# MIT Joint Program on the Science and Policy of Global Change


## Global Aerosol Health Impacts: Quantifying Uncertainties

*Noelle E. Selin, Sergey Paltsev, Chien Wang, Aaron van Donkelaar, and Randall V. Martin*

**Report No. 203**
**August 2011**

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To inform processes of policy development and implementation, climate change research needs to focus on improving the prediction of those variables that are most relevant to economic, social, and environmental effects. In turn, the greenhouse gas and atmospheric aerosol assumptions underlying climate analysis need to be related to the economic, technological, and political forces that drive emissions, and to the results of international agreements and mitigation. Further, assessments of possible societal and ecosystem impacts, and analysis of mitigation strategies, need to be based on realistic evaluation of the uncertainties of climate science.

This report is one of a series intended to communicate research results and improve public understanding of climate issues, thereby contributing to informed debate about the climate issue, the uncertainties, and the economic and social implications of policy alternatives. Titles in the Report Series to date are listed on the inside back cover.

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# Global Aerosol Health Impacts: Quantifying Uncertainties

Noelle E. Selin[*], Sergey Paltsev[§], Chien Wang[§], Aaron van Donkelaar[†], and Randall V. Martin[†#]

**Abstract**

*Atmospheric fine particulate matter <2.5 μm ($PM_{2.5}$) can cause cardiovasculatory and respiratory damages and mortalities. Assessing population exposure to and damages from $PM_{2.5}$ is important for policy, but measurement networks are only available in a few regions. We assess variation resulting from using different sources of concentration information to constrain $PM_{2.5}$ exposure worldwide, and compare the magnitude of this variation to uncertainties in epidemiological exposure-response functions and economic valuation of health impacts. We find that only 10% of global population is in areas constrained by ground-based data. We calculate and compare regionally-averaged population-weighted concentrations using two atmospheric models: the MIT/NCAR CAM3 aerosol-climate model, and the GEOS-Chem atmospheric chemistry model; and a satellite-derived $PM_{2.5}$ product. We examine the contributions of different aerosol components to population-weighted $PM_{2.5}$, and find large differences in exposure between U.S. and global populations. We use the MIT Emissions Prediction and Policy Analysis Health Effects model (EPPA-HE) to assess global health impacts and related economic costs, and conduct probabilistic uncertainty analysis of concentration-response functions. We use these combined approaches to project uncertainty ranges for health impacts and related economic costs from present-day $PM_{2.5}$. We find large uncertainties in simulated $PM_{2.5}$, especially globally; the magnitude of concentration variation among estimation methods is comparable to uncertainties in epidemiological functions and economic valuations. We identify major contributors to concentration variation, notably the parameterization of atmospheric dust. We estimate an annual global welfare cost of present-day (2000-2005) $PM_{2.5}$ of US $280 billion (range US $120 – 510 billion), and related annual mortalities at 1.3 million per year (630,000 – 2.1 million).*

**Contents**



## 1. INTRODUCTION

Atmospheric fine particulate matter <2.5 μm ($PM_{2.5}$) is extensively regulated due to its potential to harm human health. Evaluating population exposure to and potential damages from

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$PM_{2.5}$ is a critical first step in designing policies to mitigate damages. However, measurement networks for $PM_{2.5}$ are available only in a few developed regions such as the U.S., Canada, and Europe. Atmospheric models and/or satellite data can be used to assess concentrations outside these regions, but ground-based measurement data constraints on these methods cover a limited fraction of the global population. Globally, quantifying the impacts of $PM_{2.5}$ is characterized by uncertainties along the entire causal chain from concentrations to exposure to damages. Here, we assess the variation in using atmospheric models and satellite information to constrain $PM_{2.5}$, focusing on the challenge of estimating exposure to the majority of the world's population, which is outside regions covered by ground-based measurement data. We compare the magnitude of variation in concentration estimates to uncertainties in epidemiological concentration-response functions and economic valuation of health impacts. Atmospheric aerosols come from multiple sources and are a mixture of sizes and compositions (including sulfate, organic carbon, black carbon, nitrates, sea salt and mineral dust). We focus on fine particulate matter ($PM_{2.5}$) consistent with data availability for exposure analysis (Russell and Brunekreef, 2009). We use this information to project a range of economic damages from present-day $PM_{2.5}$.

There is substantial and growing interest in using model-based analyses to assess health and economic impacts of present and future air pollution. Previous studies have estimated the number of mortalities and quantified uncertainties associated with present-day $PM_{2.5}$ exposure using atmospheric models or concentration estimates. Liu *et al.* (2009) estimated that 380,000 excess mortalities globally in 2000 were associated with intercontinental transport of $PM_{2.5}$. They consider uncertainties from aerosol concentrations (expressed as a uniform distribution within a factor of 2) and concentration-response functions (normally distributed) to estimate using Monte Carlo sampling an uncertainty range between 18-240% of estimated deaths. Anenberg *et al.* (2010) calculated the global burden of anthropogenic PM on premature mortalities as 3.5 ± 0.9 (standard deviation, SD) million excess cardiopulmonary and 220,000 ± 80,000 (SD) lung cancer mortalities. They used a SD of 25% for present-day anthropogenic aerosol concentrations, as well as uncertainty in concentration-response functions, to calculate uncertainty ranges using a Monte Carlo approach. Cohen *et al.* (2005) used econometric urban aerosol projections to estimate that the global $PM_{2.5}$ burden results in 800,000 annual excess mortalities, with a stated uncertainty interval of 50%.

The U.S. EPA evaluated prospective air pollution-related damages associated with PM as part of their periodic assessments of the benefits and costs of the Clean Air Act (section 812). They conducted an uncertainty analysis using the Community Multiscale Air Quality (CMAQ) modeling system and the Environmental Benefits Mapping and Analysis Program (BenMap). They did not quantitatively assess the impact of air pollution modeling on uncertainty quantification, but they estimate that air quality estimation very likely contributes >10% to overall uncertainty in benefits assessment. However, their analysis is limited to the U.S., where an extensive database of $PM_{2.5}$ measurements is available for model validation and calibration (U.S. EPA, 2010).

