Associations between exposure to landscape fire smoke and child mortality in low-income and middle-income countries: a matched case-control study

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Summary

Background The prevalence of landscape fires has increased, particularly in low-income and middle-income countries (LMICs). We aimed to assess the impact of exposure to landscape fire smoke (LFS) on the health of children.

Methods We conducted a sibling-matched case-control study and selected 552 155 children (aged <18 years) from Demographic and Health Surveys in 55 LMICs from 2000 to 2014. Each deceased child was matched with their sibling(s). The exposure indicators were fire-sourced $PM_{2·5}$ and dry-matter emissions. We associated these exposure indicators with child mortality using conditional regressions, and derived an exposure–response function using a non-linear model. Based on the association, we quantified the global burden of fire-attributable child deaths in LMICs from 2000 to 2014.

Findings Each 1 μg/m³ increment of fire-sourced $PM_{2·5}$ was associated with a 2·31% (95% CI 1·50–3·13) increased risk of child mortality. The association was robust to different models. The exposure–response function was superlinear and suggested per-unit exposure to larger fires was more toxic. Based on our non-linear exposure–response function, we estimated that between 2000 and 2014, the five countries with the largest number of child deaths associated with fire-sourced $PM_{2·5}$ were Nigeria (164 000 [126 000 to 209 000] annual deaths), Democratic Republic of the Congo (126 000 [95% CI 114 000 to 139 000] annual deaths), India (65 900 [–22 200 to 147 000] annual deaths), Uganda (30 200 [24 500 to 36 300] annual deaths), and Indonesia (28 900 [19 100 to 38 400]).

Interpretation Exposure to landscape fire smoke contributes substantially to the global burden of child mortality.

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Introduction

The open burning of vegetation and organic soils in landscape fires causes substantial natural disturbance to earth systems, and most landscape fires are started by complex interactions between climate change and human behaviours.[1,2] Climate change and environmental change might increase the number of landscape fires worldwide.[2,3] Changes in the way that people manage land in the tropics and subtropics, where most landscape fires occur, has great potential to alter the prevalence and magnitude of these fires.[1,4,5] Landscape fires adversely affect public health in many ways, such as promoting infectious diseases via ecological effects,[6] causing direct mortality, and destroying homes. The most harmful public health effect of landscape fires is the air pollution, and exposure to toxic fine particles, they produce.[7,8] Inhalation of these particles has been linked to adverse health outcomes, such as total mortality,[9,10] cardio-respiratory diseases,[11–13] birth defects,[14] and other health impacts.[15] The global mortality burden attributable to landscape fire smoke, based on a standard exposure–response function of fine particulate matter ($PM_{2·5}$), is currently estimated to be 0·68 million deaths annually, with almost 0·27 million of these deaths occurring in children younger than 5 years.[8] However, the constituents of particles that are generated through biomass burning could have a differential toxicity compared with those from other sources, and might contribute a different magnitude of disease burden.[16,17] Therefore, deaths associated with landscape fire smoke should be re-estimated, and the exposure–response functions should be more representative of the effects of landscape fire smoke, to better assess the true magnitude of the effect on public health.

Landscape fires can cause high levels of $PM_{2·5}$ exposure over short periods of time. Compared with the type of prolonged exposure to, for example, urban air pollution, exposure to the $PM_{2·5}$ emitted by landscape fires is

Research in context

Evidence before this study

We first searched PubMed for studies examining the association between wildfire and child mortality. We used the search terms (“wildfire” OR “open fire” OR “landscape fire”) AND (“infant mortality” OR “infant death” OR “child mortality” OR “child death”) in the title or abstract, for research articles in all languages. Searching the database from inception to Nov 14, 2020, only 2 assessment studies were returned, and neither of them examined the association. Next, we widened the criteria by searching the terms (“wildfire” OR “open fire” OR “landscape fire”) AND (“mortality”) in the title or abstract. 126 items were returned and nine of them were epidemiological studies. Three of the nine studies were focused on the mortality effect of occupational smoke exposure in firefighters, and the remaining six non-occupational studies were from the USA (four), Russia (one), and Spain (one). None of these studies examined the association between mortality and fire smoke exposure in low-income and middle-income countries. A recent systematic review found a consistent association between wildfire particulate matter and the risk of death, and suggested that children or people living in low-income areas are a vulnerable population, and should be studied as a priority group.

Added value of this study

By conducting a sibling-matched case-control analysis of 55 low-income and middle-income countries (LMICs), we found a robust association between medium-term exposure to the smoke from landscape fires and child mortality. We estimated that each 1 μg/m³ increment of fire-sourced $PM_{2\cdot5}$ was associated with a 2·31% (95% CI 1·50–3·13) increased risk of child mortality. The five countries with the largest burden were Democratic Republic of the Congo (126 000 [114 000 to 139 000] annual deaths), Nigeria (164 000 [126 000 to 209 000] annual deaths), India (65 900 [–22 200 to 147 000] annual deaths), Uganda (30 200 [24 500 to 36 300] annual deaths), and Indonesia (28 900 [19 100 to 38 400]). This study assesses the burden of child mortality based on a representative exposure–response function specifically derived for landscape fire smoke exposure in LMICs for the first time.

Implications of all the available evidence

Wildfire smoke exposure contributes considerably to the global burden of child mortality. Relevant interventions and mitigations against landscape fires should be planned to protect children’s health.

typically far less frequent and discontinuous, but is often far more intense. Although uncommon, $PM_{2\cdot5}$ concentrations in regions intensely affected by fire can sometimes reach thousands of μg/m³; far in excess of what is found in even the most polluted urban areas.[18] The exposure pattern of landscape fire smoke looks similar to the smog episodes in London, UK in 1952 and Donora, PA, USA in 1948, which have been linked to irreversible health s in residents of these cities (eg, mortality).[19,20] Furthermore, exposure to landscape fire smoke more readily induces irreversible effects in more vulnerable individuals, such as pregnant women and children. Fire exposure is associated with an increased risk of low birthweight,[14] premature birth,[20,21] and child mortality.[22–24] Therefore, vulnerable subpopulations could contribute the major share of fire-attributable disease burden.

Although wildfires in high-income countries attract much media attention, low-income and middle-income countries (LMICs) have the greatest landscape fire activity by far.[1] For example, 2·43 billion km² (70%) of all 3·48 billion km² globally burned area is in Africa,[25] and areas such as the Amazon rainforest and parts of east Asia and southeast Asia are other regions of very substantial fire activity.[25] In some LMICs, open burning is commonly done as a way to clear and manage land over large areas (eg, to clear forest for agriculture or crop residues after harvest). Imposed changes to traditional land management or land use in some LMICs has altered fire regimes and might have led to more extensive or intense burning.[4] Given the poor baseline health of people living in LMICs, compared with those living in high-income countries, there could be a larger proportion of individuals vulnerable to the effects of air pollution exposure in LMICs. LMICs have a high rate of child mortality; in 99 LMICs, there were 5·0 million (95% CI 3·8–6·6) deaths of children younger than 5 years in 2017.[26] However, the association between exposure to landscape fire smoke and child deaths in LMICs is unclear. If there is such an association, the colocalisation of fire hotspots and vulnerable children in LMICs would be an important consideration for assessing the global disease burden in children related to open biomass burning.

To address this important issue, we explored the association between child mortality and exposure to landscape fire smoke in LMICs using a sibling-matched case-control study from 2000 to 2014, based on Demographic and Health Surveys (DHS) in 55 LMICs. We assessed fire exposure using the estimated surface level concentrations of $PM_{2\cdot5}$ attributable to landscape fires (fire-sourced $PM_{2\cdot5}$), satellite data of burned areas, and estimated dry-matter emission from landscape fires (ie, dry-matter fuel consumption). Using the estimated associations, we quantified the global burden of fire-attributable child deaths.

