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Woodsmoke Health Effects: A Review

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The sentiment that woodsmoke, being a natural substance, must be benign to humans is still sometimes heard. It is now well established, however, that wood-burning stoves and fireplaces as well as wildland and agricultural fires emit significant quantities of known health-damaging pollutants, including several carcinogenic compounds. Two of the principal gaseous pollutants in woodsmoke, CO and NO_x, add to the atmospheric levels of these regulated gases emitted by other combustion sources. Health impacts of exposures to these gases and some of the other woodsmoke constituents (e.g., benzene) are well characterized in thousands of publications. As these gases are indistinguishable no matter where they come from, there is no urgent need to examine their particular health implications in woodsmoke. With this as the backdrop, this review approaches the issue of why woodsmoke may be a special case requiring separate health evaluation through two questions. The first question we address is *whether woodsmoke should be regulated and/or managed separately,* even though some of its separate constituents are already regulated in many jurisdictions. The second question we address is *whether woodsmoke particles pose different levels of risk than other ambient particles of similar size.* To address these two key questions, we examine several topics: the chemical and physical nature of woodsmoke; the exposures and epidemiology of smoke from wildland fires and agricultural burning, and related

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controlled human laboratory exposures to biomass smoke; the epidemiology of outdoor and indoor woodsmoke exposures from residential woodburning in developed countries; and the toxicology of woodsmoke, based on animal exposures and laboratory tests. In addition, a short summary of the exposures and health effects of biomass smoke in developing countries is provided as an additional line of evidence. In the concluding section, we return to the two key issues above to summarize (1) what is currently known about the health effects of inhaled woodsmoke at exposure levels experienced in developed countries, and (2) whether there exists sufficient reason to believe that woodsmoke particles are sufficiently different to warrant separate treatment from other regulated particles. In addition, we provide recommendations for additional woodsmoke research.

As the ability to control fire is often considered the characteristic distinguishing prehuman and human evolution and wood is the oldest of human fuels, it is literally true that exposure to woodsmoke* is as old as humanity itself. Even today, biomass in the form of wood and agricultural wastes is a significant source of direct human energy consumption worldwide, representing about 10% of the total. Of this, about 90% is used in its traditional forms as household heating and cooking fuels in developing countries, the rest being modern forms such as powerplant fuel, principally in developed countries (United Nations Development Programme [UNDP], 2004). Because household use dominates total fuel demand in many developing countries, particularly in rural areas where half of humanity still lives, it is likely that biomass remains the main source of energy for most of humanity.

Surprisingly, although the percentage of total fuel demand constituted by wood declines with economic development, the absolute amount remains relatively constant. For example, the average use of biomass fuel per capita in the primarily wealthy countries participating in the Organization for Economic Cooperation and Development (OECD) is quite similar to that in Asia, which has the world's largest developing nations (UNDP, 2004). Of course, per capita use varies substantially with local circumstances. Countries with ample wood supplies, such as Finland, Sweden, and Canada, burn more biomass fuel per capita than most other countries, while those with low supplies, such as South Korea and Singapore, burn less (Koopmans, 1999).

Over the past few decades, rising fossil energy costs, the availability of new technologies, and the desire to use renewable sources have led to increases in the use of wood and other biomass fuels in North America. For example, in Canada, such fuels increased at about 2.4% annually during the 1990s, more than half again as fast as overall energy demand (IEA, 2004).

During this same period, the knowledge of, and consequent concern about, the health effects of air pollution have increased dramatically around the world, leading to stricter air pollution regulation and controls. While commercial sources of wood combustion have been subject to some regulation in North America and Europe, there are still important unregulated sources of woodsmoke, including household heating stoves and fireplaces. The latter have been the target of local ordinances in a number of areas where woodsmoke dominates outdoor air pollution during some seasons. To attain standards for such important pollutants as fine particles ($PM_{2.5}$ or particulate matter less than 2.5 μm in diameter), however, additional controls of these household sources in more areas may be needed.

There are also important nonpoint sources of woodsmoke, particularly wildland fires and intentional burning of agricultural waste. The apparent increases of accidental wildfires in some areas may be due to forest management practices, climate change, and the rise in human population density near fire-prone areas. In addition, the practice of clearing forested areas through the use of fire has resulted in several spectacular long-burning conflagrations in Southeast Asia and elsewhere, which have resulted in a growing concern about the potential health impacts of such events.

The sentiment that woodsmoke, being a natural substance, must be benign to humans is still sometimes heard. It is now well established, however, that wood-burning stoves and fireplaces as well as wildland and agricultural fires emit significant quantities of known health-damaging pollutants, including several carcinogenic compounds (e.g., polycyclic aromatic hydrocarbons, benzene, aldehydes, respirable particulate matter, carbon monoxide [CO], nitrogen oxides [NO_x], and other free radicals) (Tuthill, 1984; Koenig & Pierson, 1991; Larson and Koenig, 1994; Leonard et al., 2000; Dubick et al., 2002; Smith, 1987; Traynor et al., 1987). Many of these toxic pollutants present in woodsmoke are listed in Table 1.

Two of the principal gaseous pollutants in woodsmoke, CO and NO_x, add to the atmospheric levels of these regulated gases emitted by other combustion sources. Health impacts of exposures to these gases and some of the other wood smoke constituents (e.g., benzene) are well characterized in thousands of publications. As these gases are indistinguishable no matter where they come from, there is no urgent need to examine their particular health implications in woodsmoke. There are reasons, however, why woodsmoke may be a special case requiring separate health evaluation.

1. At the point of emissions, woodsmoke contains a vast array of solid, liquid, and gaseous constituents that change, sometimes rapidly, with time, temperature, sunlight, and interaction with other pollutants, water vapor, and surfaces. Many constituents are known to be hazardous to human health, but are not specifically regulated or even fully evaluated. Current methods of health-effects assessment do poorly in estimating impacts by summing the effects of separate constituents. The best approach, therefore, is to examine the toxicity of the entire mixture, as has been done with the most well-studied biomass smoke, that from tobacco burning. Although there have been more than 4000 compounds identified

*Here, we use the term "smoke" to refer to the entire mixture of gases, solid particles, and droplets emitted by combustion.

TABLE 1
Major health-damaging pollutants from biomass combustion

Compound	Examples[a]	Source	Notes	Mode of toxicity
Inorganic gases	*Carbon monoxide (CO)*	Incomplete combustion	Transported over distances	Asphyxiant
	Ozone (O_3)	Secondary reaction product of nitrogen dioxide and hydrocarbons	Only present downwind of fire, transported over long distances	Irritant
	Nitrogen dioxide (NO_2)	High-temperature oxidation of nitrogen in air, some contribution from fuel nitrogen	Reactive	Irritant
Hydrocarbons	Many hundreds	Incomplete combustion	Some transport—also react to form organic aerosols. Species vary with biomass and combustion conditions	
	Unsaturated: 40+, e.g., *1,3-butadiene*			Irritant, carcinogenic, mutagenic
	Saturated: 25+, e.g., n-*hexane*			Irritant, neurotoxicity
	Polycyclic aromatic (PAHs): 20+, e.g., *benzo[a] pyrene*			Mutagenic, carcinogenic
	Monoaromatics: 28+, e.g., *benzene, styrene*			Carcinogenic, mutagenic
Oxygenatated organics	Hundreds	Incomplete combustion	Some transport—also react to form organic aerosols. Species vary with biomass and combustion conditions	
	Aldehydes: 20+, e.g., *acrolein*, *formaldehyde*			Irritant, carcinogenic, mutagenic
	Organic alcohols and acids: 25+, e.g., *methanol* acetic acid			Irritant, teratogenic
	Phenols: 33+, e.g., *catechol, cresol (methylphenols)*			Irritant, carcinogenic, mutagenic, teratogenic
	Quinones: *hydroquinone,* fluorenone, anthraquinone			Irritant, allergenic, redox active, oxidative stress and inflammation, possibly carcinogenic
Chlorinated organics	*Methylene chloride*, *methyl chloride*, *dioxin*	Requires chlorine in the biomass		Central nervous system depressant (methylene chloride), possible carcinogens

(*Continued on next page*)

TABLE 1
Major health-damaging pollutants from biomass combustion *(Continued)*

Compound	Examples[a]	Source	Notes	Mode of toxicity
Free radicals	Semiquinone type radicals	Little is known about their formation		Redox active, cause oxidative stress and inflammatory response, possibly carcinogenic
Particulate matter (PM)	*Inhalable particles (PM_{10})*	Condensation of combustion gases; incomplete combustion; entrainment of vegetation and ash fragments	Coarse[b] + fine particles. Coarse particles are not transported far and contain mostly soil and ash	Inflammation and oxidative stress, may be allergenic
	Respirable particles	Condensation of combustion gases; incomplete combustion	For biomass smoke, approximately equal to fine particles	[See below]
	Fine particles ($PM_{2.5}$)	Condensation of combustion gases; incomplete combustion	Transported over long distances; primary and secondary production[c]	Inflammation and oxidative stress, may be allergenic

[a]Compounds in italics either are criteria air pollutants or are included on the list of hazardous air pollutants specified in Section 112 of the U.S. Clean Air Act. At least 26 hazardous air pollutants are known to be present in woodsmoke.
[b]Coarse particles are defined as those between 2.5 and 10 μm in size.
[c]Particles are created directly during the combustion process and also formed later from emitted gases through condensation and atmospheric chemical reactions.

in tobacco smoke, many dozens of which possess toxic properties, there are few well-understood links between individual constituents and many of the health effects known to be caused by exposure to this mixture.

The first question we address, therefore, is *whether separate regulation/management of woodsmoke should be considered*, even though some of its separate constituents are already regulated in many jurisdictions.

2. Fine particles are thought to be the best single indicator of the health impacts of most combustion sources. Although woodsmoke particles are usually within the size range thought to be most damaging to human health, their chemical composition is different from those derived from fossil fuel combustion, on which most health-effects studies have focused. Because their composition differs from those produced by fossil fuel combustion, woodsmoke particles may not produce the same health effects per unit mass as other combustion particles. Currently, however, except for size, national regulations and international guidelines do not distinguish particles by composition.

The second question we address, therefore, is *whether woodsmoke particles pose different levels of risk than other ambient particles of similar size.*

To address these two key questions, we examine several topics:

- The chemical and physical nature of woodsmoke.*
- The exposures and epidemiology of smoke from wildland fires and agricultural burning, and related controlled human laboratory exposures to biomass smoke.
- The epidemiology of outdoor and indoor woodsmoke exposures from residential woodburning in developed countries.
- The toxicology of woodsmoke, based on animal exposures and laboratory tests.

A short summary of the exposures and health effects of biomass smoke in developing countries is provided as an additional line of evidence. At the end we provide recommendations for additional woodsmoke research.

*Although "woodsmoke" is the substance of primary interest in this report, evidence related to smoke from other biomass (agricultural residues, grass, etc.) is also examined where relevant.

Although cancer-related epidemiology and toxicology are discussed, we do not attempt a judgment because the International Agency for Research on Cancer (IARC) has just completed its Monograph #95, which includes an assessment of the carcinogenicity of household biomass fuel combustion. It was categorized as Category 2A, probably carcinogenic in humans, with limited human evidence although supporting animal and mechanistic evidence (Straif et al., 2006).

BRIEF SUMMARY OF METHODS

The authors searched available biomedical and scientific literature databases in English for articles dealing with controlled human exposure, occupational, and epidemiologic health-effects studies, and toxicologic investigations dealing with woodsmoke; biomass smoke; forest, vegetation, and wildland fires; agricultural burning; and related terms under developed-country conditions. Because of the scattered nature of the literature, however, each author also used his or her knowledge of the literature to identify other papers that did not show up in searches and material in the gray literature. We believe that the result is a nearly complete review of the major relevant publications on these subjects and that there was no bias in selecting papers to review, although we were not able to apply specific inclusion/exclusion criteria.

We did not attempt to search for or review all the literature on the physical and chemical nature of woodsmoke, its environmental concentrations and human exposures, or its health effects in developing-country conditions, such as indoor burning for cooking. In these arenas, we only try to summarize major findings by others.

CHEMICAL COMPOSITION OF BIOMASS SMOKE

Wood consists primarily of two polymers: cellulose (50–70% by weight) and lignin (approximately 30% by weight) (Simoneit et al., 1998). Other biomass fuels (e.g., grasses, wheat stubble) also contain these polymers, although their relative proportions differ. In addition, small amounts of low-molecular-weight organic compounds (e.g., resins, waxes, sugars) and inorganic salts are also present in wood. During combustion, pyrolysis occurs and the polymers break apart, producing a variety of smaller molecules. Biomass combustion is typically inefficient, and a multitude of partially oxidized organic chemicals are generated in biomass smoke. Biomass smoke contains a large number of chemicals, many of which have been associated with adverse health impacts. The major health-damaging particulate and gaseous chemicals present in biomass smoke are listed in Table 1, along with some of their main modes of toxic action.

Tables 2 and 3 summarize the major chemical classes detected in woodsmoke; detailed chemical speciation of the several hundred individual compounds that have been detected in smoke samples is reported in the original references (Rogge et al., 1998; Schauer et al., 2001; Fine, et al., 2002; McDonald et al., 2000; Oros & Simoneit, 2001). The studies cited in Tables 2 and 3 by Rogge et al., Schauer et al., Fine et al., and McDonald et al. all attempted to recreate conditions of residential wood combustion. In contrast, the studies by Oros et al. aimed at being more representative of wildfire emissions. More recently, Lee et al. have also described comprehensive chemical composition of smoke from prescribed burns (Lee & Baumann, 2005). Although less well characterized, a similar mixture of chemicals is reported in smoke emissions from other types of biomass, including grasses, rice straw, sugarcane, and ferns (Simoneit et al., 1993, 1998; Rinehart et al., 2002).*

TABLE 2
Fine particle emissions and bulk chemical composition in woodsmoke

Compound class	Concentration	References
Fine particle emissions rate (g/kg of wood burned)	1.6–9.5	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Organic carbon (wt% of fine particle mass)	12–101	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Elemental carbon (wt% of fine particle mass)	0.65–79	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Ionic species (wt% of fine particle mass)	0.014–1.7	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Elemental species (wt% of fine particle mass)[a]	0.01–4.0	(Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)

[a]Chloride included as an element.

In general, it is difficult to make quantitative comparisons among emission factors for specific organic compounds reported by different authors. This is because many of the reports are semiquantitative and the analytical methods used were not comprehensively validated for each analyte, authentic standards were frequently not available to calibrate instrument response, variable combustion conditions (fuel type, moisture content, combustion device) were used, and emission factors were reported in a variety of units.

*It should be noted that most studies have used gas chromatography/mass spectrometry (GC/MS) to characterize the chemical content of woodsmoke. GC is a very efficient tool for separating complex mixtures of organic chemicals. Combined with MS, the technique allows for highly sensitive, specific and accurate detection and quantification of a range of organic chemicals in environmental samples. GC/MS fails to detect compounds that are nonvolatile or thermally labile, however. The application of novel methods, such as liquid chromatography–mass spectrometry (LC/MS), that are appropriate for analysis of nonvolatile or thermally labile compounds will further expand the list of chemicals known to be present in biomass smoke.

TABLE 3
Emissions by chemical class for particle and vapor constituents in woodsmoke

Chemical	Particle-phase (mg/kg wood burned)	References	Vapor-phase (mg/kg wood burned)	References
Carbon monoxide	—		130,000	(McDonald et al., 2000)
		Hydrocarbons		
Alkanes (C2–C7)	0.47–570	(Rogge et al., 1998; Fine et al., 2002)	1.01–300	(Schauer et al., 2001; McDonald et al., 2000)
Alkenes (C2–C7)	0.58–280	(Rogge et al., 1998; Fine et al., 2002)	92–1300	(McDonald et al., 2000)
Polycyclic aromatic hydrocarbons (PAHs) and substituted PAHs	5.1–32,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998; McDonald et al., 2000)	43.4–355	(Schauer et al., 2001; McDonald et al., 2000)
Methane	—		4100	(Schauer et al., 2001)
Total nonmethane hydrocarbons C2–C7	[Included in vapor phase]		390–4000	(Schauer et al., 2001; McDonald et al., 2000)
Unresolved complex mixture (UCM)	300–1,130,000	(Oros & Simoneit, 2001; Fine et al., 2002)		
		Oxygenated organics		
Alkanols	0.24–5400	(Oros & Simoneit, 2001; Fine et al., 2002)	120–9200	(McDonald et al., 2000)
Carboxylic acids	6200–755,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998)	2.4	(Schauer et al., 2001)
Aldehydes and ketones	[Included in vapor phase]		0.94–4450	(Rogge et al., 1998)[a] (Schauer et al., 2001; Fine et al., 2002; McDonald et al., 2000)
Alkyl esters	0.37–4450	(Oros & Simoneit, 2001; Fine et al., 2002)		
Methoxylated phenolic compounds	28–1000	(Rogge et al., 1998; Fine et al., 2002; McDonald et al., 2000)	1200–1500	(Schauer et al., 2001)
		Other organics		
Other substituted aromatic compounds	5.0–120,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998)	110–3600	(Schauer et al., 2001; McDonald et al., 2000)
Sugar derivatives	1.4–12600	(Oros & Simoneit, 2001; Fine et al., 2002)		
Coumarins and flavonoids	0.71–12	(Fine et al., 2002)		
Phytosteroids	1.7–34.0	(Rogge et al., 1998; Fine et al., 2002)		
Resin acids and terpenoids	1.7–41,000	(Oros & Simoneit, 2001; Fine et al., 2002; Rogge et al., 1998)	21–430	(McDonald et al., 2000)
Unresolved compounds	1.2–120	(Fine et al., 2002)	20–600	(Schauer et al., 2001; McDonald et al., 2000)

[a]Only aldehydes reported.

Woodsmoke particles are generally smaller than 1 μm, with a peak in the size distribution between 0.15 and 0.4 μm (Kleeman et al., 1999; Hays et al., 2002). As with other combustion mixtures, such as diesel and tobacco smoke, fresh woodsmoke contains a large number of ultrafine particles, less than 100 μm, which condense rapidly as they cool and age. Indeed, most of the particle mass in aged woodsmoke has been formed by such condensation processes. Fine particles in this size range efficiently evade the mucociliary defense system and are deposited in the peripheral airways, where they may exert toxic effects. Particles in this size range are not easily removed by gravitational settling and therefore can be transported over long distances (Echalar et al., 1995). The transport of biomass combustion particles over hundreds of kilometers has been extensively documented (Andrae et al., 1988). Haze layers with elevated concentrations of CO, carbon dioxide (CO_2), ozone (O_3), and nitric oxide (NO) have been observed. During transport, many of the gaseous species are converted to other gases or into particles. The "black carbon" from biomass emissions is now thought to contribute to regional and global climate change as well as adverse health effects in some parts of the world (Venkataraman et al., 2005; Koch & Hansen, 2005).

