Indoor air pollution in developing countries and acute lower respiratory infections in children

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Abstract

***Background*—A critical review was conducted of the quantitative literature linking indoor air pollution from household use of biomass fuels with acute respiratory infections in young children, which is focused on, but not confined to, acute lower respiratory infection and pneumonia in children under two years in less developed countries. Biomass in the form of wood, crop residues, and animal dung is used in more than two fifths of the world's households as the principal fuel.**

***Methods*—Medline and other electronic databases were used, but it was also necessary to secure literature from colleagues in less developed countries where not all publications are yet internationally indexed.**

***Results*—The studies of indoor air pollution from household biomass fuels are reasonably consistent and, as a group, show a strong significant increase in risk for exposed young children compared with those living in households using cleaner fuels or being otherwise less exposed. Not all studies were able to adjust for confounders, but most of those that did so found that strong and significant risks remained.**

***Conclusions*—It seems that the relative risks are likely to be significant for the exposures considered here. Since acute lower respiratory infection is the chief cause of death in children in less developed countries, and exacts a larger burden of disease than any other disease category for the world population, even small additional risks due to such a ubiquitous exposure as air pollution have important public health implications. In the case of indoor air pollution in households using biomass fuels, the risks also seem to be fairly strong, presumably because of the high daily concentrations of pollutants found in such settings and the large amount of time young children spend with their mothers doing household cooking. Given the large vulnerable populations at risk, there is an urgent need to conduct randomised trials to increase confidence in the cause-effect relationship, to quantify the risk more precisely, to determine the degree of reduction in exposure required to significantly improve health, and to establish the effectiveness of interventions.**

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Acute respiratory infection (ARI) is the most common cause of illness in children and a major cause of death in the world. Among children under five years of age, three to five million deaths annually have been attributed to ARI, of which 75% are from pneumonia.[1] The World Health Organization estimates that approximately three million children under five died from ARI in 1993, exclusive of measles, pertussis, and diphtheria, and another 1.1 million died from conditions in association with these diseases (table 1).[2] As shown in table 2, ARI is one of the leading causes of death in the world, smaller only than heart disease, cancer, and cerebrovascular disease. In terms of lost healthy life years (measured as disability adjusted life years, DALYs), however, table 2 shows that ARI is the chief cause of global ill health today because its biggest impact is in young children.[3] ARI is also a significant cause of death at other ages, particularly in the very old.

Early in the 20th century ARI, in the form of pneumonia, was also a major cause of death in the currently developed countries, but its importance diminished dramatically during the century, partly due to the development of vaccines and antibiotics.[4] A large decline had already occurred before these medical interventions became available, however, probably largely reflecting improvements in housing environments and nutrition.

This report on indoor air pollution is part of a series of reviews of the major determinants of childhood pneumonia in developing countries that were initiated by the World Health Organization in association with the London School of Hygiene and Tropical Medicine.[5] There are a number of risk factors that affect

Table 1 Annual mortality in children aged under five years from developing countries in 1993

ARI related:	4.1 million
ARI alone	3.0
ARI with measles	0.64
ARI with pertussis	0.26
ARI with malaria or HIV	0.23
Neonatal or perinatal	3.1 million (many involving ARI)
Diarrhoea related:	3.0 million
Diarrhoea alone	2.7
Diarrhoea with measles or HIV	0.27
Measles/TB/tetanus/pertussis alone	1.2 million
Malaria alone	0.68 million
Other	0.2 million
Total	12.2 million

ARI = acute respiratory infection.
Source: World Health Organization.[2]
Other ARI information:
ARI accounts for 33% of all deaths from infectious disease in the world and for 27% of the entire burden of infectious diseases.
80% of the ARI burden occurs in children under five years from less developed countries, accounting for about 6.7% of the global burden of disease from all causes.

Table 2 Global burden of death and diseases in 1990 (%). Those categories causing at least 1% of lost DALYs

	World	*LDCs*	*MDCs*
Acute respiratory infections	8.5	9.4	1.6
Diarrhoea	7.2	8.1	0.3
Perinatal effects	6.7	7.3	1.9
Child cluster (measles, pertussis, polio, tetanus, diphtheria)	5.2	5.8	0.008
Cancer	5.1	4.0	13.7
Depression	4.7	4.4	8.5
Malnutrition/anaemia (direct effects)	3.7	4.1	0.9
Heart (ischaemic)	3.4	2.5	9.9
Tuberculosis	2.8	3.1	0.3
Cerebrovascular (stroke)	2.8	2.4	5.9
Motor vehicle accidents	2.5	2.2	4.4
Congenital (birth defects)	2.4	2.4	2.2
Malaria	2.3	2.6	0.003
Maternal	2.2	2.4	0.6
Sexually transmitted w/HIV	2.2	2.3	1.3
Chronic obstructive lung disease	2.1	2.1	2.1
Falls	1.9	2.0	1.5
War	1.5	1.5	0.7
Suicide	1.4	1.2	2.3
Violence	1.3	1.3	1.1
Alcohol (direct effects)	1.2	0.8	4.0
Drowning	1.1	1.2	0.5
Total (%)	72	73	64
Population (million)	5260	4120	1140
Lost DALYs (million)	1380	1220	160
Deaths (million)	50.5	10.9	36.6

Source: Murray and Lopez.[3]
DALYs = disability adjusted life years; MDCs = more developed countries; LDCs = less developed countries.

ARI rates in young children, including malnutrition, lack of breast feeding, and the incidence of other diseases that affect susceptibility. The child's environment also affects risk through such factors as crowding, chilling, and air pollution. This review explores what is known about the contribution of household air pollution to the risk of ARI in young children worldwide, with particular focus on less developed countries. When possible, we concentrate on pneumonia, which causes the highest case fatality rate. The review does not comprehensively address the sources and concentrations of indoor air pollutants in less developed countries; rather, in the course of examining the strength of air pollution as a risk factor, it offers an overview. More details can be found in Chen *et al*[6] and Smith.[7]

Introduction to ARI and air pollution

Early in the 20th century dramatic episodes of outdoor air pollution in developed countries showed that air pollution could cause excess deaths and that children might be at particularly increased risk during the times of high pollution.[8] For example, during the London fog of 1952, which was due mainly to smoke from coal burning household stoves,[9] several thousand excess deaths occurred. Infants and young children as well as the elderly were noted to be at higher risk than others and the proportion of deaths attributed to respiratory causes was increased in comparison with the weeks before and after the fog.[10] Outdoor air pollution has now been examined as a risk factor for respiratory morbidity and mortality in numerous epidemiological studies and the evidence continues to indicate that infants and young children are at risk for adverse effects.[8 11–14] Even though ambient pollution levels have now declined in developed countries, the epidemiological evidence continues to indicate adverse effects on both respiratory morbidity and mortality.[12 13] Indeed, new studies are indicating adverse effects of inhaled particles at levels that were previously considered to be safe and are now frequently reached in many urban areas.[12 15–17]

During the last two decades the potential significance for child health of exposures to air pollutants in indoor environments has also been recognised.[6 7 18–20] The world's children are exposed to inhaled pollutants as they breathe air in diverse indoor and outdoor locations. In considering risk to health, total personal exposure—which encompasses all exposures received to an agent, regardless of the locations and the medium—is the relevant exposure measure.[21] Total personal exposure to an air pollutant can be estimated as the weighted average of the pollutant concentrations in the environments where a child spends time; the weights are proportional to the time spent in each of these environments having distinct pollutant concentrations.[22] This concept of pollution exposure, termed the microenvironmental model, makes clear the health relevance of both indoor and outdoor pollution exposures and the potential for widely varying contributions of indoor and outdoor exposures to total personal exposures for children living in different countries throughout the world, depending on sources and time-activity patterns. It emphasises that one must be sure to examine pollution where the people spend most time, as well as in places where ambient levels are high.[23–25]

Using particulates as the indicator pollutant, for example, total population exposure globally has been estimated to be dominated by household environments in developing countries where solid fuels are used for cooking and heating.[25 26] This is because of confluence of exposure factors—that is, large populations adjacent to frequently used devices with large emission factors. Crop residues, dung, wood, and coal are widely used globally, perhaps accounting for about half of all fuels used daily to cook meals.[27] From the standpoint of particle levels, the most polluted urban outdoor environments in the world are also in developing countries—notably, but not exclusively, in the coal using cities of Asia.[26 28] Exposures to environmental tobacco smoke (ETS) track tobacco consumption; this has been dominated by developed countries but rates in these countries are now static or declining while in the developing world they are growing steadily.[26]

