

# **Health Impacts of Biomass Air pollution**

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## **I. Executive Summary**

In 1997, uncontrolled forest fires burning in Indonesia resulted in a regional air pollution episode of biomass smoke which impacted several Southeast Asian nations. Biomass smoke from the fires resulted in elevated levels of particulate air pollution for a period of approximately 2 months in many areas (beginning in late July 1997), with a severe episode occurring during most of the month of September. During this period particulate levels in some areas were up to 15 times higher than normal levels. Exposures to “haze”-type air pollution can be substantial and are of public health concern due to the large numbers of individuals who may be exposed.

Biomass smoke contains a large and diverse number of chemicals, many of which have been associated with adverse health impacts. These include both particulates and gaseous compounds such as carbon monoxide, formaldehyde, acrolein, benzene, nitrogen dioxide and ozone. Particulate matter is itself a complex mixture which is associated with a wide range of health impacts. Elevated concentrations of particulate matter are consistently observed in situations where exposure to biomass material is burned. Exposures to high concentrations of carbon monoxide and other pollutants are highly variable and only occasionally observed in individuals such as wildland firefighters and people who cook with biomass fuels. Review of the exposure and health impacts literature, as well as initial evaluation of the available air monitoring data from the 1997 “haze” episode, indicates that the pollutant variable most consistently elevated in association with biomass smoke is particulate matter. Accordingly, the emphasis throughout this manuscript and of recommended future studies will be focused on particulates.

### **Non-cancer health effects**

Studies of wildland firefighters, an occupational group exposed to high levels of biomass smoke clearly indicate an association between exposure and acute effects on respiratory health. Longer term effects, lasting for a 3-6 month firefighting season, have also been observed in most studies although these effects appear to be relatively small and may be reversible. Firefighters are an extremely fit and healthy group and cannot be considered representative of the general population. Accordingly the demonstration of health effects in this occupational group indicate the plausibility, but not the magnitude, of an association between biomass smoke exposure and adverse effects in the general population.

The health effects of biomass smoke inhalation have also been documented in developing countries where women, and in some cases, children spend many hours cooking over unvented indoor stoves. Approximately 50% of the world’s population uses biomass fuels for cooking and/or heating. In particular, exposure to smoke from cooking fires has been identified as a risk factor for acute respiratory illness, the leading cause of infant mortality in developing countries. In addition, the women who are cooking are also at risk for chronic respiratory diseases. As these exposures last for 20 or more years, they are much higher than those associated with “haze” episodes. However, the studies conducted in developing countries indicate the serious consequences of exposure to high levels of biomass smoke. Increased acute respiratory illness in children is likely a major cause of infant mortality and the development of chronic lung disease in adults is associated with premature death and increased illness.

Many recent studies have also indicated that levels of air pollution currently measured in most urban areas in the world are associated with a range of adverse health outcomes. The most startling finding of these studies is the association of particulate air pollution, with increased daily mortality. These studies have been conducted by different investigators in a variety of locations, using a variety of study designs. In nearly all cases, the studies indicate an association between particle air pollution and increased risk of death, primarily in the elderly and in individuals with pre-existing respiratory and/or cardiac illness. Recent studies have also suggested an association between particulates and infant mortality. Increased risk of hospital admissions and increased emergency room visits have also been associated with short-term increases in the levels of particle air pollution. These data strongly suggest that any combustion-source particulate air pollution, including that produced during forest fires, is associated with a whole range of adverse health outcomes.

Specific studies of exposure to biomass smoke indicate a consistent relationship between exposure and increased respiratory symptoms, increased risk of respiratory illness and decreased lung function. These studies have mainly been focused on children, although the few studies which evaluated adults also showed similar results. A limited number of studies also indicate an association between biomass smoke exposure and visits to hospital emergency rooms. There are also indications from several studies that asthmatics are a particularly sensitive group. By analogy to the findings of numerous studies associating increased mortality with urban particulate air pollution mixtures, there is no evidence that particles from different combustion sources have different impacts on health, while particles generated by natural processes such as volcanic eruptions and windblown soil do appear to have less of an impact on health. Therefore, there is little reason to expect that biomass smoke particulate would be any less harmful than other combustion-source particles and it is prudent to consider that “haze” exposure will also be related to increased mortality. The particulate studies also do not show evidence for a threshold concentration at which effects are not observed.

Nearly all of the studies of biomass smoke health effects conducted in North America evaluated impacts of concentrations which were much lower than those associated with the 1997 Southeast Asian haze episode. Similarly, these studies involved exposure durations which were of comparable length to those experienced in Southeast Asia. Based on these studies it is reasonable to expect that the Southeast Asian haze episode resulted in the entire spectrum of acute impacts, including increased mortality, as well as seasonal effects on lung function, respiratory illness and symptoms. It is not possible at this time to determine the long term effect, if any, from a single air pollution episode, although repeated yearly occurrences of haze should be cause for serious concern. Chronic (several years) exposure to particulate air pollution in urban areas, at much lower levels than experienced in Southeast Asia in 1997, has been associated with decreased life expectancy and with the development of new cases of chronic lung disease.

### **Cancer**

The available, although limited, data on biomass smoke and cancer do not indicate an increased risk even at very high levels of exposure. This evidence includes studies of long-term exposure to high levels of biomass smoke from domestic cooking in developing countries. Evidence for a relationship between urban particulate air pollution and lung cancer is also limited, but is

suggestive of a small, but measurable, increased risk. There have not been enough studies conducted to evaluate the consistency of any increased risk for different particle sources. However, while biomass smoke clearly is potentially carcinogenic, it is much less so than motor vehicle exhaust.

### **Research questions**

Given the uncertainty regarding the potential for long-term effects associated with “haze” type air pollution, it would seem reasonable to initially evaluate the acute health impacts, especially since these are likely to include severe impacts such as increased mortality. To help understand the potential for adverse health effects and to evaluate the effectiveness of various mitigation measures, there is also a need to investigate several exposure issues. Several major research questions are summarized below. However, before these questions can be addressed it will be necessary to identify the availability of data, specifically air monitoring data and valid data on health indicators such as daily mortality, hospital visits, clinic/emergency room visits, etc.

1. What were the short term human health impacts associated with exposure to biomass air pollution in Southeast Asia?

For the range of identified impacts, were the effects reversible or permanent?

2. What were the long term human health impacts (if any) associated with exposure to biomass air pollution in Southeast Asia?
3. Which (if any) population groups were especially susceptible to adverse health effects of biomass air pollution in Southeast Asia?
4. What was the size of the exposed population?

Using study results and available air monitoring data (possibly including satellite data) can the region-wide health impacts be estimated?

5. What was the relationship between differences in exposure and health impacts across the affected region?

Were there exposed areas in which health impacts were larger/smaller than others?

Can an exposure-response relationship be demonstrated throughout the region?

6. What was the effectiveness of the following health protection measures:

The use of dust masks

Advising the population to remain indoors?

7. What was the composition of the biomass air pollution which affected Southeast Asia?

Can specific biomass marker compounds be identified?

To what extent is it possible to distinguish biomass air pollution from the “background” urban air pollution?

### **Recommended studies**

With regard to the general research questions identified above, several possible study designs are proposed:

- a. Formal study of the acute impacts of forest fire-related air pollution episodes should be conducted. Ideally, these studies should be directed towards the most severe health outcomes, while considering that impacts of air pollution will be small relative to all other causes of morbidity and mortality. To the extent possible, specific study protocols should be standardized and conducted in several regions where ambient air concentrations differed.
- b. Formal study of the long-term impacts of forest fire-related air pollution may be attempted although it must be acknowledged that these studies are extremely difficult to conduct, and even the best studies are unlikely to will provide firm results.
- c. A region-wide composite database of ambient air concentrations should be developed. Estimated air pollution contour plots can be developed using available air monitoring data, and, if feasible, supplemented with airport visibility and remote sensing data. With this type of a database, the regional health impact of biomass air pollution episodes can be estimated.
- d. The effectiveness of masks for use by the general public should be evaluated. An additional aim should be an adequate understanding of the variables which determine mask effectiveness, including technical factors such as filtration efficiency and leakage, as well as non-technical issues such as population compliance and comfort. Identification of the most important variables determining mask effectiveness will enable the design of new masks that are specifically applicable for general public use.
- e. The effectiveness of remaining indoors during haze episodes should be investigated. Specifically, the effectiveness of air cleaners, air conditioners, open/closed windows within various building types (residential office, etc.) as they relate to indoor penetration of fine particles should be assessed.
- f. Detailed chemical analysis of particulate samples should be conducted to identify the proportion of various functional groups within the haze particulate. While this analysis may be useful in future risk assessment and in comparing the toxicity of these particulate samples to those collected in other locations, the current emphasis should be on identifying marker compounds which may be used to distinguish air pollution originating from biomass burning from other sources.

### **Recommended health protection measures**

Due to the limited effectiveness of other health protection measures during regional haze episodes, priority emphasis must be given to elimination of the source of the air pollution, which in this case is extinguishing fires or preventing their occurrence. Close interaction between health, environment and meteorological agencies could result in effective forecasting of future air pollution episodes, be they related to forest fires or local sources of air pollution. However, despite efforts to prevent and control fires it is acknowledged that other measures may be necessary to help mitigate public health impacts. If the control or prevention of fires is not feasible, this should be followed by exposure avoidance activities such as reduced physical activity and remaining indoors. To enhance the protection offered by remaining indoors, individuals/building managers should take actions to reduce the infiltration of outdoor air. There is evidence that air conditioners, especially those with efficient filters, will substantially reduce indoor particle levels. To the extent possible, effective filters should be installed in existing air conditioning systems and individuals should seek environments protected by such systems. There is strong evidence that portable air cleaners are effective at reducing indoor particle levels, provided the specific cleaner is adequately matched to the indoor environment in which it is placed. Unfortunately, economics will limit the distribution of such devices throughout the population. As with air conditioners the increased use of such devices by a large segment of the population may have a significant impact on energy consumption. The least desirable measure of health protection is the use of dust masks. While these are relatively inexpensive and may be distributed to a large segment of the population, at present their effectiveness for general population use must be questioned. Despite this reservation, it is likely that the benefits (even partial) of wearing dust masks will outweigh the (physiological and economic) costs. Accordingly, in the absence of other mitigation techniques, the use of dust masks is warranted. Education of the population regarding specific mask types to purchase, how to wear masks and when to replace them will increase their effectiveness as will the development of new masks designed for general population use.

## **II. Introduction**

In 1997, uncontrolled forest fires burning in **the** Indonesian states of Kalimantan and Sumatra, in combination with a severe regional drought, depressed mixing heights and prevailing winds resulted in a regional air pollution episode of biomass smoke which impacted Indonesia, Malaysia, Singapore, southern Thailand, Brunei, and the southern Philippines. In particular, several large urban areas such as Singapore, Kuala Lumpur and Kuching were affected. Biomass smoke pollution from the fires resulted in elevated levels of particulate air pollution for a period of approximately 2 months in many areas (beginning in late July 1997), with a severe episode occurring during most of the month of September. During this episode a State of Emergency was declared in Sarawak, Malaysia as 24-hour PM10 levels reached as high as 930  $\mu\text{g}/\text{m}^3$ , more than 15 times higher than normal levels. Intermittent episodes occurred in Indonesia, Malaysia and Singapore until mid-November.

Several recent review papers have discussed the health impacts and pollutants associated with wood smoke air pollution (Pierson, Koenig et al. 1989; Vedal 1993; Larson and Koenig 1994). Although the emphasis of these reviews was on North American exposures, many of the conclusions are relevant to the broader understanding of biomass air pollution, which is the subject of this paper. This document will describe material presented in these reviews as well as updated information. In addition, this paper will cover additional exposures to biomass air pollution encountered by forest firefighters and by individuals in developing countries who use biomass for cooking and heating. The available data on health impacts associated with community exposure to forest / bush fire related air pollution will also be presented. Emphasis will be placed on epidemiological studies of human health impacts and on peer-reviewed literature. The emission of pollutants from forest fires will also be addressed, with particular emphasis on tropical rainforests. As data regarding the specific concentration measurements and health impacts associated with the 1997 Southeast Asian biomass air pollution (“haze”) episode are just becoming available, these will not be addressed directly in the review portion of this document. The second part of this document will specifically be directed to the situation experienced in Southeast Asia and will cover research needs, suggest several possible research designs, and discuss measures which national governments may employ or recommend to mitigate public health impacts associated with biomass air pollution originating from forest fires.

## **III. Biomass air pollution**

Biomass smoke contains a large and diverse number of chemicals, many of which have been associated with adverse health impacts. These include both particulates and gaseous compounds such as carbon monoxide, formaldehyde, acrolein, benzene, nitrogen dioxide and ozone. Particulate matter is itself a complex mixture which is associated with a wide range of health impacts. Components of particulates such as polycyclic aromatic hydrocarbons (PAHs) are also found in biomass smoke. The transport of biomass burning emissions over hundreds of kilometers in Brazil has been extensively documented (Andrae, Browell et al. 1988). Haze layers with elevated concentrations of carbon monoxide (CO), carbon dioxide (CO<sub>2</sub>), Ozone (O<sub>3</sub>), and nitric oxide (NO) have been observed. During transport, many of the gaseous species are converted to other gases, such as ozone, or into particles, such as nitrate and organic nitrogen species.



## **A. Pollutants**

### **1. Gaseous**

The main gaseous components in smoke which are potential health hazards are carbon monoxide and aldehydes. A number of studies have also reported elevated concentrations of ozone, as well as ozone precursors, nitrogen oxides and hydrocarbons, in plumes from forest fires. In particular, fires burning in the savanna regions of Central Africa and South America have been studied in detail. In Brazil, ozone concentrations reach equilibrium values of approximately 20 ppb above background levels throughout a 2 million km<sup>2</sup> region during fire seasons (Delany, Haagenen et al. 1985).

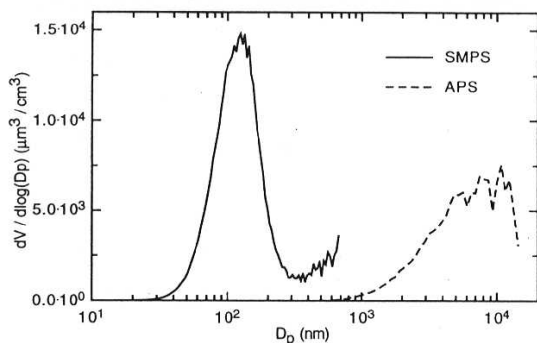
Comparisons have been made between three years of data collected at coastal site in Brazil not significantly impacted by biomass burning and measurements collected in a savanna region directly downwind of an Amazon forest region with intense burning. During the dry season elevated levels of CO and O<sub>3</sub> were measured at the savanna site, while during the wet season, levels at the two locations were nearly identical. Monthly average CO levels increased from 0.1 ppm to 0.7 and monthly average (noontime) O<sub>3</sub> levels increase from 20 ppb to 80 ppb (Kirchoff 1991). These O<sub>3</sub> levels are high relative to rural areas and are levels at which adverse health impacts have also been demonstrated.

