Quantifying the Effects of Exposure to Indoor Air Pollution from Biomass Combustion on Acute Respiratory Infections in Developing Countries

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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 (PM_{10}) generated from biomass combustion. Acute respiratory infections and acute lower respiratory infections are concave, increasing functions of average daily exposure to PM_{10}, 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]
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Acute respiratory infections (ARI) are the leading cause of the global burden of disease and account for more than 6% of the global burden of disease and mortality, mostly in developing countries (Figure 1) (*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 PM_{10} (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 in cross-sectional studies [for examples and discussion, see (*27,28–33*)]. Given the nearly universal use of biomass fuels in rural areas, this indirect approach to exposure estimation clusters many people into a single exposure category. Recent findings on large variations in emissions from individual stove types (*13,34*) and in exposure profiles within individual households (*35–37*), however, demonstrate that aggregate analysis and grouping of individuals dramatically reduces

the reliability of the estimation of the exposure–response relationship.

In this paper we report the first study that directly examines the exposure–response relationship for particulate matter from biomass combustion in a developing country. We have developed a unique data set from a field study in rural Kenya where we simultaneously collected detailed data on both exposure to indoor particulate matter and the health status of all the individuals in the study group over a period of more than 2 years; data used in this paper were collected between 1997 and 1999 as part of a long-term study of the relationship between energy technology, indoor air pollution, and public health. Detailed data on both variables at the individual level allows us to quantify the exposure–response relationship for indoor particulate matter from biomass combustion along a continuum of exposure levels. Particulate matter is only one of the pollutants in the complex mixture of biomass

smoke. Although numerous studies in industrialized and developing countries have identified particulate matter as the primary pollutant responsible for ARI, other gaseous and particulate products in biomass smoke, such as nitrogen dioxide and formaldehyde, are also known pulmonary irritants. Therefore, the results of this analysis apply to the mixture of pollutants whose effects are captured by particulate matter concentration.

Methods

Research location. The study took place at Mpala Ranch/Research Centre in Laikipia District, central Kenya (0°20´N, 36°50´E). Mpala Ranch, located on semi-arid land, is at an altitude of approximately 2,000 m, and the average monthly temperature varies between 17°C and 23°C. Cattle herding and domestic labor are the primary occupations of most of the 80–100 households residing on the ranch, with the remaining households employed as maintenance staff. The households have similar tribal backgrounds (Turkana and Samburu), economic status, and diet. The houses in both cattle-herding and maintenance villages are cylindrical with conic straw roofs. The households in the study group use unvented stoves and burn firewood or charcoal (and kerosene in the case of three or four households) for fuel. Detailed information on housing and energy technology in the study group has been previously reported (*37*).

Field research at Mpala Ranch began in 1996 and continued until late 1999. The first 6–10 months of field research involved collection of background data, including detailed demographic data for all the households residing on the ranch and surveys of energy use, energy technology, and related characteristics.

Data collection. We conducted continuous real-time monitoring of indoor air pollution [particulate matter < 10 μm in diameter (PM_{10}) and carbon monoxide] in 55 houses that were randomly selected from those households that resided on Mpala Ranch over a long fraction of the study period and from different villages and fuel types. Monitoring took place for 14–15 hr/day for more than 200 days. Studies of particulate matter pollution in both industrialized and developing countries have demonstrated correlation between concentrations of PM_{10} and $PM_{2.5}$ (which are believed to have the most important health impacts) (*23,38*), but further research on this relationship in the case of biomass smoke is needed. During these monitoring days we also recorded the location and activities of all members of the households, with emphasis on energy- and exposure-related variables. We also monitored the spatial dispersion of pollution inside the house.

