Effects of Cooking Smoke and Environmental Tobacco Smoke on Acute Respiratory Infections in Young Indian Children

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Background. Reliance on biomass for cooking and heating exposes many women and young children in developing countries to high levels of air pollution indoors. Environmental tobacco smoke also contributes to this indoor air pollution. This study estimates the effects of these sources of air pollution on acute respiratory infections (ARI) in children below 36 months of age in India.
Methods. The analysis is based on 29,768 children age 0–35 months included in India's 1998–1999 National Family Health Survey (NFHS-2). Logistic regression is used to estimate effects.
Results. In NFHS-2, children living in households using only biomass fuels for cooking or heating are almost twice as likely to have suffered from ARI during the 2 weeks before interview as children in households using cleaner fuels (OR = 1.82; 95% CI: 1.58, 2.09). This effect is somewhat reduced when exposure to environmental tobacco smoke (ETS) and a number of other factors are statistically controlled (OR = 1.58; 95% CI: 1.28, 1.95). With these other variables controlled, children in households using a mix of biomass and cleaner fuels also have a higher risk of ARI than children in households using only cleaner fuels (OR = 1.41; 95% CI: 1.17, 1.70). With controls, the effect of biomass fuel use on ARI is stronger for boys (OR = 1.68; 95% CI: 1.26, 2.23) than for girls (OR = 1.49; 95% CI: 1.11, 2.01). The effect of ETS on ARI is also positive and significant, but smaller when other variables (including type of cooking fuel) are controlled (OR = 1.12; 95% CI: 1.01, 1.23). No modifying effects of cooking smoke and ETS are observed.

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Conclusions. Findings support previous research showing positive effects of cooking smoke and ETS on ARI in young children. The relationship needs to be further investigated using more direct measures of smoke exposure and measures of outcome that are more specific to acute lower respiratory infections (ALRI), which are the most important component of overall ARI from a public health standpoint.

Medical subject headings (MeSH). air pollution; indoor; biomass; smoke; environmental tobacco smoke; respiratory tract infections; child; India.

INTRODUCTION

ARI is the leading cause of childhood illness and death worldwide, and its effects on children under age 5 years alone accounted for about 4.5% of the entire global burden of disease in 2002 (WHO, 2003). In India, as in many other developing countries, acute lower respiratory infection (ALRI), which is the more severe form of ARI, is the leading cause of childhood mortality (MOHFW, 2003). Indeed, ALRI in Indian children under five is thought to be responsible for about 490,000 deaths annually and is responsible for nearly 1.5% of the entire global burden of disease (Smith, 2003). The present study estimates the effect of household use of high-pollution biomass fuels (wood, dung, or crop residues) and environmental tobacco smoke (ETS) on ARI period prevalence in young children in India using data from a recent nationally representative household survey.

Used in simple stoves, biomass fuels are at the low end of the energy ladder in terms of combustion efficiency and cleanliness (Smith & Liu, 1994). Smoke from biomass combustion produces a large number of health-damaging air pollutants including respirable particulate matter, carbon monoxide, nitrogen oxides, formaldehyde, benzene, 1,3 butadiene, polycyclic aromatic hydrocarbons (such as benzo[a]pyrene), and many other toxic organic compounds. In developing countries, where large proportions of households rely on biomass fuels for cooking and space heating, they are typically burned in simple, inefficient, and mostly unvented household cookstoves, which, combined with poor ventilation, produce high indoor concentrations of pollutants (Bruce, Perez-Padilla, & Albalak, 2000). Moreover, these cookstoves are commonly used for several hours each day at times when people are frequently indoors, resulting in much higher exposure to air pollutants than from outdoor sources (Smith, 2002).

In such settings, daily average and peak exposures to air pollutants often far exceed safe levels recommended by the World Health Organization (WHO, 1997). A comparison of typical levels of CO, PM_{10}, and $PM_{2.5}$ in developing-country homes using biomass fuels with the United States Environmental Protection Agency's standards for 24-h average levels concluded that indoor concentrations of these pollutants in biomass-fuel-using developing-country homes usually exceed guideline levels by several fold (Bruce et al., 2000). In the case of smoke from cooking fuels, exposure levels are usually much higher among women, who tend to do most of the cooking (Behera, Dash, & Malik, 1988), and among young children, who stay indoors and who are often carried on their mother's back or lap while cooking (Albalak, Frisancho, & Keeler, 1999).

Exposure to biomass smoke from household cooking and space heating has been strongly associated with ARI in preschool age children in more than a dozen studies of different designs. For a comprehensive review of studies in developing-country households on indoor air pollution and ARI see Smith, Samet, Romieu, & Bruce (2000a). More recent studies in Zimbabwe by Mishra (2003), in Kenya by Ezzati, & Kammen (2001), and in India by Broor et al. (2001) and Mahalanabis et al. (2002) have found similar results. With varying degrees of evidence, other effects associated with biomass smoke include chronic obstructive pulmonary disease, lung cancer, cataracts, tuberculosis, stillbirth, low birth weight, and asthma (Bruce et al., 2000; Smith, 2000).

A recent quantitative review of studies on indoor air pollution and ARI (Smith, Mehta, & Maeusezahl-Feuz, 2004) includes a meta-analysis of published epidemiological studies, indicating an odds ratio of 2.3 (95% CI: 1.9, 2.7) for ALRI in children below 5 years (0–59 months). Two additional Indian hospital-based case–control studies that were published after this review was completed also show substantial effects of cooking smoke on ALRI at odds ratios of 2.5 (95% CI: 1.5, 4.2) (Broor et al., 2001) and 4.0 (95% CI: 2.7, 7.9) (Mahalanabis et al., 2002). Comparative risk studies done first for India alone (Smith, 2000) and then globally using such systematic reviews of the ALRI literature indicate that the ALRI mortality attributable to use of biomass fuels in India may have been 200,000–300,000 annually around 2000 (Smith et al., 2004).

