



ENVIRONMENTAL HEALTH PERSPECTIVES

ehponline.org

Estimated Global Mortality Attributable to Smoke from Landscape Fires

**Fay H. Johnston, Sarah B. Henderson, Yang Chen,
James T. Randerson, Miriam Marlier, Ruth S. DeFries,
Patrick Kinney, David M.J.S. Bowman, Michael Brauer**

<http://dx.doi.org/10.1289/ehp.1104422>

Online 18 February 2012



NIEHS

National Institute of
Environmental Health Sciences

National Institutes of Health
U.S. Department of Health and Human Services

Estimated Global Mortality Attributable to Smoke from Landscape Fires

Authors and Affiliations

Fay H. Johnston¹, Sarah B. Henderson^{2,3,9}, Yang Chen⁴, James T. Randerson⁴, Miriam Marlier⁵, Ruth S. DeFries⁶, Patrick Kinney⁷, David M.J.S. Bowman⁸ and Michael Brauer⁹.

1. Menzies Research Institute, University of Tasmania, Australia
2. University of Tasmania, Australia
3. British Columbia Centre for Disease Control, Canada.
4. Department of Earth System Science, University of California, Irvine, USA
5. Department of Earth and Environmental Sciences, Columbia University, USA
6. Ecology, Evolution, and Environmental Biology, Columbia University, USA
7. Mailman School of Public Health, Columbia University, USA
8. School of Plant Science, University of Tasmania, Australia
9. School of Population and Public Health, The University of British Columbia, Canada

Corresponding author

Fay Johnston, MBBS, MAE, PhD

Menzies Research Institute Tasmania

Private Bag 23, Hobart, Tasmania 7000

Ph. +613 62267726, Fax. +613 6226

Email. fay.johnston@utas.edu.au

Short title: Global Mortality from Landscape Fire Smoke

Financial Disclosure: This project was supported in part by grants from the Australian Research Council (LP LP0882048) www.arc.gov.au, the University of Tasmania (J0018237) www.utas.edu.au, and the U.S. National Aeronautics and Space Administration (NASA NNX08AF64G) www.nasa.gov. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Author Contributions: FJ conceived and led the overall project. SH led the mortality analyses. JR and YC derived the exposure estimates. MM, RD and PK led the validation studies. DB and MB advised on methods and interpretation. All authors contributed the manuscript.

Competing Interests : The authors declare that no competing interests exist.

Key words: air pollution; biomass burning; carbon cycle; deforestation; global burden of disease; landscape fire smoke; mortality.

Abbreviations

AERONET Aerosol Robotic Network

AOD Aerosol Optical Depth

EPA Environmental Protection Agency

GBD Global Burden of Disease

GFED Global Fire Emission Database

GPW Gridded Population of the World

IMPROVE Interagency Monitoring of Protected Visual Environments program

LFS Landscape Fire Smoke

MODIS MODerate resolution Imaging Spectroradiometer

MISR Multi-angle Imaging SpectroRadiometer

NASA National Aeronautics and Space Administration

PM Particulate Matter

PM_{2.5} Particulate Matter with aerodynamic diameters smaller than 2.5 micrometers

WHO World Health Organisation

Abstract

Background: Forest, grass and peat fires release approximately two petagrams of carbon into the atmosphere each year, influencing weather, climate, and air quality.

Objective: To estimate the annual global mortality attributable to landscape fire smoke (LFS).

Methods: Daily and annual exposure to particulate matter < 2.5 micrometers (PM_{2.5}) from fire emissions was estimated globally for 1997-2006 by combining outputs from a chemical transport model with satellite-based observations of aerosol optical depth. In World Health Organization (WHO) subregions classified as sporadically impacted, the daily burden of mortality was estimated using previously published concentration-response coefficients for the association between short-term elevations in PM_{2.5} from LFS (contrasted with 0 µg/m³ from LFS) and all-cause mortality. In subregions classified as chronically impacted, the annual burden of mortality was estimated using the American Cancer Society study coefficient for the association between long-term PM_{2.5} exposure and all-cause mortality. The annual average PM_{2.5} estimates were contrasted with theoretical minimum (counterfactual) concentrations in each chronically impacted subregion. Sensitivity of mortality estimates to different exposure assessments, counterfactual estimates, and concentration-response functions was evaluated. Strong La Niña and El Niño years were compared to assess the influence of inter-annual climatic variability.

Results: Our principal estimate for the average mortality attributable to LFS exposure was 339,000 deaths annually. In sensitivity analyses the interquartile range of all tested estimates was 260,000 to 600,000. The regions most affected were Sub-Saharan Africa (157,000) and Southeast Asia (110,000). Estimated annual mortality during La Niña was 262,000 compared with 532,000 during El Niño.

Conclusions: Fire emissions are an important contributor to global mortality. Adverse health outcomes associated with LFS could be substantially reduced by curtailing burning of tropical rainforests, which rarely burn naturally. The large estimated influence of El Niño suggests a relationship between climate and the burden of mortality attributable to LFS.

Introduction

Landscape fires (encompassing wild and prescribed forest fires, tropical deforestation fires, peat fires, agricultural burning, and grass fires) release approximately two petagrams (1Pg = 10^{12} kg) of carbon into the atmosphere annually (van der Werf et al. 2010). These emissions affect planetary processes such as radiative forcing (which influences average global temperatures), and hydrological cycles (which influence regional cloud formation and rainfall) (Bowman et al. 2009; Cochrane and Laurance 2008; Fargione et al. 2008; Langmann et al. 2009; Tosca et al. 2010; Yokelson et al. 2007). Most emissions originate from fires set in tropical rainforests and savannas, where they cause recurrent episodes of severe pollution that affect some of the poorest regions of the world (van der Werf et al. 2010). Despite extensive literature describing the harmful effects of air pollution, the health impacts of landscape fire smoke are rarely highlighted in discussions about fires and their role in the earth system (Lohman et al. 2007).

