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HOUSEHOLD ENERGY, INDOOR AIR POLLUTION, AND HEALTH IN DEVELOPING COUNTRIES: Knowledge Base for Effective Interventions

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■ **Abstract** Globally, almost three billion people rely on biomass (wood, charcoal, crop residues, and dung) and coal as their primary source of domestic energy. Exposure to indoor air pollution from the combustion of solid fuels is an important cause of disease and mortality in developing countries. Despite recent advances in estimating the health impacts of indoor smoke, there are limited studies targeted toward the design and implementation of effective intervention programs. We review the current knowledge of the relationship between indoor air pollution and disease, and of the assessment of interventions for reducing exposure and disease. This review takes an environmental health perspective and considers the details of both exposure and health effects that are needed for successful intervention strategies. In particular, we summarize the emerging understanding of the central role of household energy technology and day-to-day household activities in determining exposure to indoor smoke. We also identify knowledge gaps and detailed research questions that are essential in successful design and dissemination of preventive measures and policies. In addition to specific research recommendations based on the weight of recent studies, we conclude that research and development of effective interventions can benefit tremendously from integration of methods and analysis tools from a range of disciplines—from quantitative environmental science and engineering, to toxicology and epidemiology, to the social sciences.

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INTRODUCTION

Globally, almost three billion people rely on biomass (wood, charcoal, crop residues, and dung) and coal as their primary source of domestic energy (1, 2). Biomass accounts for more than 50% of domestic energy use in many developing countries and for as much as 95% in some lower income ones (1, 3). There is also evidence that in some countries the declining trend of household dependence on biomass has slowed, or even reversed, especially among poorer households (2, 4).

Biomass and coal smoke contain a large number of pollutants and known health hazards including particulate matter, carbon monoxide, nitrogen dioxide, sulfur oxides (mainly from coal), formaldehyde, and polycyclic organic matter including carcinogens such as benzo[a]pyrene (5–8). Exposure to indoor air pollution from the combustion of solid fuels has been implicated, with varying degrees of evidence, as a causal agent of several diseases in developing countries including acute respiratory infections (ARI) and otitis media (middle ear infection),[1] chronic obstructive pulmonary disease (COPD), lung cancer (especially for coal smoke), asthma, nasopharyngeal and laryngeal cancer,[2] tuberculosis, perinatal conditions and low birth weight (as a result of maternal exposure), and diseases of the eye such as cataract and blindness (9–13). Although the physiological mechanisms of the health impacts of indoor biomass smoke have not been studied in developing country settings, it is likely that some of the findings of industrialized country air pollution research also apply to this setting (10, 14).

Most current epidemiological studies on the health impacts of exposure to indoor air pollution in developing countries have focused on the first three of the above diseases (10, 11). Although low birth weight, for which there is new evidence from a recent study in Guatemala (15), can also have significant and long-term health effects, given current quantitative knowledge, acute (lower) respiratory infections (ALRI) and COPD are the leading causes of mortality and burden of disease as a result of exposure to indoor air pollution from solid fuels.[3]

[1]The middle ear is connected to and often affected by the infections of the uppermost part of the respiratory tract.

[2]The pharynx and the larynx are parts of the upper respiratory system and affected by inhaled pollutants.

[3]Burden of disease is calculated as the number of years lost from premature mortality plus the number of years lived with a disease with appropriate disability weights (16). For this reason, childhood mortality counts for a large number of years lost from premature mortality and a large contribution to burden of disease.

Conservative[4] estimates of global mortality as a result of exposure to indoor air pollution from solid fuels show that in 2000 between 1.5 and 2 million deaths were attributed to this risk factor (17, 18). This accounts for approximately 3% to 4% of total mortality worldwide. Approximately one million of the deaths were due to childhood ALRI, with the remainder due to COPD followed by lung cancer among adult women (17).

The magnitude of the health loss associated with exposure to indoor smoke as well as its concentration among marginalized socioeconomic and demographic groups (women and children in poorer households and the rural population) have recently put preventive measures high on the agenda of international development and public health organizations (11, 18–22). In this chapter we review the current knowledge of the relationship between indoor air pollution and disease (focusing on ARI, the largest contributor to the burden of disease due to this risk factor) and on the interventions for reducing exposure and disease. We also identify knowledge gaps and detailed research questions that are essential in successful design and dissemination of preventive measures and policies. In particular we argue that, given the central role of housing, household energy, and day-to-day household activities in determining exposure to indoor smoke, research and development of effective interventions can benefit tremendously from integration of methods and analysis tools from a range of disciplines—from quantitative environmental science and engineering, to toxicology and epidemiology, to the social sciences. Although our discussion of health effects focuses on acute respiratory infections, some of the findings and recommendations—in particular those on the determinants of exposure—are also applicable to other diseases caused by exposure to indoor air pollution. Our review of the health effects draws on two excellent recent review papers on the epidemiology of indoor air pollution as a risk factor (10, 11) for various diseases.

EARLY RESEARCH

Respiratory diseases have consistently been among the most prevalent diseases of developing countries. For example, Figure 1 shows the prevalence of the most common diseases in the last two decades of colonial Kenya as reported in hospital records, illustrating the consistently important role of respiratory infections.[5]

[4]Conservative assumption implies (*a*) focusing only on those outcomes with strong evidence and (*b*) using relatively low estimates of hazard size.

[5]One may expect that in the colonial period, when hospitals were less accessible, especially to the African population, only a fraction—and the most severe cases—of respiratory infections were reported, compared with other infectious and parasitic diseases that are generally more severe and likely to have had higher relative reporting rates. Therefore, there may be a downward bias in the estimates of the share of respiratory diseases. In an analysis of 1968 health statistics, Bonte also suspected a systematic under-reporting of deaths from respiratory and other diseases that are more common in poorer households (23).

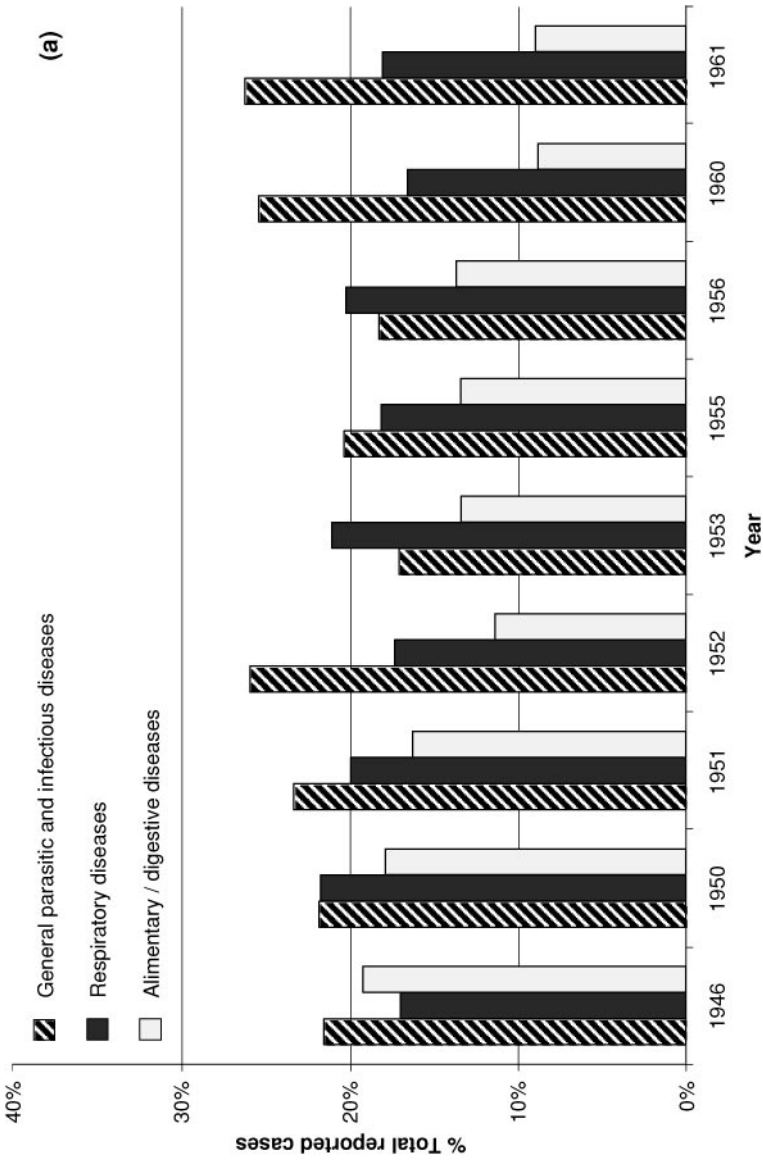


Figure 1 Common diseases of colonial Kenya (from Reference 132). (*a*) Share of total number of cases treated in hospitals. The data are the share of total inpatient and outpatient cases in hospitals except for 1960 and 1961, when data were available only for inpatient cases. (*b*) Share of inpatient mortality. Other common diseases of these years were skin diseases and injuries, which in some years had more cases than alimentary/digestive diseases.

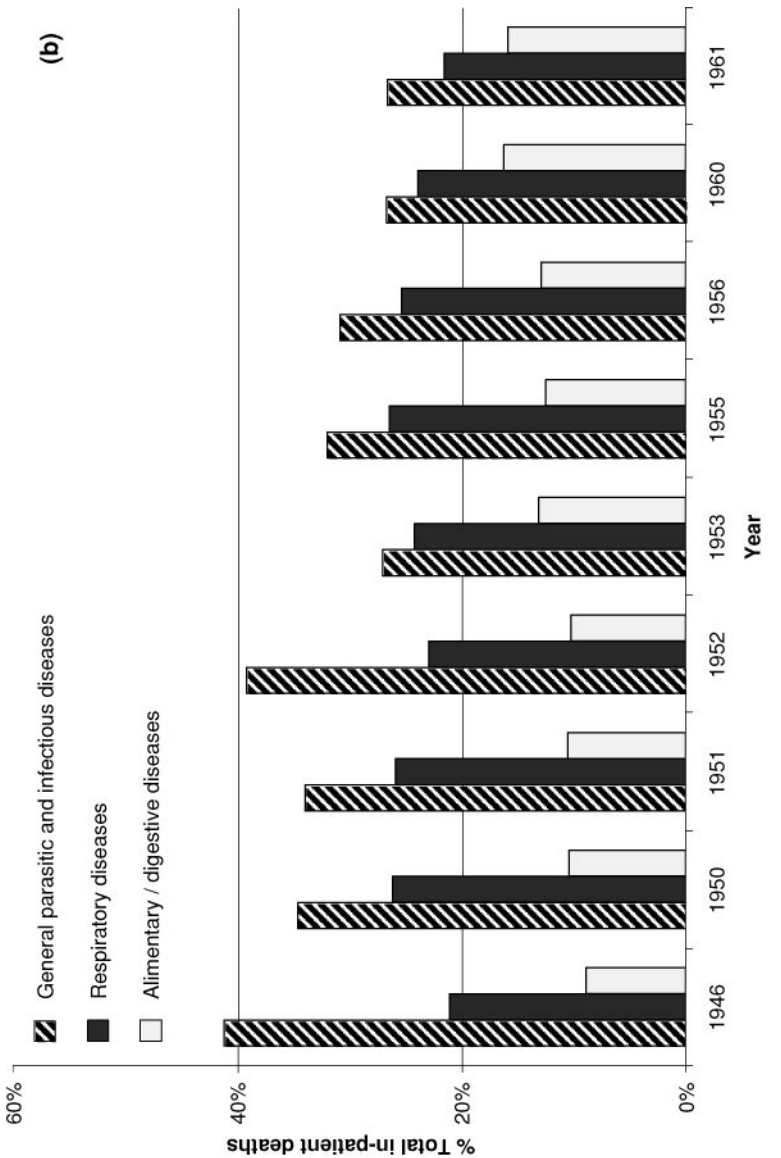


Figure 1 (*Continued*)

Respiratory diseases remained an important disease in Kenya over time. Figure 2 for example shows the distribution of the causes of death in 1968 from both hospital records and nonhospital notification of death (23), as well as the reasons for health service encounters, illustrating the sustained importance of respiratory diseases. In addition to being the leading national cause of mortality, respiratory diseases were also the first or second leading cause of mortality and important cause of hospital admissions in all provinces (23). A similar pattern existed in 1980. Acute respiratory infections and malaria led the number of cases treated in Kenyan hospitals, with a share of 21 and 18%, respectively. Infectious and parasitic diseases (20%) and respiratory diseases (18%) were the leading causes of death (24).

