

Original Contribution

Indoor Charcoal Smoke and Acute Respiratory Infections in Young Children in the Dominican Republic

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The authors investigated the effect of charcoal smoke exposure on risks of acute upper and lower respiratory infection (AURI and ALRI) among children under age 18 months in Santo Domingo, Dominican Republic (1991–1992). Children living in households using charcoal for cooking (exposed, $n = 201$) were age-matched to children living in households using propane gas (nonexposed, $n = 214$) and were followed for 1 year or until 2 years of age. Fuel use and new episodes of AURI and ALRI were ascertained biweekly through interviews and medical examinations. Household indoor-air concentration of respirable particulate matter (RPM) was measured in a sample of follow-up visits. Incidences of AURI and ALRI were 4.4 and 1.4 episodes/child-year, respectively. After adjustment for other risk factors, exposed children had no significant increase in risk of AURI but were 1.56 times (95% confidence interval: 1.23, 1.97) more likely to develop ALRI. RPM concentrations were higher in charcoal-using households ($27.9 \mu\text{g}/\text{m}^3$ vs. $17.6 \mu\text{g}/\text{m}^3$), and ALRI risk increased with RPM exposure ($10\text{-}\mu\text{g}/\text{m}^3$ increment: odds ratio = 1.17, 95% confidence interval: 1.02, 1.34). Exposure to charcoal smoke increases the risk of ALRI in young children, an effect that is probably mediated by RPM. Reducing charcoal smoke exposure may lower the burden of ALRI among children in this population.

air pollution, indoor; biomass; charcoal; Dominican Republic; particulate matter; respiratory tract infections; smoke; wood

Abbreviations: ALRI, acute lower respiratory infection; AURI, acute upper respiratory infection; CI, confidence interval; PM, particulate matter; RPM, respirable particulate matter.

Acute respiratory infections account for over 6% of the global disease burden and 7% of all deaths (1). A number of studies have suggested that exposure to domestic smoke from biomass fuels (e.g., charcoal, wood, dung, and crop residues) increases the risk of respiratory problems such as persistent cough in adults (2–5) and acute lower respiratory infections in young children (6–16). Cooking with biomass fuels inside poorly ventilated houses can result in indoor environments where daily average and peak air concentrations of pollutants such as particulate matter, nitrogen oxides, carbon monoxide, formaldehyde, benzene, 1,3-butadiene, and polycyclic aromatic hydrocarbons (17) greatly exceed levels regarded as safe (18, 19).

Charcoal combustion results in lower pollutant emission levels than combustion of other types of biomass fuels and

seems to be associated with fewer respiratory symptoms (3, 18, 20). Consequently, a shift to charcoal from other biomass fuels has been suggested as a possible approach for reducing the impact of indoor biomass smoke on respiratory illnesses (7). However, the potential reduction in children's risk of acute respiratory infection by this approach is uncertain because of the limited information on the respiratory effects of charcoal smoke. The majority of published studies have evaluated the health effects of wood smoke exposure instead of charcoal smoke (21). Moreover, proxy measures of exposure, including cooking fuel or stove type (10, 11, 15, 22–24) and regular carriage of the child on the mother's back while cooking (9, 14, 16), have often been used rather than actual measures of pollution concentrations. Although levels of respirable particulate matter

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(RPM) were measured in 2 studies (7, 13), wood was the primary fuel in both, and RPM concentrations were several times higher than those typically observed with charcoal.

We evaluated the relation between exposure to indoor charcoal smoke and risks of acute upper respiratory infection (AURI) and acute lower respiratory infection (ALRI) in a cohort of children from the Dominican Republic. We longitudinally monitored the type of fuel used, levels of indoor air pollution, and the incidence of acute respiratory infection in children under 2 years of age.

MATERIALS AND METHODS

Research location

The study took place in Domingo Savio, a low-income residential neighborhood in Santo Domingo, Dominican Republic. Santo Domingo has a tropical climate, with an average yearly rainfall of 138 cm and little variation in average monthly temperature (24°C–27°C) (<http://www.weather.com/>). Domingo Savio is characterized by a high population density, with 2- and 3-room houses scattered along dirt roads, though motor vehicle traffic is minimal. The area often floods during the rainy season, and many households lack consistent access to safe water and adequate sanitation.

Sampling frame construction

Field-workers visited each of the 8,246 households in Domingo Savio to ascertain cooking fuel type, active smoking status, and the numbers and ages of all children. We excluded households without children under 18 months of age or with a current smoker ($n = 7,355$) from the sampling frame. An additional 24 households that used neither charcoal nor propane gas for cooking and 41 households that did all of their cooking outdoors were also excluded. Out of 732 children under 18 months of age in 826 households, 275 lived in homes where charcoal was used for cooking. We selected the youngest child from each household, resulting in a cohort of 201 exposed children. From the remaining 457 children, who lived in homes where propane gas was used for cooking, we selected a nonexposed cohort of 214 children, each from a different household and individually matched within 1 month of age to an exposed child. We estimated that a sample size of 230 exposed children and an equal number of nonexposed children would provide 80% power to detect as statistically significant ($P < 0.05$) a relative risk greater than or equal to 2.0.