Smoke from the combustion of biomass is composed of hundreds of chemicals, many of which are known to be harmful to human health (Naeher et al. 2007). The most important risk-related measure of smoke is particulate matter (PM) with an aerodynamic diameter smaller than 2.5 micrometers ($PM_{2.5}$). This PM primarily consists of organic carbon and black carbon components, along with smaller contributions from inorganic species (Naeher et al. 2007; Reid et al. 2005). Particulate matter is also produced by the combustion of fossil fuels, and most health evidence for $PM_{2.5}$ comes from studies in urban environments (Pope and Dockery 2006). Urban PM has been associated with a wide range of adverse health outcomes including all-cause, neonatal and cardio-respiratory mortality, exacerbations of respiratory and cardiovascular conditions, and pathophysiological changes such as inflammation, oxidative stress, and pro-coagulation (Pope and Dockery 2006). The effects of

PM derived from burning biomass has been less extensively investigated, and much of the evidence comes from studies of air pollution from household solid fuel use (Naeher et al. 2007). A handful of toxicological studies suggest that biomass smoke particles elicit pathophysiological effects similar to those of urban PM (Barregard et al. 2006; Danielsen et al. 2009; Kocbach et al. 2008). Although there are relatively few epidemiological studies on smoke-related PM, they also report outcomes consistent with those elicited by urban PM, including increased all-cause mortality, and exacerbations of respiratory conditions (Delfino et al. 2009; Hänninen et al. 2009; Johnston et al. 2007; Johnston et al. 2011; Morgan et al. 2010; Sastry 2002). However, evidence concerning cardiovascular outcomes of smoke-related PM remains scarce and inconclusive (Naeher et al. 2007; Sanhueza et al. 2009). Results from several studies of the extensive rainforest and peat fires in Southeast Asia in 1997-1998 suggest substantial health and economic impacts of landscape fire smoke (Jayachandran 2009; Mott et al. 2005; Sastry 2002; Schweithelm et al. 1999, reprinted 2006). Further, fires are becoming more widespread and frequent in some regions (Turetsky et al. 2011; Westerling et al. 2006), and this source of air pollution is likely to continue to grow in magnitude and consequent health impacts (Confalonieri et al. 2007; Denman et al. 2007; Langmann et al. 2009). Because fire emissions contribute to radiative forcing, there is potential for the development of a positive feedback between a warming climate and increasingly severe fire events in several biomes (Bowman et al. 2009). In this context a global assessment of the mortality impacts of landscape fire smoke is required.

Methods

Studying the magnitude of health impacts from landscape fire smoke presents several technical challenges, including estimation of the exposure to smoke-specific PM for each spatial unit of analysis, selection of the most appropriate concentration response functions,

and consideration of what theoretic minimum (counterfactual) exposure values to apply. Moderate to high levels of uncertainty are associated with many of these steps, so our objectives were to provide a reasonable principal estimate given the available data, and then to evaluate the sensitivity of the principal estimate to the assumptions used in the principal analysis. The World Health Organisation (WHO) Global Burden of Disease (GBD) Comparative Risk Assessment framework provides a standard set of methods for this, and has previously been used to evaluate the annual mortality attributable to urban air pollution, and to indoor air pollution from household solid fuel use (Ezzati et al. 2002; Lopez et al. 2006a). Methods for estimating the global mortality associated with particulate air pollution are being revised in the light of new epidemiological evidence and exposure assessment methods, and new cause-specific results are expected in 2012 (Institute for Health Metrics and Evaluation 2010). However, the epidemiological evidence concerning landscape fire smoke remains limited, and evidence concerning landscape fire smoke and cause-specific mortality is not currently available. For this reason our analyses evaluate all-cause mortality.

Input data

Exposure estimates

We combined information from satellite-derived observations of global fire activity, geographic area burned, and type of vegetation burned in a global atmospheric 3-Dimensional (3-D) chemical transport model. We then combined output from that model with satellite-based measurements of aerosol optical depth (AOD) to estimate annual PM_{2.5} emissions from landscape fires. The Supplemental Material contains a detailed description of

the exposure estimates (see pages 3-8, Supplemental Table 1, and Supplemental Figures 1 and 2). A summary is presented below.

Monthly-resolved emissions estimates were obtained from the Global Fire Emission Database version 2 (GFEDv2) (van der Werf et al. 2006), which combines satellite observations of burned area (in km²) with estimates of fuel loads obtained from a biogeochemical model (Giglio et al. 2006). These emissions estimates were used in the GEOS-Chem global 3-D chemical transport model, which simulates the transport, transformation, and deposition of organic carbon and black carbon aerosols. The model had a 2° (latitude) by 2.5° (longitude) horizontal resolution (approximately 222 × 278 km at the equator) and 30 vertical layers (Bey et al. 2001). We performed two sets of GEOS-Chem simulations spanning a 10-year period (1997-2006). The first included all aerosol emission sources (fossil fuel, biofuel, landscape fires, natural dust, and sea salt) while the second excluded landscape fire emissions to separate the contribution from this source.

Finally, we scaled the modelled PM_{2.5} estimates using two sets of AOD observations from the MODerate resolution Imaging Spectroradiometer (MODIS) and the Multi-angle Imaging SpectroRadiometer (MISR) aboard the US National Aeronautics and Space Administration (NASA) Terra satellite (Martonchik et al. 2009; Remer et al. 2005). We maintained the same seasonal, regional, and vertical aerosol distributions as predicted by the GEOS-Chem simulations. Our best estimate of surface PM_{2.5} (1997-2006 average shown in Figure 1) combined information from the model estimates along with the two satellite AOD-scaled estimates as described in Equation 1.

$$\text{Landscape fire smoke PM}_{2.5} = (2*\text{MODEL} + \text{MODIS} + \text{MISR})/4 \quad [1]$$

Where MODEL is the estimate of $PM_{2.5}$ from landscape fire smoke derived from GEOS-Chem, and MODIS and MISR are the two satellite AOD-scaled estimates. We multiplied the model contribution by two so that our best estimate gave equal weight to the *a priori* atmospheric model distribution and the sum of the two satellite-scaled estimates. The total aerosol emissions from fires used in the model simulations was 23.5 Tg/yr averaged over 1997-2006. Comparable estimates for the MISR and MODIS AOD-based optimizations were 55.0 and 45.5 Tg/yr, respectively (Supplemental Table 1), and were within the range of previously published estimates (Supplemental Table 2). Our best estimate, defined according to Equation 1, was 36.9 Tg/yr.

Evaluation of exposure estimates

Surface measurements of $PM_{2.5}$ are not available for most regions with high fire emissions. To evaluate the quality of the global exposure estimates we used ground-based AOD from NASA's Aerosol Robotic Network (AERONET) (Holben et al. 1998), $PM_{2.5}$ measurements from the US EPA IMPROVE (Interagency Monitoring of Protected Visual Environments) program (Chow and Watson, 2002), and visibility data in tropical regions from the National Climatic Data Center Global Summary of the Day (NOAA, 2009). Our exposure estimates correlated well with these other measures in regions with high fire activity (see Supplemental Material, pages 9-16, Supplemental Figures 3-6). Correlations (Pearson's r) of estimated AOD with monthly mean AODs from AERONET were 0.81 in Southern Africa ($n=119$), 0.90 in Northern Africa ($n=74$) and 0.76 Southeast Asia ($n=148$) (see Supplemental Figure 4). Median correlations between $PM_{2.5}$ and visibility were 0.57 for Sub-Saharan Africa ($n=58$), 0.60 for South America ($n=47$), and 0.68 for Southeast Asia ($n=13$) (Supplemental Figure 6).

