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The Burden of Disease from Indoor Air Pollution in Developing Countries: Comparison of Estimates

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Abstract

Four different methods have been applied to estimate the burden of disease from solid fuel use in developing countries (LDCs). The largest number of estimates has involved applying exposure-response information from urban ambient air pollution studies to estimated indoor exposure concentrations of particulate air pollution. Another approach is to construct child survival curves using the results of large-scale household surveys, as has been done for India. A third approach involves cross-national analyses of child survival and household fuel use. The fourth method, which is explored in more depth here, involves applying the results of epidemiological studies done solely in LDC solid-fuel using exposure surrogates, such as fuel type, to surveys of household use to determine the impacts by disease and age group. With this method and conservative assumptions about relative risks, 4-5 percent of the global LDC totals for both deaths and DALYs can be attributed to acute respiratory infections, chronic obstructive pulmonary disease, tuberculosis, asthma, lung cancer, ischaemic heart disease, and blindness due to solid fuel use in developing countries. Acute respiratory infections in children under five years of age are the largest single category of deaths (64%) and DALYs (81%) from indoor air pollution, apparently being responsible globally for about 1.2 million premature deaths annually in the early 1990s.

Introduction

Air pollution has been consistently linked with substantial burdens of ill-health in developed and developing countries, with the bulk of research focused on urban outdoor (ambient) air pollution. With the rapid increase in vehicular and other pollution sources in urban areas of developing countries, and burgeoning numbers of epidemiological studies in developed countries showing effects as what used to be considered low levels, outdoor sources have remained the center of most air pollution research worldwide. Indeed, the first estimate of the global burden of disease from air pollution only addressed outdoor air pollution (Hong 1995).[1] This endeavor focused on the health effects of two ambient air pollutants, total suspended particulates and sulfur dioxide, to estimate that some 500,000 deaths from pneumonia, COPD, cardiovascular diseases, and all causes combined could be attributable to outdoor air pollution each year. It estimated regional urban exposures by reference to the WHO/UNEP GEMS urban air pollution database and applied available exposure-response information to determine impacts. Because few exposure-response studies had been done in developing countries, the results of those done in China were applied to the rest of the developing world. The counterfactual levels chosen were the WHO air quality guidelines (WHO 1979).

In reality, however, indoor sources of air pollution also pose substantial risks and, for some pollutants, probably dominate global human exposure. This is so even though pollutant emissions are dominated by outdoor sources. Exposures, however, are a function of the degree of pollution in places were people spend time and, globally, people spend the majority of their time indoors. As a result, a gram of pollution released indoors is likely to cause many hundreds of times more exposure than a gram released outdoors. Similarly, even outdoors, a cookfire near

[1] Summarized in (Murray and Lopez 1996)

the house will produce much more exposure per unit emissions than a vehicle or factory some distance away from places where the population spends most time (Smith 1993).

Unfortunately, there are also important indoor emission sources throughout the world and consequent significant exposures. As with outdoor air pollution, however, the bulk of indoor air pollution (IAP) research and control has focused on sources of concern in developed countries. Table 1 provides a simple categorization of indoor sources according to pollutant category worldwide.

Table 1. Indoor pollution sources by major pollutant types

Particles	Combustion Byproducts (CO, NOx)	Volatile Organics	Biologicals	Pesticides	Radon
Solid fuel combustion, ETS, cleaning	Fuel combustion, ETS	Furnishings, household products, ETS, solid fuel combustion	Furnishings, ventilation ducts, moist areas	Household products, dust from outside	Ground under building, ventilation characteristics

The important non-occupational indoor environments that might be included in a complete IAP CRA would be households, schools, and passenger compartments in vehicles. Unfortunately, however, there are too few exposure and exposure-response studies to derive reliable global risk estimates for the latter two microenvironments.

Because they contain the largest fraction of time spent by nearly all populations worldwide, household sources of pollution can dominate exposures.[2] Indeed, based on available measurements, it seems that bulk of global IAP exposures seem to be due to just two categories: the combustion of solid fuels for cooking/heating and environmental tobacco smoke (ETS). In fact, these sources probably produce more exposure for several important pollutants than all outdoor sources. Here we focus only on solid fuel use.

Nearly half of the world continues to cook with solid fuels, such as dung, wood, agricultural residues, and coal. In simple household stoves, these fuels emit substantial amounts of a number of important pollutants, including respirable particles, carbon monoxide, toxic organic compounds such as benzene, formaldehyde, and 1,3-butadiene, and polyaromatic compounds, such as benzo(α)pyrene (Smith 1987).

Different approaches used to estimate environmental burden of disease.

Known to us are four different methods that have been applied to estimate the burden of disease from solid fuel use in developing countries, each with advantages and disadvantages. Given that their results are fairly similar, taken together they provide some credibility, although by no means "proof," for the assertion that the problem is severe. Here we briefly summarize the methods and results from application of three of these methods, as done mostly by others, and

[2] We are focusing here on indoor sources, not indoor exposures. The latter is influenced by outdoor sources too, of course, since outdoor pollution penetrates indoors. Indeed, overall, the major impact of outdoor pollution is probably through the indoor exposures it causes, since such a large fraction of the population's time is spent indoors.

then explain our specific approach in some detail. A summary of the different approaches and their sources of data is given in Table 2.

Table 2. Summary of risk assessment methods applied to solid fuel use in developing countries

Approach	Methodology Utilized	Type of Data Utilized	Likely Bias
1. Pollutant-based	Exposure-response extrapolation	• Estimated exposure concentrations for indicator pollutants (usually particulates) • Exposure-response relationships from urban outdoor studies, usually in developed countries. • Current rates of morbidity and mortality	Overestimate
2. Child Survival	Survival analysis	• Survival curves for different risk factors based on household surveys • Done only for India to date	Overestimate
3. Cross-National	Regression	• Cross-country comparisons of national-level data on health and energy conditions	Overestimate
4. Exposure-based	Disease by disease summation	• Estimated distribution of exposure surrogates, usually fuel type. • Relative risk primarily from developing-country household studies of specific diseases in specific population groups experiencing exposure surrogate • Current rates of morbidity and mortality for each disease	Underestimate

A fifth approach, what might be called "Remainder-based," has been applied to make estimates for China (Florig 1997). It involves first estimating the fraction of the national burden of major categories of IAP-related ill-health that can be attributed to non-IAP factors. If there are no unaccounted major risk factors, the remainder can be considered a lower bound estimate for the fraction attributable to IAP.

Pollutant-based approach

This method involves the following steps:

--Estimate total population exposures from indoor sources to the indicator pollutant. Most estimates of this type have relied on particulate matter as the indicator pollutant and mean exposure concentrations in $\mu g/m^3$ as the metric.
--Determine best available exposure-response factors for this pollutant.
--Find the current rates of morbidity and mortality in the population of concern.
--Estimate the attributable number of deaths and diseases (see Appendix I).

Table 3 is a summary of the results from such efforts done globally and for the two largest GBD regions/nations, India and China. Shown for comparison are estimates for outdoor air pollution done using the same method. That the exposure-response relationships have been derived for outdoor air pollution in urban situations, where the chief source of particulates is fossil fuel burning, however, raises a number of questions about their suitability for application indoors mainly with rural populations relying on biomass fuels. In addition, some of the studies

Table 3. Estimate of Annual Pre-Mature Mortality from Air Pollution for World, India, and China. Most of these estimates applied the pollutant-based method discussed in the text.

Location	Outdoor Exposure ('000 deaths)	Indoor Exposure ('000 deaths)	Pollutant	Comments	Reference
World	570	---	PM & SO_x	-First draft done for GBD database using local air monitoring data and local exposure-response data where available.	(Hong 1995)
	200	2800	PM	-Using local air pollution monitoring data and MDC exposure-response information at low exposures and half risk at higher exposures.	(WHO 1997), p. 87, based on (Smith 1994)
	510	2200	PM	-Using local air pollution monitoring data and local exposure-response data where available.	(WHO 1997), p. 89, (Schwela 1996)
India	50-300	850-3300	PM	-Using urban air quality data and rural exposures from rural microenvironment studies and urban distribution; MDC exposure-response information; range comes from spread between daily & annual studies.	(Smith 1994)
	40	---	PM	-36 cities only based on MDC exposure-response data	(Brandon and Hommann 1995)
	86	---	PM & SO_x	-Uses Chinese exposure-response data since none in India.	(Hong 1995)
	84	590	PM	- Using local air pollution monitoring data and Chinese exposure-response data where since none in India.	(WHO 1997), p. 89; (Schwela 1996)
	200	2000	PM	-Based on estimates of time and exposures in major microenvironments by important population groups and MDC exposure-response data.	(Saksena and Dayal 1997)
	52		PM	-Extrapolation of (Brandon and Hommann 1995) using 1995 air pollution monitoring data.	(Kumar, Chowdhury et al. 1997)
China	68	---	PM & SO_x	-Uses exposure-response data developed in China.	(Hong 1995)
	70	370*	PM	-Uses Chinese exposure-response data.	(WHO 1997). p. 89
	17-290	720-1200*	PM	-Based on evaluation of Chinese exposure-response data for COPD, lung cancer, coronary heart disease, and childhood ARI. Combination of exposure-based and pollutant-based.	(Florig 1997)
	180	110*	PM	-Using exposure-response data from Chinese cities. Assumes only 13% of rural population exposed to IAP.	(World Bank 1997), p. 19

* All these estimates use (Sinton, Smith et al. 1996).
COPD= Chronic Obstructive Pulmonary Disease; ARI= Acute Respiratory Infections
LDC= less-developed country; MDC= more-developed country

rely mostly on developed-country studies. These characteristics raise several important questions:

1. Differences in pollutant mix due to different sources, i.e. although particulates can be used as indicator of hazard in both cases, biomass fuels as commonly used in LDC households produce relatively more organic compounds (e.g., benzene, formaldehyde, 1,3-butadiene, polyaromatic hydrocarbons) and fossil fuels more sulfur oxides. Thus risk (exposure-response) estimates derived in the latter situation may not apply to the former.
2. In a similar fashion, the chemical and other characteristics of the particles produced by biomass combustion are not the same as those produced by fossil fuel use, although of course woodsmoke is found seasonally in the outdoor air of many developed-country cities.
3. Differences in exposure patterns, i.e., indoor concentrations tend to vary much more during the day (because of household cooking and heating schedules than do outdoor urban levels.
4. Different exposure levels, i.e., the average exposure levels of concern in households using unvented biomass fuels are 10-50 times greater than the levels studied in most recent urban outdoor studies (Smith 1993). As is common with toxicants, there may be a diminishing of the effect per unit increase in exposure (shallowing of the exposure-response curve's slope) at these high levels.
5. Different populations, i.e., the patterns of disease, competing risk factors, and age distributions differ dramatically between urban developed-country populations, the world's richest, healthiest, and oldest populations, and people exposed to indoor air pollution in developing countries who tend to be the poorest, most stressed, and youngest in the world.
6. The largest number of developed-country studies are time-series studies that determine short-term changes in mortality and other endpoints in association with short-term changes in air pollution. The implication for long-term health patterns is unclear, however (McMichael, Anderson et al. 1998).
7. The few long-term cohort studies may be confounded by even slight misclassification of smokers (or other confounders), because smoking is such a powerful risk factor for the same health endpoints.
8. Research from developed countries has not focused on relevant health outcomes for developing countries. In particular, ALRI, the chief cause of ill-health globally and probably the major health impact of IAP exposures worldwide, is not a major cause of mortality in developed countries and thus has not been examined in many studies.
9. These more fundamental concerns are in addition to severe constraints imposed by incomplete information on the distribution of air pollution levels experienced indoors worldwide. There have been no studies of pollution levels in households based on stratified random sampling designs, for example[3].
10. Additional uncertainty is created because those relatively few particulate measurements done to date have been mostly with respect to total particulates, although most of the consistent exposure-response results have been with regard to smaller size fractions (PM_{10} or $PM_{2.5}$)[4]

The likely result of these problems is overestimation of impacts. As shown in Table 3, for example, applying this method directly to India results in 2 million annual excess deaths (Saksena and Dayal 1997), which is well above the available mortality in the appropriate disease

[3] Also a problem with outdoor pollution levels in LDC cities.
[4] Particles less than 10 μm or 2.5μm in mean aerodynamic diameter, respectively.

categories. To compensate for this tendency to overestimate, some of the estimates in the table arbitrarily reduced the exposure-response slope at higher concentrations, which is not a reliable or replicable approach.

