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

Majid Ezzati[1] *and Daniel M. Kammen*[2]

[1]Risk, Resource, and Environmental Management Division, Resources for the Future, Washington, DC, USA; [2]Energy and Resources Group and Goldman School of Public Policy, University of California, Berkeley, California, USA

Globally, almost 3 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 (IAP) from the combustion of solid fuels is an important cause of morbidity and mortality in developing countries. In this paper, we review the current knowledge on the relationship between IAP exposure and disease and on interventions for reducing exposure and disease. We take an environmental health perspective and consider 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 in successful design and dissemination of preventive measures and policies. In addition to specific research recommendations, we conclude that given the interaction 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 in the physical, social, and health sciences. *Key words:* **developing countries, exposure assessment, exposure–response relationship, household energy, indoor air pollution, intervention, public health.** *Environ Health Perspect* **110:1057–1068 (2002). [Online 10 September 2002]**
http://ehpnet1.niehs.nih.gov/docs/2002/110p1057-1068ezzati/abstract.html

Globally, almost 3 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 one-half of domestic energy 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 (*5–9*). Exposure to indoor air pollution (IAP) 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), chronic obstructive pulmonary disease (COPD), lung cancer (from coal smoke), asthma, cancer of the nasopharynx and larynx, tuberculosis, perinatal conditions and low birth weight, and diseases of the eye such as cataract and blindness (*9–12*).

Most current epidemiologic studies on the health impacts of exposure to IAP in developing countries have focused on the first three of the above diseases (*9,10*). Increasing evidence of the role of maternal exposure to IAP as a risk factor for low birth weight (*13*) illustrates that perinatal/neonatal conditions, in particular low birth weight, are also likely to have large and long-term health effects and to be an important source of burden of disease due to this risk factor. Given current quantitative knowledge, however, acute lower respiratory infections (ALRI) and COPD are the leading causes of mortality and burden of disease due to exposure to IAP from solid fuels.

Conservative estimates of global mortality due to IAP from solid fuels show that in 2000, between 1.5 million and 2 million deaths were attributed to this risk factor (*14,15*). This accounts for approximately 4–5% of total mortality worldwide. Approximately 1 million of these deaths were due to childhood ALRI, with the remainder due to other causes, dominated by COPD and then lung cancer, among adult women (*14,15*). Burden of disease is calculated as the number of years lost because of premature mortality plus the number of years lived with disability due to a disease, with appropriate disability weights (*16*). Therefore, childhood mortality counts for a large number of years lost because of premature mortality and a large contribution to burden of disease.

The magnitude of the health loss associated with exposure to indoor smoke and its concentration among the marginalized socioeconomic and demographic groups (women and children in poorer households and rural populations) have recently put preventive measures high on the agenda of international development and public health organizations (*10,15,17–22*). In this paper, we review the current knowledge on the relationship between IAP 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. Although our discussion of health effects mainly focuses on ARI, some of the findings and recommendations—in particular, those on the determinants of exposure—are also applicable to the other diseases caused by this risk factor. Our discussion of the health effects draws on two excellent recent reviews on the epidemiology of IAP as a risk factor (*9,10*). In addition, we used two comprehensive annotated bibliographies of IAP and ARI (*23,24*), a recent comprehensive report (*15*), and additional information from Medline (National Library of Medicine, Bethesda, MD). We also contacted other researchers in the field for articles and results since the publication of the previous reviews.

Current Research

As recently as the 1980s and 1990s, epidemiologic studies, health care manuals, and health reports focused on the biologic 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 IAP [e.g., (*25–33*)]. 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 (*34–39*). 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 (*6,15,17,40–44*).

Address correspondence to M. Ezzati, Resources for the Future, 1616 P Street NW, Washington, DC 20036 USA. Telephone: (202) 328-5004. Fax: (202) 939-3460. E-mail: ezzati@rff.org

We thank B. Singer and M. Tanner for helpful discussions on study design and program evaluation. We also thank two anonymous reviewers for valuable comments.

Received 24 September 2001; accepted 5 March 2002.

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} (particulate matter < 10 μm in diameter) to be < 150 μg/m^3 (annual average < 50 μg/m^3) (*45*). In contrast, a typical 24-hr average concentration of PM_{10} in homes using biofuels may range from 200 to 5,000 μg/m^3 or more throughout the year, depending on the type of fuel, stove, and housing (*6,8,9,40,46,47*). Concentration levels, of course, depend on where and when monitoring takes place, because significant temporal and spatial variations may occur within a house (*8,48–50*). Our field measurements (*50*), for example, recorded peak concentrations of ≥ 50,000 μg/m^3 in the immediate vicinity of the fire, with concentrations falling significantly with increasing distance from the fire. Overall, it has been estimated that approximately 80% of total global exposure to airborne particulate matter occurs indoors in developing nations (*40,47*). Levels of CO and other pollutants also often exceed international guidelines (*6,8,47,51*).

Bruce et al. (*10*) reviewed the epidemiologic evidence for the health effects of indoor smoke from solid fuels. The authors concluded that, despite some methodologic limitations, the epidemiologic studies together with experimental evidence and pathogenesis provide compelling evidence of causality for ARI and COPD, 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 epidemiologic studies (*52–55*). 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. (*14*) and Smith (*56*)], although a more recent study further confirms the relationship with low birth weight (*13*). The details of biologic mechanisms and epidemiologic studies on IAP 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.

Finally, although the physiologic mechanisms for the health impacts of indoor biomass smoke have not been studied in developing country settings, it is likely that some of the findings of air pollution research in industrialized countries also apply to these settings (*9,57*).

In the following sections, we review the methodologic and empirical characteristics of the available studies and propose directions for future research.

Emissions Monitoring and Exposure Assessment

A common characteristic of most epidemiologic 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 [e.g., (*58,59*)] or area monitors, mostly recording average daily or burning-time concentrations. Although personal monitors measure exposure directly, with current technology, exposure is aggregated over time and space. 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 have been reported by Menon (*48*), Saksena et al. (*49*), Ballard-Tremeer and Jawurek (*60*), McCracken and Smith (*61*), and Ezzati et al. (*8,50*). Menon (*48*), Ballard-Tremeer and Jawurek (*60*), and McCracken and Smith (*61*) monitored fluctuations in emission concentrations (particulate matter 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 and Jawurek (*60*) further related these fluctuations to combustion characteristics such as energy density, combustion temperature, and air flow. Our field study (*8,50*) used more recent measurement technology and conducted continuous real-time monitoring of emission concentrations under actual conditions of use in 55 households for more than 200 14-hr 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 behavior (e.g., adding or moving the fuel or cooking pot, stirring the food, etc.) during the whole day, we (*8,50*) found that the peaks in emission 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 shown in Figures 1 and 2.

In addition to studying the temporal characteristics and fluctuations of emissions, Menon (*48*), Saksena et al. (*49*), and Ezzati et al. (*50*) 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,

Figure 1. Household members involved in cooking are exposed to episodes of high pollution levels when they work directly above the fire. (*A*) Lighting the stove. (*A*) is reprinted from Ezzati et al. (*50*) with permission from *Environmental Health Perspectives*. (*B*) Preparation of ugali (see text for description). (*B*) is reprinted from Ezzati and Kammen (*11*) with permission from Elsevier Science.

Saksena et al. (*49*) 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 (~75% of exposure), followed by children (25% of exposure in winter and 40% in summer). This microenvironment made little contribution to the exposure of adolescents and almost none for men, whose exposure occurred mostly in the living room. Our measurements (*50*) and those of Menon (*48*) both considered smaller microenvironments, including dispersion within a room. These studies found that even in a single room, pollution concentrations exhibit a pronounced spatial gradient rather than instantaneous mixing (*50*). This finding implies that the exposure microenvironments for indoor smoke are considerably smaller than those reported by Saksena et al. (*49*), possibly as small as 0.5 m.