Methods

Study design and population

We used a matched case-control design to explore the association between landscape fire smoke exposure and

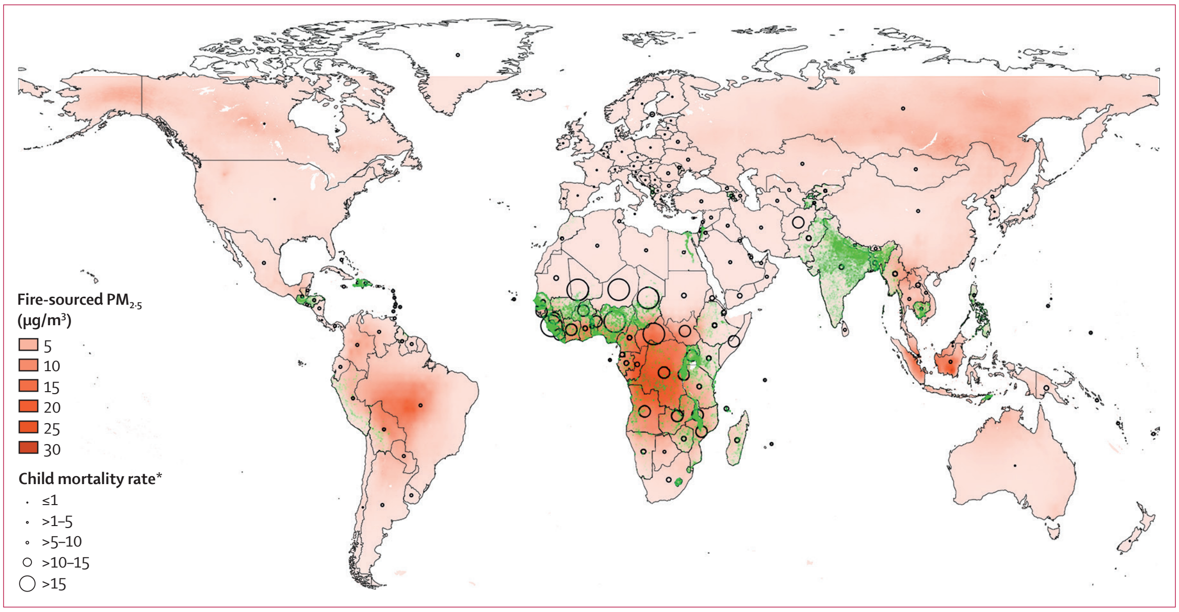

Figure 1: **Global levels of exposure to fire-sourced surface level $PM_{2·5}$ and child mortality rates, 2000–14**
Background colour represents exposure to fire-sourced surface level $PM_{2·5}$, with darker colour representing higher concentrations. Green dots represent the locations of analysed sibling-matched individual data. Black circles represent country-level or region-level total child mortality rates, with larger sizes representing higher rates. The estimates of fire-sourced $PM_{2·5}$ are not available for lowly populated northern regions. *Deaths per 1000 children.

child mortality.[27] Individual records of children (aged <18 years) and their mothers were collected from all available DHS from January, 2000, to December, 2014. DHS data are publicly available and further information is in the appendix (p 1), in our previous publications,[27] and on the DHS website. DHS are based on geocoded residences, and have been widely used in environmental health investigations. In recent surveys, the longitude and latitude of each sampling cluster (eg, residential villages) were recorded using global positioning system (GPS) devices during fieldwork. In this study, we collected all available surveys with GPS data in phases 4–7 of DHS, which covered the period of our exposure data (2000–14). The individual records of DHS contain maternal variables such as age, and child variables such as sex, birth date, survival status, and death date. We extracted individual data from the surveys relating to women who were reported as having at least one deceased child (case) and at least one alive child (control) at the time of screening. For each case, all available controls were included. On average, there were 1·9 controls matched per case. The study design is discussed in more detail in the appendix (p 2), or our previous study on fetal death.[27] Additional inclusion criteria were the availability of GPS-derived coordinates; a valid birth date of child; date of death (for cases only); and valid $PM_{2·5}$ exposure values (satellite-derived $PM_{2·5}$ surface level concentration estimates were missing for several small islands). Notably, when modelling child death, the case-control study design has a shortage: it cannot match up the age at exposure between cases and controls. Matching age-specific exposures would probably result in a highly selected sample, in which every control is required to be older than the corresponding case, to provide complete data on age-specific exposure. To avoid potential bias from such a sample selection, we have to assume the effect of landscape fire smoke is irrelevant to exposing age.

See **Online** for appendix

For more on the **DHS website** see https://www.dhsprogram.com

Procedures and outcomes

The primary indicator for landscape fire smoke exposure is the surface level concentration of fire-sourced $PM_{2·5}$, estimated from multiple sources. To separate the $PM_{2·5}$ attributable to landscape fires from other sources, we applied a zero-out approach based on GEOS-Chem (version 11.01) simulations of atmospheric components. This approach has been applied in our previous study on landscape fire smoke exposure[13] (appendix pp 1–2). Briefly, we did two GEOS-Chem model runs, identical except that the fire emissions component was switched on in the first run and switched off in the second. We estimated fire-sourced or non-fire-sourced $PM_{2·5}$ in the surface layer on the basis of a comparison between the two runs. We also obtained ambient temperature and humidity data from Modern-Era Retrospective Analysis for Research and Applications (version 2), and these monthly gridded values were downscaled into the $0·05°\times0·05°$ grid to be spatially matched with the fire-sourced $PM_{2·5}$ data.[28] We also collected two alternative indicators of landscape fire smoke: the burned area as assessed from earth observation satellite data, and the dry-matter emission from the fires derived from the

For more on **Gridded Population of the World, version 4** see https://sedac.ciesin.columbia.edu/data/collection/gpw-v4

For more on the **Global Burden of Diseases Study** see http://ghdx.healthdata.org/gbd-results-tool

global fire emission database (version 4.1s).[29] These alternative indicators provide different and complementary information about the intensity of landscape fire smoke exposure.[30] Data sources for these alternative exposure indicators are explained in the appendix (p 2). After data preparation, we assigned a monthly series of these environmental variables to each subject according to their residential address (longitude and latitude) geocoded by the DHS. A population map for the year 2000 was obtained from Gridded Population of the World, version 4. We obtained annual-level and country-level baseline mortality data in children (aged 0–18 years) for 2000–14 from the Global Burden of Diseases Study.