This review focuses on indoor exposures of the world's children to pollution from combustion of biomass fuels. (Companion reviews have also been done on ARI risks to children from indoor air pollution due to tobacco smoking and outdoor air pollution from combustion of fossil fuels.) The review does not address indoor air pollution by nitrogen dioxide from cooking stoves and space heaters. In spite of intense investigation, this indoor pollutant has not been convincingly linked to ARI, but has been inconsistently related to respiratory symptoms.[29–31] For example, a cohort

Table 3 Host defences against respiratory infections

- Anatomical barriers
- Angulation of airways
- Mucociliary clearance
- Secretory IgA
- Surfactant
- Opsonising IgG, fibronectin
- Complement
- Alveolar macrophages
- Polymorphonuclear leucocytes
- Plasma components
- Vasoactive mediators

Based on Reynolds and Elias.[36]

study of nitrogen dioxide exposure and respiratory illness during the first 18 months of life found no evidence of increased risk with exposure.[32] Ackermann-Liebrich and Rap[33] have recently reviewed the evidence on indoor exposure to nitrogen dioxide.

A brief discussion of mechanisms

ARI comprise a set of clinical conditions of various aetiologies and severities that are generally divided into two main forms: upper respiratory tract infections (URI) and lower respiratory tract infections (ALRI). The risk of severe ARI, which can be fatal, is highest in very young children and in the elderly. Clinical and epidemiological criteria are available for separating URI from ALRI but, unfortunately, worldwide there are no uniformly accepted criteria and the definitions in use are not fully consistent. For research and case management under field conditions in less developed countries the WHO defines URI to include any combination of the following symptoms: cough with or without fever, blocked or runny nose, sore throat, and/or ear discharge. URI can usually be treated successfully with supportive therapy at home. ALRI include severe ARI involving infection of the lungs, with pneumonia being the most serious form.[34] Serious infections are most commonly caused by bacteria, although they may sometimes be viral. Clinical signs of ALRI include any of the above symptoms of URI with the addition of rapid breathing and/or chest indrawing and/or stridor. Severe ALRI caused by bacteria are treated with antimicrobial therapy, without which they can sometimes be fatal.[35]

Air pollutants could increase the incidence of ARI by adversely affecting specific and non-specific host defences of the respiratory tract against pathogens (table 3).[36] The non-specific mechanisms include filtration and removal of particles by the upper airway, the mucociliary apparatus of the trachea and bronchi, phagocytosis promoting components of the epithelial lining fluid, and phagocytosis and killing of infecting organisms by cells in the airways and alveolar macrophages. The specific mechanisms involve various components of humoral and cellular immunity. Organism specific immunoglobulins promote phagocytosis; cell mediated immunity is required to kill organisms capable of living within alveolar macrophages.

Smoke from household solid fuels is a complex mixture which contains many potentially relevant components from a toxicologic perspective. These mixtures are inherently highly variable with characteristics determined by sources, materials burned, time since generation, and other factors. The chemical and physical characteristics of these mixtures have been characterised to some extent,[7 37 38] particularly in the form of wood smoke from metal heating stoves used in developed countries. Thus, only generalisations can be offered concerning mechanisms by which particular air pollutants could increase the risk for ARI and mixture-specific arguments cannot readily be developed. On the other hand, there is a sufficient basis of understanding of the toxicological properties of these mixtures to conclude that they could plausibly increase the risk of ARI.

A number of pollutants commonly found in indoor and outdoor air have been shown to adversely affect components of the defence mechanisms against infectious organisms. For example, the particulate phase of cigarette smoke and gas phase components adversely affect ciliary function in in vitro models. Gaseous components that appear to be important include nitrogen dioxide, ammonia, cyanides, aldehydes, ketones, acrolein, and acids.[39] Nitrogen dioxide has been shown to adversely affect both the mucociliary apparatus and humoral and cellular immune defences.[40] The complex mixture of sulphur dioxide and particulates may reduce the efficacy of host defences against microbial agents and respiratory tract inflammation.[13] Ozone has been shown to cause respiratory tract inflammation, increased bronchoalveolar permeability, and to impair macrophage functions.[41] In animal studies diesel exhaust has been related to chronic inflammation of the respiratory tract, epithelial cell hyperplasia, impaired alveolar clearance, pulmonary fibrosis, and compromised pulmonary function.[42]

Exposure to air pollutants might also act to increase the severity of respiratory infections and thereby increase the proportion of illnesses considered clinically to involve the lower respiratory tract, and even to increase morbidity and mortality. The increased severity might be mediated by inflammation of the epithelial surface of the tracheobronchial tree caused by the irritant pollutants. If sustained exposure to air pollutants produces chronic inflammation, then infections might become more severe as the infecting organisms further damage already inflamed and possibly narrowed airways. Recently, Thomas and Zelikoff[43] have shown that exposure of animals to wood smoke significantly altered both the local and systemic immune response associated with bacterial infection.

INDOOR AIR POLLUTION

In addition to the strength of sources, the impact of indoor emissions on air quality depends directly on ventilation and air mixing of the space. Most housing in developed countries lies at temperate latitudes and has relatively low exchange rates of indoor with outdoor air, typically one air change per hour or less.[44] Even low emission rates in such housing can result in

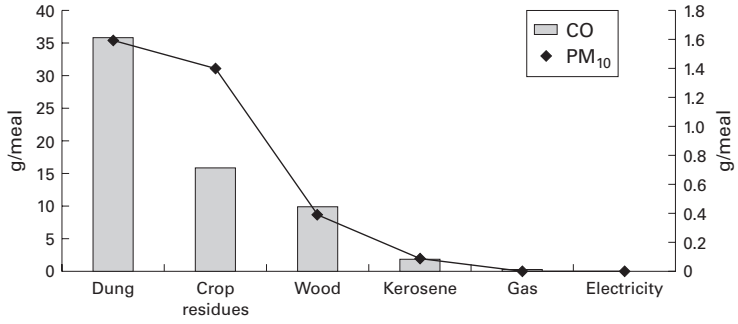

Figure 1 Emissions along the household fuel ladder. Reproduced with permission from Smith et al.[38]

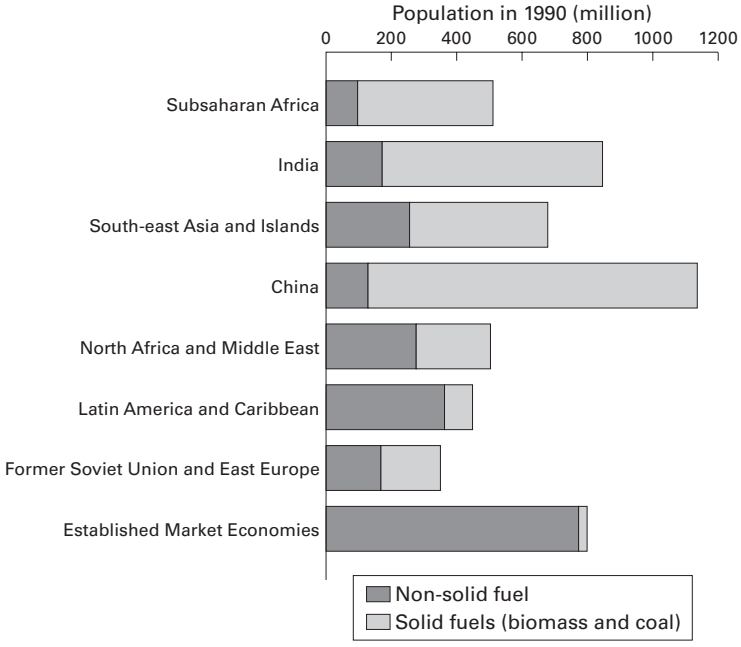

Figure 2 World distribution of household fuel use. Reproduced with permission from Reddy et al.[27]

indoor pollutant concentrations at levels of public health significance. Ventilation rates for houses in developing countries, which lie primarily in tropical and subtropical regions of the world and are often open to the outdoors, are likely to be greater. Strong sources can be readily identified in developing countries, however, including biomass (wood, crop residues, and dung) and coal burning for cooking and heating.