Here, we assess the global-scale uncertainties contributed by air quality information relative to uncertainties in health and economic benefits estimation at global scale. We use two global models and a satellite estimate of $PM_{2.5}$ to compare the sensitivity of mortality estimates on global concentration fields, and assess the magnitude of the variation contributed by various concentration estimates with the uncertainty contributed by concentration-response functions. We use our results to identify the aspects of aerosol chemistry contributing to most uncertainty in comparison with health and economic outcomes, and estimate the global health and economic burden of present-day $PM_{2.5}$.

## 2. INPUTS AND MODEL DESCRIPTION

### 2.1 Inputs and Model Description

We compare $PM_{2.5}$ estimates from three sources: 1) The GEOS-Chem global atmospheric chemistry and transport model; 2) the MIT aerosol climate model version of the NCAR Community Atmosphere Model version 3 (MIT/NCAR CAM3); and 3) a satellite $PM_{2.5}$ product generated based on information from the MODIS (Moderate Resolution Imaging Spectroradiometer) and MISR (Multiangle Imaging Spectroradiometer) satellite instruments and modeled aerosol vertical profiles.

The GEOS-Chem chemical transport model v. 8-01-04 (http://www.geos-chem.org/) (Bey *et al.*, 2001) has been used in a number of air quality investigations. The GEOS-Chem aerosol simulation has a global resolution of 2°x2.5° latitude-longitude and includes sulfate-nitrate-ammonium aerosols (Park *et al.*, 2004), sea salt (Alexander *et al.*, 2005) and secondary organic aerosol (Chung and Seinfeld, 2002). Dust is based on the mineral dust entrainment and deposition (DEAD) scheme of Zender *et al.* (2003) as implemented by Fairlie *et al.* (2007); we divide dust concentrations by two for consistency with recent updates to the GEOS-Chem dust parameterization (v. 8-03-01). To calculate $PM_{2.5}$, we combine all sulfate-nitrate-ammonium aerosols, the smallest dust size bin (<1.0 μm), 38% of the second dust bin (1.0-1.8 μm) and the smaller sea salt bin (<0.5 μm dry radius), and assume 35% relative humidity for consistency with measurements upon which exposure-response functions are based. Modeled $PM_{2.5}$ from GEOS-Chem has been compared with surface measurements in previous studies (Park *et al.*, 2004, 2006; Liao *et al.*, 2007).

The MIT/NCAR CAM3 model is a multimode, two-moment interactive aerosol-climate model (Kim *et al.*, 2008). It includes seven aerosol modes: three external mixtures of sulfate aerosol and one each for external black carbon (BC), external organic carbon (OC), sulfate/BC mixture, and sulfate/OC mixtures. We use a global resolution of 2°x2.5° latitude-longitude. To calculate $PM_{2.5}$, we assume all seven aerosol modes are <2.5 μm. We add 14.6% to sulfate mass to account for the mass of associated nitrate aerosol not simulated, based on the ratio of global burden of nitrate (Feng and Penner, 2007) to sulfate (Kim *et al.*, 2008). We adjust dry concentrations to 35% relative humidity as above. Dust is based on Mahowald (2007). Model BC, OC and sulfate were previously compared with surface observations by Kim *et al.* (2008).

Satellite $PM_{2.5}$ information is based on the product of van Donkelaar *et al.* (2010). Van Donkelaar *et al.* use total column aerosol optical depth (AOD) from MODIS and MISR and coincident aerosol vertical profiles from GEOS-Chem to generate a 0.1°x0.1° map of global ground-level $PM_{2.5}$. Geographic coverage of this product is >95%. They validate their product by comparing to a global suite of measurements and report good correlations between their product and measurements over North America (r=0.77) and elsewhere (r=0.83) (van Donkelaar *et al.*, 2010). The satellite estimate has a stated 1 SD uncertainty of 25%.

### 2.2 Economic Modeling of Health Impacts

We use the MIT Emissions Prediction and Policy Analysis model (Paltsev *et al.* 2005) with extensions to estimate and value air pollution health impacts (EPPA-Health Effects or EPPA-HE). EPPA is a computable general equilibrium (CGE) model of the world economy. Previously, EPPA-HE was applied to assess the benefits of the U.S. Clean Air Act (Matus *et al.*, 2008), the historical burden of and potential benefits of regulating European air pollution (Nam *et al.*, 2010) and the global health and economic impacts of future ozone (Selin *et al.*, 2009). The underlying economic assumptions of EPPA-HE are described in detail by Matus *et al.* (2008). The model includes sixteen global regions (see Annex Figure A1). Among other inputs, it takes as input the population-weighted pollutant concentration (here, $PM_{2.5}$) for each region. This is calculated here based on gridded 2000 population (CIESIN, 2005).

EPPA-HE calculates morbidities and mortalities from concentration-response functions for a five-year timestep (**Table 1**). Morbidities include hospital admissions, respiratory and cardiovascular endpoints in children, adults, the elderly, and the entire population. We consider mortality from both acute and chronic exposure. Concentration-response functions and associated costs are from the survey of Bickel and Friedrich (2005), adjusted from PM10 to $PM_{2.5}$ where necessary by a factor of 0.6 as recommended by Bickel and Friedrich (2005). For mortalities from acute exposure, following Bickel and Friedrich (2005), we apply a value of a statistical life year (VOLY) approach and assume that each reflects 0.5 years of life lost. Mortalities from chronic exposure are applied to adults >30 years of age using a demographic model tracking age cohort exposure. We use age-specific baseline cardio-pulmonary mortality rates (Lopez *et al.*, 2006), for high income (developed regions) and low-middle income countries (developing regions). Population age distributions are applied separately for developing and developed regions (United Nations, 2007). We track labor and leisure losses to the population through time assuming expected life span of 75 years.

