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Exposure assessment for estimation of the global burden of disease attributable to outdoor air pollution

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Abstract

Ambient air pollution is associated with numerous adverse health impacts. Previous assessments of global attributable disease burden have been limited to urban areas or by coarse spatial resolution of concentration estimates. Recent developments in remote sensing, global chemical-

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Supporting Information Additional material and descriptions of methodology along with the full dataset of concentration estimates and a brief data dictionary are provided in the Supporting Information This information is available free of charge via the Internet at http://pubs.acs.org/.

transport models, and improvements in coverage of surface measurements facilitate virtually complete spatially resolved global air pollutant concentration estimates. We combined these data to generate global estimates of long- term average ambient concentrations of fine particles ($PM_{2.5}$) and ozone at 0.1° × 0.1° spatial resolution for 1990 and 2005. In 2005, 89% of the world's population lived in areas where the World Health Organization Air Quality Guideline of 10 μg/m^3 $PM_{2.5}$ (annual average) was exceeded. Globally, 32% of the population lived in areas exceeding the WHO Level 1 Interim Target of 35 μg/m^3; driven by high proportions in East (76%) and South (26%) Asia. The highest seasonal ozone levels were found in North and Latin America, Europe, South and East Asia, and parts of Africa. Between 1990 and 2005 a 6% increase in global population-weighted $PM_{2.5}$ and a 1% decrease in global population- weighted ozone concentrations was apparent, highlighted by increased concentrations in East, South and Southeast Asia and decreases in North America and Europe. Combined with spatially resolved population distributions, these estimates expand the evaluation of the global health burden associated with outdoor air pollution.

Introduction

Ambient air pollution is associated with a considerable burden of global disease. The World Health Organization (1) estimated that exposure to fine particulate air pollution caused 800,000 deaths and 6.4 million lost years of healthy life in the world's cities in 2000. The developing countries of South-East Asia accounted for two-thirds of this burden. Because ground-level measurements of air pollution, especially particulate matter (PM) with an aerodynamic diameter smaller than 2.5 micrometers ($PM_{2.5}$), are unavailable for much of the world, the assessment of the attributable burden of disease, and subsequent assessments conducted by WHO and others, have relied largely on estimates of ambient pollution levels from either econometric or chemical transport models (1, 2)

The year 2000 assessment of the global burden of disease attributable to outdoor air pollution used the annual average concentration of $PM_{2.5}$ as the indicator of air pollution. The pollution estimates were based on modeled annual average $PM_{2.5}$ concentrations for national capital cities and urban areas with populations >100,000; only ~10% of these locations had reliable estimates of annual average concentrations from ground-level monitoring. The restriction to urban areas was necessitated by the use of an econometric model to estimate ambient concentrations of particulate matter. This urban-only model was the only feasible approach to develop global estimates at the time, but therefore excluded more than half the world's population from inclusion in the $PM_{2.5}$ burden analysis.

Recent developments in satellite-based remote sensing (3, 4), global chemical transport models (e.g.,(5)), and incremental improvements in the quality and coverage of ground-level measurements (6), however, now provide virtually complete global coverage with a high degree of spatial resolution, allowing estimation of exposure for both urban and rural populations. Applying these methods, updated estimates of the global burden of disease attributable to outdoor air pollution for the years 1990 and 2005 are being made as part of the Global Diseases, Injuries, and Risk Factors (GBD) 2010 Study (7). Here we present the

approaches used and provide estimates of exposure to $PM_{2.5}$ and ozone for the entire world's population in 1990 and 2005

Experimental

Choice of air pollution indicators

The air pollution to which individuals are exposed is multifaceted; there are no standardized approaches to characterize specific pollutant mixtures, which typically include hundreds of individual gaseous compounds and particles of complex physicochemical composition. Accordingly, indicator pollutants are often used to assess exposures for risk assessment and epidemiologic analysis. For such mixtures, the relative importance of different pollutants is a function of location-specific economic, developmental, social, and technological factors combined with meteorology, topography, geography and atmospheric transformations. Literature and measurement databases exist for a limited number of selected gaseous pollutants (ozone [O_3], nitrogen oxides [NOx ≈ NO+NO2], sulfur dioxide [SO_2], carbon monoxide [CO]) and one or more measures of PM such as Total Suspended Particles (TSP), or the mass concentration of particles with aerodynamic diameter smaller than 10 (PM_{10}) or 2.5 ($PM_{2.5}$) micrometers.