Indoor pollutants can be grouped by source into four principal classes: combustion products; semi-volatile and volatile organic compounds released by building materials, furnishings, and chemical products; pollutants in soil gas; and pollutants generated by biological processes.[45] The principal combustion pollutants include carbon monoxide, nitrogen and sulphur oxides, particles, and volatile organics. The complex mixture in indoor air produced by tobacco smoking has been referred to as environmental tobacco smoke (ETS). A wide variety of semi-volatile and volatile organic compounds can be found in indoor air; there are diverse sources of these compounds. The gas from the ground beneath a home may contain pollutants such as radon and termidicides that may adversely affect health. There are many biological agents in indoor environments including, for example, pollens and moulds, insects, viruses, and bacteria.

Although systematically collected data are unavailable, it is likely that the relative importance of the four types of indoor air pollution varies throughout the world with climate and level of development. For combustion sources, the focus of this review, some generalisations can be made. After tobacco smoking, gas stoves have been the most common indoor pollution source of concern in studies in developed countries.[20] In the global context, however, gas stoves are near the upper end of a historical evolution in the quality of household fuels, sometimes called the energy ladder.[46] On the lowest rungs are dried animal dung and scavenged twigs and grass as cooking fuels (fig 1). The next rungs in the sequence are crop residues, wood, and charcoal. The first non-biomass fuel on the ladder is kerosene or coal, and bottled and piped gases and electricity are highest. In general, each successive rung on this ladder is associated with increases in the technology of the cooking system, cleanliness, efficiency, and cost.

Biomass fuel

Nearly half the world's households are thought to cook daily with unprocessed solid fuels—that is, biomass fuels or coal (fig 2). In a significant proportion of the households using biomass fuels, the bulk of the emissions is released into the living area.[7] Although rates of exchange of indoor with outdoor air are relatively high in most housing in developing countries, the pollutant emission rates for such fuels are also high, and indoor concentrations and associated exposures can be high as a result. Compared with gas stoves, even stoves using wood, one of the cleaner biofuels, can release 50 times more pollution during cooking (fig 1). In addition, unvented space heating with biomass fuels is common in much of South Asia and in the highland areas of developing countries of Asia, Africa, Latin America and Oceania.[25] Large populations in China are exposed to smoke from coal fuels burned in simple stoves, which also have high emission rates.

Incomplete combustion of unprocessed solid fuels produces hundreds of chemical compounds under the operating conditions of simple cooking stoves. Such complex mixtures are produced by burning of both coal and biomass fuels, although the blends of compounds in the smokes are different. Unlike coal, biomass fuels generally contain few intrinsic contaminants (sulphur, trace metals, and ash) and, under proper conditions, they can be burned without releases other than the products of complete combustion (carbon dioxide and water). Unfortunately, optimum conditions for complete combustion are difficult to create with inexpensive household devices.

Smoke from cooking stoves is a complicated and unstable mixture.[7 47] Biomass fuel smoke contains significant quantities of several pollutants for which many countries have set outdoor

Table 4 Indoor air pollution from biofuel combustion in developing countries

Location and year	*Description*	*n*	*Particulate concentration ($\mu g/m^3$)*	*Reference*
Kitchen area concentration				
Papua New Guinea				
1968	Overnight at floor level	9	200–4900	Cleary & Blackburn[101]
1974	Overnight at sitting level	6	200–9000	Anderson[102]
Kenya				
1971–72	Overnight - highlands	5	2700–7900	Hofmann & Wynder[103]
	- lowlands	3	300–1500	Clifford[104]
1988	24 hours	64	1200–1900 (RSP)	Boleij *et al*[105]
India				
1982	15 min cooking - wood	22	15 800	Aggarwal *et al*[106]
	- dung	32	18 300	Patel *et al*[107]
	- charcoal	10	5500	
1988	Cooking (0.7 m to ceiling)	390	4000–21 000	Menon[108]
Nepal				
1986	Cooking - wood (geometric mean)	17	4700	Davidson *et al*[109]
China				
1987	All day - wood	?	2600 (RSP)	Mumford *et al*[110]
The Gambia				
1988	24 hours	36	1000–2500 (RSP)	Boleij *et al*[105]
Exposures during cooking (2–5 hours per day)				
India				
1983	4 villages	65	6800	Smith *et al*[111]
1987	8 villages	165	3700	Ramakrishna *et al*[56]
1987	2 villages	44	3600	Ramakrishna[112]
1988	5 villages	129	4700	Menon[108]
1991	3 villages - winter	95	6800	Saksena *et al*[113]
	- summer		5400	
	- monsoon		4800	
Nepal				
1986	2 villages[a]	49	2000	Reid *et al*[114]
1990	1 village - before[b]	20	8200 (RSP)	Pandey *et al*[115]
	- after	20	3000 (RSP)	

[a]Approximately half "improved cookstoves".
[b]Cooks' exposures measure before and after introduction of improved stoves.
The studies are not completely comparable because of different measurement protocols and equipment but, nevertheless, are fairly consistent. Area concentrations are measured with stationary instruments placed indoors at breathing height. Exposure rates were measured with instruments worn by the cook during food preparation. For comparisons, the US 24-hour standard, not to be exceeded more than once per year, has been 260 $\mu g/m^3$ and the Japanese one-hour standard is 200 $\mu g/m^3$. Some of the studies listed here also measured other pollutants, including carbon monoxide and benzo(a)pyrene, which were sometimes found in concentrations well above those found in public settings in developed countries, as well as nitrogen and sulfur oxides and formaldehyde, which were found in concentrations roughly equal to the high end of those measured in indoor developed country conditions.
Modified from Pandey *et al.*[49]

air quality standards—for example, carbon monoxide, particles, hydrocarbons, and nitrogen oxides. In addition, the aerosol contains many organic compounds considered to be toxic or carcinogenic, such as formaldehyde, benzene, and polyaromatic hydrocarbons. The composition of the smoke varies with even minor changes in fuel quality, cooking stove configuration, or combustion characteristics. There is ample evidence that particles are generally of the small sizes thought to be most damaging to health.[7 48]

Although a large scale worldwide survey of smoke concentrations has not been conducted, the findings of studies from different parts of the world provide an indication of typical indoor concentrations of the major pollutants. Table 4 lists studies that have measured particles, either total (TSP) or respirable.[49] Compared with various national standards, WHO recommendations, or even outdoor concentrations typical of the most polluted of cities, these indoor levels are dramatically high. We cannot presently derive an accurate estimate of the total population in developing countries exposed to indoor concentrations that would be considered unacceptable, nor can we readily apportion the contributions to total personal exposure of indoor and outdoor sources. Additionally, in some rural areas outdoor pollution penetrates indoors to a significant extent and fuel burning indoors may be a prominent contributor to outdoor pollution. Furthermore, there are no internationally recognised standards for pollutant concentrations indoors. Assuming that indoor standards should be at least as stringent as outdoor standards, the number of people exposed at unacceptable levels indoors is expected to rival or exceed the number exposed to unacceptable ambient concentrations in all of the world's cities.[50] Consideration of time-activity patterns, with far more time spent indoors than outdoors, suggests that the total global dose equivalent (amount actually inhaled) for indoor pollution could be an order of magnitude greater than from ambient pollution.[25]

EPIDEMIOLOGY

(For an annotated bibliography of ARI and indoor air quality (non-ETS) see McCracken and Smith.[51])

The first report in the biomedical literature to describe an association between indoor cooking smoke and childhood pneumonia in developing countries reported measurements of indoor pollution levels in the homes of infants diagnosed with bronchiolitis and bronchopneumonia at Lagos University Teaching Hospital.[52] Extremely high mean levels of various gaseous pollutants were measured and a mean exposure time of 3.1 hours per day was estimated but, unfortunately, the differences in exposure levels among households using wood, kerosene, coal, and gas were not reported and there was no control group of infants. It is thus

difficult to draw any quantitative conclusions about the relationship between exposure and the incidence of pneumonia.