Hydrocarbon and CO emissions were also measured in a study of savanna and forest regions in Brazil. Forest regions emissions of hydrocarbons were mainly alkanes (ethane and propane) and alkenes (ethylene and propylene) with smaller amounts (13%) of aromatics (benzene and toluene). Some differences in the relative composition of hydrocarbons were observed between forest fire and savanna emissions. CO levels were only slightly (0.5 ppm) increased in the atmospheric boundary layer. A recent study documented the impact of biomass pollutants transported 300 km from a fire in Alberta, Canada to the urban area of Edmonton (Cheng, McDonlad et al. 1998). Air trajectory analysis combined with monitoring of O<sub>3</sub>, nitrogen oxides and hydrocarbons, indicated that the forest fire had a significant impact on concentrations of gaseous pollutants. O<sub>3</sub> and nitrogen dioxide (NO<sub>2</sub>) concentrations were 50-150% higher than seasonal median levels.

### **2. Particulate composition and size distribution**

The size distribution of wood smoke has been measured by several investigators and indicates that nearly all particles are smaller than 1 μm, with a peak in the distribution between 0.15 and 0.4 μm. One assessment of particle size distributions of forest residue indicated that 82% of the particle mass was <1 μm and 69% <0.3 μm (Sandberg and Martin 1975). These size ranges are consistent with particle formation via condensation (Larson and Koenig 1994). Particles of this size range are not easily removed by gravitational settling and therefore can be transported over long distances. Constituents of biomass smoke may also undergo atmospheric transformations, although these have not been studied in detail. Hueglin and colleagues reported on a detailed analysis of wood smoke particle size. Particle sizes distributions were sensitive to specific combustion conditions, but generally were bimodal with a peak at approximately 0.1 – 0.2 μm, corresponding to incomplete combustion and larger (approximately 5 μm) particles consisting of unburned material. Only the smaller particles are expected to remain suspended in wood smoke

(Hueglin, Gaegauf et al. 1997). Measurements of particle size distribution in inside forest fire plumes in the Amazonian forest indicate that two particle generation mechanisms operate in forest fires: gas to particle conversion resulting in particles  $<2.0 \mu\text{m}$ , and convective dispersion of ash and semi-burned material. Due to high settling velocities of large particles, it is only the smaller particles ( $<2 \mu\text{m}$ ) which can be transported over long distances (Echalar, Gaudichet et al. 1995).



**FIGURE 4.** Particle volume concentration versus particle size measured at the stack of the wood chip burner using a SMPS and an APS. For the SMPS, particle size is given as the mobility diameter, whereas for the APS aerodynamic diameters are plotted.

FIGURE 1 (Ward and Hardy 1991)

Biomass smoke contains organic and inorganic particulates, including PAHs and a number of trace metals. Larson and Koenig recently reviewed the available information on particle composition (Larson and Koenig 1994). While, approximately 5-20% of wood smoke particulate mass is elemental carbon, the composition of the organic carbon fraction varies dramatically with the specific biomass fuel being burned and with the combustion conditions. Accordingly, profiles of specific PAHs, which are of concern for their potential carcinogenicity, are likely to be variable. For this reason, many measurements have focused on a single PAH with probable human carcinogenic properties, benzo[a]pyrene (BaP), as an example of the PAH group. Detailed analysis of organic wood smoke aerosol were conducted by Rogge et al (Rogge, Hildemann et al. 1998). Nearly 200 distinct organic compounds were measured in wood smoke, many of them derivatives of wood polymers and resins. Wood consists of cellulose (50-70%), hemicellulose (20-30%) lignin (30%), plus small amounts of resins and inorganic salts. In wood, cellulose compounds form a supporting mesh that is reinforced by lignin polymers. Together these compounds form the rigid wood structure. When burned, lignin polymers produce methoxyphenols, methoxy benzenes, phenols, catechols and benzene. Non-wood biomass does not contain lignin and therefore the methoxy phenols and methoxy benzenes are unique tracers of wood smoke combustion. Conifers (softwoods) produce large amounts of resin acids while deciduous (hardwoods) trees do not. Combustion of hardwoods produces more ash and therefore more trace elements than softwoods (Larson and Koenig 1994). Potassium is the trace element found at highest concentrations in wood smoke and has often been used as a wood smoke tracer.

Daisey et al compared concentrations of respirable particulate matter (RSP), extractable organic matter (EOM) and PAHs inside seven Wisconsin homes when the home's woodstove was

operated and when it wasn't. No statistically significant difference was observed between the two periods for RSP concentrations. Concentrations of EOM, however, were approximately two times higher, and concentrations of PAH were 2 to 46 times higher during the periods when the wood stoves were in operation. Total PAH levels were below 10 ng/m<sup>3</sup>. This study indicates that wood burning can increase indoor concentrations of particulate organic matter and PAH, due to direct indoor emissions and/or infiltration from outdoors (Daisey, Spengler et al. 1989). The atmospheric concentrations of total suspended particulate (TSP) matter and BaP were measured in a mountain community highly dependent on wood for residential space heating. BaP levels ranged from 0.6 to 14.8 ng/m<sup>3</sup>. These levels were significantly higher than BaP levels observed in metropolitan U.S. cities which are in the range of 2-7 ng/m<sup>3</sup> (Murphy, Buchan-RM et al. 1984).

Inorganic particle composition was studied inside biomass fire plumes from Amazonian forest and African and Brazilian savannas. Particles from savanna fires were enriched in K, P, Cl, Zn, Br, while tropical forest fire emissions were enriched in Si and Ca. The authors suggest that K may therefore be useful as a tracer for flaming and not smoldering fires (Echalar, Gaudichet et al. 1995). These measurements also indicated that smoldering fires contributed more than flaming fire to fine particle emissions. [Artaxo-P, 1993 #51]. Mass concentrations ranged from 30 µg/m<sup>3</sup> in areas not affected by biomass burning to 300 µg/m<sup>3</sup> in large areas (2 million km<sup>2</sup>) with intense burning. Additional studies of fine particle (<2 µm) composition associated with biomass burning in the Amazon Basin was reported by Artaxo, et al (Artaxo-P 1994). Biomass burning particulate is dominated by elemental (soot) and organic carbon, K and Cl, along with S, Ca, Mn and Zn. 24-hour average inhalable (PM<sub>10</sub>) and fine (PM<sub>2.5</sub>) mass concentrations as high as 700 and 400 µg/m<sup>3</sup>, respectively, were observed. The fine particle mass is composed of naturally released particles – organic carbon, soil dust particles and particles emitted during biomass burning. Dry season particulate levels were increased as a result of soil dust release during the entire dry season and biomass burning at the end of the dry season.

The issue of particulate air pollution and the components responsible for the observed associations between particulates and adverse health impacts is still quite controversial and currently unresolved. However, it may not be an important issue for health impacts if one agrees with the assumption that even though PM<sub>2.5</sub> (or PM<sub>10</sub>) itself may not be the agent responsible for health impacts, it is a good (the best known) surrogate for whatever components of air pollution are responsible. There is however, evidence to support a conclusion that PM<sub>2.5</sub> particulate is a better measure than PM<sub>10</sub> (Schwartz, Dockery et al. 1996) (Vedal 1997). Further, there is even reason to believe that combustion-source PM<sub>2.5</sub> itself is a responsible agent. This is based on the observation that numerous studies which demonstrate a relationship between particulate matter and health outcomes have been performed in different locations, where both the major particulate sources and the particulate composition itself are quite different. The only known common feature in these studies is the presence of combustion-source particulate air pollution.

**TABLE 1. SUMMARY OF MAJOR BIOMASS POLLUTANTS**

<b>Compound</b>	<b>Examples</b>	<b>Source</b>	<b>Notes</b>
Inorganic gases	Carbon monoxide (CO)	Incomplete combustion of organic material	Transported over distances
	Ozone (O <sub>3</sub> )	Secondary product of nitrogen oxides and hydrocarbons	Only present downwind of fire, transported over distances
	Nitrogen Dioxide (NO <sub>2</sub> )	High temperature oxidation of nitrogen in air	Reactive – concentrations decrease with distance from fire
Hydrocarbons	Benzene	Incomplete combustion of organic material	Some transport – also react to form organic aerosols
Aldehydes	Acrolein	Incomplete combustion of organic material	
	Formaldehyde (HCHO)	Incomplete combustion of organic material	
Particles	Inhalable particles (PM <sub>10</sub> )	Condensation of combustion gases; Incomplete combustion of organic material; entrainment of vegetation and ash fragments	Coarse + fine particles. Coarse particles are not transported and contain mostly soil and ash
	Respirable Particulates	Condensation of combustion gases; Incomplete combustion of organic material	For biomass smoke, approximately equal to fine particles;
	Fine particles (PM <sub>2.5</sub> )	Condensation of combustion gases; Incomplete combustion of organic material	Transported over long distances; Primary and secondary production
Polycyclic aromatic hydrocarbons (PAHs)	Benzo[a]pyrene (BaP)	Condensation of combustion gases; incomplete combustion of organic material	Specific species varies with composition of biomass

## **B. Exposures**

Exposure to biomass air pollution occurs in many settings. The highest concentrations of particles have been measured in forest fires themselves and in indoor air in developing countries where wood and other biomass is used as a cooking and heating fuel. In terms of exposure, domestic cooking and heating with biomass clearly presents the highest exposures since individuals are exposed to high levels of smoke on a daily basis for many years. Perez-Padilla and colleagues developed an index of hour-years, analogous to pack-years for smoking history. In a study of rural Mexican women who cooked with biomass, the mean hour-years was 102 (Perez-Padilla, Regalado et al. 1996). Pollutant levels measured in these settings have been described in several investigations, some of which are discussed in more detail in following sections. Daytime respirable particulate (approximately corresponding to PM<sub>3,5</sub>) measurements in China were 2600  $\mu\text{g m}^{-3}$  (Smith and Liu 1993). In Kenya (Wafula, Onyango et al. 1990) and The Gambia (Armstrong and Campbell 1991) 24-hour respirable particulate measurements were 1400 and 2100  $\mu\text{g m}^{-3}$ , respectively, while in Guatemala 24-hour PM<sub>10</sub> measurements were 850  $\mu\text{g m}^{-3}$  (Smith 1993). Brauer and colleagues measured particulate levels in rural Mexican homes. Mean PM<sub>10</sub> levels were 768  $\mu\text{g m}^{-3}$ . During cooking periods, the mean PM<sub>2.5</sub> level was 887  $\mu\text{g m}^{-3}$ , while peak (5 minute) PM<sub>2.5</sub> concentrations reached 2000  $\mu\text{g m}^{-3}$  or higher in most of the homes cooking with biomass (Brauer, Bartlett et al. 1996). During cooking periods, measurements in Brazil and Zimbabwe reported respirable concentrations of 1100 and 1300  $\mu\text{g m}^{-3}$ , respectively (Smith and Liu 1993). Even higher levels have been reported in Nepal and India, accompanied by extremely high exposures to BaP. These exposures have recently been reviewed by Smith (Smith 1993; Smith and Liu 1993).

Wildland (forest) firefighters comprise an occupational group with high exposure to biomass smoke. Exposures of wildland firefighters were recently reviewed by Reinhardt and Ottmar, and will be discussed further in the health effects section (Reinhardt and Ottmar 1997). The information regarding smoke exposures and health effects in firefighters is presented here to provide information on the plausibility of a relationship between smoke exposure and health impacts, as well as to indicate the levels of exposure encountered in this setting. It must be noted that firefighters are normally among the most physically fit in the entire population and do not normally suffer from any pre-existing health conditions. Accordingly the absence of health impacts among this group does not indicate that health impacts will not be observed in the general population. In contrast, it is reasonable to argue that the demonstration of health impacts amongst firefighters provides strong evidence that similar effects will be observed within the general population at equivalent or lower levels of exposure. Exposures of this population are seasonal (4-5 months per year) and highly variable depending upon the number of fires per season, the intensity of the fires and specific job tasks.

In a large study of 221 firefighters at 39 prescribed fires, Reinhardt and colleagues measured mean CO and PM<sub>3.5</sub> levels of 4.1 ppm (maximum = 38) and 0.63 (maximum = 6.9)  $\mu\text{g}/\text{m}^3$ , respectively. Mean formaldehyde, acrolein and benzene levels were 0.047 ppm, 0.009 ppm and 0.016 ppm, respectively. Griggs and colleagues measured CO exposures of firefighters at a peat

and ground fire in North Carolina. Downwind of the fire, CO concentrations averaged 75 ppm and peak values of 500 ppm were measured. Carbon monoxide exposure of bush firefighters were measured by Brotherhood, et al. Non-smoking individuals experienced mean exposures of 17 ppm and peak levels of 40 – 50 ppm. Smoking crew members were exposed to as much CO from their cigarettes as from the fires (Brotherhood, Budd et al. 1990). Measurements of carboxyhemoglobin in these firefighters indicated that health impacts of CO exposure were unlikely to occur. In a study of 22 firefighters, personal sampling was conducted for carbon-monoxide, sulfur-dioxide, nitrogen-dioxide, aldehydes, volatile organic compounds, total particulates, and PAHs. CO levels ranged between 4 and 8 ppm, while nitrogen dioxide concentrations were below the 0.2 ppm limit of detection. SO<sub>2</sub> concentrations ranged from non-detectable to – 1.2 ppm. Aldehyde, PAH and volatile organic compound levels were also low or below detection limits. Most total particulate concentrations were below 1.2 mg/m<sup>3</sup> although two 4 hour samples were above 15 mg/m<sup>3</sup>. It is not known what percentage of this was respirable. Across-shift symptom surveys indicated slight increases in eye, nose and throat irritation (Reh and Deitchman 1992). Materna and colleagues measured exposures of firefighters during several measurement campaigns over a three year period. Mean CO, respirable particulate, and formaldehyde exposures were 14 ppm, 1.4 mg/m<sup>3</sup>, 0.13ppm, respectively. Of the 12 specific PAHs detected, all were found at low levels (mean exposures <100 ng/m<sup>3</sup> except for phenanthrene at 380 ng/m<sup>3</sup>) (Materna, Jeffery et al. 1992). Although these data are limited, they are the most extensive available for PAH exposures.

Reh and colleagues conducted extensive exposure assessment in combination with a medical evaluation. Respirable particulate levels were 0.6 – 1.7 mg/m<sup>3</sup> and sampling for acid gases detected low levels. Lung function decreased (<3% change) and symptoms reports increased across workshifts. Electron microscopic examination of bandanas worn by the firefighters indicated that the bandanas had pore sizes of more than 100 µm, and therefore were not protective against respirable particulates or gaseous pollutants (Reh, Letts et al. 1994). Another extensive series of exposure measurements was conducted by Reinhardt and colleagues. Personal sampling was conducted on 37 firefighters. Peak (15 minute) exposures averaged 14 ppm, 2.08 mg/m<sup>3</sup>, 0.018 ppm, 0.117 ppm and 0.035 ppm, for carbon monoxide, respirable particulates, acrolein, formaldehyde and benzene, respectively Reinhardt 1995a). In summary the exposure measurements of firefighters, while variable, indicate the potential for exposure to carbon monoxide and respirable particulates at levels (above 40 ppm CO and above 5 mg/m<sup>3</sup> respirable particulate) which have been associated with adverse health impacts. These health effects will be discussed in more detail in the health effects section.

