The Health Impacts of Exposure to Indoor Air Pollution from Solid Fuels in Developing Countries: Knowledge, Gaps, and Data Needs

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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 has been implicated, with varying degrees of evidence, as a causal agent of of disease and mortality in developing countries. We review the current knowledge on the relationship between indoor air pollution and disease, and on the assessment of interventions for reducing exposure and disease. Our review takes an environmental health perspective and considers the details of both exposure and health effects that are needed for successful intervention strategies. We also identify knowledge gaps and detailed research questions that are essential for successful design and dissemination of preventive measures and policies. In addition to specific research recommendations, we conclude 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.

Key Words: Household Energy, Developing Countries, Exposure Assessment, Exposure-Response Relationship, Indoor Air Pollution, Intervention, Public Health

Contents

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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 half of national energy and as much as 95% of domestic energy in many lower-income developing countries (*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: particulate matter (PM), carbon monoxide (CO), nitrogen dioxide, sulfur oxides (mainly from coal), formaldehyde, and polycyclic organic matter, including carcinogens such as benzo[a]pyrene and benzene (*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 infection (ARI) and otitis media (middle ear infection), chronic obstructive pulmonary disease (COPD), lung cancer (for coal smoke), asthma, nasopharyngeal and laryngeal cancer, tuberculosis, perinatal conditions and low birth weight, and diseases of the eye, such as cataracts and blindness (*9-12*).

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 (*9, 10*). Although low birth weight can also have large and long-term health effects, given current

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quantitative knowledge, acute (lower) respiratory infections (ALRI) and COPD are the leading causes of mortality and burden of disease due to exposure to indoor air pollution from solid fuels.

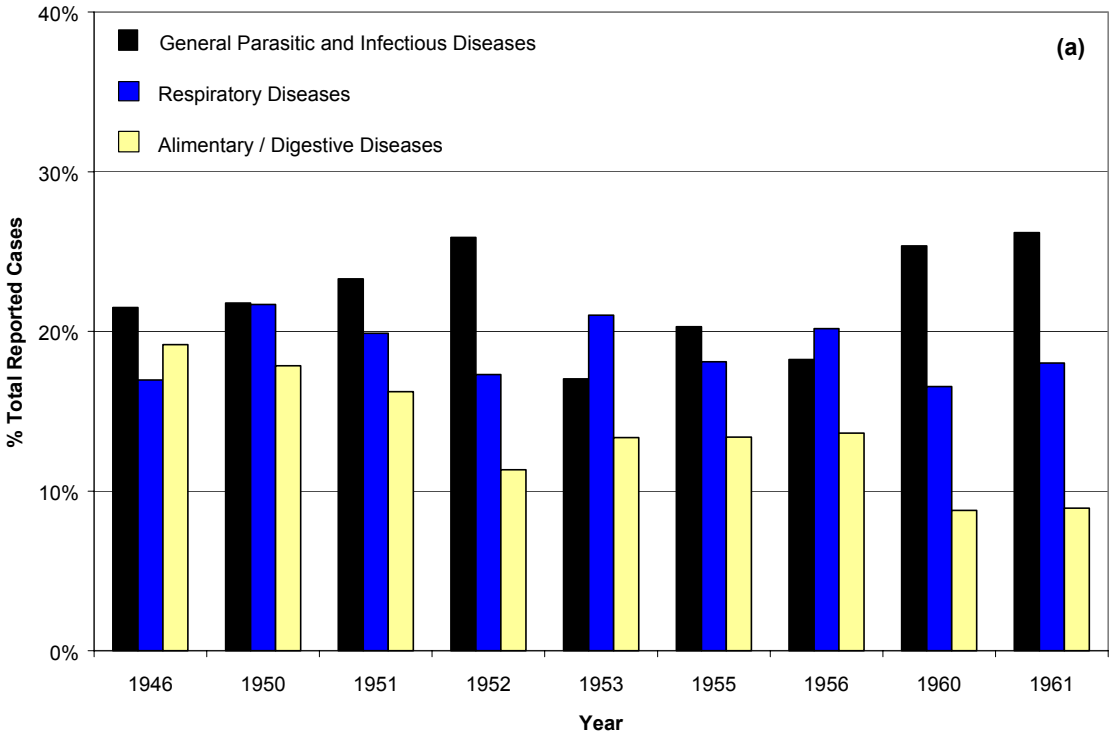
Conservative estimates of global mortality due to indoor air pollution from solid fuels show that in 2000, between 1.5 million and 2 million deaths were attributed to exposure to this risk factor (*13, 14*). This accounts for approximately 4% to 5% of total mortality worldwide. Approximately 1 million of the deaths were due to childhood ALRI, and the remainder from other causes, dominated by COPD and lung cancer among adult women (*13*).[1]

The magnitude of the health loss associated with exposure to indoor smoke as well as its concentration among the marginalized socioeconomic and demographic groups (women and children in poorer households and the rural population) have recently put preventive measures to reduce exposure to indoor air pollutionhigh on the agenda of international development and public health organizations (*10, 14, 16-19*). In this paper, we review the current knowledge on the relationship between indoor air pollution and disease (focusing on acute respiratory infection, which is the largest contributor to the burden of disease due to this risk factor), and on the assessment of 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 some of the other diseases caused by this risk factor. Our review of the health effects draws on two excellent recent review papers on the epidemiology of indoor air pollution as a risk factor (*9, 10*).

[1] Burden of disease is calculated as the number of years lost due to premature mortality plus the number of years lived with disability due to a disease with appropriate disability weights (*15*). For this reason, childhood mortality accounts for a large number of years lost due to premature mortality and a large contribution to burden of disease.

Historical Research

Respiratory diseases have consistently been among the most prevalent diseases of developing countries. Figure 1, for example, 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.[2]



[2] One may expect that in days when hospitals were less accessible, especially to the African population, only a fraction—and the most severe cases—of respiratory infections were reported; other infectious and parasitic diseases are generally more severe and likely to have had higher relative reporting rates. There may therefore 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 underreporting of deaths from respiratory and other diseases that are more common in poorer households (*20*).