An extensive epidemiological literature relates $PM_{2.5}$ to adverse health impacts (8-10). In epidemiologic cohort studies of long term exposure (which form the basis of the exposure-response functions used in health impact assessment) $PM_{2.5}$ is the most robust indicator of adverse (mortality) impacts (11). The epidemiologic observations of adverse health impacts associated with elevated ambient $PM_{2.5}$ concentrations is supported by toxicological experiments, epidemiologic analyses of acute exposures and controlled exposure studies. In populated regions, a large fraction of $PM_{2.5}$ originates from combustion processes and includes both primary PM (direct emissions) and secondary PM (resulting from atmospheric transformations).

Ozone represents a pollutant mixture that is somewhat different from that associated with PM. This gaseous pollutant is derived from a series of atmospheric photochemical reactions of primary air pollutants, including nitrogen oxides and volatile organic compounds (VOCs). The seasonal, spatial and temporal patterns of surface ozone concentrations are often distinct from those of PM, as are the relative importance of emissions source categories of ozone precursors. Epidemiologic associations have been observed between elevated ozone concentrations and premature mortality that are independent of associations between PM and mortality (12-15). There is also an extensive literature on adverse respiratory impacts resulting from ozone exposure in randomized controlled exposure studies (16). As such, estimates of the global burden of disease attributable to outdoor air pollution are further enhanced by the inclusion of ozone in addition to $PM_{2.5}$. By including both metrics, the GBD analysis is also compatible with recent national and regional analyses of air pollution health and economic impacts (e.g. (17)).

Data sources

In evaluating various approaches to developing global estimates of air pollution concentrations for assessment of health burden we considered the: i) global applicability , ii) spatial resolution and coverage relative to population , iii) accuracy and precision, iv) applicability to both $PM_{2.5}$ and ozone, v) ability to estimate exposure in 2005 and 1990 and vi) sensitivity to assumptions.

While direct measurements of urban background ambient pollutant concentrations are usually the basis for exposure metrics used in epidemiologic analyses, these could not satisfy all of the above criteria. Most importantly, surface measurement data (for PM and even more so for ozone) are still far too sparse in most of the high concentration regions for direct use in exposure assessment throughout the world (6). Even in areas with increasingly extensive measurement coverage, recent measurements would not be applicable to estimates for 1990. However, as described below, our approach does incorporate available PM measurement data along with other information sources. Here, as in most epidemiologic analyses, ambient concentrations are used as proxies for personal exposure to air pollution of outdoor origins. The relationship between ambient monitoring measurements and exposure, and the representativeness of ambient monitoring data as exposure proxies (for example, the importance of within-city variability in concentrations that is not reflected by ambient by monitoring data) has been discussed in more detail elsewhere (18, 19) and are not considered further in these analyses. However, given that our objective was to develop estimates of ambient concentrations for linkage to population information to estimate exposure, concentration estimates at a spatial resolution similar to that of population data would presumably lead to a reduction in exposure misclassification.

Four candidate approaches were considered, in addition to the use of available measurement data:

- The TM5 global atmospheric model that has been previously used in a number of evaluations of air pollution control strategy scenarios (5, 20-22), (23) and model intercomparisons (5, 21, 24). While other global atmospheric models are available with similar capability to provide ozone and/or $PM_{2.5}$ concentration estimates (e.g GEOS-Chem:(25); MOZART:(26)) the general approach is similar to that of TM5 and alternative global models were not considered.
- A method (SAT) based upon satellite observations of aerosol optical depth that are related to ground-level concentrations of $PM_{2.5}$ through spatially referenced factors derived from the global chemical transport model GEOS-Chem (27)
- The GMAPS econometric model that was used in the previous global disease burden estimates (1)
- Airport observations of visual range as a surrogate for concentrations of atmospheric aerosols (28).

Significant practical limitations were identified for both GMAPS and the visual range approach for this application and were not considered further. Specifically, the GMAPS model is not applicable to rural areas or for ozone. Although the visual range data provide a

widely available and potentially useful index of outdoor $PM_{2.5}$ exposure, raw visibility data need to be filtered to eliminate and correct for weather influences (fog, precipitation, and humidity) and visual range thresholds. Further, these data would require extensive new analysis to link with available measures of $PM_{2.5}$, and are only applicable to $PM_{2.5}$, not ozone.

Therefore, we used a combination of TM5 and SAT estimates with a PM measurement database developed for this study. Only the TM5 model was used to estimate ozone concentrations and no alternative methods for ozone were evaluated. All of these approaches estimate ambient concentrations and not concentrations indoors resulting from infiltration of polluted outdoor air.

TM5

TM5 is a nested 3-dimensional global atmospheric chemistry transport model, which simulates ozone and aerosol components 1°x1° resolution. To better evaluate urban $PM_{2.5}$, a sub-grid parameterization is applied to redistribute the computed concentrations. The current model version has been included in a large number of assessments and intercomparisons (20-22, 29-35). Further details, along with a description of the urban sub-grid parameterization, and emission inputs, are given in the Supporting Information.