Despite their public health importance, with the exception of tuberculosis, diseases of the respiratory system have received mixed attention in developing countries. As early as the turn of the century, detailed research on the prevalence, causation, and management of pneumonia and other respiratory diseases was conducted in developing countries [see e.g., (25, 26)]. However, in many studies of tropical health respiratory diseases were hardly mentioned or were not discussed at length [see e.g., (27, 28–34)].

Manderson also noticed this systematic lack of attention to respiratory and diarrheal diseases in colonial Malaya, which she attributed to the "metaphoric weight" of other diseases, especially those of an epidemic nature (35). An equally important factor in this lack of attention may be traced to the evolution of medical science in the late nineteenth century and much of the twentieth century. The search for disease vectors and parasites, and for curative approaches that would eliminate them, dominated biomedical sciences in this period. This rise of "germ theory" in medicine, and in particular in tropical medicine (which took place in a geographical context that was perceived to be ecologically and socially suitable for the spread of germs), shifted the attention of health authorities to those diseases that could be dealt with using modern biomedical tools. This dominance was intensified by the fact that colonial tropical medicine was much influenced by military doctors, whose biomedical approaches had achieved a great deal of success in combating disease among European troops overseas in the nineteenth and twentieth centuries (36, 37). In this intellectual and professional context of tropical health, "neither tuberculosis nor pneumonia appeared to 'yield' to [the dominant] methods of control . . . " (p. 62, 35).

The tendency to couple disease with germs, especially in tropical settings, was also the likely reason that even when respiratory infections received attention in the medical community, no reference to the role of air pollution in their incidence was made. In colonial Malaya, where the medical services were "rather more successful for curative than preventive purposes," it was believed that "there was likely to be little change [in tuberculosis or pneumonia] under existing social and economic circumstances" (p. 28, pp. 62–63, 35). In Malaya, as in other places, respiratory disease was almost exclusively associated with overcrowding of houses and other factors that would facilitate the spread of germs, rather than with air pollution. In "The Roots of Backwardness," a chapter in *Africa Emergent,* W.M. Macmillan cited the 1928 *Annual Medical Report* of Kenya:

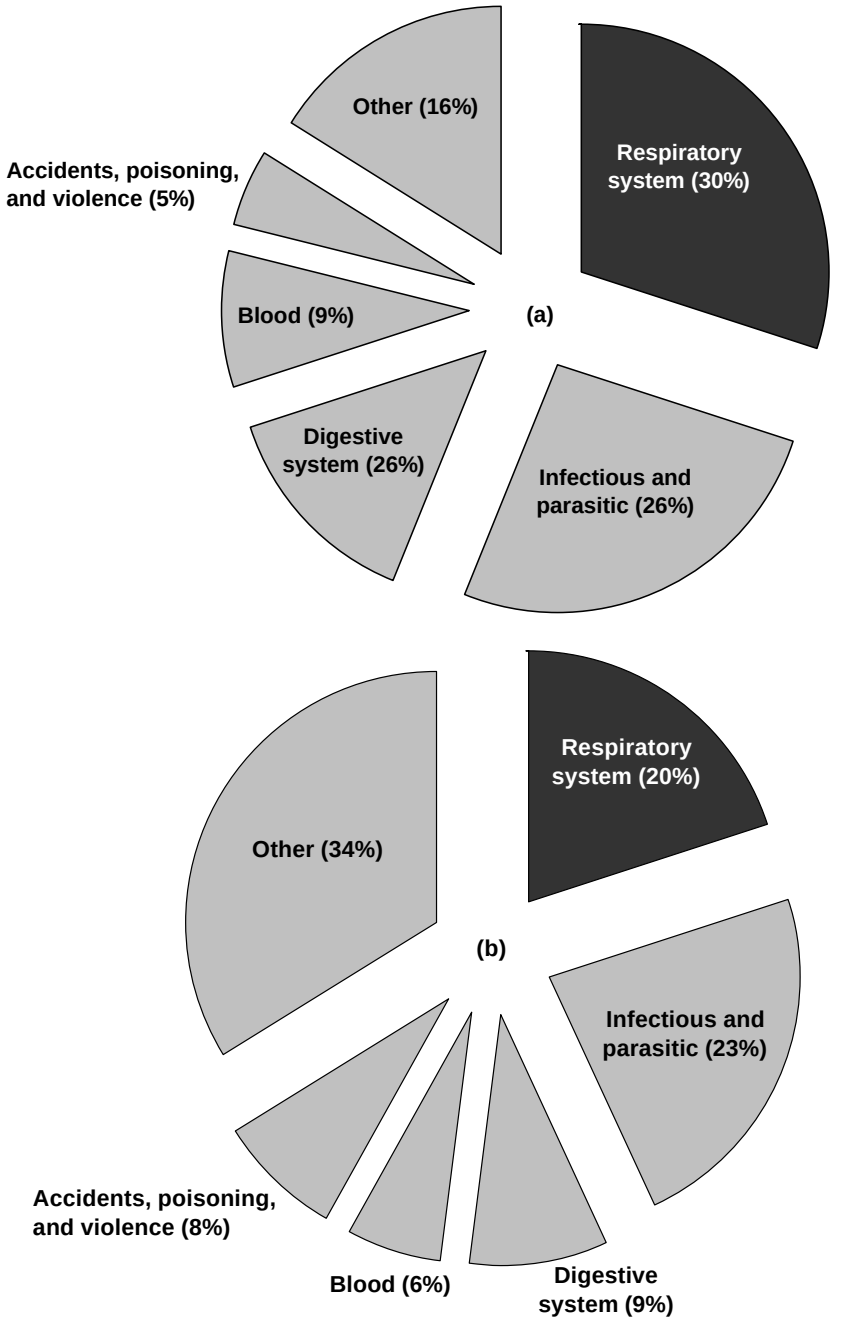


Figure 2 The distribution of mortality and health service encounters in Kenya in 1968 (from Reference 23). (*a*) Hospital death records from an estimated 9 million outpatients and 320,000 inpatients; (*b*) nonhospital notification of death records; (*c*) admissions; (*d*) outpatient attendances.

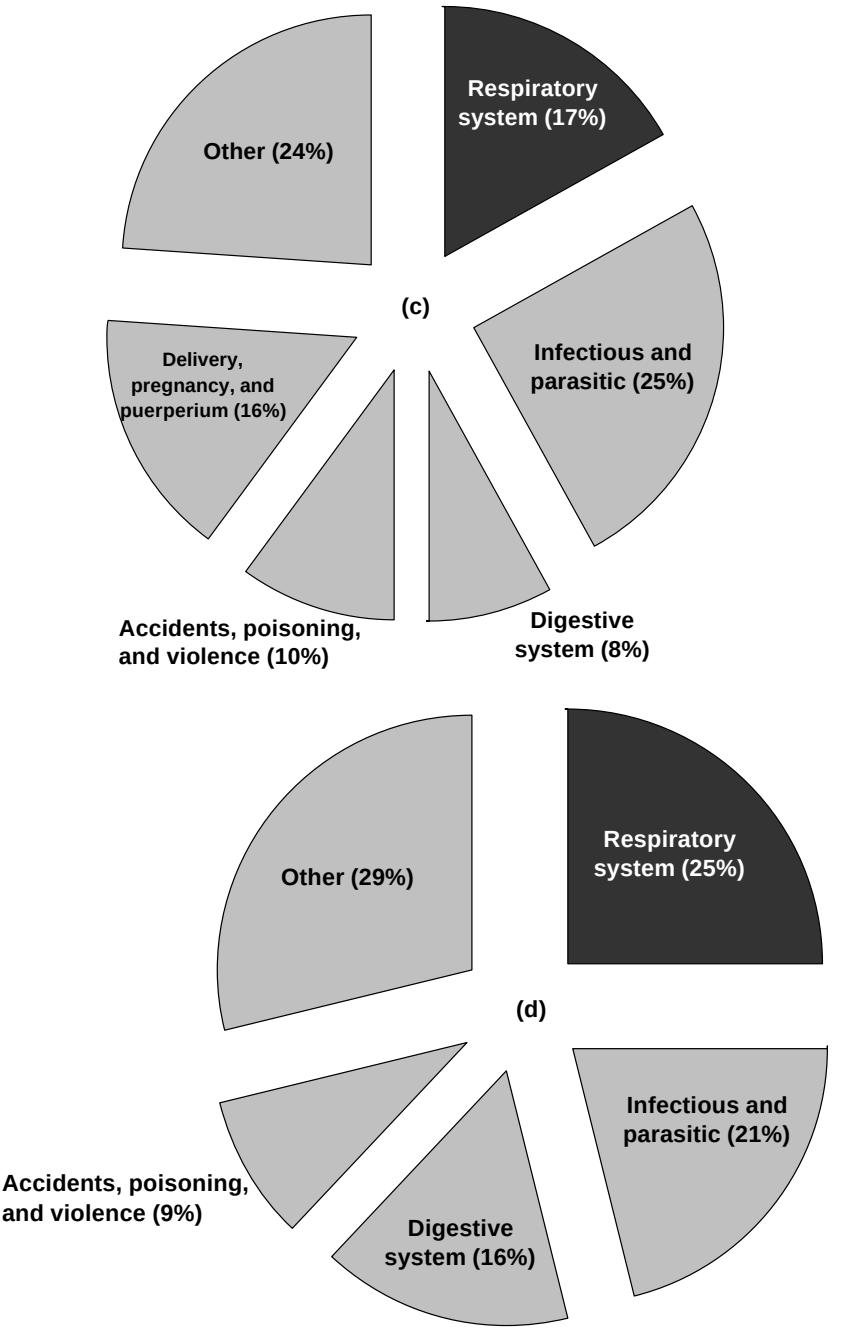


Figure 2 (*Continued*)

> Pneumonia, broncho-pneumonia, and tuberculosis take a large toll of life. The circumstances of the people are such that they live under conditions which are admirably suitable for the existence and spread of the causal agents of disease or of their animal hosts. Even where huts and villages are not overcrowded with humans, they are always overcrowded with the causative organisms of disease or the carriers of these organisms, so that escape from infection is for the great majority of people impossible (38).