	Total (n=552 155)	Cases (n=188 516)	Controls (n=363 639)
Sex			
Male	283 013 (51·3%)	101 721 (54·0%)	181 292 (49·9%)
Female	269 142 (48·7%)	86 795 (46·0%)	182 347 (50·1%)
Region			
Africa, Europe, and central Asia	361 660 (65·5%)	123 583 (65·6%)	238 077 (65·5%)
East Asia and Asia-Pacific	20 839 (3·8%)	7238 (3·8%)	13 601 (3·7%)
Latin America	22 013 (4·0%)	7649 (4·1%)	14 364 (4·0%)
South Asia	147 643 (26·7%)	50 046 (26·5%)	97 597 (26·8%)
Environmental variables			
Fire-sourced $PM_{2·5}$, µg/m³	4·06 (7·95, 0·37–3·81)	4·40 (8·83, 0·24–4·06)	3·88 (7·44, 0·43–3·66)
Non-fire-sourced $PM_{2·5}$, µg/m³	44·29 (44·37, 9·83–66·64)	38·51 (39·04, 9·03–55·71)	47·28 (46·61, 10·32–74·13)
Temperature, °C	22·83 (5·76, 19·41–26·55)	24·58 (5·45, 21·73–27·77)	21·93 (5·71, 17·79–25·99)
Humidity, g/kg	11·45 (5·10, 6·55–15·83)	12·30 (4·94, 8·32–16·49)	11·01 (5·12, 5·94–15·22)
Length of survival, months	53·10 (49·76, 8–90)	11·38 (19·74, 0–13)	74·73 (46·78, 36–110)
Birth order	3·93 (2·38, 2–5)	3·56 (2·41, 2–5)	4·13 (2·34, 2–5)
Length of survival, months	53·10 (49·76, 8–90)	11·38 (19·74, 0–13)	74·73 (46·78, 36–110)
Birth order	3·93 (2·38, 2–5)	3·56 (2·41, 2–5)	4·13 (2·34, 2–5)
Birth			
Multiple birth	31 352 (5·7%)	16 991 (9·0%)	14 361 (3·9%)
Singleton	520 803 (94·3%)	171 525 (91·0%)	349 278 (96·1%)
Birth year			
2000	46 210 (8·4%)	22 168 (11·8%)	24 042 (6·6%)
2001	38 932 (7·1%)	17 052 (9·0%)	21 880 (6·0%)
2002	46 906 (8·5%)	19 110 (10·1%)	27 796 (7·6%)
2003	47 140 (8·5%)	18 025 (9·6%)	29 115 (8·0%)
2004	48 569 (8·8%)	17 713 (9·4%)	30 856 (8·5%)
2005	46 724 (8·5%)	16 090 (8·5%)	30 634 (8·4%)
2006	45 830 (8·3%)	15 181 (8·1%)	30 649 (8·4%)
2007	43 416 (8·2%)	13 776 (7·3%)	29 640 (8·2%)
2008	41 044 (7·4%)	12 311 (6·5%)	28 733 (7·9%)
2009	35 313 (6·4%)	10 329 (5·5%)	24 984 (6·9%)
2010	32 677 (5·9%)	8828 (4·7%)	23 849 (6·6%)
2011	25 955 (4·7%)	6442 (3·4%)	19 513 (5·4%)
2012	23 259 (4·2%)	5463 (2·9%)	17 796 (4·9%)
2013	17 385 (3·1%)	3881 (2·1%)	13 504 (3·7%)
2014	12 795 (2·3%)	2147 (1·1%)	10 648 (2·9%)

(Table continues on next page)

The primary timeframe for exposure to environmental variables (ie, fire-sourced $PM_{2·5}$, non-fire-sourced $PM_{2·5}$, temperature, and humidity) was selected as the month of health outcome (ie, death of the case, or month of the survey for controls), so as to include as many individuals as possible, given the small temporal coverage of the exposure data. We also set alternative timeframes: during the 2, 3, and 4 months before the health outcome, between 12 months and the date of the health outcome, the lifetime (ie, from the birth month to the month of the health outcome), the entire pregnancy (ie, the 9 months before birth of the case or control), and the lifetime plus the entire pregnancy. Our study period was determined by the simultaneous availability of the multiple inputs into the major exposure indicator (from January, 2000, to December, 2014), annual concentrations of $PM_{2·5}$ (estimated from multiple satellites from 2000),[31] and the Community Emissions Data System inventories used to simulate fire-sourced concentrations of $PM_{2·5}$ (until December, 2014). We used three binary variables as secondary outcomes: all-child mortality (deceased or not deceased before aged 18 years), infant mortality (deceased or not deceased before aged 1 year), and under-5 mortality (deceased or not deceased before aged 5 years).

Statistical analysis

According to the case-control matched design of the study, we used a conditional regression to associate environmental exposures with child mortality. The adjusted covariates were maternal age at the child's birth year, sex of the child, multiple births (yes or no), concentration of non-fire-sourced $PM_{2·5}$, splines of birth order (5 degrees of freedom [DF]), splines of temperature (3 DF), splines of humidity (3 DF), splines of calendar year (5 DF), splines of month index (4 DF), and a random effect of country-year. The non-linear effects of month controls for the seasonal periodic variation in child health, and year controls for the long-term trend in child health. The random effect captures country-specific trends. As the primary outcome, the dependent variable was child survival status. Therefore, the model was specified as a conditional Cox regression, and the association between fire-sourced $PM_{2·5}$ exposure and child mortality was evaluated by the hazard ratio (HR) for each unit increment in the exposure indicator (ie, 1 µg/m³ of fire-sourced $PM_{2·5}$, 1% of burned area as observed by satellite, or 10 g/m³ per month of dry-matter emission). For the secondary outcomes, the dependent variable was binary. Therefore, the model was specified as a conditional logit regression, and the association was evaluated using the odds ratio (OR).

In the main analysis, we applied the adjusted model to each of the 12 exposure–response pairs (3 exposures and 4 outcomes), and the HR per 1 μg/m³ increment in fire-sourced $PM_{2·5}$ was selected as the major outcome. The sensitivity analyses focused on the associations between the four child mortality metrics and concentrations of fire-sourced $PM_{2·5}$. We explored how the associations varied with different sets of adjusted covariates, different timeframes of exposure, different subpopulations, and different exposure levels (ie, the non-linear exposure–response associations). Additionally, we also estimated the associations between child mortality and four subtypes of fire (temperate forest fires; Savanna, grassland, and shrubland fires; deforestation and degradation; and agricultural waste burning), as indicated by dry-matter emissions. When multiple exposure–response pairs were tested simultaneously, the estimated CIs were adjusted using the Bonferroni approach. Because the primary hypothesis was set before performing the sensitivity analyses, we report the unadjusted CIs of the HR per 1 μg/m³ increment in fire-sourced $PM_{2·5}$. Finally, to examine whether the estimated association is attributable to landscape fire smoke or other fire-correlated factors (eg, harvesting and property loss), we defined transported landscape fire smoke as the concentration of fire-sourced $PM_{2·5}$ in pixels in which the satellite data indicated no burned area during the exposure timeframe. We repeated our linear and non-linear association models for the major health outcome using a subset, in which all cases and controls were exposed to the transported landscape fire smoke. Chemical transport models provide a comprehensive way to model how climate variables (eg, three-dimensional wind speeds) affect landscape fire smoke plumes, and so the transported landscape fire smoke variable derived from this study is estimated similarly to the wind-based instrument variable, which has been successfully used to examine the causality in the estimated effect of landscape fires.[31,32]

To evaluate child deaths attributable to landscape fire smoke exposure, we calculated the attributable fraction (AF) as: $AF=1–1÷f$, in which f(fire-sourced $PM_{2·5}$) denotes the non-linear exposure–response function, linking HR and the monthly surface level concentration of fire-sourced $PM_{2·5}$ derived from GEOS-Chem. We applied the approach by pixels, and then derived the population-weighted AF (PAF) in each country. We calculated the number of fire-attributed deaths using the annual-level and country-level baseline mortalities in children (aged 0–18 years) in 2000–14 multiplied by PAF. Uncertainty ranges were quantified using Monte Carlo simulations.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