Gridded mortality estimates

Country-specific estimates for all-cause all-age mortality in the year 2002 were obtained from the WHO Global Health Observatory. Estimates from the Gridded Population of the World (GPWv3) project were used to map country-specific mortality onto the $2^{\circ} \times 2.5^{\circ}$ exposure cells (Sociodemographic Data and Applications Centre 2011). The spatial resolution of the GPWv3 data is 2.5 arc-minutes (approximately 4.6×4.6 km at the equator), meaning that each exposure cell encompassed 2880 population cells. To distribute mortality between the population cells we assigned each cell to the underlying country that contained most of it, summed the GPWv3 population for each country and calculated the percent of the total population in each cell, and then assigned that percentage of the national mortality to the cell. In the <1% of cases where population cells were assigned to countries that do not belong to the WHO we followed the same steps for the 21 WHO subregions and assigned those values instead. The mortality in each exposure cell was estimated by summing the mortality in the 2880 underlying population cells.

Global burden calculations

Pattern of exposure: subregions of sporadic and chronic impact

Fire activity varied widely across the globe during the 1997-2006 period. Some areas were affected sporadically, with a limited number of smoky days in any given year; some areas were affected chronically, with whole seasons being smoke-impacted in multiple years. Our principal analysis treats these areas as fundamentally different because acute and chronic PM exposures have independent health effects (Pope and Dockery 2006; Schwartz 2000). We began by classifying each of the 21 WHO subregions as *sporadically impacted* or *chronically impacted*.

The complete set of smoke-specific $PM_{2.5}$ estimates (12 months \times 10 years \times 4208 exposure cells = 504,960) was log-normally distributed with a 90th percentile value of $3 \mu\text{g}/\text{m}^3$. When concentration estimates were rounded to integers, most exposure cells had a value of zero in most months (331,035 out of 504,960), indicating low smoke-specific $PM_{2.5}$. An exposure cell with a one-month smoke-specific $PM_{2.5}$ estimate $>3 \mu\text{g}/\text{m}^3$ was classified as being smoke-affected during that month. Exposure cells with ≥ 3 smoke-affected months in ≥ 5 of the years were classified as chronically impacted (732 out of 4,208). Exposure cells that were not chronically impacted were classified as sporadically impacted (3,476 out of 4,208). A WHO subregion was classified as chronically impacted if $>50\%$ of its population and/or $>50\%$ of its land area was covered by chronically impacted exposure cells (7 out of 21, Figure 2). All other WHO regions were classified as sporadically impacted (14 out of 21, Figure 2).

Burden for sporadically impacted subregions

For sporadically impacted subregions we estimated effects of short-term (daily) fluctuations in smoke-specific $PM_{2.5}$ concentrations on mortality. Daily output from GEOS-Chem was used to estimate the number of days per year that $PM_{2.5}$ concentrations exceeded a set of threshold values (300, 200, 100, 50, 40, 30, 20, 10, 5, 4, 3, 2 and $1 \mu\text{g}/\text{m}^3$). These threshold values were chosen to provide a range of possible concentrations for sensitivity analyses, and because they reflect clinically relevant increments ($10 \mu\text{g}/\text{m}^3$, $100 \mu\text{g}/\text{m}^3$) reported in the literature.

The annual mortality attributable to landscape fire smoke in each sporadically impacted $2^\circ \times 2.5^\circ$ exposure cell was calculated as shown in Equation 2, where: PM is one smoke-specific $PM_{2.5}$ threshold concentration out of n possible threshold values (see above); D_{PM} is the number of days between PM and the next highest concentration; M is the annual number of

deaths in the exposure cell; and RR_{SI} is a relative rate estimate for all-cause mortality due to short-term PM exposure. Although annual mortality is not evenly distributed between the 365 days of the year, there are insufficient data to estimate seasonal mortality on a global scale.

$$\text{Sporadically impacted attributable mortality} = \sum_{PM} \left[D_{PM} * \frac{M}{365} * (RR_{SI}(PM) - 1) \right] \quad [2]$$

For the principal analysis a linear RR_{SI} estimate of 0.11% (95% CI = 0 – 0.26%) per increase of $1 \mu\text{g}/\text{m}^3$ was used with minimum and maximum concentrations of 5 and $200 \mu\text{g}/\text{m}^3$. This means that cells with daily exposure estimates of $<5 \mu\text{g}/\text{m}^3$ were not included, and cells with exposure estimates $> 200 \mu\text{g}/\text{m}^3$ were fixed at a value of $200 \mu\text{g}/\text{m}^3$. The RR_{SI} was calculated using the average (weighted by the inverse of the standard errors) of values from studies reporting associations between all-cause mortality and short-term elevations of ambient PM_{10} during fire events (Morgan et al. 2010; Sastry 2002) and $\text{PM}_{2.5}$ (Hänninen et al. 2009).

Associations with ambient PM_{10} were converted to associations with $\text{PM}_{2.5}$ by assuming that 75% of all particles less than 10 microns were also less than 2.5 microns. This is halfway between the 90% ratio measured during fire events (Ward and Hardy 1991) and the 60% ratio used by Cohen *et al* (Cohen et al. 2004) in the initial GBD estimate for urban air pollution.

Burden for chronically impacted subregions

No studies have yet reported on the mortality impacts of long-term exposure to landscape fire smoke. As such, we estimated all-cause mortality in chronically impacted exposure cells by assuming the effects of smoke-related PM to be the same as those of urban PM. Specifically, for the principal analysis we assumed a linear 0.64% (95% CI = 0.35 – 0.94%) increase in

annual all-cause mortality for each $1 \mu\text{g}/\text{m}^3$ increase in the long-term smoke-specific $\text{PM}_{2.5}$ average, as reported in the American Cancer Society study on urban air pollution (Pope et al. 1995). This is one of the most conservative concentration-response estimates that has been reported in multiple studies of urban PM (Pope and Dockery 2006). The maximum concentration of effect was assumed to be $50 \mu\text{g}/\text{m}^3$. This means that cells with annual exposure estimates $>50 \mu\text{g}/\text{m}^3$ were fixed at a value of $50 \mu\text{g}/\text{m}^3$. The annual mortality attributable to landscape fire smoke in each chronically impacted exposure cell was calculated as shown in Equation 3 where: PM is the estimated average annual smoke-specific $\text{PM}_{2.5}$ concentration in the exposure cell based on estimates for 1997 through 2006; CF is the counterfactual concentration for the WHO subregion in which the exposure cell was located; M is the annual number of deaths in the exposure cell; and RR_{CF} is the relative rate of all-cause mortality for long-term PM exposure (i.e. 0.64% for the principal analysis).

$$\text{Chronically impacted attributable mortality} = M * \left((RR_{CF}(PM - CF)) - 1 \right) \quad [3]$$

The counterfactual concentration is the theoretical minimum annual smoke-specific $\text{PM}_{2.5}$ concentration under ideal conditions. For example, if landscape fires were completely eliminated worldwide, the global counterfactual value would be zero. Given that fire is a natural part of the earth system we used a more data-driven approach to set counterfactual values for chronically impacted WHO subregions. We used a subregion-wide approach because emissions from similar landscapes in neighbouring countries can vary widely due to different land management practices, so the theoretical minimum exposure estimated for a single exposure cell might not truly reflect the minimum exposure possible for that particular landscape (Bowman et al. 2011). Specifically, we determined the smallest 12-month running average smoke-specific $\text{PM}_{2.5}$ concentration for each exposure cell within a WHO subregion,

and averaged the minimum annual concentrations across all exposure cells to determine the counterfactual value for that WHO subregion.

