International Journal of Epidemiology 2003;**32**:847–853
DOI: 10.1093/ije/dyg240

RESPIRATORY

Indoor air pollution from biomass combustion and acute respiratory illness in preschool age children in Zimbabwe

Vinod Mishra

Accepted 14 May 2003

Background Reliance on biomass for cooking and heating exposes many women and young children in developing countries to high levels of air pollution indoors. This study investigated the association between household use of biomass fuels for cooking and acute respiratory infections (ARI) in preschool age children (<5 years) in Zimbabwe.

Methods Analysis is based on 3559 children age 0–59 months included in the 1999 Zimbabwe Demographic and Health Survey (ZDHS). Children who suffered from cough accompanied by short, rapid breathing during the 2 weeks preceding the survey were defined as having suffered from ARI. Logistic regression was used to estimate the odds of suffering from ARI among children from households using biomass fuels (wood, dung, or straw) relative to children from households using cleaner fuels (liquid petroleum gas [LPG]/natural gas, or electricity), after controlling for potentially confounding factors.

Results About two-thirds (66%) of children lived in households using biomass fuels and 16% suffered from ARI during the 2 weeks preceding the survey interview. After adjusting for child's age, sex, birth order, nutritional status, mother's age at childbirth, education, religion, household living standard, and region of residence, children in households using wood, dung, or straw for cooking were more than twice as likely to have suffered from ARI as children from households using LPG/natural gas or electricity (OR = 2.20; 95% CI: 1.16, 4.19).

Conclusions Household use of high pollution biomass fuels is associated with ARI in children in Zimbabwe. The relationship needs to be further investigated using more direct measures of smoke exposure and clinical measures of ARI.

Keywords Indoor air pollution, biomass, smoke, respiratory tract infections, child, Zimbabwe

Acute respiratory infections (ARI) are a leading cause of childhood illness and death worldwide, accounting for an estimated 6.5% of the entire global burden of disease.[1] In Zimbabwe, as in many other developing countries, ARI are the leading cause of childhood mortality.[2] This study examines the association between household use of high pollution biomass fuels (wood, dung, or straw) and ARI prevalence in children in Zimbabwe using data from a recent nationally representative household survey.

Biomass fuels are at the low end of the energy ladder in terms of combustion efficiency and cleanliness.[3] Smoke from biomass combustion produces a large number of health-damaging air pollutants including respirable particulate matter, carbon monoxide (CO), 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, concentrations of these air pollutants tend to be highest indoors.[4] The fuels are typically burned in simple, inefficient, and mostly unvented household cookstoves, which, combined with poor ventilation, generate large volumes of smoke indoors. Moreover, cookstoves are typically used for several hours each day at times when people are present indoors, resulting in much higher exposure to air pollutants than from outdoor sources.[5]

Population and Health Studies, East-West Center, 1601 East-West Road, Honolulu, HI 96848–1601, USA. E-mail: MishraV@EastWestCenter.Org

In such settings, daily average and peak exposures to air pollutants often far exceed safe levels recommended by the World Health Organization.[6] A comparison of typical levels of carbon monoxide (CO), particulate matter ⩽10 μm (PM_{10}), and ⩽2.5 μm ($PM_{2.5}$) in developing-country homes using biomass fuels with the US Environmental Protection Agency's standards for 24-hour average concluded that indoor concentrations of these pollutants in biomass-fuel-using developing-country homes usually exceed the guideline levels several-fold.[4] Exposure levels are usually much higher among women who tend to do most of the cooking[7] and among young children who stay indoors and who are often carried on their mother's back or lap while cooking.[8]

A recent baseline survey conducted in two districts of Zimbabwe found that women and young children spend an average of 5 hours per day in the kitchen area, where air pollution levels from biomass fuel combustion for cooking tend to be very high. The measured levels of CO in the kitchen were in the range of 300–1000 p.p.m.[9]

Exposure to biomass smoke has been strongly associated with ARI in preschool age children[10–14] and with chronic obstructive pulmonary disease (COPD) or chronic bronchitis in women.[15–21] However, a few studies of preschool age children[22–23] and several studies of school age children[24–26] have failed to find a relationship between biomass smoke and ARI. For a comprehensive review of studies on indoor air pollution and ARI see Smith *et al.*[27] Biomass smoke has also been associated with other major respiratory diseases including tuberculosis[28–30] and asthma,[31–36] but the evidence for tuberculosis is limited and the evidence for asthma is conflicting.

