ABSTRACT

DARCY L. CAMPBELL. Indoor Air Pollution Effects on Low Socioeconomic Status Americans. (Under the direction of DR. MORRIS A. SHIFFMAN.)

Americans are exposed to air pollution outside, and in their places of work, at school, and in their homes. Low socioeconomic status Americans have traditionally been at greater risk to all forms of pollution, and indoor air pollution is no exception. Protection against indoor air pollution is one of the six necessary requirements for healthy housing. There are 33 million children and adults below the poverty level in this country who are more likely to suffer ill effects due to indoor air pollution, primarily because more pollutant sources are present in their homes than in the homes of middle to upper income Americans. The use of combustion appliances as primary or secondary heat sources, the presence of asbestos insulation, lead-based paint, and biological contaminants in older homes, along with frequent pesticide use and the influx of ambient air pollutants all increase the risk of health effects ranging from mucous membrane irritation to increased risk of developing cancer. The current Federal legislation regarding indoor air pollution will not sufficiently protect these people; a more active program is needed. Strict enforcement of existing legislation, development of new legislation, and the provision of information and financial assistance is required. This report is the first of its kind designed to inform and alert environmental agencies to the needs of low socioeconomic status groups regarding the hazards of indoor air pollution, and the knowledge gained through this assessment can be used to stimulate public policy actions.

## Table of Contents

## Tables and Figures

## Conversion Factors

| | | |
|---|---|---|
| $NO_2$: | 1 $mg/m^3$ = 0.532 ppm | 1 ppm = 1.9 $mg/m^3$ |
| CO: | 1 $mg/m^3$ = 0.873 ppm | 1 ppm = 1.145 $mg/m^3$ |
| HCHO: | 1 $mg/m^3$ = 0.813 ppm | 1 ppm = 1.23 $mg/m^3$ |
| $SO_2$: | 1 $mg/m^3$ = 0.382 ppm | 1 ppm = 2.62 $mg/m^3$ |

# I. INTRODUCTION

> "There were rats in Harlem for decades before the air became hard to breathe on Park Avenue- and no one was concerned. Black children in the rural South had to drink contaminated water from filthy drainage ditches-and still do-for years before oil spills killed the waterfowl or annoyed swimmers in Santa Barbara. The rural poor of Mississippi are not particularly interested in the ultimate disposal of plastic containers, glass bottles,or beer cans even though these are lofty present-day environmental goals. For the rural poor, the issue is not smog. It is survival."
>
> Andrew B. James, Assistant Professor of Community Health and Social Medicine, Tufts University Medical School (Sherrill, 1971).

Low income Americans have traditionally been exposed to more adverse conditions than Americans of the middle and upper classes. Adequate housing conditions should be available to every American, regardless of their socioeconomic status. The World Health Organization (1987) lists some of the necessary requirements for healthy housing as: 1) adequate water supply, 2) avoidance of overcrowding, 3) adequate solid wastes and excreta disposal, 4) access to community services, 5) adequate structural features, and 6) protection against indoor air pollution. It is this last requirement for a healthful living environment, protection against indoor air pollutants, which will be the focus of this paper.

Exposure to air pollution occurs outside, in the workplace, in the school, and in the home. Federal regulations exist to protect against ambient and workplace exposures, but very few protect against pollutants in the home, where most people spend the majority of their time. WHO

(1987) states that the knowledge, attitudes, and practices of residents are important factors in the extent to which these hazards affect health. This paper is the first of its kind to discuss the extent of the indoor air pollution problem in the homes of low socioeconomic status Americans, and it will demonstrate that indoor air pollution is more of a problem for these people than for the middle and upper classes. An evaluation will be made of the adequacy of the existing Federal regulations in dealing with the problem and protecting against the adverse health effects which result from exposures. Recommendations will be made so that low socioeconomic status Americans may be better protected from the avoidable effects of indoor air pollution. Finally, a check list will be provided as an output of this report to aid in the identification of potential or existing indoor air pollution problems so that the knowledge gained through this assessment can be used to stimulate public health actions.

It has long been recognized that healthful living conditions are an integral part of public health. The first public policies in the United States concerning housing were established in the 1600's, and by the 1850's it was known that sanitary and uncrowded living conditions were a necessity (Mood, 1969). The Tenement Act of 1901 in New York City was used as an example by other large cities to establish housing laws. In 1934, the National Housing Act was passed, and the Federal Housing Administration was created to carry out its objectives, including the development of minimum property standards for the determination of insurability of Federal mortagages (Mood, 1969). The most significant piece of public housing legislation was the Housing Act of 1949, which established a goal of providing "a decent home and a suitable living environment for every American family." The American Public Health

Association published A Proposed Housing Ordinance, which was used as a model for city and state housing codes (Mood, 1969).

In 1968 the nation's attention turned to housing as it never had before, and Congress passed the most sweeping housing legislation since 1949, which introduced a variety of federal assistance mechanisms (Urban America Inc., 1975). Two Presidential review bodies, the Douglas Commission and the Kaiser Commission, made recommendations to improve the housing conditions of low socioeconomic status Americans.

In 1970, the Department of Health, Education, and Welfare published the Neighborhood Environmental Evaluation and Decision System (NEEDS), which was designed to provide information on environmental problems so that action could be taken, the effects could be evaluated over time, and to increase the effectiveness and financial efficiency of money spent. NEEDS was also intended to provide researchers with associations and correlations between the prevalence of health problems and the quality of the residential environment. Finally, NEEDS was designed to better understand an area's problems, to test alternatives, and to match existing services to an area's needs (U.S. DHEW, 1970).

The check list provided in Section VIII can be considered to be an extension of the NEEDS analysis, which focuses specifically on the problems caused by the presence of indoor air pollutants and their sources. This check list will also help researchers identify relationships between the prevalence of health problems and residential conditions. Like the original NEEDS assessment, the health problems to be examined most closely are those of infants, pre-school children, and the elderly.

## II. IDENTIFICATION OF INDOOR AIR POLLUTION HAZARDS TO LOW INCOME AMERICANS

The effects of indoor air pollution on lower income families are more severe than the effects on middle and upper income families for several reasons. Primarily this group is more at risk because there are more pollutant sources present, hence more exposure occurs. For example, the type of heating method is an important factor in the potential exposure to nitrogen dioxide ($NO_2$), carbon monoxide (CO), sulfur dioxide ($SO_2$), volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs), and particles. Less affluent families are more likely to use wood or coal burning heating appliances and kerosene or gas-fired space heaters as either their primary heat source or as a secondary source. The presence of a combustion appliance is especially important to consider when examining the types of environments where these appliances are most commonly used. The Energy Information Administration (1986) found in their Residential Energy Consumption Survey that almost half of all kerosene heaters are used in multi-family dwellings, many of which have only one exterior wall and may have close to zero air changes per hour. Another third of all kerosene heaters are used in mobile homes, which also have low air exchange rates. In addition, the use of cheaper K-2 kerosene fuel will increase the sulfur emissions from kerosene heaters. It is also known that some Americans rely on their kitchen stoves for supplemental heat (Repace, 1982). In

the case of gas stoves, this may be a source of acute exposure to $NO_2$ and CO.

Low income families are the most common occupants of mobile homes, where formaldehyde exposures are of special concern because these environments have lower air exchange rates than most homes, are of smaller volume, and contain more sources of volatile organic compounds (VOCs) than other types of housing (Spengler and Sexton, 1983). These sources include materials in the mobile home such as particleboard and plywood, and carpets and furnishings. New mobile homes are usually purchased by retired persons on fixed incomes and families with young children, and these groups are hypersensitive to the health effects of indoor air pollutants.

Another common source of indoor air pollution is the presence of asbestos in ceiling tiles, floor tiles, spackling, insulation, and cement. Homes built before 1970 can be assumed to have asbestos present unless renovation has been done. Older, poorly maintained homes are often inhabited by fixed- or low-income families, thus this socio-economic group is more likely to be exposed to asbestos fibers. Asbestos exposure also increases during and immediately after removal occurs, especially if removal is handled improperly.

Older homes are also likely to be a source and breeding ground of biological contaminants. Bacteria, viruses, molds and their spores, mildew, and animal and insect parts and excreta are included in this pollutant category. High humidity is the most important factor which encourages the growth and dissemination of these contaminants (Burge, 1985). A living environment which is not tightly weatherized encourages the introduction of these contaminants from the ambient environment.

Homes without central air conditioning to maintain lower summer temperatures and humidities are likely to contain molds and dust mites, which can also be a problem in homes which are so tightly weatherized that humidity increases through the use of showers and appliances. Rats, mice, dust mites, and cockroaches also contribute biological contaminants through the dissemination of their feces, urine, and body parts (U.S. EPA, 1987).

It is probable that the presence of the above mentioned biological contaminants causes lower income families to be exposed regularly to pesticides. In low-rent apartment complexes or public tenements, the residents may have little or no control over the frequency or amount of pesticides applied, and may apply additional pesticides themselves. The use of insecticides, fungicides, and termiticides, indoors or out-of-doors, may result in increased exposure to chlordane, diazinon, chlorpyrifos, heptachlor, and propoxur, among others (Lewis et al., 1986).

Lead is another indoor air pollutant to which low income Americans may be exposed. Exposure can occur via several routes, such as inhalation of contaminated household dust or soil, inhalation of ambient air near a stationary source such as a smelter, or drinking or eating lead-contaminated water or food. Ninety-nine percent of all homes built before 1940, 70% of homes built between 1940 and 1959, and 20% of the homes built from 1959 to 1974 have paint with lead concentrations greater than 0.7 $mg/m^2$ (ATSDR, 1988). It is well documented that lead poisoning often occurs when children ingest lead-containing paint chips (Lin-Fu, 1982), but exposure also occurs through inhalation of airborne lead-containing house dust and by mouthing or sucking hands or objects which have been exposed to the lead-containing dust (Charney et al., 1980).

Figure 1 is a graphic representation of the various exposure routes for lead. It is well documented that lead poisoning is more prevalent among economically deprived children, especially black children (Chatterjee and Gettman, 1972; ATSDR, 1988). Charney and co-workers (1980) found that high blood-lead levels were associated with household lead dust levels. Exposure to lead may also occur when a lead-based paint on an outdoor surface is peeling or decaying (Ter Haar and Aronow, 1974). Ingestion of soil or mouthing or sucking of soil covered objects may occur, or lead may be inhaled along with dirt particles (Ter Haar and Aronow, 1974). Exposure to airborne lead is also often increased during renovation or removal processes (Lin-Fu, 1982; Inskip and Atterbury, 1983).

Homes which are not tightly weatherized encourage the entrance of many ambient pollutants into the indoor environment. Aside from externally applied pesticides, homes in high-traffic areas are besieged with automotive pollutants such as $NO_2$, CO, particles, benzene, and diesel emissions. Rose and co-workers (1985) state that central metropolitan areas receive higher traffic density than other parts of the city, and low income households tend to be located in these central areas. Esmen (1985) estimates that 20% of the homes constructed before 1970 have air exchange rates greater than 1.5 air changes per hour (ACH). This level compares to new homes which have 0.2 to 0.5 ACH.

Other pollutants to be discussed here are radon and environmental tobacco smoke. Although low income residents are not necessarily at greater risk through higher exposures, it is probable that they are not as aware of the problems associated with these pollutants as they should be, or as likely to be able to afford even minimal mitigation methods for radon. In addition, these pollutants act synergistically with other indoor air pollutants in the health effects they cause.