In one of the few measurements of rural community air pollution associated with large tropical forest fires, Reinhardt and Ottmar measured formaldehyde, acrolein, benzene, CO and respirable particulates (PM<sub>3.5</sub>) in a rural area of Rondonia Brazil during the peak of the biomass burning season in 1996 (Reinhardt 1996). Of the species measured, respirable particulate levels were significantly elevated, with mean levels of 190 µg/m<sup>3</sup>, with levels as high as 250 µg/m<sup>3</sup> measured during several of the 12-hour sampling periods. The authors estimated that background levels of particulates in non-burning periods were 10-20% of the levels measured during their study period. Particulate levels were also highly correlated with carbon monoxide concentrations, suggesting that carbon monoxide could be used as a surrogate measurement of smoke exposure. Similar correlations have been observed in studies of North American wildland

firefighters (Reinhardt and Ottmar 1997). The mean CO level was 4 ppm, which is similar to levels measured in moderately polluted urban areas and below the level expected to be associated with acute health impacts. The authors also reported increased levels of formaldehyde (average ambient levels of 16  $\mu\text{g}/\text{m}^3$ ) and benzene. Benzene levels (11  $\mu\text{g}/\text{m}^3$  average) were found to be higher than those measured in rural areas in other parts of the world and were comparable to those measured in many urban areas.

Another population with biomass pollution exposure are residents of North American communities where wood burning is prevalent. These elevated levels of ambient air pollution are seasonal (3-8 months depending upon the climate) and variable as they are strongly influenced by local meteorology. PM10 concentrations as high as 800  $\mu\text{g}/\text{m}^3$  have been measured in these communities, although peak levels (24-hour averages) of 200 – 400  $\mu\text{g}/\text{m}^3$  are more common (Heumann, Foster et al. 1991; Larson and Koenig 1994). PM10 measurements in British Columbia communities where wood smoke is the primary source of particulates indicate 24-hour averages of 2 – 420  $\mu\text{g}/\text{m}^3$  (Vedal 1993). Larson summarized several studies of PM measurements in communities with wood smoke as a major particulate source. In the reported studies, PM10 concentrations as high as 150  $\mu\text{g}/\text{m}^3$  and PM2.5 levels of 86  $\mu\text{g}/\text{m}^3$  were measured in cases where wood smoke contributed more than 80% of the particulate mass (Larson and Koenig 1994). As wood smoke is generally emitted outdoors and since people spend most of their time indoors, indoor penetration is an important variable for exposure assessment and will be discussed in more detail in following sections. It is estimated that approximately 70% of wood smoke particulate penetrates indoors (Larson and Koenig 1994), although this estimate is based upon a limited number of measurements in North American winter conditions.

### **Summary of biomass smoke exposures**

Elevated concentrations of particulate matter are consistently observed in situations where exposure to biomass combustion occurs. Due to the size distribution of biomass particulates, essentially all will be contained in the PM2.5 fraction, while the PM10 fraction will include additional particulates from resuspension of soil and ash. The highest concentrations are associated with indoor biomass combustion in developing countries and with exposures of wildland firefighters. These levels are 10 – 70 times above those observed in urban areas. Lower concentrations have been observed in ambient air within communities where wood burning is common and in plumes associated with large-scale tropical forest fires. These levels are 2 – 15 times those observed in urban areas. Domestic biomass burning in developing countries has also been associated with extremely high BaP levels (4000 times levels in urban air), while ten-fold lower exposures have been measured in wildland firefighters and even lower concentrations measured in community wood smoke (100 times urban air levels). Exposures to high concentrations of carbon monoxide are highly variable and only occasionally observed in wildland firefighters and in those exposed to domestic biomass smoke. Concentrations associated with tropical forest fires and community wood smoke are similar or slightly higher than those associated with motor vehicle emissions in urban areas. Large-scale tropical biomass fires are also associated with the production of ozone. Concentrations similar to those often measured in urban smog episodes have been measured in remote rural areas. Review of the exposure and health impacts literature, as well as initial evaluation of the available air monitoring data from the 1997 “haze” episode, indicates that the pollutant variable most consistently elevated in association with biomass smoke is particulate matter. Accordingly, the

emphasis throughout this manuscript and of recommended future studies will be focused on particulates.

## **IV. Acute and chronic health impacts**

### **A. Experimental and animal toxicology studies**

Many of the constituents present in wood smoke have been studied for their abilities to irritate mucous membranes and aggravate respiratory disease. Relatively few studies have evaluated the effects of whole wood smoke. Several studies have found an overall depression of macrophage activity as well as increases in albumin and lactose dehydrogenase levels, indicating damage to cellular membranes. Epithelial cell injury has also been demonstrated. A study in dogs indicated an increase in angiotensin-1-converting enzyme, a possible indication of an initial step towards pulmonary hypertension (Larson and Koenig 1994).

Two preliminary reports suggest that wood smoke exposure may lead to increased susceptibility to lung infections (Stone 1995). These observations lend support to epidemiological associations between wood smoke exposure and respiratory illnesses in young children, as discussed below. In one study, Mary Jane Selgrade of the U.S. EPA compared infectivity of *Streptococcus zooepidemicus* aerosols exposure in mice exposed previously to clean air, oil furnace emissions and wood smoke. The *Streptococcus zooepidemicus* causes severe respiratory infections. Two weeks post-exposure, 5% of the mice in the control and oil furnace groups died, compared to 26% of the wood smoke exposed group. Judith Zelikoff and colleagues at New York University exposed rats nasally to 800 µg/m<sup>3</sup> red Oak smoke for one hour. The rats were then exposed to *Staphylococcus aureus*, a respiratory pathogen. These bacteria were more virulent in rats exposed to the smoke relative to controls, although the rats' lungs did not show any signs of inflammation. The researchers suggested that the wood smoke suppressed macrophage activity.

These studies are best viewed as indications of plausibility for observed epidemiological associations and to help understand the mechanisms by which biomass smoke exposure may lead to adverse health outcomes. To demonstrate that adverse impacts of biomass smoke exposure in humans does occur, we will first evaluate population groups which are exposed to high levels, forest firefighters and developing country exposures where biomass is used for cooking and/or heating.

### **B. Epidemiological studies of non-cancer health risks**

Smoke from biomass is a complex mixture of particles and gases. Although the composition and concentrations of specific contaminants in smoke may vary by specific sources, an association between adverse health effects, particularly amongst children and the elderly, has been documented in numerous studies. Little is known about the toxicology of biomass smoke as a complex mixture, although of the constituents of biomass smoke, the epidemiological findings are most consistent with those found for particulate matter.



## 1. Wildland firefighters

Several studies have evaluated impacts of biomass smoke exposure on wildland (forest) firefighters. These studies are summarized in Table 2, and several are discussed in more detail in the following section.

A study of 76 firefighters in the U.S. Pacific Northwest evaluated cross-shift and cross-season respiratory effects. No significant increase or decrease in respiratory symptoms were observed across the firefighting season. The cross-shift and cross-season analysis identified significant mean individual declines in lung function. Although annual lung function changes for a small subset ( $n = 10$ ) indicated reversibility of effect, this study suggests a concern for potential adverse respiratory effects in forest firefighters. These firefighters worked an average of 15 fires during the season (Betchley, Koenig et al. 1997). Sutton et al, measured CO levels of 4-200 ppm and 24-hour TSP levels of approximately 0.5 mg/m<sup>3</sup> at a firefighter camp in California. Health assessment of the firefighters indicated a high prevalence of headaches (59%), cough (66%), shortness of breath (38%), lightheadedness (32%) and wheezing (31%) (Sutton, Castorina et al. 1988).

Letts and colleagues evaluated cross season changes in lung function and respiratory symptoms in 78 Southern California firefighters. Overall the mean cross season changes for lung function were -0.5% FEV1 (forced expiratory volume), 0.2% FVC (forced vital capacity) and -0.5% in the FEV1/FVC ratio. No significant increase in the prevalence of respiratory symptoms was noted cross seasonally and those which did occur were not associated with exposure. The authors conclude that there was limited evidence that forest fire fighting results in cross season changes in lung function, although the firefighters themselves indicated that the season of measurements contained fewer fires than was typical. (Letts, Fidler et al. 1991).

Rothman and colleagues studied cross-seasonal changes in pulmonary function and respiratory symptoms in 52 wildland firefighters in Northern California. The mean cross-seasonal change in FEV1 was -1.2%, with a corresponding mean change in FVC of -0.3%. Decreases in FEV1 and FVC were most strongly associated with hours of recent fire-fighting activity. When the study group was divided into three categories based on recent fire-fighting activity, firefighters in the high activity category (mean  $\pm$  SE, 73  $\pm$  7 hours of fire-fighting in previous week) had a -2.9% change in FEV1 and a -1.9% change in FVC. There was a significant cross-seasonal increase in most respiratory symptoms evaluated. Several symptoms (eye irritation, nose irritation, and wheezing) were associated with recent fire-fighting. These findings suggest that wildland firefighters experience a small cross-seasonal decline in pulmonary function and an increase in several respiratory symptoms (Rothman, Ford et al. 1991).

Liu and colleagues studies cross-season lung function and airways responsiveness in 63 wildland fire fighters during a 5-month season of active fire fighting. There were significant mean individual declines in postseason lung function, compared with preseason values. There was also a statistically significant increase in airway responsiveness when comparing preseason methacholine dose-response slopes with postseason dose-response slopes. The increase in airway responsiveness appeared to be greatest in fire fighters with a history of lower respiratory symptoms or asthma, but it was not related to smoking history. These data suggest that wildland

firefighting is associated with decreases in lung function and increases in airway responsiveness independent of a history of cigarette smoking (Liu, IRA B. Tager et al. 1992).

A recent study compared lung function of wildland firefighters in Sardinia with a control group of policemen, in an attempt to evaluate chronic impacts of firefighting exposure. On average, the firefighters worked during the 4 month fire season for 16 years. The firefighters had significantly lower levels of lung function (after controlling for age, height and smoking). Lung function measurements were conducted 10-11 months after the conclusion of the previous fire season. No relationship was observed between years of firefighting and lung function leading the authors to suggest that the adverse effect was due to repeated episodes of acute intoxication (Serra, Mocci et al. 1996).

### **Summary of wildland firefighter studies**

In summary, these studies clearly indicate an association between exposure and acute effects on respiratory health. Cross-seasonal effects have also been observed in most studies although these effects appear to be relatively small and may be reversible. As stated earlier, firefighters are an extremely fit and healthy group and cannot be considered representative of the general population. Accordingly the demonstration of acute and sub-chronic effects in this occupational group indicate the plausibility, but not the magnitude of an association between biomass smoke exposure and adverse effects in the general population.

## SUMMARY OF EPIDEMIOLOGICAL STUDIES

TABLE 2. Occupational exposures of Wildland Firefighters

<b>Study design</b>	<b>Endpoints measured</b>	<b>Results</b>	<b>Reference</b>
Longitudinal	Symptoms, lung function	Decreased cross-shift and cross-season lung function	(Betchley, Koenig et al. 1997)
Prevalence	Symptoms	High prevalence of headaches, lightheadedness, cough, shortness of breath, wheeze	(Sutton, Castorina et al. 1988)
Longitudinal	Symptoms, lung function	Slightly decreased cross-season lung function. No increase in symptoms	(Letts, Fidler et al. 1991)
Longitudinal	Symptoms, lung function	Increase in cross-season symptoms. Slight decrease in cross-season lung function. Increased symptoms associated with increased recent firefighting.	(Rothman, Ford et al. 1991)
Longitudinal	Lung function, airways responsiveness	Cross-season increase in airways responsiveness and decreased lung function	(Liu, IRA B. Tager et al. 1992)
Cross-sectional	Lung function	Decreased lung function in firefighters measured 11 months post-exposure relative to unexposed control group. No association between years of firefighting and lung function.	(Serra, Mocci et al. 1996)

## 2. Indoor air pollution in developing countries

The health effects of biomass smoke inhalation have been documented in developing countries where women, and in some cases, children spend many hours cooking over unvented indoor stoves. On a global basis it is the rural population in developing countries who are most highly exposed to fine particulates (Smith 1993). Approximately 50% of the world's population uses biomass fuel for cooking and/or heating.

The potential health effects associated with exposure to biomass combustion products in developing countries are widespread and have recently been reviewed (Smith 1993). In particular, exposure to biomass combustion products has been identified as a risk factor for ARI. ARI are the leading cause of infant mortality in the developing countries. In addition to the risks of infants, the women who are cooking are also at risk for chronic respiratory diseases as well as adverse pregnancy outcomes.

A number of studies have reported associations of health impacts with use of biomass fuels, although few have directly measured exposure. These studies have been reviewed in detail by Smith (Smith 1993) and (Chen, Hong et al. 1990) and are summarized in Table 3. Several of the more recent studies, including some in which exposures were measured will be discussed further. A case control study conducted in Zimbabwe found a significant association between lower respiratory disease and exposure to atmospheric wood smoke pollution in young children. Air sampling within the kitchens of 40 children indicated very high concentrations (546-1998  $\mu\text{g}/\text{m}^3$ ) of respirable particulates. Blood COHb was determined for 170 out of 244 children confirming that they did experience smoke inhalation (Collings, Sithole et al. 1990).

The association between exposure to air pollution from cooking fuels and health aspects was studied in Maputo, Mozambique. Personal air samples for particulate (roughly equivalent to PM10) were collected when four types of fuels (wood, charcoal, electricity, and liquified petroleum gas (LPG) were used for cooking. Wood users were exposed to significantly higher levels of particulate pollution during cooking time (1200  $\mu\text{g}/\text{m}^3$ ) than charcoal users (540  $\mu\text{g}/\text{m}^3$ ) and users of modern fuels (LPG and electricity) (200-380  $\mu\text{g}/\text{m}^3$ ). Wood users were found to have significantly more cough symptoms than other groups. This association remained significant when controlling for a large number of environmental variables. There was no difference in cough symptoms between charcoal users and users of modern fuels. Other respiratory symptoms such as dyspnea, wheezing, and inhalation and exhalation difficulties were not associated with wood use (Ellegard 1996). Lifetime exposure from cooking fuels was estimated by multiplying the exposure level (1200  $\mu\text{g}/\text{m}^3$  for wood) by years of exposure (23 for wood), duration of daily exposure (3 hours) and a use intensity factor (proportion of respondents using wood on the day of the measurement). The mean lifetime exposure variable was 2800 exposure years for those currently using wood as the principal fuel. For comparison to other studies, the wood exposure group had 69 hour-years of exposure.

A recent case-control study of Mexican women reported an increased risk of chronic bronchitis and obstructive airways disease associated with cooking with wood (Perez-Padilla, Regalado et al. 1996). The risk of chronic bronchitis was linearly associated with hour-years of cooking with

biomass. Crude odds ratio for chronic bronchitis and chronic bronchitis and obstructive airways disease with wood smoke exposure were 3.9 and 9.7, respectively. Adjusted odds ratios ranged from 1.6 – 8.3, and 1.1 – 2.0 for chronic bronchitis and obstructive airways disease, respectively, depending upon the specific control group used for comparison. The median duration of wood smoke exposure were 25 and 28 years for the chronic bronchitis and chronic bronchitis/obstructive airways disease groups, respectively. The median hours per day of wood smoke exposure was 3 in the case groups. Interestingly, the same research group conducted a cross-sectional study of Mexican women currently exposed to varying levels of biomass smoke indicated an association between biomass exposure and increased phlegm production and reduced lung function. Although these adverse effects were observed, they were smaller than expected based on the results of the case control study. Possible explanations include different study designs, bias in the case control study and the development of resistance in women repeatedly exposed (Regalado, Perez-Padilla et al. 1996).

