Indoor air pollution in developing countries: a major environmental and public health challenge

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Around 50% of people, almost all in developing countries, rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. These materials are typically burnt in simple stoves with very incomplete combustion. Consequently, women and young children are exposed to high levels of indoor air pollution every day.

There is consistent evidence that indoor air pollution increases the risk of chronic obstructive pulmonary disease and of acute respiratory infections in childhood, the most important cause of death among children under 5 years of age in developing countries. Evidence also exists of associations with low birth weight, increased infant and perinatal mortality, pulmonary tuberculosis, nasopharyngeal and laryngeal cancer, cataract, and, specifically in respect of the use of coal, with lung cancer. Conflicting evidence exists with regard to asthma. All studies are observational and very few have measured exposure directly, while a substantial proportion have not dealt with confounding. As a result, risk estimates are poorly quantified and may be biased. Exposure to indoor air pollution may be responsible for nearly 2 million excess deaths in developing countries and for some 4% of the global burden of disease.

Indoor air pollution is a major global public health threat requiring greatly increased efforts in the areas of research and policy-making. Research on its health effects should be strengthened, particularly in relation to tuberculosis and acute lower respiratory infections. A more systematic approach to the development and evaluation of interventions is desirable, with clearer recognition of the interrelationships between poverty and dependence on polluting fuels.

Keywords: air pollution, indoor – adverse effects; fossil fuels – toxicity; lung diseases; smoke inhalation injury; cataract; developing countries.

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Introduction

Indoor air pollution can be traced to prehistoric times when humans first moved to temperate climates and it became necessary to construct shelters and use fire inside them for cooking, warmth and light. Fire led to exposure to high levels of pollution, as evidenced by the soot found in prehistoric caves (*1*). Approximately half the world's population and up to 90% of rural households in developing countries still rely on unprocessed biomass fuels in the form of wood, dung and crop residues (*2*). These are typically burnt indoors in open fires or poorly functioning stoves. As a result there are high levels of air pollution, to which women, especially those responsible for cooking, and their young children, are most heavily exposed. (Fig. 1).

In developed countries, modernization has been accompanied by a shift from biomass fuels such as wood to petroleum products and electricity. In developing countries, however, even where cleaner and more sophisticated fuels are available, households often continue to use simple biomass fuels (*3*). Although the proportion of global energy derived from biomass fuels fell from 50% in 1900 to around 13% in 2000, there is evidence that their use is now increasing among the poor (*1*). Poverty is one of the main barriers to the adoption of cleaner fuels. The slow pace of development in many countries suggests that biomass fuels will continue to be used by the poor for many decades.

Notwithstanding the significance of exposure to indoor air pollution and the increased risk of acute respiratory infections in childhood, chronic obstructive pulmonary disease and lung cancer (*3, 4*), the health effects have been somewhat neglected by the research community, donors and policy-makers. We present new and emerging evidence for such effects, including the public health impact. We consider the prospects for interventions to reduce exposure, and identify priority issues for researchers and policy-makers.

Biomass fuel is any material derived from plants or animals which is deliberately burnt by humans. Wood is the most common example, but the use of animal dung and crop residues is also widespread (*5*). China, South Africa and some other countries also use coal extensively for domestic needs.

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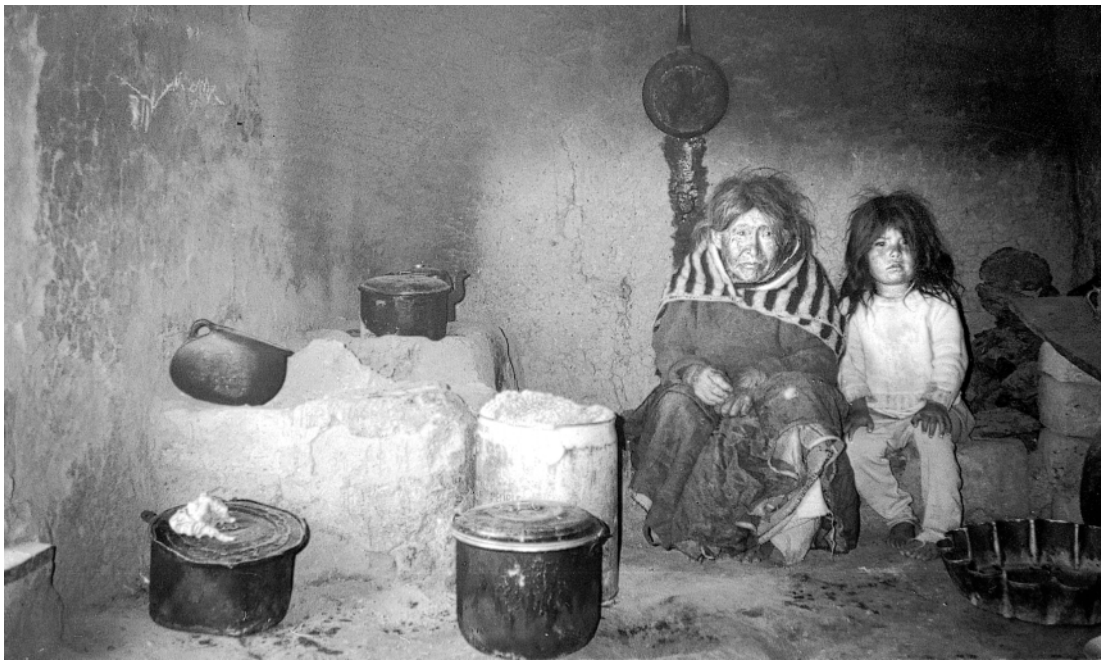
Fig. 1. **A rural home in the highlands of Bolivia with walls blackened by smoke from an open wood fire**



In general the types of fuel used become cleaner and more convenient, efficient and costly as people move up the energy ladder (*6*). Animal dung, on the lowest rung of this ladder, is succeeded by crop residues, wood, charcoal, kerosene, gas and electricity. People tend to move up the ladder as socioeconomic conditions improve. Other sources of indoor air pollution in developing countries include smoke from nearby houses (*6*), the burning of forests, agricultural land and household waste, the use of kerosene lamps (*7*), and industrial and vehicle emissions. Indoor air pollution in the form of environmental tobacco smoke can be expected to increase in developing countries. It is worth noting that fires in open hearths and the smoke associated with them often have considerable practical value, for instance in insect control, lighting, the drying of food and fuel, and the flavouring of foods (*3*).

Many of the substances in biomass smoke can damage human health. The most important are particles, carbon monoxide, nitrous oxides, sulphur oxides (principally from coal), formaldehyde, and polycyclic organic matter, including carcinogens such as benzo[*a*]pyrene (*5*). Particles with diameters below 10 microns (PM_{10}), and particularly those less than 2.5 microns in diameter ($PM_{2.5}$), can penetrate deeply into the lungs and appear to have the greatest potential for damaging health (*8*).