Resources used for health care associated with $PM_{2.5}$ morbidities are unavailable to the rest of the economy. Values associated with health endpoints reflect both treatment costs and willingness-to-pay (WTP) to avoid damages. Because information is not available on health costs in all regions, we calculate values from European costs for developed regions, and from China for developing regions (Selin *et al.*, 2009). We use purchasing power parity (Heston *et al.*, 2002) to adjust costs for local conditions in each region. Labor and leisure lost from chronic

**Table 1.** Concentration-response functions, costs and uncertainties. Concentration-response functions are in cases per (μg $m^{-3}$) except where noted. Based on Bickel and Friedrich (2005), converted from $PM_{2.5}$ using factor of 0.6.

| | Concentration-response function | 5%-95% confidence interval | Cost (US $ year 2000) | Standard Error Cost ($) |
|---|---|---|---|---|
| **ENTIRE POPULATION** | | | | |
| Respiratory hospital admissions | 1.17E-05 | (6.38E-06, 1.72E-05) | 2000 | 670 |
| Cerebrovascular hospital admissions | 8.40E-06 | (6.47E-07, 1.62E-05) | 2000 | 670 |
| Cardiovascular hospital admissions | 7.23E-06 | (3.62E-06, 1.09E-05) | 2000 | 670 |
| Mortality from acute exposure | 0.10% | (0.07%, 0.13%) | 250000 | 1850 |
| Mortality from chronic exposure | 0.42% | (0.03%, 0.80%) | Calculated in the model | |
| **CHILDREN** | | | | |
| Chronic bronchitis | 2.68E-03 | (2.07E-04, 5.17E-03) | 360 | 123 |
| Chronic cough | 3.45E-03 | (2.65E-04, 6.63E-03) | 38 | 13 |
| Respiratory symptoms days | 3.10E-01 | (3.10E-01, 1.53E-01) | 38 | 13 |
| Bronchodilator usage | 3.00E-02 | (-1.15E-01, 1.77E-01) | 1 | 0.33 |
| Cough | 2.22E-01 | (3.83E-02, 4.05E-01) | 38 | 13 |
| Lower respiratory symptoms (wheeze) | 3.10E-01 | (1.53E-01, 4.62E-01) | 38 | 13 |
| **ADULTS** | | | | |
| Restricted activity day | 9.02E-02 | (7.92E-02, 1.01E-01) | 82 | 27 |
| Minor restricted activity days | 5.77E-02 | (4.68E-02, 6.87E-02) | 38 | 13 |
| Respiratory symptoms days | 2.17E-01 | (2.50E-02, 4.05E-01) | 38 | 13 |
| Chronic bronchitis | 4.42E-05 | (-3.17E-06, 9.02E-05) | 190000 | 63000 |
| Bronchodilator usage | 1.52E-01 | (-1.52E-01, 4.62E-01) | 1 | 0.33 |
| Cough | 2.80E-01 | (4.85E-02, 5.12E-01) | 38 | 13 |
| Lower respiratory symptoms (wheeze) | 2.17E-01 | (2.50E-02, 4.05E-01) | 38 | 13 |
| **OVER AGE 65** | | | | |
| Congestive heart failure | 1.11E-05 | (8.52E-07, 2.14E-05) | 12000 | 925 |
| Ischaemic heart disease | 1.05E-05 | (8.10E-07, 2.02E-05) | 12000 | 925 |

exposure mortalities is valued endogenously by EPPA at the wage rate, which differs over time and among regions.

For the analysis presented here, $PM_{2.5}$ is assumed constant from 2000-2005, and costs are calculated based on the 2005 global economy. We consider here only damages from $PM_{2.5}$ based on exposure in 2000-2005 and thus set prior concentrations to zero (we incorporate prior concentrations in sensitivity analysis). We calculate the effect on economic welfare (defined as consumption plus the value of leisure time) in year 2000 US $. Uncertainties in concentration-response and cost estimates are based on the literature (Table 1).

### 2.3 Uncertainty Evaluation

We use a Monte-Carlo based approach (n=400) to quantitatively assess the uncertainties in concentration-response functions and economic valuation of health impacts. We assume that exposure-response functions and costs are normally distributed. We conduct uncertainty analysis similarly to the methodology used by Webster *et al.* (2008) and Selin *et al.* (2009). We apply Latin Hypercube sampling (Iman and Conover, 1982) to select from probability distributions of concentration-response functions and valuations, running EPPA-HE with these sets of inputs to calculate global economic welfare. Table 1 shows uncertainty ranges used to construct input parameter probability distributions. In sampling, we assume correlation at r=0.9 between concentration-response functions and among cost estimates to prevent sampling physically unrealistic combinations of very low response/cost for one parameter and very high for another.

## 3. VARIATION IN CONCENTRATION ESTIMATES

We assess the variation in population-weighted concentration using the three sources of concentration information. Population-weighted $PM_{2.5}$ is an imperfect exposure estimate, but it approximates large-scale monitoring data used to develop concentration-response functions. Population-weighted averages have different characteristics than the area-weighted averages more commonly assessed in the atmospheric literature. Our approach differs from previous analyses of model and satellite error and uncertainty (e.g. Park *et al.*, 2004; Kim *et al.*, 2008; van Donkelaar *et al.*, 2010) because it considers population-weighted effects.

We first assess constraints on population-weighted exposure from ground-based measurements. We use data compiled by van Donkelaar *et al.* (2010), including data from the U.S. Interagency Monitoring of Protected Visual Environments (IMPROVE) (http://vista.cira.colostate.edu/improve/Data/data.htm), the U.S. Environmental Protection Agency Air Quality System Federal Reference Method sites (http://www.epa.gov/air/data/index.html), the Canadian National Air Pollution Surveillance Network (http://www.etc.cte.ec.gc.ca/NAPS/index_e.html), and 244 annually representative, ground-based $PM_{2.5}$ measurements from published and unpublished data outside the U.S. and Canada. **Table 2** shows the percentage of population covered by ground-based measurements, assuming each measurement is representative of a 1˚x1˚ gridsquare (roughly 95x95 km at 45˚ N) or a 0.1˚x0.1˚ gridsquare (roughly 10x10 km). Globally, measurement data represent only 10% of the population assuming a 1˚x1˚ gridsquare and 2% assuming a 0.1˚x0.1˚ gridsquare. There

are substantial regional differences – while 81% of the US population is within the same 1˚x1˚ gridsquare as a ground-based $PM_{2.5}$ measurement, less than 1% of the African population is.