Child Survival Approach

Here we summarize briefly the results of an analysis of National Family Health Survey (NFHS) data done under the auspices of the South Asia Office of the World Bank. The National Family Health Survey is part of a series of Demographic and Health Surveys (DHS) funded primarily by USAID and undertaken in about three dozen countries, that focused on fertility, family planning, mortality, family planing, and child health. As one of the prime consultants on the NFHS, the East-West Center initiated the inclusion of additional questions on fuel use and respiratory health endpoints. A more detailed description of the NFHS methodology and objectives is given in Appendix II.

Although as yet unpublished, this analysis has undergone substantial review both inside and outside the Bank (Hughes and Dunleavy 2000). It determines the survival curves for Indian children 0-5 years under different household conditions, with careful attention to control for potential confounders, such as house type, mother's education, parity, caste, household size, etc. Comparisons among the curves thus indicate the impact on child mortality of differences in those conditions. A total of nearly 60,000 children is included in the analysis, about 3200 of whom died before age 5. (Since the cause of death for newborns is difficult to determine and may be due to quite different risk factors, deaths before 7 days are excluded.)

As shown in Figure 1, compared to households with clean fuels, children in households using dirty fuels had a substantially lower (higher?) mortality rate. Indeed, the negative effect of dirty fuels in the model exceeded that of lack of private water supplies and/or private toilet facilities. The relative risks (RR) for using unclean fuels (here, clean fuels were defined as electricity, kerosene, LPG, biogas, and charcoal) were 2.0 (95% confidence level: 1.4-2.8) and 1.22 (1.004-1.5) for rural and urban children respectively. It is interesting to note the much smaller effect observed in urban areas. This could be partially because NFHS collected information only on the primary fuel used in households, although a significant proportion of urban households are known to use a mixture of fuels. People living in rural areas would not likely have access to as wide a range of fuel types.

Extrapolating to India as a whole using under-five child mortality calculated from the National Census, the model indicates potential mortality reductions from a switch to clean fuels as shown in Table 4. Calculated by us in the table are the rough associated loss of DALYs, which are equivalent to about 7 percent of the national total or 15 percent of the total lost by children under 5 years.[5] Note the extreme domination of total DALYs by YLLs (years of life lost) compared to YLDs (years loss to disability), a ratio of 32:1. This is because most childhood diseases produce relatively few days of illness compared to the years of lost life and also because, in the GBD child illness days are heavily discounted by age weighting.

[5] The DALY (disability-adjusted life year) is one type of Quality Adjusted Life Year (QALY), which is an index combining mortality and morbidity using lost healthy years as the measure. Its derivation and potential problems are discussed in (Murray and Lopez 1996)

Table 4. Estimated annual child mortality due to not using clean fuels in India (Hughes & Dunleavy, 2000).

Ages	Urban	Rural	All India	YLL*	DALY**
7 days <Age <1 year	10,000	385,000	395,000	13.2 million	13.6 million
1 year >Age<5 years	4,000	172,000	176,000	6.2 million	6.4 million
Total (7 days <Age<5 years)	14,000	557,000	571,000	19.4 million	20 million

Clean fuels defined as electricity, kerosene, LPG, biogas, and charcoal.
* Using Years of Life Lost Table 1.1 in Murray and Lopez (1996)
** Using the ratio of DALY/YLL for ALRI in Indian children under five (1.03) from Tables 7c and 9c in Murray and Lopez (1996).

The NFHS questionnaire was not specific enough to allow this model to determine the mix of causes of death, which is in any case notoriously difficult to determine by survey. An examination of this mix would perhaps serve as a test as to biological plausibility of attributing these deaths to IAP. On the other hand, even if a significant portion was not due to direct IAP impacts, such as ALRI, it could still be causal through the important indirect route of pre-natal exposure to the mother leading to adverse pregnancy outcomes such as low birth weight (LBW). LBW is a risk factor for a range of childhood mortality that would not be associated directly with air pollution, including diarrhea, which is the chief cause of death in this age group after ALRI. As with all observational studies, there can always be questions about whether all potential confounders have been sufficiently accounted, but this study has made prodigious attempts to do so. In addition, of course, this approach does not address the potential impacts on other population groups, particularly women.

In order to facilitate comparison of this method with the other methodologies described here, we took the relative risk estimates from Hughes & Dunleavy (Hughes and Dunleavy 2000) and extrapolated them to India, Sub-Saharan Africa, and the other developing-country regions using mortality and population data in the GBD and solid fuel use from our global household fuel database. Results of this extrapolation are shown in Table 5.

Table 5: Apparent Under Five Mortality Attributable to Use of Unclean Fuels Using Hughes & Dunleavy Relative Risks in all LDC Regions (Hughes and Dunleavy 2000)

Region	Rural	Urban	Total
India	1,136,823	67,561	1,204,384
China	308,412	10,233	318,644
Other Asia & Pacific Islands	457,153	38,072	495,225
Sub-Saharan Africa	1,313,108	84,641	1,397,749
Latin America	65,411	5,696	71,107
Mid-East and North Africa	319,466	106,867	426,333
All LDCs	**3,600,000**	**310,000**	**3,900,000**

- Excess significant figures retained to reduce rounding errors
- All cause mortality for children less than five years of age from (Murray and Lopez 1996)
- Due to differences in access to health facilities and services, child immunization, and maternal education, rural areas are likely to bear a larger share of under-five mortality. In the absence of information on how to accurately allocate mortality into rural and urban regions, however, we chose the approach of assuming equal mortality rates. This probably overestimates the urban deaths but underestimates the totals.

Note the much larger morality for India in Table 5 than in Table 4. This is because overall under five mortality estimated in India by the GBD is much larger (+50%) than estimated by Hughes & Dunleavy using National Census data. Given that the estimates in the GBD are derived from a number of sources, including the census, and are exposed to validation discipline by being part of a coherent database containing all ages, it would seem likely to be more accurate. In addition, accurate child mortality statistics are notoriously difficult to determine by simple survey, such as the census.

The global LDC totals in Table 5 are staggering, more than 30 percent of all under five deaths or about 90 percent of all ALRI. Even considering that some of the mortality may be expressed in indirect pathways that affect diarrhea and other major non-respiratory childhood diseases, it is difficult to accept such large attributable mortality to use of solid fuels alone. In addition to residual confounding, of course, some of the apparent overestimate may be due to the different cultural/household/cooking/climate conditions in other regions that produce lower relative risks for solid fuel use than found in India.

Cross-national Comparisons

Another approach is to develop a regression model of demographic and health statistics cross-nationally corrected for confounders as has been done for 122 nations in recent work by Bloom

Table 6: Demographic indicators in relationship to biomass use (Bloom and Zaidi, 2000)

	Percent Biomass (of total fuel use)				
	0-20	20-40	40-60	60-80	80+
Number of Countries	(70)	(12)	(14)	(10)	(16)
Female Life Expectancy	74.7	68.8	62.0	56.1	48.3
Life Expectancy	71.5	66.5	59.9	54.5	47.0
Male Life Expectancy	68.5	64.	57.8	53.0	45.8
Infant Mortality Rate	22.5	46.6	64.7	82.6	116.8
Child Mortality Rate	27.5	59.3	93.0	135.3	173.0
Total Fertility Rate	2.51	3.26	4.64	5.35	6.33
Crude Birth Rate	19.2	26.2	35.0	39.1	45.0
Crude Death Rate	8.6	7.6	10.9	12.8	18.1
Annual Population Growth Rate	1.00	1.61	2.43	2.74	2.52
Life Expectancy Gap (F-M)	6.2	4.5	4.2	3.1	2.6

Sources: Biomass data from United Nations, Energy Statistics Yearbook, 1993. Demographic data from World Bank, World Development Indicators, 1998

and Zaidi (Bloom, Rosenfield et al. 2000 (in press)). The input data are shown Table 6 and the results of the model shown in Table 7. It would seem to indicate that about half of the under five childhood mortality difference between countries could be attributed to difference in percent of total fuel use from biomass. Additional analysis is needed to convert these results into burdens of disease but the size of the effect would seem roughly similar to the child survival results above.[6]

Until the full method used for this analysis is published it is difficult to interpret these results. In general, of course, such studies suffer from the lack of specificity common to all ecological

[6] Complete details of this study will not be available until published later in 2000.

Table 7: Association of demographic indicators and biomass use (Bloom and Zaidi, 2000)

Dependent Variable	Constant	Percent Traditional Biomass Use	Log GNP Per Capita	Inverse Log GNP Per Capita	R^2
Female Life Expectancy	231.647**	-0.102**	-6.234	-889.108**	0.86
Life Expectancy	213.215**	-0.088**	-5.453	-816.024**	0.86
Male Life Expectancy	195.649**	-0.076**	-4.708	746.366**	0.84
Infant Mortality Rate	-795.305**	0.247**	37.945**	4203.384**	0.83
Under 5 Mortality Rate	-1377.804**	**0.494****	67.613**	7066.313**	0.82
Total Fertility Rate	-0.011	0.025**	-0.213	37.326	0.78
Crude Birth Rate	66.412	0.176**	-5.336	-6.581	0.77
Crude Death Rate	-227.919**	0.007	13.247**	1044.726**	0.54
Population Growth Rate	3.031	0.021**	-0.184	-3.546	0.43
Life Expectancy Gap (F-M)	35.998	-0.026**	-1.526	-142.741	0.35

** indicates statistical significance at the 5% level
Data from 1993 and surrounding years. Traditional Fuel includes fuelwood, bagasse, charcoal, animal wastes, vegetable wastes, and other wastes. Traditional fuel use is expressed as a percentage of total fuel use.
Source: Traditional fuel use data from United Nations *Energy Statistics Yearbook 1993*.
Demographic data from *World Development Indicators 1998,* World Bank CD ROM.

studies, which examine relationships on a population basis without linking exposure and effect at the household or individual level. In addition, such broad-scale analyses must inevitably rely on parameters that are widely available and thus have a significant chance for residual confounding. In addition, the exposure measure, "percent traditional fuel use," is difficult to interpret with regard to the parameter of particular interest here because it is percent of total fuel use in the economy, not just of households.