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 emission concentrations, are important determinants of exposure. For example, one of the most common foods in East Africa, especially in rural areas, is ugali, 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 1B). As water evaporates and the mixture hardens, stirring becomes increasingly vigorous until the "dough" hardens. The process takes 15–40 min, 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 smoldering (and hence very smoky) phase.

To characterize this complexity of personal exposure to indoor smoke, in a previous study (*50*) we 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 exposure patterns. In brief, we divided the time budget of household members into the following activities: cooking, non-cooking household tasks, warming around the stove, playing, resting and eating, and sleeping. We also considered the set of potential microenvironments where each activity takes place (one outside microenvironment plus six microenvironments inside the house). 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, etc.); t_{ij} represents time spent in the jth microenvironment in the ith period, and w_j is the conversion (or dilution) factor for the jth microenvironment that 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 the 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), we (*50*) used the following two descriptive statistics for characterizing human exposure (i.e., to characterize c_i in Equation 1): *a*) the mean above the 75th percentile ($m_{>75}$) was used to account for the fact that some household members are closest to the stove during high-pollution episodes caused by cooking activities; and *b*) the mean below the 95th percentile ($m_{<95}$) was used to eliminate the effect of large instantaneous peaks that especially occur when lighting or extinguishing the fire, or when fuel is added.

The value of concentration, c_i, in Equation 1 was then chosen from $m_{>75}$, m, and $m_{<95}$ based on a set of decision rules [obtained from daily time–activity data and that we described in Table 5 in an earlier paper (*50*)]. For example, for cooking very close to the stove when emissions are highest, c_i was $m_{>75}$ of the burning period. On the other hand, for sleeping at night, when the stove is smoldering and not disturbed, c_i was $m_{<95}$ of the smoldering period.

Figure 3 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. In Figure 4 these values are compared 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 shown in Figure 4, 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 these ratios among the demographic groups indicates that ignoring the spatial distribution of pollution and the role of activity patterns in exposure not only could result in inaccurate estimates of exposure but also—and possibly more importantly—could bias

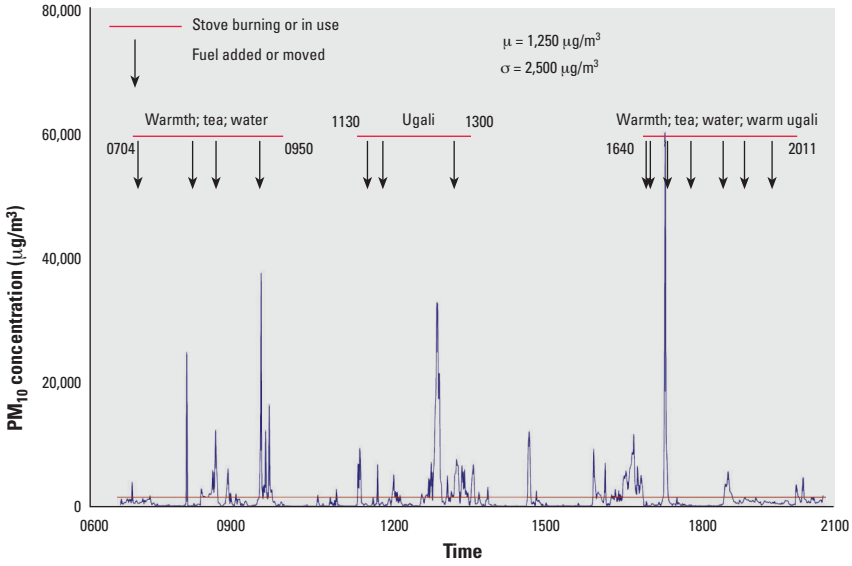

Figure 2. Day-long monitoring of pollution and cooking activities showing PM_{10} concentration (at distance and height of 0.5 m) in a household that used a three-stone stove inside. The uses of the stove are indicated above the horizontal lines (see text for description of ugali). The lower horizontal line indicates the mean pollution for the day. As shown by the data, mean concentration is a poor indicator of the patterns of exposure. μ is the sample mean, and σ is the SD.

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 epidemiologic studies on the health impacts of exposure to indoor smoke and the benefits of interventions share the following characteristics [see Table 5 in Smith et al. (*9*) and Bruce et al. (*62*)]: *a*) the use of indirect exposure proxies such as fuel type, housing characteristics, or aggregate measures of time spent near fire; *b*) case definitions of disease based on short-term monitoring, dividing the study group into those affected by disease (e.g., ARI or ALRI) and those not affected; and *c*) emphasis on randomization as the "gold standard" for hazard assessment [this has been recommended for future studies (*9,62*)].

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

The use of exposure proxies. Partially because of 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. This interest is exemplified by the 1999 World Health Organization's *WHO Air Quality Guidelines* (*44*), which states 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.

Given that some of the early studies of indoor biomass smoke focused on pollution measurement and innovative approaches to detailed exposure characterization (*35,36*), technology has not been the only cause of this interest in simple exposure indicators. Cost and time requirements may have been another consideration (*63*). Given the value of characterizing exposure, there is still a serious underrepresentation of studies that pay attention to details of exposure patterns and determinants.

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. As important, with indirect exposure proxies in epidemiologic studies, the study group could often be divided only into the broad categories of exposed and nonexposed. As a result, little is learned about the details of 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 (e.g., 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 and alternative population distributions of exposure that may not coincide with complete risk removal (*64*). For example, in an earlier study using data on time–activity budgets and emissions from different stove–fuel combinations (*65*), we 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 would therefore not be able to capture the whole range of health benefits offered by the interventions.

A further limitation of simple 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 and season to season, because of fuel characteristics (e.g., moisture content or density), air flow, type of food cooked, or if the household uses multiple stoves or fuels. Using analysis of variance, for example, we (*50*) found that, although considerably smaller than interhousehold variation, variations in individual household emissions in rural central Kenya were significant 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 provide useful information on the most important determinants of exposure and disease, not only on average but also during different days or seasons, as it has the case of ambient air pollution (*66*).

The alternative to exposure proxies. Yerushalmy and Palmer (*67*) and Murray and Lopez (*64*) discussed the multiple levels of causality in risk assessment; Yerushalmy and Palmer (*67*) referred to the factors at different causality levels as agents and vectors of disease, and Murray and Lopez (*64*) divided the levels of causality into distal, proximal, and pathophysiologic. Further, using historical analysis of research on disease causation, Evans (*68,69*) 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 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 and local ecology, at the most distal level; housing and ventilation, energy technology, and time–activity budget, at a more proximal

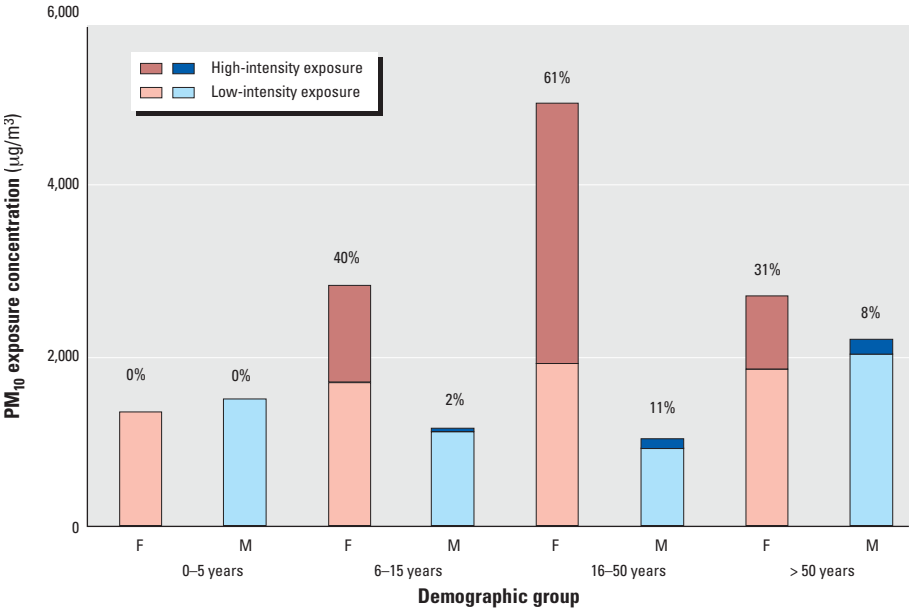

Figure 3. Components of total daily exposure to PM_{10} into high-intensity and low-intensity exposure by age category. Abbreviations: F, female; M, male. For each demographic subgroup, the total height of the column is the group average exposure concentration divided into average for high- and low-intensity components. The percentages indicate the share of total exposure from high-intensity exposure. The high-intensity component of exposure occurs in < 1 hr, emphasizing the intensity of exposure in these episodes. See Ezzati et al. (*50*) for details.

level; 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 separate cooking quarters exist. Even using some of the more proximal factors as hazard indicators, such as 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 (*63*), needs to take into account specific exposure conditions. Both physical analysis of the combustion process (*60*) 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 4) and because temporal and spatial patterns for CO (a gas) differ from those of particles, even correlation between average concentrations will make CO only a crude measure of individual exposure to particulate matter.