Sensitivity analyses

There are several sources of uncertainty in our inputs, and we addressed these through multiple sensitivity analyses. First, we assumed both linear and log-linear forms for the concentration-response functions (i.e. RR_{SI} in Equation 2 and RR_{CI} in Equation 3). Although there is increasing evidence of a log-linear association for cardiovascular mortality related to urban air pollution (Pope et al. 2011), we used the linear assumption for the principal analysis because studies on the cardiovascular effects of landscape fire smoke have been inconclusive. We also tested a range of different exposure limits. For the sporadic assumption the minimum concentration was varied between 1 and 10 $\mu\text{g}/\text{m}^3$ and the maximum was varied between 50 and 300 $\mu\text{g}/\text{m}^3$. For the chronic assumption five counterfactual definitions (listed in Table 2.) were tested with maximum yearly average concentrations at 30 and 50 $\mu\text{g}/\text{m}^3$. We repeated analyses using the GEOS-Chem and satellite AOD-scaled exposure estimates separately. To assess the effect of our assumptions concerning the combination of sporadic and chronic exposures, all analyses were repeated with all subregions classified as being sporadically impacted and with all subregions being classified as chronically impacted. There is large inter-annual variation in emissions from landscape fires mostly driven by changes in climatic conditions (van der Werf et al. 2008). To assess the influence of inter-annual climatic variability analyses were repeated with concentration estimates for a strong El Niño year that occurred between September 1997 and August 1998 (inclusive) and a strong La Niña year that occurred between September 1999 and August 2000 (inclusive) (van der Werf et al. 2004).

Results

Exposure

Estimated annual average concentrations ranged from 0 to 45 $\mu\text{g}/\text{m}^3$ annually (mean = 1.8 $\mu\text{g}/\text{m}^3$) (Figure 1). The population-weighted annual average was 2.1 $\mu\text{g}/\text{m}^3$, ranging from 0.2 $\mu\text{g}/\text{m}^3$ in the Caribbean subregion to 12.2 $\mu\text{g}/\text{m}^3$ in Sub-Saharan Africa. The population-weighted average number of annual days over 5 $\mu\text{g}/\text{m}^3$ was 28, ranging from 6 in the Caribbean subregion to 141 in Sub-Saharan Africa.

Burden of mortality

Our principal estimate for the average annual mortality associated with exposure to landscape fire smoke was 339,000 world-wide, including 157,000 in Sub-Saharan Africa and 110,000 in Southeast Asia (Figure 3). The estimates for mortality due to landscape fire smoke exposure when compared with no landscape fire smoke exposure at all (i.e. a zero exposure counterfactual) were 286,000 in Sub-Saharan Africa and 119,000 in Southeast Asia, reflecting much higher background fire activity in Sub-Saharan Africa than in Southeast Asia. During the El Niño year, the estimated mortality was higher, particularly in Southeast Asia where El Niño is associated with dry conditions and more fires (Table 1).

Outputs from all tested models (N= 2,192) had a median of 379,000 and inter-quartile range of 260,000 – 600,000 (Supplemental Material, Figure 7). Results of the sensitivity analyses are shown in Table 2. If a log-linear, rather than linear, concentration response function was assumed the mortality estimates more than doubled. The results were also sensitive to the exposure estimates, the assumed pattern of exposure (sporadic vs. chronic), and the choice of the counterfactual exposure estimation, all of which caused the estimated mortality to vary between 0.41 and 1.54 times the principal estimate (Table 2). Results were minimally

influenced by the maximum and minimum exposures of effect, which caused the estimates to vary just 0.98 to 1.01 times the principal estimate (Table 2).

Discussion

Our estimate of 339,000 annual deaths attributable to exposure to landscape fire smoke is lower than estimates for urban air pollution (800,000) and much lower than estimates for household solid fuel use (1,600,000)(Lopez et al. 2006b). Similar to other environmental risk factors such as unsafe water, indoor and urban air pollution, the mortality burden attributable to landscape fire smoke falls disproportionately on low income regions of the world (Figure 4) (Ezzati et al. 2002).

The major strengths of these analyses lie in the use of existing global datasets for terrestrial fire emissions, meteorology, population density, and mortality. Using the WHO geographic subregions and mortality estimates helped make our findings comparable with previously reported estimates for other environmental risk factors. However, there are many limitations inherent in compiling and modelling data at a global scale. A major source of uncertainty comes from the emission factors for fire-derived aerosols that were used to model the exposure estimates. We used emission factors at the lower end of the range in the literature (Supplemental Table 2) even though larger emission factors have been shown to improve model estimates of PM_{2.5} as compared with satellite and surface network observations (Chin et al. 2009; Reid et al. 2009). In addition, the sum of the black carbon and organic carbon emissions factors was often lower than the observed PM_{2.5} emissions factors, likely resulting in GEOS-Chem underestimates of smoke specific PM_{2.5}. We also chose to be conservative in applying a linear concentration response function as other studies have suggested higher slopes at lower PM_{2.5} concentrations (Pope et al. 2009).

In the absence of empirical PM data for many regions most severely affected by landscape fire smoke, we evaluated our results against global datasets of visibility and ground based aerosol optical depth, both of which are proxies for particulate air pollution. Although there was considerable regional variation in the degree of correlation with these independent measures, the estimated PM_{2.5} performed comparatively well in Sub-Saharan Africa and Southeast Asia (the two global regions with highest mortality contributions). Further reductions in uncertainty of the daily exposures could be achieved with the use of higher temporal resolution fire emission inventories. For example, Mu et al. used active fire observations from Aqua, Terra, and GOES satellites to develop a daily and 3-hourly fire emissions product for the 2002-2010 period (Mu et al. 2011).

The WHO subregions with the highest mortality were those we identified as being chronically impacted by landscape fire smoke (Figure 2). The principal estimate of 339,000 annual deaths is composed of 81% mortality due to chronic exposure and 19% due to sporadic exposure. When the analysis was run under the sporadic-only and chronic-only assumptions (Table 2), WHO subregions identified as chronically-impacted contributed 53% of the total estimates (138,000 and 520,000, respectively) in both cases.