	Total (n=552 155)	Cases (n=188 516)	Controls (n=363 639)
(Continued from previous page)			
Residence			
Rural	433 818 (78·6%)	147 201 (78·1%)	286 617 (78·8%)
Urban	118 337 (21·4%)	41 315 (21·9%)	77 022 (21·2%)
Education			
None	283 884 (51·4%)	96 242 (51·1%)	187 642 (51·6%)
Primary	173 980 (31·5%)	58 688 (31·1%)	115 292 (31·7%)
Secondary	85 332 (15·5%)	30 290 (16·1%)	55 042 (15·1%)
Higher	8940 (1·6%)	3291 (1·7%)	5649 (1·6%)
Unknown	19 (<0·1%)	5 (<0·1%)	14 (<0·1%)
Sex of head of household*			
Female	92 940 (16·8%)	32 511 (17·2%)	60 429 (16·6%)
Male	459 215 (83·2%)	15 6005 (82·8%)	303 210 (83·4%)
Household wealth quintile†			
1	183 286 (33·2%)	61 044 (32·4%)	122 242 (33·6%)
2	138 659 (25·1%)	47 323 (25·1%)	91 336 (25·1%)
3	105 177 (19·0%)	36 113 (19·2%)	69 064 (19·0%)
4	78 798 (14·3%)	27 522 (14·6%)	51 276 (14·1%)
5	45 875 (8·3%)	16 334 (8·7%)	29 541 (8·1%)
Unknown	360 (<0·1%)	180 (0·1%)	180 (<0·1%)
Type of cooking energy used			
Unclean	486 251 (88·1%)	164 925 (87·5%)	321 326 (88·4%)
Clean	50 160 (9·1%)	17 799 (9·4%)	32 361 (8·9%)
Unknown	15 744 (2·9%)	5792 (3·1%)	9952 (2·7%)
Mother's age at birth, years	26·01 (6·38, 21–30)	25·22 (6·59, 20–30)	26·42 (6·23, 22–31)
Mother's body-mass index			
Underweight	57 338 (10·4%)	19 780 (10·5%)	37 558 (10·3%)
Healthy	235 806 (42·7%)	81 071 (43·0%)	154 735 (42·6%)
Overweight	58 659 (10·6%)	20 242 (10·7%0	38 417 (10·6%)
Obese	22 637 (4·1%)	7811 (4·1%)	14 826 (4·1%)
Unknown	177 715 (32·2%)	59 612 (31·6%)	118 103 (32·5%)
Mother's smoking status			
Never smoker	433 155 (78·4%)	148 009 (78·5%)	285 146 (78·4%)
Smokes	41 469 (7·5%)	14 273 (7·6%)	27 196 (7·5%)
Unknown	77 531 (1·4%)	26 234 (13·9%)	51 297 (14·1%)
Mother's employment status			
Employed	291 482 (52·8%)	99 747 (52·9%)	191 735 (52·7%)
Unemployed	125 657 (22·8%)	42 621 (22·6%)	83 036 (22·8%)
Unknown	135 016 (24·5%)	46 148 (24·5%)	88 868 (24·4%)
Mother's parity status			
Multiparous	471 466 (85·4%)	145 759 (77·3%)	325 707 (89·6%)
Nulliparous	80 689 (14·6%)	42 757 (22·7%)	37 932 (10·4%)

Data are n (%), n (SD, IQR), or n (SD, 2·5–97·5% percentile range). IQRs are presented for all continuous variables, except for dry-matter emission and satellite burn area; for these two variables, 2·5–97·5% percentile ranges are presented because their IQR boundaries equal zero due to highly skewed distributions. Percentages might not sum to 100% due to rounding. *As decided by the respondents. †1=poorest, 5=richest.

Table: **Population characteristics of cases and controls**

Results

On May 8, 2020, we collected 552 155 child records affiliated with 149 173 mothers from 132 individual surveys of 55 LMIC countries, from January, 2000, to

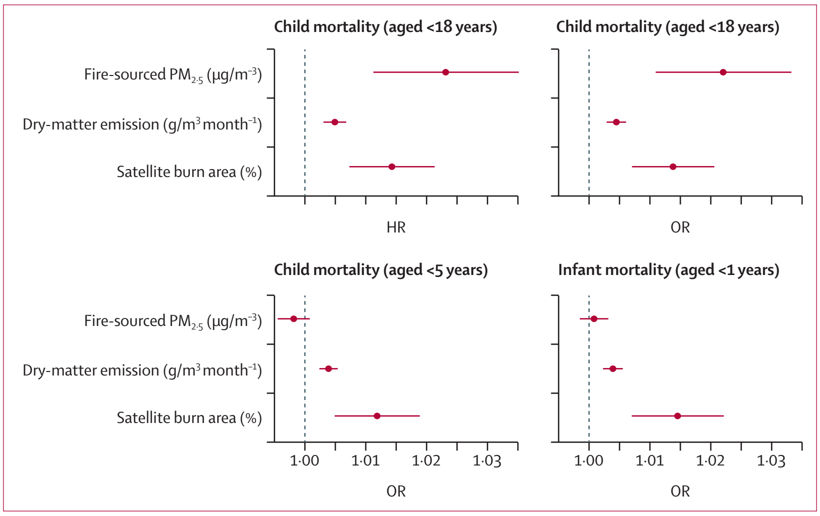

Figure 2: **Estimated associations between monthly exposure to landscape fire smoke and child mortality**
Figure shows hazard ratio (HR) or odds ratio (OR) per unit increment in exposure indicator. CIs were corrected using the Bonferroni approach.

December, 2014 (figure 1; table; appendix pp 6–7). Of the 552 155 child records, 188 516 (34·1%) recorded a death (cases). 101721 (54·0%) of 188 516 cases and 181 292 (49·9%) of 363 639 controls were male. The mean age of cases was 11 months (SD 20 months) and the mean age of controls was 6 years and 3 months (SD 3 years and 11 months). We observed hotspots of landscape fire smoke exposure in areas of low latitude, including in the Congo river basin (Africa), the Amazon (South America), and east Asia (eg, India; figure 1). The analysed individual data covered all three of these hotspots, particularly the Congo river basin. The mean concentration of fire-sourced $PM_{2·5}$ that cases were exposed to at the month of death was 4·40 μg/m³ (SD 8·83; IQR 0·24–4·06); whereas, for the controls, the mean level at the month the survey was taken was 3·88 μg/m³ (7·44; 0·43–3·66).

After adjusting for the multiple potential confounders, each 1 μg/m³ increment in monthly exposure to fire-sourced $PM_{2·5}$ was associated with an increment of 2·31% (95% CI 1·50–3·13%) in child mortality. The association between middle-term fire-sourced $PM_{2·5}$ exposure and child death was robust to differences in adjusted covariates (appendix p 16), exposure timeframes (appendix p 16), exposure indicators (figure 2), and assumption of a linear or non-linear relationship (figure 3). When the health outcome was alternatively modelled as a binary variable instead of the survival duration, the estimated associations were not considerably changed (figure 2). According to stratification analyses (appendix p 17), the association was significant in all subpopulations, except for the children of unemployed mothers (HR 1·01; 95% CI directly estimated as 1·00–1·02, but widened to 0·99–1·02 after Bonferroni correction for multiple effect modifiers) and children from Latin America (HR 1·05; 95% CI 0·95–1·15, before Bonferroni). The large uncertainty range for the estimates in Latin America were caused by the small sample size of individual data. Although there might be spatial heterogeneity of the association, because of the sibling-matched design, the sample size by country was relatively small (appendix pp 6–7) and so we were not able to further explore the geographical variations in the effect of landscape fire smoke. Of note, all of the major subtypes of fires were consistently associated with child mortality (appendix pp 2–3, 17). The non-linear analysis indicates a superlinear curvature of the association (figure 3), suggesting that the effect of landscape fire smoke on child mortality increases as the concentration increases. This finding was consistent with the strong association estimated when using the satellite or dry-matter emission indicator for exposure, or the non-significant results estimated via long-term exposure to fire-sourced $PM_{2·5}$ (appendix p 3). Additionally, we found a potential association between child mortality and maternal exposure to landscape fire smoke; each 1 μg/m³ increment in exposure to fire-sourced $PM_{2·5}$ during pregnancy was associated with increased child mortality with an HR of 1·00 (95% CI 1·00–1·01 [without Bonferroni correction] and 95% CI 0·998–1·009 [with Bonferroni correction]). The effect of maternal exposure to landscape fire smoke should therefore be further explored.