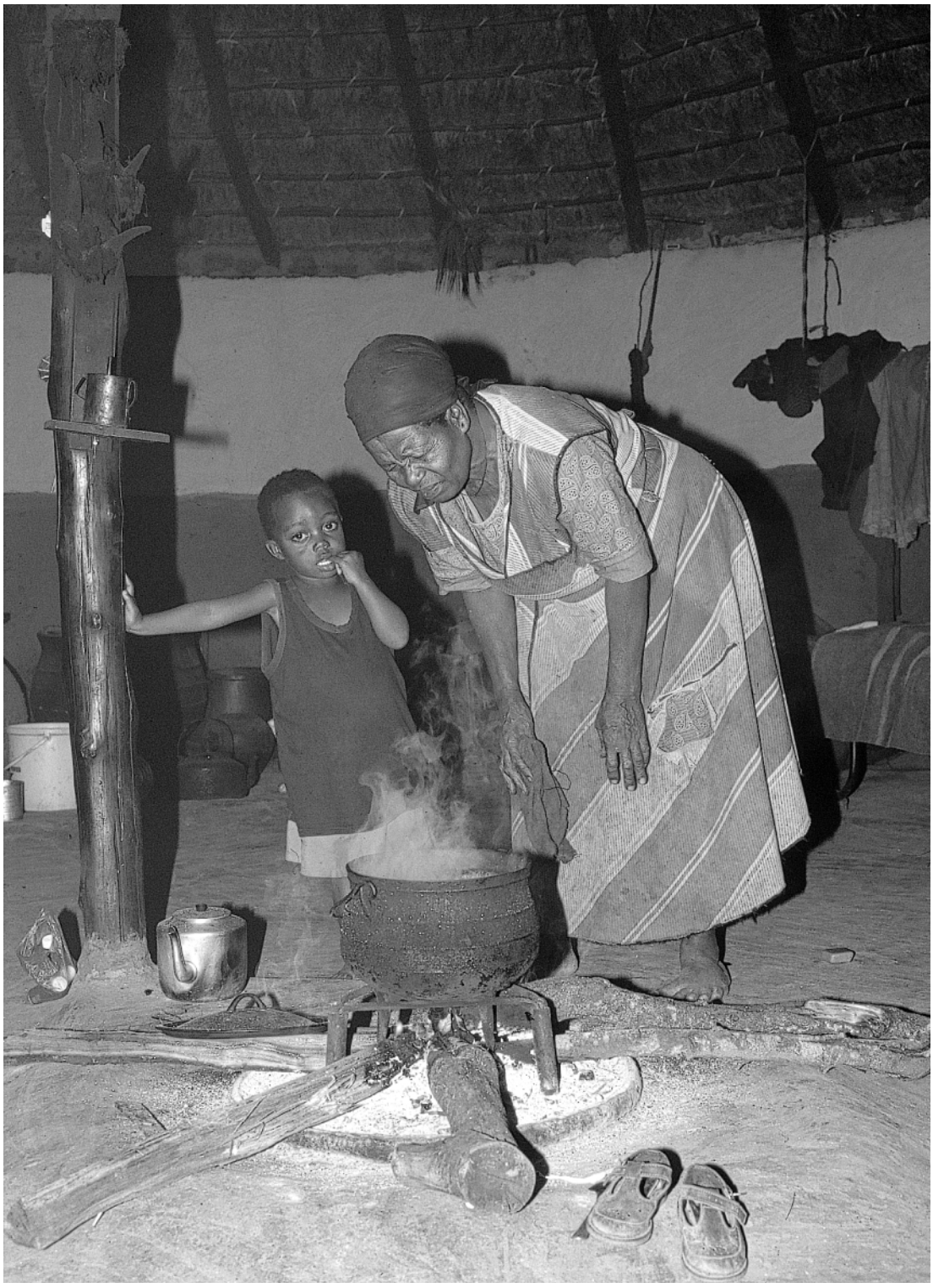
The majority of households in developing countries burn biomass fuels in open fireplaces, consisting of such simple arrangements as three rocks, a U-shaped hole in a block of clay, or a pit in the ground, or in poorly functioning earth or metal stoves (*3*) (Fig. 2). Combustion is very incomplete in most of these stoves, resulting in substantial emissions which, in the presence of poor ventilation, produce very high levels of indoor pollution (*9*). Indoor concentrations of particles usually exceed guideline levels by a large margin: 24-hour mean PM_{10} levels are typically in the range 300–3000 $\mu g/m^3$ and may reach 30 000 $\mu g/m^3$ or more during periods of cooking (*6, 7, 9–20*).

The United States Environmental Protection Agency's standards for 24-hour average PM_{10} and $PM_{2.5}$ concentrations are 150 $\mu g/m^3$ and 65 $\mu g/m^3$ respectively (*8*). The mean 24-hour levels of carbon monoxide in homes using biomass fuels in developing countries are in the range 2–50 ppm; during cooking, values of 10–500 ppm have been reported. The United States Environmental Protection Agency's 8-hour average carbon monoxide standard is 9 ppm or 10 mg/m^3 (*8*).

A health effect is determined not just by the pollution level but also, and more importantly, by the time people spend breathing polluted air, i.e. the exposure level.[a] Exposure refers to the concentration of pollution in the immediate breathing environment during a specified period of time. This can be measured either directly through personal monitoring or indirectly by combining information on pollutant concentrations in each microenvironment where people spend time with information on activity patterns (*21*). Information on such patterns is very important for understanding the dynamic relationship between levels of pollution and behaviour. As pollution levels are reduced it is possible that people will spend more time indoors or nearer the sources of pollution. If this happens a reduction in ambient

[a] Strictly, the dose that determines the health effect. In practice this is a complex issue that is difficult to assess. It is not considered further in this review.

Fig. 2. **A traditional home in KwaZula, Natal, South Africa with an open wood fire**



pollution will not necessarily result in a proportionate decrease in exposure, and there will be important implications for interventions.

People in developing countries are commonly exposed to very high levels of pollution for 3–7 hours daily over many years (*22*). During winter in cold and mountainous areas, exposure may occur over a substantial portion of each 24-hour period (*13*). Because of their customary involvement in cooking, women's exposure is much higher than men's (*23*). Young children are often carried on their mothers' backs while cooking is in progress and therefore spend many hours breathing smoke (*1*).

We concentrate on exposure associated with the use of biomass fuel in populations of developing countries. However, where evidence is particularly limited, we include information concerning relevant exposures to outdoor and indoor air pollution and to

environmental tobacco smoke. We consider respiratory illness, cancer, tuberculosis, perinatal outcomes including low birth weight, and eye disease.