For focused attention in this review we were able to identify 13 more recently published studies which quantitatively addressed the relationship between exposure to household biomass smoke and ALRI in young children in developing countries (table 5) in which ALRI case selection reasonably corresponded to established WHO or other authoritative criteria in use at the time the study was done. Such criteria have evolved over time and thus, as discussed below, have not been entirely consistent among the studies. It is our judgement, however, that the protocols in these 13 studies have been sufficiently rigorous to warrant treating them as part of the same evidence pool. Nine were conducted in Africa and one each in India, Brazil, Argentina, and Nepal. Only one dealt with case fatality and the others dealt with morbidity. In addition, we found two studies from a developed country (USA) of the relationship between household wood smoke and ALRI among Navaho children (table 6). These 15 studies are chosen for particular attention because they address actual ALRI, although confirmed by different means, in children under five years old and involve indoor exposures to biomass fuel smoke. Each is sufficiently quantitative to allow calculation of odds ratios and confidence intervals. Table 7 summarises the results of these 15 studies.

Some related studies, although discussed briefly below, are not examined in detail here because they only meet some of the criteria—for example, addressing risks to older children, addressing respiratory symptoms but not confirmed ALRI, or not providing enough information to calculate odds ratios.

Incidence of ALRI in young children of developing countries

Outcome measures differed among the 13 studies (table 5). Two of the cohort studies[53 54] and the one prospective case-control study[55] used reported shortness of breath to screen for children with lower respiratory disease. The first two assessed severity by counting respiratory rate and assessing for chest indrawing and signs of cardiorespiratory failure. O'Dempsey *et al*[55] confirmed cases by laboratory tests and radiography. Pandey *et al*[53] presented analyses for moderate and severe lower respiratory infections (grade II and grade III/IV ARI, respectively). In an expanded study of the same region in the Gambia studied by Campbell *et al*[54] pneumonia was confirmed radiologically in 50% of children with symptoms and signs of lower respiratory disease.[56] The remaining studies were based on children with pneumonia, severe wheezy bronchitis, or bronchiolitis diagnosed clinically or according to WHO recommendations in a hospital setting, or by verbal autopsy. These outcome measures would tend to include children with more severe illness.

Exposure to household smoke pollution was also assessed using different approaches. Broadly, the studies examined the relationship

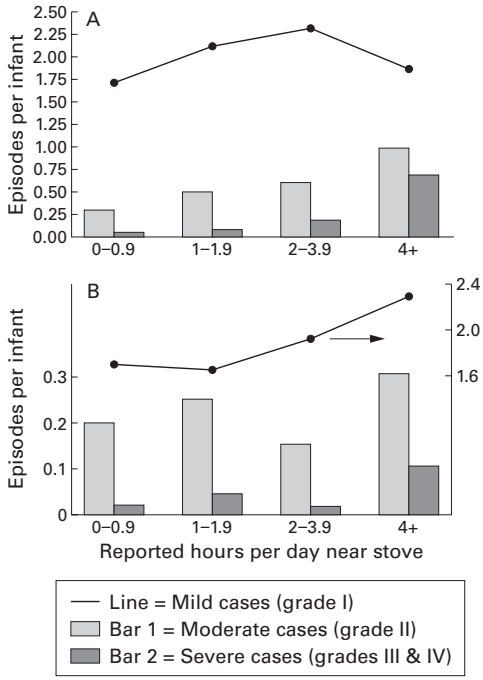

Figure 3 Acute respiratory infections (ARI) and exposure to biomass smoke in Nepal. (A) Based on six months data in about 250 infants in early 1984. (B) Based on three months data in late 1984 and early 1985 in same population but with separate teams diagnosing ARI and determining smoke exposure. Trends for moderate and severe cases are significant in both studies. Reproduced with permission from Pandey et al.[53]

of the outcome measure with cooking practices such as use of an open wood fire compared with cleaner fuels such as kerosene,[57] behavioural practices—for example, carriage on mother's back while cooking[53 55 56 58–61]—or presence of sources.[62–64] Pandey *et al*,[53] for example, used maternally reported time spent near the cooking stove as a categorical exposure measure in exploring the dose-response relationship of exposure to smoke with lower respiratory disease in children (fig 3). Only one study actually measured pollutant levels and only in a subset of study households.[57]

Air pollution studies in Kenya and the Gambia suggested that conditions were not favourable for detecting a relationship between concentrations of pollutants and lower respiratory disease in children because of the homogeneity of levels among households.[65] Collings *et al*,[57] however, found a significant difference in levels of total particles during cooking in households of 20 children with lower respiratory disease and 20 with upper respiratory disease, but few details were provided. The possibility of using carboxyhaemoglobin as a marker of smoke exposure was also explored in one study, but proved unsatisfactory because of the difficulty of controlling for time since exposure.[57]

All but one of the eight morbidity studies finding significant associations were in Africa. The data in the one non-African study (Nepal[53]) were consistent with larger relative risks for more severe disease, but the numbers were too small to exclude chance as an explanation. Age specific data available in the

Table 5 Biomass fuel use and ALRI in children under 5 in developing countries

Study	*Design*	*Case definition*	*Exposure*	*Confounding adjusted*	*Comments*	*OR (95% CI)*
Rural South Africa (1980) Natal (Kossove)[58]	Case control, 0–12 months, 132 cases, 18 controls	Outpatient cases: Wheezing, bronchiolitis & ALRI; Clinical + *x* ray. Controls: Non-respiratory problems	Asked: "Does the child stay in the smoke?" Prevalence = 33%	Routine data collection: • number of siblings • economic status Examined, not adjusted	Only 63% of 123 *x* rayed had pneumonic changes. Control group was small. Exposure assessment was vague.	4.8 (1.7 to 13.6)
Rural Nepal (1984–85) Kathmandu Valley (Pandey *et al*)[53]	Cohort, 0–23 months, 780 (study 1), 455 (study 2)	Two-weekly home visits: ARI grades I–IV (Goroka) Breathlessness	Asked mothers for average hours per day the child near fireplace. In study 1, same team asked about exposure and ARI > possible bias 77% exposed over 1 hour	Since homes were "homogeneous" confounding not taken into account	Dose response relationship found Exposure assessment not validated	2.2 (1.6 to 3.0)
Rural Gambia (1987–88) Basse (Campbell *et al*)[54]	Cohort, 0–11 months, 280	Weekly surveillance. Mother's history of "difficulty with breathing" over subsequent 3 month period	Reported carriage of child on the mother's back Prevalence = 37%	Adjusted for • birth interval • parental ETS • crowding • socioeconomic score • nutritional indicators • vaccination status • no. of health centre visits • ethnic group • maternal education • other	Father's ETS only other significant factor. Cautious about interpretation, ability to deal with confounding, and to establish causation where exposure and incidence high	2.8 (1.3 to 6.1)
Urban, Argentina (1984–87) Buenos Aires (Cerqueiro *et al*)[67]	Case-control, 0–59 months Cases: 516 inpatients; 153 outpatients, Controls: 669	Three hospitals: Cases: ALRI within previous 12 days Controls: well baby clinic or vaccination, matched by age, sex, nutritional status, socioeconomic level, date of visit, and residence.	Interview with mother: Household heating by charcoal; heating with any fuel; bottled gas for cooking	None, but success of matching verified. Multivariate analysis "currently underway"	No data available re charcoal heating in outpatient households. Chimney smoke nearby found to be associated (OR 2.5–2.7) with ARLI in both kinds of patients. ETS not significant for either	9.9 (1.8 to 31.4) for charcoal heat for inpatients 1.6 (1.3 to 2.0) for any heating fuel in inpatients 2.2 (1.2 to 3.9) for gas cooking in outpatients
Rural Zimbabwe (?) Marondera (Collings *et al*)[57]	Case control, 0–35 months, 244 cases, 500 controls	Hospital: Cases: Hosp ALRI, clinical and *x* ray. Controls: Local well baby clinic	(a) Questionnaire on cooking/exposure to woodsmoke (b) COHb (all) (c) TSP (2 h during cooking): 20 ALRI and 20 AURI cases 73% exposed to open fire	Questionnaire: • maternal ETS • overcrowding • housing conditions • school age sibs • paternal occupation not adjusted	Confounding: only difference was number of school age sibs, but not adjusted. COHb not different between ALRI and AURI. TSP means: ALRI (n=18) 1915 $\mu g/m^3$ AURI (n=15) 546 $\mu g/m^3$	2.2 (1.4 to 3.3)
Rural Gambia (?) Upper River Division (Armstrong and Campbell)[56]	Cohort, 0–59 months, 500 (approx.)	Weekly home visits: ALRI clinical and *x* ray	Questionnaire: Carriage on mother's back while cooking	Questionnaire: • parental ETS • crowding • socioeconomic index • number of siblings • sharing bedroom • vitamin A intake • no. of wives • no. of clinic visits Adjusted in MLR	Boy/girl difference could be due to greater exposure. Report carriage on back quite a distinct behaviour so should define the two groups fairly clearly with low level of misclassification	Approach (1) (all episodes) M: 0.5 (0.2 to 1.2) F: 1.9 (1.0 to 3.9) Approach (2) (1st episode) M: 0.5 (0.2 to 1.3) F: 6.0 (1.1 to 34.2)
Urban Nigeria (1985–86) Ibadan (Johnson and Aderele)[62]	Case control, n=103+103, 0–59 months	Cases: Hospitalised for ALRI (croup, bronchiolitis, pneumonia, empyema thoracis) based on clinical, *x* ray, and biolab workup. Controls: infant welfare clinic, age and sex matched, no respiratory disease	Interview Type of cooking fuel used at home (wood, kerosene, gas)	None	Age, nutritional status, ETS, crowding, and location of cooking area also not significant.	NS

