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WOOD SMOKE

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Since the early 1970s, use of wood as a heating fuel has increased substantially. In 1981 approximately 7 percent of homes in the United States used wood as the major or supplementary source of heat, compared with only 1 percent in 1970 (Annandale, Duxbury, and Newman 1983). Increased residential wood use has significantly degraded ambient air quality in several areas of the country, most notably in towns and cities prone to wintertime inversions, such as Vail and Denver, Colorado (Lewis and Einfeld 1985), and Portland, Oregon (Cooper 1980). Research on wood combustion emissions, prompted by the resurgence in wood use, has provided inventories of the pollutants emitted by wood combustion, but data needed for understanding the health consequences of wood burning are still lacking. Few investigations of the effects of wood combustion on indoor air quality have been performed, and data concerning the health effects of domestic exposure to wood smoke remain extremely limited.

Wood is composed primarily of carbon, oxygen, and hydrogen. Under ideal conditions with complete combustion, water and carbon dioxide (CO_2) are the primary end products. Nitrogen oxides and sulfur oxides are also formed; nitrogen oxides are primarily from the combination of atmospheric nitrogen with oxygen during high temperature combustion, and sulfur oxides are from oxidation of sulfur in the wood. Incomplete combustion results in emissions of other pollutants such as particulates, organic compounds including polycyclic organic materials, and carbon monoxide (CO) (Quraishi 1985). Emission rates for these pollutants are highly variable and depend on such factors as burn rate, amount of air available for combustion, size of fuel charge, type and size of wood, and the moisture content of the wood (Quraishi 1985).

DeAngelis and colleagues (1980) sampled flue gases from two airtight cast iron stoves (one baffled and one nonbaffled) and a zero-clearance fireplace and per-

Table 9.1 Studies of the Impact of Residential Wood Burning (WB) on Indoor Air Quality

Study	Findings	Conclusions
One home with wood stove, 2 with fireplaces (1 WB day in each fireplace), Boston, Mass. (Moschandreas, Zabransky, and Rector 1980)	Indoor TSP[a] and RSP[b] four times higher on WB days; BaP[c] increased fivefold in home with wood stove, and tenfold on 1 WB day in home with fireplace; CO peaks during loading and refiring.	Wood burning a major source of particulate matter emissions in residential environment
19 WB homes, 5 non-WB homes; monitored for 2 weeks, Waterbury, Vt. (Sexton, Spengler, and Treitman 1984)	RSP levels: all NWB homes, 24 μg/m; NWB homes with unexplainably high levels removed, 18 μg/m; WB homes without kerosene heater, 24 μg/ml; WB homes with kerosene heater, 34 μg/m.	Airtight wood stoves can be installed and operated with negligible release of particles into indoor air; particle concentrations in general vary widely among homes.
Experimental house, with 3 airtight and 1 non-airtight wood stove; monitored at least 5 h per test, Truckee, Calif. (Traynor et al. 1987)	Airtight stoves caused short-term CO and particulate peaks during door opening; pollutants emitted by non-airtight stove dependent on operation of stove; submicron particles, 210–1,900 μg/m; CO, 1.8–14 ppm; BaP, 5.4–150 μg/m.	Airtight stoves not an important source of pollutants; non-airtight stoves can be a moderately heavy source depending on their mode of operation.
20 homes with wood stoves monitored twice during stove operation and twice when not operating, Madison, Wisc. (Kaaraka, Kanarek, and Lawrence 1987)	CO levels slightly higher during WB (2.2 versus 1.8 ppm); on average, RSP levels were unchanged, but indoor and outdoor RSP increased in rural homes during WB; only 2 BaP measurements above limits of detection (0.32 and 0.12 ng/m).	Use of airtight stoves with adequate drafts not a source of pollution; poor stove quality or loading habits can lead to high short-term levels of contamination.
Experimental house with airtight wood stove and fireplace, Norway (Alfheim and Ramdahl 1984)	WB increased PAH[d] level from background of 1–16 ng/m with stove, 206 ng/m with fireplace; stove did not increase mutagenic activity of air; fireplace increased it substantially.	Wood stove had little impact on PAH levels or mutagenic activity; fireplace had considerable impact but still less than tobacco smoke.
12 homes with wood stoves, 12 with fireplaces, sampled during 1 WB and 1 non-WB week, Netherlands (Van Houdt et al. 1986)	WB caused increase in mutagenic activity of indoor air in 8 out of 12 homes with stoves, 12 out of 12 with fireplaces.	Fireplaces consistently cause increased mutagenicity, wood stoves less frequently; reasons for variability are unclear.