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 Acute Respiratory Infection 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,
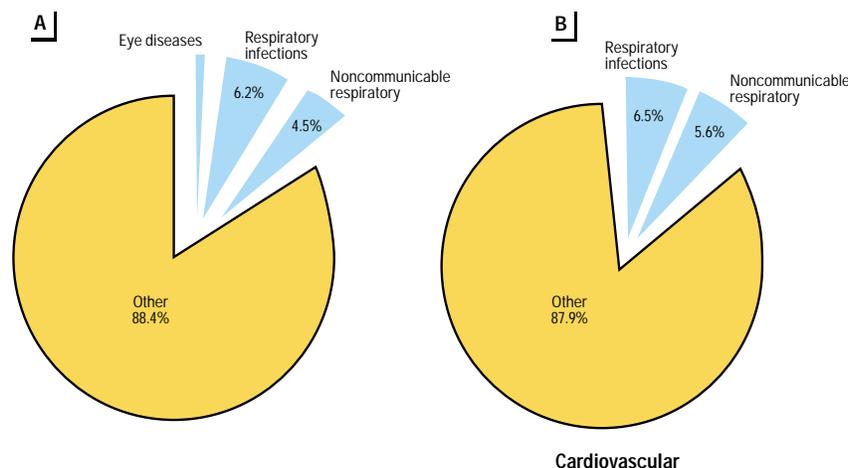
Figure 1. The share of global disease partially associated with exposure to air pollution (1998). Data from WHO (*1*) and Smith (*47*). (*A*) Global burden of disease [as percentage of disability-adjusted life years (DALYs)]; 1.38 billion DALYs were lost globally in 1998. (*B*) Mortality (as percentage of total mortality); there were 53.9 million global deaths in 1998. Noncommunicable respiratory diseases include chronic obstructive pulmonary disease (COPD) and asthma. Perinatal conditions and respiratory tract cancer have also been linked to exposure to indoor air smoke from biomass, but the evidence is weaker. Cardiovascular diseases have been linked to air pollution, but only in studies in industrialized countries.

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*)]:

$$\mathbf{y} = \mathbf{X} \times \beta + \mathbf{u}, \qquad [1]$$

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), β is the vector of coefficients, and **u** is the vector of independent, normally distributed errors.

$$\mathbf{y} = F(\mathbf{X} \times \beta + \mathbf{u}), \qquad [2]$$

where **y**, **X**, and β are defined as above, and *F* is the cumulative logistic distribution defined as:

$$F(z) = \frac{\exp(z)}{1 + \exp(z)} \qquad [3]$$

[In a logit or logistic regression model, the left hand side of Equation 2 is the probability of an event **y** (such as illness) or Pr{**y**}. Here, 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 obtained model parameters using ordinary-least-squares (OLS) regression for model 1 (Equation 1) with clustering in households and robust standard error estimates that account for outliers. For model 2 (Equation 2), we used a *blogit* regression using maximum-likelihood estimation. *blogit* 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. Illness rate, *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 2 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 ($p < 0.01$) and 0.024 ($p = 0.18$), 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 ($p = 0.49$) and 0.02 for ALRI ($p = 0.57$); 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 ($p < 0.0001$) and 0.011 ($p < 0.01$), respectively, between the lowest exposure group and 2,000 µg/m³, compared to 0.053 ($p < 0.001$) and 0.025 ($p < 0.001$), 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

Table 1. Demographic characteristics of the study group.

Age group	No. of individuals	Fraction female	Age (mean ± SD)
0–4 years	93	0.56	3.0 ± 1.4
5–14 years	109	0.56	9.7 ± 2.7
15–49 years	120	0.54	29.4 ± 10
≥ 50 years	23	0.65	63.8 ± 9.4
Total	345	0.56	18.3 ± 17.6

The mean age reflects the age at the end of the study. We chose these age divisions because children under 5 years of age have additional susceptibility to ARI; at higher ages, chronic conditions begin to appear. We chose to divide those between the ages of 5 and 49 years at the age of 15, when it is common for people to enter the workforce or to get married.

Table 2. Average daily exposure for demographic subgroups.

Age group	No. of individuals		Daily exposure (mg/m³)[a]	
	Female	Male	Female	Male
0–4 years	52	41	1.3 ± 1.2	1.4 ± 1.1
5–14 years	61	48	2.8 ± 2.1*	1.1 ± 0.6*
15–49 years	65	55	4.9 ± 3.7*	1.0 ± 1.0*
≥ 50 years	15	8	2.6 ± 1.5	2.2 ± 1.0

See Ezzati et al. (*37*) for details of methodology. Exposure values indicate the mean ± SD for all individuals in each demographic subgroup. The exposure values are relative to factory calibration of the measurement instrument, which is based on light-scattering properties of a standard mixture (dry Arizona road dust) with an uncertainty of 20% for wood smoke. The emission and exposure values reflect both emissions inside the house and contributions from ambient air including wind-blown dust and smoke from neighboring houses. Due to the extremely low housing density, the latter is likely to be negligible. [a]Based on a 24-hr period.*Difference between male and female rates significant at $p < 0.0001$.

Table 3. Number of health reports for the study group between early 1997 and June 1999.

