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Association Between Fire-Related Particulate Matter Exposure and Childhood Asthma in Peru: A Burden of Disease Assessment

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ABSTRACT. We explore the connection between exposure to particulate matter from forest fire emissions in the Peruvian Amazon and pediatric asthma incidence. The bulk of research and media coverage surrounding the Amazon Rainforest fires has focused on important environmental issues, yet the direct impact that these fires have on the health of children living nearby remains under-explored. We conducted a burden of disease assessment using publicly available data to estimate the number of incident pediatric asthma cases attributable to long term exposure to ambient particulate matter smaller than 2.5 microns ($PM_{2.5}$) resulting from increased forest fires in the Peruvian Amazon. Our model compares pediatric asthma burden that would have resulted from a more "typical" fire year, such as 2009, with that from 2016, a severe fire year, by applying $PM_{2.5}$ concentrations from each of those years to the same 2016 population. We estimate that 75,160 (95 % CI 28,638, 121,682) pediatric asthma cases in 2016 were attributable to $PM_{2.5}$, whereas counterfactually applying the 2009 $PM_{2.5}$ concentrations would have resulted in 9,636 (95 % CI 5,657, 13,615) fewer attributable cases. Thus, our results suggest that increased forest fire emissions have led to a notable increase in pediatric asthma burden in Peru.

1. Introduction

Over the past few decades, the raging fires in the Amazon Rainforest have been upheld as a global symbol for a suffering planet. The bulk of research and media coverage surrounding these fires has focused primarily on important environmental issues, yet the impact that these fires have had on the health of children remains under-explored. The children living near the Amazon fires, which have been intentionally perpetrated, in part, by slash-and-burn cattle ranching practices, face health consequences such as severe asthma, bronchitis, and other diseases (Jacobson et al., 2014; Ignotti et al., 2010).

The increase in Amazon fires is one of the most pressing environmental disasters in the world. As of 2019, Brazil alone lost four million acres of forest, and this number has since increased drastically (Gibbens, 2019). These fires have great potential to detrimentally affect ecosystems, endangered or undiscovered animal species, oxygen production, air quality, and public health. The Amazon fires, unlike more localized forest fires, are an international issue that garner attention from all countries around the world. Every nation has a card on the table because the global environmental benefits that the Amazon Rainforest has always provided are now in jeopardy. The most recent devastating Amazon fires in 2019 and 2020, although highly publicized, were not the only recent major fire events in the Amazon. Other years, such as 2016, have been similarly problematic, particularly in Peru. According to the Global Fire Emissions Database (2022), 2016

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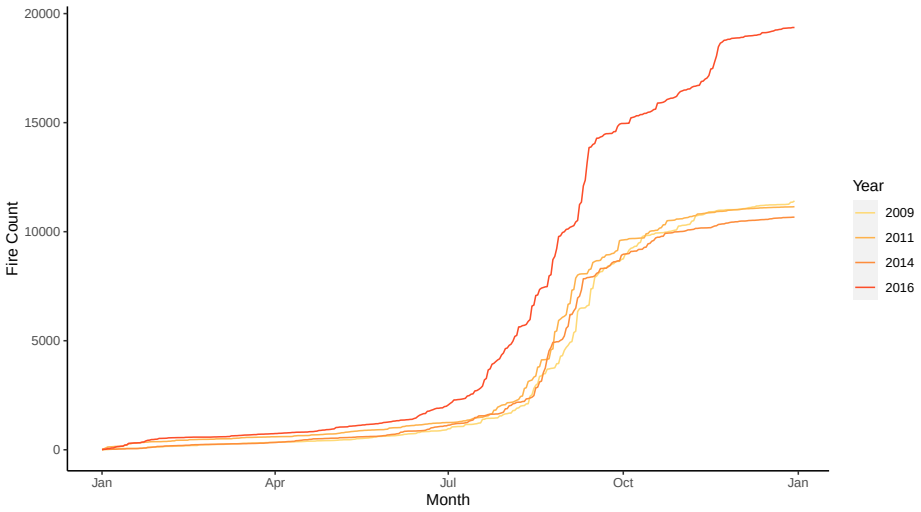

FIGURE 1.1. Cumulative daily fire counts in Peru in 2009, 2011, 2014, and 2016, from Global Fire Emissions Database

saw the highest cumulative number of fires in the Peruvian Amazon over the last decade, even more than 2019 or 2020. As seen in Figure 1.1, Peru experienced almost double the amount of fires in 2016 as it had in 2009, 2011, or 2014, which is why we selected these three years for comparison in our analysis.

