

United States
Environmental Protection
Agency

Office of Air Quality
Planning and Standards
Research Triangle Park, NC 27711

EPA-453/R-93-036
December 1993

Air



A SUMMARY OF THE EMISSIONS CHARACTERIZATION AND NONCANCER RESPIRATORY EFFECTS OF WOOD SMOKE



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**A SUMMARY OF THE EMISSIONS CHARACTERIZATION AND
NONCANCER RESPIRATORY EFFECTS OF WOOD SMOKE**

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Prepared for:

Air Risk Information Support Center (Air RISC)
US Environmental Protection Agency

Co-sponsored by:

Office of Air Quality Planning and Standards
Office of Air and Radiation
Research Triangle Park, NC 27712

Environmental Criteria and Assessment Office
Office of Health and Environmental Assessment
Office of Research and Development
Research Triangle Park, NC 27712

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EXECUTIVE SUMMARY

During the past twenty years, the use of wood has become popular as an alternative to conventional home heating fuels. This report summarizes the current state of knowledge concerning chemical composition of wood smoke, emission rates from different wood burning devices, impacts from wood burning on airborne particle levels, and the human respiratory responses to inhaled wood smoke.

Wood consists of approximately 50 to 70 weight percent cellulose and hemicellulose, which are polysaccharides, and about 30 weight percent lignin, which is a skeletal network of branch-chain polymers that provide structural integrity. Cordwood heaters burn wood with a deficit of oxygen and readily generate products of incomplete combustion, including carbon monoxide and numerous organic compounds. If these vapors are not immediately oxidized, they cool as they are exhausted to the atmosphere with subsequent formation of fine particles rich in relatively high molecular weight organic compounds.

Exposure to wood smoke can occur via a number of pathways. In addition to outdoor exposure, indoor exposure to elevated levels of wood smoke can occur either from the use of a wood burning appliance or by infiltration of outdoor air. In the past ten years, there have been a number of studies documenting the outdoor levels of airborne particles resulting from wood burning. The average night time levels of fine particle wood smoke vary from location to location. As expected, levels are higher in residential areas than in downtown urban or industrial areas and generally higher at night than during the day. The agreement between different source apportionment methods, when compared, is good. Although smoke levels in outdoor air are important, most people spend a majority of their time indoors, especially at night in residential areas.

Epidemiological investigations of adverse respiratory effects of wood smoke emissions in the US have centered on either symptomatology or pulmonary function. The symptoms measured have been the traditional respiratory disease outcomes; that is, cough, wheeze, and upper or lower respiratory infection. Pulmonary functions measured have been FEV₁, a measure of air flow limitation caused by obstruction in the airways, or FVC, a measure of the total amount of air that can be forcibly exhaled from the lungs. There are eight published reports of associations between residential wood smoke and lung function in children studied in the field and one study of responses in adult subjects. All but one of the studies found adverse respiratory outcomes associated with exposure to wood smoke. The adverse respiratory effects noted were increased respiratory symptoms, increased lower respiratory infection and decreased pulmonary function. These different endpoints

show a coherence of the data which is all the more remarkable when one considers that only one of the eight studies targeted a susceptible population of children (asthmatics). This coherence supports the plausibility of a case-effect relationship. However, a biological gradient (dose response) has not been shown, most likely due to the lack of exposure assessment data in most of the available studies.

INTRODUCTION

During the past twenty years, the use of wood has become popular as an alternative to conventional home heating fuels. Part of this movement has been due to uncertainty about the availability of fossil fuels. About ten percent of space heating in urbanized areas of the northern U.S. is from wood burning, with up to fifty percent in some smaller, rural towns (Skog and Wattersen, 1983; Lipfert and Dungan, 1983; Hough, 1988; Pierson et al, 1989). Wood is obviously a renewable resource. This attraction is offset, however, by the increased air pollution emissions from wood heating devices compared with devices fueled with oil or gas. As noted initially by Cooper (1981), particle and organic carbon emission rates can be as much as one to two orders of magnitude larger in wood heating devices than in oil or gas heating units. Not surprisingly, legislation restricting the sale of conventional wood stoves first appeared in Oregon in 1984, followed by nationwide restrictions in 1988. Although rapid progress has reduced emissions in some types of modern wood heaters, older "conventional" wood stoves and fireplace inserts are still the predominant appliance in use today. As discussed later in this document, a number of communities continue to experience elevated levels of wood smoke during the winter heating season. In addition, elevated indoor air pollution levels have been observed in homes with non-airtight or improperly operated wood stoves. As a result, there has been an ongoing interest in the potential health effects of exposure to wood smoke. Several reviews of the health effects of wood smoke have been prepared (Ammann, 1986; Koenig et al, 1988; Anderson, 1989; Pierson et al, 1989; Marbury, 1991; Dost, 1991). The present review will attempt to summarize the available information on the chemical and physical properties of wood smoke, levels of wood smoke in both indoor and outdoor environments, emission data and the adverse respiratory responses in animal toxicity studies and epidemiologic studies of human populations.

WOOD COMBUSTION AND WOOD SMOKE

Most wood burned for heat is cordwood with some increasing use of wood pellets. Cordwood heaters burn wood with a deficit of oxygen and readily generate products of incomplete combustion, including carbon monoxide and numerous organic compounds. If these vapors are not immediately oxidized, they cool as they are exhausted to the atmosphere with subsequent formation of fine particles rich in relatively high molecular weight organic compounds. A "conventional" wood stove fits this description. In order to reduce emissions from cordwood heaters, these vapors are oxidized directly downstream of

the combustion zone either by using a noble metal catalyst to more completely combust vapors at the lower exhaust temperatures, or by using an insulated secondary combustion chamber in order to maintain a high exhaust temperature while mixing the gases with a separate stream of additional combustion air. The former method is employed in "catalytic" stoves, the latter in "non-catalytic" or "high technology" stoves. In contrast to cordwood heaters, pellet stoves take advantage of the larger wood surface area per unit mass of wood. Consequently, the higher heat transfer rates from the combustion gases to adjacent, uncombusted wood results in efficient vaporization of the wood prior to combustion. Mixing these vapors with excess combustion air at the top of a pellet bed results in much more complete combustion than that in conventional stoves.

Table 1 compares the emission rates from each of these wood heating devices. Values are summarized from both controlled, laboratory tests and also from actual in-home, field tests. As expected, conventional stoves emit greater amounts of particulate matter and carbon monoxide per kilogram of wood than do catalytic, non-catalytic and pellet stoves. The pellet stove appears to be the consistently lowest emitter in both laboratory and field tests. Notice, however, that both the non-catalytic and pellet stoves, which burn wood at relatively high temperatures, emit greater amounts of nitrogen oxides than do the conventional or catalytic stoves, which burn wood at relatively lower temperatures. Higher temperatures favor formation of nitrogen oxides and oxidation of organic vapors, including both condensible organic compounds as well as volatile organic compounds (VOCs).

The large variability in emission rates for a given appliance is due to a number of factors including stove design, wood moisture content, and burn rate. High wood moisture content reduces combustion temperatures and efficiency. In conventional stoves, increasing burn rate increases combustion temperatures and efficiencies, but in catalytic and non-catalytic devices the higher burn rates actually decrease combustion efficiency by decreasing the times in the secondary combustion zone. Increased burn rates also increase PAH emissions independent of stove type (McCrillis and Burnet, 1990). Pellet stoves operate at a fixed burn rate and are not as susceptible to operator variability.

Compared to wood stoves, we know little about fireplace emissions. Here we distinguish fireplaces from conventional fireplace inserts. Inserts are a home heating device with emissions similar to conventional wood stoves. Standard, open fireplaces can be a net home cooling device because of the large amounts of air they draw from outside during maximal burn rates. In general, conventional fireplaces emit comparable amounts of particulate matter and less carbon monoxide per kg wood burned compared to conventional wood stoves. However, fireplaces usually operate at higher wood burn rates and for shorter time periods than most wood heating devices.

It is clear from Table 1 that we know relatively little about the chemical composition of wood stove exhaust as a function of stove type. Most studies of the chemical composition of wood smoke have been conducted using conventional wood stoves. Despite this, we can make a number of general conclusions based upon our knowledge of the wood combustion process and the measurements of exhaust from conventional stoves. Table 2 summarizes the reported constituents in wood smoke and provides quantitative information on their emission rates.

Wood consists of approximately 50 to 70 weight percent cellulose and hemicellulose, which are polysaccharides, and about 30 weight percent lignin, which is a skeletal network of branch-chain polymers that provide structural integrity. In addition, there are small amounts of resinous materials and inorganic salts. The lignin polymer consists of two main monomers, a guaiacyclopropane structure and a syringylpropane structure. Upon heating, these structures break apart producing a large variety of smaller molecules, many of which are part of the general class of oxygenated monoaromatics (Steiber et al, 1992). Included in this class are methoxy phenols and methoxy benzenes, as well as phenols and catechols. This decomposition also produces benzene and alkyl benzenes. The presence of guaiacol, syringol and their derivatives as a group are unique to the burning of wood because they are a direct consequence of the destruction of the unique lignin structure. In contrast, phenols, catechols, benzene and alkyl benzenes are not unique to wood combustion and have been found in the exhaust gases of other combustion sources.

Hawthorne (1988, 1989) has identified approximately 30 guaiacol and syringol derivatives in wood smoke. Guaiacol was found in the vapor phase, substituted guaiacols in both vapor and particulate phases, and syringol and derivatives predominately in the particulate phase. These two classes of compounds comprised as much as twenty percent of the particulate carbon in the wood smoke. These structures can also be transformed at higher temperatures to naphthalene and substituted naphthalenes, the predominant PAHs found in wood smoke. The thermal destruction of polysaccharides are thought to be responsible for the formation of furan and its derivatives, including benzofuran and furfural (Edye and Richards, 1991). These same researchers point to the acetyl ester groups found in the hemicelluloses as the source of acetic acid in wood smoke.

Retene has also been proposed as a unique marker of wood smoke (Ramdahl, 1983). Retene is produced from the decomposition of abietic acid (a polar cyclic terpenoid), a resinous component of wood. However, other aromatic rich fuels will also produce condensed ring aromatics. Therefore retene may not be unique to wood burning.

Abietic acid and dehydroabietic acid as well as other cyclic di- and tri-terpenoids have also been identified in wood smoke. Woody plants also contain terpenes (C₁₀ cyclo and bicyclo-alkanes and alkenes). These compounds can boil off a low temperature wood fire and are found in wood smoke (Steiber and Dorsey, 1988). Because they are relatively volatile, they are also emitted directly into the air by trees and thus are not necessarily only produced by the combustion of wood.

The size distribution of wood smoke particles has been measured by several investigators (Dasch, 1982; Kamens et al, 1984; Kleindeinst et al, 1986). The particle volume peaks at between 0.15 and 0.4 micrometers, with essentially no particles greater than one micrometer. This is consistent with the fact that the majority of the mass is formed by condensation processes in the exhaust. Owing to their relatively small size, they are very efficient at reducing visibility and are not readily removed by inertial and gravitational processes.

PAHs and oxy-PAHs are formed during the combustion of a variety of fuels including wood. As mentioned previously, PAH emission rates increase with increasing burn rate, implying that the combustion conditions in the wood burner determine their formation and emission rates. However, it has also been observed that PAH emissions are higher in conventional stoves burning pine than in similar stoves burning hard wood, implying that fuel type is also a factor (Steiber et al, 1992; McCrillis and Burnet, 1990).

Of the trace elements, potassium is found at relatively high levels in wood smoke. Combustion of hardwoods produces more ash (and thus higher levels of trace elements) than does combustion of softwoods. The particulate elemental carbon levels reported in wood smoke are somewhat controversial. Some researchers claim that up to 95 weight percent of the total particle mass is extractable in dichloromethane and/or methanol (Steiber and Dorsey, 1988), while others claim that up to 50 percent of the total particulate carbon is elemental carbon as measured by optical and thermal methods (Rau, 1989). It seems reasonable to conclude that 5 to 20 percent of the total particulate mass is unextractable and that this unextractable fraction contains elemental carbon.