[a]TSP, total suspended particles.
[b]RSP, respirable suspended particles.
[c]BaP, benzo[α]pyrene
[d]PAH, polycyclic aromatic hydrocarbon.

formed a detailed characterization of the emissions. They measured more than one hundred chemical compounds. In addition to the four criteria pollutants (particulate matter, CO, nitrogen oxides, and sulfur oxides) they identified seventeen priority pollutants of the U.S. Environmental Protection Agency, fourteen carcinogenic compounds, six cilia-toxic and mucus-coagulating agents, and four co-carcinogenic agents. Other respiratory irritants, including aldehydes, phenols, and furans, were also detected (Cooper 1980). Emissions of CO and polycyclic organic compounds were higher from the stoves, and emissions of nitrogen oxides were higher from the fireplace. Ramdahl and colleagues (1982) reported that emissions of CO, hydrocarbons, aldehydes, and volatile organic compounds were higher under starved air conditions than under normal conditions.

EXPOSURE TO WOOD SMOKE

The impact on indoor air quality of residential wood combustion in general, and specific combustion appliances in particular, has not yet been well described (Table 9.1). Indoor air concentrations of wood combustion emissions could be increased directly through leakage of pollutants from appliances or indirectly by entrainment of polluted outdoor air into the indoor environment. The newer airtight stoves operate under negative pressure and would not be expected to leak combustion by-products into the home under normal operating conditions. However, under non-airtight conditions and during starting, stoking, and reloading, pollutants may be emitted indoors.

Moschandreas and co-workers (1980) studied two residences with fireplaces and one with a wood stove over a two-week period. Twenty-four-hour levels of respirable suspended particles were higher inside on wood-burning days than outside on non-wood-burning days. During one wood-burning day, respirable suspended particulate levels ranged from 14.3 to 72.5 $\mu g/m^3$ in the home with the wood stove and averaged 159.9 and 67.6 $\mu g/m^3$ in the two homes with a fireplace. Benzo[a]pyrene levels were five times higher on wood-burning days in the home with the wood stove, averaging 4.7 ng/m^3. On the wood-burning day in one of the homes with a fireplace, benzo[a]pyrene levels reached 11.4 ng/m^3, compared with an outside concentration of 0.6 ng/m^3. CO levels were reported only for the home with the wood stove; although generally below 1 ppm, peaks exceeding 5 ppm were noted during stoking and reloading. The authors concluded that wood burning in a stove or a fireplace could be a significant source of indoor air pollution.

In a more extensive survey, Sexton and co-workers (1984) measured respirable suspended particles inside and outside twenty-four homes, nineteen with wood stoves and five without. When one non-wood-burning home that had exceptionally high levels from an unknown source was removed from the analysis, average concentrations of respirable suspended particles were 18 $\mu g/m^3$ in homes without wood-burning stoves, 24 $\mu g/m^3$ in homes with wood-burning stoves but without kerosene heaters, and 34 $\mu g/m^3$ in homes with both stoves and kerosene heaters. Although the airtightness of the stoves was not discussed explicitly in the text, the

authors concluded that their "findings suggest that airtight stoves can be installed, operated, and maintained in such a way that direct release of particles to the indoor environment is negligible."