Although infant or under-5 child mortality was potentially linked to exposure to landscape fire smoke, particularly when quantified using satellite-derived burned area and dry-matter emission data (figure 2), the association was sensitive to model settings, such as the adjusted covariates (appendix p 16) and exposure timeframes (appendix p 16). These uncertainties might be caused by small sample sizes of individual data relating to infants or children younger than 5 years. Therefore, the effects need to be further examined using other data.

Given that the association between total $PM_{2·5}$ exposure and child mortality has been explored using DHS data in detail by previous studies,[33] we didn't focus on such analyses in this study. For instance, we can repeatedly examine the association between total $PM_{2·5}$ and infant mortality using our sibling-matched model, which reports an OR (0·94; 95% CI 0·84–1·06 for a 10 μg/m³ increment of $PM_{2·5}$) comparable to previous estimates.[33] We only used the effect of non-fire-sourced $PM_{2·5}$ as a referent to evaluate the exposure–response function for fire-sourced $PM_{2·5}$ exposure (appendix p 18). Considering different timeframes of effective exposure, we compared the non-linear associations of monthly and lifelong exposure to $PM_{2·5}$ from landscape fire smoke with monthly and lifelong exposure to $PM_{2·5}$ from non-fire sources. We found very weak associations for non-fire-sourced $PM_{2·5}$ exposure or total $PM_{2·5}$ exposure, which supports previous results.[33] Therefore, we only included fire-sourced $PM_{2·5}$ exposure in the following risk assessment. We restricted the individual data to those who had been exposed to transported landscape fire smoke only, and found similar associations (appendix p 19). On the basis of the fully adjusted linear models, the HR was estimated as 1·03 (95% CI 1·02–1·04), which is comparable to the result based on general landscape fire smoke (1·02, 1·02–1·03). The comparison suggests that airborne exposure to landscape fire smoke might be the major effective pathway on child mortality.

Based on the estimated non-linear exposure–response function (figure 3), between 2000 and 2014, we estimated that global exposure to fire-sourced $PM_{2·5}$ contributed 12·91 million (95% CI 11·40–14·46) premature deaths, 0·86 million (95% CI 0·76–0·96) child deaths per year, or 9·1% (8·0–10·1) of all child mortality (142 million [95% 142–143]). Of these total deaths, 5·42 million (5·10–5·82) occurred in low-income countries, 6·53 million (5·05–7·95) in lower-middle-income countries, 0·92 million (0·59–1·26) in upper-middle-income countries, and 0·04 million (0·01–0·07) in high-income countries. 12·87 million (11·35–14·40; 99·7%) of all 12·91 million deaths were due to exposure to fire-sourced $PM_{2·5}$ in LMICs. The five countries with the largest burden were Democratic Republic of the Congo (126 000 [114 000 to 139 000] annual deaths), Nigeria (164 000 [126 000 to 209 000] annual deaths), India (65 900 [–22 200 to 147 000] annual deaths), Uganda (30 200 [24 500 to 36 300] annual deaths), and Indonesia (28 900 [19 100 to 38 400]) annual deaths (appendix pp 8–14).

Because of the improvement in baseline child health from 2000 to 2014, the total number of fire-attributable child deaths showed a decreasing trend (figure 4; appendix p 19). However, the PAF remained at similar levels, and in fact slightly increased, from 8·9% (4·7–13·1) in 2000 to 9·3% (5·2–13·4) in 2014. Although the trend in total number of fire-attributable deaths varied between regions (figure 4), the PAF, as well as the concentration of fire-sourced $PM_{2·5}$ exposure (appendix p 19), fluctuated during the 15 years. These temporal trends suggest landscape fire smoke exposure could be a neglected risk factor in previous public interventions on child health.

The fire-attributable child deaths show geographical inequality. First, the fire events themselves were unevenly distributed between different regions (figure 1), and the effects of landscape fire smoke are insubstantial in many countries, where large fires occurred infrequently (appendix pp 9–14). Based on the estimated exposure–response curve, only the fires that generated high concentrations of $PM_{2·5}$ substantially contributed to child deaths (figure 3). For the countries that have a low level of exposure to fire-sourced $PM_{2·5}$, there is a large range of uncertainty around the estimated numbers of attributable deaths. Second, most countries that are threatened by exposure to fire-sourced $PM_{2·5}$ are LMICs, and thus have rather scarce resources with which to reduce their baseline risk of child mortality, which

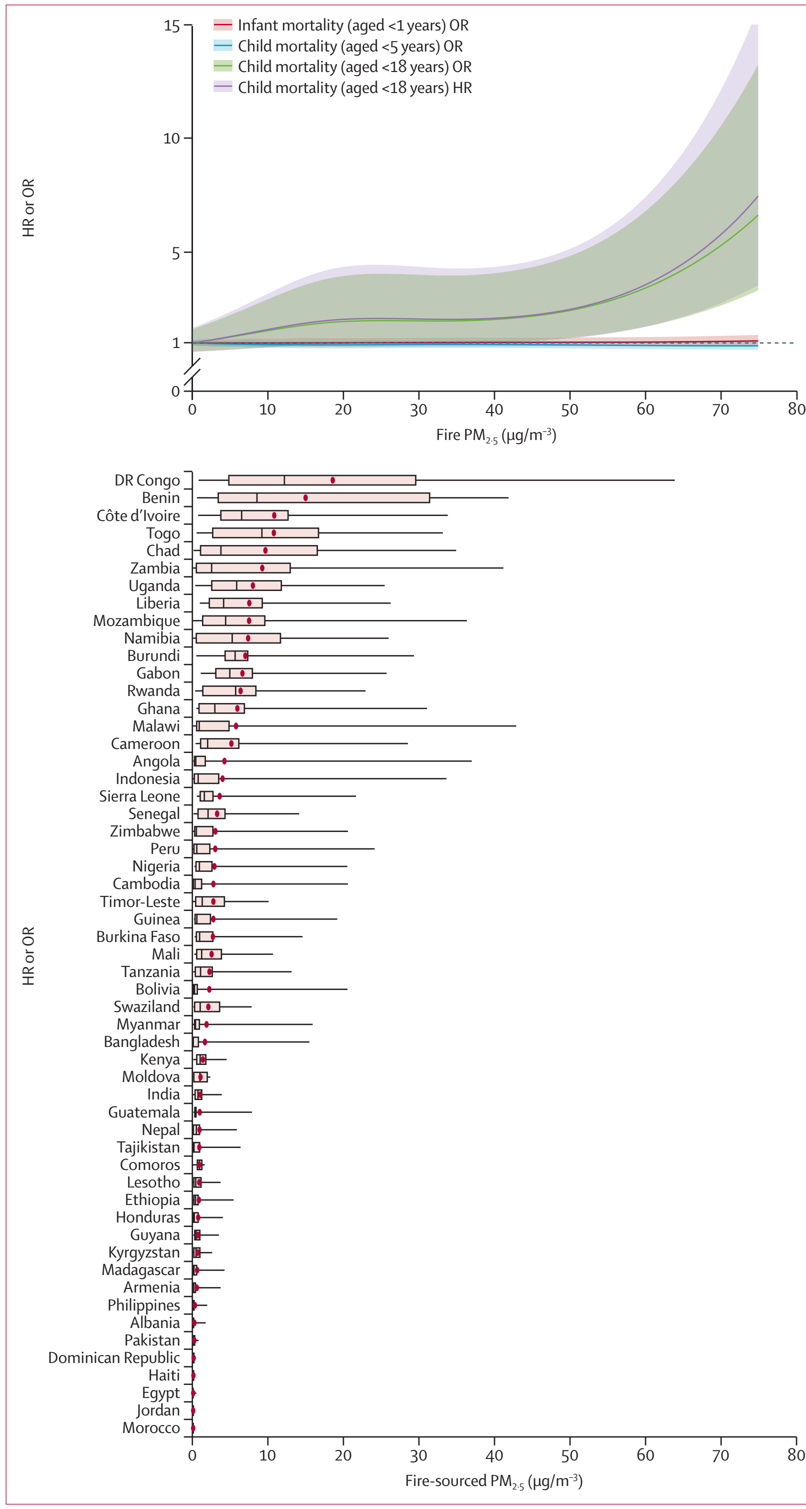

Figure 3: **Exposure–response functions between fire-sourced $PM_{2·5}$ and child mortality, estimated by non-linear models**