Although approximately 5–20% of woodsmoke particulate mass consists of elemental carbon, the composition of the organic carbon fraction varies dramatically with the specific fuel being burned and with the combustion conditions. Detailed analysis of organic woodsmoke aerosol were conducted by Rogge et al. (1998), who measured nearly 200 distinct organic compounds, many of them derivatives of wood polymers and resins (Rogge et al., 1998). Since profiles of specific polycyclic aromatic hydrocarbons (PAHs) are likely to vary, many measurements have focused on benzo[a]pyrene (BaP), a probable human carcinogen.

A number of toxic or carcinogenic compounds are present in biomass smoke, including free radicals, PAHs, and aldehydes, as shown in Table 1 (Leonard et al., 2000; Pryor, 1992; Schauer et al., 2001). Organic extracts of ambient particulate matter (PM) containing substantial quantities of woodsmoke are 30-fold more potent than extracts of cigarette smoke condensate in a mouse skin tumor induction assay (Cupitt et al., 1994), and are mutagenic in the *Salmonella typhimurium* microsuspension and plate incorporation assays (Claxton et al., 2001). Few, if any, reports exist in which the toxicity of smoke from different biomass sources was compared and related to differences in the chemical composition of each smoke type.

Woodsmoke is enriched with several chemicals relative to pollutant mixtures from other sources of air pollution. Examples include potassium, methoxyphenols, levoglucosan, retene, and specific resin acids (e.g., abietic acid) (Khalil & Rasmussen, 2003; Fine et al., 2001, 2002; Schauer et al., 2001; Rogge et al., 1998; Hawthorne et al., 1992). Many of these chemicals have been used either individually or in multivariate analyses to quantify woodsmoke emissions (Khalil & Rasmussen, 2003; Schauer & Cass, 2000; Larsen & Baker, 2003).

Levoglucosan is sugar anhydride derived from the pyrolysis of the major wood polymer cellulose. Levoglucosan is one of the most abundant organic compounds associated with particles in woodsmoke (Fine et al., 2001, 2002). It is stable in the environment and has been used extensively to estimate woodsmoke levels in ambient PM samples (Schauer & Cass, 2000; Katz et al., 2004; Larson et al., 2004). Levoglucosan is present in other biomass smoke samples, including smoke from tobacco, grasses, and rice straw (Sakuma & Ohsumi, 1980; Simoneit, et al., 1993). Under conditions in which woodsmoke dominates the biomass smoke contribution to ambient aerosol, however, levoglucosan can be considered a unique tracer for woodsmoke (Schauer & Cass, 2000).

Methoxyphenols are a class of chemicals derived from the pyrolysis of the wood polymer lignin. This class of chemicals spans a range of volatilities from relatively volatile (e.g., guaiacol) to exclusively particle-associated (e.g., sinapinaldehyde). These chemicals are relatively abundant in woodsmoke, although the most abundant compounds are predominantly in the vapor phase (Hawthorne et al., 1989; Schauer et al., 2001). Accurate chemical analysis of the methoxyphenols, however, has proved to be an analytical challenge, and many of the methoxyphenols were found to be chemically reactive—a property that would undermine their suitability as tracers for biomass smoke (Simpson et al., 2005). Methoxyphenols have been used as woodsmoke tracers in multivariate source apportionment models to determine the proportion of urban fine PM derived from woodburning (Schauer & Cass, 2000).

The organic chemical composition has been used to distinguish smokes from different biomass fuels. Smoke from hardwood versus softwood burning can be distinguished by the relative proportions of substituted guaiacols compared to syringols (Hawthorne et al., 1989; Oros & Simoneit, 2001; Schauer & Cass, 2000). Mono- and dimethoxyphenols are also present in small amounts in grass and grain smokes, but the major phenolic compounds in grass smoke are *p*-coumaryl derivatives (Simoneit, et al., 1993). Diterpenoids (e.g., dehydroabietic acid) are abundant in smoke from gymnospems (conifers) compared to angiosperms (Schauer et al., 2001). Certain chemicals may even be unique to smoke from specific tree species (e.g., juvabione from balsam fir), although the atmospheric stability of such compounds and hence their utility as source-specific exposure markers has not been established (Fine et al., 2001; Oros & Simoneit, 2001).

Emission factors for fine particles are highly dependent on the fuel characteristics and burn conditions (smoldering vs. flaming). Similarly, emission factors for specific organic chemicals are influenced by fuel moisture content and burn conditions, although the relationships may not parallel those observed for fine particles (Khalil & Rasmussen, 2003; Guillen & Ibargoitia, 1999).

FOREST FIRE AND AGRICULTURAL BURNING: EXPOSURE AND HEALTH STUDIES

In contrast to the large amount of information relating urban PM to human health impacts, there is only a limited number of studies directly evaluating the community health impacts of air pollution resulting from the burning of biomass. Several reviews have discussed the health impacts and pollutants associated with woodsmoke air pollution (Larson & Koenig, 1994; Pierson et al., 1989; Vedal, 1993; Boman et al., 2003, 2006). Although the emphasis of these reviews was on community exposures resulting from burning of wood in fireplaces and wood stoves, many of the conclusions are relevant to the broader understanding of vegetation fire air pollution. The World Heath Organization (WHO) has published a document describing Health Guidelines for Vegetation Fire Events,* which also contains a review of evidence linking air pollution from vegetation fires with human health effects. Specific information relating agricultural and forest/brush burning with human health effects is summarized next and presented in Table 4.

On a regional basis, during vegetation fire episodes PM is the air pollutant most consistently elevated in locations impacted by fire smoke (Sapkota et al., 2005). For example, during fires in southern California, PM_{10} concentrations were 3–4 times higher than during nonfire periods, while particle number, and CO and NO concentrations were increased by a factor of 2. The concentrations of NO_2 and O_3 were essentially unchanged or even lower (Phuleria et al., 2005). Further, measurements indicate that that biomass combustion emissions can be transported over hundreds of kilometers such that local air quality is degraded even at great distances from fire locations (Sapkota et al., 2005). Smoke from African and Brazilian savanna fires has been shown to contain substantial quantities of fine particles (Artaxo et al., 1991; Echalar et al., 1995). Mass concentrations ranged from 30 μg/m^3 in areas not affected by biomass burning to 300 μg/m^3 in large areas (2 million km^2) 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. (1994), who found 24-h average PM_{10} and $PM_{2.5}$ mass concentrations as high as 700 and 400 μg/m^3, respectively (Artazo et al., 1994). 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 PM ($PM_{3.5}$) in Rondonia, Brazil, during the peak of the 1996 biomass burning season (Reinhardt et al., 2001). Of the species measured, respirable particle levels were elevated 5-10 times above background, with mean levels of 190 μg/m^3 and levels as high as 250 μg/m^{3}measured during several of the 12-h sampling periods. The mean CO level was 4 ppm, which is similar to levels measured in moderately polluted urban areas, but below the level expected to be associated with acute health impacts. Benzene levels (11 μg/m^3 average) were higher than those measured in other rural areas and were comparable to those measured in cities.

Measurements from Southeast Asia also indicate that particles are the main air pollutant elevated during periods of vegetation fire-related air pollution (Radojevic & Hassan, 1999). For example, during a 2- to 3-mo period in 1994, 24-h PM_{10} levels up to 409 μg/m^3 were recorded in Kuala Lumpur (Hassan et al., 1995), and levels ranged from 36 to 285 μg/m^3 (unspecified average time) in Singapore (Nichol, 1997). In a 1997 vegetation fire episode, PM_{10} levels as high as 930 and 421 μg/m^3 were measured in Sarawak (Malaysia) and Kuala Lumpur, respectively, while 24-h levels in Singapore and southern Thailand were somewhat lower (Brauer, 1998). Closer to the fire source in Indonesia, PM_{10} concentrations as high as 1800 μg/m^3 were measured over an unspecified period (Kunii et al., 2002). In February–May 1998 a more limited vegetation fire episode affected regions of Borneo. In Brunei, 24-h PM_{10} levels as high as 440 μg/m^3 were measured during this period (Radojevic & Hassan, 1999).

Wildland Firefighters

In general, wildland firefighters experience greater exposure from forest fire smoke than members of the general public. Patterns of exposure can be intense in initial fire-suppression efforts or in situations involving thermal inversions. Workshifts are frequently 12 to 18 h and can last for more than 24 h. In large fires, prolonged work shifts can last for many days. In wildland firefighting, it is not feasible to use a self-contained breathing apparatus; often the only respiratory protection used is a cotton bandana tied over the nose and mouth. Moreover, many of the tasks in wildland firefighting are physically demanding and require elevated pulmonary ventilation rates, which can result in substantial doses of smoke to the respiratory tract. Off-shift smoke exposures may occur as well, depending on the location of the base camp (where firefighters eat and sleep) in relation to the fire and the prevailing meteorology. With the intensity of smoke exposures, it is not surprising that respiratory problems accounted for about 40% of all medical visits made by wildland firefighters during the Yellowstone firestorm of 1988 (U.S. Department of Agriculture, 1989).

There have been several investigations of both exposures and health impacts of smoke exposure among wildland firefighters. Exposure assessment can represent a major logistical challenge, considering that the work often takes place on steep terrain in remote locations and may involve extreme physical exertion. In addition, exposure assessment must of necessity be limited to relatively few of the thousands of substances in biomass smoke. By extension, the few health studies that have been undertaken have not involved concurrent exposure assessment, but have focused on cross-shift or cross-seasonal respiratory effects.

Reinhardt and Ottmar (2000) undertook an exposure assessment of breathing-zone levels of acrolein, benzene, carbon dioxide, CO, formaldehyde, and $PM_{3.5}$ among firefighters at 21 wildfires in California between 1992 and 1995. Interestingly,

*www.who.int/docstore/peh/Vegetation_fires/Health_Guidelines_final_3.pdf

TABLE 4
Summary of selected epidemiologic studies of large-scale vegetation fires

Population	Endpoints measured	Results	Reference
All ages	Emergency room visits	Increased respiratory visits in communities exposed to fire smoke	(Duclos et al., 1990)
All ages	Emergency room visits, hospital admissions	Increased emergency-room visits and hospital admissions for asthma and bronchitis during fire period relative to same period in previous year	(Sorenson et al., 1999)
All ages	Acute respiratory distress hospital visits	Increase in acute respiratory distress inhalation therapy visits associated with indirect measure (sedimentation) of air pollution during sugar-cane burning season in Brazil	(Arbex et al., 2000)
All ages	Outpatient visits	Increased visits for asthma, upper respiratory tract symptoms, and rhinitis during vegetation fire episode periods of elevated PM_{10} in Malaysia	(Brauer, 1998)
All Ages	Outpatient visits, hospital admissions, mortality	Increase in PM_{10} from 50 to 150 $\mu g/m^3$ during vegetation fire episode periods associated with increase in outpatient visits in Singapore for upper respiratory tract symptoms (12%), asthma (37%), and rhinitis (26%). No increase in hospital admissions or mortality	(Emmanuel, 2000)
All Ages	Emergency room visits	Increased asthma visits with PM_{10} during episode of exposure to biomass burning emissions in Singapore	(Chew et al., 1995)
All Ages	Emergency room visits	No increase in asthma visits with PM_{10} during episode of exposure to bushfire emissions in Australia	(Copper et al., 1994)
All Ages	Emergency room visits	No increase in asthma visits with PM_{10} During episode of exposure to bushfire emissions in Australia	(Smith et al., 1996)
All Ages	Emergency room visits	Increased asthma visits associated with PM_{10}, especially for concentrations exceeding 40 $\mu g/m^3$	(Johnston et al., 2002)
All Ages	Physician visits for respiratory, cardiovascular, and mental illness	A 46 to 78% increase in physician visits for respiratory illness during a 3-wk forest fire period in Kelowna, British Columbia	(Moore et al., 2006)
All Ages	Hospital admission for respiratory illness	Daily hospital emission rates for respiratory illness increased with levels of PM_{10} for bushfire and nonbushfire periods	(Chen et al., 2006)
All ages, >65 yr	Mortality	0.7% (all ages) and 1.8% (ages 65–74) increases in adjusted relative risk of nontrauma mortality per 10-$\mu g/m^3$ increase in PM_{10} Kuala Lumpur, Malaysia, for 1996-1997, including vegetation fore episode period	(Sastry, 2002)
Adults with COPD	Symptoms	Significant increase in symptom index (dyspnea, cough, chest tightness, wheezing, sputum production) on two days of elevated PM2.5 (65 $\mu g/m^3$) relative to control days (14 $\mu g/m^3$). Days of elevated PM attributed to fire smoke by satellite imaging	(Sutherland, 2005)
Adults	Asthma medication, lung function, asthmatic and other respiratory symptoms	Increased prevalence of respiratory symptoms and various asthma indicators, decreased lung function post-rice stubble burning period relative to period prior to burning in three communities in Iran	(Golshan et al., 2002)
Adult military recruits	Blood markers of inflammation	Bone marrow stimulated to release immature polymorphonuclear leukocytes into blood during period of exposure to forest fire smoke relative to period following smoke exposure	(Tan et al., 2000)
Children	Respiratory hospital admissions	Increased pediatric respiratory hospital admissions associated with increased biomass smoke markers (potassium and black carbon) during sugar-cane burning season in Brazil	(Cancado et al., 2002)
Children	Lung function	Decreased lung function in children during vegetation fire episode compared to preepisode measurements	(Hisham-Hashim et al., 1998)

exposures to the gases were generally well below time-weighted average occupational health standards. However, some of the fires resulted in high-level peak exposures to heavy smoke. Respirable particle ($PM_{3.5}$) exposures on multiday fires averaged 0.72 mg/m^3 on the fireline, and 0.5 mg/m^3 over the work shift, with peak concentrations of 2.3 and 2.93 mg/m^3. The corresponding exposures to CO were 4.0 and 2.8 ppm, with peak (2-h time-weighted average [TWA]) exposures of 38.8 ppm and 30.5 ppm. The particle concentrations are about 10 to 30 times higher than 24-h average ambient air quality standards for $PM_{2.5}$ (currently 65 μg/m^3 in the United States).

Materna et al. (1992) also found extremely high particle exposures among California wildland firefighters during the 1987–1989 fire seasons. Table 5 presents their data on PM exposures. These investigators also sampled for 12 PAHs and found all below 1 μg/m^3. The highest CO levels were associated with tending gasoline-powered pumping engines rather than from smoke exposure per se. An aldehyde screen detected formaldehyde, acrolein, furfural, and acetaldehyde. Most levels were well below occupational exposure limits; however, formaldehyde (which was detected in all samples) in several instances exceeded such limits (maximum TWA [226 min] = 0.42 mg/m^3). In general, these studies demonstrate that of the various measured constituents of smoke, PM tends to be the most consistently elevated during wildland firefighting in relation to health-based exposure standards.

In the first report of cross-seasonal changes in respiratory symptoms and lung function in wildland firefighters, Rothman et al. (1991) examined 69 Northern California firefighters who were nonsmokers or former smokers who had not smoked in at least 6 mo. There were significant cross-seasonal increases in reported cough, phlegm, wheeze, and eye and nasal irritation. Only eye irritation, however, was significantly associated with firefighting activity ($r = .48$, $p < .001$), while the association of wheeze with firefighting in the last 2 wk of the study was of borderline significance ($r = .25$, $p = .07$). There were small, but statistically significant, declines in several measures of pulmonary function across the season, with the strongest relationships for the highest exposure category in the final week preceding the follow-up spirometry. The associations became weaker and less significant with the progressive inclusion of additional weeks prior to the spirometry. Across the 8-wk study, several lung function metrics exhibited significant declines, including FEV1 (−1.2%, confidence interval [CI]* −0.5, −2.0%), FEV1/FVC (−0.006, CI −0.001, −0.01), and although FVC also declined, this change was not significant. Those in the highest category for hours worked in the week preceding spirometry experienced larger decrements in lung function (FEV1 = −2.9% [130 ml] and FVC = −1.9% [101 ml]). These changes were not affected by adjustment for potential confounders (not specified). The use of a cotton bandana for respiratory protection was not associated with any measurable protection.

TABLE 5
Personal TWA particle exposures among California wildland firefighters

Particle metric	Site/activity	Mean (mg/m^3)	Range (mg/m^3)
TSP	Base camp/waiting in staging area	3.3	1.8–4.4
TSP	Fireline/mop-up	9.5	2.7–37.4
Respirable	Fireline/mop-up	1.8	0.3–5.1
Respirable	Prescribed burn	1.2	0.2–2.7

Note. Modified from Materna et al. (1992).

Liu et al. (1992) examined cross-season changes in pulmonary function and airway hyperresponsiveness in 63 wildland firefighters in northern California and Montana in 1989. They were tested before the start of the fire season and within 2 wk of discharge from service. Though pre- and post-season spirometric measurements were within the normal range for all participants, there were significant cross-seasonal declines in FVC, FEV1, and FEF25–75 of 0.09 L, 0.15 L, and 0.44 L/s, respectively. There was no significant relationship with any of the covariates measured, including smoking status, history of allergy, asthma, or upper/lower respiratory symptoms, specific firefighting crew membership, or seasonal versus full-time employment. Airway responsiveness to methacholine increased significantly across the fire season, which was not affected by gender, history of smoking, allergy, full-time versus seasonal employment, or crew membership. This study suggests that, in addition to persistent cross-seasonal changes in lung function, firefighting may also be associated with increased airway hyperresponsiveness, although the effect was not significant.

Letts et al. (1991) conducted a health survey of 78 wildland firefighters in Southern California. There were no changes in symptom prevalence cross-seasonally, nor were there any significant associations with exposure (defined as low, medium, and high, based on hours of work and weighted by visual estimates of smoke intensity). There were small, nonsignificant changes in FEV1 and FVC. The decrements in FEF25–75 and FEV1/FVC, however, were both significant (−2.3%, CI −4.2, −0.5% and −0.5%, CI −1.0, −0.1%). The changes in FEF25–75 showed a nonsignificant exposure-response trend ($p = 0.08$) of: 0.5%, −1.9%, and −4.7% for the low-, medium-, and high-exposure groups, respectively. Interestingly, however, there were no associations with the number of seasons of firefighting, days since the last fire, or age. Although these investigators concluded that there was limited evidence of cross-seasonal effects of firefighting on lung function, they indicated that the season in which their survey was conducted involved an atypically low number of firefighting hours. Moreover, the baseline was established in June, reportedly "before significant smoke exposure occurred,"

*All confidence intervals reported here are at the 95% level.

though the extent of firefighting preceding the initial measurement was not documented.