Satellite-Derived $PM_{2.5}$ (SAT)

In the SAT approach (27), satellite observations of Aerosol Optical Depth (AOD), a measure of light extinction by aerosols in the total atmospheric column, are used to calculate ground-level concentrations of fine particulate matter ($PM_{2.5}$). The AOD data, provided by NASA from two instruments (MODIS: (36); and MISR: (37)), indicate how aerosols modify the radiation exiting the top of the atmosphere after being scattered by the Earth's atmosphere and surface. The AOD retrievals from both instruments were combined and applied to calculate ground-level concentrations of $PM_{2.5}$:

$$\text{Satellite}-\text{derived } PM_{2.5}=\eta \cdot AOD$$

where the relationship between AOD and $PM_{2.5}$, η, accounts for local variation in vertical structure, meteorology and aerosol type. The η parameter is calculated as the coincident ratio of $PM_{2.5}$/AOD at 2° × 2.5° simulated with a global chemical transport model (GEOS-Chem, v8-01-04) (25)brought to 0.1° × 0.1° resolution using bilinear interpolation prior to multiplication with satellite AOD retrievals to estimate $PM_{2.5}$ discussed further in Supporting Information).

PM Measurement Database

Even in the case of PM for which there is a growing global database of available measurements, directly using such data incorporates complications that may be problematic for global estimates of air quality and/or its impact on health. Specifically, i) the geographic distribution of measurements is heavily biased towards North America and Europe, ii)measurement protocols and techniques are not standardized globally, with different quality control programs and different numbers of samples to arrive at annual averages, and

iii)even for measurements made by (similar) filter-based approaches, filters are equilibrated at different relative humidity conditions prior to weighing (e.g. 35%, 40%, and 50% RH in in the U.S., Canada and the EU, respectively), and therefore not completely equivalent.

Despite these limitations, surface monitoring is used in most epidemiologic studies to estimate exposure. We used existing data to assemble a georeferenced global $PM_{2.5}$ measurement database of 2005 annual average concentrations from available national/regional/local air quality monitoring reports and from published literature (Supporting Information). The database is largely based on measured $PM_{2.5}$ from North America (US and Canada), Europe (EU), Australia and New Zealand, plus limited additional measurements and $PM_{2.5}$ estimated from measured PM_{10} and $PM_{2.5}$:PM_{10} ratios for other locations.

Combining multiple methods and data sources

Both TM5 and satellite-based $PM_{2.5}$ estimates showed similar agreement with the available measurement data, possessed unique strengths and limitations, and have been used previously in impact assessments (23, 27, 38). As such, we used a simple data fusion approach to develop the final $PM_{2.5}$ estimates from the combined information sources, as described in more detail in the Supporting Information. Briefly, satellite-derived and TM5 estimates were averaged at a 0.1° × 0.1° grid cell resolution (equivalent to approximately 11 km × 11 km at the equator). For grid cells where data from surface monitors with concentrations > 10 μg/m^3 were available (either directly measured $PM_{2.5}$ or $PM_{2.5}$ estimates from measured PM_{10}) the average of the SAT and TM5 estimates (AVG) were entered into a regression model (forced through 10 μg/m3) with the surface monitoring value as the dependent variable. From this model a prediction equation: $PM_{2.5} = 1.32*AVG^{0.922}$ was derived and ultimately applied to the AVG values to produce the final concentration estimate for each grid cell. In this way, the AVG values were calibrated to the available measurements.

Surface ozone estimates were calculated with the TM5 model. The effect of urban titration by NO_x was not included in the estimates as this involves small-scale chemical processes that cannot be resolved by the model. However, the model does exhibit titration in locations with high regional NOx emissions. Epidemiologic studies of chronic exposure to ozone (15) typically use a seasonal (summer) average and we therefore aligned our estimate accordingly. Since the ozone (summer) season varies throughout the globe, we calculated a running 3-month average (of daily 1 hour max values) for each grid cell over a full year and selected the maximum of these values. Given the scarcity of surface ozone measurements throughout the world and the complexity of accessing hourly data from available monitoring sites to develop the desired metric, we did not attempt to utilize surface ozone measurements in developing the global estimates.

To estimate disease burden the concentration estimates are compared with a ‘theoretical minimum risk exposure distribution,’ which may be a single counterfactual value or a distribution (Supporting Information).