Although the amounts and types vary depending on the biomass being burned, the most prominent pollutants directly emitted from forest fires include ozone, carbon dioxide, carbon monoxide, methane, nitrogen oxides, ammonia, non-methane hydrocarbon, and particulate matter (Carvalho et al., 2011). Among these, particulate matter has great potential to affect human health, especially in children. Notably, particulate matter is one of the main causes of childhood asthma (Anenberg et al., 2018). When referring to particulate matter, it will be commonly denoted as $PM_{2.5}$ or PM_{10}, where the subscript referes to the diameter of the particles, in microns. For example, $PM_{2.5}$ are particles that have diameters that are smaller than 2.5 microns. Radke et al. (1978) suggested that the average forest fire can emit pollutants at a rate of about 20 kg/s. Because of this, massive quantities of gases, $PM_{2.5}$, and PM_{10} are released into the atmosphere each year. According to the EPA, $PM_{2.5}$ poses as the most dangerous form of particulate matter because these tiny particulates can easily be inhaled and end up in the lungs, or even in the bloodstream (USEPA, 2021).

Children are among the most vulnerable groups in the human population (Bearer, 1995). Children living in and near the Amazon are in immediate danger of having severe health issues caused by the smoke and particulate matter emitted from fires. Emitted $PM_{2.5}$ can get into the respiratory tract, causing asthma, acute bronchitis, and chronic obstructive pulmonary disease (Caamano-Isorna et al., 2011). These issues are exacerbated in children because of their smaller organs, as well as their higher metabolic rates and higher consumption of oxygen relative to their size (Bearer, 1995). In children under six years of age, their lungs have not developed full functionality and their immune system is still in development. These effects are even more severe in children who have been already diagnosed with asthma and can lead to chronic episodes of asthma attacks and pneumonia, as well as overall decreased lung function (Schwartz, 2004). We will focus on pediatric asthma, one of the most common chronic diseases among children (Romani et al., 2020).

The prevalence of asthma in Peru is one of the highest worldwide with "48% of children in Peru reporting lifetime wheeze" (Robinson et al., 2012). Peru's capital city of Lima alone has asthma prevalence estimated to be around 20%, one of the highest in the world, largely due to traffic-related pollution (Romani et al., 2020). We will conduct a burden of disease assessment to estimate the burden of pediatric asthma incidence in Peru due to ambient $PM_{2.5}$ exposure, specifically as a result of increased Amazon fires. Burden of disease assessments are an appropriate and effective method to estimate health burden, and "[they] have become increasingly popular and have been more commonly used to assess the burden of mortality attributable to air pollution at the global, national, regional and local scales" (Khreis et al., 2021, pg. 77). Childhood asthma is one health outcome that has recently been given more attention through the use of burden of disease assessments (Khreis et al., 2021). Burden of disease assessments are especially useful when using large publicly available data sources to calculate an estimate, rather than collecting primary data.

This research will explore the relationship between $PM_{2.5}$ exposure from increased forest fires in the Peruvian Amazon and pediatric asthma incidence. Following an approach similar to that described by Achakulwisut et al. (2019), we will use a burden of disease model to estimate the number of incident pediatric asthma cases attributable to ambient $PM_{2.5}$ exposure. We will then use our model to quantify the effects that increasing Amazon fires have had on childhood health in Peru. This research can provide a basis to further investigate this relationship in other parts of the world recently severely afflicted by wildfires, such as Australia and California. The purpose of this research is to shine a light on this increasing threat to children's health, thus emphasizing that Amazonian forest fires are not just a major environmental problem.

2. Methods

2.1. Burden of Disease Model

Our overall goal in this burden of disease assessment is to estimate the number of incident pediatric asthma cases attributable to ambient $PM_{2.5}$ exposure from increasing forest fires in the Peruvian Amazon. To do this, we utilized a burden of disease model that compares pediatric asthma burden between "typical" Amazon fire years (2009, 2011, and 2014) and a severe fire year, 2016. We selected the year 2016 because, at the time, this was the most recent severe fire year with available $PM_{2.5}$ data and because, as previously noted, 2016 saw the greatest cumulative number of fires in Peru over the past decade (Global Fire Emissions Database, 2022). We compared 2016 with 2009, 2011, and 2014 by counterfactually applying $PM_{2.5}$ concentration distributions for those years to the same 2016 population. We chose to compare 2016 to "typical" fire years rather than to a hypothetical year with no or minimal fires because, given the unfortunate ubiquity of these fires, it is difficult to imagine what $PM_{2.5}$ concentrations might look like in the Amazon in the absence of fires.