In addition to examining cross-seasonal lung function changes, Betchley et al. (1997) also examined cross-shift changes among forest firefighters in the Cascade Mountains of Oregon and Washington (Betchley et al., 1997). Among 76 workers examined at the beginning and immediately after prescribed burns, mean declines in FVC, FEV1, and FEF25–75 were 0.065 L, 0.150 L, and 0.496 L/s, respectively. These changes were significant even after adjusting for respiratory infections in the preceding 4 wk, smoking status, any "lung condition," and allergy. Examining cross-seasonal changes in 53 firefighters, the values for these same measures were 0.033 L, 0.104 L, and 0.275 L/s, respectively. The changes for FEV1 and FEF25–75 were significant, and remained so even after adjustment for the same potential confounders and effect modifiers. There were no significant cross-seasonal changes in respiratory symptoms. The cross-seasonal lung function measurements and symptom reports were taken, on average, 78 days after the last occupational firefighting activities of the season. In a subsequent analysis of a subset of these workers ($n = 65$) who had been working when several combustion products were measured, the lung function decrements observed were not found to be specifically associated with $PM_{3.5}$, acrolein, carbon monoxide, or formaldehyde (Slaughter et al., 2004).

Investigators in Sardinia compared lung function among 92 wildland firefighters with a "control" group of policemen (Serra et al., 1996). The testing was undertaken in late spring, just prior to the onset of the principal fire season. The two groups had identical mean values for FVC and TLC,* and showed no significant differences for FRC,† DLCO, or DLCO‡/TLC. The firefighters, however, demonstrated modestly lower lung function test results for FEV1, FEV1/FVC, FEF50, FEF25, and RV.** Although there were significant differences in age and height between the two groups (the firefighters were older and shorter, both of which would favor lower mean lung function), the significant differences in lung function remained after multivariate control for age, height, smoking status, and pack-year history for current smokers. The investigators found no relationship of pulmonary function with years of service or with the number of fires extinguished over their careers. Cough and expectoration were more common among firefighters, but these differences were not significant.

*Total lung capacity (TLC) is the volume of air contained in the lungs after maximal inhalation.

†Functional residual capacity (FRC) measures the amount of air remaining in the lungs after a normal tidal expiration.

‡Carbon monoxide diffusing capacity (DLCO) provides an assessment of the ability of gases to diffuse across the blood–gas barrier, that is, from the alveoli into the blood.

**Residual volume (RV) is the amount of air remaining in the lungs after a maximal exhalation.

Wildland firefighting can involve intermittent prolonged exposures to high concentrations of respirable particles, which consist of mixtures unique to each situation. Exposures to elevated levels of CO and respiratory irritants such as formaldehyde also occur, but respirable particles probably represent the principal exposure of concern. The few health studies conducted on such workers have documented cross-seasonal decrements in lung function, increased airway hyperresponsiveness, and increased prevalence of respiratory symptoms. Rothman et al. (1991) demonstrated that recent cumulative exposures were more strongly associated with greater changes in lung function than were more remote exposures. At least one study has also shown acute cross-shift spirometric changes as well (Liu et al., 1992). There has been no long-term follow-up of the respiratory health of wildland firefighters, however. Among municipal firefighters, chronic pulmonary dysfunction may result from repeated smoke exposure, particularly among those who do not use respiratory protective devices (Tepper et al., 1991; Sparrow et al., 1982). It is unknown whether cessation of exposure among wildland firefighters during the off-season may allow for recovery and reversibility of effects, in contrast to municipal firefighters, who can be exposed year-round. In any case, the relatively small effects demonstrated among firefighters cannot be quantitatively extrapolated to nonoccupational exposures, as the demands of the job require a degree of physical fitness and resilience far beyond that found in most of the general population.

Forest and Brush Fires

Several studies in North America have evaluated the health impacts associated with forest and brush fires. In the first study examining the effect of wildfire smoke on the general population, Duclos and colleagues evaluated the impact of a numerous large forest fires on emergency room (ER) visits to 15 hospitals in 6 counties in California (Duclos et al., 1990). The authors calculated observed-to-expected ratios of ER visits, based on the numbers of visits during two reference periods. During the approximately $2\frac{1}{2}$-wk period of observation, ER visits for asthma and chronic obstructive pulmonary disease increased by 40% ($p < .001$) and 30% ($p = .02$), respectively. Significant increases were also observed for bronchitis (observed [O]/expected [E] = 1.2, $p = .03$), laryngitis (O/E = 1.6, $p = .02$), sinusitis (O/E = 1.3, $p = .05$), and other upper respiratory infections (O/E = 1.5, $p < .001$). Exposure assessment was problematic, however, as few PM_{10} or other monitors were located downwind of the fires. The highest PM_{10} concentration measured was 237 $\mu g/m^3$. In contrast, several measurements of total suspended particles (TSP) exceeded 1000 $\mu g/m^3$; the highest recorded value was 4158 $\mu g/m^3$. Exposure to forest fire smoke can be unpredictable, changing with wind direction, intensity of the fire, precipitation, and other variables. The few air quality measurements available to these investigators could not serve to reliably characterize population exposures, which is a general limitation of all wildfire studies. In addition, this study was subject to other typical limitations of ER analyses related

to behavioral and economic factors (e.g., perceptions of illness severity, access to other health care providers, and availability of health insurance, with the latter more problematic in the U.S. than elsewhere).

Although no air pollutant concentrations were reported, the impact of wildfires in Florida on ER visits to eight hospitals in 1998 were compared to visits during the same 5-wk period in the previous year. From 1997 to 1998, ER visits increased substantially for asthma (91%), bronchitis with acute exacerbation (132%), and chest pain (37%), while visits decreased for painful respiration (27%) and acute bronchitis (20%). Though based on smaller numbers, there were modest changes in the number of hospital admissions (increases of 46% for asthma and 24% for chest pain) (Sorenson et al., 1999). Although this study suggests that wildfire smoke exposure resulted in increased ER visits for respiratory disease and symptoms, no firm conclusions are possible. There was only one reference period selected, which might not provide a stable basis for comparison, and no statistical testing was undertaken.

In a retrospective evaluation of the health impacts of a large wildfire in a northern California Native American reservation, visits to the local medical clinic for respiratory illness increased by 52% over the same period the prior year (Mott et al., 2002). During the ten weeks that the fire lasted, PM10 levels exceeded 150 μg/m^3 (24-h average) 15 times, and on 2 days the levels exceeded 500 μg/m^3. Weekly concentrations of PM_{10} were strongly correlated with weekly visits for respiratory illness during the fire year ($r = .74$), but not in the prior year ($r = -.63$). In a community survey of 289 respondents, more than 60% reported respiratory symptoms during the smoke episode; 20% reported symptoms persisting at least 2 wk after the smoke cleared. Individuals with preexisting cardiopulmonary diseases reported significantly more symptoms before, during, and after the fire than those without such illnesses. The investigators also retrospectively evaluated the efficacy of several public health interventions in symptom reduction: (1) filtered and unfiltered masks distributed free of charge; (2) vouchers for free hotel accommodations in towns away from the smoke to assist evacuation efforts; (3) high-efficiency particulate air (HEPA) cleaners distributed for residential use; and (4) public service announcements (PSAs) about exposure reduction strategies. Mott and colleagues found that increased duration of use of a residential HEPA air cleaner was associated with decreased odds of reporting increased symptoms (odds ratio [OR] 0.54, CI 0.32, 0.89), with an inverse trend of symptom reporting with increasing duration of use. Similarly, ability to accurately recall a PSA was also associated with reduced odds for respiratory symptoms. In contrast, there was no detectable beneficial effect of evacuation from smoky areas or of the use of masks. However, the timing and duration of evacuation were not optimal. On the days with the highest recorded smoke concentrations, over 80% of the subjects had not evacuated. That mask use was not protective is not surprising; the masks were distributed without fit testing and had variable filtration efficiencies. Moreover, none of the interventions was randomized, and in fact individuals with smoke-related health effects or a prior diagnosis of respiratory or cardiovascular disease were given priority to receive hotel vouchers and HEPA air cleaners. Finally, due to the retrospective nature of the investigation, recall bias may have affected the results based on the survey.

More recently, Sutherland and colleagues reported an increase in an index of respiratory symptoms (dyspnea, cough, chest tightness, wheezing, and sputum production) among a panel of 21 subjects with COPD associated with 2 days of elevated ambient particle levels resulting from a forest fire near Denver, CO. On the 2 days in which symptom scores were increased, average $PM_{2.5}$ concentrations increased to 63 μg/m^3 relative to an average of 14 μg/m^3 on control days (Sutherland et al., 2005). During this same fire as well as several other fires in Colorado, the indoor infiltration of particulate matter was measured and the effectiveness of room HEPA-filter air cleaners was assessed in a total of eight homes. A decrease in $PM_{2.5}$ concentrations of 63–88% was measured in homes in which air cleaners were operated, relative to homes without air cleaners. In the homes without the air cleaners, measured indoor $PM_{2.5}$ concentrations were 58–100% of the concentrations measured outdoors (Henderson et al., 2005).

Moore and colleagues assessed the impact of elevated concentrations of $PM_{2.5}$ associated with forest fires on outpatient physician visits for respiratory disease. Two large fires burning adjacent to urban areas in British Columbia, Canada, resulted in intermittent elevations (140–200 μg/m^3) in daily average $PM_{2.5}$ concentrations over a 5-wk period in August and September 2003. In the city with the highest levels of PM and that was closest to a fire, weekly physician visits for respiratory disease were increased approximately 45–80% relative to average rates corresponding to those weeks during the previous 10 yr. No statistically significant increases were observed in the city with lower fire-related PM increases and neither city experienced elevated physician visits for cardiovascular diseases (Moore et al., 2006). However, as many patients experiencing symptoms characteristic of acute cardiovascular events go directly to a hospital emergency department, it is possible that the health database used in this investigation may not have been capable of identifying circulatory outcomes of interest during the study period.

During 1994, bush fires near Sydney, Australia led to elevated PM_{10} levels (maximum hourly values of approximately 250 μg/m^3) for a 7-day period. Two studies of asthma emergency room visits during the bushfire smoke episode failed to detect any association with air pollution (Copper et al., 1994; Smith et al., 1996). The report by Copper et al. (1994) was in the form of a letter to *The Lancet*, with few details provided. The investigators examined only three inner-city hospitals, preferring to avoid the influence of "patients who presented with direct effects of smoke inhalation," which might have occurred had they included hospitals with catchment areas closer to the fires. They compared the numbers of asthma ER visits for the week before the bushfires (January 1–8), the fire period (January

9–20), and afterward (January 21–31), and found no difference among the 3 periods. These comparisons were based on relatively small numbers, however, with fewer than 100 visits for asthma during the entire month for all 3 hospitals. The report by Smith et al. (1996) involved a comparison of the proportions of asthma to total ER visits to seven hospitals during the week of high smoke levels compared to the same week the prior year. There was no difference in these proportions, nor was there a relationship between the maximum daily nephelometric particle measurement and the number of asthma ER visits in multiweek regression models. Although it appears that the bushfire smoke did not have an impact on asthma ER visits, this study is limited by the use of a single reference period. In addition, the regression analysis is likely to have had very limited statistical power, with relatively few days of observation.

A recent analysis of these same fires and lung function (measured as peak expiratory flow rate [PEFR]) did not detect any association between either PM_{10} levels or an indicator variable representing the fire period and evening PEFR in 25 asthmatic children, although 20 children without airway hyperreactivity showed a significant decrease in PEFR with increasing same-day PM_{10} concentrations (Jalaludin et al., 2000). Whether this represents a true lack of association or an artifact of experimental design is difficult to ascertain. Thirty-two children in this analysis were recruited during the week of the fire. There did not seem to be any examination of whether there was a learning period for these children (during which the initial PEFR measurements might have been more variable), nor was there any discussion of the quality control for recording the measurements, or even what the PEFR protocol was. Of the 32 children (mean age = 9.2 yr), 25 had a physician's diagnosis of asthma; however, only 12 of the 32 had evidence of airway hyperresponsiveness, which is considered a hallmark of asthma. Although the regression model included indicator variables for use of asthma medications, there could nonetheless still have been residual confounding by medication use. In other words, the use of asthma medications might still have had enough of an effect on lung function to obscure a relationship between PEFR and smoke exposure, despite the attempt to control for this influence statistically. Finally, due to the timing of subject recruitment, it is not clear how many child-days of observation during the fires actually contributed to the analysis. The reported data suggest that this study is likely to have had very limited statistical power.

The results from these studies appear to conflict with those conducted in North America. As noted earlier, however, all have significant limitations that suggest caution in generalizing the results. It is also possible that there is less respiratory toxicity from bushfire smoke than from forest fire smoke due to chemical and physical differences between the two. Two more recent studies from Australia have reported associations between bushfire smoke and health impacts. For example, a study undertaken at the only hospital in Darwin (northwestern Australia) evaluated the association between daily asthma ER visits (adjusted for influenza and day-of-week effects) and measured PM_{10} over a 7-mo period, which included 2 bushfire smoke episodes. Bushfires represent the principal regional source of significant levels of air pollution in Darwin during the dry season in which this investigation took place. Increased asthma visits were associated with PM_{10} concentrations, especially for days on which PM_{10} concentrations exceeded 40 $\mu g/m^3$ (Johnston et al., 2002). The adjusted rate ratio per 10-$\mu g/m^3$ increase in PM_{10} was 1.20 (CI 1.09, 1.34). The largest association was observed for a 5-day lag, comparing days when PM_{10} exceeded 40 $\mu g/m^3$ with those on which PM_{10} was less than 10 $\mu g/m^3$ (adjusted rate ratio = 2.56 [CI 1.60, 4.09]). Unlike the prior studies of biomass smoke conducted in Australia, this investigation clearly had adequate statistical power to detect an association between PM and asthma visits. Though the time-series analysis did not control for pollen or mold, which are not routinely monitored in Darwin, the investigators considered it "extremely unlikely" that either of these would vary systematically with bushfire smoke. This assessment by the authors is probably true, but without analyzing the smoke for these bioaerosols, it is not possible to state definitively that they did not confound the results.

Chen et al. (2006) evaluated the relationship between respiratory hospital admissions in Brisbane, Australia, and particulate matter (PM_{10}) for a $3\frac{1}{2}$-yr period that included 452 days (35% of the study period) categorized as days with bushfires (>1 ha burned) in the study region, based on review of fire records. During the bushfire periods, the median of daily respiratory hospital admissions in Brisbane was 34 (range: 9–76) and the daily mean PM_{10} was 18.3 $\mu g/m^3$ (range: 7.5–60.6 $\mu g/m^3$), compared to a median of 32 respiratory hospital admissions per day (range: 7–91) and daily mean PM_{10} of 14.9 $\mu g/m^3$ (range: 4.9–58.1 $\mu g/m^3$) during non-bushfire days. The authors categorized PM_{10} values into low (<15), medium (15–20) and high (>20), rather than using a continuous variable for PM_{10}. This may have resulted in a loss of information about the potential impacts of extreme values and possibly a bias toward (or away from) the null hypothesis of no effect (Dosemeci et al., 1990). In addition, the authors noted that the single PM_{10} monitor used in this study was upwind of many of the fires, indicating that the populations affected were exposed to higher PM_{10} and smoke concentrations than those reported, which could have resulted in an overestimate of the magnitude of effect. Nonetheless, for both bushfire and nonbushfire periods, increased PM_{10} concentrations were associated with increased relative risks for respiratory hospital admissions, with some suggestion of slightly stronger associations on the days with the highest daily PM_{10} concentrations (i.e., >20 $\mu g/m^3$) on bushfire (RR = 1.19, CI 1.09, 1.30, for same-day PM_{10} concentrations) versus nonbushfire (RR = 1.13, CI 1.06, 1.23) days. The results of this study are consistent with many other time-series investigations of PM and, at a minimum, indicate that the associations between PM_{10} and respiratory health admissions on bushfire days were at least as great as those on days when other sources of PM_{10} predominated.

Major regional episodes of air pollution from vegetation fires in Southeast Asia have been the subject of several investigations

and surveillance programs. An analysis of emergency room visits for asthma in Singapore during a 1994 episode of regional pollution resulting from forest and plantation fires reported an association between PM_{10} and emergency room visits for childhood asthma. During the "haze" period, mean PM_{10} levels were 20% higher than the annual average. Although a time-series analysis was not conducted, the authors suggested that the association remained significant for all concentrations above 158 $\mu g/m^3$ (Chew et al., 1995).

Reports from surveillance monitoring activities conducted during major Southeast Asian episodes in 1997 and 1998 also indicated effects on health care utilization. In Singapore, for example, there was a 30% increase in hospital attendance for "haze-related" illnesses: A time-series analysis indicated that a PM_{10} increase of 100 $\mu g/m^3$ was associated with 12%, 19%, and 26% increases in cases of upper respiratory tract illness, asthma, and rhinitis, respectively. It is not clear why rhinitis constituted a separate diagnostic category in this investigation, rather than being included with upper respiratory tract illness. This analysis did not observe any significant increases in hospital admissions or mortality (Emmanuel, 2000). Similar findings were also observed in Malaysia (Brauer, 1998; Leech et al., 1998).

Preliminary results from a study of 107 Kuala Lumpur schoolchildren found statistically significant decreases in lung function between preepisode measurements in June–July 1996 and measurements conducted during the haze episode in September 1997 (Hisham-Hashim et al., 1998). A convenience sample questionnaire survey conducted in Indonesia during the 1997 haze episode also suggested acute impacts on respiratory and cardiovascular symptoms (Kunii et al., 2002). Of 539 interviewees, 91% reported respiratory symptoms (cough, sneezing, runny nose, sputum production, or sore throat), 44% reported shortness of breath on walking, 33% reported chest discomfort, and 23% reported palpitations. Although the numbers were small, respondents with asthma or heart disease tended to experience a greater proportion of moderate and severe symptoms relative to those without preexisting disease. Despite these findings, however, the cross-sectional nature of the sampling and reporting and the absence of an unexposed reference population weaken any inference of a causal relationship between the smoke and these symptoms.