Exposure-based Approach

The following steps summarize the "bottom-up" (disease by disease) approach taken here to estimate the burden of disease from indoor pollution in solid-fuel-using households. In parallel to the pollutant-based approach, the exposure-based approach utilizes relative risk estimates for health outcomes that have been associated with exposures to indoor air pollution from solid fuel use. In contrast to the pollutant-based approach, which focuses on specific indicator pollutants that occur as a result of combustion, the exposure-based approach takes advantage of the large number of available LDC epidemiological studies that have been conducted that treat exposure to indoor air pollution from solid fuel use as a single category of exposure. A description of the methodology used in the exposure-based approach is provided below.

A. Using data from the International Energy Agency, UN statistical office, World Bank, FAO, national censuses and specific fuel-use surveys in developing countries, the sizes of the exposed and non-exposed populations, which are defined simply as those using solid fuels and those not, are determined by region.

B. Using the results of epidemiological studies in biomass-burning households in South Asia, Latin America, Sub-Saharan Africa, and elsewhere, appropriate risk factors (relative risks) for specific diseases in specific age groups are determined. Such studies are available in sufficient quantity and quality only for adult women and children under 5, who have the highest exposures to stove emissions.
C. Using the regional population and burden of disease (death and disability) database from the GBD, the current patterns of these diseases in these population groups are determined
D. Using the standard procedure for determining the population attributable fraction, the total disease burden attributable to use of household fuels is determined by region.
E. Using the known mortality-morbidity relationships for specific diseases for each age group in each region, an estimate of the total lost life years and total sick days attributable to indoor air pollution is estimated.

This method addresses all the concerns listed above with the pollutant-based method This approach, although not without weaknesses, substantially reduces all the problems noted above for the pollutant-based approach (numbered as before):

1-4. Being based solely on studies done in biomass-using households, the differences in pollutant mix, particle composition, exposure patterns, and exposure levels should be substantially reduced.
5. The studies were all done in poor, mostly rural, developing-country populations presumably much more similar to the exposed LDC populations than urban developed-country populations.
6. The studies address directly the specific health endpoints over time periods appropriate to the each and thus do not reflect the possible "harvesting" that may be seen in time-series studies.
7. Confining the assessment to women, who have very low smoking rates in most rural LDC areas, and children under 5, greatly reduces possible confounding by smoking.
8. Since the epidemiological studies compare actual exposed versus less-exposed populations (with different stoves or fuels), there is no need to define an arbitrary baseline value.
9-10. Because the epidemiological studies used use binary exposure variables, i.e., exposed or less-exposed, it is not necessary to extrapolate quantitative pollution exposures from incomplete data or to estimate the relative contribution of different particle size fractions.

The method is explained in more detail in (Smith 1998) and (Smith and Mehta 2000). It has remaining weaknesses, however, which are explored in Appendix V. Some of these would tend to bias the estimates upwards, for example residual confounding, although others would tend to lead to underestimates, such as exposure misclassification. In general, of course, since the method only addresses effects in children under 5 years and adult women, it tends to be an underestimate of the population total. Since these two groups experience the greatest exposures, it does not seem the resulting underestimate is likely to be large. Perhaps the most important possibility for underestimation stems from the method's current inability to address the effects of in utero exposure on birth outcomes, such as low birth weight, that might affect overall child (and adult) disease burdens. This inability is due to a lack of available studies on these endpoints.

Method of Assessment

Here we summarize the data used according to the method sequence above:

A. **Exposure assessment**: The population exposed to indoor air pollution from solid fuel use was derived from the percentage of households using solid fuels -- see Appendix III. The relative risk for solid fuel use and lung cancer only applies to coal. Therefore, lung cancer attributable risk was calculated only for China and India, since accurate quantitative information on domestic coal use is unavailable elsewhere.[7,8]

B. **Epidemiological evidence**: Based on a review of literature for epidemiological studies done in developing country solid-fuel households for specific disease endpoints and with sufficient sample sizes and methodological care to derive quantitative odds ratios, the range of risks in Table 8 were derived for the major disease categories known to be related to indoor air pollution. The reviews of the epidemiological literature and derivation of the range of odds ratios are found in (Smith 1998; Smith, Samet et al. 2000) and (Smith and Mehta 2000).[9] The diseases are ranked in order of the strength of available evidence.

Strong Evidence **(from studies of outdoor air pollution, active and passive smoking, and multiple studies in LDC solid-fuel-using households)**

Acute Respiratory Infections, a class that includes infections from a range of viruses and bacteria, but with similar symptoms and risk factors. At one-eighth of the total burden, ARI is the largest single disease category for India, as well as for the world at large where it causes about one-twelfth of the burden. Evidence from 10 studies in developing countries indicate a odds ratio range of 2-3, i.e., young children living in solid-fuel using households have 2-3 times more risk of serious ARI than unexposed children (McCracken and Smith 1997; Smith, Samet et al. 2000).

Chronic Obstructive Pulmonary Disease (COPD), such as chronic bronchitis, in women accounts for about 1.5 percent of deaths in India; 16 percent in China. Evaluation of 4 studies in developing countries indicate an odds-ratio range of 2-4 for women cooking over biomass fires for many years (Smith 1998).

Lung Cancer in women is a well-demonstrated outcome of cooking with open coal stoves in China, but there is little evidence of its connection to biomass fuel. Typical range of odds ratios for non-smoking women in 20+ Chinese studies is 3-5 (Smith and Liu 1994).

Moderate Evidence **(from studies of outdoor air pollution, smoking, and at least 2 studies in biomass-using households)**

Blindness. An odds ratio of 1.3 for blindness in women was found in analysis of the NFHS-I corrected for socio-economic confounding (Mishra, Retherford et al. 2000). A Delhi clinical case-control study found similar risks for cataract-caused blindness (Mohan, Sperduto et al. 1989).

Tuberculosis: Analysis of NFHS-I also found a TB risk of nearly 3.0 for women, after correcting for confounders. (Mishra, Retherford et al. 1999). A clinical study in Lucknow, India, found similar risks (Gupta, Mathur et al. 1997).

[7] It is known, however, that there is significant household coal use in Pakistan and South Africa as well as other parts of Africa and the Caribbean. More data are being sought.

[8] Since the Chinese economy has been growing rapidly since 1990, it is likely that the fraction of households using solid fuels has declined. Updated data are being sought.

[9] See also the review done by Bruce et al. for this meeting.

Limited Evidence **(from studies of outdoor air pollution and smoking but no studies yet done in biomass-using households)**
Ischaemic Heart Disease (IHD) and *asthma* are known to be related to outdoor air pollution and active and passive smoking in developed countries, but do not seem to have been yet studied in developing-country solid-fuel-using households. As a result, it is necessary to extrapolate from developed-country outdoor urban studies to determine risks, not an entirely satisfactory procedure as indicated above (Ostro 1996).

Insufficient Evidence**:** All categories of studies available, e.g., (Mavalankar, Trivedi et al. 1991) (Boy, Delgado et al. 1998) but too few in LDC households to derive quantitative RR estimates.
Adverse pregnancy outcomes such as stillbirth and low birth weight
Perinatal effects (during first 2 weeks after birth): Some known to be due to ARI

Used in this analysis are only those studies applying strict diagnostic criteria for the disease endpoint, study designs allowing for quantitative comparisons, and sufficient sample sizes and detected differential rates to derive statistically significant relative risks. Most corrected for confounders as well. A number of other studies are available that do not meet one or another of these criteria, for example examining respiratory symptoms without explicit diagnosis for ARI or COPD (Smith 1998; Smith, Samet et al. 2000).

Table 8: Relative Risk Estimates Used in Deriving Population Attributable Risk for Solid Fuel Use.

Disease	Population	Relative Risk-Low	Relative Risk-High	Strength of Evidence	Studies in LDC Households
ARI	<5	2.0	3.0	Strong	15
COPD	F≥15	2.0	4.0	Strong	5
Lung Cancer*	F≥15	3.0	5.0	Strong	20+
TB	F≥15	1.5	3.0	Moderate	2***
Cataracts	F≥15	1.3	1.6	Moderate	2***
Ischaemic Heart	F≥15	1.096	1.42	Limited**	0
Asthma	<5, F≥15	1.05	1.31	Limited**	0
Adverse Pregnancy Outcomes	<7 days	--	--	Insufficient	

*Coal smoke only. **Exposure-response results from urban outdoor studies in developed countries were used for these endpoints (Ostro 1996) ***Including NFHS-I analyses done by the East-West Center (Mishra, Retherford et al. 1999; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 2000).

C. Existing patterns of disease: The existing age-specific patterns of mortality and morbidity for the diseases in Table 8 are taken from the GBD (Murray and Lopez 1996). See Appendix IV.

D-E. Attributable burden: Using the formula for population attributable fraction (Appendix 1) The total regional burden is calculated for the each particular disease and population group. Thus, the range of odds ratios noted above has been combined with the estimated exposed percentage and regional disease burdens to determine mortality for each disease attributable to use of household solid fuel (Appendix VI.) To derive point estimates for each disease, we took the results from the low end of the relative risk for those in the moderate and limited evidence categories and the geometric mean of the low and high estimates for the strong evidence category in Table 8.

Summary estimates of burdens

Since the ratio total illnesses and total DALYs to mortality is assumed to be constant for all causes of a disease in a specific age group in a specific region (but different among age groups and regions), DALYs can be simply determined from the attributed mortality. Table 9 thus shows total deaths, disease incidence, and DALYs as well as the derived index of sick days by region.[10]

Table 9. Burden of Disease from Solid Fuel Use, early 1990s

Region	Deaths	Illness Incidence	DALYs	Sick Days
India	534,573	448,400,500	16,268,713	1,562,854,585
China	536,545	209,753,986	9,480,883	775,862,768
Other Asia & Pacific Islands	224,408	306,374,971	6,710,468	626,189,657
Sub-Saharan Africa	436,336	350,713,488	14,396,472	1,405,953,493
Latin America	32,003	58,250,599	941,710	85,136,260
Mid-East and North Africa	177,916	64,166,271	5,731,768	549,260,524
LDC Total	**1,900,000**	**1,400,000,000**	**54,000,000**	**5,000,000,000**

Excess significant figures retained to reduce rounding errors.

Since ARI represents the largest burden, Table 10 shows the IAP impact by LDC region on ARI in children under 5. The 1.2 million deaths are somewhat fewer than 10 percent of all deaths in LDC children of these ages. The DALYs lost represent nearly 4 percent of the global LDC total for all ages.

