DISCUSSION PAPER

September 2014 ■ RFF DP 14-30

# How Should the World Bank Estimate Air Pollution Damages?

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## Abstract

We evaluate the approach currently used by the World Bank to measure exposure to outdoor air pollution and associated economic costs, as reported in the World Development Indicators database. We recommend that current exposure estimates, based on an econometric model, be replaced by estimates used in computing the Global Burden of Disease (GBD). The GBD combines satellite data with chemical transport models to provide global estimates of fine particle exposure. We recommend that the World Bank also use estimates of disability-adjusted life years (DALYs) lost due to outdoor air pollution produced by the GBD. DALYs should continue to be monetized using the value of a statistical life year, which is currently transferred from a US value of a statistical life (VSL) using an income elasticity of one. Going forward, it would be desirable to allow the income elasticity of the VSL to vary with income and to revisit the choice of baseline VSL.

**Key Words:** air pollution exposure, valuing DALYs, Global Burden of Disease

**JEL Classification Numbers:** Q51, Q53, Q56

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# How Should the World Bank Estimate Air Pollution Damages?

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## I. Introduction and Overview

### *A. Motivation*

One of the indicators the World Bank uses to measure sustainability of a country's growth is adjusted net savings (ANS). The ANS indicator accounts for pollution damages by estimating the costs of health damages from exposure to particulate matter. This pollution damage indicator is published annually in the World Development Indicators database along with $PM_{10}$ (particulate matter less than 10 microns in diameter) concentrations. $PM_{10}$ concentrations, published at the country level, are based on estimates of annual average $PM_{10}$ in cities of 100,000 or more. Estimates of the health damages associated with $PM_{10}$ exposure are reported annually in the *Little Green Data Book*. Health damages are the monetized value of disability-adjusted life years (DALYs) associated with $PM_{10}$ exposure in these cities, expressed as a percentage of each country's gross national income (GNI).

This paper reviews the current methodology for estimating the cost of air pollution damages and identifies better data sources to enable more accurate estimates of pollution exposure and health costs. This effort involves a comprehensive review of the current methodology as well as a justification for a proposed new approach. The project has involved consultations with experts in economics, atmospheric science, geographic information systems, and epidemiology.

### *B. Summary of Recommendations*

We believe the current approach to modeling exposure to outdoor air pollution has several shortcomings. Because they focus on cities with populations of 100,000 or more, current estimates ignore approximately 5 billion people who live outside the 3,224 cities considered.

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Although outdoor air pollution has historically been considered a problem in urban areas, it would be preferable to estimate exposure for persons in rural areas as well. For example, a comparison of Brauer et al. (2012) and World Bank estimates suggests that half of deaths due to outdoor air pollution occur in rural areas. Another problem with the current approach is that the estimates for urban $PM_{10}$ come from an econometric model (Global Model of Ambient Particulates, or GMAPS) that estimates $PM_{10}$ concentrations at the city level using data on fuel consumption at the country level. This model is estimated using data from 98 of the 196 countries for which $PM_{10}$ projections are made. Few observations are available for Latin America and the Caribbean (LAC), the Middle East and North Africa (MENA), or Sub-Saharan Africa (SSA).[1] Some of the model's predictions for African countries are clearly suspect. Botswana is estimated to be the second most polluted country in the world, and urban areas in Senegal and Zimbabwe are estimated to have higher particulate concentrations than urban areas in China.

Current estimates of health damages follow the approach set forth in Cohen et al. (2004). The concentration-response functions used to estimate adult premature mortality associated with particulate concentrations are based on the American Cancer Society study of Pope and colleagues (2002). Following Cohen et al. (2004), GMAPS converts $PM_{10}$ estimates to estimates of $PM_{2.5}$, the pollutant associated with mortality by Pope et al. (2002). This literature is, however, changing fast. In the 2010 Global Burden of Disease (GBD) study (Lim et al. 2012), estimates of mortality and morbidity attributable to air pollution are based on a meta-analysis of eight studies (Burnett et al. 2014). The 2013 Global Burden of Disease will incorporate additional studies.

We believe that it would be advisable for the World Bank to use estimates of population exposures to outdoor air pollution and estimates of the health effects of air pollution produced by the GBD team at the Institute for Health Metrics and Evaluation (IHME) rather than producing estimates using the current approach. This would have several advantages. Estimates of air pollution health effects published in the GBD are based on estimates of global population exposures to $PM_{2.5}$. This is accomplished by combining satellite measurements of aerosol optical depth (AOD) with atmospheric chemistry models to produce estimates of annual average $PM_{2.5}$ at a 10 km × 10 km resolution for all countries in the world (Brauer et al. 2012). Within-sample

[1] Only 1.6 percent of observations come from LAC, 0.6 percent from MENA, and 0.1 percent from SSA.

comparisons of $PM_{2.5}$ estimates from the GBD with monitored data produce average percentage errors that are comparable to within-sample errors from GMAPS. GBD estimates, however, have the advantage of global coverage. GBD estimates of health effects are produced for exposure to ozone as well as $PM_{2.5}$ and are updated to reflect the rapidly changing epidemiological literature. Use of these figures by the World Bank will guarantee that the Bank is publishing results that reflect the state of the art in both exposure measurement and epidemiology.

The GBD team plans to update estimates of population exposures to $PM_{2.5}$ and ozone and corresponding health effects at least every three years. Health impacts are reported as (a) premature deaths; (b) years of life lost (YLLs); and (c) disability-adjusted life years (DALYs) associated with $PM_{2.5}$ and ozone.[2] Estimates of $PM_{2.5}$ and ozone exposure are available at a 10 km ×10 km resolution. Both health impacts and exposure estimates could be reported by the World Bank at the country level.[3] For example, exposure could be described as the percentage of a country's population exposed to various annual average $PM_{2.5}$ levels.

The health damages associated with outdoor air pollution are currently monetized for inclusion in ANS using a value per DALY. This is calculated by dividing a $3.6 million (1990 US$) value of a statistical life (VSL) by 22 DALYs lost and is interpreted as representing willingness to pay to avoid a DALY by a person in the United States.[4] It is converted to current dollars using the US gross domestic product (GDP) deflator. Willingness to pay (WTP) to avoid a DALY is estimated for other countries by multiplying the US figure by the ratio of the country's per capita GNI to GNI per capita for the United States, measured at market exchange rates.

We believe that the current approach to monetization is basically sound and should be continued. Going forward, if the GBD estimates are used, most of the DALYs associated with outdoor air pollution will be life years lost, rather than years lost due to disability (YLD).[5] We recommend using the current approach to valuation, which is equivalent to the value of a statistical life year (VSLY) approach. For reasons explained more fully below, we believe that

[2] Both mortality and morbidity impacts are reported for $PM_{2.5}$. Only premature mortality is reported for ozone.

[3] The IHME website reports health impacts at the country level. Colleagues at IHME have indicated that exposure data is also available at the country level.

[4] It is estimated that the average person in US studies used to estimate the VSL has approximately 22 DALYs remaining.

[5] In the 2010 GBD, 94 percent of DALYs are years of life lost (YLLs) rather than YLDs.

this is preferable to applying a constant value per statistical life (VSL) to people of all ages. Over the longer run, we advise the World Bank to revisit the methods it uses to value mortality risks and to apply them consistently across all its operational work.

The remainder of the paper is organized as follows. Section II describes the current procedures used to calculate the monetary value of air pollution damages in the ANS. Section III describes how population exposures to $PM_{2.5}$ and ozone are measured in the GBD, and section IV describes how health effects are computed. In section V, we discuss various approaches to monetizing health damages and comment on the current approach. Section VI discusses the feasibility of including other pollutants and other damage categories in the ANS.