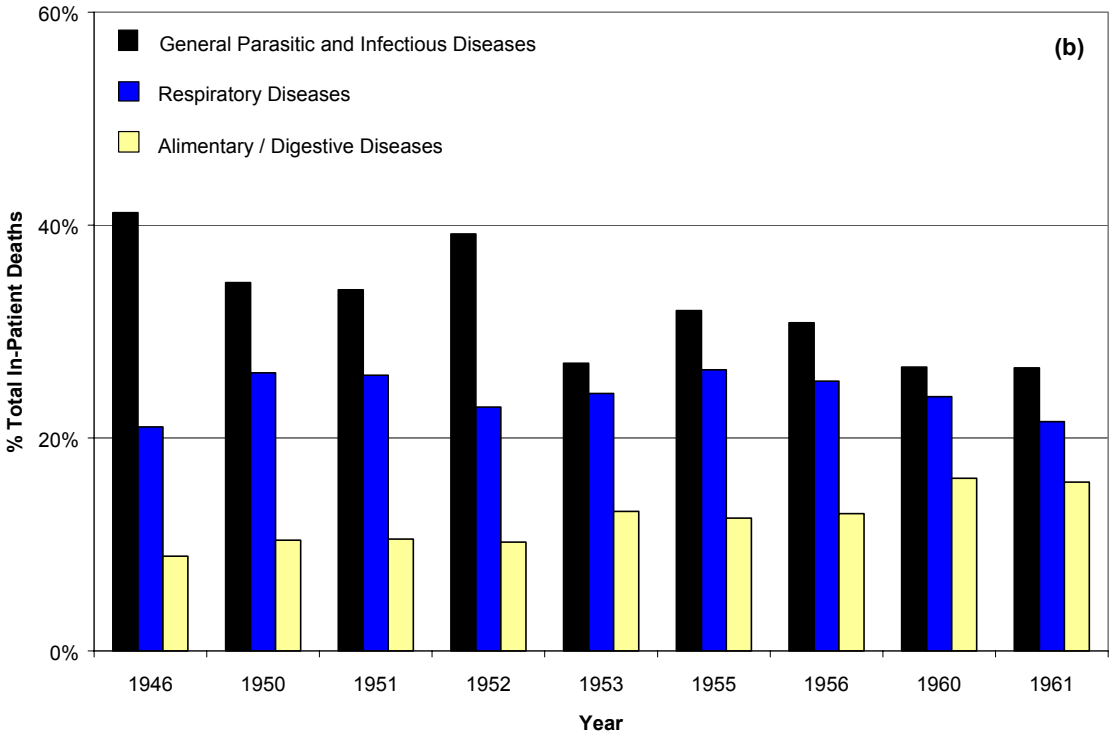


Figure 1. Common diseases of colonial Kenya. (a) Share of total number of cases treated in hospitals. The data are the share of total inpatient and outpatient cases in hospitals except for 1960 and 1961, when data were available only for inpatient cases. (b) Share of inpatient mortality. Other common diseases of these years were skin diseases and injuries, which in some years had more cases than alimentary and digestive diseases (*21*).

Respiratory infections remained an important disease in Kenya over time. In 1968, hospital records of the causes of death for the estimated 9 million outpatients and 320,000 inpatients showed the following distribution for the five most common causes of death: diseases of respiratory system (30%), infectious and parasitic diseases (26%), diseases of the digestive system (14%), blood diseases (9%), and accidents, poisoning, and violence (5%) (*20*). In nonhospital notification-of-death records the distribution of causes was diseases of respiratory system (20%), infectious and parasitic diseases (23%), diseases of the digestive system (9%),

blood diseases (6%), and accidents, poisoning, and violence (8%). In addition to being the leading national cause of mortality, respiratory diseases were also the first or second leading cause of mortality in all provinces (*20*). The contribution of respiratory diseases to morbidity in 1968 was of similar importance. Among outpatients they ranked first, with 25% of all cases, followed by infectious and parasitic diseases (21%), diseases of the digestive system (16%), and accidents, poisoning, and violence (9%). In outpatient admissions, respiratory diseases ranked second, with 17% of all cases, after infectious and parasitic diseases (25%), and followed by delivery, pregnancy, and puerperium (16%), accidents, poisoning, and violence (10%), and diseases of the digestive system (8%). The importance of respiratory diseases in hospital admissions in different provinces was consistent with the national ranking (*20*). 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 (*22*).

At the same time, with the exception of tuberculosis, diseases of the respiratory system have received mixed attention in developing countries. On the one hand, 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 for example*23, 24*). On the other, in many studies of tropical health, respiratory diseases were hardly mentioned or were not discussed at length (see for example *25, 26-32*).

Manderson also notices this systematic lack of attention to respiratory and diarrheal diseases in colonial Malay, which she attributes to the "metaphoric weight" of other diseases, especially those of an epidemic nature (*33*). An equally important factor for this lack of attention may be traced to the evolution of medical sciences in the late 19th century and much of the 20th century. The search for disease vectors and parasites, and for curative approaches that would eliminate them, dominated biomedical sciences in this period. The rise of "germ theory" in medicine, and in particular tropical medicine—which took place in a geographical context that was perceived as 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. Moreover, colonial tropical medicine had a strong presence and contribution from military doctors, whose biomedical approaches had achieved a great deal of success in combating disease among European troops overseas in the 19th and 20th centuries (*34, 35*). In this intellectual and professional context of tropical health, "neither tuberculosis nor pneumonia appeared to 'yield' to [the dominant] methods of control…" (*33*).

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 Malay, 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" (*33*). But in Malay, as in other places, this was almost exclusively associated with overcrowding of houses and other factors that would facilitate the transmission 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:

> 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 (*36*).

Other accounts of the "native huts" by health personnel also referred to crowding with people and objects and odors (*37*) but not smokiness. Even when the relationship between air pollution and respiratory health was discussed in the context of occupational health (*38*), 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 for example e.g., *20, 39–46*).

Current 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 (*47–52*). 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, 14, 16, 53–57*).

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} to be below 50 $\mu g/m^3$. In contrast, typical 24-hour average concentration of PM_{10} (particulates smaller than 10 microns in diameter) in homes using biofuels may range from 200 to 5,000 $\mu g/m^3$ or more, depending on the type of fuel, stove, and housing (*6, 8, 9, 53, 58, 59*). Concentration levels, of course, depend on where and when monitoring takes place, since within a house, and even from room to room, significant temporal and spatial variations may occur (*8, 60–62*). Ezzati et al. (*62*), 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. Overall it is estimated that approximately 80% of total global exposure to air-borne particulate matter occurs indoors in developing nations (*53, 59*). Levels of carbon monoxide and other pollutants also often exceed international guidelines (*6, 8, 59, 63*).

Bruce et al. (*10*) have reviewed the epidemiological evidence for the health effects of indoor smoke from solid fuels. The authors concluded that despite the limitations of methodology, the combination of epidemiological studies as well as 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 (*64–67*). For other health outcomes, including asthma, upper aerodigestive cancer, interstitial lung disease, low birth weight, perinatal mortality, tuberculosis, and eye diseases, Bruce et al. (*10*) classified the evidence as more tentative (moderate or weak, as classified by Smith et al. (*13*)). The details of biological mechanisms and epidemiological studies on indoor air pollution and childhood ARI were reviewed by Smith et al. (*9*), 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" (*9*). 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 on 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, for example, *68, 69*) or area monitors, mostly recording average daily or burning-time concentrations. Although personal monitors measure exposure directly, exposure is usually aggregated over time and space. This lack of detail limits a predictive assessment of the impacts of various intervention strategies on individual exposure and prevents the inclusion of the high-intensity emissions episodes that commonly occur during the combustion of biomass fuels.

Important alternatives to those approaches to pollution or exposure monitoring have been undertaken, however. Menon (*60*), Ballard-Tremeer and Jawurek (*70*), and McCracken and Smith (*71*) monitored fluctuations in emissions concentrations (PM or CO) for Indian and South African cookstoves over a period of a few hours and found that emissions from biomass stoves vary greatly over short intervals. The thorough work of Ballard-Tremeer and Jawurek further related these fluctuations to combustion characteristics, such as energy density, combustion temperature, and air flow. Ezzati et al. (*8, 62*), using more recent measurement technology, conducted continuous real-time monitoring of emissions concentrations under actual conditions of use in 55 households for more than 200 14-hour days. By also recording the status of fire (whether it was off, starting, burning, or smoldering), the type of food prepared, and other energy-use or cooking behaviors—such as adding or moving of fuel or cooking pot, stirring of food, and so on during the whole day—the authors found that the peaks in emissions concentrations 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 Figures 2 and 3.

Figure 2. Household members involved in cooking are exposed to high pollution levels when they work directly above the fire.