Age group	Mean	Median	SD
0–4 years	72.2	85	23.9
5–14 years	82.2	88	16.3
15–49 years	80.5	87.5	17.7
≥ 50 years	73.9	82	19.1
Total	78.4	87	19.7

The numbers include only visits for which an adult member of the household was present.

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 $\mu g/m^3$ 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 $\mu g/m^3$ increase in average exposure is equivalent to 4 min of active inhalation of cigarette smoke, with an estimated particulate matter concentration of 400,000 $\mu g/m^3$.]

Weight 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 PM_{10} from biomass combustion and both ARI and ALRI is increasing, but the rate of increase declines at average daily exposures above 2,000 $\mu g/m^3$. For ALRI, the rate
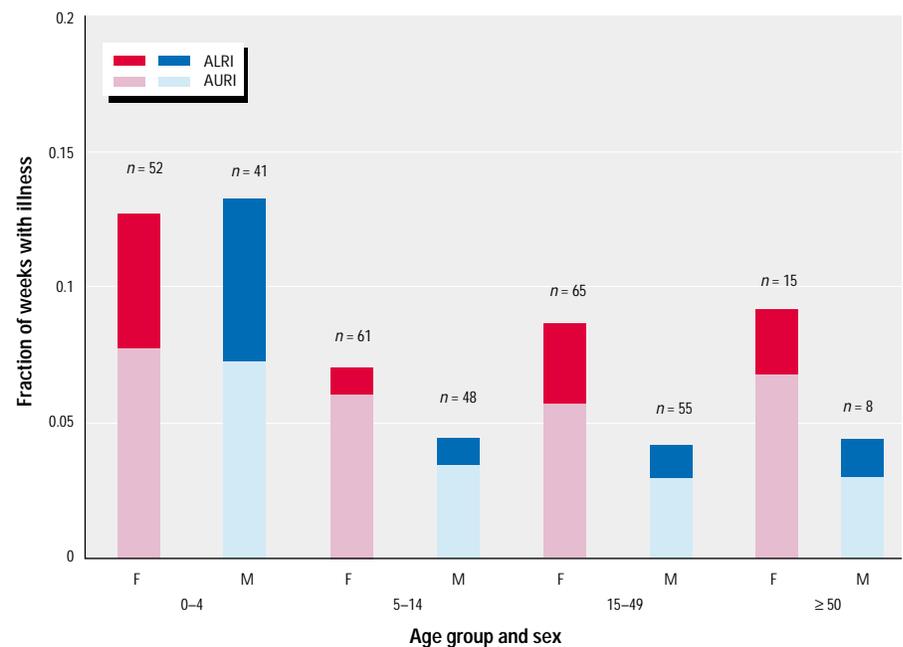

Figure 2. Demographic distribution of ARI at Mpala Ranch. The health end point is the fraction of weekly examinations in which an individual was diagnosed with ARI, divided into ALRI (including bronchitis, pneumonia, and bronchopneumonia) and acute upper respiratory infections (AURI) (*48*). The *p*-values for the differences between female (F) and male (M) ARI rates using two-sided two sample *t*-test with unequal variances are $p = 0.78$ for 0–4 years, $p = 0.01$ for 5–14 years, $p < 0.0001$ for 15–49 years, and $p = 0.01$ for ≥ 50 years. The *p*-values for the differences between female and male ALRI rates are $p = 0.36$ for 0–4 years, $p = 0.95$ for 5–14 years, $p = 0.0001$ for 15–49 years, and $p = 0.14$ for ≥ 50 years. Statistical significance is not sensitive to the use of unequal variances.

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, there is a slight lowering of ARI risk with increasing age for this group, which cannot be explained by known physiologic mechanisms, except for a potential increase in immunity, which is not expected to continue in higher ages.