## II. Summary of Current Approach to Measuring PM Exposure and Monetizing Health Damages

### *A. Exposure Measurement Using the GMAPS Model*

Population exposure to $PM_{10}$ is currently estimated using GMAPS, a regression model that predicts annual average $PM_{10}$ concentrations at the city level using information on population, income level, economic activity, energy mix, climate, and geomorphology.[6] The model explains the logarithm of annual average $PM_{10}$ concentration in city *i* in year *t* as a function of (a) country-level per capita energy consumption by fuel type, population density, and per capita GDP in year *t*; (b) city-level population, population density, and GDP/km$^2$ in year *t*; (c) time-invariant meteorological variables, including temperature and topography; (d) city-specific time trends; and (e) country fixed effects. (The appendix contains a formal description of the model.) The model is estimated using data from 98 countries. Because model parameters are used to project $PM_{10}$ concentrations for 196 countries, it is necessary to estimate country fixed effects (i.e., country-specific intercepts) for countries not used to estimate the model. This is accomplished using a second equation that regresses country fixed effects from the first equation on the variables in list (a), averaged over the sample period. This equation is used to predict country-specific intercepts for each of the 98 countries not used estimating the model.

The model is currently estimated using data from 1,195 cities in 98 countries, covering the period from 1985 through 2011. Table A1 in the appendix lists the sources of data used to

[6] The GMAPS model, as originally developed by Pandey et al. (2006), is described in detail in Cohen et al. (2004). It was used to estimate population exposure to PM in GBD 2000.

estimate the model. Table 1 below shows the sources of data by World Bank region. More than half of the observations used to estimate the model come from cities in North America and Western Europe. Cities in East Asia and the Pacific (primarily China) constitute approximately one-quarter of the observations. As noted above, a very small fraction of the total sample observations comes from LAC, MENA, and SSA.

**Table 1. Geographic Distribution of $PM_{10}$ Measurements Used to Estimate GMAPS**

| Region | Percentage of total observations, 1985 to 2011 | Percentage of observations reported in 2005 or later |
|---|---|---|
| East Asia & Pacific (EAP) | 24.2 | 30.1 |
| Europe & Central Asia (ECA) | 12.4 | 14.9 |
| Latin America & Caribbean (LAC) | 1.6 | 0.9 |
| Middle East & North Africa (MENA) | 0.6 | 0.7 |
| North America (NA) | 16.3 | 11.4 |
| South Asia (SA) | 7.7 | 8.1 |
| Sub-Saharan Africa (SSA) | 0.1 | 0.15 |
| Western Europe (WE) | 36.9 | 33.8 |
| Total observations | 8,335 | 5,234 |

The within-sample fit of the model is good. Table 2 compares model predictions for the most recent year for which data are available with actual monitoring data for cities in the WHO monitoring network.[7] The average percentage error in estimating annual average $PM_{10}$ is 7.3 percent unweighted and 20 percent when weighted by $PM_{10}$ concentrations. Model fit improves when model residuals are used in predicting PM concentrations. What cannot be measured is how well the model predicts ambient concentrations in the 2,000 cities and 98 countries for

[7] Because the model was estimated using all available monitoring data, data from these cities were used in estimating the model, so no out-of-sample predictions have been made.

which monitoring data are unavailable.[8]

**Table 2. Within-Sample Comparison of GMAPS Predictions Using Data from Cities in the WHO Monitoring Network**

| Prediction errors | ANS14 (*not* including residuals) | ANS14 (including residuals) |
|---|---|---|
| Overall | | |
| RMSE | 465 | 192 |
| Average error (%), weighted by concentration | –0.8% | 1.9% |
| Average error (%), unweighted | 7.3% | 5.4% |
| Average magnitude of error (% over or under), weighted by concentration | 20.0% | 10.1% |
| WHO-monitored cities for which GMAPS predictions are available | 653 | 653 |
| Total population of WHO-monitored cities for which GMAPS predictions are available | 788,732,237 | 788,732,237 |
| Population-weighted error ($\mu g/m^3$) | –2.0 | –1.9 |
| Population-weighted error (%) | 3.2% | 0.7% |

### *B. Evaluation in Terms of Level of Coverage, Update Frequency, Quality of Data, and Errors in Estimation*

The GMAPS model performs well in explaining variation in annual average $PM_{10}$ for cities for which monitoring readings are available. The model was originally estimated using data for the period 1985–1999 (Pandey et al. 2006). These estimates of model parameters were used to predict $PM_{10}$ concentrations for use in the GBD 2000 (Cohen et al. 2004). The model has recently been estimated by Chris Sall (2013) using updated population and more recent monitoring data. As noted above, within-sample fit of the model is good; however, the model is being used to project $PM_{10}$ readings for more than 2,000 cities and 98 countries for which no monitoring data exist.

[8] Out-of-sample predictions cannot be compared with actual monitoring data since the model is currently estimated using all available monitoring data. Out-of-sample predictions using the original GMAPS model, estimated using data from 1985 to 1999, are described in Cohen et al. (2004).

Many of the model's predictions are problematic. For example, GMAPS predicts that Botswana, Senegal, and Zimbabwe are among the 13 most polluted countries in the world—more polluted than cities in China. In the case of Botswana and Senegal, this occurs because there is only one observation for each country among the 8,300 observations in the dataset. With country fixed effects, the model will always use these observations to predict $PM_{10}$ in each country.[9] Basing a forecast for a country on a single observation is less than desirable. Zimbabwe is an out-of-sample prediction. How reasonable the model's out-of-sample predictions are can be gauged by comparing them with estimates from other sources.

A comparison with Brauer et al. (2012) suggests that GMAPS does not predict well in regions not used to estimate the model. Urban areas in Zimbabwe are predicted to be more polluted than urban areas in China by GMAPS but not by Brauer and colleagues. Furthermore, Brauer et al. estimate $PM_{2.5}$ concentrations in Botswana and Senegal to be in the neighborhood of 2–5μg/$m^3$ and 20–40μg/$m^3$, respectively.[10]

The main shortcoming of the GMAPS model is, however, its limited coverage of the world's population. Restricting the analysis to cities with populations over 100,000 fails to include the more than 5 billion people who reside in smaller cities and rural areas. It would be more informative to estimate the percentage of people in each country exposed to various levels of air pollution, as is currently being done in exposure estimates prepared for the GBD (Brauer et al. 2012). Moreover, the model measures exposure to $PM_{10}$, whereas it is fine particles ($PM_{2.5}$) that have consistently been linked to serious health effects.

### *C. Calculation of Health Impacts Using GMAPS*

The health damages from outdoor air pollution are calculated using a generalization of the approach outlined in the 2000 Global Burden of Disease study (Cohen et al. 2004). Estimates of the burden of disease from outdoor air pollution are based on mortality and morbidity from cardiopulmonary disease (CPD) and lung cancer in adults and acute respiratory infections (ARI) in children five years or younger. Attributable deaths and years of life lost (YLLs) for adults are estimated using risk coefficients from a large US cohort study of adults (Pope et al. 2002) and a meta-analysis of five time-series studies of ARI mortality in children. Premature deaths and

[9] The observations are a $PM_{10}$ level of 216 μg/$m^3$ for Botswana (2005) and a $PM_{10}$ level of 146 μg/$m^3$ for Senegal (2010). There are 17 countries with only one observation in the dataset.