An earlier study in Zimbabwe compared 244 children under age 3 who visited a hospital with lower respiratory disease with 500 children of similar nutritional and socioeconomic background reported to a local Well Baby Clinic and found a significant association between the presence of woodsmoke pollution in the house and lower respiratory disease.[37] This hospital-based study of 244 ARI cases is the only study to date that has tried to link biomass smoke with ARI in children in Zimbabwe.

Methods

The analysis presented in this paper is based on information on 3559 children under age 5 years included in the fourth Zimbabwe Demographic and Health Survey (ZDHS) conducted in 1999. The ZDHS collected demographic, socioeconomic, and health information from a nationally representative probability sample of 6369 households and 5907 women age 15–49 in the sample households, representing all 10 provinces of Zimbabwe. The sample is a two-stage cluster sample with an overall response rate of 97.8%. In the first stage, 230 enumeration areas were selected with equal probability. In the second stage, households were selected using probability proportional to size (PPS) sampling, after a complete household listing and mapping of the selected enumeration areas. To allow estimation of key demographic and health parameters separately for the 10 provinces, the sampling design allowed for over sampling of smaller strata. The survey questionnaires were carefully translated into *Shona* and *Ndebele* languages, and back translated to ensure that the questions measured what they intended to measure. The questionnaires were field tested in April 1999 in the Bulawayo and Gweru areas using 150 *Shona* and *Ndebele* households. Training for the field staff for the main survey was conducted over a 4-week period in July 1999. Data were collected during 15 August to 30 November 1999. Further details about the sampling design, survey management, and quality control are provided in the basic survey report.[2]

For each child under age 5, the mother was asked if the child had been ill with a cough in the 2-week period preceding the survey interview. For children who had been ill with cough in the last 2 weeks, the mother was additionally asked if the child, when ill with cough, breathed faster than usual with short, rapid breaths. Children who suffered from cough accompanied by short and rapid breathing at any time during the last 2 weeks are defined as having suffered from an acute respiratory infection. This reported prevalence of ARI is the response variable in our analysis.

Exposure to cooking smoke was ascertained indirectly by type of fuel used for cooking. The survey used an eightfold classification of main cooking fuel—wood, dung, straw, charcoal, kerosene, electricity, liquid petroleum gas (LPG)/natural gas, and a residual category of other fuels. The question was, 'What type of fuel does your household mainly use for cooking?' Information on fuel types was used to group households into three categories representing the extent of exposure to cooking smoke—high pollution fuels (wood, dung, or straw), medium pollution fuels (kerosene or charcoal), and low pollution fuels (LPG/natural gas or electricity). The small residual category of other fuels (0.1% of the sample) was excluded from the analysis due to unknown nature of fuels in that category.

A number of multivariate logistic regression models were estimated to assess the effects of cooking fuel type (representing exposure to cooking smoke) and other factors on ARI. The analysis was carried out using the GENMODE procedure in the SAS System.[38] Control variables, identified in previous research as covariates of ARI, included age of child in months (0–5, 6–11, 12–23, 24–35, 36–59), 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 (15–24, 25–34, 35–49), mother's education in completed years of schooling (<3, 3–6, 6+), mother's religion (Christian, non-Christian), household standard of living (low, medium, high), and region of residence (Manicaland, Mashonaland Central, Mashonaland East, Mashonaland West, Matabeleland North, Matabeleland South, Midlands, Masvingo, Harare, Bulawayo). See footnotes to Table 1 for definitions of child's nutritional status and household standard of living variables.

A weighing factor was applied to all observations to compensate for over-sampling of certain categories of respondents in the study design. Results are presented in the form of odds ratios (OR) with 95% CI. The estimation of CI takes into account design effects due to clustering at the household level and at the level of the primary sampling unit. The adjustments for clustering at the household and primary sampling unit levels were done using the GEE technique for correlated data in the SAS System.[38]

Human subjects' informed consent

The analysis presented in this paper is based on secondary analysis of existing survey data with all identifying information

Table 1 Sample distribution and reported prevalence of acute respiratory infections (ARI) among children under age 5 during the 2 weeks preceding the survey, by selected characteristics, Zimbabwe, 1999