Upon release to the environment, many of the compounds in wood smoke are expected to undergo some degree of chemical transformation in the atmosphere. The relatively few studies of these transformations are summarized in Table 3. In fact, the only laboratory study of actual wood stove exhaust that was designed to observe the transformation of selected organic compounds was done under simulated daytime conditions in the presence of intentionally added nitrogen oxides (Kleindeinst et al, 1986). Under these photochemically active conditions, there was rapid destruction of the alkenes and furans as well as production of aldehydes including formaldehyde.

The nature and extent of these transformations in the dark without added nitrogen oxides has not been reported. We have only indirect evidence largely obtained from field measurements of selected compounds. These field studies compare the levels of a given compound or compounds in both the stack and the ambient air relative to each other and/or to a reasonably conservative tracer such as fine particle mass. If the relative amounts are similar in both the stack and the environment, then this constitutes evidence that the chemical transformations are relatively minor over the time of the measurement. If order-of-magnitude changes occur, then this implies chemical transformation has also occurred. The night time field studies listed in Table 3 found that the methoxy phenols and the total levels of one, two and three oxygen atom monoaromatics remain relatively constant relative to particle mass and to each other respectively. These observations imply that these compounds undergo relatively little atmospheric transformation during the night in winter. This result is interesting in light of the potential for substituted phenols to undergo relatively rapid reaction with nitrate radicals (Carter et al, 1981). In contrast to the stability of these selected compounds, the acidity of wood smoke particles appears to degrade upon release to the night time environment (Lindell, 1991). The specific acids that contribute to particle acidity are unknown. We do know, however, that some of the constituents of wood smoke are found in fogwater (Muir, 1991) and rainwater (Luenberger et al, 1985). This may be an important scavenging route for these compounds in the atmosphere.

EXPOSURE TO WOOD SMOKE

Exposure to wood smoke can occur via a number of pathways. In addition to outdoor exposure, indoor exposure to elevated levels of wood smoke can occur either from the use of a wood burning appliance or by infiltration of outdoor air. In the past ten years, there have been a number of studies documenting the outdoor levels of airborne particles resulting from wood burning. These studies are summarized in Table 4. We have included only those studies in this table that quantified the levels of airborne particles using one of several chemical tracer methods. There have also been a large number of studies that have documented elevated levels of particulate matter in residential communities where wood burning is prevalent, but have not employed receptor models to estimate the wood burning fraction. Perhaps the most notable of these are the measurements taken in Klamath Falls, Oregon that have exceeded $600 \mu\text{g}/\text{m}^3$ on a 24 hour basis during the winter (Hough, 1988). Based upon inventories of fuel use, wood smoke may account for as much as 80 percent of the airborne particle concentrations during the winter (Heumann et al, 1991). The Klamath Falls studies emphasize another important point- the location of the air

monitoring device. There is up to a four fold difference between various parts of town, with the highest readings in the residential area (Heumann et al, 1991). This same spatial variability was observed by Larson et al (1990) using a mobile nephelometer. They found that the nighttime drainage flow tended to concentrate the wood smoke at valley floors, with a consistent factor of two to three difference between ridge line and valley flow smoke levels. With this caveat in mind, we can see from Table 4 that the average night time levels of fine particle wood smoke vary from location to location. As expected, levels are higher in residential areas than in downtown urban or industrial areas and generally higher at night than during the day. The agreement between different source apportionment methods, when compared, is good.

Several studies are of interest to later discussions of the health effects of wood smoke. In addition to the Klamath Falls studies discussed above, the limited measurements by Carlson (1982) in Missoula, Montana indicated that the majority of the fine particle mass was due to wood burning. The measurements taken in Boise, Idaho also found that the majority of the extractable organic material found in fine particles was from wood burning, with the remainder due to mobile sources (Klouda et al, 1991; Lewis et al, 1991). Finally, the measurements taken in Seattle, Washington (Larson et al, 1990; Larson et al, 1992) indicate not only that there are elevated levels of wood smoke particles during winter evening periods at a residential location, but also that the majority of the fine particle mass at this location is due to wood burning all weeks of the year. The fine particle mass concentrations at this site are low in the summer, and therefore the absolute concentrations due to wood burning are about an order of magnitude less in summer than in winter. Open burning restrictions went into effect in this area in September, 1992. Thus wood burning is not expected in the summer from yard waste burning and land clearing fires.

Although smoke levels in outdoor air are important, most people spend a majority of their time indoors, especially at night in residential areas. Indoor exposure can occur not only from infiltration of outdoor air, but also from emissions into the home from a wood burning appliance. Table 5 summarizes our current knowledge of indoor wood smoke levels. Emissions occur into the home during fueling of the stove and may also occur during stove operation. More modern, airtight stoves generate less emissions directly into the home than older non-airtight stoves or improperly operated and/or maintained stoves. To put the emission rates listed in Table 5 into perspective, consider a 100 cubic meter room. If seven tenths of the volume of air in the room is exchanged with outside air every hour, and if 70 percent of the fine particles from the outside air penetrate into the home, then for a typical outdoor level of $20 \mu\text{g}/\text{m}^3$ of wood smoke particles there is an effective infiltration rate of $20 \times 0.7 \times 0.7 \times 100 = 1$ milligram per hour of fine particle mass. Higher

outdoor levels or more rapid air exchange rates would give larger infiltration rates. For a majority of the studies of fine particle mass in homes with airtight stoves, the indoor/outdoor ratios are at or below 1.0, implying that infiltration is important even in homes with stoves. Recently unpublished work in nine homes in Seattle, Washington without stoves and without smokers also found a median indoor/outdoor ratio less than 1.0 for over 3000 hours of continuous nephelometer data, with elevated indoor levels in wood burning neighborhoods (J. Anusewski, M.S.E. thesis, Univ. of Washington, 1992). As also shown in Table 5, the indoor/outdoor ratios are much higher for carbon monoxide and formaldehyde, two species that have a number of indoor sources in addition to wood stoves. The data for formaldehyde are particularly striking, implying that although wood stoves emit formaldehyde, their emissions are not a major determinant of overall exposure to this specie.

HEALTH EFFECTS

Many of the constituents of wood smoke described earlier are known to aggravate respiratory disease and irritate mucous membranes. Knowledge of the toxicity of a compound usually depends on data from three sources, animal toxicology, controlled human studies, and epidemiology. In this section, we review the available information on wood smoke exposures, with emphasis on studies of human subjects.

Animal toxicology:

We restrict our discussion of animal toxicology to those studies employing whole wood smoke; we do not discuss data on individual compounds found in wood smoke. Unfortunately there is a paucity of data on acute effects and no data on chronic effects of inhalation of wood smoke in animals. One study investigated the pulmonary toxicity of respirable combustion products from residential wood and coal stoves in guinea pigs (Beck and Brain, 1982). Suspensions of the respirable fraction of particles from wood and coal smoke were instilled in the lungs of guinea pigs who were then sacrificed. The lungs were lavaged and assayed for biochemical or cellular indicators of pulmonary damage. The response to wood smoke products was less than that from coal-derived products; however adverse effects were seen. There was an overall depression in macrophage activity, increases in albumin and lactose dehydrogenase (LDH) levels (both indicating damage to cellular membranes), and a large increase in red blood cell numbers.

A morphological study of injury from inhalation of white pine wood smoke in rabbits showed a reproducible, necrotizing tracheobronchial epithelial cell injury (Thorning et al, 1982). The exposure was achieved by combusting a 30.4 cm by 1.9 cm square piece

of white pine wood. Air pumped through the combustion chamber was delivered to the exposure chamber for head-only exposure of the rabbit. Carbon monoxide and aldehydes were chosen as the indicator species in this study; the concentration of particulate matter was not measured. Aldehyde levels ranged from 285 to 1707 ppm total aldehyde. The pattern of injury first appeared six hours after exposure and, at 24 hours after the exposure, ciliated and secretory cells were mostly destroyed. By 72 hours, cells began to recover their normal structural appearance. Another pathological result from wood smoke exposure comes from an investigation of smoke from burning pine wood delivered to the lungs of dogs (Brizio-Molteni et al, 1984). Significant increases in angiotensin-1-converting enzyme, a substance which regulates vasomotor activity in endothelial cells, was measured immediately after exposure and was even higher 30 minutes post exposure. This pathological change could be an initial step toward pulmonary hypertension which is a suggested risk factor for a myocardial infarction.

Two animal studies of toxicity of wood smoke were reviewed by Marbury (Marbury, 1991). She cited a study by Fick and co-workers (Fick et al, 1984) who studied the effects of wood smoke from Douglas fir on pulmonary macrophages in rabbits. In this study, young adult New Zealand white rabbits were exposed to smoke from pyrolysis of Douglas fir using a smoke combustion chamber. Three rabbits were exposed simultaneously; their heads were held in a plexiglass restraint which directed their noses into the smoke stream. These researchers reported significant changes in the numbers and functions of the macrophages after smoke exposure compared with control. There were a greater number of cells but their adherence and antibacterial activity was depressed. The other study, carried out by Wong and co-workers (Wong et al, 1984), demonstrated a blunted respiratory response to CO₂ in wood smoke exposed guinea pigs which may indicate disruption of respiratory neural control.

Clark and co-workers (Clark et al, 1990) studied the distribution of extravascular lung water after acute smoke inhalation in mongrel dogs. The exposures were for 2 hours. Extravascular lung water, determined using Evans dye which binds quickly to plasma albumin, was increased in the smoke exposed dogs compared with controls. Wood smoke was generated by burning a standard mixture of fir plywood sawdust and kerosene. Whether the plywood contained epoxys and the contribution of toxicity from kerosene were not discussed.

Extrapolation of the results of these animal studies to human populations living in areas with elevated wood smoke concentrations is very difficult. Instillation of material directly into the lung certainly is different from inhalation. Also inhalation from a smoke stream would result in breathing considerably higher concentrations of smoke than seen in

neighborhoods in the human breathing zone. None of the animal studies evaluated pulmonary function or symptoms of respiratory illness, the end points assessed in epidemiological studies.

Controlled laboratory studies:

No controlled laboratory studies of human subjects exposed to wood smoke per se have been reported. There are some related studies with formaldehyde and environmental tobacco smoke but these are not discussed here.

Studies of wildfire and prescribed burn fire-fighters:

Municipal fire-fighters are exposed primarily to smoke from synthetic products and these studies are not discussed in this report. Forest fire-fighters of wildfires or prescribed burns are exposed to very high concentrations of carbon monoxide, aldehydes, volatile organic compounds and particulate matter (Reinhardt, 1991). There are very few data describing the health effects of this level of exposure. Rothman and co-workers (Rothman et al, 1991) studied cross seasonal changes in pulmonary function and respiratory symptoms in 52 wildfire fighters in California. Both the forced expiratory volume in one second (FEV₁) and the forced vital capacity (FVC) were significantly decreased at the end of the season compared to values before the season began. Individual functional decreases appeared to be related to the duration of exposure. Significant changes in eye irritation and phlegm were also seen from beginning to end of the fire fighting season. No air monitoring data were given.

Studies in Developing Countries:

The health effects of inhalation of wood smoke has been documented in third world countries where women spend many hours close to an open unvented indoor fire used for cooking (Ammann, 1986; Marbury, 1991). Increased respiratory symptoms, decreased pulmonary function and large increases in the prevalence of chronic bronchitis have been reported in New Guinea, India and Nepal. However, measurements of particulate matter have not been reported in most of these studies. Other studies, such as one in Nepal, compared indoor particulate matter concentrations in huts using traditional cooking methods to those using an improved cookstove (Reid et al, 1986). Concentrations of total suspended particulate matter in the huts using the traditional method averaged 2.7 mg/m³; a similar average concentration associated with the improved cook stove was 1.0 mg/m³, still much higher than concentrations of particulate matter to which US populations currently are exposed. A recent report of indoor air pollution in a similar situation in China (indoor use of an open cooking fires) measured concentrations up to 25 mg/m³ PM₁₀ (Harris et al, 1992).