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 combustion products from other forms of biomass. At the same time, smoking has been causally linked with many 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
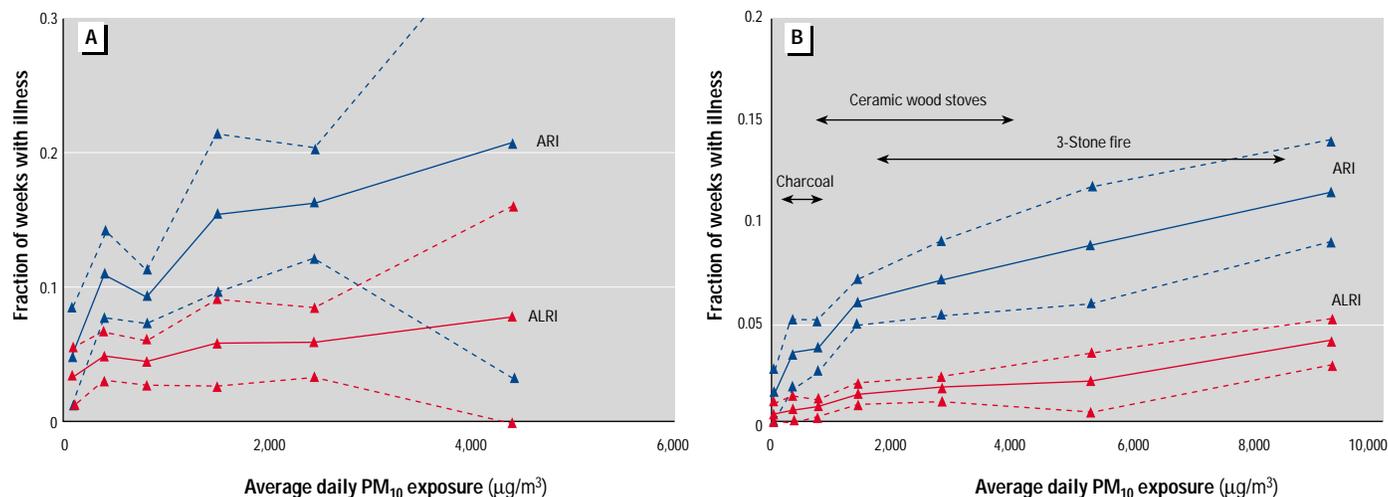

Figure 3. Unadjusted exposure–response relationship for ARI and ALRI (see Tables 4 and 5 for the adjusted relationship). (*A*) Age: 0–4 years (*n* = 93 individuals). (*B*) Age: 5–49 years (*n* = 229 individuals). Each group is divided into exposure categories to reflect the day-to-day variability of individual exposure. The exposure categories for (*A*) are < 200 μg/m³, 200–500 μg/m³, 500–1,000 μg/m³, 1,000–2,000 μg/m³, 2,000–3,500 μg/m³, and > 3,500 μg/m³. The exposure categories for (*B*) are < 200 μg/m³, 200–500 μg/m³, 500–1,000 μg/m³, 1,000–2,000 μg/m³, 2,000–4,000 μg/m³, 4,000–7,000 μg/m³, and > 7,000 μg/m³. Mean ARI and ALRI rates for each exposure category are plotted against the average exposure of the category. The shape of the curve is not sensitive to marginal modifications in exposure categories or to the use of median ARI and ALRI rates (instead of mean). The dotted lines connect the 95% confidence intervals (CI) for the mean ARI and ALRI in each exposure category. The larger CI for the last exposure category among infants and children (*A*) is due to the small number of children (*n* = 5) in the highest exposure category. In (*B*), the arrows indicate the interquartile range of exposures resulting from different fuel combinations obtained from comparison of stove emissions using previously described multiple descriptive statistics (*13*). "3-Stone fire" refers to the traditional open fire. Ceramic wood stoves are improved (high-efficiency and low-emission) wood stoves with a ceramic liner and metal body. "Charcoal" refers to all groups of charcoal stoves, including the older metal stove and improved models with an insulated liner. Among charcoal stoves, the improved models have lower emissions than the older metal stoves (*13*).

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 ARI 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 ARI ($p > 0.10$) beyond its contribution to total (or average) exposure. The coefficients of participation in household tasks are not jointly significant for ARI 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 *blogit* 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 ARI, 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 ARI, 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 $\mu g/m^3$. This result is robust to the choice of statistical model: the

Table 4. Parameters of the exposure–response relationship for ARI and ALRI using OLS regression (Equation 1).