Characteristic	Sample distribution (%)	ARI prevalence (%)
Zimbabwe	–	15.82
Cooking fuel type		
High pollution fuel	66.08	17.94
Medium pollution fuel	10.60	15.03
Low pollution fuel	23.32	10.09
Age of child (in months)		
0–5	10.31	13.77
6–11	9.68	20.59
12–23	21.90	20.57
24–35	19.15	16.38
36–59	38.96	12.00
Sex of child		
Boy	48.66	16.04
Girl	51.34	15.59
Birth order		
1	32.87	14.87
2	22.71	17.36
3	14.55	17.95
4+	29.86	14.61
Nutritional status of child		
Stunted[a]	26.45	16.98
Not stunted	73.55	17.06
Mother's age at childbirth		
15–24	53.62	15.99
25–34	34.45	16.28
35–49	11.92	13.81
Mother's education (years)		
<3	26.91	17.39
3–6	42.18	14.70
6+	30.91	16.04
Mother's religion		
Christian	81.03	17.72
Non-Christian	18.97	15.40
Household standard of living[b]		
Low	33.20	17.98
Medium	38.88	17.06
High	27.92	11.43
Residence		
Urban	32.55	11.27
Rural	67.45	18.05
Region		
Manicaland	16.10	16.17
Mashonaland Central	9.49	27.83
Mashonaland East	8.79	17.07
Mashonaland West	9.80	9.62
Matabeleland North	5.22	5.45
Matabeleland South	6.25	14.63
Midlands	12.84	22.56
Masvingo	10.07	17.54
Harare	15.51	13.33
Bulawayo	5.91	4.21
No. of children[c]	3559	3269

[a] 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.

[b] Household standard of living index (SLI) is calculated by adding the following scores: 3 for a car or tractor; 2 each for a scooter/motorcycle, TV, telephone, refrigerator, piped/public tap water, flush toilet, electricity, wood/vinyl/asphalt/ceramic/cement/carpet of main floor material; 1 each for a bicycle, radio. Index scores range from 0–2 for low SLI, 3–8 for medium SLI, 9–21 for high SLI.

[c] Number of children varies slightly depending on the number of missing cases at each variable.

removed. The survey obtained informed consent from each respondent (in this case, mothers of the children included in the analysis) before asking questions.

Results

About two-thirds (66%) of children live in households that rely primarily on high pollution biomass fuels (wood, dung, or straw) for cooking, 11% live in households using medium pollution fuels (charcoal or kerosene), and the remaining 23% live in households using low pollution fuels (LPG/natural gas or electricity) (Table 1). Children are more or less evenly distributed by age and sex. The proportion of children in the sample declines by birth order, from 33% at birth order 1 to 23% at birth order 2, and 15% at birth order 3. Twenty-six per cent of the children are stunted, 54% are born to mothers aged 15–24, 31% have mothers with ⩾6 years of education, and 19% have non-Christian mothers. Thirty-three per cent live in low standard of living households and 28% live in high standard of living households. Thirty-three per cent live in urban areas and 67% live in rural areas. By region of residence, the largest proportions are from Manicaland and Harare provinces (16% each) and smallest from Matabeleland North (5%), Bulawayo (6%), and Matabeleland South (6%) provinces.

Sixteen per cent of children under age 5 suffered from an ARI during the 2 weeks preceding the survey. The reported prevalence of ARI was much higher among children living in biomass-fuel-using households (18%) than among those living in households using low pollution fuels (10%) (Table 1). Children aged 6–23 months were more likely to have suffered from ARI than children under 6 months of age or older children. This pattern of childhood disease rates peaking at 6–23 months is typical for ARI and diarrhoea in many developing countries. This may be partly due to start of supplementary feeding around 6 months of age, which increases the likelihood of consuming contaminated foods and removes the protection provided by breast milk. Also, children start crawling around this age and are more likely to be carried outdoors, which exposes them to infections. The disease rate typically declines as children grow older and start developing resistance.

Children from high standard of living households were considerably less likely to have had ARI (11%) than those from low or medium standard of living households (17–18%). The prevalence of ARI is much lower in urban areas (11%) than in rural areas (18%). By region of residence, the prevalence of ARI ranged from Bulawayo (4%) and Matabeleland North (5%) at the low end to Midlands (23%) and Mashonaland Central (28%) at the high end. The pattern of ARI prevalence by region of residence is generally consistent with the patterns of diarrhoea prevalence and infant and child mortality rates in Zimbabwe, and may reflect differential access to health care and climatic conditions. Prevalence of ARI did not vary much by sex of child, birth order of child, nutritional status of child, mother's age at childbirth, mother's education, or mother's religion. Children of birth order 2 and 3, children of younger mothers (aged 15–34), children of mothers with <3 years of education, and children of Christian mothers were somewhat more likely to have suffered from ARI than other children.