Results

$PM_{2.5}$

Figure 1 displays available 2005 annual average $PM_{2.5}$ surface monitoring (including $PM_{2.5}$ estimates from PM_{10} concentrations) for each of the 21 regions of the world. The regions are based on a combination of geography and disease burden and used in the GBD (Supporting Information). The vast majority of measured concentrations were from Europe and North America, whereas, other regions either had estimated concentrations or none whatsoever. Within this database of PM measurements, the highest directly measured annual (2005) average concentration of $PM_{2.5}$ was 58 μg/m^3 (Beijing, China), with the highest estimated (from PM_{10} measurements) concentration of 121μg/m^3 (Datong, China – a coal mining center in Shanxi Province). The lowest measured concentration was 4 μg/m^3 (Morella, Spain). Observed $PM_{2.5}$:PM_{10} ratios ranged from 0.13 (Puerto Rico) to 0.94 (North Carolina, USA).

Our $PM_{2.5}$ concentration estimates for 2005 are shown in Figure 2. Similar to other global assessments (2, 27) and the available measurements, high concentrations are evident in south and east Asia. High concentrations (annual averages > 50 μg/m^3) are also apparent in North Africa, Central Asia and Saudi Arabia, which result primarily from airborne mineral dust, rather than combustion emissions (39).

By linking our concentration estimates with urbanization measures from the GPW3 population estimates (Supporting Information), we stratified each of the 21 regions by urban and rural grid areas. Figure 3 shows examples of stratified distributions of estimated $PM_{2.5}$ annual average concentrations. Both urban and rural concentrations were higher in Asia than in North America or Europe. Urban concentrations tend to be more normally distributed and greater than the highly right-skewed rural concentrations in these regions.

Table 1 (and Figure S8, Supporting Information) describes regional population-weighted mean concentration estimates for 1990 and 2005. Differences in regional estimates for these periods are derived from changes in estimates of emissions, and may not accurately reflect localized trends in ambient concentrations. However, comparison of differences and ratios between the periods (Figure 4) indicates a general global decrease in $PM_{2.5}$ concentrations, but an increase in the global population-weighted mean concentration (Table 1). Decreases in concentrations in North America and Europe are clearly evident while increases are apparent in rapidly developing economies in East, South and Southeast Asia, which also have experienced large population growth. In some regions (e.g. Australasia and rural areas of North America) with relatively low concentrations in 1990, the apparent increases likely reflect uncertainty in the estimation rather than true increases.

An important observation (Table 1) is that the regional population-weighted concentration estimates derived from the SAT and TM5 approaches are remarkably similar, despite very different methodologies and different input parameters. The mean (absolute value) difference in 2005 across all regions was 2.7 μg/m^3, with a maximum concentration difference of 8 μg/m^3 (25%) in South Asia. In 2005, TM5 estimates were greater than the SAT estimates in 16 of the 21 regions. In 1990, the differences between the two approaches

were slightly larger with a mean difference across all regions of 3.6 μg/$m^3$and a maximum difference of 13 μg/m^3 (41%) in South Asia. As in 2005, TM5 estimated somewhat higher concentrations than did SAT in most (18 of 21) regions. Global maps of absolute and proportional differences between TM5 and SAT (Supporting Information) indicate that the largest differences are observed in desert areas, likely due to uncertainty in simulated emissions of windblown mineral dust and the impact of bright surfaces on satellite AOD retrievals. The similarity in the estimates derived from two fundamentally different approaches strengthens confidence in their use for the GBD. In a general sense, the range of concentrations derived from the two approaches provides an indication of the uncertainty related to the choice of method. Formal uncertainty analysis in the GBD incorporates this uncertainty as well as error derived from the prediction model (Supporting Information).

Ozone

As expected for this secondary pollutant, ozone concentration spatial variability is less pronounced than that of $PM_{2.5}$ (Figure 5, Table 1) and are not as systematically higher in rapidly developing countries of Asia. The highest levels are evident in North and Latin America, Europe and South and East Asia as well as parts of Africa. Ozone ratios between 1990 and 2005 (Figure 6) indicate small decreases throughout most of the world, especially in Europe and North America. Increased concentrations are evident for much of South and East Asia.

Discussion

As part of the global estimation of the disease burden attributable to outdoor air pollution, we estimated outdoor $PM_{2.5}$ and O_3 exposures for the Earth's entire human population, thereby allowing the inclusion of populations in smaller cities and rural areas in air pollution disease burden estimates for the first time, and more than doubling the number of persons to be considered in the Global Burden of Disease Project's outdoor air pollution evaluation. This was accomplished by combining two fundamentally different approaches with a database of available PM measurements to estimate $PM_{2.5}$ concentrations throughout the world at a resolution of 0.1° × 0.1°. For ozone, a single model (TM5) was used due to the absence of substantially different alternative estimation approaches and the general lack of measurement data outside of North America and the European Union for the period of interest. Future assessments of ozone, which may become increasingly relevant given projected increases, could fuse multiple chemical transport models and utilize the growing number of measurements available globally (e.g. (5)).