Data collection

Field research began in 1991 and continued until late 1992. The study was approved by the institutional review board of the Johns Hopkins School of Hygiene and Public Health (Baltimore, Maryland), and written informed consent was obtained from the parents or legal guardians of study children, resulting in a 100% parental consent rate. We collected baseline data on sociodemographic variables, cooking fuel use, and amounts of time the child spent in the kitchen, in the bedroom, and outside. We verified information on birth date and birth weight using birth identification

tags and hospital records. We then conducted a follow-up evaluation once every 2 weeks to ascertain changes in cooking fuel, passive smoking, or breastfeeding status and any incident episodes of acute respiratory infection occurring during the previous 2 weeks. Body weight was measured every 2 months. Each child was followed for 1 year or until he or she reached 2 years of age.

Exposure

During the follow-up evaluations, we collected data on cooking fuel type and amounts of time the child had spent in the kitchen, in the bedroom, and outdoors since the last visit. We conducted bimonthly measurements of indoor concentrations of respirable suspended particles—that is, particles meeting a size selection criterion specified by the American Conference of Governmental Industrial Hygienists (25). Respirable dust samples have a 50% cut size of 4 μm (particulate matter with an aerodynamic diameter less than or equal to 4 μm (PM_4)). Half of the RPM measurements were conducted in the kitchen and half in the child's bedroom. We sampled for approximately 10 hours per sampling day, using 37-mm polyvinyl chloride filters and a 10-mm nylon cyclone preselector connected to a battery-operated Gilian sampling pump (Sensidyne, Inc., Clearwater, Florida). The data were analyzed according to the US National Institute of Occupational Safety and Health's Method 0600 (26). Pumps were calibrated before each sampling day using a Minibuck soap-bubble air-flow calibrator (A. P. Buck, Inc., Orlando, Florida). Gravimetric analysis was performed using a Cahn Electrobalance (Cahn Instruments, Paramount, California) with an accuracy of 0.1 μm at the Johns Hopkins School of Hygiene and Public Health for determination of RPM mass, which was then used to calculate a time-weighted average concentration of RPM (expressed as $\mu\text{g}/\text{m}^3$) for each room sampled. We submitted 12% of the samples as field blanks.

We estimated the average biweekly personal exposure (E_{ij}) of each child (i) at each follow-up visit (j) on the basis of mean RPM concentration in the kitchen and the bedroom (RPM_{ijk}) and the average amount of time the child spent in these microenvironments and outside the home (i.e., $E_{ij} = \sum_k (\text{RPM}_{ijk} \times \text{time in environment } k)$). We used quintiles of E_{ij} for data analysis. We also defined 3 exposure classifications using survey data: 1) baseline exposure, 2) follow-up exposure, and 3) overall exposure. Baseline exposure was defined as the type of cooking fuel reported at baseline (charcoal vs. gas). Follow-up exposure was defined as the type of cooking fuel used during the 2 weeks preceding a follow-up visit (charcoal vs. gas). Overall exposure was defined as the general pattern of cooking fuel used throughout the entire follow-up period (i.e., households that used charcoal at least 70% of the time were defined as charcoal users; those that used gas at least 70% of the time were defined as gas users; and those that alternated between charcoal and gas at least 30% of the time were defined as charcoal/gas users).

Health outcomes

We employed 8 physicians as field-workers and trained them to identify signs and symptoms of acute respiratory

infection using World Health Organization protocols (27). We examined children whose mothers reported them sick or who were found to have a cough, fever, or diarrhea during a follow-up visit and referred seriously ill children to the Robert Reid Cabral Children's Hospital for pediatric care. We determined respiratory rate (breaths per minute) by averaging 3 1-minute measurements of respiratory frequency. An ALRI episode was defined as an illness characterized by wheezing, tachypnea, retraction, or stridor ascertained by physical examination. An AURI episode was defined as the presence of at least 2 of the following signs or symptoms: runny nose, cough, fever, or ear secretion, as reported by the mother or identified by physical examination in the absence of ALRI signs or symptoms. These definitions are similar to those used in other studies (7, 28, 29). A pediatrician reviewed each episode and made the diagnosis and classification of all episodes of respiratory illness.

Statistical analysis

We estimated the incidences of AURI and ALRI by allocating person-time at risk for each child up to the final observation day (excluding time during an AURI or ALRI episode). Consecutive episodes of acute respiratory infection in the same child were considered independent if they were separated by 3 or more symptom-free days. We used multiple logistic regression to examine the relation between exposure and risks of AURI and ALRI in a given 2-week period of follow-up, while accounting for other risk factors. Age was included in all models to account for the matching design. Sex, birth weight, numbers and ages of siblings, mother's age, crowding (number of people per sleeping room), breastfeeding (yes/no), nutritional status (weight-for-age score), socioeconomic status (mother's education and household income), previous episodes of acute respiratory infection, and passive smoking (presence of a smoker in the household) were evaluated as potential confounders. Only variables that were significantly associated with risks of ALRI and AURI ($P < 0.05$) or that confounded the exposure-disease association were retained in the final models.