In another study of the 1997 Southeast Asia haze episode, Tan and colleagues (2000) obtained blood samples at weekly intervals from 30 Singaporean military recruits who followed standardized outdoor routines during the episode. The mean 24-h PM_{10} level during the episode was 125.4 $\mu g/m^3$. Analyzing the numbers of immature inflammatory cells (polymorphonuclear cells or PMNs) in the subjects' blood in relation to daily measures of several pollutants, these investigators found the strongest relationship with same-day PM_{10}, though a 1-day lag of this metric was also statistically significant. Although these results are insufficient to establish a causal relationship, they suggest that smoke inhalation stimulated the bone marrow to eject immature PMNs into the circulation.*

Recently, Mott et al. reported several related examinations of the Indonesian fires on hospitalizations and survival (Mott et al., 2005). In analyses of the fire period (August through October 1997) compared with a 31-mo baseline period (January 1995 through July 1997), they reported fire-related increases of 50% and 83% for admissions due to COPD and asthma among individuals aged 40 to 64, and an increase of 42% for COPD among individuals aged 65 and older. In a time-series analysis in which the baseline period was used to generate predicted numbers of hospitalizations by age group for the fire period, the observed admissions were significantly elevated for several respiratory categories (asthma and COPD), principally among the 40–64 yr age stratum. There was no significant elevation of admissions for total circulatory diseases, though observed ischemic heart disease (IHD) admissions ($n = 6$) for the 18–39 yr age stratum were slightly above the 95% upper limit predicted ($n = 5.7$). However, the small numbers involved, coupled with the absence of a significant elevation of IHD admissions in older age groups, suggest caution in interpreting this relationship. Finally, Mott and colleagues examined repeat hospitalizations and survival during the fire period compared with the corresponding periods in 1995 and 1996. Individuals over age 65 with prior hospitalizations for any cardiorespiratory disease, any respiratory disease, or COPD in particular were more likely to be re-hospitalized during the fire period, especially for respiratory causes, compared with the corresponding periods in 1995 and 1996. In particular, individuals with a prior history of hospitalization for COPD were more likely to be rehospitalized for COPD or die from any cause during the fire period (an approximately 44% increase for both outcomes combined); this phenomenon was only manifest when smoke levels exceeded approximately 150 $\mu g/m^3$.

Only one other study has evaluated the impacts of air pollution from vegetation fires on mortality. Sastry (2002) evaluated the population health effects in Malaysia of air pollution generated by a widespread series of fires that occurred mainly in Indonesia between April and November 1997. The results showed that the haze from these fires was associated with deleterious effects on population health in Malaysia and were in general agreement with the mortality impacts associated with particles in urban air (Sastry, 2002). A 10-$\mu g/m^3$ increase in PM_{10} measured in Kuala Lumpur was associated with 0.7 % (all ages) and 1.8% (ages 65–74) increases in adjusted relative risks of nontraumatic mortality. Visibility-based estimates of PM concentrations in Kuching, a city closer to the fire sources, were also associated with increased mortality.

*In a subsequent toxicological examination involving rabbits, these same investigators found that repeated PM_{10} instillations into the respiratory tract resulted in increased production of PMNs in the bone marrow and an acceleration of their release into the blood, both of which were associated with the numbers of particles ingested by the animals' alveolar macrophages (Mukae et al., 2001).

With the exception of three of the Australian bushfire investigations, all of which have significant structural limitations, the epidemiologic studies of indoor and community exposure to biomass smoke indicate a generally consistent relationship between exposure and increased respiratory symptoms, increased risk of respiratory illness, including hospital admissions and emergency room visits, and decreased lung function. Several studies suggest that asthmatics are a particularly susceptible subpopulation with respect to smoke exposure, which is consistent with the results of many studies of the impacts of ambient air pollution. The effects of community exposure to biomass air pollution from wildfires on mortality have not been sufficiently studied to support general conclusions.

Agricultural Burning

There have been few studies of the impacts of agricultural burning, despite growing concern about its potential impact on human health (Tenenbaum, 2000). In one Canadian study, 428 middle-aged subjects with slight-to-moderate airway obstruction were surveyed about respiratory symptoms during a 2-wk period of exposure to straw and stubble combustion products. During the exposure period, 24-h average PM_{10} levels increased from 15–40 $\mu g/m^3$ to 80–200 $\mu g/m^3$. One-hour levels of CO and nitrogen dioxide reached 11 ppm and 110 ppb, respectively. Total volatile organic compound levels increased from preepisode levels of 30–100 $\mu g/m^3$ to 100–460 $\mu g/m^3$ during the episode. Although 37% of subjects were not bothered by smoke at all, 42% reported that several respiratory 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. Subjects with asthma and chronic bronchitis were more likely to be affected, and women appeared to be more susceptible than men for several symptoms (cough, shortness of breath, nocturnal awakening) (Long et al., 1998). In contrast, current cigarette smokers reported significantly fewer symptoms than the former smokers constituting the rest of the study population. This study indicates that, besides woodsmoke, biomass air pollution from agricultural burning is associated with increased respiratory symptoms among a susceptible population with preexisting lung disease.

A time-series study in California suggested that agricultural burn smoke was associated with serious exacerbations of asthma. The association between asthma hospital admissions and the burning of rice field stubble and waste rice straw was examined in Butte County, California, over a 10-yr period (Jacobs et al., 1997). Although burning was not associated with any measurements of major air pollutants (probably because monitors were not sited to provide optimal measurement of burn smoke), burn acreage was significantly associated with an increased risk of asthma hospitalization and showed an exposure-response trend. The greatest risk of hospitalization was observed on days when 500 or more acres were burned (relative risk [RR] 1.23, CI 1.09, 1.39).

A recent cross-sectional study in three rural villages in Iran also evaluated the relationship between rice stubble burning and respiratory morbidity, especially asthmatic symptoms (Golshan et al., 2002). During a burning period lasting several weeks, PM_{10} concentrations doubled. Based on responses to a physician-administered survey before and after this episode, the investigators reported significant increases in the prevalence of asthma attacks, use of asthma medications, the occurrence of nocturnal sleep disturbances, and other respiratory symptoms among 994 residents of an agricultural region. Several measures of pulmonary function also decreased significantly.

The relationship of rice stubble burning with asthma was also studied in Niigata prefecture, Japan (Torigoe et al., 2000). In this study, measured PM_{10} concentrations were associated with monthly asthma hospital admissions and ER visits in a region where rice straw burning emissions led to high particle concentrations during the September–October burning season. During the period 1994–1998, both asthma ER visits and hospitalizations were significantly higher in September than in almost all other months of the year except October and November (for ER visits; hospitalizations in the month of December were also not significantly different from September). Although PM_{10} levels were not associated with monthly ER visits for asthma, the investigators reported a significantly higher number of asthma ER visits on days when rice straw burning occurred and the following 2 days (7.1 ± 3.9) versus other days (4.5 ± 3.3). The latter comparison would have better time resolution than an analysis of monthly average of asthma exacerbations, and should probably be accorded greater weight. Although this investigation also involved a parental questionnaire suggesting more asthma exacerbations in children during the rice burning season than at other times of the year, an autumn peak in asthma flares is also common in other parts of the world where rice burning does not occur. In general, multiple findings in this investigation are suggestive of a rice smoke effect on asthma, but several limitations of the study design constrain both causal inference and the generalizability of the findings.

A metric commonly used as a surrogate of exposure to biomass smoke is the amount of agricultural land burned, as in the Butte County, California, study mentioned earlier (Jacobs et al., 1997). Norris (1998) evaluated the association between acres of grass seed residues burned around Spokane, WA, and visits to local emergency departments for asthma. (Norris, 1998). During one burning event, peak PM_{10} concentrations in Spokane reached 100 $\mu g/m^3$ (Figure 1). Using a bivariate indicator (20 days with >499 acres burned) for the exposure surrogate, an association with increased emergency department visits for children was observed (RR 1.30, CI 1.08, 1.58).

A few studies have specifically examined air pollution and health effects associated with the burning of sugar cane. In Brazil, daily indirect measurements (sedimentation of particle mass) of air pollution during the sugar cane burning season in 1995 were associated with the number of patients visiting hospitals for inhalation therapy for acute respiratory distress (Arbex

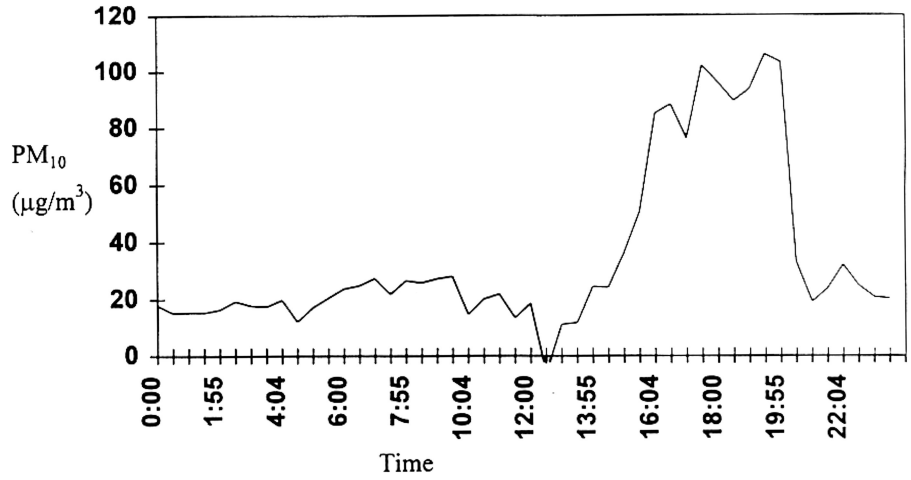

FIG. 1. PM_{10} measured downwind of a grass-burning event in Spokane, WA, at the Rockwood residential monitoring site (September 1994). *Note*: Modified from Norris (1998).

et al., 2000). The relative risk of such a hospital visit associated with an increase of 10 mg in the sediment was 1.09 (1–1.19); this association displayed an exposure-response relationship as well. Boopathy and colleagues presented a descriptive analysis of asthma hospital visits to a medical center in Houma, LA, during 1998–1999 (Boopathy et al., 2002). The area served by this medical center accounted for approximately 27% of Louisiana's sugar-cane cultivation during this period. Although no air pollution measurements were available, asthma hospital visits increased dramatically during the October–December sugar-cane burning season. As noted earlier, however, an autumn peak in asthma exacerbations is common, and respiratory infections (the main precipitating factor for severe asthma attacks) also typically increase in frequency during this time. Therefore, it would be inappropriate to infer a causal relationship between sugar-cane burning and asthma hospital visits based on this descriptive study. Boeniger and coworkers (1991) conducted an exposure assessment of smoke during sugar-cane harvesting in Hawaii in 1987 (Boeniger et al., 1991). They collected both area and personal samples. The concentration of PM increased by at least 20 and up to 70 times the measured background levels at the sampling sites chosen, but were highly variable, making exposure assessment difficult. A subsequent study of Hawaiian sugar-cane workers, however, reported no elevated morbidity or mortality rates or decreased lung function (Miller et al., 1993).

Together, these epidemiologic studies suggest that exposure to products of agricultural burning, specifically the burning of rice stubble/straw, may be associated with exacerbation of asthma. In a chamber study of smoke generated by controlled burning of rice stubble straw, Solomon and colleagues exposed 13 adults with allergic rhinitis (age range 24–55) at rest to filtered air, rice-straw smoke (RSS) at 200 $\mu g/m^3$ or at 600 $\mu g/m^3$ for 30 min, or RSS at 200 $\mu g/m^3$ on 3 consecutive days. Bronchoalveolar lavage (BAL) was conducted at 6 h postexposure. Of a variety of cell types and cytokines measured in BAL fluid, the investigators found a near doubling of epithelial cells only after the 3-day exposure, but no difference from filtered air exposures in total white blood cells, macrophages, PMNs, lymphocytes, eosinophils, or interleukin-8 under any of the RSS exposure conditions. Interestingly, this effect was not observed at a higher concentration (600 $\mu g/m^3$) delivered over a shorter time interval, suggesting that repeated exposures may be necessary, at least among individuals with allergic rhinitis (Solomon, 2003).

Several studies have also reported an increased risk (odds ratios of 1.5–2.5) of lung cancer and mesothelioma among sugar-cane workers, although specific job activities were not evaluated and exposure measurements were not made (Rothschild & Mulvey, 1982; Brooks et al., 1992). A case-control study (118 histologically confirmed lung cancer cases and 128 controls with other cancers matched by age, sex, district of residence, and timing of diagnosis) in India found an increased risk of lung cancer in sugar-cane workers associated with postharvest burning (odds ratio = 1.8, 95% CI 1.0−3.3) (Amre et al., 1999). It has been suggested that this association may be due to the liberation of asbestos-like biogenic silica fibers in sugar cane smoke.

RESIDENTIAL WOODSMOKE IN DEVELOPED COUNTRIES: EXPOSURE AND HEALTH STUDIES

During winter in areas where wood is available, woodburning is common in essentially every part of the developed world for household heating. It is also popular for recreational use in fireplaces. This has implications for area-wide ambient levels and indoor pollution as well as what can be called "neighborhood" pollution, outdoors but sometimes localized in neighborhoods where woodstoves are in use. Here we do not attempt to summarize the evidence on the contribution of woodsmoke to ambient pollution in the developed world, but provide typical examples in different regions.

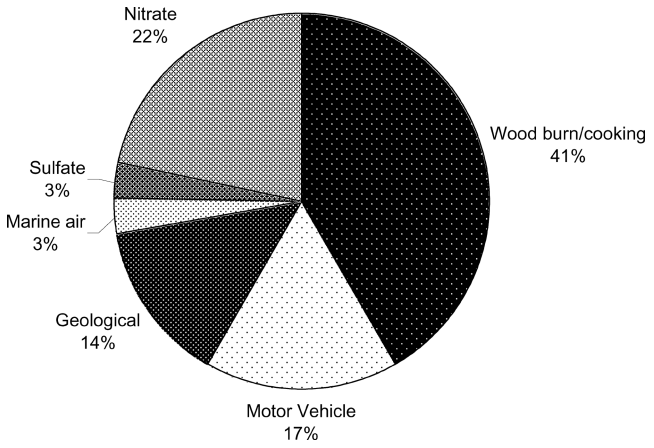

FIG. 2. Source apportionment results for wintertime PM_{10} in San Jose, CA (1993–1994). *Note*: Modified from Fairley (1990).

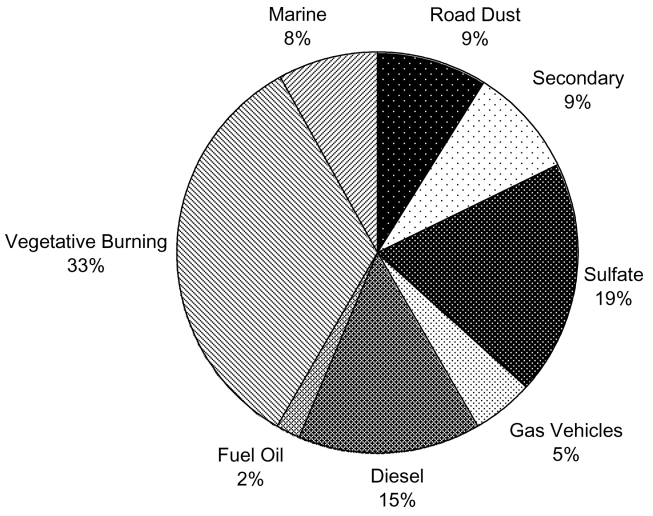

FIG. 3. Source apportionment results for PM_{10} in Seattle, WA (1996–1999). *Note*: Modified from Maykut et al. (2003).

Ambient and Neighborhood Levels

Source apportionment studies indicate that woodsmoke is a major source of ambient PM during winter months in several parts of the United States. Figure 2 shows data from San Jose, CA, that indicate that 42% of the PM_{10} during winter months could be attributed to wood burning (Fairley, 1990). Chemical mass balance receptor modeling of fine particles in Fresno and Bakersfield, CA during wintertime identified both hardwood and softwood as sources of PM and organic compounds (Schauer & Cass, 2000), which were likely to have been due to residential woodburning.

Outdoor PM levels in Seattle, WA; are also heavily influenced by residential woodstoves. Data from 3 years of sampling in Seattle were analyzed for sources using positive matrix factorization (PMF) (Maykut et al., 2003). The PMF analysis found that vegetative burning contributed 34% to the total sources of PM in Seattle over 3 yr (Figure 3).

Another study utilized a large data set from a 2-year exposure assessment and health effects panel study in Seattle during September 2000–May 2001. Indoor, outdoor, personal, and fixed-site PM monitoring data were available. The samples were analyzed for elements using XRF, and positive matrix factorization (PMF) was used to apportion sources (Larson et al., 2004). Five sources contributed to indoor and outdoor samples: vegetative burning, mobile emissions, secondary sulfate, a chlorine source, and a crustal-derived source. Vegetative burning contributed the largest fraction of PM mass in all the samples (35%, 49%, and 62% in indoor, outdoor, and personal mass, respectively).

The distribution of particle-phase organic compounds has been measured in communities with children participating in the Southern California Children's Health Study (CHS) (Manchester-Neesvig et al., 2003). Concentrations of levoglucosan, a good tracer for woodsmoke aerosol, were seen in all 12 CHS communities (Figure 4). The average concentration increased substantially in the winter, as would be expected for woodsmoke emissions. The concentrations of levoglucosan were highest at the Atascadero site, which is about 15 miles inland. Earlier, these investigators identified two additional sugar anhydride tracers of woodsmoke (galactosan and mannosan) in a study of urban sites in the San Joaquin Valley, California (Nolte et al., 2001). These data may allow a separate estimation of the effects of woodsmoke exposure on health outcomes.

In Canada, with cold winters and abundant forests, woodsmoke is a major source of particle emissions. Figure 5 shows that household woodsmoke is responsible for more than 30% of annual PM emissions in 8 provinces and more than 10% in the remaining 4. It is also more responsible for a significant fraction of VOC emissions.*

Christchurch, New Zealand, is another city impacted by woodsmoke. It is estimated that more than 90% of wintertime ambient PM comes from heating stoves and open fires burning wood (McGowan et al., 2002). Frequent periods of air stagnation compound the problem by trapping PM near the ground, and local meteorologists estimate that the relatively even mixing results in fairly homogeneous PM exposure to the population.

Emissions inventories in Launceston, Australia, indicate that household woodburning accounted for 85% of annual PM_{10} emissions in 2000 and that a 50% reduction would be needed in order the city to meet air quality standards.

Source apportionment studies in Denmark show that household woodburning was responsible for 47% of national $PM_{2.5}$ emissions in 2002. In addition, household woodburning grew by about 50% during the 1990s, as compared to only 7% for total energy use.