To first find the number of incident asthma cases attributable to $PM_{2.5}$ concentrations for each year, we used the following epidemiological burden of disease model described by Anenberg et al. (2018) and Achakulwisut et al. (2019):

$$B = \sum_{a=1}^{4} e^{\gamma_a} \sum_{i \in P} N_{ai}(1 - e^{-\beta X_i}) \, . \tag{2.1}$$

Here, B represents incident asthma cases attributable to ambient $PM_{2.5}$ for a specific year, γ_a represents the natural logarithm of the asthma incidence rate for age group a (0-4, 5-9, 10-14,

TABLE 2.1. Estimates of the relative risk of pediatric asthma incidence for each 10 $\mu g/m^3$ increase in ambient $PM_{2.5}$, along with 95% confidence intervals (from Anenberg et al., 2018)

Article Source	Estimated Relative Risk	95% Confidence Interval
Anderson et al., 2013	1.34	(0.96, 1.86)
Khreis et al., 2017	1.34	(1.11, 1.63)

or 15-19 years), N_{ai} is the predicted number of people in age group a within grid cell i, β is a concentration-response function relating $PM_{2.5}$ concentrations and pediatric asthma incidence, X_i represents gridded $PM_{2.5}$ data for the specific year of interest in grid cell i, and P is the set of all grid cells within Peru. An outcome of one iteration of this model will provide an estimate for the number of pediatric asthma cases in Peru attributable to $PM_{2.5}$ exposure. To compare the burdens of disease for two different years, we use the following difference function:

$$\Delta = \sum_{a=1}^{4} e^{\gamma_a} \sum_{i \in P} N_{ai}(e^{-\beta X_{t,i}} - e^{-\beta X_{2016,i}}) \,, \tag{2.2}$$

where Δ represents the difference in pediatric asthma burden attributed to $PM_{2.5}$ between two different years, and $X_{t,i}$ represents the gridded $PM_{2.5}$ data for year t in grid cell i. An outcome of one iteration of this model will provide an estimate for the difference in asthma cases in the same 2016 population between two years of counterfactually applied $PM_{2.5}$ concentrations.

To create and execute our burden of disease model, we used the R programming language, version 4.0.2. We used a statistical approach called the delta method to derive approximate 95% confidence intervals for Equations (2.1) and (2.2) (see Appendix A). We used a variety of data sets from different sources as inputs for our model, and these are detailed in the following subsections.

2.2. Estimates for Relative Risk and β

For our model, we used estimates of β taken from previous studies. Table 2.1 shows the relative risk estimates from two relevant meta-analyses of epidemiological studies relating ambient $PM_{2.5}$ exposure and pediatric asthma incidence, along with their corresponding 95% confidence intervals (Anenberg et al., 2018). Note that Anenberg et al. (2018) standardized all relative risk estimates into units of 10 $\mu g/m^3$ to allow for easier comparisons.

To get an estimate, $\hat{\beta}$, to use in our model, we took the natural log of each relative risk estimate in Table 2.1 and divided by 10 to convert it to a relative risk corresponding to a 1 $\mu g/m^3$ increase in $PM_{2.5}$. For example: $\hat{\beta} = \ln(1.34)/10 \approx 0.029$, which suggests that for every 1 $\mu g/m^3$ increase in annual $PM_{2.5}$ exposure, the risk of asthma incidence increases by a factor of $e^{0.029} \approx 1.029$. We performed a similar calculation on the respective confidence limits. Because the confidence intervals were asymmetric, we calculated the approximate variance for each side of the interval, and averaged those variances to derive an approximate variance for $\hat{\beta}$. We then used this approximate variance as an input into our variance estimation using the delta method (see Appendix A).

