The health effects of indoor air pollution exposure in developing countries

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by

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PREFACE

The provision of air that is safe to breathe is just as important as safe water or food. Yet many millions of people, predominantly women and children in the poorest developing countries, are obliged to breathe air that is heavily polluted with biomass emission products.

Air pollution in general and indoor air pollution in particular have been associated in many people's minds with industrialization and urbanization and thus with the cities of developed countries. The WHO Air Monitoring Information System (AMIS) has demonstrated, however, that the worst ambient conditions reported today exist in the cities of developing countries. Similarly, although most studies of indoor air quality have been carried out in developed-country buildings, the greatest indoor concentrations of and exposures to many important pollutants are found in both rural and urban households of developing countries. On a global basis, it is estimated that approximately half of the world's households cook daily with unprocessed solid fuels, i.e. biofuels (such as wood, crop residues and dung) or coal. A significant proportion of this activity takes place in conditions where much of the airborne effluent is released into the living area. Although ventilation rates are often relatively high, emission factors for such fuels are so great that indoor concentrations and exposures can still be significant. Compared with gas stoves, for example, even stoves using one of the cleaner biofuels - wood - typically release 50 times more particulate matter, carbon monoxide and hydrocarbons in cooking an equivalent meal. Taking into account other biofuels such as dung or crop residues, the resulting human exposures exceed recommended World Health Organisation levels by factors of 10, 20 or more.

Exposure to biomass smoke is a significant cause of health problems such as acute respiratory infections (ARI) in children, chronic obstructive lung diseases (such as chronic bronchitis and asthma), lung cancer and pregnancy-related outcomes. Global estimates show that about 2.5 million deaths each year result from indoor exposures to particulate matter in rural and urban areas in developing countries, representing 4-5% of the 50-60 million global deaths that occur annually.

The document presented here is the extended version of a paper published by the same authors in the Bulletin of the World Health Organization (*Indoor air pollution in developing countries: a major environmental and public health challenge*, 2000, 78(9)). From January to May 2002, the English and Spanish versions of this article were downloaded 12 000 times from the WHO web site, making it the most popular Bulletin article during this period. In response to the considerable demand, we have decided to publish the full, slightly updated version of the original article. The objective is to provide the interested reader with a literature summary of the major health effects caused by indoor air pollution resulting from the combustion of biomass for cooking and heating purposes with a focus on the situation in developing countries.

This document is a contribution by WHO's Department for the Protection of the Human Environment and WHO's Department for Child and Adolescent Health and Development to the *WHO Strategy on Air Quality and Health*. This approach puts emphasis on vulnerable groups, such as children and the poor, and the types of exposure that have the largest impacts on health.

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SUMMARY

Background

Around half of the world's population, some 3 billion people, rely on biomass fuels such as wood, animal dung, and crop residues, and coal for domestic energy needs. Over the past 25 years, transition to cleaner fuels among the poor has slowed dramatically and there is evidence that reliance on biomass is increasing in some parts of the world. Typically burnt in open fires or poorly functioning stoves, the use of these fuels leads to very high levels of indoor air pollution. Smoke exposure affects mainly women and young children who accompany their mothers during cooking and other households activities. Studies from several countries report average particulate levels that exceed United States Environmental Protection Agency standards 20 times or more.

Scope

This review examines the evidence that indoor air pollution increases the risk of a range of health problems. It does not attempt to describe current work seeking to quantify the global burden of disease arising from this exposure.

Summary of findings

Studies of the health effects of biomass smoke exposure in developing countries are being reviewed, but where their availability is limited, studies of wood smoke, environmental tobacco smoke (ETS) and ambient (outdoor) pollution from developed countries are also included. There is now consistent evidence that biomass smoke exposure increases the risk of childhood acute respiratory infections, particularly pneumonia, and probably otitis media. An association between smoke exposure with chronic bronchitis (assessed by symptoms) and chronic obstructive pulmonary disease (assessed clinically and by spirometry) is well established, in particular among women. In line with findings for ETS, there is emerging evidence that exposure during pregnancy reduces birth weight, possibly mediated through carbon monoxide. Infant and perinatal mortality may also be increased. Furthermore, biomass smoke exposure is likely to exacerbate asthma, although the evidence is limited and conflicting. A number of studies have also shown evidence of an increased risk of tuberculosis. Finally, human and animal studies suggest an increased risk of cataract, which is supported by evidence from studies on ETS.

Limitations of study methods

All of the reported studies are observational and very few measure exposure directly, instead relying on proxies such as fuel type, stove type or reported time spent near the fire. Furthermore, a substantial proportion does not adequately deal with confounding. Thus, despite mounting evidence that biomass smoke exposure increases the risk of a range of diseases with important implications for public health, these methodological limitations imply that risk estimates are poorly quantified and may be subject to bias.

Need for a household energy perspective

The risks associated with indoor air pollution need to be put into the context of other health problems in relation to household energy use. These include accidents such as burns and kerosene ingestion, the consequences of local and global environmental damage, the implications for poverty and income generation, and the opportunity costs attributable to time spent collecting fuel. Efforts to understand and alleviate the health effects of indoor air pollution should therefore adopt a holistic approach.

Conclusions

Indoor air pollution is a major environmental and public health hazard for many of the world's poorest, most vulnerable people. However, current evidence is based on a limited number of studies, few of which have measured smoke exposure directly. It is important to strengthen both the amount and quality of this evidence to support advocacy, to plan prevention programmes for specific settings, to make an economic case for potential interventions, and to motivate the health sector to contribute more actively to multi-sectoral action.

Further research should pay particular attention to the valid quantification of exposure and confounding. There are a number of health problems for which the available evidence is very limited or inconsistent. For these, well-conducted observational studies represent the next step. Efforts should also be made to strengthen emerging exposure-response relationships, particularly for common and serious health outcomes such as acute lower respiratory infections. Intervention studies, while complex, would provide the strongest evidence on health risks, although case control or cohort studies would also be useful under conditions where exposure levels and confounding factors are not strongly associated. Further work is also required to develop practical, robust and valid methods for measuring exposure levels and patterns for both field studies of health risk and the evaluation of interventions.

INTRODUCTION – INDOOR AIR POLLUTION EXPOSURE IN DEVELOPING COUNTRIES

Indoor air pollution can be traced to prehistoric times when humans first moved to temperate climates approximately 200,000 years ago. These cold climates necessitated the construction of shelters and the use of fire indoors for cooking, warmth and light. Ironically, fire, which allowed humans to enjoy the benefits of living indoors, resulted in exposure to high levels of pollution as evidenced by the soot found in prehistoric caves (Albalak, 1997). It has been estimated that approximately half the world's population, and up to 90% of rural households in developing countries, still rely on biomass fuels (WRI, 1999). Typically burnt indoors in open fires or poorly functioning stoves, this leads to levels of air pollution that are among the highest ever measured. Figure 1 shows a typical kitchen in a rural developing country setting, with blackened timbers and roof.

Figure 1:
A rural home in Kwa-Zulu Natal

In developed countries, modernization has without exception been accompanied by a shift from biofuel to petroleum products (kerosene, LPG) and electricity. In developing countries, even where cleaner more sophisticated fuels are available, households often continue to use biomass (Smith, 1987). Although the portion of global energy derived from biofuel has fallen from 50% in 1900 to around 13% currently, this trend has leveled and there is evidence that biofuel use is increasing among the poor (WRI, 1999). Poverty is one of the main barriers to the adoption of cleaner fuels and slow pace of development in many countries implies that biofuels will continue to be used by the poor for many decades.

Despite the magnitude of the problem of indoor air pollution, the health impact of this environmental exposure has been relatively neglected by research, donors and policy makers.

We begin by discussing the extent of the indoor air pollution problem in developing countries, then review the evidence on health risks, the global health impact and the prospects for interventions to reduce exposure. We conclude by considering the implications for research and policy.

Sources and uses of biomass fuels

Biomass fuel, or biofuel, refers to any plant or animal based material deliberately burned by humans. Wood is the most common biofuel, but use of animal dung and crop residues is also widespread (De Koning et al., 1985). Some countries, including China and South Africa also use coal extensively for domestic needs.

The types of fuels used typically increase in cleanliness, convenience, efficiency and cost as people move up what has been termed the “energy ladder” - see Figure 2 (Smith et al., 1994).