For reasons of cost and simplification of research and program evaluation, it is necessary to develop 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 is an area that has been successfully pursued in research on ambient air pollution (*57,70*) and more recently on IAP (*50,71–74*). 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, in which the subjects have been divided into those who are affected by disease and those who are not [see Table 5 in Smith et al. (*9*) for a summary of the studies]. Although this approach can readily capture mortality or chronic conditions (e.g., 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 (vs. 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, a severity measure can be added to incidence and duration, or alternatively, ALRI and acute upper respiratory infections (AURI) can be analyzed separately. [ALRI, which include bronchitis, pneumonia, and bronchopneumonia, are generally significantly more severe than AURI, which include infections of the upper sections of the respiratory tract, including the larynx, pharynx, tonsillar glands, eustachian tube, nasal cavities, and sinuses (*9,29,75*). ARI mortality is predominantly due to pneumonia.] 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.

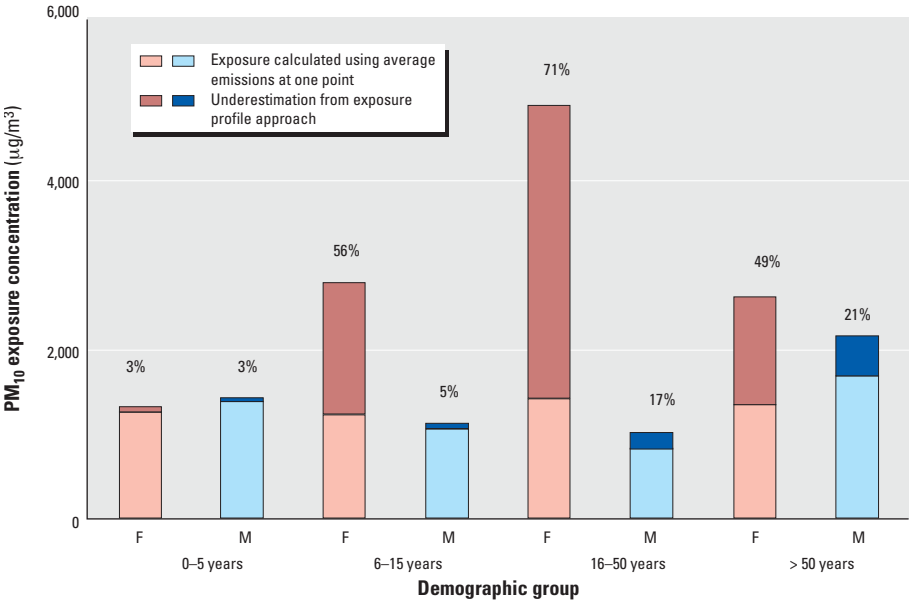

Figure 4. Comparison of exposure values that take into account temporal and spatial characteristics of pollution concentration and individual time–activity budgets versus values based on average emissions at a single point and time spent inside (without accounting for spatial dispersion and activity). Abbreviations: F, female; M, male. For each demographic group, the height of the column is the group average (from Figure 3). The underestimation of exposure using this method relative to the exposure profile approach is also shown as a percentage. See Ezzati et al. (*50*) for details.

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 epidemiologic "gold standard" (*9,10,62*).

Heckman and Smith (*76*) and Britton et al. (*77*) 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 (*78*) (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 hence may influence the outcome of or participation in an intervention). For example, socioeconomic variables are likely to be correlated with exposure to indoor smoke and also to determine nutritional status and access to medical services for case management that affect the same disease (*62,79,80*). 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. Intervention trials, however, cannot address a number of important questions:

- Because intervention studies take a long time to show effects when disease risk is

dependent on accumulated exposure (e.g., COPD or lung cancer), they cannot readily address the issue of chronic risk. This issue, however, also applies to prospective cohort studies. The only alternatives for short- and medium-term research are therefore studies with retrospective data on exposure histories.

- 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 (*81,82*). The program evaluation literature in public health sciences has traditionally avoided the determinants of this difference between efficacy and community-based effectiveness and has focused on its magnitude in order 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, 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 (*76,77,83–85*).
- 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 (*86*). This is a critical shortcoming of intervention trials, especially because (as discussed 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 use of multiple stoves or fuels; Figure 5) (*87,88*)] are likely scenarios that require a better understanding of the exposure determinants and designing new intervention packages to reduce adverse health effects.

Figure 5. Traditional open fire and ceramic woodstove used simultaneously. Because households may use combinations of different fuels, stoves, cooking locations, and other energy-related behaviors, focusing on individual interventions in randomized controlled trials may not provide realistic estimates of program effects after implementation.

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 (vs. choice) of better interventions and impacts of partial exposure reduction. As important, in assessing the benefits of interventions, randomization creates a "randomization bias" in which effects on the randomized group may be different from the benefits to participants after actual implementation (*76,89*). 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 IAP is different from interventions such as vitamin A or iodine supplementation, where fairly uniform and widespread implementation may be possible. As discussed by Heckman and Smith (*76*), selection bias and confounding arise from lack of data, and the best way to handle this is to collect 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 (*90*). 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. (*9*) on 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 IAP 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 studies with retrospective exposure and other supplemental data for chronic conditions. Finally, epidemiologic research on the exposure–response relationship should be complemented with an understanding of the pathophysiologic 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 (*91*). 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. We carried out 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 (*11,12*). Using detailed monitoring of individual-level exposure to indoor PM_{10} from biomass combustion, longitudinal data on ARI, and demographic and socioeconomic data, we quantified the exposure–response relationship for ARI (*11,12*). Using both linear and logistic risk models, this analysis (*11,12*) showed that the relationship between average exposure to indoor PM_{10} and the fraction of time that a person has an 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 1,000–2,000 μg/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 6 shows the unadjusted exposure–response relationship graphically. The relationship after adjusting for age and a number of covariates is given in Table 1.

In addition to quantifying the exposure–response relationship along a continuum of exposure levels, an important finding of this analysis was on 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} (*50*), we (*11,12*) found that males and females had similar responses (i.e., coefficients of the female variable were not statistically significant). On the other hand, when exposure was estimated (results not shown) only from average daily PM_{10} concentration and time spent indoors (i.e., without accounting for the specific activities and movement patterns of individuals), females > 5 years of age had excess risk of ARI and ALRI.

As shown in Figure 4, this latter (and 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 years, when females actually take part in household activities, a finding that further confirms this bias.

Finally, to further consider the role of exposure patterns, in a previous study (*11,12*) we used two variables that were indicators of the length and intensity, respectively, of exposure to high concentrations of PM_{10}. These were the amount of household cooking tasks that 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 2), for the highest exposure groups (notably, the individuals who cook) approximately one-half of daily exposure occurs during high-intensity episodes (Figure 3). 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 had 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 we previously described (*50*) that accounts for higher exposure during cooking periods. Investigation of the last 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 that of people 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 (*92*) has provided important understanding of the health impacts of alcohol and the benefits of interventions (*93*). Similar research on the role of exposure patterns for IAP will be equally valuable.