[10] $PM_{10}$ concentrations would be approximately twice as high.

years of life lost due to disability (YLDs) are calculated for each disease and each country by combining the air pollution estimates with the corresponding dose-response coefficients taken from the GBD project, baseline health data from the World Health Organization (WHO), and United Nations (UN) demographic data. Since Pope et al. (2002) find premature mortality to be related to exposure to $PM_{2.5}$, a conversion factor of 0.5 is applied to derive $PM_{2.5}$ concentrations from $PM_{10}$ estimates.[11]

Because baseline disease data are not available at the city level, average population-weighted urban $PM_{10}$ concentrations are calculated for each country, and estimates of deaths and YLDs due to ARI, COPD, and lung cancer are calculated for each demographic group using population data for urban areas covered by GMAPS. Using a conversion table that relates deaths at a given age to YLL, the corresponding disability-adjusted life years (DALYs) are calculated for each demographic group and health outcome. These are added to obtain the total number of DALYs per year lost in each country due to anthropogenic air pollution.

### *D. Evaluation in Terms of Update Frequency and Correspondence with International Practice*

As noted above, the calculation of DALYs in GMAPS follows the protocols used for GBD 2000 (Cohen et al. 2004). The calculation of DALYs in GBD 2010 differs, however, in several respects from the GBD 2000 protocol. DALYs are now calculated based on estimates of world population exposure rather than exposure of urban populations in cities of 100,000 or more. This has resulted in much larger estimates of the health burden associated with outdoor air pollution: 3.2 million estimated deaths in GBD 2010 compared with 800,000 deaths in GBD 2000. GMAPS, in contrast, estimates approximately 1.66 million deaths due to air pollution in 2010.Additionally, in calculating years of life lost per death, life years lost are no longer age weighted or discounted.[12] This has increased the average number of life years lost per death. Finally, the concentration-response functions used to estimate health impacts have been updated to reflect advances in the epidemiological literature.

[11] Cohen et al. (2004) estimate that, worldwide, the average ratio of $PM_{2.5}$ to $PM_{10}$ is approximately 0.5.

[12] The practice of weighting years lost due to death and disability was an arbitrary one (i.e., based on the opinions of experts), as was discounting life years lost at a rate of 3 percent.

### *E. Valuation of Health Impacts in GMAPS*

In GMAPS, health impacts from outdoor air pollution are monetized by multiplying aggregate DALYs by an appropriate dollar value per DALY to yield economic damages due to air pollution in the urban areas of each country. DALYs are valued using a value of a statistical life (VSL) for the United States of $3.6 million (1990 US$).[13] This is converted to current dollars using a GDP deflator; for example, the VSL is $5.64 million in 2010 US$. Assuming 22 DALYs per life at risk in the studies that led to the US VSL estimate, the value of a statistical DALY is estimated to be $3.6 million/22, or $163,636 (1990 US$).

The value of a DALY is estimated for other countries by multiplying the US estimate by the ratio of per capita GNI for the country in question by US per capita GNI. Since health impacts are expressed as a percentage of GNI at market exchange rates, calculations are made using per capita GNI calculated using market exchange rates.[14] In the ANS, total damages are expressed as a percentage of a country's GNI.

### *F. Evaluation in Terms of International Practice*

Despite the criticism of placing a monetary value on DALYs, we believe that the current GMAPS practice is defensible. The criticism of monetizing DALYs (or quality-adjusted life years, QALYs) is that DALYs (QALYs) evaluate health outcomes by the time spent in various health states, independently of an individual's income or wealth (Hammitt 2002). In practice, however, DALYs (QALYs) are often monetized, implying that people are willing to trade money for health risks.[15] When this is done, the approach is to use the value placed on risk of death (the VSL) and using it to calculate the value of a statistical life year (VSLY).

The VSL is based on the idea that individuals will trade income for small changes in their risk of dying; in fact, this implicitly occurs in labor markets when workers receive compensating wage differentials for working in riskier jobs. The VSL is the sum of what individuals would pay

[13] The source of this figure is unclear in the original GMAPS documentation (Pandey et al. 2006).

[14] While in many cross-country comparisons, health impacts are valued in purchasing power parity terms, DALYs are valued in the ANS at market exchange rates, since the figures are reported as a percent of GNI, which is also measured at market exchange rates.

[15] The literature on risk–risk trade-offs (Krupnick and Cropper 1992) provides a possible link between the two approaches. In this literature, respondents trade more of one risk (e.g., risk of chronic bronchitis) for less of another (e.g., risk of death in an auto accident). The researcher then values changes in the risk of chronic bronchitis using the VSL.

to avoid mortality risks that sum to one (statistical) life lost. The VSLY is computed by dividing the VSL by the average discounted remaining life expectancy of the persons whose choices led to the empirical estimate of the VSL.[16] How the VSLY is calculated is also controversial (Krupnick 2007), as we discuss in section IV; however, the practice is widely used in Europe and the United States to value mortality risks. In the GBD 2010, 94 percent of the DALYs associated with air pollution are life years lost. Use of a VSLY to value DALYs is therefore in keeping with international practice.

## III. Exposure Measurement and Calculation of Health Impacts in the GBD

### *A. Exposure Measurement in the GBD*

GBD 2010 uses satellite-based estimates of $PM_{2.5}$ in combination with simulations from the TM5 chemical transport model to generate global estimates of long-term average concentrations of fine particles and ozone at a 0.1° × 0.1° spatial resolution (Brauer et al. 2012; Lim et al. 2012). Estimates are made for 1990 and 2005, and results are extrapolated to 2010 using the 1990 to 2005 trend. We briefly describe the TM5 and satellite sources of data and how they were combined to estimate population exposures.

TM5 is a nested, three-dimensional global atmospheric chemistry transport model that simulates ozone and aerosol components at a 1° × 1° resolution. The TM5 model uses meteorological data from the European Centre for Medium-Range Weather Forecasts (ECMWF; six hours Integrated Forecasting System [IFS] forecast). The model runs for 1990 and 2005 use meteorological data from 2001, considered to be a representative period. In addition to meteorological data, key inputs to TM5 are global gridded emissions of chemically active species such as $SO_2$, $NO_x$, and VOCs, as well as PM, at a spatial resolution of 1° × 1°. The 1990 and 2005 model runs used emissions inventories from the International Institute for Applied Systems Analysis (IIASA) Greenhouse Gas and Air Pollution Interactions and Synergies (GAINS) model.

[16] Remaining life expectancy is discounted because it is assumed that individuals discount future monetary rewards. As explained in section V below, the VSLY assumes that each year of life is equally valuable in monetary terms, and that the individual discounts this value by his probability of surviving to each future year, as well as by the rate of interest.

To better capture urban exposure to $PM_{2.5}$, a subgrid parameterization is applied to redistribute concentrations among urban and rural areas, while maintaining the average concentration of the grid cell. This is accomplished using a high-resolution (0.042° × 0.042°) population dataset to subdivide the 1° × 1° grid in 24 × 24 subgrids. Population density is used as a proxy to identify high emission areas within each 1° × 1° grid cell. If the population density exceeds 600/km$^2$, the subgrid is labeled as "urban." Otherwise, it is labeled "rural." Once the model is resolved, the TM5 1° × 1° fields are interpolated to a 0.1° × 0.1° resolution, and for the relevant grid cells, the urban and rural $PM_{2.5}$ are assigned to the respective subgrid cells based on population density.

The time resolution of the output from TM5 is matched to the definition of the exposure metrics: for PM, monthly means are stored and averaged annually. Ozone is stored as hourly means for the calculation of exposure metrics based on daily maximum levels.

The satellite-based approach (SAT) combines measures of aerosol optical depth (AOD) from multiple satellite instruments and converts them to estimates of ground-level $PM_{2.5}$ concentrations using the Goddard Earth Observing System (GEOS) chemical transport model GEOS-Chem, v8-01-04. The Moderate Resolution Imaging Spectroradiometer (MODIS) and Multi-angle Imaging SpectroRadiometer (MISR), on board NASA's Terra satellite, provide measurements of aerosol optical death, a measure of light extinction by aerosols in the earth's atmospheric column.[17] The relationship between AOD measurements and surface air quality depends on the composition, size distribution, and water composition of atmospheric aerosol.