To account for the effect of repeated observations and multiple ALRI episodes occurring in the same child, we conducted logistic regression analyses of all ALRI episodes using generalized estimating equations (30) and logistic regression restricted to the follow-up time contributed by each child up to the first ALRI episode, or up to the end of follow-up if no episode occurred. To estimate the absolute impact of charcoal exposure on ALRI risk, we calculated the fraction of cases that would not have occurred in the absence of exposure (attributable fraction) (31). We conducted a similar analysis for AURI episodes. All analyses were conducted in SAS (32) and Stata (33).

RESULTS

We followed 201 children from households using charcoal at baseline and 214 children from households using propane gas at baseline. Sixty children (14.5%) were lost to follow-up, but the losses did not differ appreciably by exposure status (15.4% in the charcoal group and 13.6%

in the gas group) or by child's age, sex, nutritional status, or crowding (data not shown). We completed a total of 7,218 interviews, corresponding to 276.8 child-years of follow-up (131.7 in the charcoal group and 145.1 in the gas group, by baseline exposure classification), and an average of 18 follow-up visits per child.

Exposed children lived in houses with fewer rooms, and their families had a lower monthly income (Table 1). They were also more likely to have low birth weight and malnutrition at baseline, to be breastfed, to have an illiterate mother, and to have a mother under 20 years of age. In contrast, the level of crowding and the child's history of vaccination and hospitalization due to respiratory illness were similar in exposed and nonexposed children.

Charcoal-burning cookstoves were typically unvented, with a side opening for adding fuel. Households using charcoal for cooking had higher indoor RPM concentrations than those using gas (geometric mean concentrations of $27.9 \mu\text{g}/\text{m}^3$ and $17.6 \mu\text{g}/\text{m}^3$, respectively; $P < 0.01$). Further, while households using gas showed little variation in RPM by area sampled, those using charcoal had higher concentrations of RPM in the kitchen than in other household areas (geometric mean concentrations of $38.7 \mu\text{g}/\text{m}^3$ and $27.3 \mu\text{g}/\text{m}^3$, respectively; $P < 0.01$).

The incidence rates of AURI and ALRI were 4.4 and 1.4 episodes/child-year, respectively (Table 2). The incidence of AURI was lowest in the first 6 months of life (2.6 episodes/child-year) and peaked at 12–17 months of age (4.8 episodes/child-year), whereas ALRI incidence was highest among infants under age 6 months (4.0 episodes/child-year) and progressively declined with increasing age. The incidence of AURI was not significantly associated with either cooking fuel type or personal RPM exposure level. However, the crude incidence of ALRI was significantly higher in children living in charcoal-using households than in children living in gas-using households with all 3 exposure classifications (Table 3).

When classified by their baseline or follow-up exposure, the incidences of ALRI in children from charcoal-using households were 1.58 (95% confidence interval (CI): 1.29, 1.96) and 1.49 (95% CI: 1.21, 1.84) times higher, respectively, than those in children from households using gas. Similarly, for the overall exposure classification, the crude incidence of ALRI increased with higher levels of charcoal exposure. Specifically, the unadjusted rate ratio was 1.27 (95% CI: 0.97, 1.66) for the "charcoal/gas" group and 1.37 (95% CI: 1.09, 1.74) for the "charcoal only" group, as compared with the propane gas group (trend test: $P = 0.02$).

Results from the analysis based on estimated personal RPM exposure also suggested a dose-response relation (Table 4). Compared with children in the lowest quintile of pollution exposure, the risk of ALRI was increased 15% among those in the second quintile, 43% among those in the third quintile, 53% among those in the fourth quintile, and almost 80% among those in the highest quintile (trend test: $P < 0.01$).

Logistic regression analyses of ALRI and the various exposure classifications yielded models with similar sets of explanatory variables and parameter estimates (Table 5). After adjustment for child's age, nutritional status, and

Table 1. Characteristics of Children Under 2 Years of Age by Baseline Exposure to Cooking Fuels, Santo Domingo, Dominican Republic, 1991–1992

