and population models to estimate annual premature mortality from these emissions. The paper compares a 2012 *No Control* scenario (assuming a global average of 2.7% or 27 000 ppm S) with three emissions control scenarios. The first two control scenarios represent cases where marine fuel is limited to 0.5% S (5000 ppm) and 0.1% S (1000 ppm) content, respectively, within 200 nm of coastal areas. The third control scenario represents a case where sulfur content is limited to 0.5% S globally. In this way, we provide a global estimate of some human health benefits associated with different control options. Other impacts, such as the effects of these policies on atmospheric aerosol burdens and the Earth's radiation budget, are discussed in a related paper (9).

The next section of the paper discusses our analytical approach, including the process of generating geospatially resolved emissions inventories for ships, processing these inventories through ECHAM5/MESSy1-MADE to calculate PM<sub>2.5</sub> concentrations, and applying these concentrations to risk functions to estimate health impacts. This is followed by a discussion of our results and the implications of this study.

### Analytical Approach

**Emissions Inventories and Concentrations.** We employ the approach taken in previously published work and described in more detail in related papers (3, 9). This approach involves (1) generating geospatial emissions inventories for ships

**TABLE 1. Relative Emissions by Pollutant for Each of Our 2012 Scenarios Compared to the No Control Case<sup>a</sup>**

ship emission scenario	fuel sulfur content	NO <sub>x</sub>	SO <sub>2</sub>	primary SO <sub>4</sub>	CO	BC	POM
2012 <i>No Control</i>	2.7%	100%	100%	100%	100%	100%	100%
2012 <i>Coastal 0.5</i>	0.5%	100%	18.5%	18.5%	100%	100%	32.1%
2012 <i>Coastal 0.1</i>	0.1%	100%	3.7%	3.7%	100%	100%	20.0%
2012 <i>Global 0.5</i>	0.5%	100%	18.5%	18.5%	100%	100%	32.1%

<sup>a</sup> Emission reductions are applied within 200 nautical miles of coastal areas in the *Coastal* scenarios and globally in the *Global* scenario. Source: ref 9

under different regulatory mandates (e.g., fuel sulfur content controls); (2) processing these emissions inventories with global inventories from other sources through a global atmospheric aerosol/chemistry model to determine the geospatial concentrations of pollutants attributed to shipping; and (3) applying concentration-risk (C-R) functions and population models to determine health impacts from these concentrations.

We compare health impacts from a 2012 “*No Control*” scenario against three emissions control scenarios. Therefore, as mentioned above, we have four scenarios for our analysis: (1) *No Control*, representing a case where marine fuel is 2.7% S globally; (2) *Coastal 0.5*, representing a case where marine fuel is 0.5% S within 200 nm of coastal areas; (3) *Coastal 0.1*, representing a case where marine fuel is 0.1% S within 200 nm of coastal areas; and (4) *Global 0.5*, representing a case where marine fuel is 0.5% S globally.

We employ the same geospatial inventory (10), atmospheric model, uncertainty factors, and health risk functions as reported previously (3). A detailed discussion of uncertainty is provided in the Supporting Information (SI). Where earlier work demonstrated mortality results that were consistent with previous scientific studies at both a global and regional scale (11, 12), this paper compares control scenarios with our prior study to assess health benefits of fuel sulfur control.

PM<sub>2.5</sub> emissions estimates were obtained based on a global representation of ship emissions inventories of sulfur, black carbon, and organic carbon. We use the previously derived 2002 emissions inventories based on the International Comprehensive Ocean-Atmosphere Data Set (ICOADS) and the Automated Mutual-Assistance Vessel Rescue System (AMVER) (10). These data sets combine detailed information about vessel characteristics with vessel traffic densities to determine emissions geospatially. All oceangoing commercial ship types are included in these data sets, with some sampling bias based on over-reporting of some vessel types, depending on the data set. More details on both data sets and a comparison are given by Wang et al. (2008) and our companion paper (9).

Ship emissions for 2012 were estimated from current inventories using a uniform global annual compound growth rate of 4.1% to establish our *No Control* scenario (13). To create inventories for our *Coastal 0.5*, *Coastal 0.1*, and *Global 0.5* cases, we reduced emissions of sulfur and particulate organic matter (POM) to correspond with properties for different marine fuel types, as shown in Table 1. Aside from the reductions in fuel sulfur content made plain by our scenario titles, we also reduced POM emissions by ~68% for marine diesel oil at 0.5% S and by ~80% for marine gas oil at 0.1% S to correspond to expected differences among residual and distillate fuel PM<sub>2.5</sub> measurements (14).