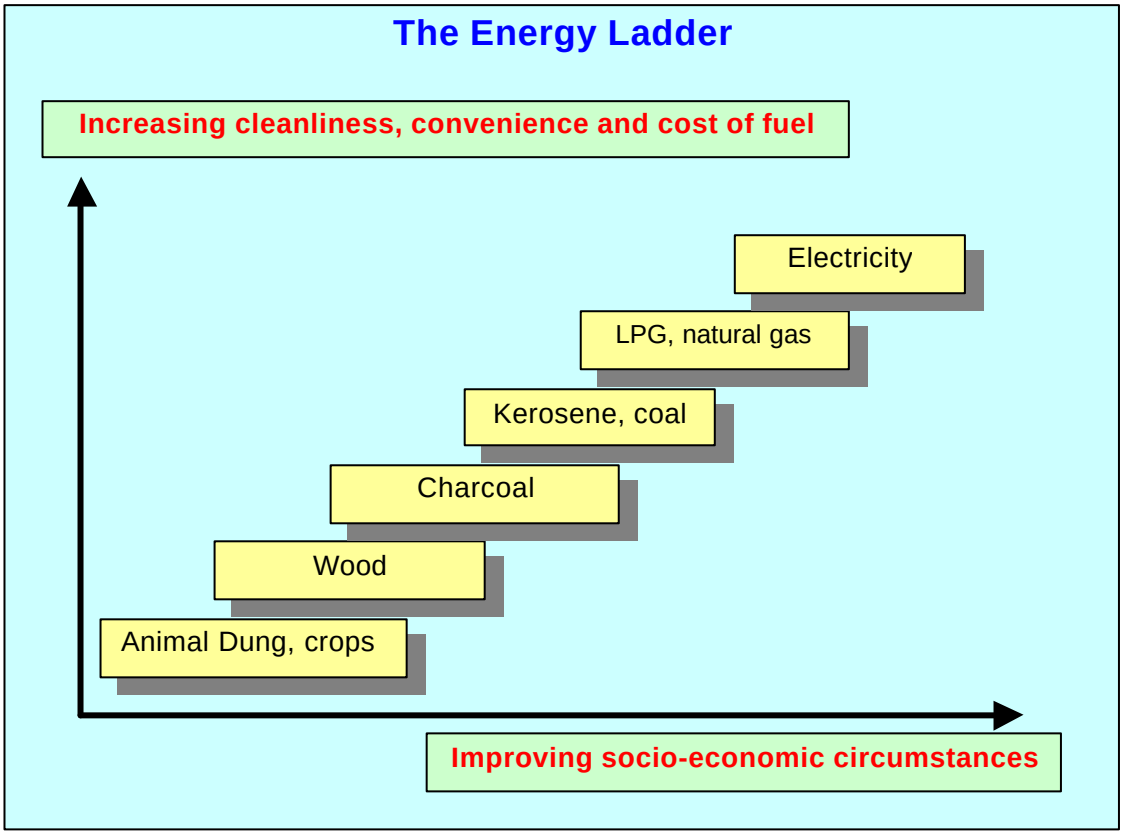

Figure 2: The Energy Ladder (adapted from Smith et al., 1994)

Animal dung is on the lowest rung of the ladder progressing to crop residues, wood, charcoal, kerosene, gas, and finally electricity. People generally move up the ladder as socio-economic conditions improve. Other sources of indoor air pollution in developing countries include smoke entering the home from nearby houses (Smith et al., 1994), burning of forests, agricultural land and household waste, the use of kerosene lamps (McCracken & Smith, 1998), and industrial and vehicle pollution. Environmental tobacco smoke (ETS), is another source of indoor air pollution exposures which can be expected to increase in importance in developing countries. It is important also to recognize that the open hearth and resulting smoke often have considerable cultural and practical value in the home, including control of insects lighting, drying food, fuel and housing materials and for flavouring foods (Smith, 1987).

Pollutant emissions and pollution exposure

Biomass smoke contains many thousands of substances, many of which damage human health. Most important are particulates, carbon monoxide, nitrous oxides, sulphur oxides (more with coal), formaldehyde, and polycyclic organic matter which includes carcinogens such as benzo[a]pyrene (DeKoning et al., 1985). Small particles of diameter less than 10 microns (termed PM_{10}), and in particular those less than 2.5 microns ($PM_{2.5}$), are able to penetrate deep into the lungs and appear to have the greatest health-damaging potential (USEPA, 1997). Table 1 summarizes the most important health damaging pollutants in biomass and coal smoke, the mechanisms involved and potential health consequences. These mechanisms are discussed further in the next section as the evidence for health effects is reviewed, although in general there is a scarcity of studies on the toxicology of particulate matter and particularly that arising from biomass combustion

Table 1: Mechanisms by which some key pollutants in smoke from domestic sources may increase risk of respiratory and other health problems

Pollutant	Mechanism	Potential health effects
Particulate matter: small particles less than 10 microns, and particularly those less than 2.5 microns aerodynamic diameter	• Acute: bronchial irritation, inflammation and increased reactivity • Reduced muco-ciliary clearance • Reduced macrophage response and (?) reduced local immunity • (?) Fibrotic reaction • Autonomic imbalance, pro-coagulant activity, oxidative stress	• Wheezing, exacerbation of asthma • Respiratory infections • Chronic bronchitis and COPD • Exacerbation of COPD • Excess mortality, including from cardiovascular disease
Carbon Monoxide	• Binding with Haemoglobin (Hb) to produce COHb which reduced O2 delivery to key organs and the developing fetus.	• Low birth weight (fetal COHb 2-10%, or higher) • Increase in perinatal deaths
Benzo[a]pyrene	• Carcinogenic (one of a number of carcinogenic substances in coal and biomass smoke)	• Lung cancer • Cancer of mouth, nasopharynx, and larynx
Formaldehyde	• Nasopharyngeal and airways irritation • (?) Increased allergic sensitisation	• (?) increased susceptibility to infections • (?) May lead to asthma
Nitrogen dioxide	• Acute exposure increases bronchial reactivity • Longer term exposure increases susceptibility to bacterial and viral lung infections	• Wheezing and exacerbation of asthma • Respiratory infections • Reduced lung function (children)
Sulphur dioxide	• Acute exposure increases bronchial reactivity • Longer term: difficult to dissociate from particulate effects	• Wheezing and exacerbation of asthma • Exacerbation of COPD, CVD
Biomass smoke (component uncertain)	• Absorbtion of toxins into lens, leading to oxidative changes	• Cataract

In most of these stoves, combustion is very incomplete and results in high emissions which combine with often poor ventilation to produce very high levels of indoor pollution . The most recent review of these pollution levels is included in the revised WHO Air Quality Guidelines (WHO, 1999). Appendix Table A summarizes additional studies either not included in the WHO review or published later, and shows concentrations for the most commonly measured pollutants: total suspended particulate matter (TSP), PM_{10}, $PM_{2.5}$, CO and benzo[a]pyrene. Many early studies measured TSP, as it was not appreciated that the smaller particles were more

important. Although these findings are not always comparable because of different measurement protocols and environmental conditions, they uniformly reveal exceedingly high concentrations. For comparison, the US Environmental Protection Agency's (USEPA) 99th percentile values for 24-hour average PM_{10} and $PM_{2.5}$ concentrations are 150 $\mu g/m^3$ and 65 $\mu g/m^3$, respectively. The USEPA 8-hour average CO standard is 9 ppm or 10 mg/m^3. Most US cities rarely exceed these PM standards whereas in rural homes in developing countries, these levels are exceeded on a daily basis by a factor of ten, twenty and sometimes more. It should be noted that the revised WHO air quality guidelines do not quote values for PM_{10}, because there is growing evidence that there is no safe lower limit of exposure. Accordingly, exposure-response data for mortality and morbidity outcomes are presented.

Table 2 summarizes typical values of PM_{10} and CO in developing country homes using simple stoves and compares these with WHO and United States Environmental Protection Agency's (USEPA) air quality guidelines. Urban and peri-urban settings in developing countries often demonstrate high indoor levels from domestic fuels use including biomass, together with ambient (outdoor) pollution levels from a variety of domestic, industrial and transport sources which are far higher than those experienced in developed countries.

Table 2: Comparison of typical levels of PM_{10} and CO in developing country homes with WHO and USEPA guidelines

Pollutant	**Range of ambient levels in LDC studies, for simple stoves**		**WHO guidelines** (WHO, 1999)		**US EPA guidelines** (USEPA. 1997)	
	Period	**Level**	**Period**	**Level**	**Period**	**Level**
Particles (PM_{10}) ($\mu g/m^3$)	**Annual**	Not available, but expect similar to 24 hr	**Annual**	Guidelines presented as exposure-response relationships. Levels as low as 10 $\mu g/m^3$ associated with excess risk.	**Annual**	50
	24 hour	300 - 3,000+	**24 hour**		**24 hour**	150 (99th per-centile)
	During use of stove	300 - 20,000+ some 30,000+				
Carbon Mon-oxide (ppm)	**24 hour**	2 - 50+	**8 hour**	10	**8 hour**	9
	During stove use	10 - 500+	**1 hour**	30	**1 hour**	35
			15 mins	100		
	COHb (%)	1.5 - 13%	**COHb (%)**	Critical level < 2.5% Typical non-smoker 0.5 - 1.5%. Typical smoker 10%		
	During use of stove	1000+				

Health effects are determined not just by the pollution level, but more importantly by the time people spend breathing polluted air – in other words the exposure level[a]. Exposure refers to the concentration of pollution in the immediate breathing environment over a specified time interval. This can be measured either directly through personal monitoring or alternatively indirectly by combining information on pollutant concentrations in each microenvironment where people spend time with information on activity patterns (Lioy, 1990). Information on activity patterns is very important for understanding the dynamic relationship between levels of pollution and behaviour. Thus, although poorly studied to date, it is quite possible that as pollution levels are reduced, people spend more time indoors or nearer the pollution source so reducing ambient pollution will not necessarily result in a proportionate decrease in exposure - a situation which has important implications for interventions.

In developing countries, individuals are typically exposed to these very high levels of pollution for between 3 and 7 hours each day over many years (Engel et al., 1998). During winter in the many cold and mountainous areas, exposure may occur over a substantial portion of each 24 hour period (Norboo et al., 1991a & b). Cultural practices common in developing countries may promote exposure of infants, women, the elderly and the sick. Since it is the women who generally cook, their exposure is much higher than men's (Behera et al., 1988). Young children are often carried on their mother's back while she is cooking, so that from early infancy, children spend many hours breathing smoke (Albalak, 1997).

Although, as shown in Appendix Table A, there are a fair number of studies that have measured pollution and personal exposure, almost none of the published studies examining health risks have done so. As will be discussed in the next section, this is one of the principal limitations of the available evidence on the health impact of indoor air pollution exposure in developing countries.

HEALTH EFFECTS - INTRODUCTION

Context

In the first section of this report we have seen how very large numbers of already vulnerable women and young children are exposed each day to levels of air pollution well in excess of WHO and EPA air quality guidelines. These guidelines are based on extensive research into the health effects of pollution, albeit in developed countries. As a result, there can be little doubt that this exposure to indoor air pollution in developing countries presents a major public health threat for many poor urban and rural communities in Asia, Africa and the Americas, and demands concerted action to reduce exposure. It is reasonable therefore, to ask why we should invest further time and resources in studying the evidence for associations between specific disease problems and IAP exposure.