Previous estimates of the global mortality associated with urban air pollution (Cohen et al. 2005) and smoke from household solid fuel use (Lopez et al. 2006b) assumed purely chronic exposure to PM. Our distinction between chronic and sporadic impacts is a departure from this approach, reflecting the current state of epidemiologic evidence and the nature of landscape fire smoke exposure. On one hand, only a few studies have reported on the mortality effects of landscape fire smoke (Hänninen et al. 2009; Morgan et al. 2010; Sastry 2002), and all have estimated associations with short-term fluctuations in PM concentrations. On the other hand, urban air pollution studies have clearly demonstrated that chronic exposure to PM is associated with greater increases in mortality than short-term fluctuations

(Pope and Dockery 2006). Landscape fire smoke is episodic in many parts of the world, and annual average exposures are not appropriate for estimating smoke-related mortality in those regions. Similarly, fire smoke exposure is more chronic (due to high seasonal averages) in some regions and mortality estimates based on short-term fluctuations might be overly conservative. To date the short-term mortality impacts for PM from landscape fires have been consistent with those of urban PM. As such, we considered it reasonable to estimate the chronic effects of PM from landscape fire smoke using conservative values for the chronic effects of PM from urban sources until more specific evidence becomes available. We were also unable to account for different population responses to air pollution. While our coefficient for acute exposure was driven by a study in Southeast Asia, no studies conducted in Sub-Saharan Africa were available.

Estimates of counterfactual exposures are highly uncertain. Human influence on landscape fire activity varies considerably between eco-climatic regions. We set the theoretical minimum for PM_{2.5} from landscape fire smoke as the lowest estimated for each chronically impacted WHO subregion over the decade-long study period. However, variation in fire activity during the last decade will not necessarily capture the reduction in fire activity that could be achieved in each environment. For example, tropical rainforests and peat swamps, the primary source of fire emissions in Southeast Asia, rarely burn without human instigation. If such deforestation fires were to be halted, fire activity in this region (and the associated mortality) would be minimal. However, the role of human fire management in savannas, the primary source of emissions in Africa, is less well understood because fire is an integral part of these landscapes (van der Werf et al. 2008). The large estimated influence of El Niño on mortality related to landscape fire smoke implies that the burden may change in the future if climate change modifies the El Niño Southern Oscillation or drier conditions occur in places with adequate fuels and ignition sources.

Landscape fire activity has been recognized as a global-scale environmental challenge because plumes transgress international boundaries and component gases and particles contribute to climate change (Bowman et al. 2009; Pope and Dockery 2006; van der Werf et al. 2008). This first attempt to quantify the global burden of mortality attributable to landscape fire smoke has demonstrated important impacts at regional and global scales. We anticipate that subsequent estimates will be improved by better exposure assessment (particularly as empirical PM data become more globally available), further epidemiologic studies on mortality and morbidity associated with landscape fire smoke (particularly in regions with high exposure), and improved understanding of how fire regimes can be modified to reduce smoke emissions. Reducing population level exposure to air pollution from landscape fires is a worthwhile endeavour that is likely to have immediate and measurable health benefits. Such interventions could also potentially provide benefits for the mitigation of climate change and slowing the loss of biodiversity.

References

- Barregard L, Sallsten G, Gustafson P, Andersson L, Johansson L, Basu S, et al. 2006. Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. *Inhal Toxicol* 18(11):845-863.
- Bey I, Jacob DJ, Yantosca RM, Logan JA, Field BD, Fiore AM, et al. 2001. Global modeling of tropospheric chemistry with assimilated meteorology: Model description and evaluation. *Journal of Geophysical Research-Atmospheres* 106(D19):23073-23095.
- Bowman DMJS, Balch JK, Artaxo P, Bond WJ, Carlson JM, Cochrane MA, et al. 2009. Fire in the Earth System. *Science* 324(5926):481 - 484.
- Bowman DMJS, Balch J, Artaxo P, Bond WJ, Cochran MA, D'Antonio CM, et al. 2011. The human dimension of fire regimes on Earth. *Journal of Biogeography* 38(12):2223-2236.
- Chin M, Diehl T, Dubovik O, Eck TF, Holben BN, Sinyuk A, et al. 2009. Light absorption by pollution, dust and biomass burning aerosols: A global model study and evaluation with AERONET data *Annales Geophysicae* 27:3439-3464.
- Chow JC, Watson JG. 2002. PM_{2.5} carbonate concentrations at regionally representative Interagency Monitoring of Protected Visual Environment sites. *Journal of Geophysical Research-Atmospheres* 107(D21):8344.
- Cochrane MA, Laurance WF. 2008. Synergisms among Fire, Land Use, and Climate Change in the Amazon. *AMBIO: A Journal of the Human Environment* 37(7):522-527.
- Cohen AJ, Anderson HR, Ostro B, Pandey KD, Krzyzanowski M, Kuenzli N, et al. 2004. Mortality impacts of urban air pollution. In: *Comparative quantification of health risks: Global and regional burden of disease due to selected major risk factors*, Vol. 2, (Ezzati M, Lopez AD, Rodgers A, Murray CUJL, eds). Geneva:World Health Organization.
- Cohen AJ, Ross Anderson H, Ostro B, Pandey KD, Krzyzanowski M, Kunzli N, et al. 2005. The global burden of disease due to outdoor air pollution. *Journal of Toxicology & Environmental Health Part A* 68(13-14):1301-1307.
- Confalonieri U, Menne B, Akhtar R, Ebi KL, Hauengue M, Kovats RS, et al. 2007. Human health, climate change 2007: Impacts, adaptation and vulnerability. In: *Contribution of working group II to*

the fourth assessment report of the Intergovernmental Panel on Climate Change, (Parry ML, Canziani OF, Palutikof JP, van der Linden PJ, Hanson CE, eds). Cambridge, UK:Cambridge University Press, 402.

Danielsen PH, Loft S, Kocbach A, Schwarze PE, Møller P. 2009. Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines. *Mut Res-Genetic Toxicology and Environmental Mutagenesis* 674(1-2):116-122.

Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M, et al. 2009. The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. *British Medical Journal* 66(3):189.

Denman KL, Brasseur G, Chidthaisong A, Ciais P, Cox PM, Dickinson RE, et al. 2007. Couplings Between Changes in the Climate System and Biogeochemistry. In: *Climate Change 2007: The Physical Science Basis Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*, (Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, et al., eds). Cambridge, United Kingdom and New York, NY, USA.:Cambridge University Press, 555.

Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. 2002. Selected major risk factors and global and regional burden of disease. *Lancet* 360(9343):1347-1360.

Fargione J, Hill J, Tilman D, Polasky S, Hawthorne P. 2008. Land clearing and the biofuel carbon debt. *Science* 319(5867):1235.