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 LPG/natural gas or electricity for cooking (OR = 1.95, 95% CI: 1.40, 2.71) (Table 2, Model 1). As expected, children from households using medium pollution charcoal or kerosene also had higher ARI prevalence (OR = 1.58, 95% CI: 0.96, 2.71) than those from households using LPG/natural gas or electricity. The effect of biomass fuel use on ARI remains large and statistically significant when child's age, sex, birth order, and nutritional status are controlled in Model 2 (OR = 2.13, 95% CI: 1.45, 3.12). Controlling for child's demographic and physical growth characteristics sharpens the effect of biomass fuel use to some extent. When mother's age at childbirth, mother's education, mother's religion, and household living standard are additionally controlled in Model 3, the effect of cooking with biomass fuels remains virtually unchanged—large and statistically significant (OR = 2.12; 95% CI: 1.19, 3.77). In the full model (Model 4), when region of residence is additionally controlled, the effect of cooking with biomass fuels on ARI prevalence sharpens further, and remains large and statistically significant (OR = 2.20, 95% CI: 1.16, 4.19).

Table 2 Odds ratio estimates of effects of cooking fuel type and other factors on acute respiratory infection (ARI) prevalence among children under age 5, Zimbabwe, 1999

	Model 1	**Model 2**	**Model 3**	**Model 4**
Characteristic	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR 95% CI)
Cooking fuel type				
High pollution fuel	1.95 (1.40, 2.71)	2.13 (1.45, 3.12)	2.12 (1.19, 3.77)	2.20 (1.16, 4.19)
Medium pollution fuel	1.58 (0.96, 2.59)	1.42 (0.77, 2.64)	1.43 (0.70, 2.92)	1.33 (0.64, 2.77)
Low pollution fuel[a]	–	–	–	–
Age of child (months)				
0–5[a]	–	–	–	
6–11		2.22 (1.38, 3.56)	2.20 (1.37, 3.53)	2.24 (1.37, 3.65)
12–23		1.87 (1.19, 2.92)	1.83 (1.16, 2.88)	1.90 (1.19, 3.03)
24–35		1.33 (0.84, 2.13)	1.26 (0.78, 2.02)	1.27 (0.78, 2.07)
36–59		1.00 (0.65, 1.52)	0.96 (0.63, 1.48)	1.00 (0.65, 1.55)
Sex of child				
Boy[a]		–	–	–
Girl		1.04 (0.82, 1.31)	1.03 (0.81, 1.31)	1.04 (0.81, 1.32)
Birth order				
1[a]		–	–	–
2		1.08 (0.78, 1.50)	1.04 (0.75, 1.45)	1.08 (0.76, 1.53)
3		1.17 (0.83, 1.65)	1.19 (0.81, 1.75)	1.26 (0.85, 1.87)
4+		0.84 (0.62, 1.14)	0.92 (0.59, 1.44)	0.97 (0.61, 1.53)
Nutritional status of child				
Stunted		0.90 (0.69, 1.17)	0.92 (0.71, 1.20)	0.93 (0.71, 1.22)
Not stunted[a]		–	–	–
Mother's age at childbirth				
15–24[a]			–	–
25–34			0.95 (0.67, 1.34)	0.94 (0.66, 1.34)
35–49			0.74 (0.44, 1.25)	0.70 (0.41, 1.20)
Mother's education (years)				
<3[a]			–	–
3–6			0.88 (0.65, 1.20)	0.91 (0.66, 1.25)
6+			0.85 (0.62, 1.18)	0.90 (0.65, 1.25)
Mother's religion				
Christian[a]			–	–
Non-Christian			0.97 (0.71, 1.34)	0.99 (0.71, 1.37)
Household standard of living				
Low[a]			–	–
Medium			1.03 (0.78, 1.35)	0.95 (0.71, 1.27)
High			1.01 (0.59, 1.73)	0.98 (0.56, 1.73)
Region				
Manicaland				0.73 (0.35, 1.50)
Mashonaland Central				1.45 (0.71, 2.96)
Mashonaland East				0.78 (0.37, 1.63)
Mashonaland West				0.38 (0.17, 0.82)
Matabeleland North				0.21 (0.09, 0.51)
Matabeleland South				0.44 (0.21, 0.91)
Midlands				1.12 (0.57, 2.21)
Masvingo				0.79 (0.38, 1.62)
Harare[a]				–
Bulawayo				0.37 (0.17, 0.82)
No. of children	3266	2482	2451	2451

[a] Reference category.