We believe that there are a number of compelling reasons why this evidence and efforts to strengthen it are still very important, although because of the overwhelming evidence of high levels of exposure, this should be carried out in parallel with (and not delay) the development and implementation of interventions to reduce exposure (Bruce, 1999):

- More solid evidence that IAP exposure substantially increases the risk of a number of serious and common disease conditions would provide a powerful and much-needed advocacy tool. Evidence that lowering exposure reduces the incidence of diseases such as childhood pneumonia would provide valuable additional impact.

[a] Strictly, it is the dose that determines the health effect. In practice, this is a complex issue and difficult to assess, and will not be addressed further in this review.

- Health systems, which have responsibility for the control and prevention of these conditions, need reliable information to plan preventive programmes for specific conditions.
- Such evidence will provide the basis for impact (burden) and economic analyses that can contribute to the assessment of relative cost-effectiveness of household energy and related interventions capable of reducing exposure. Economic arguments are having a growing influence on the allocation of resources and the absence of this information may deny such interventions the attention and resources they deserve.
- Reliable evidence, particularly on the effect of interventions, will help encourage the health sector to put its weight behind the multi-sectoral initiatives required to address health problems associated with poor home environments.

Sources of evidence

In contrast to the situation for developed country populations, there is currently relatively little published evidence on the health effects of air pollution, both outdoor and indoor in developing countries. Although the emphasis for this review is on biofuel exposure is LDC populations, where published evidence is particularly limited, information from relevant exposures to outdoor and indoor air pollution, and to environmental tobacco smoke (ETS) - which is a form of biomass air pollution - has been included. The review will begin with respiratory illness, and then consider cancer, perinatal outcomes and birth weight, and eye disease.

Childhood acute respiratory infections (ARI)

Acute lower respiratory infections

Acute lower respiratory infections (ALRI) remain the single most important cause of death globally in children under 5 years and account for around 2 million deaths annually in this age group. There are some sixteen studies in LDCs which have reported on the association between IAP exposure and ARLI (Kossove, 1982; Pandey et al., 1989; Campbell et al., 1989; Cerquiero et al., 1990; Collings et al., 1990; Armstrong & Campbell, 1991, Mtango et al., 1992, Johnson & Aderele, 1992; de Francisco et al., 1993; Shah et al., 1994; Victora et al., 1994; Depmsey et al., 1996, Wesley & Loening, 1996, Lopez-Bravo et al., 1997, Ezzati and Kammen 2001a,b) and two further studies among Navajo Indians in the US (Morris et al., 1990; Robin et al., 1996). Comment will be restricted to the studies listed as these have all used definitions of ALRI which conform reasonably closely to current WHO criteria (or other definitions that were accepted at the time the study was carried out) and/or include radiographic evidence. A detailed review of this topic has recently been published (Smith et al., 2000) and a summary is presented here. The study by Ezzati and Kammen was published after the review by Smith et al, and is described in more detail below.

Of the sixteeen LDC studies, 10 are case-control designs (two mortality studies), 5 cohort studies (all morbidity), and one a case-fatality study. In contrast to the relatively robust definitions of ALRI, the measurement of exposure has relied in almost all studies on proxies, including the type of fuel and stove (Cerqueiro et al., 1990; Collings et al., 1990; Johnson & Aderele, 1992; Mtango et al., 1992; Victora et al., 1994; Shah et al, 1994; O'Dempsey et al., 1996, Wesley & Loening, 1996; Lopez-Bravo et al., 1997), whether the child stays in the smoke during cooking (Kossove, 1982, Mtango et al., 1992, Victora et al., 1994), reported hours spent near stove (Kossove, 1982; Pandey et al., 1989), and whether the child is carried on the mother's back during cooking (Campbell et al., 1989; Armstrong & Campbell, 1991; de Francisco et al., 1993). Apart from Ezzati's study (see below), only one previous study made direct measurements of pollution (particulates) and exposure (COHb) in a subsample (Collings et al., 1990). In that study, respirable particulates in the kitchens of cases were substantially higher than for controls (1998 $\mu g/m^3$ vs. 546 $\mu g/m^3$; $p<0.01$), but there was no significant difference in COHb levels.

Five studies reported no significant association between ALRI incidence and exposure (Johnson & Aderele, 1992; Victora et al., 1994; Shah et al., 1994; Wesley & Loening, 1996, Lopez-Bravo et al., 1997), but the remainder reported significantly elevated ORs (for incidence or deaths) in the range 2-8. Not all however, have dealt adequately with confounding factors (Pandey te al., 1989; Kossove, 1982; Cerquiero et al., 1990; Collings et al, 1990; Johnson & Aderele, 1992), although accounting for confounding in studies of this exposure may in any case be problematic (Armstrong & Campbell, 1991; Bruce et al., 1998). In several studies finding no association, relatively small proportions of the samples were exposed. Thus, in urban Brazil only 6% of children were exposed to indoor smoke (Victora et al., 1994) and in another south American study 97% of homes used gas for cooking, although 81% used polluting fuels (kerosene, wood, coal) for heating (Lopez-Bravo et al., 1997). This study also excluded neonates with birth weight <2,500 gms – the group most vulnerable to ALRI. In the study from Durban, only 19% of cases and 14% of controls used wood or coal stoves (Wesley & Loening, 1996). In the study reported by Shah, a so-called 'smokeless chullah' was used as an indicator of lower exposure, but such stoves can be little better than traditional ones (Smith, 1989).

The most recent report on this topic, by Ezzati and Kammen, describes a cohort study of 345 rural Kenyan people (of which 93 were aged less than 5 years), living in 55 homes on a rural cattle ranch (Ezzati and Kammen 2001a; Ezzati and Kammen 2001b). Households used mainly wood or charcoal, in open fires and improved (chimneyless) stoves. Detailed personal exposure assessment was combined with weekly (initially bi-weekly) health outcomes assessment for adults and children using WHO criteria for ALRI. This is the first study that has reported (and presented) exposure-response relationships for particulates and incidence of ALRI, in children (less than 5 years) and adults. The trend of increasing risk with higher levels of exposure was highly significant. Socio-economic status and birth weight were not adjusted for, but the authors reported that income, housing and nutrition varied little due to the social organisation of the ranch community. The incidence of ALRI in young children in this study was considerably higher than previously reported in similar populations.

The Navajo studies in North America used case-control designs, reported fuel type (wood vs. cleaner) as a proxy for exposure and adjusted for confounding (Morris et al., 1990; Robin et al., 1996). Both reported elevated ORs of approximately 5, although non-significant in one (Robin et al., 1996). This latter study also carried out 15 hour PM_{10} measurements, but found minimal differences between cases and controls, while the actual levels (median 15 hr PM_{10} 22.4 $\mu g/m^3$, range 3.2 - 186.5) were relatively low. However, children living in homes with $PM_{10} \geq 65$ $\mu g/m^3$ had an OR 7.0 (95% CI 0.9-56.9) times that for children with levels < 65 $\mu g/m^3$ (Robin et al., 1996).

Upper respiratory infection, and otitis media

Several studies have reported an association between biofuel smoke exposure and general acute respiratory illness in children, mostly upper respiratory illness (URI). We will focus on middle ear infection (otitis media), which although rarely fatal causes a great deal of morbidity, (including deafness) and considerable demands on the health system. Untreated, it may progress to mastoiditis. Evidence from LDCs is very limited, but there is good reason to expect an association. There is now strong evidence that ETS exposure causes middle ear disease: a recent meta-analysis reported an OR of 1.48 (1.08-2.04) for recurrent otitis media if either parent smoked, and 1.38 (1.23-1.55) for middle-ear effusion (Strachan et al., 1998a). A clinic-based case-control study of children in rural New York State, reported an adjusted OR for otitis media (two or more separate episodes) of 1.73 (1.03-2.89) for exposure to woodburning stoves (Daigler et al., 1991).

Chronic pulmonary disease

Chronic Obstructive Pulmonary Disease (COPD)

In developed countries, smoking explains most cases of (over 80%) chronic bronchitis (CB – inflammation of the lining of the bronchial tubes) emphysema (over-inflation and destruction of the air sacs in the lungs) and chronic obstructive pulmonary disease (COPD – progressive and incompletely reversible airflow obstruction). It has been recognised for some time that these diseases occur in regions were smoking is infrequent. Several early studies described patients with chronic lung disease in communities heavily exposed to indoor biofuel pollution in New Guinea. Anderson (1979a) found in adults older than 45, a high prevalence of respiratory symptoms and disease, similar in men and in women, and that 20% of men and 10% of women had airflow obstruction defined as an FEV_1/FVC lower than 60 percent. The clinical presentation was as COPD with a few patients having focal lung fibrosis and bronchiectasis (permanent damage and dilatation of bronchii with recurrent infection) and disease was attributed to IAP and repeated infections (Anderson, 1979b). Most cases were smokers of home grown tobacco, inhaled in a similar way to cigars, but no association with smoking was found for airflow obstruction or mortality (Anderson et al., 1988).