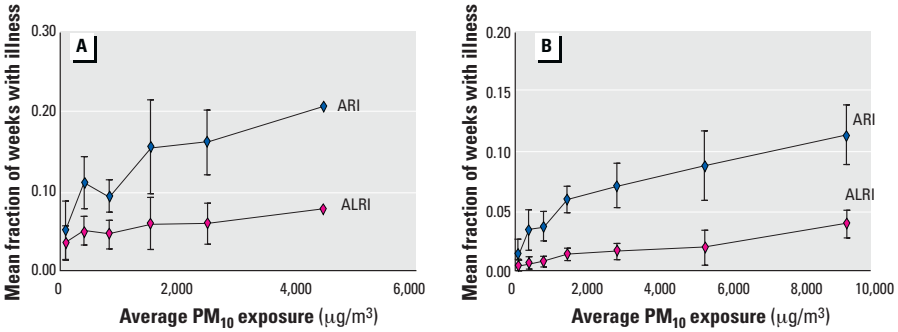

Figure 6. Unadjusted exposure–response relationship for ARI (including otitis media, which was often accompanied by ARI symptoms) and ALRI (see Table 1 for adjusted relationship). (*A*) 0–4 years of age (n = 93 individuals). (*B*) 5–49 years of age (n = 229 individuals). Each group is divided into exposure categories to reflect the day-to-day variability of individual exposure. The exposure categories are as in Table 1. 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). Error bars represent the 95% confidence interval (CI) for illness rates. The CI for the highest exposure category among infants and children (*A*) is considerably larger than that for the other categories because of the small number of children (n = 5) for the highest exposure category. See Ezzati et al. (*11,12*) for details.

Table 1. Adjusted odds ratios (OR) for different factors affecting ARI (including otitis media) and ALRI rates using b-logit regression.

	ARI		ALRI	
Exposure category, factor	OR (95% CI)	*p*-Value	OR (95% CI)	*p*-Value
0–4 years of age				
< 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[a]	0.99 (0.83–1.17)	0.88	0.84 (0.65–1.10)	0.21
Age[b]	0.88 (0.83–0.94)	< 0.001	0.76 (0.70–0.84)	< 0.001
Village type[c]	1.29 (0.99–1.67)	0.06	1.18 (0.79–1.77)	0.41
Number of people in the house[c]	1.00 (0.95–1.05)	0.99	0.98 (0.91–1.06)	0.70
5–49 years of age				
< 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[a]	1.24 (1.01–1.52)	0.04	1.21 (0.78–1.88)	0.39
Age[b]	0.99 (0.99–1.00)	0.02	1.01 (1.00–1.02)	0.02
Smoking[c]	1.48 (1.07–2.04)	0.02	1.53 (0.82–2.85)	0.18
Village type[c]	0.92 (0.76–1.12)	0.41	0.93 (0.62–1.40)	0.74
Number of people in the house[c]	0.96 (0.93–1.00)	0.04	0.99 (0.92–1.07)	0.75

CI, confidence interval. See Ezzati et al. (*11,12*) for details of methods and analysis.
[a]Variable that was assigned a value of 1 if the person was female and 0 if male; therefore, the coefficient of this variable is the odds ratio for illness among women relative to men when all other factors have been accounted for. [b]The coefficient of the age variable indicates the odds ratio of being diagnosed with illness with each additional year of age. [c]A value of 1 was assigned if a person smoked or lived in a maintenance village (vs. a cattle compound), and 0 was assigned otherwise; the coefficients of these variables are the odds ratios for illness among smokers or those living in a maintenance village relative to others, when all other factors have been accounted for. *Jointly significant (p < 0.01).

Research on Interventions and Intervention Programs

Although reducing exposure to IAP from solid fuels can be achieved through interventions in emissions source and energy technology, housing and ventilation, and behavior and time–activity budget (*15*), most current research has focused on the first method with focus on improved (high-efficiency and low-emissions) 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 (*46,94–97*). 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 (*94,98,99*). Initial research and development 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 (*96,97*). 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 (*87,100*). As a result of these studies, the initial large potential benefits from improved stoves have been questioned (*60,101*), most recently resulting in reconsidering the continuation of the apparently unsuccessful Indian improved-stove program.

Ballard-Tremeer and Jawurek (*60*), McCracken and Smith (*61*), Ezzati et al. (*8,50,65*) and Albalak et al. (*102*) are among the recent studies that have considered performance of exposure reduction interventions under actual conditions of use. McCracken and Smith (*61*) and Albalak et al. (*102*) found that the Guatemalan improved stove (plancha) provides significant reductions in average pollution concentration. Further, Albalak et al. (*102*) found that the benefits of the plancha stove persisted 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 and Jawurek (*60*) conducted a novel analysis of stove performance coupled with the thermodynamics of the combustion process. This analysis not only measured the performance of various stoves (efficiency and emissions) but also allowed identifying the factors besides stove type that influence performance. In a previous study (*8*), we used continuous real-time monitoring of emission concentrations under actual conditions of use in 55 households for more than 200 14-hr days to compare various stove–fuel combinations based on average burning-time emissions as well as other characteristics affecting personal exposure (Figure 7). 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 (*8*). This variability illustrates that how a stove is used may be as important a determinant of its emissions as is the stove type. Our field results under actual conditions of use (*8*) confirm the laboratory findings of Ballard-Tremeer and Jawurek (*60*) 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, we previously 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 compared to indoor use of traditional open fires (*65*). Using the exposure–response relationship of Table 1, we also estimated the reductions in disease associated with these interventions. In particular, we found that, on average, the range of interventions considered could reduce the fraction of times that infants and children younger than 5 years of age 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 years and highly depended on the time–activity budgets of individuals. These reductions in infant and child ALRI, due to environmental management, are similar in magnitude to those achieved by more costly medical interventions (*103–108*).

Beyond technical performance, some of the issues surrounding the success of intervention programs after community implementation (vs. technology performance) have been discussed by Agarwal (*87*), Barnes et al. (*98*), Ezzati (*85*), Kammen (*109*), Hoiser and Dowd (*110*), Manibog (*97*), Smith et al. (*99*), and von Schirnding et al. (*15*) using a limited number of available case studies in various countries. One reason for the lack of