A case-control study conducted in Colombia identified a similar risk of obstructive airways disease in women who cooked with biomass. Univariate analysis showed that tobacco use (OR = 2.22;  $p < 0.01$ ), wood use for cooking (OR = 3.43;  $p < 0.001$ ) and passive smoking (OR = 2.05;  $p = 0.01$ ) were associated with OAD. The adjusted odds ratio for obstructive airways disease and wood use (adjusted for smoking, gasoline and passive smoke exposure, age and hospital) was 3.92. The mean number of years of wood smoke exposure was 33 in the cases. The authors suggested that wood smoke exposure in these elderly women was associated with the development of OAD and may help explain around 50% of all OAD cases (Dennis, Maldonado D et al. 1996).

A recent clinical report describes a group of 30 non-smoking patients with lung disease thought to be associated with biomass smoke exposure during cooking. The patients had abnormal chest x-rays and evidence of pulmonary arterial hypertension. Their pulmonary function was consistent with mixed obstructive-restrictive disease (Sandoval, Salas et al. 1993).

Cassano and colleagues reported on a cross-sectional study of approximately 8000 individuals in rural areas of China with 58% wood use as domestic cooking fuel and 77% use of vented stoves. Vented stoves were associated with increased lung function and time spent cooking was related to decreased lung function. County-wide COPD mortality data were inversely related to lung function data. These findings were similar for all fuel types and suggest a link between wood use as a cooking fuel and COPD (Cassano, Hu et al. 1994).

### **Summary of studies of indoor air pollution in developing countries**

Studies in developing countries indicate that biomass smoke exposure is associated with both acute respiratory illness in children and the development of chronic lung disease in adults. As these exposures are much higher than would occur as a result of short-term exposure to biomass air pollution associated with forest fires, direct comparisons are difficult to make. More so than with studies of wildland firefighters, the studies conducted in developing countries indicate the serious consequences of exposure to high levels of biomass air pollution. Increased acute respiratory illness in children associated with biomass smoke exposure is a likely cause of infant mortality while the development of chronic lung disease in adults is associated with premature mortality and substantial morbidity.



## SUMMARY OF EPIDEMIOLOGICAL STUDIES

TABLE 3. Indoor exposure (high level)

<b>Population</b>	<b>Study design</b>	<b>Endpoints measured</b>	<b>Results</b>	<b>Reference</b>
Children in Papua New Guinea	Cross-sectional	Symptoms	Increased cough and rhinitis in high exposure group. Increased wheeze in low exposure group.	(Anderson 1978)
Children in Papua New Guinea	Prospective	Symptoms	No difference in symptoms between the two exposure groups	(Anderson 1978)
Adult women in Papua New Guinea	Cross-sectional	Lung function	10% of women > 45 years had FEV1/FVC < 60%. No control group	(Anderson 1979)
Children in South Africa	Cross-sectional	Respiratory illness	Increased serious lower respiratory illness in exposed group	(Kossove 1982)
Adults in Nepal	Cross-sectional	Respiratory illness	Increased chronic bronchitis prevalence with increasing hours of exposure	(Pandey 1984)
Children in Nepal	Cross-sectional	Respiratory illness	Increased severe respiratory illness with increased hours of exposure	(Pandey, Neupane et al. 1989)
Children in Malaysia	Cross-sectional	Lung function	Decreased lung function with home wood stove	(Azizi and Henry 1990)
Children in Malaysia	Cross-sectional	Symptoms	Slight increase in cough and phlegm prevalence in exposed group	(Azizi and Henry 1991)
Children in Kenya	Cross-sectional	Respiratory illness	No increase in illness rates for exposed children	(Wafula, Onyango et al. 1990)
Children in The Gambia	Cross-sectional	Respiratory illness	Increased acute respiratory infection risk in girls exposed while carried on mothers' back. No effect in boys.	(Armstrong and Campbell 1991)

TABLE 3. Indoor exposure (high level) (continued)

<b>Population</b>	<b>Study design</b>	<b>Endpoints measured</b>	<b>Results</b>	<b>Reference</b>
Children in Zimbabwe	Cross-sectional	Respiratory illness	Increased lower respiratory illness with wood smoke exposure (blood COHb)	(Collings, Sithole et al. 1990)
Adult, non-smoking women in India	Cross-sectional	Lung function	Reduced FEV1/FVC with increased exposure (expired CO)	(Norboo, Yahya et al. 1991)
Adult women in Mexico	Case series	COPD	COPD in non-smoking women	(Sandoval, Salas et al. 1993)
Adults in China	Cross-sectional	Lung function	Increased lung function in adults with vented stoves. Decreased lung function with time spent cooking. County-wide COPD mortality highest in counties with lowest lung function.	(Cassano, Hu et al. 1994)
Adult women in Mexico	Case control	COPD	COPD in non-smoking women	(Perez-Padilla, Regalado et al. 1996)
Adult women in Mexico	Cross-sectional	Symptoms, lung function	Slightly reduced lung function and increased cough and phlegm in women with highest PM10 exposure.	(Regalado, Perez-Padilla et al. 1996)
Adult women in Mozambique	Cross-sectional	Symptoms	Increased cough symptoms in wood smoke exposed group (relative to charcoal, gas, electric). No increase in other respiratory symptoms (wheeze, difficulty breathing, etc.)	(Ellegard 1996)
Adult women in Colombia	Case-control	COPD	COPD in non-smoking women	(Dennis, Maldonado D et al. 1996)



### 3. Community/cohort indoor and ambient air pollution studies

#### a) Particulates - overview

This paper will not discuss the voluminous particulate epidemiology literature in detail as a book (Wilson and Spengler 1996) and several review articles have recently been published (Dockery and Pope 1994) (Pope, Bates et al. 1995) (Vedal 1997). Instead we will present an overview of findings, discuss examples of the major study types and their findings, with emphasis on the time series studies of acute health impacts and the recent prospective cohort studies of chronic exposure impacts. We will then discuss in detail the available evidence associating biomass air pollution with adverse health outcomes.

Early air pollution disasters, such as the London Fog of 1952, were dramatic examples of the impact of air pollution on mortality and other health effects (Logan 1953; Schwartz and Marcus 1990). These air pollution episodes were the motivation for regulations and consequent air quality improvements in the past 30-40 years. However, recent studies have indicated that current levels of air pollution are associated with adverse health outcomes. The most startling finding of these studies is the association of particulate air pollution, with increased daily mortality (Dockery, Pope et al. 1993) (Schwartz 1991) (Dockery, Schwartz et al. 1992; Pope, Schwartz et al. 1992; Schwartz and Dockery 1992; Schwartz and Dockery 1992; Dockery, Pope et al. 1993; Schwartz 1993; Spix, Heinrich et al. 1993; Pope, Thun et al. 1995). These studies have been conducted by different investigators in a variety of locations, using a variety of study designs. In nearly all cases, the studies indicate an association between particle air pollution and increased risk of death, primarily in the elderly and in individuals with pre-existing respiratory and/or cardiac illness (Schwartz 1994; Schwartz 1994). Recent studies have also suggested an association between particulates and infant mortality (Bobak and Leon 1992) (Woodruff, Grillo et al. 1997) as well as with low birth weight (Wang, Ding et al. 1997). Increased risk of hospital admissions and increased emergency room visits have also been associated with short-term increases in the levels of particle air pollution (Pope 1989; Pope 1991; Schwartz, Slater et al. 1993; Dockery and Pope 1994; Schwartz 1994; Schwartz 1994; Schwartz 1995; Schwartz 1996). Table 4 summarizes the results of these studies for the various health outcomes assessed. One common feature of the study locales is that the ambient particulate are produced in combustion processes. Studies of naturally-produced particles (such as those generated from windblown soil or volcanic eruptions) show a much smaller impact on health outcomes for an equivalent particle concentration (Hefflin, Jalaludin et al. 1994) (Buist, Johnson et al. 1983) (Dockery and Pope 1994). These data support the hypothesis that any combustion-source particulate air pollution is associated with adverse health outcomes. The implications of this hypothesis are far-reaching, as they suggest that particulates are associated with adverse health effects in essentially all urban areas.

**TABLE 4. Combined effect estimates of daily mean PM<sub>10</sub>**

	<b>% Change per each 10 µg/m<sup>3</sup> increase in PM<sub>10</sub></b>
<b>Increase in daily mortality</b>	
Total deaths	1.0
Respiratory deaths	3.4
Cardiovascular deaths	1.4
<b>Increase in hospital usage (all respiratory)</b>	
Admissions	.8
Emergency department visits	1.0
<b>Exacerbation of asthma</b>	
Asthmatic attacks	3.0
Bronchodilator	2.9
Emergency department visits*	3.4
Hospital admissions	1.9
<b>Increase in respiratory symptom reports</b>	
Lower respiratory	3.0
Upper respiratory	0.7
Cough	1.2
<b>Decrease in lung function</b>	
Forced expiratory volume	0.15
Peak expiratory flow	0.08

From (Dockery and Pope 1994)

The majority of the particulate epidemiology studies have evaluated the acute impacts of particulate air pollution with time-series study designs. Only a limited number of studies have investigated long-term effects. Of these, the most significant are the prospective cohort studies in which the analyses can control for individual differences in risk factors such as smoking. Dockery, et al, studied over 8,000 adults in 6 cities with different levels of air pollution over a period of 16 years. The adjusted mortality risk was 26% higher in the most polluted city relative to the least polluted city. Survival decreased with increasing particulate levels (Dockery, Pope et al. 1993). In a study of more than 500,000 adults with 8-year follow-up, Pope and colleagues found a significant association between fine particles and particle sulfate with cardiopulmonary mortality after controlling for smoking, education and other potential confounding factors (Pope, Thun et al. 1995). The adjusted mortality risk was 15 – 25% higher in cities with the highest particulate levels relative to the cities with the lowest levels. Together these studies indicate that long term exposure to particulate air pollution has a significant impact on survival. When viewed in

conjunction with the numerous studies indicating an association between particulates and acute mortality, the literature suggests that the acute effect of particulates is to significantly reduce survival, rather than to simply advance deaths which would have otherwise occurred by several days. The results of the cohort studies of Dockery and Pope were used to estimate the reduction in life expectancy associated with long-term particulate exposure. For a  $10 \mu\text{g}/\text{m}^3$  difference in long-term exposure to  $\text{PM}_{2.5}$ , the relative risk of mortality is 1.1. When applied to a 1992 life table for Dutch men, the estimated effects of life expectancy is 1.1 years for each  $10 \mu\text{g}/\text{m}^3$  difference in long-term exposure to  $\text{PM}_{2.5}$  [World Health Organization, 1995 #135].

In the only cohort study of morbidity associated with long-term particulate exposure, Abbey and colleagues studied a cohort of nearly 4000 non-smoking Seventh Day Adventists in California. The relative risks of developing new cases of chronic respiratory disease were significantly associated with particulate levels. Figure 2 indicates the relative risk of developing obstructive airways symptoms at different levels of average annual exposure for a 4 year period. For TSP levels above  $100 \mu\text{g}/\text{m}^3$  (in this case similar risks were observed for  $\text{PM}_{10}$  levels above  $80 \mu\text{g}/\text{m}^3$ ), significantly elevated risks were observed for as few as 500 hours (21 days) of exposure per year, for 4 years (Abbey, Mills et al. 1991; Abbey, Moore et al. 1991). Increased risks were observed for longer duration and higher levels of exposure. A significant risk for the development of asthma was associated with long-term exposure to TSP above  $150 \mu\text{g}/\text{m}^3$ . Similar analyses were also conducted for estimated and measured concentrations of  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ . While the risk of asthma development was not evident in these analyses, significant risks for the development of new cases of chronic bronchitis and obstructive airways disease were found annual average  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  levels of 20 -  $100 \mu\text{g}/\text{m}^3$  (Abbey, Ostro et al. 1995). Increased symptoms severity was associated with annual average concentrations of 20 -  $40 \mu\text{g}/\text{m}^3$  and 40-50  $\mu\text{g}/\text{m}^3$  for  $\text{PM}_{2.5}$  and  $\text{PM}_{10}$ , respectively.

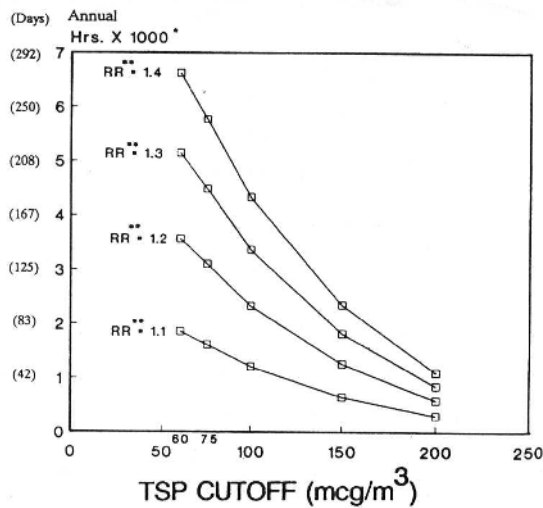


Fig. 2. Relative risk contour plots for new cases of definite symptoms of airway obstructive disease associated with total suspended particulates (\* Average annual hours, 1973-1977. \*\* Relative risks for cutoffs of 60 and  $75 \mu\text{g}/\text{m}^3$  are not statistically significant.)

FIGURE 2 (Abbey, Petersen et al. 1993)

While the vast majority of studies have measured PM<sub>10</sub>, there is evidence to support regulating PM<sub>2.5</sub> levels. Schwartz compared PM<sub>2.5</sub> and the coarse fraction of PM<sub>10</sub> (PM<sub>10</sub> – PM<sub>2.5</sub>) as indicators of mortality. A significant relationship between PM<sub>2.5</sub> and mortality, but not with coarse particles, was found (Schwartz, Dockery et al. 1996). These results are also consistent with our understanding of particle deposition since coarse particles are efficiently removed in the upper respiratory tract, while fine particles penetrate deep into the lung. A recent analysis of insoluble particles in autopsy lungs found that 96% of the particles were smaller than 2.5 µm (Churg and Brauer 1997). The PM<sub>2.5</sub> fraction also contains primarily particles produced in combustion processes, while the coarse fraction contains soil and crustal material that is not as toxicologically reactive.

Currently inhalable particulates are regulated in many countries. Since 1987, the U.S. EPA standard has been 150 µg/m<sup>3</sup> and 50 µg/m<sup>3</sup> for 24-hours and one year, respectively. California has set a standard of 50 and 30 µg/m<sup>3</sup> for 24 hour and annual averages, respectively. The WHO Air Quality Guidelines for Europe declined to recommend specific guidelines for particulate matter as the available studies do not indicate an obvious exposure concentration and duration that could be judged a threshold [World Health Organization, 1995 #135]. The document argues that the available data suggest a continuum of effects with increasing exposure. Recently the U.S. EPA set the first standard for PM<sub>2.5</sub> as 65 µg/m<sup>3</sup> and 15 µg/m<sup>3</sup> for 24 hour and annual averages, respectively.

