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Authors
Ezzati, M
KAMMEN, Daniel M.

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Acute respiratory infections (ARI) are the leading cause of burden of disease worldwide and have been causally linked with exposure to pollutants from domestic biomass fuels in developing countries. We used longitudinal health data coupled with detailed monitoring and estimation of personal exposure from more than 2 years of field measurements in rural Kenya to estimate the exposure–response relationship for particulates < 10 µm diameter (PM10) generated from biomass combustion. Acute respiratory infections and acute lower respiratory infections are concave, increasing functions of average daily exposure to PM10, with the rate of increase declining for exposures above approximately 1,000–2,000 µg/m³. This first estimation of the exposure–response relationship for the high-exposure levels characteristic of developing countries has immediate and important consequences for international public health policies, energy and combustion research, and technology transfer efforts that affect more than 2 billion people worldwide. Key words: acute respiratory infections, Africa, biomass combustion, developing countries, exposure–response relationship, field study, indoor air pollution, particulate matter, public health. Environ Health Perspect 109:481–488 (2001). [Online 4 May 2001]

http://ehpnet1.niehs.nih.gov/docs/2001/109p481-488ezzati/abstract.html

Acute respiratory infections (ARI) are the leading cause of burden of disease and mortality, mostly in leading cause of the global burden of disease (1). Between 1997 and 1999, acute lower respiratory infections (ALRI) were the leading cause of mortality from infectious diseases, with an estimated 3.5–4.0 million deaths worldwide (1–3). Exposure to indoor air pollution, especially to particulate matter, from the combustion of biofuels (wood, charcoal, agricultural residues, and dung) has been implicated as a causal agent of respiratory diseases in developing countries (4–9). This association, coupled with the fact that globally more than 2 billion people rely on biomass as the primary source of domestic energy, has put preventive measures to reduce exposure to indoor air pollution high on the agenda of international development and public health organizations (10–13). The evaluation of the benefits and effectiveness of measures that aim to reduce these negative health impacts, such as design and dissemination of improved stoves and fuels, requires knowledge of the exposure–response relationship between indoor particulate matter from biomass combustion and ARI.

Epidemiologic and physiologic studies over the past two decades in urban areas of industrialized countries have resulted in significant progress in identifying and quantifying the health impacts of outdoor (ambient) particulate matter (14–24). These results, however, are applicable to a small range of exposures, generally below 200 µg/m³, which are primarily of concern in industrialized countries (12). The latest U.S. Environmental Protection Agency National Ambient Air Quality Standards, for instance, required the concentration of PM10 (particulate matter < 10 µm) to achieve a 24-hr average <150 µg/m³). There is little information on the shape of the exposure–response relationship at concentrations of hundreds to thousands of micrograms per cubic meter that are commonly observed in indoor environments in developing countries (13). This is a critical gap in our understanding of the role of exposure to particulate matter as a causal agent of ARI, and thus as a contributor to the global burden of disease, because approximately 80% of total global exposure to this pollutant occurs indoors in developing nations (25,26).

Research on the health impacts of indoor air pollution in developing countries has been hindered by a lack of detailed data on both exposure and illness outcomes. In these settings, many epidemiologic studies have used indirect and often inaccurate measures, such as fuel or housing type, as proxies for personal exposure to indoor particulate matter. However, these proxies are hindered by a lack of detailed data on both variables at the individual level. The findings and conclusions in this paper are those of the authors and do not reflect the views of the World Health Organization. Received 2 October 2000; accepted 21 November 2000.

Address correspondence to M. Ezzati, Global Programme on Evidence for Health Policy, World Health Organization, CH-1211 Geneva 27, Switzerland. Telephone: 41 22 791 2369. Fax: 41 22 791 4328. E-mail: ezzatim@who.ch

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This research was supported by grants from the MacArthur Foundation, Social Science Research Council, and the Princeton University Council on Regional Studies and Center of International Studies (through a grant from the MacArthur Foundation). This research was approved by the Institutional Review Panel for Human Subjects of the University Research Board, Princeton University (Case #1890) and by the Government of Kenya, under the Office of the President Research Permit OP/13/001/25C 167. It has followed all the human subject guidelines, including consent of subjects to data collection. The findings and conclusions in this paper are those of the authors and do not reflect the views of the World Health Organization.
We complemented these data with extensive interviews with household members and local extension workers on household energy technology and time-activity budget.