With cooking fuel type and other variables controlled, child's age and region of residence are the only variables that have statistically significant effects on ARI in children. As in the case of unadjusted ARI prevalence levels reported in Table 1, adjusted odds of ARI in Model 4 are much higher at ages 6–23 months than at ages 0–5 months or 24–59 months. By region, adjusted odds of ARI are lowest in Matabeleland North, Bulawayo, and Mashonaland West provinces and highest in Mashonaland Central and Midlands provinces.

Discussion

ARI are a serious problem in Zimbabwe. Results of this study suggest that exposure to cooking smoke from biomass combustion is significantly associated with ARI prevalence in young children, independent of child's age, nutritional status, maternal education, household living standard, and other factors (OR = 2.20; 95% CI: 1.16, 4.19). These results are consistent with the earlier hospital-based case-control study of young Zimbabwean children[37] and provide further evidence that cooking with high pollution unprocessed biomass fuels can increase the risk of ARI in young children.

The mechanism by which cooking smoke can increase the risk of ARI is not fully understood. Exposure to biomass smoke has been associated with compromised pulmonary immune defence mechanisms.[39–43] Tobacco smoke also has been shown to cause depressed immune system responses.[44–48] Of 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 the cardiorespiratory morbidity.[49–52] 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.[53–55] 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.[56,57] It is, therefore, possible that extended exposure to high levels of cooking smoke can impair the pulmonary defence mechanisms, compromise the lung function, and render children more susceptible to ARI.

Several measurement constraints should be kept in mind when considering the findings of this study. First, because the survey did not collect information on tobacco smoking by household members, we were unable to control for environmental tobacco smoke (ETS), which is a known risk factor for acute respiratory illness in children.[58] Although data on mother's smoking were collected, maternal smoking is not a significant confounding factor because women rarely smoke in Zimbabwe (only 1.47% in the ZDHS sample).

Second, many households in Zimbabwe typically use a combination of cooking fuels, whereas we have information only on the primary cooking fuel. Our estimated effects are attenuated to the extent that a mix of biomass fuels and cleaner fuels is actually used by many households instead of biomass fuels alone.

Third, there is a possibility of some selection in the sample due to ARI-related mortality. 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 are downwardly biased. However, given high prevalence of ARI and relatively small number of deaths in the sample, the impact of this bias on our estimated effect is likely to be small.

Fourth, there is also a possibility of underreporting of ARI due to lack of awareness that the child had the disease during the 2-week reference period. To the extent that underreporting due to lack of awareness is greater among those living in households using biomass fuels, it would contribute to underestimation of the effect of cooking smoke on the prevalence of ARI.

Fifth, we were unable to control directly for urban/rural residence in the analysis owing to its collinearity with cooking fuel type (r = 0.95). However, our analysis includes controls for measures of socioeconomic status (SES) such as child's nutritional status, mother's education, and household living standard, which may provide some gradient of exposure within urban and rural populations. Also, the analysis controls for residence in the 10 provinces of Zimbabwe, including two exclusively urban provinces of Harare and Bulawayo. We feel that controlling for SES and region adequately controls for potential confounding effects of urban/rural residence. Moreover, other similar studies of national survey data have found no statistically significant effects of urban/rural residence on respiratory health after controlling for SES, and in these studies controlling for urban/rural residence did not alter the effects of cooking fuel.[28,31]

Sixth, we were unable to control directly for crowding in the household because the ZDHS did not collect data on number of rooms in the household. Indoor crowding tends to be correlated with biomass fuel use and may affect the risk of ARI. However, indoor crowding is controlled in our analysis to the extent it is correlated with birth order of child and with various measures of SES included in the analysis.

Finally, we were also not able to control directly for extent of use of medical services, because the survey did not collect any information on this subject. However, our set of control variables includes measures of SES, which are correlated with access to and use of medical services.