#### **b) wood and other biomass smoke**

Epidemiological studies of wood smoke in North America have focused on symptoms and/or lung function as the main outcome measures. The majority of studies have focused on children, due to the assumption that children are susceptible due to small lung volumes and incompletely developed immune systems. Children are also somewhat simpler to study, as cigarette smoking or occupation does not confound their exposures.

Several early studies focused on the presence of a wood burning stove in the home as a risk factor. Several studies indicate that wood stoves, especially older varieties can emit smoke directly into the home (Larson and Koenig 1994). Newer airtight stoves emit less smoke into the homes, but indoor exposure still occurs due to infiltration of smoke emitted outdoors back into the home. Therefore, while these earlier studies strongly suggest that there are adverse impacts associated with wood smoke exposure, their crude exposure assessment precludes more specific conclusions. The Harvard University Six Cities Study reported that wood stove use was associated with an increased risk of respiratory illness in children [Dockery, 1987 #117]. Honicky and colleagues studied 34 children living in homes with wood stoves compared to 34 with other heating sources, mainly gas. Occurrence of wheeze and cough was much greater in the group of children living in homes with wood stoves, although no measurements were made of wood smoke (Honicky-RE 1985). The study of Honicky was motivated by a case report of a 7 month old infant hospitalized with serious respiratory disease, which was associated with the family's purchase of a wood burning stove (Honicky-RE 1983). Another clinical case report strongly argues for the biological relationship between wood smoke exposure and lung disease, in this case, interstitial disease, and not the obstructive lung disease commonly associated with biomass smoke exposure in developing countries (Pandey 1984). Ramage and colleagues reported on the case of a 61-year-old woman with interstitial lung disease. Bronchoalveolar

lavage revealed numerous particulates and fibers, as well as cellular and immunoglobulin abnormalities. The particles were shown to be carbonaceous by energy dispersive X-ray analysis (EDXA). Inflammation and fibrosis were found surrounding them on open biopsy. The particle source was traced to a malfunctioning wood-burning heater in the patient's home (Ramage, Roggli et al. 1988).

Tuthill and colleagues measured respiratory symptoms and disease prevalence in 258 children living in homes with wood stoves compared to 141 children in homes without wood stoves. A slight, but not statistically significant elevated risk of symptoms was found in this study. No exposures were measured (Tuthill 1984). Butterfield and colleagues monitored 10 respiratory disease symptoms in 59 1 – 5 ½ year old children, again comparing those living in homes with and without wood stoves. Wheeze and cough symptoms were associated with living in a home with a wood stove. Although no measurements were made of wood smoke exposure, a study conducted during the following winter indicated monthly mean outdoor PM levels of approximately 50-65 µg/m<sup>3</sup> while source apportionment studies indicated that approximately 70% of the winter particulate was from wood burning (Cupitt, Glen et al. 1994). A case control study of 59 matched pairs of native American children less than 2 years old indicated increased risk of lower respiratory tract infection for children living in homes with wood stoves (Morris, Morgenlander et al. 1990).

A similar case-control study conducted among Navajo children evaluated the association between wood smoke exposure and acute lower respiratory illness (ALRI). In a significant improvement from earlier studies, indoor particulate levels were measured in this investigation. Forty-five 1 – 24 month old children hospitalized with an ALRI were compared with age and gender matched controls who had a health record at the same hospital and had never been hospitalized for ALRI. Home interviews of parents of subjects elicited information on heating and cooking fuels and other household characteristics. Indoor PM<sub>10</sub> sampling was conducted in the homes of all cases and controls. Matched pair analysis revealed an increased risk of ALRI for children living in households that cooked with any wood or had indoor particle concentrations greater than or equal to 65 µg/m<sup>3</sup>. The indoor particle concentration was positively correlated with cooking and heating with wood (geometric mean levels of approximately 60 µg/m<sup>3</sup>) but not with other sources of combustion emissions (Robin 1996). In the only study to date to evaluate impacts of wood burning on adult asthma, Ostro and colleagues measured symptoms in a panel of 164 asthmatics. Exposure to indoor combustion sources, including wood stoves was associated with increased asthma exacerbation (Ostro, Lipsett et al. 1994). The studies of low-level indoor exposure are summarized in Table 5.

Several other studies, summarized in Table 6, have evaluated health outcomes in communities where wood smoke is a major, although not the only, source of ambient particulate. Heumann studied lung function in 410 school children in Klamath Falls Oregon during a winter season. Children from schools in high and low exposure areas were studied. In Klamath Falls, it has been estimated that wood smoke accounts for as much as 80% of winter period PM<sub>10</sub>. Winter period PM<sub>10</sub> levels in the high exposure area ranged from approximately 50 – 250 µg/m<sup>3</sup> while levels in the low exposure area ranged from 20 – 75 µg/m<sup>3</sup>. Lung function decreased during the wood burning season for the children in the high exposure area, but not in the low exposure area.(Heumann, Foster et al. 1991) Two studies were conducted in Montana to evaluate acute

changes in lung function in children within a single community at different levels of air pollution, and also to evaluate cross-sectional differences in lung function between communities with different air quality levels, as an indication of chronic impacts (Johnson, Gideon et al. 1990). Acute lung function decrements measured in 375 children were associated with increased levels of particulates. 24-hour averages ranged from 43-80  $\mu\text{g}/\text{m}^3$  and 14-38  $\mu\text{g}/\text{m}^3$  for PM10 and PM2.5, respectively. The chronic impact study also associated small decrements in lung function with residence in communities with higher levels of air pollution. Although particle composition was not measured directly in this study, measurements conducted in the acute study community during the same period, attributed 68% of the PM3.5 to wood smoke (Larson and Koenig 1994).

A questionnaire study of respiratory symptoms compared residents of 600 homes in a high wood smoke pollution area of Seattle with 600 homes (questionnaires completed for one parent and two children in each residence) of a low wood smoke pollution area. PM10 concentrations averaged 55 and 33  $\mu\text{g}/\text{m}^3$  in the high and low exposure areas, respectively. When all age groups were combined, no significant differences were observed between the high and low exposure areas. However, there were statistically significant higher levels of congestion and wheezing in 1-5 year olds between the two areas for all three questionnaires (1 baseline questionnaire and two follow-up questionnaires which asked about acute symptoms). This study, supports the other investigations suggesting that young children are particularly susceptible to adverse effects of wood smoke (Browning, Koenig JQ et al. 1990).

A more comprehensive study in the same high exposure Seattle area was initiated in 1988. In these residential areas in Seattle, 80% of the particle are from wood smoke (Larson and Koenig 1994). Lung function was measured in 326 (including 24 asthmatics) elementary school children before, during and after two wood burning seasons. Fine particulates were measured continuously with an integrating nephelometer. Significant lung function decrements were observed in the asthmatic subjects, in association with increased wood smoke exposure. The highest (nighttime 12-hour average) PM2.5 level measured during the study period was approximately 195  $\mu\text{g}/\text{m}^3$  and PM10 levels were below the U.S. National Ambient Air Quality Standard of 150  $\mu\text{g}/\text{m}^3$  during the entire study period (Koenig, Larson TV et al. 1993). For the asthmatic children FEV1 and FVC decreased by 17 and 18.5 ml for each 10  $\mu\text{g}/\text{m}^3$  increase in PM2.5, while no significant decreases in lung function were observed in the non-asthmatic children. A companion study evaluated the impact of particulate matter on emergency room visits for asthma in Seattle (Schwartz, Slater et al. 1993). In this study a significant association was observed between PM10 particle levels and emergency room visits for asthma. The mean PM10 level during the 1-year study period was 30  $\mu\text{g}/\text{m}^3$ . At this concentration, PM10 appeared to be responsible for 125 of the asthma emergency room visits. An exposure response relationship was also observed down to very low levels of PM10, with no evidence for a threshold at concentrations as low as 15  $\mu\text{g}/\text{m}^3$ . The authors indicate that on an annual basis 60% of the fine particle mass in Seattle residential neighborhoods is from wood burning.

Two time series studies have been conducted in Santa Clara County, California, an area in which wood smoke is the single largest contributor to winter PM10, accounting for approximately 45% of winter PM10. Particulate levels are highest during the winter in this area. The first study was one of the initial mortality time series studies which indicated an association between relatively low PM10

levels and increased daily mortality (Fairley 1990). A recent study of asthma emergency room visits in Santa Clara County and winter PM10 found a consistent relationship. Specifically, a 10  $\mu\text{g}/\text{m}^3$  increase in PM10 was associated with a 2 – 6 % increase in asthma emergency room visits (Lipsett, Hurley et al. 1997). These results demonstrate an association between ambient wintertime PM10 increased daily mortality and exacerbations of asthma in an area where one of the principal sources of PM10 is residential wood smoke.

Two other studies conducted in North America focused on other sources of biomass burning besides wood burning stoves or fireplaces. In a small study of 7 medicated asthmatics, subjects were walked 0.5 mile with and without exposure to burning leaves on the same day, under very similar environmental conditions. Significant decreases in lung function were observed in the asthmatics within 30 min of exposure to leaf burning, while lung function of non-asthmatics was not affected (From, Bergen et al. 1992). In a recent study, 428 subjects with moderate to severe airways obstruction were surveyed for their respiratory symptoms during a 2-week period of exposure to combustion products of agricultural burning (straw and stubble). During the exposure period, 24-hour average PM10 levels increased from 15-40  $\mu\text{g}/\text{m}^3$  to 80-110  $\mu\text{g}/\text{m}^3$ . 1-hour level of carbon monoxide and nitrogen dioxide reached 11 ppm and 110 ppb, respectively. Total volatile organic compound levels increased from 30-100  $\mu\text{g}/\text{m}^3$  before the episode to 100-460  $\mu\text{g}/\text{m}^3$  during the episode. While 37% of subjects were not bothered by smoke at all, 42% reported that symptoms (cough, wheezing, chest tightness, shortness of breath) developed or became worse due to the air pollution episode and 20% reported that they had breathing trouble. Those with symptoms were more likely to be female than male and were more likely to be ex-smokers than smokers. Subjects with asthma and chronic bronchitis were also more likely affected (Long, Tate et al. 1998). The results of these studies indicate that other forms of biomass air pollution, in addition to wood smoke, are associated with some degree of impairment, and suggest that individuals with pre-existing respiratory disease are particularly susceptible.

Perhaps the study with the most relevance to the issue of biomass air pollution in Southeast Asia is an analysis of emergency room visits for asthma in Singapore during the 1994 “haze” episode (Chew, Ooi et al. 1995). The study, described briefly in a letter to *Lancet*, indicates an association between PM10 and emergency room visits for childhood asthma. During the “haze” period, mean PM10 levels were 20% higher than the annual average. Although a time series analysis was not conducted, the authors suggest that the association remained significant for all concentrations above 158  $\mu\text{g}/\text{m}^3$ .

Two studies have been conducted regarding asthma emergency room visits and PM10 levels associated with smoke from bushfires in Sydney Australia (Copper, Mira et al. 1994; Smith, Jalaludin et al. 1996). During 1994, PM10 levels were elevated (maximum hourly values of approximately 250  $\mu\text{g}/\text{m}^3$ ) for a 7-day period. Ozone levels were not elevated during the period in which smoke impacted Sydney. Neither study detected any increase in asthma emergency room visits during the bushfire smoke episode. Both studies used relatively simple analyses. One study had little power to detect small changes in emergency room visits as they related to air pollution and the other only used relatively short periods for comparison, neither detected any association. These results appear to conflict with results of studies conducted in North America. Possible reasons are differences in study design and sample size as well as differences in

chemical composition of the particulates and differences in the relative toxicity of the specific particle mixture.

In a similar analysis to the studies of Australian bushfires, Duclos and colleagues evaluated the impact of a number of large forest fires in California on emergency room visits (Duclos, Sanderson et al. 1990). During the approximately 2 ½ week period of the fires, asthma and chronic obstructive pulmonary disease visits increased by 40 and 30%, respectively. PM10 concentrations as high as 237 µg/m<sup>3</sup> were measured. Based on TSP concentrations, PM10 levels were significantly higher in other regions without PM10 monitoring.

### **Summary of wood and biomass community/cohort studies**

The epidemiological studies of indoor and community exposure to biomass smoke indicates a consistent relationship between exposure and increased respiratory symptoms, increased risk of respiratory illness and decreased lung function. These studies have mainly been focused on children, although the few studies which evaluated adults also showed similar results. A limited number of studies also indicate an association between biomass smoke exposure and visits to hospital emergency rooms. A notable exception is the analysis of populations exposed to large bushfires in Australia which did not show any association between PM10 and asthma emergency room visits. There are also indications from several studies that asthmatics are a particularly sensitive group. No studies have explicitly evaluated the effect of community exposure to biomass air pollution on hospitalizations or mortality, although one study indicated a relationship between PM10 and mortality in an area where wood smoke is a major contributor to ambient PM10. By analogy to the findings of numerous studies associating increased mortality with urban particulate air pollution mixtures it is reasonable to conclude that similar findings would also be observed in locations exposed to biomass smoke. From the vast number of particulate studies, there is no evidence that airborne particles from different combustion sources have different impacts on health. Particles generated by natural processes such as volcanic eruptions and windblown soil do appear to have less of an impact on health. Therefore, there is little reason to expect that biomass smoke particulate would be any less harmful than other combustion-source particles and it is prudent to consider that biomass smoke exposure is also related to increased mortality. The particulate mortality studies also do not show evidence for a threshold concentration at which effects are not observed. If such a threshold level does exist it is likely to be at a very low level, below those levels measured in most urban areas of the world.

Nearly all of the low-level indoor and community biomass smoke studies mentioned above evaluated impacts of concentrations which were much lower than those associated with the 1997 Southeast Asian haze episode. Similarly, the studies of seasonal exposure to wood smoke involved exposure durations which were of comparable length to those experienced in Southeast Asia. Based on these studies it is reasonable to expect that the Southeast Asian haze episode resulted in the entire spectrum of acute impacts, including increased mortality, as well as sub-chronic (seasonal) effects on lung function, respiratory illness and symptoms. It is not possible at this time to determine the long term effect, if any, from a single air pollution episode, although repeated yearly occurrences of high biomass smoke exposure should be cause for serious concern. Chronic (several years) exposure to particulate air pollution in urban areas, at much lower levels than experienced in Southeast Asia in 1997, has been associated with decreased life expectancy and with the development of new cases of chronic lung disease.



