$\mu g/m^3$ (21). Brauer and colleagues (22) measured particulate levels in rural Mexican homes. Mean PM_{10} levels were 768 $\mu g/m^3$. During cooking periods, the mean $PM_{2.5}$ level was 887 $\mu g/m^3$, while peak (5 minutes) $PM_{2.5}$ concentrations reached 2000 $\mu g/m^3$ or higher in most of the homes cooking with biomass (22). During cooking periods, measurements in Brazil and Zimbabwe reported respirable concentrations of 1100 and 1300 $\mu g/m^3$, respectively (18). 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 (18, 21).

Wildland (forest) firefighters comprise an occupational group with high exposure to biomass smoke. Exposures of wildland firefighter were recently reviewed by Reinhardt and Ottmar (23), and will be discussed further in the health effects section. 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 the firefighter 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 Ottmar (23) measured mean CO and $PM_{3.5}$ levels of 4.1 ppm (maximum=38) and 0.63 (maximum=6.9) $\mu g/m^3$, respectively. Mean formaldehyde, acrolein and benzene levels were 0.047 ppm, 0.009 ppm and 0.016 ppm, respectively. Griggs and colleagues (24) 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 (25). 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. 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, sulphur 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_2 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^3, although two 4 hour samples were above 15 mg/m^3. It is not known what percentage of this was respirable. Across-shift symptom surveys indicated slight increases in eye, nose and throat irritation (26). Materna and colleagues (27) 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^3 and 0.13 ppm, respectively. Of the 12 specific PAHs detected, all were found at low levels (mean exposures <100 ng/m^3 except for phenanthrene at 380 ng/m^3). Although these data are limited, they are the most extensive available for PAH exposures.

Reh and colleagues (28) conducted extensive exposure assessment in combination with a medical evaluation. Respirable particulate levels were 0.6 - 1.7 mg/m^3 and sampling for acid gases detected low levels. Lung function decreased (<3 per cent 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 (28). 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^3, 0.018 ppm, 0.177 ppm and 0.035 ppm, for carbon monoxide, respirable particulates, acrolein, formaldehyde and benzene, respectively (29). 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 for CO and above 5 mg/m^3 for 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 (29) measured formaldehyde, acrolein, benzene, CO and respirable particulates ($PM_{3.5}$) in a rural area of Rondonia, Brazil, during the peak of the biomass burning season in 1996. Of the species measured, respirable particulate matter levels were significantly elevated, with mean levels of 190 $\mu g/m^3$, and with levels as high as 250 $\mu g/m^3$ measured during several of the 12-hour sampling periods. The author estimated that background levels of particulates in non-burning periods were 10-20 per cent of the levels measured during their study period. Particulate matter levels were also highly correlated with carbon monoxide concentrations, suggesting that carbon monoxide could be used as surrogate measurement of smoke exposure. Similar correlations have been observed in studies of North American wildland firefighters (23). 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 $\mu g/m^3$) and benzene. Benzene levels (11 $\mu g/m^3$ 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. 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. PM_{10} concentrations as high as 800 $\mu g/m^3$ have been measured in these communities, although peak levels (24-hour averages) of 200-400 $\mu g/m^3$ are more common (3, 30). PM_{10} measurements in British Columbia communities where wood smoke is the primary source of particulates indicate 24-hour averages of 2-420 $\mu g/m^3$ (2). Larson and Koenig (3) summarised several studies of PM measurements in communities with wood smoke as a major particulate source. In the reported studies, PM_{10} concentrations as high as 150 $\mu g/m^3$ and $PM_{2.5}$ levels of 86 $\mu g/m^3$ were measured in cases where wood smoke contributed more than 80 per cent of the particulate mass. 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 per cent of wood smoke particulate penetrates indoors (3), 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 $PM_{2.5}$ fraction, while the PM_{10} 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 literature on exposure and health impacts, 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 paper and of recommended future studies will be focused on this compound.

ACUTE AND CHRONIC HEALTH IMPACTS

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 (3).