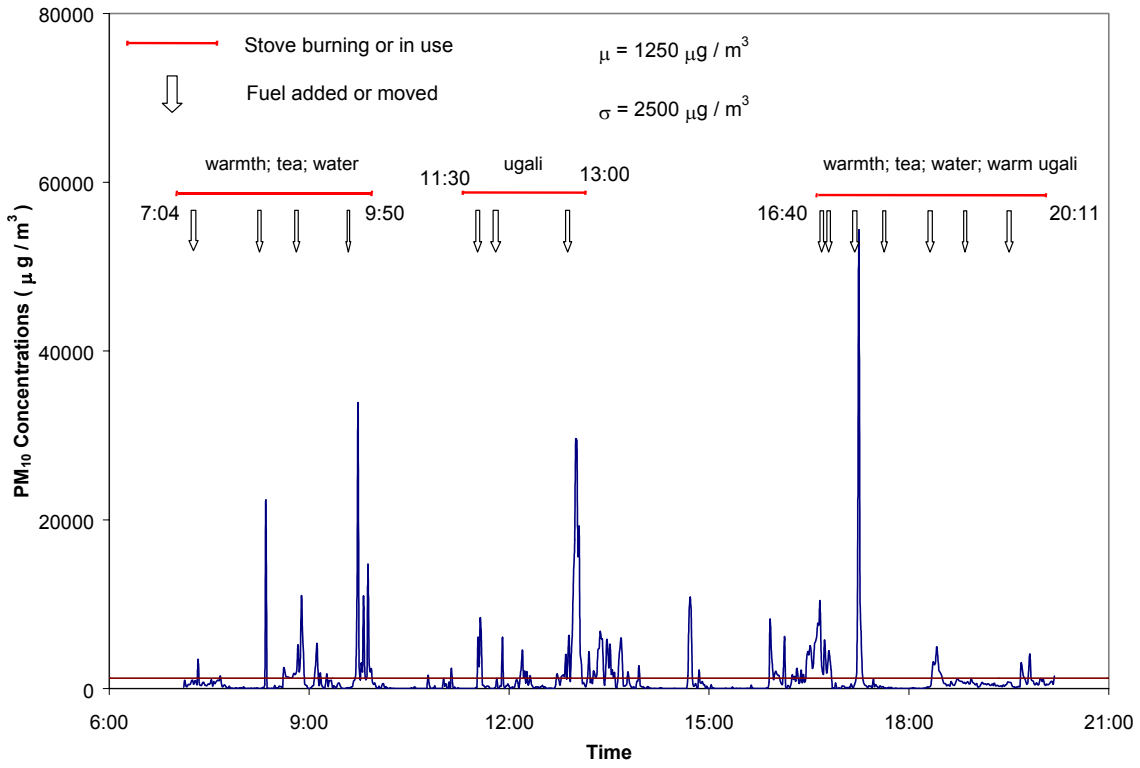


Figure 3. Day-long monitoring of pollution and cooking activities. PM_{10} concentration (at a distance of 0.5 meter) in a household that used a three-stone stove inside. The uses of the stove are indicated above the horizontal lines. The lower horizontal line indicates the mean pollution for the day. As seen, mean concentration is a poor indicator of the patterns of exposure. Ugali is a common Kenyan food made from maize or sorghum flour.

In addition to studying the temporal characteristics and fluctuations of emissions, Menon (*60*), Saksena et al. (*61*), and Ezzati et al. (*62*) 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. (*61*) estimated the contribution of each microenvironment to personal exposure. These authors found large variability among demographic subgroups in the contributions of different microenvironments, with kitchen during cooking being the largest contributor to the exposure of women (approximately 75% of exposure), followed by children (25% of exposure in winter and 40% in summer). This microenvironment made little contribution to the exposure of youth and almost none for men, who were mostly exposed in the living room microenvironment. The measurements by Menon (*60*) and Ezzati et al. (*62*) 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 (*62*)). This finding implies that the exposure microenvironments for indoor smoke are considerably smaller than those of Saksena et al. (*61*), possibly as small as 0.5 meter. Coupled with the large variability of emissions from biofuels over short 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 concentrations, are important determinants of exposure. To characterize this complexity of personal exposure to indoor particulate matter, Ezzati et al. (*62*) 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 that account for individual patterns of exposure, including daily and day-to-day variability. In particular, with continuous data on instantaneous pollution levels, this work went beyond the single measure of average daily concentration and developed *individual exposure profiles* using other descriptive statistics of emissions data that better characterize human exposure.

Figure 4 shows exposure estimates obtained using this method, which considers the full exposure patterns and profile of individuals, and decomposed into exposure during high-intensity and low-intensity episodes, respectively. Figure 5 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).

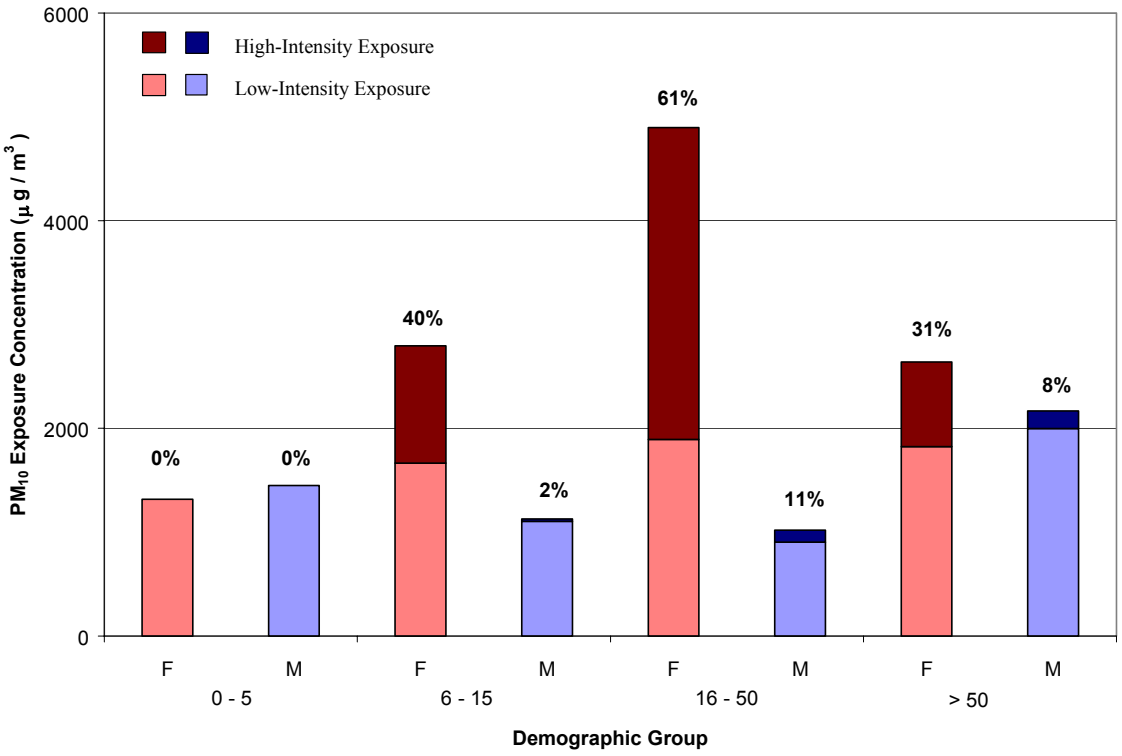


Figure 4. Breakdown of total daily exposure to PM_{10} into high-intensity (darker shade) and low-intensity (lighter shade) exposure. For each demographic subgroup the total height of the column is the group average exposure concentration divided into averages for high- and low-intensity components. The percentages indicate the share of total exposure from high-intensity exposure. The high-intensity component of exposure occurs in less than one hour, emphasizing the intensity of exposure in these episodes. See (*62*) for details.