A recent clinical report describes a group of 30 nonsmoking patients whose lung disease may be due to wood smoke exposure (Sandoval et al, 1992). These individuals were seen in Mexico City and all had a history of living in the countryside away from urban air pollution. The smoke exposure was the result of the use of wood and biomass for home cooking. These patients had abnormal chest X-ray scans showing a diffuse, bilateral, reticulonodular pattern and evidence of pulmonary arterial hypertension. Their pulmonary function tests were consistent with a mixed restrictive-obstructive disease diagnosis. The authors suggest that this group of patients was suffering from wood-smoke inhalation-associated lung disease (WSIALD) (Sandoval et al, 1992).

Epidemiology:

Epidemiological investigations of adverse respiratory effects of wood smoke emissions in the US have centered on either symptomatology or pulmonary function. The symptoms measured have been the traditional respiratory disease outcomes; that is, cough, wheeze, upper or lower respiratory infection. Pulmonary functions measured have been FEV₁, a measure of air flow limitation caused by obstruction in the airways, or FVC, a measure of the total amount of air that can be forcibly exhaled from the lungs. All but one of the available studies have been carried out in children, most likely on the assumption that children are most at risk for adverse effects from inhaled irritants due to the small size of their lungs and also due to the immature nature of their immune system. Other advantages of children as subjects in studies of respiratory effects of air pollution are the relative lack of confounders such as years of cigarette smoking or occupational exposure. There is good precedence for suspecting that young children are vulnerable to inhaled agents from the numerous studies of the effects of environmental tobacco smoke on children's respiratory health. There are eight published reports of associations between lung function and wood smoke in children studied in the field and one study of responses in adult subjects. The results of these studies are summarized in Table 4. An additional study of the association between visits to emergency departments for asthma and fine particulate matter (Schwartz et al, 1993) is included since this study was conducted in Seattle where a considerable percentage of fine particles are produced by residential wood burning.

The earliest report of adverse health effects from exposure to wood smoke in the US came from Michigan. Honicky and co-workers studied 31 young children who lived in homes with wood stoves and compared them to 31 children who lived in homes with other sources of home heating (Honicky et al, 1985). They recorded respiratory symptoms over the telephone using a modified Epidemiology Standardization Project Children's Questionnaire (Ferris, 1978). The occurrence of cough and wheeze was much greater in children from the homes with stoves and in general, both moderate and especially severe

symptoms of respiratory diseases were significantly greater in the wood smoke exposed children ($p < 0.001$). No measurements of wood smoke were reported.

Previously Tuthill reported the results of an investigation of symptoms of respiratory illness and respiratory disease prevalence associated with wood smoke and formaldehyde exposure (Tuthill, 1984). Symptoms were collected using a questionnaire; chronic respiratory illness was defined as physician-diagnosed chronic bronchitis, asthma or allergies. The subjects were 399 children from kindergarten through the sixth grade. Two hundred fifty eight lived in homes with wood stoves and 141 lived in homes without stoves. Although he found increased risk ratio = 2.4 (confidence intervals 1.7-3.4) for exposure to formaldehyde (from off-gassing of building materials after new construction or remodeling, from foam insulation, or from wood burning), the risk ratio for exposure to wood smoke of 1.1 (0.76-1.7) was not significant. The difficulty assigning formaldehyde exposure to sources other than wood burning was not discussed, although it is consistent with data from studies reviewed in Table 3.

The effects of wood stoves on general respiratory health in preschool age children was studied by Butterfield and others (Butterfield et al, 1989). Ten symptoms of respiratory disease were tracked in 59 children during the 1985-86 winter heating season in the Boise, ID area. The ages of the children ranged from 1 to 5 1/2 years. The symptoms which were significantly associated with living in a home with a wood stove in use were frequency of wheeze, severity of wheeze, frequency of cough, and waking up at night with cough. An independent study of sources of extractable organic material in ambient particles in Boise during the 1986-87 heating season showed an average of 67% due to wood burning (Lewis et al, 1991).

Another study compared the incidence of lower respiratory tract infection in American Indian children with presence of a wood stove in the home (Morris et al, 1990). The children lived on the Navajo reservation in Arizona. Cases were children less than 24 months of age with lower respiratory tract infection (bronchiolitis or pneumonia) who were matched with a control case visiting the clinic as part of a well-child program. Fifty-eight age and gender matched pairs participated in the study. Forty-nine percent of the cases lived in home using wood-burning for heat, whereas only 33 percent of the control children lived in such home. In this study, living in a home with a wood burning source of heat was a risk factor for lower respiratory tract infection (odds ratio = 4.2, $p < 0.001$).

Heumann and co-workers (Heumann et al, 1991) studied pulmonary function in a group of elementary school children in Klamath Falls, Oregon using standard spirometric values. Pulmonary function test data were collected on 410 children in grades 3 through 6 at three time periods during the 1990 heating season. There was a significant decrease in

average FEV₁ and FVC among children who had the highest exposure to wood smoke. A preliminary report of this study was presented (Heumann et al, 1991); analysis is still ongoing.

The 1977 Montana legislature funded an extensive Montana Air Pollution Study (Johnson et al, 1990) which was designed to evaluate whether air pollution was associated with adverse health effects in urban centers. The study involved third, fourth, and fifth grade children in five Montana cities. It measured lung function both within and between communities. Thus lung function of school children living in communities with different levels of air pollution was ascertained. Also comparisons of lung function changes of school children and air quality within a single community were evaluated. Each child served as his or her own control and analysis of co-variance was used to test for statistical significance in the acute study within a single community. In the multi-city study, linear regressions and principal components techniques were used and appropriate adjustments were made for factors such as altitude which varied from city to city. Three-day averages of the pollutants were used. Both studies detected significant lung function effects associated with total suspended particulate matter (TSP) and both fine and coarse respirable particulate matter. Pulmonary function decrements ranged from 1% to 10%, 24-hour average TSP ranged from 24 to 128 $\mu\text{g}/\text{m}^3$ during the study period. Sources of the particulate matter were not identified, however the authors state that the particulate matter essentially was from wood burning and entrained dust. Measurements of fine particles (PM_{3.5}) during this period found 68% by weight attributable to wood smoke in Missoula, Montana (Carlson, 1982).

One study in Denver, CO of a panel of adult subjects with asthma was conducted evaluating the presence of a wood stove or fireplace in the home and symptoms of respiratory disease and shortness of breath (Lipsett et al, 1991). Using logistic regression analysis, the presence of a wood stove in the home was associated with shortness of breath in females and both shortness of breath and moderate or severe cough in males ($p < 0.01$ in all cases).

Two studies of the health effects of wood smoke have been conducted at the University of Washington. The first was a questionnaire study of respiratory health in areas of high and low ambient wood smoke pollution (Browning et al, 1990). The communities were chosen based on extensive air monitoring of wood smoke distributions in the greater Seattle area (Larson et al, 1990). Six hundred residences in each community were sent questionnaires and asked to answer for one adult and one children at each address. The initial questionnaire asked about chronic symptoms of respiratory disease, in mild, moderate and severe categories (Ferris, 1978). Two follow-up mailings asked about acute

symptoms over the past two weeks. During the study period, PM₁₀ concentrations in the low wood smoke area averaged 33 µg/m³; in the high wood smoke area, the average for the three months of the study was 55 µg/m³. Questionnaire responses were stratified by age; 1-5; 6-14; 14-44; 45-64; and ≥65. There were no statistically significant differences between residents of the high and low wood smoke communities, however there was a pattern of increased symptoms and chronic illness in children aged 1-5 in the area with high wood smoke.

These suggestive data stimulated another study in the same air shed. In this second study, pulmonary function was measured in third through sixth grade children in two elementary schools in the area characterized as being impacted by wood smoke (Koenig et al, in press). FEV₁ and FVC were measured before, during, and after the heating season in 326 children during 1988-89 and in just 26 children with asthma in 1989-90. Wood smoke was assessed using an integrating nephelometer, a light scattering device. In this airshed there is a high correlation between light scattering coefficient and PM₁₀. Analyses show that greater than 80% of particles in residential neighborhoods are from wood burning during winter months (Larson et al, 1992). Random and mixed effects models of statistical association were used to evaluate the relationships between lung function and wood smoke concentrations. Lung function measurements were compared with wood smoke concentrations for the previous 12 hour period from 7 PM to 7 AM. Statistically significant decrements in both FEV₁ and FVC were seen in young children with asthma, both at the p < 0.05 level. FEV₁ and FVC dropped an average of 34 ml and 37 ml respectively for each unit of light scattering coefficient (1 X 10⁻⁴ m⁻¹). During the study period the PM₁₀ levels were over 90 µg/m³ on four nights in 1988-89 but not above 110; the highest value during 1989-90 was 103 µg/m³. Thus the National Ambient Air Quality Standard for PM₁₀ was not violated during either heating season. It was concluded that wood smoke is significantly associated with respiratory function decrements in young children with asthma.

A study of the relationship between fine particulate matter and emergency room visits for asthma in the metropolitan Seattle area was designed to help confirm whether air pollution was a risk factor for asthma (Schwartz et al, 1993). Using Poisson regressions controlling for weather, season, time trends, age, hospital, and day of the week, a significant association (p < 0.005) was found between fine particles measured at the residential monitoring station used in the studies described above and visits to emergency departments in eight participating hospitals. Analyses show that between 60 and 90% of particles in residential neighborhoods measured either gravimetrically or by nephelometers are from wood burning year round (Larson et al, 1992).

Suspended particulate air pollution is associated with decreased lung function and increased prevalence of respiratory disease symptoms in young children under 12 years of age (Dockery et al, 1989). In this two-year study of the relationship between pulmonary function changes in third and fourth grade children and air pollutant alerts in Steubenville, Ohio, researchers found a decline in pulmonary function tests associated with increasing 24-hour concentrations of total suspended particulate matter (TSP). Peak values of TSP ranged from 27 $\mu\text{g}/\text{m}^3$ to 422 $\mu\text{g}/\text{m}^3$. The pulmonary function declines were small but persisted for up to two weeks. The elimination of children with reported prevalence of coughs, colds and other respiratory symptoms did not change the estimated mean effect. Similar findings were reported from the Netherlands (Dassen et al, 1986) in a study of children aged 6-11 years before and during an air stagnation episode, although the effects of allergy and chronic respiratory disease were not evaluated. More recently, Dockery and co-workers (Dockery et al, 1982) have reported increased rates of cough, bronchitis and chest illness in children exposed to inhaled particulate pollution.

It certainly is biologically plausible that wood smoke could cause adverse respiratory effects. The average size of the particles ($< 1 \mu\text{m}$) is such that these will travel deep into the lower respiratory tract (Ammann, 1986). Some of the chemical species in wood smoke are chemically reactive and thus present a risk to respiratory tissues. The complex mixture of wood smoke allows deposition of reactive chemical onto particles which then can be carried into the alveolar region of the lung. As stated by Ammann (Ammann, 1986), "irritants such as phenols, aldehydes, and quinones, as well as nitrogen oxides and sulfur oxides, in smoke may also contribute to both acute and chronic health problems. Generally irritants interfere with ciliary activity ... and hence the flow of the particle-trapping mucous stream. Inflammation, with all of its sequelae, also results."

In the earlier Six City Study report of children in Steubenville (Dockery et al, 1989), a group median estimate of the slope between FVC and total suspended particulate was $-0.081 \text{ mL}/\mu\text{g}/\text{m}^3$ for all children. When the estimate of a similar relationship (FVC/measure of fine particle concentration) is made using the Seattle data (Koenig et al, in press), the estimated mean FVC decrease per unit increase of $\text{PM}_{2.5}$ is $-1.8 \text{ mL}/\mu\text{g}/\text{m}^3$ and $+0.34 \text{ mL}/\mu\text{g}/\text{m}^3$ for asthmatic and nonasthmatic children respectively. The FVC change per unit increase in $\text{PM}_{2.5}$ for the asthmatic children in our study is sufficiently pronounced as to suggest that fine particulate matter measured with a nephelometer may be more irritating than general industrial TSP. However, the difference between the two studies may be due solely to a increased sensitivity to airborne irritants in children with asthma. Based on prior work by Larson (Larson et al, 1990), the fine particulate matter measured

on winter nights in this Seattle residential area is almost exclusively the result of residential wood-burning.