Characteristic	Propane Gas (n = 214)		Charcoal (n = 201)		Total (n = 415)		P Value ^a
	No.	%	No.	%	No.	%	
Household							
≥3 rooms	120	56.1	53	26.4	173	41.7	<0.001
≥4 people sleeping/room	120	56.1	115	57.7	226	56.9	0.83
Monthly income >US\$160	79	36.9	35	17.4	114	27.5	<0.001
Child							
Male sex	107	50.0	110	54.7	217	52.3	0.34
Birth weight <2,500 g	17	8.1	24	12.1	41	10.0	0.18
History of breastfeeding ^b	73	34.9	97	48.7	170	41.0	<0.01
Malnourished ^c	22	10.0	32	16.2	54	13.0	0.09
History of wheezing	44	20.6	48	23.9	92	22.2	0.42
Poliomyelitis vaccine	197	92.1	177	88.1	374	90.8	0.22
Diphtheria-pertussis-tetanus vaccine	137	87.8	110	85.9	247	87.0	0.64
Mother							
Illiterate	16	7.6	26	13.3	48	11.8	0.06
Hospitalized for chest illness	11	5.1	9	4.5	20	4.8	0.75
Age <20 years	26	12.3	48	24.0	74	18.0	<0.01

^a Derived using a 2-sided chi-square test.^b A child was considered not being breastfed when breastfeeding was suspended for at least 3 consecutive follow-up visits.^c Children with weight-for-age scores less than 2 standard deviations from the US National Center for Health Statistics mean (50) were considered malnourished.

crowding, children living in charcoal-using households had a significantly higher risk of ALRI than children living in gas-using households. The multivariate adjusted odds ratios for baseline and follow-up exposure to charcoal smoke were 1.56 (95% CI: 1.23, 1.97) and 1.34 (95% CI: 1.07, 1.69), respectively. For overall exposure, children in the “charcoal/

gas” group were 1.16 (95% CI: 0.85, 1.58) times more likely to develop ALRI, and those in the “charcoal-only” group were 1.38 (95% CI: 1.06, 1.81) times more likely to develop ALRI (trend test: $P = 0.03$). Moreover, exposure to respirable particles resulted in a statistically significant 17% increased risk of ALRI for each 10- $\mu\text{g}/\text{m}^3$ increase in RPM

Table 2. Age-Specific Incidence Rates of Acute Upper and Lower Respiratory Infection Among Children Under 2 Years of Age, Santo Domingo, Dominican Republic, 1991–1992

Infection and Child's Age, months	No. of Cases	Child-Years of Follow-Up	No. of Episodes Per Child-Year	95% Confidence Interval
Acute upper respiratory infection				
0–5	30	11.7	2.6	1.7, 3.7
6–11	220	54.8	4.0	3.5, 4.6
12–17	467	97.6	4.8	4.4, 5.2
18–23	376	87.2	4.3	3.9, 4.8
Total	1,093	251.4	4.4	4.1, 4.6
Acute lower respiratory infection				
0–5	46	11.4	4.0	3.0, 5.4
6–11	123	56.8	2.2	1.8, 2.6
12–17	128	106.3	1.2	1.0, 1.4
18–23	70	94.9	0.7	0.6, 0.9
Total	367	269.4	1.4	1.2, 1.5

Table 3. Unadjusted Rate Ratios for Acute Upper and Lower Respiratory Infection Among Children Under 2 Years of Age in Relation to Exposure to Charcoal Smoke, Santo Domingo, Dominican Republic, 1991–1992^a

Exposure	Acute Upper Respiratory Infection				Acute Lower Respiratory Infection			
	No. of Cases	Rate ^b	RR	95% CI	No. of Cases	Rate ^b	RR	95% CI
Baseline								
Gas	545	4.09	1.00		152	1.07	1.00	
Charcoal	548	4.61	1.12	1.00, 1.27	215	1.69	1.58	1.29, 1.96
Follow-up								
Gas	683	4.35	1.00		198	1.17	1.00	
Charcoal	397	4.34	1.00	0.88, 1.13	166	1.71	1.49	1.21, 1.84
Overall								
Gas	593	4.34	1.00		174	1.35	1.00	
Gas/charcoal	192	4.05	0.93	0.79, 1.09	76	1.51	1.27	0.97, 1.66
Charcoal	308	4.57	1.05	0.92, 1.21	117	2.31	1.37	1.09, 1.74

Abbreviations: CI, confidence interval; RR, rate ratio.

^a Children exposed to propane gas were used as the reference group.^b Number of episodes/child-year.

(odds ratio = 1.17, 95% CI: 1.02, 1.34), with an apparent threshold level of 19 $\mu\text{g}/\text{m}^3$ (data not shown).

Results from the logistic regression analyses accounting for repeated ALRI episodes in the same child yielded estimates of exposure effect similar to those of the logistic models limited to the initial ALRI episode. For example, for baseline exposure, the multivariate adjusted odds ratios were 1.56 (95% CI: 1.23, 1.97) for the model accounting for

repeated episodes and 1.35 (95% CI: 1.02, 1.79) for the model restricted to the initial ALRI episode. Corresponding estimates for follow-up exposure and overall exposure, as well as for RPM levels, were practically identical for all models (data not shown).

According to the 2002 Dominican Republic Demographic and Health Survey, 10.4% of all households in the Dominican Republic cook with charcoal or wood (34).