More than twenty studies, including cross-sectional and case control designs, have reported on the association between exposure to biomass smoke and CB or COPD (Appendix Table B) (Padmavati & Pathak, 1959; Master, 1974; Anderson, 1976; Anderson, 1979b; Pandey, 1984a; Pandey, 1984b; alik, 1985; Pandey et al, 1988; Behera et al., 1991; Norboo et al., 1991a; Perez-Padilla et al., 1993; Dossing et al., 1994; Behera et al., 1994; Qureshi, 1994; Menezes et al, 1994; Regalado et al., 1996; Dennis et al., 1996; Dutt et al., 1996; Ellegard, 1996; Albalak et al., 1999). In Nepal, the prevalence of CB was similar in men and women (18.9%) which is unexpected if cigarette smoking, more common in men, were the main cause (Pandey, 1984a, Pandey, 1984b). Prevalence of CB was also considerable in women in Ladakh, where few women smoke (Norboo et al., 1991a), also in Pakistan (Qureshi, 1994). Biomass exposure has been reported to be more frequent in people with airflow obstruction in hospital based case-control studies (Dennis et al., 1996, Perez-Padilla et al., 1993) but also in some community studies (Pandey et al., 1985; Regalado et al., 1996; Behera et al., 1994). In hospital based studies, obstruction was often severe and the association with exposure strong (odds ratios in range 1.8 to 9.7, one reporting 49). In community studies however, the reported differences in lung function associated with wood smoke exposure have usually been relatively small (Appendix Table B). A study from rural Mexico found biomass use was associated with a 4% decrease in FEV1/FVC, while an increase in the kitchen particulate concentration of 1,000 $\mu g/m^3$ was associated with a reduction of 2% in FEV_1 (Regalado et al., 1996). In India, cases using biomass had lower FVC than those using kerosene, gas and mixed fuels (Behera et al., 1994). Pandey reported an exposure-response relationship with FEV_1 and FVC decreasing as the reported hours of exposure increased, although non-significant in non-smokers (Pandey et al., 1985). Based on experience in cigarette smokers, it is likely that only a small susceptible population, less than 15% of those with long-term exposure to wood smoke, develop clinically significant COPD (a higher proportion develop CB), although this may depend on the level of exposure. Indoor pollution is considered now a risk factor for COPD by the Global Strategy for the Diagnosis Management, and Prevention of Chronic Obstructive Lung Disease (Global Initiative for Chronic Obstructive Lung Disease, 2001).

Exposure was usually estimated from questionnaires as present or absent, as hours by the wood stove, or hours multiplied by years of exposure. Few studies measured kicthen particulate levels (Regalado et al., 1996, Ellegard, 1996, Albalak, 1999), but those doing so confirmed very high concentrations. This was combined with time-budget assessment in one study (Albalak et al., 1999). Norboo et al (1991a) reported the use of kitchen and personal (exhaled) CO levels, but otherwise personal measurements of exposure have not been used to our knowledge. Chronic

bronchitis has generally been determined by questionnaire, with spirometry for airflow obstruction and COPD, but for many of the studies there is little account of quality control.

Clinical characteristics of lung disease

The most common presentation in both community and referral hospital studies is chronic airways disease, specially CB. Airflow obstruction and shortness of breath (dyspnoea) are hallmarks of patients seen in referral hospitals (Dossing et al., 1994; Moran, 1992). Chronic respiratory failure may ensue in patients with severe airflow obstruction with pulmonary hypertension or cor pulmonale (Pandey, 1984a). In a review of 29 patients with CB exposed to wood smoke, 20 had electrocardiographic or radiographic signs of pulmonary hypertension (Moran, 1992). Lung function of patients presenting to referral hospitals may have changes similar to smokers, ranging from normal to severe airflow obstruction. Some patients have classical characteristics of emphysema (Pandey, 1984a; Moran, 1992) but restrictive changes have also been reported (Appendix Table C). In a referral hospital study in Mexico, no significant differences in lung function, clinical symptoms or radiographic features were found between patients with CB exposed to biomass smoke compared with tobacco smokers (Moran, 1992), however, in patients with COPD due to biomass smoke exposure, bullous emphysema is very uncommon.

Experimental evidence and pathogenesis

Acute massive exposure to wood smoke, as in forest fires, can be rapidly lethal. Besides asphyxia and carbon monoxide intoxication, severe damage to the respiratory epithelium with airway and pulmonary oedema can result. Lesser degrees of wood smoke exposure in guinea pigs produces broncho-constriction and increases the response to a subsequent exposure (Hsu et al., 1998). Exposure to wood smoke has also produced chronic lung disease in experimental animals: after exposure to wood smoke for 3 hours a day for 3 months, Guinea pigs developed mild emphysema (Juarez-Ceron, 1996). Rats exposed intermittently to wood smoke for 75 minutes daily for 15 days had mononuclear bronchiolitis and mild emphysema, more severe in animals exposed for 30 and 45 days (Lal et al., 1993). A fibrotic lung reaction simulating silicosis has also been produced experimentally in animals exposed to wood smoke (Restrepo et al., 1983).

Although the mechanisms by which smoke causes emphysema and airways disease are uncertain, there are some clues. Oxidative stress may be a component, as oxidizing radicals are present in tobacco and biomass smoke and also released by inflammatory cells (Repine et al., 1997). Risk factors for COPD associated with tobacco smoking include bronchial hyperreactivity (Villars et al., 1995), atopy (Gottlieb, 1996) and genetic susceptibility (Silverman, 1998), all of which could apply to biomass smoke exposure. Predisposition to COPD later in life may result from impaired lung growth in infancy resulting in reduced adult lung function. Exposure to smoke during pregnancy and infancy, either tobacco or biomass, may therefore increase the risk of COPD.

One consistent finding in the patients exposed to biomass is substantial deposition of carbon in the lung (anthracosis), Appendix Table C. In 18 necropsies of women with cor pulmonale who never smoked but most of whom were exposed to biomass smoke, all had emphysema, 11 bronchiectasis, 5 CB and 2 tuberculosis (Padmavati & Joshi, 1964). Several studies have described lung fibrosis which resembles pneumoconiosis (a chronic reaction of the lung to dust inhalation, usually involving fibrosis) (Appendix Table C), including cases with progressive massive fibrosis among in subjects exposed to wood smoke. Exposure to inorganic or organic dusts may coexist in these patients, but evidence of bronchial disease is present and in most cases predominates. Non-occupational silicosis has also been reported in developing countries, and attributed to sand storms, but these subjects are often also exposed to biomass smoke (Norboo et al., 1991a; Norboo et al., 1991b).

There is some evidence also that wood smoke exposure may be associated with interstitial lung disease (inflammation of the lung structure leading to fibrosis) in developed countries (Appendix Table C). In a small case control study, Scott found patients with cryptogenic fibrosing alveolitis were more likely to have lived in a house heated by a wood fire (Scott et al., 1990).

Asthma

International variations in asthma prevalence (ISAAC, 1998), together with recent increases in many countries, has focused attention on the role of air pollution. Asthma in poor rural communities in LDCs has been little studied, and the relationship between asthma and environment still less. The influence of air pollution on the development of asthma is complex and still controversial. While some assert that air pollution (including ETS) may be one factor sensitising genetically susceptible individuals during early life to allergens (Bjorksten, 1999), a recent systematic review does not support this view for ETS. There is however more consistent evidence that air pollution and ETS do trigger asthma in sensitised individuals (Strachan et al., 1998b; Bjorksten, 1999).

Studies of biomass smoke and asthma in developing countries in both children and adults have yielded mixed findings (Appendix Table D). A questionnaire survey of 9-12 year olds in Turkey, which included spirometry, found coal users had more day/night cough ($p<0.05$) and those using wood-burning stoves had the lowest values of FVC, FEV1, PEFR and FEF_{25-75} (Guneser et al., 1994), but adjustment for confounding was not carried out. A matched case-control study of 11-17 year olds in rural Nepal using ISAAC (International Study of Asthma and Allergies in Children) survey methods found an adjusted OR of 2.2 (1.2-4.8) for asthma among those using wood fires or stoves without flues, compared to those using stoves with flues, gas or kerosene (Melsom 2001). Findings of a cross-sectional study of 1058 4-6 year olds in rural Guatemala also using ISAAC methods found adjusted ORs of 1.81 (1.04-3.12) for ever wheeze, and 2.35 (1.08-5.13) for wheeze in the last 12 months (Schei 2002). In Jordan, a cross-sectional study of lung function in 7-13 year olds found significantly reduced FVC, FEV1, PEFR and FEF_{25-75} for exposure to wood/kerosene stoves and ETS, but no adjustment was made for confounding (Gharaibeh, 1996). A case-control study of school children in Nairobi also found increased exposure to wood smoke in asthmatics (Mohammed et al., 1995).

Several studies have reported no association, however. A case-control study of children aged 1 month to 5 years hospitalised with asthma in Kuala Lumpur found that kerosene or wood stove use was not independently associated with asthma though mosquito coil smoke was (Azizi et al., 1995). Noorhassim et al. (1995) also found no association between asthma and biomass smoke in a cross-sectional study of 1,000 children in Malaysia. A study in urban Maputo found no association after adjustment between fuel type and either wheeze or PEFR (Appendix Table B) (Ellergard, 1996). Qureshi (1994) found no association in rural Pakistan, although the number with asthma was small (Appendix Table B).

Evidence in adults comes from a large study of nearly 29,000 adults in rural China which reported adjusted ORs for wheezing and asthma for the group with *occupational* exposure to wood/hay smoke of 1.36 (1.14-1.61) and 1.27 (1.02-1.58), respectively (Xu et al., 1996). Since 93% of the sample used wood/hay for cooking, the relationship with asthma was studied among the 39% of women and 21% of men exposed occupationally. Similarly elevated ORs were reported for those using coal for cooking.

Mixed findings have also been reported from developed countries, with several studies reporting positive associations (Pistelly, 1997), and some finding no association – for example among 5-9 year old Seattle children (Maier et al., 1997). There is evidence of biomass smoke being

associated with a lower risk, reflecting a possible protective effect. Von Mutius et al. (1996) found the risk of hay fever, atopy and bronchial reactivity to be lower in rural German children aged 9-11 years whose homes were heated by coal or wood. Similar evidence has been reported from urban Australia (Volkmer et al., 1995).

Cancer

Lung cancer

Tobacco smoke is the most important risk for lung cancer and explains most cases in developed countries. However in developing countries, non smokers, frequently women, form a much larger proportion of patients with lung cancer. Thus, around two thirds of women with lung cancer from China (Gao, 1996), Mexico (Medina et al., 1996) and India have been found to be non-smokers (Gupta RC et al., 1998). In a review of studies from China, odds ratios for lung cancer among women exposed to coal smoke at home, particularly so-called “smokey” coal, were in the range 2-6 (Smith &Liu, 1993).