A recent phenomenon in the United States has been the use of backyard wood-fired boilers for heating homes, which have not

*http://www.ec.gc.ca/science/sandejan99/article1_e.html

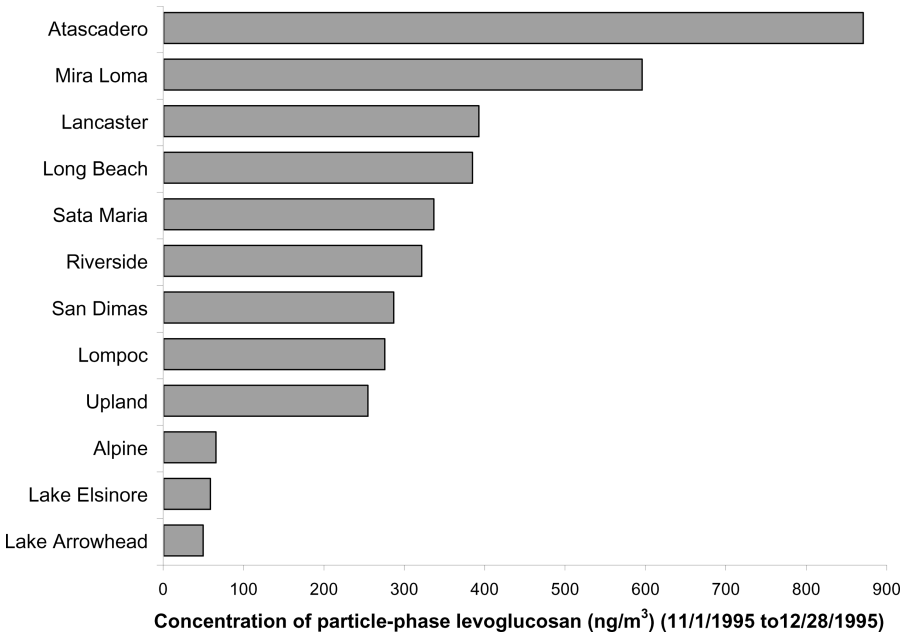

FIG. 4. Spatial distribution of winter time levoglucosan in Southern California (1995–1996). *Note*: Modified from Manchester-Neesvig et al. (2003).

been regulated and often produce substantial pollution locally (Johnson, 2006).

Indoor Levels

Relatively few measurements seem to have been reported of indoor concentrations of woodsmoke in developed-country households. A case-control study of woodstoves and health in Navajo children in Arizona did include measurements of indoor concentrations of respirable particles (PM_{10}) in 90 households (Robin et al., 1996). Cases were children from birth to 24 months of age hospitalized with acute respiratory illnesses and controls that were not hospitalized. Sixty-three percent of the cases had wood stoves in their homes, compared with 51% of the controls. TWA concentrations (15-h) ranged from 22.2 μg/m^3 in houses that used gas or electricity to 100 μg/m^3 in homes that heated with wood alone.

Early studies of woodsmoke health effects often used the presence or absence of a wood stove in the home as the

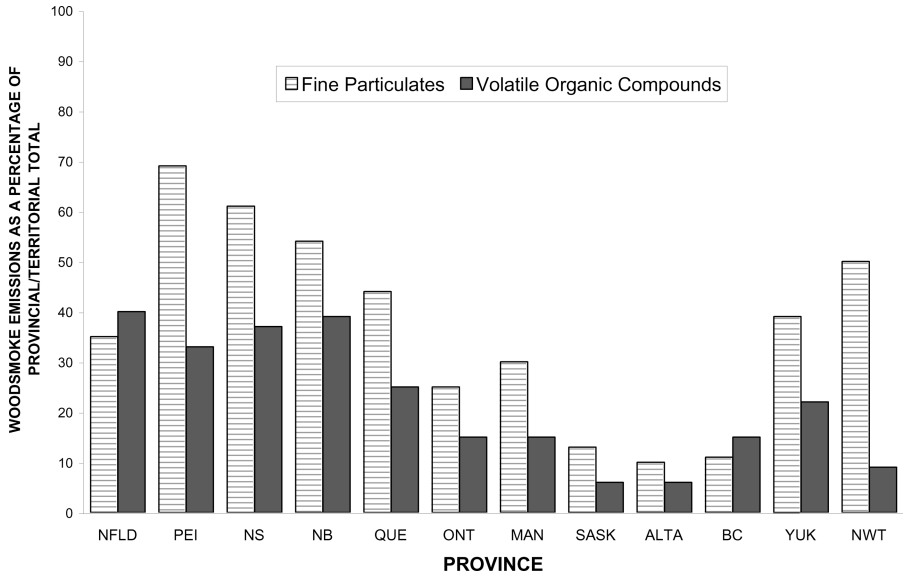

FIG. 5. Importance of woodsmoke emissions in Canada by province. *Note*: Data from Health Canada. See footnote on page 86.

indicator of exposure (see next section). Due to penetration of woodsmoke particles indoors, these exposures may not be due exclusively to indoor sources of woodsmoke. It has been shown in a woodsmoke-impacted community that particles readily penetrate inside residences (Anuszewski et al., 1998). The contribution from outdoor-generated particles to indoor and personal exposure in Seattle, WA, residences has been estimated using a recursive model (Allen et al., 2003, 2004). Nonlinear regression was used to estimate particle penetration, particle decay rate, and particle infiltration. Estimates of particle infiltration agree well with those derived from sulfur-tracer methods ($R^2 = .78$) (Sarnat et al., 2002). In a sample of 44 residences, outdoor-generated particles accounted for an average of $79 \pm 17\%$ of the indoor PM concentration. These data suggest that in epidemiologic studies of associations between health outcomes and outdoor PM, much of the exposure to outdoor particles can occur inside the home. Other factors, such as the age of the house, opening of windows, and air conditioning, can affect penetration. In one study, home air conditioning was associated with lower penetration of outdoor particles; moreover, the associations between PM_{10} and hospital admissions were lower in cities with a higher prevalence of air conditioning (Janssen et al., 2002). These findings imply that even woodstoves and fireplaces operating well that vent most smoke outside may produce substantial exposures through penetration back into the house, a characteristic of "neighborhood pollution."

Health Effects of Residential Woodburning*

To date, only a single controlled exposure study of human exposure to woodsmoke itself seems to have been published (Barregard et al., 2006; Sallsten et al., 2006). Thirteen subjects were exposed to realistic concentrations of woodsmoke (200–300 $\mu g/m^3$ $PM_{2.5}$) generated under controlled conditions for two 4-h sessions, spaced 1 wk apart. In this study, exposure to woodsmoke resulted in small exposure-related changes in levels of inflammatory mediators and coagulation factors. In addition, evidence of increased free radical-mediated lipid peroxidation was observed in 9 of the 13 subjects. Although this is the only controlled study of woodsmoke exposure published to date and it observed a small number of subjects, it is suggestive of woodsmoke-associated systemic inflammatory effects.

The majority of information regarding direct human health effects associated with woodsmoke exposure is derived from a relatively large number of epidemiologic studies have documented respiratory effects of residential woodburning, especially in children. One of the earliest studies was conducted in Michigan by Honicky et al., who compared respiratory symptoms in 31 children who lived in homes with wood stoves with 31 children who lived in homes without wood stoves (Honicky et al., 1985). Symptoms were categorized as mild, moderate, and severe. The two groups did not differ with respect to mild symptoms, but differed significantly for severe symptoms ($p < .001$). A similar study was conducted in Boise, ID, by Butterfield et al., where respiratory symptoms were tracked in 59 children under the age of $5\frac{1}{2}$ years during a winter season (Butterfield et al., 1989). Symptoms such as wheeze, cough, and nocturnal awakening were associated with presence of a woodstove.

Morris et al. (1990) evaluated the impact of indoor woodsmoke child health on a Navajo reservation in Arizona by assessing use of a well-child clinic (Morris et al., 1990). For 58 case-control pairs, the odds ratio (OR) for a serious acute lower respiratory infection (ALRI: bronchiolitis or pneumonia) associated with the presence of a wood stove was 4.2 ($p < .0012$). A more recent case-control study among slightly younger (1–24 mo) Navajo children reached similar, but nonsignificant conclusions (OR 5.0, CI 0.6, 42.8) (Robin et al., 1996). Measured 15-h PM_{10} levels above 65 $\mu g/m^3$ were more frequent in households with wood cookstoves (OR 7.0, CI 0.9 to 56.9). Adjustment for potential confounders (including the number of children living in the house, lack of running water or electricity, difficulty with transportation to the clinic, type of home, and the temperature on the PM_{10} sampling day) had relatively little effect on the magnitude of the associations. The low number of cases (45) likely affected the precision of the estimates, reducing the investigators' ability to detect significant associations between use of wood-burning devices and respiratory infections. It is noteworthy, however, that the magnitude of effect exceeds those generally found in developing-country studies of ALRI in children (discussed later).

A questionnaire study of respiratory symptoms compared residents of 600 homes in a high woodsmoke area of Seattle, WA, with 600 homes (questionnaires completed for one parent and two children in each residence) of a low woodsmoke area (Browning et al., 1990). PM_{10} concentrations averaged 55 and 33 $\mu g/m^3$, respectively. When all age groups were combined, no significant differences were observed between the high- and low-exposure areas. There were, however, statistically significantly higher levels of congestion and wheezing in 1- to 5-year-olds between the 2 areas for all three questionnaires (1 baseline questionnaire and 2 follow-up questionnaires which asked about acute symptoms). This study supports findings from the other investigations suggesting that young children are particularly susceptible to adverse effects of woodsmoke.

In Seattle, WA, 326 elementary school children were studied during the heating seasons of 1988–1989 and 1989–1990 (Koenig et al., 1993). Monthly or bimonthly spirometry values were collected during the school year. PM exposure was measured by light scattering using nephelometers. The exposure metric used was the 12-h nighttime average (7 p.m. to 7 a.m.) to reflect the hours when woodsmoke is most elevated. A random-effects statistical model compared changes in FEV_1 and FVC with changes in the light-scattering coefficient. The 26 children with asthma showed a significant decrement (18 ml/$\mu g/m^3$

*A thorough summary of emissions from woodsmoke was published several years ago (Larson & Koenig, 1994).

$PM_{2.5}$) for both measures of lung function. Children without asthma showed no significant changes in lung function associated with PM values.

A companion study evaluated the impact of particulate matter on emergency room visits for asthma in Seattle (Schwartz et al., 1993). A significant association was observed between PM_{10} particle levels and emergency room visits for asthma. The mean PM_{10} level during the 1-yr 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% of the fine particle mass in Seattle residential neighborhoods is from woodburning.

Overall, health effects research in Seattle shows associations between $PM_{2.5}$ and lung function decrements in children (Koenig et al., 1993), visits to emergency departments for asthma (Norris et al., 1999), hospitalizations for asthma (Sheppard et al., 1999), and increases in asthma symptoms in children (Yu et al., 2000), as well as increases in exhaled nitric oxide (Koenig et al., 2003, 2005). Since woodburning is the primary source of fine particles in the Seattle airshed, the health effects studies suggest a causal relationship.

Lung function in 410 schoolchildren in Klamath Falls, OR, was studied during winter in high- and low-exposure areas were studied where it has been estimated that woodsmoke accounts for as much as 80% of winter period PM_{10} (Heumann et al., 1991). Winter PM_{10} levels in the high exposure area ranged from approximately 50 to 250 $\mu g/m^3$, while levels in the low exposure area ranged from 20 to 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.

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 et al., 1990). Acute lung function decrements measured in 375 children were associated with increased levels of particulates. The 24-h averages ranged from 43 to 80 $\mu g/m^3$ and from 14 to 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% of the $PM_{3.5}$ to woodsmoke (Larson & Koenig, 1994).

Another study examined the relationship of woodstoves to otitis media and asthma in a case-control study of home environmental air pollutants in Springville, NY (Daigler et al., 1991). That study found use of woodstoves was more likely to be present in homes of children with otitis media (OR 1.7, CI = 1.03, 2.89).

In contrast, in a larger, prospective study of 904 infants in Connecticut and Virginia, Pettigrew et al. found no relationship between either woodstove or fireplace use and either single episodes of otitis media or recurrent otitis media, which was defined as 4 or more episodes during 1 yr (Pettigrew et al., 2004). Data on infant respiratory symptoms (in this case, a physician's diagnosis of an ear infection) and hours of use of secondary heating sources were collected in telephone interviews with the mothers every 2 wk for 1 yr. Although both woodstove and fireplace use were significantly associated with the outcomes in bivariate models, these associations were absent in multivariate models that adjusted for gender, race, day care, number of children in the household, duration of breast-feeding, winter heating season, use of gas appliances, season of birth, maternal education, maternal history of asthma and allergy, and pets. On the other hand, in the same study, woodstove but not fireplace use was associated with total days of cough in these infants (RR 1.08, CI 1.00,1.16) (Triche et al., 2002).

In a panel study of adults (ages 18–70) in Denver, CO (Ostro et al., 1991), the use of a fireplace or woodstove was associated with an increase in daily moderate or severe shortness of breath (OR 1.3, CI 1.1, 1.4). Use of woodstoves or fireplaces was second only to the presence of smokers in the home, and more strongly associated with shortness of breath than use of gas stoves or occupational exposures. As this study included only subjects with moderate to severe asthma, however, the findings may not be generalizable across the entire clinical spectrum of asthma.

In a study of 888 women living in nonsmoking households in Connecticut and Virginia, Triche and colleagues analyzed daily respiratory symptom data collected during the winter heating seasons of 1994–1996 (Triche et al., 2005). Using Poisson regression and controlling for age, race, allergic status, number of children, education, type of dwelling (single-family vs. multi-unit), and state of residence, these investigators found that each hour-per-day use of a fireplace was associated with several reported respiratory symptoms, including cough (RR 1.05, CI 1.01, 1.09), sore throat (RR 1.04, CI 1.00, 1.08), chest tightness (RR 1.05, CI 0.99, 1.12), and phlegm (RR 1.04, CI 0.99, 1.09). These results suggest that use of a fireplace for 4 h would increase the risk of such symptoms by about 16–20%. No such associations were found for woodstove use, which the investigators suggested may have been due to greater indoor emissions from fireplaces.

Several large time-series studies have been conducted in communities with known woodsmoke sources. The first was conducted in Seattle over a 1-yr period (September 1989 September 1990) (Schwartz et al., 1993), during which there were 2955 emergency department visits for asthma to 8 hospitals. PM_{10} TWA over 24 h ranged from 6 to 103 $\mu g/m^3$, with a mean of 29.6. In Poisson regressions controlling for weather, season, time trends, age, hospital, and day of the week, the daily counts of emergency room visits for persons under age 65 were significantly associated with PM_{10} exposure on the previous day. The relative risk for a 30 $\mu g/m^3$ increase in PM_{10} was 1.12

TABLE 6
Woodsmoke in developed countries: A sample of studies

Location	Woodsmoke concentration	Source
Outdoors		
Santa Clara County, CA	42% of CMB	(Fairley, 1990)
Seattle, WA	49% of total $PM_{2.5}$ mass	(Larson et al., 2004)
Atascadero, CA	Levoglucosan	(Manchester-Neesvig et al., 2003)
Atlanta, GA	11% of total $PM_{2.5}$ mass	(Polissar et al., 2001)
Vermont	10–18% of $PM_{2.5}$	(Polissar et al., 2001)
Christchurch, New Zealand	90% of $PM_{2.5}$ in winter	(McGowan et al., 2002)
Indoor/personal		
Seattle, WA; personal	62% of total $PM_{2.5}$ mass	(Larson et al., 2004)
Seattle, WA; indoor	35% of total $PM_{2.5}$ mass	(Larson et al., 2004)
Fort Defiance, AZ	Indoor PM_{10} dominated by woodstove smoke	(Robin et al., 1996)

(CI 1.04, 1.2). A significant exposure-response trend was found up to nearly 60 $\mu g/m^3$. Woodsmoke contributed approximately 85% of the wintertime PM in residential areas during the study period.

Two time-series studies have been conducted in Santa Clara County, California, an area in which woodsmoke is the single largest contributor to winter PM_{10} (see Table 6). 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 PM_{10} levels and increased daily mortality (Fairley, 1990). A study of asthma emergency room visits in Santa Clara County and winter PM_{10} found a relative risk for an emergency visit, adjusted to a 60-$\mu g/m^3$ increase in PM_{10}, to be 1.4 (CI 1.2, 1.7) at 20°F (Lipsett et al., 1997).

A study in Christchurch, New Zealand, examined the association between hospital admissions and PM_{10} for the period 1988–1998. Ambient PM_{10} levels during the study period averaged 25 $\mu g/m^3$, with a maximum of 283 $\mu g/m^3$. The results were stratified into total cardiac and total respiratory admissions. The estimated percentage increases per interquartile increase in PM_{10} (approximately 15 $\mu g/m^3$) for all age groups was 3.37 (CI: 2.3–4.4) for respiratory admissions and 1.26 (CI: 0.3–2.2) for cardiac admissions, but with no increase for ischaemic heart disease (McGowan et al., 2002). As noted in Table 6, woodsmoke makes up 90% of wintertime PM_{10}. One interpretation of these data is that fine particles from wood burning are more closely associated with adverse respiratory effects than adverse cardiovascular effects. Data from Seattle, WA; support this interpretation, as studies show $PM_{2.5}$ in Seattle associated with asthma aggravation (Koenig et al., 1993, 2003) but do not find similar associations with cardiac events such as myocardial infarction (Sullivan et al., 2005) or sudden cardiac address (Levy et al., 2001).

On the other hand, several studies have failed to find associations between woodstove use and respiratory health (Tuthill, 1984; Eisner et al., 2002). The Tuthill study evaluated health outcomes associated with woodsmoke or formaldehyde exposures in children. An association was seen between respiratory symptoms and prevalence of respiratory disease and estimated exposure to formaldehyde but not seen between these endpoints and estimated exposure to wood smoke. Eisner et al. (2002) studied asthma outcomes in adult subjects exposed to combustion sources indoors that included woodsmoke and environmental tobacco smoke. Although higher use of woodstoves and fireplaces was associated with more severe asthma at baseline, there was no association between use of wood burning devices and asthma aggravation after the 18-mo follow-up.

Summary of Residential Woodsmoke Epidemiology

The studies discussed in this section do have some limitations. For instance, in common with most nonoccupational air pollution epidemiologic studies, few had personal exposure information. These studies, however, do encompass a gradient of health impacts associated with woodsmoke and PM. The indicators of adverse effects run from increases in respiratory symptoms to lung function decreases to visits to emergency departments and finally hospitalizations. It is highly unlikely that this pyramid of adverse effects could be built if the associations reported were not real.

In assessing the "strength" of air pollution health effects data, Bates (1992) concluded that the question of coherence is crucial. He went on to state that such coherence may exist at different levels: within epidemiologic data, and between epidemiologic and toxicological data, and between epidemiologic data and controlled studies. Woodsmoke exposure of residents inside their homes is supported by the infiltration data discussed earlier in this section. Therefore, it is reasonable to conclude that exposure to the concentrations and durations of woodsmoke associated with residential woodburning is likely to cause a variety of adverse respiratory health effects. The biological plausibility for this conclusion is supported both by the toxicology literature, limited controlled exposure studies, and the wealth of data on

health effects of biomass burning in developing countries reviewed below.

Other reviewers have come to similar conclusions (McGowan et al., 2002). Boman et al reviewed the literature relating to adverse health effects from ambient exposure to woodsmoke and, comparing the results of studies of acute exposure to those done in areas without much woodsmoke, concluded that there was no reason to think that the adverse impacts of acute woodsmoke exposure would be less than those associated with other sources of ambient PM (Boman et al., 2003).