The most well studied form of biomass smoke is that from tobacco, which has also been shown to be a risk factor for ARI in young children through exposure to environmental tobacco smoke (ETS), i.e., passive smoking (Strachen & Cook, 1997). In a meta-analysis of such studies, mostly done in developed countries, Janet, Jennifer, Xuan, & Berry (1999) found that young children whose parents smoke tobacco are at about twice the risk of having a serious respiratory tract infection requiring hospitalization.

The biological mechanisms by which biomass smoke can increase the risk of acute respiratory infections are not fully understood. Exposure to biomass smoke has been associated with compromised pulmonary immune-defense mechanisms (Thomas & Zelikoff, 1999). Tobacco smoke also has been shown to cause depressed immune system responses (Johnson, Houchens, Kluwe, Craig, & Fisher, 1990). Among the specific pollutants in biomass smoke, exposure to respirable particulate matter (PM_{10}) has been shown to induce a systemic inflammatory response that includes stimulation of the bone marrow, which can contribute to the pathogenesis of cardiorespiratory morbidity (Mukae et al., 2001; van Eeden et al., 2001). Other evidence indicates that exposure to polycyclic aromatic hydrocarbons (PAH)—especially benzo[a]pyrene (B[a]P), which is found in large quantities in biomass smoke—can cause immune suppression and can increase the risk of infection and disease (Kong, Luster, Dixon, O'Grady, & Rosenthal, 1994). Moreover, acute and long-term exposures to oxides of nitrogen, commonly found in biomass smoke, can increase bronchial reactivity and susceptibility to bacterial and viral infections (Samet & Spengler, 1991). Young children, with immature immune systems and rapidly developing lungs, may be particularly vulnerable to adverse effects of air pollution exposures, which may impair their pulmonary defense mechanisms and render them more susceptible to acute respiratory infections.

METHODS

Data

Data are from India's second National Family Health Survey (NFHS-2) conducted in 1998–1999. NFHS-2 collected demographic, socioeconomic, and health information from a nationally representative probability sample of 92,486 households. All states of India are represented in the sample, covering more than 99% of the country's population. The sample is a multi-stage cluster sample with an overall household response rate of 98% and an overall woman response rate of 96%. The data were collected during November 1998 to December 1999. Details of sample design, including sampling frame and sample implementation, are provided in the basic survey report for all India (IIPS & ORC Macro, 2000). The analysis here is based on 29,768 children 0–35 months old living in the sample households.

Response Variable

For each child under age 36 months, the mother was asked, "Has [the child] been ill with a cough at any time in the last two weeks?"(Q#465). If the answer was yes, the mother was additionally asked, "When [the child] was ill with a cough, did he/she breathe faster than usual with short, rapid breaths?" (Q#466). Children whose mothers responded positively to both questions are defined here as having suffered from an acute respiratory infection. This is the response variable in our analysis (1 if the child suffered from ARI, 0 otherwise).

Predictor Variables

Exposure to cooking smoke is ascertained indirectly by type of fuel used for cooking or heating. Measurements in India show that the emission of pollutants from household stoves directly varies along the "energy ladder," with solid fuels producing substantially more pollution per meal cooked than liquid or gaseous fuels (Smith et al., 2000b). Fuel type has also been shown in India to be a good predictor of indoor pollution levels in households (Mehta, 2002). The NFHS-2 used a ten-fold classification of cooking fuel—wood, crop residues, dung cakes, coal/coke/lignite, charcoal, kerosene, electricity, liquid petroleum gas, biogas, and a residual category of other fuels. The question was, "What type of fuel does your household mainly use for cooking?" (Q#37) followed by the above list of fuels. The survey also included a second question, "What other types of fuel does your household commonly use for cooking or heating?" (Q#38)—with the same ten-fold classification of fuels. This second question was a multiple-response question, so that a respondent could choose more than one fuel.

We use information from these questions to group households into three categories representing the extent of exposure to cooking smoke—a high-exposure group (households using only biomass fuels: wood, crop residues, or dung cakes), a low-exposure group (households using only cleaner fuels: electricity, liquid petroleum gas, biogas, or kerosene), and a medium-exposure group (a mix of biomass fuels and cleaner fuels or coal/coke/lignite/charcoal). This three-category classification of fuels is the principal predictor variable in our analysis. The small number of cases in the residual category of "other fuels" were excluded from the analysis.

The survey also collected information on tobacco smoking. The NFHS-2 asked the household respondent in a sampled household, "Does anyone listed smoke?" (Q#26) Exposure to environmental tobacco smoke was defined as 1 if at least one person in the household currently smoked at the time of the survey and 0 otherwise. All types of tobacco smoking were included, commercial and non-commercial.