Our estimations of global $PM_{2.5}$ concentrations and the spatial patterns are very similar to those reported by van Donkelaar and colleagues (27), which is not surprising because our approach includes their methodology. Globally, in 2005, 89% of the world's population lived in areas where the WHO Air Quality Guideline of 10 μg/m^3 (annual average) was exceeded. In South and East Asia, this proportion was 99%, with lower proportions in Western Europe (92%) and North America (76%). Population-weighted mean concentrations exceeded the WHO Guideline in all regions of the world except Australasia, Oceania, parts of Latin America (Andean, Southern, Tropical) and southern Sub-Saharan

Africa. Globally, 32% of the population lived in areas exceeding the WHO Level 1 Interim Target of 35 μg/m^3, mainly due to high proportions in East (76% in areas exceeding the interim target) and South (26%) Asia. The ozone metric reported here was selected to conform to that used in epidemiologic analyses of chronic exposure impacts on mortality (15) and therefore differs from the WHO Air Quality Guideline (100 μg/m3 daily maximum 8-hour mean) so that direct comparisons were not made. However, in comparison to exposure levels in the Jerrett et al. (15) epidemiologic analysis, 27% of the world's population would be in the upper exposure quartile (>62.4 ppb), with 45% and 61% in the upper two (>57.4 ppb) and three (>53.1 ppb) quartiles, respectively.

Although we do not rigorously evaluate the change in estimated concentrations over the 1990 to 2005 period, our estimates are generally consistent with other reports. Specifically, we observed large reductions in ambient concentrations of $PM_{2.5}$ in Europe (40) and, to a lesser degree, in North (41) and Central America, while increased concentrations in South, East and Southeast Asia are consistent with emissions estimates (42)(43). Measured concentrations of PM_{10} from cities in these parts of Asia do, however, suggest decreases in some large cities. One possible explanation is that PM_{10} concentrations may be decreasing, while $PM_{2.5}$ levels are increasing. In addition, evidence suggests that emissions, while increasing in general, have shifted from large/capital cities to smaller cities and rural areas in this region (42). For ozone the observed patterns follow the success of air quality management programs in North America and Europe, the increases in emissions of ozone precursors in Asia (42) and increases in global background ozone concentrations in some rural regions (44). These estimates also agree with more detailed trend evaluations based on surface monitoring, including the increase in global background ozone concentrations, which is apparent in some high latitude rural regions, and from measurements at background stations on the coasts of North America and Europe (5). Decreases in South America and Central Africa are thought to result from declining biomass burning in these regions. As with other global models, our estimates of ozone do not incorporate within-city titration of ozone by NOx and may therefore over-estimate exposures for urban populations. This limitation is common to most epidemiologic studies as they typically average concentrations across multiple urban background monitors within an urban area.

Previous global health impact assessments have generally relied on chemical transport models and used emissions inventories to evaluate source-sector impacts. For example, Anenberg et al. (2) used MOZART-2 to estimate anthropogenic contributions to ozone and $PM_{2.5}$ compared with preindustrial emissions. Concentration estimates at a spatial resolution of 2.8° × 2.8° were combined with regional baseline disease incidence and demographic data to estimate the global attributable mortality Liu et al. (45) used MOZART-2 to estimate the health impacts of the inter-continental transport of $PM_{2.5}$ on mortality. Corbett and colleagues (46) used GEOS-Chem and ECHAM5/ MESSy1-MADE along with ship emissions inventories to estimate the global contribution to $PM_{2.5}$ concentrations from shipping at a resolution of ~2.5° × ~3°. Annual average shipping contributions of as much as 2 μg/m^3 were estimated. The estimated concentration increases were interpolated to 1°×1° resolution and combined with population data at the same spatial resolution to estimate the proportion of the population exposed to air pollution from shipping and associated mortality impacts. Barrett et al. (47) used GEOSChem and emissions inventories to estimate a global

contribution of aircraft emissions of <0.2 ug/m^3. Van Aardenne et al. (23) used the TM5 model to estimate air quality and health impacts related to a number of air quality and climate change policy scenarios.

While both TM5 and GEOS-Chem (used in the SAT estimates) also have the capability of estimating concentrations of PM arising only from anthropogenic source contributions, as in the above examples, we developed estimates based upon combined natural and anthropogenic source contributions. This was based on evidence of population health effects resulting from windblown mineral dust exposure (48)(49), and evidence that increasing dust concentrations can be related to anthropogenic activity (50). Further, the proportion of dust in all locations from which epidemiologic concentration response estimates were derived is not known so these response functions likely incorporate some impact of dust exposure.