These ship emissions inventories are overlaid on global emissions inventories without ship traffic to obtain the increased PM<sub>2.5</sub> pollution attributed to vessel operations. Inventories for nonship sources are for the year 2000 and therefore do not capture changes in nonship emissions that

may occur between 2000 and 2012. A sensitivity study with scaled-up nonship emissions showed that our approach may introduce an uncertainty of about 5–15% to the calculated number of premature deaths. This is similar to the uncertainty associated with the geographical distribution of the ship emissions (AMVER, ICOADS) investigated in this study. Additional details can be found in the SI.

The fate and transport of particles emitted or formed from gaseous ship emissions were then summed into PM<sub>2.5</sub> concentration data (in μg/m<sup>3</sup> dry weight) from atmospheric modeling using ECHAM5/MESSy1-MADE (referred to as E5/M1-MADE (15)), an aerosol microphysics module (MADE) coupled to a general circulation model (ECHAM5 (16)), within the framework of the Modular Earth Submodel System MESSy (17). Along with global PM<sub>2.5</sub> concentrations attributed to nonship sources, the E5/M1-MADE model provides ambient concentrations of BC, POM, sulfates (SO<sub>4</sub>), nitrates (NO<sub>3</sub>), and ammonium ion (NH<sub>4</sub>) aerosols. This model was run under similar input assumptions as previously published (15), and described in greater detail in related papers (3, 9). Model output at 2.8 × 2.8° was interpolated to a 1 × 1° global resolution; therefore, we report our results at the global and continental scales.

Because health-risk studies have concentrated on long-term mortality impacts, we aggregated monthly PM<sub>2.5</sub> data into annual averages. Monthly variations in total ship activity and near coastlines are small, and seasonal variation in open-ocean shipping lanes can be neglected at this model resolution, especially given that we are primarily interested in the *change* across scenarios (10). For example, although some variation does exist over open oceans (due to seasonal shifts in shipping routes and varying meteorological conditions), concentration variation on land is much smaller because nearly 70% of global ship emissions are within 400 km of land, and because ship activity and emissions near shore remain relatively constant throughout the year (18). We do not expect modest seasonal variations near coastlines represented in ICOADS or AMVER global shipping patterns to affect long-term mortality results at the model resolution used in this global study.

Comparing results of each scenario with and without ship inventories of PM<sub>2.5</sub> components and precursor gases, we quantify changes in ambient concentrations of PM<sub>2.5</sub> due to marine shipping. Figure 1 presents globally distributed PM<sub>2.5</sub> concentrations due to ships on a 1 × 1° global grid for each of our scenarios using the ICOADS inventory data set (a similar map for AMVER may be found in the SI).

## Health Impacts

We calculate health impacts due to these PM<sub>2.5</sub> concentrations by applying a health effects model that incorporates demographic data, background incidence data, and concentration-risk (C-R) functions for cardiopulmonary and lung cancer premature mortality. We use population forecasts obtained in a 1 × 1° format from the Socioeconomic Data and Applications Center at Columbia University (19). Acquiring values for 2010 and 2015, we linearly interpolate values for 2012. We also used U.S. Census Bureau International Database numbers to derive, by continent, the percent of population between 30 and 99 years of age (the age group of concern for the examined mortality impacts).

The incidence rates of these health impacts (necessary for calculating the increased risk due to ship emissions, as shown below) were estimated using World Health Organization (WHO) data. We use 2002 WHO estimates for causes of death by age to derive the incidence rates by WHO region for each type of mortality examined. United States cardiopulmonary incidence values obtained from Environmental Benefits Mapping and Analysis Program (BenMAP) technical