TABLE 2.2. Inputs for Global Burden of Disease Results Tool

Input Parameter	Value
GBD Estimate	Cause of death or injury
Measure	Incidence
Metric	Number, Percent, Rate
Cause	Asthma
Location	Peru
Age	<5 years, 5-9 years, 10-14 years, 15-19 years
Sex	Both
Annual Rate of Change	Unselected
Year	2016

2.3. Population Data

For the population data in Model (2.1), we used estimated population count raster data for four age groups in Peru for the year 2016 from WorldPop.org. Raster data are a type of data set that is comprised of a spatial grid of cells. Each cell contains data called attributes; in this case, the attribute is the estimated population count for a particular age group and sex. Raster data sets allow for efficient spatial analysis and mapping capabilities. In their raster data sets, WorldPop uses "random forest regression tree-based mapping approaches" to get predicted population counts (WorldPop.org, 2018). The raster data that we used covered the entire country of Peru. The four age groups included were 0-4, 5-9, 10-14, and 15-19 years, and they were split into different raster sets by sex. For each age group, we added the male and female raster sets together, as we were only interested in total population cell counts for each age group. These data sets were originally in 100m $\times$ 100m resolution, but we aggregated them to 1km $\times$ 1km resolution to align them with the other data in our model using the "resample" function from the raster package (version 3.5-15) in R (Hijmans et al., 2022). The result was a raster data set with 3,346,878 1km $\times$ 1km predicted population cell counts for each age group in Peru. We used the same 2016 population data for all calculations to minimize fluctuations in asthma incidence not due to $PM_{2.5}$ exposure. This also allows us to counterfactually apply different years' $PM_{2.5}$ exposures to the exact same population to observe what would have happened had the $PM_{2.5}$ distribution been other than it actually was.

2.4. Asthma Incidence Estimates

For the age-group-specific asthma incidence rates, we used estimated asthma incidence rates per 100,000 people in Peru sourced from the Institute for Health Metrics and Evaluation's (IHME) Global Burden of Disease (GBD) data (IHME, 2019). Using the GBD Results Tool, we were able to enter specific inputs to obtain the data we needed (Table 2.2).

This provided us with the necessary asthma incidence estimates for children in Peru (Table 2.3). We divided these asthma incidence rates by 100,000 to get individual-level rates. We used the confidence intervals from these rates to extrapolate variances for $\hat{\gamma}_a$, the log of the individual-level rate, by using a similar process for asymmetric confidence intervals as described in Section 2.2. We then multiplied these asthma incidence rates by the age group population raster data sets to get estimated incident asthma case counts for each grid cell in Peru.

TABLE 2.3. GBD asthma incidence rates (per 100,000) and confidence intervals for Peru in 2016, by age group

Age Group	Estimated Incidence Rate	95% Confidence Interval
0-4 years	3663.8	(2237.6, 5554.4)
5-9 years	1932.9	(903.9, 3399.0)
10-14 years	922.5	(479.1, 1454.7)
15-19 years	388.2	(210.9, 628.7)

2.5. $PM_{2.5}$ Data

The final input for our burden of disease model was gridded $PM_{2.5}$ data for a given year in each 1km $\times$ 1km cell. We used NASA's Global Annual $PM_{2.5}$ Grids, which are estimated from a model using aerosol optical depth (Hammer et al., 2022). These estimates are "annual concentrations (micrograms per cubic meter) of ground-level fine particulate matter ($PM_{2.5}$), with dust and sea-salt removed" (Hammer et al., 2022). These $PM_{2.5}$ data are from all sources, such as vehicle emissions, and not just forest fires. However, when running our comparison models, we assume that these other sources remain fairly stable from year to year. For our model, we used the resample function from the raster package (version 3.5-15) in R (Hijmans et al., 2022) on these data to align the resolution with the Peruvian population data, and then created a $PM_{2.5}$ risk data set, called the attributable fraction, using $1 - e^{-\beta X}$. We calculated incident asthma cases attributable to $PM_{2.5}$ exposure for each age group in each cell in the raster grid by multiplying the attributable fraction and the respective asthma incidence data sets. This process was completed separately using $PM_{2.5}$ data from all four years. By comparing the estimated asthma incidence due to $PM_{2.5}$ concentrations from a year with increased forest fire activity, such as 2016, with that obtained using $PM_{2.5}$ concentrations from a year with fewer fires, such as 2011, on the same population, we can estimate the burden of pediatric asthma incidence attributable to increased fire-related $PM_{2.5}$ in Peru.

3. Results

Figure 3.1 shows the WorldPop.org raster data from 2016 that breaks population data down into our four age groups. The 0-4 year age group has a total population of 3,125,721, the 5-9 year age group has a total population of 2,998,168, the 10-14 year age group has a total population of 2,907,767, and the 15-19 year age group has a total population of 2,860,034. The darker blue cells represent more children located in each 1km $\times$ 1km cell, while the lighter blue cells represent fewer children per cell. Overall, there are no major apparent differences in population distribution between the four age groups, with the largest notable differences being between Age Groups 1 (0-4 years) and 4 (15-19 years).