## SUMMARY OF EPIDEMIOLOGICAL STUDIES

TABLE 5. Indoor exposure (low level)

<b>Population</b>	<b>Study design</b>	<b>Endpoints measured</b>	<b>Results</b>	<b>Reference</b>
Children	Cross-sectional	Symptoms	No association between respiratory illness and home wood burning	(Tuthill 1984)
Children	Cross-sectional	Symptoms	Increased cough, wheeze, allergic symptoms with home wood burning	(Honicky-RE 1985)
Children	Cross-sectional	Symptoms, respiratory illness	increased history of chest illness in past year with home wood burning; no effect on symptoms	(Dockery, Spengler et al. 1987)
Children	Longitudinal	Symptoms	increased frequency of wheeze and cough with increased hours of wood stove use	(Butterfield, LaCava et al. 1989)
Children < 2 years	Longitudinal	Respiratory illness	increased risk of lower respiratory with wood burning	(Morris, Morgenlander et al. 1990)
Children	Cross-sectional	Symptoms, respiratory illness, lung function	No increased symptoms or illness and no decreased lung function with home wood burning	(Vedal 1993)
Children	Case-control	Hospitalization for respiratory illness	increased hospitalization with home wood burning – results dependent upon control group	(Vedal 1993)
Adult asthmatics	Longitudinal	Symptoms	increased cough, shortness of breath on days with home wood burning	(Ostro, Lipsett et al. 1994)
Children < 2 years	Case-control	Respiratory illness	Increased acute respiratory illness in wood burning homes with PM10 > 65 µg/m <sup>3</sup>	(Robin 1996)

SUMMARY OF EPIDEMIOLOGICAL STUDIES

TABLE 6. Ambient exposure

<b>Population</b>	<b>Study design</b>	<b>Endpoints measured</b>	<b>Results</b>	<b>Reference</b>
All ages > 1	Cross-sectional	Symptoms, respiratory illness	No significant effects. Trend for children 1-5	(Browning, Koenig JQ et al. 1990)
Children	Longitudinal	Lung function	Decreased lung function during and after wood burning season in exposed community but not in control community	(Heumann, Foster et al. 1991)
Children	Longitudinal	Lung function	Decreased winter lung function in exposed community but not in control community	(Johnson, Gideon et al. 1990)
Children	Longitudinal	Spirometry	Decreased lung function and fine particles in asthmatics	(Koenig, Larson TV et al. 1993)
All ages	Longitudinal	Emergency room visits	Increased asthma visits with fine particles in areas where wood smoke accounts for 80% of PM2.5	(Schwartz, Slater et al. 1993)
All ages	Longitudinal	Emergency room visits	Increased asthma visits with PM10 in area where wood smoke accounts for 45% of winter PM10	(Lipsett, Hurley et al. 1997)
All ages	Longitudinal	Mortality	Increased daily mortality with PM10 in area where wood smoke accounts for 45% of winter PM10	(Fairley 1990)
All ages	Longitudinal	Emergency room visits	Increased respiratory visits in community exposed to fire smoke	(Duclos, Sanderson et al. 1990)
Adult asthmatics	Experimental	Lung function	Decreased lung function following exposure to burning leaves in asthmatics, but not in non-asthmatics	(From, Bergen et al. 1992)
Adults with airways obstruction	Prevalence	Symptoms	42% of population reported increased or worsened symptoms during episode of exposure to agricultural burning emissions. 20% reported breathing trouble	(Long, Tate et al. 1998)

TABLE 6. Ambient exposure (continued)

<b>Population</b>	<b>Study design</b>	<b>Endpoints measured</b>	<b>Results</b>	<b>Reference</b>
All ages	longitudinal	Emergency room visits	Increased asthma visits with PM10 during episode of exposure to biomass burning emissions in Singapore	(Chew, Ooi et al. 1995)
All ages	longitudinal	Emergency room visits	No increase in asthma visits with PM10 during episode of exposure to bushfire emissions in Australia	(Copper, Mira et al. 1994)
All ages	longitudinal	Emergency room visits	No increase in asthma visits with PM10 during episode of exposure to bushfire emissions in Australia	(Smith, Jalaludin et al. 1996)

### C. Cancer

Pope and Cohen recently reviewed the evidence associating air pollution with lung cancer. Studies suggest rather consistently that ambient air pollution resulting from fossil fuel combustion is associated with increased rates of lung cancer. Two recent prospective cohort studies observed 30-50% increases in lung cancer rates associated with exposure to respirable particulates, best viewed as a complex mixture originating from diesel exhaust, coal, gasoline and wood. One of these studies suggested that sulfate particles (likely originating from coal and diesel combustion) appeared to be more strongly associated with lung cancer than fine particles. The results of these cohort studies are generally consistent with other types of exposure to combustion-source pollution such as occupational exposure and environmental tobacco smoke. While biomass smoke may be similar in some respects to cigarette smoke, the excess lung cancer risk associated with ambient air pollution (relative risks of 1.0 – 1.6) is small compared with that from cigarette smoking (relative risks of 7 – 22) but comparable to the risk associated with long term environmental tobacco smoke exposure (relative risk of 1.0 – 1.5).

There is little direct information regarding the human cancer risks associated with biomass air pollution. The U.S. EPA studied the contribution of wood smoke and motor vehicle emissions to the mutagenicity of ambient aerosols in Albuquerque, NM. This study found that, despite wood smoke being the major contributor to the mutagenicity of ambient particulate matter, it was 3 times less potent as a mutagen than extractable organics associated with vehicle emissions (Lewis, Baumgardner et al. 1988). The mutagenic potency of air samples decreased linearly with increased fraction of samples originating from wood smoke.

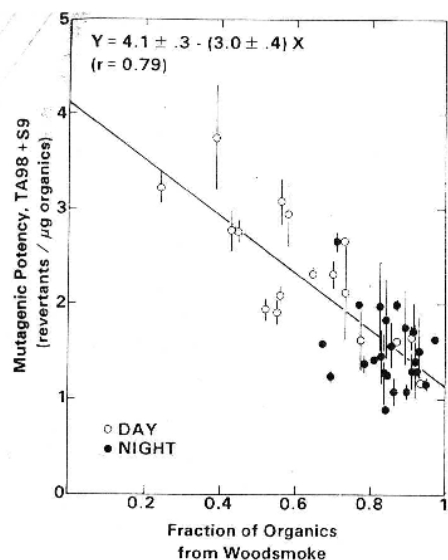


Figure 1. Mutagenic potency (*S. typhimurium* TA98 with S9 exogenous activation) of dichloromethane-extractable organics in ambient fine-particle samples versus fraction of extractable organics due to woodsmoke. Error bars are equal to half the difference in duplicate bioassay results.

FIGURE 3 (Lewis, Baumgardner et al. 1988)

In an application of this and other work, the estimated lifetime cancer risk associated with 70 years of exposure to air pollution dominated by wood smoke (80%) was calculated to be approximately 1 in 2,000. This calculation assumes lifetime exposure to PM10 levels of 25-60 µg/m<sup>3</sup> of which wood smoke is a major component for approximately 3 months every year. Extrapolating these estimates to an environment of 100% wood smoke estimates an individual lifetime cancer risk of approximately 1 in 10,000 (Cupitt, Glen et al. 1994)). It must be emphasized that these risk estimates do not mean that out of every 10,000 exposed people, 1 will develop cancer, but rather serve as estimates upon which different exposure scenarios and pollutants can be compared and to evaluate whether certain exposures can be considered as significant risks. For environmental exposure, regulatory agencies often consider lifetime cancer risks greater than 1 in 1 million to be significant.

BaP is a probable human carcinogen as defined by the International Agency for Research on Cancer (IARC) and is present in biomass smoke at high levels. Smith and Liu review studies of BaP levels in biomass smoke and discuss studies which have evaluated lung cancer risks (Smith and Liu 1993). Despite high exposures to a known human carcinogen, there is relatively little evidence for a relationship between lung cancer and biomass smoke exposure. If any effect does exist it is thought to be small, relative to other risk factors such as diet or exposure to air pollution from coal burning. Lung cancer is itself relatively rare in areas of biomass fuel use, even if age-adjusted cancer rates are analyzed (Smith and Liu 1993). A similar argument is presented for nasopharyngeal cancers, which are also rare in areas of biomass smoke use [Dekoning , 1985 #225].

The findings of relatively low mutagenicity for wood smoke, have, to some extent, been validated in an ongoing study of indoor environmental exposure risks and lung cancer in China. Cross-sectional comparisons of population subgroups in Xuan Wei, China, an area noted for high mortality from respiratory disease and lung cancer, suggested that the high lung cancer rates cannot be attributed to smoking or occupational exposure. Since residents of Xuan Wei, especially women, are exposed to high concentrations of coal and wood combustion products indoors, a study was undertaken to evaluate the lung cancer risks of these exposures. On average women and men in Xuan Wei spend 7 and 4 hours per day, respectively, near a household fire. A 1983 survey indicated that the lung cancer rate in Xuan Wei was strongly associated with the proportion of homes using smoky coal in 1958 (Mumford 1990). No relationship was observed between lung cancer and the percentage of homes using wood.

A follow-up study compared exposures in two otherwise similar Xuan Wei communes, one with high lung cancer mortality (152/100,000) where smoky coal was the major fuel, and another with low lung cancer mortality (2/100,000) where wood (67%) and smokeless coal (33%) were used. Lung cancer mortality was strongly associated with indoor burning of smoky coal and not with wood burning. This association was especially strong in women who had low smoking and were more highly exposed to cooking fuel emissions than men (Chapman, Mumford et al. 1988). Indoor PM10 concentrations measured during cooking were extremely high (24, 22 and 1.8 mg/m<sup>3</sup> for smoky coal, wood and smokeless coal, respectively). In contrast to other studies of wood smoke particle size distribution, measurements in Xuan Wei indicated that only 6% of the particles emitted during wood combustion were smaller than 1µm in size, whereas 51% of the smoky coal particles were submicron. Mutagenicity tests of particulates collected from the

various combustion processes indicated that smoky coal was approximately 5 times more mutagenic than wood (Mumford, He XZ et al. 1987). This study suggests that there was little association between open-fire wood smoke exposure and lung cancer, despite very high exposures with long duration (women generally start cooking at age 12). One possible explanation is the relatively low biological activity of wood smoke particulate combined with less efficient deposition of the larger particles.

The available, although limited, data on biomass smoke and cancer do not indicate an increased risk even at very high levels of exposure. This evidence includes studies of long-term exposure to high levels of biomass smoke from domestic cooking in developing countries. Evidence for a relationship between urban particulate air pollution and lung cancer is also limited, but is suggestive of a small, but measurable, increased risk. There have not been enough studies conducted to evaluate the consistency of any increased risk for different particle sources. However, while biomass smoke is clearly mutagenic, it is much less so than motor vehicle exhaust, on a comparable mass basis.

## **V. Research needs**

The studies discussed above indicate that biomass air pollution is clearly associated with some degree of adverse health outcome. By analogy to the general particulate epidemiology, it is likely that the exposures encountered during biomass air pollution episodes in Southeast Asia will result in acute health impacts spanning the entire range of severity, from sub-clinical impacts on lung function to increased daily mortality. Shorter duration episodes at lower air pollution concentrations have been linked with adverse impacts, while chronic exposure to higher levels of biomass air pollution and to lower levels of urban air pollution have been associated with development of chronic lung disease and decreased life expectancy. Therefore, the Southeast Asian biomass air pollution scenario falls into a relatively unique exposure category in which severe acute effects are highly probable but exposures are much lower (especially in terms of duration) than those experienced in studies which have demonstrated chronic impacts. Given the uncertainty regarding the potential for chronic effects, it would seem reasonable to initially evaluate the acute health impacts, especially since these are likely to include severe impacts such as increased mortality. Further, acute impacts are expected to be much easier to detect with a high degree of confidence than chronic health endpoints. To help understand the potential for adverse health effects and to evaluate the effectiveness of various mitigation measures, there is also a need to investigate several exposure issues. Several major research questions are summarized below. However, before these questions can be addressed it will be necessary to identify the availability of data, specifically air monitoring data and valid data on health indicators such as daily mortality, hospital visits, clinic/emergency room visits, etc.

1. What were the short term human health impacts associated with exposure to biomass air pollution in Southeast Asia?

For the range of identified impacts, were the effects reversible or permanent?

2. What were the long term human health impacts (if any) associated with exposure to biomass air pollution in Southeast Asia?
3. Which (if any) population groups were especially susceptible to adverse health effects of biomass air pollution in Southeast Asia?
4. What was the size of the exposed population?

Using study results and available air monitoring data (possibly including satellite data) can the region-wide health impacts be estimated?

5. What was the relationship between differences in exposure and health impacts across the affected region?

Were there exposed areas in which health impacts were larger/smaller than others?

Can an exposure-response relationship be demonstrated throughout the region?

6. What was the effectiveness of the following health protection measures:

The use of dust masks

Advising the population to remain indoors?

7. What was the composition of the biomass air pollution which affected Southeast Asia?

Can specific biomass marker compounds be identified?

To what extent is it possible to distinguish biomass air pollution from the “background” urban air pollution?

## VI. Possible research designs

In developing study designs for epidemiological investigations, it is important to note that, in general, the more serious the outcome measure, the smaller the affected population will be. In turn, the more serious the outcome measure, the greater the availability of data. The pyramid below (Figure 4) illustrates these tradeoffs, with the size of each level representative of the number of people affected. Severe outcomes such as mortality will only be seen in a relatively small group of people, and will therefore require a large sample size to detect an effect. However, such information is often easily available from administrative databases (death registries, for example). Less severe outcomes such as reduced lung function will generally be evident in a larger segment of the population, therefore requiring a smaller sample population to detect an effect. To obtain this information, however, requires individual assessment.

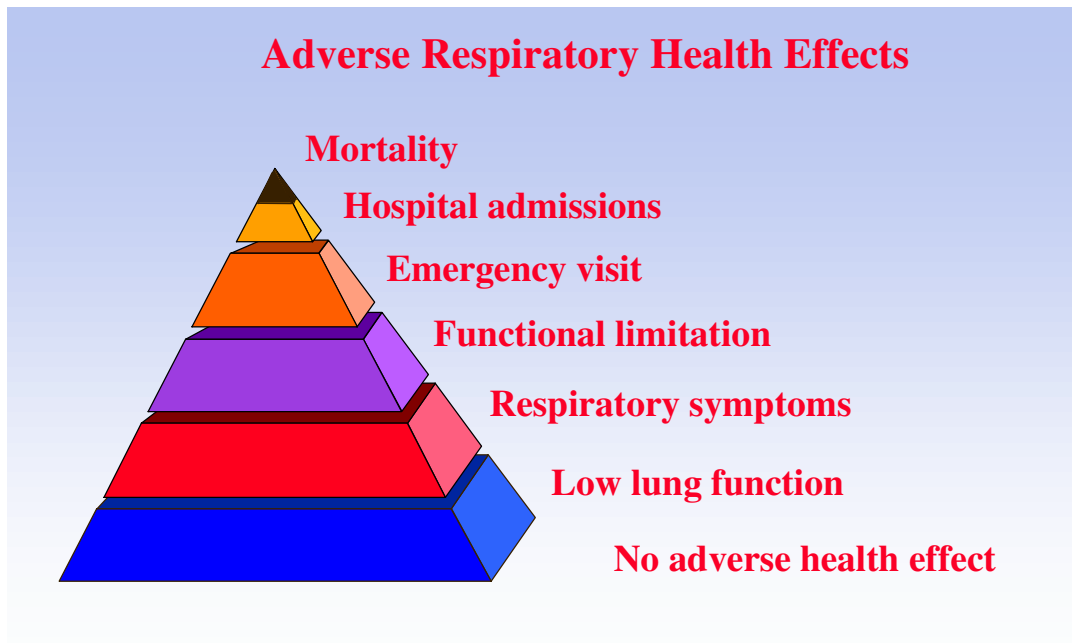


FIGURE 4

A similar set of trade-offs occurs when selecting exposure measurements. At one extreme, regional data on air quality can be obtained from remote sensing data relatively inexpensively and efficiently. These data are imprecise and only provide a crude, but still quantitative, estimate of exposure. It is possible to estimate concentrations for several gases and for particulates by this technique. No detailed information on particle composition can be obtained and the measurements are “snapshots” of selected time intervals. This approach may be most useful for evaluating impacts in rural areas without ambient monitoring stations. Ground-based ambient air monitoring can either provide a continuous or time-integrated assessment of particulate and gaseous pollutants. Integrated particulate measurements have the advantage of being feasible for chemical analysis of particles, post-sampling. The usefulness of these data are determined by the extent of regional coverage of a monitoring network. In the absence of these measurements,



airport visibility data has been used as a surrogate measure of particulate concentrations (Abbey, Ostro et al. 1995). For particular study populations, specific ambient monitoring stations that more closely reflect the populations' exposure may be required – such as placing specific monitors in selected neighborhoods.