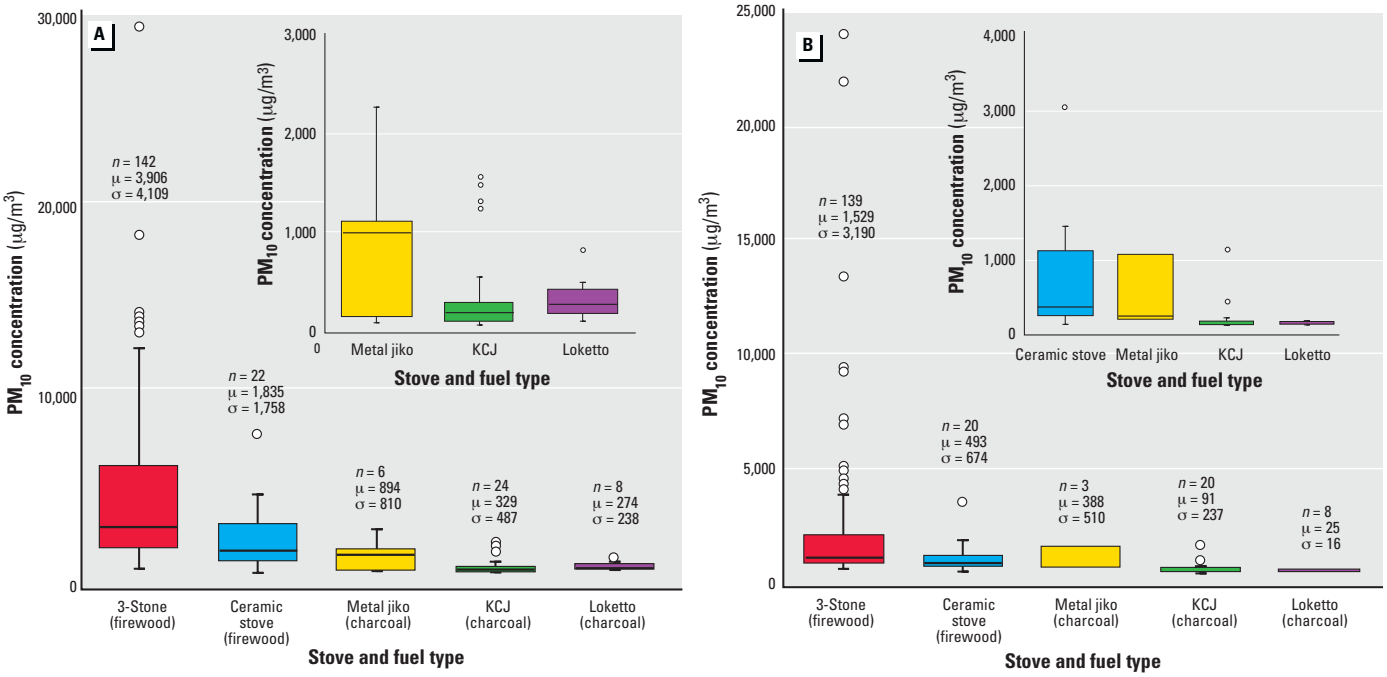

Figure 7. Day-long average of PM_{10} concentration for various stove and fuel combinations, calculated over the burning period (*A*) and smoldering period (*B*). The insets provide more details for the last three or four stoves. Abbreviations: KCJ, Kenya ceramic jiko; μ, sample mean; *n*, number of days of measurement; σ, SD. The box plots summarize the distribution of the variable: The top and bottom of the boxes show the 25th and 75th percentiles, respectively, and therefore enclose the middle half of the distribution. The middle line is the median.

systematic studies of such programs may be that, with the central role of energy technology in household livelihood, the adoption of interventions is more likely to vary from setting to setting and even household to household (*88*). Therefore, research on the design of programs for reducing the health impacts of IAP from solid fuels must still address three key questions: First, although the benefits of adopted interventions may be known, as illustrated by varying levels of success of different stove programs, it is not entirely clear what factors motivate households to adopt any intervention or suite of interventions and what the required institutions are (*85,87,97,98,110*). Second, long-term performance of interventions in exposure reduction have not been monitored, with the exception of the recent work of Albalak et al. (*102*), which ensured proper maintenance. The recent study of Lan et al. (*111*) in rural China is, to the best of our knowledge, the only work to estimate the long-term health benefits of improved stoves on a chronic disease (lung cancer). This retrospective cohort study showed that Chinese farmers using vented stoves had significantly lower incidence of lung cancer (RR = 0.59 for men; RR = 0.59 for women) over a 16-year follow-up period compared to those using open coal fires. 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 (*65*), 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 (*112*). Further, the political economy of charcoal production and markets has been found to be complex, influencing access to this fuel for different sectors of the society (*113*).

Based on the above discussion, some important issues for future research include the following:

- Conditions of exposure should be incorporated into intervention design and evaluation. For example, given the important role of peak emissions in total daily exposure (Figure 3), the design of new interventions, such as new stove technology, should give as much attention to "worst-scenario" emissions (e.g., emissions during lighting, extinguishing, or moving of fuel) as to average emission levels.
- The complex nature of household energy use should be acknowledged and scenarios that include potential energy–housing–behavior combinations, including multiple-stove and multiple-fuel scenarios, should be considered.
- Longitudinal monitoring of both technical performance and adoption, including the role of community networks in facilitating or impeding technology adoption, should be carried out.
- The social, economic, and environmental implications of each intervention strategy, beyond its impacts on exposure reduction, should be monitored or anticipated.
- The factors that facilitate or impede the development of entrepreneurial networks for designing and marketing locally manufactured energy technology or housing designs should be examined.

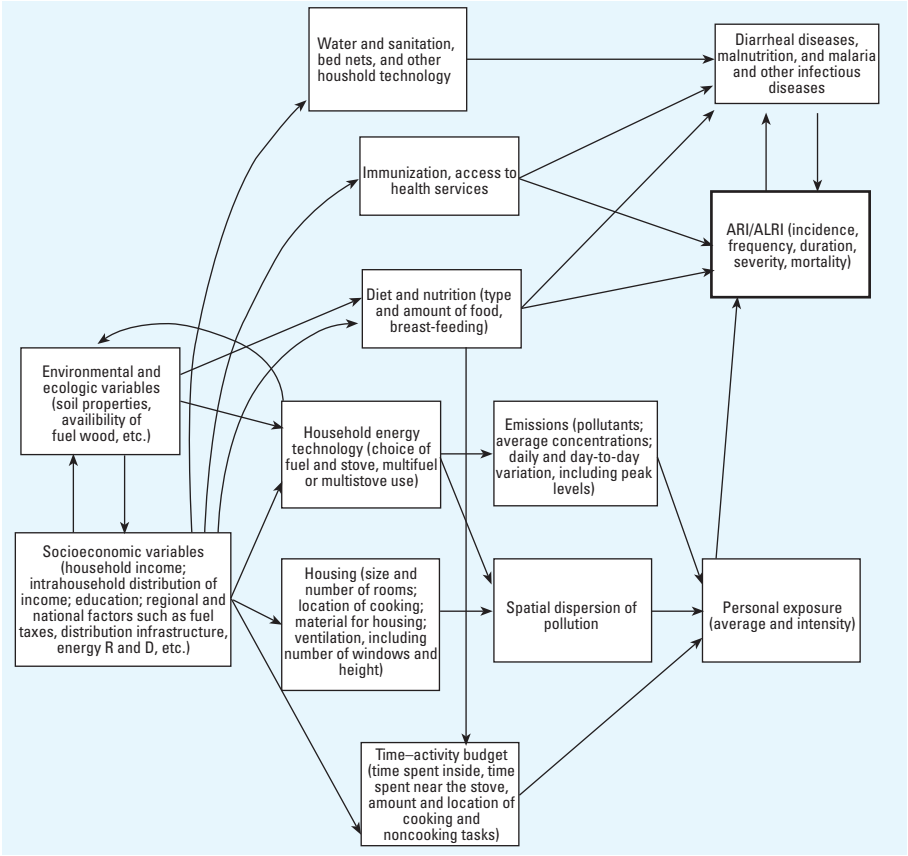

Figure 8. Important research areas, questions, and links for a predictive understanding of the health impacts of indoor smoke from household energy use. R and D, research and development. The choice of household technology and housing will also depend on successful implementation of intervention programs. 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.

Conclusions and Recommendations

We have argued that solid fuel combustion and other determinants of exposure to indoor smoke are complex phenomena, and we 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:

- 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 (e.g., the size of the house and the material it is built from, the number of windows, and the arrangement of rooms), and behavioral factors (e.g., the amount of time spent indoors or near the cooking area).
- What is the quantitative relationship between exposure to IAP and the incidence of disease (i.e., the exposure–response relationship)?
- Which determinants of human exposure will be influenced, and to what extent, through any given intervention strategy?
- What are the impacts of any intervention on human exposure and on health outcomes, and how would these impacts persist or change over time?
- What are the broader environmental effects of any intervention, its costs, and the social and economic institutions and infrastructure required for its success?

Figure 8 illustrates the research areas and questions needed for effective interventions in reducing the disease burden associated with indoor solid-fuel smoke. In addition to the variables discussed in this review, data must be collected on other important determinants of ARI, such as nutritional status (including breast-feeding for infants) (*79,114*), which may not only act as confounding variables but also, and possibly more important for risk management, interact with and modify the effects of exposure to indoor smoke. 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 of health services and case management as additional tools for disease reduction. Finally, because comorbidity is very common among different childhood (infectious) diseases (*79,115*), 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 IAP.