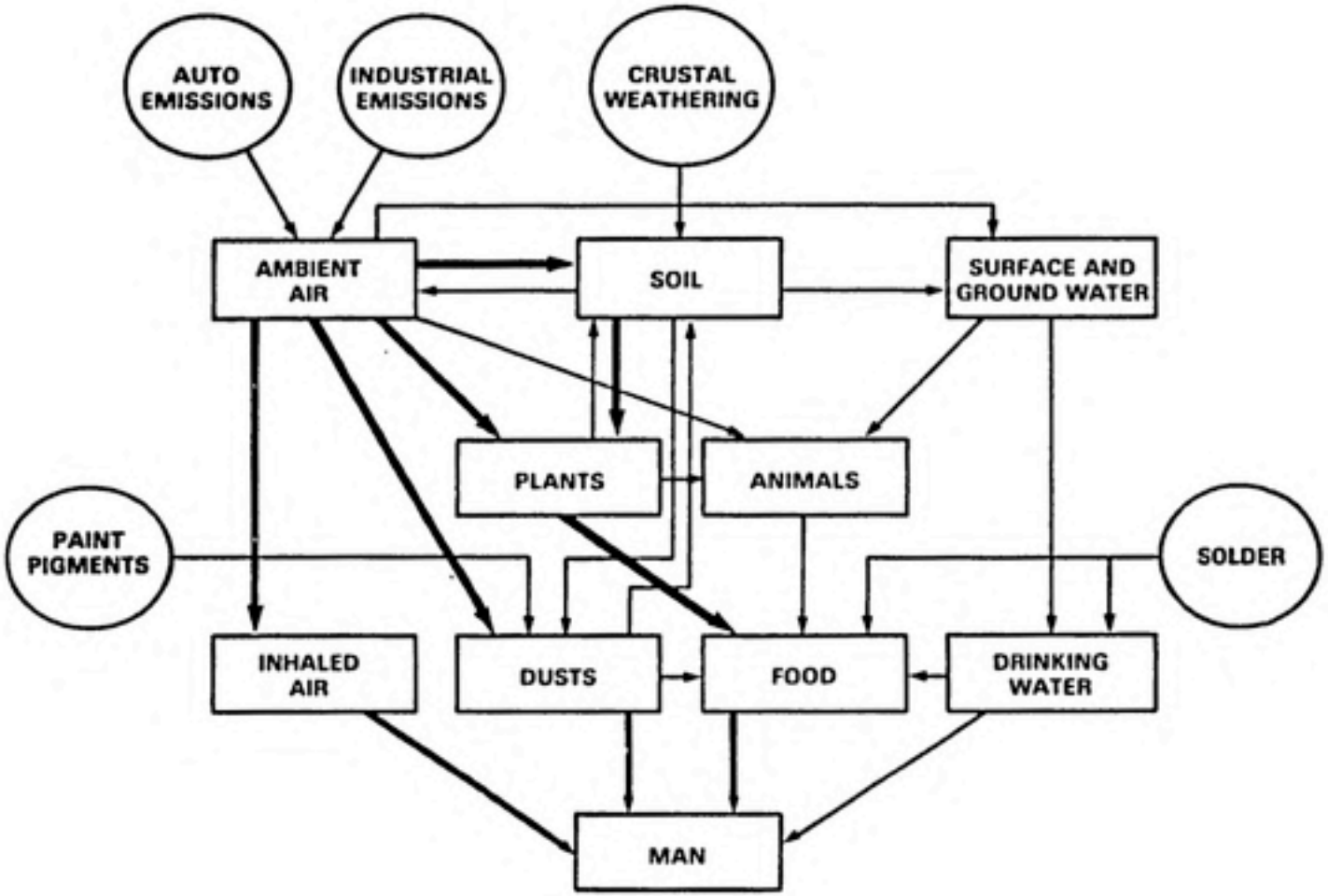


Figure 1. Routes of Lead Exposure.

Source: U.S. EPA (1986b).

The final aspects in this discussion of increased indoor air pollution risks concern the amount of time individuals are exposed, their treatment options once health problems arise, and their mitigation options. Although it is known that the average American spends up to 16 hours a day at home, young children, the infirm, and the elderly most assuredly spend even more time in their homes. It is often this group that is hypersensitive to the pollutants discussed above (Ammann, 1987).

Studies show that low income families are less likely to seek medical treatment than middle or upper income families. Newacheck and Starfield (1988) compared morbidity and physician visits for low and high income children and found that on the average, children from families with annual incomes of less than $10,000.00 had more bed days due to multiple health problems, yet used fewer physician services than higher income children in similar health.

The lack of information distributed to this group may also result in obvious signs and symptoms of pollution exposures to be ignored. An example of this are the flu-like symptoms of nausea, dizziness, and headache which occur following exposure to carbon monoxide (Dolan, 1985). If these symptoms are ignored, and the exposed individual remains home in order to "recover," the consequences may be fatal. An individual who becomes ill due to exposure to an indoor air pollutant usually relies on the medical profession to diagnose the cause of their ailment. This diagnosis is made more difficult by the presence of multiple pollutant sources. If a diagnosis is made, however, that an indoor air pollutant is responsible, the next question is what can be done to eliminate exposure. Economic limitations and the fact that the residence is not owner-occupied may prevent or hinder even the most simple mitigation efforts. In the U.S., 34% of all housing units are renter occupied, and

33% of the renter-occupied units house families which make less than $10,000.00 per year (U.S. Bureau of the Census, 1985). Product substitution, such as purchasing a newer-model combustion appliance or replacing old furnishings, may be economically unfeasible. In extreme cases, where the residence cannot be altered to reduce pollutant levels, a family of low socioeconomic status may not have the luxury to consider relocating.

## III. EXPOSURE LEVELS EXPECTED

### Combustion Appliances

The use of combustion appliances as a primary or secondary heat source often results in exposures to nitrogen oxides, sulfur dioxide, acid aerosols, particulate matter (including soot), volatile organic compounds (i.e., formaldehyde), polycyclic aromatic hydrocarbons (i.e., benzo(a)pyrene), and carbon monoxide (U.S. EPA, 1987). Combustion appliances include wood stoves, fireplaces, kerosene space heaters, and gas-fired space heaters and appliances.

As shown in the following table, test results from experimental chambers and actual in-home measurements give emission rates and pollutant levels for the compounds discussed above (Table 1). The emissions generated from combustion appliances can vary greatly according to the type of appliance, the appliance's age and operating condition, the type of fuel used, and the ventilation conditions in the room and house. For example, unvented kerosene space heaters can be convective, radiant, radiant/radiant, radiant/convective, or wickless (U.S. EPA, 1987). Higher flame heights during operation will increase the emission of nitrous oxide, while lower flames result in increased emissions of carbon monoxide, formaldehyde, and particulate matter (Mueller Associates, 1985). The emission of sulfur dioxide from kerosene heaters is dependent on the amount of sulfur in the fuel. In general, older convective kerosene heaters emit the most carbon monoxide, nitrogen

Table 1. Exposure Levels and Emission Rates from Combustion Appliances

| Pollutant | Source | Max. Concentration | Reference |
|---|---|---|---|
| $NO_2$ | Gas kitchen stove | 1 $mg/m^3$ (in kitchen) | U.S. EPA (1987) |
| $NO_2$ | Gas Range (1 burner) | 18-430 mg/hr | Mueller Associates (1985) |
| $NO_2$ | Gas oven | 67-270 mg/hr | Mueller Associates (1985) |
| $NO_2$ | Kerosene heater (radiant) | 16-38 mg/hr | Mueller Associates (1985) |
| $NO_2$ | Kerosene heater (convective) | 33-530 mg/hr | Mueller Associates (1985) |
| $NO_2$ | Gas space heater | 3-1225 mg/hr | Mueller Associates (1985) |
| $NO_2$ | Wood heater | 1.3-7.0 mg/hr | Mueller Associates (1985) |
| $SO_2$ | Kerosene heater (low sulfur fuel) | 0.1-2.0 ppm (12 hr average) | U.S. EPA (1987) |
| $SO_2$ | Kerosene heater (radiant) | 31-109 mg/hr | Mueller Associates (1985) |
| $SO_2$ | Kerosene heater (convective) | 37-94 mg/hr | Mueller Associates (1985) |
| $SO_2$ | Wood stove (non-air tight) | 42.4 ppb (hourly concen.) | TVA (1985) |
| CO | Wood stove (non-air tight) | 9.1 ppm (1 hr. concen.) | TVA (1986) |
| CO | Wood heater | 70-375 mg/hr | Mueller Associates (1985) |
| CO | Kerosene heater (radiant) | 281-542 mg/hr | Mueller Associates (1985) |
| CO | Kerosene heater (convective) | 35-635 mg/hr | Mueller Associates (1985) |

Table 1. continued...

| Pollutant | Source | Max. Concentration | Reference |
|---|---|---|---|
| CO | Gas space heater | 12-5004 mg/hr | Mueller Associates (1985) |
| CO | Gas range (1 burner) | 191-2700 mg/hr | Mueller Associates (1985) |
| CO | Gas oven | 195-3564 mg/hr | Mueller Associates (1985) |
| CO | Gas stove | 120.0 ppm (in kitchen, in use 20 min.) | Sterling and Sterling (1979) |
| Particles | Wood heater | 2.6 mg/hr | Mueller Associates (1985) |
| Particles | Kerosene heater (old-radiant) | 0.16 mg/hr | Traynor et al. (1983) |
| Particles | Kerosene heater (old-convective) | 0.034 mg/hr | Traynor et al. (1983) |
| Particles | Gas range (1 burner) | 1.9 to 30 mg/hr | Mueller Associates (1985) |
| Particles | Gas oven | 0.1 mg/hr | Fisk et al. (1985) |
| Particles | Unvented gas space heater | 0.2 to 3.2 mg/hr | Fisk et al. (1985) |
| Benzo(a) pyrene | Unvented gas range | $3.0 \times 10^{-3}$ ng/m$^3$ | Moschandreas et al. (1986) |
| Benzo(a) pyrene | Wood stove | 4.7 ng/m$^3$ | Moschandreas et al. (1980) |
| Benzo(a) pyrene | Fireplace | 11.4 ng/m$^3$ | Moschandreas et al. (1980) |

dioxide, and formaldehyde, while radiant heaters produce the highest levels of particles (Traynor et al., 1983). Pollutant emissions from gas-fired space heaters also vary according to usage pattern, brand of heater, burner design, size of heater, and the fuel-to-air mixture (Mueller Associates, 1985). Wood burning stove emissions and fireplace emissions into the indoor environment vary because of improper installation, cracks or leaks in the stove pipe, negative air pressure in the room, downdrafts, and refueling (U.S. EPA, 1987). Use patterns, appliance type, and type of wood burned also affect emission rates (U.S. EPA, 1987). Gas-stove emissions are affected most by the presence or absence of an outdoor-ventilated fan and whether the appliance has a pilot light or a newer electric starter. The age of the stove, the burner design, the number of burners in use, and the flame intensity have not been shown to affect pollutant emissions (GEOMET Technologies, Inc., 1976). Pilot lights produce pollutants during use and non-use periods (GEOMET Technologies, Inc., 1976).

Sterling and Kobayashi (1981) found that CO concentrations may reach 25 to 50 ppm in homes in which the gas range is used for heating purposes. They also investigated the amount of time the gas ranges were used per day, and found an average of two hours per weekday and three hours per weekend day. The National Ambient Air Quality Standard (NAAQS) for carbon monoxide is 35 ppm for 1 hour, and 9 ppm for an 8 hour average.

In addition to the factors mentioned above, the number of combustion appliances in use will affect pollutant levels. Leaderer and co-workers measured levels of $NO_2$ in homes with kerosene heaters and gas stoves, and found average $NO_2$ levels of 7.4 $ug/m^3$ (4 ppb) in homes with neither appliance, 36.8 $ug/m^3$ (19 ppb) in homes with one kerosene heater and no gas stove, 34.3 $ug/m^3$ (18 ppb) in homes with no kerosene heater and a gas

stove, and $NO_2$ levels of 66.8 ug/$m^3$ (36 ppb) in homes with one kerosene heater and a gas stove. Parkhurst et al. (1988) monitored $NO_2$ levels in public housing units with gas stoves, gas water heaters, and one or more vented gas heaters. They found that 47% of the 185 units had at least one weekly $NO_2$ measurement greater than 100 ppb, and 7% had at least one 200 ppb measurement or higher. The highest $NO_2$ level encountered was a weekly average of 1402 ppb and a unit average of 989 ppb. These concentrations can be compared to the primary National Ambient Air Quality Standard of 50 ppb (annual average) for $NO_2$. The 50 ppb annual average is the maximum ambient concentration to which Americans can be exposed for 24 hours per day for 365 days per year. It should be kept in mind that most Americans spend most of their time indoors rather than outdoors, and while the 50 ppb level may not be exceeded continually, during the heating season (up to six months of the year) this level may be greatly exceeded for 24 hours per day.

Several studies have shown that although combustion appliances are known to emit formaldehyde, these emissions do not significantly affect indoor levels. Girman and co-workers (1982) predicted average formaldehyde steady-state levels of 65 ug/$m^3$ for well tuned, unvented gas-fired space heaters, and levels of 391 ug/$m^3$ for poorly tuned heaters. Leaderer et al. (1984) found that formaldehyde levels were low (27.5 ug/$m^3$) in homes with various combustion sources present and remained low even in the presence of two kerosene heaters and a gas stove.

Mobile Homes

Mobile homes have been found to often have elevated concentrations of volatile organic compounds because of the presence of many material sources, their small volume, and their low air exchange rates. The U.S.

EPA (1987) reviewed the formaldehyde levels in mobile homes and homes with urea formaldehyde foam insulation, many of which were based on complaint investigations. The formaldehyde levels found in these environments ranged from 0.0 to 9.8 $mg/m^3$. The highest value will only occur rarely, however, typical concentrations range from 0.1 to 1.0 $mg/m^3$ (Ammann et al., 1986). The NRC (1981a) reviewed formaldehyde levels in mobile homes after complaints and found a range of 0.03 to 3 $mg/m^3$, unrelated to ambient formaldehyde levels.

Asbestos

Asbestos used in floor and ceiling tiles, insulation, and other materials may be in the form of chrysotile, amphibole, or actinolite. Chrysotile is the most common asbestos form found in the U.S. (U.S. EPA, 1987). Exposure usually occurs when the asbestos-containing material is physically damaged, but may also occur because of degradation, humidity variations, or vibration (U.S. EPA, 1978). The presence of asbestos in an indoor environment does not necessarily imply that exposure is occuring. Intact materials may not increase asbestos concentrations. Samples taken in a building with asbestos cement wall and roofing materials showed that following a heavy rain, asbestos concentrations ranged from 20 to 4500 $ng/m^3$ (Nicholson, 1978). The wearing of asbestos flooring tiles has been shown to cause asbestos concentrations of 170 $ng/m^3$, and Constant et al. (1983) measured asbestos levels of 183 $ng/m^3$ in school areas with asbestos surfacing materials. Nicholson et al. (1978) sampled 10 schools and found crysotile levels ranging from 9 to 1950 $ng/m^3$, with an average of 217 $ng/m^3$. The 1950 $ng/m^3$ measurement occured following floor sweeping in an area with a water-damaged asbestos surface with no visible sign of asbestos fibers in the air.