Because the effects of exposure to cooking smoke, as well as tobacco smoke, on the prevalence of ARI are likely to be confounded with the effects of other risk factors, it is necessary to statistically control for such factors to the extent possible. Our control variables, all of which have been identified in previous research as covariates of ARI, include age of child in months (0–5, 6–11, 12–23, 24–35), sex of child (boy, girl), birth order of child (1, 2, 3, 4+), nutritional status of child (stunted, not stunted), mother's age at childbirth (13–24, 25–34, 35–49), mother's education (illiterate, <middle school, middle complete or higher), religion of household head (Hindu, Muslim, other), caste/tribe status of household head (scheduled caste or scheduled tribe, other backward class, other), house type (*pucca*, semi-*pucca*, *kachha*–see explanation in Table 1), availability of a separate kitchen (yes, no), household crowding (<3 persons per room, 3+ persons per room), household standard of living (low, medium, high), residence (urban, rural), and region of residence (North, Central and West, East and Northeast, South). In one of our initial models, we also included an interaction term between biomass fuel use and ETS to test whether exposure to tobacco smoke modifies the effect of cooking smoke or *vice versa*, but the interaction effect was not significant and did not alter the independent effects of cooking smoke and ETS. In the final analysis, therefore, the interaction term was dropped from the models. Table 1 provides further detail on definitions of variables.

Method of Analysis

Because our response variable—prevalence of ARI—is dichotomous, we use logistic regression to estimate the effects of cooking smoke (from biomass fuel use relative to cleaner fuel use) and environmental tobacco smoke on ARI prevalence in the 2 weeks preceding the NFHS-2. A number of multivariate logistic regression models are estimated using different combinations of the 14 potentially confounding variables mentioned above. Because of gender differences in feeding, treatment, and care of young children in India, boys and girls may be exposed to different levels of air pollutants from cooking and smoking, and the effects of such exposure may vary by sex of child. For these reasons, we also estimate separate models for boys and girls.

In the survey, certain states and certain categories of households were over-sampled and non-response rates varied from one geographical area to another. In our analysis, weights are used to restore the representativeness

TABLE 1

Sample Distribution and Reported Period Prevalence of Acute Respiratory Infections (ARI) in Children Age 0–35 months during the 2 weeks Preceding the Survey by Selected Characteristics, India, 1998–1999

Characteristic	Sample distribution (%)	ARI prevalence (%)
India	–	19.6
Cooking smoke[a]		
Biomass fuels	64.2	21.2
Fuel mix	23.5	18.4
Cleaner fuels	12.3	13.0
Environmental tobacco smoke[b]		
Yes	51.1	21.2
No	48.9	17.9
Age of child (in months)		
0–5	16.5	17.5
6–11	16.1	23.9
12–23	33.8	20.3
24–35	33.6	17.8
Sex of child		
Boy	51.8	20.9
Girl	48.2	18.1
Birth order		
1	26.8	20.4
2	25.5	18.7
3	17.9	19.0
4+	29.8	19.9
Nutritional status of child		
Stunted[c]	46.0	21.0
Not stunted	54.0	19.4
Mother's age at childbirth		
13–24	60.7	20.3
25–34	34.8	18.2
35–49	4.5	20.7
Mother's education		
Illiterate	60.4	20.6
Literate, <middle complete	17.5	20.5
Middle complete or higher	22.1	16.0
Religion		
Hindu	79.0	19.3
Muslim	15.9	21.2
Other[d]	5.0	19.2
Caste/tribe[e]		
Scheduled caste/scheduled tribe	30.7	20.4
Other backward class	32.2	19.1
Other	37.2	19.3

TABLE 1 (*Continued*)

Characteristic	Sample distribution (%)	ARI prevalence (%)
House type[f]		
Pucca	25.9	16.4
Semi-*pucca*	37.3	20.4
Kachha	36.8	21.0
Separate kitchen		
Yes	45.1	18.3
No	54.9	20.6
Crowding		
<3 persons per room	44.6	18.7
≥3 persons per room	55.4	20.3
Standard of living[g]		
Low	46.8	21.0
Medium	40.0	19.5
High	13.2	15.1
Residence		
Urban	22.3	16.3
Rural	77.7	20.5
Region[h]		
North	26.9	19.5
Central and West	28.5	20.0
East and Northeast	26.3	22.4
South	18.3	15.1
Number of children[i]	29,081	27,095

[a] Biomass fuels: wood, animal dung, or crop residues; fuel mix: mix of biomass fuels and cleaner fuels, or coal/coke/lignite; cleaner fuel: kerosene, petroleum gas, biogas, or electricity.

[b] Child lives in a household where one or more persons currently smoke.

[c] Stunting is a measure of linear growth retardation in children. A child whose height-for-age is more than two standard deviation units below the median of the International Reference Population is defined as stunted.

[d] Sikh, Buddhist, Christian, Jain, Jewish, Zoroastrian, etc.

[e] Scheduled castes (SC), scheduled tribes (ST), and other backward classes are those castes and tribes designated by the Government of India as socially and economically backward and in need of protection from social injustice and exploitation.

[f] *Kachha* houses are made from mud, thatch or low-quality materials. *Pucca* houses are made from high-quality materials (such as bricks, tiles, cement, and concrete) throughout, including roof, walls, and floor. Semi-*pucca* houses are made from partly low-quality materials and partly high-quality materials.

[g] Standard of living index (SLI) is calculated by adding the scores assigned to the durable goods in the household as following: 4 for a car or tractor; 3 each for a moped/scooter/motorcycle, telephone, refrigerator, or color television; 2 each for a bicycle, electric fan, radio/transistor, sewing machine, black and white television, water pump, bullock cart, or thresher; and 1 each for a mattress, pressure cooker, chair, cot/bed, table, or clock/watch. Index scores range from 0 to 5 for low SLI, 6 to 15 for medium SLI, 16 to 42 for high SLI.