Other accounts of the "native huts" by health personnel also included descriptions of their crowdedness with people or objects and their various odors (39) but not of their smokiness. Even when the relationship between air pollution and respiratory health was discussed in the context of occupational health (40), it was ignored in residential settings and cooking activities, a trend that continued until recent decades. In fact, as recently as the 1980s and 1990s, epidemiological studies, health care manuals, and health reports focused on the biological mechanisms of infection and biomedical management of respiratory infections, with some consideration of the role of temperature and crowding but little mention of the role of indoor air pollution [see, e.g., (23, 41–48)].

THE EVOLUTION OF EPIDEMIOLOGICAL RESEARCH

More detailed research on exposure to indoor smoke and its impacts on respiratory diseases in developing countries began in the 1960s and 1970s in India, Nigeria, and Papua New Guinea (49–54). Thanks to an increasing number of research projects in the 1980s, the public health importance of this risk factor has recently appeared on the agenda of research and policy communities (6, 18, 19, 55–59).

Monitoring of pollution and personal exposures in biomass-burning households has shown concentrations many times higher than those in industrialized countries. The latest National Ambient Air Quality Standards of the U.S. Environmental Protection Agency, for instance, required the daily average concentration of PM_{10} (particulates below 10 microns in diameter) to be below 150 $\mu g/m^3$ (annual average below 50 $\mu g/m^3$). In contrast, the typical 24-hour average concentration of PM_{10} in homes using biofuels may range from 200 to 5,000 $\mu g/m^3$ or more throughout the year, depending on the type of fuel, stove, and housing as shown by measurements in settings as diverse as China, India, Kenya, Papua New Guinea, Nepal, and The Gambia (6, 8, 10, 55, 60). Concentration levels of course depend on where and when monitoring takes place, because significant temporal and spatial variations may occur within a house (8, 61–63). Ezzati et al. (63) for example have recorded peak concentrations of 50,000 $\mu g/m^3$or more in the immediate vicinity of the fire, with concentration levels falling significantly with increasing distance from the fire. Menon (61) and Saksena et al. (62) have measured high average concentrations (20,000 $\mu g/m^3$ or more) near the cooking location using Indian stoves, with lower concentrations in other parts of the house or kitchen. Overall it is estimated that ~80% of total global exposure to airborne particulate matter occurs indoors in

developing nations (55, 60). Levels of carbon monoxide and other pollutants also often exceed international guidelines (6, 8, 60, 64).

Bruce et al. (11) have reviewed the epidemiological evidence of the health effects of indoor smoke from solid fuels. They concluded that, despite some methodological limitations, the epidemiological studies together with experimental evidence and pathogenesis provide compelling evidence of causality for acute respiratory infections and chronic obstructive pulmonary disease, particularly in conjunction with findings for environmental tobacco smoke and ambient air pollution. The relationship between coal smoke (but not biomass) and lung cancer has also been consistently established in a number of epidemiological studies (65–68). For other health outcomes, including asthma, upper aerodigestive cancer, interstitial lung disease, low birth weight, perinatal mortality, tuberculosis, and eye diseases, Bruce et al. (11) classified the evidence as more tentative [moderate or weak as classified by Smith & Mehta (17)]. The details of biological mechanisms and epidemiological studies of indoor air pollution and childhood acute respiratory infections (ARI) were reviewed by Smith et al. (10), who concluded that "when interpreted in 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 characterized" (p. 530). In the following sections we review the methodological and empirical characteristics of these and some recent studies and propose directions for future research.

EMISSIONS MONITORING AND EXPOSURE ASSESSMENT

A common characteristic of most epidemiological studies of the health impacts of indoor smoke has been the use of indirect measures of exposure, such as fuel type, housing characteristics, or aggregate measures of time spent near fire. In studies that focus on emissions and exposure assessment, the alternative to indirect exposure measures has been the use of personal monitors [see, e.g., (69, 70)] or area monitors, mostly recording average daily or burning-time concentrations. Although personal monitors measure exposure directly, with current personal monitoring technology exposure is aggregated over time and space (i.e., a single measure of exposure for the individual that sums their exposure in all locations and times). This lack of detail leaves out the patterns of exposure (including the high-intensity emission episodes that commonly occur during the combustion of biomass fuels) and limits a predictive assessment of the impacts of various intervention strategies on individual exposure.

Important alternatives to these approaches to pollution and exposure monitoring are the works of Menon (61) for Indian cookstoves, Saksena et al. (62) in the Indian Himalayas, Ballard-Tremeer & Jawurek (71) in South Africa, McCracken & Smith (72) in Guatemala, and Ezzati et al. (8, 63) in Kenya. Menon (61), Ballard-Tremeer & Jawurek (71), and McCracken & Smith (72) monitored fluctuations in concentrations (PM or CO) for Indian, South African, and Guatemalan cookstoves over a period of a few hours and found that emissions from biomass stoves vary

greatly over short time intervals. The thorough work of Ballard-Tremeer & Jawurek (71) further related these fluctuations to combustion characteristics, such as energy density, combustion temperature, and air flow. Ezzati et al. (8, 63), using more recent measurement technology, conducted continuous real-time monitoring of concentrations under actual conditions of use in 55 households for more than 200 14-hour days. By also recording the status of the fire (whether it was off, starting, burning, or smoldering), the type of food prepared, and other energy-use or cooking behavior (such as adding or moving of fuel or cooking pot, stirring of food, etc.) during the whole day, the authors found that the emission peaks commonly occur when fuel is added or moved, the stove is lit, the cooking pot is placed on or removed from the fire, or food is stirred, as seen in Figure 3 and Figure 4.

In addition to studying the temporal characteristics and fluctuations of emissions, Menon (61), Saksena et al. (62), and Ezzati et al. (63) also monitored the spatial patterns (dispersion) of pollution in different microenvironments in the house and found a spatial gradient for pollution concentration. Using data on microenvironment concentration, daily time budget, and daily personal exposure, Saksena et al. (62) estimated the contribution of each microenvironment to personal exposure. The authors found large variability among demographic subgroups in terms of contributions of different microenvironments, with kitchen during cooking being the largest contributor to the exposure of women ($\sim$75% of exposure), followed by children (25% of exposure in winter and 40% in summer). The kitchen microenvironment made little contribution to the exposure of youth and almost none for men, whose exposure (significantly lower than women) occurred mostly in the living room. The measurements by Menon (61) and Ezzati et al. (63) both considered smaller microenvironments, including dispersion within a room. Their results show that even in a single room, pollution concentrations exhibit a pronounced spatial gradient rather than instantaneous mixing [see Figure 1 in (63)]. This finding implies that the exposure microenvironments for indoor smoke are considerably smaller than those of Saksena et al. (62), possibly as small as 0.5 m. Coupled with the large variability of emissions from biofuels over short time periods—with the instantaneous peaks coinciding with household members who cook being consistently closest to the fire—this indicates that the complete time-activity budgets of individuals, in relation to emissions, are important determinants of exposure. For example, one of the most common foods in East Africa, in particular in rural areas, is *ugali*. Ugali is a porridge made from maize or sorghum flour thickened into a "cake." After adding flour to boiling water, the cook continuously stirs the mixture (Figure 3*B*). As water evaporates and the mixture hardens, stirring becomes increasingly vigorous, finally turning into folding the hardened dough. The process takes 15–40 minutes, during which the cook is very close to the fire. Throughout cooking, heat is controlled by increasing the burning rate or putting the fire into a smoldering (and hence very smoky) phase.

To characterize this complexity of personal exposure to indoor smoke, Ezzati et al. (63) used continuous monitoring of PM_{10} concentration, data on spatial dispersion of indoor smoke, and detailed quantitative and qualitative data on time-activity budgets to construct measures of exposure that account for individual

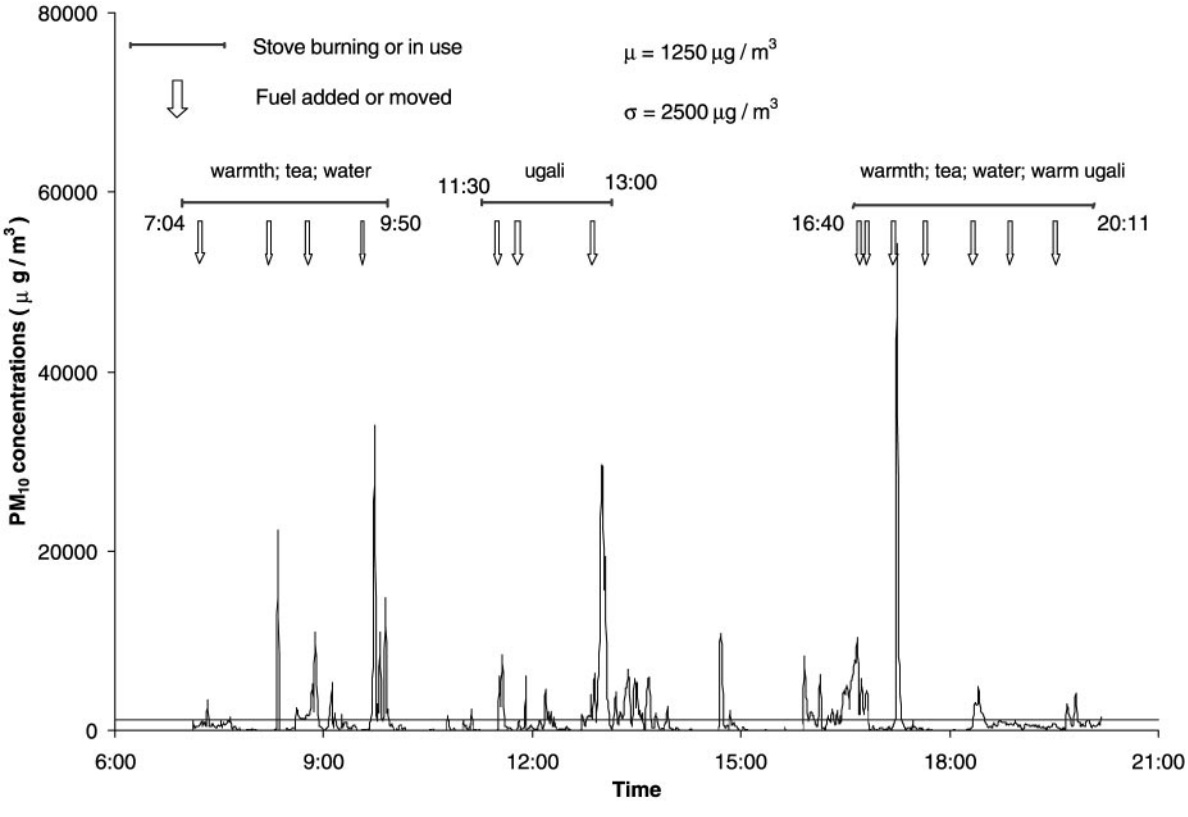


Figure 4 Day-long monitoring of pollution and cooking activities. PM_{10} concentration (at distance of 0.5 m) in a household that used a three-stone stove inside. The uses of the stove are indicated above the horizontal lines (see text for a description of ugali). The lower horizontal line indicates the mean pollution for the day. As seen, mean concentration is a poor indicator of the patterns of exposure.

exposure patterns. In brief, the authors divided the time budget of household members into the following activities: cooking, non-cooking household tasks, warming around the stove, resting and eating, playing, and sleeping. They also considered the set of potential microenvironments where each activity takes place (a total of seven microenvironments: outside plus six microenvironments inside). For example, playing or resting may take place inside the house or outside, cooking activities directly above the fire or slightly farther away, and so on. Daily exposures were then obtained using the following relationship:

$$E = \sum_{i=1}^{n} \sum_{j=1}^{7} w_j t_{ij} c_i, \qquad 1.$$

where c_i is the emission concentration in the ith period of the day, with each period corresponding to one type of activity and n representing the total number of activities for each individual (therefore the two summations together represent all the activity-location pairs for each individual, such as playing outside, cooking inside near fire, resting inside away from fire, and so on), t_{ij} time spent in the jth microenvironment in the ith period, and w_j the conversion (or dilution) factor for the jth microenvironment, which converts the emission concentration measurements to concentration at the jth microenvironment.

