Health effects of cookstove emissions

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Despite the fact that the knowledge that air pollution promotes adverse health effects is not new, studies on pollution derived from indoor sources has received considerably less attention, mostly because its effects are less evident in areas where "hard science" is produced. In this study, the health consequences of prolonged exposure to indoor pollution generated by biomass-burning stoves are summarised. This kind of exposure is associated with respiratory, cardiovascular, reproductive and cancer outcomes. Considering that the use of biomass as fuel for cooking is almost entirely restricted to developing countries, some projections on the costs due to health consequences of this practice indicate that procedures must be implemented not only to avoid suffering caused to the population but also to remove the extra burden on frail economies.

1. Introduction

Significant health damage costs can be associated with indoor air pollution arising from combustion of biomass and coal for cooking, water-heating, and space-heating. The substitution of cleaner fuels for direct solid fuels used for cooking and heating in developing countries is essential to address poverty and public health issues in rural and some urban areas. Sustainable development must aim at creating universal access to cleaner household fuels at least to the extent required to satisfy basic human needs. Evaluation and quantification of health damage costs are important to help perform techno- and socio-economic analyses, and developing policies towards the replacement of solid fuels with cleaner fuels.

In the first half of the 20th century the episodes of severe air pollution in the Meuse Valley [Firket, 1931], Donora [Ciocco and Thompson, 1961] and London [Logan and Glasg, 1953] clearly demonstrated acute health effects of ambient air pollution and promoted public awareness and the promulgation of laws regulating emissions from stationary sources. As a consequence of such measures, air pollution decreased in most large Western urban centres, attaining levels below the limits prescribed by air quality standards. During the same time window, pollution derived from indoor sources received considerably less attention, mostly because its effects are less evident in areas where "hard science" is produced. In fact, for the majority of the developed nations, indoor pollution is represented almost exclusively by passive smoking. However, if one considers the number of people exposed to emissions derived from low-technology stoves burning biomass-derived fuels, and the high levels of air pollution reached in these homes, it is clear that energy and resources should be directed to study and correct this situation. The present study plans to conduct the reader across some basic questions on the subject that deserve further attention.

- What is the magnitude of the health effects of indoor pollution generated by burning biomass for either cooking or heating?
- What are the main acute outcomes?
- What are the consequences of prolonged exposure to such pollution?
- What are the possible mechanisms responsible for the observed health effects?

To achieve the aforementioned objectives, this paper will touch on concepts of epidemiology, pathology and cell biology. For the sake of clarity, narrow technical/medical wording will be deliberately avoided.

2. How severe is indoor pollution due to biomass use and how many people are exposed to it?

It is estimated that, globally, almost two and half billion people use biomass, including wood, charcoal, crop residues and dung, as their primary source of energy [Reddy et al., 1997]. In several lower-income developing countries, biomass use still accounts for as much as 95 % of domestic energy [Arungu-Olede, 1984]. Although globally the use of biomass is declining, there is clear evidence that its use is increasing amongst the poorest segments of the world's population [Bruce et al., 2000]. Thus, the number of people exposed to severe indoor pollution derived from biomass-burning probably exceeds the number under the influence of severe outdoor pollution, which, even in developing countries, results mostly from the burning of fossil fuels for powering transportation or industrial processes.