Personal exposures were calculated from these data and accounted for daily and day-to-day variability of exposure, time budget and activities of individuals, and spatial dispersion of pollution in the house. Measurement and data analysis methods for personal exposure values have been previously discussed (37). Demographic information for the individuals in the 55 households in the study group are presented in Table 1. Table 2 provides summary statistics for personal exposure values.

For collection of health data, two community nurses from Nanyuki District Hospital visited all the households in the study group on a regular basis. The nurses had received training from the National ARI Programme (designed in consultation with the World Health Organization (WHO)) on the WHO protocols for clinical diagnosis of ARI. In the initial months of the program, each village was visited once every 2 weeks. The visits then increased to once per week. In the initial months, one of the coordinators of the National ARI Programme from the Department of Paediatrics of the Kenyatta National Hospital accompanied the visiting nurses to the villages to ensure the proper execution of diagnosis protocols. In each visit at least one adult member from each household reported to the nurse on the health status of the household members, with specific emphasis on the presence of cough and other respiratory ailments. The responses were collected in the language of choice of the respondents and recorded in English by the nurses, who spoke Swahili and Turkana.

The nurse then clinically examined all of the individuals who were reported with symptoms, and recorded the relevant clinical information, including symptoms and diagnosis. The reporting process also included information on visits to any other health facility since the nurse’s last visit. Therefore, the health data include a 2-year array of weekly health records for each individual in the study group. Depending on the severity, the cases were treated with the standardized treatment of the National ARI Programme, which also resulted in standardization of treatment in the study group. Treatments included drugs that were readily available in the nearest town (Nanyuki) which were dispensed by the nurses for more severe cases. The nurses also provided assurance or recommended home remedies for minor cases. The extreme, and potentially fatal, cases were referred to one of the hospitals in Nanyuki. No information was recorded for those households for which no adult member was present or for household members who were away from home during the day of the visit. Table 3 provides summary statistics on the number of health reports for the individuals in the study group.

The health status of the individuals in the study group was likely to have been affected by the medical treatment provided during the collection of health data. In additional to ethical considerations, this provision standardized treatment in the whole study group and prevented confounding due to factors such as access to health care facilities. At the same time, if the treatment affected the cases differently in a way that is correlated with exposure, this could modify the shape of the exposure–response curve (the so-called Hawthorne Effect). Therefore,
the relationships obtained in this analysis are based on the presence and use of a small level of health care.

**Statistical models.** We estimated the parameters of the exposure–response relationships using two models [the properties of additive and multiplicative risk models have previously been discussed (39,40):]

\[
y = X \times \beta + u,
\]

where \(y\) is the vector of illness rates for all of the individuals in the study group, \(X\) is a matrix of characteristics for the individuals in the study group (i.e., the above explanatory and control variables), \(\beta\) is the vector of coefficients, and \(u\) is the vector of independent, normally distributed errors.

\[
y = F(X \times \beta + u),
\]

where \(y\), \(X\), and \(\beta\) are defined as above, and \(F\) is the cumulative logistic distribution defined as:

\[
F(z) = \frac{\exp(z)}{1 + \exp(z)}
\]

([In a logit or logistic regression model, the left hand side of Equation 2 is the probability of an event such as illness or Pr\(\{y\}\). Hence, since the outcome is defined as the fraction of time with illness, therefore equivalent to rate or probability of illness, the left hand side is simply \(y\).]