Table 10: Acute Respiratory Infections in Children <5 Attributable to Solid Fuel Use

Region	Disease	Ages	Deaths	Incidence of Illness	DALYs	Sick Days
IND	ARI	<5	400,922	447,809,193	13,942,987	1,382,417,067
CHN	ARI	<5	129,977	208,602,534	4,745,261	446,986,402
OAI	ARI	<5	157,580	306,050,104	5,631,211	546,384,260
SSA	ARI	<5	366,103	350,458,723	12,779,786	1,274,524,604
LAC	ARI	<5	20,630	58,190,722	750,585	71,641,189
MEC	ARI	<5	148,133	64,021,724	5,238,285	513,116,986
LDC TOTAL	ARI	<5	**1,200,000**	**1,400,000,000**	**43,000,000**	**4,200,000,000**

Excess significant figures retained to reduce rounding errors

Table 11 shows the total burden of disease from solid fuel use as a proportion of the total burden of disease experienced in each region. Compared to China, a larger percentage of India's DALYs compared to deaths can be attributable to solid fuel use because young children account for a larger proportion of the deaths in India, while women in China experience a larger burden of COPD and lung cancer, which occur at older ages. The table also shows the percentage in each category due to ARI, which correspondingly form a much smaller fraction of the burden due to solid fuels in China than in the rest of the world. (See Appendix VI for more details.)

[10] Sick Days are determined by dividing the YLL by the severity factor and multiplying by 365

Table 11. Percentage of Total LDC Burden Attributable to Solid Fuel Use

Region	Deaths	Percent ARI	DALYs	Percent ARI
India	5.7%	75%	5.7%	86%
China	6.0%	24%	4.5%	50%
Other Asia & Pacific Islands	4.1%	70%	3.8%	84%
Sub-Saharan Africa	5.3%	84%	4.9%	89%
Latin America	1.1%	64%	1.0%	80%
Mid-East and North Africa	3.9%	83%	3.8%	91%
LDC Total	4.9%	64%	4.5%	81%

Table 12 compares these figures for solid fuels in LDCs with other major LDC risk factors. Note that solid fuel use is the third largest factor of those major factors thought possible to manipulate. Of course, unlike Unsafe Sex (HIV) and Tobacco, it is not increasing rapidly.

Table 12. Major Risk Factors in LDCs

Risk Factor	Percent of Total LDC Deaths	Percent of Total LDC DALYs
Malnutrition	14.9%	18%
Water/Hygiene/Sanitation	6.7%	7.6%
Solid Fuel Use	**4.9%**	**4.5%**
Unsafe Sex/Unwanted Pregnancies	2.5%	3.7%
Alcohol	1.6%	2.7%
Occupation	2.3%	2.5%
Traffic Accidents	1.8%	2.2%
Tobacco	3.7%	1.4%
Hypertension	3.8%	0.9%
Illicit Drugs	0.2%	0.4%
Outdoor Air Pollution	0.7%	0.4%

In common with all the other approaches, the estimates from this approach could be subject to overestimate because of residual confounding in the individual studies making up the risk estimates. The fact that they are composites of several separate studies in different places and done by different investigators perhaps gives some confidence that such residual confounding is not too strong. In addition, this approach is likely to underestimate the LDC total burden because it addresses only two population groups, children under 5 years and women above 15 years. These two groups receive the highest exposures because of their household roles, but youths (5-15) and adult men undoubtedly experience some exposure and risk as well. In particular, in many LDCs girls begin cooking duties well before age 15. In addition, of course, this disease-by-disease approach does not capture effects due to disease risks indirectly increased through such pathways as low birth weight. In addition, due to the way the GBD database was constructed, there are reasons to think that the TB and cataract burdens are underestimated - see Appendix VII.

Research recommendations

Here we divide potential research into three areas (Smith 1999).

Exposures: In addition to a lack of studies on the risks of exposures, there is a dearth of studies on the distribution and magnitude of exposure to IAP in developing countries. Such information is needed both to estimate the total burden of disease and to identify the regions and populations where interventions need to be focused. Compared to the thousands of studies on outdoor and indoor air pollution in developed-country urban settings, only a handful of studies (perhaps 3 dozen) have been done in biomass-using households of the developing world, most of which are in rural areas. (By contrast, more than 100 such studies have been done in Chinese coal-burning households.) Although striking because of the high exposures to major pollutants that have been demonstrated, the available studies have not been conducted in a fashion that allows the results to be confidently extrapolated to large populations. Even for secondary indicators of IAP, e.g., distribution of fuel use, stove type, ventilation conditions, etc., the data are poor in most countries.

Risk Studies: Consider, by comparison to the several dozen or so small studies relating IAP and disease in developing countries, the thousands of rigorous studies that have been done linking smoking with ill-health, or linking such major risk factors as hypertension and cholesterol with heart disease. Tens of billions of research dollars have been and are still being spent understanding these connections. Given the potential scale of impact and the particularly vulnerable nature of the populations affected by IAP (poor women and children in developing countries), therefore, it would seem appropriate to undertake a much more complete and scientifically sophisticated research program designed to reduce the most important uncertainties in the risk estimates (the quantitative connection between IAP exposure and various diseases).

Interventions: In spite of the clear need to conduct more research on risks and exposures, the current, if imperfect, knowledge of the health burden imposed by IAP in developing countries argues that action is warranted now. Unfortunately, however, there also is a severe lack of good information on the interventions that might be best applied to effectively reduce the risks. Better ventilation, better stoves, better fuels, and behavioral changes would seem to encompass the range of potential interventions, but remarkably little systematic work has been done on any of these, considering again the potential scale of the problem and consequent potential benefit. Although, for example, there have been several hundred improved stove programs worldwide including the large Indian and Chinese efforts, there has been no systematic and independent evaluation since 1990 and no effort ever to actually conduct measurements and surveys to assess their effectiveness in reducing IAP exposures.

Since interventions are being addressed by another paper in this workshop, we do not discuss them further here.[11]

Exposure Assessment

There is a clear need for more information about the exposure situation and trends internationally. To be considered might be to

:

[11] Ballard-Tremeer and Mathee.

- Develop exposure atlases for particular regions or nations detailing regions of highest exposure and best potential for exposure reduction. This might involve:

  --Collecting and organizing information on fuel-use patterns, rural and urban, and identifying gaps in order to implement needed additional data gathering.

  --Collecting similar information on household conditions relevant to exposures, including systematic time-activity surveys.

  --Coordinating with agencies taking data on outdoor pollution levels.

  --Validating the estimates by conducting IAP measurements in a stratified random sample of households.

- It might be possible to add relevant questions to national census questionnaires or other national surveys such as the NFHS, as has been done so usefully in India.

- Consider promoting environmental indicators for access to clean household air equivalent to the commonly used indicators for access to clean water and sanitation. Possibilities include:

  --Access to clean fuels (stoves for using liquid and gaseous fuels; local availability of fuels below defined sulfur levels)

  --Access to ventilation (chimneys, hoods, or outside cooking)

Risk Factor Studies

Given what is known about the relative importance of the major air-pollution-related diseases in developing countries, the risks from existing studies, and the relative difficulties of measuring effects in studies of reasonable duration, the following kinds of studies would seem to have the highest priority. Here under each category, the diseases are listed with the highest priorities first:

- Case-control studies of acute respiratory infections (ARI) in young children, tuberculosis (TB), adverse pregnancy outcomes (APO), and cardiovascular disease (CVD). Such studies should be done carefully to assure that socio-economic factors do not confound the results. In other words, since poverty is associated with both biomass-fuel use and the prevalence of these diseases, the effect of fuel quality must be distinguished from the relationship solely due to poverty. There are means to do this in the design of the studies, methods of choosing controls, and the way the data are analyzed, but they add to the resources and sophistication required. They also need to be done with clinically confirmed health impacts, i.e., the disease condition should be confirmed using standard international clinical criteria by trained health personnel with a sub-sample verified by medical staff. Exposures to air pollution must be done carefully, particularly in TB and CVD studies where current disease status is a function of many years past exposure.

ARI is given first priority because of its huge impact on lost life years globally and TB because of its particular importance for developing-country women in the middle productive (and child rearing) years and because it is on the rise in many parts of the world due to the HIV epidemic.

- Intervention studies of ARI and adverse pregnancy outcomes. The "gold standard" of risk studies is the randomized double-blind intervention trial. This is considered the best way to actually demonstrate causality, i.e., to show that a statistical association is due to one factor physically causing the disease outcome and not a result of some third factor causing both. Such intervention trials, for example, are nearly universally required before a new drug is approved for use. They are randomized because the choice of which person to receive the drug and which to receive the placebo is made randomly. They are double-blind if neither the researcher administering the medication nor the patient knows whether they are receiving the drug or the placebo. In this way, there is less chance for bias to be introduced by the behavior or attitude of the participants.

  Randomized intervention trials would bring the evidence for the relationship of IAP with ARI and APO much closer to the standards of causality expected in the public health community. Such trials could be done by randomizing households for introduction of improved stoves or fuels, for example, and then following the intervention and control households to see if the ARI and APO rates diverged. It is difficult, however, to envision a double-blind study (placebo stoves?). Nevertheless, researcher and participant bias can be reduced by careful design and implementation of such studies.

  Although such intervention trials might also provide excellent evidence of causality for IAP with TB and CVD, they are not practical for these diseases. This is because of the long latency periods involved – a change in exposures today would not be manifested in changed disease rates for many years, even decades. Changes in ARI and APO rates, on the other hand, should be discernable within a few months.

  Preferably, such studies would be done in such a way that exposure-response relationships for major indicator pollutants (particulates, for example) can be established. Accepted exposure-response relationships of this kind would be quite valuable in assessing some kinds of interventions.

Such studies would go a long way in making the argument about causality to health ministries and international agencies that support them, who usually have very limited resources to deal with a number of large health problems. They have the results of rigorous studies focused on other means of dealing with these same diseases (e.g., antibiotics and vitamin-A for ARI; DOTS treatment for TB). At present, since they can be much more certain of the effectiveness and cost of such measures, IAP interventions have little attraction. On the other hand, the available interventions are clearly imperfect and will not serve to entirely control the diseases. Thus, unlike for example such diseases as measles where vaccination is essentially a magic bullet, there is need to find additional weapons.

***Causal Webs*:** Because all important disease conditions thought to have IAP as a risk factor have other important risk factors in LDCs, to realistically determine the impact of interventions on one risk factor it is necessary to know the interactions among risk factors. A way to do this is to develop causal web models of the relationships among risk factors, intervening parameters, and disease states. Figures 2-4 illustrate draft causal webs for the three most important age exposed age groups: pre-natal; children under 5; and adult women. More work is needed to identify the most important pathways in these webs and to attempt quantitative estimates of their magnitude based on existing studies. Those pathways for which existing data are incomplete but seem to be important for estimating the impact of feasible interventions would then be priority candidates for future research.