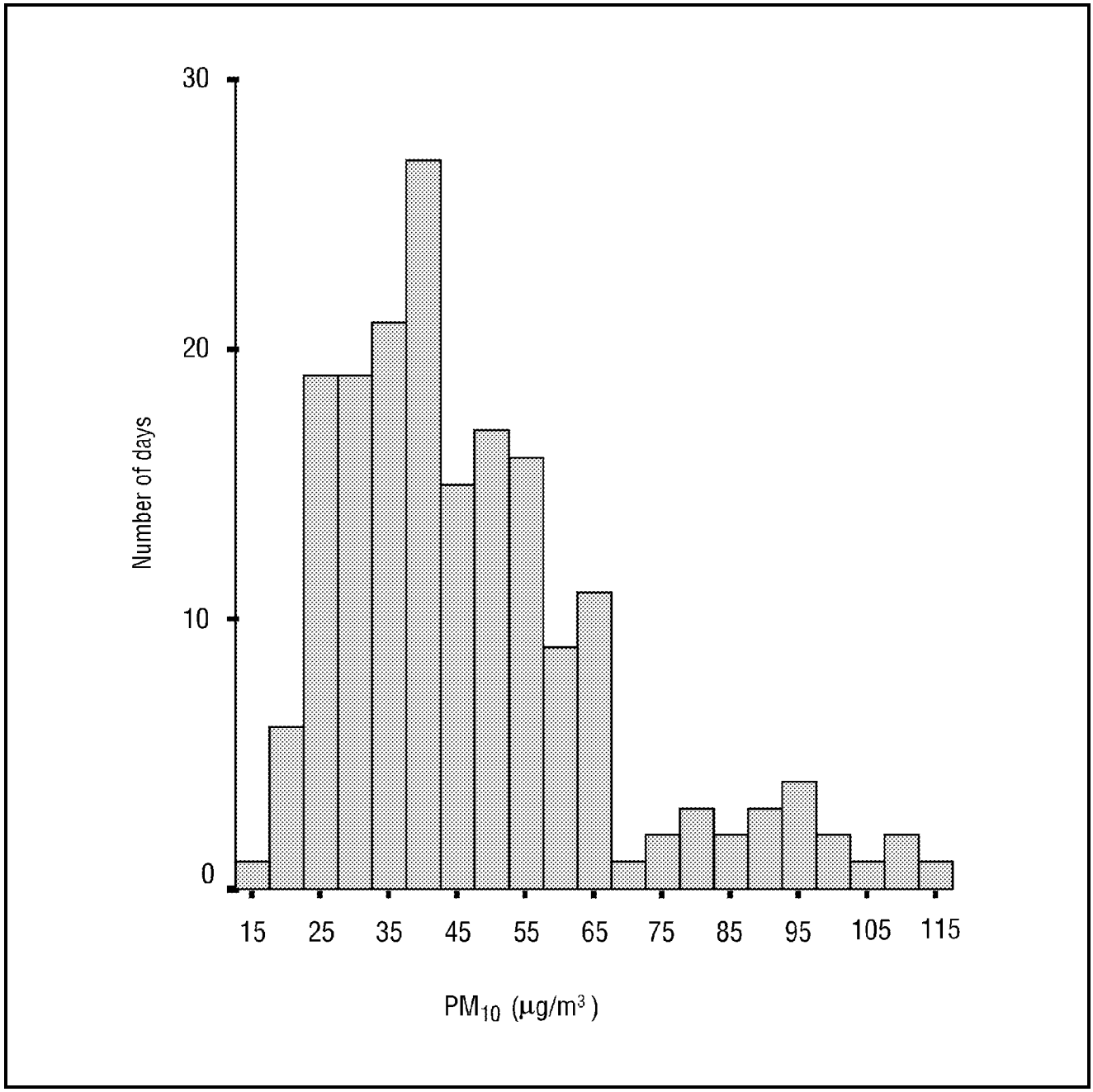

Figure 1. Frequency distribution of ambient concentrations of PM_{10} in São Paulo during the first 6 months of 2000. This scenario is characteristic of the air pollution levels found in many large cities of developing countries, where air pollution is dominated by automotive or industrial sources. Elaborated from [CETESB, 2001]

The indoor concentration of a given pollutant is established by the rates of its production and removal from the environment. Both of the aforementioned factors favour the accumulation of air pollutants when biomass is employed for cooking. First, the air exchange rate of an indoor environment is considerably smaller than for an outdoor setting. Solid fuel stoves also tend to be inefficient combustion devices. Burning of biomass leads to the formation of several pollutants, such as carbon monoxide, nitrogen oxides, aldehydes, methane, polycyclic aromatic hydrocarbons and primary and secondary particles. Primary particles are usually composed of a core of carbon which has several compounds adhering to its surface, whereas secondary particles are formed mainly by gas conversion to particles, such as nitrogen oxides to nitrates. All of these components are harmful to health to some degree or other and may exhibit a high concentration inside homes during the cooking process.