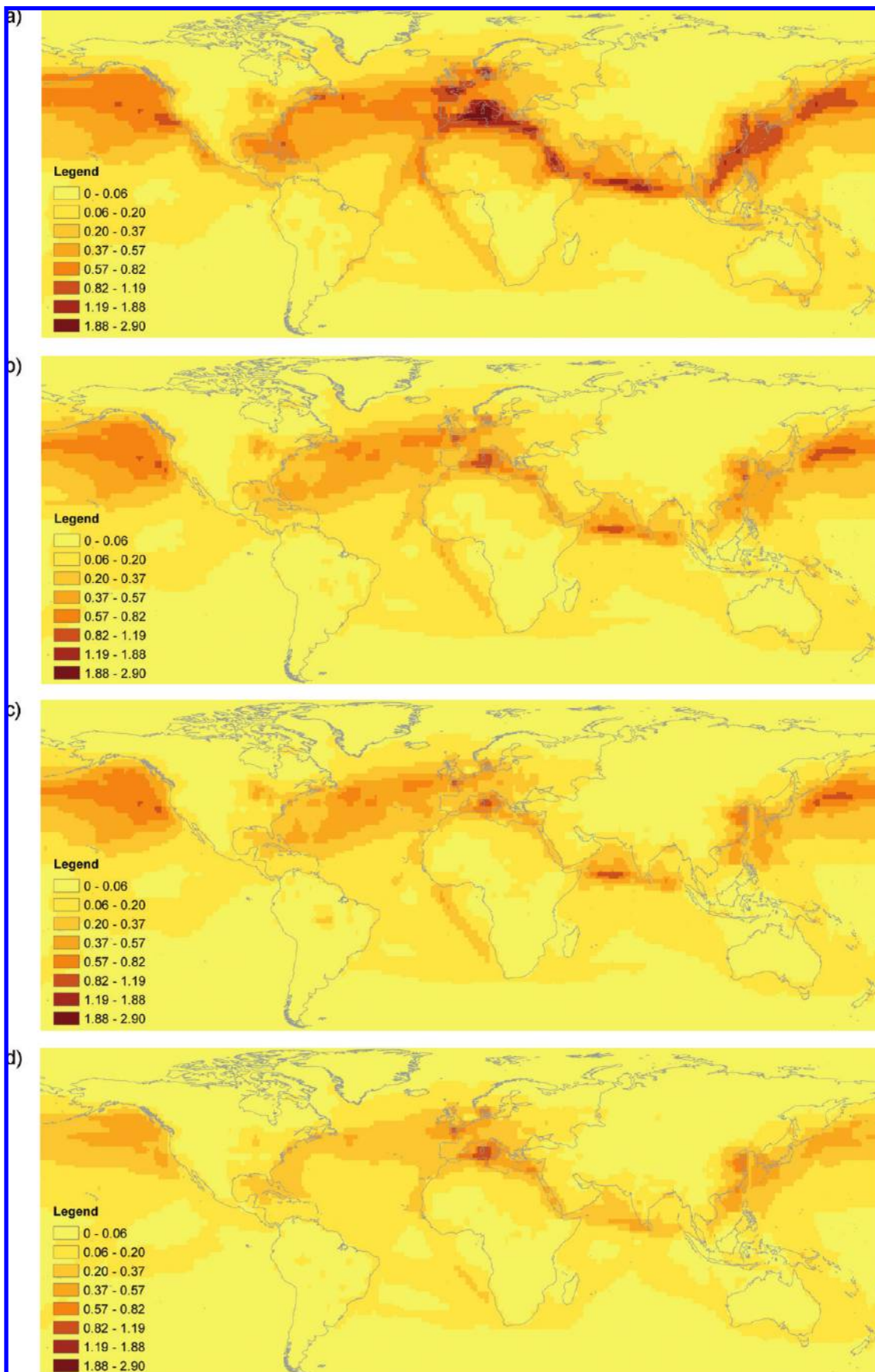


FIGURE 1. Concentrations of  $PM_{2.5}$  for four scenarios with ICOADS data in micrograms per cubic meter. Shows geospatial distribution of  $PM_{2.5}$  pollution due to oceangoing vessels in the 2012 *No Control* scenario and the three control scenarios explored here; values represent concentrations due to shipping (compared to an environment without shipping emissions). (a) *No Control* scenario; (b) *Coastal 0.5%* scenario; (c) *Coastal 0.1%* scenario; (d) *Global 0.5%* scenario.