Conclusion

The four methods that have been applied to estimate health impacts from solid fuel use in developing countries have different strengths and weaknesses. The two using calculations of the impact on overall child mortality arrive at extremely high burdens, exceeding 3 million deaths per year in children under five alone. The estimates using particle pollution exposure-response data from developed countries also derive large estimates, for example over 2 million deaths in India alone, unless the exposure-response curves are arbitrarily made shallow at high exposures. The estimates based on the disease-by-disease application of epidemiology done solely in LDC household derive much smaller, although still substantial, results, 1.8 million premature deaths in women and children under 5. By its nature, it is probably an underestimate, but seems unlikely to be greatly so since it deals with the main diseases and principally exposed population groups.

An important point to remember, of course, is that most of the methods are actually determining the impact of solid fuel use, not of air pollution. It is likely that IAP is the chief way in which such fuels affect health, but there are other pathways as well that may be significant, such as the extra physical burden of collecting such fuels compared to modern (cleaner) fuels.

All the methods depend on exposure estimates that are extremely crude. Clearly, given the potential scale of the effect indicated by all these methods and the vulnerability of the population at risk, substantially more work is warranted to pin down the exposures as well as the risks in more detail. In the interim, however, more effort is needed as well to implement and validate a range of interventions.

Acknowledgments

We appreciate comments on earlier drafts by Sydney Rosen and Keith Florig.

Appendices

Appendix I: Deriving Population Attributable Risk (PAR)

Population attributable risk is defined here as the proportion of disease thought to be attributable to the exposure of interest. This has been calculated as follows:

$$PAR = \frac{Pe(RR-1)}{1 + Pe(RR-1)} \qquad \text{where } RR = \frac{\text{Incidence in exposed}}{\text{Incidence in unexposed}}$$

Where

PAR = Population attributable risk
Pe = population exposed
RR = relative risk

Then, the

Disease Burden due to Exposure = PAR*NBD

Where NDB is the total national burden due to the particular disease.

Taking, for illustration, an odds ratio of 2 for ALRI incidence from smoke in India and a population of one million rural children under 5, 75% of whom are exposed to biomass smoke, the following calculation shows how the number attributable to ALRI is determined.

PAR = (.75*(2-1))/(1+(.75*(2-1))=42.9%
If annual ALRI deaths (assumed to be directly proportional to incidence for all ALRI risk factors) = 9400/million (Murray & Lopez, 1996a) then
PAR*NBD = 42.9%*9400, so that 4029 deaths are attributable to smoke exposure

Appendix II. National Family Health Surveys in India

Summary: In 1992/3, the first National Family Health Survey (NFHS-I) was undertaken in India funded primarily by USAID. Covering nearly 90 thousand households and 515 thousand people, the survey was one of the largest detailed social-science surveys ever conducted. The NFHS is part of the USAID-funded program of Demographic and Health Surveys (DHS) in about 3 dozen countries, which all focused on issues related to reproductive health and fertility. Due to the involvement of the East-West Center, Honolulu, in the NFHS, however, a few questions on household fuel use and respiratory diseases were added to the extensive questionnaire for India. Several analyses of NFHS-I data relevant to IAP health effects have been published to date (Mishra and Retherford 1997; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 1999; Mishra, Retherford et al. 2000). More refined questions on health and fuel use were substituted in the recently finished round two of the survey (NFHS-II), which should allow even more useful analyses.

Details: The first National Family Health Survey (NFHS-1) was conducted in three phases during 1992-93. A core questionnaire was administered in a probability sampling framework covering each of 24 states and in the National Capital Territory of Delhi. Certain state-specific questions were also asked in some states. In all states, a household schedule was used to identify members of the household and to select eligible respondents for the individual interview. These selected ever-married women (age 13-49) were then interviewed using an individual questionnaire. In addition, data were collected at the community level in the form of a village questionnaire. In all, 88,562 households with 514,827 people were covered representing 99% of the Indian population (not Kashmir and Sikkim). This survey was one of the largest social-science surveys undertaken anywhere. The data have been widely disseminated and are being used to develop relevant policies and programs in India.

The second National Family Health Survey (NFHS-2) has recently been completed (Nov 1999) in all 25 states of India and the National Capital Territory of Delhi. Like NFHS-1, NFHS-2 is a massive undertaking; about 100,000 married women in their reproductive years were interviewed.

NFHS was designed to provide policy-relevant information on a wide variety of topics, including:

- Nutrition
- Reproductive and health conditions
- Maternal mortality
- Infant and child mortality
- Child health
- Knowledge of HIV/AIDS
- Vaccinations
- Fertility and family planning
- Fertility preferences
- Marriage patterns
- Household and individual characteristics, including fuel use

Data from NFHS-II will be useful in tracking progress since NFHS-II on these indicators. In addition, NFHS-II is covering new areas, such as:

- Quality of health and family planning services
- Reproductive tract infections
- Maternal anthropometry, vitamin A administration to children >3, maternal and child night blindness, estimation of maternal levels of hemoglobin
- Jaundice and asthma for all household members
- Marketing of oral contraceptives, oral rehydration solution, and immunization services
- Women's autonomy and domestic violence
- Life-style (smoking, alcohol consumption)
- Women's employment
- Educational aspirations for children

NFHS is funded by USAID with supplementary funding for the nutrition component being provided by UNICEF. International Institute for Population Sciences (IIPS) is the nodal implementing agency and the East-West Center/MACRO International provides technical assistance. The NFHS is one of a series of Demographic and Health Surveys (DHS) undertaken in 35 countries by MACRO for USAID (www.macroint.com/dhs).

Data are available from the NFHS archive at IIPS (iips.nfhs@axcess.net.in), Mumbai, for each state as well as the nation as a whole. These data are available in hierarchical, flat or rectangular file formats. Separate files are available for both the household and individual data files, as well as the village level data. Information about the survey and summaries of analyses derived from it are found in the *NFHS Bulletin,* a joint publication of IIPS and the East-West Center, Honolulu (poppubs@ewc.hawaii.edu).

Appendix III: Household Solid Fuel Use in LDCs

	Biomass			Coal			Non-Solid		
Region	**Urban**	**Rural**	**Total**	**Urban**	**Rural**	**Total**	**Urban**	**Rural**	**Total**
Sub-Saharan Africa	32%	92%	74%	18%	4%	8%	50%	4%	18%
India	38%	92%	78%	5%	1%	2%	57%	7%	20%
Other Asia and Pacific Islands	35%	73%	62%	3%	0%	1%	62%	27%	37%
China	15%	73%	58%	82%	13%	31%	3%	14%	11%
Mid-East and North Africa	30%	69%	46%	0%	0%	0%	70%	31%	54%
Latin America	5%	55%	19%	0%	0%	0%	95%	45%	81%
Developing Country Total	**24%**	**79%**	**59%**	**19%**	**5%**	**10%**	**57%**	**16%**	**31%**

Source: (Smith, Bailis et al. 2000)

Appendix IV. Population and Disease Data Relevant to IAP Risks in LDCs

Region	Health Outcome	Population	Burden of Disease Indicators (thousands), 1990 *				
			DEATHS	YLL	YLD	DALYs	Episodes
IND	Acute Respiratory Infections	Children < 5	777	26215	807	27022	867870
IND	Asthma	Females ≤ 15	9	94	336	430	512
IND	Chronic Obstructive Pulmonary Disease	Females ≤ 15	53	422	550	972	167
IND	Ischaemic Heart Disease	Females ≤ 15	555	4068	461	4529	708
IND	Lung Cancer	Females ≤ 15	6	64	2	66	7
IND	Tuberculosis	Females ≤ 15	232	3895	425	4320	549
IND	Blindness (Cataracts)	F≥15	0	0	1278	1278	1455
CHN	Acute Respiratory Infections	Children < 5	291	9792	832	10624	467033
CHN	Asthma	Females ≤ 15	15	153	743	896	1308
CHN	Blindness (Cataracts)	F≥15	3	26	506	532	626
CHN	Chronic Obstructive Pulmonary Disease	Females ≤ 15	685	3715	4212	7927	1813
CHN	Ischaemic Heart Disease	Females ≤ 15	377	2459	274	2733	498
CHN	Lung Cancer	Females ≤ 15	66	588	47	635	95
CHN	Tuberculosis	Females ≤ 15	100	1292	221	1513	300
OAI	Acute Respiratory Infections	Children < 5	329	11162	595	11757	638980
OAI	Asthma	Females ≤ 15	9	97	298	395	357
OAI	Blindness (Cataracts)	F≥15	0	0	595	595	680
OAI	Chronic Obstructive Pulmonary Disease	Females ≤ 15	27	184	218	402	84
OAI	Ischaemic Heart Disease	Females ≤ 15	227	1666	175	1841	305
OAI	Lung Cancer	Females ≤ 15	21	209	11	220	23
OAI	Tuberculosis	Females ≤ 15	155	2153	429	2582	547
SSA	Acute Respiratory Infections	Children < 5	736	25071	621	25692	704549
SSA	Asthma	Females ≤ 15	5	65	326	391	460
SSA	Blindness (Cataracts)	F≥15	0	0	803	803	363
SSA	Chronic Obstructive Pulmonary Disease	Females ≤ 15	36	308	265	573	99
SSA	Ischaemic Heart Disease	Females ≤ 15	113	1006	127	1133	159
SSA	Lung Cancer	Females ≤ 15	5	53	3	56	4
SSA	Tuberculosis	Females ≤ 15	164	3786	287	4073	415
LAC	Acute Respiratory Infections	Children < 5	99	3364	238	3602	279253
LAC	Asthma	Females ≤ 15	6	61	229	290	421
LAC	Blindness (Cataracts)	F≥15	0	0	236	236	433
LAC	Chronic Obstructive Pulmonary Disease	Females ≤ 15	24	159	229	388	72
LAC	Ischaemic Heart Disease	Females ≤ 15	168	1201	121	1322	231
LAC	Lung Cancer	Females ≤ 15	9	84	6	90	12
LAC	Tuberculosis	Females ≤ 15	30	622	66	688	130
MEC	Acute Respiratory Infections	Children < 5	384	13015	564	13579	165961
MEC	Asthma	Females ≤ 15	4	45	145	190	238
MEC	Blindness (Cataracts)	F≥15	0	0	427	427	502
MEC	Chronic Obstructive Pulmonary Disease	Females ≤ 15	26	194	271	465	82
MEC	Ischaemic Heart Disease	Females ≤ 15	291	2158	206	2364	372
MEC	Lung Cancer	Females ≤ 15	6	65	3	68	5
MEC	Tuberculosis	Females ≤ 15	35	687	77	764	153

Total LDC	Acute Respiratory Infections	Children < 5	2616	88619	3657	92276	3123646
Total LDC	Asthma	Females ≤ 15	48	515	2077	2592	3296
Total LDC	Blindness (Cataracts)	F≥15	3	26	3845	3871	4059
Total LDC	Chronic Obstructive Pulmonary Disease	Females ≤ 15	851	4982	5745	10727	2317
Total LDC	Ischaemic Heart Disease	Females ≤ 15	1731	12558	1364	13922	2273
Total LDC	Lung Cancer	Females ≤ 15	113	1063	72	1135	146
Total LDC	Tuberculosis	Females ≤ 15	716	12435	1505	13940	2094
***Deaths, YLL, YLD, and DALYs from Global Burden of Disease; Episodes from Global Health Statistics**							