SAT estimates of $PM_{2.5}$ use the GEOS-Chem model to simulate the relationship between AOD ground-level concentrations of $PM_{2.5}$. GEOS-Chem simulates transport of directly emitted $PM_{2.5}$ and secondary particles at a time step of 15 minutes using meteorological datasets, emissions inventories, and equations representing the physics and chemistry of the atmosphere. Global emissions for 2005 are based on the Emission Database for Global Atmospheric Research (EDGAR) emissions inventory, supplemented by additional data sources. The ratio of $PM_{2.5}$ to AOD ($\eta$) is estimated at a resolution of 2.5° × 2.5° using GEOS-Chem, and then interpolated to a 0.1° × 0.1° resolution for use in the GBD. Satellite-derived $PM_{2.5} = \eta \cdot AOD$.

[17] AOD = –ln($x$), where $x$ is the fraction of light not scattered by aerosols. A value of $x$ = 0.9 implies an AOD of 0.1, which would correspond to a sunny day. Annual average values of AOD for the United States lie in the range of 0.1–0.15.

Satellite-derived and TM5 estimates are averaged at a 0.1° × 0.1° grid cell resolution. For grid cells where data from surface monitors with concentrations exceeding 10 μg/m$^3$ are available, the average of the SAT and TM5 estimates (AVG) is entered into a regression model with the surface monitoring value as the dependent variable. From this model, a prediction equation, $PM_{2.5} = PM2.5 = 1.32 \cdot AVG^{0.922}$, is derived and applied to the AVG values to produce the final concentration estimate for each grid cell.[18] Thus AVG values are calibrated to the available ground-level measurements. Population weighting is conducted using the 1990 and 2005 Gridded Population of the World database. These methods are discussed in further detail in Brauer et al. (2012, Supporting Information). For the 2010 GBD, 2005 TM5 and satellite-based measurements were extrapolated to 2010 based on the 1990–2005 trend line. It was assumed that the change from 2005 to 2010 relative to 2005 was one-third as large as the change from 1990 to 2005 relative to 1990.

According to IHME, a similar approach will be adopted for the 2013 GBD (Cohen 2014), which will include year-specific satellite-based estimates of $PM_{2.5}$ for 2000–2012 (three-year averages of retrievals centered on the year of interest) and an improved estimation algorithm. For 1990 and 1995, the estimates from 2000 will be scaled back using emissions ratios. New TM5-FASST (reduced-form version of TM5) simulations for 1990, 2000, and 2010 will be included using an updated set of emissions and meteorological inputs. Similarly for ozone measurement, the 2013 GBD will use TM5-FASST simulations for 1990, 2000, and 2010, as well as interpolated values for 1990 and 2000 and extrapolated values for 2013.

### *B. Evaluation in Terms of Level of Geographic Coverage, Update Frequency, Quality, and Availability of Data*

The GBD estimates are global in nature and are produced at fine (0.1° × 0.1°) spatial resolution. They could be presented at the country level as population-weighted averages or by describing the percentage of the population exposed to different levels of $PM_{2.5}$, as in van Donkelaar et al. (2010).

An effort is being undertaken by the GBD authors to update measurement data for 2010–2013 using a variety of sources described in Brauer et al. (2012), as well as new data from China and India. Regression calibration approaches will be used to combine satellite-based estimates,

[18] The equation is estimated using only observations above 10 μg/m$^3$.

TM5-FASST simulations, and ground-level measurements to produce final global estimates at a 0.1° × 0.1° grid cell resolution. Beginning with estimates for the year 2013, the GBD will be updated on an annual basis. This will incorporate updates to the exposure estimates, including more recent ground-level measurement data.

Estimates of population-weighted exposure to $PM_{2.5}$ at the country level will be made available to the World Bank. Estimates of deaths, YLLs, and DALYs at the country level are publicly available on the IHME website.

### *C. Quantification of Errors in Estimating Exposure*

As in the case of GMAPS, monitoring data is used to calibrate estimates of PM rather than to validate the model. Table 3 lists the number of monitoring stations by region used to estimate the regression model that combines TM5 and SAT estimates of $PM_{2.5}$ to produce estimates of population exposures. Within-sample errors from the regression model, measured as the ratio of prediction error to predicted value, range from 8.3 to 15.9 percent for concentrations above 10 $\mu g/m^3$.

In terms of model validation, estimates of $PM_{2.5}$ produced by combining satellite measurements of AOD with GEOS-Chem have been validated against monitoring data (van Donkelaar et al. 2010). As stated by van Donkelaar and colleagues, "Our evaluation of the satellite-derived estimate with ground-based *in situ* measurements indicates significant spatial agreement with North American measurements ($r$ = 0.77; slope = 1.07; $n$ = 1057) and with noncoincident measurements elsewhere ($r$ = 0.83; slope = 0.86; $n$ = 244)." To our knowledge, predictions from TM5 have not been compared with ground-level monitoring data; however, the TM5 model has been compared with other CTMs to see if it produces similar results, and individual components of the model have been compared against observational data (Brauer et al. 2012, Supporting Information).

**Table 3. Regional Distribution of Ground-Based PM Monitoring Stations in the PM Measurement Database (Brauer et al. 2012, Supporting Information)**

| Region | Measured $PM_{2.5}$ | $P_{2.5}$ Estimated from $PM_{10}$ |
|---|---|---|
| Asia Pacific, high income | 2 | 5 |
| Asia, East | 2 | 115 |
| Asia, South | 3 | 18 |
| Asia, Southeast | 9 | 7 |
| Australasia | 23 | 0 |
| Europe, Central | 26 | 0 |
| Europe, Eastern | 0 | 0 |
| Europe, Western | 141 | 0 |
| Latin America, Central | 3 | 16 |
| Latin America, Southern | 0 | 5 |
| Latin America, Tropical | 1 | 0 |
| North Africa/Middle East | 2 | 7 |
| North America, high income | 263 | 23 |
| Sub-Saharan Africa, Southern | 0 | 7 |
| Sub-Saharan Africa, Western | 0 | 1 |

## IV. Calculation of Health Impacts in the GBD

### *A. Description of Current Methodology, Including Health Endpoints Covered*

Risks associated with exposure to $PM_{2.5}$ and ozone are estimated using methods described in Burnett et al. (2014) and Lim et al. (2012). These studies develop relative risk (RR) functions over the entire global range of ambient $PM_{2.5}$ for five leading causes of death: ischemic heart disease (IHD), cerebrovascular disease (stroke), chronic obstructive pulmonary disease (COPD), lung cancer (LC), and acute lower respiratory infections (ALRI) in children ages 0–4.

The risk functions are then used to estimate mortality and life years lost.[19] New cohort studies will be added to the integrated exposure response (IER) functions in the 2013 GBD in order to include risk estimates from other sources of $PM_{2.5}$ such as secondhand smoke (SHS), household air pollution (HAP), and active smoking (AS). Adding these sources can better capture the shape of the exposure-response curve at higher ambient concentrations. SHS, HAP, and AS exposures are converted to estimate annual $PM_{2.5}$ exposure equivalents using inhaled doses of particle mass.

Epidemiological studies conducted in the United States have reported the association of ambient average concentrations of $PM_{2.5}$ (approximately 5–30 $\mu g/m^3$) with increased mortality from heart and lung disease (Chen et al. 2008; EPA 2009). However, there is little information on the association of long-term exposure to $PM_{2.5}$ with mortality from chronic cardiovascular and respiratory disease in areas where ambient concentrations exceed 100 $\mu g/m^3$, such as in South and East Asian cities (Health Effects Institute 2010; Brauer et al. 2012). While previous efforts to estimate the global burden attributable to ambient air pollution assume that risk functions for cardiopulmonary mortality can be extrapolated linearly in RR from 7.5 to 50 $\mu g/m^3$ (Cohen et al. 2004), the recent literature does not restrict the relationship to a linear function over the global range of exposure.