We shall illustrate the severity of air pollution in this scenario by focusing on particles with an aerodynamic diameter below 10 μm, also known as PM_{10}, i.e., the fraction of aerosol that penetrates into the lungs. The average daily outdoor concentration allowed by international standards is 150 $\mu g/m^3$ and the annual average concentration must not exceed 50 $\mu g/m^3$. For instance, the mean concentration of PM_{10} in São Paulo during the first 6 months of 2000 was 47 $\mu g/m^3$, following a distribution depicted in Figure 1.

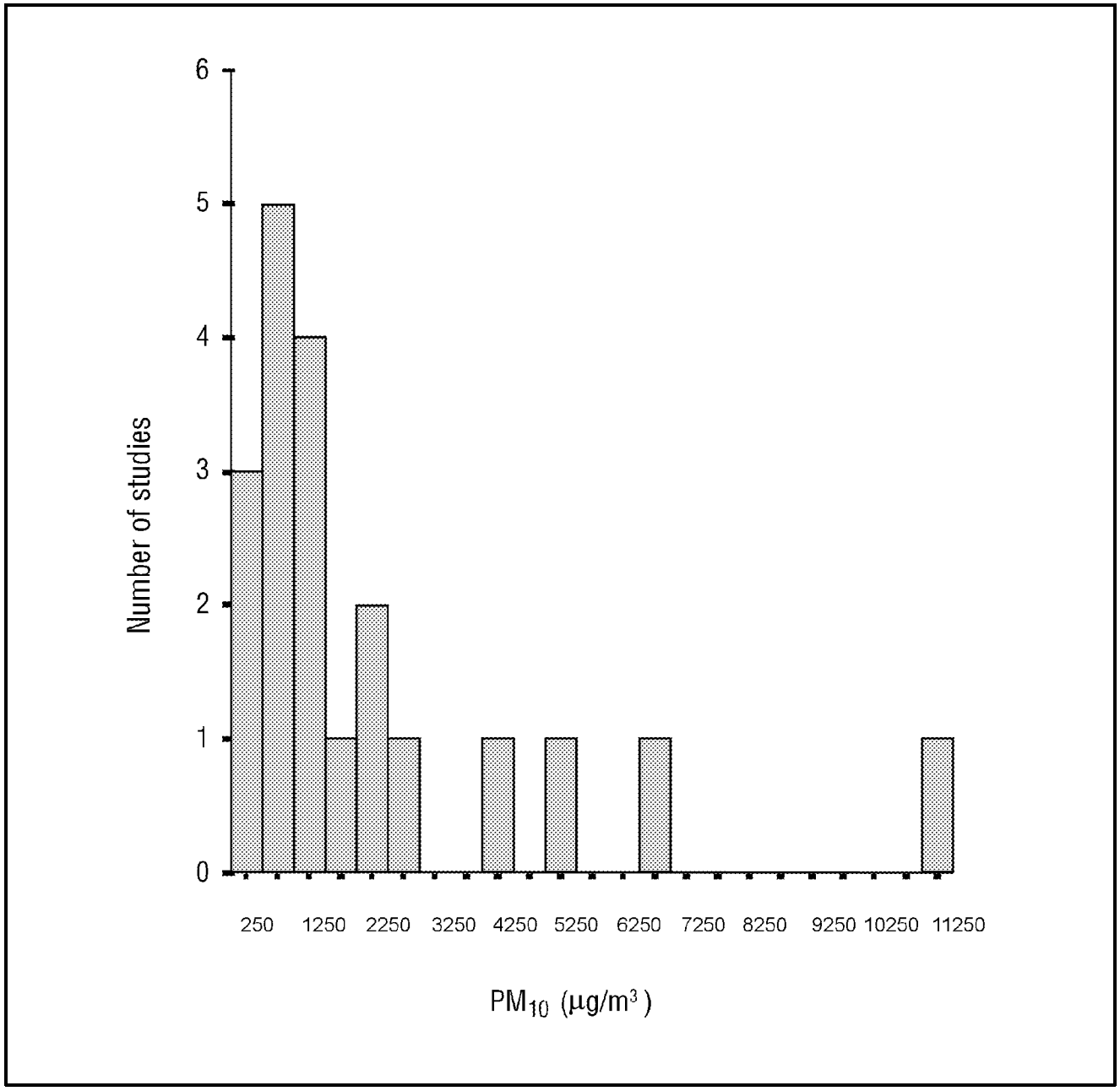

Figure 2. Frequency distribution of indoor concentrations of PM_{10} from biomass combustion in developing countries [Reddy et al., 1997]. The values were obtained from studies made in the Pacific (2 studies), South Asia (15 studies), China (8 studies), Africa (8 studies) and Latin America (5 studies).

If we make the same distribution plot using the concentrations reported in one study that measured indoor particulate concentrations from biomass combustion, the levels are significantly higher than those presented in Figure 1, attaining a mean concentration of about 2,200 $\mu g/m^3$ (Figure 2). These levels are not uncommon in many homes burning biomass or coal for cooking or heating in developing countries. If the same levels were reached in an urban outdoor setting, the situation would be considered a severe episode of air contamination and immediate measures would be adopted to preserve public health.

In developing countries, cooking is an activity almost exclusively performed by women. The same environment, most probably, is shared by their children and elderly people. Thus, these are the segments of the population with higher exposure.

3. What are the health consequences associated with indoor biomass-burning?

Most knowledge about the health effects of air contamination is derived from epidemiological studies. In the case of air pollution, the majority of studies available in the literature focus on outdoor air pollution. In this section, we will employ data gathered from either outdoor or indoor pollution studies, on the basis of two