Respiratory illness

Childhood acute respiratory infections

Acute lower respiratory infections. Acute lower respiratory infections are the single most important cause of mortality in children aged under 5 years, accounting for around 2 million deaths annually in this age group. Various studies in developing countries have reported on the association between exposure to indoor air pollution and acute lower respiratory infections (*11, 16, 24–37*). We restrict comment to the studies listed, as these have all used definitions of such infections which conform reasonably closely to current WHO criteria (*38*) or to other definitions that were accepted at the time the studies were carried out and/or include radiographic evidence. A detailed review of this topic has recently been published (*39*).

Ten studies had case-control designs (two were mortality studies), four were cohort studies (all concerned with morbidity), and one was a case-fatality study. Whereas acute lower respiratory infections were relatively robustly defined, the measurement of exposure relied in almost all studies on proxies, including the types of fuel and stove (*11, 27, 29, 30, 32–36*), whether a child stayed in the smoke (*24, 29, 33)* and whether it was carried on the mother's back (*26, 28, 31*) while cooking was in progress, and reported hours spent near the stove (*24, 25*). In the only study in which direct measurements were made of pollution and exposure in a subsample, respirable particles in the kitchens of cases were substantially higher than for controls (1998 $\mu g/m^3$ versus 546 $\mu g/m^3$; $p < 0.01$) but there was no significant difference in carboxyhaemoglobin levels (*11*).

Five studies reported no significant association between the incidence of acute lower respiratory infections and exposure (*30–33, 35, 36*), but the remainder reported significantly elevated odds ratios in the range 2–5 for incidence or deaths. Not all, however, dealt adequately with confounding factors (*11, 24, 25, 27, 30*), although accounting for confounding in studies of this exposure may in any case be problematic (*28, 40*). However, odds ratios in studies that adjusted for confounding were similar in range to those in unadjusted studies.

In several studies in which no association was found, relatively small proportions of the samples were exposed. In urban Brazil, for instance, only 6% of children were exposed to indoor smoke (*33*); in another South American study, 97% of homes used gas for cooking, although 81% used polluting fuels for heating, namely kerosene, wood and coal (*36*). In the latter study, neonates with a birth weight below 2500 g — the group most vulnerable to acute lower respiratory infections — were excluded. In Durban only 19% of cases and 14% of controls used wood or coal stoves (*35*). A so-called smokeless *chullah* (mud hearth) was used in one study as an indicator of lower exposure (*32*), but such stoves can be little better than traditional ones (*41*).

Studies in Navajo communities used case-control designs, reported fuel type (wood versus cleaner) as a proxy for exposure and adjusted for confounding (*16, 37*). They reported elevated odds ratios of approximately 5, although these were not statistically significant in one of the studies (*16*). The latter study also involved measuring 15-hour PM_{10} levels: there were minimal differences between cases and controls, and the actual levels (median 15-hour PM_{10} = 22.4 $\mu g/m^3$, range 3.2–186.5 $\mu g/m^3$) were relatively low. However, children living in homes with PM_{10} levels of 65 $\mu g/m^3$ and above had an odds ratio that was 7.0 times higher than for children with levels below 65 $\mu g/m^3$ (95% confidence interval = 0.9–56.9).

Upper respiratory infection and otitis media. Several studies have reported an association between exposure to biomass fuel smoke and general acute respiratory illness in children, mostly of the upper respiratory tract. Middle ear infection (otitis media) is rarely fatal but causes much morbidity, including deafness, and makes demands on the health system. Untreated, it may progress to mastoiditis. Evidence from developing countries is very limited, but there is good reason to expect an association. There is strong evidence that exposure to environmental tobacco smoke causes middle ear disease: a recent meta-analysis reported an odds ratio of 1.48 (1.08–2.04) for recurrent otitis media if either parent smoked, and one of 1.38 (1.23–1.55) for middle ear effusion in the same circumstances (*42*). A clinic-based case-control study of children in rural New York State reported an adjusted odds ratio for otitis media, involving two or more separate episodes, of 1.73 (1.03–2.89) for exposure to wood-burning stoves (*43*).

Chronic pulmonary disease

Chronic obstructive pulmonary disease. In developed countries, smoking is responsible for over 80% of cases of chronic bronchitis, i.e. inflammation of the lining of the bronchial tubes, and for most cases of emphysema (overinflation of the air sacs in the lungs) and chronic obstructive pulmonary disease (progressive and incompletely reversible airflow obstruction). However, these diseases occur in regions where smoking is infrequent. Patients with chronic lung disease have been reported in communities heavily exposed to indoor biomass smoke pollution in New Guinea. Adults aged over 45 years had a high prevalence of respiratory symptoms and disease, similar in men and in women, and 20% of men and 10% of women had an FEV1/FVC (forced expiratory volume in one second / forced vital capacity) below 60% (*44*). The clinical presentation was as chronic obstructive pulmonary disease with, in a few patients, local lung fibrosis and bronchiectasis (localized destruction and infection of the lung) (*45*), and disease was attributed to indoor air pollution and

repeated infections. Most patients 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 (*46*).

Numerous studies, including ones with cross-sectional and case-control designs, have reported on the association between exposure to biomass smoke and chronic bronchitis or chronic obstructive pulmonary disease (*13, 15, 18, 47–63*). In Nepal, the prevalence of chronic bronchitis was similar in men and women (18.9%); this would not have been expected if cigarette smoking, being commoner in men, had been the main cause (*50, 51*). The prevalence of chronic brochitis was also greater in women in Ladakh, where few women smoke (*13*), and in Pakistan (*59*). Exposure to biomass smoke has been reported as more frequent in people with airflow obstruction in hospital-based case-control studies (*56, 57, 62*) and some community studies (*52, 58, 61*). In hospital-based studies, obstruction was often severe and the association with exposure was strong, adjusted odds ratios being in the range 1.8–9.7. One community study reported an adjusted odds ratio of 2.5 (*18*), but in spirometric studies the reported differences in lung function associated with exposure to wood smoke have usually been relatively small, probably reflecting the selection of much more severe cases in hospital studies. In rural Mexico the use of biomass was associated with a 4% decrease in FEV1/FVC, while an increase in the kitchen particle concentration of 1000 $\mu g/m^3$ was associated with a reduction of 2% in FEV1 (*61*). In India, patients using biomass had lower FVC than those using kerosene, gas and mixed fuels (*58*). Pandey reported an exposure-response relationship with FEV1 and FVC which decreased as the reported hours of exposure increased; it was not statistically significant in non-smokers (*52*). Experience with cigarette smokers suggests that fewer than 15% of people exposed to wood smoke are likely to develop chronic obstructive pulmonary disease or chronic bronchitis, although this may depend on the level of exposure.