While our estimation approach is conceptually similar to those described above, it included a number of significant enhancements. For $PM_{2.5}$ we used a chemical transport model combined with surface monitoring data and PM concentration estimates derived from satellite retrievals. The use of two separate approaches in combination with a prediction model to calibrate to measurements provides an indirect assessment of uncertainty in the estimates, which also incorporates the inherent uncertainty in both the satellite-derived method and TM5 (Supporting Information).In populated areas, as indicated by the population-weighted regional averages (Table 1) and the distribution of absolute and proportional differences (Supporting Information), the differences between SAT and TM5 are remarkably small as was the calibration factor introduced by including measurements. For ozone, while only a single approach was used to generate estimates, previous comparisons with surface measurements (38) indicate that monthly mean concentrations are well-characterized by TM5, except for northern India and Central-West and Southern Africa. This may reflect uncertainties in emissions or poor representation of regional ozone concentrations by the limited monitoring data available in these regions. To reduce uncertainty in future assessments, more complex data assimilation approaches could be employed. In addition, uncertainty will likely be reduced as more surface monitoring data becomes available and with improved resolution obtained from the next generation of satellite instruments.

In addition to the use of multiple data sources in our estimates, we took advantage of high resolution population data and the ability to develop concentration estimates at 0.1° × 0.1° to improve the spatial alignment of estimated concentrations with population data. From the perspective of estimating health impacts, the high spatial resolution allows us to estimate full distributions of population exposure within each region of the world. Although this improved spatial resolution is an important component of our approach, we still are likely unable to reliably estimate concentrations in smaller high concentration cities located in regions with low average concentrations due to averaging within each pixel. Together, these features were designed to reduce exposure misclassification, to provide robust estimates of global exposure and to allow for explicit estimation of uncertainty in both concentration and burden of disease estimates. The application of these estimates to both urban and rural regions of the world is a major enhancement compared to the previous estimates (1) that were restricted to large urban areas.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This study was conducted in the context of the Global Burden of Disease (GBD) 2010 Project (7). This is the first major effort since the original 1990 global burden of disease study (1) to carry out a complete systematic assessment of the data on all diseases and injuries, and produce comprehensive and comparable estimates of the burden of diseases, injuries and risk factors for multiple time periods. Members of the Outdoor Air Pollution Expert Working Group of the Global Burden of Disease Project are: H Ross Anderson (co-Chair), Markus Amann, Richard Atkinson, Michelle Bell, Michael Brauer, Bert Brunekreef, Richard Burnett, Aaron Cohen (co-Chair), Frank Dentener, Bryan Hubbell, Kan Haidong, Michal Krzyzanowski, Stephanie London, Randall Martin, Sumi Mehta, Bart Ostro, Kiran Dev Pandey, C. Arden Pope III, Beate Ritz, Isabelle Romieu, Amir Sapkota, Kirk Smith, George Thurston, Rita Van Dingenen, Aaron van Donkelaar.

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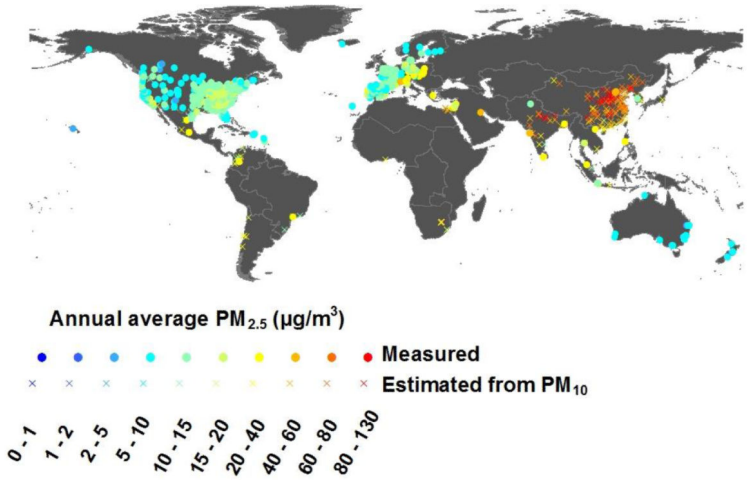

Figure 1.
Available measured (and estimated from PM_{10} measurements) annual (2005) average $PM_{2.5}$ concentrations ($\mu g/m^3$).