Figure 3.2 shows raster data from NASA's Global Annual $PM_{2.5}$ Grids. The darker pink cells represent heavier concentrations of particulate matter. Later years tend to have more ambient $PM_{2.5}$, and 2016 clearly has the highest particulate matter concentrations compared to every other year shown. This visualization suggests that the increase in forest fires most likely was one of

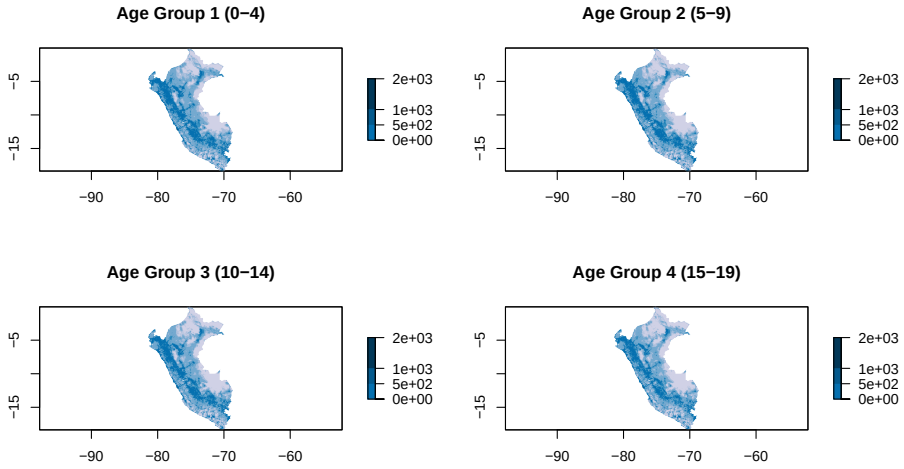

FIGURE 3.1. Distribution of pediatric population in Peru, by age group, for 2016 from WorldPop.org

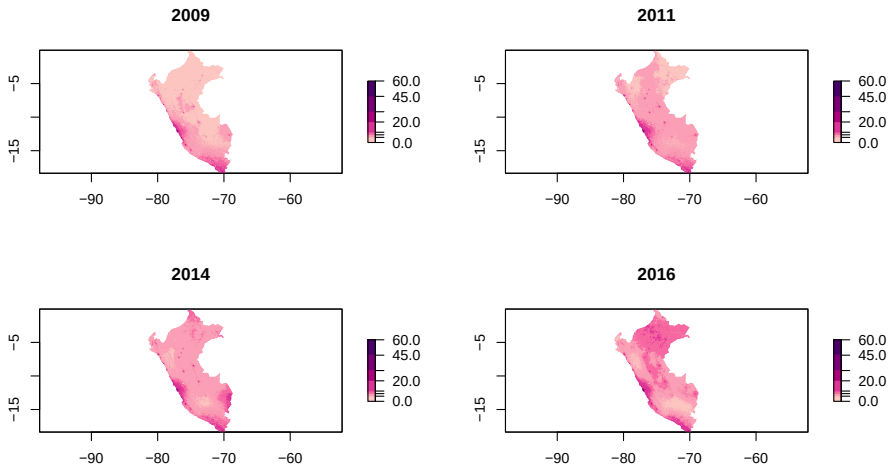

FIGURE 3.2. Distribution of $PM_{2.5}$ ($\mu g/m^3$) in Peru over four selected years from NASA's Global Annual $PM_{2.5}$ Grids

the causes of the higher levels of particulate matter in 2016 compared to the three other years of interest, since much of the increase occurred in the Amazon basin.

Figure 3.3 shows the calculated asthma incidence for the four age groups in 2016. These visualizations represent asthma incidence rates multiplied by the age group population data. As would be expected, it is clear that the youngest age groups tend to have more asthma incidence, which is confirmed in the Table 3.1. Children under the age of 10 are more likely to be afflicted by asthma incidence than their older counterparts. The results in Table 3.1 are significant because they show our calculated asthma cases for the four age groups compared with the 2016 Global Burden of Disease estimates from the IHME. Our estimates are all within $\pm 1\%$ of the Global Burden of Disease