Table 4. Incidence Rates and Unadjusted Rate Ratios for Acute Upper and Lower Respiratory Infection Among Children Under 2 Years of Age According to Level of Exposure to Respirable Particulate Matter, Santo Domingo, Dominican Republic, 1991–1992

Infection and RSP Exposure Level ($\mu\text{g}/\text{m}^3$)	No. of Episodes ^a	No. of Episodes Per Child-Year	Incidence Rate Ratio	95% Confidence Interval
Acute upper respiratory infection^b				
≤5.9	205	4.24	1.00	
>5.9–12.4	225	4.70	1.11	0.92, 1.35
>12.4–18.7	194	4.00	0.94	0.78, 1.16
>18.7–30.3	217	4.49	1.06	0.88, 1.29
>30.3	206	4.28	1.01	0.84, 1.23
Acute lower respiratory infection^c				
≤5.9	51	0.97	1.00	
>5.9–12.4	58	1.12	1.15	0.80, 1.73
>12.4–18.7	72	1.39	1.43	1.02, 2.12
>18.7–30.3	77	1.49	1.53	1.10, 2.27
>30.3	88	1.73	1.77	1.29, 2.60

Abbreviation: RSP, respirable particulate matter.

^a The total number of episodes in this table is slightly lower than that in Table 2 because RSP data were not available for all households at all times and a few episodes of acute respiratory infection could not be grouped by RSP exposure level.

^b Trend test: $P = 0.57$.^c Trend test: $P < 0.01$.

Table 5. Multivariate Adjusted Odds Ratio for Acute Lower Respiratory Infection Among Children Under 2 Years of Age According to Risk Factors Significantly Associated with Disease Incidence and Exposure to Charcoal Smoke, Santo Domingo, Dominican Republic, 1991–1992

Risk Factor	No. of Episodes	Odds Ratio ^a	95% Confidence Interval
Child's age, months ^b			
0–5	46	1.00	
6–11	123	0.51	0.35, 0.73
12–17	128	0.27	0.19, 0.39
18–23	70	0.17	0.11, 0.25
No. of children aged >5 years in household ^b			
1	158	1.00	
2	148	1.34	1.06, 1.70
≥3	61	1.34	0.98, 1.83
Nutritional status ^{b,c}			
Not malnourished	300	1.00	
Malnourished	67	1.39	1.05, 1.85
Exposure to charcoal			
Baseline			
Propane	152	1.00	
Charcoal	215	1.56	1.23, 1.97
Follow-up			
Propane	198	1.00	
Charcoal	166	1.34	1.07, 1.69
Overall exposure			
Propane gas	174	1.00	
Charcoal/gas	76	1.16	0.85, 1.58
Charcoal	117	1.38	1.06, 1.81
Respirable particulate matter (10- $\mu\text{g}/\text{m}^3$ increase)	346	1.17	1.02, 1.34

^a Adjusted for all variables included in the table.

^b Corresponds to model including baseline exposure.

^c Children with weight-for-age scores less than 2 standard deviations from the US National Center for Health Statistics mean (50) were considered malnourished.

Assuming that the observed association between charcoal smoke exposure and ALRI is causal (21) and that wood smoke exposure and charcoal smoke exposure have similar respiratory effects, we estimate that 18.2% (95% CI: 15.4, 20.9) of ALRI cases in children under 2 years of age in this population could be prevented by eliminating this exposure.

DISCUSSION

In this cohort study, we found that children living in households where charcoal was used for cooking had a significantly higher risk of ALRI than children living in house-

holds where propane gas was used. Further, we found that charcoal-using households had higher concentrations of RPM than those using gas and that ALRI risk increased progressively with increasing RPM exposure level. The incidence of ALRI in our study (1.4 episodes/child-year) was within the range of the 0.9–3.8 episodes/child-year observed in other developing countries (35).

Our findings are consistent with those from a recent meta-analysis of studies on solid fuel smoke and ALRI in children (odds ratio = 1.78, 95% CI: 1.45, 2.18) (36), as well as previous cohort studies that used indirect exposure measures to evaluate the effect of wood smoke (37, 38). In those studies, children who were exposed to wood smoke were 2.28 times more likely to suffer severe episodes of acute respiratory infection (95% CI: 1.70, 3.05; our calculations). However, our estimates of the effect of charcoal smoke exposure on ALRI incidence are lower than those from previous wood-smoke studies. One possible explanation for this contrast is differences in accounting for potential confounders. For example, incomplete control of confounding by poverty and other factors associated with use of biomass fuels (21) could result in overestimation of the effect of wood smoke on ALRI. In our study, comparison groups came from the same low-income neighborhood, and we accounted for apparent differences in socioeconomic status (mother's education, average household income, and quality of household building materials). A likely and potentially more important explanation is that charcoal combustion emits considerably less smoke and should therefore result in less exposure and lower ALRI risk than combustion of other biomass fuels (7, 39).