Investigators in Wisconsin measured nitrogen dioxide (NO_2), CO, respirable suspended particles, and benzo[a]pyrene on four separate occasions in twenty homes with wood stoves, twice during the winter when the stove was in use and twice during the summer (Kaaraka, Kanarek, and Lawrence 1987). Most of the stoves were airtight. Indoor NO_2 levels were higher during wood burning (13.3 versus 9.8 $\mu g/m^3$), but this increase in levels was attributed to higher outdoor concentrations rather than direct emissions. CO levels were also slightly elevated during wood burning (2.2 versus 1.8 ppm), but again, the increase was not completely attributable to wood burning. One site had a peak of 25.3 ppm which was traced to an incident during which loading the stove had taken an unusually long time. Indoor levels of respirable suspended particles were not significantly different over the whole set of homes during the winter and summer periods although rural homes had increased indoor and outdoor concentrations during wood burning. The investigators could not determine whether the increased indoor concentrations were due to direct emissions or infiltration from outside. Only two sites had detectable benzo[a]pyrene concentrations, 0.32 and 0.12 ng/m^3, respectively. Both of these concentrations were detected during non-burn periods.

Traynor and colleagues (1987) measured the effect on indoor air quality of three airtight and one non-airtight wood-burning stoves under experimental conditions. With use of the airtight stoves, the average concentrations of CO varied from 0.4 to 2.8 ppm and of submicron particles from 11 to 36 $\mu g/m^3$. In contrast, average emissions from the non-airtight stove ranged from 1.8 to 14 ppm for CO and 210 to 1,900 $\mu g/m^3$ for submicron particles, with a peak of 10,000 $\mu g/m^3$. Indoor concentrations of five different polycyclic aromatic hydrocarbons were higher when the non-airtight stove was used. Differences in operating conditions caused much greater variability in emissions from the non-airtight wood stove although all the stoves demonstrated higher emissions during starting and reloading.

These studies suggest that airtight wood stoves contribute substantially to ambient air pollution but little to indoor concentrations. Airtight wood stoves are more thermally efficient than other wood-burning appliances. Their slower burn rate results in less complete combustion, causing greater release of CO, particulates, and polycyclic organic materials into the ambient air. However, since airtight stoves operate under negative pressure, pollutant emissions from airtight stoves into indoor air are substantially less than from other wood-burning appliances. Although combustion is more complete in fireplaces, fireplace emissions into indoor air have not been characterized adequately. Non-airtight wood stoves are probably intermediate in completeness of combustion and have the potential to cause significant degradation of indoor air quality although the magnitude of their impact can be significantly affected by the way in which they are operated.

HEALTH EFFECTS OF WOOD SMOKE

Wood smoke is an extremely complex mixture, both in its physical and chemical properties and in its toxicologic properties. The toxicology of certain individual components of wood smoke, such as benzo[a]pyrene, other polycyclic organic compounds, and nitrogen oxides, has been studied extensively. Little research, however, has been directed toward the toxicology of wood smoke as a complex mixture. *In vitro* experiments demonstrate that emissions from a wood stove induce sister chromatid exchange (Hytonen, Alfheim, and Sorsa 1983) and are mutagenic, as assessed by the Ames *Salmonella* assay (Alfheim and Ramdahl 1984; Van Houdt et al. 1986). Alfheim and Ramdahl (1984) found that mutagenic activity in indoor air increased slightly during heating with an airtight wood stove but increased substantially when wood was burning in an open fireplace. Van Houdt and co-workers (1986) found some increase in the mutagenic activity in indoor air in eight of twelve homes with wood stoves and substantial increases in twelve of twelve homes with fireplaces.

ANIMAL STUDIES

Animal studies have addressed the respiratory effects of wood smoke. Using a rabbit model, Fick and colleagues (1984) studied the effects of wood smoke on pulmonary macrophages. Following acute exposure to Douglas fir wood smoke, respiratory tract cells were recovered by bronchoalveolar lavage, and the functional properties of the alveolar macrophages were studied. Significantly more cells were recovered on lavage of exposed rabbits, compared with controls, and defects in macrophage phagocytic activity, bacterial uptake, and surface adherence were noted. Cell differential counts and macrophage viability and bactericidal processing were not affected. Wong and co-workers (1984) evaluated the response of guinea pigs to Douglas fir smoke with repeated CO_2 challenges. Exposure resulted in a decreased ventilatory response to CO_2 as measured by changes in tidal volume and respiratory frequency, with complete recovery within three days.