As mentioned earlier, information on ARI is based on mothers' reports and no clinical measurements were undertaken, and smoke exposure was ascertained from type of fuel used for cooking. Although the symptomatic definition used here is intended to measure acute lower respiratory infections (ALRI) in children, some acute upper respiratory illness may have been included in the reported prevalence. Because it is not possible to separate ALRI from these data, we use the term ARI in this study, not ALRI. In developing countries such as Zimbabwe, where clinical data on ARI are usually not available or very weak, the symptomatic definition of illness used here has been shown to provide a fairly accurate assessment of ARI in the population.[59] Moreover, indoor air pollution measurements in several developing countries have shown fuel type to be the best single indirect indicator of household pollution levels.[60] 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, with direct measures of smoke exposure and clinical measures of ARI. Such research is important because a large proportion of households in Zimbabwe and other developing countries rely on biomass fuels for household energy and ARI are 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 Zimbabwe, the findings of this study have important implications for public health programmes and policy. 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 Zimbabwe that currently rely on biomass fuels are unlikely to be able to afford cleaner fuels any time soon, and given poor infrastructure to supply cleaner fuels to most rural households, widespread adoption of cleaner fuels is unlikely to occur in the short term. Therefore, the efforts need to focus on providing improved cookstoves designed to reduce exposure to smoke by means of improved combustion and improved venting, and designing public information campaigns to inform people about the health risks of exposure to indoor smoke. For such programmes to be effective, local needs and community participation should receive high priority.

Acknowledgements

The author is thankful to Sally Dai and Gayle Yamashita for assistance in data analysis. Thanks are also due to Kirk Smith, Robert Retherford, and four anonymous reviewers for useful comments on an earlier draft of this paper.

KEY MESSAGES

- Household use of unprocessed biomass fuels (wood, dung, crop residues/grasses) exposes many women and young children in developing countries to high levels of toxic air pollutants indoors.
- In Zimbabwe, children in households using biomass fuels for cooking are more than twice as likely to suffer from acute respiratory infections as those in households using cleaner fuels (liquid petroleum gas [LPG]/natural gas, electricity).
- Governments need to promote widespread use of cleaner fuels, provide improved cookstoves, and inform people about health risks of indoor smoke.

References

1 World Health Organization. *World Health Report 2001*. Geneva: WHO, 2001.

2 Central Statistical Office (CSO) Zimbabwe and Macro International Inc. *Zimbabwe Demographic and Health Survey 1999.* Calverton, MD: Central Statistical Office and Macro International Inc., 2000.

3 Smith KR, Liu Y. Indoor air pollution in developing countries. In: Samet JM (ed.). *Epidemiology of Lung Cancer, Lung Biology in Health and Disease Series* 1994;**74:**151–84.

4 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:**1080–92.

5 Smith KR. Indoor air pollution in developing countries: recommendations for research. *Indoor Air* 2002;**12:**1–7.

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

7 Behera D, Dash S, Malik SK. Blood carboxyhaemoglobin levels following acute exposure to smoke of biomass fuel. *India J Med Res* 1988;**88:**522–42.

8 Albalak R. *Cultural Practices and Exposure to Particulate Pollution from Indoor Biomass Cooking: Effects on Respiratory Health and Nutritional Status among the Aymara Indians of the Bolivian Highlands* [PhD Thesis]. Ann Arbor, Michigan: University of Michigan. 1997.

9 Mika L. Addressing health and household energy problem in Zimbabwe. Paper presented at the 9th International Conference on Indoor Air Quality and Climate, Monterey, California. June 30–July 5 2002.

10 Ezzati M, Kammen D. Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: an exposure-response study. *Lancet* 2001;**358:**619–24. Erratum in *Lancet* 2001;**358:**1104.

11 Robin LF, Less PS, Winget M *et al.* Wood-burning stoves and lower respiratory illnesses in Navajo children. *Pediatr Infect Dis J* 1996;**15:** 859–65.

12 Armstrong JR, Campbell H. Indoor air pollution exposure and lower respiratory infections in young Gambian children. *Int J Epidemiol* 1991;**20:**424–29.

13 Pandey MR, Boleji J, Smith KR, Wafula E. Indoor air pollution in developing countries and acute respiratory infection in children. *Lancet* 1989;**25:**427–29.

14 Kossove D. Smoke-filled rooms and lower respiratory disease in Infants. *S Afr Med J* 1982;**63:**622–24.

15 Albalak R, Frisancho AR, Keeler GJ. Domestic biomass fuel combustion and chronic bronchitis in two rural Bolivian villages. *Thorax* 1999;**54:**1004–08.