[h] North: Jammu & Kashmir, Himachal Pradesh, Haryana, Punjab, Delhi, Uttar Pradesh; Central and West: Maharashtra, Gujarat, Madhya Pradesh, Rajasthan; East and Northeast: Bihar, West Bengal, Orissa, Assam, Arunachal Pradesh, Manipur, Meghalaya, Mizoram, Nagaland, Sikkim, Tripura; South: Andhra Pradesh, Karnataka, Kerala, Tamil Nadu, Goa.

[i] Number of children varies slightly for individual variables depending on the number of missing values.

of the sample (IIPS & ORC Macro, 2000). Results are presented in the form of odds ratios (OR) with 95% confidence intervals (95% CI). The estimation of confidence intervals takes into account design effects due to clustering at the level of the primary sampling unit (village or urban block). The analysis is restricted to last births in order to avoid clustering at the mother level. Logistic regression models were estimated using the STATA statistical software package (Stata Corporation, 2003).

RESULTS

Table 1 shows how the sample of children is distributed on the variables included in the analysis. This table also shows ARI two-week period prevalence (raw prevalence, with no controls) for each category of each predictor variable.

Effects of Cooking Smoke and Environmental Tobacco Smoke

Table 2 shows the main results of the logistic regression analysis, based on all children regardless of their sex. In the table, Model 1 shows unadjusted odds ratios (no controls) indicating the effect of cooking smoke on ARI, and Model 2 shows unadjusted odds ratios indicating the effect of ETS on ARI. Model 3 includes both cooking smoke and ETS as predictors. Models 4, 5, and 6 progressively add child characteristics, mother's characteristics, and housing and geographic characteristics as additional predictors, which function as controls.

Model 1 shows that the unadjusted odds of having suffered from ARI are almost two times higher among children living in households using high-pollution biomass fuels than among those living in households using low-pollution cleaner fuels, including electricity, LPG, biogas, or kerosene, for cooking (OR = 1.82; 95% CI: 1.58, 2.09). Children in households using a mix of biomass fuels and cleaner fuels or coal/coke/lignite/charcoal also have higher ARI prevalence (OR = 1.52; 95% CI: 1.30, 1.77) than children in households using cleaner fuels only. Model 2 shows that the unadjusted odds of ARI are also higher for children exposed to ETS than children not so exposed (OR = 1.22; 95% CI: 1.12, 1.33). Model 3 shows that the unadjusted effects of cooking smoke and ETS are both reduced slightly when cooking smoke and ETS are both included in the model.

The effects of cooking smoke and ETS are not reduced much when child's age, sex, birth order, and nutritional status are additionally

TABLE 2

Unadjusted and Adjusted Effects (OR, 95% CI) of Cooking Smoke, Environmental Tobacco Smoke, and other Factors on ARI Period Prevalence in Children Age 0–35 months, India, 1998–1999

Characteristic	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Cooking smoke												
Biomass fuels	1.82	(1.58, 2.09)			1.75	(1.52, 2.03)	1.80	(1.54, 2.10)	1.61	(1.36, 1.90)	1.58	(1.28, 1.95)
Fuel mix	1.52	(1.30, 1.77)			1.49	(1.28, 1.73)	1.51	(1.29, 1.77)	1.41	(1.20, 1.66)	1.41	(1.17, 1.70)
Cleaner fuels[a]	1.00	–			1.00	–	1.00	–	1.00	–	1.00	–
Environmental tobacco smoke												
Yes			1.22	(1.12, 1.33)	1.14	(1.04, 1.24)	1.13	(1.03, 1.24)	1.11	(1.01, 1.22)	1.12	(1.01, 1.23)
No[a]			1.00	–	1.00	–	1.00	–	1.00	–	1.00	–
Age of child (in months)												
0–5[a]							1.00	–	1.00	–	1.00	–
6–11							1.39	(1.21, 1.59)	1.41	(1.22, 1.62)	1.44	(1.26, 1.66)
12–23							1.11	(0.97, 1.27)	1.13	(0.99, 1.29)	1.17	(1.03, 1.34)
24–35							0.98	(0.85, 1.12)	0.99	(0.86, 1.14)	1.02	(0.89, 1.17)
Sex of child												
Boy[a]							1.00	–	1.00	–	1.00	–
Girl							0.82	(0.76, 0.89)	0.82	(0.75, 0.89)	0.82	(0.76, 0.89)

TABLE 2 (*Continued*)

Birth order			
1[a]	1.00 –	1.00 –	1.00 –
2	0.90 (0.80, 1.01)	0.91 (0.81, 1.02)	0.91 (0.81, 1.03)
3	0.86 (0.75, 0.97)	0.85 (0.74, 0.98)	0.83 (0.73, 0.96)
4+	0.91 (0.81, 1.02)	0.94 (0.81, 1.08)	0.89 (0.77, 1.03)
Nutritional status of child			
Stunted	1.09 (1.00, 1.20)	1.06 (0.97, 1.16)	1.02 (0.93, 1.12)
Not stunted[a]	1.00 –	1.00 –	1.00 –
Mother's age at childbirth			
13–24[a]		1.00 –	1.00 –
25–34		0.88 (0.79, 0.98)	0.88 (0.79, 0.98)
35–49		0.94 (0.76, 1.16)	0.94 (0.76, 1.17)
Mother's education			
Illiterate[a]		1.00 –	1.00 –
Literate, <middle complete		0.95 (0.85, 1.07)	0.98 (0.87, 1.11)
Middle complete or higher		0.79 (0.69, 0.91)	0.87 (0.75, 1.00)
Religion			
Hindu[a]		1.00 –	1.00 –
Muslim		1.07 (0.93, 1.22)	1.08 (0.94, 1.24)
Other		1.04 (0.86, 1.26)	1.08 (0.89, 1.31)