The exact mechanism by which biomass smoke increases risk and severity of ALRI in young children remains unclear. The effect may be mediated in part by inhalation and deposition of particulate matter in the lower, nonciliated region of the respiratory tract. Particulate matter is a complex mixture of organic compounds similar to those found in tobacco smoke (18), which has been shown to cause depressed immune system responses (40). In particular, particulate matter may increase the attachment of bacteria to the respiratory mucosa and reduce resident macrophage bactericidal activity (41) and chemotaxis movement (40, 42). Further, charcoal smoke inhalation may lead to the deposition of larger particles in the conducting airways, resulting in inflammation and impaired mucociliary activity (43, 44) and thus a reduction of lung clearance capacity that could occur hours or days after an acute exposure. Notably, the results of our study suggest that both short- and long-term effects of particulate matter on ALRI are likely, given that children in households using charcoal were more likely to develop ALRI for "baseline" and "overall" exposure, which indicates long-term effects, as well as for "follow-up" exposure, which suggests short-term effects.

Potential sources of bias should be taken into consideration while interpreting our findings. Approximately 15% of the children in our cohort were lost to follow-up. However, it is unlikely that selection bias resulted from these losses, since they were not associated with exposure status or other risk factors for acute respiratory infection. Moreover, we used multiple regression analysis to account for the

potentially confounding effects of known risk factors for acute respiratory infection, such as sex, nutritional status, crowding, and mother's age and education.

Nondifferential errors in measurement of RPM exposure probably occurred as a consequence of errors in estimating the average amount of time each child spent in each micro-environment. Similarly, children living in households where gas was used may have been exposed to charcoal smoke on occasion and vice versa. It is unlikely, however, that exposure misclassification resulted in a spurious association, since its effect would have been to lessen between-group differences in actual exposure and therefore attenuate an association between charcoal smoke and ALRI. Further, the consistency of the results for the various exposure classifications suggests that the observed association was unlikely to have been due to measurement error.

Differential ascertainment of acute respiratory infection could have occurred if knowledge of the child's exposure status influenced parents' recall of respiratory signs or the quality of interviews and health examinations conducted by the field-workers. We attempted to control for this by 1) standardizing the collection of information on respiratory signs and symptoms, 2) collecting information on respiratory signs and symptoms biweekly to minimize recall errors, and 3) eliciting information on respiratory signs and symptoms rather than allowing the field-workers to diagnose cases of acute respiratory infection.

Our study showed strong and significant respiratory effects from charcoal smoke at much lower pollution concentrations than in many other studies conducted in developing countries. The RPM levels associated with charcoal use in our study were considerably lower than the levels of $PM_{7.1}$ (particulate matter with an aerodynamic diameter less than or equal to $7.1\text{ }\mu\text{m}$) reported for households burning charcoal (mean $PM_{7.1}$ level = $543\text{ }\mu\text{g}/\text{m}^3$) in Maputo, Mozambique (3) and the levels of PM_{10} (particulate matter with an aerodynamic diameter less than or equal to $10\text{ }\mu\text{m}$) reported for charcoal-burning stoves in rural Kenya (45). This may be attributable to differences in stove type, cooking practices, ventilation, presence of tobacco smoke, and ambient pollution levels and to differences in sampling methods. The average RPM level found in charcoal-using households in our study ($27.9\text{ }\mu\text{g}/\text{m}^3$) was similar to the concentration of PM_{10} reported in households with 1 smoker in the United States: $24.4\text{ }\mu\text{g}/\text{m}^3$ (46). Incidentally, the effect of charcoal smoke exposure on ALRI observed in our study (odds ratio = 1.56) coincides with the effect of passive smoking on lower respiratory illness in children under age 2 years derived from a meta-analysis of 24 studies conducted in both developed and developing countries (odds ratio = 1.57, 95% CI: 1.40, 1.77) (47).

Our findings support the results of previous studies suggesting that particulate matter is the pollutant(s) most likely to be associated with the observed respiratory effects. More recent studies of air pollution have focused on fine particulate matter (particulate matter with an aerodynamic diameter less than or equal to $2.5\text{ }\mu\text{m}$ ($PM_{2.5}$)). Combustion of solid fuels such as charcoal primarily produces fine particulate matter. Respirable dust sampling, used in this study, is designed to collect data on particulate matter that deposits in

the respiratory epithelium and is more commonly used in occupational studies. There are likely to be minor differences in particle mass concentrations between $PM_{2.5}$ and PM_4 air samples in these environments. If the observed association between charcoal smoke and ALRI is causal, then controlling exposure to indoor biomass smoke will help reduce the risk of ALRI in young children. In the long run, minimizing exposure might be best achieved by transitioning to cleaner-burning fuels. Carefully conducted observational studies show that the largest exposure reduction (~90%) could be achieved by transitioning from wood to charcoal (39). However, charcoal use may be more environmentally damaging, since traditional production techniques convert just 20%–25% of wood to charcoal. Further, the lower exposure levels achieved with charcoal still increase ALRI risk in young children by over 50% in comparison with propane gas.