We estimated model parameters using ordinary-least-squares (OLS) regression 

\[
y = X \times \beta + u,
\]

where \(y\) is a vector of illnesses and \(X\) is a matrix of characteristics for the individuals in the study group (i.e., the above explanatory and control variables), \(\beta\) is the vector of coefficients, and \(u\) is the vector of independent, normally distributed errors.

\[
y = F(X \times \beta + u),
\]

where \(y\), \(X\), and \(\beta\) are defined as above, and \(F\) is the cumulative logistic distribution defined as:

\[
F(z) = \frac{\exp(z)}{1 + \exp(z)}
\]

\[
\text{logit}(y) = X \times \beta + u,
\]

where \(\logit(y)\) is the logit transformation of \(y\), \(X\) is a matrix of characteristics for the individuals in the study group (i.e., the above explanatory and control variables), \(\beta\) is the vector of coefficients, and \(u\) is the vector of independent, normally distributed errors.

\[
y = \exp(X \times \beta + u),
\]

where \(y\), \(X\), and \(\beta\) are defined as above, and \(\exp\) is the exponential function.

Where \(y\), \(X\), and \(\beta\) are defined as above, and \(F\) is the cumulative logistic distribution defined as:

\[
F(z) = \frac{\exp(z)}{1 + \exp(z)}
\]

([In a logit or logistic regression model, the left hand side of Equation 2 is the probability of an event such as illness or Pr\(\{y\}\). Hence, since the outcome is defined as the fraction of time with illness, therefore equivalent to rate or probability of illness, the left hand side is simply \(y\).]

We estimated model parameters using ordinary-least-squares (OLS) regression for model 1 (Equation 1) with clustering in ordinary-least-squares (OLS) regression for model 2 (Equation 2), we used a logit regression using maximum-likelihood estimation. Logit regression also accounts for the increasing confidence in illness rates with the increasing number of health exams. (The number of times that an individual is diagnosed with illness in \(n\) examinations has a binomial distribution. The probability of illness, \(y\), defined as the fraction of examinations with illness, is then an estimate for the probability of being diagnosed with illness, \(p\). The confidence interval for \(p\) is obtained from an approximately normal distribution around \(y\) with variance \(y(1 - y)/n\). The variance and the confidence interval are therefore decreasing functions of the number of visits, \(n\).)

### Results and Discussion

**Distribution of ARI and ALRI with demographic characteristics and exposure.** Figure 2 shows ARI and ALRI rates—defined as the fraction of weeks that an individual is diagnosed with ARI and ALRI—for different demographic subgroups of the study group. For a disease such as ARI, whose episodes have a limited and short duration, disease episode and case have interchangeable definitions. As a result, all episodes in a time interval count toward disease incidence, and the fraction of weeks diagnosed with disease is an aggregate measure of both incidence and duration.

The female–male comparisons in Figure 4 illustrate that, once exposed to higher PM_{10} emissions through greater cooking and other domestic activities at later ages, women are approximately twice as likely as men to be diagnosed with ARI or ALRI.

Figure 3 shows the ARI and ALRI rates for infants and children (0–4 years of age Figure 3A) and young and adult individuals (5–49 years of age Figure 3B) plotted against average daily exposure to PM_{10}. No analysis was conducted for the ≥ 50 age group because of the small sample size. Personal exposure to biomass smoke varies from day to day because of the variations in both pollution levels and time-activity budget (37). To account for this variability, as well as any error or uncertainty in the estimates of average exposure, we assigned individuals to exposure categories.

For both age groups, ARI and ALRI rates rise more rapidly for exposures < 2,000 µg/m³. For children 0–4 years of age (Figure 3A), ARI and ALRI rates in the < 200 µg/m³ exposure category are 0.11 (< 0.01) and 0.024 (< 0.18), respectively, lower than those in the 1,000–2,000 µg/m³ group. The increase between the latter group and the highest exposure category (> 3,500 µg/m³) is only 0.05 for ARI (< 0.49) and 0.02 for ALRI (< 0.57); in this specific comparison, although the large p-values are partially due to the small fraction of children in the highest exposure category, they are also a reflection of the smaller slope of the exposure–response relationship. In Figure 3B, ARI and ALRI rates increased by 0.048 (< 0.0001) and 0.011 (< 0.01), respectively, between the lowest exposure group and 2,000 µg/m³, compared to 0.053 (< 0.0001) and 0.025 (< 0.001), respectively, between the 2,000 µg/m³ group and the > 7,000 µg/m³ category in an exposure range four times as large.