TABLE 2 (*Continued*)

Characteristic	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Caste/tribe						
Scheduled caste/scheduled tribe					1.00 (0.89, 1.13)	1.02 (0.90, 1.15)
Other backward class					0.97 (0.87, 1.09)	1.04 (0.93, 1.17)
Other[a]					1.00 –	1.00 –
House type						
Pucca						1.06 (0.92, 1.22)
Semi-*pucca*						1.08 (0.97, 1.21)
Kachha[a]						1.00 –
Separate kitchen						
Yes						0.98 (0.88, 1.09)
No[a]						1.00 –
Crowding						
<3 persons per room[a]						1.00 –
≥3 persons per room						0.99 (0.90, 1.09)
Standard of living						
Low[a]						1.00 –
Medium						0.95 (0.85, 1.06)
High						0.83 (0.69, 1.00)

TABLE 2 (*Continued*)

Residence							
Urban						1.03	(0.89, 1.19)
Rural[a]						1.00	–
Region							
North						1.51	(1.26, 1.81)
Central and West						1.55	(1.31, 1.82)
East and Northeast						1.57	(1.34, 1.85)
South[a]						1.00	–
Number of children	24,667	24,789	24,667	20,023	19,872	19,790	

Note: For variable definitions, see Table 1.

[a] Reference category.

controlled in Model 4, when mother's age at childbirth, education, religion, and caste/tribe are additionally controlled in Model 5, and when house type, separate kitchen, crowding, household living standard, urban/rural residence, and geographic region are additionally controlled in Model 6, which is the full model. In Model 6, the effect of using biomass fuels only, relative to cleaner fuels, is still large (OR = 1.58; 95% CI: 1.28, 1.95), as is the effect of fuel mix (OR = 1.41; 95% CI: 1.17, 1.70). The effect of ETS is affected hardly at all by the additional controls (OR = 1.12; 95% CI: 1.01, 1.23).

Effects of Other Factors

With cooking smoke, ETS, and other variables controlled, child's age, sex, birth order, mother's age at childbirth, household living standard, and geographic region have statistically significant effects on childhood ARI (Model 6 in Table 2). The odds of ARI are higher at ages 6–23 months than at ages 0–5 months or 24–35 months. Girls are significantly less likely to have suffered from ARI than boys (OR = 0.82; 95% CI: 0.76, 0.89). First-order births and children of younger mothers (age 13–24 at the time of childbirth) are somewhat more likely to have suffered from ARI than other children. Children from households with a high standard of living have significantly lower prevalence of ARI than children in poorer households. The prevalence of ARI is significantly lower in the South region than in other regions of India. None of the other background factors included in the analysis show any statistically significant effects on childhood ARI.

Separate Analysis by Sex of Child

Table 3 repeats the full model (Model 6) in Table 2 separately for boys and girls to test whether the effects of cooking smoke and ETS are different for boys and girls. The effect of biomass fuel use on childhood ARI is positive and significant for both boys and girls, but somewhat greater for boys (OR = 1.68; 95% CI: 1.26, 2.23) than for girls (OR = 1.49; 95% CI: 1.11, 2.01). The effect of ETS is also somewhat stronger for boys (OR = 1.18; 95% CI: 1.04, 1.33) than for girls (OR = 1.05; 95% CI: 0.91, 1.21). The effect of ETS is not statistically significant at the 5% level for girls.

Among the control variables, only child's age and geographic region have statistically significant effects for both boys and girls. Boys and girls age 6–11 months had significantly higher prevalence of ARI than children of other ages, and boys and girls in the South region had significantly lower

TABLE 3

Adjusted Effects (OR, 95% CI) of Cooking Smoke, Environmental Tobacco Smoke, and other Factors on ARI Period Prevalence in Children Age 0–35 months, by Sex of Child, India, 1998–1999

Characteristic	Boy		Girl	
	OR	(95% CI)	OR	(95% CI)
Cooking smoke				
Biomass fuels	1.68	(1.26, 2.23)	1.49	(1.11, 2.01)
Fuel mix	1.39	(1.08, 1.79)	1.46	(1.12, 1.91)
Cleaner fuels[a]	1.00	–	1.00	–
Environmental tobacco smoke				
Yes	1.18	(1.04, 1.33)	1.05	(0.91, 1.21)
No[a]	1.00	–	1.00	–
Age of child (in months)				
0–5[a]	1.00	–	1.00	–
6–11	1.41	(1.17, 1.70)	1.48	(1.21, 1.82)
12–23	1.19	(1.00, 1.42)	1.15	(0.94, 1.41)
24–35	1.00	(0.83, 1.21)	1.05	(0.85, 1.30)
Birth order				
1[a]	1.00	–	1.00	–
2	0.92	(0.79, 1.08)	0.90	(0.75, 1.09)
3	0.78	(0.65, 0.94)	0.90	(0.73, 1.11)
4+	0.75	(0.62, 0.91)	1.11	(0.89, 1.40)
Nutritional status of child				
Stunted	0.95	(0.84, 1.08)	1.11	(0.96, 1.27)
Not stunted[a]	1.00	–	1.00	–
Mother's age at childbirth				
13–24[a]	1.00	–	1.00	–
25–34	1.02	(0.89, 1.17)	0.73	(0.62, 0.86)
35–49	0.95	(0.71, 1.27)	0.90	(0.66, 1.24)
Mother's education				
Illiterate[a]	1.00	–	1.00	–
Literate, <middle complete	0.97	(0.83, 1.14)	0.98	(0.82, 1.17)
Middle complete or higher	0.82	(0.67, 0.99)	0.92	(0.74, 1.14)
Religion				
Hindu[a]	1.00	–	1.00	–
Muslim	1.13	(0.95, 1.36)	1.02	(0.83, 1.24)
Other	1.07	(0.83, 1.37)	1.11	(0.84, 1.47)
Caste/tribe				
Scheduled caste/scheduled tribe	1.09	(0.93, 1.28)	0.93	(0.78, 1.11)
Other backward class	1.18	(1.02, 1.37)	0.89	(0.75, 1.06)
Other[a]	1.00	–	1.00	–
House type				
Pucca	1.26	(1.04, 1.52)	0.85	(0.70, 1.04)