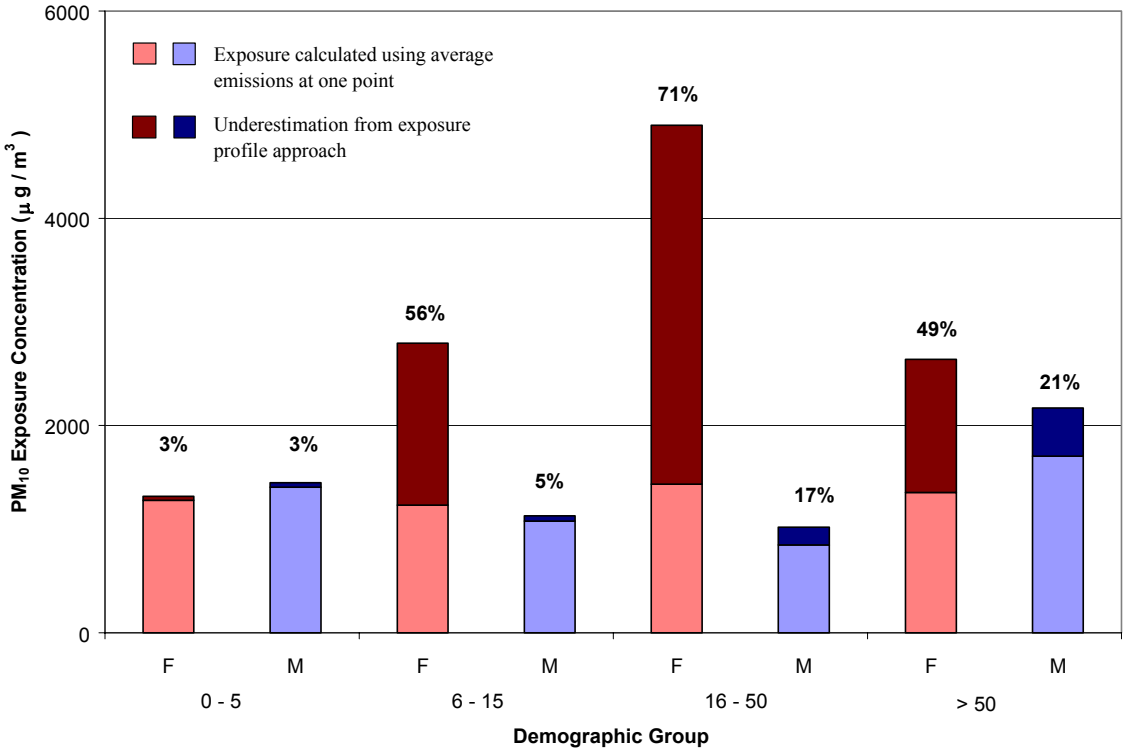


Figure 5. Exposure values that take into account temporal and spatial characteristics of pollution concentration and individual time-activity budgets, compared with those using average emissions at a single point and time spent inside (without accounting for spatial dispersion and activity). For each demographic group the height of the column is the group average (from Figure 4). The lighter shade is exposure calculated using average emissions at a single point. The darker shade is thus an underestimation of exposure using this method relative to the exposure profile approach, also shown as a percentage. See (*62*) for details.

As seen in Figure 5, 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—bias the relative exposure levels for

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 on the health impacts of exposure to indoor smoke and the benefits of interventions share the following characteristics (see Table 5 in (*9*) and (*72*)):

- use of indirect exposure proxies, such as fuel type, housing characteristics, or aggregate measures of time spent near fire;
- case definitions of disease based on short-term monitoring, dividing the study group into those affected by disease and those not affected; and
- emphasis on randomization as the "gold standard" for hazard assessment.

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 motivated by limits and complexities of measurement technology, there has been a continued interest in the use of simple exposure proxies for determining the health impacts of indoor smoke from solid fuel use.[3] This interest is exemplified by 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" (*57*).

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

[3] Given that some early studies of indoor biomass smoke focused on pollution measurement and innovative approaches to detailed exposure characterization (*48, 49*), technology has not been the only cause of this interest in simple exposure indicators. Cost and time requirements may have been another consideration (*73*), but there is still a serious underrepresentation of studies that pay attention to details of exposure patterns and determinants.

groups. Moreover, with indirect exposure proxies in epidemiological studies, the study group is often divided only into the broad categories of exposed and nonexposed, and as a result, little is learned about the quantitative relationship between exposure and health risks. Although this categorical approach to exposure may be appropriate for risk factors where interventions result in risk removal (such as prevention or cessation of smoking), it does not in general consider the impacts of interventions that can result in a continuum of exposure levels or alternative population distributions of exposure that may not coincide with complete risk removal (*74*). For example, using data on time-activity budgets and emissions from different stove-fuel combinations, Ezzati and Kammen (*75*) estimate that various energy- or behavior-based interventions can result in a 35% to 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 easily track day-to-day or 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), air flow, type of food cooked, or type of stove or fuel. Using analysis-of-variance, Ezzati et al. (*62*) found that although considerably smaller than interhousehold 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 can identify the most important determinants of exposure and disease—not only in average but also in different days or seasons, as has been conducted in the case of ambient air pollution (*76*).

The Alternative to Exposure Proxies

Yerushalmy and Palmer (*77*) and Murray and Lopez (*74*) discuss the multiple levels of causality in risk assessment.[4] Further, using historical analysis of research on disease causation, Evans (*78, 79*) finds that best available measurement and monitoring technology plays an important role in studying and identifying causal agents at different causality levels. Although

[4] Yerushalmy and Palmer (*77*) refer to the factors at different causality levels as agents and vectors of disease. Murray and Lopez (*74*) divide the levels of causality into distal, proximal, and pathophysiological.

much of this discussion has focused on causation, the results can be extended to the quantitative relationship between exposure and health outcome.

The relevant risk factors for the health impacts of exposure to indoor smoke from solid fuels include, at the most distal level, socioeconomic status, housing and ventilation, energy technology, and time-activity budgets, plus more proximal factors—stove emissions, and finally the exposure and dose of the numerous pollutants or combinations of pollutants that are present in smoke. Using each of the distal factors alone as an exposure indicator will mask the fact that individual exposure is often determined by their interactions, which change over time and from place to place, motivating different intervention strategies. For example, the choice of wood as 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 the use of the more proximal factors as hazard indicators, such as using carbon monoxide (CO) concentration as a proxy for particulate concentration (itself a proxy for health effects), which has been advocated based on arguments about cost of measurement (*73*), needs to take into account specific exposure conditions. Both physical analysis of the combustion process (*70*) 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 5) and because temporal and spatial patterns for CO (a gas) and particulate matter differ, even with correlation between average concentrations, the former will form only a crude measure of individual exposure to the latter.

In summary, 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 measurement technology, it is crucial to estimate and calibrate the parameters determining the relationship between the indicator (whether distal or proximal) and exposure—and consider potential sources of uncertainty. This is an area which has been successfully pursued in research on ambient air pollution (*80, 81*) and more recently indoor air pollution (*62, 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, case definition has often been based on incidence or prevalence, in which the study group has been divided into those who are affected by disease and those who are not (see Table 5 in (*9*) for a summary of the studies). Although this approach can readily capture mortality or chronic conditions, such as COPD, it is less suited for short-duration and episodic diseases, such as 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 only. To provide an even more complete indicator of the burden of disease, in addition to incidence and duration, a severity measure can be added (alternatively, ALRI and 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 experimental studies that allow randomization of the study group, especially randomized intervention studies, as the epidemiological "gold standard" (*9, 10, 72*).

Heckman and Smith (*86*) and Britton et al. (*87*) review 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*)—that is, 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 be correlated with exposure to indoor smoke and also affect nutritional status or access to medical services for case management that affects the same disease (*72, 89, 90*).