Statisticians are attempting to derive models that will allow source apportionment data to be added to health endpoint analysis without creating undue bias (Lumley & Liu, 2003). Creation of such models will help apportion specific health outcomes to specific sources such as woodburning.

This short summary of published studies shows that significant exposures to ambient woodsmoke do occur in developed countries and that important health effects have been demonstrated to result.

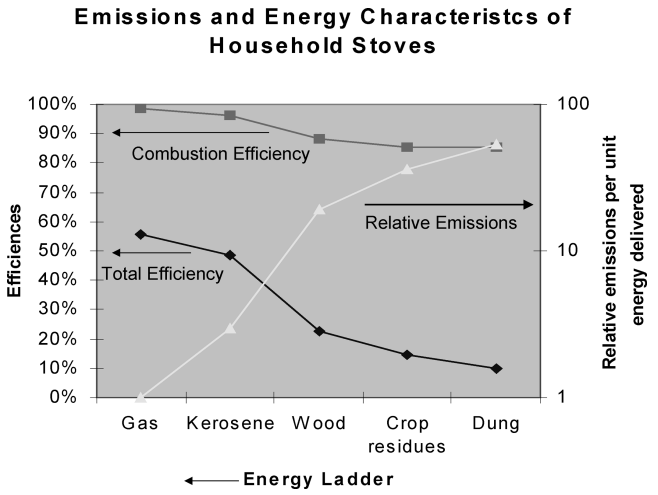

FIG. 7. Emissions and energy characteristics of typical Indian cookstoves. Note improvement in combustion and total efficiency moving from solid to liquid and gaseous fuels and great reduction in emissions per unit energy delivered. Source: Data from Smith et al. (2000).

BIOMASS USE IN DEVELOPING COUNTRIES

Indoor Air Pollution From Household Fuels

Throughout human history, the largest exposures to particle air pollution probably occurred in households through use of wood and other forms of biomass as sources of cooking, drying, and space-heating energy. Even today, such uses probably account for the majority of human exposure to respirable PM worldwide because of the continued high dependence on such household fuels (Smith, 1993). As shown in Figure 6, for example, about half of the world's households are still thought to cook with solid fuels on a daily basis (Smith et al., 2004). Of this, about 95% consists of wood and agricultural residues. Household use of mineral coal for cooking, which makes up the remainder, is mainly confined to China.

In simple devices, like the household stoves commonly used in developing countries, biomass fuel does not combust cleanly. Systematic emissions studies in India and China, for example, have generally validated the so-called "energy ladder" concept with regard to the emissions from combustion of household fuels (Smith et al., 1994). As shown in Figure 7, the energy ladder

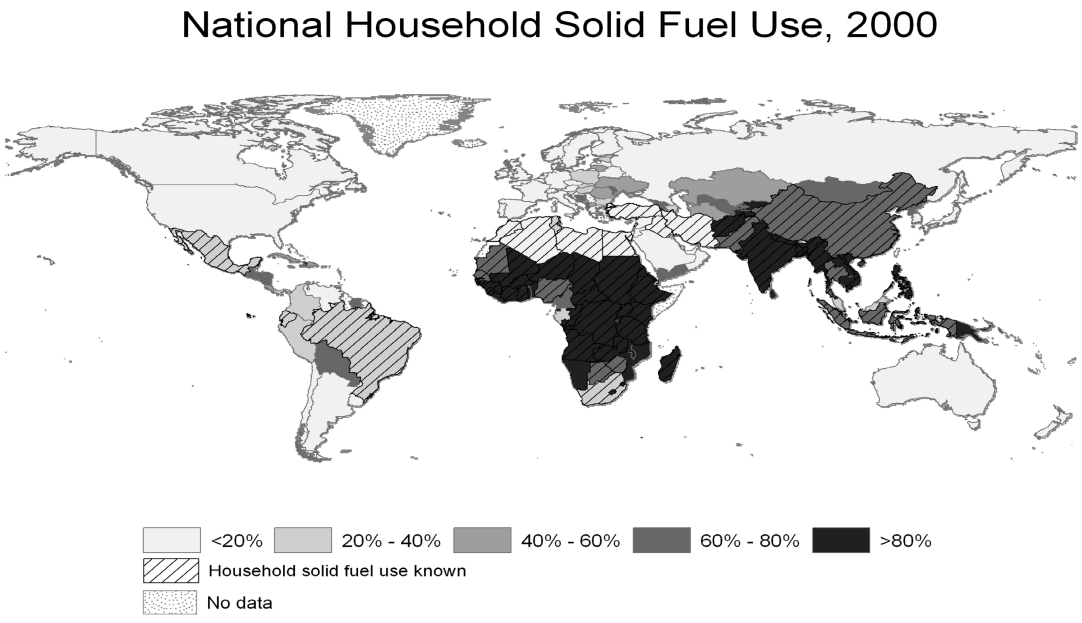

FIG. 6. Map of solid fuel use. Source: Smith et al. (2004).

starts at the bottom with low-quality biomass fuels, such as cow dung, moves up through crop residues, to wood. Further up the ladder lie liquid and then gaseous fuels (kerosene and liquefied propane gas, LPG), with electricity being at the top (i.e., with the lowest emissions). Nominal combustion efficiency (percent of fuel carbon emitted as CO_2) is as low as 80% for the poorer fuels and reaches more than 99% with gaseous fuels (Smith et al., 2000). In combination with the low thermal efficiency of solid fuel stoves, the result is differences in emissions per meal of nearly two orders of magnitude between gaseous and solid fuels. In addition to cleanliness, the cost, complexity, and ease of household use generally increase as one moves up the ladder (Office of Technology Assessment, 1992). Broadly, as average household income increases in societies, usage tends to move up the ladder, although not always to the last rung (electricity). This is shown by econometric studies at the national level (Mehta, 2003). In individual communities, however, the situation is often more complicated, particularly during transition phases, when households may straddle several rungs of the ladder at once by using multiple fuels depending on prices, seasons, availability, and so forth (Sinton et al., 2004).

As noted elsewhere in this report, poor combustion efficiency creates high emission factors for wood and other biomass across a wide range of health-damaging pollutants. High emissions, however, do not necessarily lead to high exposures unless they reach human breathing zones. Unfortunately, however, conditions in hundreds of millions of Third World households are nearly ideal to maximize exposures from emissions. A large, but unknown, fraction of daily cooking is done in unvented stoves, that is, stoves in which the emissions are released directly into the living area and not vented through a chimney or hood. Although there are not systematic surveys in developing-country settings, about 200 studies of indoor air quality (IAQ) measurements in households using solid fuels have been published, more than half from China.* Table 7 shows a summary of these studies for the two most widely measured pollutants, PM and CO. These studies have been published between 1968 and 2003. The studies in South Asia were mainly conducted in Nepal and in India, with only one reported from Bangladesh. The studies in Africa come mainly from Kenya, Gambia, and South Africa. Most of the Latin American studies have been conducted in Guatemala and Mexico.

Most of these studies were conducted in rural settings and attempted to characterize the distribution of concentration levels in the kitchens, with the earlier studies reported from the highlands in different parts of the world. Also, there is little information available on seasonal effects or differences across the various meals cooked in a day. Meal cooking time varied from study to study, generally between 30 min and 3 h, with one study reporting up to 8 h. Although several studies made comparisons between the traditional and the improved stoves, in this summary table that distinction has not been made.

A highly polluting source releasing pollution indoors at times and places when people are always present (household cooking) has a potential to produce high exposure. Put another way, the associated *intake fraction (*fraction of material released that is actually inhaled by someone) is orders of magnitude higher for indoor than for outdoor sources of air pollution (Bennett et al., 2002). Although the uncertainties are large, the available evidence would indicate that the total exposure to combustion-derived fine particles from indoor solid fuel use is larger than that from all outdoor sources of pollution in the world (Smith, 1993).†

Even in communities where most households use chimneys, however, the intake fraction can be substantially higher than for typical outdoor sources since the smoke may sit in the area among the houses in what is called "neighborhood pollution." Such pollution may not be fully reflected by ambient monitoring data, but may nevertheless substantially influence local exposures (Smith et al., 1994). This same phenomenon exists in developed countries as well, for example, from household fireplaces, as discussed earlier. Because of their almost universal role as household cooks, the highest exposures from household use of solid fuels, however, seem usually to occur to women and their youngest children who are with them during cooking, although significant exposures can accrue to other household members as well (Balakrishnan et al., 2004).

Although few studies have linked linked IAQ measurements to ill health, a growing number of epidemiologic studies have found significant risks of various exposure indicators and ill health in developing-country biomass-using households. Such exposure indicators include use of solid or "dirty" fuel versus liquid/gas "clean" fuel; using a stove with a flue or without; years cooking with solid fuel, and, for infants, being carried on their mother's back while cooking or not. Taking advantage of the increasing number of such studies, the recent global Comparative Risk Assessment (CRA) managed by the WHO included indoor as well as outdoor air pollution among the 26 risk factors examined (Ezzati et al., 2002).

The available epidemiologic evidence was divided into three categories, as shown in Table 8. Considered sufficient in quantity and quality to justify inclusion in the global CRA was evidence only in the top category: acute lower respiratory infections (ALRI: pneumonia) in young children, chronic obstructive pulmonary disease (COPD) in adults, and lung cancer in adults (for coal smoke only). The odds ratios shown in the table are the result of meta-analyses of the data in published studies that met the criteria for inclusion (Smith et al., 2004).

A number of epidemiologic studies have also been published for these populations in relation to other important diseases, however. Shown in the second category ("Moderate" evidence) are simple means of odds ratios in available studies

*See the Chinese IAQ database (Sinton et al., 1996) and the non-Chinese IAQ database (Saksena et al., 2003) both available at http://ehs.sph.berkeley.edu/krsmith.

†See also Table 10, which shows the estimate for total global health effects for indoor and outdoor air pollution.

TABLE 7
Summary of indoor air quality studies: Household pollution levels in developing countries outside of China. Values are indicative only as they were determined with various times and measurement methods.

	Number of studies	Total number of samples	TSP ($\mu g/m^3$)		PM_{10} ($\mu g/m^3$)		$PM_{2.5}$ ($\mu g/m^3$)[c]		CO (mg/m^3)	
			Meal	Daily	Meal	Daily	Meal	Daily	Meal	Daily
Bangladesh	1	53							15–26	
Bolivia	1	169			3700					
Brazil	1	23			530					
Burundi	1	2								42
Ethiopia	1	N/A							48	
Gambia	1	12				1600-2200				
Ghana	1	21			590				9	
Guatemala	7	768		280–840		190–1200	450–27,000	97–1900	2–149	1.2–17
India	13	1009	646–16,000		900–1100	506	110–2100	1300–1500	5–216	
Kenya	4	199	3776			1800–3900		630–3500		5–60
Malaysia	1	10		300						3
Mexico	5	191			280–1200	97–290		890	10–22	
Mozambique	1	114			1200				48	
Nepal	5	127	710–8800	4600–8420	4700		1700–5700		14–360	14–52
New Guinea	1	9		360–670						13–24
Nigeria	1	28		108					1076	
South Africa	1	20		1725					79–180	92
Zambia	1	89			890				10	
Zimbabwe	1	34	1357							

Note. From Saksena et al. (2003).
Unspecified averaging time for CO measurements.

TABLE 8
Health effects of use of solid household fuels in developing countries

Disease	Population affected	Relative risk (95% confidence interval)	Strength of evidence
COPD	Females >15 yr	3.2 (2.3, 4.8)[a]	Strong
	Males >15 yr	1.8 (1.0, 3.2)[a]	Intermediate
ALRI	Children <5 yr	2.3 (1.9, 2.7)[a]	Strong
Lung cancer (coal only)	Women >15 yr	1.9 (1.1, 3.5)[a]	Strong
	Men >15 yr	1.5 (1.0, 2.5)[a]	Intermediate
Blindness (cataracts)	Females >15 yr	1.3–1.6[b]	Intermediate
Tuberculosis	Females >15 yr	1.5–3.0[b]	Intermediate

Note: From Smith et al. (2004).
[a]Based on formal meta-analysis.
[b]Range of results in published studies.

showing significant impacts of use of solid fuels on tuberculosis and cataracts. There is also evidence of the impact of biomass smoke exposures on lung cancer (Behera & Balamugesh, 2005; Hernandez-Garduno et al., 2004). Although consistent, the number and character of these studies was not considered sufficiently persuasive to include these diseases in the CRA.* Similarly, although studies of outdoor air pollution, ETS, and other sources of particle exposure indicate impacts on asthma and heart disease, no convincing studies are yet available in the populations of interest for household solid fuel use.†

Using only the "Strong" evidence category in Table 8 and the distribution of solid fuel use shown in Figure 6, the total impact of IAQ from solid fuel use calculated in the CRA is shown and compared to other environmental risk factors in the CRA in Table 9.

At 1.6 (0.8–2.4) million deaths and 2.6% of the global burden of disease (as measured in lost life-years), IAQ ranks second only to poor water/sanitation/hygiene among environmental health risk factors. Among all major policy-relevant risk factors, indoor air pollution from solid fuel is tenth globally, and fourth in least-developed countries.‡ See Figure 8.

Biomass Smoke in LDC Cities

Ambient air pollutants come primarily from combustion of fossil fuel. In many cities and rural areas in developing countries, residential space heating and cooking with solid fuels, mostly biomass and coal, can also contribute significantly to the ambient pollution. Several studies have been conducted in developing countries quantifying the contribution of biomass smoke in cities. Begum et al. (2004) report contribution from biomass combustion to be 12% in Dhaka and 50% in Rajshahi, Bangladesh (Begum et al., 2004). From a study conducted by Zheng et al. (in preparation) in three sites in Hong Kong, they find that 9–10% of the organic carbon in $PM_{2.5}$ comes from biomass smoke. Chowdhury et al. report contributions from biomass in three Indian cities by season as seen in Table 10. (Chowdhury et al., 2005.).

High concentration of biomass smoke in the colder months compared to the warmer months can be explained by the regional meteorology where the monsoon rains are dominant in the summer months followed by the dry winter, when pollutants are trapped inside the inversion layer. Also, during the winter months there is a tendency to use biomass fuel for heating purposes, leading to the higher concentrations in Delhi and Kolkata, 66/51 $\mu g/m^3$ $PM_{2.5}$, representing 29/17% of the total $PM_{2.5}$. Although a bit higher in absolute concentrations, this winter pattern is also found in developed-country urban areas using wood fuels.

TOXICOLOGICAL EFFECTS OF WOODSMOKE EXPOSURE

Although studying the effects of air pollutants directly on humans offers a number of advantages, epidemiologic and controlled clinical studies are often limited by societal concerns, ethical and legal issues, as well as cost. Because of these difficulties, predictive health assessments associated with inhaled woodsmoke need to include information gained from animal exposure studies and, in some cases, *in vitro*/*ex vivo* assay systems. Furthermore, animal studies also have the potential to help uncover information concerning the mechanisms of toxicity and relative toxicity of different mixtures and sources. The discussion that follows summarizes some of the principal published toxicologic studies of woodsmoke.

*A similar conclusion was reached in 2006 in IARC Monograph #95, in which household combustion of biomass was rated as Category 2A, limited human evidence with supporting animal evidence (Straif et al., 2006).

†A recent conference abstract, however, has shown a clear effect of lowered blood pressure from women whose woodsmoke exposures were lowered in a randomized clinical trial of improved stoves with chimneys in Guatemala (McCracken et al., 2005).

‡The summary results of the CRA were released in the World Health Report (WHO, 2002; Ezzati et al., 2002) and were published in detail in Smith et al. (2004).

TABLE 9
Global burden of disease and premature death due to major environmental risk factors in 2000

Parameter	Poor countries	Mid-income countries	Rich countries	World
Population (millions)	2343	2424	1358	6125
DALYs (million disability-adjusted life years)[a]	846	406	214	1,467
Unsafe water, sanitation, and hygiene	5.5%	1.8%	0.4%	3.7%
Indoor smoke from solid fuels	3.6%	1.9%	0.3%	2.6%
Occupational risks	1.1%	2.4%	1.5%	1.5%
Lead exposure	0.7%	1.4%	0.6%	0.9%
Urban air pollution	0.3%	1.0%	0.5%	0.5%
Climate change	0.6%	0.1%	0.0%	0.4%
Total environmental burden of disease, % of total for region	11.8%	8.5%	3.4%	9.7%
Deaths (thousands)	26,700	16,000	13,000	55,700
Unsafe water, sanitation, and hygiene	1538	172	20	1730
Indoor smoke from solid fuels	1039	558	22	1619
Occupational risks	2393	640	176	3209
Lead exposure	93	69	72	234
Urban air pollution	220	426	154	800
Climate change	148	5	0	153
Total environmental mortality in region (thousands)	5431	1870	444	7745

Note: From WHO (2002).

[a]DALYs are calculated as the sum of lost years from premature mortality and lost years of illness and injury weighted by a severity factor.

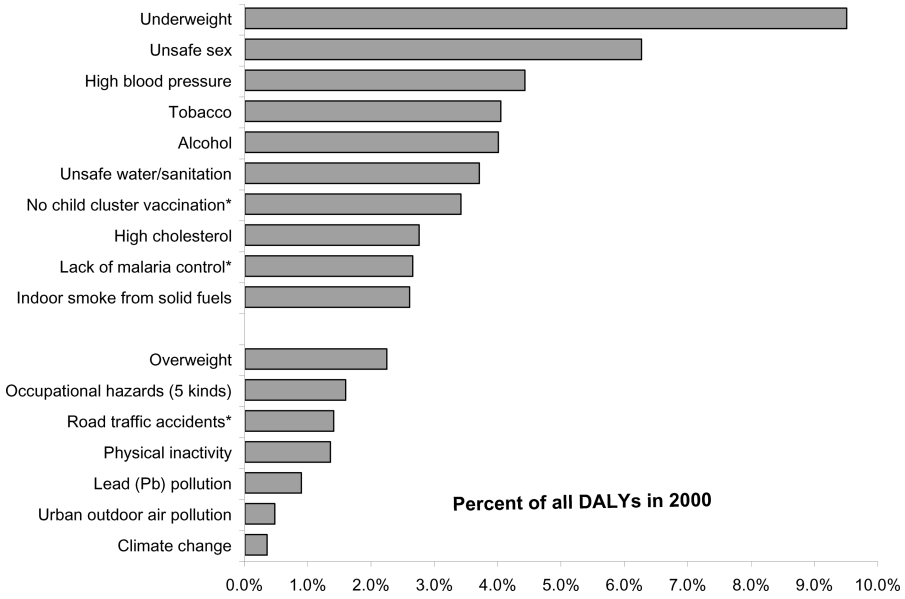

FIG. 8. Estimated burden of disease in 2000 measured as lost healthy life years (DALYs) from major preventable risk factors. Note importance of indoor smoke from solid fuels, which are mostly biomass. Those marked with asterisk are based on outcomes in the WHO Global Burden of Disease database. *Note*: Created from WHO data by Smith and Ezzati, 2005.

