Reduced lung function due to biomass smoke exposure in young adults in rural Nepal

Om P. Kurmi*, Graham S. Devereux#, W. Cairns S. Smith#, Sean Semple#, Markus F.C. Steiner#, Padam Simkhada¶, Kin-Bong Hubert Lam* and Jon G. Ayres*

ABSTRACT: This study aimed to assess the effects of biomass smoke exposure on lung function in a Nepalese population, addressing some of the methodological issues seen in previous studies.

We carried out a cross-sectional study of adults in a population exposed to biomass smoke and a non-exposed population in Nepal. Questionnaire and lung function data were acquired along with direct measures of indoor and outdoor air quality.

Ventilatory function (forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and forced expiratory flow at 25–75% of FVC) was significantly reduced in the population using biomass across all age groups compared to the non-biomass-using population, even in the youngest (16–25 yrs) age group (mean FEV_1 (95% CI) 2.65 (2.57–2.73) *versus* 2.83 (2.74–2.91) L; p=0.004). Airflow obstruction was twice as common among biomass users compared with liquefied petroleum gas users (8.1% *versus* 3.6%; p<0.001), with similar patterns for males (7.4% *versus* 3.3%; p=0.022) and females (10.8% *versus* 3.8%; p<0.001), based on the lower limit of normal. Smoking was a major risk factor for airflow obstruction, but biomass exposure added to the risk.

Exposure to biomass smoke is associated with deficits in lung function, an effect that can be detected as early as the late teenage years. Biomass smoke and cigarette smoke have additive adverse effects on airflow obstruction in this setting.

KEYWORDS: Airflow obstruction, biomass, indoor air pollution, lung function

About half the world's population, mostly in developing countries, use solid fuels (biomass and coal) [1] as their main energy source, resulting in potentially harmful exposures. Chronic obstructive pulmonary disease (COPD) is as much a disease of developing countries as of the developed world [2] with the World Health Organization estimating that ~700,000 of the 2.7 million global deaths due to COPD are attributable to indoor air pollution from solid fuels [3], particularly in females. A meta-analysis has reported a three-fold increase in risk for COPD in populations exposed to solid-fuel smoke; the effect being dependent on fuel type, with those exposed to wood smoke having the greatest effect (relative risk 4.3) compared with animal dung/crop residues (relative risk 2.5) and coal/charcoal (relative risk 1.5–1.8) [4]. Studies from developing countries, including Nepal, have shown higher prevalence of respiratory symptoms and reduced lung function associated with solid fuel use both in children and in adults, particularly in females involved in cooking [4]. However, many of these studies from Nepal have suffered from inadequate methodology, including the use of non-validated questionnaires, poor-quality spirometry results, lack of a non-exposed population, compromised validity of exposure assessment and limited control for confounding factors such as cigarette smoking. All of these shortfalls could have potentially resulted in over-estimation of effect sizes.

This cross-sectional study aimed to use validated objective and subjective measures of lung disease to test the hypothesis that exposure to the products of biomass fuel use is associated with reduced lung function and increased airflow obstruction when compared with exposure to liquefied petroleum gas (LPG). A secondary aim was to relate real-time particle exposures to lung function.