SUMMARY

In conclusion, this review summarizes extensive information about the constituents and fate of wood smoke but, due to limited data, less information about the health effects. Animal toxicological studies show that wood smoke exposure can disrupt cellular membranes, depress macrophage activity, destroy ciliated and secretory respiratory epithelial cells and cause aberrations in biochemical enzyme levels. With respect to the human epidemiological data, the literature summarized in Table 4 show a coherence of the data from young children, with seven of eight studies reporting increased respiratory symptoms, lower respiratory infection and decreased pulmonary function as a result of exposure to wood smoke. The findings were especially pronounced in the study of children with asthma. As Bates (1992) has discussed the coherence of the data, although not amenable to statistical tests, carries the weight of linkage and plausibility. These adverse respiratory effects associated with wood smoke exposure also comply with many of Brandon Hill's aspects of association necessary to establish causation (Hill, 1965). There is strength of association, consistency (seven of eight studies showing positive associations), temporality, plausibility, coherence, and analogy (using ETS exposure; Nat. Res. Council, 1986; US EPA, 1992). A biological gradient (or dose response) has not been shown, although one is suggested in the study of pulmonary function in wildfire fighters. We conclude that the preponderance of the data suggest a causal relationship between elevated wood smoke levels and adverse respiratory health outcomes in young children.

References

- Alfheim I, Becker G, Hongslo, JK, Ramdahl, T. 1984. Mutagenicity testing of high performance liquid chromatography fraction from wood stove emission samples using a modified salmonella assay requiring smaller sample volumes. *Env. Mut.* 6: 91-102
- Ammann H. 1986. Health implications of wood smoke. International Congress on Residential Wood Energy; Conferences and Institutes, Reno. WSU WWREC pp. 331-48
- Anderson N. 1989. Risk assessment document for residential wood combustion emissions. Maine Department of Human Services
- Baechler MC. The compatibility of house tightening and residential wood burning: A case study of Bonneville power administration's analyses and programs. *Proc. Int. Conf. Residential Wood Energy*, Reno. pp. 423-30
- Bardana EJ Jr, Mantanaro A. 1991. Formaldehyde; an analysis of its respiratory, cutaneous and immunologic effects. *Ann. Allergy* 66: 441-52
- Barnett SG, Roholt R. 1990. In home performance of advanced technology woodstoves during the 1988-89 heating season in Glens Falls, New York. Presented at 83rd Annual Meeting Air Waste Manage. Assoc. Pittsburgh
- Barnett SG, Houck JE, Roholt RB. 1991. In-home performance of pellet stoves in Medford and Klamath Falls, Oregon. Presented at 84th Annual Meeting Air Waste Manage. Assoc. Vancouver
- Barnett SG, McCrillis RC, Crooks RB. 1992. Evaluation of emissions from masonry heaters and masonry fireplaces in homes. Presented at 85th Annual Meeting Air Waste Manage. Assoc. Kansas City
- Bates DV. 1992 Health indices of the adverse effects of air pollution: The question of coherence. *Env. Res.* 59: 336-49
- Beck BD, Brain JD. 1982. Prediction of the pulmonary toxicity of respirable combustion products from residential wood and coal stoves. *Proc.: Residential wood and coal combustion. Specially Conference (SP 45) Air Pollut. Control Assoc.*, Pittsburgh

- Benedict R, Naylor M. 1988. Fine particulate receptor modeling in Las Vegas Using combined gaseous and particulate source profiles. In Transactions, PM10 Implementation and Standards, ed., C.V. Mathai, D.H. Stonefield. Air Pollut. Control Assoc. Pittsburgh , 518-30 pp.
- Benton G, Miller DP, Reimold M, Sisson R. 1982. A Study of Occupant Exposure to Particulates and Gases from Woodstoves in Homes. In Residential Solid Fuels: Environmental Impacts and Solutions, ed. J. A. Cooper, D. Malek, pp. 539-50. Beaverton: Oregon Grad. Center. pp 539-50
- Brizio-Molteni L, Piano G, Rice PL, Warpeha R, Fresco R, et al. 1984. Effect of wood combustion smoke inhalation on angiotensin-1-converting enzyme in the dog. Ann. Clin. Lab. Sci. 14: 381-89
- Browning KG, Koenig JQ, Checkoway H, Larson TV, Pierson WE. 1990. A questionnaire study of respiratory health in areas of high and low ambient wood smoke pollution. Pediat. Asthma All. Immunol. 4:183-91
- Burnet P, Edmisten NG, Tiegs PE, Houck JE, Yoder RA. 1986. Particulate, carbon monoxide, and acid emission factors for residential wood burning stoves. J. Air. Pollut. Control Assoc. 36: 1012-18
- Burnet PG. 1987. Performance monitoring of advanced technology woodstoves: field testing for fuel savings, creosote buildup, and emissions. EPA-600/7-87-026a and EPA-600/7/87-026b, Research Triangle Park: U.S. EPA
- Butcher SS, Sorenson EM. 1979. A study of wood stove particulate emissions J Air Pollut. Control Assoc.. 29: 724-28
- Butterfield P, LaCava G, Edmundson E, Penner J. 1989. Woodstoves and indoor air: The effects on preschoolers' upper respiratory systems. J. Env. Health 52: 172-73
- Calloway CP, Li S, Buchanan JW. 1989. A refinement of the potassium tracer method for residential wood smoke. Atmos. Environ. 23: 67-69

- Carlson JH. 1982. Residential wood combustion in Missoula, Montana: An overview of its air pollution contributions, health effects, and proposed regulatory solutions. In Residential Solid Fuels: Environmental Impacts and Solutions, ed. J. A. Cooper, D. Malek, pp. 539-50. Beaverton: Oregon Grad. Center.
- Carter WPL, et al. 1981. Environ. Sci. Technol. 15: 829-831
- Childers JW, Wilson NK, Barbour RK. 1988. Identification of semivolatile organic compounds in selected air sample extracts by gas chromatography/matrix isolation infrared spectrometry. Proc. EPA/Air Pollut. Control Assoc. Sym. Measure. Toxic Related Air Pollut., pp. 15-20. APCA Publication VIP-10 (EPA 600/-88-015)
- Chow JC, Watson JG, Frazier CA, Egami RT, Goodrich A, Ralph C. 1988. Spatial and temporal source contributions to PM₁₀ and PM_{2.5} in Reno, NV. In: Transactions, PM₁₀ Implementation and Standards, ed. C.V. Mathai, D.H. Stonefield, Air Pollut. Control Assoc. Pittsburgh, pp. 438-57.
- Claessens HA, Lammerts van Bueren LGD, van de Ven PM. 1986. Extraction and determination of polycyclic hydrocarbons from domestic stove aerosol. J. Aerosol Sci. 17: 639-42
- Clark WR, Nieman G, Hakim TS. 1990. Distribution of extravascular lung water after acute smoke inhalation. J. Appl. Physiol. 68: 2394-402
- Clement RE, Tosine HM, Ali B. 1985. Levels of polychlorinated dibenzo-p-dioxins and dibenzofurans in wood burning stoves and fireplaces. Chemosphere 14: 815-19
- Colome SD, Spengler JD. 1982. Residential indoor and matched outdoor pollutant measurements with special consideration of wood-burning homes. In Residential Solid Fuels: Environmental Impacts and Solutions, ed. J. A. Cooper, D. Malek, pp. 455-55. Beaverton: Oregon Grad. Center.
- Cooke WM, Allen JM. 1982. Characterization of emission from residential wood combustion sources. In Residential solid fuels. Eds. Cooper J, Malek D. Oregon Grad. School pp. 139-63

- Cooper JA, Currie LA, Klouda GA. 1981. Assessment of contemporary carbon combustion sources to urban air particulate levels using carbon-14 measurements. *Env. Sci. Tech.* 15: 1045-50
- Core JE, Cooper JA, Neulicht RM. 1984. Current and projected impacts of residential wood combustion on Pacific Northwest air quality. *J Air Pollut. Control Assoc.* 34: 138-43
- Core JE. 1989. Receptor modeling source profile development for the Pacific Northwest States: The Pacific Northwest source profile library. EPA Region X, States of Oregon, Washington, Idaho, Puget Sound Air Pollut. Control Agency and Lane Regional Air Pollut. Control Authority
- Dasch JM. 1982. Particulate and gaseous emissions from wood-burning fireplaces. *Env. Sci. Technol.* 16: 639-45
- Dassen W, Brunekreef B, Hoek G et al. 1986. Decline in children's pulmonary function during an air pollution episode. *J. Air Pollut. Control. Assoc.* 36: 1223-27
- DeAngelis DG, Ruffin DS, Reznik RB. 1980. Preliminary characterization of emissions from wood-fired residential combustion equipment, EPA-600/7-80-040, Research Triangle Park: U.S. EPA
- DeCesar RT, Cooper JA. 1982. The quantitative impact of residential wood combustion and other vegetative burning sources on the air quality in Medford, Oregon. In *residential Solid Fuels: Environmental Impacts and Solutions*, ed. J.A. Cooper, D. Malek, 1271 pp. Beaverton: Oregon Grad. Center.
- Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. 1989. Effects of inhalable particles on respiratory health of children. *Am. Rev. Respir. Dis.* 139: 587-94
- Dockery DW, Ware JH, Ferris BG Jr., et al. 1982. Change in pulmonary function in children associated with air pollution episodes. *J. Air Pollut. Control. Assoc.* 32: 937-42
- Dost FN. 1991. Acute toxicology of components of vegetation smoke. *Rev. Env. Contam. Toxicol.* 119: 1-46

- Dresser AL, Baird BK. 1988. A dispersion and receptor model analysis of the wintertime PM₁₀ problem in Telluride, Colorado. In: Transactions, PM₁₀ Implementation and Standards, ed. C.V. Mathai, D.H. Stonefield. Air Pollut. Control Assoc. Pittsburgh, pp. 458-471
- Edye LA, Richards GN. 1991. Analysis of condensates from wood smoke: Components derived from polysaccharides and lignins. *Env. Sci. Technol.* 25: 1133-37
- Ferris BG. 1978. Epidemiology standardization project. *Am. Rev. Respir. Dis.* 118: 1-53
- Fick RB, Paul ES, Merrill WW, Reynolds HY, Loke JSO. 1984. Alterations in the antibacterial properties of rabbit pulmonary macrophages exposed to wood smoke. *Am. Rev. Respir. Dis.* 129: 76-81
- Frigas E, Filley WV, Reed CE. 1984. Bronchial challenge with formaldehyde gas: lack of bronchoconstriction in 13 patients suspected of having formaldehyde-induced asthma. *Mayo. Clin. Proc.* 59: 295-99
- Green DJ, Bascom R, Healey EM, Hebel JR, Sauder LR. 1989. Acute pulmonary response in healthy nonsmoking adults to inhalation of formaldehyde and carbon. *Toxicol. Indust. Health* 28: 261-75
- Harris BB, Chapman RS, Mumford JL. 1992. Battery powered PM-10 indoor air samplers applied to unvented Third World residential sources. Presented at 85th Annual Meeting Air Waste Manage. Assoc. Kansas City.
- Hawthorne SB, Krieger MS, Miller DJ, Mathiason MB. 1989. Collection and quantitation of methoxylated phenol tracers for atmospheric pollution from residential wood stoves *Env. Sci. Technol.* 23: 470-75
- Hawthorne SB, Miller DJ, Krieger MS. 1989. Collection, identification, and quantitation of methoxylated phenols as tracers of wood smoke pollution in urban air. *Abs. Pap. ACS* 198: 116
- Hawthorne SB, Miller DJ, Barkley RM, Krieger MS. 1988. Identification of methoxylated phenols as candidate tracers for atmospheric wood smoke pollution. *Env. Sci. Technol.* 22: 1191-96