Exposure was usually estimated from questionnaires as present or absent, as hours spent close to a wood stove, or as hours multiplied by years of exposure. The studies measuring particle levels in kitchens confirmed very high concentrations (*15, 18, 61*); a time-budget assessment was also made in one of these studies (*18*). Norboo reported the use of kitchen and exhaled personal carbon monoxide levels (*13*). Chronic bronchitis has generally been determined by questionnaire, while spirometry has been employed to determine airflow obstruction and chronic obstructive pulmonary disease. In many of the studies there has been scant attention to quality control.

Clinical characteristics of lung disease. The most common presentation in both community and referral hospital studies of adults is chronic airways disease, particularly chronic bronchitis. Airflow obstruction and shortness of breath (dyspnoea) are typical of patients seen in referral hospitals (*57, 64*). Chronic respiratory failure may ensue in patients having severe airflow obstruction together with pulmonary hypertension or right heart failure (*50*). Of 29 patients with chronic bronchitis who were exposed to wood smoke, 20 had electrocardiographic or chest X-ray signs of pulmonary hypertension (*64*). Lung function in patients presenting to referral hospitals may have changes similar to those in smokers, ranging from normal to severe airflow obstruction. Some patients had classic characteristics of emphysema (*50, 64*) but restrictive changes have also been reported. A referral hospital study in Mexico found no significant differences between patients with chronic bronchitis who were exposed to biomass smoke and tobacco smokers in respect of lung function, clinical symptoms or radiographic features (*64*).

Experimental evidence and pathogenesis. Acute massive exposure to wood smoke, as in forest fires, can be rapidly lethal. Besides asphyxia and carbon monoxide intoxication there may be severe damage to the respiratory epithelium , with airway and pulmonary oedema. Lesser degrees of wood smoke exposure in guinea pigs produces bronchoconstriction and increases the response to subsequent exposure (*65*). After exposure to wood smoke for 3 hours a day for 3 months, guinea-pigs developed mild emphysema (*66*). Rats exposed intermittently to wood smoke for 75 minutes daily for 15 days had mononuclear bronchiolitis and mild emphysema; these conditions became more severe following exposure for 30 and 45 days (*67*). A fibrotic lung reaction simulating silicosis has been produced experimentally in animals exposed to wood smoke (*68*).

There is some uncertainty about the mechanisms whereby smoke causes emphysema and airway disease. Oxidative stress may be a component, as oxidizing radicals are present in tobacco and biomass smoke and are released by inflammatory cells (*69*). Risk factors for chronic obstructive pulmonary disease associated with tobacco smoking include bronchial hyperreactivity, atopy and genetic susceptibility, all of which could apply to biomass smoke exposure. A predisposition to chronic obstructive pulmonary disease later in life may result from impaired lung growth in infancy, leading to reduced adult lung function. Exposure to tobacco smoke or biomass smoke during pregnancy and infancy may therefore increase the risk of such disease.

Substantial deposition of carbon in the lung (anthracosis) occurred consistently in patients exposed to biomass. Necropsies of non-smoking women with cor pulmonale, most of whom were exposed to biomass smoke, revealed that all had emphysema, 11 had bronchiectasis, 5 had chronic bronchitis and 2 had tuberculosis (*70*). Several studies have described lung fibrosis, resembling pneumoconiosis (a chronic reaction of the lung to dust inhalation, usually involving fibrosis), including cases with progressive massive fibrosis, 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 sandstorms, but frequently the subjects were also exposed to biomass smoke (*13, 71*).

There is some evidence that exposure to wood smoke may be associated with interstitial lung disease (inflammation of the lung structure leading to fibrosis) in developed countries (*48, 68, 71–76*). In a small case-control study it was found that patients with cryptogenic fibrosing alveolitis had a heightened probability of having lived in a house heated by a wood fire (*76*). Exposure to wood smoke was more likely in 10 non-smoking cases with eosinophilic granuloma than in 36 controls with other interstitial lung diseases studied in Mexico City (odds ratio 5.6, 95% confidence interval = 1.04–30) (*77*). Cases associated with wood smoke exposure also demonstrated S-100 proteins, a marker of this disease.

Asthma. International variations in the prevalence of asthma (*78*), together with recent increases in many countries, have focused attention on the role of air pollution. The complex influence of air pollution on the development of asthma is a matter of controversy. While some assert that air pollution, including environmental tobacco smoke, may be a factor sensitizing genetically susceptible individuals to allergens in early life (*79*), a recent systematic review does not support this view in so far as environmental tobacco smoke is concerned (*80*). There is more consistent evidence that air pollution and environmental tobacco smoke trigger asthma in sensitized individuals (*79, 81*).

In developing countries, studies on biomass smoke in relation to asthma in children and adults have yielded mixed findings. A questionnaire survey of children aged 9–12 years in Turkey, which included spirometry, found that coal users had more day/night cough ($p < 0.05$) and that those using wood-burning stoves had the lowest values of FVC, FEV1, PEFR (peak expiratory flow rate) and FEF_{25} (forced expiratory flow rate at 25% of lung volume) (*82*); however, there was no adjustment for confounding. A matched case-control study of people aged 11–17 years in rural Nepal found an adjusted odds ratio of 2.3 (1.2–4.8) for asthma among those using wood fires or stoves compared to gas or kerosene (Schei, personal communication). In Jordan a cross-sectional study of lung function in children aged 11–13 years found significantly reduced FVC, FEV1, PEFR and $FEF_{25–75}$ for exposure to wood/kerosene stoves and environmental tobacco smoke, but no adjustment was made for confounding (*83*). A case-control study of schoolchildren in Nairobi found increased exposure to wood smoke in asthmatics (*84*).

Several studies, however, have reported no association. A case-control study of children aged between 1 month and 5 years who were hospitalized with asthma in Kuala Lumpur found that the use of kerosene or wood stoves was not independently associated with asthma, but that there was an association between mosquito coil smoke and this disease (*85*). Noorhassim found no association between asthma diagnosed by doctors or reported wheeze and biomass smoke in a cross-sectional study of 1007 children aged 1–12 years in Malaysia (*86*). A study in urban Maputo found no association after adjustment between fuel type and either wheeze or peak flow (*15*). Qureshi found no association in rural Pakistan, although the number of people with asthma was small (*59*). Preliminary findings of another cross-sectional study of 1058 children aged 4–6 years in rural Guatemala, in which the methods of the International Study of Asthma and Allergy in Childhood (ISAAC) were used, suggest a possible protective effect. The use of an open fire was associated with a non-significantly reduced risk of asthma (prevalence 5.9% for open fire versus 7.3% for all subjects, odds ratio = 0.64, 95% confidence interval = 0.21–1.91). However, there was a significant difference for exercise-induced asthma (prevalence 2.3% open fire vs. 3.7% total, OR=0.42, 95% CI: 0.21–0.82) (Schei, personal communication).