This article has supplementary material available from www.erj.ersjournals.com

METHODS

Sampling frame and participants

Between April 2006 and February 2007, a biomass-exposed population (98.9% used wood) were sampled from two village development committees (VDCs) in the Kathmandu Valley, Nepal. Four wards (out of nine) in each VDC were randomly selected and all individuals in the selected wards aged $\geqslant 16$ yrs were eligible if they met the inclusion criteria (no doctor-diagnosed respiratory or cardiovascular health problems and agreement to 24-h exposure monitoring in their homes). The non-exposed population (98.4% used LPG) were selected from six wards (from a total of 35) in the Kathmandu municipality: three selected randomly on the periphery of a ring road and the other three selected from 1–2 km inside the ring road. The non-exposed sample lived around 10–12 km to the south-west of the biomass-exposed sampling sites. All locations were 1,300–1,600 m above sea level. The majority of the houses in the biomass-exposed sample were constructed from a mud-based material with a thatched or tiled roof, whereas in the non-exposed areas, houses were made of brick and cement. The non-exposed population lived in close proximity to main roads, while the biomass-exposed population lived in rural areas with negligible traffic or industrial activities. However, the latter regularly travelled to areas near the ring road to sell their agricultural products in the early mornings (when traffic is minimal). The study protocol was approved by the Nepal Health Research Council (Kathmandu, Nepal). Written consent was obtained from all participants.

Sample size

As published lung function data from Nepal have been limited, sample size was calculated assuming a prevalence of COPD of 10% in the non-exposed population and 20% in the exposed population, the latter being twice the reported prevalence in previous Nepalese studies [5, 6]. For 80% power at a 5% significance level and allowing for 10% dropout or refusal, a sample size of 396 females and 396 males in each exposure group was needed.

Particle exposure measurement

Full details have been reported elsewhere [7]. Levels of respirable dust and particulate matter with a diameter $\leqslant 2.5$ μm ($PM_{2.5}$) were measured over a 24-h period in most dwellings (n=490) using a photometric device (DustTrak 8520 and SidePak AM510; TSI Inc., Shoreview, MN, USA). All data obtained by the photometric devices were multiplied by a calibration factor obtained using a standard gravimetric technique [7]. Indoor carbon monoxide levels over 24 h were measured by HOBO CO loggers (MicroDAQ, Contoocook, NH, USA). Mean 24-h exposure data are expressed as geometric mean and geometric standard deviation unless indicated.

Lung function

All participants underwent spirometry using the EasyOne spirometer (ndd Medizintechnik AG, Zurich, Switzerland) at their homes in a standing position, in accordance with the latest American Thoracic Society (ATS)/European Respiratory Society (ERS) guidelines [8] without the use of a bronchodilator. The quality of the spirometry data was assessed at the Institute of Occupational and Environmental Medicine (University of Birmingham, Birmingham, UK) by one of the authors (J.G.

Ayres) by inspection of both volumetric and flow–volume traces. Unacceptable traces were removed. The best of three reproducible values was used in the analysis. We defined airflow obstruction as forced expiratory volume in 1 s (FEV_1) to forced vital capacity (FVC) ratio less than the lower limit of normal (LLN) to minimise over- or under-diagnosis [9]. Predicted values were calculated by the reference equations from the European Community for Steel and Coal with a 10% reduction for non-Caucasians [9]. Height was measured by measuring tape, and weight was measured without footwear and with very light clothing, using a digital scale. For comparability with some previous studies, we repeated the analysis by redefining airflow obstruction as $FEV_1/FVC <70\%$.

Questionnaire

An interviewer-administered questionnaire was used to collect data on smoking, socioeconomic status, kitchen characteristics, cooking details, literacy and history of fuel use.

Statistical analysis

Statistical analyses were performed using STATA (version 11; Stata Corp., College Station, TX, USA). Baseline demographic characteristics were compared between biomass-exposed and non-exposed samples by regression for survey data, taking into account household clustering. Linear and logistic regression models were built to evaluate the effect of the use of biomass and the exposure levels on lung function indices and airflow obstruction, respectively. All known and potential risk factors not collinear with biomass use were routinely adjusted to obtain regression coefficient (β), with robust variance estimates to allow for a household clustering effect. In addition, we calculated three measures of biological interaction (on the additive scale): relative excess risk due to interaction (RERI), attributable proportion due to interaction (AP) and the synergy index (SI), where in the absence of interaction, both RERI and AP are 0 and SI is 1 [10, 11]. A positive interaction indicates that the combined effect of exposures is larger than the sum of the individual effects, and a negative interaction represents a smaller combined effect.