Since individuals spend the majority of time indoors, more precise exposure estimates are obtained from indoor monitoring. The cost of this enhancement is that an individual monitor must be placed in each residence/workplace of the study population. Often indoor and outdoor measurements can be combined with information about an individual's activity patterns (how much time they spend in particular locations) to estimate their exposure. Large scale models have been developed to estimate population exposure based on census data, time-activity surveys of the population, and information on indoor and outdoor relationships for various pollutants within the various "microenvironments" where individuals spend time throughout their lives (home, work, school, etc.). The U.S. EPA has developed models for CO and O<sub>3</sub>. Currently researchers at Harvard University and The University of British Columbia are working together to develop models for PM<sub>10</sub> and PM<sub>2.5</sub> exposure. The most precise exposure measure is obtained by actually monitoring personal exposure – having an individual wear a monitor as they move from microenvironment to microenvironment throughout the day. While it is possible with this approach to accurately measure exposure of a representative population sample, the extent of such monitoring is limited by financial and logistical constraints. Further, such monitoring is often inconvenient for subjects and the measurement technology may be constrained to the extent that precision is affected.

With regard to the general research questions identified above, several possible study designs are proposed:

#### **A. Acute health impacts**

Formal study of the acute impacts of forest fire-related air pollution episodes should be conducted. Ideally, these studies should be directed towards the most severe health outcomes, while considering that impacts of air pollution will be small relative to all other causes of morbidity and mortality. Accordingly, the use of adequately large sample sizes is critical, as is the selection of appropriately sensitive statistical modelling techniques. Examples of studies are:

- (a) a time-series study of hospital visits in one or more major metropolitan areas with air monitoring and complete hospital visit data. In these studies daily counts of deaths/hospital admissions are compared to daily air pollution concentrations
- (b) a time-series study of acute mortality in one or more major metropolitan areas
- (c) Where feasible, additional time series studies on emergency visits, clinic visits, respiratory symptoms, work or school absenteeism can be conducted.

To the extent possible, individual factors such as age, disease status, socioeconomic factors, etc. should be evaluated in the analysis, both to control for potential confounding as well as to evaluate the existence of susceptible population sub-groups.

Further, to the extent possible specific study protocols should be standardized and conducted in several regions where ambient air concentrations differed.

## ***B. Chronic health impacts***

Formal study of the long-term impacts of forest fire-related air pollution may be attempted although it must be acknowledged that these studies are extremely difficult to conduct, and even the best studies will provide equivocal results due to issues of confounding variables and misclassified exposure. These issues are particularly complicated in the study of impacts of episodic air pollution events. Furthermore, the cross-sectional and semi-individual study designs depend upon the identification of measurable variability in exposure, and in this case, that the impact of the specific haze episode(s) resulted in variability in exposure. Possible study designs might include:

- (a) continuation of any ongoing cohort studies of health status (in which individual-level data are available) in locations where air monitoring data are available or where concentrations can be reliably estimated. Studies of large populations with varying exposure to biomass air pollution would be particularly useful. If available, such databases can be linked retrospectively with air pollution data.
- (b) cross-sectional comparisons of respiratory/cardiovascular disease incidence and mortality in areas with differing exposure to biomass air pollution
- (c) semi-individual studies in which a members of a demographically homogeneous population can be individually evaluated for health outcomes (lung function measurements for example) and in which potential confounding variables (smoking status, for example) can be measured. The measured health outcome is then compared to individual exposure profiles which are determined retrospectively by combining subject interviews with ambient air monitoring data. Examples of populations studied by this method are military recruits and entering university students.
- (d) Case-control studies have also been used in the past to estimate the risk of chronic health impacts. In these studies, individuals with some well-defined health outcome are identified and the exposure of these “cases” is compared to a suitable control group which is similar to the case group, except for the presence of the health outcome. It is quite difficult to conduct these studies, primarily since the selection of a suitable control group is critical. If an inappropriate control group is used, biased results can be obtained. To estimate the impact of biomass-related air pollution with this study type would require the identification of a control group which was similar (in terms of age, socioeconomic status, smoking status, etc.) to a group treated for some respiratory illness after the haze episode.

## ***C. Exposure issues***

### **Regional exposure**

A region-wide composite database of ambient air concentrations should be developed. Estimated air pollution contour plots can be developed using available air monitoring data, and, if feasible, supplemented with airport visibility and remote sensing data. Using such a database combined region-specific demographic data and with exposure-response relationships determined from epidemiological studies, the regional impact of the biomass air pollution episode can be estimated.

### **Mask effectiveness**

The effectiveness of masks for use by the general public should be evaluated. An additional aim should be an adequate understanding of the variables determining mask effectiveness, including technical factors such as filtration efficiency and leakage, as well as non-technical issues such as population compliance and comfort. Identification of the most important variables determining mask effectiveness will enable the design of new masks that are specifically applicable for general public use.

### **Indoor penetration**

The effectiveness of air cleaners, air conditioners, open/closed windows within various building types (residential office, etc.) as they relate to indoor penetration of fine particles should be assessed. Perhaps the simplest investigation would be to measure air exchange rates in representative building types under different scenarios. As discussed in the Mitigation Measures section, once air exchange rate information is known the infiltration of outdoor particles can be calculated. Verification of these calculated values could then be undertaken on a smaller set of buildings.

### **Biomass smoke composition**

Detailed chemical analysis of particulate samples should be conducted to identify the proportion of various functional groups (PAHs, elemental carbon, trace metals, etc.) within the haze particulate. While this analysis may be useful in future risk assessment and in comparing the toxicity of these particulate samples to those collected in other locations, the current emphasis should be on identifying marker compounds which may be used to distinguish air pollution originating from biomass burning from other sources. If identified, tracer compound(s) may be used to refine exposure assessment in epidemiological studies and to specifically evaluate indoor penetration of biomass particulate. Additional efforts should be directed to the determination of the size distribution of the biomass particulate and to a formal analysis of impacts of the haze episode on other routinely monitored ambient air pollutants, in particular, carbon monoxide and ozone.

## **VII. Mitigation measures**

Due to the limited effectiveness of exposure avoidance activities during regional haze episodes, priority emphasis must be given to elimination of the source of the air pollution, which in this case is extinguishing fires or preventing their occurrence. Close interaction between health, environment and meteorological agencies could result in effective forecasting of future air pollution episodes, be they related to forest fires or local sources of air pollution. However, despite efforts to prevent and control fires it is acknowledged that other measures may be necessary to help mitigate public health impacts. Following from basic principles of exposure control, if source control is not feasible then administrative or engineering controls receive priority, followed by personal protective equipment such as dust masks. In this exposure situation, administrative controls might include recommendations to the population to reduce their level of physical activity, while engineering controls include the use and/or enhancement of air conditioning or indoor air cleaning. Reduction of physical activity will certainly reduce the dose of inhaled air pollutants and will likely reduce the risk of health impacts, although no

formal studies have been conducted for particulate matter. Other mitigation measures are discussed in more detail in the following sections.

### **A. Dust masks**

During the 1997 haze episode, one of the major government and commercial efforts to mitigate public health impacts was the distribution of facial masks. Many different types of masks with variable filtration effectiveness were used. Several of the most effective masks have been tested to meet older United States of America National Institute of Occupational Safety and Health (NIOSH) standards for dust respirators. These masks passed a test procedure which uses 0.5  $\mu\text{m}$  silica particles and have been demonstrated to filter more than 99% of challenge particles. However, in order for these masks to reduce human exposure by the same degree, the masks must provide an airtight seal around the face. As all masks are designed for use by adult workers, the effectiveness of even the highest quality masks for use by the general public (including children) has not been evaluated. It is unlikely that they will provide more than partial protection. Lower quality masks will offer even less protection. Further, while it is expected that such masks would also filter a high percentage of the smaller ( $<0.1 \mu\text{m}$ ) particles present in biomass smoke, no performance data are available.

Wake and Brown evaluated nuisance dust masks, which are generally not approved by health and safety authorities, for their filtration efficiency. These masks are designed for coarse dusts and not for the fine particles present in biomass smoke. Handkerchiefs and tissues were also tested. Although the smallest particle size used in testing was 1.5  $\mu\text{m}$ , they found no difference between wet and dry handkerchiefs and in general found the penetration of 1.5  $\mu\text{m}$  particles to be quite high (60-90%) for all dust masks tested. Penetration for handkerchiefs and tissues was 70-97%. In all case, higher airflow was associated with increased filtration (Wake and Brown 1988).

Qian and colleagues evaluated dust/mist respirators which met older NIOSH regulations and surgical masks, and compared these to new N95 respirators which, by definition, meet newer NIOSH regulations (Qian, Willeke et al. 1998). In 1995 NIOSH issued new regulations for non-powered particulate respirators. The regulations indicate 9 classes of filters (3 efficiency levels - 95, 99, 99.97% and 3 series of filter degradation resistance). Criteria are met by testing with aerosols of the most penetrating size (0.1 – 0.3  $\mu\text{m}$ ) at an 85 l/min flow rate. N95 respirators from different companies were found to have different particle penetration for the most penetrating sizes (0.1 – 0.3  $\mu\text{m}$ ), but all were  $>95\%$  efficient. For particles larger than 0.3  $\mu\text{m}$ , the filtration increased with increasing size such that 99.5% of 0.75  $\mu\text{m}$  particles are removed. For welding fumes with sizes  $<1 \mu\text{m}$ , approximately 1.8% of the mass penetrated the respirator, indicating excellent protection if a good face seal exists. Minimum efficiencies at the most penetrating particle sizes were 96, 82 and 71% for the N95, dust mask and surgical mask, respectively.

The devices were also tested at a lower flow rate, which may be more representative of general population use. Under these conditions, efficiencies increased due to the increased time available for particle removal by the electrostatic material in the masks. Efficiencies were 98.8, 86 and 80% for the N95, dust masks and surgical masks, respectively. It should be noted that these efficiencies do not consider face seal leakage. As filter material is loaded the pressure drop across it increases, encouraging air to bypass the filter material through any leaks that are

present. Dust masks meeting the older NIOSH certification were used in Malaysia during the 1997 haze. It is unclear whether any N95 respirators were used. Although the N95 respirators have higher collection efficiencies, the dust masks, and even surgical masks will provide a high degree of protection, provided there is an adequate seal around the face and provided that they are changed once loaded.

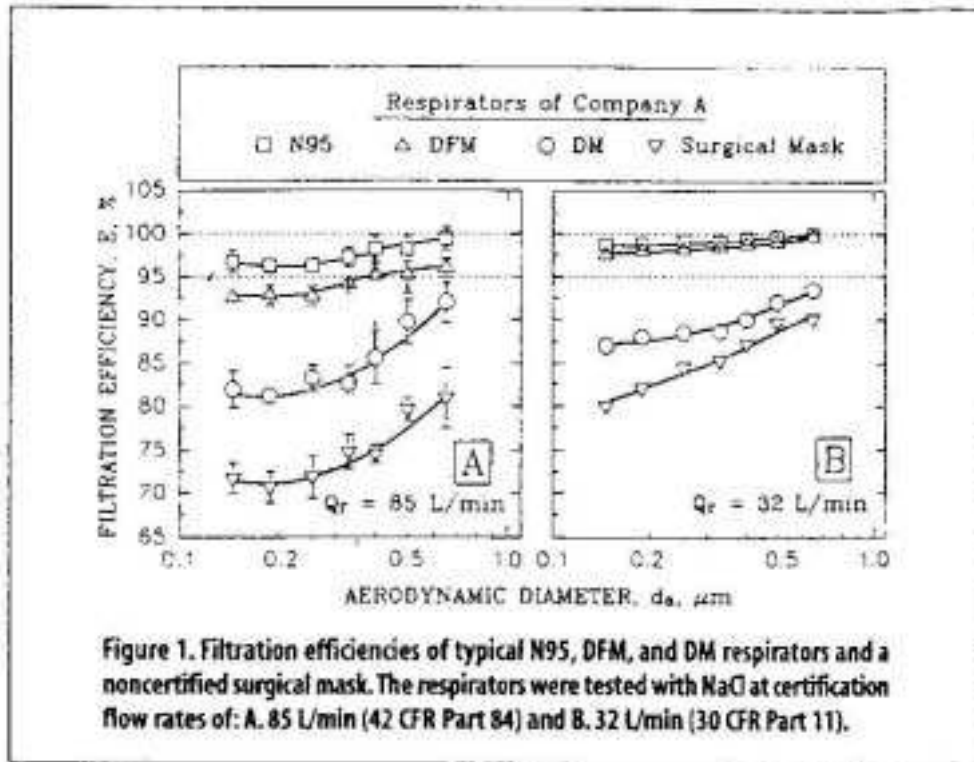


FIGURE 5 [Qian, Willeke et al. 1998]

Chen and colleagues evaluated dust masks and surgical masks for their filtration efficiency of 0.8 $\mu\text{m}$  polystyrene latex spheres at a flow rate of 46 l/min. As with the other studies, the effect of facial fit was not addressed. NIOSH has estimated that at least 10-20% leakage occurs in masks not fitted to the wearers' face (CDC in Chen) and measurements have confirmed this problem for dust masks (Tuomi 1985). One of the masks tested by Chen, et al was used in Malaysia during the 1997 haze. This mask had a mean efficiency of 96% while the surgical mask had an efficiency of 96% (Chen, Vesley et al. 1994). The same mask was tested by Hinds and Kraske at a number of flow rates and particle sizes (Hinds and Kraske 1987). As indicated in the Figure, efficiency decreased with decreasing particle size and increasing flow rate. At normal resting or moderately active respiratory rates and for the particle sizes present in biomass smoke, this mask type would be expected to be 80-90% efficient. However, despite these relatively high filtration efficiencies, the magnitude of the face seal leakage (up to 100% for sub-micron particles) indicates that fit testing and selection of tight-fitting masks is essential for protection. Surgical

masks, in contrast to approved dust masks, are not designed for fit testing or for an adequate face seal.