No association has been reported so far between lung cancer and wood smoke exposure (Smith &Liu , 1993) but there is little evidence available. Rates of lung cancer from rural areas where exposure to wood smoke is common do tend to be low. This could be due to a variety of factors associated with the rural environment, and it would be unwise at this stage to conclude that biomass smoke does not incease lung cancer risk – particularly since exposure to known carcinogens in biomass smoke is intense. For example, it has been estimated that in some homes, 3 hours of cooking per day exposes women to similar amounts of benzo[a]pyrene as smoking 2 packs of cigarettes daily (Smith & Liu, 1993). If exposure to all carcinogens in wood smoke parallels exposure to particulates, cooking with traditional biomass stoves is equivalent to smoking several cigarettes per day (Smith & Liu, 1993).

A history of previous lung disease has been found to be a risk factor for lung cancer in women (Wu et al., 1995). In developing countries, previous lung disease due to tuberculosis and other lung infections could contribute to lung cancer development in never smokers. In fact COPD is associated with an increase in cancer risk even taking into account age, sex, occupation and smoking (Samet et al., 1986). This suggests a parallel exposure to lung toxins and carcinogens, or else, that chronically inflammed or injured tissue is more prone to developing cancer. Whatever the mechanism, exposure to biomass smoke remains a potential risk factor for lung cancer.

Nasopharyngeal and laryngeal cancer

Biomass smoke has been implicated as a cause of nasopharyngeal carcinoma (Clifford, 1972) although this is not a consistent finding (Yu et al., 1985). A case-control study from Brazil found that oral cancer was associated to tobacco, alcohol and having a wood stove (Franco et al., 1989). Another case control study from S America of 784 cases of oral, pharyngeal and laryngeal cancer, reported an adjusted odds ratio of 2.68 (95% CI: 2.2-3.3) for exposure to wood smoke compared to cleaner fuels (Pintos et al., 1998). Significant associations were demonstrated separately for mouth, laryngeal and pharyngeal carcinomas, and the authors estimate that wood smoke exposure explains about one third of upper aero-digestive tract cancers in the region.

Pulmonary Tuberculosis

Analysis of data from 200,000 Indian adults found an association between self-reported TB and exposure to wood smoke at interview in the 1992-93 National Family Health Survey (Mishra et al., 1999). Persons living in households burning biomass reported TB more frequently compared to persons using cleaner fuels, with an OR to 2.58 (1.98-3.37) after adjustment for a range of

socio-economic factors. These findings are similar to those of a study in north India which reported an association between use of biofuel and TB defined by clinical measures (Gupta & Mathur, 1997), although adjustment was made only for age.

Additional evidence for this association with TB comes from a hospital based case-control study in Mexico (Perez-Padilla et al. 2001) which reported on 288 cases with active smear positive or culture positive TB and 545 controls with ear, nose and throat ailments and no evidence of chest disease studied concurrently. Exposure to wood smoke was assessed as yes or no, by asking about use of traditional wood stoves. The odds ratio for exposed subjects living in the Mexico Metropolitan area (the power for rural subjects in the study was too low), was 2.4 (95% CI: 1.04–5.6) adjusted for a range of socio-economic factors (including crowding, zone of residence).

An increase in risk of TB may result from reduced resistance to infection, as exposure to smoke interferes with mucociliary defences (Houtmeyers et al., 1999) and decreases antibacterial properties of lung macrophages. Tobacco smoke also decreases cellular immunity, antibody production and local bronchial immunity and has been associated with tuberculosis (Altet et al., 1996). Although such widespread immuno-suppression has not been reported with biomass smoke, an increase in risk of TB is quite feasible.

If confirmed, the public health implications of this association would be very substantial. Using prevalence data from the 1992-93 survey, Mishra et al. (1999) report that biomass smoke exposure can explain about 59 percent of rural and 23 percent of urban cases of TB in India (95% CI not given). Biomass smoke exposure may be an additional factor in the well-established relationship between poverty and TB, hitherto explained by malnutrition, overcrowding and less access to health care.

Low birth weight and infant mortality

A recently published study in rural Guatemala found that babies born to women using wood fuel, compared to those using gas and electricity, were 63 gm lighter (95% CI 0.35-125.6; p=0.049) after adjustment for socio-economic and maternal factors (Boy et al., 2002). Although we are not aware of any other similar reports, evidence from active smoking and ETS (Windham et al., 1999) strongly suggests this effect – quite possibly mediated through carbon monoxide - is likely to be real. Levels of CO in homes where biofuels are used are high enough. 24 hour means in the range 5-10 ppm, and 20-50 ppm or more during use of the fire (Dary et al, 1981; Norboo et al., 1991a&b, Naeher et al., 1996) and COHb levels of between 1.5-2.5% (Dary et al., 1981) to as high as 13% (Behera et al., 1988) have been reported. These levels of COHb are comparable with ETS exposure, and in some cases with active smoking (WHO, 1999).

There is also evidence linking ambient air pollution with reduced birth weight (Wang et al., 1997; Ritz & Yu, 1999; Bobak & Leon, 1999), although only one of these studies has specifically reported the association with CO (Ritz & Yu, 1999). In judging the likely public health impact of IAP through this effect on birth weight, it is important to recognise that exposure is greatest among poor women of childbearing age living in communities often with an already high prevalence of LBW.

Only one study has reported an association between perinatal mortality and IAP exposure in a developing country, with an OR of 1.5 (1.0-2.1) for still births following adjustment for a wide range of factors (Mavlankar et al, 1991). A univariate association with early neonatal deaths did not persist after adjustment. Supportive evidence comes from outdoor air pollution studies. A time series study in Mexico City examined the relationship between fine particulate matter and IMR (Loomis et al., 1998). The strongest effect was with $PM_{2.5}$ 3-5 days prior to death, where a 10 $\mu g/m^3$ increase was associated with a 6.9% (2.5-11.3) excess IMR. Analysis of US infant

mortality also showed an excess perinatal mortality associated with higher PM_{10} levels after adjustment: OR of 1.10 (1.04-1.16) for the high pollution group (mean 44.5 $\mu g/m^3$) vs. the low pollution group (mean 23.6 $\mu g/m^3$). In normal birthweight infants, high exposure was associated with respiratory mortality [OR 1.40 (1.05-1.85)] and SIDS [OR 1.26 (1.14-1.39)]. On the other hand, in an ecological study of pollution and stillbirths in the Czech Republic, no association was found between any measure of pollution (TSP, SO_2, NO_x) and stillbirths – despite the association with LBW reported above (Bobak & Leon, 1999).

Cataract

There is no question that biofuel pollution causes eye irritation (Ellegard, 1997), but there is also evidence that it may cause cataract. In a hospital-based case control study in Delhi, when compared to those using cow dung and wood, use of LPG was associated with an adjusted OR of 0.62 (0.4-0.98) for cortical, nuclear and mixed (but not posterior subcapsular) cataracts (Mohan et al., 1989). Animal studies have shown that wood smoke condensates do, like cigarette smoke, damage the lens of rats, causing discolouration, opacities and particulate debris - the mechanism is thought to involve absorbtion and accumulation of toxins which then lead to oxidation (Rao et al., 1995). The growing evidence that ETS causes cataracts is supportive (West, 1992).

A broader perspective on household energy and health

Indoor air pollution is one aspect of the supply, collection and use of household energy within the community and household environment, although from a health perspective it does appear to be the most important. There are however a number of other health consequences directly associated with household energy in developing countries including burns (Onuba & Udoidiok, 1987; Courtright et al., 1993) and kerosene ingestion (Yach, 1994; Gupta S et al., 1998). In addition, there are a range of issues encompassing the use of women's time, injuries associated with the collection and carrying of fuel (mainly wood), use of fires and smoke in the home (food preservation, control of insects), opportunities for small scale commercial activities, effects on the local and global environment and so on which can also impact - directly or indirectly - on health (WHO, 1992).

Although these health outcomes do not result from human exposure to IAP, efforts to control that exposure will impact on these other consequences. For example, an additional benefit of the transition from open fires to enclosed stoves or alternative fuels may be to reduce the incidence of serious burns. On the other hand, the growing use of kerosene for example in South Africa (Yach, 1994; Reed & Conradie, 1997), and India (Gupta S et al., 1998) may increase the burden of accidental poisoning, unless active measures are taken to prevent this (Yach, 1994; Ellis et al., 1994; Krug et al., 1994, Reed & Conradie, 1997).

As a result of these many inter-related household, development and environmental issues, it is helpful to consider both the health effects of IAP and approaches to alleviating exposure within an holistic view of household energy at community, national and indeed international levels.

DISCUSSION

The existing studies on indoor air pollution in developing countries, while providing important evidence of associations with a range of serious and common health problems, suffer from a number of methodological limitations, namely (a) the lack of detailed and systematic pollution exposure determination, (b) the fact that all studies to date have been observational and (c) that many have dealt inadequately with confounding.

Few studies have measured pollutant concentrations and even fewer have measured exposure either directly or indirectly. Most have used proxy measures such as type of fire, reported hours spent near stove, and carriage of the baby on the mother's back. Not only does this open up the possibility of serious exposure misclassification, but also means that little data on exposure levels are available to describe the relationships between exposure level (for example, particulates in $\mu g/m^3$) and risk (for example, the relative risk estimate). This has important implications for assessing the health impact of exposure levels in various populations, as well as in estimating the potential health gain that might result from different amounts of exposure reduction. The recently reported cohort study from Kenya has however started to address this (Ezzati and Kammen 2001a,b), but further work is required.

The observational nature of most studies presents a particular problem in terms of confounding since households adopting less polluted stoves and/or behaviours generally do so following improvements in their socio-economic circumstances – factors that are known to strongly influence many of the health outcomes studied (Bruce, et al. 1998). This, together with the lack of adjustment for confounding in a substantial minority of studies further compromises our ability to obtain unbiased risk estimates.