By avoiding selection bias and confounding, randomization (especially randomized controlled trials) 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. Intervention trials, however, cannot address a number of important issues:

- 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 do not readily address the issue of chronic risk.
- More importantly, a randomized trials do not show the benefits of an intervention on those who choose to participate in large-scale intervention programs. 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 the magnitude in order of the difference between efficacy and community-based effectiveness to readjust the estimates of the former. But in practice these determinants are likely to be important components of the underlying social and economic system and constraints that can affect the success of large-scale intervention efforts, as illustrated by analogous research in the social sciences on program evaluation and a limited number of examples in public health and medicine (*86, 87, 93–95*).
- 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 design 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 in different sectors (*96*). This is a critical shortcoming of intervention trials, especially because (as we discuss below) 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 the use of multiple stoves and fuels; see Figure 6; (*97, 98*)) are likely scenarios that require a better understanding of the exposure determinants and design of new intervention packages to reduce the adverse health effects.

Figure 6. 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 under actual conditions.

In summary, randomization addresses questions of selection bias and confounding in estimating hazards but provides little information on many questions of interest, particularly patterns and determinants of exposure that can lead to design (versus choice) of better interventions and impacts of partial exposure reduction. As importantly, in assessing the benefits of interventions, randomization creates a "randomization bias" (*86*), in which effects on the randomized group may be different from those on participants after actual implementation (*99*). Given the central role of household energy technology and housing in daily life, this differential

participation is an important factor. As discussed by Heckman and Smith (*86*), selection bias and confounding arise from 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*).

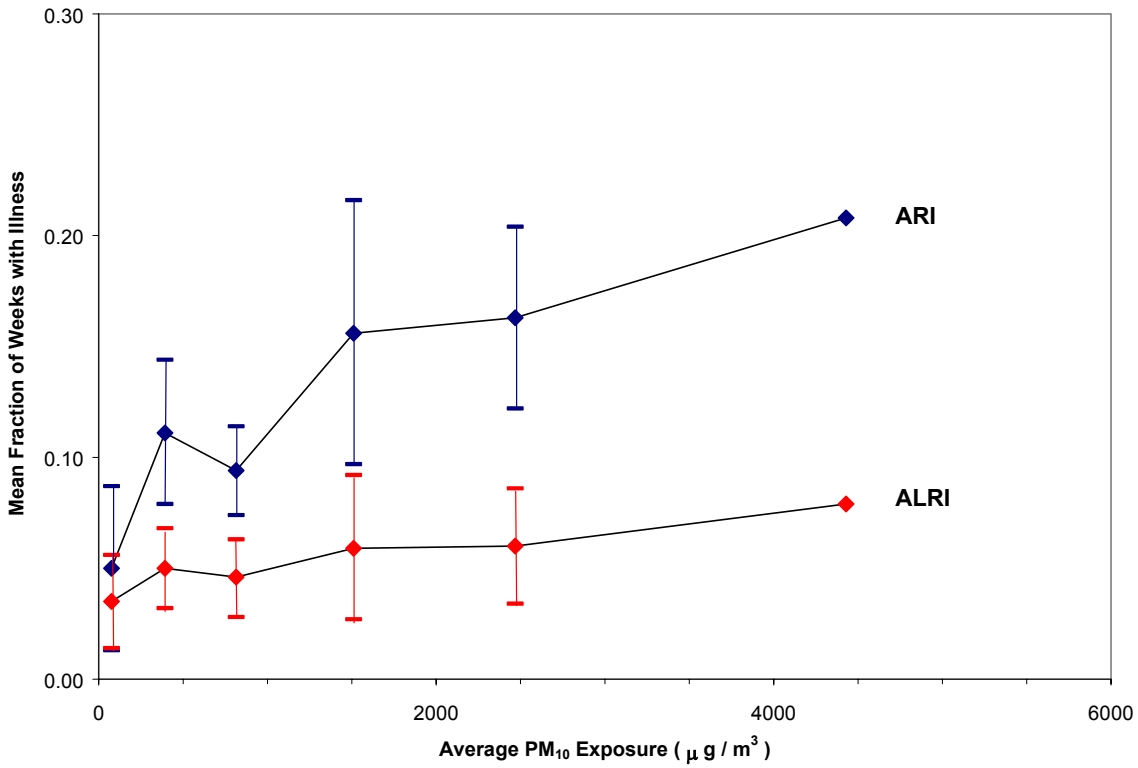
Therefore, we go beyond the suggestion of Smith et al. (*9*) on supplementing randomized studies with other data, and recommend collecting better data on exposure and other factors for ARI and using randomization only as a supplement to more detailed nonexperimental data. In the short run, research should 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 such controls as the proportional mortality approach, used by Liu et al. (*67*), or spousal control, which reduces the effects of some confounding factors.

Finally, epidemiological research on the exposure-response relationship should be complemented with an understanding of the pathophysiological 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 Hazard Assessment

One of the first studies to consider the exposure-response relationship for indoor smoke along a continuum of exposure levels and over a relatively long period of health monitoring is the work of Ezzati and Kammen (*11, 12*). Using detailed monitoring of individual-level exposure to indoor PM_{10} from biomass combustion, longitudinal data on ARI, and demographic and socioeconomic information, 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 that a person has ARI (or the more severe ALRI) is an increasing function. Based on the best estimate of exposure-response relationship, the rate of increase is higher for daily exposures below 1,000–2,000 $\mu g/m^3$ 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 7 shows the unadjusted exposure-response relationship graphically; the relationship after adjusting for age and a number of covariates is given in Table 1.