**Table 2.** Percentage of population covered by ground-based measurement data. Calculated for each EPPA region, assuming data points are representative of a 1˚x1˚grid square or a 0.1˚x0.1˚grid square. A map with regional abbreviations is provided in the Appendix.

| EPPA region | % of population at 1˚x1˚ | % of population at 0.1˚x0.1˚ |
|---|---|---|
| AFR | <1% | <1% |
| ANZ | 56% | 8% |
| ASI | 12% | 2% |
| CAN | 74% | 15% |
| CHN | 1% | <1% |
| EET | 19% | 2% |
| EUR | 41% | 4% |
| FSU | <1% | <1% |
| IDZ | <1% | <1% |
| IND | <1% | <1% |
| JPN | 23% | <1% |
| LAM | 2% | <1% |
| MES | 6% | 1% |
| MEX | 8% | 1% |
| ROW | 4% | <1% |
| USA | 81% | 13% |

**Figure 1** shows population-weighted $PM_{2.5}$ for each EPPA region, for each of the concentration sources, plus an estimate from ground-based data covering the fraction of population where data are available (Table 2). The largest concentrations are in developing regions such as China, Africa, the Middle East, India, and the Rest of the World (ROW) region that mostly includes less-developed economies of South/Central Asia. We find large differences (standard deviations among the different estimates up to 100%) in population-weighted $PM_{2.5}$, especially outside data-constrained regions. The difference in $PM_{2.5}$ alone is greater than uncertainty estimates in some previous literature. This is also comparable to the overall 25%-200% uncertainty ranges previously assumed for estimated mortalities, which take into account not only concentration uncertainty but also epidemiological uncertainties (Anenberg *et al.*, 2010; Liu *et al.*, 2009). We adopt this range as a lower bound of potential simulated uncertainty, as different simulations share some assumptions about emissions and processes and therefore cannot be interpreted as covering a true uncertainty range. We focus on the uncertainty in the simulations, as they provide the best source of information on global $PM_{2.5}$ composition, and composition may affect the dose-response curve as discussed below.

Also shown in Figure 1 are GEOS-Chem and MIT/NCAR CAM3 simulations without fine mineral dust. A substantial portion of the difference between these two model estimates comes from dust, which is poorly constrained. These results are consistent with a recent global

intercomparison of dust aerosol optical depth (Huneeus *et al.*, 2010), which found that models can differ from surface measurements by up to two orders of magnitude. It is unknown whether mineral dust in the $PM_{2.5}$ range causes similar health outcomes as industrial aerosol. Anenberg *et al.* (2010) avoid some of this uncertainty as they consider only $PM_{2.5}$ from anthropogenic sources and exclude mineral dust and sea salt. We show, however (section 4.3), that this omits the majority of global $PM_{2.5}$ exposure. Even omitting mineral dust from our analysis, however, concentration differences between the two models are up to 150% for some regions, far exceeding the 25-50% used in previous studies.

All three concentration sources have shown acceptable agreement with measurement data in previous studies (Kim *et al.*, 2008; Park *et al.*, 2004, 2006; Liao *et al.*, 2007; van Donkelaar *et al.*, 2010). With the same surface data set, comparing only in areas with available ground-based measurements, van Donkelaar *et al.* (2010) found significant spatial agreement with their satellite product. Our analysis is not a model performance evaluation. Comparisons with measurements in areas where surface measurements are available are an important constraint on model performance; however, as Table 2 shows, the use of surface measurements to estimate population-based exposure is severely constrained by data availability. Thus, none of the points in Figure 1 is intended to represent the "true" estimate; rather, we use different sources to illustrate the range, and do not intend to portray one source as performing better or worse. We note, however, that the satellite product is constrained by additional global-scale information (total-column aerosol optical depth from satellite), making it the only concentration source that incorporates data over the entire global population.

## 4. SOURCES OF CONCENTRATION VARIATION

We examined the sources and contributions of variation contributing to the differences among three $PM_{2.5}$ estimates, including emissions, interannual variability, and various chemical components of aerosol.

### 4.1 Emissions

Different assumptions about emissions and atmospheric processing can explain some differences between the two models (GEOS-Chem and MIT/NCAR), though the satellite product is less dependent on this information. As used here, GEOS-Chem applies global emissions from the EDGAR FT2000 inventory (Olivier *et al.*, 2001) for NOx, CO and $SO_2$ and the GEIA inventory for VOCs. These emissions are replaced with improved data for the U.S., Canada, Mexico and East Asia as described by van Donkelaar *et al.* (2008). For global BC and OC, the Bond *et al.* (2006) emissions are used except for the U.S. and Canada, where the Cooke *et al.* (1996) inventory is used. Interannual scaling is applied for emissions relative to the base year of the simulation (van Donkelaar *et al.*, 2008). The MIT/NCAR model uses the EPPA inventory for BC, OC and $SO_2$ (Mayer *et al.*, 2000; Babiker *et al.*, 2001; Wang, 2004; Asadoorian *et al.*, 2006) and the GEIA inventory for biogenic VOCs. The EPPA inventory, using emission factors from Cooke *et al.* (1999), estimates substantially higher emissions for BC (14.4 Tg $y^{-1}$) and OC (54.4