assumptions. First, among the constituents of indoor and outdoor pollution are many common compounds, the main differences being due to the higher concentration of some primary pollutants (such as CO, NO_2 and PM_{10}) and smaller amounts of secondary oxidants, i.e., reactive compounds such as ozone and components of photochemical smog formed in the outdoor environment by chemical reactions powered by sunlight. Second, despite the molecular

structure of wood or charcoal being probably simpler than that of gasoline or diesel, studies performed in areas where outdoor pollution is highly influenced by biomass-burning reveal that particles generated from biomass-burning exhibit similar toxicity to those produced by combustion of petroleum-derived fuels [Arbex et al., 2000].

Epidemiologists are responsible for characterising health effects of air pollution. The largest amount of information in this area can be obtained by following a given community of individuals across a defined period of time and correlating changes in measures of air pollution to variations of an indicator of health – usually mortality or morbidity counts. This type of epidemiological design is known as *time-series study.* Another way of getting useful information on air pollution effects is by comparing two or more individuals living in environments with different pollution backgrounds, in *cross-sectional or case-control studies.* Finally, we will discus data derived from the follow-up of a group of individuals living in an environment with a special profile of pollution or exhibiting a peculiar health condition that makes them more susceptible to the action of air contaminants. This approach is known as *cohort study* (if a population is the subject of investigation) or a *panel study* (in the case that a group of individuals is being studied). Of course, these three epidemiological designs are not mutually exclusive. They may be combined to increase the power of the study. For example, it is possible to incorporate a time-series within a cross-sectional study, or follow a group of patients with chronic bronchitis (panel study) in different communities.