A study of nearly 29 000 adults in rural China reported that the adjusted odds ratios for wheezing and asthma for a group with occupational exposure to wood or hay smoke were 1.36 (1.14–1.61) and 1.27 (1.02–1.58) respectively (*87*). Since 93% of the sample used wood or hay for cooking the relationship with asthma was studied among the 39% of women and 21% of men exposed occupationally. Similarly elevated odds ratios were reported for those using coal for cooking.

Mixed findings have also been reported from developed countries, several studies having found positive associations (*88*) and some having found no association, as with children aged 5–9 years in Seattle (*89*). There is evidence that biomass smoke is associated with reduced risk, reflecting a possible protective effect. Von Mutius found the risk of hay fever, atopy and bronchial reactivity to be reduced in rural German children aged 9–11 years whose homes were heated by coal or wood (*90*). Similar evidence has been reported from urban Australia (*91*).

Overall, the evidence on exposure to biomass smoke and asthma in developing countries is limited and inconsistent. Although asthma is less common among rural populations where biomass fuels are used most, it should not be assumed that smoke exposure is protective in these settings.

Cancer

Lung cancer

Tobacco smoke is the most important risk for lung cancer and explains most cases in industrialized countries. In developing countries, non-smokers, frequently women, form a much larger proportion of patients with lung cancer. Some two-thirds of women with lung cancer in China (*92*), India (*93*) and Mexico (*94*) were non-smokers. In China, odds ratios for lung cancer among women exposed to coal smoke at home, particularly that of so-called smoky coal, were in the range 2–6 (*95, 96*). Smoky coal has been found

to be more carcinogenic than cleaner coal and wood smoke when tested on mouse skin (*97*).

No association has been reported between lung cancer and exposure to wood smoke (*95*). Rates of lung cancer in rural areas, where such exposure is common, tend to be low. This could be attributable to various factors associated with the rural environment, and it would be unwise to conclude that biomass smoke does not incease the risk of lung cancer, especially as there is intense exposure to known carcinogens in biomass smoke. In some homes, cooking for three hours per day exposes women to similar amounts of benzo[*a*]pyene as smoking two packets of cigarettes daily (*95*). If exposure to all carcinogens in wood smoke parallels exposure to particles, cooking with traditional biomass stoves is equivalent to smoking several cigarettes per day.

A history of previous lung disease is a risk factor for lung cancer in women (*98*). In developing countries, previous lung disease attributable to tuberculosis and other lung infections could contribute to lung cancer development in persons who have never smoked. Chronic obstructive pulmonary disease is associated with an increase in cancer risk, even when age, sex, occupation and smoking are taken into account (*99*). This suggests either that there is a parallel exposure to lung toxins and carcinogens or that chronically inflamed or injured tissue is more prone than normal tissue to develop cancer. Whatever the mechanism, exposure to biomass smoke is a potential risk factor for lung cancer.

Nasopharyngeal and laryngeal cancer

Biomass smoke has been implicated as a cause of nasopharyngeal carcinoma (*100*), although this is not a consistent finding (*101*). A case-control study in Brazil found that oral cancer was associated with tobacco, alcohol and the use of wood stoves (*102*). Another case-control study from South America of 784 cases of oral, pharyngeal and laryngeal cancer reported an adjusted odds ratio of 2.68 (95% confidence interval = 2.2–3.3) for exposure to wood smoke as compared with cleaner fuels (*103*). Significant associations were demonstrated separately for mouth, laryngeal and pharyngeal carcinomas and it was estimated that exposure to wood smoke explained about a third of upper aerodigestive tract cancers in the region.

Pulmonary tuberculosis

An analysis of data on 200 000 Indian adults found an association between self-reported tuberculosis and exposure to wood smoke (*104*). Persons living in households burning biomass reported tuberculosis more frequently than persons using cleaner fuels, with an odds ratio of 2.58 (1.98–3.37) after adjustment for a range of socioeconomic factors. These findings were similar to those of a study in north India, which reported an association between the use of biomass fuel and tuberculosis defined by clinical measures (*105*), although adjustment was made only for age.

This effect of wood smoke may result from reduced resistance to lung infection. Exposure to smoke interferes with the mucociliary defences of the lungs (*106*) and decreases several antibacterial properties of lung macrophages, such as adherence to glass, phagocytic rate and the number of bacteria phagocytosed (*107, 108*). Chronic exposure to tobacco smoke also decreases cellular immunity, antibody production and local bronchial immunity, and there is increased susceptibility to infection and cancer (*109*). Indeed, tobacco smoke has been associated with tuberculosis (*110, 111*). Although such widespread immunosuppression has not been reported with biomass smoke, an increase in the risk of tuberculosis is quite conceivable.

This association, if confirmed, would have substantial implications for public health. Exposure to biomass smoke can explain about 59% of rural cases and 23% of urban cases of tuberculosis in India (*104*). Such exposure may be an additional factor in the relationship between poverty and tuberculosis, hitherto explained by malnutrition, overcrowding and inadequate access to health care.

Low birth weight and infant mortality

In rural Guatemala, babies born to women using wood fuel were 63 g lighter ($P < 0.049$) than those born to women using gas and electricity, after adjustment for socioeconomic and maternal factors (*112*). Although we are not aware of any other similar reports, evidence relating to active smoking and environmental tobacco smoke (*113*) strongly indicates the probability of this effect, possibly mediated by carbon monoxide. Levels of carbon monoxide in homes using biomass fuels are high enough. Mean 24-hour values in the range 5–10 ppm, means of 20–50 ppm or more during the use of a fire (*13, 114, 115*), and carboxyhaemoglobin levels between 1.5% and 2.5% (*114*) and rising to 13% (*23*) have been reported. These levels are comparable with those associated with exposure to environmental tobacco smoke, and in some cases with active smoking (*9*).

There is evidence linking ambient air pollution with reduced birth weight (*116–118*), although only one study has specifically reported the association with carbon monoxide (*117*). In judging the potential public health impact of indoor air pollution through this effect on birth weight it is important to recognize that exposure is greatest among poor women of childbearing age who live in communities where there is frequently a high prevalence of low birth weight.

