El Niño and health risks from landscape fire emissions in Southeast Asia

Miriam E. Marlier[1*], Ruth S. DeFries[2], Apostolos Voulgarakis[3†], Patrick L. Kinney[4], James T. Randerson[5], Drew T. Shindell[3], Yang Chen[5], and Greg Faluvegi[3]

1. Department of Earth and Environmental Sciences, Lamont-Doherty Earth Observatory of Columbia University, Palisades, NY, 10964, USA
2. Department of Ecology, Evolution, and Environmental Biology, Columbia University, New York, NY, 10027, USA
3. NASA Goddard Institute for Space Studies and Columbia University, New York, NY, 10025, USA
4. Mailman School of Public Health, Columbia University, New York, NY, 10032, USA
5. Department of Earth System Science, University of California, Irvine, CA, 92697, USA
* Email: marlier@ldeo.columbia.edu
† Now at: Department of Physics, Imperial College London, London, SW7 2AZ, UK

Emissions from landscape fires affect both climate and air quality[1]. In this study, we combine satellite-derived fire estimates and atmospheric modeling to quantify health effects from fire emissions in Southeast Asia from 1997 to 2006. This region has large interannual variability in fire activity due to coupling between El Niño-induced droughts and anthropogenic land use change[2,3]. We show that during strong El Niño years, fires contribute up to 200 μg/m^3 and 50 ppb in annual average fine particulate matter ($PM_{2.5}$) and ozone (O_3) surface concentrations near fire sources, respectively. This corresponds to a fire contribution of 200 additional days per year that exceed the World Health Organization (WHO) 50 μg/m^3 24-hour $PM_{2.5}$ interim target (IT-2)[4] and an estimated 10,800 (6,800-14,300) person (~2%) annual increase in regional adult cardiovascular mortality. Our results indicate that reducing regional deforestation and degradation fires would improve public health along with widely established benefits from reducing carbon emissions, preserving biodiversity, and maintaining ecosystem services.

Fires are pervasive instruments of land management in the tropics for clearing debris in the process of deforestation and agricultural management. These fires enable an economical and effective method for expanding and maintaining agricultural production, but release gases (including O_3 precursors) and aerosols (mostly black and organic carbon) that interact with the climate system[5], degrade surface air quality[1], and jeopardize public health[6]. Fires associated with deforestation emitted ~1.0 Pg C/yr from 2000-10[7], with considerable interannual variability from droughts in tropical forests[8]. Fires also contribute to $PM_{2.5}$ and O_3 increases, which are both detrimental to public health[1,4].

Projections of greater fire activity in a warming climate[9] suggest increasing contributions to atmospheric concentrations and population exposure.

Globally, most fires occur in Africa and South America[8], but recent studies have highlighted the importance of Southeast Asia because of high population densities near high fire activity[10]. Regional emissions may differ by a factor of 50 between opposite phases of the El Niño-Southern Oscillation (ENSO). In the Global Fire Emissions Database version 3 (GFED3), regional fire emissions were 1069 Tg C during the 1997 El Niño but only 21 Tg C during the 2000 La Niña[8], illustrating the nonlinearity between fires and drought[11]. During the warm phase of ENSO and the Indian Ocean Dipole, cool sea surface temperature anomalies near Indonesia decrease regional rainfall[2,12]. Landowners ignite fires to clear land and manage agricultural areas[3], and although typically too wet to combust, deforestation and degradation have enhanced the susceptibility of peatland forests (with carbon-rich peat deposits) to human-ignited fire during droughts[13].

$PM_{2.5}$ and O_3 exposure increases hospital admissions and mortality from respiratory and cardiovascular diseases, even at low concentrations[4]. During the 1997-98 fires in Southeast Asia, daily ground-level PM concentrations reached hazardous levels[6], with concomitant negative impacts on respiratory and general health[14]. Increases in respiratory illnesses were also reported in Singapore from transported emissions[15]. While these studies offer some information on the health effects of fires, they have been confined to specific locations or time periods by limited data availability.

We expand on these local studies by using satellite data and two atmospheric models, NASA GISS-E2-PUCCINI general circulation model (GCM) and Harvard University's GEOS-Chem chemical transport model (CTM) to estimate pollutant concentrations and

corresponding regional mortality from 1997 to 2006, applying existing concentration-response functions from the epidemiological literature (See Methods). Atmospheric models simulate the transport of fire emissions and formation of pollutants, offer a continuous spatiotemporal dataset in a region with limited ground monitoring but large rural populations, and allow us to examine how climate and emissions influence aerosol and trace gas concentrations interannually.