RESULTS

A total of 1,648 participants were enrolled, of whom 1,392 (656 males and 736 females) had valid spirometry results and were used in analysis. Of these, 49.9% (n=695) were exposed to biomass smoke and remaining 50.1% (n=697) used non-biomass fuel (primarily LPG) for domestic purposes (table 1). Biomass-exposed males and females were significantly shorter, weighed less and were more likely to be illiterate than their non-biomass-using counterparts. The biomass-exposed groups had much lower annual incomes compared with the other groups (median US$ 744 versus $2,496; p<0.001) and had a higher proportion of current smokers, especially among females (table 1).

Exposures

Across the dataset, the geometric mean of the 24-h indoor $PM_{2.5}$ concentrations in biomass using homes (455 μg·m^{-3}, 95% CI 426–485 μg·m^{-3}) was significantly greater than in LPG using homes (101 μg·m^{-3}, 95% CI 96–106 μg·m^{-3}; p<0.001). Indoor $PM_{2.5}$ concentrations in ventilated kitchens where biomass was burned (448 μg·m^{-3}, 95% CI 405–495 μg·m^{-3}) were significantly higher than where LPG was used either with

TABLE 1	Demographic data of 1,392 Nepalese adults aged $\geqslant$16 yrs according to type of fuel use by sex					
	Males			**Females**		
	Biomass	**Non-biomass**	**p-value**	**Biomass**	**Non-biomass**	**p-value**
Subjects n	326	330		369	367	
Age yrs	35.2±16.7	36.3±14.9	0.636	34.8±16.0	34.8±14.8	0.991
Height cm	162.2±7.3	165.7±6.7	0.029	150.1±5.6	153.1±6.2	0.037
Weight kg	52.8±8.2	62.5±10.0	0.003	46.6±7.0	57.0±10.0	0.002
Body mass index kg·m^{-2}	20.0±2.6	22.8±3.4	0.005	20.7±2.8	24.3±3.8	0.002
Literate	274 (84.1)	321 (97.3)	0.015	174 (47.2)	295 (80.4)	0.024
Smoking status						
Non-smoker	181 (55.5)	210 (63.6)	0.070	242 (65.6)	336 (91.6)	0.012
Ex-smoker	26 (8.0)	56 (17.0)	0.197	42 (11.4)	19 (5.2)	0.232
Current smoker	119 (36.5)	64 (19.4)	0.119	85 (23.0)	12 (3.3)	0.020

Data are presented as mean±SD or n (%), unless otherwise specified. p-values were derived from regression analysis of survey data taking into account the clustering of households.

ventilation (99 µg·m^{-3}, 95% CI 94–104 µg·m^{-3}; p<0.001) or without ventilation (120 µg·m^{-3}, 95% CI 99–144 µg·m^{-3}; p<0.001). The 24-h time-weighted average carbon monoxide concentrations measured in kitchens were significantly (p<0.001) higher where biomass fuel was used (13.4 ppm, 95% CI 11.7–15.4 ppm) compared with where LPG fuel was used (2.0 ppm, 95% CI 1.9–2.2 ppm).

Lung function parameters

In general, univariate analysis suggested that biomass smoke-exposed individuals had poorer lung function than the non-exposed individuals. Deficits in FEV_1, FVC, FEV_1/FVC and forced expiratory flow at 25–75% of FVC ($FEF_{25-75\%}$) were found in both males and females, across the age range (table S1). FEV_1, FEV_1/FVC and $FEF_{25-75\%}$ remained lower in biomass fuel-using participants, even after adjusting for potential confounders including height, age, sex, body mass index (BMI), literacy, income, ever smoking history and environmental tobacco smoke exposure. Similar findings were obtained when the data were re-analysed after stratifying for sex (table 2). In the youngest age group of 16–25 yrs, FEV_1 was 0.225 L (95% CI 0.106–0.344 L; p<0.001) less, while $FEF_{25-75\%}$ was 0.510 L·s^{-1} (95% CI 0.263–0.757 L·s^{-1}; p<0.001) lower in participants exposed to biomass smoke, having adjusted for confounders. However, there was no significant association between FVC and biomass use. Conversely, none of the lung function parameters measured was significantly associated with carbon monoxide and $PM_{2.5}$ (table 2).