**Issues in estimation of the exposure–response relationship.** In determining the exposure–response relationship, it is important to account for the range of possible confounding and contributing factors, especially the potential correlation between exposure and other determinants of health, such as socioeconomic status and nutrition (33). In particular, there is evidence that poorer households, who may have additional susceptibility to disease, use more polluting sources of energy for cooking and live in poorer housing conditions. Although empirical research has demonstrated that the household choice of energy technology is influenced by a range of social and cultural factors (41), income is indeed an important determinant of exposure (25,42).

Incomes are similar among the residents of Mpala Ranch, except for a few skilled differences.
workers. Further, because part of the income is paid in-kind as food, there is little variation in nutrition. Incomes are similar between the two groups of villages (maintenance and cattle-herding), and workers are moved between types of villages at the instruction of ranch management with no change in earnings. Houses are assigned by the management and are nearly identical within each village type. Therefore, village type and housing are not endogenous variables and are not correlated with income.

With the exception of the occasional use of paraffin, firewood and charcoal are the exclusive fuels at Mpala Ranch. The most important determinant of access to charcoal is contact with traders from a neighboring community where charcoal is produced. Therefore, with the relatively small range of incomes, the choice of charcoal or wood is mostly determined by the location of specific village where a family lives, which is decided by the ranch manager and is therefore exogenous. It may nonetheless be possible that other factors also influence the choice of fuel, especially because there is variation in fuel use within individual villages. If these factors are not correlated with health (such as how the type of fuel affects the preference for a specific flavor of food), the issue of endogenous exposure is not a concern. If some of the determinants of fuel use are correlated with health, such as the education of the mother, the problem of endogeneity remains. In our interviews on fuel use, the commonly stated reasons for choice of fuel were uncertainty about future access, the taste of food, the cost of charcoal (a large bag of charcoal sufficient for approximately 1 week for an average household costs approximately 1.5 times the daily wage), and difficulty of wood collection. Because no household level variable that is correlated with health could be specified as the determinant of fuel choice and because few households used charcoal exclusively (almost all charcoal users had a mixed-fuel profile), the choice of fuel in this setting is exogenous to other determinants of health. We nonetheless controlled for the type of village where a household lives to account for any potential unobservable differences between them.

Clustering of observations is another important methodologic issue in estimation of the exposure–response relationship because the determinants and outcome of health status are likely to exhibit similarity within a single household. We accounted for the clustering of observations in units of households and used robust estimates of variance to correct for this and any statistical outliers in estimation of standard errors.

Estimation of model parameters. In addition to exposure, the main explanatory variable, we controlled for the following variables:

- Sex: We controlled for sex to account for potential female–male susceptibility differences.
- Age: To account for effects of age on immunity or the chronic impacts of long-term exposure, we controlled for age.
- Village type: Although income and nutritional status are similar between the residents of maintenance and cattle-herding villages, there may be differences that are unobservable to the researcher that can influence disease rates. These differences would result in a statistically significant coefficient of this variable.
- Number of people residing in the house: Because of the communicable nature of ARI, living in more crowded environments would be expected to facilitate transmission. Because house sizes are standardized within each village type, the number of residents living in each house is a proxy for crowding. The mean, median, and standard deviation of the number of people living in a house were 7.0, 7.0, and 2.2, respectively, in the cattle-herding villages and 5.3, 5.0, and 2.0, respectively, in the maintenance villages.
- Smoking: Tobacco smoking is a known causal agent of respiratory diseases. The number of smokers at Mpala Ranch was low (13 in the sample of households used in this analysis) and they smoked infrequently, because of the cost of cigarettes and because a more accessible alternative (chewing the leaves of a specific plant) exists. We treated smoking in two different ways: first, as a separate variable without considering its contribution to exposure, and second, as a source of exposure to particulate matter from tobacco (itself biomass) combustion. [For the smokers (all male) in the group (n = 13), exposure was increased by 1,000 µg/m³ from those estimated by Ezzati et al. (37) to reflect exposure to particulate matter as a result of combustion of biomass in cigarettes. A 1,000 µg/m³ increase in average exposure is equivalent to 4 min of active inhalation of cigarette smoke, with an estimated particulate matter concentration of 400,000 µg/m³.]