Burnett et al. (2014) assume that RR must be equal to 1 when $PM_{2.5}$ concentrations are below some threshold that reflects a counterfactual low exposure, and that RR increases monotonically with exposure. Although the authors assume that the RR changes with the magnitude of exposure, they also assume that, at a specific exposure level, toxicity is largely equivalent among all sources and temporal patterns. Motivated by the results of Pope et al. (2011), the IER can take on various shapes and is modeled as follows:

$$\mathrm{RR}_{\mathrm{IER}}(z) = \begin{cases} 1, & z < z_{cf} \\ 1 + \alpha(1 - e^{-\gamma(z - z_{cf})^{\delta}}), & z \geq z_{cf} \end{cases}$$

where z is exposure to $PM_{2.5}$ measured in $\mu g/m^3$ and $z_{cf}$ is the counterfactual exposure below which there is no risk of death from exposure to air pollution. A power of $\delta$ is included to predict risk at higher concentrations. $\gamma$ can be interpreted as the ratio of RR at low to high exposures.

[19] Relative risks from mortality studies are in some cases used to predict the relative risk of morbidity from stroke and ischemic heart disease.

The parameters α, γ, and δ are estimated using nonlinear regression methods, where the set of RR estimates is weighted by the inverse of the estimated variance of the logarithm of the RR to reflect uncertainty.

For the 2013 GBD, minor modifications are made to the methodology adopted in Burnett et al. (2014). The theoretical minimum risk exposure distribution (TMRED) for $PM_{2.5}$ is recalculated by excluding three cohort studies whose minimum exposure concentration was greater than 8.8 μg/$m^3$. It is believed that these studies do not provide clear information on the association between mortality risk and exposure at lower concentrations. Four more studies have been added to the list of cohort studies that are used to fit the risk models.

Population attributable fractions (PAFs) associated with ambient $PM_{2.5}$ exposure are calculated for each of the 187 countries included in the 2010 GBD. Using the surface-level $PM_{2.5}$ concentrations estimated on a global scale at a resolution of 0.1° × 0.1° in Brauer et al. (2012), Burnett et al. (2014) are able to predict an RR for each grid cell within a country. A population-weighted attributable fraction (*PAF*) is then constructed for each country *i*:

$$PAF_i = 1 - \frac{1}{\sum_{j=1}^{J_i} w_{ij} RR_{IER}(z_{ij})}$$

where $z_{ij}$ is the $PM_{2.5}$ concentration in the $j^{th}$ of $J_i$ cells in the $i^{th}$ country, and $w_{ij} = p_{ij}\left(\frac{1}{\sum_{j=1}^{J_i} p_{ij}}\right)$ with $p_{ij}$ the population in the $j^{th}$ cell of the $i^{th}$ country.

The authors find that the proportion of deaths due to ambient air pollution exposure (*PAF*) varies across countries from 2 to 41 percent for IHD, less than 1 to 21 percent for COPD, 1 to 43 percent for stroke, less than 1 to 25 percent for LC, and less than 1 to 38 percent for ALRI.

### *B. Evaluation in Terms of Geographic Coverage, Update Frequency, Quality and Availability of Data*

Despite the lack of long-term cohort studies for chronic cardiovascular and respiratory disease in East and South Asia and the Middle East, where ambient concentrations are significantly higher and where the burden of disease attributable to specific sources of air pollution differs from North America and Europe, the IER model adopted for the 2013 GBD attempts to derive the shape of the exposure response curve at higher ambient concentrations by

incorporating the RR of mortality from exposure to second-hand smoke, household air pollution and active smoking.

Burnett et al. (2014) compare the ability of the IER model to estimate RR in areas of high levels of ambient air pollution by comparing it to Cao et al. (2011), who report increased risk of mortality from cardiovascular and respiratory disease from long term exposure to TSP in 71,000 residents in 31 Chinese cities. Cao et al. (2011) use a 3/1 ratio to convert TSP to PM2.5 based on current and historical Chinese data. By calculating the geometric averages of the three RRs between consecutives quartiles of PM2.5 concentrations for both cohorts, Burnett et al. find that the RRs of the Chinese cohort and those predicted by the IER model are similar for IHD, stroke and LC, suggesting that the IER model produced reasonable predictions of RR at higher PM2.5 concentrations than are typically observed in North America and Europe. However, the authors note that, compared to PM2.5, TSP is a poor predictor of cardiovascular mortality in U.S. cohort studies. There is also uncertainty regarding the temporal and spatial validity of the TSP/PM2.5 conversion ratio of 3/1.

Additional uncertainties may exist due to scarce information on exposure from specific sources such as second hand smoke (Pope et al. 2011), misclassification and differences in the duration of exposure among sources. These uncertainties can be minimized by improving the precision of the actual estimates of RR from the epidemiological literature used to develop the IER model.

In terms of update frequency, the GBD recognizes that the risk measurement literature is changing quickly. With each successive study, the team is updating the cohort studies that are used in the IER model based on the following criteria: (i) exposure estimation based on measured and modeled PM2.5, (ii) exposure estimates based on a scale consistent with the GBD estimates (i.e. 10km X 10km resolution), and (iii) controls for potential confounders. The team added four cohort studies to the 2013 GBD based on these criteria.

## V. Monetization of Health Impacts

### *A. What Is to Be Balued*

Estimates of the health effects of air pollution include estimates of lives lost, by age and country, as a result of exposure to air pollution. Estimates are also made of new cases of

cardiovascular disease associated with air pollution exposure, by age and country.[20] In the GBD, estimates of lives lost are converted to years of life lost (YLLs) by calculating the remaining life expectancy of each statistical life lost.[21] Historically, life years lost were assigned age weights and discounted using an annual discount rate of 3 percent. This is no longer being done, beginning with GBD 2010. YLDs associated with cases of cardiovascular disease are estimated based on age at onset and DALY weights, which describe the fraction of a healthy year lost due to illness.[22] Historically, age weighting and discounting were used in computing DALYs; however, this is no longer the case.[23]

The GBD 2010 estimates that over 3 million lives and over 68 million DALYs were lost due to exposure to outdoor air pollution in 2010. Ninety-four percent of the DALYs are YLLs associated with premature mortality. When, historically, age weighting and discounting were used in constructing YLLs and DALYs, YLDs constituted a much higher fraction of DALYs. Because the vast majority of DALYs associated with outdoor air pollution are life-years lost, we focus on valuing lives and life-years lost. Table 4 indicates the percentage of deaths, DALYs, YLLs and YLDs attributable to ambient PM pollution in the 2010 GBD, by age group.[24]

### *B. Approaches to Valuing Lives and Life-Years Lost*

In benefit-cost analyses of health and safety regulations conducted in the United States and the European Union, and in many other countries around the world, mortality risks are valued using the amount that people are willing to pay to reduce these risks. Typically, the amounts that people will pay for small risk reductions that sum to one statistical life are termed the Value per Statistical Life (VSL). To illustrate, if each of 10,000 people were willing to pay $500 per year to reduce their annual risk of dying by 1 in 10,000, we would say that the Value

[20] As noted in footnote 18, RRs for mortality are also used to estimate cardio-vascular morbidity, which is expressed in terms of years lost due to disability (YLDs).

[21] These calculations are made using Japanese life tables, rather than country-specific life tables. They are therefore likely to overstate estimates based on country-specific life tables.