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 technologic 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 at the population level, 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 the indicators and their interactions locally. The exact choice of the appropriate indicators itself requires detailed pilot projects that illustrate the strength of different variables as predictive indicators of exposure and health impacts.

An important implication of the above discussions is that, given the central role of housing, household energy, and day-to-day household activities in determining exposure to indoor smoke, research and reliable data on even the most quantitative variables, such as exposure, require an integration of methodology and concepts from a variety of disciplines, ranging from quantitative environmental science and engineering, to toxicology and epidemiology, to the social 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 (*116–120*). These experiences, and more recent ones with improved stove programs, 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 may either face resistance during implementation or not achieve their intended goals (*46,85,87*).

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 review 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. Addressing the research needs at various scales, from epidemiology to risk analysis to intervention assessment, will provide the knowledge base for expanding the limited number of current interventions and creating effective programs to reduce disease burden from IAP in developing countries.

References and Notes

1. Reddy AKN, Williams RH, Johansson TB, eds. Energy after Rio: Prospects and Challenges. New York:United Nations Publications, 1996.
2. World Resources Institute, United Nations Environment Programme, United Nations Development Programme, World Bank. World Resources 1998–1999: A Guide to the Global Environment. New York:Oxford University Press, 1998.
3. Arungu-Olende S. Rural energy. Nat Resour Forum 8:117–126 (1984).
4. WHO. Health and Environment in Sustainable Development. WHO/EHG/97.8. Geneva:World Health Organization, 1997.
5. De Koning HW, Smith KR, Last JM. Biomass fuel combustion and health. Bull WHO 63:11–26 (1985).
6. Smith KR. Biofuels, Air Pollution, and Health: A Global Review. New York:Plenum Press, 1987.
7. Zhang J, Smith KR. Indoor air pollution: formaldehyde and other carbonyls emitted from various cookstoves. In: Indoor Air '96: Proceedings of the 7th International Conference on Indoor Air Quality and Climate, Nagoya, Japan, 21–26 July 1996. Tokyo, Indoor'96, 1996;85–90.
8. Ezzati M, Mbinda BM, Kammen DM. Comparison of emissions and residential exposure from traditional and improved biofuel stoves in rural Kenya. Environ Sci Technol 34:578–583 (2000).
9. Smith KR, Samet JM, Romieu I, Bruce N. Indoor air pollution in developing countries and acute lower respiratory infections in children. Thorax 55:518–532 (2000).
10. Bruce N, Perez-Padilla R, Albalak R. Indoor air pollution in developing countries: a major environmental and public health challenge. Bull WHO 78:1078–1092 (2000).
11. Ezzati M, Kammen DM. Indoor air pollution from biomass combustion as a risk factor for acute respiratory infections in Kenya: an exposure-response study. Lancet 358:619–624 (2001).
12. Ezzati M, Kammen DM. Quantifying the effects of exposure to indoor air pollution from biomass combustion on acute respiratory infections in developing countries. Environ Health Perspect 109:481–488 (2001).
13. Boy E, Bruce N, Delgado H. Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. Environ Health Perspect 110:109–114 (2002).
14. Smith KR, Mehta S, Feuz M. The global burden of disease from indoor air pollution: results from comparative risk assessment. In: Indoor Air 2002: Proceedings of the 9th International Conference on Indoor Air Quality and Climate, Monterey, CA, 30 June–5 July 2002, Vol 4. Santa Cruz, CA:Indoor Air 2002, 2002;10–19.
15. von Schirnding Y, Bruce N, Smith KR, Ballard-Tremeer G, Ezzati M, Lvovsky K. Addressing the impact of household energy and indoor air pollution on the health of the poor—implications for policy action and intervention measures. In: Working Group 5 (Improving the Health Outcomes of the Poor), Commission on Macroeconomics and Health. Available: http://www.cmhealth.org/wg5.htm [cited 1 March 2002].
16. Murray CJL, Lopez AD, eds. The Global Burden of Disease. Cambridge, MA:Harvard School of Public Health, on behalf of the World Health Organization and the World Bank, 1996.
17. World Bank. World Development Report: Investing in Health. New York:Oxford University Press, 1993.
18. McMichael AJ, Smith KR. Seeking a global perspective on air pollution and health. Epidemiology 10:1–4 (1999).
19. WHO European Office. The Right to Healthy Indoor Air. Report on a WHO Meeting. WHO/PEP/92.3A. Bilthoven, The Netherlands:World Health Organization, European Office, 2000.
20. Rahman Q, Nettesheim P, Smith KR, Prahlad KS, Selkirk J. International conference on environmental and occupational lung disease. Environ Health Perspect 109:425–431 (2001).
21. Bruce N. Household energy, health, and development. J Epidemiol Commun Health 55:221–222 (2001).
22. Torres A. A glance at child health. J Epidemiol Commun Health 55:610 (2001).
23. McCracken JP, Smith KR. An Annotated Bibliography on Prevention of Acute Respiratory Infections (ARI) and Indoor Air Pollution. Washington, DC: Environmental Health Project and U.S. Agency for International Development, 1997. Available: http://www.ehproject.org/PDF/ARIbibs/ARIBib1.pdf [cited 1 March 2002].
24. Kammen DM, Wahhaj G, Yiadom MY. Broad-Search Annotated Bibliography on Acute Respiratory Infections (ARI) and Indoor Air Pollution. Washington, DC: Environmental Health Project and U.S. Agency for International Development, 1998. Available: http://www.ehproject.org/PDF/ARIbibs/ARIBib2.pdf [cited 1 March 2002].
25. Bonte J. Patterns of mortality and morbidity. In: Health and Disease in Kenya (Vogel LC, Muller AS, Odingo RS, Onyango Z, De Geus A, eds). Nairobi:Kenya Literature Bureau, 1974.
26. Odhiambo O, Voorhoeve AM, van Ginneken JK. Age-specific infant and childhood mortality and causes of death. In: Maternal and Child Health in Rural Kenya: An Epidemiological Study (van Ginneken JK, Muller AS, eds). London:Croom Helm, 1984;213–222.
27. Voorhoeve AM, Nordbeck HJ, Lakhani SA. Factors related to infant mortality. In: Maternal and Child Health in Rural