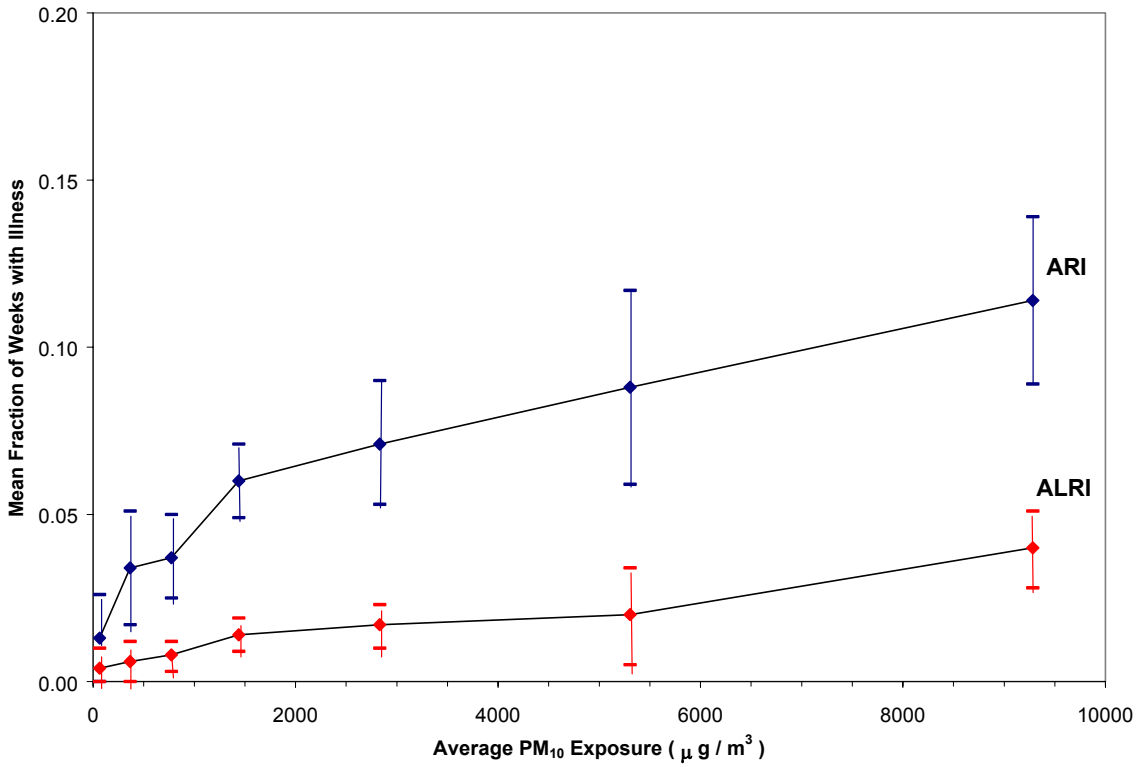


Figure 7. Unadjusted exposure-response relationship for ARI and ALRI (see Table 1 for adjusted relationship). (**a**) Age: 0–4 years (n = 93 individuals). (**b**) Age: 5–49 years (n = 229 individuals). Each group is divided into exposure categories to reflect the day-to-day variability of individual exposure. The exposure categories for ages 0–4 years (panel a) are < 200 $\mu g.m^{-3}$ 200–500 $\mu g.m^{-3}$, 500–1,000 $\mu g.m^{-3}$, 1,000–2,000 $\mu g.m^{-3}$, 2,000–3,500 $\mu g.m^{-3}$, > 3,500 $\mu g.m^{-3}$. The exposure categories for ages 5–49 years (panel b) are < 200 $\mu g.m^{-3}$, 200–500 $\mu g.m^{-3}$, 500–1,000 $\mu g.m^{-3}$, 1,000–2,000 $\mu g.m^{-3}$, 2,000–4,000 $\mu g.m^{-3}$, 4,000–7,000 $\mu g.m^{-3}$, > 7,000 $\mu g.m^{-3}$. 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 (panel a) is due to the small number of children (n = 5) for the highest exposure category. See (*11, 12*) for details.

TABLE 1

In addition to quantifying the exposure-response relationship along a continuum of exposure levels, this analysis explored the role of exposure assessment methodology. Once patterns of individual exposure (including their time-activity budgets and the spatial dispersion

of smoke in the house) were included in calculating daily exposure to PM_{10} (*62*), Ezzati and Kammen found that males and females had similar response (i.e., coefficients of *Female* were not statistically significant). On the other hand, when exposure was estimated only from average daily PM_{10} concentration and time spent indoors (i.e., without accounting for the specific activities and movement patterns of individuals), the authors found that females older than 5 had additional risk of ARI and ALRI.

As seen in Figure 5, this latter (and commonly used) method of exposure estimation underestimated the exposure of women, who regularly cook. The analysis of hazard size shows that this 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.

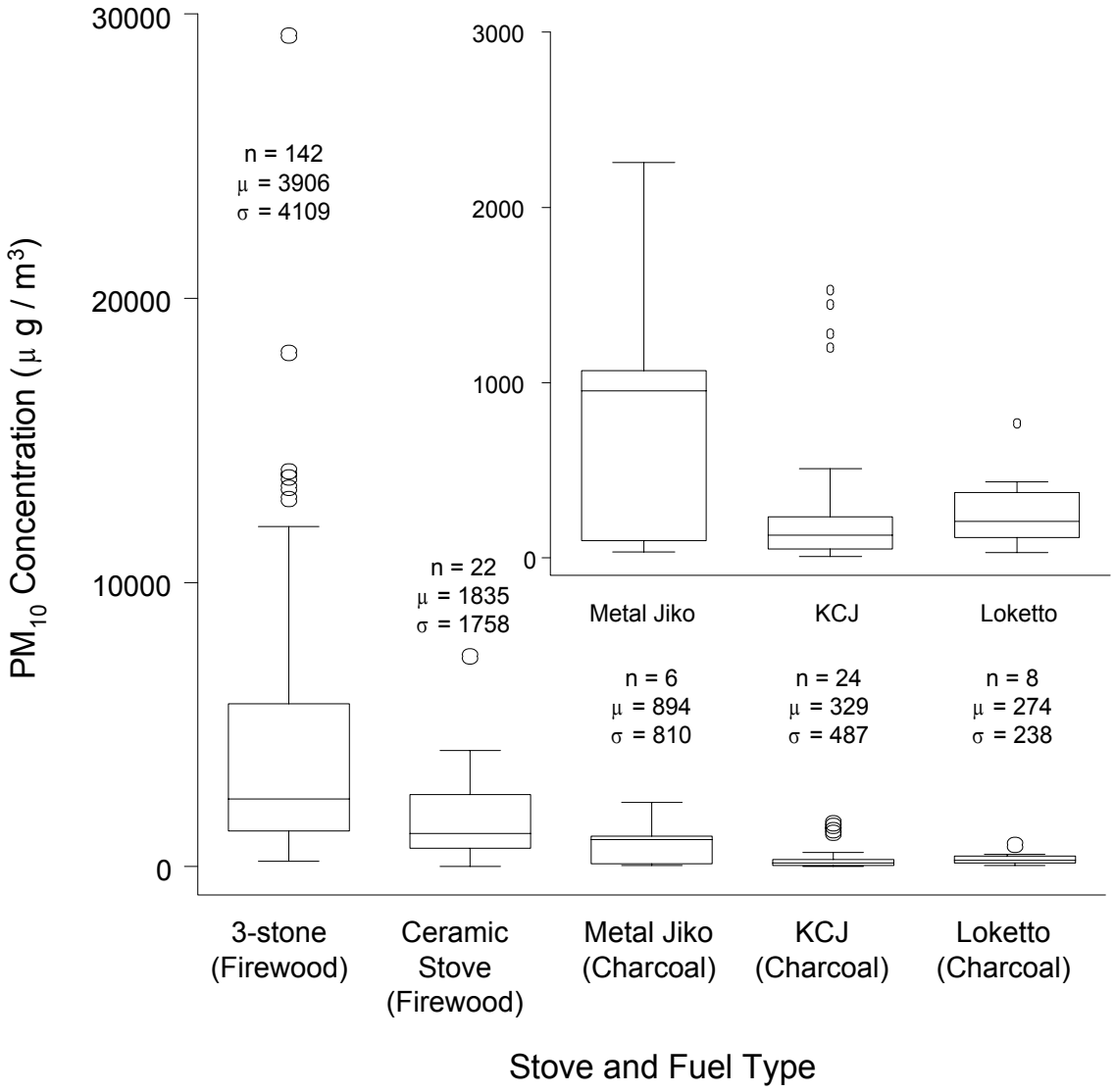
Research on Interventions and Intervention Programs

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 (*14*), most current projects have focused on the first method—using improved stoves and fuels, which provide more affordable options in the near future than a complete change 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 zeal for increased efficiency (*58, 102–105*). 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 (*102, 106, 107*). Initial research efforts on the benefits of improved stoves, 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 (*104, 105*). More recently, the attention of researchers 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, 108*). As a result of these studies, the initial high level of benefits from improved stoves has been questioned (*70, 109*).

Ballard-Tremeer and Jawurek (*70*), McCracken and Smith (*71*), Ezzati et al. (*8, 62, 75*) and Albalak et al. (*110*) are among the recent works that have considered performance of exposure reduction interventions under actual conditions of use. McCracken and Smith (*71*) and Albalak et al. (*110*) found that the Guatemalan improved stove (Plancha) provides significant reductions in average pollution concentration. Further, Albalak et al. (*110*) find that the benefits of Plancha persist over the eight-month period of monitoring under normal conditions of use with proper maintenance. Instead of focusing on statistical comparison of pollution measurements, Ballard-Tremeer and Jawurek (*70*) conducted a novel analysis of stove performance coupled with the thermodynamics of the combustion process; this allowed them not only to illustrate the efficiency and emissions performance of various stoves but also to discuss what factors besides the choice of stove could affect performance. Using continuous real-time monitoring of emissions concentrations under actual conditions of use in 55 households for more than 200 14-hour days, Ezzati et al. (*8*) compared various stove-fuel combinations for average burning-time emissions and other characteristics affecting personal exposure. With a relatively large sample size the authors also found that all stove-fuel combinations considered (and in particular the traditional three-stone fire) exhibit large variability of emissions concentrations. How a stove is used, they conclude, may be as important a determinant of emissions as its type. Their field results confirm the laboratory finding of Ballard-Tremeer and Jawurek (*70*) on the overlap between the range of emissions from open fires and ceramic stoves, although the latter on average achieved large, statistically significant reductions. The comparison of different stove-fuel combinations for average burning and smoldering time emissions concentrations is shown in Figure 8.