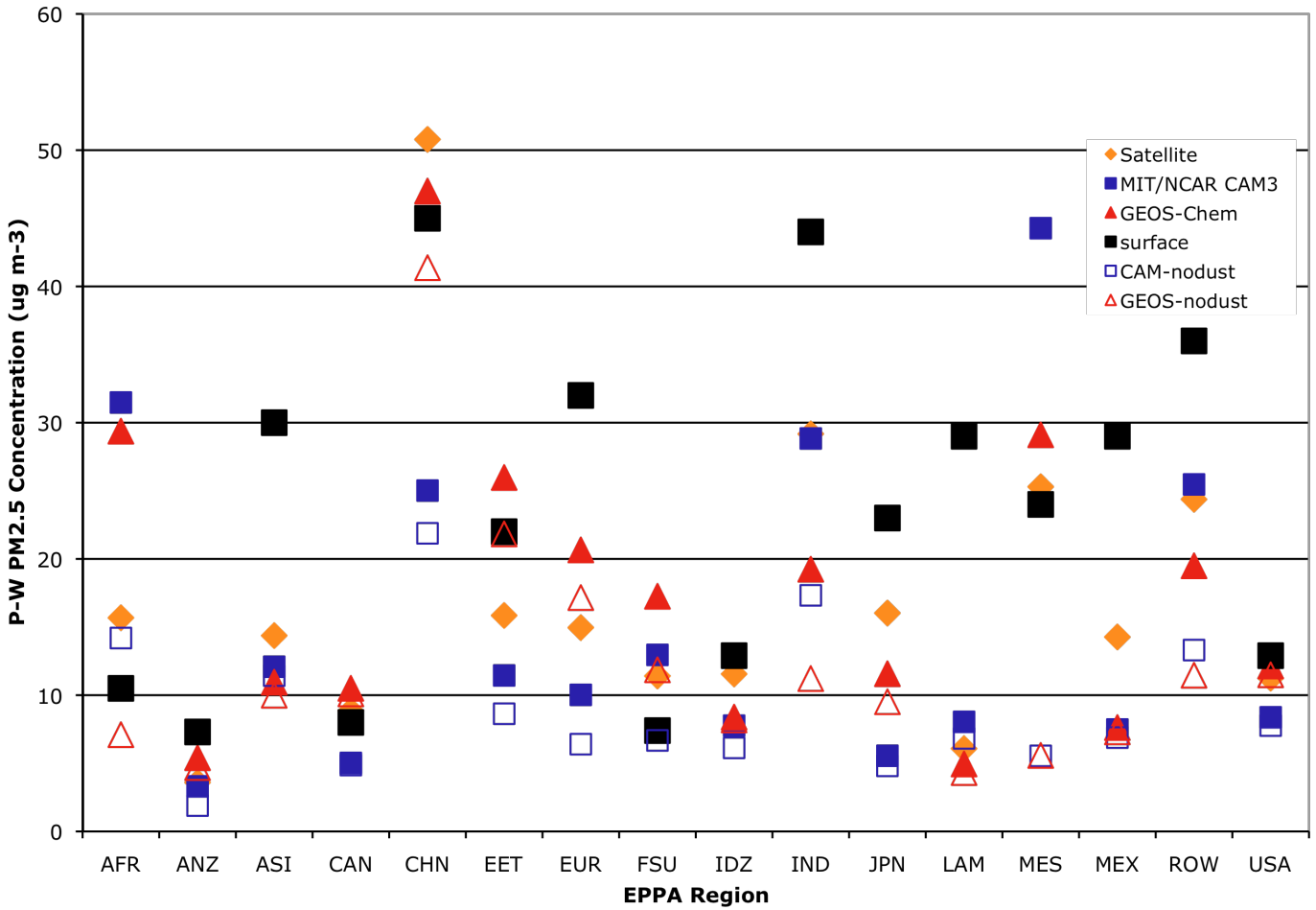


**Figure 1**. Variation in population-weighted (P-W) $PM_{2.5}$ concentration (μg $m^{-3}$) calculated for each EPPA region from two models (GEOS-Chem, filled orange triangles, and MIT/NCAR CAM3, filled blue squares) and a satellite product (orange diamonds). Also shown are population-weighted concentrations estimated from surface data (black squares) that only cover a subset of the regional population (see Table 2). Open blue squares and open red triangles reflect CAM and GEOS-Chem runs without contributions from dust. A map with regional abbreviations is provided in the Appendix.

Tg $y^{-1}$) than Bond *et al.* (8.0 and 33.8 Tg $y^{-1}$, respectively). Previously, Kim *et al.* (2008) compared the sensitivity of modeled aerosol to emissions, using the Bond *et al.* and EPPA inventories. They reported differences up to 20% in total sulfate mass, and changes in BC and OC radiative forcing up to 57% and 30% respectively between the two inventories. This suggests a substantial combined influence on concentration variation among models due to emissions uncertainty in different regions.

### 4.2 Interannual Variability

To assess the contribution to variation from interannual variability, we calculated population-weighted $PM_{2.5}$ for each EPPA region from GEOS-Chem runs for 2001-2006, including both meteorological differences and year-to-year emissions variation. For 13 of 16 regions, the interannual difference is small; in Asia and Australia/New Zealand, interannual variation was within 40%. Interannual differences in the latter two regions are from differences in emissions associated with biomass burning. We conclude that interannual variability contributes only a small amount to uncertainty and variation in population-weighted $PM_{2.5}$.

### 4.3 Aerosol Components

We assessed the influence of variation and uncertainty resulting from different $PM_{2.5}$ components. Recent work has suggested that different components such as BC and OC and some transition metals contribute most to overall PM toxicity (Lippmann and Chen, 2009). Because epidemiological studies relating $PM_{2.5}$ to toxicity have been conducted in the U.S. based on bulk aerosol to which U.S. populations are exposed, comparing differences in exposure to aerosol components provides insight into the uncertainty contributed by applying U.S. epidemiological functions elsewhere.

To assess the differences between U.S. and global aerosol composition, we used GEOS-Chem to calculate the regional population-weighted contribution of different aerosol components to $PM_{2.5}$. Compared with area-weighting, population-weighting weights urban aerosol more heavily and is more relevant to estimating exposure.

Shown in **Figure 2** are population-weighted contributions for the U.S. (panel a) and the entire globe (panel b) for total $PM_{2.5}$. This comparison shows that the $PM_{2.5}$ to which the average global citizen is exposed is very different from that which the average U.S. resident encounters. Specifically, >30% of global population-weighted $PM_{2.5}$ is dust. Combined with the conclusion above that the largest contribution to population-weighted concentration variation results from dust, which is poorly understood, this suggests that constraining non-anthropogenic aerosol is of primary importance in assessing global $PM_{2.5}$ impacts.