Our study region is a 50°x30° area (92.5°E-142.5°E, 20°N-10°S) encompassing the Association of Southeast Asian Nations (ASEAN). In 2005, the population was approximately 540 million (Fig. 1a). Fire activity, predominately in the Indonesia islands of Sumatra and Borneo (Fig. 1b), peaks during the dry southern monsoon of September and October, along with potential spring burning[2,6].

The additional contribution of fires to annual surface $PM_{2.5}$ and O_3 concentrations in 1997, a strong El Niño year, greatly increases the number of days that exceeded the WHO interim targets of 50 μg/m^3 24-hour $PM_{2.5}$ (IT-2) and 80 ppb 8-hour maximum O_3 (IT-1), which are both twice the WHO's air quality guidelines (Fig. 2; Supplementary Table S1). In 1997, both models show two distinct areas of fire-derived $PM_{2.5}$ over Sumatra and Borneo with concentrations elevated by 50-200 μg/m^3 and with increases of 50-150 days over the WHO interim targets. O_3, in contrast, had widely distributed increases of 25-50 ppb and up to 150 exceedance days. Corresponding results with all sources in 1997 are in Supplementary Fig. S1, this simulation captured the general temporal evolution seen in ground observations (Supplementary Figs. S2, S3, S4; Supplementary Table S2).

We explored how modeled concentrations with and without fire emissions affect population exposure to WHO interim targets (Supplementary Table S1)[4]. Decadal exposure

85 over these interim targets, along with the fraction of exposure due to fire, shows how the
86 major influence of fires was not confined to the 1997-98 El Niño (Fig. 3). Interannual
87 variability in exposure for both short- and long-term guidelines is dominated by the fire
88 contribution of $PM_{2.5}$ and O_3; the WHO's 25 $\mu g/m^3$ annual $PM_{2.5}$ interim target (IT-2) is
89 never exceeded without including fire emissions.
90 We also tested the sensitivity of regional health impacts, including exceedances and
91 cardiovascular disease mortality, to using the original model or satellite-scaled model $PM_{2.5}$
92 estimates (Supplementary Figure S5). The mortality estimates combine modeled pollutant
93 concentration changes from fires with published epidemiological relationships between
94 exposure to O_3 or $PM_{2.5}$ total mass and cause-specific mortality (See Methods). In Table 1,
95 1997 and 2000 highlight the considerable differences in health effects between years with
96 high and low fire contributions. For example, $PM_{2.5}$ annual exposure in 2000 hardly
97 exceeds the WHO interim target and O_3 exposure is 100 times lower than in 1997. During
98 high fire years, fire emissions increase the adult cardiovascular disease mortality burden
99 by approximately 10,800 (6,800-14,300) annual deaths from $PM_{2.5}$ exposure and an
100 additional 4,100 (2,300-5,900) annual deaths from O_3.
101 Modeled annual adult cardiovascular disease mortality shows a strong correlation
102 with the multivariate El Niño Index (MEI)[16], which was averaged over the July to October
103 dry season (Fig. 4). We present the most conservative mortality estimates, but this
104 relationship holds with varying relative risk (RR) relationships or durations of exposure
105 (Supplementary Fig. S6). Reduced convection during El Niño years likely increases
106 exposure by elevating emissions[5,11] and increasing aerosol lifetimes by reducing wet
107 deposition..

Uncertainty in our health effect estimates comes primarily from: 1) the fire emissions dataset, 2) atmospheric modeling, and 3) concentration-response equations. First, van der Werf et al. (2010) estimated fire carbon emissions uncertainty at 20% globally, though higher in equatorial Asia due to peat carbon stock uncertainties and in years before MODIS data[8]. Second, although the lack of ground stations precludes an in-depth evaluation, available ground data and satellite AOD indicate that both models are likely conservative (Supplementary Figs. S3, S4, S5). The range between the two model scenarios ($PM_{2.5}$ and O_3) and two satellite AOD optimized results ($PM_{2.5}$ only) provide some insight about uncertainty related to transport and deposition processes. There is up to a factor of two difference between models (less among satellite-optimized estimates), but this range is expected given previous findings that model physics and parameterizations drive more variation in aerosols than emissions[17]. Differences in our $PM_{2.5}$ concentrations are primarily driven by lower precipitation and wet deposition in the GISS model, which increase aerosol lifetime relative to GEOS-Chem (data not shown). However, for the purposes of health impacts the results are much closer (Table 1). This is due to the nonlinear relationship between the RR and exposure, which reduces differences between mortality estimates at high concentrations. Finally, we address mortality equation uncertainties through 95% confidence intervals around the concentration-response estimates (Table 1) and various estimates of the RR and $PM_{2.5}$ exposure relationship (Supplementary Tables S3 and S4; Supplementary Fig. S6). Additional epidemiological factors that we did not address are extrapolation of RR equations to high concentrations and applying equations developed in the U.S. to non-U.S. populations. In addition, evidence for potential differences in $PM_{2.5}$ toxicity between urban pollution in U.S. cities and