TABLE 3 (*Continued*)

Characteristic	Boy		Girl	
	OR	(95% CI)	OR	(95% CI)
Semi-*pucca*	1.11	(0.96, 1.28)	1.06	(0.91, 1.24)
Kachha[a]	1.00	–	1.00	–
Separate kitchen				
Yes	0.97	(0.85, 1.10)	0.99	(0.84, 1.16)
No[a]	1.00	–	1.00	–
Crowding				
<3 persons per room[a]	1.00	–	1.00	–
≥3 persons per room	1.05	(0.92, 1.20)	0.92	(0.80, 1.07)
Standard of living				
Low[a]	1.00	–	1.00	–
Medium	0.91	(0.78, 1.06)	0.99	(0.85, 1.16)
High	0.82	(0.64, 1.05)	0.85	(0.65, 1.11)
Residence				
Urban	1.05	(0.88, 1.27)	0.99	(0.81, 1.21)
Rural[a]	1.00	–	1.00	–
Region				
North	1.59	(1.27, 1.99)	1.42	(1.11, 1.81)
Central and West	1.60	(1.31, 1.96)	1.49	(1.19, 1.86)
East and Northeast	1.57	(1.28, 1.92)	1.58	(1.26, 1.97)
South[a]	1.00	–	1.00	–
Number of children	10,535		9,255	

Note: For variable definitions, see Table 1.
[a] Reference category

prevalence of ARI than in other regions. Effects of all other variables included in the analysis are generally small and non-significant, except the effects of birth order, mother's education, and house type for boys and the effect of mother's age at childbirth for girls.

DISCUSSION

Acute respiratory infections are a serious problem in India. One in five children under age 36 months suffered from ARI as defined in this study during the 2 weeks preceding NFHS-2. The survey also shows that almost two-thirds of children under age 36 months live in households that use only unprocessed biomass fuels for cooking. Our logistic regression analysis shows that exposure to smoke from biomass fuels, relative to cleaner fuels, has significant effects on ARI period prevalence in young children, independent of environmental tobacco smoke, child's age, nutritional status, maternal education, household living standard, and other factors (OR = 1.58; 95% CI: 1.28, 1.95). Children in households that use a mix of biomass fuels and cleaner fuels also have significantly higher prevalence of ARI than children in households using only cleaner fuels (OR = 1.41; 95% CI: 1.17, 1.70). These results are consistent with previous research, and provide further evidence that cooking with high pollution fuels can increase the risk of acute respiratory infections in young children. With other factors (including type of cooking fuel) controlled, ETS also has a significant effect on ARI (OR = 1.12; 95% CI: 1.01, 1.23). Perhaps because ventilation is typically higher in Indian households than in the developed-country settings where most ETS studies have been done, the size of the effect of ETS on childhood ARI, measured as presence of smokers in the household, is smaller than in other studies (Janet et al., 1999).

In NFHS-2, prevalence of ARI is somewhat higher among boys than among girls, and our analysis shows that the effects of cooking smoke and ETS are also somewhat higher among boys than among girls. These results are consistent with a previous study of effect of biomass fuel use on childhood ARI in India, based on the 1992–1993 National Family Health Survey (Mishra, Retherford, & Smith, 2002). A lower prevalence of ARI among girls could occur if there is more underreporting of ARI for girls than for boys, and a lower effect of cooking smoke on ARI for girls, relative to boys, could occur if underreporting of ARI for girls is greater in biomass-fuel-using households than in cleaner-fuel-using households. Sex differentials could also occur if, as is common in India, mothers are more likely to carry young boys or keep them in the kitchen area while cooking than young girls, thereby inadvertently exposing boys to higher levels of air pollution. In this case, ironically, discrimination against girls would work to their advantage.

Several measurement constraints should be kept in mind when considering the findings of this study. ARI is composed of two major components, acute lower respiratory infections (ALRI) and acute upper respiratory infections (AURI), which often share superficial symptoms. The incidence of AURI in Indian children, however, is thought to be about 15 times that for ALRI (Murray & Lopez, 1996). Unlike ALRI, AURI is common in children throughout the world, with probably less than a factor-of-two difference in incidence between rich and poor countries, compared with much larger differences for ALRI. In contrast to the high incidence of AURI, the case fatality rate for AURI is very low (approximately 1/1500 as high as the case fatality rate for ALRI in Indian children). Because of this and its generally low severity and short duration, AURI is not considered an important public health burden, accounting for much less than 1% of the burden of ALRI in Indian children (WHO, 2003).