16 Bruce N, Neufeld L, Boy E, West C. Indoor biofuel air pollution and respiratory health: the role of confounding factors among women in highland Guatemala. *Int J Epidemiol* 1998;**27:**454–58.

17 Perez-Padilla R, Regalado J, Vedal S *et al.* Exposure to biomass smoke and chronic airway disease in Mexican women: A Case-control Study. *Am J Respir Crit Care Med* 1996;**154:**701–06.

18 Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. Woodsmoke exposure and risk for obstructive airways disease among women. *Chest* 1996;**109:**115–19.

19 Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 1991;**100:**385–88.

20 Malik SK. Exposure to domestic cooking fuels and chronic bronchitis. *Indian J Chest Dis Allied Sci* 1985;**27:**171–74.

21 Pandey MR. Domestic smoke pollution and chronic bronchitis in a rural community of the hill region of Nepal. *Thorax* 1984;**39:**337–39.

22 Aldous MB, Holberg CJ, Wright AL, Martinez FD, Taussig LM. Evaporative cooling and other home factors and lower respiratory tract illness during the first year of life. *Am J Epidemiol* 1996;**143:** 423–30.

23 Samet JM, Lambert WE, Skipper BJ *et al.* Nitrogen dioxide and respiratory illness in children. Part I: Health outcomes. *Res Rep Health Eff Inst* 1993;**58:**1–32; discussion 51–80.

24 Azizi BH, Henry RL. The effects of indoor environmental factors on respiratory illness in primary school children in Kuala Lumpur. *Int J Epidemiol* 1991;**20:**144–50.

25 Tuthill, RW. Woodstoves, formaldehyde, and respiratory disease. *Am J Epidemiol* 1984;**120:**952–55.

26 Anderson HR. Respiratory abnormalities in Papua New Guinea children: the effects of locality and domestic wood smoke pollution. *Int J Epidemiol* 1978;**7:**63–72.

27 Smith KR, Samet JM, Romieu I, Bruce N. Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax* 2000;**55:**518–32.

28 Mishra VK, Retherford RD, Smith KR. Biomass cooking fuels and prevalence of tuberculosis in India. *Int J Infect Dis* 1999;**3:**119–29.

29 Mishra VK, Retherford RD, Smith KR. Cooking smoke can increase the risk of tuberculosis. *Boiling Point* 2000;**44:**11–13.

30 Perez-Padilla R, Perez-Guzman C, Baez-Saldana R, Torres-Cruz A. Cooking with biomass stoves and tuberculosis: A case-control study. *Int J Tuberc Lung Dis* 2001;**5:**1–7.

31 Mishra V. Indoor air pollution from biomass combustion and prevalence of asthma in the elderly. *Environ Health Perspect* 2003;**111:**71–77.

32 Schei MA, Hessen JO, McCraken J, Lopez V, Bruce N, Smith KR. Asthma and indoor air pollution among indigenous children in Guatemala. Paper presented at the 9th International Conference on Indoor Air Quality and Climate, Monterey, California. June 30–July 5 2002.

33 Thorn J, Brisman J, Toren K. Adult-onset asthma is associated with self-reported mold or environmental tobacco smoke exposures in the home. *Allergy* 2001;**56:**287–92.

34 Pistelly R. Wood smoke and asthma: a controversial issue. *Am J Respir Crit Care Med* 1997;**155:**A941.

35 Xu X, Niu T, Christiani DC *et al.* Occupational and environmental risk factors for asthma in rural communities in China. *Int J Occup Environ Health* 1996;**2:**172–76.

36 Mohammed N, Ng'ang'a L, Odhiambo J, Nyamwaya J, Menzies R. Home environment and asthma in Kenyan school children: a case-control study. *Thorax* 1995;**50:**74–78.

37 Collings DA, Sithole SD, Martin KS. Indoor woodsmoke pollution causing lower respiratory disease in children. *Trop Doct* 1990;**20:** 151–55.

38 SAS Institute Inc. 2001. *SAS System for Windows, Release 8.02*. Cary, NC: SAS Institute Inc.

39 Zelikoff JT. Woodsmoke Emissions: Effects on host pulmonary immune defense. *CIAR Currents* 1994;**Nov:**1–3.

40 Thomas P, Zelikoff J. Air pollutants: moderators of pulmonary host resistance against infection. In: Holgate ST, Samet JM, Koren HS, Maynard RL (eds). *Air Pollution and Health*. San Diego: Academic Press.