Only one study has reported an association between perinatal mortality and exposure to indoor air pollution in a developing country, with an odds ratio of 1.5 (1.0–2.1) for still births following adjustment for a wide range of factors (*119*). 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 particles and the infant mortality rate (*120*). The strongest effect was with $PM_{2.5}$ at 3–5 days before death, when an increase of 10 $\mu g/m^3$ was associated with a 6.9% (95% CI: 2.5–11.3) excess infant mortality rate. Infant mortality in the USA showed an excess perinatal mortality associated with higher PM_{10} levels after adjustment: an odds ratio of 1.10 (1.04–1.16) for the high pollution group (mean 44.5 $\mu g/m^3$) versus the low pollution group (mean 23.6 $\mu g/m^3$) (*121*). In infants of normal birth weight, high exposure was associated with respiratory mortality (odds ratio = 1.40 (1.05–1.85)) and sudden infant death syndrome (SIDS) (odds ratio = 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 low birth weight (*118*).

Cataract

Pollution attributable to the use of biomass fuel causes eye irritation (*17*) and may cause cataract. In a hospital-based case-control study in Delhi the use of liquefied petroleum gas was associated with an adjusted odds ratio of 0.62 (0.4–0.98) for cortical, nuclear and mixed, but not posterior subcapsular cataracts in comparison with the use of cow dung and wood (*122*). An analysis of over 170 000 people in India (*123*) yielded an adjusted odds ratio for reported partial or complete blindness of 1.32 (1.16–1.50) in respect of persons using mainly biomass fuel compared with other fuels, and there were significant differences between men and women and between urban and rural residents. Adjustment was made for a number of socioeconomic, housing and geographical variables, although there was a lack of information on smoking, nutritional state, episodes of diarrhoea and other factors that might have influenced the prevalence of cataract. On the other hand, the crude method of classifying exposure could be expected to result in an underestimation of the effect.

Animal studies have shown that wood smoke condensates, like cigarette smoke, damage the lens in rats, producing discoloration, opacities and particles of debris. The mechanism is thought to involve absorbtion and accumulation of toxins that lead to oxidation (*123*). The growing evidence that environmental tobacco smoke causes cataracts is supportive (*124*, *125*).

Table 1 summarizes the possible mechanisms by which the most important pollutants in biomass and coal smoke may cause cataract and the other health effects reviewed above.

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

Pollutant	Mechanism	Potential health effects
Particules (small particles less than 10 microns, and particularly less than 2.5 microns aerodynamic diameter)	• Acute: bronchial irritation, inflammation and increased reactivity • Reduced mucociliary clearance • Reduced macrophage response and (?) reduced local immunity • (?) Fibrotic reaction	• Wheezing, exacerbation of asthma • Respiratory infections • Chronic bronchitis and chronic obstructive pulmonary disease • Exacerbation of chronic obstructive pulmonary disease
Carbon monoxide	• Binding with haemoglobin to produce carboxy haemoglobin, which reduces oxygen delivery to key organs and the developing fetus.	• Low birth weight (fetal carboxy-haemoglobin 2–10% or higher) • Increase in perinatal deaths
Polycyclic aromatic hydrocarbons, e.g. benzo[*a*]pyrene	• Carcinogenic	• Lung cancer • Cancer of mouth, nasopharynx and larynx
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 in children
Sulphur dioxide	• Acute exposure increases bronchial reactivity • Longer term: difficult to dissociate from effects of particles	• Wheezing and exacerbation of asthma • Exacerbation of chronic obstructive pulmonary disease, cardiovascular disease
Biomass smoke condensates including polycyclic aromatics and metal ions	• Absorption of toxins into lens, leading to oxidative changes	• Cataract

The health impact of indoor air pollution in developing countries

Attempts have been made to quantify the impact of exposure to air pollution, including that arising from indoor air pollution, globally (*126, 127*) and in India (*128*). Broadly, two approaches have been adopted (Table 2). Despite the limitations of the evidence, particularly concerning exposure levels and risk estimates, both methods have resulted in remarkably consistent estimates of just under 2 million excess deaths (Table 3). An error factor of two in either direction was suggested. For India, Smith reported between 410 000 and 570 000 premature deaths among adult women and children aged under 5 years arising from exposure to indoor air pollution, on the basis of data on risk and exposure derived principally from studies carried out in the country (*128*). The most striking conclusion from these studies is that by far the greatest burden of mortality arises from indoor exposures in rural areas of developing countries. Estimates of the global burden of disease suggest that indoor air pollution is responsible for just under 4% of the disability-adjusted life years lost, meaning that its consequences are comparable with those of tobacco use and that they are only exceeded by those of malnutrition (16%), unsafe water and sanitation (9%) and unsafe sex (4%) (*127*). By far the largest contribution to the disability-adjusted life years lost arises from acute respiratory infections because of their high incidence and the mortality for which they are responsible among young children (*128*).

Prospects for interventions

The goal of interventions should be to reduce exposure to indoor air pollution, while meeting domestic energy and cultural needs and improving safety, fuel efficiency and environmental protection. Interventions should be affordable, perhaps requiring income generation and credit arrangements, and they should be sustainable. The evaluation of interventions should take into consideration all these criteria in addition to emphasizing the importance of reducing exposure to indoor air pollution.

Table 2. **Summary of approaches for estimating excess deaths attributable to exposure to indoor air pollution** (*126*)

Smith's method	Schwela's method
The mean risk of death per unit increase in the concentration of ambient particles is applied to population numbers at risk, using the following information. • The risk estimate is derived from urban studies on ambient pollution, and yields a range of 1.2–4.4% increase per 10 μg/m^3 PM_{10}. • Levels of pollution are obtained from studies of mean particle concentrations indoors in urban and rural settings in developed and developing countries. • A number of assumptions are made, including: that the lowest risk estimate (1.2%) is used; that this risk is halved above 150 μg/m^3; that PM_{10} levels are 50% of total suspended particles; and that risk estimates derived from developed country urban studies apply to other populations.	Analysis is carried out in six major economic areas, using air pollution (suspended particle matter) data derived from GEMS and AMIS and estimates of increased mortality associated with pollution. • The number of people at risk is determined on the basis of numbers exposed to annual mean levels of suspended particle matter exceeding the 1987 WHO guidelines. • The mortality rate/100 000 is determined without air pollution influences (levels below WHO guidelines). • The estimate of increase in mortality attributable to air pollution is taken as 100 μg/m^3 suspended particle matter, based on data from China, Central and Eastern Europe and the Established Market Economies.

Table 3. **Numbers of deaths attributable to indoor particles air pollution, by setting** (*126*)

Author	Total deaths attributable to indoor particles air pollution	Excess mortality by setting (deaths and % of total)			
		Developed countries		Developing countries	
		Urban	Rural	Urban	Rural
Smith	2.8 million	640 000 23%	1 800 000 67%	250 000 9%	30 000 1%
Schwela	2.7 million	363 000 13%	1 849 000 68%	511 000 19%	Not calculated

Exposure can be reduced by means of improved stoves, better housing, cleaner fuels and behavioural changes. Cleaner fuels, especially liquefied petroleum gas, probably offer the best long-term option in terms of reducing pollution and protecting the environment, but most poor communities using biomass are unlikely to be able to make the transition to such fuels for many years.