As described above, stove emissions exhibit large temporal variability throughout the day including intense peaks of short duration, and some household members are consistently closest to the fire when pollution level is the highest. These episodes typically occur when fuel is added or moved, the stove is lit, the cooking pot is placed on or removed from the fire, or food is stirred. This indicates that average daily concentration alone is not a sufficient measure of exposure. Therefore, in addition to mean daily concentration (m), Ezzati et al. (63) used the following two descriptive statistics for characterizing human exposure (i.e., to characterize c_i in Equation 1):

1. Mean above the 75th percentile ($m > 75$): to account for the fact that some household members are closest to the stove during high-pollution episodes caused by cooking activities.
2. Mean below the 95th percentile ($m < 95$): to eliminate the effect of large instantaneous peaks that especially occur when lighting or extinguishing the fire or when fuel is added.

Therefore the value of concentration, c_i, in Equation 1 was chosen from $m > 75$, m, and $m < 95$ based on the criteria in Table 5 in (63). For example, for cooking very close to the stove when emissions are highest, c_i was $m > 75$ of the burning period. However, for sleeping at night, when the stove is smoldering and not disturbed, c_i was $m < 95$ of the smoldering period.

Figure 5 shows exposure estimates obtained using this method, which considers the full exposure patterns and profile of individuals and is decomposed into exposure during high-intensity and low-intensity episodes. Figure 6

compares these values with the exposure estimates obtained using only average pollution concentration at a single point and time spent inside (i.e., without taking into account either the spatial distribution of pollution or the role of activity patterns).

As seen in Figure 6, the ratios of exposure estimates using average concentration at a single point to those using the exposure profile approach for the four age groups are 0.97, 0.44, 0.29, and 0.51 for females and 0.97, 0.91, 0.83, and 0.79 for males. The large variation of this ratio among the demographic groups indicates that ignoring the spatial distribution of pollution and the role of activity patterns in exposure could not only result in inaccurate estimates of exposure but also—and possibly more importantly—could bias the relative exposure levels of various demographic groups. The exposure of women, who cook and are most affected by high-intensity pollution episodes, would be underestimated most severely by using average pollution alone. This could in turn result in systematic bias in assessing the health impacts of exposure and benefits from any intervention strategy.

HEALTH IMPACT (HAZARD) ASSESSMENT

Most of the epidemiological studies of the health impacts of exposure to indoor smoke and the benefits of interventions share the following characteristics [see Table 5 in (10); (73)]:

1. the use of indirect exposure proxies such as fuel type, housing characteristics, or aggregate measures of time spent near fire;
2. case definitions of disease based on short-term monitoring, dividing the study group into those affected by disease (for example ARI or ALRI) and those not affected; and
3. emphasis on randomization as the "gold standard" for hazard assessment as a guide for design of future studies.

In the following sections we discuss the implications of each of these methodological characteristics and offer extensions or alternatives for future research when appropriate.

The Use of Exposure Proxies

Partially owing to limits and complexities of measurement technology, there has been a continued interest in the use of simple exposure proxies for studying the health impacts of indoor smoke from solid fuel use.[6] This interest is exemplified by

[6]Given that some of the early studies of indoor biomass smoke focused on pollution measurement and innovative approaches to detailed exposure characterization (50, 51) technology has not been the only cause of this interest in simple exposure indicators. Cost and time requirements may have been another consideration (74), but given the value of characterizing exposure there is still a serious under-representation of studies that pay attention to details of exposure patterns and determinants.

the 1999 Air Quality Guidelines of the World Health Organization, which state that "although work on simple exposure indicators urgently needs to be encouraged, realistically it is likely to be some years before sufficient environmental monitoring can be undertaken in most developing countries" (59, p. 134).

As discussed above, indirect exposure indicators mask the complexities of exposure to indoor smoke and may result in incorrect estimates of exposure with bias among demographic groups. Equally important, with indirect exposure proxies in epidemiological studies, the study group could often be divided only into the broad categories of exposed and nonexposed, and as a result, little is learned about the details of the quantitative relationship between exposure and health risks. Whereas this categorical approach to exposure may be appropriate for risk factors for which interventions result in risk removal (such as vitamin A and iodine supplementation or interventions that result in prevention or cessation of smoking), it does not in general allow consideration of the impacts of interventions that can result in a continuum of exposure levels or alternative population distributions of exposure that may not coincide with zero exposure (75). For example, using data on time-activity budgets and emissions from different stove-fuel combinations, Ezzati & Kammen (76) estimated that various energy- or behavior-based interventions can result in a 35%–95% reduction in exposure to PM_{10} for different demographic subgroups in rural Kenya. A two-category division of exposure would necessarily assign each intervention to one of the two categories and therefore not be able to capture the whole range of health benefits offered by the interventions.

A further limitation of exposure proxies is their inability to readily track day-to-day and seasonal variations in exposure. Emissions in a single household can vary from day to day, or season to season, because of fuel characteristics (such as moisture content or density), airflow, type of food cooked, or use of multiple stoves or fuels. Using analysis-of-variance, Ezzati et al. (63) for example found that, although considerably smaller than inter-household variation, emissions in individual households in rural central Kenya varied significantly from day to day. Activity patterns can also vary because of the seasonal nature of work and school, illness, market days, and so on. When coupled with disease patterns over time (see below), such a longitudinal analysis, as has been conducted in the case of ambient air pollution (77), can provide useful information on the most important determinants of exposure and disease, not only on average but also in different days or seasons.

The Alternative to Exposure Proxies

Yerushalmy & Palmer (78) and Murray & Lopez (75) discussed the multiple levels of causality in risk assessment.[7] Further, using historical analysis of research on disease causation, Evans (79, 80) found that best available measurement and monitoring technology plays an important role in studying and identifying causal agents at different causality levels. Although much of this discussion has focused

[7]Yerushalmy & Palmer (78) referred to the factors at different causality levels as agents and vectors of disease. Murray & Lopez (75) divided the levels of causality into distal, proximal, and patho-physiological.

on causation, the results can be extended to the quantitative relationship between exposure and health outcome.

For exposure to solid fuel smoke, the relevant risk factors include socioeconomic status at the most distal level; housing and ventilation, energy technology, and time-activity budget at a more proximal level; stove emissions; and finally the exposure and dose of the numerous pollutants or combinations of pollutants in smoke. Using each of the distal factors alone as an exposure indicator will mask the fact that human exposure is often determined by their interactions, which change over time and from place to place, requiring different intervention strategies. For example, the choice of wood for fuel is likely to result in considerably higher infant and child exposure where cooking and living areas are the same or where infants are carried on their mothers' backs than where a separate cooking quarter exists. Even if using some of the more proximal factors as exposure indicators, such as carbon monoxide (CO) concentration as a proxy for particulate concentration (itself a proxy for the hazards of multiple pollutants that are present in biomass smoke), which has been advocated based on arguments about cost of measurement (74), specific exposure conditions must be accounted for. Both physical analysis of the combustion process (71) and statistical analysis of the relationship between CO and PM_{10} concentrations (8) have shown that the relationship between the two pollutants is highly dependent on the fuel-stove combinations and conditions of cooking and therefore requires local calibration. Moreover, because average concentration may be an inadequate indicator of exposure (Figure 6) and because temporal and spatial patterns for CO (a gas) and particulate matter differ, even correlation between average concentrations will make the former only a crude measure of individual exposure to the latter.

For reasons of cost and simplifying research and program evaluation, it is necessary to develop a set of indicators for exposure to indoor smoke, especially in lower-income developing countries. At the same time, given the complexities of exposure and the state of available measurement technology, it is crucial that the parameters determining the relationship between the indicator (whether distal or proximal) and exposure are estimated and calibrated in local pilot projects with potential sources of uncertainty identified. This area has been successfully pursued in research on ambient air pollution (14, 81) and more recently indoor air pollution (63, 82–85). Further, as the emphasis for exposure proxies moves toward more distal risk factors such as stove-fuel combination, housing, and time-activity budgets, multiple indicators representing multiple risk factors should be combined to provide a matrix of exposure determinants and levels.

Case Definition

In studying the health effects of solid fuel smoke, even when using systematic diagnostic criteria, case definition has often been based on incidence or prevalence, by dividing the study group into those who are affected by disease and those who are not at one point in time [see Table 5 in (10) for a summary of the studies]. Although

this approach can readily capture mortality or chronic conditions [such as chronic obstructive pulmonary disease (COPD)], it is less suited for short-duration and episodic diseases such as acute respiratory infections (ARI), which affect a large proportion of the population at some frequency and severity. For common, short-duration, and episodic diseases a more useful measure of disease is the frequency of illness or fraction of time affected by disease (which combines incidence with duration of each episode) over an extended period. Such a time-based (versus event-based) measure allows each individual to be in a continuous range between 0 and 1 rather than in either 0 or 1. To provide an even more complete indicator of the burden of disease, in addition to incidence and duration, a severity measure can be added, or alternatively acute lower respiratory infections (ALRI) and acute upper respiratory infections (AURI) can be analyzed separately. An additional advantage of a longitudinal approach to disease monitoring and measurement is that, if coupled with corresponding longitudinal data on exposure (as described above), it can show how exposure fluctuations over a period from a few days to a season can affect disease patterns.

Emphasis on Randomization

Recent emphasis in study design for understanding and quantifying the health impacts of exposure to indoor smoke and the benefits of interventions has been on the need for experimental studies that allow randomization of the study group, especially randomized intervention studies, as the epidemiological gold standard (10, 11, 73).