Despite these limitations, the evidence for two of the most important conditions – ALRI and CB/COPD – is compelling and suggestive of causality, particularly when viewed in conjunction with findings of ETS and ambient air pollution. A similar level of certainty applies to lung cancer risk and coal smoke. With these outcomes, the major weakness in the evidence is quantification of the exposure-response relationship. For other health outcomes including asthma (exacerbation), otitis media, lung cancer (arising from biofuel smoke exposure), nasopharyngeal and laryngeal cancer, ILD, low birth weight, perinatal mortality, TB and cataract, evidence must be seen as more tentative – although for most of these outcomes there is support from a variety of studies of ETS, ambient pollution, and animal experiments. Evidence of an association with cardiovascular disease has not been reviewed here since there are no studies relating to biomass smoke exposure in developing countries. However, the considerable body of evidence on the effects on cardiovascular disease of particulate and gaseous outdoor air pollution (Burnett et al., 1999; Schwartz, 1999) and ETS (He et al., 1999) raises this as a potentially important area for future work.

In Table 3, the Bradford-Hill criteria for assessing causality in an observed association are reviewed for three of the conditions studied - ALRI, low birth weight and TB. Table 4 summarizes the overall status of the evidence for all conditions discussed in this review.

Table 3: Criteria for judging causation (Bradford-Hill criteria) applied to three selected health outcomes

Criterion	ALRI	Low birth weight	Tuberculosis
Appropriate time sequence	Even though assessed crudely, exposure results from consistent, routine daily activities	Even though assessed crudely, exposure results from consistent, routine daily activities	Even though assessed crudely, exposure results from consistent, routine daily activities
Biologically plausible mechanism	Through effects of small PM and gas irritants on reducing resistance to infection	Potential mechanism through CO exposure and COHb levels in range 2-10+%	Through effects of small PM and gas irritants on reducing resistance to infection
Strength of association	RR/OR in range 2-4, though some studies no effect	One study found 63gm (95% CI: 0.35-125.6) adjusted difference	OR in range 2-3 for the few studies reported.
Consistency across populations with different exposure	Potentially major problem of confounding with comparisons of this type (e.g. comparing populations with high or low ALRI rates)	Potentially major problem of confounding with comparisons of this type	Potentially major problem of confounding with comparisons of this type
Consistency across studies in different settings	Fair degree of consistency, though some studies found no effect.	Consistent with systematic review (meta-analysis) of ETS	Consistent for the few studies reported
Dose-response relationship	Some evidence, but hard to assess due to lack of exposure measurement in most studies	Too little evidence	Too little evidence
Independence from confounding	Where adjusted for, effect remained, but some studies did not adjust. Tendency for socio-economic conditions to be strongly related to choice of fuel and stove	Adjustment carried out in the one study reported	Adjustment for range of factors in 2 studies, only for age in one other.
Supportive animal evidence	Indirect: airways damage, impairment of lung defences	From studies of cigarette smoke	Indirect: airways damage, impairment of lung defences
Removing exposure reduces risk	No intervention studies available. Feasible given short duration to outcome.	No intervention studies available. Feasible given short duration to outcome.	No intervention studies available. May not be feasible as usually long duration to outcome.

Table 4: Summary of status of available evidence on risk of specific disease conditions associated with indoor air pollution exposure

Condition	Status of evidence
ALRI in young children	Some 16 community and hospital-based studies with well defined outcomes, but very few with exposure assessment, the exception being a recent study from Kenya. The majority of published studies find RR/OR in range 2-4. Confounding inadequately dealt with in some. ETS evidence supportive but mainly from developed countries where the epidemiology of lower respiratory illness differs. Overall strongly suggests IAP exposure increases risk, but RR probably poorly estimated.
Otitis Media	Very few studies, but positive findings strongly supported by evidence from ETS.
Chronic lung disease (CB, COPD, Emphysema, Cor Pulmonale	Around 20 community and hospital based studies with various outcomes that include symptom of chronic bronchitis, clinical examination and lung function. Majority of studies find associations, although not reported in consistent manner. As with studies of ALRI in children, very few carried out exposure assessment and confounding was dealt with inadequately in some. Strongly supported by evidence from active smoking and ETS. Overall, strongly suggests IAP exposure increases risk of CB progressing to more serious COPD, emphysema and cor pulmonale in some. The RRs may be poorly estimated.
Lung fibrosis and Interstitial Lung Disease	A handful of studies to date are suggestive of this, but development of disease may also be related to silica containing dusts.
Asthma	Still relatively few published studies from developing countries and outcome definitions not well standardised. Exposure not measured and confounding not dealt with in some. Overall, evidence from developing countries suggests IAP may increase risk, but a number of studies found no effect. Studies of ETS and ambient pollution suggest that wood smoke pollution does exacerbate asthma in sensitised people.
Tuberculosis	Only three studies have found increased risk of TB, none having measured exposure directly and confounding not fully accounted for in one. Mechanism of reduced resistance plausible, and some supportive evidence from smoking.
Lung Cancer	A number of studies have found that coal smoke exposure increases risk of lung cancer, but none for biomass smoke. High levels of carcinogens present in wood and coal smoke.
Cancer of Nasopharynx and larynx	Only a few studies available, not all consistent, and rely on history of exposure. However, one recent study reported a highly significant adjusted odds ratio.
Low birth weight	Only one study in developing country community (submitted for publication). Exposure not assessed directly, but adjusted for confounding. Other sources indicate CO and COHb levels similar to passive and active smoking for which evidence strong. Ambient pollution studies in developed countries provide additional support.
Perinatal mortality	Only one study in developing country. Exposure not assessed directly, but adjusted for confounding. Supportive evidence from ambient pollution studies in developed countries.
Cataract	Eye irritation widely reported. Only one hospital-based study in a developing country reported so far that has shown increased risk of cataract. Supported by evidence from animal studies of effects of wood smoke, and from studies of smoking and cataract.

CONCLUSION

Indoor air pollution appears to be a major environmental and public health hazard for large numbers of the world's poorest, most vulnerable people. The evidence from which this conclusion is derived, however, is based on studies with limitations that limit our ability to quantify the risk and burden of ill-health accurately, and for a number of health outcomes there are still very few studies. It is important therefore to strengthen both the amount and quality of this evidence, paying particular attention to more valid quantification of exposure and ensuring that confounding is adequately dealt with.

The disease conditions or health problems for which the available evidence is extremely limited or inconsistent and conflicting are listed below. We are not aware of any reports linking cardiovascular disease to biomass smoke exposure in developing countries, although this has been established for ambient pollution in developed countries. For these conditions, well-conducted observational studies which include exposure assessment and deal with confounding should be the next step.

Conditions for which there is a paucity of evidence

- Otitis media
- Low birth weight, perinatal mortality
- Tuberculosis
- Cataract
- Asthma
- Lung cancer in biomass using areas
- Cancer of the nasopharynx and larynx
- Lung fibrosis, Interstitial lung disease
- Cardiovascular disease

There is also a strong case for carrying out a limited number of intervention studies, particularly for the most common and serious health outcomes such as ALRI where there is already a reasonably consistent set of observational studies. The acute nature of ALRI makes it amenable to an intervention study in a way that more chronic conditions such as established COPD in older people, TB or cancer would not be. Such studies may adopt a randomised controlled trial design which will provide the most powerful evidence and remove the problem of confounding, or be based on natural experiments if appropriate settings are found. Case-control or cohort studies may also serve this purpose if suitable areas can be identified where a good range of exposure levels are distributed fairly evenly and confounding factors are not too strongly associated with exposure.

Finally, the emphasis that future studies should pay more attention to exposure assessment implies a need to further develop practical, robust and valid methods for measuring the exposure levels and patterns - particularly for women and young children. As has been noted, ambient (room) pollution levels, while better than proxies such as stove type, are still a relatively poor indicator of personal exposure.

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Appendix Table A. Survey of studies on indoor air pollution levels in developing countries

Location	Ref #	Duration of sample and size fractions (TSP, PM10, or PM2.5)	Fuel type	Cooking conditions and/or comments	PM (mean or range) in mg/m^3	CO (ppm)
Africa						
Lusaka, Maputo, and Hanoi	Ellergard (1997)	Duration not clear/PM10	Mixed	Range of means	531 - 1038	
Maputo, Mozambique	Ellergard (1996)	Cooking period/PM10	Wood Charcoal Coal		1200 540 940	
Zimbabwe	Martin (1991)	Cooking period/ unclear which size fraction	Wood	children with ALRI children with URI	1,998 546	
Zimbabwe	Collings et al. (1990)	Blood sample/HbCO	Wood			6.45%
Asia						
Bangkok	Smith et al. (1994)	12-24 hours/PM10	Charcoal	Indoor Personal monitoring	330 550	1.6 8.5
Beijing	Smith et al. (1994)	12-24 hours/PM10	Coal (vented)	Indoor Personal Monitoring	550 1900	20
Beijing	Smith et al. (1994)	Estimated daily exposure PM10 (µg-h/m3)	Coal (vented)		23000-35000	
Garhwal Himalaya	Saksena te al. (1992)	Cooking period in kitchen for TSP and instantaneous for CO	Wood and shrubs	Geometric mean	4500	10
Garhwal Himalaya	Saksena et al. (1992)	Estimated total exposure	Wood and shrubs			
Himalayan village, northern India	Norboo et al. (1991(a)		Wood and Yak dung	Summer Winter		14.9 26.2
Pune	Smith et al. (1994)	12-24 hour period/PM10	Wood Wood	Area monitoring Personal monitoring	2000 1100	 10
Americas						
Navajo children	Robin et al.	15 hours/PM10	Wood		3-186	