[22] Information on DALY weights is available at http://www.who.int/healthinfo/global_burden_disease/GBD2004_DisabilityWeights.pdf?ua=1

[23] Life years lost due to illness and premature mortality were originally assigned age weights and discounted. For example, a year lost at age 60 was considered equal to 0.X of a year lost at age 25. Future life years were discounted at 3%. Both procedures were arbitrary: age weights were determined by a panel of experts; and, discounting, while appropriate for monetary values, is not necessarily appropriate for discounting future life years.

[24] Note that no deaths or DALYs are attributed to outdoor air pollution for persons 5-14.

per Statistical Life is $5,000,000 (= $500 x 10,000).

**Table 4. Percentage of Deaths, DALYs, YLLs and YLDs Attributable to Ambient PM Pollution in the 2010 GBD by Age Group**

| Age group | Deaths | DALYs | YLLs | YLDs |
|---|---|---|---|---|
| 1-4 years | 1.2 | 4.7 | 4.8 | 2.3 |
| 15-49 years | 9.5 | 21.4 | 20.7 | 33.5 |
| 50-69 years | 35.5 | 45.7 | 45.8 | 42.9 |
| 70+ years | 53.8 | 28.2 | 28.6 | 21.3 |
| | 100.0 | 100.0 | 100.0 | 100.0 |

### *C. The VSL*

The economic theory behind the VSL is the lifecycle consumption-saving model with uncertain lifetime (Yaari 1965) which has been adapted to analyze mortality risk valuation by Shepard and Zeckhauer (1982, 1984), Cropper and Sussman (1990), Cropper and Sahin (2009) and others. The amount that a rational person would pay to reduce his risk of dying should reflect the enjoyment he will receive over the remainder of his lifetime. This, in turn, should depend on his current age and remaining life expectancy, his expected consumption over the remainder of his lifetime and factors that affect his utility of consumption.

According to the lifecycle model, the amount an individual would pay today (e.g., at age *40*) to reduce his probability of dying over the coming year, $D_{40}$, is equal to the product of the rate at which he is willing to trade wealth $W$ for a change in $D_{40}$, which is termed the Value per Statistical Life, $VSL_{40}$, times the size of the change in $D_{40}$, $WTP_{40} = VSL_{40}dD_{40}$.

When the individual can borrow and lend at the riskless rate of interest $r$ the VSL is proportional to discounted utility of consumption over the remainder of the individual's life, where $q_{40,t}$ is the probability of surviving to age t, conditional on being alive at age *40* and $U_t(C_t)$ denotes utility of consumption at age t,

$$VSL_{40} = \frac{1}{1-D_{40}} \sum_{t=41}^{T} q_{40,t}(1+r)^{40-t}\left[U_t(C_t)/U_t^{'}(C_t)\right]. \qquad (1)$$

Under certain simplifying assumptions, the theory has direct implications for how the VSL should vary with consumption and life expectancy. If we assume that utility of consumption is iso-elastic—a common assumption the Economics literature—and independent of age, i.e., $U_t(C_t) = C_t^{\beta}$, $1 \geq \beta \geq 0$, then the VSL simplifies to (2),[25]

$$VSL_{40} = \frac{1}{1-D_{40}} \sum_{t=41}^{T} q_{40,t}(1+r)^{40-t}\left[C_t / \beta\right]. \qquad (2)$$

The VSL is proportional to discounted expected utility of consumption, where the factor of proportionality, $1/\beta$, reflects the individual's risk aversion. For example, if $\beta = 0.2$, the VSL should equal five times the present value of discounted expected lifetime consumption.[26]

The VSL may be further simplified if annual consumption is approximately constant, i.e., $C_t = \overline{C}$ in which case the VSL is proportional to discounted remaining life expectancy, $\sum_{t=41}^{T} q_{40,t}(1+r)^{40-t}$,

$$VLS_{40} = \frac{1}{1-D_{40}} \overline{C}\beta^{-1} \sum_{t=41}^{T} q_{40,t}(1+r)^{40-t}.^{27} \qquad (3)$$

Equation (3) implies that the VSL should vary in proportion to consumption (which may, in turn, be proportional to per capita income), but that the VSL may also differ due to differences in risk preferences and discounted life expectancy.

### *D. The Value of a Statistical Life Year (VSLY)*

Equation (3) implies that the VSL is proportional to remaining life expectancy and, hence, that the VSL can be written as the product of discounted remaining life expectancy and a Value per Life Year Saved (VSLY) ($\overline{C} / \beta$).[28] This is a convenient assumption that is frequently

[25] The standard form for the iso-elastic (Constant Relative Risk Aversion) utility function must be modified in this case to make utility of consumption while alive greater than utility of death.

[26] This assumes that $1/(1\text{-}D_{40}) \approx 1$. Equation (2) also implies that the VSL is likely to exceed the present value of expected lifetime earnings, provided the individual is not a net borrower.

[27] It should be noted that, formally, (3) requires extreme assumptions ($r = \delta$ in the lifecycle model). However, it underlies the common practice (discussed below) of apportioning the VSL into a constant value per life year saved (VSLY).

[28] In practice, the VSLY is derived by taking the VSL from an empirical study and dividing the VSL by the discounted expected number of life years remaining for the average individual studied.

used in policy analysis, especially to value QALYs and DALYs. The empirical literature on the effects of age on WTP does not support the notion that WTP falls in proportion to remaining life expectancy, although it suggests that WTP does decline after some age. (Alberini et al. (2004), for example, find that the VSL declines by about 30 percent after age 70 in stated preference studies conducted in the US and Canada.) In spite of lack of empirical support for a constant VSLY, it is often used in practice (Robinson 2007).

### *E. Empirical Estimation of the VSL and VSLY*

Empirical estimates of the VSL most frequently come from hedonic wage studies, which estimate compensating wage differentials in the labor market, or from contingent valuation surveys in which people are asked directly what they would pay for a reduction in their risk of dying. There are many recent surveys of this literature, including Cropper, Hammitt and Robinson (2011) who discuss methodological issues in estimating the VSL using each approach and provide surveys of recent meta-analyses of the literature, focusing on estimates for the US.

The empirical VSL literature is expanding rapidly worldwide; however, it remains the case that there are many more studies in high-income countries than in high-middle-income or low-middle-income countries (using World Bank country definitions). To our knowledge, there is no published study in a low-income country. A recent OECD survey of stated preference (contingent valuation) studies (Braathen et al. 2009), that value mortality risks in the context of traffic safety, environment or health, reviews 18 studies in high-income countries, 4 in high-middle-income countries, 1 in low-middle income countries and 1 in low-income countries.[29] Viscusi and Aldy's (2003) review of wage-risk studies includes 18 studies in OECD and 5 studies in non-OECD countries outside of the US. What is clear is that the developing country literature at this point is not sufficiently mature to provide estimates for individual countries. This suggests transferring estimates from countries where better studies exist to countries for which there are no empirical estimates of the VSL.

### *F. The Standard Approach to Benefits Transfer*

Due to the limited number of empirical estimates of the VSL in developing countries, economists frequently use benefits transfer to estimate developing country VSLs. Most transfers

[29] Not all of these studies have been published.

are based on income differences between countries: As the lifecycle model suggests, the VSL should depend on consumption and, hence, on income. If risk preferences, discount rates and survival probabilities are the same in all countries, equation (3) implies that the VSL should be proportional to consumption/income. The standard approach to benefits transfer treats the ratio of the VSL to per capita income as constant across countries, and uses estimates of the VSL from high income countries to estimate the VSL in middle and low income countries. We discuss the implications of this approach and then ask whether the empirical evidence from mortality risk studies in middle income countries supports it.