EPIDEMIOLOGIC STUDIES

The epidemiologic evidence on the health effects of wood smoke is also limited (Table 9.2). Most of the available information is derived from investigations in developing countries, where intense smoke exposure results from the use of cooking fires in poorly ventilated dwellings. Smith (1987) has reviewed air pollution and biomass fuels comprehensively from a global perspective. Domestic exposure to smoke from biomass fuels, which include dung and straw in addition to wood, has been implicated as a cause of chronic obstructive lung disease, particularly among women who are responsible for cooking. Master (1974) evaluated a random sample of ninety-four New Guinea natives for the presence of respiratory disease. He reported that the prevalence of chronic lung disease, as measured by the presence of chronic productive cough, dyspnea, and/or abnormal pulmonary findings on physical examination, increased with age and reached 78 percent in

Table 9.2 Studies of the Relationship of Domestic Smoke Exposure to Respiratory Illness

Study	Findings	Conclusions
Adults		
Prevalence survey of chronic respiratory disease, evaluated by questionnaire and spirometry, 1,284 adults, Papua, New Guinea (Anderson 1979)	Increased chronic respiratory symptoms and decreased pulmonary function with age; 20% of males, 10% of females over 45 years had FEV/FVC < 60%; smoking not associated with pulmonary function.	Exposure to smoke not inquired about; raised as a possible etiologic factor based on its widespread prevalence and exclusion of other factors.
Prevalence survey of chronic respiratory disease, evaluated by questionnaire with follow-up spirometry and evaluation on subgroup, 2,826 adults; Nepal (Pandey 1984a, 1984b)	Prevalence of chronic bronchitis was 17.6% among men, 18.9% among women; prevalence increased with increasing hours of exposure to domestic smoke in both sexes regardless of smoking status.	Exposure assessed by questionnaire on average number of hours per day spent near the fireplace; socioeconomic status was homogeneous.
Prevalence survey of chronic respiratory disease, evaluated by questionnaire and spirometry, 446 adults, India (Malik 1985)	Overall prevalence of chronic bronchitis 21.7% in men, 19.0% in women; prevalence was 5.8% in nonsmoking men, 5.9% in nonsmoking women.	Exposure to smoke not inquired about; raised as a possible etiologic factor due to large number of hours spent in crowded poorly ventilated dwellings.
Prevalence survey of chronic respiratory disease, evaluated by questionnaire and peak flow meters, 2,180 adults, India (Malik 1985)	Prevalence of chronic bronchitis varied with type of cooking fuel: 5% with cow dung and firewood; 2.6% coal and kerosene oil; 1.3% kerosene oil; 1.6% low-pressure gas.	Exposure assessed by type of cooking fuel; smoking and socioeconomic status not addressed.
Children		
Prevalence survey of respiratory symptoms among 807 children from the Highlands, 843 from a coastal area; supplemented by a 30-week prospective study of 112 children, Papua, New Guinea (Anderson 1978)	Prevalence of most respiratory symptoms similar between the two groups; prospective study showed no differences between 87 children exposed to domestic smoke at night and 25 children who lived in smokefree houses.	Degree of smoke exposure not ascertained in either study; in prospective study, lack of comparability between the two groups not addressed.
Case-control study of serious lower respiratory illness in infants under 13 months, 132 cases, 18 controls, South Africa (Kossove 1982)	Cases more likely to be exposed to smoke than controls (70 versus 33%); no significant differences in cigarette smoking or other sociodemographic factors; cases 2 months older than controls.	Smoke exposure from open cooking fires averaged 6–7 h/day; some degree of confounding by age likely.
Prevalence study comparing respiratory symptoms of 31 children from homes with, and 31 children from homes without, wood stoves, Michigan (Honicky, Osborne, and Akpom 1985)	Prevalence rate ratio, comparing exposed to nonexposed children, was 2.7 for moderate respiratory symptoms and 28.0 for severe symptoms; parental smoking, socioeconomic status similar in both groups.	Measure of exposure a dichotomous variable, reflecting presence or absence of woodstove
Prospective follow-up of above study group for three years, by interview at end of each winter period; 10% annual attrition (Osborne and Honicky 1986)	Prevalence of coughing at night, coughing more than 4 days per week, and wheezing ranged from 30–85% in exposed, 0–16% in control.	Analyzed cross-sectionally each year, not longitudinally; measure of exposure as before; data available only in abstract form.
Prevalence study comparing respiratory symptoms and acute respiratory illnesses over the past 3 months, 258 schoolage children from homes with wood stoves and 141 from homes without, Massachusetts (Tuthill 1984)	Excess illness (defined as two or more episodes) not associated with wood stove use; also no excess of specific symptoms; no covariates related to either exposure or illness.	Specific questions asked relating to use of wood stove; cigarette smoking not related to respiratory illness, but indices of exposure to formaldehyde were related.
Prospective study of about 450 children, aged 0–2 years, exposed to smoke from fireplaces in poorly ventilated huts without chimneys, followed up over two time periods (Pandey, Neupane, and Gautam 1987)	Both studies showed association between increasing hours spent near the fireplace and severe acute respiratory illness; less consistent association seen with milder forms of illness.	Illness and exposure history gathered biweekly by trained lay reporters; measures of effect not calculable from data provided in preliminary report.
Prevalence study of respiratory symptoms and medical history, 5,338 children from six U.S. cities (Dockery et al. 1987)	Chest illness in past year associated with presence of wood stove in all six cities (prevalence ratio, 1.32); wheezing and coughing not associated	Exposure variable dichotomous reflecting presence or absence of wood stove; preliminary report.