Location	Ref #	Duration of sample and size fractions (TSP, PM10, or PM2.5)	Fuel type	Cooking conditions and/or comments	PM (mean or range) in mg/m^3	CO (ppm)
in Arizona	(1996)					
Rural Bolivia	Albalak et al. (1999)	6 hour/PM10	Mostly cow dung	Indoor kitchen Outdoor kitchen Geometric means	1830 280	
Rural Bolivia	Albalak et al. (1999)	Estimated daily exposure				
Rural Guatemala	McCracken & Smith (1998)	Experimental conditions: standardized cooking test/PM2.5	Wood	Open fire Improved stove	27,200 450	
Oceania						
Eastern Highlands, PNG	Anderson (1978)	6 pm – 4 am/TSP	Wood		600 – 2000	
Other						
Hypothetical Village kitchen in developing countries	Zhang et al. (1999)	1 hour	Charbiquette Charcoal Brush wood Dung Crop residue Fuel wood			562 528 511 464 241 150

Appendix Table B: Studies of chronic bronchitis or COPD and exposure to biomass smoke

Study & Location	Study design and numbers	Measure of exposure	Measure of health outcome	Findings	Comments confounding etc.
Padmavati & Pathak (1959) Delhi	127, Case series of cor pulmonale	Cooking fuel extracted from charts, information not available in all	Clinical diagnosis of cor pulmonale	Most exposed to cow dung and from rural origin.	No control of confounding.
Master (1974) Highlands, Papua New Guinea	94 adults selected randomly, clinically evaluated	All living in relatively closed huts burning biomass	Clinical assessment	Small closed huts with smoky fires to keep warm. Many were smokers. 78% of those studied had chronic lung disease, mainly obstructive. Smoking technique similar to cigar smoking.	No formal control for confounding. Equal distribution among gender. Smoking, malnutrition, poor sanitation considered as contributing to lung disease.
Anderson (1979a&b) Highlands, Papua New Guinea	Cross sectional study of 1284 adults, 11 villages, 1800 m altitude	Questionnaire	Spirometry	Airflow obstruction (FEV1/FVC<60%) in 20% of men and 10% of women. No risks estimated. Lung disease not associated with smoking. Disease attributed to wood smoke and acute chest infections.	Prevalence reported stratified by age and gender and smoking habits Most men and women were smokers of home grown tobacco (smoked without inhalation).
Anderson (1976) Karkar Island, Papua New Guinea	Cross sectional study of 87% of residents, total 1026 subjects living in 3 villages.	Questionnaire	Spirometry, questionnaire	Prevalence of respiratory abnormalities similar in men and women, with airflow obstruction.	Control for gender, age and smoking. Smoking resembles cigar smoking. Authors suggest repeat chest infections also a cause of Chronic obstructive lung disease.
Pandey (1984a) Rural hill region, Nepal	Cross sectional study of 2826 adults. 57% of those with CB had airflow obstruction. 93% of cases with CB were confirmed in hospital, also some cases of emphysema and cor pulmonale	Questionnaire, hours spent near fire.	Questionnaire, clinical evaluation, chest X-rays, pulmonary function tests	Prevalence of CB 18.3%, similar in men and women. No analysis of association between exposure and emphysema or cor pulmonale or airflow obstruction reported.	Stratified analysis by age. Similar prevalence in four ethnic groups and in both sexes. No influenza epidemics detected during the study. Sputum analyzed for tuberculosis in patients with chronic cough.
Pandey (1984b) Rural hill region, Nepal	Cross sectional study of 1375 adults aged 20 plus. Included 1077 smokers, 223 non smokers, 75 past	Questionnarie, type of stove, hours spent near fire.	Questionnaire	Prevalence of CB increases with hours near the fireplace in smokers and non smokers. Aditive effect with tobacco	Control for smoking, gender and age

Study & Location	Study design and numbers	Measure of exposure	Measure of health outcome	Findings	Comments confounding etc.
	smokers.				
Pandey et al. (1985) Rural area near Kathmandu, Nepal	Cross-sectional study of 150 women from a rural area, similar number of smokers and non smokers.	Questionnaire to assess number of hours cooking, separated in 3 levels	Spirometry	Group with most exposure had a 16% lower FEV1 and FVC in smokers, and a 7% lower FEV1 and 4% lower FVC in non smokers. Differences in non smokers were not statistically significant.	Stratified by smoking status. No control outdoor pollution.
Malik (1985) Chandigarh, India	Cross sectional study of 2180 women older than 20	Questionnaire, type of fuel used	Symptoms by questionnaire, spirometry	CB in group exposed to cow dung and wood, 5.0%, in LPG 1.6%, paraffin oil 1.3%, coal+paraffin 2.6%. PEFR was lower in the group exposed to biomass (Chulla stove)	No control for confounding reported
Pandey et al. (1988) Plains and hill regions, Nepal	Cross sectional study of adults: 652 from the plains, 641 from mountains	Questionnaire, type of fuel used.	Questionnaire	Prevalence of CB 13.1% in plains, 30.9% in mountains. Similar in men and women. Difference attributed to lesser ventilation and more exposure in mountains.	Control for gender, age and smoking.
Anderson et al. (1988) Papua New Guinea	2026 highland, 1734 coast, 15 year mortality follow up after surveys in 1970-71	Questionnaire	Mortality, spirometry	Increased mortality in subjects short of breath, wheezers, and those with reduced lung function, but not in local tobacco smokers	Control for age, height and smoking.
Norboo et al. (1991a) Rural Ladakh, N India	Cross-sectional study of 208 women and 156 men aged 20 and over, carried out in summer and winter on same subjects. Alt > 3000 m	Type of stove and fuel, ambient and personal exhaled air CO level.	MRC (UK) questionnaire for chronic bronchitis, spirometry	Inverse relationship between personal CO and FEV1/FVC ratio ($p<0.05$ for women). Significant increase in exposure (assessed by room CO and personal exhaled CO) between summer and winter. Fall in individual FEV1 between summer and winter associated with increase in personal CO ($p<0.01$).	Adjusted for age and height. Very few women smoked, and smoking men excluded from analysis.
Behera et al. (1991) Rural India	Cross sectional study of 3701 women, of whom only 93 were smokers.	Questionnaire, type of fuel	Symptoms by questionnaire, spirometry	CB in 1.9% of the sample, 2.9% of those using biomass (Chulla). Total symptoms more common in mixed fuel and biomass than in gas. Reduction in FEV1 (3%) and FVC (2%) in biomass users compared to LPG. No RR reported	No adjustment for confounding reported

Study & Location	Study design and numbers	Measure of exposure	Measure of health outcome	Findings	Comments confounding etc.
Dossing et al. (1994) Saudi Arabia	Case (n=50) control (n=71) study. Cases were of COPD (FEV1<70% predicted, FEV1/FVC<0.70, non reversible. Healthy controls	Questionnaire, type of stove	Spirometry	Association in non smoking women with indoor wood fire (OR=49: 95% CI). COPD not associated with incense burning.	Passive smoking and occupational expossure to dust "equally distributed" among cases and controls. No other comments on control of confounding.
Menezes et al. (1994) Urban Pelotas, Brazil	Cross-section study of 1053 adults aged over 40 years. Chronic bronchitis in 12.7%	Questionnaire	Symptoms by questionnaire	Increased risk of CB with higher indoor pollution, OR=1.9 (1.2-3.0).	Multivariate analysis used. Low family income, no education, smoking, and respiratory illnesses in infancy also risk factors.
Qureshi (1994) Rural Kasmir, Pakistan	Cross-sectional study of all residents over 15 years in 2 villages, one biomass using, the other using mainly kerosene, LPG and electricity. Alt 1600 m.	Interview and observation of ventilation, smoking, fuel type and average time per day near fireplace.	Symptoms of CB and asthma by questionnaire, spirometry	7.7% with CB, 2.0 with asthma. Higher prevalence in women and in biomass using village. CB in women positively associated with time spent near fire ($p<0.01$), but asthma not associated (small numbers). Association between lung function and exposure not reported.	CB associated with smoking, but very few women smoked. CB also associated with quality of housing and ventilation, but these factors not adjusted for.
Dennis et al. (1996) Colombia	Hospital based case (n=104) control (n=104) study. Cases had COPD, FEV1/FVC<70, FEV1<70% predicted. Controls	Questionnaire, type of stove	Spirometry	Crude OR for wood smoke 3.4, adjusted 3.9. Also significant for tobacco 2.6 and ETS 2.0	Stratified analysis by age, hospital, smoking, use of alcohol, ocupational exposures. Multivariate analysis with smoking, passive smoking, age, hospital.
Perez Padilla et al. (1996) Mexico	Hospital based case (n=127) control (n=375) study. Cases were CB or COPD (FEV1<70% predicted). Controls (4 groups) had ILD, ENT and TB as diagnoses, and visitors.	Questionnaire, type of stove	Spirometry for COPD and questionnaire to define CB	Crude OR 3.9 for CB, 9.7 for CB+COPD and 1.8 for pure COPD. Adjusted ORs similar to unadjusted.	Control of confounding by stratified analysis and logistic regression which iIncluded age, smoking, place of birth and residence, SE status, level of education.
Dutt et al. (1996) Urban slum, Pondicherry, India	Stratified random sample of 1117 women, drawn from 1560 subjects identified in a survey. Included 105 using biofuels, 105 kerosene, and (number) LPG.	Questionnaire, type of fuel	Questionnaire, spirometry	Respiratory symptoms in 26% of those using biofuels, 13% for kerosene and 8% for LPG. Lung function lower in biomass users as well. No RR reported	No adjustment for confounding reported.