Biological Contaminants

An estimate of exposure to biological contaminants is more difficult than for the other indoor air pollutant categories discussed here. Because little is known about the occurrence and concentration levels, and because monitoring methods are not standardized to permit comparison of results, this discussion will simply detail the specific biological contaminants which are known to occur in indoor environments and discuss the factors that are known to affect concentrations. Molds and their spores, bacteria, viruses, protozoans, algae, insect body parts and excreta, pollen, and animal excreta make up this group of indoor air pollutants (U.S. EPA, 1987). Some of the molds identified indoors to date include: Aspergillis and Penicillium, and the bacteria include Legionella, Staphyllococcus, and Streptococcus (Burge, 1986). Insect parts and excreta are commonly found in house dust, especially dust mite feces (Andersen and Korsgaard, 1986). The concentrations of biological contaminants usually vary with season, and are affected by the indoor/outdoor pressure gradient, temperature, and humidity (Burge, 1985). Andersen and Korsgaard (1986) found that the optimal environment for most biological contaminants is a temperature around 25 degrees Centigrade and 75 to 80% relative humidity. Many of these contaminants enter from the outside, such as pollen, algae, and mold spores, thus the tightness of the home can directly affect the pollutant concentrations (Burge, 1985). Once inside, contaminants such as house dust mites, bacteria, and algae breed in such places as air conditioners, humidifiers, refrigerator drain pans, insulation, bedding, carpets, and upholstery (Platts-Mills and Chapman, 1987). Solomon (1975) reports that under poor hygienic conditions, concentrations of fungal spores indoors may be 200 to 400 percent above ambient levels. Table 2 lists some

Table 2: Biological Contaminant Concentrations

| Contaminant | Measurement | Concentration Indoor / Outdoor | | Building Type |
|---|---|---|---|---|
| Bacteria | Colonies/sample | 27 | 16 (fall) | Apartment |
| Bacteria | Colonies/sample | 40 | 43 (spring) | Apartment |
| Bacteria | Colonies/sample | 71 | 6 (fall) | House |
| Fungal spores | Colonies/sample | 12 | 38 | House |
| Fungal spores | Colonies/sample | 60 | 76 | House |
| Fungal spores | Colonies/sample | 55 | 13 | House |
| Pollen | Grains/$m^3$ | 11 | 205 (no AC) | House |

Adapted from: Samfield (1985)

measurements of bacteria, fungal spores, and pollen taken from homes and apartments by various researchers in the U.S., England, Denmark, and Japan.

Pesticides

People are exposed to pesticides via ingestion, inhalation, and dermal exposures (Lewis et al., 1986). Lewis and co-workers (1986) investigated inhalation exposures in a nine-home pilot study. The most common pesticides they encountered in their sampling were chlorpyrifos, diazonon, chlordane, propoxur, and heptachlor. Chlorpyrifos is an insecticide used in the control of mosquitos, cockroaches, termites, and lice, among others (Lewis et al., 1986), and diazinon is an insecticide used to control soil and household insects and flies. Chlordane and heptachlor are termiticides whose use was restricted in 1987 and banned in 1988 (Cohen, 1987), and propoxur is an insecticide for controlling

cockroaches, flies, and mosquitos (Lewis et al., 1986). The concentrations measured for these five compounds were usually one order of magnitude higher than for any other pesticides found, and the indoor levels measured were generally one to two orders of magnitude higher than outdoor levels (Lewis et al., 1986). Table 3 is a summary of the monitoring data for these five pesticides.

Lillie and Barnes (1987) examined 5000 homes for airborne chlordane levels and found concentrations above 5 ug/$m^3$ in 26% of the homes. The termiticide had either been applied to the soil in homes with crawl spaces or via subslab injection in homes built on slabs. This study revealed that 19% of the homes with crawl spaces exceeded that 5 ug/ $m^3$ level, and that chlordane levels in the home were directly correlated to the crawl space temperature. At the time this study was published, the National Academy of Sciences had recommended the 5 ug/$m^3$ level as an interim guideline for the home environment.

Table 3: Concentrations of the Most Common Pesticides

| Pesticide | Concentration (ug/$m^3$) Indoor / Outdoor |
|---|---|
| Chlorpyrifos | 2.4 / 0.059 |
| Diazinon | 1.4 / 0.11 |
| Chlordane | 0.51 / 0.058 |
| Propoxur | 0.042 / 0.0034 |
| Heptachlor | 0.088 / 0.016 |

Adapted from: Lewis et al. (1986)

Davies et al. (1975) reported that significant exposure to DDT can occur through house dust contamination; like chlordane and heptachlor, DDT usage has been banned in this country, however. Louis and Kisselbach (1987) found that pesticide concentrations may vary during sampling because of temperature, humidity, and air movement caused by heating and cooling systems.

Lead-Based Paints

As discussed previously, lead-based paints often cause high lead levels in household dust. Many studies have been undertaken to investigate lead poisoning in children, and very often lead-based paints are responsible. The Agency for Toxic Substances and Disease Registry (1988) estimates that about three million tons of lead remain in paint which is accessible to children in the U.S. Laxen et al. (1987) examined blood-lead levels and lead levels in household dust, and found a significant relationship between the two. Dust-lead levels ranged from 48 to 13,600 ug/g, with a median of 503 ug/g, while blood-lead levels ranged from 3.3 to 10.7 ug/dl with a median of 10.1 ug/dl. They also found that the highest airborne dust-lead levels were due to redecoration activities during the year before sampling. Clark et al. (1985) compared lead levels in different types of housing in Cincinnati, Ohio. They found that lead content of interior surface dust was the lowest for public and rehabilitated housing (350 and 623 ug/g, respectively) and highest for older private homes in dilapidated and deteriorating condition (2200 and 3000 ug/g). Even private nonrehabilitated homes which were judged visually to be in satisfactory condition had lead levels of 1410 ug/g. Blood-lead levels of children living in dilapidated/deteriorated homes were two to three times higher than levels of children living in public housing.

The NAAQS for lead is currently 1.5 ug/$m^3$ (quarterly average), and there is no U.S. standard for lead in dust. Laxen et al. (1987) recommended that a lead-dust level be established in the Netherlands, and calculated a standard of 1020 to 1750 ug/g based on comparison with the U.S. ambient air standard.

Ter Haar and Aronow (1974) measured lead levels around the perimeter of brick and frame homes in Detroit, Michigan. They found the average soil-lead concentration near brick homes ranged from 351 to 595 ppm within 2 feet of the house, and 156 ppm in the front yard (nearest to the street) and 200 ppm in the back yard. The lead in dirt within 2 feet of frame houses averaged over 2000 ppm, and front and back yard levels averaged 436 ppm. The authors concluded that exterior paint rather than vehicle exhaust was the source of high soil-lead levels.

Automotive Pollutants

Interior and exterior paints have not always been the main source of indoor lead concentrations. The U.S. Environmental Protection Agency lowered the acceptable lead level in gasoline on the basis that there was a strong relationship between leaded gasoline use and blood-lead levels. Figure 2 represents the EPA's interpretation of the second National Health and Nutrition Examination Survey data. The EPA then promulgated standards to reduce the lead content of gasoline (F.R., 1982, October 29; F.R., 1985, March 7). The U.S. EPA (1986) stated that ambient air lead levels have declined from 1.23 ug/$m^3$ in 1975 to 0.36 ug/$m^3$ in 1984. Lynam et al. (1983) found that overall mean blood-lead levels decreased 37% during this period, from 15.8 to 10.0 ug/dl. The ATSDR (1988) report concluded that past gasoline lead inputs produced airborne lead that significantly added to blood lead by direct and indirect routes, and that as expected, blood-lead levels have decreased as airborne lead has decreased.

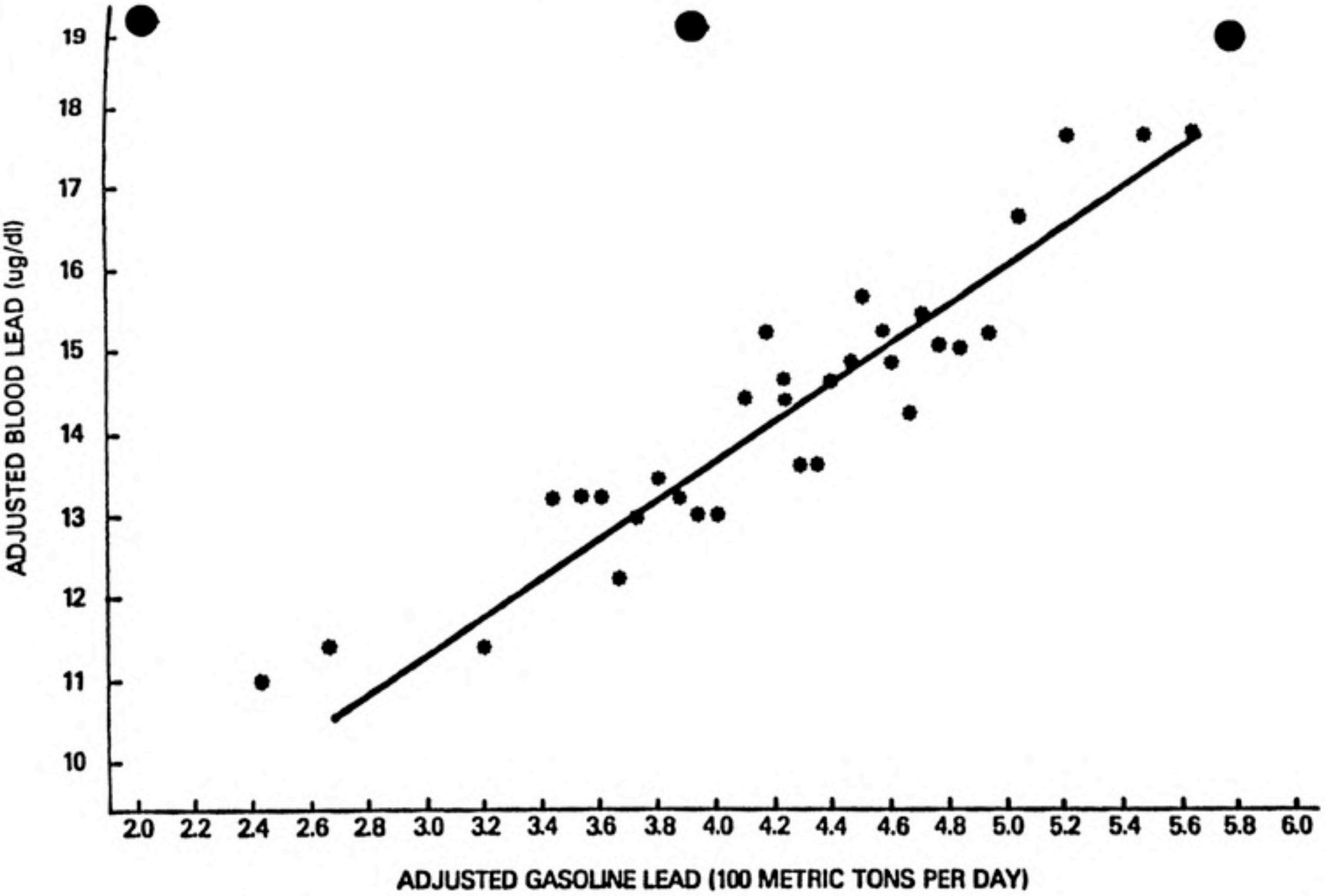


Figure 2. Relationship Between Lead in Gasoline and Blood Lead Levels.

Source: U.S. EPA (1985).

The infiltration of ambient air pollutants may also result in increased exposures to other automotive pollutants, such as diesel fuel emissions, nitrogen oxides, carbon monoxide, and benzene. Holmberg and Ahlborg (1983) estimated the emissions from automotive sources as shown in Table 4. The particles emitted from this source are small, usually less than 2 um in size. Diesel engines emit particles an order of magnitude higher than regular catalyst-equipped engines.

Radon

Radon enters dwellings primarily through soil gas or water. Indoor levels of radon measured range from 500 to 2 million $pCi/m^3$ (U.S. EPA, 1987). The presence of radon in the soil beneath a structure varies according to the geological formations in the area. High radium levels and porous soil or fractured rock structures facilitate the movement of radon and its progeny through the soil, while foundation cracks and negative pressure gradients move the gas into the dwelling (U.S. EPA, 1987). Some well water supplies are also known to be high in radon, but most are estimated to have radon concentrations of less than 2000 pCi/liter (U.S. EPA, 1987). Walsh et al. (1984) report some wells with radon levels exceeding 100,000 pCi/liter, however. Radon present in well-water supplies is distributed to the indoor air when it is heated or divided into small droplets, as in showering.