Heckman & Smith (86) and Britton et al. (87) reviewed the conceptual arguments for and against randomization (or randomized social experiments). The most compelling reason for randomized studies is avoiding selection bias and confounding (88) (i.e., removing the effect of variables that may be correlated with the risk factor of interest—in this case exposure to indoor smoke—and influence the outcome or participation in an intervention). For example, socioeconomic variables are likely correlated with exposure to indoor smoke and also determine nutritional status or access to medical services for case management, which affect the same disease (73, 89, 90).

By avoiding selection bias and confounding, randomization (especially randomized controlled trials of interventions) will, first, persuade the most skeptical analysts of the causal relationship between exposure to indoor solid fuel smoke and disease and, second, provide an indication of the mean effect of exposure or an (existing) intervention on the average participant. Randomized intervention trials, however, cannot address a number of important questions:

1. Because intervention studies take a long time to show effects when disease risk is dependent on accumulated exposure (as for COPD or lung cancer), they cannot readily address the issue of chronic risk.
2. More importantly, randomized trials do not show the benefits of an intervention on those who choose to participate in intervention programs when

they are implemented in large scale. This shortcoming is a well-known phenomenon in research on the health effects of risk factors and interventions for which program participation is highly dependent on individual behavior, such as treatment and counseling for problem drug users (91, 92). The program evaluation literature in public health sciences has traditionally avoided the determinants of this difference between efficacy and community-based effectiveness and focused on its magnitude in order to re-adjust the estimates of the former. In practice, however, these determinants are likely to be important components of the underlying social and economic variables, which can affect the success of large-scale intervention efforts, as illustrated by the analogous research in the social sciences on program evaluation and a limited number of examples in public health and medicine (86, 87, 93–95).

3. Finally, intervention trials do not capture the complex determinants and patterns of exposure that are crucial for designing new interventions or combinations of interventions. Rather, a randomized experimental study can consider only the effects of current interventions (often one at a time or in limited combinations) but not the potential benefits from interventions in energy, housing, or behavioral research and development or from combining efforts from different sectors (96). This is a critical shortcoming of intervention trials, especially because, as we discuss below, in general the menu of affordable interventions for reducing the health impacts of indoor smoke is limited and based on historical trial-and-error. Given the central role of cooking in daily life, various exposure circumstances (including multi-stove or multi-fuel use (Figure 7) (97, 98) are likely scenarios that require a better understanding of the exposure determinants and new intervention packages to reduce adverse health effects.

In summary, randomization addresses questions of selection bias and confounding in estimating hazards but provides little information on many questions of interest in public health, particularly patterns and determinants of exposure that can lead to design (versus choice) of better interventions and impacts of partial exposure reduction. Equally important, in assessing the benefits of interventions, randomization creates a "randomization bias," in which effects on the randomized group may be different from those on participants after actual implementation (86, 99). Given the central role of household energy technology and housing in daily life, this differential participation is an important factor. In this manner, the role of randomized trials in informing program design for indoor air pollution is different from interventions such as vitamin A or iodine supplementation for which fairly uniform and widespread implementation may be possible. As discussed by Heckman & Smith (86), selection bias and confounding arise from a lack of data, and the best way of handling it is collecting better data. Similarly, it has been found that with proper measurement and control for various explanatory variables and with similar exclusion criteria, the results of randomized and nonrandomized studies are similar (100). The cofactors for the diseases affected by exposure to

indoor smoke are often well understood and measurable in well-designed data collection schemes and surveys, allowing nonexperimental studies to readily control for these variables.

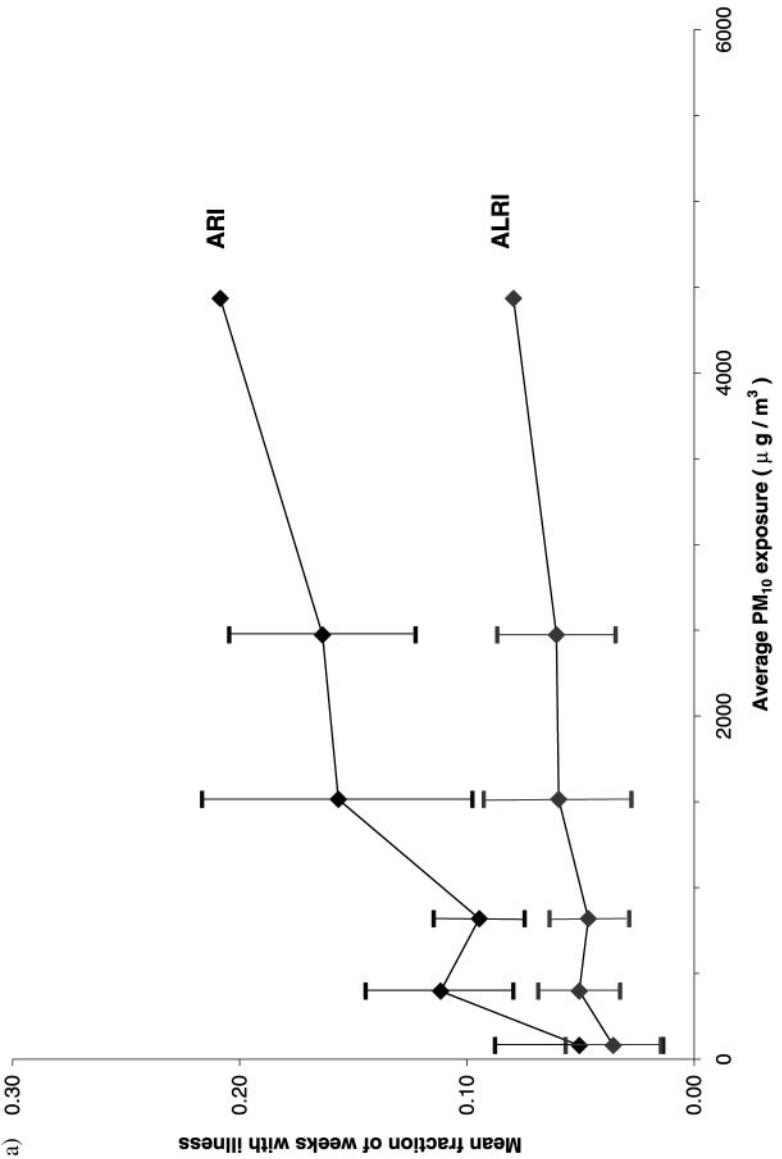
Therefore, in contrast to the suggestion of Smith et al. (10) of supplementing randomized studies with other data, we recommend the collection of better data on exposure and other factors for ARI and using randomization only as a supplement to more detailed nonexperimental data for research on indoor air pollution and health. In the short term research should include longitudinal prospective cohort studies with detailed monitoring of exposure, health, and other covariates for acute conditions, and case-control studies with retrospective exposure and other supplemental data for chronic conditions. The findings of case-control studies can be further strengthened with proper choice of controls and methods such as the proportional mortality approach used by Liu et al. (68) or spousal control, which reduces the effects of some confounding factors.

Finally, epidemiological research on the exposure-response relationship should be complemented with a better understanding of the patho-physiological mechanisms of effect. In particular, the role of high-intensity exposure raises a research question about inhalation and pulmonary deposition of particulate matter under different exposure circumstances. Important recent work has shed new light on the dispersion of aerosol bolus in human airways (101). New research that integrates modeling, laboratory testing, and field trials is needed to consider dispersion, deposition, and health impacts as a function of pollution intensity.

Recent Work on Health Impact (Hazard) Assessment

One of the first studies to consider the exposure-response relationship for indoor smoke and ARI along a continuum of exposure levels and over a relatively long period of health monitoring was that of Ezzati and Kammen (12, 13). Using detailed monitoring of individual-level exposure to indoor PM_{10} from biomass combustion, longitudinal data on ARI, and demographic and socio-economic data, the authors quantified the exposure-response relationship for ARI. Using both linear and logistic risk models, this analysis showed that the relationship between average exposure to indoor PM_{10} and the fraction of time a person has ARI (or the more severe ALRI) is an increasing function. Based on the best estimate of the exposure-response relationship, the rate of increase is higher for daily exposures below 1000–2000 $\mu g/m^3$. Although this concave shape was within the uncertainty range of the parameters of the exposure-response relationship, it was also confirmed in analysis with a continuous exposure variable for adults (for both ARI and ALRI) and total ARI in children. Figure 8 shows the unadjusted exposure-response relationship. The relationship after adjusting for age and a number of covariates is given in Tables 1*a*,*b*.

In Table 1 *female* is a variable that takes a value of 1 if the person is female and 0 if male. Therefore the coefficient of *female* is the odds ratio for illness among women relative to men when all other factors have been accounted for. *Smoking*



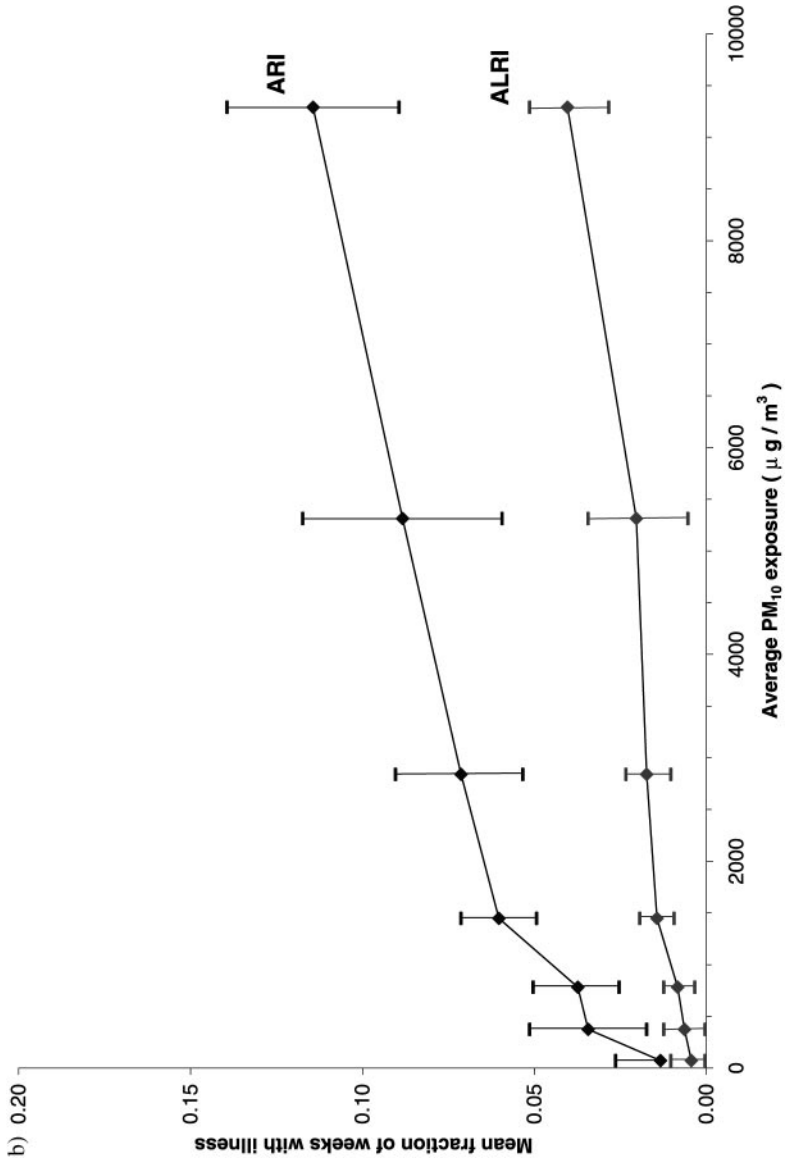


Figure 8 (*Continued*)

and *village type* take a value of 1 if a person smokes or lives in a maintenance village (versus a cattle compound) respectively, and 0 otherwise; these coefficients have an interpretation similar to *female*. The coefficient of *age* indicates the odds ratio of being diagnosed with illness with each additional year of age. In the first 60 months after birth age had an overall downward effect on susceptibility to ARI and ALRI, consistent with previous work (130, 131). Village type had no statistically significant effect after exposure and other factors were accounted for, which we attribute to comparable income levels and diets in the two village types. In fact, the type of village that a household resides in is decided by the ranch management and is not correlated with income. A part of wages is paid as food rations (milk and staple food items), which equalizes food consumption among households. Number of people in the house was not statistically significant, possibly because with a pastoralist life-style, for most of the day activity patterns are a more important determinant of the amount of time spent inside together (and therefore crowding) than the number of household members.