Airflow obstruction

The prevalence of airflow obstruction in the biomass smoke-exposed population (8.1%) was significantly higher than that in the non-biomass-exposed group (3.6%) with similar findings for males (7.4% versus 3.3%: p=0.022) and females (10.8% versus 3.8%; p<0.001) separately (table S2). Those aged 16–25 yrs and exposed to biomass smoke (n=253) had a significantly higher prevalence of airflow obstruction compared with non-exposed individuals of the same age (n=223) regardless of smoking status (3.6% versus 0.5%; p=0.018). When restricting to lifelong

non-smokers, airflow obstruction was also more common in the biomass smoke exposed group (5.2% versus 1.8%, p=0.004).

Multivariate analysis (adjusted for height, age, sex, BMI, literacy, income, smoking history and environmental tobacco smoke) shows that airflow obstruction was significantly higher in the biomass smoke-exposed population compared with those using LPG fuel (table 2). A similar magnitude of association was obtained when stratified for sex, although being statistically nonsignificant, possibly due to a lack of power. Redefining airflow obstruction as FEV_1/FVC <70% did not alter the conclusions.

Interaction

We did not find any positive interaction between smoking history and biomass smoke exposure but found some evidence of negative interaction for airflow obstruction between ever-smokers and biomass smoke exposure (male: RERI -2.57, 95% CI -9.98-4.83; female: RERI -2.70, 95% CI -0.86-3.18) and also negative interaction in females for airflow obstruction between current smoking and biomass smoke exposure (RERI -4.10, 95% CI -13.63-5.43). Nevertheless, none of them were statistically significant (table S3).

DISCUSSION

This is the first population-based study of lung function in Nepal studying two very distinct populations: biomass and non-biomass fuel users. Different indices of lung function were significantly lower in both males and females in the biomass smoke exposed group compared to the non-exposed group and this difference was evident even in the youngest age group studied (16–25 yrs), suggesting a possible detrimental effect of biomass smoke exposure on lung growth in early life. The prevalence of airflow obstruction in the biomass smoke-exposed group was twice than that found in the non-biomass exposed group and was higher in the youngest age group.

The prevalence of airflow obstruction based on LLN, regardless of smoking status, was 8.1% in the biomass smoke exposed group and 3.6% in the non-exposed participants, being higher

TABLE 2 Regression coefficients (lung function indices) and odds ratios using robust variance estimates comparing use of biomass fuel with use of liquefied petroleum gas stoves (referent category)