Urban Nigeria (1985–86) Ibadan (Johnson and Aderele)[62]	Case fatality, n=103, 0–59 months	Cases: Death in hospital among ALRI patients (see above)	Interview Type of cooking fuel used at home (79 = kerosene, gas = 5, wood = 16, other = 3)	None	Overall case fatality rate = 7.8%. 5 of 8 deaths were from wood burning homes; one additional death had partial exposure to wood smoke. Poor nutrition (1.8×), low income (1.5×), low maternal literacy (2.1×) were more frequent in wood burning homes. ETS rates were similar. Yet paternal income, maternal education, household crowding, ETS not related to case fatality rate	12.2 (p<0.0005) for those exposed to wood smoke compared with those exposed to kerosene and gas
Rural Tanzania (1986–87) Bagamoyo District (Mtango *et al*)[60]	Case-control Cases: ALRI deaths = 154 Other deaths = 456 Controls = 1160 0–59 months	Cases: Verbal autopsy certified by physician of all deaths in period. Controls: Multistage sampling (40 of 76 villages). Children with ALRI were excluded	Household interview; ● Child sleeps in room where cooking is done ● Cook with wood	Village, age, questionnaire respondent, maternal education, parity, water source, child eating habit, whether mother alone decides treatment.	About 95% of all groups cook with wood. No tendency to be different distances from road. Perhaps confusion of ALRI with other diseases (e.g. measles). Water not from tap had OR = 11.9 (5.5 to 25.7). Models with all deaths, pneumonia deaths, and non-pneumonia deaths all had same significant risk factors. No difference in source of treatment by location where child sleeps. Maternal education, religion, crowding, and ETS, not significant	All deaths: 2.8 (1.8 to 4.3) for sleeping in room with cooking. 4.3 for pneumonia only. 2.4 for other deaths
Rural Gambia Upper River Division (de Francisco *et al*)[61]	Case-control Cases: 129 ALRI deaths Controls: 144 other deaths 270 live controls 0–23 months	Cases: Verbal autopsy confirmed by 2 of 3 physicians. Controls: Matched by age, sex, ethnic group, season of death, and geographic area	Indoor air pollution index based on location and type of stove, carrying of child while cooking, and parental ETS (details not provided)	Cases vs. live controls: Adjusted for significant factors in univariate analysis: socioeconomic score, crowding, parental ETS, and nutrition indicators plus maternal education. No significant factors for cases vs. dead controls.	Only other significant risk factor remaining after multiple conditional logistic regression was whether child ever visited welfare clinic OR = 0.14 (0.06 to 0.36) Misclassification of ALRI deaths (e.g. confusion with malaria) is possible reason for lack of significant difference between cases and dead controls.	5.2 (1.7 to 15.9) for cases vs. live controls
Urban Brazil (1990) Porto Alegre (Victora *et al*)[64]	Case control, 0–23 months, 510 cases, 510 controls	Cases: ALRI admitted to hospital, clinical and X-ray. Controls: Age matched, neighborhood	Trained field worker interview: ● Any source of indoor smoke (open fires, woodstoves, fireplaces) ● Usually in kitchen while cooking	Interview: ● cigarettes smoked ● housing quality ● other children in hh ● income/education ● day centre attendance ● history of respiratory illness ● (other) Hierarchical model/MLR	Only 6% of children exposed to indoor smoke. Urban population with relatively good access to health care. Not representative of other settings in developing countries	Indoor smoke: 1.1 (0.61 to 1.98) Usually in the kitchen: 0.97 (0.75 to 1.26)
Urban and rural India (1991) South Kerala-Trivandrum (Shah *et al*)[63]	Case control, 2–60 months, 400 total	Hospital: Cases: Admitted for severe/very severe ARI (WHO definition). Controls: Outpatients with non-severe ARI	History taken, including ● type of stove, with "smokeless" category ● outdoor pollution	History: ● smokers in house ● number of siblings ● house characteristics ● socioeconomic conditions ● education ● birth weight etc. Adjusted in MLR	This is a study of the risk factors for increased severity, as the controls have ARI (non-severe). On MLR, only age, sharing a bedroom, and immunisation were significant. Exposure assessment was vague and invalidated	"Smokeless" stove: 0.82 (0.46 to 1.43).
Rural Gambia (1989–1991) Upper River Division (O'Dempsey *et al*)[55]	Prospective case-control, n=80+159, 0–59 months	Attending clinic. Cases: if high respiratory rate, transported to Medical Research Council where physician diagnosed pneumonia after lab tests and *x* ray. Controls: selected randomly from neighbourhood of cases, matched by age	Household questionnaire: Mother carries child while cooking	Adjusted for mother's income, ETS, child's weight slope, recent illness, and significant illness in last six months.	No effect of bednets, crowding, wealth, parental education, paternal occupation, age of weaning, and nutritional status. ETS OR = 3.0 (1.1 to 8.1). Aetiological (preventive) fraction for eliminating maternal carriage while cooking = 39%; for eliminating ETS in house = 31%. May be reverse causality, i.e. sick children being more likely to be carried.	2.5 (1.0 to 6.6)

This list is confined to quantitative studies that have used internationally standardised criteria for diagnosing ALRI. There are additional studies that have noted a relationship with various respiratory symptoms including cough, runny nose, noisy respiration, and sore throat—for example, the study in Lucknow, India by Awasthi *et al*[68] which is discussed in the text.

Table 6 Wood burning and ALRI in children under five in developed countries

Study	*Design*	*Case definition*	*Exposure*	*Confounding adjusted*	*Comments*	*OR (95% CI)*
Navaho reservation (1988) Tuba City, Arizona, USA (Morris *et al*)[69]	Case-control 0–24 months n=58+58	Hospital: Cases: ALRI, bronchiolitis, pneumonia clinical and *x* ray Controls: Age-sex matched, well child clinic	Interview: Primary energy source for heating and cooking	Family history of asthma, recent exposure to respiratory disease, dirt floor, presence of running water.	Wood burning stoves with chimneys but exposure levels not validated. Recent exposure to respiratory disease only other factor remaining significant (OR 1.4) after multivariate analysis. Humidifiers, ETS, pets, crowding, and house type not significant.	4.8 (1.7 to 12.9)
Navaho reservation (1993) Fort Defiance, Arizona, USA (Robin *et al*)[70]	Case-control 1–24 months n=45+45	Hospital: Cases: ALRI, bronchiolitis, pneumonia Controls: Age-sex matched, sought care not for other conditions	Interview: cook with wood Measured 15 h PM_{10} levels (5 pm–8 am)	Interview • children/hh • running water • electricity • difficulty of transport to clinic • ETS • house type	No variation in PM_{10} levels with ETS, type of home, etc. Type of cooking/heating only explained 10% of variance. Median PM_{10} levels 24 μg/m^3 (cases), 22 μg/m^3 (controls). No effect for coal use or wood for heating, but sample sizes small	Cook with wood 5.0 (0.6 to 43) PM >65 μg/m^3 7.0 (0.9 to 57)

As in table 5, this list is confined to those quantitative studies using standardised protocols for determining ALRI. Other studies have just looked at the relationship of wood burning with respiratory symptoms, e.g. Honicky *et al*,[71] Butterfield *et al*,[72] and Browning *et al*[73] which are discussed in the text.