Direct comparison of the results with other epidemiological studies of indoor smoke and childhood ALRI is not possible because much greater detail on both exposure and health outcomes is provided in this work. At the same time, qualitative comparisons show consistency in the magnitude of odds ratio (OR) for the higher exposure groups in the Kenyan study and two cohort studies from rural Nepal (102) (OR $=$ 2.2) and rural Gambia (103) (OR $=$ 2.8), which used qualitative measures of exposure based on the time spent near fire or on mother's back during cooking. A second cohort study from the Gambia (104) shows similar results for

Figure 8 Unadjusted exposure-response relationship for acute respiratory infections (ARI) (including otitis media) and acute lower respiratory infections (ALRI) (see Tables 1*a*,*b* for adjusted relationship). ALRI, which include bronchitis, pneumonia, and broncho-pneumonia are generally significantly more severe than acute upper respiratory infections, which include infections of the upper sections of the respiratory tract including the larynx, pharynx, tonsilar glands, eustachian tube, nasal cavities, and sinuses (10, 44, 133). (*A*) Age: 0–4 years ($n = 93$ individuals). (*B*) Age: 5–49 years ($n = 229$ individuals). Each group is divided into exposure categories to reflect the day-to-day variability of individual exposure. The exposure categories for ages 0–4 years (*A*) are <200 μg/m^3, 200–500 μg/m^3, 500–1000 μg/m^3, 1000–2000 μg/m^3, 2000–3500 μg/m^3, >3500 μg/m^3. The exposure categories for ages 5–49 years (*B*) are <200 μg/m^3, 200–500 μg/m^3, 500–1000 μg/m^3, 1000–2000 μg/m^3, 2000–4000 μg/m^3, 4000–7000 μg/m^3, >7000 μg/m^3. Mean ARI and ALRI rates for each exposure category are plotted against the average exposure of the category. The shape of the curve is not sensitive to marginal modifications in exposure categories or the use of median ARI and ALRI rates (instead of mean). The larger confidence interval for the last exposure category among infants and children (*A*) is due to the small number of children ($n = 5$) for the highest exposure category. See (12, 13) for details.

TABLE 1*a* Adjusted odds-ratios for different factors affecting acute respiratory infection (ARI) (including otitis media) and acute lower respiratory infection (ALRI) rates in individuals aged 0–4 years using *b-logit* regression [see (12, 13) for details of methods and analysis]. (See text for details.) Reprinted with permission from Elsevier Science (12).

Factor	ARI OR (95% confidence interval)	*P*	ALRI OR (95% confidence interval)	*P*
Exposure category				
<200 μg/m^3	1.00	—	1.00	—
200–500 μg/m^3	2.42 (1.53–3.83)	<0.001*	1.48 (0.83–2.63)	0.18*
500–1000 μg/m^3	2.15 (1.30–3.56)	0.003*	1.40 (0.74–2.67)	0.30*
1000–2000 μg/m^3	4.30 (2.63–7.04)	<0.001*	2.33 (1.23–4.38)	0.009*
2000–3500 μg/m^3	4.72 (2.82–7.88)	<0.001*	1.93 (0.99–3.78)	0.05*
>3500 μg/m^3	6.73 (3.75–12.06)	<0.001*	2.93 (1.34–6.39)	0.007*
Female	0.99 (0.83–1.17)	0.88	0.84 (0.65–1.10)	0.21
Age	0.88 (0.83–0.94)	<0.001	0.76 (0.70– 0.84)	<0.001
Village type	1.29 (0.99–1.67)	0.06	1.18 (0.79–1.77)	0.41
Number of people in house	1.00 (0.95–1.05)	0.99	0.98 (0.91–1.06)	0.70

*Jointly significant ($p < 0.01$); OR, odds ratio.

TABLE 1*b* Adjusted odds-ratios for different factors affecting acute respiratory infection (ARI) (including otitis media) and acute lower respiratory infection (ALRI) rates in individuals aged 5–49 years using *b-logit* regression [see (12, 13) for details of methods and analysis]. (See text for details.)

Factor	ARI OR (95% confidence interval)	*P*	ALRI OR (95% confidence interval)	*P*
Exposure category				
<200 μg/m^3	1.00	—	1.00	—
200–500 μg/m^3	3.01 (1.59–5.70)	0.001*	1.65 (0.50–5.45)	0.41*
500–1000 μg/m^3	2.77 (1.49–5.13)	0.001*	1.87 (0.61–5.71)	0.27*
1000–2000 μg/m^3	3.79 (2.07–6.92)	<0.001*	2.74 (0.93–8.12)	0.07*
2000–4000 μg/m^3	4.49 (2.43–8.30)	<0.001*	3.28 (1.09–9.85)	0.03*
4000–7000 μg/m^3	5.40 (2.85–10.22)	<0.001*	3.21 (1.01–10.24)	0.05*
>7000 μg/m^3	7.93 (4.11–15.27)	<0.001*	7.10 (2.26–22.32)	0.001*
Female	1.24 (1.01–1.52)	0.04	1.21 (0.78–1.88)	0.39
Age	0.99 (0.99–1.00)	0.02	1.01 (1.00–1.02)	0.02
Smoking	1.48 (1.07–2.04)	0.02	1.53 (0.82–2.85)	0.18
Village type	0.92 (0.76–1.12)	0.41	0.93 (0.62–1.40)	0.74
Number of people in house	0.96 (0.93–1.00)	0.04	0.99 (0.92–1.07)	0.75

*Jointly significant ($p < 0.01$); OR, odds ratio.

girls (OR = 1.9) but not boys, who did not have a significant odds ratio. This was attributed to potential differences in maternal behavior with respect to female and male children. The case control studies, summarized in Smith et al. (10), are from a number of settings including Argentina, Brazil, the Gambia, India, Nigeria, South Africa, and Tanzania. Most of these studies show increased risk of childhood ARI or ALRI as a result of exposure to indoor smoke, but the range of hazard size varies. This variability is likely to reflect the differences in exposure in different settings (caused by differences in emissions, housing and ventilation, or time-activity budgets) as much as the lack of detail in measures of exposure or health outcome.

In addition to quantifying the exposure-response relationship along a continuum of exposure levels, an important finding of the Kenyan study involved the role of exposure-assessment methodology. Once patterns of exposure (including time-activity budgets and spatial dispersion of smoke in the house) were included in the estimates of daily exposure to PM_{10} (63), Ezzati & Kammen (12, 13) found that adult males and females had similar responses (i.e., coefficients of *female* were not statistically significant, as seen in Table 1). However, when exposure was estimated only from average daily PM_{10} concentrations and time spent indoors (i.e., without accounting for the specific activities and movement patterns of individuals), females older than 5 years had excess risk of ARI and ALRI (regression coefficients not reported here).

As seen in Figure 6, this commonly used method of exposure estimation underestimates the exposure of women, who regularly cook, more than men. The analysis of hazard size shows that this differential underestimation results in systematic bias in assessment of the exposure-response relationship. Controlling for the amount of cooking activity eliminated the statistical significance of sex, confirming that the role of sex was a substitute for exposure patterns (i.e., a proxy for the omitted variable of high-intensity exposure) when average daily PM_{10} concentration was used. Finally, when estimating exposure using average daily PM_{10} concentration and time alone, the role of sex appears only after the age of 5, when females actually take part in household activities, a finding that further confirms this bias.

Finally, to further consider the role of exposure patterns Ezzati & Kammen (12, 13) used two variables that were indicators of the length and intensity of exposure to high concentrations of PM_{10}. These were the amount of household cooking tasks a person performs (none, low, medium, high) and the intensity of exposure (defined as concentration during those times when a person is close to the stove and emissions are the highest). Exposure intensity did not have a statistically significant association with the incidence of ARI beyond its contribution to total (or average) exposure. At the same time, because combustion of biomass results in highly volatile pollution profiles (Figure 4), for the highest-exposure groups (notably individuals who cook) approximately half of daily exposure occurs during high-intensity episodes (Figure 5). This implies an important role for measures that reduce total exposure by reducing peak emissions.

The coefficients of the categories of participation in household tasks were not jointly significant for ARI or ALRI. However, the group that regularly participated in cooking-related tasks has additional risk of ALRI that was significant. This result implies that either long periods of exposure to very high levels of PM_{10} cause (either short-term or chronic) damage to the lower respiratory system beyond that described by the average exposure-response relationship, or the exposure of this group is underestimated even by the approach described in Reference 63 that accounts for higher exposure during cooking periods. Investigation of this hypothesis would be possible with more detailed monitoring of personal exposure. Studying the chronic impacts of high-intensity exposure would require knowledge of the history of exposure of individuals. Alternatively, it is possible to compare ALRI incidence among people who have cooked for many years with those who have just begun to cook. Finally, research on dispersion and deposition of particulates in the airways as a function of pollution intensity can shed light on the acute impacts of high-intensity exposure. Research on the role of drinking patterns has provided important understanding of the health impacts of alcohol and the benefits of interventions (105, 106). Similar research on the role of exposure patterns for indoor air pollution will be equally valuable.

RESEARCH ON INTERVENTIONS AND INTERVENTION PROGRAMS

Although reducing exposure to indoor air pollution from solid fuels can be achieved through interventions in emissions source and energy technology, housing and ventilation, and behavior and time-activity budget (18), most current research has focused on the first method with emphasis on improved stoves and fuels, which provide more affordable options in the near future than a complete shift to nonsolid fuels.