The simplest approach to benefits transfer is illustrated by equation (4), which says that the VSL in India—measured in US dollars—equals the VSL in the US, multiplied by the ratio of per capita income in India ($Y_{India}$) to per capita income in the US ($Y_{USA}$). $\varepsilon$ is the income elasticity of the VSL. The equation assumes that preferences, e.g., attitudes

$$VSL_{India} = VSL_{USA} * (Y_{India}/Y_{USA})^{\varepsilon} \quad (4)$$

towards risk, are the same in both countries. When $\varepsilon = 1$, the equation implies that the ratio of the VSL to per capita income is the same in both countries:

$$VSL_{USA}/Y_{USA} = VSL_{India}/Y_{India} . \quad (5)$$

Equation (5) is consistent with the simplified version of the life cycle model (equation (3) above) if consumption is proportional to per capita income. The life cycle model implies that the VSL is proportional to current consumption *if people have the same discount rates, survival probabilities and the same risk preferences in both countries.*

But, discount rates, survival probabilities and risk preferences do differ among countries. Cropper and Sahin (2009) and Hammitt and Robinson (2008, 2011) both suggest that the VSL/Y ratio varies with income. They argue, based on what empirical evidence is available, that the VSL/Y ratio is likely to be lower at low incomes than at high incomes. This is consistent with the simplified life-cycle model: people who are less risk-averse, have higher discount rates and shorter life expectancies should have a lower VSL/Y ratio. Cropper and Sahin (2009) suggest, based on empirical evidence, that the VSL/Y ratio is approximately 140 in high-income countries and 80 in middle income countries, implying that the income elasticity of the VSL rises as incomes fall. Hammit and Robinson (2009, 2011) suggest using an income elasticity of 1.5, rather than 1, in transferring the VSL to countries in Sub-Saharan Africa.

### *G. Evaluation of Current GMAPS Procedures*

The approach currently taken in GMAPS is to use a VSL from the US of $3.6 million (1990 USD), which is divided by 22 to compute a value per DALY of $163,636 (1990 USD).[30] This value is updated to adjust for inflation using the US GDP price deflator and transferred to other countries assuming an income elasticity of 1. DALYs associated with PM in each region are multiplied by the transferred value per DALY. As noted above, this practice is generally in accordance with international practices. One criticism that could be made of current practice is that the baseline US VSL has not been adjusted for income growth over the period since 1990, only for inflation. Thus, the US VSL/Y ratio has fallen from 149 in 1990 to 114 in 2010. There are, however, countervailing factors—e.g., the use of an income elasticity of the VSL of 1—that suggest that the VSL may be over-estimated for some countries. On balance, we believe the current practice passes muster.

It should also be noted that the current approach transfers a VSL from the US to other countries using per capita GNI at market exchange rates. We believe that this is the correct approach. Equation (4) can be used in benefits transfer using either Y at market exchange rates or Y adjusted for purchasing-power parity (i.e., in International dollars).[31] If Y is measured in PPP terms, then so is the VSL. If the VSL for India is measured in International dollars, then it is imperative, in a cost-benefit analysis, that costs should also be measured in International dollars, rather than measuring costs using market exchange rates. Because PM damages are reported as a percent of GNI, measured at market exchange rates, VSL transfer should be conducted using market exchange rates. If it is not, the ratio of the VSL to income will be much greater than 1 in poor countries.[32]

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[30] Twenty-two years represents the number of DALYs lost by a 43-year-old male, using the Japanese life table, a discount rate of 3 percent and age weights; i.e., the approach used by WHO to calculate DALYs in the 2000 GBD. It is, coincidentally, the number of discounted life year lost by a 43-year-old male in the US using US life tables and a 3 percent discount rate.

[31] Note that per capita GNI for the US is the same whether measured in International dollars or at market exchange rates.

[32] To illustrate, the VSL for India is about 3 times greater in PPP dollars than when measured using market exchange rates. But, if the VSL for India in PPP dollars is divided by the GNI for India at MER, the ratio of VSL to income will be 3 times greater than is implied by an income elasticity of 1.

### *H. Recommendations Going Forward*

If the GBD estimates of PM damages are adopted by the World Bank it will still be necessary to monetize damages in order to compute Adjusted Net Savings. We recommend that the Bank value DALYs associated with PM and ozone exposures using a VSLY transferred to developing countries from the US, and that it continue to conduct transfers using per capita GNI measured using market exchange rates. In the short term, transfers will likely be made using an income elasticity of 1. As noted above, the current monetization procedure implies that the VSL is about 114 times per capita income, and that the VSLY is about 5.18 times per capita income (=114/22). Using a human capital approach to valuation would imply setting the VSLY equal to per capita income. These estimates might be presented as a lower bound to the value of health damages.

In the long run, we believe that the World Bank should consider allowing the income elasticity of the VSL to vary with income. We also recommend that the Bank revisit the issue of how the baseline VSL (currently the US VSL) is chosen, and that the baseline VSL be updated to reflect income growth in the country from which the estimate is derived.[33]

The World Bank estimates of the value of mortality risk for many of its operations and analytical work. However, there is great variation in the methods and assumptions used. It would be useful to review the methods and come to agreement on a consistent methodology to be used in all World Bank work.

## VI. Feasibility of Including Other Pollutants and Damage Categories

We were asked to consider whether other categories of damages associated with particulate matter should be included in ANS estimates and also whether damages from other pollutants (e.g., black carbon and ozone) might be included in the ANS. There are two issues here: one is whether it is possible to obtain damage estimates on a global scale; the second is whether the magnitude of these damages warrant the effort involved in estimating them. Damages to field crops associated with ozone and to forests due to acid rain could be estimated on a global scale. Studies by the USEPA, however, suggest that these damages are likely to be

[33] The current estimate of 5.46 million (2009 USD) is in line with meta-analyses of the VSL for the US reported in Cropper, Hammitt and Robinson (2011); however, no justification for the original point estimate is given by Pandey et al. (2006).

small relative to health damages.[34] Damages to buildings and materials from outdoor air pollution and ecosystem damages, such as acidification of lakes and excess nutrients in surface water bodies are likely to be difficult to estimate on a global scale, as are health damages due to water pollution.[35]

[34] In The Benefits and Costs of the Clean Air Act 1990 to 2010 (USEPA 1999), the USEPA, using methods similar to those in GMAPS, values the health damages associated with outdoor air pollution at $100 billion (1990 USD), damages to field crops at $550 million (1990 USD) and damages to timber yields at $600 million (1990 USD).

[35] The GBD (Lim et al. 2012) estimates health damages due to lack of access to potable water and improved sanitation. The majority of these damages represent diseases transmitted by the oral-fecal contamination route, rather than diseases associated with pollution externalities. The GBD does not estimate health damages due to water pollution externalities.

## Appendix: Description of GMAPS (written by Chris Sall)

GMAPS is a two-stage, fixed-effects regression model that predicts the city annual average concentration of $PM_{10}$ (particulate matter particulate matter with an aerodynamic diameter of less than 10 μm) based on information on population, income level, economic activity, use of different types of energy sources, climate and geomorphology. The model parameters are estimated using average annual monitored concentrations of particulate matter with an aerodynamic diameter of less than 10 μm ($PM_{10}$) in cities from 1985 to 2011. Monitored data for total suspended particulates (TSP) and $PM_{2.5}$ are excluded from the re-estimated model.