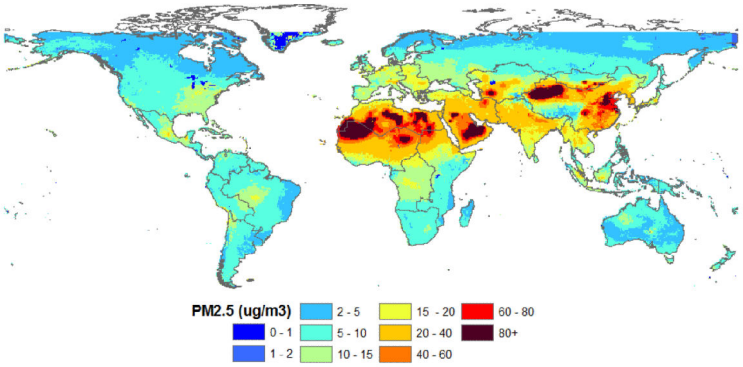

Figure 2.

Estimated 2005 annual average $PM_{2.5}$ concentrations ($\mu g/m^3$). The $PM_{2.5}$ estimates are generated from the grid cell average of SAT and TM5 and calibrated with prediction model incorporating surface measurements.

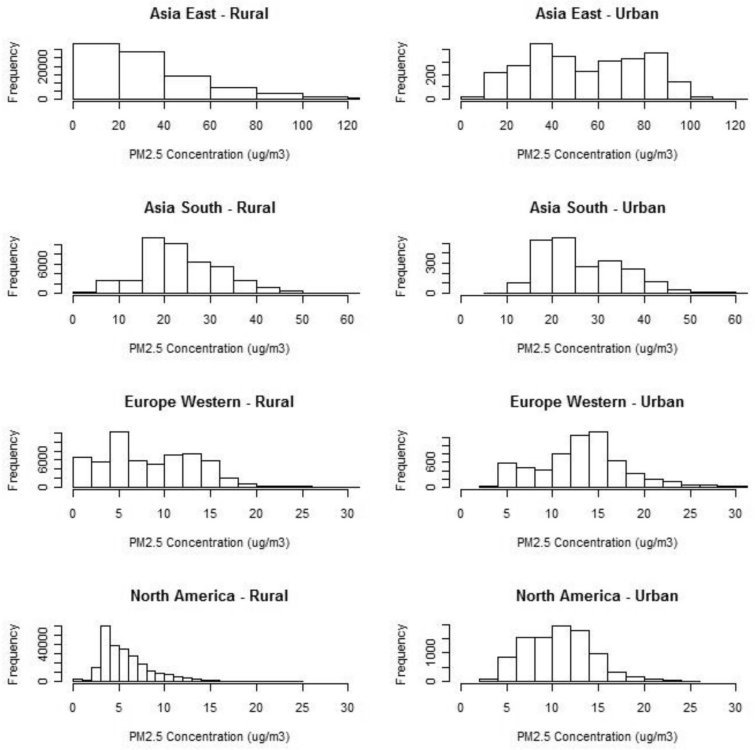

Figure 3.

Histograms of selected regional (2005) annual average $PM_{2.5}$ concentrations for urban and rural grid cells. The regions are described in the Supporting Information and the urban and rural characterization based on the GPW3 population database (Supporting Information). Frequency denotes the number of grid cells with concentrations in a given range. Note difference in scales between regions.

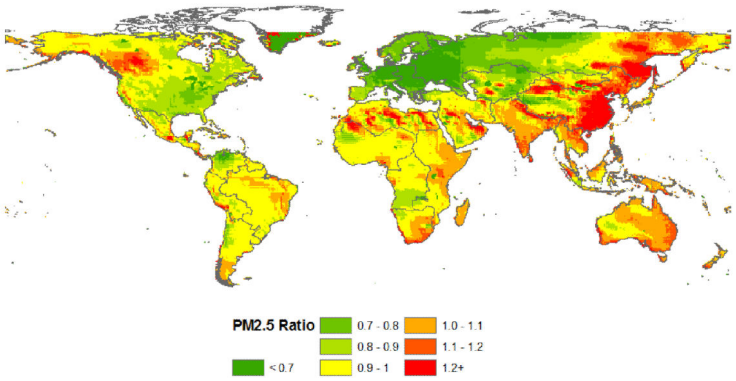

Figure 4.

Ratio of 2005:1990 annual average $PM_{2.5}$ concentrations. Concentrations are estimated to have increased in areas denoted by orange and red, while concentrations decreased in areas of yellow and green color.