Two preliminary reports suggest that wood smoke exposure may lead to increased susceptibility to lung infection (31). 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 US EPA compared infectivity of Streptococcus zooepidemicus aerosol 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 per cent of the mice in the control and oil furnace groups died, compared to 26 per cent of the wood smoke exposed group. Judith Zelikoff and colleagues at New York University exposed rats nasally to 800 $\mu g/m^3$ 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 marcrophage 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 do occur, we will first evaluate population groups which are exposed to high levels; i.e. forest firefighters and those exposed to indoor air pollution in developing countries where biomass is used for cooking and/or heating.

Epidemiological studies of non-cancer health risks

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

Wildland firefighters

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

A study of 76 firefighters in the US Pacific Northwest evaluated cross-shift and cross-season respiratory effects. No significant increase or decrease in respiratory symptoms was 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 (31). Sutton et al (33) measured CO levels of 4-200 ppm and 24-hour TSP levels of approximately 0.5 mg/m^3 at a firefighter camp in California. Health assessment of the firefighters indicated a high prevalence of headaches (50 per cent), cough (66 per cent), shortness of breath (38 per cent), lightheadedness (32 per cent) and wheezing (31 per cent).

Letts and colleagues (34) 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 per cent FEV_1 (forced expiratory volume), 0.2 per cent FVC (forced vital capacity) and -0.5 per cent in the FEV_1/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 concluded 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 (34).

Rothman and colleagues (35) studied cross-seasonal changes in pulmonary function and respiratory symptoms in 52 wildland firefigthers in Northern California. The mean cross-seasonal change in FEV_1 was -1.2 per cent, with a corresponding mean change in FVC of -0.3 per cent. Decreases in FEV_1 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 +/- SE, 73+/- hours of fire fighting in previous week) had a -2.9 per cent change in FEV_1 and a -1.9 per cent 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 (35).

Liu and colleagues (36) studied 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 post-season lung function, compared with pre-season values. There was also a statistically significant increase in airway responsiveness when comparing pre-season methacholine dose-response slopes with post-season 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 (36).

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 firefigthing exposure. On average, the firefighters worked during the 4-month fire seasons 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 (37).

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 indicates the plausibility, but not the magnitude of an association between biomass smoke exposure and adverse effects in the general population.

Indoor air pollution in developing countries

The health effects of biomass smoke inhalation have been documented in developing countries where women and 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 (22). Approximately 50 per cent 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 (21). In particular, exposure to biomass combustion products has been identified as a risk factor for acute respiratory infections (ARI). ARI are the leading cause of infant mortality in 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 (21, 38) and are summarised 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 $\mu g/m^3$) of respirable particulates. Blood carboxyhaemoglobin (COHb) was determined for 170 out of 244 children confirming that they did experience smoke inhalation (39).

The association between exposure to air pollution from cooking fuels and health aspects was studied in Maputo, Mozambique (40). Personal air samples for particulate matter (roughly equivalent to PM_{10}) 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 $\mu g/m^3$) than charcoal users (540 $\mu g/m^3$) and users of modern fuels (LPG and electricity)(200-380 $\mu g/m^3$). 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 (40). Lifetime exposure from cooking fuels was estimated by multiplying the exposure level (1200 $\mu g/m^3$ for wood) by years of exposure (23 for wood), duration of daily exposure (3 hours) and 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 group using wood for burning 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 (41). The risk of chronic bronchitis was linearly associated with hour-years of cooking with biomass. Crude

odds ratio for chronic bronchitis and chronic bronchitis/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 obstructive airways disease groups, respectively. The median hours per day of wood smoke exposure was 3 in the groups. Interestingly, the same research group who 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 design, bias in the case-control study and the development of resistance in women repeatedly exposed (41).

A case-control study conducted in Colombia identified a similar risk of obstructive airways disease (OAD) 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 OAD 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 per cent of all OAD cases (42).

A recent clinical report described 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 (43).