Disability Weights and Treatment Proportions Used in Calculation of Sick Days*

REGION	OUTCOME	DISABILITY WEIGHT		
		TREATED	UNTREATED	% TREATED
IND	ARI	0.280	0.280	-
IND	TB	0.264	0.264	-
IND	COPD	0.388	0.428	0.200
IND	ASTHMA	0.059	0.099	0.450
IND	LUNG CANCER	0.146	0.146	0.200
IND	BLINDNESS (CATARACTS)	0.480	0.600	0.300
IND	ISCHAEMIC HEART DISEASE	0.300	0.400	0.200
CHN	ARI	0.280	0.280	-
CHN	TB	0.264	0.264	-
CHN	COPD	0.388	0.428	0.500
CHN	ASTHMA	0.059	0.099	0.650
CHN	LUNG CANCER	0.146	0.146	0.300
CHN	BLINDNESS (CATARACTS)	0.480	0.600	0.300
CHN	ISCHAEMIC HEART DISEASE	0.300	0.400	0.500
OAI	ARI	0.280	0.280	-
OAI	TB	0.264	0.264	-
OAI	COPD	0.388	0.428	0.400
OAI	ASTHMA	0.059	0.099	0.550
OAI	LUNG CANCER	0.146	0.146	0.250
OAI	BLINDNESS (CATARACTS)	0.480	0.600	0.300
OAI	ISCHAEMIC HEART DISEASE	0.300	0.400	0.350
SSA	ARI	0.280	0.280	-
SSA	TB	0.264	0.264	-
SSA	COPD	0.388	0.428	0.100
SSA	ASTHMA	0.059	0.099	0.200
SSA	LUNG CANCER	0.146	0.146	0.100
SSA	BLINDNESS (CATARACTS)	0.480	0.600	0.050
SSA	ISCHAEMIC HEART DISEASE	0.300	0.400	0.150

LAC	ARI	0.280	0.280	-
LAC	TB	0.264	0.264	-
LAC	COPD	0.388	0.428	0.500
LAC	ASTHMA	0.059	0.099	0.650
LAC	LUNG CANCER	0.146	0.146	0.450
LAC	BLINDNESS (CATARACTS)	0.480	0.600	0.500
LAC	ISCHAEMIC HEART DISEASE	0.300	0.400	0.600
MEC	ARI	0.280	0.280	-
MEC	TB	0.264	0.264	-
MEC	COPD	0.388	0.428	0.450
MEC	ASTHMA	0.059	0.099	0.550
MEC	LUNG CANCER	0.146	0.146	0.400
MEC	BLINDNESS (CATARACTS)	0.480	0.600	0.400
MEC	ISCHAEMIC HEART DISEASE	0.300	0.400	0.500

*Disability weights and percent treated based on Global Burden of Disease.

Appendix V. Weaknesses of Exposure-based Method

Although a major improvement over application of the pollutant-based method using developed-country data, the approach presented here has important remaining weaknesses:

1. All the epidemiological studies relied upon for the estimates here have been observational, i.e. they examined the health conditions of populations with existing differences in exposure patterns. Such studies are always subject to potential bias due to confounders, i.e., some third factor may be responsible for the effect. For example, since both solid fuel use and poor nutrition are a consequence of being poor, it may be that poor health in solid-fuel using households is due mainly to poor nutrition, lack of education, or other factors associated with poverty. Most of the epidemiological studies reviewed above attempted to check and/or adjust for confounders, but it is never possible to be sure that all potential confounders have been adequately accounted.

   In epidemiology, the "gold standard" for arguing causality is the prospective randomized double-blind intervention, where the researchers randomly allocate the exposure-reducing treatment within a population and follow the resulting difference in health conditions between the intervention group and the controls. Done well, it is thought that such a study design essentially eliminates the possibility that some unknown confounders have been operating.

   It might be noted, however, that all epidemiological studies of air pollution, including those in developed-country urban settings, have been observational. Apparently, no randomized studies have ever been done. Even so, society has been able to derive risks and set standards in spite of not reaching the "gold standard," which is required for drug trials, for example. Given careful accounting for confounders in sufficient numbers of studies by different investigators in different settings and backed by other evidence, such as animal tests and plausible physiological mechanisms, observational data by themselves are often adequate for establishing causality in practical terms.

   Randomized trials would at best be difficult and in practical terms are impossible for many air pollution endpoints. It is not feasible to impose a treatment and wait for 25 years to detect a difference in lung cancer rates, for example. Furthermore, it is difficult to imagine how one would randomize cities or parts of cities with regard to some intervention related to outdoor air pollution.

   In this respect, household air pollution in developing countries offers a research opportunity not available in developed countries, i.e., to conduct randomized trials in ways that could provide large exposure differences between intervention and control groups. Improved fuels, stoves, or ventilation could be randomly allocated at the household level, thus providing an opportunity to move air pollution epidemiology toward the "gold standard"[12] Not only do such study designs have scientific advantages, they have important policy merits because they reveal much more convincingly how much health improvement can be achieved by a particular intervention. Endpoints that would seem most appropriate for such trials are ARI, low birth weight, perinatal effects, and, perhaps, TB and asthma.

2. Essentially all the studies relied on here focused only on morbidity, e.g., they monitored the difference in incidence or prevalence of ARI, COPD, or TB between exposed and un-exposed populations. Much of the overall burden from these diseases, however, is due to mortality, which was not measured directly, but was estimated by using morbidity as an indicator. We assumed, for illustration, that a case of ARI attributed to air pollution in children under five in India carries the same mortality risk as the average case of ARI from all causes, i.e. that the case fatality rate for air-pollution induced ARI is no different from the average. This may well be a conservative assumption, however, for the one relevant study (Johnson and Aderele 1992) indicates that the case-fatality rate may be much higher in smoke-exposed infants. Clearly, however, more work of this kind is needed to pin down this relationship for all the major health outcomes.

[12] It would difficult, however, to meet the further requirement of the "gold standard" that the studies be double-blind, i.e., that neither householders nor researchers knew which households were receiving the intervention and which not (controls).

The availability of information on fuel use from the fuel use databases and on exposure-response relationships from the literature mandated use of a simple binary variable for exposure, i.e., exposed or not to smoke from household solid fuels. In reality of course, there is a continuum of exposures from high to low and the binary indicators used are only imperfect indicators.[13] For example, households using solid fuels have different ventilation conditions, different family behavior patterns, different solid fuel characteristics (biomass/coal type, moisture, contaminant content, etc.), different distances from neighbors using smoky fuels, and may use different mixtures of solid and other fuels over the year even if relying principally on one or the other. Consequently, there is undoubtedly a substantial amount of "nondifferential misclassification bias (NMB)," meaning that some households classified as exposed actually had low exposures and vice versa. Because it dilutes the real differences in health effects, the most likely result of NMB is an underestimate of the risks and thus an underestimate of the NBD (Mazzati).

NMB could be reduced by a number of means. Some are relatively easy. For example, more detailed fuel use questions could be asked at each household to determine whether a mixture of fuels is used, what kinds of solid fuels are used, the condition of stoves, degree of ventilation, etc. To reduce NMB to a minimum, however, it would be important to actually measure exposures in the households participating in a epidemiological study. Done with care, such efforts could also lead to better understanding of the actual shape of the exposure-response relationship over a wide range of exposures.[14] They might also be able to distinguish whether measures of mean or peak exposures best reflect risk.

To go from better risk estimates to better estimates of the burden will require better national estimates of exposure as well. For this purpose, additional household fuel questions have been added to the 1991 Indian Census questionnaire to determine fuel quality and the use of mixtures. If buttressed with random stratified measurements of pollution levels in different settings or validated household exposure models, such information could greatly improve NBD estimates. Similar surveys are needed in other parts of the world.

3. Although by broad comparison with what is known in developed countries, there is need for further study of all the health outcomes noted above, perhaps the most egregious gaps exist for TB and heart disease. TB is the chief outcome of AIDS in developing countries, and because of the alarming rise in HIV rates, TB is expected to continue to grow rapidly in India and elsewhere. It would be quite valuable to know how much this burden might be blunted by household environmental improvements.

   Heart disease is one of the main outcomes of smoking and of air pollution exposures in developed countries, but no studies have been done of the risks from indoor air pollution in developing countries. The background rate is expected to rise in India as incomes rise and thus it is becoming increasingly important to know the incremental burden from air pollution (both indoor and outdoor).

   In addition, perinatal conditions represent a significant fraction of the burden in India and other developing countries (xx% for all developing countries, xx% of the burden in developing country children under 5). Because of their relatively acute nature compared to COPD or heart disease, for example, these disease conditions would not be difficult to study and could be done so using the more powerful randomized intervention design.

4. Attributable risk calculations are usually done with the assumption that all other risks remain constant (xxx). Thus, a number of separate attributable risks calculated for a population can add up to more than the actual total burden of disease. What this means is that the risks and the diseases they produce are not completely independent. In the case of indoor air pollution, for example, some of the resulting IHD may have been induced or exacerbated by COPD. As a result, the disease-by-disease method has the potential of some double counting.

5. On the other hand, there are three kinds of reasons to think that the burden estimated here might be understated

[13] For example, see Naeher et al. and Mazzati xxx.

[14] Developing-country settings offer not only the opportunity to study higher exposure levels than now exist in developed countries, but also to explore larger ranges within the same population, because typical exposure distributions overlap with the upper end of those in developed countries.

for women and children:

- The GBD framework accounts TB secondary to HIV as HIV cases. Thus, actual TB cases are understated in the TB row of the tables. Since IAP apparently acts to suppress respiratory immunity, however, it probably produces more TB in HIV victims as well as in normal TB-positive persons, but this is not calculated in the results here, which start with the TB as listed in the GBD. See Appendix VII.
- Few deaths are directly accounted to cataracts in the GBD. There is evidence, however, that blind people in LDCs have substantially higher general mortality rates than the non-blind (). Including this indirect impact of cataracts would increase the total deaths and DALYs due to IAP. (Appendix VII).
- The likely impact of IAP on birth outcome, including birthweight, will not only have an effect on perinatal death, but also on a range of other disease rates of childhood and later. Thus, for example, some diarrhea may be accounted to IAP exposures during pregnancy.

7. Lastly, because of data limitations, the analysis in this study only provides estimates for women and young children. No attempt has been made to calculate the disease burden for youths or adult men. It is likely that the relative impact is smaller in these populations because of their exposure patterns. Given the known impact of particulate air pollution at even relatively low levels (by developing country norms), however, the impact may be still be important. It may thus be useful to focus a few future studies on these groups, particularly on female youths (5-15) who, because of their household roles as daughters and young wives, may experience significant exposures.