Nepal study did not show greater effects in infants than during the second year of life. In a detailed analysis of data from the Gambia, Armstrong and Campbell[56] found that the risk of pneumonia in association with smoke exposure was increased in girls but not in boys. The authors suggested that this difference resulted from greater exposure of females and not from biological differences between the sexes. The studies variably considered potential confounding in their design and analytical approaches (tables 6 and 7). Inadequate control of confounding is likely to result in an overestimate of the odds ratios, since the use of open fires and biomass fuels is associated with poverty and associated risk factors for ALRI.

Other possible sources of bias include misclassification of exposure through recall bias. Case-control studies are more likely to be subject to recall bias, although such bias can also occur in prospective studies when collection of exposure data follows the occurrence of illness. For example, in the study of Zulu children in Natal by Kossove[58] the reported duration of smoke exposure was remarkably similar in cases and controls, though the proportion of women reporting exposure of the child to smoke (determined by questionnaire) was much higher in cases.

Table 7 Summary of studies of ALRI in young children and indoor biomass smoke in developing countries

Case-control studies (n = 9)	
(South Africa, Zimbabwe, Nigeria, Tanzania, Gambia (2), Brazil, India, Argentina)	
6 adjusted for confounders	n=4311
3 not significant	Odds ratios = 2.2–9.9
Cohort studies (n = 4)	
(Nepal, Kenya, Gambia (2))	
2 adjusted for confounders	n=910
1 not significant	Odds ratios = 2.2–6.0
Case-fatality study (n = 1)	
(Nigeria)	
Hospitalised patients	n=103
	Odds ratio = 4.8
Developed countries (n = 2)	
(USA (2))	
Case-control	n=206
Adjusted for confounders	Odds ratios = 4.8–7.0

The dividing line between developed and developing countries = $1000 per capita purchasing power in 1995 (UNDP, 1998).[116]

Bias in case-control studies from differential use of health services

Differential use of health services could introduce bias if the subjects who use health services for serious paediatric illnesses, but not for mild illnesses or preventive care, are also those who use unprocessed biomass fuels for cooking and who take no measures to avoid exposing their young children to the smoke. In one of the case-control studies from Africa, but not the others, breast feeding patterns and socioeconomic status of cases and controls differed.[57] In the case-control study by Kossove[58] it is not clear whether the clinic controls were less sick. This approach to control selection could introduce bias if caretakers whose children were more heavily exposed to indoor air pollution were less likely than others to bring their children to these services when they were only mildly unwell or for preventive care, but were just as likely as others to bring their children when seriously ill. This would result in heavily exposed children being underrepresented in the control groups and bias of the odds ratio away from unity. This situation could arise due to distance of such households from the clinics, inability to afford the cost of transport, or from other constraints associated with poverty. In principle, this scenario is quite possible since those households with the highest exposures are also those most likely to be poor, with less access to transport, etc.

In practice it is difficult to assess whether this bias has contributed to the risk estimates in the studies quoted since care seeking has not been studied directly. From information available on socioeconomic circumstances in three studies, however, there appears to be little difference between cases and controls.[57 58 62] This source of bias was discussed by Morris *et al*[69] as in their study there was some evidence that socioeconomic circumstances (dirt floor, lack of running water) were poorer among cases. It was reported, however, that over 90% of children born in the catchment area of the hospital completed routine immunisation, suggesting that the control sample from the well baby clinic was likely to represent the population.

The study by Shah *et al*[63] provided limited information on socioeconomic circumstances for cases and controls, and in any case did not report an increased odds ratio for smoke exposure. The study from urban Argentina by Cerqueiro *et al*[67] matched on five factors including socioeconomic status and district of residence. Overall, it appears that this bias was probably not important in this group of case-control studies, although without specific information on care seeking it remains a possible source of error.

The study by Cerqueiro *et al*[67] found a large odds ratio (9.9, 95% CI 1.8 to 31) for home heating with "charcoal" in patients with hospital diagnosed ALRI compared with controls matched by socioeconomic level, nutritional status, and other factors often addressed only by multivariate analysis in other studies (table 5). No pollution measurements were reported and little information was provided about the type of stove and fuel involved. Cooking with gas (rather than electricity) also produced a significant odds ratio (2.2, 95% CI 1.2 to 3.9).

It is intriguing to note that the three studies that found no significant association were the only ones which relied on questionnaires to determine what type of cooking stove or fuel was used at home without additional information about family behaviour patterns. In Kerala, India the measure of exposure was a question about the existence of a "smokeless" stove (with a flue) at home.[63] Unfortunately, however, such stoves in India often do not actually lower indoor air pollution levels.[66] The Brazil study took place in a city where the prevalence of household cooking with wood was quite low (6%).[64] The case-control study reported by Johnson and Aderele in Nigeria found no significant association of ALRI morbidity with reported type of household fuel, but did find a strong relationship of fuel type with case fatality.[62]

Other studies

A study of ARI in infants aged less than one year in India,[74] which did not qualify for table 5 because of its broad definition of ALRI, found somewhat conflicting results in urban slum communities where some households used biomass fuels and others kerosene. This was possibly due to strong interference by large scale urban outdoor pollution and local outdoor "neighbourhood" pollution from the cooking stoves themselves and other neighbourhood sources. Another study not qualifying for inclusion in table 5 because of its inclusive case definition was an observational study of 650 randomly chosen pre-school children aged 1–59 months in Lucknow, India, 14.5% of whom were found to have respiratory disease as defined by runny nose, cough, sore throat, breathlessness, or noisy respiration.[68] After adjusting for age, weight, sex, income, and house type, use of dung as cooking fuel (OR 2.7, 95% CI 1.4 to 5.3) and crowding (OR 1.2, 95% CI 1.1 to 1.4) were associated with one or more of these respiratory symptoms. The location of the child during cooking, ETS, and cooking with coal, kerosene, or wood were not associated with respiratory symptoms in this study. Somewhat different results were obtained in the previously discussed six month prospective study of 650 children aged 1–53 months in the same area. With fortnightly household visits, a significant association of symptoms and/or duration of symptoms was found with outdoor TSP measurements.[75] After multivariate analysis, cooking with any of the solid fuels (ORs 1.3 (wood); 1.6 (coal); 1.5 (dung)) or kerosene (OR 1.4) and being indoors while cooking took place (OR 2.0, 95% CI 1.7 to 2.4) were also significantly associated. Morbidity due to "probable pneumonia" was also determined by cough and difficulty in breathing and was found only to be weakly but significantly related to the use of dung fuel (OR 1.01, 95% CI 1.00 to 1.02).

A study of 658 children aged 0–6 years in Jakarta found that, although respiratory symptom rates were, after multivariate analysis, related to evidence of uncollected refuse around the house (OR 1.6), they were not related to the type of cooking fuel used.[76] The author speculates that the sample size of households using wood burning stoves (not given) was too small to find an effect, and that the impact of the refuse may be a result of the smoke generated by its frequent burning.[76]

A large national household survey in India found a statistically significant relationship (OR 1.3) between reported use of household biomass fuel and reported incidence of respiratory infection in the previous week among children under five years.[77] Since the survey did not distinguish cases by ALRI, URI, or severity, however, it probably is not a good predictor of the risk of severe, life threatening ALRI.

Mortality from pneumonia in developing countries

An association between exposure to household biomass pollution and mortality from pneumonia has been shown in one study of ALRI in Nigeria.[62] Although a case-control study in the same hospital did not reveal a relationship between type of cooking fuel and hospital admissions for ALRI, the children with ALRI who came from homes that burnt wood were 12.2 times ($p<0.0005$) more likely to die than those coming from homes using kerosene or gas (table 5). Even though wood burning homes were characterised as a group by poorer nutritional status, lower income, and less maternal literacy, neither these factors nor crowding nor smoking were related to case fatality rates. Unfortunately, no multivariate analysis was reported and the case sample size was small (eight deaths in 100 ALRI cases).