	FEV1		FVC		FEF25–75%		FEV1/FVC		Airflow obstruction[#]	
	β (95% CI)[¶]	p-value	β (95% CI)[¶]	p-value	β (95% CI)[¶]	p-value	β (95% CI)[¶]	p-value	OR (95% CI)[¶]	p-value
Combined data for males and females										
Use of biomass	-0.103 (-0.167–-0.039)	0.002	-0.045 (-0.118–0.028)	0.224	-0.355 (-0.493–-0.217)	<0.001	-2.881 (-4.127–-1.636)	<0.001	2.06 (1.16–3.67)	0.014
PM2.5 mg·m^{-3}	0.001 (-0.035–0.037)	0.961	4.26×10^{-6} (-3.31×10^{-5}–4.16×10^{-5})	0.822	-5.03×10^{-6} (-1.26×10^{-4}–1.16×10^{-4})	0.935	-3.68×10^{-5} (-0.001–0.001)	0.944	1.00 (0.99–1.00)	0.482
Carbon monoxide ppm	-0.001 (-0.008–0.004)	0.488	0.001 (-0.004–0.006)	0.698	-0.009 (-0.021–0.003)	0.126	-0.139 (-0.261–-0.016)	0.027	1.02 (0.99–1.06)	0.234
Inadequate ventilation	-0.020 (-0.082–0.041)	0.517	0.009 (-0.052–0.072)	0.752	-0.104 (-0.240–0.032)	0.132	-1.004 (-2.299–0.292)	0.129	1.19 (0.73–1.93)	0.482
Sleeping in the kitchen	0.028 (-0.043–0.099)	0.437	0.048 (-0.023–0.119)	0.182	-0.016 (-0.173–0.140)	0.839	-0.385 (-1.836–1.066)	0.602	1.21 (0.60–2.46)	0.589
Female data only										
Use of biomass	-0.074 (-0.148–-0.001)	0.046	-0.034 (-0.113–0.044)	0.393	-0.304 (-0.466–-0.142)	<0.001	-2.586 (-4.244–-0.927)	0.002	2.38 (0.94–5.99)	0.067
PM2.5 mg·m^{-3}	-0.002 (-0.043–0.038)	0.908	-4.66×10^{-6} (-3.45×10^{-5}–2.52×10^{-5})	0.759	-8.56×10^{-6} (-1.70×10^{-4}–1.87×10^{-4})	0.925	0.001 (-0.001–0.002)	0.885	1.00 (0.99–1.00)	0.586
Carbon monoxide ppm	-0.005 (-0.012–0.002)	0.174	-0.002 (-0.008–0.005)	0.627	-0.013 (-0.027–0.001)	0.077	-0.214 (-0.933–-0.036)	0.019	1.04 (0.98–1.09)	0.199
Inadequate ventilation	0.002 (-0.074–0.079)	0.956	0.013 (-0.075–0.102)	0.770	-0.042 (-0.197–0.113)	0.597	-0.630 (-2.307–1.046)	0.460	1.09 (0.52–2.27)	0.824
Sleeping in the kitchen	0.015 (-0.079–0.109)	0.755	0.021 (-0.073–0.115)	0.660	-0.003 (-0.200–0.194)	0.977	-0.030 (-2.268–2.208)	0.979	0.97 (0.35–2.68)	0.950
Male data only										
Use of biomass	-0.0127 (-0.223–0.030)	0.010	-0.055 (-0.165–0.056)	0.330	-0.397 (-0.600–-0.193)	<0.001	-2.953 (-4.644–-1.261)	0.001	1.74 (0.75–4.04)	0.199
PM2.5 mg·m^{-3}	0.007 (-0.060–0.073)	0.845	1.85×10^{-5} (-4.95×10^{-5}–8.65×10^{-5})	0.593	-2.42×10^{-5} (-1.26×10^{-4}–7.78×10^{-5})	0.641	-0.0003 (-0.001–0.0007)	0.563	1.00 (0.99–1.00)	0.606
Carbon monoxide ppm	-2.79×10^{-5} (-0.007–0.007)	0.993	0.003 (-0.004–0.009)	0.415	-0.006 (-0.019–0.006)	0.316	-0.067 (-0.186–0.051)	0.260	0.99 (0.94–1.06)	0.978
Inadequate ventilation	-0.047 (-0.137–0.042)	0.300	-0.001 (-0.108–0.105)	0.982	-0.174 (-0.348–-0.0003)	0.050	-1.231 (-2.727–0.265)	0.106	1.29 (0.60–2.89)	0.515
Sleeping in the kitchen	0.026 (-0.074–0.125)	0.613	0.060 (-0.051–0.170)	0.290	-0.044 (-0.250–0.161)	0.672	-0.761 (-2.372–0.851)	0.354	1.79 (0.73–4.39)	0.202