Figure 2 also shows the contribution of different aerosol components to anthropogenic $PM_{2.5}$ (where anthropogenic is defined as excluding dust and sea salt) (panels c,d). While contributions to global total population-weighted $PM_{2.5}$ are very different from those in the U.S., contributions to anthropogenic population-weighted $PM_{2.5}$ are more similar to the U.S. The largest difference is for nitrate.

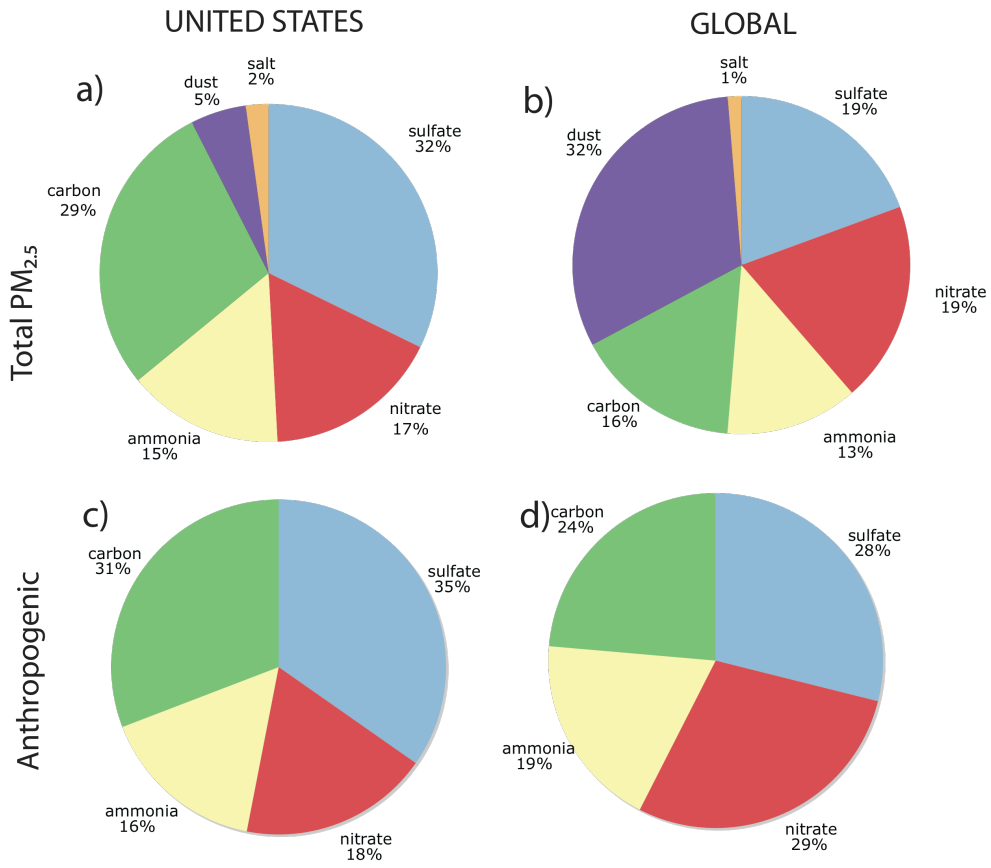


**Figure 2.** Contribution to population-weighted $PM_{2.5}$ by aerosol components in GEOS-Chem. Top row shows contributions to total $PM_{2.5}$ including dust and sea salt for a) the U.S. and b) global average. Bottom row shows contributions excluding dust and sea salt ("anthropogenic") for c) the U.S. and d) the globe.

This analysis suggests that applying concentration-response functions from the U.S. may be unsuitable where total $PM_{2.5}$ measurements include dust and sea salt. It is unknown whether dust in the $PM_{2.5}$ range has similar health effects to other PM sources (Perez *et al.*, 2008). Dust exposure has been associated in epidemiological studies with asthma (Bener *et al.*, 1996), though this may be due to microorganisms present in dust rather than size (Griffin and Kellogg, 2004). Dust can also contain metals such as iron that may influence toxicity (Prospero, 1999).

## 5. COSTS, MORTALITIES AND ASSOCIATED UNCERTAINTIES

We quantitatively assess the influence of variation in concentration on assessment of $PM_{2.5}$ health impacts. We first use the EPPA-HE model to calculate global economic welfare losses associated with population-weighted $PM_{2.5}$ estimated by the two models and the satellite product. We then use a Monte Carlo approach (Section 2.3) to quantitatively assess uncertainties in concentration-response functions and economic valuation of health impacts.

In our Monte Carlo analysis, we use deterministic population-weighted $PM_{2.5}$ from each of the three sources, and vary concentration-response functions and associated economic valuations of case endpoints. We then run EPPA-HE for each of the sampled sets of inputs, and record global pollution-related welfare loss as the difference between EPPA-HE runs with and without pollutant damages.

We show in **Figure 3** (vertical lines) global welfare losses from EPPA-HE using concentrations from the two models and mean values of the epidemiological and economic parameters. Model values are shown with and without contributions from dust, which contributes substantially to the range in concentration as discussed above. Red columns represent the frequency (# of runs out of 400) where Monte Carlo analysis varying epidemiological and economic parameters resulted in global welfare loss in specified ranges, using satellite concentrations. The median annual global welfare loss from present-day $PM_{2.5}$, calculated using satellite $PM_{2.5}$ is US $340 billion; the 95% uncertainty range taking into account variation in epidemiological and economic parameters is US $190-540 billion.