In light of the economic constraints in many developing countries, more practical approaches than changes in fuel type can be implemented in the short term. These include educating child caregivers on the importance of keeping small children away from the cooking hearth or stove, encouraging ventilation, and increasing the availability and appropriate use of high-efficiency, low-emission cookstoves (18, 48). Unfortunately, wood stoves may provide limited benefit in preventing ALRI in young children, since they reduce pollution exposure to levels that are still at least an order of magnitude higher than those observed in our study (45, 49).

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REFERENCES

1. World Health Organization. *The World Health Report 2002. Reducing Risks, Promoting Healthy Life*. Geneva, Switzerland: World Health Organization; 2002.
2. Norbooo T, Yahya M, Bruce N, et al. Domestic pollution and respiratory illness in a Himalayan village. *Int J Epidemiol*. 1991;20(3):749–757.
3. Ellegård A. Cooking fuel smoke and respiratory symptoms among women in low-income areas in Maputo. *Environ Health Perspect*. 1996;104(9):980–985.
4. Regalado J, Pérez-Padilla R, Sansores R, et al. The effect of biomass burning on respiratory symptoms and lung function in rural Mexican women. *Am J Respir Crit Care Med*. 2006; 174(8):901–905.
5. Bruce N, Neufeld L, Boy E, et al. Indoor biofuel air pollution and respiratory health: the role of confounding factors among women in highland Guatemala. *Int J Epidemiol*. 1998;27(3): 454–458.
6. Pandey MR, Boleij JS, Smith KR, et al. Indoor air pollution in developing countries and acute respiratory infection in children. *Lancet*. 1989;1(8635):427–429.
7. Ezzati M, Kamman D. Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: an exposure-response study. *Lancet*. 2001;358(9282):619–624.
8. Kossove D. Smoke-filled rooms and lower respiratory disease in infants. *S Afr Med J*. 1982;61(17):622–624.
9. Armstrong JR, Campbell H. Indoor air pollution exposure and lower respiratory infections in young Gambian children. *Int J Epidemiol*. 1991;20(2):424–429.
10. Morris K, Morgenlander M, Coulehan JL, et al. Wood-burning stoves and lower respiratory tract infection in American Indian children. *Am J Dis Child*. 1990;144(1):105–108.
11. Mishra V. Indoor air pollution from biomass combustion and acute respiratory illness in preschool age children in Zimbabwe. *Int J Epidemiol*. 2003;32(5):847–853.
12. Robin LF, Less PS, Winget M. Wood-burning stoves and lower respiratory illnesses in Navajo children. *Pediatr Infect Dis J*. 1996;15(10):859–865.
13. Collings DA, Sithole SD, Martin KS. Indoor woodsmoke pollution causing lower respiratory disease in children. *Trop Doct*. 1990;20(4):151–155.
14. Campbell H, Byass P, Greenwood BM. Acute lower respiratory infections in Gambian children: maternal perception of illness. *Ann Trop Paediatr*. 1990;10(1):45–51.
15. Cerqueiro MC, Murtagh P, Halac A, et al. Epidemiologic risk factors for children with acute lower respiratory tract infection in Buenos Aires, Argentina: a matched case-control study. *Rev Infect Dis*. 1990;12(suppl 8):S1021–S1028.
16. O'Dempsey T, McArdle T, Morris J, et al. A study of risk factors for pneumococcal disease among children in a rural area of West Africa. *Int J Epidemiol*. 1996;25(4):885–893.
17. de Koning HW, Smith KR, Last JM. Biomass fuel combustion and health. *Bull World Health Organ*. 1985;63(1):11–26.
18. Smith K. *Biofuels, Air Pollution, and Health: A Global Review*. New York, NY: Plenum Press; 1987.
19. Bruce N, Perez-Padilla R, Albalak R. Indoor air pollution in developing countries: a major environmental and public health challenge. *Bull World Health Organ*. 2000;78(9):1078–1092.
20. Ezzati M, Kammen D. Evaluating the health benefits of transitions in household energy technologies in Kenya. *Energy Policy*. 2002;30(10):815–826.
21. Smith KR, Samet JM, Romieu I, et al. Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax*. 2000;55(6):518–532.
22. Sharma S, Sethi GR, Rohtagi A, et al. Indoor air quality and acute lower respiratory infections in Indian urban slums. *Environ Health Perspect*. 1998;106(5):291–297.
23. Johnson AW, Aderele WI. The association of household pollutants and socio-economic risk factors with the short-term outcome of acute lower respiratory infections in hospitalized pre-school Nigerian children. *Ann Trop Paediatr*. 1992; 12(4):421–432.
24. Victora CG, Fuchs SC, Flores JA, et al. Risk factors for pneumonia among children in a Brazilian metropolitan area. *Pediatrics*. 1994;93(6 pt 1):977–985.
25. American Conference of Governmental Industrial Hygienists. *2008 Threshold Limit Values (TLVs) and Biological Exposure Indices (BEIs)*. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists; 2008.
26. National Institute for Occupational Safety and Health. NIOSH Method 0600. In: Eller PM, ed. *NIOSH Manual of Analytical Methods*. 3rd ed. Cincinnati, OH: National Institute for Occupational Safety and Health; 1984. (DHHS/NIOSH publication no. 84-100).
27. Programme for Control of Acute Respiratory Infections, World Health Organization. *Acute Respiratory Infections in Children: Case Management in Small Hospitals in Developing Countries: A Manual for Doctors and Other Senior Health Workers*. Geneva, Switzerland: World Health Organization; 1990.
28. Tupasi TE, de Leon LE, Lupisan S, et al. Patterns of acute respiratory tract infection in children: a longitudinal study in a depressed community in Metro Manila. *Rev Infect Dis*. 1990; 12(suppl 8):S940–S949.
29. Borrero I, Fajardo L, Bedoya A, et al. Acute respiratory tract infections among a birth cohort of children from Cali, Colombia, who were studied through 17 months of age. *Rev Infect Dis*. 1990;12(suppl 8):S950–S956.
30. Liang KY, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrika*. 1986;73(1):13–22.
31. Greenland S. Applications of stratified analysis methods. In: Rothman K, Greenland S, eds. *Modern Epidemiology*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1998:253–279.
32. SAS Institute Inc. *SAS System for Windows, Release 8.02*. Cary, NC: SAS Institute Inc; 2001.
33. Stata Corporation. *Stata Statistical Software. Release 10*. College Station, TX: Stata Corporation LP; 2007.
34. Molina-Achecar M, Ramírez N, Polanco J, et al. *República Dominicana. Encuesta Demográfica y de Salud 2002*. Calverton, MD: Macro International, Inc; 2003.
35. Selwyn B. The epidemiology of acute respiratory tract infection in young children: comparison of findings from several developing countries. Coordinated Data Group of BOSTID Researchers. *Rev Infect Dis*. 1990;12(suppl 8):S870–S888.
36. Dherani M, Pope D, Marenhas M, et al. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. *Bull World Health Organ*. 2008;86(5):390C–398C.
37. Pandey M, Neupane R, Gautam A, et al. Domestic smoke pollution and acute respiratory infections in a rural community of the hill region of Nepal. *Environ Int*. 1989;15:337–340.
38. Campbell H, Armstrong J, Byass P. Indoor air pollution in developing countries and acute respiratory infection in children [letter]. *Lancet*. 1989;1(8645):1012.
39. Ezzati M, Mbida BM, Kammen DM. Comparison of emissions and residential exposure from traditional and improved cookstoves in Kenya. *Environ Sci Technol*. 2000;34(4): 578–583.
40. Gardner D. Alteration in host bacteria interactions by environmental chemicals. In: Lee S, Mudd J, eds. *Assessing the*