Boxplots represent the country-specific ranges of exposure concentrations in the analysed samples. HR=hazard ratio. OR=Odds ratio.

means that the absolute effect of fire-sourced $PM_{2\cdot5}$ on child mortality is increased. For instance, the share of the global burden borne by high-income and upper-middle-income countries decreased from 9·1% to 6·6% between 2000 and 2014. In 2000, 14·4% of fire-associated child deaths occurred in the most intense exposure hotspot, Democratic Republic of the Congo, which accounts for 1·12% of the global child population. In 2014, this contribution increased to 15·7%; a result of unequal progress in child health. However, there was a lower concentration of fire-sourced $PM_{2\cdot5}$ in 2014 than in 2000 (population-weighted concentration decreased from 24·1 μg/m³ in 2000 to 17·9 μg/m³ in 2014).

Discussion

We did a sibling-matched case-control study of the association between exposure to fire-sourced $PM_{2\cdot5}$ and child mortality. We found that landscape fire smoke significantly and robustly increased the risk of premature death in children in LMICs. This study is the first to establish a representative exposure–response function between child mortality and landscape fire smoke exposure in LMICs. Few reports have associated landscape fire smoke exposure with child mortality or other relevant early-life outcomes.[14,22–24,34,35] An unpublished study reported that, compared with unexposed controls, exposure to five or more upwind agricultural fires increases mortality by more than 2·72 neonatal deaths, 3·03 infant deaths, and 2·98 under-5 deaths per 1000 births.[34] The extreme Indonesian fires of 1997 were some of the most severe and polluting landscape fires ever witnessed,[36,37] and were estimated to be associated with 15 600 infant and fetal deaths.[23] In Kuala Lumpur, Malaysia, 14 fire-caused smoky days between 1994 and 1997 (defined by visibility <0·91 km) were also significantly associated with infant mortality, with an adjusted relative risk of 1·65 (SD 0·37).[22] By contrast, no significant association between landscape fire smoke exposure and non-traumatic mortality was detected in a 2006–17 case-crossover study in Washington, WA, USA in children aged 0–4 years (OR 0·97 [95% CI 0·77–1·22]) or in children aged 5–14 years (OR 0·95 [0·64–1·41]).[24] The weak associations in the Washington study might be a result of the relatively small sample size in the subgroup analyses and should not be interpreted as evidence against an effect of landscape fire smoke on

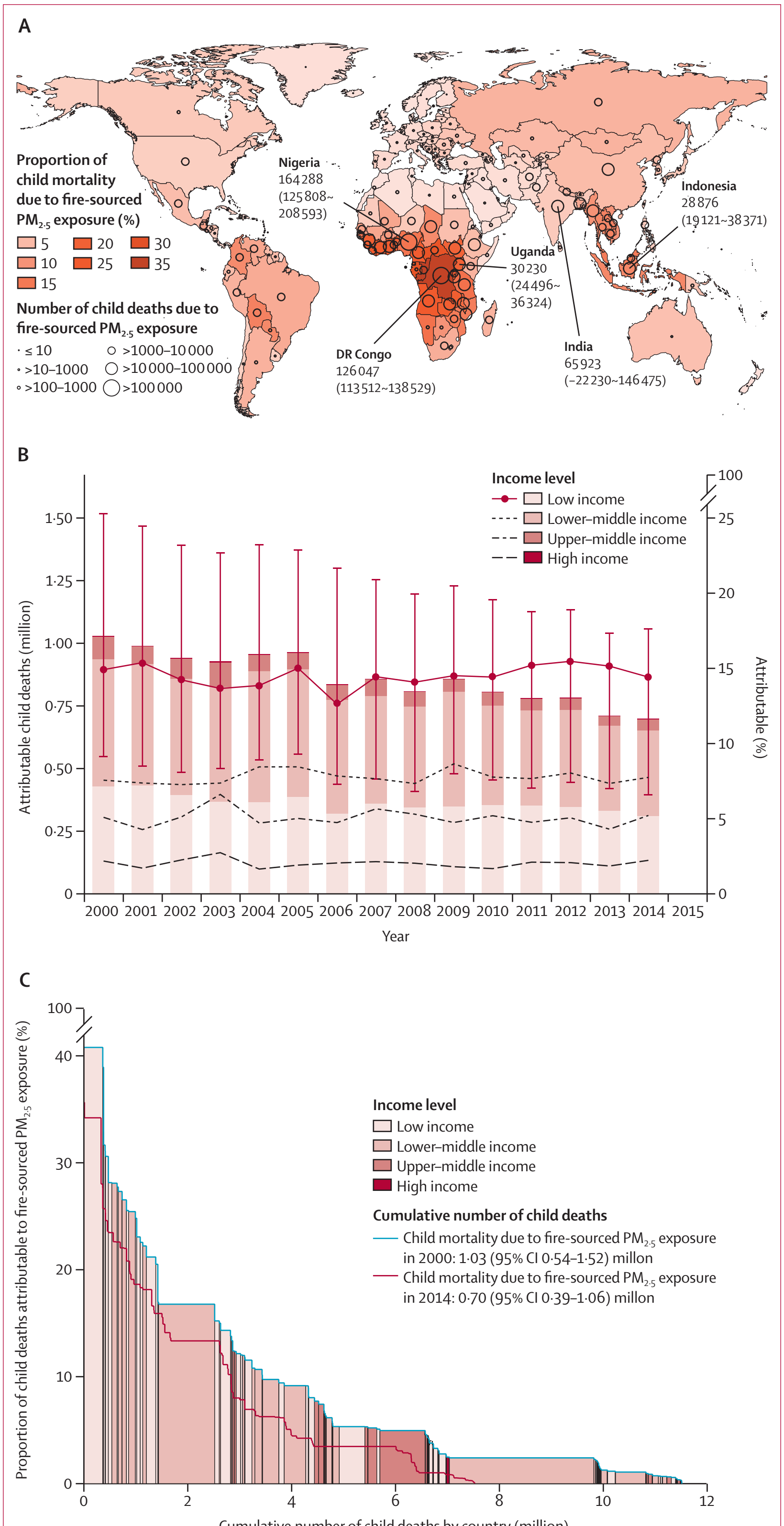

Figure 4: **Global burden of child deaths attributable to fire-sourced $PM_{2\cdot5}$ exposure, from 2000 to 2014.**
(A) Global map of the burden, with the percentage of fire-attributable child deaths as a proportion of total child deaths per country, and the number of total child deaths per country, with five hotspot areas indicated. (B) Temporal trends in the annual numbers (columns with error bars) or percentages (lines) of child deaths attributable to fire-sourced $PM_{2\cdot5}$ by income level. (C) Distribution of the burden in 2000 and 2014 by country, in which each country is represented by a bar and the area of each bar is proportional to the annual number of fire-associated child deaths. Relative distributions of these burdens are in the appendix (p 20).

child mortality. Therefore, our results are comparable with previous findings, and the association we found was robust to the use of different model settings, subgroups, and exposure timeframes. Previous reports of the acute effects of landscape fire smoke on eye and respiratory symptoms or adverse birth outcomes (eg, low birthweight) in children[14,35] suggest the underlying mechanisms of the effect, and indicate that an effect on mortality is biologically plausible. Total mortality has been well associated with landscape fire smoke exposure (appendix p 3), which also aligns with our findings.