Environmental Tobacco Smoke

Environmental tobacco smoke contributes many compounds into the indoor environment, including nicotine, particles, PAHs (including B(a)P), benzene, formaldehyde, nitrogen oxides, and carbon monoxide (Samfield, 1985; Repace, 1982; Girman et al., 1982). Up to 3800 compounds have been detected in sidestream smoke (emitted from the burning end of the cigarette, pipe, or cigar) and the smoke which is

Table 4. Automobile Emissions

| Pollutant | Leaded | Unleaded | Diesel |
|---|---|---|---|
| Particles (mg/km) | 50-100 | 5-10 | 750-1500 |
| Benzene (mg/km) | 50-100 | 1-15 | 10-20 |
| Formaldehyde (mg/km) | 20-50 | 1-3 | 10-15 |
| Benzo(a)pyrene (ug/km) | 1-10 | 0.1-1 | 1-10 |
| PAH (ug/km) | 35-170 | 3 | 500-1000 |

Adapted from: Holmberg and Ahlborg (1983)

exhaled by the smoker (mainstream smoke). Of great concern from a health standpoint is the respirable particulate matter (RSP) which may reach very deep into the lungs of an exposed person. Respirable particulate matter is made up of particles which have a mean mass diameter of 2.5-3.5 microns or less. Samfield (1985) conducted a literature review of the available information on ETS, and concluded that if cigarette smoking is excluded, the respirable particulate matter found indoors is largely influenced by outdoor levels. Spengler et al. (1981) measured concentrations (annual mean) of RSP outdoors (21.1 $ug/m^3$), indoors with no smoking (23.4 $ug/m^3$), indoors with one smoker (36.5 $ug/m^3$), and indoors with two smokers (70.4 $ug/m^3$).

The following table gives emissions and concentration data available for a few compounds contained in environmental tobacco smoke (Table 5). Air dilution can greatly change the makeup of ETS, and a high degree of dilution can reduce particles from 500 $mg/m^3$ to very few (NRC, 1986). Dilution will also affect the volatilization of nicotine. As ETS ages in an indoor environment, nitric oxide will be oxidized to form nitrogen

dioxide (NRC, 1986). Burning temperature, type and blend of tobacco, the presence of substitutes or additives, cigarette length, and use of filters will also affect the chemical composition of environmental tobacco smoke (Esmen, 1985).

Table 5. Environmental Tobacco Smoke Pollutant Concentrations

| Pollutant | Concentration | Reference |
|---|---|---|
| CO | 8.3 mg/cigarette (sidestream) | Sandia National Laboratories (1982) |
| Respirable Particles | 10 mg/cigarette (sidestream) | Girman et al. (1982) |
| Total Suspended Particulates | 901-1318 ug/mg tobacco | Lewtas et al. (1987) |
| Nicotine | 1.6-2.4 mg/g tobacco | Lewtas et al. (1987) |
| Nicotine | 5-50 $ug/m^3$ | NRC (1986) |
| $NO_2$ | 7.31 $ug/m^3$ (compared to 6.23 w/ no smoker present) | Leaderer et al. (1987) |
| $NO_2$ | 21.3 $ug/m^3$ (winter) (compared to 17.5 w/ no smoker present) | Good et al. (1982) |
| $NO_2$ | 16.5 $ug/m^3$ (summer) (compared to 12.4 w/ no smoker present) | Good et al. (1982) |
| Benzene | 12-48 ug/cigarette (mainstream) | NRC (1986) |
| Formaldehyde | 70-100 ug/cigarette | NRC (1986) |
| B(a)P | 20-40 ng/cigarette (mainstream) | NRC (1986) |

## IV. DOSE-RESPONSE ASSESSMEMT

The information presented in this section will consist of the available dose-response information for individual indoor air pollutants, and when available, information for pollutant mixtures will be given. This pollutant mixture information will be of value because it will alleviate the need to assume an additive or synergistic relationship between pollutants. Dose-response information is not available, however, for the majority of indoor air pollutants.

### Combustion Appliance Pollutants

The primary mechanism by which carbon monoxide causes damage is via its interference with oxygen transport by binding to hemoglobin (U.S. EPA, 1984b). This results in less oxygen being released to tissues, which is especially harmful to organs with high oxygen demand, such as the brain and the heart (U.S. EPA, 1987). Neurological effects range from behavioral alterations to coma (Dolan, 1985; Ginsberg, 1985), and there is empirical evidence to suggest neurobehavioral function decrements at COHb levels below 5% (U.S. EPA, 1984b). Cardiac effects include arrhythmias and myocardial infarction (Dolan, 1985). Exposure to 25 ppm CO for extended periods of time (1 to 8 hrs) results in carboxy-hemoglobin (COHb) levels of 2-3% among nonsmokers. This level of COHB results in decreased exercise and work capacity. The population especially at risk from CO exposure is chronic angina patients (U.S. EPA, 1987). The U.S. EPA (1984b) states that frequency and duration of angina attacks increase at 2.9 to 4.5% COHb, and may occur at even lower COHb levels. Other risk groups are the elderly, fetuses and infants, and

people with severe respiratory diseases, chronic bronchitis, and emphysema. People with congestive heart failure, anemia, or those taking drugs which depress the central nervous system are also at greater risk (U.S. EPA, 1984b).

Exposure to $NO_2$ at levels greater than 3.7 $mg/m^3$ for less than 2 hour exposures causes pulmonary function changes in healthy subjects (U.S. EPA, 1982a). Bylin et al. (1985), however, found pulmonary function changes at 0.4 $mg/m^3$. Asthmatics have been identified as a susceptible subpopulation, and the U.S. Department of Energy (1986) states that exposure for 15 minutes to 0.5 to 5.0 $mg/m^3$ will increase airway resistence in asthmatics. The DOE (1986) also indicated that in healthy individuals, exposure for 15 minutes at 7.5 to 9.4 $mg/m^3$ will decrease pulmonary diffusing capacity, and exposure for 5 minutes to 11.3 to 15.2 $mg/m^3$ will increase airway resistance in both asthmatics and healthy adults.

The effects seen in gas stove epidemiologic studies are presumed to be due to $NO_2$. Melia et al. (1979) studied school children exposed to gas stove emissions as compared to children living in homes with electric stoves, and found a higher prevalence of bronchitis, coughing, and colds going to the chest in the children in homes with gas stoves. Speizer et al. (1980) found an association between serious respiratory illness before the age of two in children in homes with gas stoves. The $NO_2$ levels measured ranged from 14.7 to 54.3 $ug/m^3$, with one short term peak of 1100 $ug/m^3$ in a kitchen. Ogston et al. (1985) studied the effects of gas stove emissions on infants and found no association with illness or hospitalizations, but Ekwo et al. (1983) studied children ages 6-12 and found a significant relationship beween gas stove usage and hospitalization for respiratory illnesses. Studies of adults and gas stove

emissions have not found any significant increases in acute respiratory illnesses (U.S. EPA, 1987). The U.S. EPA (1987) concluded that although the health effects information may be inconclusive, $NO_2$ is likely to have additive or synergistic effects with other indoor pollutants, and the $NO_2$ levels observed in some homes can decrease pulmonary function in asthmatics.

The effects of sulfur dioxide alone (not associated with particles) are most serious in asthmatics, and at 0.75 ppm asthmatics will experience a doubling of airway resistance (Horstman et al., 1986). The effects of particles include irritant effects, altered mucociliary transport, changes in alveolar macrophage activity, airway constriction, decrements in lung function, and increased respiratory infection in children (U.S. EPA, 1987). The health effects of soot are primary due to the attached polycyclic aromatic hydrocarbons (PAHs). Soot has been found to be mutagenic in short-term bioassays, and some of the attached PAHs are known to be carcinogenic, such as the dinitropyrenes which have been found in the emissions of kerosene heaters (Tokiwa et al., 1985). Occupational studies with coal soot show an increased risk for lung, larynx, and skin cancer (U.S. EPA, 1987). Groups especially sensitive to $SO_2$ and particulate matter exposures are asthmatics, the elderly, and those with preeexisting cardiovascular or respiratory diseases (U.S. EPA, 1986c).

The air sampled from homes with wood stoves in use has been found to have more mutagenic activity than the air taken from homes without wood stoves (Lewtas, 1985), and wood smoke has also been found to contain some compounds which are known carcinogens. Benzo(a)pyrene is an example (U.S. DHHS, 1985). Calle and Zeighami (1985) estimate that exposure for life to 1 $ng/m^3$ of B(a)P will cause from 0.2 to 5.0% excess lung cancer deaths.

Other PAHs act as procarcinogens and cocarcinogens.

Formaldehyde

Formaldehyde exposure causes irritation of the mucous membranes of the eyes and upper respiratory tract (U.S. EPA, 1987). The following table shows the dose-response information for acute exposures (Table 6). Formaldehyde is a known animal carcinogen and causes chronic reductions of lung function and asthma in humans (U.S. EPA, 1987). Formaldehyde is thought to cause allergenic responses in 8% of the population (U.S. EPA, 1987).

Asbestos

Asbestos is a known human carcinogen which causes lung cancer and mesothelioma, the two most important causes of death due to asbestos exposure (Nicholson, 1986). Mesothelioma is a rare cancer of the membrane which lines the chest and abdomen. Asbestos also causes asbestosis, a non-cancerous respiratory disease, and cancers of the gastrointestinal tract. It is thought at this time that chrysotile is not as likely to cause lung tumors as some of the other forms of asbestos, particularly amosite and crocidolite (Nicholson, 1986). Chrysotile is important as a cause of mesothelioma, however. In general, the effects of asbestos exposure are not apparent for up to 20 years following exposure.

Biological Contaminants

Because indoor environments are contaminated with a great number of biological contaminants, this discussion will focus on those for which some type of health effects information is available and those which are likely to be encountered in the types of housing which are available for low income families. Fungi are most commonly associated with allergenic reactions indoors, including allergic rhinitis and bronchial asthma (U.S.

Table 6. Formaldehyde Dose-Response Information

| Concentration ($mg/m^3$) | Estimated Median | Reported Effects |
|---|---|---|
| 0.06-1.2 | 0.1 | Odor threshold for 50% of people |
| 0.01-1.9 | 0.5 | Eye irritation threshold |
| 0.1-3.1 | 0.6 | Throat irritation threshold |
| 2.5-3.7 | 3.1 | Biting sensation in the nose, eye |
| 5-6.7 | 5.6 | Tearing eyes, long term lung effects |
| 12-25 | 17.8 | Tolerable for 30 minutes with strong flow of tears for one hour |
| 37-60 | 37.5 | Inflammation of lung (pneumonitis), edema, respiratory distress, danger to life |
| 60-125 | -- | Death |

Adapted from: Lucier and Hook (1984)

EPA, 1987). Other fungi, however, produce potent mycotoxins which may be carcinogenic (e.g., aflatoxin), mutagenic, affect immunosuppression, or act as nephrotoxins, heptatoxins, or neurotoxins (U.S. EPA, 1987). Airborne molds which are known to be toxigenic include Aspergillis, Penicillium, Fusarium, and Cladosporium (Burge, 1985). Viruses spread through indoor environments include measles, chickenpox, and respiratory diseases such as colds and influenza (U.S. EPA, 1987). Tuberculosis is caused by an airborne mycobacterium, and inhalation exposure to just one infective unit may cause infection. Legionella is a soil bacterium which

can become airborne and cause Pontiac Fever, a flu-like syndrome, or Legionnaires disease, an often fatal pneumonia (U.S. EPA, 1987). Platts-Mills and Chapman (1987) reviewed the health effects information on house dust mite allergens, and found that they can contribute to asthma, perennial rhinitus, and atopic dermatitis. Mites of the genus Dermatophagoides produce the most common allergen, and there is a high prevalence of mite allergy in asthmatics. Mathison et al. (1982) states that aeroallergen-provoked asthma is more of a problem for children and young adults than for other adults. Korsgaard (1983) estimates that 100 mites/gram of dust is the risk factor for sensitization and the development of asthma.

The National Health Education Committee (1976) states that viruses and bacteria which cause pneumonia, influenza, and other respiratory diseases are the fifth leading cause of death in the U.S., and the most important cause of acute disabling illnesses.

Pesticides

The health effects information for the pesticides of interest in low socioeconomic housing units is generally from oral and dermal toxicity studies. This discussion will evaluate the available inhalation studies, and where information is lacking the oral $LD_{50}$ and acceptable daily intake (ADI) information will be given.

Most organophosphate compounds are irreversible acetylcholinesterase inhibitors. Berteau and Deen (1978) report an inhalation $LD_{50}$ of 94 mg chlorpyrifos/kg for mice. The World Health Organization (1973) concluded that chlorpyrifos had no reproductive or teratogenic effects in rats, nor was any effect found in mice given 10.0 mg/kg/day orally. Like most of the organophosphate pesticides, chlorpyrifos and diazinon consistently give negative mutagenicity results in Salmonella typhimurium and

Escherichia coli, with and without activation. Diazinon did not increase the number of chromosomal aberrations or induce sister chromatid exchanges (U.S. EPA, 1988). The many chronic and subchronic feeding studies with organophosphates have not shown any carcinogenic activity. The primary effects of these compounds on humans is neurotoxicity. The initial symptoms of acute exposure are intestinal cramps, tightness in the chest, blurred vision, sweating, headache, and diarrhea (U.S. EPA, 1988). The second stage of intoxication results in symptoms of weakness, fatigue, cyanosis, and tremors. The oral risk reference dose for chlorpyrifos in humans is 0.003 mg/kg/day (U.S. EPA, 1988). Studies of workers exposed to up to 27.6 $ug/m^3$ and diazinon up to 41.0 $ug/m^3$ indicate a significant inhibition of plasma cholinesterase but not red blood cell cholinesterase (Hayes et al., 1980). The EPA (1984) found that diazinon's no-observed-effect-level (NOEL) was 0.9 ug/kg bw/day. Murphy (1986) gives an ADI of 0.002 mg/kg for humans.