FEV1: forced expiratory volume in 1 s; FVC: forced vital capacity; FEF25–75: forced expiratory flow at 25–75% of FVC; PM2.5: particulate matter with a diameter $\leqslant$2.5 µm. [#]: defined as FEV1/FVC below the lower limit of normal; [¶]: adjusted for height, age, sex, literacy, income, body mass index, smoking history ("ever-smoker": $\geqslant$20 packs of cigarettes or 12 oz (360 g) tobacco in a lifetime, or $\geqslant$1 cigarette per day or 1 cigar a week for 1 yr) and environmental tobacco smoke ("yes": regularly exposed to other people's tobacco smoke where "regularly" is on most days and nights).

in females than in males. Studies from Turkey [12], Nepal [13], China [14], Spain [15] and Columbia [16] have reported positive associations between biomass smoke exposure and COPD, although the quantified risk varies across a wide range [4] of effect sizes. However, the odds ratio for airflow obstruction in the biomass smoke-exposed population in this study was around half that found in a meta-analysis of studies of COPD in biomass smoke-exposed populations (OR for wood smoke exposure 3.96) [4]. This difference may be due to aspects of study design or the definition of airflow obstruction. First, we used quality-assured lung function testing, adhering strictly to the ATS/ERS guidelines [8]. Biomass smoke exposure is almost uniform in the rural areas in Nepal; ''normal'' lung function values, even if they did exist, would be difficult to interpret given the likely effects of this exposure on lung function. This can be addressed by comparing exposed with non-exposed populations, but this has often been absent in previous studies. Secondly, relevant confounding factors have not always been addressed in previous work. In this respect, we have accounted for environmental tobacco smoke exposure (self-report) and have used literacy and household income as proxies for socioeconomic status, although we cannot rule out the possibility of other unmeasured confounding factors.

Airflow obstruction was related to smoking in the non-biomass smoke exposed population (8% in current smokers, 12% in ex-smokers and 2% in never-smokers), while in the rural area, smoking and biomass exposure appeared to have a multiplicative effect in terms of airflow obstruction. This coheres with earlier work from Nepal [13], which found significantly lower lung function in both smokers and non-smokers among rural, biomass smoke-exposed dwellers compared with non-biomass smoke-exposed urban dwellers. Our findings are qualitatively similar to previous work in Ecuador [17], India [18] and Turkey [19], where populations using biomass fuel had lower lung function compared with relatively clean fuel users, irrespective of smoking status.

The deficit in FEF$_{25-75\%}$ both in females and males is consistent with our previous finding from a pilot study [20] carried out in the same area. While FEF$_{25-75\%}$ is not recommended in clinical practice for the diagnosis of small airway obstruction [21, 22], its deficit provides additional evidence for the presence of airflow obstruction.

Our study has some limitations. For practical and clinical reasons, post-bronchodilator lung function was not measured, hence we were not able to differentiate whether airflow obstruction was a manifestation of COPD or asthma. A further limitation is the near perfect concordance between the use of biomass fuel, lower socioeconomic status and rural dwelling. Although it is likely that the associations reported here between rural dwelling and reduced lung function result from lifetime exposure to biomass smoke, it is not possible to rule out confounding by other closely associated influences such as diet [23], respiratory infection [24] and low birth weight [25], all closely associated with low socioeconomic status. The exposed population being slightly shorter compared to the non-exposed population could be the result of malnourishment during development and, hence, the lower lung function observed. We had anticipated that objective measures of

indoor airborne pollution (carbon monoxide and PM$_{2.5}$) would clarify the issue, but only the association between FEF$_{25-75\%}$ and carbon monoxide was significant (p=0.027). It is likely that a ''one-off'' measurement of indoor air pollution fails to quantify the effects of a lifetime of variable exposure to biomass fuel. A previous study has suggested that a large day-to-day variability of exposures exists within a home [26]. Our findings appear to suggest that repeated measurements of exposure for a longer duration and also in different seasons are essential to understand the dose–response relationship. A detailed record of time activities along with measurements of exposure in different micro-environments such as kitchen, bedroom, living area, outdoor and workplace environments will be required to better quantify the exposures because static measurements in the kitchen only will not provide sufficient information to estimate the personal exposure with better accuracy. Ultimately, these issues can only be resolved by interventional trials, such as the work in Mexico showing significant attenuation of FEV$_1$ and fewer respiratory symptoms with the use of the improved cooking stoves [27].