Morbidity studies indicate that smoke pollution is a risk factor for both milder and more severe cases of lower respiratory disease. Effective strategies for pneumonia case management will modify the relationship between the incidence of pneumonia and mortality.[53] As long as pneumonia fatality rates remain high, an association between pneumonia mortality and exposure to smoke pollution will remain of concern.

Incidence of ALRI in young children from developed countries

Studies of the health effects of biomass smoke in developed countries have focused on households using enclosed metal heating or cooking stoves with chimneys. The indoor pollutant concentrations are normally substantially less than those found in village homes using open fires.[47] Peak indoor particulate concentrations resulting from leaky heating stoves in developed country homes are at most several hundred μg/m^3 and are typically much less than the peak values of many thousand μg/m^3 in village homes cooking with biomass fuel.[25] Nevertheless, the impact on ALRI has been shown in the two studies summarised in table 6.

Although not increased to the extent suffered by children from developing countries, the age adjusted ALRI mortality rate of Native American children has been some six times that of non-Hispanic white children. A study of young Navaho children in Arizona found that household cooking/heating with woodstoves (with flues) produced a significant odds ratio (4.8, 95% CI 1.7 to 12.9) after multivariate analysis for physician-confirmed ALRI in hospitalised patients using radiographs.[69] Cases were thus confirmed as bronchiolitis or pneumonia, but no information was given on the mix. A second study was designed to address factors not covered in the first—in particular, to include the difficulty of reaching the clinic in the multivariate analysis and to actually measure indoor air pollution levels.[70] Cases included LRI, pneumonia, and bronchiolitis, ascertained from the hospital's inpatient records. In this case a similar but non-significant odds ratio was found (5.0, 95% CI 0.6 to 43). The median 15 hour PM_{10} levels in both sets of households (measured once), however, were quite similar (table 6) and were much lower than those found in developing countries. Further analysis revealed that PM_{10} levels over 65 μg/m^3 (90th percentile) were related to ALRI, but with a broad confidence band (OR 7.0, 95% CI 0.9 to 57), and that type of cooking/heating only explained 10% of the variance.

Three other US studies of respiratory symptoms in young children exposed to wood smoke did not qualify for inclusion in table 6. Honicky *et al*[71] performed a historical prospective study of 68 preschool children, half of whom came from homes with wood heating stoves, and found significantly more respiratory symptoms in the exposed group. Careful matching was done to assure that the groups did not differ by income, ETS, residence, etc, but no multivariate analysis was reported. Butterfield *et al*[72] found a significant correlation between hours of reported wood stove use and five of 10 respiratory symptoms in 59 children aged less than 66 months. Browning *et al*,[73] on the other hand, found no significant relationship between respiratory symptoms in 823 children aged over one year and location in high or low wood smoke neighbourhoods. However, a non-significant trend was observed in those aged 1–5 years. Ambient monitoring showed an approximate difference of 20 μg/m^3 in PM_{10} levels between the two neighbourhoods.

Morbidity in school aged children

Exposure to pollution from wood stoves has been associated with chronic respiratory symptoms, changes in lung function, and/or hospital visits in studies of school aged children in the USA exposed to wood smoke from stoves in their own home and/or their neighbourhoods. Larson and Koenig[78] reviewed six such studies in school aged children, two of which dealt with asthmatics and five of which found significant risks. (An earlier review can be found in Honicky and Osborne.[79]) The one study lacking statistical significance[80] was based on telephone interviews with 399 households. In addition, the Harvard Six Cities study of air pollution found use of wood stoves to be associated with a 30% increase in respiratory illness (chronic cough, bronchitis, chest illness, wheeze or asthma) in a large sample of children aged 7–10 years.[81] Such studies suggest an adverse effect of biomass pollution on lung function and are consistent with irritation and inflammation of airways and impaired host defences.

Similar associations were not found in two studies in Malaysia. In a study involving 12147 12-year-old children the presence of a wood or kerosene stove in the home was inversely associated with the forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1), but in a multiple logistic regression analysis that included passive smoking the association with stove type was no longer significant.[82] A more detailed analysis of 1501 7–12-year-old children found, after multivariate analysis, that use of mosquito coils was significantly related to maternally reported wheeze (OR 1.4) and asthma (OR 1.4) and ETS with chest illness (OR 1.7). No association was found with type of cooking fuel.[83]

In Papua New Guinea Anderson[84] also failed to show a difference in rates of respiratory symptoms or lung function in studies comparing children exposed to different levels of smoke. In the first study of 1650 highland and lowland children under 10 years, both groups were found to have similar rates of loose cough, adventitia and past chest illness, despite higher levels of smoke exposure in the highlands. In fact, higher rates of asthma and wheeze were found in coastal children. In a much smaller study involving 112 highland children, those exposed to smoke in their village homes were found to have the same prevalence of respiratory symptoms and similar lung function as their counterparts living in nearby government housing.

More recent studies in developing countries, however, have found effects in school aged children. A study in Adana, Turkey found by questionnaire in a group of 617 9–12-year-olds that those in homes heated with coal had significantly more cough than those using kerosene, oil, or electricity.[85] The lowest statistically different lung functions (FVC, FEV_1, PEFR, FEF_{25}) were in children from wood burning homes. A similar study of 1905 7–13-year-olds in Jordan found that open wood

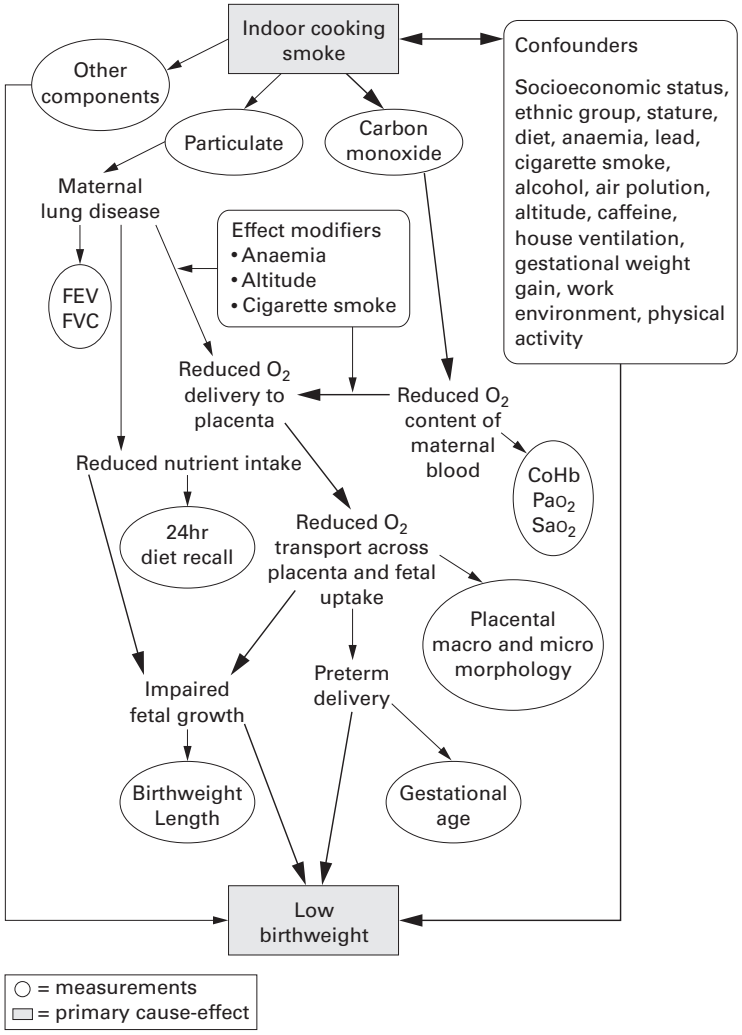

Figure 4 Pathways relating smoke exposure and childhood health. Reproduced with permission from Hass JD, 'Potential mechanisms for the effect of indoor cooking smoke on fetal growth'. Invited paper presented at WHO Workshop on 'The Impact of Indoor Cooking Smoke on Health', Geneva, Switzerland, 26–29 February 1992.

and/or kerosene burning was statistically related to lower lung function with about twice the negative impact of ETS.[86] No multivariate analyses were reported for these studies.