30000
20000
10000
0
3000
2000
1000
0
PM10 Concentration (μ g / m3)
n = 142
μ = 3906
σ = 4109
n = 22
μ = 1835
σ = 1758
n = 6
μ = 894
σ = 810
n = 24
μ = 329
σ = 487
n = 8
μ = 274
σ = 238
Metal Jiko
KCJ
Loketto
3-stone (Firewood)
Ceramic Stove (Firewood)
Metal Jiko (Charcoal)
KCJ (Charcoal)
Loketto (Charcoal)
Stove and Fuel Type

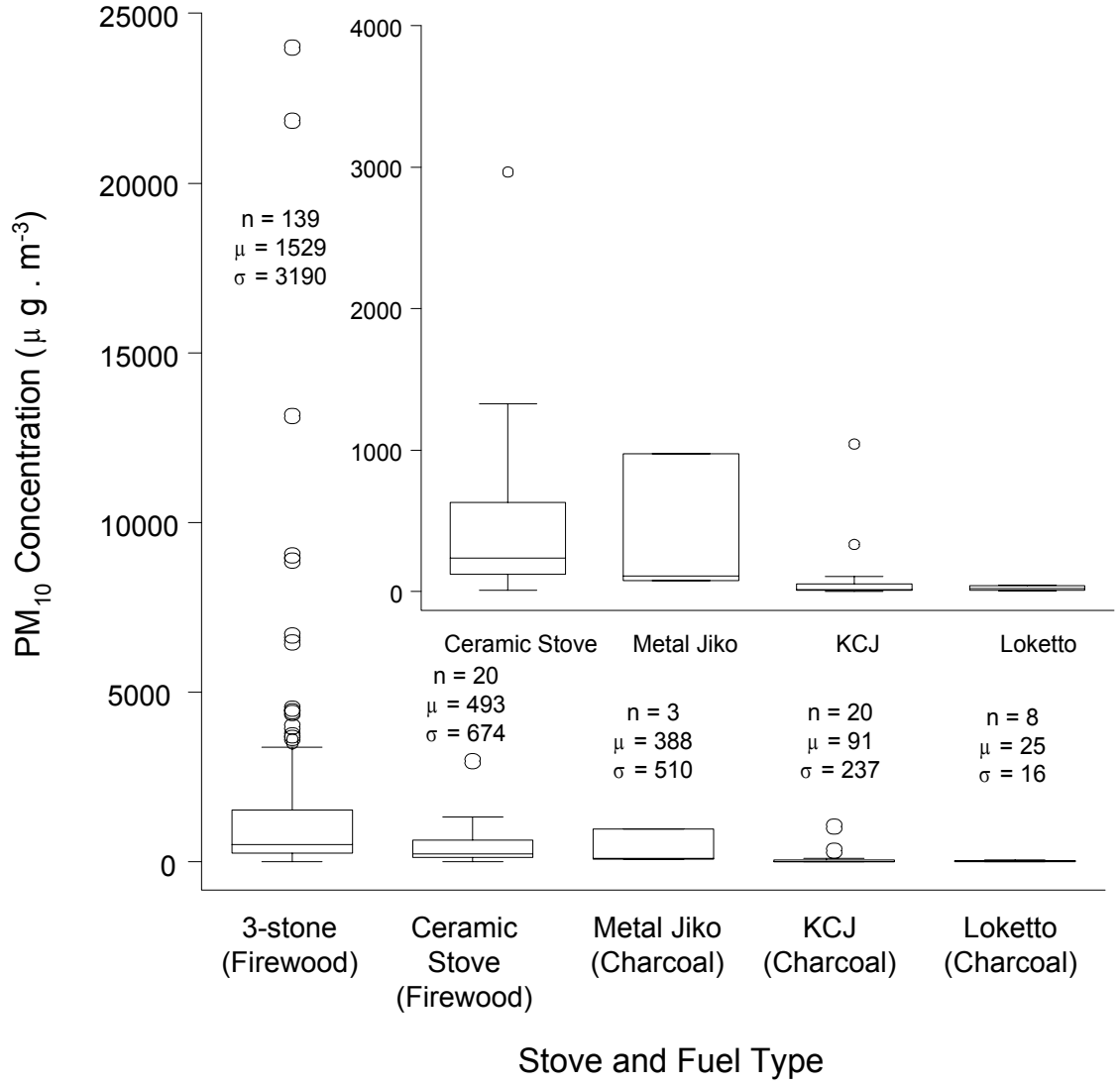


Figure 8. Day-long average of PM_{10} concentration for various stove and fuel combinations, calculated over (a) burning period and (b) smoldering period. The diagram in the upper right-hand corner is a more detailed version of the plot for the last three or four stoves. *n* refers to the number of days of measurement; μ is the sample mean, and σ is the standard deviation. The box plot 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.

Using these data and complete determinants of exposure as discussed above, Ezzati and Kammen (*75*) estimated that various energy- or behavior-based interventions can result in a 35% to 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 could reduce the number of times that infants and children under 5 are diagnosed with disease by 24–64% for ARI and 21–44% for ALRI. The range of reductions was larger for those older than 5 and highly dependent on the time-activity budgets of individuals. These reductions due to environmental management in infant and child ALRI are of similar magnitude to those achieved by more costly medical interventions.

Beyond technical performance, some of the issues surrounding the success of intervention programs after community implementation (versus technology performance) have been discussed by Agarwal, Kammen, Smith et al., and von Schirnding et al. using a limited number of available case studies (*14, 58, 97, 106, 107, 111*). One reason for the lack of systematic studies of such programs may be that the adoption of interventions is likely to vary from setting to setting and even household to household (*98*). Despite recent advances in program monitoring for household energy interventions, the design of programs for reducing the health impacts of indoor air pollution from solid fuels must still address three issues. First, although the benefits of adopted interventions may be known, it is not entirely clear what factors motivate households to adopt any intervention or suite of interventions. Second, the long-term performance of interventions in exposure reduction is uncertain—a question only recently addressed in work by Albalak et al. (*110*). Third, knowledge is scarce about the wider environmental and socioeconomic implications and sustainability of proposed interventions. For example, encouraging a shift to charcoal, which offers significant health benefits compared with wood, could lead to more severe environmental degradation because current charcoal production methods are inefficient in their use of wood (*112*). Further, the political economy of charcoal production and markets has been found to be complex, and access varies for different sectors of society (*113*).

Based on the above discussions, we can list some of the important issues for consideration in future research:

- *Incorporating the conditions of exposure in intervention design and evaluation.* For example, given the important role of peak emissions periods in determining total daily exposure (Figure 4), the design of an intervention scheme involving new stove technology should give “worst-scenario” emissions—such as emissions during the lighting, extinguishing, or moving of fuel—as much attention as average emission levels.

- *Acknowledging the complex nature of household energy use.* Researchers should consider scenarios that include potential energy-housing-behavior combinations, including multistove and multifuel scenarios.
- *Longitudinal monitoring of both technical performance and adoption.* This includes the role of community networks in facilitating or impeding technology adoption.
- *Anticipating the social, economic, and environmental implications of each intervention strategy.* An intervention may have consequences beyond its impact on exposure reduction.
- *Examiningfactors that encourage or discourage entrepreneurial networks for designing and marketing locally manufactured energy technology or housing.*

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. 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 means that unless they are explicitly related to and calibrated against local parameters, simple indicators are likely to miss important information about individuals' exposure and the 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 individual exposure? These factors include energy technology (stove-fuel combination), housing characteristics (size and material of the structure, number of windows, arrangement of rooms), and behavioral factors (amount of time spent indoors or near the cooking area).
2. What is the quantitative relationship between exposure to indoor air pollution and the incidence of 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 and the social and economic institutions and infrastructure required for its success?