Cassano and colleagues (44) reported on a cross-sectional study of approximately 8000 individuals in rural areas of China with 58 per cent wood use as domestic cooking fuel and 77 per cent use of vented stoves. Vented stoves were associated with increased lung function and time spent

cooking was related to decreased lung function. Countywide chronic obstructive pulmonary disease (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.

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.

Community/cohort indoor and ambient air pollution studies

Particulates - overview

This paper will not discuss the voluminous particulate epidemiology literature in detail as a book (45) and several review articles have recently been published (16,46,47). Instead, an overview of the findings, and examples of the major study types and their findings, will be discussed with emphasis on the time series studies of acute health impacts and the recent prospective cohort studies of chronic exposure impacts. The available evidence associating biomass air pollution with adverse health outcomes will then be discussed in detail.

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 (48,49). 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 matter air pollution, with increased daily mortality (50-58). Different investigators have conducted these studies 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 (59,60). Recent studies have also suggested an association between particulates and infant mortality (61,62) as well as with low birth weight (63). 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 (46, 59, 64-68). Table 4 summarizes the results of these studies for the various health outcomes assessed. One common feature of the study locales is that ambient particulate matter is 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 (46, 69, 70). 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.

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 (50) studied over 8,000 adults in six US cities with different levels of air pollution over a period of 16 years. The adjusted mortality risk was 26 per cent higher in the most polluted city relative to the least polluted city. Survival decreased with increasing particulate levels. 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 (58). The adjusted mortality risk was 15-25 per cent 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 reduction of life expectancy. The results of the cohort studies of Dockery and Pope (46) were used to estimate the reduction in life expectancy associated with long-term particulate exposure. For a 10 $\mu g/m^3$ difference in long-term exposure to $PM_{2.5}$, the relative risk of mortality is 1.1. When applied to a 1992 life expectancy for Dutch men, the estimated effects is a 1.1 years reduction for each 10 $\mu g/m^3$ difference in long-term exposure to $PM_{2.5}$.

In the only cohort study of morbidity associated with long-term particulate exposure, Abbey and colleagues (71, 72) 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. For TSP levels above 100 $\mu g/m^3$ (in this case similar risks were observed for PM_{10} levels above 80 $\mu g/m^3$), significantly elevated risks were observed for as few as 500 hours (21 days) of exposure per year, for 4 years (71,72). 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 $\mu g/m^3$. Similar analyses were also conducted for estimated and measured concentrations of PM_{10} and $PM_{2.5}$. 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 at annual average PM_{10} and $PM_{2.5}$ levels of 20-100 $\mu g/m^3$ (73). Increased symptoms severity was associated with annual average concentration of 20-40 $\mu g/m^3$ and 40-50 $\mu g/m^3$ for $PM_{2.5}$ and PM_{10}, respectively.

While the vast majority of studies have measured PM_{10}, there is evidence to support regulating $PM_{2.5}$ levels. Schwartz et al (74) compared $PM_{2.5}$ and the coarse fraction of PM_{10} (PM_{10}-$PM_{2.5}$) as indicators of mortality. A significant relationship between $PM_{2.5}$ and mortality, but not with coarse particles, was found. These results are also consistent with our understanding of particle deposition since coarse particles are efficiently removed in the upper respiratory tract, while fine particle penetrate deep into the lung. A recent analysis of insoluble particles in autopsy lungs found that 96 per cent of the particles were smaller than 2.5 $\mu g/m$ (75). The $PM_{2.5}$ fraction also contains primarily particles produced in combustion processes, while the coarse fraction contains solid and crustal material that is not as toxicologically reactive.

Currently inhalable particulate matter is regulated in many countries. Since 1987, the US EPA standard for PM_{10} has been 150 $\mu g/m^3$ and 50 $\mu g/m^3$ for 24-hours and one year, respectively. California has set a standard of 50 and 30 $\mu g/m^3$ 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. The document argues that the available data suggest a continuum of effects with increasing exposure. Recently the US EPA set the first standard for $PM_{2.5}$ as 65 $\mu g/m^3$ and 15 $\mu g/m^3$ for 24 hour and annual averages, respectively.