Appendix VI. Detailed Results of Exposure-based Method:

Burden of Disease from Solid Fuel Use, 1990

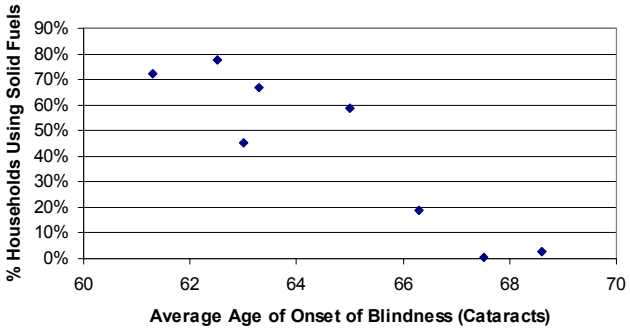
REGION	OUTCOME	Deaths	Episodes	DALYs	Sick Days
CHN	ARI	129,977	208,602,534	4,745,261	446,986,402
CHN	ASTHMA	452	39,423	27,005	122,871
CHN	BLINDNESS (CATARACTS)	449	93,644	79,582	800,664
CHN	COPD	332,528	880,107	3,848,103	268,565,134
CHN	ISCHAEMIC HEART DISEASE	20,090	26,538	145,641	16,740,317
CHN	LUNG CANCER	30,379	43,727	292,280	14,422,755
CHN	TB	22,671	68,013	343,011	28,224,626
CHN	TOTAL FROM FUELS	536,545	209,753,986	9,480,883	775,862,768
IND	ARI	400,922	447,809,193	13,942,987	1,382,417,067
IND	ASTHMA	356	20,265	17,020	110,000
IND	BLINDNESS (CATARACTS)	-	275,216	241,736	-
IND	COPD	29,327	92,408	537,847	35,797,007
IND	ISCHAEMIC HEART DISEASE	38,552	49,180	314,598	39,193,250
IND	LUNG CANCER	467	545	5,140	265,601
IND	TB	64,949	153,693	1,209,386	105,071,660
IND	TOTAL FROM FUELS	534,573	448,400,500	16,268,713	1,562,854,585
LAC	ARI	20,630	58,190,722	750,585	71,641,189
LAC	ASTHMA	59	4,164	2,869	16,077
LAC	BLINDNESS (CATARACTS)	-	23,175	12,631	-
LAC	COPD	5,745	17,234	92,870	5,667,511
LAC	ISCHAEMIC HEART DISEASE	2,986	4,106	23,497	2,649,133
LAC	LUNG CANCER	.	.	.	.
LAC	TB	2,584	11,197	59,258	5,162,350
LAC	TOTAL FROM FUELS	32,003	58,250,599	941,710	85,136,260
MEC	ARI	148,133	64,021,724	5,238,285	513,116,986
MEC	ASTHMA	94	5,599	4,470	29,752
MEC	BLINDNESS (CATARACTS)	-	60,241	51,241	-
MEC	COPD	11,040	34,818	197,443	12,327,298
MEC	ISCHAEMIC HEART DISEASE	12,168	15,554	98,845	11,527,128
MEC	LUNG CANCER	.	.	.	.
MEC	TB	6,482	28,334	141,484	12,259,360
MEC	TOTAL FROM FUELS	177,916	64,166,271	5,731,768	549,260,524
OAI	ARI	157,580	306,050,104	5,631,211	546,384,260
OAI	ASTHMA	308	12,225	13,527	93,358
OAI	BLINDNESS (CATARACTS)	-	113,670	99,461	-
OAI	COPD	13,966	43,449	207,937	14,312,417
OAI	ISCHAEMIC HEART DISEASE	13,700	18,407	111,108	13,395,277
OAI	LUNG CANCER	.	.	.	.
OAI	TB	38,854	137,115	647,225	52,004,345
OAI	TOTAL FROM FUELS	224,408	306,374,971	6,710,468	626,189,657
SSA	ARI	366,103	350,458,723	12,779,786	1,274,524,604
SSA	ASTHMA	184	16,934	14,394	79,478
SSA	BLINDNESS (CATARACTS)	-	64,563	142,822	-
SSA	COPD	19,271	52,994	306,722	25,515,279
SSA	ISCHAEMIC HEART DISEASE	7,316	10,295	73,357	9,153,046
SSA	LUNG CANCER	.	.	.	.
SSA	TB	43,462	109,980	1,079,390	96,681,086
SSA	TOTAL FROM FUELS	436,336	350,713,488	14,396,472	1,405,953,493
LDC Total	ARI	1,223,344	1,435,133,000	43,088,114	4,235,070,509
LDC Total	ASTHMA	1,454	98,611	79,284	451,536
LDC Total	BLINDNESS (CATARACTS)	449	630,509	627,474	800,664
LDC Total	COPD	411,876	1,121,010	5,190,921	362,184,646
LDC Total	ISCHAEMIC HEART DISEASE	94,812	124,080	767,047	92,658,152
LDC Total	LUNG CANCER	30,846	44,272	297,420	14,688,356
LDC Total	TB	179,000	508,331	3,479,755	299,403,426
LDC Total	TOTAL FROM FUELS	1,941,781	1,437,659,814	53,530,014	5,005,257,288

Appendix. VII. Reasons Why the TB and Blindness Burdens May be Underestimated

Blindness: Fewer than 500 deaths from blindness could be directly attributable to indoor air pollution using the distribution of death and disability in the GBD (Appendix VI). Studies have suggested, however, that the risk of death from all causes is 2-3 times higher in blind individuals (Evans in (Murray and Lopez 1996), p. 250). The 700,000 or so annual incident cases of blindness from cataracts could thus be a proximal cause of a much larger burden of death and disability than has been estimated here.

Additional evidence beyond the two Indian studies used to derive the cataract risks in Table 8 is shown in Figure VII-1. Crudely, the use of solid fuels seems to be correlated with an earlier age of onset of cataracts, which might argue a connection. More detailed analysis would have to be done, however, before any firm conclusions can be drawn.

Figure VII-1: Exploring the relationship: Solid Fuel Use and Blindness (Cataracts)



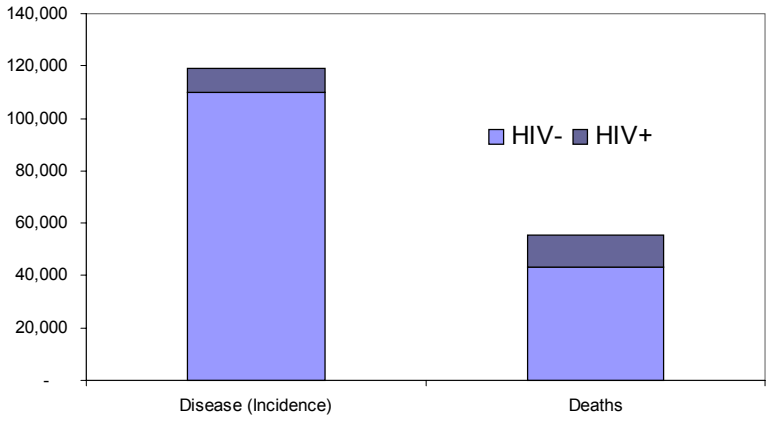
*Sources: (Smith, Bailis et al. 2000); (Murray and Lopez 1996)

In addition to cataracts, indoor air pollution may be linked as well to blindness through trachoma (Preuss and Mariotti 2000). Two separate studies in Tanzania found such a link (West, Lynch et al. 1989) (Taylor, West et al. 1989) although another study in Ethiopia found cooking in a central room to be protective, perhaps through reduction of flies (Sahlu and Larson 1992). The total global burden of trachoma, however, is only about 15% that of cataracts (Murray and Lopez 1996).

HIV and Tuberculosis: The estimates of the burden of disease from TB do not include TB in individuals who are HIV sero-positive. This is because the GBD categorizes deaths and opportunistic infections among HIV sero-positive individuals as part of the HIV burden of disease. The prevalence of HIV/TB co-infection is not insignificant, however, and is growing worldwide.

Global Health Statistics (Murray and Lopez, 1996b) provides estimates of the number of TB deaths and cases among HIV sero-prevalent individuals (see Figure VII-2). The reported incidence of co-infection in 1990 is, of course, quite low in all regions except for Sub-Saharan Africa. As reflected in the projected values for 2000, however, co-infection of TB and HIV will continue to rise with the AIDS epidemic. Co-infection with HIV is likely to be a greater risk factor for tuberculosis mortality than exposure to indoor air pollution, but it is not unreasonable to hypothesize that the association between solid fuel use and increased incidence of tuberculosis could due to some mechanism of immuno-suppression (Mitra et al., 1999). Therefore, the same level of risk associated with solid fuel use could be seen in HIV sero-prevalent cases. Including these cases in the attributable risk calculations would result in additional 12,000 incident cases and 9,000 deaths from TB attributable to solid fuel use in Sub-Saharan Africa alone.

Figure VII-2: Tuberculosis in SSA Attributable to Solid Fuel Use, 1990



Figures

Figure 1: Estimated child survival in India under different household environmental conditions (Hughes and Dunleavy, 2000)