The initial emphasis of research on household energy in developing countries was on environmental impacts of biomass use, such as impacts on deforestation and desertification, resulting in a level of zeal for increased efficiency (107–111). The public health benefits from reduction in exposure to indoor smoke as well as the reduction in carbon emissions became the subject of attention soon after. This “double-dividend”—improving public health while reducing adverse environmental impacts—focused a great deal of effort on the design and dissemination of improved stoves (108, 112, 113). Initial improved stove research and development efforts, however, were often marked by a lack of detailed data on stove performance. Efficiencies and emissions, for example, were often measured in controlled environments with technical experts using the stoves under conditions very dissimilar to those in the field (110, 111). More recently, the attention of the research community has shifted from such ideal operating conditions to monitoring stove performance under actual conditions of use, taking into account the various social and physical factors that would limit the use of these stoves altogether, or

result in suboptimal performance (97, 114). As a result of these studies the initial large potential benefits from improved stoves have been questioned (71, 115).

Ballard-Tremeer & Jawurek (71), McCracken & Smith (72), Ezzati et al. (8, 63, 76) and Albalak et al. (116) are among those who have recently considered performance of exposure-reduction interventions under actual conditions of use in South Africa, Kenya, and Guatemala. McCracken & Smith (72) and Albalak et al. (116) found that the Guatemalan improved stove (Plancha) provides significant reductions in average pollution concentration. Further, Albalak et al. (116) found that the benefits of the Plancha persist over the 8-month period of monitoring under normal conditions of use with proper maintenance. Instead of focusing on statistical comparison of pollution measurements, Ballard-Tremeer & Jawurek (71) conducted a novel analysis of stove performance coupled with the thermodynamics of the combustion process for a number of South African Stoves. This analysis not only measured the performance of various stoves (efficiency and emissions) but also allowed identification of the factors besides stove type that influence performance. Using continuous real-time monitoring of emissions under actual conditions of use in 55 households for more than 200 14-hour days, Ezzati et al. (8) compared various stove-fuel combinations in Kenya based on average burning-time emissions as well as other characteristics affecting personal exposure. The comparison of different stove-fuel combinations for average burning- and smoldering-time emission concentrations is shown in Figure 9. With a relatively large sample size, this analysis also showed that all stove-fuel combinations considered (and in particular the traditional three-stone fire) exhibit large variability of emissions. For example, analysis-of-variance (ANOVA) shows that the fraction of variance of average emission concentrations for both burning and smoldering period (Figure 9) explained by interhousehold variation is approximately 18 times the fraction explained by interstove variability. Although the estimate is particularly dominated by the large variability in the emissions of the three-stone open fire,[8] this variability illustrates that how a stove is used may be as important a determinant of its emissions as the stove type. Their field results under actual conditions of use confirm the laboratory finding of Ballard-Tremeer & Jawurek (71) on the overlap between the range of emissions from open fire and ceramic stoves, although the latter on average achieved large, statistically significant reductions.

Using these data and complete determinants of exposure as discussed above, Ezzati & Kammen (76) estimated that various energy- or behavior-based interventions can result in 35–95% reduction in exposure to PM_{10} for different demographic subgroups in rural Kenya. Using the exposure-response relationship of Table 1, the authors also estimated the reductions in disease associated with these interventions. In particular, they found that, on average, the range of interventions considered

[8]Without the three-stone fire the fraction of variance of average emission concentrations for burning period (Figure 9*a*) explained by interhousehold variation is approximately five times the fraction explained by interstove variability. The ratio is approximately nine for smoldering period (Figure 9*b*).

could reduce the fraction of times that infants and children below 5 are diagnosed with disease by 24–64% for ARI and 21–44% for ALRI among those who used an open wood fire inside. The range of reductions was larger for children older than 5 and highly dependent on the time-activity budgets of individuals. These reductions as a result of environmental management in infant and child ALRI are of similar magnitude to those achieved by more costly medical interventions. Although direct extrapolation of these results to a global scale with limited data on exposure is

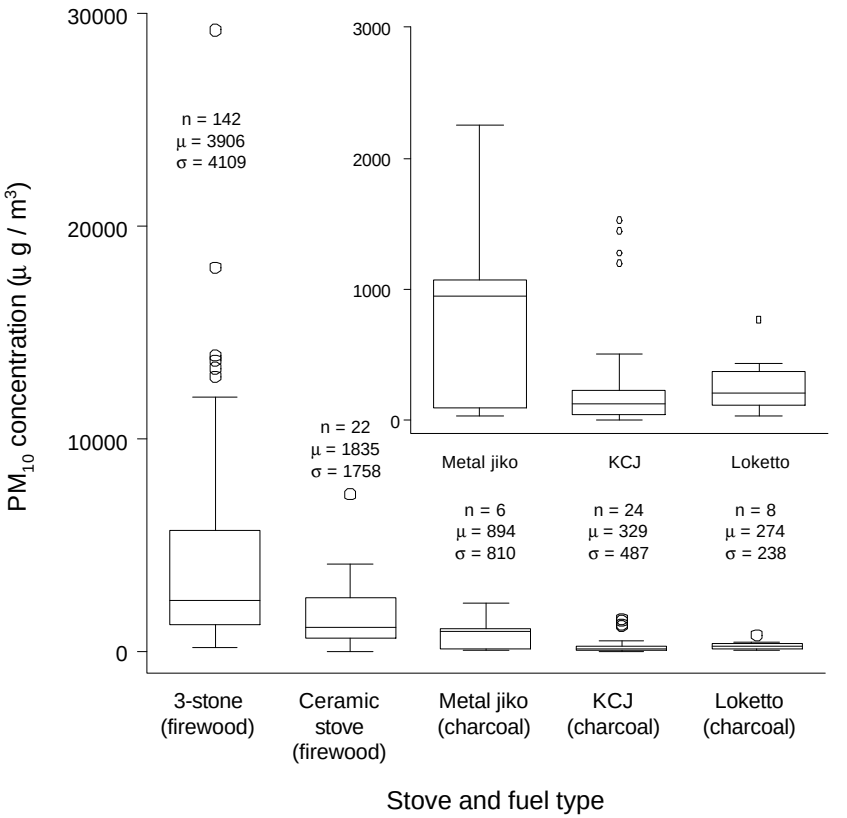


Figure 9 Day-long average of PM_{10} concentration for various stove and fuel combinations, calculated over (*a*) burning period and (*b*) smoldering period. The diagrams in the upper right hand corners are more detailed versions of the plot for the last three or four stoves. *n*, number of days of measurement; μ, sample mean; σ, standard deviation. The box-plot used in this figure shows a summary of the distribution of the variable. The lower and upper sides of the rectangle show the 25th and 75th percentiles and therefore enclose the middle half of the distribution. The middle line, which divides the rectangle in two, is the median. Reprinted with permission from (8). Copyright (2000) American Chemical Society.

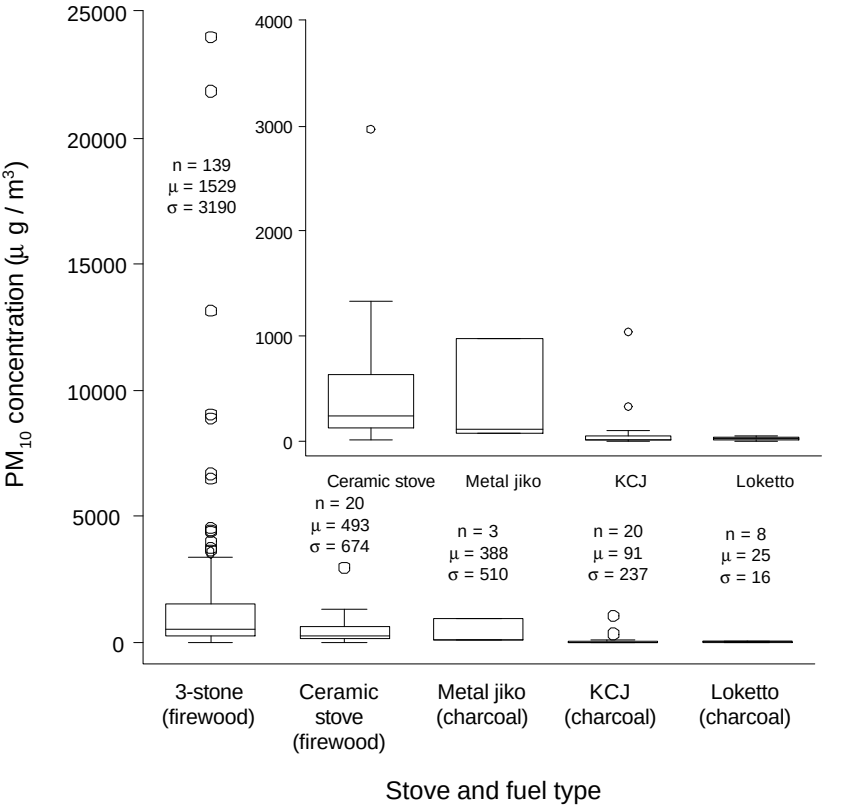


Figure 9 (*Continued*)

difficult, the consistency of epidemiological studies in different settings and the important role of ALRI in developing country mortality and burden of disease would imply large expected health benefits as a result of transitions from wood to cleaner fuels (or alternatively combinations of energy, housing, and behavioral interventions that would have equivalently large reductions in exposure) from reductions in both childhood ALRI and other diseases that have not been quantified here.

Beyond technical performance, some of the issues surrounding the success of intervention programs after community implementation (versus technology performance) have been discussed by Agarwal (97), Barnes et al. (112), Ezzati (95), Kammen (117), Hosier & Dowd (118), Manibog (111), Smith et al. (113), and von Schirnding et al. (18) using a limited number of available case studies in various countries. One reason for the lack of systematic studies of such programs may be that, with the central role of energy technology in household livelihood, the adoption of interventions is likely to vary from setting to setting and even household to household (98). Therefore, despite recent advances in program

monitoring for household energy interventions, research on the design of programs for reducing the health impacts of indoor air pollution from solid fuels must still address three key questions: First, although the benefits of adopted interventions may be known, there is little systematic evidence of what factors motivate households to adopt any intervention or suite of interventions and what the required institutions are, as illustrated by varying levels of success of different stove programs (95, 97, 111, 112, 118). Second, long-term performance of interventions in exposure reduction has not been monitored, with the exception of the recent work of Albalak et al. (116), which ensured proper maintenance. Third, knowledge is scarce about the wider environmental and socio-economic implications and sustainability of proposed interventions. For example, encouraging a shift to charcoal, which offers significant health benefits compared with wood (76), could lead to more severe environmental degradation because, given the current charcoal production methods, more wood may be needed per meal when cooking with charcoal than with wood (119). Further, the political economy of charcoal production and markets has been found to be complex, influencing access to this fuel for different sectors of society (120).

Based on the above discussions some important issues for future research include:

1. Incorporating the conditions of exposure into intervention design and evaluation. For example, given the important role of peak emissions in total daily exposure (Figure 5), the design of new interventions such as new stove technology should give as much attention to "worst-scenario" emission—such as emission during lighting, extinguishing, or moving of fuel—as average emission levels.
2. Acknowledging the complex nature of household energy use and considering scenarios that include potential energy-housing-behavior combinations, including multi-stove and multi-fuel scenarios.
3. Longitudinal monitoring of both technical performance and adoption, including the role of community networks in facilitating or impeding technology adoption.
4. Monitoring or anticipating the social, economic, and environmental implications of each intervention strategy beyond its impacts on exposure reduction.
5. Examining the factors that facilitate or impede the development of entrepreneurial networks for designing and marketing locally manufactured energy technology or housing designs.