Giglio L, van der Werf GR, Randerson JT, Collatz GJ, Kasibhatla P. 2006. Global estimation of burned area using MODIS active fire observations. *Atmospheric Chemistry and Physics* 6:957-974.

Hänninen OO, Salonen RO, Koistinen K, Lanki T, Barregard L, Jantunen M. 2009. Population exposure to fine particles and estimated excess mortality in Finland from an East European wildfire episode. *Journal of Exposure Science and Environmental Epidemiology* 19(May/June):414-422.

Holben BN, Eck TF, Slutsker I, Tanre D, Buis JP, Setzer A, et al. 1998. AERONET - A federated instrument network and data archive for aerosol characterization. *Remote Sens Environ* 66(1):1-16.

Institute for Health Metrics and Evaluation. year. *Global Burden of Diseases Study*. Available: www.globalburden.org/ [accessed 2 August 2011].

- Jayachandran S. 2009. Air Quality and Early-Life Mortality During Indonesia's Massive Wildfires in 1997. *J Human Resources* 44(4):916-954.
- Johnston FH, Bailie R, Pilotto L, Hanigan I. 2007. Ambient biomass smoke and cardio-respiratory hospital admissions. *BMC Public Health* 7(1):240.
- Johnston FH, Hanigan IC, Henderson SB, Morgan GG, Bowman DMJS. 2011. Extreme air pollution events from bushfires and dust storms and their association with mortality in Sydney, Australia 1994-2007. *Environmental Research* 111:811-816.
- Kocbach A, Namork E, Schwarze PE. 2008. Pro-inflammatory potential of wood smoke and traffic-derived particles in a monocytic cell line. *Toxicology* 247(2-3):123-132.
- Langmann B, Duncan B, Textor C, Trentmann J, van der Werf GR. 2009. Vegetation fire emissions and their impact on air pollution and climate. *Atmospheric Environment* 43(1):107-116.
- Lohman DJ, Bickford D, Sodhi NS. 2007. The burning issue. *Science* 316(5823):376.
- Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJL. 2006a. Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. *The Lancet* 367(9524):1747-1757.
- Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJL, Abramson M, eds. 2006b. *Global burden of disease and risk factors*. New York:Oxford University Press and World Bank Publications.
- Martonchik JV, Kahn RA, Diner DJ. 2009. Retrieval of aerosol properties over land using MISR observations. In: *Satellite Aerosol Remote Sensing Over Land*, (Kokhanovsky AA, de Leeuw G, eds). Berlin Springer, 267-293.
- Morgan G, Sheppard V, Khalaj B, Ayyar A, Lincoln D, Jalaludin B, et al. 2010. The effects of bushfire smoke on daily mortality and hospital admissions in Sydney, Australia, 1994 to 2002. *Epidemiology* 21(1):47-55.
- Mott JA, Mannino DM, Alverson CJ, Kiyu A, Hashim J, Lee T, et al. 2005. Cardiorespiratory hospitalizations associated with smoke exposure during the 1997, Southeast Asian forest fires. *International Journal of Hygiene and Environmental Health* 208(1-2):75-85, .

Mu M, Randerson JT, van der Werf GR, Giglio L, Kasibhatla P, Morton DC, et al. 2011. Daily and hourly variability in global fire emissions and consequences for atmospheric model predictions of carbon monoxide. . *Journal of Geophysical Research-Atmospheres* in press.

Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, et al. 2007. Woodsmoke health effects: a review. *Inhalation Toxicology* 19(1):67-106.

NOAA. 2009. National Climatic Data Center. Available at www.ncdc.noaa.gov/oa/ncdc.html Date accessed 17 May 2011.

Pope CA, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, et al. 2009. Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: shape of the exposure-response relationship. *Circulation* 120(11):941.

Pope CA, Burnett RT, Turner MC, Cohen AJ, Krewski D, Jerrett M, et al. 2011. Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: shape of the exposure-response relationships. *Environ Health Perspect*.

Pope CA, Dockery DW. 2006. 2006 Critical Review. Health effects of fine particulate air pollution: Lines that connect. *Journal of the Air and Waste Management Association* 56:709-742.

Pope CAI, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, et al. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med* 151:669-674.

Reid JS, Hyer EJ, Prins EM, Westphal DL, Zhang J, Wang J, et al. 2009. Global monitoring and forecasting of biomass-burning smoke: Description of and lessons from the Fire Locating and Modeling of Burning Emissions (FLAMBE) program. *IEEE Journal of Selected Topics in Applied Earth Observations and Remote Sensing* 2(3):144-162.

Reid JS, Koppmann R, Eck TF, Eleuterio DP. 2005. A review of biomass burning emissions part II: intensive physical properties of biomass burning particles. *Atmospheric Chemistry and Physics* 5:799-825.

Remer LA, Kaufman YJ, Tanré D, Mattoo S, Chu DA, Martins JV, et al. 2005. The MODIS Aerosol Algorithm, Products, and Validation. *Journal of the Atmospheric Sciences* 62:947-973.

Sanhueza PA, Torreblanca MA, Diaz-Robles LA, Schiappacasse LN, Silva MP, Astete TD. 2009. Particulate Air Pollution and Health Effects for Cardiovascular and Respiratory Causes in Temuco,

Chile: A Wood-Smoke-Polluted Urban Area. *Journal of the Air & Waste Management Association* 59(12):1481-1488.

Sastry N. 2002. Forest fires, air pollution, and mortality in southeast Asia. *Demography* 39(1):1-23.

Schwartz J. 2000. Harvesting and long term exposure effects in the relation between air pollution and mortality. *American Journal of Epidemiology* 151(5):440-448.

Schweithelm J, Glover D, Jessup T. 1999, reprinted 2006. Chapter six. Conclusions and Policy Recommendations. In: *Indonesia's fire and haze: The Cost of Catastrophe*, (Glover D, Jessup T, eds). Ottawa and Singapore: International Development Research Centre and the Institute of Southeast Asian Studies.

Sociodemographic Data and Applications Centre. 2011. Gridded Population of the World. Available at <http://sedac.ciesin.columbia.edu/gpw/> Date accessed 17 May 2011. Colombia University.

Tosca MG, Randerson JT, Zender CS, Flanner MG, Rasch PJ. 2010. Do biomass burning aerosols intensify drought in equatorial Asia during El Nino? *Atmospheric Chemistry and Physics* 10:3515-3528.