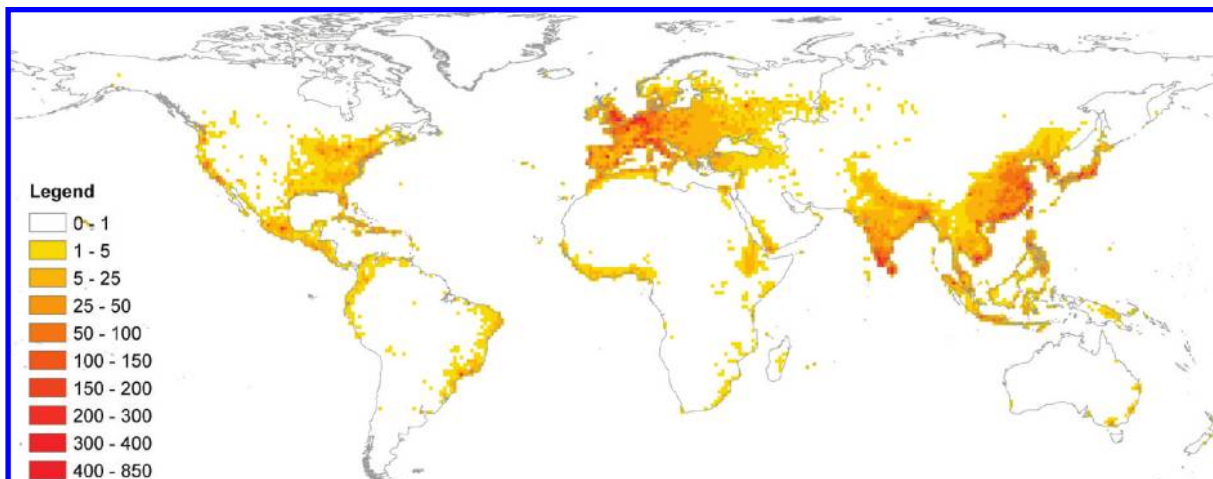


FIGURE 2. Annual premature mortality for the *No Control* scenario compared to a “no shipping” case using ICOADS data.

documentation of the U.S. Environmental Protection Agency (EPA) were used for North American incidence estimates (20).

Mortality impacts are estimated using C-R functions from Pope et al. (2002), the same epidemiological study used to estimate mortality impacts in the U.S. EPA’s regulatory analysis of controlling emissions from nonroad diesel engines. Here, we assume that the global PM<sub>2.5</sub> concentration-mortality association is equivalent to that of the United States. As noted in other work (21), epidemiological studies have found a relatively consistent association between short-term PM<sub>2.5</sub> exposure and mortality across several countries—from South America to Western Europe. By analogy, we assume long-term PM<sub>2.5</sub> exposures will be similarly consistent—an assumption made by other experts in air pollution health-risk assessments (22). Functional coefficient values were obtained for cardiopulmonary and lung cancer mortality from Ostro (2004).

We employed log-linear C-R functions to estimate changes in relative risk of mortality, as recommended in Ostro (2004). The relative risk is calculated considering the C-R function and change in PM<sub>2.5</sub> concentration, and is given by

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

where  $\beta = 0.1551$  (95% confidence interval (CI) = 0.05624, 0.2541) for cardiopulmonary mortality,  $\beta = 0.232179$  (95% CI = 0.08563, 0.37873) for lung cancer related mortality,  $X_0$  represents the concentration ( $\mu\text{g}/\text{m}^3$ ) of the base case, and  $X_1$  represents the concentration ( $\mu\text{g}/\text{m}^3$ ) of the case under evaluation (22). Ostro (2004) calls this the *log-linear* formulation because it relates risk to a logarithmic function of concentration.

We applied changes in relative risk, population, and existing incidence rates to calculate the change in mortality due to ship pollution for each grid cell. Ship pollution incrementally increases ambient PM<sub>2.5</sub> conditions over a base pollution concentration attributed to nonshipping activities. Our formulation does not include a threshold effect for PM<sub>2.5</sub> and there has been some discussion in the literature as to whether a threshold effect exists for PM<sub>2.5</sub> at low concentrations (23–25). However, we decided to use the previously published nonthreshold models given that most impacts will be seen over populated regions where PM<sub>2.5</sub> concentrations are relatively high already (i.e., above potential thresholds). Thus, the total effect ( $E$ ) of additional PM<sub>2.5</sub> concentration is given by