Weights at birth would be another important control variable for the 0–4 age group if data were available.

Tables 4 and 5 present the parameters of the exposure–response relationship for the models of Equations 1 and 2, respectively. The coefficients of exposure in Tables 4 and 5 confirm the relationship seen in Figure 3: The exposure–response relationship for indoor PM10 from biomass combustion and both ARI and ALRI is increasing, but the rate of increase declines at average daily exposures above 2,000 µg/m³. For ALRI, the rates...
of increase rises again at the highest exposure levels for both age groups, > 3,500 µg/m³ for infants and children and > 7,000 µg/m³ for young and adult individuals.

In the first 60 months after birth, age has an overall downward effect on susceptibility to ARI and ALRI, which is consistent with the findings of Cruz et al. (43) and Oyejide and Osinusi (44); each year of age decreases the likelihood of being diagnosed with ARI and ALRI by 0.009 (p = 0.08) and 0.01 (p = 0.002), respectively. If the population as a whole is considered (regression results not shown), on average, infants and children < 5 years of age have an additional risk of 0.08 (p < 0.001) for being diagnosed with ARI (0.05 for ALRI; p < 0.001) compared to those between the ages of 5 and 49, after controlling for exposure and other factors. This is consistent with the described susceptibility-reducing role of age among infants and children.

After the age of 5, age increases the probability of being diagnosed with ALRI, potentially due to chronic effects of earlier exposure. In the OLS model (Table 4), age does not affect susceptibility to ARI for ages ≥ 5; in the blogit model, this is consistent with the described susceptibility-reducing role of age among infants and children.

We found no statistically significant effect for village type (p > 0.40) after accounting for exposure and other factors; we attribute this to comparable income levels and diets in the two village types, as explained above. The number of people in the household was not statistically significant (p ≥ 0.45). With a pastoral lifestyle, activity patterns are a more important determinant of the amount of time spent inside together for most of the day than the number of household members.

When considered independently, smoking increases the risk of ARI by 0.02 (p = 0.04) in the OLS model and with an odds ratio of 1.48 (p = 0.02; 95% confidence interval (CI), 1.07–2.04) in the logistic model. The increase in ALRI risk from smoking is not statistically significant. When smoking is considered a source of exposure to particulate matter from combustion of tobacco, which is a form of biomass, the coefficient of smoking is no longer significant. The remainder of the results were not sensitive to the method of including smoking in the analysis. This illustrates that the impacts of smoking on ARI may be similar to other health hazards (45), some of which may be similar to other biomass products and others, in particular lung cancer, may be different.

The implications of exposure assessment methodology. The role of sex is particularly important and has implications for exposure assessment methodology and public health measures. Exposure values in this analysis account for the actual patterns of exposure of individuals, including their time budget and activities, and the spatial dispersion of smoke in the house (37). Once these patterns are included in calculating daily exposure to PM_10, males and females have similar responses. In Table 4, coefficients for females are statistically not significant; in Table 5 the odds ratios for females are statistically not different from 1.0, except in the case of ARI for age ≥ 5 years, with a 95% CI of 1.01–1.52.

In contrast, if exposure is calculated from average daily PM_10 concentrations and time spent indoors only (i.e., without accounting for the specific activities and movement patterns of individuals), females > 5 years of age have additional risk of ARI and ALRI. Using this method of exposure calculation in the OLS model, being female increases the probability of ARI by 0.03 (p < 0.001) and ALRI by 0.01 (p < 0.01); in the blogit model, the odds ratios for the risk associated with being female are 1.74 (p < 0.001; 95% CI, 1.48–2.04) for ARI and 1.94 (p < 0.001; 95% CI, 1.38–2.72) for ALRI. In an earlier study (37), we demonstrated that this latter and commonly used method of exposure estimation underestimates the exposure of women more than men because women cook more often than men. The current analysis shows that this underestimation results in systematic bias in assessment of the exposure–response relationship. Controlling for the amount of cooking activity that a person performs eliminates the statistical significance of sex.
of sex, confirming that sex is a substitute for exposure patterns (i.e., a proxy for the omitted variable of high-intensity exposure) when average daily PM$_{10}$ concentration is used. Finally, this bias is further confirmed by noting that when estimating exposure using average daily PM$_{10}$ concentration and time alone, the role of sex appears only after the age of 5 years when females actually take part in household activities. For those <5 years of age, the coefficient of sex remains insignificant ($p = 0.87-0.88$ for AR1 and $p = 0.21-0.47$ for ALRI).