- Hawthorne SB, Miler DJ, Langenfeld JL, Krieger MS. 1992. PM-10 high-volume collection and quantitation of semi- and nonvolatile phenols, methoxylated phenols, alkanes, and polycyclic aromatic hydrocarbons from winter urban air and their relationship to wood smoke emission. *Environ. Sci. Technol.* 26: 2251-62
- Heumann MA, Foster LR, Johnson L, Kelly LE. 1991. Woodsmoke air pollution and changes in pulmonary function among elementary school children. Presented at 84th Annual Meeting Air Waste Manage. Assoc. Vancouver, BC
- Highsmith R, Merrill R, Zweidinger R. 1988a. Characterization of the indoor and outdoor air associated with residences using woodstoves in Raleigh, *Env. Int.* 14: 213-19
- Highsmith R, Rodes C, Zweidinger R, Lewtas J. 1988. Influence of residential wood combustion on indoor air quality of Boise, ID, residences. *Proc. EPA/Air Pollut. Control Assoc. Sym. Measure. Toxic Related Air Pollut.*, pp. 804-13. APCA Publication VIP-10 (EPA 600/9-88-015)
- Highsmith VR, Rodes CE, Zweidinger RB, Merrill RG. 1987. The collection of neighborhood air samples impacted by residential wood combustion in Raleigh, NC and Albuquerque, NM. *Proc. EPA/Air Pollut Control Assoc. Sym. Measure. Toxic Related Air Pollut.*, pp. 562-72. APCA Publication VIP-8 (EPA 600/9-87-010)
- Hill AB. 1965. The environment and disease: association or causation? *Proc. R. Soc. Med.* 58: 295-300.
- Honicky RE, Osborne JS III, Akpom CA. 1985. Symptoms of respiratory illness in young children and the use of wood-burning stoves for indoor heating. *Pediatrics* 75: 587-93
- Hornig JF, Soderberg RH, Larsen D, Parravano C. 1982. Ambient air assessment in rural village and small town locations in New Hampshire where wood is an important fuel. In *Residential Solid Fuels: Environmental Impacts and Solutions*, ed. J. A. Cooper, D. Malek, pp. 539-50. Beaverton: Oregon Grad. Center. pp 506-19

- Hough M. 1988. Oregon's approach to reducing residential woodsmoke as part of the PM-10 strategy. In: Transactions PM₁₀ Implementation and Standards, pp. 646-53. Air Pollut Control Assoc. Specialty Conference, San Francisco
- Imhoff RE. 1982. Final Report on a Study of the Ambient Impact of Residential Wood Combustion in Petersville, Alabama. In: Residential Wood and Coal Combustion Air Pollut. Control Assoc. Specialty Conference Proceedings SP-45, pp 161-88
- Jaasm DR, Gundappa M, Champion MR, McCrillis RC. 1991. Field performance of woodburning stoves in Crested Butte, Colorado. Presented at 84th Annual Meeting Air Waste Manage. Assoc. Vancouver
- Johnson KG, Gideon RA, Loftsgaarden DO. 1990. Montana air pollution study: Children's health effects. *J Official Stat.* 5: 391-407
- Kalman D, Larson TV. 1987. Puget Sound receptor modeling feasibility study final report. Submitted to the Puget Sound Air Pollution Control Agency
- Kamens RM, Rives GD, Perry JM, Bell DA, Paylor RF Jr., et al. 1984. Mutagenic changes in dilute wood smoke as it ages and reacts with ozone and nitrogen dioxide: An outdoor chamber study *Env. Sci. Technol.*, 18: 523-30
- Khalil MAK, Edgerton SA, Rasmussen RA. 1983. A gaseous tracer model for air pollution from residential wood burning. *Env. Sci. Technol.* 17: 555-59
- Kleindienst TE, Shepson PB, Edney EO, Claxton LD, Cupitt LT. 1986. Wood smoke: measurement of the mutagenic activities of its gas- and particle-phase photooxidation products. *Env. Sci. Technol.* 20: 493-501
- Klouda GA, Barraclough D, Currie LA, Zweidinger RB, Lewis, CW, Stevens RK. 1991. Source apportionment of wintertime organic aerosols in Boise, ID by chemical and isotopic(¹⁴C) methods. Presented at 84th Annual Meeting Air Waste Manage. Assoc., Vancouver, BC
- Klouda GA, Currie LA, Sheffield AE, Wise SA, Benner BA, et al. 1987. The Source Apportionment of Carbonaceous Combustion Products by Micro-Radiocarbon Measurements for the Integrated Air Cancer Project. *Proc. EPA/Air Pollut. Control*

- Assoc. Sym. Measure. Toxic Related Air Pollut., APCA Publication VIP-8 (EPA 600/9-87-010), pp. 573-78
- Knight CV, Humphreys MP, Kuberg DW. 1985. Summary of three-year study related to wood heater impacts on indoor air quality. Proc. Int. Conf. Residential Wood Energy, pp. 409-22. Reno.
- Koenig JQ, Covert DS, Larson TV, Maykut N, Jenkins P, Pierson WE. 1988. Wood smoke: Health effects and legislation. NW Env. J. 4: 41-54
- Koenig JQ, Larson TV, Hanley QS, Rebolledo V, Dumler K, et al. Pulmonary function changes in children associated with fine particulate matter. Env. Res. In press
- Kowalczyk JF, Greene WT. 1982. New Techniques for Identifying Ambient Air Impacts from Residential Wood Heating In Residential Solid Fuels: Environmental Impacts and Solutions, ed. J. A. Cooper, D. Malek, pp. 539-50. Beaverton: Oregon Grad. Center. pp.469-94
- Krzyzanowski M, Quackenboss JJ, Lebowitz MD. 1990. Chronic respiratory effects of indoor formaldehyde exposure. Env. Res. 52: 117-25
- Larson TV, Kalman D, Wang S, Nothstein G. 1990. Urban air toxics mitigation study. Report submitted to the Puget Sound Air Pollution Control Agency, June
- Larson TV, Koenig J Q. 1993. A summary of the chemistry, emissions, and non cancer respiratory effects of wood smoke. EPA 453/R-93-008
- Larson TV, Yuen P F, Maykut N. 1992. Weekly composite sampling of PM_{2.5} for total mass and trace elemental analysis. In: Proceedings of the Air Waste Manage. Assoc. Specialty Conference on Fugitive Emissions and PM₁₀ Control Strategies, Scottsdale. ISBN 0-92-32-04-09- Pittsburgh
- Lewis C, Baumgardner R, Stevens R, Claxton L, Lewtas J. 1983. The contribution of wood smoke and motor vehicle emissions to ambient aerosol mutagenicity. Env. Sci. Technol. 22: 968-71

- Lewis CW, Baumgardner RE, Stevens RK, Rosswurm GM. 1986. Receptor modeling of Denver winter haze. *Env. Sci. Technol.* 20: 1126-36
- Lewis CW, Stevens RK, Zweidinger RB, Claxton LD, Barraclough D, Klouda, GA. 1991. Source apportionment of mutagenic activity of fine particle organics in Boise, Idaho. Presented at 84th Annual Meeting Air Waste Manage. Assoc., Vancouver, BC
- Lindell AK. 1991. Acidity of airborne particles from residential wood burning. Presented at 84th Annual Meeting Air Waste Manage. Assoc. Vancouver
- Lipari F, Dasch JM, Scruggs, WF. 1984. Aldehyde emissions from wood-burning fireplaces. *Env. Sci. Technol.* 18: 326-30
- Lipfert FW, Dungan JL. 1983. Residential firewood use in the United States. *Science* 219: 1425-27
- Lipsett M, Ostro B, Mann J, Wiener M, Selner J. 1991. Effects of exposures to indoor combustion sources on asthmatic symptoms. Presented at 84th Annual Meeting Air Waste Manage. Assoc. Vancouver, BC
- Lofroth JE, Lazaridi G, Rudling L. 1986. L-Mutagenicity assay of emission extracts from wood stoves- comparison with other emission parameters. *Sci. Total E.* 58: 199-208
- Luenberger C, et al. 1985. *Environ. Sci. Technol.* 19: 1053-60
- Magliano KL. 1988. Level 1 PM₁₀ assessment in a California air basin. In: *Transactions, PM₁₀ Implementation and Standards*, ed. C.V. Mathai, D.H. Stonefield. Air Pollut. Control Assoc. Pittsburgh, pp. 508-517
- Marbury MC. 1991. Wood smoke. In *Indoor air pollution*, ed. J.M. Samet, J.D. Spengler, Ch 9. Baltimore: Johns Hopkins Press
- McCrillis RC, Burnet PG. 1990. Effects of burnrate, wood species, altitude, and stove type of woodstove emissions. *Toxicol. Indust. Health* 6: 95-102
- McCrillis RC, Butts NL. 1991. Advancing woodstove secondary combustion state-of-the-art. Presented at 84th Annual Meeting Air Waste Manage. Assoc. Vancouver

- Menon PK, Stankus RP, Rando RJ, Salvaggio JE, Lehr SB. 1991. Asthmatics responses to passive cigarette smoke: persistence of reactivity and effect of medications. *J. Allergy Clin. Immunol.* 88: 861-69
- Merrill R, Zweidinger R, Martz R, Koinis R. 1988. Semivolatile and condensable organic materials distribution in ambient and woodstove emissions. *Proc. EPA/ Air Pollut. Control Assoc. Sym. Measure. Toxic Related Air Pollut.*, pp. 828-34. APCA Publication VIP-10 (EPA 600/9-88-015)
- Miller DJ, Hawthorne SB, Langenfeld JJ. 1990. PM-1- hi-vol collection and quantitation of semi-volatile methoxylated phenols as tracers of wood smoke pollution in urban air. *Abs. Pap. ACS* 198: 105
- Morris K, Morganlander M, Coulehan JL, Gahagen S, Arena VC. 1990. Wood-burning stoves and lower respiratory tract infection in American Indian children *Am. J. Dis. Child* 144: 105-08
- Moschandreas DJ, Zabransky J, Rector HE. 1980. The effects of woodburning on the indoor residential air quality. *Environ. Int.* 4: 463-68
- Muir PS. 1991. fogwater chemistry in a wood-burning community, Western Oregon. *J. Air Waste Manage.* 41: 32-38
- Murphy D, Buchan RM, Fox DG. 1982. Ambient particulate and benzo(a)pyrene concentrations from residential wood combustion, in a mountain resort community. In *Residential Solid Fuels: Environmental Impacts and Solutions*, ed. J. A. Cooper, D. Malek, pp. 539-50. Beaverton: Oregon Grad. Center. pp. 495-505
- NAS Environmental Tobacco Smoke; Measuring exposures and assessing health effects. National Research Council, Washington DC, 1986.
- Naylor MN. 1985. Air pollution from fireplaces in Las Vegas, Nevada. Presented at 78th Annual Meeting Air Pollut. Control Assoc. Detroit