Previous assessments of the global mortality burden of landscape fire smoke were based on the exposure–response associations of common particulate matters (eg, $PM_{2·5}$ or PM_{10}), rather than those related to fire-specific particles. For instance, a 2021 study estimated that landscape fire smoke contributed 0·68 million deaths per year.[8] However, particulate matters are mixtures of various chemical components, with toxicities that can vary depending on their source.[17] Particles emitted by the open burning of vegetation and organic soil are rich in toxic components such as black carbon and polycyclic aromatic hydrocarbons, with their exact nature depending on the type of fuel and combustion that is occurring.[18,38] Therefore, the effect of per-unit exposure to fire-sourced $PM_{2·5}$ might be stronger than the effect of exposure to non-fire-sourced $PM_{2·5}$. Two studies from 2018 and 2020 estimated the association between long-term $PM_{2·5}$ exposure and infant mortality by performing a cross-sectional analysis of the DHS database, and reported an OR of 0·99 (95% CI 0·89–1·12) for each 10 μg/m³ increment in total $PM_{2·5}$.[33,39] The authors of these studies also found that carbonaceous $PM_{2·5}$ was more toxic than dust and sea salt particles. We repeatedly examined the association between total $PM_{2·5}$ and infant mortality using our epidemiological model of DHS and estimated the OR as 0·94 (0·84–1·06), which is consistent with the finding of the previous studies. In comparison, the OR for infant death was estimated by the same model as 1·15 (0·97–1·36) for each 10 μg/m³ in lifelong exposure to fire-sourced $PM_{2·5}$. Although long-term exposure to fire-sourced $PM_{2·5}$ was not significantly associated with infant death, its effect size was larger than that of exposure to general $PM_{2·5}$. Therefore, exposure–response functions based on total $PM_{2·5}$ might underestimate the disease burden of landscape fire smoke exposure. Our analysis has developed the first tool to evaluate the specific effect of landscape fire smoke on child health in LMICs.

Most research to date has focused on the public health effect of landscape fire smoke in developed countries. However, most fires occur in LMICs, and in this study we found that over 99% of fire-associated child deaths occurred in LMICs. Given the colocalisation of high levels of landscape fire smoke exposure and poor baseline public health (appendix p 21), LMICs might also have high rates of deaths attributable to other fire-related diseases, such as respiratory diseases in adults. The unequal development rates between LMICs and high-income countries has the potential to enlarge the gap in public health and the share of the burden of disease attributable to open biomass burning. Therefore, future epidemiological studies should be done in LMICs.

Environmental change and modifications to the way people manage land is a key driver of open biomass burning in LMICs. Climate change might also alter some of the drivers of landscape fires, such as the frequency and intensity of the tropical droughts that are induced by the El Niño climate pattern.[40] Therefore, fire-attributable diseases in LMICs need to be evaluated to better mitigate the effects of climate and environmental change on health.

Prolonged exposure to $PM_{2·5}$, found in urban air pollution for example, certainly affects long-term health, whereas fire-sourced $PM_{2·5}$ concentrations peak intermittently and so might effect health in the short or medium term. For instance, in our study, child mortality was more strongly associated with exposure to fire-sourced $PM_{2·5}$ on a monthly scale than it was on an annual scale. Therefore, prevention strategies to reduce exposure to landscape fire smoke must be different from those focusing on other types of $PM_{2·5}$ pollution. Because the adverse effects of fire-sourced smoke can occur within a narrow timeframe, the wearing of effective face masks, the limiting of outdoor activities, and the temporary migration of the vulnerable out of the most affected areas can be effective in reducing the health effects. However, here, we examined only the effect of fire-sourced $PM_{2·5}$ on child mortality, and so the effective timeframes for other health outcomes should be explored when planning interventions.

This study had several limitations. First, although we controlled for potential confounders (eg, adjusting for multiple covariates and matching samples by their mothers), risk factors for child mortality could have been missed, hampering the analysis of the causality of the associations. Detailed discussions on the potential confounders are documented in the appendix (p 4). Second, as stated in the methods section, the matched design, which helps to control for unmeasured confounders, means we have been unable to explore age-specific effects of $PM_{2·5}$ in this study. Third, although we assessed landscape fire smoke exposure using three indicators, none were perfect. Any one indicator could overlook the mechanisms underlying the association between landscape fire smoke exposure and child mortality. For instance, in the estimates of fire-sourced $PM_{2·5}$ concentrations, the uncertainty embedded within the GEOS-Chem model simulations could result in exposure misclassifications. Future studies should explore how different exposure assessments, such as chemical transport model simulations based on different fire inventories (eg, the Quick Fire Emissions Dataset biomass burning emissions), will affect the association between landscape fire smoke and child death. Fourth, the health outcomes were self-reported, so could potentially be influenced by recall bias. These issues might cause misclassification of health outcomes, which would also bias the estimated associations. Fifth, although previous studies[32] suggest that evaluating the mortality risk of fire on a monthly scale is appropriate, such analyses can ignore detailed temporal patterns on the effect of landscape fire smoke. For instance, because of the low time-resolution in our health data, we were unable to examine whether there was a lag (eg, a few days) between landscape fire smoke exposure and child death. Finally, because the exposure–response association was derived from LMICs, it might be not be fully representative of high-income countries. Although landscape fires mainly occur in LMICs, leading to the vast majority of fire-associated child mortality, the global assessment results should be interpreted cautiously. Further epidemiological studies should be done in high-income countries to accurately assess the global burden.

Contributors

TX and TZ were responsible for study design and conception. TX, GG, JL, QG, HW, and BJ were responsible for data preparation. TX, GG, and JL were responsible for data analysis. TX, YH, FJK, MJW, BW, and XD were responsible for interpreting the results. TX, GG, JL, and TZ wrote the first draft. YH, FJK, and MJW wrote the second draft. All coauthors revised the manuscript together and approved the final version. TX, GG, JL, and QG had access to and verified the underlying data. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Declaration of interests

We declare no competing interests.

Data sharing

All datasets relevant to this study are publicly available. DHS data, satellite fire data (MODIS MCD64A1), global fire emission database (GFED, version 4.1s), anthropogenic emission inventory of Community Emissions Data System (CEDS), Modern-Era Retrospective analysis for Research and Applications Version 2 (MERRA-2) data, and satellite-based $PM_{2·5}$ data are available from https://www.dhsprogram.com/, https://lpdaacsvc.cr.usgs.gov/appeears/, http://globalfiredata.org/, https://esgf-node.llnl.gov/search/input4mips/, https://disc.gsfc.nasa.gov/, and http://fizz.phys.dal.ca/~atmos/martin/?page_id=140, respectively. The fire $PM_{2·5}$ data that support the findings of this study are derived from the GFED, CEDS, MERRA-2, and satellite-based $PM_{2·5}$ data using a standard model of GEOS-Chem (version 11–01, freely available from http://acmg.seas.harvard.edu/geos/).