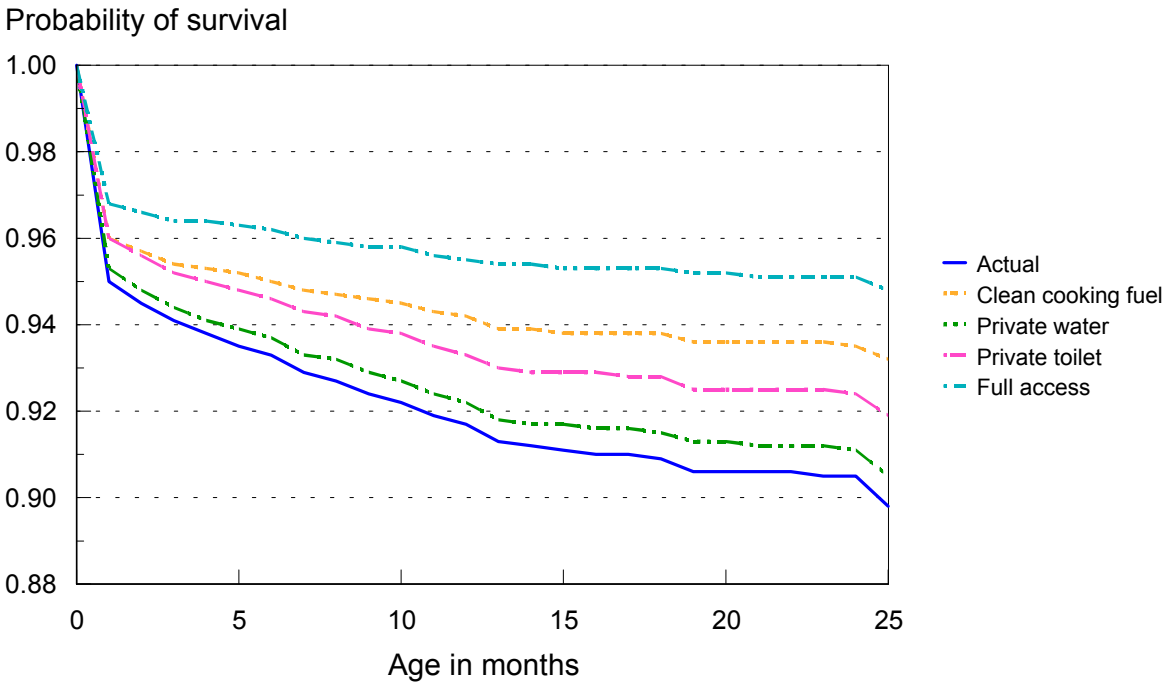
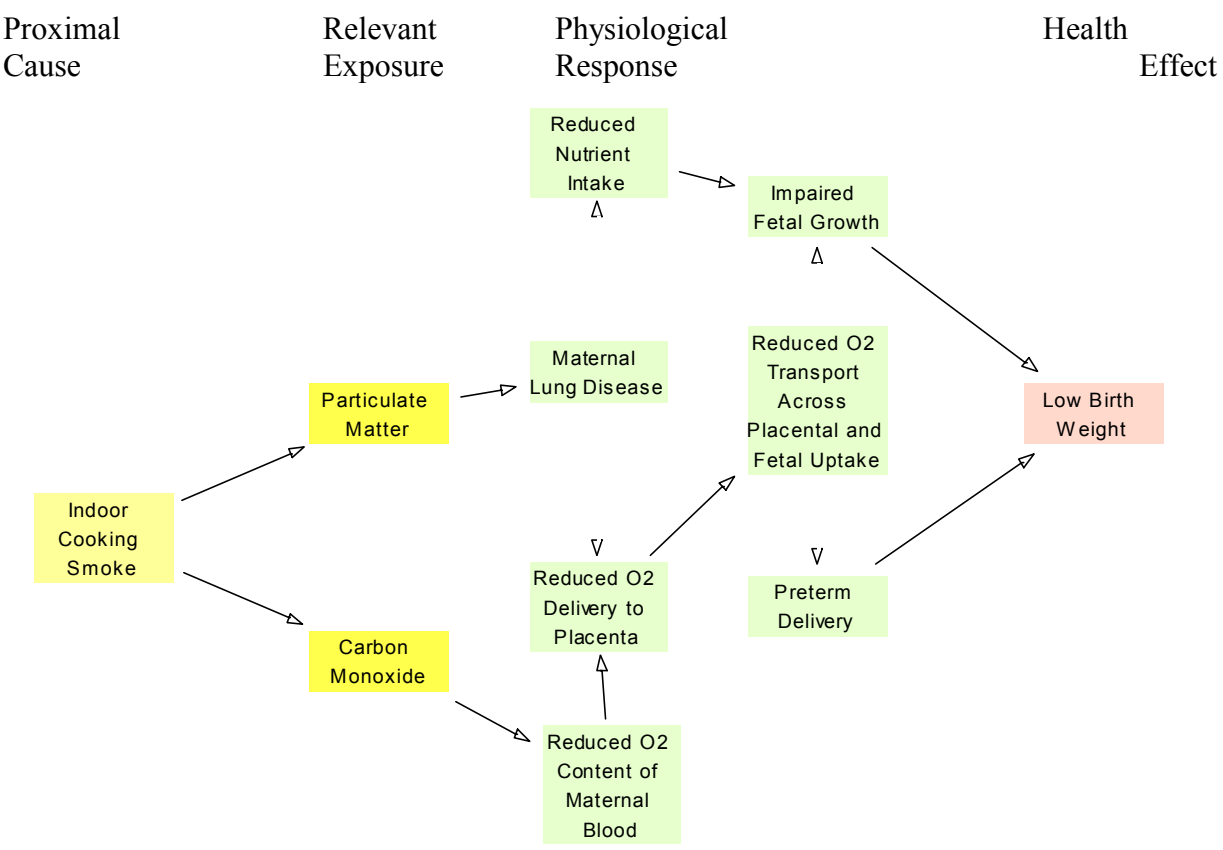


Figure 2: Indoor Air Pollution and Low Birth Weight*



*Adapted from Jere D. Haas' schematic diagram of causal pathway for indoor cooking smoke and birthweight (Smith, Samet et al. 2000).

Figure 3: Indoor Air Pollution from Solid Fuel Use and Health of Children ≤ 5 years of age)

Proximal Causes | Physiological / Pathophysiological Response | Health Effect

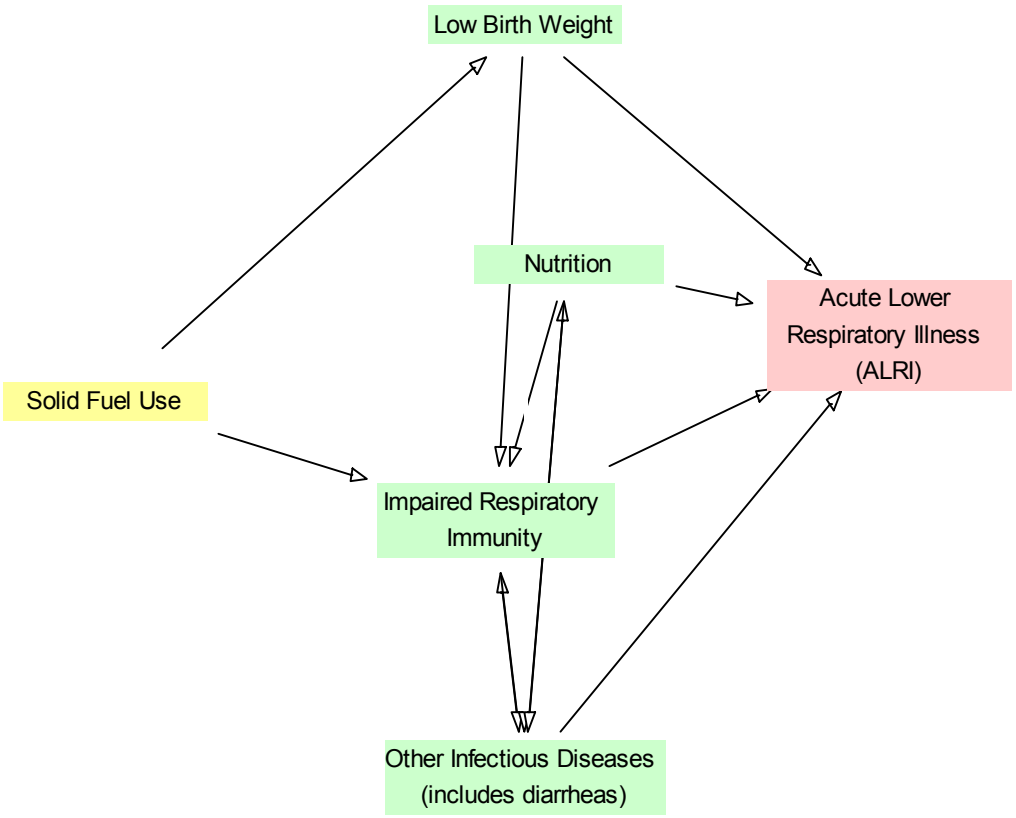
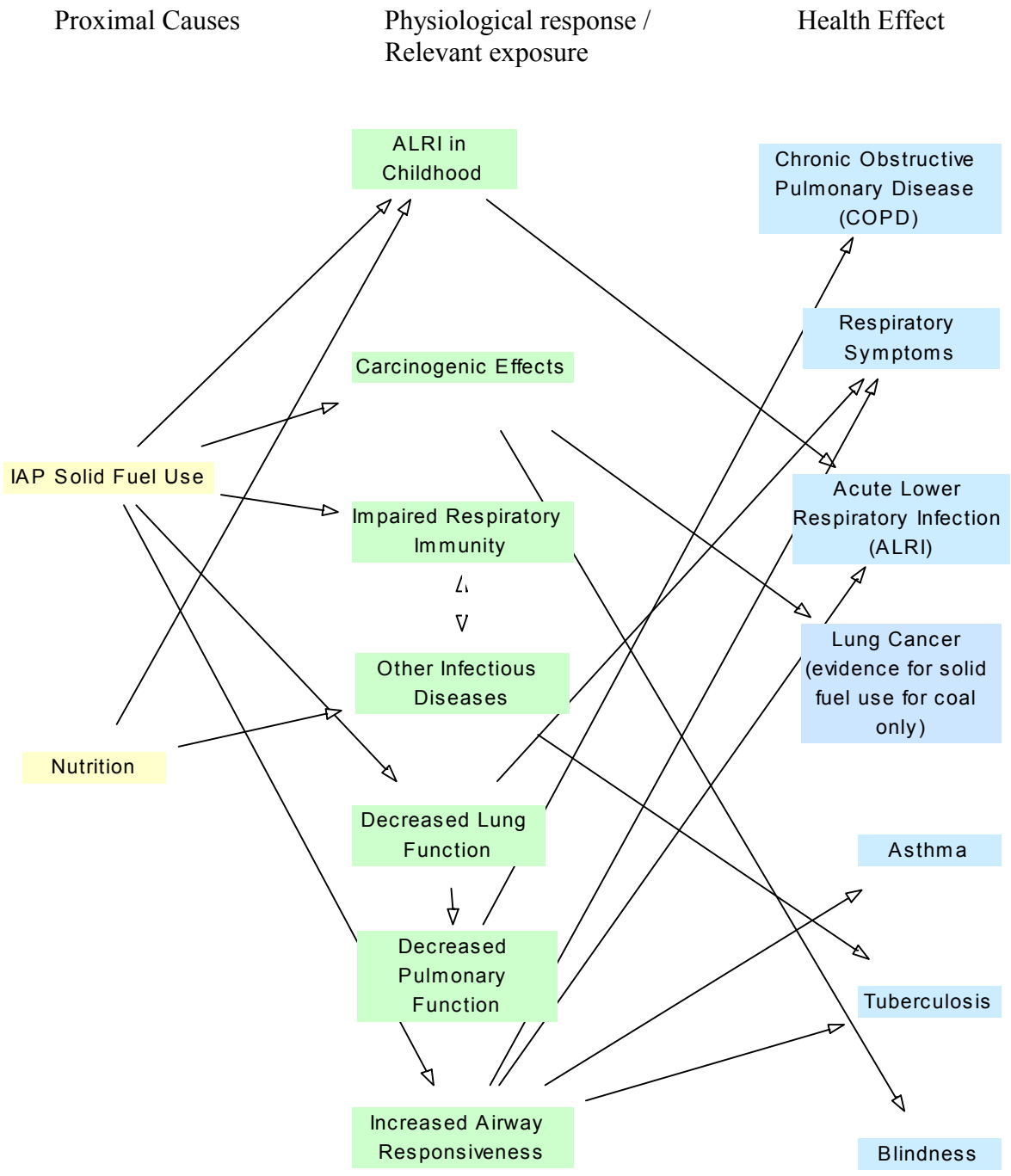


Figure 4: Indoor Air Pollution from Solid Fuel Use and Health of Adult Population



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