TABLE 10
Contribution of biomass smoke into urban ambient $PM_{2.5}$ in India

	Spring		Summer		Autumn		Winter	
Location	$\mu g/m^3$	% of $PM_{2.5}$	$\mu g/m^3$	% of $PM_{2.5}$	$\mu g/m^3$	% of $PM_{2.5}$	$\mu g/m^3$	% of $PM_{2.5}$
Delhi	25	22%	5	10%	33	21%	66	29%
Mumbai	5	13%	N/A	N/A	14	21%	11	13%
Kolkata	10	19%	6	24%	14	32%	51	17%

Note: From Chowdhury et al. (2005). Based on five to seven 24-h samples per season.

In Vivo Inhalation Studies

Although woodsmoke can be delivered to animals by a variety of methods, including open-chest ventilation, only those studies that employed exposure routes most relevant to the human woodsmoke experience (i.e., nose-only/whole body inhalation in conscious animals) are reviewed herein.

Single Woodsmoke Exposures

Acute lung injury. Because of increasing interest in the mechanisms of damage in firefighting and other high-exposure situations, the majority of toxicologic studies reviewed for this document fell within this category. As the exposure levels used for these studies are usually much greater than those to which the general public in developed countries is exposed, these studies will serve primarily to demonstrate the effects that could, potentially, occur as a result of lower level, repeated exposures. In 1982, an inhalation study by Thorning et al. (1982) described the effects of inhaled woodsmoke on pulmonary lung cell injury (as determined by changes in lung morphology). Two combustion products (i.e., CO and organic aldehydes) were monitored as a measure of woodsmoke exposure. Total aldehyde concentrations in the chamber ranged from 285 to 1707 ppm for the 25- to 45-min exposures. Smoke-exposed rabbits exhibited necrotizing tracheobronchial epithelial cell injury that peaked by 24 h post-exposure. The authors concluded that the acute lung injury and early reactions to smoke damage observed in rabbits closely resembled those pulmonary lesions seen in smoke-injured victims, and that these injuries could affect pulmonary host resistance. Particle-adsorbed aldehydes were suggested to play the major role in such injuries.

In another study using rabbits, Loke et al. (1984) investigated the effects of a 60-min inhalation of Douglas fir-generated woodsmoke (mean carboxyhemoglobin [COHb] level = 16.4%) on alveolar macrophage (Mø) response and tracheobronchial morphology. Smoke injury to the proximal tracheal lining cells was severe, with major epithelial cell loss observed in exposed animals. In addition, mucociliary escalator dysfunction was also observed. Similar to the cellular alterations observed in long-term cigarette smokers (Rasp et al., 1978), alveolar Mø from woodsmoke-exposed rabbits were flatter and contained less surface ruffling (a marker of cell activation) than their unexposed counterparts. In addition, Mø numbers were increased within the alveoli, suggesting an inflammatory response in the smoke-exposed rabbits.

In a study by Fick et al. (1984), rabbits were exposed acutely (i.e., 30–120 min) to smoke from the pyrolysis of Douglas fir wood and effects on Mø functional activity were examined immediately following exposure. At a smoke concentration yielding a COHb level of 7% and no evidence of thermal injury, pulmonary Mø-mediated bacterial phagocytosis and intracellular killing of the gram-negative bacterial pathogen *Pseudomonas aeruginosa* was dramatically reduced. Although an inflammatory response was not observed, smoke-exposed animals demonstrated a significantly greater lavageable cell yield than the unexposed controls. This investigation provided the first evidence that lower levels of woodsmoke could produce subclinical effects and alter lung properties in the absence of any acute lung injury. This well-executed toxicologic study employed the most current inhalation procedures of their time and evaluated effects in a dose-response manner based on increasing COHb levels. The authors concluded that inhalation of woodsmoke, at a relatively low level, had the potential to alter host pulmonary immune defense mechanisms in such a way as to lead to an increased susceptibility to infectious lung disease.

Woodsmoke-induced alterations in phagocyte-mediated oxidative stress response and antioxidant status were studied in a rat model designed to simulate an inhalation injury as might be encountered by firefighters and/or burn victims (Dubick et al., 2002). In this study, rats, either previously scalded or sham-burned, were exposed for approximately 16 min to clean air or smoke generated from the burning of Western bark wood (i.e., fir and pine); smoke exposure was assessed by measuring COHb levels in exposed hosts (19 ± 2%). At different time points post-exposure, animals were sacrificed and either their lungs were lavaged for evaluation of total protein and airway cellular/lung water content, or their tissues were recovered for measurements of antioxidant enzyme activities and lipid peroxidation (measured by thiobarbituric acid-reactive substances [TBARS]) (Dubick et al., 2002). Similar to that seen in other smoke injury studies, Dubick et al. (2002) observed that acute inhalation of woodsmoke produced areas of tracheal erosion resulting in the loss of epithelium. At 24 h postexposure, lipid peroxidation was increased two- to three-fold above control values in the smoke-only and burn/smoke-exposure groups; TBARS

declined after 48 h in the smoke-only group. Other investigators have also reported oxidative stress as a result of woodsmoke exposure (Demling & LaLonde, 1990; Demling et al., 1994; Lalonde et al., 1994). Minor changes in lung antioxidant enzyme activities were also observed in this study. However, in contrast to the dramatic inflammatory response observed in previous studies examining acute smoke-induced lung injury (Traber & Herndon, 1986; Hubbard et al., 1991), pulmonary immune cell infiltration was not observed. Given that leukocyte infiltration appears to "track" with woodsmoke-induced pulmonary injury, a lack of neutrophil influx was not surprising, given the modest level of lung injury produced in the aforementioned study.

In addition to tracheobronchial alterations and changes in immune cell morphology, acute inhalation (30 min) of Douglas fir-generated woodsmoke has been shown to diminish the ventilatory response of exposed guinea pigs. In a study by Wong et al. (1984), animals were exposed for 30 min to increasing woodsmoke concentrations generated by burning different amounts of wood chips. Just prior to exposure and at various time points post-exposure, guinea pigs challenged with CO_2 were placed in a whole-body plethysmograph and lung compliance (elastic properties of the lungs) was measured. Exposure to moderate levels of smoke increased baseline ΔP (change in lung relaxation pressure), but only 0.5 h after exposure. None of the exposure groups varied significantly from controls with respect to ΔP after this early time point. Reduction in pulmonary compliance has also been shown in woodsmoke-exposed dogs (Stephenson et al., 1975) and in human victims of smoke inhalation (Garzon et al., 1970). Wong et al. (1984) concluded that acute inhalation of woodsmoke can alter lung function, but that recovery occurs within several days after exposure.

Woodsmoke-induced alterations in airway responsiveness to bronchoconstrictor challenge have also been observed more recently in other studies using guinea pigs. Exposure to a PM concentration of 25 mg/m^3 produced airway hyperreactivity in response to challenge with the bronchoconstrictors substance P, capsaicin, and prostaglandins (Hsu et al., 1998a, 1998b; Hsu & Kou, 2001; Lin & Kou, 2000; Lin et al., 2001). Despite the "artificial" exposure route used to deliver woodsmoke in these studies, the results provide compelling evidence regarding the adverse effects of woodsmoke on airway responsiveness. Support for these findings in guinea pigs comes from repeated exposure studies by Tesfaigzi et al. (2002), who demonstrated a significant increase in total pulmonary resistance and dynamic lung compliance in brown Norway rats exposed by inhalation (whole body) to lower concentrations (1 or 10 mg/m^3) of woodsmoke generated from the burning of *Pinus edulis* wood for either 4 or 12 wk (3 h/day, 5 days/wk).

Some of the same investigators who examined bronchoconstriction in guinea pigs also performed studies in rats to evaluate the role of sensory receptors and nerve fibers in pulmonary ventilation following woodsmoke exposure (Kou & Lai, 1994; Kou et al., 1995, 1997, 1999; Wang et al., 1996; Lin & Kou, 1997; Lai & Kou, 1998a, 1998b, 1998c; Lin et al., 2000; Ho & Kou, 2000). For these studies, rats were exposed to either particle-free (i.e., gas only) or whole woodsmoke effluents. Results demonstrated that (among other things) woodsmoke-induced slowing of respiration is a reflex resulting from stimulation of bronchopulmonary C-fiber nerve endings (unmeylinated sensory neurons that conduct nerve impulses slowly) induced by the woodsmoke gas phase. These studies further revealed that increased hydroxyl radical (·OH) burdens following smoke exposure were actively involved in evoking the acute irritant effects of woodsmoke. Investigations by Ho and Kou (2002) also demonstrated that woodsmoke exposure increased nasal airway resistance and airway reactivity in rats exposed to woodsmoke via the nose.

In a thorough, well-executed inhalation study by Matthew et al. (2001), exposure of mice to high doses of woodsmoke (COHb level of 50% immediately after exposure): altered pulmonary histology; induced an inflammatory response; increased static lung compliance; and increased lavageable cytokine levels and cell counts (Matthew et al., 2001). Given that smoke inhalation damage is mediated in part via an upregulated immune response, increased numbers of lavageable immune cells are consistent with the observed lung pathology. The authors speculated that smoke-associated PM, with or without adhered noxious gases, were likely responsible for the majority of observed lung pathologies. This notion was supported by the studies of Zelikoff (2002), who demonstrated diminished immunotoxicity of inhaled woodsmoke effluents in rats following removal of the particulate smoke phase (Thomas & Zelikoff, 1999).

Repeated and Subchronic Woodsmoke Exposures

Pulmonary and systemic effects . Inhalation studies utilizing scenarios other than acute single-exposure regimes are extremely limited. Long-term investigations that more closely reflect smoke exposures associated with domestic pollution (i.e., home heating and cooking) are needed to assess potential long-term risks. Lai et al. (1993) examined the hematological and histopathologic responses of rats exposed repeatedly to smoke generated from the combustion of wood dust. Despite the primitive nature of the exposure system, the lack of information concerning smoke concentration and wood type, and the lack of data regarding thermal burns, many of the same smoke-induced pathologies observed in this study (i.e., epithelial lining cell desquamation, pulmonary edema, and peribronchiolar and perivascular infiltration of polymorphonuclear neutrophils [PMN]) have also been reported in the studies of acute smoke exposures (Lal et al., 1993). Bronchiolitis, parenchymatous blood vessel congestion, hyperplasia of lymphoid follicles, and mild emphysema were also observed after 15 days of smoke exposure. Although the emphysematic changes remained constant over time, other pulmonary lesions worsened dramatically with increasing exposure duration. In addition, marginal alterations were observed in hemoglobin levels, sedimentation rate, packed cell volume, and total and differential leukocyte counts from animals exposed to smoke for 15 days. Eosinophilia was also observed, but only in rats exposed for 30 and 45 days. Results

of this study demonstrated that woodsmoke-induced pulmonary lesions are progressive with repeated smoke exposures. Moreover, given that domestic woodsmoke pollution has been associated with chronic obstructive pulmonary disease (e.g., chronic bronchitis and emphysema) in developing countries and that emphysematic changes were observed in this toxicologic study, the authors concluded that the rat model of acute lung injury may prove useful for assessing the toxicologic impact and human health outcomes of inhaled woodsmoke.

Studies from this group (Zelikoff, 2000) have demonstrated that repeated short-term (1 h/day for 4 days), nose-only inhalation exposure of rats to woodsmoke generated from the burning of red oak wood (i.e., 750 μg PM/m^3, <2 ppm CO, 3 ppb NO_x, and 1.5 ng total PAH/m^3) inhibited pulmonary clearance of intratracheally instilled *Staphylococcus aureus*; the smoke concentration used in this study is reflective of that found indoors during operation of a poorly vented fireplace, a non-U.S. Environmental Protection Agency (EPA)-certified wood-burning device, or under extreme residential conditions where open fires are used for heating and cooking. The effect on bacterial clearance was observed in the absence of any lung pathology, lung cell damage (as measured by total protein and lactate dehydrogenase release) or inflammation. The lack of pulmonary injury and/or inflammation was similar to that observed in a more recent study by Reed et al. (2006) in which rats and mice were exposed (by whole-body inhalation) for longer time durations (1 wk or 6 mo) to hardwood smoke (HWS) generated from an uncertified wood stove.

In the aforementioned studies, suppressed bacterial clearance began as early as 3 h postexposure and persisted for almost 2 wk (Thomas & Zelikoff, 1999). Interestingly, similar dramatic effects on pulmonary bacterial clearance were not observed for rats exposed to particle-free woodsmoke effluents. This response demonstrates the importance of the woodsmoke-associated PM in bringing about the observed time-related effects on pulmonary host resistance. In the same study (Thomas & Zelikoff, 1999), woodsmoke exposure also suppressed production of pulmonary Mø-mediated superoxide anion ($O_2^{\cdot -}$), a reactive oxygen species critical for the intracellular killing of *S. aureus*. The authors suggested that reduced production of $O_2^{\cdot -}$ might (in part) be responsible for the observed woodsmoke-induced decrease in pulmonary host resistance against this particular pathogen. Taken together with results from earlier studies, the authors concluded that short-term, repeated inhalation of woodsmoke can compromise pulmonary immune mechanisms that are critical for host protection against infectious lung pathogens. Moreover, they concluded that the pulmonary Mø represents a sensitive target for the toxic effects of inhaled woodsmoke.

A more recent study has examined the effects of inhaled woodsmoke at 1 or 10 mg PM/m^3 on rats exposed to smoke generated from the burning of *Pinus edulis* for 4 or 12 wk (Tesfaigzi et al., 2002). In the absence of any effects on lung-associated T-lymphocyte proliferation or lavageable cytokine levels, repeated smoke inhalation produced a modest but significant reduction of CO-diffusing capacity (as demonstrated by an impairment of gas exchange) in the high-dose exposure group; increased dynamic lung compliance also in the 10-mg PM/m^3 group; and mild chronic inflammation and squamous-cell metaplasia in the larynx of all groups of exposed rats. The mucous-cell metaplasia observed after 30 days of woodsmoke exposure was transient and resolved after 90 days. The severity of alveolar Mø hyperplasia and pigmentation increased with smoke concentration and length of exposure. However, some dose-response inconsistencies and the absence of a significant change in quasistatic compliance, a more specific measure of lung elastic recoil, led the authors to conclude that the impact of woodsmoke in this study was small and, except for the observed reduction in gas exchange, of little clinical importance. Though one could dispute the investigators' conclusions regarding the nominal importance of these findings and the insensitivity of the immune system for assessing the health impacts of inhaled woodsmoke, the study was well executed, demonstrated a dose-response relationship for some endpoints, employed a well-described generation/exposure system, and incorporated extensive chemical characterization of the woodsmoke effluents.

Two rodent studies, recently published by investigators at the Lovelace Respiratory Research Institute (LRRI), examined the health effects of repeated hardwood smoke (HWS) exposure using a range of exposure concentrations at or just above those commonly experienced in the indoor and/or outdoor U.S. environment (30–1000 μg PM/m^3) (Reed et al., 2006; Barrett et al., 2006). Specifically, studies by Barrett et al. (2006) investigated the ability of short-term, repeated exposure to HWS to exacerbate allergic airway responses in already sensitized mice; two different sensitization paradigms were examined. Findings from this study demonstrated that in the absence of tissue inflammation or altered Th1/Th2 cytokine levels, a 3-day exposure to HWS following the final allergen challenge could exacerbate some indices of allergic airway inflammation, such as lavageable numbers of eosinophils and serum OVA-specific immunoglobulin E (IgE). The authors concluded that the effects of HWS on allergic airway parameters were relatively mild, but were comparable to those responses observed with other pollutant mixtures such as diesel exhaust.

A large companion study published by some of the same LRRI investigators examined the effects of longer exposure durations (1 wk or 6 mo) to HWS on general indicators of toxicity (i.e., body and lymphoid organ weights, clinical chemistry and hematology), bacterial clearance, cardiac function, and carcinogenic potential using both mice and rats (Reed et al., 2006). A range of woodsmoke levels (30–1000 μg PM/m^3) and both genders of two rodent species were examined. Exposure-related effects included increases in blood platelets; decreases in blood urea nitrogen and serum alanine aminotransferase; changes in liver, spleen and thymus weight; and increased circulating white blood cell (WBC) counts. No effects were observed upon micronuclei formation, tumorigenesis, cardiac parameters, or pulmonary clearance of the bacteria *Pseudomonas aeruginosa*. The

lack of any effects on bacterial clearance is in contrast to those effects reported by Zelikoff (2000), who demonstrated that inhalation of woodsmoke at 750 $\mu g/m^3$ for 4 days significantly reduced pulmonary clearance of *S. aureus* in exposed rats. Differences between the studies may have been due to any one of a number of factors, including disparity between rodent models, wood type, burning conditions, and/or possible adaptation of the mice to the long-term exposure scenario (6 mo). Another important difference between the two studies is the bacterial species used for challenge. *Pseudomonas* is removed mainly in the infected host by the bactericidal mechanisms of neutrophils, while those mechanisms used to remove the gram-positive cocci *S. aureus* are primarily mediated by Mø, a sensitive target for the toxic effects of woodsmoke, Thus, differences in bacterial clearance mechanisms could have played a role in the disparity observed between the studies. Reed et al. (2006) concluded, based upon observed sex and exposure duration inconsistencies, that at the woodsmoke concentrations utilized in these studies, the observed effects "posed little to small hazard with respect to clinical signs, lung inflammation and cytotoxicity, blood chemistry, hematology, cardiac effects, bacterial clearance and carcinogenic potential." While this study represents an eloquently executed investigation, the authors seem to have minimized the observed smoke-induced effects which could result in an underestimation of the actual risks associated with such exposures. The potential short- and long-term health risks associated with some of the observed effects (i.e., increased platelet number, reduced liver weight and increased spleen weight) appear worthy of further consideration, particularly in light of the recent experimental study which demonstrates that healthy humans exposed to wood smoke at 250–280 micrograms/m3 during two 4h sessions increases the levels of serum amyloid A (a cardiovascular risk factor) and plasma factors important for maintaining the balance of coagulation factors (Barregard et al., 2006).