Group/explanatory variable	ARI	ALRI
0–4 year group		
Constant	0.05 ($p = 0.45$)	0.07 ($p = 0.06$)
Exposure category		
< 200 $\mu g/m^3$	Reference category	Reference category
200–500 $\mu g/m^3$	0.06 ($p = 0.002$)*	0.01 ($p = 0.16$)
500–1,000 $\mu g/m^3$	0.06 ($p = 0.04$)*	0.01 ($p = 0.24$)
1,000–2,000 $\mu g/m^3$	0.13 ($p = 0.001$)*	0.03 ($p = 0.05$)
2,000–3,500 $\mu g/m^3$	0.14 ($p = 0.001$)*	0.03 ($p = 0.16$)
> 3,500 $\mu g/m^3$	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 $\mu g/m^3$	Reference category	Reference category
200–500 $\mu g/m^3$	0.027 ($p = 0.003$)*	0.0037 ($p = 0.48$)*
500–1,000 $\mu g/m^3$	0.022 ($p = 0.06$)*	0.0043 ($p = 0.32$)*
1,000–2,000 $\mu g/m^3$	0.039 ($p = 0.002$)*	0.011 ($p = 0.03$)*
2,000–4,000 $\mu g/m^3$	0.052 ($p = 0.001$)*	0.011 ($p = 0.03$)*
4,000–7,000 $\mu g/m^3$	0.064 ($p = 0.002$)*	0.013 ($p = 0.09$)*
> 7,000 $\mu g/m^3$	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 ARI and ALRI rates (defined as the fraction of weeks with ARI/ALRI). The lowest exposure category (< 200 $\mu g/m^3$) 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 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.
*Jointly significant ($p < 0.01$).

linear probability model with OLS estimation or the *blogit* 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 particulate 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 ARI and ALRI using *blogit* regression (Equation 2).

Explanatory variable	ARI		ALRI	
	OR	CI	OR	CI
0–4 year group				
Exposure category				
< 200 μg/m³	Reference category		Reference category	
200–500 μg/m³	2.42 ($p < 0.001$)*	1.53–3.83	1.48 ($p = 0.18$)*	0.83–2.63
500–1,000 μg/m³	2.15 ($p = 0.003$)*	1.30–3.56	1.40 ($p = 0.30$)*	0.74–2.67
1,000–2,000 μg/m³	4.30 ($p < 0.001$)*	2.63–7.04	2.33 ($p = 0.009$)*	1.23–4.38
2,000–3,500 μg/m³	4.72 ($p < 0.001$)*	2.82–7.88	1.93 ($p = 0.05$)*	0.99–3.78
> 3,500 μg/m³	6.73 ($p < 0.001$)*	3.75–12.06	2.93 ($p = 0.007$)*	1.34–6.39
Female	0.99 ($p = 0.88$)	0.83–1.17	0.84 ($p = 0.21$)	0.65–1.10
Age[a]	0.88 ($p < 0.001$)	0.83–0.94	0.76 ($p < 0.001$)	0.70–0.84
Village type	1.29 ($p = 0.06$)	0.99–1.67	1.18 ($p = 0.41$)	0.79–1.77
No. of people in household[a]	1.00 ($p = 0.99$)	0.95–1.05	0.98 ($p = 0.70$)	0.91–1.06
5–49 year group				
Exposure category				
< 200 μg/m³	Reference category		Reference category	
200–500 μg/m³	3.01 ($p = 0.001$)*	1.59–5.70	1.65 ($p = 0.41$)*	0.50–5.45
500–1,000 μg/m³	2.77 ($p = 0.001$)*	1.49–5.13	1.87 ($p = 0.27$)*	0.61–5.71
1,000–2,000 μg/m³	3.79 ($p < 0.001$)*	2.07–6.92	2.74 ($p = 0.07$)*	0.93–8.12
2,000–4,000 μg/m³	4.49 ($p < 0.001$)*	2.43–8.30	3.28 ($p = 0.03$)*	1.09–9.85
4,000–7,000 μg/m³	5.40 ($p < 0.001$)*	2.85–10.22	3.21 ($p = 0.05$)*	1.01–10.24
> 7,000 μg/m³	7.93 ($p < 0.001$)	4.11–15.27	7.10 ($p = 0.001$)	2.26–22.32
Female	1.24 ($p = 0.04$)	1.01–1.52	1.21 ($p = 0.39$)	0.78–1.88
Age[a]	0.99 ($p = 0.02$)	0.99–1.00	1.01 ($p = 0.02$)	1.00–1.02
Smoking	1.48 ($p = 0.02$)	1.07–2.04	1.53 ($p = 0.18$)	0.82–2.85
Village type	0.92 ($p = 0.41$)	0.76–1.12	0.93 ($p = 0.74$)	0.62–1.40
No. of people in household[a]	0.96 ($p = 0.04$)	0.93–1.00	0.99 ($p = 0.75$)	0.92–1.07

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 coefficient for "Age" indicates the odds ratio of being diagnosed with illness with each 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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