Kenya: An Epidemiological Study (van Ginneken JK, Muller AS, eds). London:Croom Helm, 1984;257–270.
28. WHO. Antibiotics in the Treatment of Acute Respiratory Infections in Young Children. WHO/ARI/90.10. Geneva:World Health Organization, Programme for Control of Acute Respiratory Infections, 1990.
29. WHO. Acute Respiratory Infections in Children: Case Management in Small Hospitals in Developing Countries: A Manual for Doctors and Other Senior Health Workers. WHO/ARI/90.5. Geneva:World Health Organization, Programme for Control of Acute Respiratory Infections, 1990.
30. WHO. Technical Basis for the WHO Recommendations on the Management of Pneumonia in Children at First-Level Health Facilities. WHO/ARI/91.20. Geneva:World Health Organization, Programme for Control of Acute Respiratory Infections, 1991.
31. WHO. Division of Diarrhoeal and Acute Respiratory Disease Control: 1994–1995 Report. WHO/CHD/96.1. Geneva:World Health Organization, 1996.
32. LeVine RA, LeVine S, Leiderman PH, Brazelton TB, Dixon S, Richman A, Keefer CH. Child Care and Culture: Lessons from Africa. Cambridge, UK:Cambridge University Press, 1994.
33. Stanfield P, Balldin B, Versluys Z, eds. Child Health: A Manual for Medical and Health Workers in Health Centres and Rural Hospitals. Nairobi, Kenya:African Medical and Research Foundation, 1997.
34. Rice DT. Less smoke in the cook-house. Rural Health Digest 2:214 (1960).
35. Clearly GJ, Blackburn RB. Air pollution in native huts in the highlands of New Guinea. Arch Environ Health 17:785–794 (1968).
36. Sofoluwe GO. Smoke pollution in dwellings of infants with bronchopneumonia. Arch Environ Health 16:670–672 (1968).
37. Woolcock AJ, Blackburn RB. Chronic lung disease in the territory of Papua and New Guinea—an epidemiological study. Australas Ann Med 16:11–19 (1967).
38. Anderson HR. Respiratory abnormalities in Papua New Guinea children: the effects of locality and domestic wood smoke pollution. Int J Epidemiol 7:63–72 (1978).
39. Anderson HR. Chronic lung disease in the Papua New Guinea highlands. Thorax 34:647–653 (1979).
40. Smith KR. Fuel combustion, air pollution exposure, and health: situation in developing countries. Annu Rev Energy Environ 18:529–566 (1993).
41. Smith KR. The Most Important Chart in the World. United Nations University Lecture Series No. 6. Tokyo:United Nations University, 1994.
42. Smith KR. Indoor air pollution in developing countries: growing evidence of its role in the global burden of disease. In: Proceedings of the 7th International Conference on Indoor Air Quality and Climate, Nagoya, Japan, 21–26 July 1996. Tokyo, Indoor'96, 1996;33–44.
43. WHO. Epidemiological, Social, and Technical Aspects of Indoor Air Pollution from Biomass Fuel: Report of a WHO Consultation. WHO/PEP/92.3A. Geneva:World Health Organization, 1991.
44. WHO. WHO Air Quality Guidelines. Geneva:World Health Organization, 1999.
45. U.S. Environmental Protection Agency. National Ambient Air Quality Standards (NAAQS). Available: http://www.epa.gov/airs/criteria.html [cited 15 August 2002].
46. Kammen DM. Cookstoves for the developing world. Sci Am 273:63–67 (1995).
47. Smith KR. Air pollution: assessing total exposure in developing countries. Environment 30:16–34 (1988).
48. Menon P. Indoor Spatial Monitoring of Combustion Generated Pollutants (TSP, CO, and Bap) by Indian Cookstoves. UHMET 88–01. Honolulu, HI:Department of Meteorology, University of Hawaii, 1988.
49. Saksena S, Prasad R, Pal RC, Joshi V. Patterns of daily exposure to TSP and CO in the Garhwal Himalaya. Atmos Environ 26A:2125–2134 (1992).
50. Ezzati M, Saleh H, Kammen DM. The contributions of emissions and spatial microenvironments to exposure to indoor air pollution from biomass combustion in Kenya. Environ Health Perspect 108:833–839 (2000).
51. Terblanche P, Nel R, Golding T. Household Energy Sources in South Africa: An Overview of the Impact of Air Pollution on Human Health. Pretoria, South Africa:CSIR Environmental Services and Department of Mineral and Energy Affairs, 1994.
52. Smith KR, Liu Y. Indoor air pollution in developing countries. In: Epidemiology of Lung Cancer: Lung Biology in Health and Disease (Samet J, ed). New York:Marcel Dekker, 1993;151–184.
53. Du YX, Cha Q, Chen XW, Chen YZ, Huang LF, Feng ZZ, Wu XF, Wu JM. An epidemiological study of risk factors for lung cancer in Guangzhou, China. Lung Cancer 14:S9–S37 (1996).
54. Wang TJ, Zhou BS, Shi JP. Lung cancer in nonsmoking Chinese women: a case control study. Lung Cancer 14:S93–S98 (1996).
55. Liu BQ, Peto R, Chen ZM, Boreham J, Wu YP, Li JY, Campbell TC, Chen JS. Emerging tobacco hazards in China: 1. Retrospective proportional mortality study of one million deaths. Br Med J317:1411–1422 (1998).
56. Smith KR. The national burden of disease from indoor air pollution in India. Proc Natl Acad Sci USA 97:13286–13293 (2000).
57. Wilson R, Spengler JD, eds. Particles in Our Air: Concentrations and Health Effects. Cambridge, MA:Harvard University Press, 1996.
58. Reid HF, Smith KR, Sherchand B. Indoor smoke exposures from traditional and improved cookstoves: comparisons among rural Nepali women. Mount Res Dev 6:293–304 (1986).
59. Ellegard A. Cooking fuel smoke and respiratory symptoms among women in low-income areas in Maputo. Environ Health Perspect 104:980–985 (1996).
60. Ballard-Tremeer G, Jawurek HH. Comparison of five rural, wood-burning cooking devices: efficiencies and emissions. Biomass Bioenergy 11:419–430 (1996).
61. McCracken JP, Smith KR. Emissions and efficiency of improved woodburning cookstoves in highland Guatemala. Environ Int 24:739–747 (1998).
62. Bruce N, Neufeld L, Boy E, West C. Indoor biofuel air pollution and respiratory health: the role of confounding factors among women in highland Guatemala. Int J Epidemiol 27:454–458 (1998).
63. Naeher LP, Smith KR, Leaderer BP, Neufield L, Mage DT. Carbon monoxide as a tracer for assessing exposures to particulate matter in wood and gas cookstove households of highland Guatemala. Environ Sci Technol 35:575–581 (2001).
64. Murray CJL, Lopez AD. On the comparable quantification of health risks: lessons from the global burden of disease. Epidemiology 10:594–605 (1999).
65. Ezzati M, Kammen DM. Evaluating the health benefits of transitions in household energy technology in Kenya. Energy Policy 30:815–826 (2002).
66. Pope CA III, Dockery DW, Spengler JD, Raizenne ME. Respiratory health and PM_{10} pollution: a daily time-series analysis. Am Rev Respir Dis 144:668–674 (1991).
67. Yerushalmy J, Palmer CE. On the methodology of investigations of etiologic factors in chronic diseases. J Chronic Dis 108:27–40 (1959).
68. Evans AS. Causation and disease: the Henle-Koch postulates revisited. Yale J Biol Med 49:175–195 (1976).
69. Evans AS. Causation and disease: a chronological journey. Am J Epidemiol 108:249–258 (1978).
70. Levy JI, Houseman EA, Ryan L, Richardson D, Students from the 1998 Summer Program in Biostatistics, Spengler JD. Particle concentrations in urban microenvironments. Environ Health Perspect 108:1051–1057 (2000).
71. Baughman AV, Gadgil AJ, Nazaroff WW. Mixing of a point source pollutant by natural convection flow within a room. Indoor Air: Int J Indoor Air Qual Climate 4:114–122 (1994).
72. Dresher AC, Lobascio C, Gadgil AJ, Nazaroff WW. Mixing of a point source pollutant by forced convection. Indoor Air: Int J Indoor Air Qual Climate 5:204–214 (1995).
73. Lai ACK, Thatcher TL, Nazaroff WW. Inhalation transfer factors for assessing human health risks from air pollutant sources. In: Indoor Air '99: Proceedings of the 8th International Conference on Indoor Air Quality and Climate, Edinburgh, UK, 8–13 August 1999. Edinburgh,UK:Indoor Air '99, 1999;193–198.
74. Woodward A, al-Delaimy W. Measures of exposure to environmental tobacco smoke: validity, precision, and relevance. Ann NY Acad Sci 895:156–172 (1999).
75. Graham NMH. The epidemiology of acute respiratory infections in children and adults: a global perspective. Epidemiol Rev 12:149–178 (1990).
76. Heckman JJ, Smith JA. Assessing the case for social experiments. J Econ Perspect 9:85–110 (1995).
77. Britton A, McKee M, Black N, McPherson K, Sanderson C, Bain C. Threats to applicability of randomised trials: exclusion and selective participation. J Health Serv Res Policy 4:112–121 (1999).
78. Rothman KJ, Greenland S. Moden Epidemiology. Philadelphia:Lippincott-Raven, 1998.
79. Rice AL, Sacco L, Hyder A, Black RE. Malnutrition as an underlying cause of childhood deaths associated with infectious diseases in developing countries. Bull WHO 108:367–376 (2000).
80. Cerqueiro MC, Murtagh P, Halac A, Avila M, Weissenbacher M. Epidemiologic risk factors for children with acute lower respiratory tract infection in Buenos Aires, Argentina: a matched case-control study. Rev Infect Dis 12:S1021–S1028 (1990).
81. Singer BH. Self-selection and performance-based ratings: a case study in program evaluation. In: Drawing Inferences from Self-Selected Samples (Wainer H, ed). New York: Springer-Verlag, 1986;29–49.
82. Rhodes F, Wood MM, Booth RE. Efficacy and effectiveness issues in the NIDA cooperative agreement: interventions for out-of-treatment drug users. J Psychoact Drugs 30:261–268 (1998).
83. Brook RH, Lohr KN. Efficacy, effectiveness, variations, and quality: boundary-crossing research. Med Care 23:710–722 (1985).
84. Fienberg SE, Singer BH, Tanur JM. Large-scale social experimentation in the U.S.A. In: International Statistical Institute Centenary Volume: A Celebration of Statistics (Atkinson A, Fienberg SE, eds). New York:Springer-Verlag, 1985;287–326.
85. Ezzati M. The missing costs and benefits in the application of cost-benefit analysis to the evaluation of household level technology. Presented at The Cost-Benefit Analysis Dilemma: Strategies and Alternatives, October 1999, New Haven, CT. Available: http://www.rff.org/~ezzati/Household-CBA.pdf [cited 15 August 2002].
86. Pearson TA, Feinberg W. Behavioural issues in efficacy versus effectiveness of pharmacologic agents in the prevention of cardiovascular disease. Ann Behav Med 19:230–238 (1997).
87. Agarwal B. Diffusion of rural innovations: some analytical issues and the case of wood-burning stoves. World Dev 11:359–376 (1983).
88. Masera OR, Saatkamp BD, Kammen DM. From linear fuel switching to multiple cooking strategies: a critique and alternative to the energy ladder model. World Dev 28:2083–2103 (2000).
89. McKee M, Britton A, Black N, McPherson K, Bain C, Sanderson C. Interpreting the evidence: choosing between randomized and non-randomized studies. Br Med J 319:312–315 (1999).
90. Horwitz RI, Viscoli CM, Clemens JD, Sadock RT. Developing improved observational methods for evaluating therapeutic evidence. Am J Med 89:630–638 (1990).
91. Sarangapani R, Wexler AS. Modeling aerosol bolus dispersion in human airways. J Aerosol Sci 30:1345–1362 (1999).
92. Puddey IB, Rakic V, Dimmitt SB, Beilin LJ. Influence of pattern of drinking on cardiovascular disease and cardiovascular risk factors—a review. Addiction 94:649–663 (1999).
93. Britton A, McKee M. The relationship between alcohol and cardiovascular disease in Eastern Europe: explaining the paradox. J Epidemiol Commun Health 54:328–332 (2000).
94. Kammen DM. From energy efficiency to social utility: improved cookstoves and the Small Is Beautiful model of development. In: Energy as an Instrument for Social Change (Goldemberg J, Johansson TB, eds). New York:United Nations Development Programme, 1995;50–62.
95. Karekeizi S. Disseminating renewable energy technologies in sub-Saharan Africa. Annu Rev Energy Environ 19:387–421 (1994).
96. Krugmann H. Review of Issues and Research Relating to Improved Cookstoves. IDRC-MR152e. Ottawa, Ontario, Canada:International Development Research Centre, 1987.
97. Manibog FR. Improved cooking stoves in developing countries: problems and opportunities. Annu Rev Energy 9:199–227 (1984).
98. Barnes DF, Openshaw K, Smith KR, van der Plas R. What Makes People Cook with Improved Biomass Stoves? A Comparative International Review of Stove Programs. Washington, DC:The World Bank, 1994.
99. Smith KR, Shuhua G, Kun H, Daxiong Q. One hundred million improved cookstoves in China: how was it done? World Dev 21:941–961 (1993).
100. Ravindranath NH, Ramakrishna J. Energy options for cooking in India. Energy Policy 25:63–75 (1997).
101. Wallmo K, Jacobson SK. A social and environmental evaluation of fuel-efficient cook-stoves and conservation in Uganda. Environ Conserv 25:99–108 (1998).
102. Albalak R, Bruce N, McCracken JP, Smith KR, de Gallardo T. Indoor respirable particulate matter concentrations from an open fire, improved cookstove, and LPG/open fire