The two questions in NFHS-2 were designed to capture ALRI period prevalence, but because of the inherent limitations of such large surveys (restricted number of questions about any one issue; self-reporting of health conditions; non-medical survey personnel, etc.), they probably do not have high specificity for ALRI; i.e., a significant proportion of the reported prevalence may actually have been AURI (WHO, 2003). Evidence for this low specificity is that the period prevalence as estimated by this study of approximately one-fifth translates crudely into an annual incidence of about five for children 0–35 months, some 10 times greater than the annual incidence rate of ALRI in young Indian children (0.45) and not too different from what is thought to be the rate for all ARI (AURI plus ALRI) for 0–5-year-olds in India (Murray & Lopez, 1996). Thus, it is clear that the questions used in the questionnaire were not specific to ALRI and picked up much AURI as well. The question about fast breathing (Q#466) is designed to differentiate ALRI and AURI, but judgment about fast breathing is difficult to apply by recall. Even trained field workers with stopwatches often have difficulty in determining fast breathing accurately. In addition, the rate of breathing considered ''fast'' is a function of the age of the baby (Lanata et al., 2004).

There is also a possible loss of sensitivity (i.e., some of the actual ALRI may have been missed) due to the use of only a single question on fast breathing. The official recommendations for determining ALRI include three possible indicators: fast breathing, chest indrawing, and/or ''too sick to feed'' (Lanata et al., 2004). Since two of these questions were not asked, there is a possibility that some ALRI was missed.

In summary, although the symptomatic definition used here was intended to measure ALRI in children, considerable AURI is probably included in the reported prevalence and some ALRI was probably missed. Consequently, we use the term ARI in this study. The Indian studies of ALRI, where trained fieldworker determination based on symptoms was validated using medical personnel, often in conjunction with X-rays, have been able to show up to about 90% specificity and about 90% sensitivity, but of course, because of the intrinsic trade-off between the two, when one is higher in a particular study, the other tends to be lower (Lanata et al., 2004). As noted above, specificity in our study is likely to be substantially lower than these best-case situations.

To the extent that children living in poorer biomass-fuel-using households are more likely to die from ARI, our estimates of effect of cooking smoke on ARI may be biased downwards. Given the high prevalence of ARI and relatively small number of deaths in the sample, however, the impact of this bias on our estimated effect is likely to be small. Moreover, our estimated effects may be biased due to seasonal variations in the type of fuel use and in the prevalence of ARI. However, because the survey period covered an entire year (November 1998 to December 1999), any effect of seasonality on the estimated effects is likely to be small. Also, we were not able to control directly for extent of use of medical services. Our set of control variables includes measures of SES, however, which are correlated with access to and use of medical services.

The advantage of this household survey, of course, is the large representative sample size and the ability of the researcher to control for a wide range of possible confounders. The disadvantage of being part of a large survey without validation, however, is a relatively low specificity, although perhaps reasonable sensitivity, compared with the best published studies. The somewhat lower odds ratios estimated in this study, compared with odds ratios in smaller and more focused ALRI studies (Smith et al., 2004), may well be due to the dilution of measured effect on ALRI by the large number of AURI cases picked up by the questionnaire. Nearly all such studies to date have relied on indirect indicators of exposure, such as solid fuel use as used here, but better measures would undoubtedly reduce the effects of exposure misclassification (Smith et al., 2004).

Despite these problems in the measurement of smoke exposure and ARI, the consistency in the size of crude and adjusted effects of biomass fuel use on childhood ARI suggests a possible 'exposure–response' relationship. To validate this relationship, our research needs to be followed by carefully designed epidemiological studies, preferably with direct measures of smoke exposure and validated measures of ALRI. Such research is important, both because a large proportion of households in India and other developing countries rely on biomass fuels for household energy, and because ALRI is a leading cause of ill health and death in young children.

Given heavy reliance on biomass fuels for household energy and high prevalence of ARI in children in India, the findings of this study add weight to the to growing evidence from around the world of significant health impacts.

The most important intervention to reduce exposure to indoor air pollution is to promote widespread use of cleaner fuels, such as LPG and electricity. However, given that many poor households in India that currently rely on biomass fuels are unlikely to be able to afford cleaner fuels soon, and given the poor infrastructure for supplying cleaner fuels to rural households, widespread adoption of cleaner fuels is unlikely to occur in the short term. Therefore, efforts need to focus on providing improved cookstoves designed to reduce exposure to smoke by means of improved combustion and improved venting, and on designing and implementing public information campaigns to inform people about the health risks of exposure to indoor smoke. In addition to action and resources by international and national authorities, local needs and community participation should receive high priority if such programs are to be effective.

HUMAN SUBJECTS INFORMED CONSENT

The analysis presented in this paper is based on secondary analysis of existing survey data with all identifying information removed. The survey obtained informed consent from each respondent (in this case, mothers of the children included in the analysis) before asking questions.

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REFERENCES

Albalak, R., Frisancho, A. R., & Keeler, G. J. (1999). Domestic biomass fuel combustion and chronic bronchitis in two rural Bolivian villages. *Thorax, 54*, 1004–1008.

Behera, D., Dash, S., & Malik, S. K. (1988). Blood carboxyhaemoglobin levels following acute exposure to smoke of biomass fuel. *Indian Journal of Medical Research, 88*, 522–542.

Broor, S., Pandey, R. M., Ghosh, M., Maitreyi, R. S., Lodha, R., Singhal, T., & Kabra, S. K. (2001). Risk factors for severe acute lower respiratory infection in under-five children. *Indian Pediatrics, 38*, 1361–1369.

Bruce, N., Perez-Padilla, R., & Albalak, R. (2000). Indoor air pollution in developing countries: A major environmental and public health challenge. *Bulletin of the World Health Organization, 78,* 1080–1092.

Ezzati, M., & Kammen, D. (2001). Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: An exposure–response study. *Lancet, 358,* 619–624. Erratum in *Lancet, 358,* 1104.