adults over forty years of age. In contrast to the usual male preponderance of chronic respiratory disease, the prevalence among women was similar to that of men. Although direct measures of smoke exposure were not obtained, Master concluded from the pathologic changes found on autopsy of one young man that smoke exposure was the most important factor in the early development of respiratory disease.

The work of Anderson in New Guinea (1979), of Pandey in Nepal (1984a, 1984b), and Malik in India (1985) supports these conclusions. Anderson (1979) conducted a prevalence survey of respiratory disease among 1,284 adults in Papua, New Guinea. Chronic respiratory symptoms increased with age, and 20 percent of men and 10 percent of women over the age of forty-five years demonstrated an obstructive defect on spirometry. A measure of exposure to wood smoke was not incorporated into the study design, but the author concluded that smoke exposure was a potential etiologic agent for the excess respiratory symptoms and obstruction.

Pandey (1984a) administered a standardized respiratory symptoms questionnaire in a house-to-house survey of 2,826 permanent residents, who were 20 years of age or older, of two villages in Nepal. Subjects who reported more than an occasional cough were referred for further evaluation. The overall prevalence of chronic bronchitis, which was confirmed in the follow-up examination in 94 percent of these subjects, was 18.3 percent, and chronic bronchitis was equally prevalent in men and women. Data on 1,375 individuals were analyzed further to examine the relationship between prevalence of chronic bronchitis and exposure to domestic smoke, as measured by the average time spent near the fireplace on a daily basis (Pandey 1984b). A dose-response relationship was observed between the prevalence of chronic bronchitis and hours of exposure. This relationship was independent of smoking status. Pandey and colleagues (1985) also evaluated the pulmonary function status of 150 women aged thirty to forty-four from two rural villages in Nepal. Pulmonary function declined as hours of smoke exposure increased in cigarette smokers but not in nonsmokers.

Malik (1985) evaluated the respiratory status of 2,180 adult women with a standardized respiratory questionnaire. Pulmonary function was measured with a peak flow meter. Although the overall prevalence of chronic bronchitis reported in this study was lower than in previous studies, the prevalence of chronic bronchitis among women who cooked on chullas (traditional stoves) with cow dung and firewood was higher (5 percent) than among women who cooked with coal and kerosene (2.6 percent), kerosene oil alone (1.3 percent), or low-pressure petroleum gas (1.6 percent). The peak expiratory flow rate was also lower among the group of asymptomatic women who cooked on chullas. Smoking and socioeconomic status were not reported in this study although most of the women who cook with biomass fuels live in rural areas.

The relationship between domestic smoke exposure and acute respiratory illness has also been examined in children from less developed countries. Anderson (1978) examined 112 children from the highlands of Papua, New Guinea, on a weekly basis for thirty weeks. Children who lived in villages were considered to be exposed to smoke, and children who lived in government housing at the village station were not. The weekly prevalence rates of loose cough and nasal discharge were similar in the two groups, but confounding by socioeconomic status cannot be excluded.