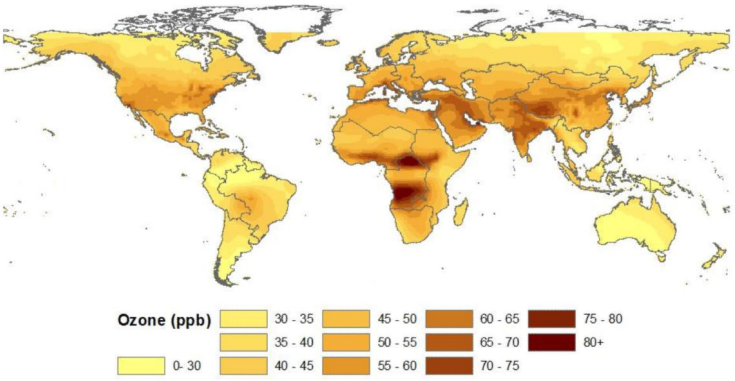

Figure 5.
Estimated (2005) seasonal (3-month) hourly maximum ozone concentrations (ppb).

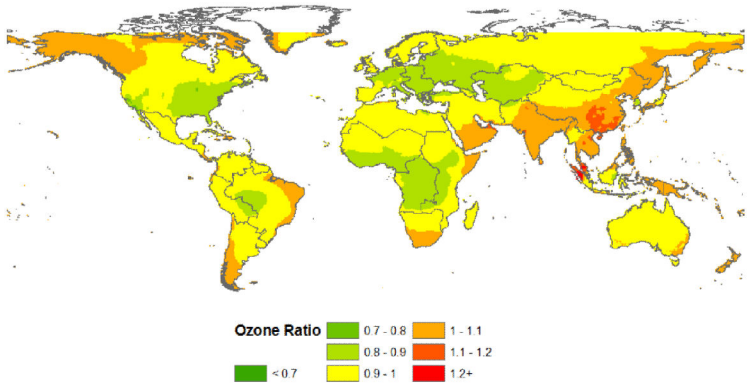

Figure 6.

Ratio of 2005:1990 seasonal (3-month) hourly maximum ozone concentrations. Concentrations are estimated to have increased in areas denoted by orange and red, while concentrations decreased in areas of yellow and green color.

Table 1

Population-weighted regional annual average $PM_{2.5}$ concentrations (μg/m^3) for 1990 and 2005 estimated from TM5, the satellite-based (SAT) method, the average of the two methods and the calibrated values (combined) approach. Population-weighted regional seasonal (3-month) hourly maximum ozone concentrations (ppb) for 1990 and 2005 from TM5. N: number of 0.1°× 0.1° grid cells per region used to estimate population- weighted concentrations. POP: regional population from summation of all grid cells within each region. The population-weighting is described in more detail in the Supporting Information.

Region	N grid cells (× 10^4)	1990 POP (× 10^6)	TM5 1990	SAT 1990	AVG 1990	$PM_{2.5}$ 1990	OZONE 1990	2005 POP (× 10^6)	TM5 2005	SAT 2005	AVG 2005	$PM_{2.5}$ 2005	OZONE 2005
Asia Pacific, High Income	0.48	160	31	23	27	27	53	170	27	20	23	24	52
Asia Central	6.7	68	23	19	21	22	60	79	20	16	18	19	53
Asia East	9.7	1200	45	42	43	43	52	1300	56	58	57	55	55
Asia South	4.5	1100	19	32	26	26	58	1400	23	31	27	28	62
Asia Southeast	3.8	430	15	14	14	15	40	550	14	15	15	16	41
Australasia	7.3	19	8	3	5	6	32	22	8	4	6	7	32
Caribbean	0.57	28	12	8	10	11	46	33	12	8	10	11	46
Europe Central	1.4	120	28	25	26	27	61	120	16	15	16	17	53
Europe Eastern	26.75	220	18	15	16	17	53	210	11	10	10	11	46
Europe Western	65	370	20	23	21	22	59	380	14	15	14	16	53
Latin America Andean	2.2	37	7	9	8	9	35	49	7	10	8	9	34
Latin America Central	3.8	160	13	12	13	14	49	220	12	11	11	12	47
Latin America Southern	3.8	47	7	7	7	8	35	57	6	6	6	7	34
Latin America Tropical	7.4	150	7	4	6	7	40	180	7	4	6	7	39
North Africa Middle East	10.80	290	27	25	26	27	65	400	26	24	25	26	62
North America, High Income	24.91	270	19	13	16	17	67	320	13	11	12	13	57
Oceania	4.4	4.9	7	4	5	6	29	6.8	7	4	5	6	29
Subsaharan Africa Central	4.0	53	14	12	13	14	75	85	13	12	12	13	65
Subsaharan Africa East	7.1	200	11	10	11	12	53	300	12	10	11	12	48
Subsaharan Africa Southern	2.7	51	8	6	7	8	50	64	9	6	7	8	49
Subsaharan Africa West	6.5	190	26	21	23	24	66	280	24	22	23	24	58
Global	141.3	5124	25	25	25	25	55	6283	26	27	27	27	54