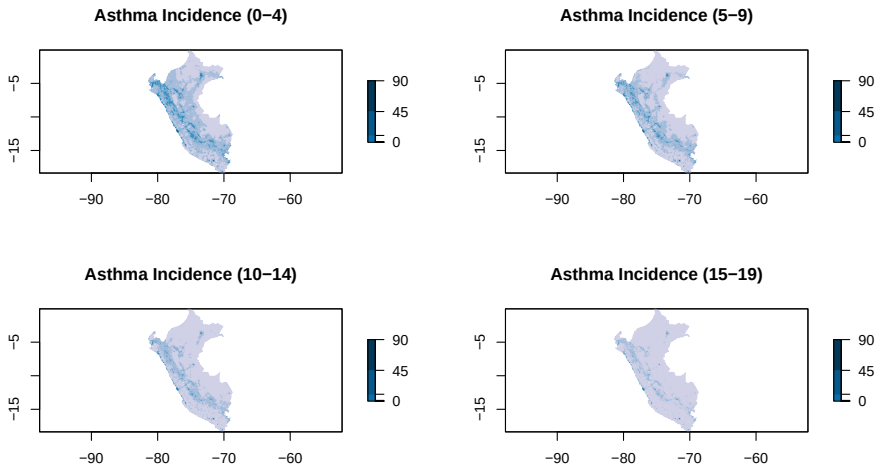

FIGURE 3.3. Distribution of calculated counts of incident asthma cases in Peru in 2016, by age group

TABLE 3.1. Comparison between calculated estimated incident asthma cases and Global Burden of Disease (GBD) estimated incident cases (IHME, 2019) for Peru in 2016

Age Group	Estimated Asthma Cases	GBD Cases	% Difference
0-4 years	114520.5	114692.6	-0.150%
5-9 years	57950.6	57742.3	0.361%
10-14 years	26824.4	26828.5	-0.015%
15-19 years	11102.5	11211.8	-0.975%

estimated counts, which helps to validate our results. The estimated asthma cases are the sums of the cells from Figure 3.3 within each age group.

Table 3.2 provides our point estimates of incident asthma cases attributable to $PM_{2.5}$ exposure, along with 95% confidence intervals, using $PM_{2.5}$ data for each of the years in our burden of disease model (2.1). As a sensitivity check, we report confidence intervals calculated using estimated variances for $\hat{\beta}$ from two different sources, Anderson et al. (2013) and Khreis et al. (2017). The results from Khreis et al. (2017) shown in Table 2.1 lead to an approximate standard error for $\hat{\beta}$ of 0.0098, while the results from Anderson et al. (2013) lead to a larger approximate standard error of 0.01687. The point estimate, $\hat{\beta}$, obtained from both of these papers was 0.0293. To interpret the results for the first row in Table 3.2, we estimate that 65,524.3 incident pediatric asthma cases in 2016 would have been attributable to $PM_{2.5}$ had the distribution of $PM_{2.5}$ in 2016 been the same as it was in 2009. Using the Khreis et al. (2017) results from Table 2.1, we can be 95% confident that the number of attributable incident pediatric cases would have been between 19,821.4 and 111,227.2 had the distribution of $PM_{2.5}$ in 2016 been the same as it had been in 2009. The remaining results can be interpreted similarly.

TABLE 3.2. Point estimates for incident asthma cases attributable to $PM_{2.5}$ exposures from different years along with confidence intervals calculated using two different estimates of relative risk variance

$PM_{2.5}$ Year	Estimated Attributable Asthma Cases	95% Confidence Interval using Khreis et al. (2017) Variance for $\hat{\beta}$	95% Confidence Interval using Anderson et al. (2013) Variance for $\hat{\beta}$
2009	65524.3	(19821.4, 111227.2)	(2172.7, 128875.9)
2011	71248.5	(24890.3, 117606.7)	(5676.1, 136821.0)
2014	72550.3	(26074.5, 119026.2)	(6546.7, 138554.0)
2016	75160.2	(28638.1, 121682.4)	(8724.6, 141595.8)

TABLE 3.3. Point estimates and confidence intervals for excess asthma cases calculated from the difference between burden of disease models using $PM_{2.5}$ values from different years

Comparison: $PM_{2.5}$ Data Years	Estimated Difference in Attributable Asthma Cases	95% Confidence Interval using Khreis et al. (2017) Variance for $\hat{\beta}$	95% Confidence Interval using Anderson et al. (2013) Variance for $\hat{\beta}$
2016 vs. 2009	9635.9	(5657.2, 13614.7)	(4299.3, 14972.5)
2016 vs. 2011	3911.7	(2462.5, 5360.9)	(2120.7, 5702.7)
2016 vs. 2014	2609.9	(1694.9, 3524.8)	(1536.7, 3683.1)