The number of affordable and effective interventions is currently 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 and their marketing, 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 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

Figure 9 illustrates the research areas and questions needed for effective interventions in reducing the disease burden associated with indoor solid fuel smoke. 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 calibrate any indicator locally and to use an array whenever the indicators consist of more distal factors. The exact choice of the indicators requires a number of detailed pilot projects that illustrate the strength of different variables as predictive indicators of exposure and health impacts.

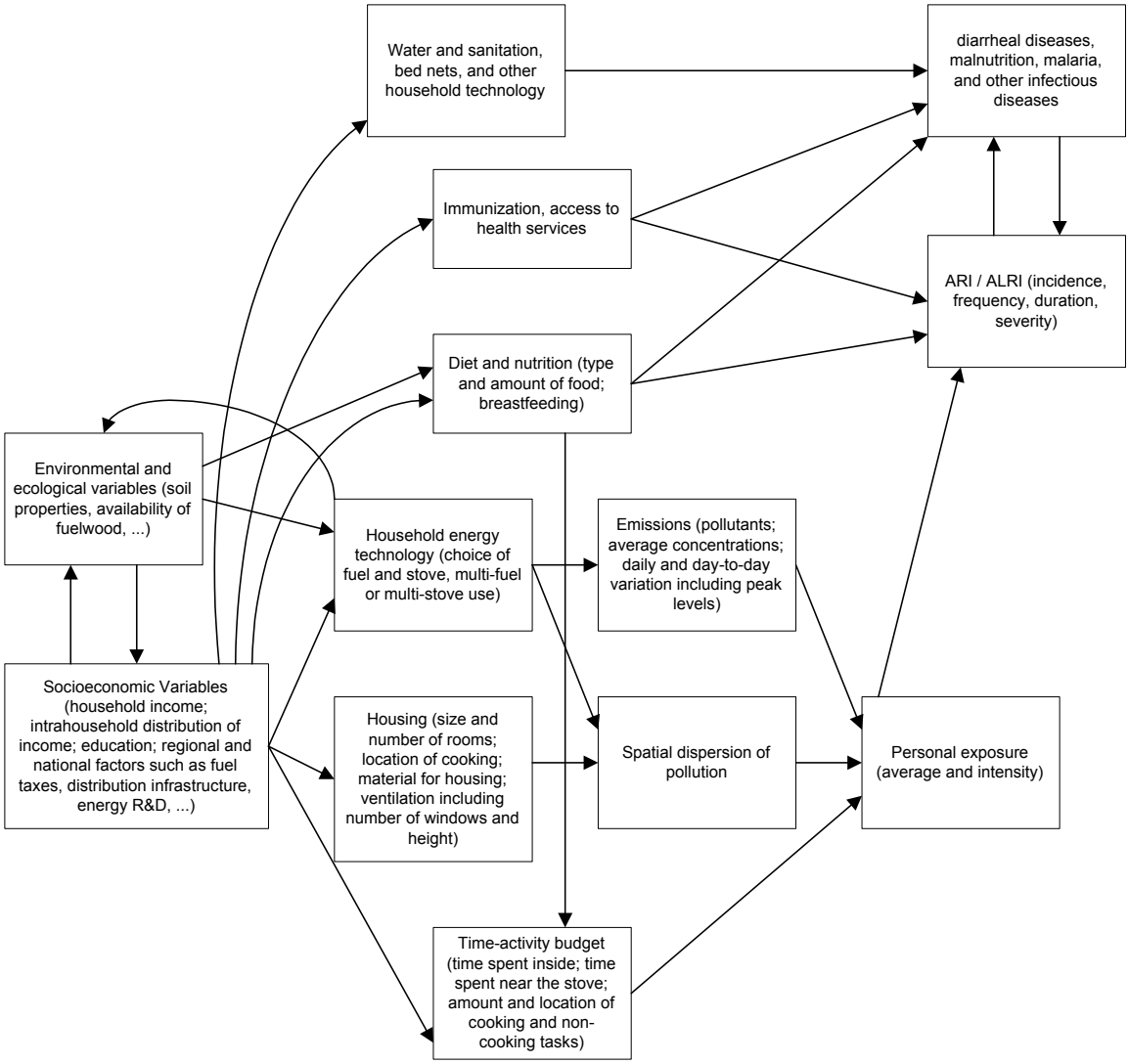


Figure 9. Important research areas, questions, and links for a predictive understanding of the health impacts of indoor smoke from household energy use. The choice of household technology and housing will also depend on successful implementation of intervention programs. For many of the variables in the system, longitudinal data are required. The relationship between other household technologies (water and sanitation, etc.) and health is also dependent on exposure variables (source and storage of water, boiling of water, etc.) through similar causal links.

In addition to the variables discussed in this paper, data must be collected on other important determinants of ARI, such as nutritional status (including breastfeeding for infants) (*89, 114*), 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 (*115, 116*), 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 (*117*). 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 temporal patterns of these variables, including seasonal changes, which are important for planning health services and case management.

An important implication 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 detail (*118–122*). These experiences, and more recent ones with improved stoves, show how ignoring 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 either face resistance during implementation or not do achieve their intended goals (*58, 95, 97*). Similar analysis has been conducted for mathematical models used to study malaria and other infectious diseases, illustrating that overlooking the complexity of the disease etiology systems in data collection and analysis can result in limited predictive power (*117*).

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 interventions and creating effective programs to reduce diseases from indoor air pollution in developing countries.

Tables

Table 1: Adjusted odds ratios for different factors affecting ARI and ALRI rates using *blogit* regression (see (*11, 12*) for details of methods and analysis). (**a**) Age: 0–4 years. (**b**) Age: 5–49 years. *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* 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; the coefficients have an interpretation similar to *Female*. Coefficient of *Age* indicates the odds ratio of being diagnosed with illness with each additional year of age.

(a)

Factor	**ARI** OR (95% CI)	*p*	**ALRI** OR (95% CI)	*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 *
500–1,000 μg/m^3	2.15 (1.30–3.56)	0.003 *	1.40 (0.74–2.67)	0.30 *
1,000–2,000 μg/m^3	4.30 (2.63–7.04)	< 0.001 *	2.33 (1.23–4.38)	0.009 *
2,000–3,500 μg/m^3	4.72 (2.82–7.88)	< 0.001 *	1.93 (0.99–3.78)	0.05 *
> 3,500 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 the house	1.00 (0.95–1.05)	0.99	0.98 (0.91–1.06)	0.70

* Jointly significant ($p < 0.01$).

(b)

Factor	**ARI** OR (95% CI)	*p*	**ALRI** OR (95% CI)	*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 *
500–1,000 μg/m^3	2.77 (1.49–5.13)	0.001 *	1.87 (0.61–5.71)	0.27 *
1,000–2,000 μg/m^3	3.79 (2.07–6.92)	< 0.001 *	2.74 (0.93–8.12)	0.07 *
2,000–4,000 μg/m^3	4.49 (2.43–8.30)	< 0.001 *	3.28 (1.09–9.85)	0.03 *
4,000–7,000 μg/m^3	5.40 (2.85–10.22)	< 0.001 *	3.21 (1.01–10.24)	0.05 *
> 7,000 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)	$p = 0.02$
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 the house	0.96 (0.93–1.00)	0.04	0.99 (0.92–1.07)	0.75

* Jointly significant ($p < 0.01$).

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