International Institute for Population Sciences (IIPS) and ORC Macro. (2000). *National Family Health Survey (NFHS-2), 1998–99: India.* Mumbai: International Institute for Population Sciences.

Janet, S. M .L., Jennifer, K. P., Xuan, W., & Berry, G. (1999). Meta analysis on the association between environmental tobacco smoke (ETS) exposure and the prevalence of lower respiratory tract infection in early childhood. *Pediatric Pulmonology, 27,* 5–13.

Johnson, J. D., Houchens, D. P., Kluwe, W. M., Craig, D. K., & Fisher, G. L. (1990). Effects of mainstream and environmental tobacco smoke on the immune system in animals and humans: A review. *Critical Reviews in Toxicology, 20,* 369–395.

Kong, L. Y., Luster, M. I., Dixon, D., O'Grady, J., & Rosenthal, G. J. (1994). Inhibition of lung immunity after intratracheal instillation of benzo(a)pyrene. *American Journal of Respiratory and Critical Care Medicine, 150,* 1123–1129.

Lanata, C. F., Rudan, I., Boshci-Pinto, C., Tomaskovic, L., Cherian, T., Weber, M., & Campbell, H. (2004). Methodological and quality issues in epidemiological studies of acute lower respiratory infections in children in developing countries. *International Journal of Epidemiology, 33,* 1–11.

Mahalanabis, D., Gupta, S., Paul, D., Gupta, A., Lahiri, M., & Khaled, M. A. (2002). Risk factors for pneumonia in infants and young children and the role of solid fuel for cooking: A case–control study. *Epidemiology and Infections, 129,* 65–71.

Mehta, S. (2002). *Characterizing Exposures to Indoor Air Pollution From Household Solid Fuel Use in India, PhD Dissertation.* Berkeley: University of California.

Ministry of Health and Family Welfare (MOHFW). (2003). *Annual Report 2002–03.* New Delhi: Government of India Press.

Mishra, V. (2003) Indoor air pollution from biomass combustion and acute respiratory illness in preschool age children in Zimbabwe. *International Journal of Epidemiology, 32,* 847–853.

Mishra, V., Retherford, R. D., & Smith, K. R. (2002). Indoor air pollution: The quiet killer. *Asia-Pacific Issues, 8,* 1–8.

Mukae, H., Vincent, R., Quinlan, K., English, D., Hards, J., Hogg, J. C., & van Eeden, S. F. (2001). The effect of repeated exposure to particulate air pollution (PM10) on the bone marrow. *American Journal of Respiratory and Critical Care Medicine, 163,* 201–209.

Murray, C. J. L., & Lopez, A. D. (1996). *Global Health Statistics.* Cambridge, MA: Harvard University School of Public Health for the World Health Organization, 906.

Samet, J. M., & Spengler, J. D., (Eds.). (1991). *Indoor Air Pollution: A Health Perspective.* Baltimore, MD: Johns Hopkins University Press.

Smith, K. R. (2000). National burden of disease in India from indoor air pollution. *Proceedings of the National Academy of Sciences, 97,* 13286–13293.

Smith, K. R. (2002). Indoor air pollution in developing countries: Recommendations for research. *Indoor Air, 12,* 1–7.

Smith, K .R. (2003). Indoor air pollution and acute respiratory infections. *Indian Pediatrics, 40,* 815–819.

Smith, K. R., & Liu, Y. (1994). Indoor air pollution in developing countries. In: J. M. Samet (Ed.), *Epidemiology of Lung Cancer, Lung Biology in Health and Disease Series,* pp. 151–184.

Smith, K. R., Mehta, S., & Maeusezahl-Feuz, M. (2004). Indoor smoke from household solid fuels. In: M. Ezzati, A. D. Rodgers, A. D. Lopez, & C. J. L. Murray (Eds.). *Comparative Quantification of Health Risks: Global and Regional Burden of Disease due to Selected Major Risk Factors.* Geneva: World Health Organization, *2,* 1437–1495.

Smith, K. R., Samet, J. M., Romieu, I., & Bruce, N. (2000a). Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax, 55,* 518–532.

Smith, K. R., Zhang, J., Uma, R., Kishore, V. V. N., Joshi, V., & Khalil, M. A. K. (2000b). Greenhouse implications of household fuels: An analysis for India. *Annual Review of Energy and the Environment, 25*, 741–763.

Stata Corporation. (2003). *Stata/SE 8.2 for Windows, Release 8.02*. College Station, TX: Stata Corporation.

Strachan, D. P., & Cook, D. G. (1997). Health effects of passive smoking. 1. Parental smoking and lower respiratory illness in infancy and early childhood. *Thorax, 52*, 905–914.

Thomas, P., & Zelikoff, J. (1999). Air pollutants: modulators of pulmonary host resistance against infection. In: S. T. Holgate, J. M. Samet, H. S. Koren, & R. L. Maynard (Eds.). *Air Pollution and Health,* pp. 357–380. San Diego, CA: Academic Press.

van Eeden, S. F., Tan, W. C., Suwa, T., Mukae, H., Terashima, T., Fujii, T., Qui, D., Vincent, R., & Hogg, J. C. (2001). Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM_{10}). *American Journal of Respiratory and Critical Care Medicine, 164*, 826–830.

World Health Organization (WHO). (2003). *GBD Database, 2002rev.* http://www3.who.int/whosis/menu.cfm?path=whosis,burden,burden_estimates,burden_estimates_2002Rev,burden_estimates_2002Rev_Subregion&language=english.

World Health Organization (WHO). (1997). *Health and Environment for Sustainable Development: Five Years After the Earth Summit*. Geneva: World Health Organization.