- Nestrick TJ, Larparski LL. 1982. Isomer-specific determination of chlorinated dioxins for assessment of formation and potential environmental emission from wood combustion. *Anal. Chem.* 54: 2292-99
- Neulicht RM, Core J. 1982. Impact of residential wood combustion appliances on indoor air quality. In: Residential Wood and Coal Combustion. Air Pollut. Control Assoc. Specialty Conference Proceedings SP-45, pp 240-52.
- OMNI Environmental Services. 1988. Environmental impacts of advanced residential and institutional (woody) biomass combustion systems. Final Report to U.S. DOE Pac. NW and Alaska Regional Biomass Energy Program Contract: DE AC79-86 BP61196
- Pierson WE, Koenig JQ, Bardana EJ Jr. 1989. Potential adverse health effects of wood smoke. *West J. Med.* 151: 339-42
- Pope CA III, Dockery DW, Spengler JD, Raizenne ME. 1991. Respiratory health and PM10 pollution; a daily time series analysis. *Am. Rev. Respir. Dis.* 144: 668-72
- Ramdahl T, Schjoldager J, Currie LA, Hanssen JE, Moller M, et al. 1985. Ambient impact of residential wood combustion in Elverum, Norway. *Sci. Total Env.* 36: 81-90
- Ramdahl T. 1983. Retinene - a molecular marker of wood combustion in ambient air. *Nature* 306: 580-82
- Rau JA. 1989. Composition and size distribution of residential wood smoke particles *Aerosol Sci. Technol.* 10:181-92
- Reid HF, Smith KR, Sherchand B. 1986. Indoor smoke exposures from traditional and improved cookstoves: Comparisons among rural Nepali women. *Mountain Res. Devel.* 6: 293-304
- Reinhardt TE. 1991. Monitoring firefighter exposure to air toxins at prescribed burns of forest and range biomass. U.S. Dept. Agriculture, Paper PNW-RP-441
- Romero AE, Buchman RM, Fox DG. 1978. A study of air pollution from fireplace emissions at Vail Ski Resort. *Env. Health* 41: 117-19

- Rothman N, Ford P, Baser ME, Hansen JA, O'Toole, T, et al. 1991. Pulmonary function and respiratory symptoms in wildland firefighters. *J. Occup. Med.* 33: 1163-71
- Sandoval J, Salas J, Martinez-Garcia M, Gomez A, Martinez C, et al. 1992 Pulmonary arterial hypertension and cor pulmonale associated with chronic domestic woodsmoke inhalation. *Chest* 103: 12-20
- Sauder LR, Chatham MD, Green DJ, Kulle TJ. 1986. Acute pulmonary response to formaldehyde exposure in healthy nonsmokers. *J. Occup. Med.* 28: 420-24
- Schachter NE, Witek TJ JR, Tosun T, Beck GJ. 1986. A study of respiratory effects from exposure to 2 ppm formaldehyde in healthy subjects. *Arch. Env. Health* 41: 229-39
- Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ. 1993. Particulate air pollution and hospital emergency visits for asthma in Seattle. *Am. Rev. Respir. Dis.* 147: 826-831.
- Sexton K, Spengler JD, Treitman RD, Turner WA. 1984. Winter air quality in a wood-burning community: A case study in Waterbury, Vermont. *Atmos. Env.* 18: 1357-70
- Sexton K, Spengler JD, Treitman RD. 1984. Effects of residential wood combustion on indoor air quality: A case study of Waterbury, VT. *Atmos. Env.* 18: 1371-83
- Sheppard D, Eschenbacher WL, Epstein J. 1984. Lack of bronchomotor response to up to 3 ppm formaldehyde in subjects with asthma. *Env. Res.* 35: 133-39
- Simons CA, Christiansen PD, Houck JE, Prithcett LC. 1989. Woodstoves emission sampling methods comparability analysis and in-situ evaluation of new technology woodstoves. EPA-600/7/89-002, Research Triangle Park: U.S. EPA
- Skog EK, Wattersen IA. 1983. Survey completion report, residential fuel wood use in the United States: 1980-81, U.S. Department of Agriculture, Forest Service Rep.
- Standley LJ, Simoneit BRT. 1990. Preliminary correlation of organic molecular tracers in residential wood smoke with the source of the fuel. *Atmos. Env. B.* 24: 67-73
- Steiber R, Dorsey J. 1988. GC/MS analysis of stove emissions and ambient samples from a woodsmoke impacted area. *Proc. EPA/APCA Sym. Measure. Toxic Related Air Pollut.*, pp. 828-34. Air Pollut. Control Assoc. Publication VIP-10 (EPA 600/9-88-015)

- Steiber RS, McCrillis RC, Dorsey JA, Merrill RG Jr. 1992. Characterization of condensable and semivolatile organic materials from Boise woodstove samples. Presented at 85th Annual Meeting Air Waste Manage. Assoc., Kansas City
- Stern CH, Jaasma DR, Shelton JW, Satterfield G. 1992. Parametric study of fireplace particulate matter and carbon monoxide emissions. *J. Air Pollut. Control Assoc.* 42: 777-83
- Thorning DR, Howard ML, Hudson LD, Schumacher RL. 1982. Pulmonary responses to smoke inhalation: Morphological changes in rabbits exposed to pine wood smoke. *Human Pathol.* 13: 355-64
- Traynor GW, Apte MG, Carruthers AR, Dillworth JF., Grimsrud DT, Gundel LA. 1987. Indoor air pollution due to emissions from wood-burning stoves. *Env. Sci. Technol.* 21: 691-97
- Truesdale RS. 1984. Characterization of emissions from a fluidized bed wood chip home heating furnace. NTIS #PB84-179878
- Tuthill RW. 1984. Woodstoves, formaldehyde, and respiratory diseases. *Am. J. Epidemiol.* 120: 952-55
- Uba g, Pachorek D, Bernstein J, Garabrant DH, Balmes JR, et al. 1989. Prospective study of respiratory effects of formaldehyde among healthy and asthmatic medical students. *Am. J. Indust. Med.* 15: 91-101
- US EPA. 1992. Respiratory health effects of passive smoking: Lung cancer and other disorders. 600/6-90/006F
- van Houdt JJ, Daenen CMJ, Boleij JSM, Alink GM. 1986. Contribution of wood stoves and fire places to mutagenic activity of airborne particulate matter inside homes. *Mut. Res.* 171: 91-98
- Watson JG. 1979. Chemical element balance receptor-model methodology of assessing the sources of fine and total suspended particulate matter in Portland, Oregon. PhD thesis. Env. Science, Oregon Grad. Center, Portland

- Wolff GT, Countess RJ, Groblicki PJ, Ferman MA, Cadle SH, Muhlbaier JL. 1981. Visibility-reducing species in the Denver "Brown Cloud"-II. sources and temporal patterns. *Atmos. Env.* 15:2485-502
- Wong KL, Stock MF, Malek DE, Alarie Y. 1984. Evaluation of the pulmonary effect of wood smoke in guinea pigs by repeated CO₂ challenges. *Toxicol Appl. Pharmacol* 75: 69-80
- Zeedik IH. 1986. Polycyclic aromatic hydrocarbon concentrations in smoke aerosol of domestic stoves burning wood and coal. *J. Aerosol Sci.* 17: 635-38
- Zweidinger, Tejada S, Highsmith R, Westburg H, Gage L. 1988. Volatile organic hydrocarbon and aldehyde distribution for the IACP Boise, ID, Residential Study. *Proc. EPA/ Air Pollut. Control Assoc. . Sym. Measure. Toxic Related Air Pollut.*, pp. 814-20. APCA Publication VIP-10 (EPA 600/9-88-015)

Table 1. Emission Rates from Different Wood Burning Appliances

Appliance	Particulate Matter	Carbon Monoxide	Nitrogen Oxides	VOCs	Benzene	PAHs	B(a)P
Conventional Wood Stove							
<i>Laboratory Measurements</i> ^{2,3}	7-30	125-300	0.2	20-52	0.6 - 4	0.1 - 6	1x10 ⁻³
<i>Field Measurements</i> ⁴	22	113	--	--	--	--	--
Catalytic Wood Stove							
<i>Laboratory Measurements</i> ^{2,5}	1.6 - 2.3	7 - 42	0.4	5 - 13	0.06-0.5	0.10-0.13	n.d.
<i>Field Measurements</i> ⁴	10.6	51	--	--	--	--	--
Non-Catalytic Wood Stove							
<i>Laboratory Measurements</i> ⁶	0.5 - 22	7 - 140	0.7 - 40	--	--	--	--
<i>Field Measurements</i> ^{4,7}	4-12	76	--	--	--	--	--
Pellet Stove							
<i>Laboratory Measurements</i> ²	0.5 - 2.3	0.2 - 1.7	0.9	0.5 - 0.9	n.d.	0.006-0.5	1.6x10 ⁻²
<i>Field Measurements</i> ⁸	0.23-5.5	12-45	--	--	--	(ref. 7)	n.d.
Conventional Fireplace ⁹	15 - 32	80 - 120	--	--	--	--	--
Advanced Tech Fireplace ⁹	1.4 - 5.8	41 - 83	--	--	--	--	--
Masonry Heater ⁹	9 - 11	44 - 72	--	--	--	--	--

¹ n.d. = not detected; when only one value is shown, only one value was reported in reference

² OMNI (1988)

³ Controlled laboratory measurements of conventional stove burning douglas fir with 17-18% moisture content at 0.7 - 4 kg/hr.

⁴ Jaasma et al (1991); average values in Crested Butte, CO in winters of 1988-89 and 1989-90.

⁵ Controlled laboratory measurements of catalytic stove burning douglas fir with 17-18% moisture content at 0.8 - 2.3 kg/hr. EPA's field test value for 1988 NSPS Certified/Certifiable Catalytic Stoves is 6.6 grams of particulate matter per kilogram of wood.

⁶ McCrillis and Butts (1991); hydrocarbon measurements as total hydrocarbons

⁷ Barnett and Roholt (1990), values from Glens Falls, NY.

⁸ Barnett et al (1991) field measurements in Klamath Falls, OR (3 stoves) and Medford, OR (3 stoves); in-stack PAH concentrations ranged from 1-10 µg/M³.

⁹ Barnett et al (1992); field measurements in Portland, OR; five conventional fireplaces and two advanced technology fireplaces; the latter have a secondary combustion zone

Table 2. Chemical Composition of Wood Smoke

Species ¹	g/kg wood ²	Physical State ³	Reference
Carbon Monoxide	80-370	V	4,5
Methane	14-25	V	5
VOCs (C ₂ -C ₇)	7-27	V	5
<i>Aldehydes</i>	0.6-5.4	V	4,6
Formaldehyde	0.1-0.7	V	4,6
Acrolein	0.02-0.1	V	6
Propionaldehyde	0.1-0.3	V	4,6
Butryaldehyde	0.01-1.7	V	4,6
Acetaldehyde	0.03-0.6	V	4,6
Furfural	0.2-1.6	V	7,8
Substituted Furans	0.15-1.7	V	7,8
Benzene	0.6-4.0	V	5
<i>Alkyl Benzenes</i>	1-6	V	9
Toluene	0.15-1.0	V	9
Acetic Acid	1.8-2.4	V	7
Formic Acid	0.06-0.08	V	7
<i>Nitrogen Oxides</i> (NO,NO ₂)	0.2-0.9	V	4,5
Sulfur Dioxide	0.16-0.24	V	4
Methyl chloride	0.01-0.04		10
Napthalene	0.24-1.6	V	9
<i>Substituted Napthalenes</i>	0.3-2.1	V/P	9
<i>Oxygenated Monoaromatics</i>	1 - 7	V/P	9
Guaiacol (and derivatives)	0.4-1.6	V/P	11
Phenol (and derivatives)	0.2-0.8	V/P	11
Syringol (and derivatives)	0.7-2.7	V/P	11
Catechol (and derivatives)	0.2-0.8	V/P	11
Total Particle Mass	7-30	P	5
Particulate Organic Carbon	2-20	P	12
<i>Oxygenated PAHs</i>	0.15-1	V/P	9
<i>PAHs</i>			
Fluorene	4x10 ⁻⁵ - 1.7x10 ⁻²	V/P	13
Phenanthrene	2x10 ⁻⁵ - 3.4x10 ⁻²	V/P	13
Anthracene	5x10 ⁻⁵ - 2.1x10 ⁻²	V/P	13