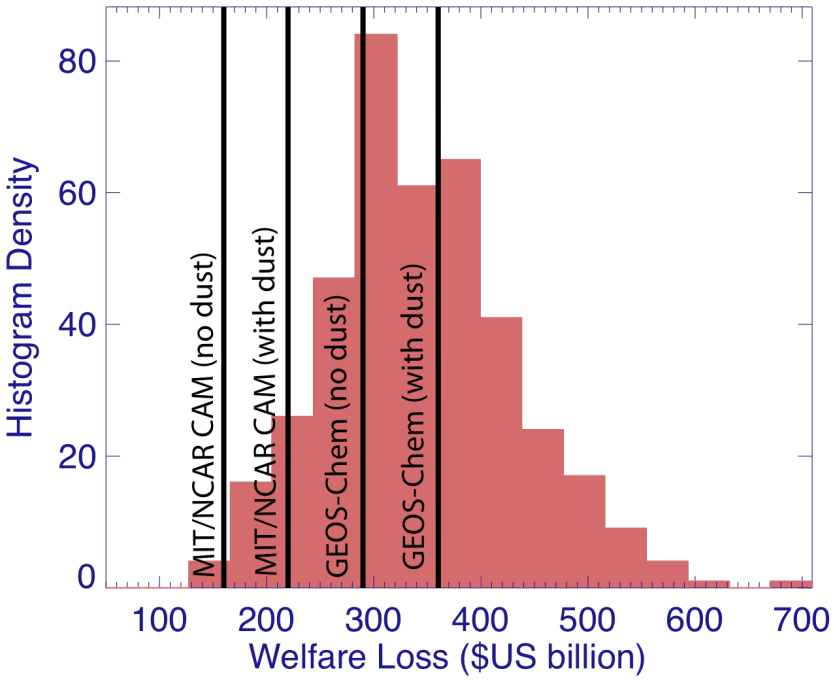


**Figure 3.** Uncertainty in welfare loss (US $billion) from $PM_{2.5}$ due to variation in concentration estimates and uncertainty in concentration-response functions and economic costs. Black vertical lines show welfare loss associated with median values of ensembles using CAM and GEOS-Chem models, with and without dust. Histogram shows frequency distribution of welfare loss for a Monte Carlo simulation (number of simulations where total n=400), varying epidemiological and economic assumptions, using the satellite concentration estimate.

Table 3 shows welfare losses and uncertainty ranges for each concentration assumption. We estimate from this ensemble an overall uncertainty range for welfare loss due to present-day $PM_{2.5}$ of median US $280 billion, with a range of US $120-510 billion per year. This is about 0.3-1.1% of total 2005 global welfare.

**Table 3.** Uncertainty ranges for annual global welfare cost (US $billion) and mortalities due to present-day $PM_{2.5}$ for different concentration inputs. Mortalities include both those from chronic exposure (resulting from additional 2000-2005 exposure only) and acute exposure.

| **Concentration Input** | **Welfare Cost (US $billion)** | | | **Mortalities (thousands)** | | |
|---|---|---|---|---|---|---|
| ***Confidence interval*** | **2.5%** | **50%** | **97.5%** | **2.5%** | **50%** | **97.5%** |
| Satellite | 190 | 340 | 540 | 960 | 1600 | 2200 |
| GEOS-Chem (no dust) | 170 | 290 | 470 | 630 | 1050 | 1500 |
| GEOS-Chem (with dust) | 210 | 360 | 580 | 930 | 1300 | 2000 |
| MIT/NCAR CAM (no dust) | 90 | 160 | 260 | 520 | 860 | 1200 |
| MIT/NCAR CAM (with dust) | 130 | 220 | 370 | 830 | 1400 | 1900 |

Table 3 also shows confidence intervals for our estimates of mortalities due to both acute exposure as well as the chronic exposure resulting from present-day (2000-2005) $PM_{2.5}$. We calculate a median estimate of total annual mortality from $PM_{2.5}$ of 1.3 million per year (range 630,000-2.1 million). This is within the range of previous estimates.

We find that the range of global mean welfare loss resulting from different $PM_{2.5}$ estimates is roughly the same magnitude as the range in global mean welfare loss due to uncertainty in health impacts and valuation. Further, our comparison of three concentration estimates does not cover the full uncertainty range in simulating atmospheric concentrations – thus we view this as a lower bound for simulated concentration uncertainties.

We conduct sensitivity analyses to assess the influence of past concentrations on present-day mortalities, by setting past concentrations equal to present-day concentrations. This takes into account additional present-day deaths due to past exposure, but not the continuing economic effects of previous years' deaths on the present-day economy. This increases our median and range of welfare cost to US $360 billion (US $150-$640 billion), and mortalities to 4.7 million (960,000-10 million). Fully estimating present-day costs of past $PM_{2.5}$ requires concentration and economic information for all regions for 40+ years; such analyses have been conducted for the U.S. (Matus *et al.*, 2008), Europe (Nam *et al.*, 2010) and China (Matus *et al.*, 2011). A previous study with EPPA-HE found that 89% of costs related to chronic PM exposure were from premature deaths occurring in previous years (Nam *et al.*, 2010); our analysis only incorporates cumulative loss beginning in 2000. However, our analysis is perhaps more policy-relevant, as it better reflects the potential benefits from reducing present-day PM as policies cannot affect previous exposure.

## 6. CONCLUSIONS

We assessed the relative importance of errors from concentration estimates relative to those from concentration-response functions and health impact costs in calculating $PM_{2.5}$ impacts. We compared three estimates of population-weighted $PM_{2.5}$ globally to quantify and assess their variation. We used these concentration inputs to calculate an uncertainty range using Monte Carlo simulation for global mortalities and economic costs associated with $PM_{2.5}$ health damages. We concluded that variation in atmospheric concentration estimates contributes comparable uncertainty to variation in concentration-response functions and economic data, and we estimated an uncertainty range for global $PM_{2.5}$ health and economic damages.

We used three different concentration estimates: the GEOS-Chem global atmospheric chemistry and transport model; the MIT/NCAR CAM3 model, and a satellite $PM_{2.5}$ product based on information from the MODIS and MISR satellite instruments and modeled aerosol and vertical profiles. We calculated population-weighted $PM_{2.5}$ for each estimate to drive the EPPA-HE model, which calculated health and related economic damages based on chronic and acute exposure to $PM_{2.5}$ for 2000-2005. We used Monte Carlo analysis to assess the influence of epidemiological and economic cost uncertainty on our results.