A retrospective study of workers exposed to the chlorinated insecticides chlordane and heptachlor by Wang and MacMahon (1979) found no increase in deaths due to cancer, but found an increase in deaths from cerebrovascular disease. Murphy (1986) reviewed the oral $LD_{50}$ toxicity information for various pesticides, and listed heptachlor's oral $LD_{50}$ is 100 mg/kg, with an ADI of 0.0005 mg/kg/day for humans. The oral $LD_{50}$ for chlordane is 335 mg/kg for male rats, and the ADI is 0.001 mg/kg/day. Murphy (1986) also lists chlordane and heptachlor as pesticides reported to have effects on host defense mechanisms. Propoxur (Baygon) was listed with an oral $LD_{50}$ of 83 mg/kg for male rats.

Lead

The Agency for Toxic Substances and Disease Registry (1988) recently reviewed the health effects associated with lead exposure, and their

information will be summarized here. The groups especially at risk for lead poisoning are fetuses and young children. Young children are at risk because of their developmental physiology and their frequency of contact with lead contaminated objects. Children ingest and absorb a larger amount of lead per unit body measure and retain a larger fraction than adults. Children also have higher metabolism and respiration rates to enhance lead uptake. Fetuses are at risk because lead is readily transferred across the placenta. The primary target for toxicity is the brain or central nervous system. Other targets are the heme-forming system and the vitamin D regulatory system. Lead exposure is often characterized by concentration of lead in blood (ug/dl), and 10-15 ug/dl is associated with the onset of effects which may be biomedically adverse. Lynam et al. (1983) found that the indicated response of blood-lead concentration to inhaled air lead is about 1 ug/dl per 1 $ug/m^3$. The ATSDR (1988) report gives the following dose-response information (Table 7). One factor of great importance when evaluating lead toxicity is its accumulation in the body. Low exposure levels can cause lead to build up significantly over time, with resulting injuries to the central nervous system of children which are irreversible. Some of the effects of lead exposure may be reversible, such as the effect on heme biosynthesis, but these effects can only be alleviated if exposure is discontinued. In the case of low income families, this may not occur.

Automotive Pollutants

The health effects associated with automotive pollutants will include the effects already discussed for carbon monoxide, nitrogen oxides, lead, formaldehyde, PAHs, and particles. In addition, benzene and diesel emissions may have substantial health effects if encountered in high enough concentrations. Holmberg and Ahlborg (1983) evaluated

Table 7. Dose-Response Information for Blood-Lead Levels

| Pb-B (ug/dl) | Health Effect |
|---|---|
| 10-15 (prenatal and postnatal) | Reduced gestational age and weight at birth, neurobehavioral and growth deficits, reduced size up to age 7 |
| 15-20 | Adverse impacts on heme biosynthesis, and vitamin D and calcium synthesis |
| 25 and below | Deficits in IQ scores in children, slowed reaction time |
| 30 | Slowed nerve conduction velocity |
| 40 and below | Peripheral nerve dysfunction in children |
| 40 | Reduced hemoglobin synthesis |
| 40-80 | Peripheral neuropathy and frank anemia |
| 80 | Severe poisoning (coma, convulsions, mental retardation, death) |
| 80-100 | Encephalopathy |

Adapted from: ATSDR (1988)

---

the health effects of auto emissions and found that gasoline emissions contain much less nitrated PAHs (e.g. dinitropyrenes) than diesel emissions. Both gasoline and diesel emissions caused mutations in short-term bioassays. Gasoline extracts were found to be more mutagenic than diesel extracts, but diesel engines emit more particles and associated organic matter. The organics from motor vehicle emissions contain known human carcinogens, and there is evidence to support a relationship between benzene exposure and leukemia (U.S. DHHS, 1985).

Radon

The health effect associated with radon exposure is an increased risk of lung cancer (U.S. EPA, 1987). The EPA (1986a) predicts that 5000 to 20,000 lung cancer deaths per year are due to radon exposure, and the EPA has established a recommended "action" level of 4 pCi/l (0.02 working levels) in homes (U.S. EPA, 1986a). The EPA estimates that exposure to 4 pCi/l for 70 years will result in between 1 and 5 lung cancer deaths out of 100, while exposure to 200 pCi/l is estimated to cause between 14 and 42 deaths in 100.

Environmental Tobacco Smoke

Many pollutants are contained in environmental tobaccco smoke, as discussed in Section III. This discussion will focus on the whole mixture of ETS, rather than each individual compound. Eye, nose, and throat irritation are the most common acute effects of ETS (U.S. EPA, 1987). Reviews by the EPA (1987), Schenker et al. (1983), and Ware et al. (1984), among others, have found a strong correlation between ETS and the occurrence of lower respiratory tract illness in children. Increased frequency of bronchitis, pneumonia, and other respiratory symptoms have also been found in children during the first year of their lives (U.S. EPA, 1987). The health effects in adults are not as well established as those in children, but the NRC (1986) and the Surgeon General (1986) both concluded that epidemiologic studies indicate an association between involuntary smoking and lung cancer. Repace and Lowrey (1985) estimate that 5000 lung cancer deaths per year are due to ETS exposure. The associations between ETS and asthma and ETS and cardiovascular disease have not been definitely defined.

## V. ESTIMATED NUMBER OF LOW INCOME AMERICANS EXPOSED TO THESE POLLUTANTS

Although it is difficult to predict the number of people exposed to the specific pollutant concentrations given in Section III, an estimate can be made of the number of people potentially exposed. This information will be gathered primarily from census data and consumer purchasing information. An estimate can be made of the potential exposure which low-income families receive, but as discussed previously, many factors will influence pollutant levels, such as activity patterns, ventilation practices, and condition of the home or the pollutant source.

Census figures indicate that there are 234.5 million people in the U.S., and 14.4% of these individuals fall below the weighted poverty level, that is, 33.7 million people (U.S. Bureau of the Census, 1985). The Census Bureau uses a weighted poverty level based on the number of family members and annual income. For a 1 person household, $5300.00 and below is the poverty level, while for a family of 7 or more persons an annual income of $14,000.00 is considered to be the poverty level. In cases where specific information is not available for these low-income individuals, it will be assumed that the same percent of low-income individuals are exposed to indoor air pollutants as in the general population. In many cases this may underestimate exposures because of the factors discussed previously. In cases where the census data was presented by household, it will be assumed that there is an average of 2.5 residents per household.

Beginning with combustion appliances, estimates are made of the number of low-income Americans potentially exposed to emissions. These estimates are derived as discussed above; 14.4% of the number of appliances in use is multiplied by an average of 2.5 persons per dwelling. The number of kerosene heaters used by low socioeconomic status Americans is based on the estimate of 7 million kerosene heaters in use in the U.S. By examining kerosene fuel consumption patterns, it is seen that 43% of the fuel purchased is used in homes whose average annual income is less than $10,000.00 (Energy Information Administration, 1986). An estimate is made then, that 3 million low income families use kerosene heaters as primary or secondary heat sources.

Gas ranges: 14 million

Gas space heaters: 2.9 million

Wood stoves: 1.9 million

Fireplaces: 6.5 million

Kerosene heaters: 7.5 million

Sterling and Kobayashi (1981) evaluated the prevalence of gas stove usage for heating and found that 55% of the homes they studied in New York City used the gas oven as a heat source. In addition to the volatile organic compound exposures which individuals receive from material sources in mobile homes, a suprising number of kerosene heaters are used in these environments. The Energy Information Administration (1986) estimates that one third (2.3 million) of all kerosene heaters are used in mobile homes. Because of the factors discussed previously, this could mean that this population is exposed to very high concentrations of formaldehyde, carbon monoxide, nitrogen oxides, and particles, among others. Again assuming that there are 2.5 residents per home, an estimated 5.8 million Americans may be living in mobile homes and using

kerosene heaters.

The NRC (1981) estimates that 11 million people live in mobile homes which contain substantial amounts of particleboard and plywood. Even if a combustion appliance is not present, these people are exposed to higher than average concentrations of volatile organic compounds, especially formaldehyde.

An estimate of the number of people exposed to asbestos fibers will be made solely on the basis of the number of occupied homes built before 1970. Although this estimate may include some homes which have been renovated and had the asbestos removed, or homes in which the asbestos is intact and not friable, there is no way to estimate the number of these cases. The fact that renovation was undertaken may not mean that asbestos exposure is decreased, as careless handling may only serve to increase the amount of airborne fibers. In the case of children exposed, it must be kept in mind that exposure may also occur at school (Nicholson et al., 1978). The U.S. Bureau of the Census (1985) found that 68 million homes in the U.S. were built before 1970, so an estimated 170 million people, or 72% of the population, live in homes where asbestos insulation, spackling, cement, or floor tiles may be present. Although it may be a gross underestimation, if 14.4% of these people fall below the poverty level, then 24.5 million low-income Americans live in these homes.

An estimate of the number of people potentially exposed to high concentrations of biological contaminants can be made generally by estimating the number of homes which may have favorable conditions for their growth. The presence of old insulation and the lack of air conditioning in humid areas of the country are examples of these conditions. Another important factor to consider with regards to

biological contaminants is the presence of cockroaches and other insects. The U.S. Bureau of the Census (1985) lists approximately 14 million homes in the South and the Northeast which do not have central or room air conditioning. If 14.4% of these families are below the poverty level, then 2 million homes of low income Americans have the potential for increased exposure to biological contaminants. In addition to favorable conditions, the number of people who suffer from asthma, legionellosis, and allergic rhinitis (hay fever) can be used as an indication of the number of people who are sensitive to biological contaminants. Mathison et al. (1982) report that asthma afflicts 10 million Americans, and the U.S. Bureau of the Census (1985) recorded 852 cases of legionellosis (up from 654 cases in 1982). The U.S. Bureau of the Census also reports that 19 million people suffer with allergic rhinitis.

Although almost all Americans use pesticides both in and around their homes, the population of concern here are those who are exposed involuntarily. This assessment will thus focus on the number of people living in public housing tenements, where they have no control over the frequency of pesticide application, and where the concentration of pesticides applied probably exceeds that to which the average person is exposed. In the U.S. there are over 3.5 million families living in public or subsidized rental housing units (U.S. Bureau of the Census, 1985). Assuming 2.5 persons per household, there are an estimated 8.7 million people potentially exposed to high pesticide levels on a regular basis.

The ATSDR (1988) estimates that a total of 42 million homes occupied today have paint with lead levels exceeding 0.7 $mg/m^2$. The total number of children less than 7 years of age in these homes is 12 million, and

this group of Americans is at the greatest risk for lead toxicity. Of these 12 million young children, 1.8 million live in deteriorating lead-painted homes (ATSDR, 1988). They also concluded that 2.4 million children living in Standard Metropolitan Statistical Areas already have unacceptable blood-lead levels above 15 ug/dl. As expected, the ATSDR report found that the "traditional" high-risk groups, e.g. poor, inner-city black children, figured prominently in the outcomes. The ATSDR (1988) evaluated blood-lead levels in children of families whose income is less than $15,000.00, and found that a total of 1.5 million children under the age of 5 have blood-lead levels exceeding 15 ug/dl.

Exposure to high levels of automobile exhaust is estimated by the number of low-income residents in metropolitan areas. The U.S. Bureau of the Census (1985) states that there are over 6.6 million low-income families living in metropolitan areas. A survey of all low-income households shows that only 16% have central air conditioning. This indicates that 13.9 million low-income Americans live in metropolitan areas without central air conditioning.

The U.S. EPA (1987) estimates that 2.5 million people are exposed to radon in the U.S., thus 360,000 low-income residents have the potential for high exposure levels. This may be an overestimate due to the fact that many low income Americans may live in homes which have not been weatherized to be airtight. The EPA (1987) also states that 124 million people, including smokers, are exposed to environmental tobacco smoke. If 14.4% of these people are below the poverty level, then 17.8 million are exposed to ETS.

Although these are very crude estimates of the number of low-income Americans exposed to indoor air pollutants, they indicate the potential for a large number of health-related problems. It is essential to keep

in mind when examining these estimates that multiple exposures will also occur. For example, of the homes built before 1940 in the U.S., the U.S. Bureau of the Census (1985) found that 20% use fuel oil or kerosene as the primary heat source, and wood or coal stoves are used in 2 million of these homes. Residents of these homes thus have potential exposures to lead-based paints, asbestos, combustion appliance emissions, and biological contaminants. As discussed in Section III, the presence of more than one combustion source results in additive exposure levels. Many indoor air pollutants also act synergistically in the health effects they cause, such as asbestos and ETS or radon and ETS.

The information presented in this section may also be examined by comparing the number of low income Americans exposed to these pollutants with the number of non-low income Americans exposed. For example, 16% of all low income households rely on kerosene heaters as a primary or secondary heat source, while only 6% of other households use these heaters. In other words, 43% of all kerosene heaters are used by only 14.4% of the population. Gas ranges are present in 55% of all low income residences, but are present in only 40% of other households. The ATSDR's (1988) estimate of 1.5 million low income children whose blood-lead levels exceed 15 ug/dl (29% of all low-income children less than 5 years old) can be compared to their estimate that 880,000 of the 8 million children from non-low income families will have blood-lead levels >15 ug/dl (or 10% of these children).