The role of intense episodes of exposure.

In a previous study (37) we demonstrated that, for individuals who cook, approximately one-half of total daily exposure occurs within a short period when stove emissions are the highest and the individual is closest to the stove. To see whether such episodes of intense exposure have health effects beyond their contribution to total daily exposure, we considered the following two variables for age > 5 years (because children <5 years of age do not participate in household tasks and infants are not carried on their mothers’ backs during housework, these variables do not apply to the pattern of exposure for children <5 years of age).

- Participation in household tasks is a categorical variable that divides individuals into four groups: those who do not perform any household tasks; those who participate in some household tasks, such as water collection or cleaning the house, but none that involve the use of the stove; those who sometimes use or tend the stove, but not on a regular basis; and individuals who participate in cooking-related tasks regularly.

- Exposure intensity is defined as the concentration during an individual’s most intense exposure episode. For those who participate in household tasks, this equals the pollution concentration in the area immediately around the stove during the times the stove has its highest pollution level [i.e., in its top 25th percentile as defined by Ezzati et al. (37)]. For those who do not participate in cooking-related tasks, exposure intensity is simply their average daily exposure. Smokers are included with those who have the highest exposure intensity due to the high concentration of particulate matter in cigarette smoke.

Therefore, these two variables are indicators of the length and intensity, respectively, of exposure to high concentrations of PM$_{10}$. This analysis shows that exposure intensity does not have a statistically significant association with the incidence of AR1 ($p > 0.10$) beyond its contribution to total (or average) exposure. The coefficients of participation in household tasks are not jointly significant for AR1 or ALRI. However, the group that regularly participates in cooking-related tasks has an additional risk of ALRI that is significant. In the OLS model, the ALRI rate for this group is higher by 0.02 ($p = 0.03$); in the logit model, the odds ratio for the ALRI risk associated with regular cooking is 2.40 ($p = 0.03; 95\% CI, 1.10–5.25$).

This result implies that either long periods of exposure to very high levels of PM$_{10}$ cause (either short-term or chronic) damage to the lower respiratory system beyond that described by the average exposure–response relationship, or the exposure of this group is underestimated even by the approach described previously (37) that accounts for higher exposure during cooking periods. Investigation of the last hypothesis would be possible with more detailed monitoring of personal exposure. Studying the chronic impacts of high-intensity exposure would require knowledge of the history of exposure of individuals. Alternatively, it is possible to compare ALRI incidence among people who have cooked for many years with those who have just begun to cook after controlling for age, which was not possible in our study due to sample size. Finally, research on dispersion and deposition of particulates in the airways as a function of pollution intensity can shed light on the acute impacts of high-intensity exposure.

**Conclusions**

Monitoring and estimating individual-level exposure to indoor PM$_{10}$ from biomass combustion, longitudinal data on AR1, and demographic information have enabled us to quantify the exposure–response relationship for one of the most common diseases in developing nations. This analysis shows that the relationship between daily exposure to indoor PM$_{10}$ and the fraction of time that a person has AR1, or the more severe ALRI, is an increasing function. Based on the best estimate of the exposure–response relationship, the rate of increase is higher for daily exposures < 2,000 µg/m$^3$. This result is robust to the choice of statistical model; the