combination in a rural Guatemalan community. Environ Sci Technol 35:2650–2655 (2001).
103. Pandey MR, Sharma PR, Gubhaju BB, Shakya GM, Neupane RP, Gautam A, Shrestha IB. Impact of a pilot acute respiratory infection (ARI) control programme in a rural community of the hill region of Nepal. Ann Trop Paediatr 9:212–220 (1989).
104. Kirkwood BR, Gove S, Rogers S, Lob-Levyt J, Arthur P, Campbell H. Potential interventions for the prevention of childhood pneumonia in developing countries: a systematic review. Bull WHO 73:793–798 (1995).
105. Lye MS, Nair RC, Choo KE, Kaur H, Lai KPF. Acute respiratory tract infection: a community-based intervention study in Malaysia. J Trop Pediatr 42:138–143 (1996).
106. Bang AT, Bang RA, Tale O, Sontakke P, Solanki J, Wargantiwar R, Kelzarkar P. Reduction in pneumonia mortality and total childhood mortality by means of community-based intervention trial in Gadchiroli, India. Lancet 336:201–206 (1990).
107. Mtango FDE, Neuvians D. Acute respiratory infections in children under five years: control project in Bagamoyo District, Tanzania. Trans R Soc Trop Med Hyg 80:851–858 (1986).
108. van Ginneken JK, Lob-Levyt J, Gove S. Potential interventions for preventing pneumonia among young children in developing countries: promoting maternal education. Trop Med Int Health 1:283–294 (1996).
109. Kammen DM. Research, development, and commercialization of the Kenya ceramic jiko. In: Technology, Humans, and Society: Toward a Sustainable World (Dorf RC, ed). San Diego:Academic Press, 2001;310–321.
110. Hosier RH, Dowd J. Household fuel choice in Zimbabwe. Res Energy 9:347–361 (1987).
111. Lan Q, Chapman RS, Schreinemachers DM, Tian L, He X. Household stove improvement and risk of lung cancer in Xuanwei, China. J Natl Cancer Inst 94:826–835 (2002).
112. Dutt GS, Ravindranath NH. Bioenergy: direct applications in cooking. In: Renewable Energy: Sources for Fuels and Electricity (Johansson T, Kelly H, Reddy AKN, Williams RH, eds). Washington, DC:Island Press, 1993;653–697.
113. Ribot JC. From exclusion to participation: turning Senegal's forestry policy around? World Dev 23:1587–1599 (1995).
114. Cesar JA, Victora CG, Barros FC, Santos IS, Flores JA. Impact of breast feeding on admission for pneumonia during post-neonatal period in Brazil: nested case-control study. Br Med J 318:1316–1320 (1999).
115. Snow RW, Armstrong JRM, Forster D, Winstanley MT, Marsh VM, Newton CR, Waruiru C, Mwangi I, Winstanley PA, Marsh K. Childhood deaths in Africa: uses and limitations of verbal autopsies. Lancet 340:351–355 (1992).
116. Drangert J-O. Who Cares About Water? A Study of Household Water Development in Sukumaland, Tanzania. Linkoping, Sweden:Linkoping University, 1993.
117. Cassman KG, Pingali PL. Extrapolating trends from long-term experiments to farmers' fields: the case of the irrigated rice systems in Asia. In: Agricultural Sustainability: Economic, Environmental, and Statistical Considerations (Barnett V, Payne R, Steiner R, eds). London:John Wiley and Sons, 1995;63–84.
118. Frossard D. Peasant Science: Farmer Research and Philippine Rice Development [PhD Thesis]. Irvine, CA:University of California, Irvine, 1994.
119. Scott JC. Seeing Like a State: How Certain Schemes to Improve the Human Condition Have Failed. New Haven, CT:Yale University Press, 1998.
120. Williams B, Campbell C, Williams R. Broken houses: science and development in the African savannahs. Agric Hum Values 12:29–38 (1995).