## VI. PUBLIC HEALTH IMPLICATIONS

Based on the information presented in Sections III, IV, and V, an estimate can be made of the magnitude of the indoor air pollution problem for the 33 million low socioeconomic status children and adults living in this country. Because of the nature of the existing information, a quantitative risk characterization of the health effects from indoor pollution is not possible. The exposure information available is rarely consistent with existing dose-response information, thus making it impossible to make more than a qualitative estimate of the number of low socioeconomic status Americans who will suffer the adverse health effects of indoor air pollution. This qualitative assessment adequately describes the indoor air pollution problems faced by low income Americans, however, and the information thus synthesized is sufficient to lead to public policy considerations, as will be discussed in Section VIII.

Table 8 summarizes the indoor air pollutant concentrations reported in various studies. It must be kept in mind that every household will have different pollutants present in different concentrations, even if the same sources are present. Table 9 indicates the potential number of people who may harmed in some manner by the indoor air pollutants present in their homes. As discussed previously, many of these estimates may be gross underestimates. Low income Americans are more likely to use combustion appliances in their homes, and these appliances may be older and less efficient than the same appliances in middle and upper income homes. Low income Americans are more likely to live in older homes with

Table 8. Summary of Indoor Air Pollutant Concentration Levels

| Pollutant | Source | Max. Concentration | Reference |
|---|---|---|---|
| $NO_2$ | Gas kitchen stove | 1 $mg/m^3$ | U.S. EPA (1987) |
| $NO_2$ | One kerosene heater | 36.8 $ug/m^3$ | Leaderer et al. (1984) |
| $NO_2$ | Gas kitchen stove | 34.3 $ug/m^3$ | Leaderer et al. (1984) |
| $NO_2$ | One kerosene heater and one gas stove | 66.8 $ug/m^3$ | Leaderer et al. (1984) |
| $NO_2$ | One gas stove, one gas water heater, and one vented gas heater | 2.67 $mg/m^3$ (weekly avg.) | Parkhurst et al. (1988) |
| $SO_2$ | Kerosene heater (low sulfur fuel) | 0.1-2.0 ppm (12 hr. avg.) | U.S. EPA (1987) |
| CO | Gas range used for heating | 25-50 ppm | Sterling and Kobayashi (1981) |
| CO | Wood stove (non-air tight) | 9.1 ppm (1 hr. concen.) | TVA (1986) |
| CO | Gas stove | 120.0 ppm (in kitchen, in use 20 min.) | Sterling and Sterling (1979) |
| Benzo(a) pyrene | Wood stove | 4.7 $ng/m^3$ | Moschandreas et al. (1980) |
| Benzo(a) pyrene | Fireplace | 11.4 $ng/m^3$ | Moschandreas et al. (1980) |
| HCHO | Mobile home (Complaint investigation) | 0.03-3 $mg/m^3$ | NRC (1981a) |
| Asbestos | Materials | 20-4500 $ng/m^3$ | Nicholson (1978) |
| Chlorpyrifos | Pesticide | 2.4 $ug/m^3$ | Lewis et al. (1986) |
| Diazinon | Pesticide | 1.4 $ug/m^3$ | Lewis et al. (1986) |

Table 8. continued...

| Pollutant | Source | Max. Concentration | Reference |
|---|---|---|---|
| Chlordane | Pesticide | 0.51 ug/$m^3$ | Lewis et al. (1986) |
| Propoxur | Pesticide | 0.042 ug/$m^3$ | Lewis et al. (1986) |
| Heptachlor | Pesticide | 0.088 ug/$m^3$ | Lewis et al. (1986) |
| Lead | Dust | 2200-3000 ug/g | Clark et al. (1985) |
| Radon | Soil gas | 500-2 million pCi/$m^3$ | U.S. EPA (1987) |
| RSP | ETS | 36.5 ug/$m^3$ (one smoker) | Spengler et al. (1981) |
| RSP | ETS | 70.4 ug/$m^3$ (two smokers) | Spengler et al. (1981) |
| Nicotine | ETS | 5-50 ug/$m^3$ | NRC (1986) |
| $NO_2$ | ETS | 21.3 ug/$m^3$ | Good et al. (1982) |
| Benzene | ETS | 12-48 ug/cigarette | NRC (1986) |
| HCHO | ETS | 70-100 ug/cigarette | NRC (1986) |
| B(a)P | ETS | 20-40 ug/cigarette | NRC (1986) |

Table 9. Estimated Number of Low Income Americans Affected by Indoor Air Pollution-Related Health Problems

| Pollutant Source | No. of People | Health Effect |
|---|---|---|
| Fireplace | 6.5 million | Exposure to carcinogenic B(a)P and soot, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Wood stove | 1.9 million | Exposure to carcinogenic B(a)P and soot, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Kerosene heater | 7.5 million | Doubling of airway resistance in asthmatics, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Gas space heater | 2.9 million | Pulmonary function changes, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Gas range | 14 million | Increased prevalence of respiratory illnesses in children. |
| Gas range for heating | 7.7 million | Decreased work capacity, neurological damage, exacerbation of angina symptoms. |
| Mobile home | 11 million | Mucous membrane irritation, asthma, chronic lung function reductions, long term lung damage. |

Table 9. continued...

| Pollutant Source | No. of People | Health Effect |
|---|---|---|
| Mobile home with kerosene heater | 5.8 million | Mucous membrane irritation, asthma, chronic lung function reductions, long term lung damage, doubling of airway resistance in asthmatics, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Home built before 1940 with wood or coal stove | 2 million | Potential exposure to lead, asbestos, and biological contaminants, exposure to carcinogenic B(a)P and soot, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Home built before 1970 | 24.5 million | Lung cancer, mesothelioma, asbestosis. |
| Home with high level of biologicals | 2 million | Nephrotoxicity, neurotoxicity, hepatoxicity, tuberculosis, respiratory diseases, Legionellosis. |
| Home with high rate of pesticide application | 8.7 million | Neurotoxicity. |
| Children with blood lead levels above 1.5 ug/dl | 2.4 million | Neurobehavioral and growth deficits. |
| Home in high traffic area with no A/C | 13.9 million | Exposure to carcinogenic benzene, formaldehyde, B(a)P, and PAHs, irritation, altered mucociliary transport, changes in alveolar macrophage activity. |
| Radon | 360,000 | Lung cancer. |
| Environmental tobacco smoke | 17.8 million | Increased respiratory disease occurrence and severity, lung cancer. |

older insulation and furnishings, and the presence of lead-based paints is made more serious if the home is in need of repair. Pesticide usage in public tenements is more frequent than in other types of housing, and the lack of air conditioners in homes in high traffic areas is a more common phenomenon for low socioeconomic status residents than for other residents.

Low socioeconomic status Americans are also at greater risk to have existing health problems, as pointed out by Mitchell and Dawson (1973), who found that more children of semi- and unskilled manual workers suffered from severe asthma than children from other social classes, and these children were frequently absent from school because of acute attacks or exacerbations of asthma. Platts-Mills (1988, personal communication) believes that inner cities represent a major risk factor because of increased levels of dust mite, cockroach, and rodent proteins which cause immediate hypersensitivity and asthma. The increased occurrence and severity of respiratory illnesses in children is an important factor to keep in mind when examining school absences. The neurological deficits which result from lead exposure may have a dramatic impact on the mental abilities of low socioeconomic status children.

Another very important factor which must be taken into consideration in viewing Table 9 is the exposure of hypersensitive populations. The elderly, children, and those people with preexisting health problems are particularly at risk to these pollutants, and may spend more time in the residence than average healthy individuals.

## VII. REVIEW OF EXISTING FEDERAL LAWS REGARDING INDOOR AIR POLLUTION

It has now been demonstrated that low socioeconomic status Americans are at greater risk to suffer the adverse health effects due to the presence of indoor air pollution than are middle and upper income Americans. Based on this conclusion, then, an evaluation will be made of the Federal government's ability to protect against these risks. This section will review the legislation which is intended to protect all Americans from the hazards of indoor air pollution, and the following section will evaluate the actual protection which will be provided to low income Americans.

There are several existing Federal laws which have been or may be used in controlling indoor air pollutants. This discussion will focus on these laws and the Federal Agencies to which they pertain, and will give examples of how these standards have been used in the past to control the indoor air pollutants which are of concern to low income Americans.

The Clean Air Act Amendments of 1977 are interpreted by the EPA as pertaining to the ambient environment. In the case of automotive pollutants, the reduction of lead in gasoline and the requirement for catalytic converters have reduced lead, particles, and CO emissions. This legislation has also been used to ban asbestos in certain situations (i.e., sprayed on insulation and troweled-on decorations). Under the Toxic Substances Control Act of 1976, EPA can prohibit or limit manu-

facture, processing, or distribution of any chemical substance and require manufacturers to list potentail health risks, provide instructions for use, or replace the substance. EPA can also require that manufacturers conduct health testing.

According to the Federal Insecticide, Fungicide, and Rodenticide Act, EPA can restrict the use and manufacture of pesticides. An example of this is the Agency's banning of the termiticides chlordane and heptachlor (Cohen, 1987). The major objectives of this Act are to protect public health from unreasonable pesticide risks, taking into consideration the economic and social costs and benefits (Bureau of National Affairs, 1982a). Actions might include warning labels on products, and manufacturers are required to test for acute and chronic effects in laboratory animals. The Safe Drinking Water Act enables the EPA to set maximum contaminant levels for bacteriological and microbiological quality which can affect the dissemination of these pollutants into indoor air. Interim Primary Drinking Water Regulations also include pesticide and radionuclide contaminants and have been in effect since 1977 (Bureau of National Affairs, 1982b).

The 1974 Consumer Product Safety Act allows the Consumer Product Safety Commission (CPSC) to regulate any product used in or around a home, and it can completely ban products deemed hazardous. An example of this is the CPSC's ban of artificial emberizing materials containing respirable free-form asbestos for use in fireplaces (CFR, 1988a), and their ban of asbestos-containing patching compounds (CFR, 1988b). Another example is the CPSC's attempt at banning urea formaldehyde foam which was overturned in the courts. The use of lead-based paint was also banned via this legislation.

As discussed previously, the Housing Act of 1949 is directed towards

the elimination of poor housing and is intended to provide a decent home and suitable living environment for Americans (U.S. DHEW, 1968). It is through this piece of legislation that housing codes are established.

The Office of Housing and Urban Development requires that each manufactured home have a health notice of formaldehyde emissions which informs people of the immediate irritating symptoms, lists the sensitive populations, and indicates that research is continuing to examine the long term health effects (CFR, 1987a). HUD also has established emission levels for plywood and particleboard materials installed in manufactured homes: 1) plywood material can't emit formaldehyde in excess of 0.2 ppm, and 2) particleboard materials can't emit formaldehyde in excess of 0.3 ppm (CFR, 1987b).

The Uranium Mill Tailings Radiation Control Act applies to uranium mill tailings, especially as they are used for landfill in residential areas or in the construction of dwellings.

The Federal Trade Commission ensures that products are labeled accurately and truthfully. They recently charged two manufacturers of air cleaners with falsely advertising their products. Other existing Federal legislation includes limits the radiation levels of homes built on uranium and phosphate mining sites, and $NO_x$ emission limitations on gas fired appliances.

The final piece of Federal legislation to be discussed here is the 1986 Radon Gas and Indoor Air Quality Research Act of the Superfund Amendment and Reauthorization Act (SARA) which calls for the establishment an indoor air quality research plan by all affected Federal agencies. Research is designed to 1) evaluate the health problems associated with indoor pollutants, 2) assess the appropriate Federal government actions, 3) identify, characterize, and monitor sources,

4) develop control technologies and design measures to prevent or avoid indoor air pollution, and 5) disseminate information to the public.

The Federal agencies have opted to concentrate on the information dissemination programs required under SARA. For the general public, the EPA has printed A Citizen's Guide to Radon, Removal of Radon From Household Water, Radon Reduction Methods, and Radon Reduction in New Construction. For technical groups the EPA has published Radon Reduction Techniques for Detached Houses and Radon/Radon Progeny Measurement Proficiency Program. The EPA has also put together several "Fact Sheets" dealing with indoor air pollution, and together with the Consumer Product Safety Commission published a brochure on asbestos.

# VIII. CONCLUSION

Except for the examples given in Section VII, almost all of the Federal agencies involved in some aspect of indoor air pollution have opted toward the information dissemination programs. The primary problem with these types of programs, especially with regards to low income Americans, is that they require: 1) some means for the Federal government to disseminate health effects and mitigation information to the low socioeconomic status Americans, 2) an understanding of the information by those receiving it, 3) an incentive among those receiving the information to protect themselves, and 4) the financial resources to correct the problems. Finally, these programs provide almost no protection for those people who live in non-owner occupied units.

Others have reviewed the options available for dealing with indoor air pollution problems, but not specifically in the protection of the low socioeconomic status people. Sexton (1986) lists the alternative government responses as: no action, more research, public information, economic incentives, moral persuasion, legal liability, guidelines, and rules and regulations. He indicates that the factors which will affect individual choices are the perceived costs and benefits and awareness of health risks. He also states that in private residences, government decisions aimed at improving personal decisions about indoor air quality may be preferable to rules and regulations (such as simple warning devices, product labeling, or information programs).