Occupational exposures also contribute to the development of airflow obstruction but the extent has not been assessed in this study. The biomass-exposed group recruited in the rural areas was exposed to dust and organophosphate pesticides from agricultural activities, the former being a recognised risk factor for airway inflammation [28] and the latter being purported to be linked with reductions in lung function [29]. However, urban dwellers who did not use biomass fuel were exposed to higher levels of ambient air pollution from traffic sources. Moreover, some of the current non-users had previously lived in rural areas and had thus been exposed to biomass smoke. However, these effects would tend to reduce the comparative impact on lung function and airflow obstruction in this study.

Despite the relatively few female smokers compared to males, lung function was consistently worse in females, perhaps because females were more likely to do the cooking. Lung function in biomass smoke exposed, lifelong non-smoking and younger individuals (16–25 yrs) of both sexes was significantly lower compared to their non-exposed counterparts, as was the prevalence of airflow obstruction. Rural dwellers are exposed to biomass smoke from birth and females in Nepal start cooking regularly when they are 13–14 yrs old, thus increasing their exposures at critical times in lung development. It is thus reasonable to assume that biomass smoke exposure could retard lung growth although other factors, such as environmental tobacco smoke exposure and a diet deficient in nutrients [30], may also play a role. The fact that for rural dwellers smoking appeared to have less influence on lung function, particularly in females, may imply a swamping effect of biomass smoke.

We found evidence suggestive of negative multiplicative interactions between ever smoking and biomass smoke on airflow obstruction, possibly suggesting either a survival effect or that the combined effect of biomass and smoking is more than the individual effect of smoking and biomass but less than the multiple of biomass and smoking.

In summary, we have shown that exposure to biomass smoke results in a doubled risk of airflow obstruction in a biomass

smoke-exposed population in Nepal after allowing for other factors, but that cigarette smoking has an additive effect. The observation that these associations were evident by the late teenage years suggests that biomass smoke exposure during childhood may impair lung growth.