The use of improved biomass stoves has given varying results and has often been unsuccessful. However, evaluation has been very limited and has not considered the range of criteria outlined above. Indeed, until recently, the main emphasis of stove programmes has been to reduce the use of wood, and consequently there has been relatively little evaluation of reductions in exposure (*129*). Nevertheless, there are examples of large-scale rural stove programmes, for instance in China (*130*). Under the Chinese programme, which began in 1980, improved stoves had been installed in over 172 million homes by the end of 1995. Smaller programmes, for example in western Kenya, have been enthusiastically adopted, mainly because of the participation of local women in construction and dissemination (*131*). Although improved stoves are usually capable of reducing ambient pollution and personal exposure, the residual levels for stoves in regular use are still high, mostly in the range 500 to several thousand $\mu g/m^3$ TSP or PM_{10}) (*115, 132, 133*).

Relatively little information is available on the potential of other types of intervention, including the use of cleaner fuels, particularly for poor rural communities. A study of patterns of fuel use in households following electrification in a traditionally wood-burning area of South Africa showed that, while there was a shift to the use of electricity, the more polluting fuels continued to be used, particularly for cooking and heating (*134*). The main reasons for not using electricity more were its cost and that of electrical appliances, although other factors, such as seasonal energy requirements and cultural beliefs, are also important in this connection.

In the field of development, household energy is important from the health, environmental and economic standpoints. This is consequently a very important field for interventions, and one in which technical and policy research needs to be closely linked to development work in a range of countries and settings.

Discussion

Evidence on health effects

This review of the health effects of indoor air pollution in developing countries confirms the findings of previous reviews (*3, 4*) and provides further evidence of associations with a range of serious and common health problems. The most important appear to be childhood acute lower respiratory infections, which remain the single most important cause of death for children aged under 5 years in developing countries. Nevertheless, the evidence has significant limitations: a general paucity of studies for many conditions, a lack of pollution/exposure determinations, the observational character of all studies, and the failure of too many studies to deal adequately with confounding.

That few studies have measured pollution or exposure presents the possibility of serious misclassification of exposure, and means that very little information is available to quantify the relationships between exposure level and risk. This has important implications for assessing the health impact of exposure levels in various populations, as well as in estimating the potential health gains that might result from reducing exposure by different amounts. In particular, it should be noted that where interventions (mainly stoves) have been evaluated the residual levels of pollution are still well above those indicated in current air quality guidelines. The observational nature of most studies presents a problem in relation to confounding since households adopting less polluted stoves and/or behaviour generally do so following improvements in their socioeconomic circumstances, which strongly influence many health outcomes (*40*). This, together with inadequate adjustment for confounding in a substantial proportion of studies, is likely to result in biased risk estimates.

Despite these limitations, the evidence for two of the most important conditions — acute upper respiratory infections and chronic obstructive respiratory disease — is compelling and suggestive of causality, particularly in conjunction with findings for environmental tobacco smoke and ambient pollution. With these outcomes, the major weakness in the evidence relates to the quantification of the exposure-response relationship. For other health outcomes, including asthma, otitis media, lung cancer (particularly in relation to biomass fuel smoke) and nasopharyngeal/laryngeal cancer, interstitial lung disease, low birth weight, perinatal mortality, tuberculosis and cataract, the evidence must be seen as more tentative. The evidence of an association with cardiovascular disease has not been reviewed in detail 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 (*135, 136*) and environmental tobacco smoke (*137*) suggests that this is a potentially important area for future work.

Conclusion

Indoor air pollution is a major public health hazard for large numbers of the world's poorest, most vulnerable people and may be responsible for a similar proportion of the global burden of disease as risk factors such as tobacco and unsafe sex. The greatest contribution to this burden results from childhood acute lower respiratory infections. The evidence on which these estimates are based, however, is rather limited. It is

important to extend and strengthen it, particularly for the most common and serious conditions including acute lower respiratory infections and tuberculosis, to quantify exposure, and to ensure that confounding is adequately dealt with. A few well-conducted randomized controlled studies on the health impact of reducing exposure would markedly strengthen the evidence, and should be feasible at the household level. For conditions where the evidence is very limited (e.g. low birth weight) or where a long latent period would make an intervention study impractical (e.g. tuberculosis, cataract), further observational studies are desirable.

Although work on interventions to reduce exposure has given mixed results, there is a wide range of possibilities and there has been some success in terms of both exposure reduction and uptake. The development and evaluation of interventions should take account of the many aspects of household energy supply and utilization, and should include assessment of pollution and exposure reductions, fuel efficiency and impact on the local and global environment, safety, capacity to meet household needs, affordability and sustainability. There is a need for a coordinated set of community studies to develop and evaluate interventions in a variety of settings, together with policy and macroeconomic studies on issues at the national level, such as fuel pricing incentives and other ways of increasing access by the poor to cleaner fuels. Also required is a systematic, standardized approach to monitoring levels and trends of exposure in a representative range of poor rural and urban populations.

Finally, it is necessary to keep in mind the close interrelationship between poverty and dependence on polluting fuels, and consequently the importance of socioeconomic development, which should be at the core of efforts to achieve healthier household environments. ■

Résumé

Pollution atmosphérique à l'intérieur des locaux : un problème majeur pour l'environnement et la santé publique

Plus de la moitié de la population mondiale utilise la biomasse (bois, déjections animales, résidus végétaux) pour produire de l'énergie domestique. Ces matériaux sont classiquement brûlés dans des feux ouverts ou des poêles défectueux, ce qui entraîne des taux très élevés de pollution à l'intérieur des habitations. Des études en provenance de nombreux pays font état de taux moyens de particules au moins 20 fois supérieurs aux normes fixées par l'United States Environmental Protection Agency. L'exposition à cette pollution touche principalement les femmes et les jeunes enfants qui leur tiennent compagnie pendant la préparation des repas.