Turetsky MR, Kane ES, Harden JW, Ottmar RD, Manies KL, Hoy E, et al. 2011. Recent acceleration of biomass burning and carbon losses in Alaskan forests and peatlands. *Nature Geoscience* 4:27-31.

van der Werf G, Randerson J, Giglio L, Gobron N, Dolman A. 2008. Climate controls on the variability of fires in the tropics and subtropics. *Global Biogeochemical Cycles* 22(3).

van der Werf GR, Randerson JT, Collatz GJ, Giglio L, Kasibhatla PS, Arellano AF, et al. 2004. Continental-scale partitioning of fire emissions during the 1997 to 2001 El Nino/La Nina period. *Science* 303(5654):73-76.

van der Werf GR, Randerson JT, Giglio L, Collatz GJ, Kasibhatla PS, Arellano Jr AF. 2006. Interannual variability in global biomass burning emissions from 1997 to 2004. *Atmospheric Chemistry and Physics* 6:3423-3441.

van der Werf GR, Randerson JT, Giglio L, Collatz GJ, Mu M, Kasibhatla PS, et al. 2010. Global fire emissions and the contribution of deforestation, savanna, forest, agricultural, and peat fires (1997–2009). *Atmospheric Chemistry and Physics* 10:11707-11735.

Ward DE, Hardy CC. 1991. Smoke emissions from wildland fires. *Environmental International* 17:117-134.

Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW. 2006. Warming and Earlier Spring Increase Western US Forest Wildfire Activity. *Science* 313(5789):940.

Yokelson RJ, Karl T, Artaxo P, Blake DR, Christian TJ, Griffith DWT, et al. 2007. The Tropical Forest and Fire Emissions Experiment: overview and airborne fire emission factor measurements. *Atmospheric Chemistry and Physics* 7:5175-5196.

Table 1. Estimates of the global and regional annual mortality attributable to landscape fire smoke and estimates from two years that corresponded with strong El Niño and La Niña conditions. Results are shown for the three most severely smoke impacted regions. These estimates are based on the assumptions used in the principal analysis (see Table 2).

| Scenario | Global | Sub-Saharan Africa^a | Southeast Asia^b | South America^c |
|-------------------------------------------|---------------|---------------------------------------|-----------------------------------|----------------------------------|
| Annual average (1997-2006) | 339,000 | 157,000 | 110,000 | 10,000 |
| EL Niño year (September 1997-August 1998) | 532,000 | 137,000 | 296,000 | 19,000 |
| La Niña year (September 1999-August 2000) | 262,000 | 157,000 | 43,000 | 11,000 |

a WHO subregions 18-21

b WHO subregion 5 only

c WHO subregions 11-14

Table 2. Results of sensitivity analyses indicating the influence of varying individual assumptions on annual global mortality estimates. These are expressed as a ratio of the principal estimate.

| Source of uncertainty | Description of assumption used in the principal analysis followed by variations used in the sensitivity analyses. | Proportion of principal estimate of annual mortality, when all other principal analysis assumptions are held constant |
|--------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------|
| Estimated PM _{2.5} concentrations | Principal analysis: Landscape fire smoke PM _{2.5} concentrations estimated from a combination of a global chemical transport model GEOS-CHEM and satellite-derived aerosol data from MODIS and MISR $PM_{2.5} = (2 * MODEL + MODIS + MISR) / 4$ | 1.00 |
| | MODEL: PM _{2.5} concentrations estimated from the GEOS-CHEM global chemical transport model | 0.68 |
| | MODIS: MODEL estimate optimised using satellite-derived aerosol data from MODIS | 1.47 |
| | MISR: MODEL estimates optimised using satellite-derived aerosol data from MISR | 1.20 |
| Pattern of exposure | Principal analysis: Mortality in sporadically affected subregions was estimated using daily average exposure estimates and response functions. Mortality in chronically affected WHO subregions was estimated using yearly mean exposure estimates and response functions. | 1.00 |
| | Sporadic only: Mortality in all subregions was estimated using daily average exposure estimates and response functions | 0.41 |
| | Chronic only: Mortality in all subregions was estimated using yearly average exposure estimates and response functions | 1.54 |
| Shape of concentration-response function | Principal analysis: The mortality response was calculated as a linear function of the PM _{2.5} concentration | 1.00 |
| | Log-linear: The mortality response was calculated as a function of the logarithm of the PM _{2.5} concentration | 2.31 |

Table 2 (continued)

| Source of uncertainty | Description of assumption used in the principal analysis followed by variations used in the sensitivity analyses. | Proportion of principal estimate of annual mortality, when all other principal analysis assumptions are held constant |
|--------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------|
| Counterfactual exposure estimates for chronically impacted regions | Principal analysis: The counterfactual was estimated for each WHO subregion as the mean of the minimum 12-month running average smoke-specific PM _{2.5} concentration for each exposure cell within the subregion. | 1.00 |
| | Zero: A global value of 0 µg/m ³ . | 1.44 |
| | La Niña: Cell-by-cell average for a La Niña year, September 1999 - August 2000 inclusive. | 0.45 |
| | La Niña regional average: Regional average of the values from La Niña. | 0.81 |
| | Cell-by-cell minimum: Minimum of the 12-month running averages of each cell | 0.78 |
| | Cell-by-cell categorization: Global categorization of the values above at the 90 th , 97 th and 99 th percentiles, applying the average of the category to all cells in the category. | 0.82 |
| Maximum yearly average exposure used for estimating chronic mortality impacts | Principal analysis: 50 µg/m ³ was the maximum exposure used for estimating the mortality associated with chronic exposure | 1.00 |
| | 30 µg/m ³ was the maximum exposure used for estimating the mortality associated with chronic exposure | 0.99 |
| Range of minimum and maximum daily exposures used for estimating sporadic exposure impacts | Principal analysis: The range of exposure assessed was 5 (min) to 200 (max) µg/m ³ | 1.00 |
| | 10 to 100 µg/m ³ : Most restrictive range tested. | 0.98 |
| | 1 to 300 µg/m ³ : Least restrictive range tested. | 1.01 |

Figure Legends

Figure 1: Estimated annual average (1997-2006) PM_{2.5} concentrations from landscape fires, combining estimates from the GOES-Chem model with the MODIS and MISR optimizations.

Figure 2 World Health Organization subregions classified as sporadically and chronically impacted.

Subregions were classified as chronically impacted if $\geq 50\%$ of their populations and/or $\geq 50\%$ of their land areas were covered by smoke-affected exposure cells for at least three months per year for five or more years. The theoretical minimum annual average (counterfactual) concentration used for chronically impacted subregions was calculated by taking the mean of the minimum 12-month running average (over 120 months) of all exposure cells in the subregion. The remaining subregions were classified as sporadically impacted. The theoretical minimum daily average (counterfactual) concentration used for sporadically impacted subregions was zero.

Figure 3. Map of the principal estimate of the annual average (1997-2006) global mortality attributable to landscape fire smoke.

Figure 4. Annual mortality estimate for landscape fire smoke in the context of estimates for other modifiable risk factors assessed as part of the World Health Organisation Global Burden of Disease Studies. Adapted from Ezzati et al. (2002).