The estimating equation (A1) focuses on the anthropogenic sources of pollution and the dissipitative/dispersive capacity of the natural environment. Its determinants include the scale and composition of economic activity, the energy mix, the strength of local pollution regulation, and geographic and atmospheric conditions that affect pollutant transport. The model incorporates information on factors such as fuel mix, level of economic development, demographics, climate, geography, and topography to predict ambient concentrations of $PM_{10}$ in urban areas.

$$C_{jkt} = \sum_{k=1}^{K} \beta_k Z_k + \sum_{f=1}^{F} \beta_{Ef} E_{fkt} + \sum_{g=1}^{G} \beta_{Mg} M_{gjk} + \beta_R R_{kt} + \beta_N N_{jkt} + \beta_D D_{jk} + \beta_{Scale} Scale_{jkt} + \beta_Y Y_{kt} + \beta_T Trend_{jkt} + \beta_{YT} Y_{kt} Trend_{jkt} + u_{jkt} \quad \text{(A1)}$$

where the β's are the parameters that are estimated by the model, and:

$C_{jkt}$=log of average $PM_{10}$ concentration in city j, country k, in year t

$Z_k$= binary variable for country k

$E_{fkt}$= log of per capita energy consumption of energy source type f in country k in year t

$M_{gjk}$= log of time-invariant meteorological/geographic factors g for city j in country k

$R_{kt}$= log of population density of country k in year t

$N_{jkt}$= log of population of city j in country k in year t

$D_{jk}$= log of local population density in the vicinity of city j in country k

$Scale_{jkt}$= log of scale of economy for city j in country k in year t (local GDP per $km^2$)

$Y_{kt}$= log of income per capita (lagged three year moving average) in k at t

$Trend_{jkt}$=time trend

$u_{jkt}$ =error term for city j in country k in year t

GMAPS estimates a total of 37 slope coefficients related to $PM_{10}$ plus the country-level fixed effect ($Z_k$). Whereas some variables take on different values for each year, the geographic, topographic, and climatological variables are assumed to be time-invariant. In order to avoid making predictions based on exogenous variables that are outside of the range defined by the cities that are basis of GMAPS, the data is truncated at the 1st and 99th percentile of the distribution. The estimated $PM_{10}$ concentration in a city for a given year is calculated by multiplying the exogenous data matrix from any city with these slope coefficients and adding the country-level fixed effect and predicted residual term for each city, which is the mean ( $\bar{\rho}_k$ ) of $\rho_{jkt}$ for city j in country k for all years t in equation (A2) below:

(A2) $$\rho_{jkt} = \hat{C}_{jkt} - C_{jkt}$$

where

$\rho_{jkt}$ =predicted residual for city j in country k in year t

$\hat{C}_{jkt}$ =estimated $PM_{10}$ concentration for city j in country k in year t

$C_{jkt}$ =actual monitored $PM_{10}$ concentration for city j in country k in year t

The country-specific binary variable, $Z_k$, is used to control for economic, social and natural factors that are not captured by other explanatory variables. This includes differences in ambient concentration data quality and collection methods across countries, the degree of regulatory heterogeneity within a country, the relative importance of inter-city transport, proximity of and pollution levels in neighboring cities and the composition of economic activity. The country-specific binary variables measure the average PM concentration in each country during the period from 1985 to 2009, controlling for within country variations in due to factors accounted for in the remainder of the estimating equation. In contrast, the rest of the estimation model explains the marginal contribution of the included factors to deviations in the ambient city concentration from this average level.

In order to facilitate predictions for countries not included in the first-stage regression (i.e., countries without air quality monitoring data), a second-stage regression shown in equation (A3) is introduced to predict the country-level fixed effects in equation (A1). Country-level

fixed effects can be interpreted at the average level of ambient PM concentrations in each country not considering other factors:

(A3) $$\hat{\beta}_k = A + \sum_{f=1}^{F} \gamma_{Ef} \bar{E}_{fk} + \gamma_R \bar{R}_k + \gamma_Y \bar{Y}_k + u_k$$

where the γ's are the estimated coefficients, and

$\hat{\beta}_k$ =country-specific binary variable coefficient estimated in equation (A1)

A=constant

$\bar{E}_{fk}$ = log of average per capita energy consumption of energy type f for country k during 1985-2009 (f=1,...,F)

$\bar{R}_k$ = log of average population density of country k during 1985-2009

$\bar{Y}_k$ = log of average national per capita income of country k during 1985-2009

$u_k$=error term for country k

The model has recently been re-estimated after supplementing the original PM10 monitoring data with more recent WHO data and additional monitoring data from Chinese cities, the European Environmental Agency and the U.S. EPA. Adding the new PM10 data has significantly improved the geographic and temporal coverage of the model. The re-estimated model includes 8,632 observations from 1,195 cities and 98 countries. The model parameters are estimated using average annual PM10 concentrations from 1985 to 2011. There are some important differences in the new approach:

1. In contrast to the original estimation, logged PM10 concentrations ($C_{jkt}$) are measured at the city level rather than for individual monitoring sites in cities. Since PM10 concentrations are reported at the city level by the new sources (WHO, Chinese yearbooks and Clean Air Asia), concentrations measured by individual monitoring stations in a city are aggregated to a mean value.
2. Local population density, one of the explanatory variables in the original model, is estimated from the *Gridded Population of the World* (version3), available from the Consortium for International Earth Science Information Network (CIESIN). This dataset provides the best available population data for about 120,000 administrative units, converted to a regular grid of population counts at a 5km resolution.

3. While local population density is still calculated as the average population density for all grid cells within a 20km radius of the city center, it was assumed to be unchanged since 1995 in the original model. In the re-estimated model, local population density data varies at 5-year intervals from 1990-2015. Population density for years in between data points is interpolated linearly.

## Appendix Table 1.[a] GMAPS PM10 Measurement Counts by Source

| Year | CCY | Clean Air Asia | EEA AirBase | US EPA | WB-DEC | WHO |
|---|---|---|---|---|---|---|
| **1985** | 0 | 0 | 0 | 0 | 7 | 0 |
| **1986** | 0 | 0 | 0 | 0 | 19 | 0 |
| **1987** | 0 | 0 | 0 | 0 | 12 | 0 |
| **1988** | 0 | 0 | 0 | 0 | 20 | 0 |
| **1989** | 0 | 0 | 0 | 0 | 24 | 0 |
| **1990** | 0 | 0 | 0 | 0 | 27 | 0 |
| **1991** | 0 | 0 | 0 | 0 | 36 | 0 |
| **1992** | 0 | 0 | 3 | 0 | 43 | 0 |
| **1993** | 0 | 0 | 9 | 0 | 51 | 0 |
| **1994** | 0 | 0 | 15 | 0 | 53 | 0 |
| **1995** | 0 | 0 | 20 | 0 | 55 | 0 |
| **1996** | 0 | 0 | 27 | 0 | 218 | 0 |
| **1997** | 0 | 0 | 52 | 0 | 213 | 0 |
| **1998** | 0 | 0 | 43 | 0 | 236 | 0 |
| **1999** | 0 | 71 | 85 | 0 | 216 | 0 |
| **2000** | 0 | 75 | 136 | 0 | 0 | 0 |
| **2001** | 0 | 81 | 215 | 0 | 0 | 0 |
| **2002** | 0 | 84 | 233 | 0 | 0 | 0 |
| **2003** | 0 | 87 | 279 | 0 | 0 | 6 |
| **2004** | 0 | 143 | 304 | 0 | 0 | 8 |
| **2005** | 118 | 156 | 330 | 102 | 0 | 3 |
| **2006** | 126 | 161 | 337 | 102 | 0 | 7 |
| **2007** | 141 | 152 | 334 | 102 | 0 | 29 |
| **2008** | 144 | 113 | 124 | 102 | 0 | 405 |
| **2009** | 225 | 133 | 393 | 0 | 0 | 203 |
| **2010** | 227 | 17 | 396 | 102 | 0 | 11 |
| **2011** | 238 | 0 | 393 | 0 | 0 | 0 |

[a] CCY = Chinese City Yearbooks

*Source*: Sall 2013.

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