Comparison of the variation in global population-weighted $PM_{2.5}$ from the three sources showed most variation where fewer data constraints are available. Population-weighted concentrations across regions differed substantially, far above the 25-50% variation assumed in previous literature using models. A large fraction of the variation resulted from dust in the $PM_{2.5}$ range. Variations in anthropogenic aerosol only were up to 150%. Emissions difference among models was a large influence on variability, while interannual variability was small. The global average contribution of different aerosol components to total population-weighted $PM_{2.5}$ differs greatly from the U.S. regional average, with more global $PM_{2.5}$ contributed by dust; this suggests that concentration-response functions developed for U.S. aerosol may need to be revised for global applicability. Component contributions to population-weighted anthropogenic-only (non-dust, non-sea salt) $PM_{2.5}$ are more similar between U.S. and global averages.

Estimates of global welfare (consumption plus leisure) were calculated using Monte Carlo ensembles of EPPA-HE, varying concentration-response functions and economic cost information. Median values for welfare cost using different concentration assumptions varied from US $160-360 billion. The 95% confidence interval taking into account variation in concentration-response functions and economic costs (with fixed concentrations from satellite data) was US $190-540 billion. We conclude that simulated atmospheric concentration variation contributes comparable uncertainty as concentration-response functions and economic data to global air pollution health estimation.

The range in global welfare costs of present-day $PM_{2.5}$ calculated from EPPA-HE ranged from of US $120-510 billion annually, with a median of US $280 billion. This is equivalent to about 0.3-1.1% of 2005 global welfare. We estimate 1.3 million annual mortalities associated with global $PM_{2.5}$ (with a range 630,000-2.1 million). Considering long-term damages from historical $PM_{2.5}$, median estimated mortalities increased by roughly a factor of 3. Our methodology goes

beyond the assumption of instantaneous response of mortalities to concentration changes and systematically calculates the potential economic benefits of policies to reduce chronic impacts.

We estimated that taking into account present-day deaths from past exposure would increase costs to US $350 billion (US $150-$630 billion), and mortalities to 5 million (900,000-11 million). A full accounting of welfare costs would also include losses from mortalities prior to the year 2000 and cumulative impacts of welfare losses and resource allocation prior to 2000. Our estimate, however, better reflects the potential for economic gains from reducing $PM_{2.5}$.

Our results suggest that quantifying global aerosol-related health damages, particularly using models, is as limited by atmospheric science uncertainties as by damage quantification uncertainties. Though increasing measurement network coverage can address some of these uncertainties, model information is necessary for policy scenarios or to assess the influence of changing climate. Increased model evaluation and intercomparisons for highly-populated regions in developing countries would improve our ability to use models to assess global health outcomes. We also suggest that $PM_{2.5}$ from non-anthropogenic sources may be a substantial, yet underappreciated, source of uncertainty for global health.

Ground-based stations provide few constraints on global population exposure to $PM_{2.5}$. Given the large degree of variation in model estimates of present-day population-weighted $PM_{2.5}$, despite agreement with available measurements, this suggests that measurement networks could substantially benefit from increased coverage and design improvements taking overall population distributions into account. Satellite information provides an additional data-based constraint on exposure outside these regions. Our analysis suggests that the 1 SD uncertainty of 25% in the satellite estimate (van Donkelaar *et al.*, 2010) and global coverage, if accurate, places it among the best-constrained sources of exposure information globally.

We address here only uncertainties we can quantify using models and other methods; the true uncertainty in quantifying aerosol health impacts is undoubtedly larger. Uncertainties that we cannot quantify at this time include potential error in: using area concentrations as a proxy for exposure; applying concentration-response functions from the U.S. and Europe to other countries (particularly developing countries); the degree to which damages are modified by differential access to health care; and quantifying the unknown health impacts of aerosols such as dust and sea salt. These and other uncertainties should be addressed in future research.

**Acknowledgments**

This research was supported in part by the United States Environmental Protection Agency's Science to Achieve Results (STAR) program (RD-83427901-0) and the industrial and foundation sponsors of the MIT Joint Program on the Science and Policy of Global Change. Although the research described in the article has been funded in part by the US EPA, it has not been subjected to any EPA review and therefore does not necessarily reflect the views of the Agency, and no official endorsement should be inferred.

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# APPENDIX

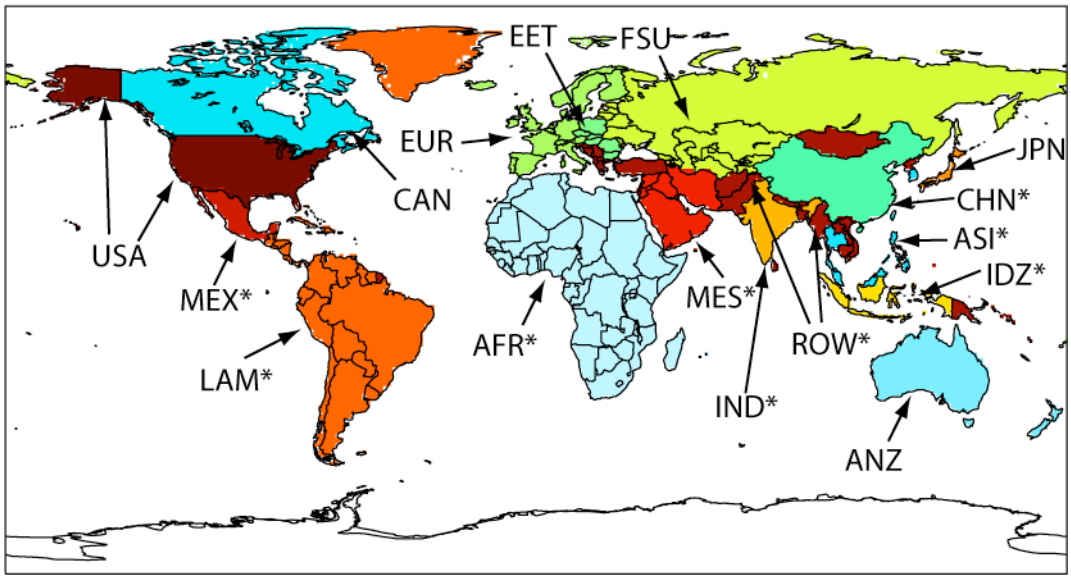


**Figure A1**. EPPA regions used in this study. Asterisks denote regions using data inputs for developing regions.

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