When analysing the literature about the health effects of indoor pollution from biomass-burning in developing countries, there are some common characteristics.

- The majority of studies have employed *cross-sectional* or *case-control* approaches, implying that there is adequate control of other confounding variables (smoking, general health, and socio-economic conditions, sanitation, etc.), which is critical to interpreting the effects of pollution.
- Measurements of exposure are made during relatively short periods of time, evaluating, predominantly, particulate matter.
- Unlike many situations in occupational medicine, where a substance (or its metabolite) or an element is specific to the emission source, there is no specific marker of exposure to biomass smoke. Thus, measurements of inflammatory markers or pulmonary function are employed to assess the risk or to quantify the magnitude of health impairment. In studies focusing on health effects of biomass-derived pollution, physiological measurements in the exposed individuals are restricted to measurements of vital capacity of forced expiratory flows, which are rather crude indicators of pulmonary function. We did not find studies employing collection of cells from the lungs or blood, or other indicators of cardiovascular impairment, such as ECG (electrocardiogram) or measurements of heart-rate variability.

The aforementioned limitations do not obscure the conclusion that indoor air pollution harms health, but clearly indicate the need to employ more sophisticated approaches to have a better picture of the magnitude of the damage, since the available data most probably underestimate its effects.

At this point, we should make the reader aware that most of the discussion about the effects of indoor air pollution on health will be related to information on the effects of particles. This choice was made for several reasons. First, there is a vast literature relating health effects to particle concentrations, derived either from indoor or outdoor sources, which largely outnumber the studies on the effects of the gaseous fraction of air contamination. In fact, when a source of air pollution operates, it simultaneously emits gases and particles with a high degree of cross-correlation and, thus, in the complex scenario of real-world air pollution, it is frequently not possible to ascribe to a single pollutant the responsibility for any observed health effect. Thus, in this paper, we will encourage the reader to understand particles as a proxy variable for overall indoor air pollution, rather than the only cause of the described effects. Also, it is necessary to clarify that we avoided including in the discussion of health effects an important (and rather extensive) source of indoor pollution: tobacco smoke. This choice was made with the aim of focusing on pollution from cooking and heating, problems with different economic and social characteristics and, consequently, deserving a strategy of intervention quite different from that needed in the case of tobacco control.

3.1. Acute respiratory infection, asthma and bronchitis

Acute respiratory infections in children exhibit a consistent association with exposure to biomass pollution [Collins et al., 1990; Kossove, 1982; Campbell et al., 1989; Armstrong and Campbell, 1991]. Although there are studies reporting no association between indoor pollution and acute respiratory infections [Johnson and Aderele, 1992], the majority report that indoor pollution entails a significant risk of respiratory morbidity. It is possible to estimate that children exposed to indoor pollution derived from biomass have a risk two to five times higher than non-exposed children of developing pulmonary infections. Increased risk of acute upper respiratory infection and otitis media (range 1.03 to 2.89) has been reported in children exposed to indoor pollution [Strachan and Cook, 1998; Daigler et al., 1991].

There are several mechanisms that make children exposed to pollution more susceptible to acute respiratory infection. First, air pollution may damage the airway defences. The mucociliary apparatus represents the first line of defence of the lungs against inhaled noxious agents, by removing particles and chemical species from the airways by means of the continuous transport of the airway mucus to the oropharynx using the mechanical input provided by the coordinated beating of the ciliated cells [Macchione et al., 1999]. In this context, the mucociliary apparatus represents a point of important interaction between

the organism defences and inhaled pollutants, and the result of such contact influences the development of adverse respiratory effects promoted by air pollution. There is evidence that exposure to air pollutants induces structural and functional abnormalities of the mucociliary apparatus mainly in the phase when such defence undergoes maturation, during which the capacity of the respiratory system to deal with inhaled pathogens is reduced [Flo-Neyret et al., 2001; Macchione et al., 1999; Lemos et al., 1994, Souza et al., 1998; Saldiva et al., 1992].