Two studies of children in less developed countries have shown associations between exposure to smoke and lower respiratory illness. Kossove (1982) performed a case-control study of 150 Zulu infants under thirteen months of age, 132 infants with severe lower respiratory disease ("wheezing bronchitis" or pneumonia), and eighteen infants without respiratory disease. More than twice as many cases as controls (70 versus 33 percent) had a history of daily heavy smoke exposure from cooking and/or heating fires. Parental smoking and socioeconomic status were similar in the two groups although the average age of controls was two months younger than cases.

Pandey and colleagues (1987) conducted a prospective study during two observation periods of children aged two years and younger. Acute respiratory illnesses were ascertained biweekly by trained lay reporters and classified by severity (grades I–IV). Although some inconsistency was found between the two periods in the relationship between mild illnesses and hours of smoke exposure, both studies showed a strong positive association between the incidence of more severe respiratory illness and hours of exposure to wood smoke.

Cancer of the respiratory tract might plausibly be associated with domestic smoke exposure, but the relevant evidence is quite limited. Ecologic studies have suggested that rates of nasopharyngeal cancer are higher in the Kenyan highlands, where cooking is performed indoors, than in hotter parts of Kenya, where cooking is performed outdoors. Other studies of nasopharyngeal cancer, however, have not demonstrated this relationship (DeKoning, Smith, and Last 1985). High lung cancer mortality rates among women living in Xuan Wei County in China prompted investigators to examine the relationship between lung cancer occurrence and fuel type (Mumford et al. 1987). Comparing lung cancer rates among communes that used different types of fuel, the investigators concluded that lung cancer was related to the burning of "smokey coal" that emits high concentrations of polycyclic aromatic hydrocarbons. Lung disease mortality was substantially lower in communes burning wood and smokeless coal. Although a relationship between cancer and wood smoke remains highly speculative, it is plausible in light of the known toxicology of the constituents of wood smoke and needs further investigation.

Although these studies implicate exposure to smoke as an important etiologic factor in the development of both acute and chronic respiratory illness in developing countries, their relevance to the U.S. experience with residential use of wood stoves and fireplaces is questionable. In addition to differences in type of fuel used, emissions from an unvented open fire are several orders of magnitude higher than emissions from modern wood combustion appliances. In a small pilot study, Smith and colleagues (1983) measured the personal exposures to particulates and ben-

zo[a]pyrene among women cooking food on simple stoves using biomass fuels. During cooking periods, total suspended particulate exposures averaged nearly 7 mg/m^3 and benzo[a]pyrene exposures about 4,000 ng/m^3, the equivalent of smoking about twenty packs of cigarettes per day. These concentrations markedly exceed the fireplace benzo[a]pyrene concentrations of 11 and 19 ng/m^3 as measured by Moschandreas and colleagues (1980).

Unfortunately, the epidemiologic data relevant to the United States are sparse and inconsistent. One case report described a seven-month old infant who had recurrent serious episodes of wheezing and pneumonia, temporally related to being in his parents' or grandparents' homes, both having wood stoves (Honicky, Akpom, and Osborne 1983) The episodes resolved when use of the wood stoves was discontinued. This case prompted the same investigators to conduct a prevalence study of respiratory symptoms among thirty-one children, aged one to seven years, living in homes with wood stoves and thirty-one children, matched for age and sex, from homes with other types of heating (Honicky, Osborne, and Apkom 1985). Using a standardized respiratory symptoms questionnaire, information on demographic factors, sources of home heat, other potential sources of indoor pollutants such as tobacco smoking, past medical history, and occurrence of respiratory symptoms over the past winter was obtained from subjects' parents. Symptoms were scored dichotomously (ever or never present) and graded according to severity. The exposed group experienced significantly more moderate and severe respiratory symptoms: 84 percent of children in the exposed group reported at least one severe symptom as compared with 3 percent of the nonexposed group. These findings were not altered by consideration of potentially confounding factors. Follow-up examination of these children after two and three years showed that the respiratory symptoms persisted among the exposed group (Osborne and Honicky 1986). Over this time span, five families stopped using a wood stove; in all five cases the children's respiratory symptoms disappeared.