Splengler and Sexton (1983) state that "consideration of voluntary and nonvoluntary risks is important for policy decisions," and that policy alternatives such as economic incentives, better definition of legal rights and liabilities, public information programs, and expanded administrative efforts based on existing legislation might be more appropriate for control of indoor air pollution than a regulatory approach.

This paper has shown that emphasis should be placed on the economic incentives, enforcement of legal rights, and expanded use of existing legislation to protect the group of Americans that is most at risk yet least helped through information dissemination programs alone. In instances of non-owner occupied housing, the strict regulatory approach is necessary to initiate action. Housing codes, ventilation standards, close examination of pesticide usage and frequency, and strict enforcement programs are needed. Drummond and Mood (1973) evaluated the corrective responses of landlords in an inner city and found the success rate based on tenent complaints ranged from 48 to 74%.

The presence of some indoor air pollutants is solely the result of personal behavior. For these pollutants, such as ETS and combustion appliance emissions, behavior modification through information or incentive programs is the only means of government action. Sexton and Repetto (1982), however, note that many of the indoor air pollutants are present in amounts below perception thresholds, so individuals have no way of determining if their environment is healthful. They also state that reliance on private choice presumes unrealistic levels of information among the general public. In the case of consumer products, regulations are again needed to protect the unsuspecting public by banning the sale of hazardous products rather than simply providing

information on the labels stating the potential risks.

If indoor air pollution information dissemination programs are to result in "behavioral adjustments", then the low income Americans must receive and understand the importance of such information. For some pollutants, stricter building codes are needed (i.e., radon, formaldehyde). But because this population is often living in older homes with older appliances and furnishings, they must still be given information concerning their risks and financial assistance to replace leaking combustion appliances, mildewed furniture, peeling lead-based paint, and friable asbestos insulation, for example. The Federal government thus must be willing to expend greater effort and finances in order to alleviate the indoor air pollution problems faced by low socioeconomic status Americans than for middle and upper class Americans.

The objective of this paper was to review the existing information on indoor air pollution, examine the severity of the effects on low income groups in the U.S., and evaluate the existing Federal legislation's ability to protect this faction of Americans who are the most at risk due to the presence of indoor air pollutants in their dwellings. It has been demonstrated that enough information is now available to initiate public policy action to provide more assistance to this group.

The following table is a summary of the information presented in this paper of indoor air pollution sources and symptoms of the health problems due to their presence (Table 10). This check list can be used as an initial indication of a potential or existing indoor air problem in the residences of low socioeconomic status Americans. If social workers, housing inspectors, teachers, employers, and landlords make use of this summary, many indoor air pollution problems will be elucidated. This check list would be particularly useful to attending

physicians who are confronted with patients whose health problems may stem from indoor air pollution exposures.

TABLE 10. Check List Of The Presence Of Indoor Air Pollution Sources And Health-Related Problems

| Items to Check | Yes | No | Number | Good | Fair | Poor | Kerosene Heater | Woodstove | Gas Space Heater | Gas Range |
|---|---|---|---|---|---|---|---|---|---|---|
| Home built before 1940 | | | | | | | | | | |
| Home built 1940-1970 | | | | | | | | | | |
| Air conditioned | | | | | | | | | | |
| High traffic area | | | | | | | | | | |
| Public tenement | | | | | | | | | | |
| Owner occupied | | | | | | | | | | |
| No. of occupants | | | | | | | | | | |
| No. of children | | | | | | | | | | |
| No. of elderly | | | | | | | | | | |
| No. of infirm | | | | | | | | | | |
| Sign of health problems | | | | | | | | | | |
| Sign of biological contaminants | | | | | | | | | | |
| Sign of pesticide over-use | | | | | | | | | | |
| Smokers present | | | | | | | | | | |
| Condition of home | | | | | | | | | | |
| Condition of furnishings | | | | | | | | | | |
| Presence of combustion appliances | | | | | | | | | | |

## IX. BIBLIOGRAPHY


Agency for Toxic Substances and Disease Registry. (1988) The nature and extent of lead poisoning in children in the United States: A report to congress. Public Health Service, U. S. Department of Health and Human Services.

Ammann, H. A.; Berry, M. A.; Childs, N. E.; Mage, D. T. (1986) Health effects associated with indoor air pollutants. In: Proceedings of the ASHRAE conference IAQ '86: managing indoor air for health and energy conservation; April; Atlanta, GA. Atlanta, GA: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.; pp. 53-70.

Ammann, H. M. (1986) Problems of determining exposure to woodsmoke. In: Proceedings of the International Conference on Residential Wood Energy; March; Reno, NV. pp. 349-358

Ammann, H. M. (1987) Effects of indoor air pollution on sensitive populations. Clin. Ecol. 5: 15-21.

Andersen, I.; Korsgaard, J. (1986) Asthma and the indoor environment: assessment of the health implications of high indoor air humidity. In: Berglund, B.; Berglund, U.; Lindvall, T.; Spengler, J.; Sundell, J., eds. Indoor air quality: papers from the third international conference on indoor air quality and climate; August 1984; Stockholm, Sweden. Environ. Int. 12: 121-127.

Berteau, P. E.; Deen, W. A. (1978) A comparison of certain metabolically active drugs and oximes on tri-o-cresyl phosphate toxicity. Toxicol. Appl. Pharmocol. 7:227-235.

Bureau of National Affairs. (1982a) Environment Reporter, pp. 31-36.

Bureau of National Affairs. (1982b) Environment Reporter, pp. 25-26.

Burge, H. A. (1985) Indoor sources for airborne microbes. In: Gammage, R. B.; Kaye, S. V., eds. Indoor air and human health: proceedings of the seventh life sciences symposium; October 1984; Knoxville, TN. Chelsea, MI: Lewis Publishers, Inc.; pp. 139-148.

Burge, H. A. (1986) Toxigenic potential of indoor microbial aerosols. Presented at: Fifth symposium on the application of short-term bioassays in the analysis of complex environmental mixtures; October; Durham, NC.

Bylin, G.; Lindvall, T.; Rehn, T.; Sundin, B. (1985) Effects of short-term exposure to ambient nitrogen dioxide concentrations on human bronchial reactivity and lung function. Eur. J. Respir. Dis. 66: 205-217.

Calle, E. E.; Zeighami, E. A. (1985) Health risk assessment of residential wood combustion. In: Walsh, P. J.; Dudney, C. S.; Copenhaver, E. D., eds. Indoor air quality. Boca Raton, FL: CRC Press, Inc.; pp. 39-53.

Charney, E.; Kessler, B.; Farfel, M.; Jackson, D. (1983) Childhood lead poisoning: a controlled trial of the effect of dust-control measures on blood lead levels. N. Engl. J. Med. 309: 1089-1093.

Charney, E.; Sayre, J.; Coulter, M. (1980) Increased lead absorption in inner city children: where does the lead come from? Pediatrics 65: 226-231.

Chatterjee, P.; Gettman, J. H. (1972) Lead poisoning: subculture as a facilitating agent? Am. J. Clin. Nutr. 25:324-330.

Chisolm, J. J., Jr.; Mellits, E. D.; Quaskey, S. A. (1985) The relationship between the level of lead absorption in children and the age, type, and condition of housing. Environ. Res. 38: 31-45.

Clark, C. S.; Bornschein, R. L.; Succop, P.; Que Hee, S. S.; Hammond, P. B.; Peace, B. (1985) Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. In: Bornschein, R. L.; Rabinowitz, M. B., eds. The second int ernational conference on prospective studies of lead; April 1984; Cincinnati, OH. Environ. Res. 38: 46-53.

Code of Federal Regulations. (1987a) 3280.309 Health notice on formaldehyde emissions. 24 CFR Ch. XX pp. 214-215.

Code of Federal Regulations. (1987b) 3208.308 Formaldehyde emission controls for certain wood products. 24 CFR Ch. XX pp. 213-214.

Code of Federal Regulations. (1988a) Part 1305-Ban of artificial emberizing materials (ash and embers) containing respirable free-form asbestos. 16 CFR pp. 316.

Code of Federal Regulations. (1988b) Part 1304-Ban of consumer patching compounds containing respirable free-form asbestos. 16 CFR pp. 309-315.

Cohen, D. (1987) Note to correspondents (chlordane). Washington, DC: U. S. Environmental Protection Agency, Press Services Division.

Constant, P. C.; Bergman, F. J.; Atkinson, G. R.; Rose, D. R.; Watts, D. L.; Logue, E. E.; Hartwell, T. D.; Price, B. P.; Ogden, J. S. (1983) Airborne asbestos levels in schools. Washington, DC: U.S. Environmental Protection Agency, Office of Toxic Substances; EPA report no. 560/5-83-003. Available from NTIS, Springfield, VA; PB84-129683/REB.

Davies, D. J. A.; Watt, J. M.; Thornton, I. (1987) Air lead concentrations in Birmingham, England - a comparison between levels inside and outside inner-city homes. Environ. Geochem. Health 9: 3-7.

Davies, J. E.; Edmundson, W. F.; Raffonelli, A. (1975) The role of house dust in human DDT pollution. Am. J. Public Health 65: 53-57.

Dolan, M. C. (1985) Carbon monoxide poisoning. Can. Med. Assoc. J. 133: 392-399.

Drummond, D. W.; Mood, E. W. (1973) Actions of residents in response to environmental hazards in the inner city. Am. J. Public Health 63: 336-340.

Ekwo, E. E.; Weinberger, M. M.; Lachenbruch, P. A.; Huntley, W. H. (1983) Relationship of parental smoking and gas cooking to respiratory disease in children. Chest 84:662-668.

Energy Information Administration. (1986) The 1984 Residential Energy Consumption Survey. Office of Energy Markets and End Use, Form EIA-457.

Esmen, N. A. (1985) The status of indoor air pollution. EHP Environ. Health Perspect. 62: 259-265.

Fisk, W. J. et al. (1985) Indoor air quality control techniques: a critical review. LBL-16493; DOE/BP-00098-2.

GEOMET Technologies, Inc. (1976) The status of indoor air pollution research 1976. Research Triangle Park, NC: U. S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory; EPA report no. EPA-600/4-77-1029. Available from: NTIS, Springfield, VA; PB-272596/6.

Ginsberg, M. D. (1985) Carbon monoxide intoxication: clinical features, neuropathology and mechanisms of injury. J. Toxicol. Clin. Toxicol. 23: 281-288.

Girman, J. R.; Apte, M. G.; Traynor, G. W.; Allen, J. R.; Hollowell, C. D. (1982) Pollutant emission rates from indoor combustion appliances and sidestream cigarette smoke. Environ. Int. 8: 213-221.

Goldstein, B. D.; Melia, R. J. W.; Chinn, S.; du ve Florey, C.; Clark, D.; John, H. H. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. II. Factors affecting nitrogen dioxide levels in the home. Int. J. Epidemiol. 8: 339-345.

Good, B. W.; Vilcins, G.; Harvey, W. R.; Clabo, D. A., Jr.; Lewis, A. L. (1982) Effect of cigarette smoking on residential NO2 levels. Environ. Int. 8: 167-175.

Halpern, M. (1978) Indoor/outdoor air pollution exposure continuity relationships. J. Air Pollut. Control Assoc. 28: 689-691.

Hammond, P. B.; Bornschein, R. L.; Succop, P. A.; Clark, C. S. (1983) Profiles of lead burdens in early childhood. A longitudinal study in a high risk environment. In: International conference: heavy metals in the environment; September; Heidelberg, West Germany. Edinburgh, United Kingdom: CEP Consultants, Ltd.; pp. 262-268.

Hayes, A. L.; Wise, R. A.; Weir, F. W. (1980) Assessment of occupational exposure to organophosphates in pest control operators. Am. Ind. Hyg. Assoc. J. 41:568-575.

Hinkle, L. E., Jr.; Murray, S. H. (1981) The importance of the quality of indoor air. Bull. N. Y. Acad. Med. 57: 827-844.

Holmberg, B.; Ahlborg, U., eds. (1983) Consensus report: mutagenicity and carcinogenicity of car exhausts and coal combustion emissions. EHP Environ. Health Perspect. 47: 1-30.

Horstman, D.; Roger, L. J.; Kehrl, H.; Hazucha, M. (1986) Airway sensitivity of asthmatics to sulfur dioxide. Toxicol. Ind. Health 2: 289-298.

Inskip, M. J.; Hutton, M. (1987) Lead-based paint in dwellings: the potential for contamination of the home environment during renovation. Environ. Geochem. and Health 9:86-92.

Inskip, M.; Atterbury, N. (1983) The legacy of lead-based paint: potential hazards to 'do-it-yourself' enthusiasts and children. In: International conference: heavy metals in the environment; September; Heidelberg, West Germany. Edinburgh, United Kingdom; pp. 286-289.