Species ¹	g/kg wood ²	Physical State ³	Reference
Methylanthracenes	$7 \times 10^{-5} - 8 \times 10^{-3}$	V/P	13
Fluoranthene	$7 \times 10^{-4} - 4.2 \times 10^{-2}$	V/P	13
Pyrene	$8 \times 10^{-4} - 3.1 \times 10^{-2}$	V/P	13
Benzo(a)anthracene	$4 \times 10^{-4} - 2 \times 10^{-3}$	V/P	13
Chrysene	$5 \times 10^{-4} - 1 \times 10^{-2}$	V/P	13
Benzofluoranthenes	$6 \times 10^{-4} - 5 \times 10^{-3}$	V/P	13
Benzo(e)pyrene	$2 \times 10^{-4} - 4 \times 10^{-3}$	V/P	13
Benzo(a)pyrene	$3 \times 10^{-4} - 5 \times 10^{-3}$	V/P	13
Perylene	$5 \times 10^{-5} - 3 \times 10^{-3}$	V/P	13
Ideno(1,2,3-cd)pyrene	$2 \times 10^{-4} - 1.3 \times 10^{-2}$	V/P	13
Benz(ghi)perylene	$3 \times 10^{-5} - 1.1 \times 10^{-2}$	V/P	13
Coronene	$8 \times 10^{-4} - 3 \times 10^{-3}$	V/P	13
Dibenzo(a,h)pyrene	$3 \times 10^{-4} - 1 \times 10^{-3}$	V/P	13
Retene	$7 \times 10^{-3} - 3 \times 10^{-2}$	V/P	14
Dibenz(a,h)anthracene	$2 \times 10^{-5} - 2 \times 10^{-3}$	V/P	13
<i>Trace Elements</i>			
Na	$3 \times 10^{-3} - 1.8 \times 10^{-2}$	P	15
Mg	$2 \times 10^{-4} - 3 \times 10^{-3}$	P	15
Al	$1 \times 10^{-4} - 2.4 \times 10^{-2}$	P	15
Si	$3 \times 10^{-4} - 3.1 \times 10^{-2}$	P	15
S	$1 \times 10^{-3} - 2.9 \times 10^{-2}$	P	15
Cl	$7 \times 10^{-4} - 2.1 \times 10^{-1}$	P	15
K	$3 \times 10^{-3} - 8.6 \times 10^{-2}$	P	15
Ca	$9 \times 10^{-4} - 1.8 \times 10^{-2}$	P	15
Ti	$4 \times 10^{-5} - 3 \times 10^{-3}$	P	15
V	$2 \times 10^{-5} - 4 \times 10^{-3}$	P	15
Cr	$2 \times 10^{-5} - 3 \times 10^{-3}$	P	15
Mn	$7 \times 10^{-5} - 4 \times 10^{-3}$	P	15
Fe	$3 \times 10^{-4} - 5 \times 10^{-3}$	P	15
Ni	$1 \times 10^{-6} - 1 \times 10^{-3}$	P	15
Cu	$2 \times 10^{-4} - 9 \times 10^{-4}$	P	15
Zn	$7 \times 10^{-4} - 8 \times 10^{-3}$	P	15
Br	$7 \times 10^{-5} - 9 \times 10^{-4}$	P	15
Pb	$1 \times 10^{-4} - 3 \times 10^{-3}$	P	15

Species ¹	g/kg wood ²	Physical State ³	Reference
Particulate Elemental Carbon	0.3 - 5	P	16
<i>Normal alkanes (C₂₄-C₃₀)</i>	1x10 ⁻³ - 6x10 ⁻³	P	17
<i>Cyclic di-and triterpenoids</i>			
Dehydroabietic acid	0.01 - 0.05	P	18
Isopimaric acid	0.02 - 0.10	P	18
Lupenone	2x10 ⁻³ - 8x10 ⁻³	P	18
Friedelin	4x10 ⁻⁶ - 2x10 ⁻⁵	P	18
<i>Chlorinated dioxins</i>	1x10 ⁻⁵ - 4x10 ⁻⁵	P	19
<i>Particulate Acidity</i>	7x10 ⁻³ - 7x10 ⁻²	P	20

¹ Some species are grouped into general classes as indicated by italics

² To estimate the weight percentage in the exhaust, divide the g/kg value by 80. This assumes that there are 7.3 kg combustion air per kg of wood. Major species not listed here include carbon dioxide and water vapor (about 12 and 7 weight percent respectively under the assumed conditions).

³ At ambient conditions; V = vapor, P = particulate, and V/P = vapor and/or particulate (i.e., semi-volatile).

⁴ DeAngelis (1980)

⁵ OMNI (1988)

⁶ Lipari (1984), values for fireplaces

⁷ Edye et al (1991), smoldering conditions; other substituted furans include 2-furanmethanol, 2 acetylfuran, 5-methyl-2-furaldehyde, and benzofuran

⁸ Value estimated for pine from Edye et al (1991) from reported yield relative to guaiacol, from guaiacol values of Hawthorne (1989) and assuming particulate organic carbon is 50% of total particle mass

⁹ Steiber et al (1992), values computed assuming a range of 3-20 g of total extractable, speciated mass per kg wood

¹⁰ Khalil (1983)

¹¹ Hawthorne (1989), values for syringol for hardwood fuel; see also Hawthorne (1988)

¹² Core (1989), DeAngelis (1980), Kalman and Larson (1987)

¹³ From one or more of the following studies: Cooke (1981), Truesdale (1984), Alfheim et al (1984), Zeedijk (1986), Core (1989), Kalman and Larson (1987); assuming a range of 7 to 30 grams of particulate mass per kg wood when values were reported in grams per gram of particulate mass. Similar assumptions apply to references 14, 15 and references 17-19

¹⁴ Core (1989), Kalman and Larson (1987)

¹⁵ Watson (1979), Core (1989), Kalman and Larson (1987)

¹⁶ Rau (1989), Core (1989)

¹⁷ Core (1989)

¹⁸ Standley and Simoneit (1990); Dehydroabietic acid values for pine smoke, lupenone and isopimaric acid values for alder smoke, and friedelin values for oak soot.

¹⁹ Nestruck and Lamparski (1982), from particulate condensed on flue pipes; includes TCDDs, HCDDs, H₇CDDs and OCDDs

²⁰ Burnet et al (1986); one gram of acid = one equivalent of acid needed to reach a pH of 5.6 in extract solution

Table 3. Summary of Wood Smoke Reactivity Studies

Investigator	Study Type	Species	Major Findings
Kamens et al (1984)	Laboratory Chamber Study. Oak smoke diluted to 1300-8000 $\mu\text{g}/\text{M}^3$ and then mixed with NO_2 and O_3 . Most tests in dark, a few in artificial sunlight. Chamber run in static mode.	Fine particles, NO, NO_2 and ozone	NO_2 reacts with O_3 in both light and dark conditions, changing the mutagenic (and therefore chemical) properties of the vapor and particle phases.
Kleindienst et al (1986)	Laboratory Chamber Study. Oak smoke diluted to $\approx 500 \mu\text{g}/\text{M}^3$. NO_x intentionally added to some runs to supplement flue gas NO_x . All tests in artificial sunlight, simulating mid-day, winter time ultraviolet intensities. Chamber run in stirred tank reactor mode with small flow in and out.	Fine particles, NO, NO_2 , ozone, alkenes (ethylene, propylene, 1-butene), aromatics (benzene, toluene), heterocyclics (furan, 2-methylfuran, 2-furaldehyde), and aldehydes (formaldehyde, acetaldehyde).	Without added NO_x , the HC/NO_x molar ratio (>100) limits photochemical reactivity. Adding NO_x ($\text{HC}/\text{NO}_x \sim 25$) enhances photochemistry, thereby generating ozone, increasing NO_2 relative to NO, producing larger particles, rapidly destroying alkenes and furans, and increasing levels of aldehydes. Reactivity in the absence of sunlight was not reported.
Steiber and Dorsey (1988)	Field measurements in winter in Boise, ID	Oxygenated monoaromatics in both vapor and particulate phases.	The mass distribution of methoxylated benzenes and phenols as a function of the number of oxygen atoms per molecule as measured in both indoor and outdoor air correlates well with the corresponding distribution in undiluted wood stove exhaust. This indicates that these compounds do not undergo significant chemical degradation at night in the environment.

Investigator	Study Type	Species	Major Findings
Miller et al (1990); Hawthorne et al (1989, 1989a) Hawthorne et al (1992)	Field measurements in winter in Grand Forks, ND, Salt Lake City, Utah and Minneapolis, MN	Methoxylated phenols in both vapor and particulate phases.	Methoxylated phenols in the atmosphere were the same compounds as those identified in wood stove exhaust. The relative proportions in the air vs. in the stack were consistent with only minor atmospheric degradation of these compounds.
Standley and Simoneit (1990)	Field study in western Oregon; 24-hour samples at three locations	Dehydroabietic acid	Relative proportions of dehydroabietic acid in particles found in ambient air was consistent with the proportions found in the exhaust of wood stoves burning pine, implying only minor atmospheric degradation of this compound.
Lindell (1991)	Field study in Seattle, WA; 35 night time samples in winter	Particle acidity	Average particle acidity levels in winter (12 nmoles/M ³) were an order of magnitude lower than the levels expected in particles freshly emitted from wood stoves (Burnet, 1986). This implies that the particle acidity of wood smoke decreases after release to the environment at night.

Table 4. Summary of Measured Wood Burning Impacts on Airborne Particle Levels

Investigator ^a	Location	Measurement ^b	Level ($\mu\text{g}/\text{m}^3$) Mean	Range	Wood Smoke (wt %)	Method ^c	Comments
Cooper (1981)	Portland, OR	PM _{2.5} total carbon	68 31.3	—	36 51	14C	Single sample at residential location in winter
Wolff et al (1981)	Denver, CO	PM _{2.5} total carbon	39.5 27	—	12 33	K/Fe 14C	Five samples during winter
Carlson (1982)	Missoula, MT	PM _{3.5}	—	7-43	68	CMB	Average of winter samples
Imhoff (1982)	Petersville, AL	PM _{2.5}	45	13-86	85	CMB	Seven residential samples in winter
Core et al (1984)	Seattle, Spokane, Tacoma, Yakima, Portland, Boise	PM _{2.5}	57	34-122	44-92	CMB	Sixty-one 24-hr samples in autumn and winter from eight sites in WA, one site in ID and one site in OR
Ramdahl et al (1985)	Medford, OR Portland, OR Elverum, Norway	PM _{2.5} PM _{2.5} total carbon (<3 μm)	17.5 3.0 20	8.8-30.2 1.5-3.9 5-50	42-66 10-19 65	14C	Annual average values for 3 sites Annual average values for 4 sites Ten 24-hr winter samples; avg PM ₁₀ = 51 $\mu\text{g}/\text{M}^3$ (range 31-101)
Naylor (1985)	Las Vegas, NV	total carbon	36	25-46	47	14C	Four 12-hr winter samples (day and night)
Lewis et al (1986)	Denver, CO	PM _{2.5}	19	?-47	8	MLR	Seventeen 12-hr daytime samples in winter
		PM _{2.5}	23	?-41	17		Nineteen 12-hr night-time samples in winter
Klouta et al (1987)	Raleigh, NC	total carbon elemental carbon	— 3.2	23-80 —	95 68	14C	Four 12-hr daytime samples in winter One 12-hr daytime sample in winter
	Albuquerque, NM	total carbon elemental carbon	— 4.6	11-71 —	75 41	14C	Six 12-hr samples (day & night) at residential site in winter Four 12-hr samples (day & night) at residential site in winter

Investigator ^a	Location	Measurement ^b	Level ($\mu\text{g}/\text{m}^3$)		Wood Smoke (wt %)	Method ^c	Comments
			Mean	Range			
Lewis et al (1988)	Albuquerque, NM	total carbon	—	—	67	MLR	Six 12-hr samples (day & night) in winter
		EOM	18.9	—	68 78	14C MLR	Forty-four 12-hr samples (day & night) in winter
Chow et al (1988)	Sparks, NV	PM ₁₀	41	?-154	30 ^d	CMB	Fifty seven 24-hr samples every 6th day for one year at a residential site
	Reno, NV		76		44 ^d		Subset of above samples from Oct-Dec period (n=15)
Benedict & Naylor (1988)	Las Vegas, NV	PM _{2.5}	30	?-99	3 ^d		Fifty six 24-hr samples every 6th day for one year at an urban site
			46		9 ^d		Subset of above samples from Oct-Dec period (n=15)
			12.5		27	CMB	One 24-hr sample during winter;
			8.7		12.9	CMB	Nine month average of every sixth day 24-hr samples (March-December)
Magliano (1988)	Bakersfield, CA	PM ₁₀	13.8	—	62.8		
		PM _{2.5}	7.1	—	16.8		
Dresser & Baird (1988)	Telluride, CO	PM ₁₀	—	—	33	CMB	Four 24-hr avg spring samples
		PM ₁₀	205	—	58		Two 24-hr holiday winter samples
Larson et al (1991)	Seattle, WA	PM ₁₀	39	9-123	6	CMB	Seven 12-hr daytime samples at industrial site in winter of 1987-88.
			30	8-61	11		The corresponding seven 12-hr nighttime samples at above site.
Klouta et al (1991)	Boise, ID	EOM	45	12-104	54		Ten 12-hr daytime samples at residential site in winter of 1987-88.
			75	5-144	82		The corresponding ten 12-hr nighttime samples at above site.
			116	75-139	82		The sixteen highest 12-hr night-time samples at the same residential site in the winter of 1988-89.
			—	—	72-89	14C	Reported range of values (avg not reported) for nine 12-hr daytime samples at a residential site in winter.
		EOM	—	—	52-83		Range of values (avg not reported) for nine 12-hr night-time samples at residential site in winter.