REFERENCES
1 Rehfuess E, Mehta S, Pruss-Ustun A. Assessing household solid fuel use: multiple implications for the Millennium Development Goals. *Environ Health Perspect* 2006; 114: 373–378.
2 Bruce N, Perez-Padilla R, Albalak R. The health effects of indoor air pollution exposure in developing countries. Geneva, World Health Organization, 2002.
3 World Health Organization. Indoor air pollution and health. www.who.int/mediacentre/factsheets/fs292/en/ Date last accessed: October 14, 2009. Date last updated: September 2011.
4 Kurmi OP, Semple S, Simkhada PP, *et al.* COPD and chronic bronchitis risk of indoor air pollution from solid fuel: a systematic review and meta-analysis. *Thorax* 2010; 65: 221–228.
5 Pandey MR. Domestic smoke pollution and chronic bronchitis in a rural community of the hill region of Nepal. *Thorax* 1984; 39: 337–339.
6 Pandey MR. Prevalence of chronic bronchitis in a rural community of the hill region of Nepal. *Thorax* 1984; 39: 331–336.
7 Kurmi OP, Semple S, Steiner M, *et al.* Particulate matter exposure during domestic work in Nepal. *Ann Occup Hyg* 2008; 52: 509–517.
8 Miller MR, Hankinson J, Brusasco V, *et al.* Standardisation of spirometry. *Eur Respir J* 2005; 26: 319–338.
9 Quanjer PH, Tammeling GJ, Cotes JE, *et al.* Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. *Eur Respir J* 1993; 6: Suppl. 16 5–40.
10 Hallqvist J, Ahlbom A, Diderichsen F, *et al.* How to evaluate interaction between causes: a review of practices in cardiovascular epidemiology. *J Intern Med* 1996; 239: 377–382.
11 Andersson T, Alfredsson L, Kallberg H, *et al.* Calculating measures of biological interaction. *Eur J Epidemiol* 2005; 20: 575–579.
12 Ekici A, Ekici M, Kurtipek E, *et al.* Obstructive airway diseases in women exposed to biomass smoke. *Environmental Research* 2005; 99: 93–98.
13 Shrestha IL, Shrestha SL. Indoor air pollution from biomass fuels and respiratory health of the exposed population in Nepalese households. *Int J Occup Environ Health* 2005; 11: 150–160.
14 Zhong N, Wang C, Yao W, *et al.* Prevalence of chronic obstructive pulmonary disease in China: a large, population-based survey. *Am J Respir Crit Care Med* 2007; 176: 753–760.
15 Orozco-Levi M, Garcia-Aymerich J, Villar J, *et al.* Wood smoke exposure and risk of chronic obstructive pulmonary disease. *Eur Respir J* 2006; 27: 542–546.
16 Dennis RJ, Maldonado D, Norman S, *et al.* Woodsmoke exposure and risk for obstructive airways disease among women. *Chest* 1996; 109: 115–119.
17 Rinne ST, Rodas EJ, Bender BS, *et al.* Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador. *Respir Med* 2006; 100: 1208–1215.
18 Saha A, Rao NM, Kulkarni PK, *et al.* Pulmonary function and fuel use: a population survey. *Respir Res* 2005; 6: 127.
19 Sumer H, Turaclar UT, Onarlioglu T, *et al.* The association of biomass fuel combustion on pulmonary function tests in the adult population of Mid-Anatolia. *Sozial-Und Praventivmedizin* 2004; 49: 247–253.
20 Kurmi OP, Gaihre S, Semple S, *et al.* Acute exposure to biomass smoke causes oxygen desaturation in adult women. *Thorax* 2011; 66: 724–725.
21 Pellegrino R, Viegi G, Brusasco V, *et al.* Interpretative strategies for lung function tests. *Eur Respir J* 2005; 26: 948–968.
22 Burgel P-R. The role of small airways in obstructive airway diseases. *Eur Respir Rev* 2011; 20: 23–33.
23 Devereux G, Seaton A. Diet as a risk factor for atopy and asthma. *J Allergy Clin Immunol* 2005; 115: 1109–1117.
24 Ezzati M, Kammen D. Indoor air pollution from biomass combustion and acute respiratory infections in Kenya: an exposure-response study. *Lancet* 2001; 358: 619–624.
25 Mishra V, Dai X, Smith KR, *et al.* Maternal exposure to biomass smoke and reduced birth weight in Zimbabwe. *Ann Epidemiol* 2004; 14: 740–747.
26 Dionisio KL, Arku RE, Hughes AF, *et al.* Air pollution in Accra neighborhoods: spatial, socioeconomic, and temporal patterns. *Environ Sci Technol* 2010; 44: 2270–2276.
27 Romieu I, Riojas-Rodriguez H, Marron-Mares AT, *et al.* Improved biomass stove intervention in rural mexico: impact on the respiratory health of women. *Am J Respir Crit Care Med* 2009; 180: 649–656.
28 Matheson MC, Benke G, Raven J, *et al.* Biological dust exposure in the workplace is a risk factor for chronic obstructive pulmonary disease. *Thorax* 2005; 60: 645–651.
29 Peiris-John RJ, Ruberu DK, Wickremasinghe AR, *et al.* Low-level exposure to organophosphate pesticides leads to restrictive lung dysfunction. *Respir Med* 2005; 99: 1319–1324.
30 McKeever TM, Scrivener S, Broadfield E, *et al.* Prospective study of diet and decline in lung function in a general population. *Am J Respir Crit Care Med* 2002; 165: 1299–1303.