41 Wang J, Hu SM. The effects of kitchen air pollution on animals' immune function [Abstract]. *Chung Hua Yu Fang I Hsueh Tsa Chih* 1992;**26:**86–88.

42 Green GM, Jakab GJ, Low RB, Davis GS. Defense mechanisms of the respiratory membrane. *Am Rev Respir Dis* 1977;**115:**479–514.

43 Taszakowski W, Dwornicki J. Effect of environmental factors on the defense system of the respiratory tract [Abstract] *Pol Tyg Lek* 1992; **47:**763–65.

44 Johnson JD, Houchens DP, Kluwe WM, Craig DK, Fisher GL. Effects of mainstream and environmental tobacco smoke on the immune system in animals and humans: a review. *Crit Rev Toxicol* 1990; **20:**369–95.

45 Chang JC, Distler SG, Kaplan AM. Tobacco smoke suppresses T cells but not antigen-presenting cells in the lung-associated lymph nodes. *Toxicol Appl Pharmacol* 1990;**102:**514–23.

46 Sopori ML, Cherian S, Chilukuri R, Shopp GM. Cigarette smoke causes inhibition of the immune response to intratracheally administered antigens. *Toxicol Appl Pharmacol* 1989;**97:**489–99.

47 Hersey P, Prendergast D, Edwards A. Effects of cigarette smoking on the immune system. Follow-up studies in normal subjects after cessation of smoking. *Med J Aust* 1983;**2:**425–29.

48 Jacob CV, Stelzer GT, Wallace JH. The influence of cigarette tobacco smoke products on the immune response. The cellular basis of immunosuppression by a water-soluble condensate of tobacco smoke. *Immunology* 1980;**40:**621–27.

49 Tan WC, Qiu D, Liam BL *et al.* The human bone marrow response to acute air pollution caused by forest fires. *Am J Respir Crit Care Med* 2000;**161:**1213–17.

50 Mukae H, Vincent R, Quinlan K *et al.* The effect of repeated exposure to particulate air pollution (PM10) on the bone marrow. *Am J Respir Crit Care Med* 2001;**163:**201–09.

51 van Eeden SF, Tan WC, Suwa T *et al.* Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM_{10}). *Am J Respir Crit Care Med* 2001;**164:**826–30.

52 Fujii T, Hayashi S, Hogg JC, Vincent R, van Eeden SF. Particulate matter induces cytokine expression in human bronchial epithelial cells. *Am J Respir Cell Mol Biol* 2001;**25:**265–71.

53 Kong LY, Luster MI, Dixon D, O'Grady J, Rosenthal GJ. Inhibition of lung immunity after intratracheal instillation of benzo(a)pyrene. *Am J Respir Crit Care Med* 1994;**150:**1123–29.

54 Hardin JA, Hinoshita F, Sherr DH. Mechanisms by which benzo-[a]pyrene, an environmental carcinogen, suppresses B cell lymphopoiesis. *Toxicol Appl Pharmacol* 1992;**117:**155–64.

55 Schnizlein CT, Bice DE, Mitchell CE, Hahn FF. Effects on rat lung immunity by acute lung exposure to benzo(a)pyrene. *Arch Environ Health* 1982;**37:**201–06.

56 Samet JM, Utell MJ. The risk of nitrogen dioxide: What have we learned from epidemiological and clinical studies? *Toxicol Ind Health* 1990;**6:**247–62.

57 Samet JM, Spengler JD (eds). *Indoor Air Pollution: A Health Perspective*. Baltimore, MD: Johns Hopkins University Press, 1991.

58 Janet SML, Jennifer KP, Xuan W, Berry G. Meta analysis on the association between environmental tobacco smoke (ETS) exposure and the prevalence of lower respiratory tract infection in early childhood. *Pediatr Pulmonol* 1999;**27:**5–13.

59 Cabaraban, MC. *Rural Mother's Management of Acute Respiratory Infection (ARI) in Region 10: Project Report to the Health Research Network*. Cagayan de Oro, Philippines: Research Institute for Mindanao Culture, Xavier University. 1993.

60 Mehta S, Smith KR, Balakrishnan K *et al.* Using household characteristics to predict respirable particulate levels in rural households in Andhra Pradesh, India. Paper presented at the 9th International Conference on Indoor Air Quality and Climate, Monterey, California. June 30–July 5 2002.