Lung cancer. A field study was carried out in which mice and rats were placed for 15 or 19 mo, respectively, in an indoor environment to inhale either air (without combustion products) or smoke generated from burning wood or coal (Liang et al., 1988). Burning was carried out in round shallow pits of individual rooms meant to simulate those of villagers in Xuan County, China, as were the patterns and intensity of burning (241 kg/mo; 12 h smoke/day). Although smoke generated from both coal and wood contained similar total suspended particulate (TSP) levels (i.e., 14.4 vs. 14.9 mg/m^3, respectively), the BaP concentration in the wood exposure room was approximately 47 times higher than that measured in the air control environment, although substantially less than that measured in the coal-using rooms. In the woodsmoke room, measured levels of CO, SO_2 and H_2SO_4 were 80 mg/m^3, 0.05 mg/m^3, and 0.27 mg/m^3, respectively. Following exposure, animals were immediately sacrificed and the incidence of nonmalignant and malignant lung tumors was evaluated. Tumors were histologically differentiated into several groups including adenomas, adenocarcinomas (AC), adenosquamous carcinomas (ASC), and squamous-cell carcinomas (SCC). Although control mice demonstrated a lung cancer incidence of 17%, mice exposed long-term to either wood or coal smoke had incidences of 45.8% and 89.5%, respectively. Although all lung cancer types were observed in coal smoke-exposed mice, those exposed to woodsmoke demonstrated far fewer SCC and ASCs. Lung cancer incidence for rats exposed to ambient air or smoke from burning wood or coal was 0, 0, and 67.2%, respectively.

The authors of the aforementioned study concluded that woodsmoke proved to be only a weak carcinogen compared to coal smoke. Differential effects between wood and coal have also been observed in a mouse skin tumorigenicity study that compared the effects of organic extracts from smoky coal and wood combustion (Mumford et al., 1990). In this case, particle extracts from smoky coal combustion proved to be a potent complete carcinogen, whereas that from woodsmoke proved relatively inactive. The relatively modest effects observed in this early study by Liang et al. (1988) are in line with those reported by Reed et al. (2006), who demonstrated that inhalation of lower concentrations of woodsmoke (30–1000 μg PM/m^3 vs. 14.9 mg/m^3) for only 6 mo failed to significantly increase lung tumors in exposed mice (compared to control).

Ex Vivo/In Vitro Woodsmoke Exposure Studies

Although toxicologic studies using routes of exposure other than inhalation were not the primary focus of this review, several *ex vivo/in vitro* studies are briefly discussed inasmuch as they might contribute to a better understanding of the potential health impacts of woodsmoke. Bhattacharyya et al. (1998) examined the effects of pine woodsmoke exposure for 5–20 min on rabbit tracheal explants. Exposure of explants for 20 min led to degeneration of the mucociliary epithelial sheath; shorter smoke exposures (i.e., 10 min) resulted in retained tissue integrity, but altered epithelial morphology. Similar woodsmoke-associated pathologies have been observed *in vivo* following acute inhalation exposure.

Exposure of cultured eye lenses to woodsmoke condensates for 10 min resulted in woodsmoke metabolite-induced opacification (Rao et al., 1995). Histological analyses of smoke-exposed lenses revealed distinct morphological changes including hyperplasia, hypertrophy, and multilayering of epithelial cells. The authors concluded that exposure to woodsmoke could contribute to progressive eye lens opacification.

Leonard et al. (2000) examined the effects of pine- and Douglas fir-generated liquefied woodsmoke on cultured mouse Mø free radical generation, DNA damage, nuclear factor (NF) κB activation and tumor necrosis factor-α (TNFα) release. These studies demonstrated that exposure to liquefied woodsmoke in combination with hydrogen peroxide (H_2O_2) resulted in hydroxyl radical ($\cdot$OH)-induced DNA damage, and that co-exposure to on an $\cdot$OH radical scavenger or a metal chelator inhibited the observed genotoxicity (Leonard et al., 2000). The authors concluded that free radicals generated by woodsmoke

through the reaction of iron with H_2O_2 could produce genetic and cellular damage. Moreover, such free radicals could also play a role in the development of woodsmoke-induced pulmonary fibrosis.

A number of genetic toxicology studies have evaluated the mutagenicity of woodsmoke condensates (Hytonen et al., 1983; Alfheim & Ramdahl, 1984; Alfheim et al., 1984; Asita et al., 1991). In all cases, woodsmoke extracts were mutagenic in bacterial systems. A number of factors, including heating conditions, type of wood-burning device, wood origin, and PAH concentration, seemed to play important roles in overall mutagenic activity.

Toxicology Summary

The majority of the toxicology studies presented in this review provide biological plausibility for the epidemiologic evidence suggesting that exposure to woodsmoke emissions adversely affects human health. These animal studies also contribute to a better understanding of the possible mechanisms(s) by which woodsmoke, and its associated PM, may act to bring about increased pulmonary morbidity in exposed individuals. It appears clear from the toxicologic studies that short-term inhalation of woodsmoke can compromise pulmonary immune defense mechanisms important for maintaining host resistance against pulmonary infections. Moreover, a likely target for woodsmoke-induced immunotoxicity seems to be the lung Mø. These immune cells, which serve as the primary defense of the deep lung, provide a link between the nonspecific and specific defense systems of the respiratory tract. These studies lend support to the notion that inhaled woodsmoke contributes to the increased incidence of infectious respiratory disease reported in children living in developing nations and/or near homes heated by woodburning devices.

Effects of inhaled woodsmoke were most dramatic after acute, high-dose exposure. While effects observed at woodsmoke concentrations of 750 μg PM/m^3 may not be especially relevant for developed nations, levels much higher than 1 mg/m^3 are commonly encountered in developing countries where about 15% of the total energy supply comes from wood. Thus, high-dose studies that examine effects related to the majority of the world's population are critical and should continue to be carried out. In addition, while some evidence is also provided that long-term exposure to lower concentrations of woodsmoke, more reflective of those encountered in North America, may also pose some health risks, more studies are needed before any definitive conclusions can be reached regarding the health risks (if any) associated with such exposures.

Making interspecies comparisons between humans and other mammalian species is complicated and needs to be approached with caution due to differences in anatomy, breathing rates, metabolism, and particle deposition. However, results of these studies lend support to the applicability of laboratory animals as a model to predict woodsmoke-induced alterations of human pulmonary health. Both the similarities and differences in woodsmoke-induced effects seen between humans and laboratory models underscore the importance of comparative studies as a basis for extrapolation modeling. Although more toxicologic studies are needed to determine the effects of long-term exposure, and to identify the woodsmoke constituents responsible for the observed toxicities, it is clear that inhalation of woodsmoke can have a significant impact on pulmonary homeostasis and/or exacerbations of ongoing lung disease processes.

SUMMARY, RECOMMENDATIONS, AND CONCLUSION

Even though woodsmoke is natural, it is not benign. Indeed, there is a considerable and growing body of epidemiologic and toxicologic evidence that both acute and chronic exposures to woodsmoke in developed country populations, as well as in the developing world, are associated with adverse health impacts.

Summary

Chemical composition. Woodsmoke contains thousands of chemicals, many of which have well-documented adverse human health effects, including such commonly regulated pollutants as fine particles, CO, and nitrogen oxides as well as ciliatoxic respiratory irritants such as phenols, cresols, acrolein, and acetaldehyde; carcinogenic organic compounds such as benzene, formaldehyde, and 1,3 butadiene; and carcinogenic cyclic compounds such as PAHs. Woodsmoke contains at least five chemical groups classified as known human carcinogens by the International Agency for Research on Cancer (IARC), others categorized by IARC as probable or possible human carcinogens, and at least 26 chemicals listed by the U.S. EPA as hazardous air pollutants. Among the currently regulated pollutants in woodsmoke, fine particles ($PM_{2.5}$) serve as the best exposure metric in most circumstances and, in addition, tend to be among the most elevated in relation to existing air quality standards.

Toxicology. Most available animal studies indicate that exposure to woodsmoke results in significant impacts on the respiratory immune system and at high doses can produce long-term or permanent lesions in lung tissues. Based on relatively few studies, these effects seem most strongly associated with the particle phase. Woodsmoke is also mutagenic and possibly carcinogenic in laboratory and field studies, but less so than coal smoke. Not enough is currently known to reliably distinguish the toxicological effects of different types of biomass smoke (e.g., smoke from combustion of wood versus agricultural wastes). More work in this area is needed so as to better understand the mechanisms by which adverse effects observed in exposed individuals might occur.

Exposures. Measured in the form of fine particles, significant woodsmoke exposures, mostly in winter, occur indoors and outdoors in all areas of the developed world where wood is used for residential heating and in fireplaces. Woodsmoke often comprises a significant fraction of ambient particle levels in such areas, on both a daily and an annual basis. In developing countries, such exposures occur indoors at concentrations that can be orders of magnitude greater than those observed in the developed

world. Wildland fires and agricultural burning can generate enormous quantities of smoke and can impact populated areas, albeit infrequently. Occupational exposures can be extremely high for wildland firefighters. Woodsmoke-specific chemical tracers provide the potential for increased understanding of the contribution of ambient woodsmoke concentrations to indoor and personal woodsmoke exposures in settings where multiple sources of fine particles are present. Continuous personal monitoring of woodsmoke pollutants (e.g., CO, PM) is useful for developing microenvironmental exposure models that could subsequently be combined with questionnaire data to more accurately predict individual-level exposures to woodsmoke when personal monitoring is not feasible.

Epidemiology: Wildland fires and agricultural burning. Although rarely combined with individual exposure assessment, a number of studies have found associations between wildfires and emergency room visits for both upper and lower respiratory tract illnesses, (including asthma), respiratory symptoms, and decreased lung function. In one study, particulate matter in wildfire smoke resulting from a major episode in Southeast Asia was associated with increased cardiopulmonary mortality, although this is the only study to have specifically evaluated mortality as an outcome in relation to wildfire smoke. Though less well documented, exposures to smoke from agricultural burning may also be linked with adverse respiratory outcomes, particularly exacerbations of asthma. Only one study has examined the efficacy of various intervention strategies to reduce exposure and possibly morbidity among the general population during wildfires. Thus, there are few data on which to base recommendations to the general population on effective measures to reduce exposures. Several studies have documented cross-seasonal effects of wildland fire smoke exposure on firefighters' lung function. Long-term consequences of repeated occupational exposures to such extraordinarily high concentrations of vegetation smoke have not been investigated, however.

Epidemiology: Residential wood combustion exposures in developed countries. Surprisingly relatively few studies examining the health impacts of woodsmoke have been conducted in developed countries, partly due to the difficulty of disentangling risks due to woodsmoke from those associated with other pollutants also present. In addition, most available studies are ecologic in design, limiting the ability to infer causality. Those that have been done, however, indicate that exposure to the smoke from residential woodburning is associated with a variety of adverse respiratory health effects, which are no different in kind and, with present knowledge, show no consistent difference in magnitude of effect from other combustion-derived ambient particles. The few studies that are available seem to indicate a somewhat smaller effect of woodsmoke on cardiovascular than respiratory effects, but the actual public health implications would depend on the background rates of these diseases as well as other factors. No studies seem to be available related to cancer endpoints in developed countries.

Exposures and epidemiology in developing countries. Exposures to biomass smoke are common in nearly half the households in the world that use wood, crop residues, or animal dung for cooking and heating. Although poorly characterized overall, such biomass smoke exposures are substantially higher than those in developed countries. In more than a dozen studies each, both chronic obstructive lung disease and acute lower respiratory tract infections have been strongly associated with these household exposures, leading to an estimate by WHO of 1–2 million premature deaths per year globally. Multiple studies have also shown relationships with tuberculosis, cataracts, adverse birth outcomes, lung cancer, and asthma. Biomass smoke is also an important part of outdoor air pollution in many developing-country cities, although no studies seem to have been done to separate out its impacts from those of other pollutants.

Hundreds of studies have examined the relationships between outdoor pollutants and disease around the world. To the extent that woodsmoke contributes to individual airborne chemicals, such as CO, nitrogen oxides, and benzene, the conclusions of these studies can be applied to those same chemicals in woodsmoke because, being specific molecules, they do not vary by source. It is less clear, however, whether woodsmoke-associated particles are differentially toxic relative to particles from other sources that have been dominant in most epidemiologic studies of ambient air pollution. Since particles are probably the single most important disease-associated constituent of woodsmoke,* an assessment of their hazard is crucial for evaluating the overall hazard of the woodsmoke mixture.

Perhaps because of long human associations with woodsmoke particles and the consequent perception that they are "natural" and thus somehow less hazardous than particles from modern sources, such as fossil fuel combustion, there has been some reluctance to treat them equally, for example, in emissions standards. This effectively constitutes a decision that woodsmoke particles are actually less hazardous per unit concentration than "average" ambient particles, such as those in diesel exhaust. Although the database is not as extensive as those for other major air pollutants, the weight of the evidence, consisting of animal and in vitro toxicology, the human exposure data, and epidemiologic studies of wildfires and of household wood combustion, indicates that woodsmoke particles are hazardous to human health. Specifically, our review suggests that there is sufficient evidence from the available literature that air pollution from biomass combustion is associated with a range of adverse respiratory health impacts and little evidence to suggest reduced or altered toxicity from these particles relative to the more commonly studied urban air PM. Most of the epidemiologic studies, however, have focused on respiratory health outcomes, in contrast to the recent emphasis on cardiovascular effects of urban

*In terms of health impact. The largest constituents of woodsmoke in terms of mass are, in order, CO_2 and CO.

and regional airborne particle exposures. Likewise, there are no toxicological data examining the effects of woodsmoke on cardiovascular outcomes. Thus, at present there are insufficient data to assess the extent to which ambient woodsmoke pollution might affect the circulatory system.

Since source apportionment studies show that woodsmoke is a major contributor to PM in many communities, it is likely that woodsmoke exposure plays a role in the spectrum of adverse effects linked to PM exposure. The large effects seen at higher exposures in the developing world provide additional evidence of the toxicity of woodsmoke. Further, there is evidence that biomass combustion globally is not decreasing and may in fact increase as a result of climate change-related increases in wildfires and shifts to the use of renewable fuels as the costs of fossil fuels, including natural gas, continue to rise. Although not reviewed here, the evidence of health impacts from exposures to the most well-studied biomass smoke (i.e., from burning tobacco) also strongly support this conclusion. Most relevant in this regard are the well-documented health impacts of environmental tobacco smoke (ETS or SHS, "second-hand smoke"), for which exposure levels are much closer to those typically experienced for woodsmoke in developed countries than exposures experienced by active smokers.

Recommendations for Further Research

Although many potential research avenues exist, here we note what we believe are the most critical gaps in our knowledge of the health effects of smoke from combustion of wood and other biomass:

Chemical composition

- Better understanding of the similarities and differences of smokes generated by combustion of different categories of biomass in different conditions, including wood and major crop residues.
- Utilization of recent advances in analytical chemistry (e.g., LC/MS techniques) to identify and quantify a wider range of chemicals in biomass smoke.
- Linkage of toxicological studies with comprehensive chemical composition measurements to identify specific chemicals or compounds classes responsible for the toxicity of biomass smoke.
- Fate of these chemicals in ambient air. Which ones break down and which ones linger?

Toxicology

- More long-term animal inhalation studies at concentrations relevant to indoor and outdoor woodsmoke levels found in both developed and developing countries, as well as comparative studies of both acute and chronic effects of exposures to particle phase woodsmoke and particles from other sources.
- Studies that provide information concerning the constituents associated with adverse health outcomes should be considered for developing intervention strategies.

Controlled human exposures

- More chamber studies that can elucidate the acute effects of high exposure to major types of biomass smokes.
- Following research in the second-hand smoke (ETS) literature, it would be useful to look for short-term physiologic responses to short-term changes in woodsmoke exposures in controlled or semicontrolled settings.

Exposure assessment

- More source and exposure apportionment studies are needed to determine the degree to which residential wood combustion contributes to both indoor and outdoor particle exposures in areas where wood smoke is likely to represent a significant source of particle exposure.
- Reliable biomarkers are needed to assist in epidemiologic studies.

Epidemiology

- Find better ways to combine source and exposure apportionment assessments in epidemiologic studies designed to determine the proportion of particle-associated health effects attributable to woodsmoke.
- Undertake studies among populations exposed primarily to woodsmoke particles, at least seasonally.
- Conduct studies focused on cardiovascular and cancer effects to compare with risks from fossil fuel-derived ambient particles, particularly for cardiovascular effects.
- More research is needed to assess both potential exposure reductions achievable through interventions and the real health impacts of such interventions to protect the public exposed to smoke from wildfires.

*Exposures and epidemiology: Developing countries**

- Accelerate efforts to quantify the potential exposure reductions and health benefits of practical interventions to control indoor exposures to biomass smoke, including better ventilation such as chimneys, better combustion, and better fuels.
- The high household exposures to biomass smoke common in developing countries present opportunities for research on heath effects of complex pollutant mixtures (fine and ultrafine PM, CO, benzene, PAH, etc.) of interest globally and to reduce significant health risks to hundreds of millions of the poorest people in the world.

*Because the primary emphasis of this article is not developing countries, only two recommendations are listed here.

Conclusions

Recognizing the limitations of current knowledge and need for additional information, we nevertheless offer preliminary answers to the questions raised in the introduction:

The hazards of woodsmoke as a mixture. Because woodsmoke is made up of such a large mixture of different chemicals, it is impossible at present to attempt to accurately assess its health impacts by simply summing the potential effects of individual constituents. (Indeed, there are few if any examples in which the effects of mixtures are fully reflected by the summed toxic potentials.) Particularly in high-exposure situations with fresh woodsmoke, as with occupational exposures or vegetation fire episodes, there may be a need to derive indices of exposure that take into account a range of toxic endpoints due to woodsmoke, for example, including acute-acting as well as chronic toxicants, so that appropriate protective actions can be adequately taken. Use of fine particles or any other single metric by itself may not be sufficient in these circumstances.

Woodsmoke particles. Nevertheless, at the present time fine particles may represent the best metric to characterize exposures to smoke from residential wood combustion and from wildfire smoke. There is no persuasive evidence that woodsmoke particles are significantly less dangerous for respiratory disease than other major categories of combustion-derived particles in the same size range. There is too little evidence available today, however, to make a judgment about the relative toxicity of woodsmoke particles with respect to cardiovascular or cancer outcomes.

Table 6 indicates that millions of people are exposed to smoke from household combustion of wood and other sources of biomass burning. Given the recent upward trend in the costs of oil and natural gas, it is likely that residential biomass combustion will become even more widespread throughout both the developed and developing world. More explicit efforts to reduce emissions from small-scale biomass smoke sources are likely to become even more important in the near future in order to meet air quality goals set to protect health.

Finally, returning to the questions posed at the start, we conclude that although there is a large and growing body of evidence linking exposure to wood/biomass smoke itself with both acute and chronic illness, there is insufficient evidence at present to support regulating it separately from its individual components, especially fine particulate matter. In addition, there is insufficient evidence at present to conclude that woodsmoke particles are significantly less or more damaging to health than general ambient fine particles.

Nevertheless, given the importance of woodsmoke as a contributor to particle concentrations in many locations, strategies to reduce woodsmoke emissions may be an effective means of lowering particle exposures. In addition, given the weight of toxicologic evidence, additional epidemiologic studies are needed to confirm our conclusions.

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