131 Southeast Asian fire emissions is too limited to warrant using separate epidemiological
132 equations[18], so we assume that total $PM_{2.5}$ mass is the most appropriate metric.
133 These uncertainties and additional factors contribute to our substantially lower
134 regional $PM_{2.5}$ mortality estimates relative to the global analysis of Johnston et al. (2012)[10].
135 The two estimates are not directly comparable. Our conservative estimates
136 (Supplementary Table S5) are based on a tailored regional analysis for ASEAN countries
137 and use updated fire emissions, multiple atmospheric models, epidemiological equations
138 developed over a wide concentration range, and cause-specific disease estimates
139 (Supplementary Table S6). We did not include children since the epidemiological equations
140 were developed for adults over 30 years; this cuts out more than half of the population and
141 ignores risks to infants and children.
142 While previous work in Borneo has emphasized the value of avoided deforestation
143 in terms of carbon emissions[19], it is important to also account for health. By demonstrating
144 the direct link between climate variability and health impacts from fire emissions
145 throughout Southeast Asia, we offer additional support for policies that use regional
146 climate forecasts to restrict burning during high fire risk seasons. Fire emissions during
147 1997 to 2006 repeatedly exposed 1-11% of the population in Southeast Asia to $PM_{2.5}$ and
148 O_3 above WHO interim targets during El Niño years. Although the regional influence of
149 climate change is uncertain[20], these observed trends would be exacerbated by the potential
150 for more frequent droughts related to El Niño and increased baseline cardiovascular
151 disease caused by demographic shifts towards sedentary lifestyles and increased animal
152 product consumption. Reducing fires from deforestation and land management would

benefit public health in addition to global-scale benefits for carbon storage and biodiversity.

Methods

Fire emissions estimates are from GFED3, a global gridded monthly emissions dataset that combines surface reflectance and active fire detection data from several satellites to detect the spatiotemporal variability of burned area[21]. This drives a biogeochemical model that estimates fuel loads, combustion completeness, and emissions[8]. GFED3 is available since 1997 at 0.5°x0.5°. We define landscape fires to include all burning sources; in Southeast Asia this includes peat, forest, agricultural waste burning, deforestation and degradation.

We use two models: the NASA GISS-E2-PUCCINI GCM from 1997-2007 and Harvard University's GEOS-Chem CTM[22] from 1997-2006. See Supplementary Information for descriptions of the models, spin-up, and boundary conditions. Both were run at 2°x2.5°, including a control run without fire emissions and a perturbed run with GFED3 emissions. We define years from July 1st to June 30th to avoid splitting a burning season into two years. Since meteorological fields for GEOS-Chem are available through December 2006, we only have a complete 2006 "fire-year" from GISS. For $PM_{2.5}$, we analyzed 24-hour and annual average concentrations. For O_3, we used 1-2pm concentrations as a proxy for the 8-hour maximum (Supplementary Fig. S4) and 24-hour concentrations for mortality calculations.

Aerosol optical depth (AOD) data from MISR and MODIS satellite instruments, available from 2001-2006, were used to scale modeled AOD; these scaling factors were then applied to modeled $PM_{2.5}$ (Supplementary Fig. S5). While AOD represents total column

aerosol loading, it is often closely related with surface abundance[23], and hence provides some measure of large-scale biases in the models. Scaling factors were applied to surface $PM_{2.5}$ for all grid boxes, maintaining the modeled spatial and temporal distribution of aerosols.