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. These studies indicate that wood stoves, especially older varieties can emit smoke directly into the home (3). Newer airtight stoves emit less smoke into the homes, but indoor exposure still occurs due to infiltration of smoke emitted outdoors 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 (76). Honicky and colleagues (77, 78) 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 (77). The study of Honicky and Osborne (78) was motivated by a case report of a 7 month old infant hospitalised with serious respiratory disease, which was associated with the family's purchase of a wood

burning stove. 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 (79). Ramage et al (80) reported on the case of 61-year-old women with interstitial lung disease. Bronchioalveolar lavage revealed numerous particulates and fibres, as well as cellular and immunoglobulin abnormalities. The particles were shown to be carboneceous 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 (80).

Tuthill (81) 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. Butterfield et al (82) monitored 10 respiratory disease symptoms in 59 one to 5½ 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 $\mu g/m^3$ while source apportionment studies indicated that approximately 70 per cent of the winter particulate was from wood burning (83). 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 stove (84).

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 hospitalised with an ALRI were compared with age and gender matched controls who had a health record at the same hospital and had never been hospitalised for ALRI. Home interviews of parents of subjects elicited information on heating and cooking fuels and other household characteristics. Indoor PM_{10} 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 $\mu g/m^3$. The indoor particle concentration was positively correlated with cooking and heating with wood (geometric mean levels of approximately 60 $\mu g/m^3$) but not with other sources of combustion emissions (85). In the only study to date to evaluate impacts of wood burning on adult asthma, Ostro et al (86) measured symptoms in a panel of 164 asthmatics. Exposure to indoor combustion sources, including wood stoves was associated with increased asthma exacerbation. The studies of low-level indoor exposure are summarized in Table 5.

Several other studies, summarised in Table 6, have evaluated health outcomes in communities where wood smoke is a major, although not the only, source of ambient particulate. Heumann et al (30) 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 per cent of winter period PM_{10}. Winter period PM_{10} levels in the high exposure area ranged from approximately 50-250 $\mu g/m^3$ while levels in the low exposure area ranged from 20-75 $\mu g/m^3$. Lung function decreased during the wood burning season for the children in the high exposure area, but not in the low exposure area (30). 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 (87). Acute lung function decrements measured in 375 children were associated with increased levels of particulates. 24-hour averages ranged from 43-80 $\mu g/m^3$ and 14-38 $\mu g/m^3$ for PM_{10} and $PM_{2.5}$, 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 per cent of the $PM_{3.5}$ to wood smoke (3).

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. PM_{10} concentration averaged 55 and 33 $\mu g/m^3$ 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 (88).

A more comprehensive study in the same high exposure Seattle area was initiated in 1988. In these residential areas in Seattle, 80 per cent of particulate matter are from wood smoke (3). Lung function was measured in 326 elementary school children (including 24 asthmatics) before, during and after two wood burning seasons. Fine particulate matters was 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 (night time 12-hour average) $PM_{2.5}$ level measured during the study period was approximately 195 $\mu g/m^3$ and PM_{10} levels were below the US National Ambient Air Quality Standard of 150 $\mu g/m^3$ during the entire study period (89). For the asthmatic children FEV_1/FVC decreased by 17 and 18.5 ml for each 10 $\mu g/m^3$ increase in $PM_{2.5}$, 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 (66). In this study a significant association was observed between PM_{10} particle levels and emergency room visits for asthma. The mean PM_{10} level during the 1-year study period was 30 $\mu g/m^3$. At this concentration, PM_{10} 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 PM_{10}, with no evidence for a threshold at concentrations as low as 15 $\mu g/m^3$. The authors indicate that on an annual basis 60 per cent of the fine particle mass in Seattle residential neighbourhood is from wood burning.

Two time series studies (90, 91) have been conducted in Santa Clara County, California, an area in which wood smoke is the single largest contributor to winter PM_{10}, accounting for approximately 45 per