| Table 4. Parameters of the exposure–response relationship for AR1 and ALRI using OLS regression (Equation 1). |
| Group/explanatory variable | AR1 | ALRI |
|-----------------------------|-----|------|
| **0–4 year group**          |     |      |
| Constant                    | 0.05 ($p = 0.45$) | 0.07 ($p = 0.06$) |
| Exposure category           |     |      |
| <200 µg/m$^3$                | Reference category | Reference category |
| 200–500 µg/m$^3$             | 0.06 ($p = 0.002$)* | 0.01 ($p = 0.16$) |
| 500–1,000 µg/m$^3$           | 0.06 ($p = 0.04$)* | 0.01 ($p = 0.24$) |
| 1,000–2,000 µg/m$^3$         | 0.13 ($p = 0.001$)* | 0.03 ($p = 0.05$) |
| 2,000–3,500 µg/m$^3$         | 0.14 ($p = 0.001$)* | 0.03 ($p = 0.16$) |
| >3,500 µg/m$^3$              | 0.18 ($p = 0.04$)* | 0.04 ($p = 0.30$) |
| Female                      | -0.0007 ($p = 0.98$) | -0.009 ($p = 0.43$) |
| Age                         | -0.009 ($p = 0.08$) | -0.01 ($p = 0.002$) |
| Village type                | 0.03 ($p = 0.42$) | 0.006 ($p = 0.70$) |
| Number of people in household | 0.0005 ($p = 0.94$) | 0.0001 ($p = 0.99$) |
| $R^2$                       | 0.20 | 0.16 |
| **5–49 year group**         |     |      |
| Constant                    | 0.03 ($p = 0.10$) | 0.0002 ($p = 0.97$) |
| Exposure category           |     |      |
| <200 µg/m$^3$                | Reference category | Reference category |
| 200–500 µg/m$^3$             | 0.027 ($p = 0.003$)* | 0.0337 ($p = 0.48$) |
| 500–1,000 µg/m$^3$           | 0.022 ($p = 0.06$) | 0.0403 ($p = 0.32$)* |
| 1,000–2,000 µg/m$^3$         | 0.039 ($p = 0.002$)* | 0.011 ($p = 0.03$)* |
| 2,000–4,000 µg/m$^3$         | 0.052 ($p = 0.001$)* | 0.011 ($p = 0.03$)* |
| 4,000–7,000 µg/m$^3$         | 0.064 ($p = 0.002$)* | 0.013 ($p = 0.09$)* |
| >7,000 µg/m$^3$              | 0.090 ($p < 0.001$)* | 0.031 ($p = 0.001$)* |
| Female                      | 0.013 ($p = 0.18$) | 0.003 ($p = 0.40$) |
| Age                         | -0.0003 ($p = 0.22$) | 0.0002 ($p = 0.03$) |
| Smoking                     | 0.02 ($p = 0.04$) | 0.004 ($p = 0.47$) |
| Village type                | -0.007 ($p = 0.54$) | -0.002 ($p = 0.53$) |
| Number of people in household | -0.002 ($p = 0.45$) | -0.0001 ($p = 0.87$) |
| $R^2$                       | 0.22 | 0.17 |

Each entry shows the contribution of the explanatory variable to AR1 and ALRI rates (defined as the fraction of weeks with AR1/ALRI). The lowest exposure category (<200 µg/m$^3$) was used as the base category. Therefore, the entries for all other exposure categories are the additional fraction of weeks with illness relative to this category. The variable “Female” = 1 if the person is female and 0 if male; therefore the coefficient for “Female” is the additional fraction of weeks of illness among women compared to men, when all other factors have been accounted for. “Smoking” and “Village type” = 1 if a person smokes or lives in a maintenance village, respectively, and 0 otherwise; the coefficients have an interpretation similar to “Female.” The coefficient for “Age” indicates additional probability of being diagnosed with illness with each additional year of age. The shape of the exposure–response relationship is confirmed by analysis using a continuous exposure variable and inverse quadratic relationship. For the 5–49 year age group, we repeated the analysis by considering smoking as a source of exposure to particulate matter. With this change, the coefficient for “Smoking” is no longer significant ($p > 0.47$). The values of other coefficients and their p-values changed very little. Statistical significance remained unchanged for all other variables.
linear probability model with OLS estimation or the binomial model with maximum likelihood parameter estimation. An important implication is that public health programs aiming to reduce the negative impacts of indoor air pollution in developing countries should focus their attention on measures that result in larger reductions in pollution, especially those that bring average exposure below 2,000 µg/m³, confirming a concern that was raised qualitatively by Bruce et al. (33).