$$E = AF \times B \times P$$

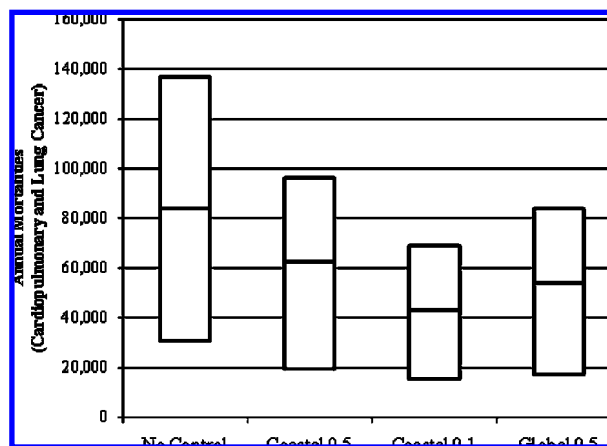


FIGURE 3. Total mortality for four different scenarios showing low, mean, and high estimates for the ICOADS data set.

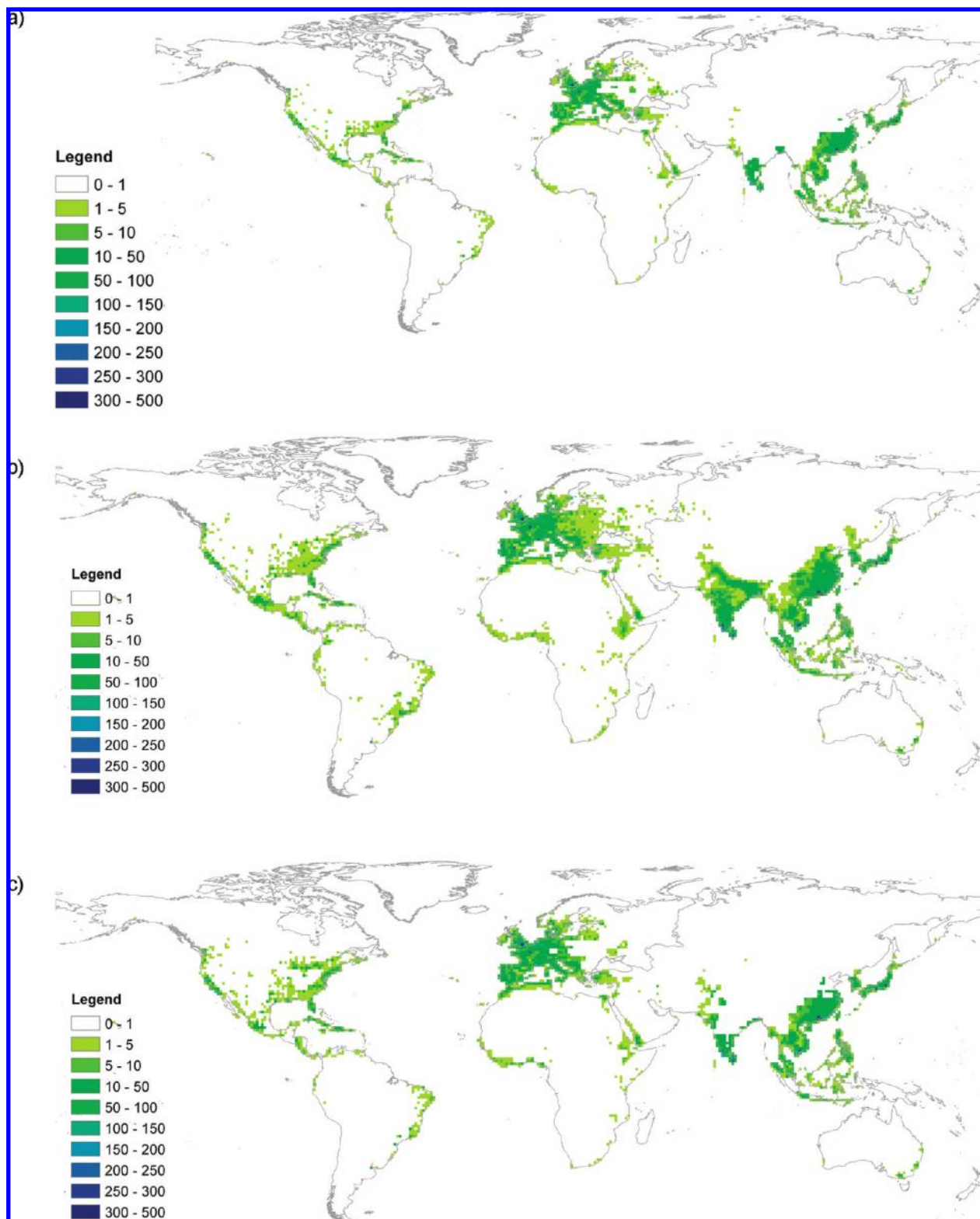
where  $B$  represents the incidence of the given health effect (e.g., deaths/1000 people);  $P$  represents the relevant exposed population; and,  $AF$  is the relative risk due to the increase in pollution, and is given by