Investigator ^a	Location	Measurement ^b	Level ($\mu\text{g}/\text{m}^3$) Mean	Range	Wood Smoke (wt %)	Method ^c	Comments
Lewis et al (1991)	Boise, ID	EOM	22	—	67 ^d	MLR	Forty 12-hr samples (day & night)
Larson et al (1992)	Seattle, WA	PM _{2.5}	14.8	6.0-32.9	71	CMB	Forty eight one-week average composite samples (Jan-Nov) at a residential site. The composite consisted of sampling for 15 minutes every two hours for the entire study period. Wood burning was the dominant source all seasons of the year, ranging from 60% in summer to 90% in winter.
Hawthorne et al (1992)	Minneapolis, MN	PM ₁₀			42-68	¹⁴ C	Four twelve-hour average samples starting either at 7AM or noon.
	Salt Lake City	PM ₁₀			15-47	MLR	Nine twelve hour average samples starting either at 7AM or noon. MLR based upon guaiacol tracers.
					16-74	¹⁴ C	
					4-55	MLR	

^a Other investigators have measured elevated levels of particulate matter in wood burning communities, but did not use one of the methods cited above to quantify the fraction attributable to wood burning. Methods not listed above include emission inventory/atmospheric dispersion modeling (Romero et al, 1978; Butcher & Sorenson, 1979; Kowalczyk & Greene, 1982; Murphy et al, 1982; Hornig et al, 1982; Imhoff, 1982; Sexton et al, 1984; Ramdahl et al, 1984; Naylor, 1985), gaseous methyl chloride tracer measurement (Khalil, 1983), time series of particle light scattering coefficient (Kowalczyk & Greene, 1982; Larson et al, 1991) and thermography (Kowalczyk & Greene, 1982).

^b PM_x = mass concentration of particles $\leq x$ μm in aerodynamic diameter; EOM = mass concentration of extractable organic matter from particles with aerodynamic diameters ≤ 2.5 μm ; total carbon = total organic and elemental carbon mass concentration in particles ≤ 2.5 μm in aerodynamic diameter unless otherwise noted.

^c ¹⁴C = isotopic carbon measurement to determine biogenic carbon concentration, i.e., contemporary carbon from biogenic material ~ 40 years or less old; CMB = chemical mass balance regression model; K/Fe = tracer enrichment method based upon the mass ratio of potassium to iron; MLR = multiple linear regression of individual tracer elements (e.g. potassium for wood and lead for motor vehicles) against mass concentration of relevant measurement listed above.

^d Estimated from reported average concentration of wood smoke divided by average concentration of total mass

Table 5. Impacts of Wood Burning Appliances on Indoor Air Quality¹

Species	Source ²	Indoor level (µg) with source	Indoor level (µg) without source	Average Indoor:Outdoor ratio with	Average Indoor:Outdoor ratio without	Estimated indoor source strength (mg/hr) ³	Reference
PM _{2.5}	a	7-40	8-32	<1	<1	—	4.5
	a,i	50-80	10-30	1.3	0.4	—	4.5
	a	3-27	—	0.3	—	—	6
	a,i	18	—	0.8	—	—	6
	a	28-38	—	1.1	—	2.3-3.6	7
PM _{3.5}	n	44-91	—	2.1	—	5.5-11.1	7
	—	13-15	8-32	>1	>1	—	8
Total suspended particles	—	25 (19)	24 (22)	1.7	1.4	—	9
	a	24-71	—	3.1	—	2.5-8.7	10
	n	28-1500	—	33.5	—	16-230	10
	b	19-24	—	1.3	—	1.1-1.6	10
Total suspended particles	a	52-65	—	1.2	—	4.1-7.5	7
	n	119-166	—	2.6	—	12-21	7
Carbon Monoxide	a	1100-1960	—	2.4	—	55-182	7
	n	2450-4410	—	6.8	—	210-530	7
	a	490-3430	—	2.2	—	12-170	10
	n	2200-17,100	—	24.5	—	270-2200	10

Species	Source ²	Indoor level (µg) with source	Indoor level (µg) without source	Average Indoor:Outdoor ratio with	Average Indoor:Outdoor ratio without	Estimated indoor source strength (mg/hr) ³	Reference
	a	1100-2700	—	1.0	—	—	6
Nitric Oxide	a	27-47	—	1.6	—	0.4-3.2	7
	n	26-67	—	4.4	—	2.0-9.4	7
Nitrogen Dioxide	a	12-25	—	0.5	—	<0-2.4	7
	n	18-26	—	0.7	—	2.1-3.6	7
Formaldehyde	a	12-51	18-25	8	5	—	11
	a,i	15-33	17-34	3	4	—	4,11
Benzo(a)pyrene	a	3x1 ⁻⁴ -3.5x10 ⁻³	—	1.6	—	2x10 ⁻⁵ -7.6x10 ⁻⁴	10
	n	2x10 ⁻³ -4.9x10 ⁻¹	—	84	—	2x10 ⁻³ -5.7x10 ⁻²	10
Extractable Organic Matter -particulate phase	a	1.4-2.3	—	0.6	—	—	6
	a,i	2.0	—	0.6	—	—	6
vapor phase (semi-volatile)	a	12-20	16-20	3.0	3.5	—	12
	a	14.4-14.5	—	2.1	—	—	6
	a,i	29.1	—	2.6	—	—	6
	a	268-308	296-308	3.0	3.5	—	12

¹ Studies listed here include simultaneous measurements of indoor and outdoor levels. Other studies of note not listed include Benton, et al (1982), Neulicht & Core (1982), Baechler (1985) and van Houtd et al (1986). These latter studies document the impacts on indoor air quality of wood burning devices by comparison with "no-burn" periods, but do not specifically control for outdoor pollution variability.

² a = airtight wood stove; i = improperly operated wood stove; n = non-airtight wood stove; f = fireplace; b = background test (no stove present).

³ Reported estimates via mass balance from indoor and outdoor levels as well as air exchange rates.

- 4 Highsmith et al (1988); values taken from Figure 3 are averages of weekday and weekend sampling periods; indoor levels from one home with excessive impacts from a humidifier are excluded.
- 5 Ten pairs of homes in Boise, ID; each pair consists of one home with one and one without a wood burning device as well as a simultaneous outdoor sample at the home without the wood burning device.
- 6 Highsmith et al (1988a); three residences in Raleigh, NC.
- 7 Knight et al (1985); data from test house in Chattanooga, TN (0.3-0.6 air changes per hour)
- 8 Colome & Spengler (1982); range of annual average values for two homes with stoves and three without stoves in Portage, WI.
- 9 Sexton et al (1984); nineteen wood burning homes and five non-wood burning homes in Waterbury, VT; standard deviation of mean levels shown in parentheses.
- 10 Traynor et al (1987); data from test house in Truckee, CA (0.3-0.9 air exchanges per hour) using one of three airtight models and one non-airtight model. Indoor levels averaged over burn time (5-16 hours).
- 11 Zweidinger et al (1988); average measurements also reported for selected VOCs and carbonyls; indoor formaldehyde levels higher on weekends than weekdays.
- 12 Merrill et al (1988); samples collected in Boise, ID; indoor values reported assuming 6 percent of total EOM is particulate and 94 percent is vapor phase; indoor/outdoor ratio estimated from ratio of averages.

Table 6. Summary of studies of respiratory effects of exposure to wood smoke.

Referene	Age of Subjects	Number of Subjects	Endpoints Measured	Results
Honicky	1-7 yr	34 w/stoves 34 without	Symptoms	More symptoms in children with stoves p <0.001.
Tuthill	5-11*yr	258 w/stoves 141 without	Symptoms	Risk ratio = 1.1, showing no sign. effect
Browning	1yr and older	455,high smoke** 368 low smoke	Symptoms Disease prevalence	No significant effects. Trend in children aged 1- 5.
Koenig (In Press)	8-11 yr	296 healthy*** 30 asthmatic	Spirometry	Significant assoc. between fine particles and lung function in asthmatics in an area heavily impacted by wood smoke, p =0.05.
Butterfield	1-5 1/2 yr	59	Symptoms	Significant corr between woodstove use and wheeze and cough frequency p = 0.01.
Morris	< 24 mo	58 pairs	Respiratory Disease	Woodstove significant risk factor for lower resp infection.
Heumann	8-11	410***	Spirometry	Significant decrease in PFTs with elevated wood smoke
Johnson	8-11	495	Spirometry	Significant relation of function decrease with increasing TSP
Lipsett	X age,46 yr	182	Symptoms	Significant assoc
Schwartz	2955 cases 3810 controls	Emergency room visits for asthma or gastroenteritis.Significant association (p < 0.005) with particle concentrations at a site heavily impacted by wood smoke year round.		

* Described as kindergarten through grade 6.

** Geographical areas chosen as having high or low wood smoke pollution.

*** Grades 3 through 6.

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1. REPORT NO. EPA-453/R-93-036	2.	3. RECIPIENT'S ACCESSION NO.
4. TITLE AND SUBTITLE A Summary of the Emissions Characterization and Noncancer Respiratory Effects of Wood Smoke	5. REPORT DATE December 1993 (Date of Approval)	6. PERFORMING ORGANIZATION CODE
	8. PERFORMING ORGANIZATION REPORT NO.	
7. AUTHOR(S) Timothy V. Larson and Jane Q. Koenig University of Washington, Seattle, WA	10. PROGRAM ELEMENT NO.	
9. PERFORMING ORGANIZATION NAME AND ADDRESS USEPA, Air Risk Information Support Center Office of Air Quality Planning and Standards Emission Standards Division Research Triangle Park, N.C. 27711	11. CONTRACT/GRANT NO. 1D3253NAEX	
	13. TYPE OF REPORT AND PERIOD COVERED Final	
12. SPONSORING AGENCY NAME AND ADDRESS	14. SPONSORING AGENCY CODE	

15. SUPPLEMENTARY NOTES

16. ABSTRACT

This report summarizes the available literature on constituents and fate of wood smoke and the health effects of wood smoke. The emission characterization of wood smoke focuses on the chemical composition, emission rates from different wood burning devices, and impacts from wood burning on airborne particle levels. The epidemiological data focus on human respiratory responses to inhaled wood smoke such as increased respiratory symptoms, lower respiratory infection and decreased pulmonary function in children, especially those with asthma. The report concludes that the data demonstrate a causal relationship between elevated wood smoke concentrations and adverse respiratory effects in children.

17. KEY WORDS AND DOCUMENT ANALYSIS		
a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Wood Smoke Respiratory Effects Emissions Characterization Exposure Assessment Lung Function		
18. DISTRIBUTION STATEMENT	19. SECURITY CLASS (This Report) Unclassified	21. NO. OF PAGES 47
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