Study & Location	Study design and numbers	Measure of exposure	Measure of health outcome	Findings	Comments confounding etc.
Ellegard (1996) Suburbs of Maputo, Mozambique	Cross sectional study in 1188 women, 218 with monitoring of air pollution	Questionnaire on fuel type use and measurement of particulates	Questionnaire of symptoms (cough symptom index), PEF with Wright mini peak flow.	Particulates in wood users 1200 $\mu g/m^3$, charcoal 540, gas and electricity 200-380. After adjustment, wood use and time cooking significantly associated with cough symptom index. Kitchen ventilation and time in house (but not fuel type) associated with PEF. No association with wheeze.	Adjustment for a range of socio-economic and environmental variables.
Regalado et al. (1996) Highland (2500m) Mexico	Cross sectional study of 871 of women aged over 38. Alt 2500 m.	Type of fuel used from questionnaire. Kitchen particulate concentration while cooking measured with nephelometer	Symptoms by questionnaire, spirometry	Biomass stove associated with increase in phlegm and 4% reduction in FEV1/FVC. Mild effect on FEV1 in homes with higher PM_{10}. At levels of PM_{10} above 2000 $\mu g/m^3$, an increase in 1000 $\mu g/m^3$ was associated with a reduction of 2% in FEV1.	76% of women cooked with gas but also with biomass, 10% only biomass. Control for age, cigarette smoking, socioeconomic status.
Albalak et al. (1999) Rural highland Bolivia	Cross sectional study of all residents (total n=241) from 2 villages, one cooking indoors and the other outdoors. Alt 4100 m.	Questionnaire to assess time activity patterns. Sampling for area PM10 measurements in 12 randomly selected homes in each village.	Questionnaire for symptoms of CB	Prevalence of CB was 22% and 13% in indoor and outdoor cooking villages respectively. OR=2.5 (95% CI) for indoor cooking. Estimates exposures for women (based on time activity and PM10 measurements) were about double in village cooking indoors.	Communities similar in SE status, climate, altitude, access to health care. Adjustment for age and sex in logistic regression.

Appendix Table C: Studies reporting interstitial and pneumoconiosis like lung changes in subjects exposed to biomass smoke.

Author	Setting	N	Description	Proposed pathogenesis	Findings
Master (1974)	Highlands in New Guinea	98	Chronic pulmonary disease	Wood smoke, poor sanitation, endemic infectious diseases, tobacco smoking	Obstructive and restrictive disease
Restrepo et al. (1983)	Mountain areas of Colombia	22	Pneumoconiosis, features of bronchitis and lung fibrosis	Inhalation of wood smoke fly ash with silicates in cold areas using biomass smoke with poor ventilation	Functional pattern obstructive and restrictive. In biopsy fibrosis and anthracosis with crystals under the polarized light
Grobbelar (1991)	Xhosas from Transkei, grind dry maize producing a fine dust	25	Hut lung	Domestically acquired pneumoconiosis due to wood smoke, plus exposure to dust containing quartz	17 non smokers, 7 previous TB 12 simple anthracosis, 6 macula formation, 7 fibrosis or mixed dust pneumoconiosis. BAL with macrophages laden with particles. Cases with PMF and coalescent nodules
Dhar (1991)	Gujjar community, Kashmir altitude, closed environment	46	Gujjar lung	Severe exposure to wood smoke	Anthracosis, 27/36 biopsies with fibrotic reaction, no particles by polarized light. PFT with obstruction and restriction. Cases with coalescent nodules
Saiyed (1991)	3 villages, Ladakh, India	449	Pneumoconiosis, including silicosis in 2%, 20% and 45%	Dust storms and smoke from domestic fuels	Cases of progressive massive fibrosis and egg-shell calcification
Norboo ET AL. (1991b)	Villages in Ladakh, N Indian Himalaya	20 men and 20 women > 50 years	Silicosis	Dust storms, quartz demonstrated in dust and lung. Also exposed to biomass smoke [see Norboo 91(a)]	Cases with progressive massive fibrosis. Silicosis in 8/20 men and 16/20 women
Sandoval (1993)	Rural Mexico, women	22	Lung disease associated with wood smoke exposure	Wood smoke	Anthracosis with fibrotic reation

*All patients had micronodular or reticular opacities in chest X-rays and several had reduced lung volumes in addition to airflow obstruction and symptoms of airways disease.

Appendix Table D: Published studies from developing countries reporting on associations between exposure to polluting household fuels which include biofuels, and wheeze and/or asthma. Listed by year of publication.

Study & Location	Study design/numbers	Measure of exposure	Measure of wheeze or asthma	Findings	Comments
Guneser ET AL. (1994) Adana, Southern Turkey	Cross-sectional survey of 9-12 year olds (n=617)	Reported fuel and stove type used at home. Annual mean (outdoor) smoke level (1988) 26 μg/m^3.	Reported symptoms including day and night cough. Spirometry: FVC, FEV_1, PEFR, FEF_{25}	Coal using group reported more day/night cough than kerosene, oil, electricity users ($p<0.05$). Lowest values of FVC, FEV_1, PEFR, FEF_{25} in wood stove group ($p<0.05$)	No adjustment for confounding reported
Mohamed et al. (1995) Urban Nairobi, Kenya	Community matched case (n=77) control (n=77) study, with cases from a prevalence survey in 9-11 year old schoolchildren. Matched for age and gender	Type of stove and fuel, "visible" indoor pollution observed at home visit	Doctors diagnosis, history of wheeze or >10%FEV1 decline after exercise.	22% (17) of cases using wood and charcoal versus 10% (8) of controls. Use of other fuels similar. Main analysis based on subjective assessment of indoor air pollution not on fuel, with a crude OR 3 (1.4-6.3). Adjusted OR for pollution 2.5 (2.0-6.4).	Adjusted for air pollution outside home, passive smoking, mud floors. Fuel type not associated with asthma.
Azizi et al. (1995) Kuala Lumpur	Hospital based case (n=158) control (n=201) study of children 1 month to 5 years. Controls age matched non-respiratory admissions.	Type of stove and fuel used at home reported by mothers at interview.	First hospitalised episode of acute asthma, with physician diagnosis. Excluded acute bronchiolitis, pneumonia, stridor and other respiratory conditions.	Unadjusted OR for asthma with wood stove was 1.4 (0.6-3.6). Also NS in multivariate model. For mosquito coil smoke adjusted OR = 1.73 (1.02-2.93).	(Multivariate analysis used).
Noorhassim et al. (1995) Rural Malaysia	Cross-sectional study of 1000 children aged 1-12 years	Questionnaire	How was asthma defined and cases ascertained?	No association. Is OR (95% CI) given?	
Gharaibeh (1996) Jordan	Cross-sectional survey of 7-13 year olds (n=1905). 72% of sample urban, 28% rural.	Reported fuel and stove type used at home, divided into exposed (wood or kerosene), and unexposed (electricity or modified kerosene stove which expels fumes outside)	Symptoms not reported. Spirometry: FVC, FEV_1, PEFR, FEF_{25-75}	Values of FVC, FEV_1, PEFR, FEF_{25} all lower in exposed children ($p<0.005$).	No adjustment for confounding, but children exposed to polluting fuels at home, and to smoking, were excluded.

Study & Location	Study design/numbers	Measure of exposure	Measure of wheeze or asthma	Findings	Comments
Xu et al. (1996) Rural China	Cross-sectional study of 28,946 adults aged 15 years and over.	Interview questionnaire on environmental exposures, including dust, fumes, outdoor and indoor air pollution. 93% of population used wood/hay for cooking, so occupational exposures used to study effect of biomass on asthma, but cooking exposures for effects of coal on asthma.	Interview questionnaire using "standard questions" for asthma symptoms, and for recall of physician diagnosed asthma.	Adjusted Ors for occupational exposure to wood/hay smoke: wheezing 1.36 (1.14-1.61); asthma 1.27 (1.02-1.58) Coal for cooking: wheezing 1.47 (1.09-1.98); asthma 1.51 (1.05-2.17)	Adjustment made for gender, age, area. Education, smoking and ambient air pollution.
Lopez-Bravo et al. (1997) Santiago, Chile	Cohort study of 437 children followed from birth to 18 months. Neonates with birth weigh <2,500 gms excluded.	Type of stove and fuel used at home reported by mothers at interview. Defined groups as polluted (use of wood, kerosene or coal for cooking or heating) and non-polluted (use of gas)	Cases seen in clinic and referred to hospital for physician diagnosis of ARI. Three groups defined: bronchitis, obstructive bronchitis syndrome (presumed to include wheeze or other evidence of asthma), pneumonia.	Percent children with ≥2 episodes of obstructive bronchitis syndrome: 47.3% for polluted homes, 34.2 for non-polluted ($p<0.01$). Difference for pneumonia (20.6% vs 15.7%) not significant.	97% homes used gas for cooking, but 81% used kerosene, wood or coal for heating. No adjustment for confounding reported.
Melsom et al (2001), urban and rural Nepal	Case-control study of 121 children aged 11-17 years with asthma and 126 controls	Interview questionnaire on type of fuels used in the home.	Standard ISAAC core questionnaire and video questionnaire	Adjusted OR for asthma associated with smoking by 2 or more family members 1.9 (1.0-3.9), for use of open fire/stove without flue 2.2 (1.0-4.5) compared to stove with flue or cleaner fuels.	Stratification by sex showed results for smoking and smoky fuels only significant for boys.
Schei et al (2002), rural Guatemala	Cross-sectional study of 4-6 year olds in 1058 homes	Interview questionnaire and observation of type of fire and fuel used: open fire (38.1%), chimney stove using wood (51.5%), gas/open fire combination (10.4%)	Standard ISAAC questionnaire to assess ever wheezed, wheezing in last 12 months, speech limiting symptoms, exercise-induced wheezing, asthma diagnosis	Overall prevalence of ever wheezing 7.3%, and 3.4% for last 12 months. In logistic regression, adjusted OR for open fire was 1.81 (1.04-3.12) for ever wheezed, and 2.35 (1.08-5.13) for wheeze in last 12 months.	Regression model included family history of wheeze (independently associated with all asthma symptoms in this study), sex and interviewer.