Although these findings are provocative, they have not yet been confirmed. Tuthill (1984), using a similar design, asked four hundred parents in western Massachusetts about their youngest elementary schoolchild's respiratory symptoms and illnesses over the previous winter months. Twenty-three percent of children from homes with wood stoves had had two or more acute respiratory illnesses, compared with 20 percent of children from homes without wood stoves. Wood stove use was also unrelated to fever, sore throat, runny nose, cough, or wheeze.

Wood stove use was also investigated in a cohort of six thousand schoolchildren participating in a nationwide study of air pollution in six cities (Dockery et al. 1987). Use of a wood stove was associated with a 32 percent increase in respiratory illness of sufficient severity to keep the child from normal activities for three or more days. This association was found in all six cities, although it was weakest in Portage, Wisconsin, the city with the highest prevalence of wood stoves. Physician-diagnosed respiratory illness before the age of two years, bronchitis in the previous year, chronic cough for three months of the year or more, and persistent wheeze were not associated with use of a wood stove.

Differences in the study populations, type of wood burned, or wood stove used or ascertainment of illness may explain the disparate findings of these studies. Honicky and co-workers (1985) studied preschool children, who usually spend more time at home than do children who are in school, and thus have higher exposures to pollutants in the home. Although questionnaires were used in each study to obtain data on recent respiratory symptoms, differences in questionnaire design might have produced different results. The lack of information on the effect of wood stoves on indoor air quality adds to the difficulty of interpreting these studies. Emission studies that have monitored particulates, CO, and NO_2 have not found a significant impact of airtight wood stoves on indoor air concentrations of these pollutants. Thus, the biologic basis for the positive findings of Honicky and colleagues (1985) and of Dockery and colleagues (1987) is uncertain. However, some unmeasured component of wood smoke, such as acrolein or another toxic aldehyde, may be emitted at sufficiently high concentrations to cause respiratory disease.

Although no epidemiologic studies have examined the effect of domestic exposure to wood smoke on adults in the United States, a case of interstitial lung disease attributed to wood smoke has been reported (Ramage et al. 1988). The subject was a sixty-one-year-old woman who developed increasingly severe shortness of breath over the period of a year. The patient's lungs contained a large number of carbonaceous particulates and fibers, with inflammation and fibrosis surrounding them. An evaluation of the patient's home implicated a malfunctioning wood-burning radiant heater as the source of the particles.

Another potential hazard of wood-burning stoves is illustrated by a case report of a Wisconsin family that experienced arsenic poisoning (Peters et al. 1984). All eight family members experienced recurring health problems involving the eyes, respiratory system, central nervous system, blood, reproductive system, skin, and hair loss. Symptoms improved during the summer but then became progressively worse each winter over a three-year period. When hair analysis revealed elevated arsenic levels, a thorough environmental evaluation of the house implicated the burning of chromium-copper-arsenate-treated wood as the major arsenic source.

Wood smoke exposure may also occur in the workplace. In a small preliminary survey of restaurant broiler cooks in Utah, the prevalence of respiratory symptoms in thirteen mesquite broiler cooks was compared with the prevalence in seventeen gas flame broiler cooks (Johns et al. 1986). Symptoms suggestive of any respiratory irritation were significantly higher in the mesquite broiler cooks. The prevalence of symptoms suggestive of either upper or lower respiratory tract irritation was also higher in the mesquite broiler group although the differences did not meet statistical significance. Although the small sample size prevented the authors from considering the effect of cigarette smoking, the differences are unlikely to be due solely to this factor.

SUMMARY

The current data base regarding the impact of wood combustion appliances on indoor air quality and the effect of these emissions on health is inadequate. Use of wood stoves may increase in the future if prices of oil and natural gas rise. Although the need to control ambient emissions became obvious in the 1970s, we do not yet have sufficient information to determine whether more effort to control indoor emissions is also warranted. A trial of discontinuance of wood stove use is certainly warranted in cases of children with recurrent respiratory symptoms. However, we do not yet have sufficient data on the relationship between respiratory symptoms and the use of a wood stove to recommend that all parents with wood stoves discontinue their use.

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