Air contaminants also interfere with the cellular and immune defences of the lungs. Oxidants, such as NO_2, decrease the ability of alveolar macrophages to phagocytose *Candida albicans* and to produce superoxide [Devlin et al., 1999], indicating that the capacity of killing microorganisms, either by phagocytosis or by the release of free radicals, is impaired in this situation. Carbon monoxide confers potent anti-proliferative effects in CD-3 activated T-lymphocytes, which are mediated by p21-dependent caspase activity, in particular caspase 8 [Song et al., 2004], suggesting that the proliferation of immune cells triggered by the presence of foreign micro-organisms may be impaired by the presence of such air pollutant.

Considering the foregoing information, there is solid biological plausibility for the observations relating indoor pollution and increased risk of acute respiratory infection and pulmonary inflammation in the exposed population. The health consequences of such alterations may be expressed either in terms of the increased morbidity air pollution produces [Braga et al., 2001; Lin et al., 1999; Martins et al., 2002; Medeiros et al., 2004; Pereira et al., 1998; Saldiva et al., 2002], or mortality [Conceição et al., 2001; Lin et al., 2004; Saldiva et al., 1994, 1995].

3.2. Cardiovascular diseases

The concept that air pollution affects cardiovascular diseases has gained strength after the publication of epidemiological studies relating changes in air pollution to cardiovascular morbidity and mortality (reviewed in [Pope, 2000]). Increases in air pollution have been associated with objective markers of cardiovascular risk, such as circulating white cell counts, plasma fibrinogen levels and decreased heart rate variability [Dockery, 2001; Medeiros et al., 2004; Schwartz, 2001]. A recent study [Brook et al., 2002] reported that the inhalation of concentrated air particles plus ozone causes narrowing of the brachial artery in humans. Concentrated air pollution particles enhance experimental cardiac ischemia in canine studies [Godleski et al., 2000; Wellenius et al., 2003]. In addition, acute exposure to concentrated air particles has been shown to induce vasoconstriction of small pulmonary arteries in normal and chronic bronchitic rats [Batalha et al., 2002]. This vasoconstriction was correlated with specific particle components, and suggests that the pulmonary vasculature might be an important target for ambient particle toxicity.

3.3. Reproductive health and pregnancy

Air pollution has been shown to be associated with decreased fertility and low birth-weight by several groups [Bobak et al., 1997; Gouveia et al., 2004; Ritz et al., 2000, 2002; Wang et al., 1997]. Pereira et al. [1998] showed an association between intrauterine mortality and air pollution in São Paulo. In addition, evidence of foetal exposure to air pollution was suggested by a significant association between ambient CO levels and the levels of carboxyhemoglobin of blood sampled from the umbilical cord in children delivered by non-smoking women. Other studies also suggest that reproductive function in females may be a target of air contaminants [Ford et al., 1995; Hughes and Cooperg, 1995; Sharara et al., 1998]. Male reproductive function has also received attention. Several studies reported a significant effect of air pollution on male fertility [Adamopoulos et al., 1996; Auger et al., 1995; Bahadur et al.,1996; Ginsburg and Hardiman, 1992; Haidl et al., 1996; Irvine et al., 1996], but negative associations in this respect were also reported [Berling and Wolner-Hanssen, 1997]. This heterogeneity of results suggests that semen production and quality may be influenced by geographic and environmental factors [Bahadur et al., 1996], including air pollution. Therefore, both male and female reproductive outcomes may be the target for this very important influence of air pollution on people.

3.4. Cancer

The exposure of women to emissions generated by biomass stoves has been shown to significantly increase the rate of lung [Smith and Liu, 1993] and laryngeal [Clifford, 1972] cancers. Based on outdoor pollution data, Pope et al. [2002] computed that each 10-g/m^3 elevation in fine particulate concentration was associated with approximately 8 % increased risk of lung cancer mortality. These findings were consistent with the increase in risk of developing lung cancer observed in the range of 2 to 6 times detected in Chinese women exposed to coal stove emissions [Smith and Liu, 1993].