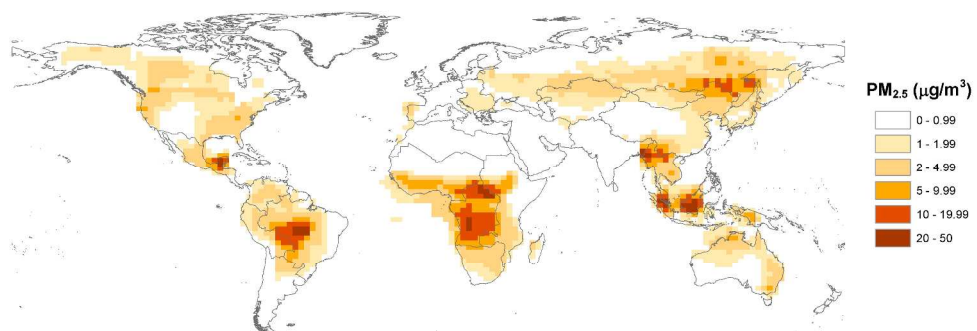


Figure 1: Estimated annual average (1997-2006) PM_{2.5} concentrations from landscape fires, combining estimates from the GOES-Chem model with the MODIS and MISR optimizations.
330x127mm (300 x 300 DPI)

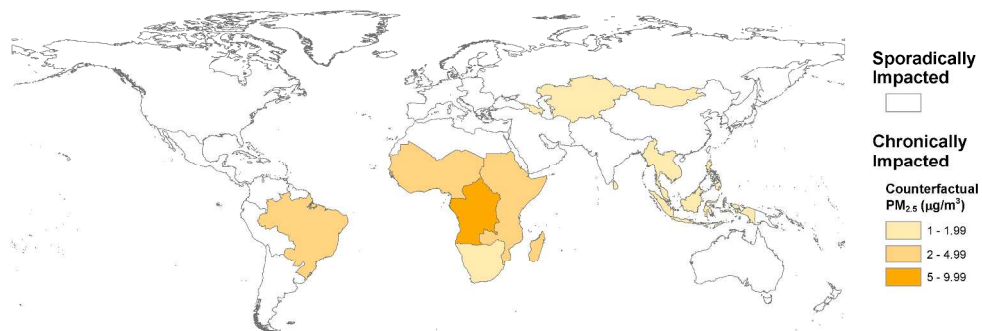


Figure 2 World Health Organization subregions classified as sporadically and chronically impacted. Subregions were classified as chronically impacted if $\geq 50\%$ of their populations and/or $\geq 50\%$ of their land areas were covered by smoke-affected exposure cells for at least three months per year for five or more years. The theoretical minimum annual average (counterfactual) concentration used for chronically impacted subregions was calculated by taking the mean of the minimum 12-month running average (over 120 months) of all exposure cells in the subregion. The remaining subregions were classified as sporadically impacted. The theoretical minimum daily average (counterfactual) concentration used for sporadically impacted subregions was zero.

330x127mm (300 x 300 DPI)

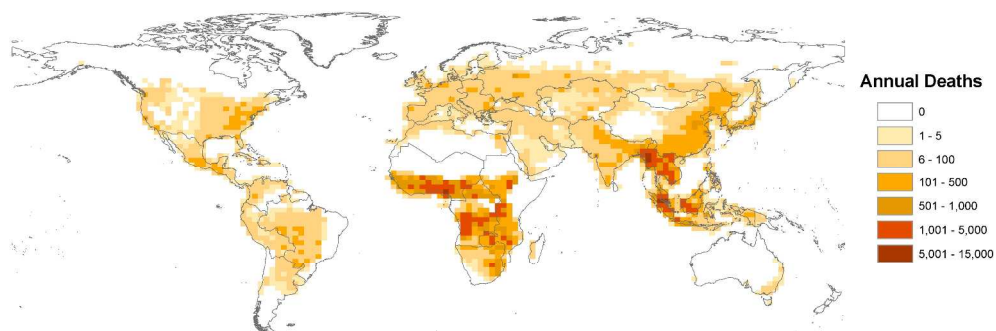


Figure 3. Map of the principal estimate of the annual average (1997-2006) global mortality attributable to landscape fire smoke.
330x127mm (300 x 300 DPI)

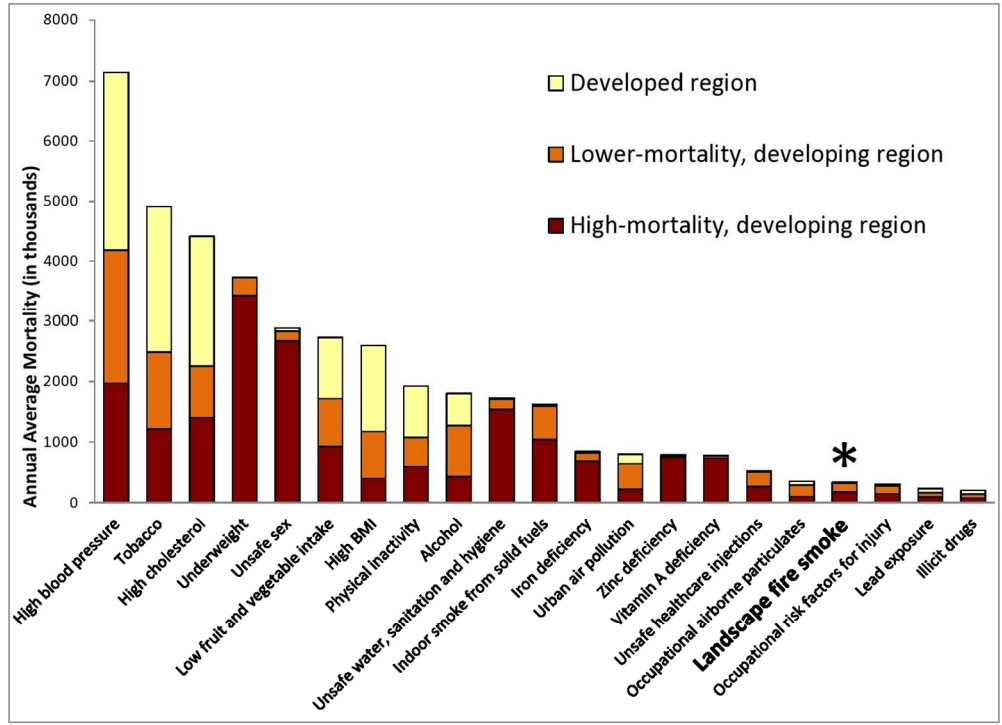


Figure 4. Annual mortality estimate for landscape fire smoke in the context of estimates for other modifiable risk factors assessed as part of the World Health Organisation Global Burden of Disease Studies. Adapted from Ezzati et al. (2002). 380x277mm (96 x 96 DPI)