Le présent article expose les résultats d'études sur les effets sanitaires de l'exposition à la fumée émise par la combustion de biomasse dans les pays en développement. Lorsque ces études étaient trop limitées, on a tenu compte de résultats d'études réalisées dans les pays industrialisés sur la fumée de bois, la fumée de tabac et la pollution atmosphérique à l'intérieur des locaux. Il est maintenant régulièrement démontré que l'exposition à la fumée de biomasse augmente le risque d'infection des voies respiratoires inférieures chez l'enfant et probablement aussi le risque d'otite moyenne. Une association avec la bronchite chronique (d'après les symptômes) et les maladies respiratoires obstructives chroniques (d'après les signes cliniques et les tests spirométriques) est bien établie, surtout chez les femmes; ces affections évoluent dans certains cas en emphysème ou en cœur pulmonaire. D'après des observations préliminaires, il y aurait également une association avec les pneumopathies interstitielles. On commence à disposer de preuves d'un effet sur le poids de naissance, très probablement dû à l'action du monoxyde de carbone, et on pourrait assister à une augmentation de la mortalité infantile et périnatale. L'exposition à la fumée de biomasse favorise probablement les crises d'asthme, même si les données en provenance des pays en développement sont parfois contradictoires. Trois études montrent une augmentation du risque de tuberculose pulmonaire. Des études chez l'homme et chez l'animal laissent à penser qu'il pourrait y avoir un risque accru de cataracte. Toutes les études rapportées sont des études d'observation et très peu d'entre elles ont mesuré directement l'exposition : des paramètres indirects ont été utilisés, et une proportion notable des études n'ont pas suffisamment tenu compte des facteurs de confusion. Malgré l'abondance de données démontrant que l'exposition à la fumée de biomasse augmente le risque de diverses maladies graves, ces insuffisances méthodologiques impliquent que les estimations du risque sont très approximatives et sujettes à des biais.

D'après des estimations de la mortalité attribuable, l'exposition à la pollution atmosphérique à l'intérieur des locaux pourrait être responsable de près de 2 millions de décès excédentaires dans les pays en développement, et d'environ 4 % de la charge mondiale de morbidité. Une vaste gamme d'interventions pourrait aider à réduire l'exposition, bien que des études d'évaluation montrent qu'il reste beaucoup à faire en ce qui concerne la lutte contre la pollution et la viabilité des mesures prises. Néanmoins, certaines interventions ont déjà conduit à une réduction sensible de l'exposition tout en étant acceptées et largement adoptées, bien que rarement en association. Cette expérience doit être poursuivie.

La pollution atmosphérique à l'intérieur des locaux constitue une menace majeure pour la santé publique, qui exige une augmentation considérable des travaux de recherche et une attention soutenue de la part des responsables politiques. Les recherches sur les effets sanitaires doivent être renforcées, en mettant l'accent sur les études d'intervention et la mesure de l'exposition.

Une approche plus systématique de l'élaboration et de l'évaluation des interventions est nécessaire, en tenant compte de la relation étroite entre la pauvreté et le recours obligé à des combustibles polluants. Les capacités locales techniques et en matière de développement devront être renforcées pour pouvoir mettre toutes ces interventions en œuvre là où elles sont le plus nécessaires.

Resumen

Contaminación del aire de locales cerrados en los países en desarrollo: un importante reto ambiental y de salud pública

Más de la mitad de la población mundial depende de la biomasa (madera, estiércol, restos de cosechas) para obtener energía doméstica. Esos productos se suelen quemar en lumbres expuestas o en estufas de funcionamiento defectuoso, lo que provoca unos niveles muy altos de contaminación del aire en locales cerrados. Estudios realizados en muchos países han detectado concentraciones promedio de partículas superiores en 20 o más veces a las establecidas como referencia por la Agencia para la Protección del Medio Ambiente de los Estados Unidos. La exposición a esa contaminación afecta principalmente a las mujeres y a los niños de corta edad que las acompañan mientras cocinan los alimentos.

Esta revisión se ha basado principalmente en estudios de los efectos sanitarios de la exposición al humo de combustibles de biomasa en los países en desarrollo. En los casos en que esos estudios son muy limitados, sin embargo, se ha recurrido a trabajos llevados a cabo en países industrializados acerca del humo de madera, el humo de tabaco ambiental y la contaminación ambiental (exterior). Disponemos hoy de pruebas bastante sólidas de que la exposición al humo de combustibles de biomasa aumenta el riesgo de infecciones agudas de las vías respiratorias inferiores en los niños, y también probablemente el de otitis media. Está razonablemente establecida la relación con la bronquitis crónica (evaluada en función de los síntomas) y con la enfermedad pulmonar obstructiva crónica (evaluada clínicamente y mediante espirometría), sobre todo entre las mujeres, algunas de las cuales acaban desarrollando enfisema o cor pulmonale. Datos preliminares sugieren también una asociación con las enfermedades que afectan al intersticio pulmonar. Cada vez son más los indicios de un efecto en el peso al nacer, mediado muy probablemente por el monóxido de carbono, y la mortalidad de lactantes y perinatal también puede verse aumentada. La exposición al humo de combustibles de biomasa exacerba probablemente el asma, si bien los datos disponibles sobre los países en desarrollo son contradictorios. Se hace referencia a tres estudios que sugieren un incremento del riesgo de tuberculosis pulmonar. Estudios realizados en el hombre y en animales apuntan también a un aumento del riesgo de catarata. Todos los estudios considerados están basados en la observación, y muy pocos han determinado la exposición directamente: se han utilizado variables sustitutivas, y en una proporción sustancial de los estudios no se han abordado debidamente los factores de confusión. Pese a la creciente evidencia de que la exposición al humo de combustibles de biomasa aumenta el riesgo de sufrir diversas enfermedades graves e importantes, las limitaciones metodológicas impiden cuantificar bien el riesgo y tienden a introducir sesgos en su estimación.

Las estimaciones de la mortalidad atribuible llevan a pensar que la exposición a la contaminación de los locales cerrados podría estar causando casi 2 millones de defunciones en los países en desarrollo, y el equivalente a aproximadamente un 4% de la carga mundial de morbilidad. La exposición puede reducirse mediante un amplio espectro de intervenciones, si bien los estudios de evaluación muestran que siguen pendientes retos importantes en lo que respecta a reducir la contaminación y asegurar la sostenibilidad de los logros. No obstante, algunas intervenciones han permitido conseguir reducciones sustanciales de la exposición, han sido bien acogidas y se han difundido ampliamente, aunque rara vez en combinación. Es necesario aprovechar esa experiencia.

La contaminación del aire en locales cerrados constituye una importante amenaza para la salud pública mundial, y exige mucha más investigación y atención por parte de los formuladores de políticas. Deberían reforzarse las investigaciones sobre los efectos sanitarios, orientándolas al estudio de las intervenciones y a la cuantificación de la exposición. Hay que enfocar de forma más sistemática el desarrollo y evaluación de las intervenciones, reconociendo claramente la estrecha relación existente entre la pobreza y la dependencia de los combustibles contaminantes. Y debe fortalecerse la capacidad técnica y de desarrollo a nivel local para apoyar la aplicación de medidas allí donde más se necesiten.

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