4. Cost of indoor pollution

The annual number of deaths attributable to biomass indoor air pollution adds up to over 1.6 million people worldwide, predominantly women and children, which corresponds to approximately 30 million disability-adjusted life-years (DALYs) a year [WHO, 2002]. DALY is a health indicator that provides measurement of the adverse effects in a time basis considering both the morbidity (years lived with disability, that is, time lived in a health status other than perfect) and mortality (years of life lost or time lost due to premature death).

The global mortality and burden of disease due to different risk factors were analyzed by Ezzati et al. [2002]. Indoor smoke from solid fuels were the eleventh and eighth highest risk factors for mortality and attributable DALY, respectively. The different ordering of risk factors in their contributions to mortality and burden of disease reflects the age profile of mortality, resulting in larger loss of healthy life-years because it is concentrated in children aged younger than 5.

In order to make a monetary estimate of this burden, we can convert DALYs to economic values through the use of the value of a statistical life (VOSL). As there is no market to determine the value of a human life, the

VOSL is usually inferred from a proxy market, such as the wage differentials between risky and less risky jobs, voluntary purchase of seat belts, etc., or obtained by surveying a strategically sampled population for their willingness-to-pay or willingness-to-accept (WTP/WTA) for a change in the probability of death due to an additional risk, say, from air pollution [Wang, 2004]. However, VOSL variations are enormous: there are several ethical and technical approaches, socio-economic differences from country to country and many other associated factors. Viscusi and Aldy [2003] have reviewed estimates of value of life comprehending different methodological approaches in many parts of the world. Half of the studies, based on the US labor market, reveal a value of a statistical life ranging from $ 5 million to $ 12 million. In addition, this review of the VOSL showed that developing countries tend to have lower values of statistical life than do developed countries. Since solid fuels are mostly used in developing countries, a VOSL estimate based on a study conducted in a developing country would better represent localities with indoor smoke from solid fuels in estimating the monetary values of the health damage.

Considering a value of statistical life (VOSL) for developing economies due to air pollution evaluated by Bowland and Beghin [1998] of US$ 519,000, but acknowledging that the range of values for VOSL is large, the annual global burden of biomass indoor pollution is estimated to be about US$ 232 billion[1]. In order to have an idea of the magnitude of this figure, we may compare it with the world's and developing countries' GDP values: US$ 36.4 and US$ 6.3 trillion, respectively [World Bank, 2004]. That is, this estimate of the cost of indoor air pollution from using solid fuels is equivalent to approximately 0.64 % of the world's GDP and 3.68 % of the developing countries' GDP. In this sense, the adverse health effects of biomass indoor air pollution could be considered a quite representative burden, even if other VOSL estimates or health valuation techniques were employed. Note that we have only considered premature deaths due to exposure to indoor air pollution from biomass-burning, not the morbidity. If the risk of morbidity is included, the total health damage resulting from the exposure to indoor air pollution would be even higher.

The actual scenario may become worse due to the high price of cleaner fuels compared to solid fuels – higher fuel prices impact on poor families which may substitute gas and electric energy with biomass-burning, especially in developing countries, increasing indoor smoke emissions. However, as suggested in the earlier discussion in this section, the cost of implementing indoor air pollution controls (e.g., through increasing use of clean fuels) is most likely to be economically justifiable.

5. Conclusion

The negative effects on human health of utilization of solid fuels for indoor cooking and heating in very large population segments worldwide are extremely important and deserve substantial mitigation efforts by national/local governments. The identification and quantification of health damage is essential to put in perspective possible actions in the way of technology development and implementation to address the problem. ■

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Note

1. Total burden = DALYs (30 million)/average life expectancy in developing countries (67 years)×VOSL (US$ 519,000)

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