We evaluate health effects by estimating: 1) exposure above WHO short-term and annual air quality targets, and 2) cause-specific adult mortality. Mortality attributable to fires combines the relative risk (RR) from changes in pollutant exposure with baseline observed mortality rates. We focus on cardiovascular disease because it is a proximal outcome from exposure that will be experienced annually. However, this underestimates total mortality due to other long-term effects and short-term exposure. The equations that we use were developed for adults (less than half of the regional population[24]).

We applied a power-law relationship between RR and $PM_{2.5}$. Due to the lack of data on differential health effects of biomass smoke particles[18], we use an equation developed for total $PM_{2.5}$ mass:

(1) Cardiovascular $RR=1+\alpha(I*C)^{\beta}$

which describes the relationship between $PM_{2.5}$ exposure and cardiovascular disease mortality risk over a large concentration range[25]. Pope et al. (2011) published values for α and β by reanalyzing previous estimates of RR and dose of $PM_{2.5}$ (in mg) from ambient air pollution, second-hand smoke, and cigarette smoke. For cardiovascular disease, $\alpha=0.2685$ and $\beta=0.2730$. Although ambient $PM_{2.5}$ concentrations from fires will not reach the cigarette smoke doses included in Pope et al. (2011), this equation was essential due to our high ambient concentrations above the range of other studies. Since 95% confidence intervals were given for each individual study but not the overall relationship, we refit a

power-law relationship to approximate the uncertainty based on the individual studies' upper or lower limits, respectively. The annual average of 24-hour total mass $PM_{2.5}$ concentrations were used for (C), assuming a constant average inhalation rate (I) of 18 m^3/day to convert to $PM_{2.5}$ dose (in mg)[25]. We separately calculated the RR using concentrations with and without fires due to the equation's nonlinearity. We then followed the approach of Ostro et al. (2004)[26] to calculate the attributable fraction (AF) and annual mortality (ΔM):

(2) $AF = (RR-1)/RR$

(3) $\Delta M_{annual} = M_b * P * (AF_{fire} - AF_{nofire})$

where the average annual baseline mortality rate (M_b) was calculated from adult deaths due to cardiovascular disease, averaged over the countries in ASEAN[27]. Population with ages greater than 30 years was from the UN Population Division[24] and CIESIN's Gridded Population of the World version 3 and Future Estimates, aggregated to the model resolution[28,29]; both were interpolated from 5 yearly data to annual estimates.

For O_3, the linear RR is given by:

(4) $\text{Cardiovascular RR} = \exp[\delta(C_{fire} - C_{nofire})]$

where δ=1.11 (0.68-1.53) is the percent increase in cardiovascular disease morality per 10 ppb increase in 24-hour O_3 concentrations, based on a meta-analysis of U.S. and non-U.S. studies[30]. Daily mortality due to fire pollution is then estimated with:

(5) $\Delta M_{daily} = (M_b/365) * P * (AF_{fire} - AF_{nofire})$

using the population characteristics described above. We assume that mortality is evenly spread throughout the year (M_b is not year-specific so we divide consistently by 365), and sum by days per year to obtain annual estimates. GEOS-Chem includes leap years, but GISS

uses a fixed 365 day calendar. Bell et al. (2005) concluded that the O_3 mortality burden was insensitive to PM^{30}, indicating that this is separate from $PM_{2.5}$ mortality.

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Additional Information

Correspondence and requests for materials should be addressed to M.E.M.

Acknowledgments

We are grateful to Prasad Kasibhatla for his help with the GEOS-Chem model runs. We also thank the local staffs at Bukit Koto Tabang and Tanah Rata for the WDCGG O_3 data, Michael

Brauer for the annual $PM_{2.5}$ data, and Klaus Wolter at NOAA for the El Niño index. This work was supported by a National Sciences Foundation graduate research fellowship and NASA award NNX11AF96G. GFED3 is publicly available at http://www.globalfiredata.org.

Author Contributions

R.S.D., A.V., and M.E.M. designed the study. G.F., A.V., and M.E.M. conducted the model runs, R.S.D., A.V., J.T.R., D.T.S., Y.C., and M.E.M. contributed to the model analysis, P.L.K. and M.E.M. conducted the health estimates. All authors contributed to the writing of the manuscript.

Figure 1. Study area population and locations of fire activity. a, 2005 population density, in persons per km^2, for countries belonging to the Association of Southeast Asian Nations (ASEAN). Data from CIESIN GPWv3[29] at 0.25° resolution. **b**, 1997-2006 mean fire emissions, in g C/m^2/month at 0.5° resolution, from the Global Fire Emissions Database version 3[8].