$$AF = \frac{RR - 1}{RR}$$

In addition, for our control scenarios, we only calculate health impacts where differences between the climatological annual mean concentrations of PM<sub>2.5</sub> in the control scenario compared to the *No Control* scenario were statistically significant at a 99% confidence level compared to their interannual variability. This trimming controls for potentially insignificant differences among the *No Control* and three control scenarios, a refinement to our previous study.

## Results

Figure 2 shows global distributions of premature mortality due to ship emissions for our *No Control* case using the ICOADS data set. Figure 3 shows premature mortality for each of our cases using a box plot, where the low, middle, and high estimates are presented. Lastly, Figure 4 presents geospatially the estimated number of *avoided premature deaths* as policy-driven controls move from the *No Control* case to each of our three control scenarios. Similar figures for AMVER can be found in the SI. We also include in Table 2 specific values for expected annual mortality due to ship emissions, and expected avoided premature mortalities under



**FIGURE 4. Annual avoided premature mortality for the three control scenarios: (a) Coastal 0.5, (b) Coastal 0.1, and (c) Global 0.5 for the ICOADS data set. Reductions in estimated premature mortality use the 50th-percentile beta values in the C-R function and are relative to the *No Control* scenario.**

our three scenarios. Tables that show disaggregated values by region are included in the SI.

We show that  $PM_{2.5}$  impacts due to ships may cause 83 500 and 76 700 premature cardiopulmonary deaths each year for AMVER and ICOADS inventories, respectively (see Table 2); Likewise ship emissions may cause 7100 and 7000 lung cancer deaths annually, depending on underlying inventory

pattern. These tables show 50th-percentile values setting  $\beta$  at its 50th percentile estimate, as well as ranges representing results based on the 95% confidence intervals for  $\beta$  from the literature as described above (22). All values are rounded to the nearest hundred.

By showing avoided deaths compared to the *No Control* scenario, we report relative health benefits (reduced pre-

**TABLE 2. Global Premature Mortality Due to Ship Emissions and the Avoided Premature Deaths Associated with Three Fuel Sulfur Control Strategies Using a Log-Linear Relative Risk Formulation<sup>a</sup>**

	cardiopulmonary		lung cancer	
	mid	range	mid	range
AMVER Inventory				
2012 <i>No Control</i> case premature mortality	83 500	30 300–136 400	7100	2600–11 500
2012 <i>Coastal 0.5</i> reduced mortality from <i>No Control</i>	33 800	12 200–55 200	2800	0–4500
2012 <i>Coastal 0.1</i> reduced mortality from <i>No Control</i>	41 600	15 100–68 000	3400	1300–5500
2012 <i>Global 0.5</i> reduced mortality from <i>No Control</i>	42 500	12,100–54,700	3500	1000–4300
ICOADS Inventory				
2012 <i>No Control</i> Case premature mortality	76 700	27 800–125 400	7000	2600–11 400
2012 <i>Coastal 0.5</i> reduced mortality from <i>No Control</i>	28 200	10 200–46 200	2500	900–4100
2012 <i>Coastal 0.1</i> reduced mortality from <i>No Control</i>	38 800	14 100–63 500	3400	1300–5500
2012 <i>Global 0.5</i> reduced mortality from <i>No Control</i>	33 500	12 200–54 800	3000	1100–4800

<sup>a</sup> Notes: This table shows results from both the AMVER and ICOADS analyses. The 2012 *No Control* case represents annual premature mortality due to PM<sub>2.5</sub> pollution from oceangoing vessels assuming fuel sulfur content of 2.7% S. The other cases represent *reduced mortality* associated with various sulfur control scenarios compared to the *No Control* case. For example, our analysis shows that a coastal 0.5% fuel sulfur limit will reduce annual cardiopulmonary premature mortality by 33 800 compared to the *No Control* case.