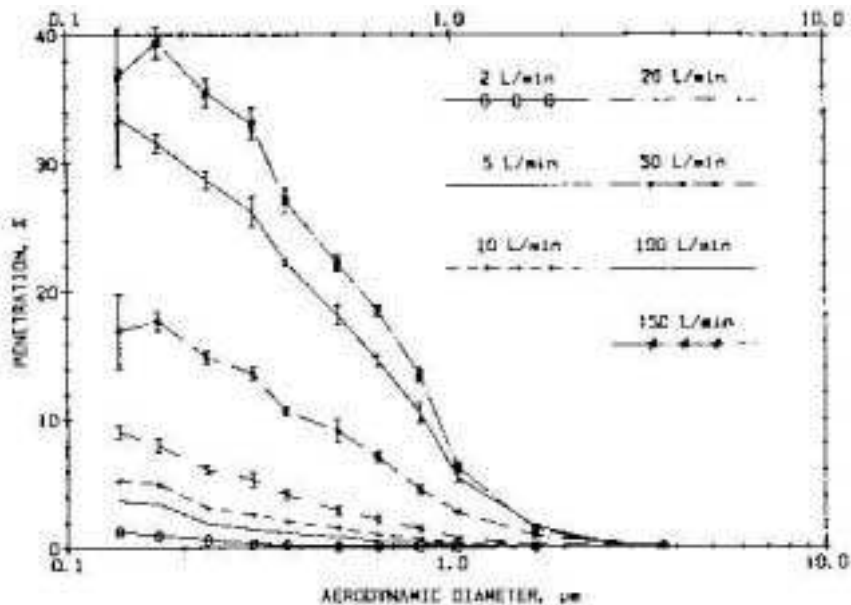


Figure 6 — Filter performance, 3M 8710 single-use respirator. Bars on 20-150 L/min lines indicate  $\pm$  standard errors of the mean for each data point.

FIGURE 6 [Hinds and Kraske 1987]

Adverse effects of wearing a disposable respirator (the same type used in Malaysia as described above) were evaluated by doing treadmill exercise. Physiological stress indicators such as heart rate, breath rate, and blood pressure were monitored as well as breath assistance and heat stress imposed by wearing respirator. Although resistance to breathing through a disposable respirator is not great, a disposable respirator imposes significant physiological stress including increased heart and respiratory rates, especially at moderate and heavy work loads (Jones 1991).

The Ministry of Environment of Singapore developed recommendations for mask use during biomass air pollution episodes. (24 Sep 97, <http://www.gov.sg/env/sprd/Rel-Mask.htm>). These recommendations suggest that surgical and other similar masks are not useful in preventing the inhalation of fine particles as they are not efficient in the filtration of particles of less than 10  $\mu\text{m}$ , such as those present in biomass smoke. Accordingly, use of these masks may provide a false sense of well-being to the users. The recommendations also suggest that respirators (which includes some types of dust masks) are able to filter 80% to 99% of particles between 0.2 and 0.4  $\mu\text{m}$ . The recommendations indicate that respirators may be useful, but are uncomfortable and increase the effort of breathing. According to some assessments, over an eight-hour period of use, a respirator of 95% efficiency can offer satisfactory filtration without undue breathing resistance to an average healthy adult. At higher efficiencies, breathing resistance increases and the user will experience more discomfort. Respirators may have a role for those with chronic cardio-respiratory illness, but should be used on the recommendation of their attending doctors. The recommendations also suggest that during periods of intense air pollution, it would be better

for the public to avoid outdoor activity than to put on a mask and stay outdoors for prolonged periods. However, for those who cannot avoid going outdoors, the use of respirators would provide some relief.

### ***B. Indoor penetration***

Another major recommendation for the population was to stay indoors. Unfortunately, this recommendation is likely to have provided only partial protection, and in some cases, no protection at all. Data from studies conducted in the U.S. for combustion-source particulates indicate that in non-air conditioned homes, approximately 88% of outdoor particles penetrate indoors during summer (Suh, Spengler et al. 1992). Limited measurements conducted in Singapore in 1994 indicate that during biomass episode periods, the increase in particle concentrations was due to particles smaller than 2  $\mu\text{m}$  and on average 60% penetrated indoors (Chia, 1995 #242). In a sample of 11 homes with air conditioning, an average infiltration factor of 44% was measured, but this is dependent upon the level of particle filtration (Suh, Spengler et al. 1992). Simultaneous indoor and outdoor measurements with continuous particle monitors were performed in Seattle areas impacted by residential wood burning. These measurements demonstrated a strong correlation between indoor and outdoor levels and an indoor:outdoor ratio of 0.98, presumably due to the high air exchange rates in these homes (Anuszewski, Larson et al. 1992).

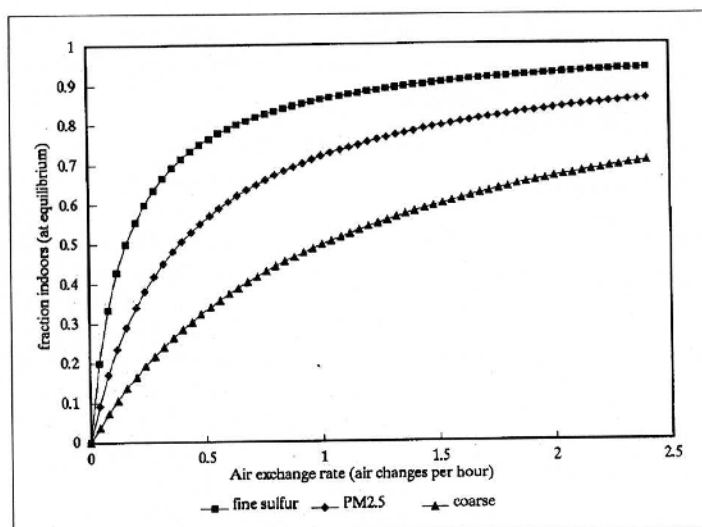
Brauer, et al measured indoor:outdoor ratios in 6 non air conditioned homes in Boston during summer. Using sulfate as a tracer of outdoor source fine particles, indoor:outdoor ratios of 0.96 was measured (Brauer, Koutrakis et al. 1991). Other studies in homes have measured indoor:outdoor sulfate ratios of approximately 0.7, while a similar value was observed in office buildings with mechanical ventilation systems. The variation between these measurements are likely due to differences in air exchange rates (Brauer, Koutrakis et al. 1991), as discussed below. The PTEAM study of nearly 300 homes in Riverside, California indicated that nearly 100% of indoor particle sulfur was of outdoor origin (Ozkaynak, Xue et al. 1996).

Since the majority of time is spent indoor, exposure indoors is an important variable to consider, even for pollutants generated outdoors. The impact of outdoor particles on indoor levels, was discussed in detail by Wallace [Wallace, 1996 #234]. Recent research has indicated that the impact of outdoor particle on indoor levels is a function of the particle penetration through the building envelope, the air exchange rate and the particle decay rate. Several studies indicate that penetration is complete for PM<sub>10</sub> and PM<sub>2.5</sub>. This means that the impact of outdoor particle on indoor levels is determined by the decay of particles indoors and by the rate of ventilation. Particle decay rates for PM<sub>10</sub> and PM<sub>2.5</sub> are also known, Therefore, the impact of outdoor particles can easily be calculated for any air exchange rate. In typical North American homes outdoor air accounts for 75% and 65% of fine and coarse particles, respectively. In North American homes, the geometric mean air exchange rates are 0.45-0.55/hr, but vary by season and specific geographic location. In general, air conditioned homes typically have lower air exchange rates than homes that use open windows for ventilation. In one study, air conditioned homes had air exchange rates of 0.8/hr, while non air-conditioned homes had rates of 1.2/hr, implying indoor fractions of outdoor PM<sub>2.5</sub> of 67 and 75%, respectively. Wallace comments that one method of reducing particle exposure would be to decrease home air exchange rates, by

weatherizing in cold seasons and by installing air conditioners for hot seasons to reduce the use of open windows.

Commercial building studies were also reviewed by Wallace, although little information is available as most studies have been directed towards the impact of smoking and not outdoor air pollution (Wallace 1996). In a study of 40 non-smoking buildings, the mean indoor:outdoor respirable particulate ratio was 0.9, although it is not possible to determine the relative importance of particles originating outdoors or indoors. The infiltration of outdoor particles into commercial buildings is likely to be highly variable as it is dependent upon the air exchange rate and specific characteristics of the ventilation system, including the efficiency of air filters.

Wallace



**Figure 7.** Expected concentration indoors under steady-state conditions for ambient sulfur and for fine and coarse particles, assuming a penetration rate of unity, and a decay rate of  $0.16 \text{ h}^{-1}$  for sulfur in the  $\text{PM}_{2.5}$  fraction,  $0.39 \text{ h}^{-1}$  for fine ( $\text{PM}_{2.5}$ ) particles and  $1.01 \text{ h}^{-1}$  for coarse particles. The curve for  $\text{PM}_{10}$  would lie between the curves for the fine and coarse particles, depending on the relative amounts of the two fractions.

FIGURE 7 [Wallace, 1996 #234])

### C. Air cleaners

Offermann and colleagues conducted chamber tests to evaluate portable air cleaners for their effectiveness in controlling indoor levels of respirable particles (Offermann, Sextro et al. 1985). Mixing fans, ion generators and small panel-filter devices were ineffective for particle removal. In contrast, electrostatic precipitators, extended surface filters and HEPA filter units worked well, with effective cleaning rates (for removal of 98% of particles in a room) of 100 – 300  $\text{m}^3/\text{hr}$ .

The Ministry of Environment of Singapore conducted an assessment of the use of portable air cleaners for homes, during periods of biomass air pollution. (26 Feb 98 <http://www4.gov.sg/env/sprd/haze-rel-22-98.htm>). The Ministry of Environment found that



several models of portable air cleaners were able to reduce the level of fine particles in a typical living room or bedroom to an acceptable level when there is an intense biomass episode. The Ministry of Environment also suggests that households can add a special filter to window or split-unit air-conditioners to achieve similar results for particle removal. For central air conditioning systems, electrostatic precipitators, high-efficiency media filters and medium-efficiency media filters can be added so that the particle level in the indoor air can be kept within acceptable levels during a prolonged biomass smoke period.

Portable air cleaners were also discussed in a U.S. EPA report (USEPA 1990). Studies have been performed on portable air cleaners assessing particle removal from the air in room-size test chambers or extensively weatherized or unventilated rooms. All of the tests addressed removal of cigarette smoke particles, which is similar in size to biomass smoke. The studies show varying degrees of effectiveness of portable air cleaners in removing particles from indoor air. In general, units containing either electrostatic precipitators, negative ion generators, or pleated filters, and hybrid units containing combinations of these mechanisms, are more effective than flat filter units in removing cigarette smoke particles. Effectiveness within these classes varies widely, however. The use of a single portable unit would not be expected to be effective in large buildings with central heating, ventilating, and air-conditioning (HVAC) systems. Portable units are designed to filter the air in a limited area only.

The effectiveness of air cleaners in removing pollutants from the air is a function of both the efficiency of pollutant removal as it goes through the device and the amount of air handled. A product of these two factors (for a given pollutant) is expressed as the unit's clean air delivery rate (CADR). The Association of Home Appliance Manufacturers (AHAM) has developed an American National Standards Institute (ANSI)-approved standard for portable air cleaners (ANSI/AHAM Standard AC-1-1988)<sup>25</sup>. This standard may be useful in estimating the effectiveness of portable air cleaners. Under this standard, room air cleaner effectiveness is rated by a clean air delivery rate (CADR) for each of three particle types: tobacco smoke, dust, and pollen. For induct systems, the atmospheric dust spot test of ASHRAE Standard 52-76 and the DOP method in Military Standard 282 may be used, respectively, to estimate the performance of medium and high efficiency air cleaners.

Table 7 shows the percentage of particles removed from indoor air in rooms of various size by rated CADR, as estimated by AHAM. The table provides estimates of the percent of particles removed by the air cleaner and the total removal by both the air cleaner and by natural settling. If the source is continual, the devices would not be expected to be as effective as suggested by the Table. In addition, the values represent performance that can be expected during the first 72 hours of use. Subsequent performance may vary depending on conditions of use.

**Exhibit 2. Estimated Percentage of Particle Removal for Portable Units by CADR and by Room Size**

		<b>PERCENTAGE OF PARTICLES REMOVED:</b>					
<b>Room Size</b>	<b>CADR</b>	<b>Smoke (20 min)</b>		<b>Dust (20 min)</b>		<b>Pollen (10 min)</b>	
		<b>AC<sup>a</sup></b>	<b>T<sup>b</sup></b>	<b>AC<sup>a</sup></b>	<b>T<sup>b</sup></b>	<b>AC<sup>a</sup></b>	<b>T<sup>b</sup></b>
5 X 6	10	49%	68%	49%	70%	—	—
	40	89%	97%	88%	98%	57%	93%
	80	95%	100%	95%	100%	75%	99%
9 X 12	40	53%	71%	52%	72%	24%	78%
	80	76%	89%	75%	89%	40%	86%
	150	89%	98%	89%	98%	58%	94%
12 X 18	80	53%	71%	52%	72%	24%	78%
	150	74%	87%	73%	88%	38%	85%
	300	89%	97%	—	—	—	—
	350	—	—	91%	99%	—	—
	450	—	—	—	—	69%	97%
18 X 24	150	51%	70%	50%	71%	23%	78%
	300	73%	87%	—	—	—	—
	350	—	—	77%	91%	—	—
	450	—	—	—	—	50%	91%
20 X 30	300	63%	79%	—	—	—	—
	350	—	—	67%	84%	—	—
	450	—	—	—	—	40%	86%

<sup>a</sup> Removal by the air-cleaning device

<sup>b</sup> Removal by the air-cleaning device plus natural settling

Note: Estimates ignore the effect of incoming air. For smoke and, to a lesser extent, dust, the more drafty the room, the smaller the CADR required. For pollen, which enters from outdoors, a higher CADR is needed in a drafty room.

**TABLE 7. Estimated Percentage of Particle Removal for Portable Units by CADR and by Room Size (USEPA 1990)**

## **VIII. Recommendations of health protection measures**

As discussed above, the hierarchy for health protection is control or prevention of fires followed by administrative controls such as reduced physical activity and remaining indoors. To enhance the protection offered by remaining indoors, individuals/building managers should take actions to reduce the air exchange rate. Clearly there are comfort and economic costs associated with reduced air exchange, as well as potential health effects due to increased impact of indoor pollution sources. It is not possible at this time to recommend more specific measures which would be feasible to employ on a population-wide basis. There is evidence that air conditioners, especially those with efficient filters, will substantially reduce indoor particle levels. To the extent possible, effective filters should be installed in existing air conditioning systems and individuals should seek environments protected by such systems. There is strong evidence that portable air cleaners are effective at reducing indoor particle levels, provided the specific cleaner is adequately matched to the indoor environment in which it is placed. Fortunately most air cleaners have been evaluated by manufacturers and their effectiveness is known. Unfortunately, economics will limit the distribution of such devices throughout the population. As with air conditioners the increased use of such devices by a large segment of the population will have a significant impact on energy consumption, and may in turn have negative impacts on ambient air quality. The least desirable measure is the use of personal protective equipment, such as dust masks. While these are relatively inexpensive and may be distributed to a large segment of the population, at present their effectiveness for general population use must be questioned. Despite this reservation, it is likely that the benefits (even partial) of wearing dust masks will outweigh the (physiological and economic) costs. Accordingly, in the absence of other mitigation techniques, the use of dust masks is warranted. Education of the population regarding specific mask types to purchase, how to wear masks and when to replace them will increase their effectiveness as will the development of new masks designed for general population use.

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