Effects of Environmental Pollutants. Ann Arbor, MI: Ann Arbor Sciences Publisher; 1979:87–103.

41. Demarest GB, Hudson LD, Altman LC. Impaired alveolar macrophage chemotaxis in patients with acute smoke inhalation. *Am Rev Respir Dis.* 1979;119(2):279–286.
42. Fick RB Jr, Paul ES, Merrill WW, et al. Alterations in the antibacterial properties of rabbit pulmonary macrophages exposed to wood smoke. *Am Rev Respir Dis.* 1984;129(1):76–81.
43. Schlesinger R. The interaction of inhaled toxicants with respiratory tract clearance mechanisms. *Crit Rev Toxicol.* 1990;20(4):257–286.
44. Thorning DR, Howard ML, Hudson LD, et al. Pulmonary responses to smoke inhalation: morphological changes in rabbits exposed to pine wood smoke. *Hum Pathol.* 1982;13(4):355–364.
45. Ezzati M, Kammen DM. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs. *Environ Health Perspect.* 2002;110(11):1057–1068.
46. Spengler JD, Dockery DW, Turner WA, et al. Long-term measurements of respirable sulfates and particles inside and outside homes. *Atmos Environ.* 1981;15:23–30.
47. Strachan DP, Cook DG. Health effects of passive smoking. 1. Parental smoking and lower respiratory illness in infancy and early childhood. *Thorax.* 1997;52(10):905–914.
48. Kammen DM. Cookstoves for the developing world. *Sci Am.* 1995;273:72–75.
49. McCracken J, Smith K, Díaz A, et al. Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. *Environ Health Perspect.* 2007;115(7):996–1001.
50. National Center for Health Statistics. *NCHS Growth Curves for Children, Birth–18 Years, United States.* Hyattsville, MD: National Center for Health Statistics; 1978.