Table 3.3 shows the excess incident pediatric asthma cases attributable to $PM_{2.5}$ when comparing burden of disease from 2016 to burden of disease that would have occurred had the $PM_{2.5}$ distribution been similar to each of the other three years. For example, when comparing 2009 and 2016, we estimated that $PM_{2.5}$ resulting from the severe fires in 2016 would lead to 9,635.9 more pediatric asthma cases than would have been observed had the $PM_{2.5}$ levels been representative of a more typical fire year, 2009. Although the widths of the confidence intervals differ depending on whether the Anderson et al. (2013) or Khreis et al. (2017) variance estimate for $\hat{\beta}$ was used, they both suggest that we can be 95% confident that there were at least 1,500-4,300 more incident asthma cases (and possibly as many as 14,000-15,000 more) in 2016 than there would have been had $PM_{2.5}$ been similar to that in a more "typical" fire year.

4. Discussion and Conclusion

We have identified a significant connection between forest fires in the Peruvian Amazon and pediatric asthma incidence. Our asthma incident case estimates for each age group lies within 1% of the 2016 Global Burden of Disease estimates from IHME. We have also found that particulate matter concentrations have steadily increased between the years 2009 and 2016. This is potentially due to a few reasons, but a main cause is almost certainly an increase in forest fires in the Amazon.

According to our burden of disease model, the estimated incident asthma cases decrease when the particulate matter concentrations for each of years 2009, 2011, and 2014 are applied to the 2016 population. The point estimates for incident asthma cases attributable to $PM_{2.5}$ range between 65,000 and 76,000. Using uncertainty estimates based on Khreis et al. (2017), we estimate that the actual attributable cases may range between 19,000 and 122,000, while using uncertainty estimates based on Anderson et al. (2013), the cases may range between 2,000 and 142,000. When we compared estimated asthma cases using 2009 and 2016 $PM_{2.5}$ data, we see the largest difference in cases, as opposed to the 2011/2016 and 2014/2016 comparisons. These differences in point estimates range between 2,600 and 9,700 estimated incident cases. Overall, our results suggest that the excessive number of Peruvian Amazon fires in 2016 resulted in increased pediatric asthma incidence.

Some recent publications, such as Lavigne et al. (2021), have studied the global relationship between particulate matter concentration and childhood asthma incidence. Other studies, such as Khreis et al. (2021), looked at the relationship between air pollution and onset of childhood asthma in the United States. Others, such as Anenberg et al. (2018) and Anderson et al. (2013), have looked at the effects of traffic and fire-related air pollution on asthma incidence. Our study attempts to look specifically at the effects of the particulate matter emissions from increasing Amazon Rainforest fires on children living in Peru. Overall, our results coincides with the results from similar recent studies.

Our approach does have some potential limitations. The first limitation has to do with the data that our model is based on. All of the data that we used, although from validated sources, are model-based estimates and not directly observed values. In calculating our confidence intervals, we were able to account for variability in both asthma incidence and the estimates of the response function, β, but not for uncertainty regarding either the population size or particulate matter data. However, our use of the delta method, which simultaneously accounted for both of these sources of variation, is a step beyond previous research (Anenberg et al., 2018; Achakulwisut et al., 2019), which each only accounted for a single source of variation. The next limitations have to do with the assumptions surrounding our model. Acknowledging that particulate matter is not the only cause of asthma, we use estimates from previous meta-analyses that quantify the relationship between particulate matter and asthma incidence to help account for this. We also understand that not all particulate matter is emitted from forest fires. However, our visualizations showing the drastic increase in forest fires in 2016 compared with other years and the corresponding increases in $PM_{2.5}$ specifically in the Amazon basin do suggest that fire is a major cause. Although we cannot control for additional sources such as traffic-related air pollution, we hope that applying $PM_{2.5}$ distributions from different years to exactly the same population can help to control for these other sources. Using the same population also ensures that there would be no confounding due to population changes over time, and made it easier to make direct comparisons between the estimated asthma incident cases across all four years of $PM_{2.5}$ data. Finally, we do not account for particulate matter coming from fires in neighboring countries, such as Brazil. Given the prevailing wind patterns, it is highly likely that fires in the Brazilian Amazon influenced air quality in Peru. Despite these limitations, we believe that our research provides valuable insights into the connection between forest fires and pediatric asthma incidence in Peru.