Exposure assessment methodology has commonly focused on average pollution levels. In the case of indoor smoke, where exposure occurs in an episodic manner, using average concentrations results in a systematic bias in assessment of exposure (37) and health impacts. We found that once total exposure is calculated to appropriately include high-intensity exposure episodes, sex does not provide an effective indicator of ARI and ALRI rates. We also found that the intensity of exposure does not contribute to the incidence of disease, once its role is accounted for in total exposure. At the same time, because combustion of biomass results in highly volatile pollution profiles (13,34), approximately one-half of daily exposure for the highest exposure groups (notably the individuals who cook) occurs during high-intensity episodes (37). This implies an important role for measures that reduce total exposure through the reduction of peak emissions.

Technology transfer programs and public health initiatives provide a variety of benefits in developing nations. With more than 2 billion people worldwide relying on biomass as their primary source of energy, efforts to introduce new energy technologies should also include detailed attention to health outcomes. A long record of national, multilateral, and private donor efforts to promote improved (high-efficiency and low-emissions) stoves exists (46). Many of these programs, although lowering average emissions, may not have reduced exposure below the 2,000 µg/m³ level (let alone to several hundreds of micrograms per cubic meter) that may provide important health benefits. The results of this analysis, for example, indicate that although improved wood stoves substantially reduce exposure, in many cases they offer smaller health benefits than a transition to charcoal, which can reduce exposure to very low levels. Other transitions through the “energy ladder” from wood to charcoal, or to kerosene, gas, and electricity, also require an evaluation of public health and environmental tradeoffs (such as impacts on vegetation and greenhouse gas emissions) of various energy technologies. In particular, armed with a richer quantitative understanding of health impacts of particular matter, development, public health, and energy research and development efforts that aim to reduce disease burden can effectively address acute respiratory infections.

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### Table 5. Odds ratios (OR) and 95% confidence intervals (CI) of the exposure–response relationship for ARIs and ALRIs using blogit regression (Equation 2).

| Explanatory variable | ARI | CI | OR | CI |
|----------------------|-----|----|----|----|
| 0-4 year group |
| Exposure category |
| <200 µg/m³ | Reference category |  | 2.42 (p < 0.01) |
| 200–500 µg/m³ | 1.53–3.83 | 1.48 (p = 0.18) |
| 500–1,000 µg/m³ | 1.30–3.56 | 1.40 (p = 0.30) |
| 1,000–2,000 µg/m³ | 2.63–7.04 | 2.33 (p = 0.009) |
| 2,000–3,500 µg/m³ | 2.02–7.88 | 2.03 (p = 0.05) |
| >3,500 µg/m³ | 1.75–12.06 | 2.93 (p = 0.007) |
| Female | 0.99 (p = 0.88) | 0.83–1.17 | 0.84 (p = 0.21) |
| Agea | 1.29 (p = 0.001) | 0.99–1.52 | 1.18 (p = 0.41) |
| Village type | 0.99 (p = 0.99) | 0.95–1.05 | 0.98 (p = 0.70) |

Each entry shows the odds ratio for the risk associated with the explanatory variable for ARI rates and ALRI rates. The lowest exposure category (<200 µg/m³) was taken as the reference category for the odds ratios of exposure groups. The variable “Female” = 1 if the person is female and 0 if male; therefore the coefficient for “Female” is the odds ratio for illness among women relative to men, when all other factors have been accounted for. “Smoking” and “Village type” = 1 if a person smokes or lives in a maintenance village, respectively, and 0 otherwise; the coefficients have an interpretation similar to “Female.” The coefficients for “Age” indicate the change in the odds of illness with the addition of an additional year of age. For the 5-49 year group, we repeated the analysis by considering smoking as a source of exposure to particulate matter. With this change, the coefficient of smoking is no longer significant (p > 0.47). The values of other coefficients and their p values changed very little. Statistical significance remained unchanged for all other variables.

*a Odds ratios for age and number of people in the household, which are both continuous variables, represent the odds ratios for two subsequent units of these variables. **Jointly significant (p < 0.01).
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