Impact on known precursors of ARI

One mechanism by which biomass smoke and other air pollution exposures could enhance the risk of ARI in young children would be by in utero exposures via their mothers who, when cooking, can be heavily exposed. Adverse pregnancy outcomes such as low birth weight are known ARI risk factors[1] through reduced immunocompetence and/or impaired lung function.[87] As shown in fig 4, there are several pathways by which low birth weight and other adverse pregnancy outcomes may result from heavy maternal air pollution exposures. Providing evidence of such in utero effects, a case-control study of 451 stillbirths in Ahmedabad, India[88] found after multivariate analysis that cooking with biomass fuel was associated with a statistically significant increased chance of stillbirth (OR 1.5, 95% CI 1.0 to 2.1). A study in Bohemia found significant intrauterine growth retardation in babies born to women exposed to increased levels of small particles.[89] A recent cohort study of nearly 75 000 births in Beijing[90] found a 6.9 g decrease in birth weight for each 100 μg/m^3 of ambient TSP, which translated to an odds ratio of 1.1 (95% CI 1.05 to 1.14) for low birthweight babies (<2500 g). A study in highland Guatemala found, after adjusting for confounding, a statistically significant decrease of 63 g in birth weight of infants born to mothers cooking with wood rather than gas.[91] Given that indoor TSP exposures in wood burning homes of highland Guatemala are probably at least 1000 μg/m^3 higher than those in homes using gas,[92] the results of these two studies are remarkably consistent. Lower birth rates have also been associated with ETS exposures to pregnant women.[93]

As indicated in fig 4, particle levels may be serving as surrogates for carbon monoxide (CO) exposures in these birth outcome studies. CO, which is also associated with biomass use, has well established mechanisms for producing low birthweight infants. Indeed, a recent cross sectional study of 125 000 birth weights in southern California found an odds ratio of 1.22 (95% CI 1.03 to 1.44) for low birthweight infants (1000–2499 g) born to mothers experiencing more than 5.5 ppm ambient CO during their last trimester. Adjustment was made for a range of socioeconomic and other potential confounders, but not active smoking or ETS.[94] A time series study in Sao Paulo found similar levels of CO to be associated with excess intrauterine mortality (0.022 increase per ppm), although even stronger associations with an index combining CO, NO_2, and SO_2 levels.[95] Given that indoor biomass use commonly results in 24 hour indoor CO levels of many tens of ppm,[7] there would seem to be potential for high in utero risks in households in less developed countries leading to, among other problems, excess disease risk in infancy.

Agreement with ETS and outdoor pollution studies

The evidence on health effects from use of biomass fuels should be interpreted with consideration of data from studies of other indoor pollutants that may act through comparable toxicological mechanisms to adversely affect respiratory illness. There is sufficient overlap between some components of biomass smoke and components of other investigated mixtures to justify considering this large additional body of evidence.[7] These other pollutants include ETS and ambient pollution with particles by fossil fuel combustion.

Smokes from biomass fuels contain particles, aldehydes, and other irritant gases[37 96 97] that are also found in ETS, which of course is also the result of burning a form of biomass. While undoubtedly there are differences between ETS and these other biomass smokes, the well documented adverse effects of ETS on the respiratory health of children complement the epidemiological findings on smoke from biomass fuel. As discussed earlier, an extensive epidemiological literature documents an association between exposure to ETS and

increased ALRI in infants and young children, an association that has been judged as causal.[32]

Similarly, a large literature, primarily based on studies of various pollutants in outdoor air in urban settings, also shows adverse effects of particles and gases on the respiratory health of children. Existing studies are likely to underestimate the size of the association between ambient pollution and health as a result of misclassification of the exposure status of individuals within populations.

Conclusions

Indoor and outdoor environments are widely contaminated by complex mixtures of gases and particles that are produced by combustion. Components of these mixtures have been shown to adversely affect host defences against respiratory infections and it is thus plausible that such pollutant mixtures increase the incidence of respiratory infections. Air pollutants might also increase the severity of respiratory infections by causing inflammation of the lung airways and alveoli. Infants and young children are particularly susceptible to these adverse effects because of the immaturity of respiratory defence mechanisms and the geometry of the airways. Patterns of time-activity, which place children near sources of pollution such as cooking stoves, cigarettes, vehicle exhaust, or other contaminated environments, may contribute to the increased risk of ARI from airborne pollutants in young children.

This review documents the potential for preventing ARI in general, and pneumonia in particular, in children by reducing exposures to air pollution. Combustion of household solid fuels in developing countries produces exposures to smoke components that are remarkably high by the standards set for outdoor air in developed countries. Adverse effects of these exposures would be anticipated on a toxicological basis. Although the epidemiological evidence on smoke from biomass fuels and pneumonia is not yet abundant, associations have been demonstrated between exposure measures and indicators of illnesses involving the lower respiratory tract. When interpreted within the broad framework of epidemiological and toxicological evidence on inhaled pollutants and ARI, the association of smoke from biomass fuels with ARI should be considered as causal, although the quantitative risk has not been fully characterised.

Risk estimates from individual studies are imprecise because of relatively small sample sizes and misclassification of exposure and outcome. Given the imprecision and uncertainty in characterising the risk of biomass smoke exposure, quantitative risk assessments cannot be offered with great confidence. On the other hand, the large population of children exposed and even our limited database on levels of exposure implies a significant burden of attributable ARI. The extent to which excess biomass smoke can be prevented is uncertain, however, because of the lack of information on exposure-response relationships. We urge further research directed at the time-activity patterns of children under the age of five years as well as studies designed to characterise total personal exposures and the contributions of indoor and outdoor pollution sources to children's exposures in developing countries. The resulting data would facilitate the design of additional case-control and cohort studies to better quantify the relationship between smoke exposure and ARI and to identify the most effective intervention strategies.

Unlike most sources of ambient air pollution, however, household sources of exposure such as cooking and heating offer the opportunity for conducting randomised trials of potential interventions, both engineering and behavioural. Thus, of even higher priority than further observational studies is the promotion of well designed randomised intervention trials in households in less developed countries in conjunction with careful exposure assessment. Data from intervention studies could quantify exposure-response relationships for ARI, convincingly demonstrate to policy makers the health benefits of practical interventions such as clean fuels, improved stoves, and householder education and, ironically, given past scientific inattention to this particular problem, move air pollution epidemiology in general closer to the "gold standard" of randomised clinical trials.

Globally, even though the attributable fraction of pneumonia/ARI mortality due to air pollution is not yet certain, it is probable that this disease outcome represents the largest class of health impacts from air pollution exposure worldwide. This is likely to be the case in terms of total morbidity and mortality but, because much of the burden falls on young children, is almost certainly the case with regard to measures of ill health that consider the lost life years involved. This is due to three factors: (1) the relatively high odds ratios apparently involved (table 5), (2) the seemingly high and prevalent exposures in less developed countries, particularly in households (fig 2), and (3) the high base rate of the disease in these nations (table 2).

Relatively recently there has been a significant increase in attention in many developed countries to issues related to "environmental justice"—that is, the unfortunate tendency for the highest exposures to environmental pollutants to be experienced by some of the most disadvantaged populations.[98] Globally, however, even more egregious examples of this injustice prevail. Indeed, few if any large groups are more disenfranchised and disadvantaged than poor rural women in developing countries and their young children, who experience the bulk of global airborne exposures to many pollutants.

Some readers may be surprised by our conclusion that ARI in children represents one of the major health consequences of air pollution globally. This conclusion contrasts with the limited epidemiological research on air pollution and ARI in developed countries. Indeed, the evidence driving policy for air pollution control at present derives largely from studies of elderly persons. We suggest that this seeming paradox reflects a failure to systematically

focus studies of air pollution and health on the populations receiving the highest exposures.[99 100] These populations may not necessarily correspond with the locations where the greatest pollutant emissions occur. We need an organised international effort to monitor, evaluate, and mitigate air pollution in the places where people live and work. A principal goal of this effort should be rapid reduction of the alarming global burden of ARI.

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