mature mortality) due to each of the control scenarios. For example, based on AMVER results reported in Table 2, a 50th-percentile  $\beta$  value yields a 33 800 decrease in premature deaths due to cardiopulmonary illness in the *Coastal 0.5* scenario, a 41 600 decrease in the *Coastal 0.1* scenario, and a 42 500 decrease in the *Global 0.5* scenario. Likewise, based on ICOADS data, these annual avoided mortalities are 28 200, 38 800, and 33 500, respectively. These values represent reductions in mortality of around 30–50% compared to the *No Control* case.

As may be expected, the *Coastal 0.1* scenario shows the greatest benefits. Using the ICOADS spatial distribution, this scenario is clearly preferred from a health standpoint, although it is almost indistinguishable from the *Global 0.5* scenario using the AMVER spatial distribution. We believe this is because ICOADS places more shipping (and ship emissions) along coastal routes frequented by containerships along coastal shipping particularly in the Europe and Mediterranean areas, whereas AMVER data places more shipping along bulk and tanker routes toward Southeast Asia and North America (10). In other words, less benefit may be expected for global control scenarios compared to coastal control scenarios under the ICOADS spatial distribution. Moreover, formation of aerosol nitrate is stronger in the ICOADS scenarios than the AMVER scenarios (9). This strong nitrate formation in the *Global 0.5* scenario may be another reason why a global emission reduction is less beneficial than coastal action under the ICOADS spatial distribution.

These benefits are not linear with respect to sulfur content reductions. For example, moving from noncontrolled sulfur levels (2.7% S) to 0.5% S represents ~80% decrease in sulfur content and provides ~50% reduction in premature mortality; yet moving from a 0.5% sulfur level to a 0.1% sulfur level (also a decrease of 80%), only provides ~30% incremental benefits in premature mortality. This nonlinear relationship may be partially due to nitrate substitution that exists when SO<sub>x</sub> emissions are reduced without concomitant reductions in nitrates. In such cases, ammonium in the atmosphere (which would preferentially combine with SO<sub>x</sub> to make ammonium-sulfate particles) instead combines with nitrate to make ammonium-nitrate particles. This effect could be further explored as potential cobenefits of reducing nitrate emissions along with sulfur emissions (9).

SI Tables S-1 and S-2 also show the global distribution of avoided mortality impacts. Europe and Asia see large benefits (measured in avoided deaths) in moving from the *No Control* scenario to any of the control scenarios, with Southeast Asia seeing some of the most significant benefits.

A comparison of the *Global 0.5* and the *Coastal 0.5* cases is also informative in terms of quantifying the marginal benefits of moving from a coastal (e.g., ECA) to a global control regulatory regime. Here we show that, at the 50th-percentile  $\beta$  value, the coastal scenario provides between ~31 000 and ~37 000 avoided deaths annually (ICOADS and AMVER, respectively), while the global scenario avoids ~36 000–46 000 deaths annually (ICOADS and AMVER, respectively). This suggests a movement from a coastal regulatory regime to a global one may avoid additional 5000–9000 premature deaths annually.

These results confirm that meaningful benefits are achieved from either a 0.5% S or 0.1% S control strategy. Our findings demonstrate that upward of 45 000 premature mortalities could be prevented annually across the globe with a movement toward lower sulfur fuels in the future. Of course, reduced premature mortality is directly, but nonlinearly, related to the geographic use of clean fuels: lower sulfur fuels lead to larger health benefits, particularly when used in a near-coastal environment.

The premature mortality impacts demonstrated in this paper are only two of many impacts that are related to shipping emissions and fuel quality. Climate change, acidification, visibility, eutrophication, and other environmental effects are closely related to the type of fuel used in ships. This paper and complementary papers on this topic, offer important input to current science and policy discussions about the application of low sulfur fuel regulations for international shipping.

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### Supporting Information Available

Additional figures corresponding to results using the AMVER dataset, additional mortality and avoided death data by world region, and presentation and discussion of uncertainty factors. This material is available free of charge via the Internet at <http://pubs.acs.org>.

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