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References

1 Hantson S, Pueyo S, Chuvieco E. Global fire size distribution is driven by human impact and climate. *Glob Ecol Biogeogr* 2015; **24:** 77–86.
2 Bowman DM, Kolden CA, Abatzoglou JT, et al. Vegetation fires in the Anthropocene. *Nat Rev Earth Environ* 2020; **1:** 500–15.
3 Sun Q, Miao C, Hanel M, et al. Global heat stress on health, wildfires, and agricultural crops under different levels of climate warming. *Environ Int* 2019; **128:** 125–36.
4 Sloan S, Locatelli B, Wooster MJ, Gaveau DLA. Fire activity in Borneo driven by industrial land conversion and drought during El Nio periods, 1982–2010. *Glob Environ Change* 2017; **47:** 95–109.
5 Bonilla-Aldana DK, Suárez JA, Franco-Paredes C, et al. Brazil burning! What is the potential impact of the Amazon wildfires on vector-borne and zoonotic emerging diseases? A statement from an international experts meeting. *Travel Med Infect Dis* 2019; **31:** 101474.
6 Hahn MB, Gangnon RE, Barcellos C, Asner GP, Patz JA. Influence of deforestation, logging, and fire on malaria in the Brazilian Amazon. *PLoS One* 2014; **9:** 1–8.
7 Xu R, Yu P, Abramson MJ, et al. Wildfires, global climate change, and human health. *N Engl J Med* 2020; **383:** 2173–81.
8 Roberts G, Wooster MJ. Global impact of landscape fire emissions on surface level $PM_{2·5}$ concentrations, air quality exposure and population mortality. *Atmos Environ* 2021; **252:** 118210.
9 Kollanus V, Prank M, Gens A, et al. Mortality due to vegetation fire-originated $PM_{2·5}$ exposure in Europe-assessment for the years 2005 and 2008. *Environ Health Perspect* 2017; **125:** 30–37.
10 Faustini A, Alessandrini ER, Pey J, et al. Short-term effects of particulate matter on mortality during forest fires in Southern Europe: results of the MED-PARTICLES Project. *Occup Environ Med* 2015; **72:** 323–29.
11 Wettstein ZS, Hoshiko S, Fahimi J, Harrison RJ, Cascio WE, Rappold AG. Cardiovascular and cerebrovascular emergency department visits associated with wildfire smoke exposure in California in 2015. *J Am Heart Assoc* 2018; **7:** e007492.
12 Alman BL, Pfister G, Hao H, et al. The association of wildfire smoke with respiratory and cardiovascular emergency department visits in Colorado in 2012: a case crossover study. *Environ Health* 2016; **15:** 64.
13 Stowell JD, Geng G, Saikawa E, et al. Associations of wildfire smoke $PM_{2·5}$ exposure with cardiorespiratory events in Colorado 2011–2014. *Environ Int* 2019; **133:** 105151.
14 Cândido da Silva AM, Moi GP, Mattos IE, Hacon SS. Low birth weight at term and the presence of fine particulate matter and carbon monoxide in the Brazilian Amazon: a population-based retrospective cohort study. *BMC Pregnancy Childbirth* 2014; **14:** 309.
15 Reid CE, Brauer M, Johnston FH, Jerrett M, Balmes JR, Elliott CT. Critical review of health impacts of wildfire smoke exposure. *Environ Health Perspect* 2016; **124:** 1334–43.
16 Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature* 2015; **525:** 367–71.
17 Kelly FJ, Fussell JC. Global nature of airborne particle toxicity and health effects: a focus on megacities, wildfires, dust storms and residential biomass burning. *Toxicol Res (Camb)* 2020; **9:** 331–45.
18 Wooster MJ, Gaveau DLA, Salim MA, et al. New tropical peatland gas and particulate emissions factors indicate 2015 Indonesian fires released far more particulate matter (but less methane) than current inventories imply. *Remote Sens (Basel)* 2018; **10:** 495.
19 Bell ML, Davis DL, Fletcher T. A retrospective assessment of mortality from the London smog episode of 1952: the role of influenza and pollution. *Environ Health Perspect* 2004; **112:** 6–8.
20 Jacobs ET, Burgess JL, Abbott MB. The Donora smog revisited: 70 years after the event that inspired the clean air act. *Am J Public Health* 2018; **108:** S85–88.
21 Ilango S, Weaver M, Benmarhnia TJEE. Extreme heat episodes, wildfires, and risk of preterm delivery in California, 2005–2013. *Environ Int* 2019; **3:** 173–74.
22 Sastry N. Forest fires, air pollution, and mortality in southeast Asia. *Demography* 2002; **39:** 1–23.

23 Jayachandran S. Air quality and infant mortality during Indonesia's massive wildfires in 1997. Jan 1, 2005. http://www.ccpr.ucla.edu/ccprwpseries/ccpr_023_05.pdf (accessed Oct 17, 2020).

24 Doubleday A, Schulte J, Sheppard L, et al. Mortality associated with wildfire smoke exposure in Washington state, 2006–2017: a case-crossover study. *Environ Health* 2020; **19:** 4.

25 Giglio L, Randerson JT, van der Werf GR. Analysis of daily, monthly, and annual burned area using the fourth-generation global fire emissions database (GFED4). *J Geophys Res Biogeosci* 2013; **118:** 317–28.

26 Burstein R, Henry NJ, Collison ML, et al. Mapping 123 million neonatal, infant and child deaths between 2000 and 2017. *Nature* 2019; **574:** 353–58.

27 Xue T, Zhu T, Geng G, Zhang Q. Association between pregnancy loss and ambient $PM_{2·5}$ using survey data in Africa: a longitudinal case-control study, 1998–2016. *Lancet Planet Health* 2019; **3:** e219–25.

28 Xue T, Geng G, Han Y, et al. Open fire exposure increases the risk of pregnancy loss in South Asia. *Nat Commun* 2021; **12:** 3205.

29 van der Werf GR, Randerson JT, Giglio L, et al. Global fire emissions estimates during 1997–2016. *Earth Syst Sci Data* 2017; **9:** 697–720.

30 Nguyen HM, Wooster MJ. Advances in the estimation of high Spatio-temporal resolution pan-African top-down biomass burning emissions made using geostationary fire radiative power (FRP) and MAIAC aerosol optical depth (AOD) data. *Remote Sens Environ* 2020; **248:** 111971.

31 Hammer MS, van Donkelaar A, Li C, et al. Global estimates and long-term trends of fine particulate matter concentrations (1998–2018) *Environ Sci Technol* 2020; **54:** 7879–90.

32 Rangel MA, Vogl TS. Agricultural fires and health at birth. *Rev Econ Stat* 2019; **101:** 616–30.

33 He GJ, Liu T, Zhou MG. Straw burning, $PM_{2·5}$ and death: evidence from China. *J Dev Econ* 2020; **145:** 102468.

34 Goyal N, Karra M, Canning D. Early-life exposure to ambient fine particulate air pollution and infant mortality: pooled evidence from 43 low- and middle-income countries. *Int J Epidemiol* 2019; **48:** 1125–41.

35 Pullabhotla H. Fires, wind, and smoke: air pollution and infant mortality. Nov 15, 2018. https://ace.illinois.edu/sites/ace.illinois.edu/files/Hemant_Pullabhotla_JMP_0.pdf (accessed Oct 17, 2020).

36 Künzli N, Avol E, Wu J, et al. Health effects of the 2003 Southern California wildfires on children. *Am J Respir Crit Care Med* 2006; **174:** 1221–28.

37 Wooster MJ, Perry GLW, Zoumas A. Fire, drought and El Nino relationships on Borneo (southeast Asia) in the pre-MODIS era (1980–2000). *Biogeosciences* 2012; **9:** 317–40.

38 Aditama TY. Impact of haze from forest fire to respiratory health: Indonesian experience. *Respirology* 2000; **5:** 169–74.

39 Jayarathne T, Stockwell CE, Gilbert AA, et al. Chemical characterization of fine particulate matter emitted by peat fires in Central Kalimantan, Indonesia, during the 2015 El Nino. *Atmos Chem Phys* 2018; **18:** 2585–600.

40 Goyal N, Karra M, Canning D. Early-life exposure to ambient fine particulate air pollution and infant mortality: pooled evidence from 43 low- and middle-income countries. *Int J Epidemiol* 2019; **48:** 1125–41.