Figure 2. Modeled annual mean 1997 surface concentrations and corresponding additional daily exceedances in 1997 due to fires only. **a**, $PM_{2.5}$ **b**, O_3 annual concentrations and daily exceedances over World Health Organization (WHO) interim targets (50 μg/m^3 daily $PM_{2.5}$ (IT-2) and 80 ppb 8-hour maximum O_3 (IT-1)). Annual concentrations are from 24-hour $PM_{2.5}$ and 8-hour maximum O_3. GISS refers to GISS-E2-PUCCINI and G-C refers to GEOS-Chem.

Figure 3. Population exposure above World Health Organization (WHO) interim targets. a, Exposure over 50 μg/m^3 24-hour $PM_{2.5}$ interim target (IT-2). **b**, Exposure over 25 μg/m^3 annual $PM_{2.5}$ interim target (IT-2). **c**, Exposure over 80 ppb 8-hour maximum O_3 interim target (IT-1). **d**, Fraction of population exposure above each WHO interim target that is attributable to fires. Each case is calculated with and without GFED3 fire emissions using GISS-E2-PUCCINI results, which was close to the average concentration estimate. Refer to Supplementary Table S1 for estimated health effects. Note the logarithmic scale for (**a**) and (**c**).

Figure 4. Additional annual cardiovascular disease (CVD) mortality from exposure to fire-contributed annual $PM_{2.5}$ and 24-hour O_3, along with the Multivariate El Niño Index (MEI)[16]. Results for 1997-2006 are from the baseline GISS-E2-PUCCINI and GEOS-Chem concentrations, with the power-law RR relationship for CVD mortality. R^2=0.87-0.91 for $PM_{2.5}$ and R^2=0.82-0.89 for O_3. AOD-scaled results and sensitivity analysis are in Table 1, Supplementary Table S4, and Supplementary Fig. S6.

Table 1. Fires-only concentration, exposure, and mortality using different models for an El Niño (1997) and La Niña (2000). Average ASEAN annual concentration due to fires only (from 24-hour $PM_{2.5}$ and 8-hour maximum O_3); additional exposure due to fires above the annual 25 μg/m^3 $PM_{2.5}$ interim target (IT-2; x10^6 person-years) and above the 80 ppb daily 8-hour maximum O_3 interim target (IT-1; x10^7 person-days); cardiovascular mortality due to fires only (x10^3 people), with the range from 95% confidence intervals from epidemiological studies. GISS refers to GISS-E2-PUCCINI and G-C refers to GEOS-Chem, also with satellite scaling factors.

a) $PM_{2.5}$

	Concentration (μg/m³)		**Exposure above IT-2 (x10⁶ person-years)**		**Mortality (x10³ people)**	
	1997	*2000*	*1997*	*2000*	*1997*	*2000*
GISS	7.8	0.3	55.6	0.0	9.9 (8.0-11.4)	1.0 (0.8-1.2)
G-C	3.7	0.2	25.8	0.0	8.7 (6.8-10.7)	1.5 (1.1-1.9)
GISS MISR	10.7	0.4	57.0	0.0	11.2 (9.6-13.5)	1.3 (1.0-1.6)
G-C MISR	7.4	0.5	59.1	4.7	10.1 (8.1-11.8)	1.7 (1.4-2.1)
GISS MODIS	12.0	0.4	66.6	0.0	12.5 (10.1-14.2)	1.5 (1.1-1.8)
G-C MODIS	8.3	0.5	50.3	0.0	12.1 (9.7-14.3)	2.3 (1.8-2.8)
AVERAGE	**8.3**	**0.4**	**52.4**	**0.8**	**10.8 (6.8-14.3)***	**1.6 (0.8-2.8)***

*Maximum error range.

b) O_3

	Concentration (ppb)		**Exposure above IT-1 (x10⁶ person-days)**		**Mortality (x10³ people)**	
	1997	*2000*	*1997*	*2000*	*1997*	*2000*
GISS	9.0	1.4	395	3.5	4.3 (2.6-5.9)	1.0 (0.6-1.4)
G-C	7.1	1.3	346	0.0	3.8 (2.3-5.2)	1.0 (0.6-1.4)
AVERAGE	**8.0**	**1.4**	**371**	**1.8**	**4.1 (2.3-5.9)***	**1.0 (0.6-1.4)***