Overall, we provide statistical evidence to suggest that the increase in Peruvian Amazon fires has caused an increase in pediatric asthma incidence. This is largely due to the particulate matter that was absorbed into the lungs of children living in Peru. With our burden of disease model, we found that the year 2016 had significantly more estimated asthma incident cases from particulate

matter than would have been observed had the particulate matter concentrations been more similar to the years 2009, 2011, or 2014.

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Appendix A. Estimating Variances using the Delta Method

We used the delta method to approximate the variance of a complex function with several random terms to allow for a more accurate confidence interval around our estimations. This approach represents an advance over previous analyses which only accounted for uncertainty in the relative risk estimates (Anenberg et al., 2018; Achakulwisut et al., 2019). Although there is also uncertainty present in the estimators of population and $PM_{2.5}$, we have no information about the variability of these estimates. However, we were able to approximate the variances for both $\hat{\beta}$ and $\hat{\gamma}_a$. Using these variances, we created 95% confidence intervals around our estimates. We then repeated this entire process for comparing the years 2009, 2011, and 2014 with 2016. We used this method to estimate the difference, along with a confidence interval, between the burden of disease models for each of the three comparisons. As a sensitivity analysis, we separately applied variance estimates for $\hat{\beta}$ from both the Anderson et al. (2013) and Khreis et al. (2017) papers to evaluate how much that would change our uncertainty intervals.

Here, we show how to derive our confidence intervals using the delta method. We first use the delta method to approximate the variance for the difference model (2.2). We start by using a first-order multivariate Taylor series expansion around the true parameters β and γ_a and rearranging terms to obtain

$$\left(\hat{\Delta} - \Delta\right) \approx \left(\hat{\beta} - \beta\right) \left\{ \sum_{a=1}^{4} e^{\gamma_a} \sum_{i \in P} N_{ai} \left(-X_{t,i}\, e^{-\beta X_{t,i}} + X_{2016,i}\, e^{-\beta X_{2016,i}} \right) \right\} + \sum_{a=1}^{4} \left(\hat{\gamma}_a - \gamma_a\right) \left\{ e^{\gamma_a} \sum_{i \in P} N_{ai} (e^{-\beta X_{t,i}} - e^{-\beta X_{2016,i}}) \right\}. \tag{A.1}$$

We then square and take the expected values of both sides to obtain (noting that $\hat{\beta}$ and $\hat{\gamma}_a$ are from different sources and so are uncorrelated):

$$\text{Var}\left(\hat{\Delta}\right) \approx \text{Var}\left(\hat{\beta}\right) \left\{ \sum_{a=1}^{4} e^{\gamma_a} \sum_{i \in P} N_{ai} \left(-X_{t,i}\, e^{-\beta X_{t,i}} + X_{2016,i}\, e^{-\beta X_{2016,i}} \right) \right\}^2 + \sum_{a=1}^{4} \text{Var}\left(\hat{\gamma}_a\right) \left\{ e^{\gamma_a} \sum_{i \in P} N_{ai} (e^{-\beta X_{t,i}} - e^{-\beta X_{2016,i}}) \right\}^2. \tag{A.2}$$

We can then use this approximate variance for $\hat{\Delta}$ to get an approximate 95% confidence interval for Δ as $\hat{\Delta} \pm 1.96 * \sqrt{\text{Var}\left(\hat{\Delta}\right)}$.

Similarly, we can approximate the variance for the burden of disease model (2.1) as

$$\text{Var}\left(\hat{B}\right) \approx \text{Var}\left(\hat{\beta}\right)\left\{\sum_{a=1}^{4} e^{\gamma_a} \sum_{i \in P} N_{ai} X_i \, e^{-\beta X_i}\right\}^2 + \sum_{a=1}^{4} \text{Var}\left(\hat{\gamma}_a\right)\left\{e^{\gamma_a} \sum_{i \in P} N_{ai}\left(1 - e^{-\beta X_i}\right)\right\}^2 \quad \text{(A.3)}$$

and use this to calculate an approximate 95% confidence interval for B.

Using the delta method, we were thus able to simultaneously account for the variances in $\hat{\beta}$ and $\hat{\gamma}_a$ when estimating the number of pediatric asthma incidence cases attributable to increased fire related $PM_{2.5}$ in Peru.

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