CONCLUSIONS AND RECOMMENDATIONS

We have reviewed the current knowledge and important gaps in understanding the health impacts of exposure to indoor smoke from solid fuels, focusing on acute respiratory infections. Epidemiological research and recent risk assessment

provide strong evidence of large health effects from this risk factor on ARI, COPD, and lung cancer (from coal), and moderate evidence or indications of impacts on other diseases. We have argued that solid fuel combustion and other determinants of exposure to indoor smoke are complex phenomena and have discussed some of the complexities of exposure patterns based on social and physical variables. This complexity illustrates that, unless they are explicitly related to and calibrated against local parameters, simple indicators are likely to overlook important information about exposure and benefits of interventions.

In broad terms, answers to five research questions are needed for understanding the health effects of exposure to indoor smoke so that appropriate interventions and policies can be designed and implemented:

1. What factors determine human exposure and what are the relative contributions of each factor to personal exposure? These factors include energy technology (stove-fuel combination), housing characteristics and ventilation (such as the size and material of the house, the number of windows, and arrangement of rooms), and behavioral factors (such as the amount of time spent indoors or near the cooking area).
2. What is the quantitative relationship between exposure to indoor air pollution and disease (i.e., the exposure-response relationship)?
3. Which determinants of human exposure will be influenced, and to what extent, by any given intervention strategy?
4. What are the impacts of any intervention on human exposure and on health outcomes, and how would these impacts persist or change over time?
5. What are the broader environmental effects of any intervention, its costs, and the social and economic institutions and infrastructure required for its success?

In addition to the variables discussed in this chapter, data must be collected on other important determinants of ARI such as nutritional status (including breastfeeding for infants) (89, 121), which may not only act as confounding variables but also, and possibly more importantly for risk management, interact with and modify the effects of exposure to indoor smoke. Also, because comorbidity is very common among different childhood (infectious) diseases (122, 123), these competing dependent risks should ideally be considered together for understanding how overall child morbidity and mortality would be affected as a result of reductions in exposure to indoor air pollution (124). Finally, in addition to the specific data required, longitudinal monitoring of emissions, exposure, and disease is needed to provide not only better estimates of average or total effects (by accounting for short- or long-term variability) but also additional insight into the temporal patterns of these variables, including seasonal changes, which are important for planning health services and case management.

The current number of affordable and effective interventions for reducing the risks associated with exposure to indoor smoke from household energy technology

in developing countries is limited. Possible causes include overlooking the complexities of household energy and exposure in designing new interventions and a lack of infrastructure to support technological innovations, marketing and dissemination, and maintenance. Even less is known about combinations of technologies that may be used by any household and the factors that motivate the households to adopt them. For this reason, randomized intervention trials, which focus on the effectiveness of a limited number of existing interventions under tightly controlled conditions, may not provide the most useful information for large-scale interventions, despite being epidemiologically convincing and suitable for risk factors that can be characterized with few variables. Randomized trials will nonetheless continue to play a very important role in verifying some of the effects estimated from nonexperimental or indirect methods. Therefore, a selected number of such studies must supplement more-detailed data collection.

Further, to realistically monitor exposure, health effects, and interventions in a large number of settings, indicators for some of the variables of interest will have to be developed. At the same time, it is important to use an array of indicators when they consist of more distal factors and to calibrate indicators locally. The exact choice of the appropriate indicators itself requires detailed pilot projects that illustrate the strength of different variables as predictive variables of exposure and health impacts. An important implication of the above discussions is that reliable data on even the most quantitative variables, such as exposure and its determinants, require an integration of methodology and concepts from a variety of disciplines in the physical, social, and health sciences. Given the fundamental interactions of these variables, integration of tools and techniques should take place early in the design of studies as well as in data collection, analysis, and interpretation.

The successes and failures of intervention programs for improving health through household and community water and sanitation programs, agricultural projects, or tropical disease management have been studied in some detail (125–129). These experiences, and more recent ones with improved-stove programs, show that overlooking the complexities of individual and household behavior when public health is interconnected with household-level technology and daily life can result in well-intended programs that may either face resistance during implementation or not achieve their intended goals (95, 97, 107).

Quantitative research on health risks and interventions should at the most fundamental level be motivated by the need to improve human health in ethical, sustainable, and cost-effective ways. The data needs raised in this paper go beyond simply identifying those most affected by exposure to indoor smoke, and describe the complex mechanisms of impact and measures for reducing negative health effects. By addressing the research needs at various scales, from epidemiology to risk analysis to intervention assessment, they provide the knowledge base for expanding the limited number of current interventions and creating effective programs to reduce disease burden from indoor air pollution in developing countries.

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A

B

Figure 3 Household members involved in cooking are exposed to episodes of high pollution levels when they work directly above or very close to the fire. (*B*) reprinted from (12) with permission from Elsevier Science.

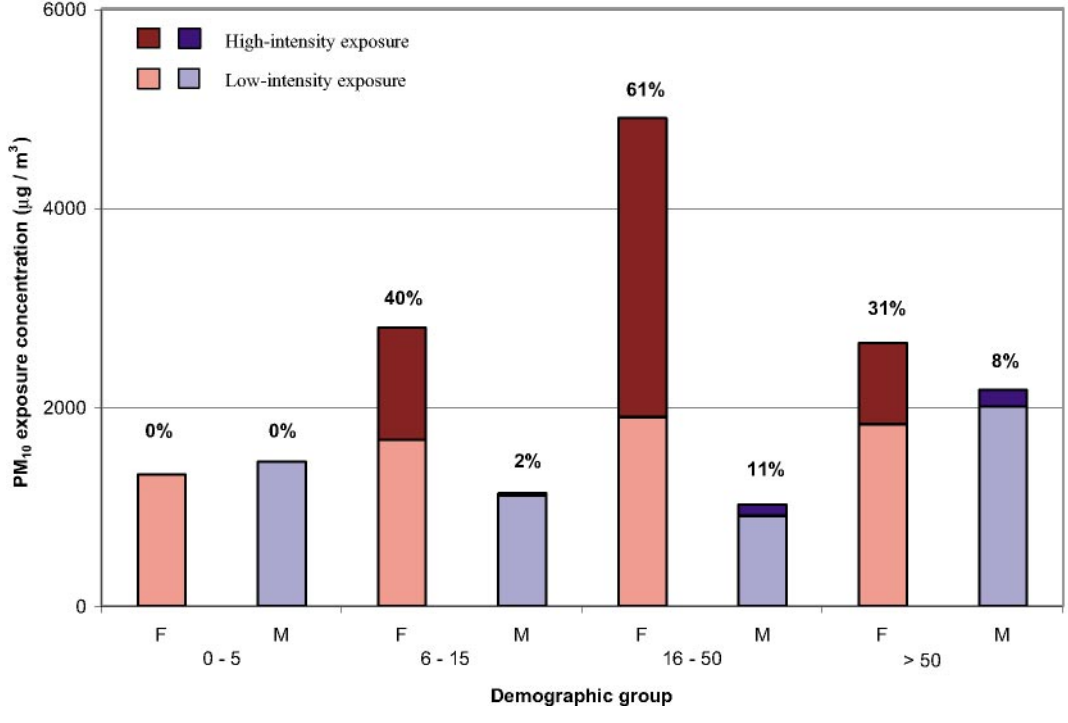

Figure 5 Breakdown of total daily exposure to PM_{10} into high-intensity (darker shade) and low-intensity (lighter shade) exposure. For each demographic subgroup the total height of the column is the group average exposure concentration divided into average for high- and low-intensity components. The percentages indicate the share of total exposure from high-intensity exposure. The high-intensity component of exposure occurs in less than one hour, which emphasizes the intensity of exposure in these episodes. See Reference (63) for details.

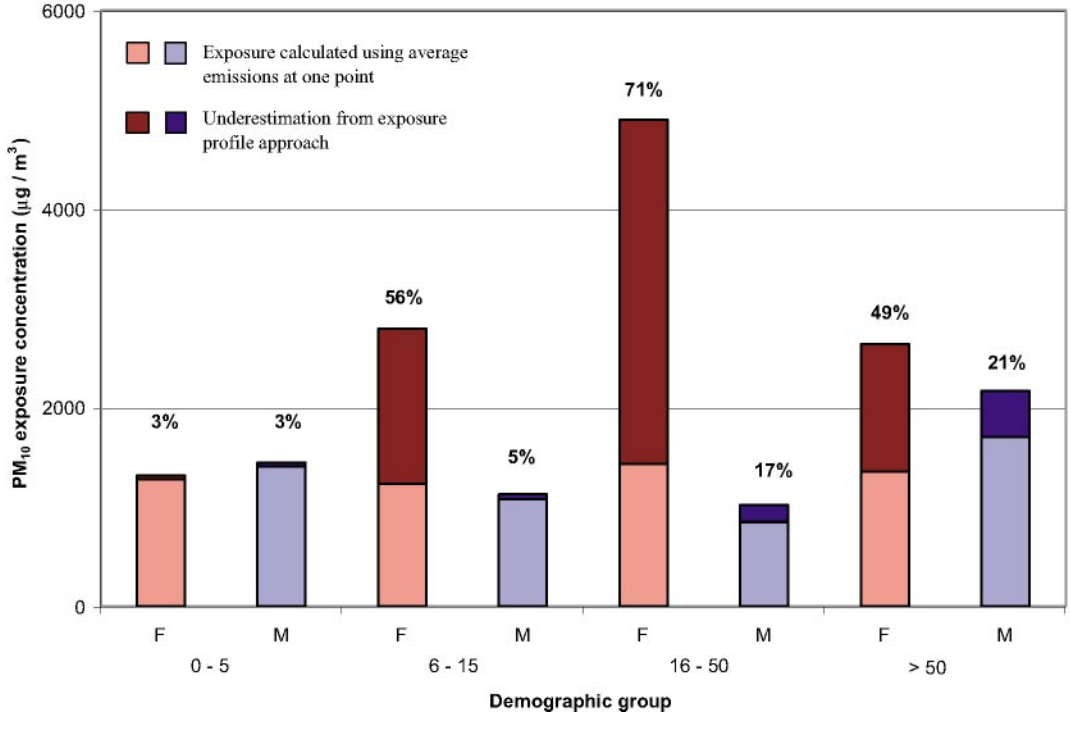

Figure 6 Comparison of exposure values that take into account temporal and spatial characteristics of pollution concentration and individual time-activity budgets with those using average emissions at a single point and time spent inside (without accounting for spatial dispersion and activity). For each demographic group the height of the column is the group average (from Figure 5). The lighter shade is exposure calculated using average emissions at a single point. Therefore, the darker shade is the underestimation of exposure using this method relative to the exposure profile approach, also shown as a percentage. See Reference (63) for details.

Figure 7 Traditional open fire and ceramic woodstove used simultaneously. Because households may use combinations of different fuels, stoves, cooking locations, and other energy-related behavior, focusing on individual interventions in randomized controlled trials may not provide realistic estimates of program effects after implementation.

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ERRATA

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