Klus, H.; Begutter, H.; Ball, M.; Intorp, M. (1987) Environmental tobacco smoke in real life situations. In: Seifert, B.; Esdorn, H.; Fischer, M.; Rueden, H.; Wegner, J., eds. Indoor air '87: proceedings of the 4th international conference on indoor air quality and climate, v. 2, environmental tobacco smoke, multicomponent studies, radon, sick buildings, odours and irritants, hyperreactivities and allergies; August; Berlin, Federal Republic of Germany. Berlin, Federal Republic of Germany: Institute for Water, Soil and Air Hygiene; pp. 137-141.

Korsgaard, J. (1983) Mite asthma and residency: a case-control study on the impact of exposure to house dust mites in dwellings. Am. Rev. Respir. Dis. 128: 231.

Laxen, D. P. H.; Raab, G. M.; Fulton, M. (1987) Children's blood lead and exposure to lead in household dust and water--a basis for an environmental standard for lead in dust. Sci. Total Environ. 66: 235-244.

Leaderer, B. P.; Stolwijk, J. A. J.; Zagraniski, R. T.; Qing-Shan, M. (1984a) A field study of indoor air contaminant levels associated with unvented combustion sources. Presented at: 77th annual meeting of the Air Pollution Control Association; June; San Francisco, CA. Pittsburgh, PA: Air Pollution Control Association; paper no. 84-33.3.

Leaderer, B. P.; Zagraniski, R. T.; Holford, T. R.; Berwick, M.; Stolwijk, J. A. J. (1984b) Multivariate model for predicting NO2 levels in residences based upon sources and source use. In: Berglund, B.; Lindvall, T.; Sundell, J., eds. Indoor air: proceedings of the 3rd international conference on indoor air quality and climate, v. 4, chemical characterization and personal exposure; August; Stockholm, Sweden. Stockholm, Sweden: Swedish Council for Building Research; pp. 43-48. Available from: NTIS, Springfield, VA; PB85-104214.

Leaderer, B. P.; Zagraniski, R. T.; Berwick, M.; Stolwijk, J. A. J.; Qing-Shan, M. (1984c) Residential exposures to NO2, SO2 and HCHO associated with unvented kerosene space heaters, gas appliances and sidestream tobacco smoke. In: Berglund, B.; Lindvall, T.; Sundell, J., eds. Indoor air: proceedings of the 3rd international conference on indoor air quality and climate, v. 4, chemical characterization and personal exposure; August; Stockholm, Sweden. Stockholm, Sweden: Swedish Council for Building Research; pp. 151-156. Available from: NTIS, Springfield, VA; PB85-104214.

Lewis, R. G.; Bond, A. E.; Fitz-Simons, T. R.; Johnson, D. E.; Hsu, J. P. (1986) Monitoring for non-occupational exposure to pesticides in indoor and personal respiratory air. Presented at: 79th annual meeting of the Air Pollution Control Association; June; Minneapolis, MN. Pittsburgh, PA: Air Pollution Control Association; paper no. 86-37.4.

Lewis, R. G.; Bond, A. E. (1987) Non-occupational exposure to household pesticides. In: Seifert, B.; Esdorn, H.; Fischer, M.; Rueden, H.; Wegner, J., eds. Indoor air '87: proceedings of the 4th international conference on indoor air quality and climate, v. 1, volatile organic compounds, combustion gases, particles and fibres, microbiological agents; August; Berlin, Federal Republic of Germany. Berlin, Federal Republic of Germany: Institute for Water, Soil and Air Hygiene; pp. 195-199.

Lewis, R. G.; Bond, A. E.; Johnson, D. E.; Hsu, J. P. (1988) Measurement of atmospheric concentrations of common household pesticides: a pilot study. Environ. Monit. Assess. 10: 59-73.

Lewtas, J. (1982) Comparison of the mutagenic and potentially carcinogenic activity of particle bound organics from wood stoves, residential oil furnaces, and other combustion sources. In: Residential solid fuels; pp. 606-619.

Lewtas, J. (1985) Combustion emissions: characterization and comparison of their mutagenic and carcinogenic activity. In: Stich, H. F., ed. Carcinogens and mutagens in the environment, v. 5. Boca Raton, FL: CRC Press, Inc.; pp. 59-74.

Lewtas, J.; Williams, K.; Lofroth, G.; Hammond, K.; Leaderer, B. (1987) Environmental tobacco smoke: mutagenic emission rates and their relationship to other emission factors. In: Seifert, B.; Esdorn, H.; Fischer, M.; Rueden, H.; Wegner, J., eds. Indoor air '87: proceedings of the 4th international conference on indoor air quality and climate, v. 2, environmental tobacco smoke, multicomponent studies, radon, sick buildings, odours and irritants, hyperreactivities and allergies; August; Berlin, Federal Republic of Germany. Berlin, Federal Republic of Germany: Institute for Water, Soil and Air Hygiene; pp. 8-12.

Lin-Fu, J. S. (1982) Children and lead: new findings and concerns. N. Engl. J. Med. 307: 615-617.

Linn, W. S.; Shamoo, D. A.; Spier, C. E.; Valencia, L. M.; Anzar, U. T.; Venet, T. G.; Avol, E. L.; Hackney, J. D. (1985a) Controlled exposure of volunteers with chronic obstructive pulmonary disease to nitrogen dioxide. Arch. Environ. Health 40: 313-317.

Linn, W. S.; Solomon, J. C.; Trim, S. C.; Spier, C. E.; Shamoo, D. A.; Venet, T. G.; Avol, E. L.; Hackney, J. D. (1985b) Effects of exposure to 4 ppm nitrogen dioxide in healthy and asthmatic volunteers. Arch. Environ. Health 40: 234-239.

Louis, J. B.; Kisselbach, K. C., Jr. (1987) Background levels of chlordane and heptachlor found in indoor air of homes treated for termites. Presented at: 80th annual meeting of the Air Pollution Control Association; June, New York, NY. Pittsburgh, PA: Air Pollution Control Association; paper no. 87-84.3

Lucier, G. W.; Hook, G. E. R. (1984) Report on the consensus workshop on formaldehyde. EHP Environ. Health Perspect. 58: 323-381.

Lynam, D. R.; Hughmark, G. A.; Fort, B. F., Jr.; Hall, C. A. (1983) Blood lead concentrations and gasoline lead usage. In: International conference: heavy metals in the environment; September; Heidelberg, West Germany. Edinburgh,United Kingdom: CEP Consultants, Ltd.; pp. 417-420.

Mathison, D. A.; Stevenson, D. D.; Simon, R. A. (1982) Asthma and the home environment. Ann. Intern. Med. 97: 128-130.

Matthews, T. G.; Reed, T. J.; Tromberg, B. J.; Daffron, C. R.; Hawthorne, A. R. (1983) Formaldehyde emissions from combustion sources and solid formaldehyde resin containing products: potential impact on indoor formaldehyde concentrations and possible corrective measures. In: Proceedings of an Engineering Foundation conference on management of atmospheres in tightly enclosed spaces; October; Santa Barbara, CA. Atlanta, GA: American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc.

McCarthy, J.; Spengler, J.; Chang, B.-H.; Coultas, D.; Samet, J. (1987) A personal monitoring study to assess exposure to environmental tobacco smoke. In: Seifert, B.; Esdorn, H.; Fischer, M.; Rueden, H.; Wegner, J., eds. Indoor air '87: proceedings of the 4th international conference on indoor air quality and climate, v. 2, environmental tobacco smoke, multicomponent studies, radon, sick buildings, odours and irritants, hyperreactivities and allergies; August; Berlin, Federal Republic of Germany. Berlin, Federal Republic of Germany: Institute for Water, Soil and Air Hygiene; pp. 142-146.

McMichael, A. J.; Baghurst, P. A.; Wigg, N. R.; Vimpani, G. V.; Robertson, E. F.; Roberts, R. J. (1988) Port Pirie cohort study: environmental exposure to lead and children's abilities at the age of four years. New England J. Med. 319:468-475.

Melia, J.; Florey, C.; Sittampalam, Y.; Watkins, C. (1983) The relation between respiratory illness in infants and gas cooking in the UK: a preliminary report. In: Air quality VIth world congress: [proceedings of the International Union of Air Pollution Prevention Association]; May; Paris, France. Paris, France: SEPIC (APPA); pp. 263-269.

Melia, R. J. W.; du ve Florey, C.; Chinn, S. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. I. Results from a national survey. Int. J. Epidemiol. 8: 333-338.

Melia, R. J. W.; du ve Florey, C.; Morris, R. W.; Goldstein, B. D.; Clark, D.; John, H. H. (1982a) Childhood respiratory illness and the home environment. I. Relations between nitrogen dioxide, temperature and relative humidity. Int. J. Epidemiol. 11: 155-163.

Melia, R. J. W.; du ve Florey, C.; Morris, R. W.; Goldstein, B. D.; John, H. H.; Clark, D.; Craighead, I. B.; Mackinlay, J. C. (1982b) Childhood respiratory illness and the home environment. II. Association between respiratory illness and nitrogen dioxide, temperature and relative humidity. Int. J. Epidemiol. 11: 164-169.

Mitchell, R. G.; Dawson, B. (1973) Educational and social characteristics of children with asthma. Arch. Dis. Childhood 48: 467-471.

Moschandreas, D. J.; Relwani, S.; Johnson, D.; Billick, I. (1986) Emission rates from unvented gas appliances. In: Berglund, B.; Berglund, U.; Lindvall, T.; Spengler, J.; Sundell, J., eds. Indoor air quality: papers from the third international conference on indoor air quality and climate; August 1984; Stockholm, Sweden. Environ. Int. 12: 247-253.

Moschandreas, D. J.; Zabransky, J.; Rector, H. E. (1980) The effects of woodburning on the indoor residential air quility. Environ. Int. 4:463.

Mueller Associates, Inc. (1985) Indoor air quality environmental information handbook: combustion sources. Washington, DC; U. S. Department of Energy, Office of Environmental Analysis; contract no. DE-AC01-81EV10450.

Murphy, S. D. (1986) Toxic Effects of Pesticides. In: Klaassen, C. D.; Amdur, M. O.; Doull, D. Eds. Casarett and Doull's Toxicology. 3rd ed. New York, NY: Macmillan Publishing Company; pp. 519-581.

National Health Education Committee. (1976) The killers and cripplers: facts on major diseases in the United States today. New York, NY: McKay.

National Research Council. (1981a) Formaldehyde and other aldehydes. Washington, DC: National Academy Press.

National Research Council. (1981b) Indoor pollutants. Washington, DC: National Academy Press.

National Research Council. (1986) Environmental tobacco smoke: measuring exposures and assessing health effects. Washington, DC: National Academy Press.

Newacheck, P. W.; Starfield, B. (1988) Morbidity and use of ambulatory care services among poor and nonpoor children. Am. J. Public Health 78: 927-932.

Nicholson, W. J. (1978) Chrysotile asbestos in air samples collected in Puerto Rico: report to the Consumer Products Safety Commission. New York, NY: City University of New York, Mount Sinai School of Medicine.

Nicholson, W. J. (1986) Airborne asbestos health assessment update. Research Triangle Park, NC: U. S. Environmental Protection Agency, Environmental Criteria and Assessment Office; EPA report no. EPA/600/8-84/003F. Available from: NTIS, Springfield, VA.

Nicholson, W. J.; Rohl, A. N.; Sawyer, R. N.; Swoszowski, E. J.; Todaro, J. D. (1978) Control of sprayed asbestos surfaces in school buildings: a feasibility study. New York, NY: Mount Sinai School of Medicine, Environmental Sciences Laboratory.

Ogston, S. A.; Florey, C. du V.; Walker, C. H. M. (1985) The Tayside infant morbidity and mortality study: effect on health of using gas for cooking. Br. Med. J. 290:957-960.

Parkhurst, W. J.; Harper, J. P.; Spengler, J. D.; Fraumeni, L. P.; Majahad, A. M.; Cropp, J. W. (1988) Indoor nitrogen dioxide in five Chattanooga, Tennessee, public housing developments. Presented at the 81st Annual Meeting of the Air Pollution Control Association, Dallas, TX; June.

Pierrard, J. M.; Pfeifer, C. G.; Snee, R. D. (1983) Assessment of blood lead levels in the USA from NHANES II data. In: International conference: heavy metals in the environment; September; Heidelberg, West Germany. Edinburgh, United Kindom: CEP Consultants, Ltd.; pp. 421-424.

Platts-Mills, T. A. E.; Chapman, M. D. (1987) Dust mites: immunology, allergic disease, and environmental control. J. Allergy Clin. Immunol. 80: 755-775.

Platts-Mills, T. A. E.; Heymann, P. W.; Pollart, S. M.; Mitchell, E. B.; Hayden, M. L.; Chapman, M. D. (1987) Indoor allergens and acute asthma in adults. Presented at: 80th annual meeting of the Air Pollution Control Association; June; New York, NY. Pittsburgh, PA: Air Pollution Control Association; paper no. 87-86.3.

Quinn, M. J. (1983) Factors affecting blood lead concentrations in the UK. In: International Conference: Heavy Metals in the Environment, Volume 1; September; Heidelberg, West Germany. Edinburgh, United Kingdom: CEP Consultants, Ltd; 1983; pp. 294-297.

Repace, J. L. (1982) Indoor air pollution. Environ. Int. 8: 21-36.

Repace, J. L.; Lowrey, A. H. (1985) A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. Environ. Int. 11: 3-22.

Rose, K.; LaBelle, S.; Winter, R.; Klein, Y. (1985) Effects on minority and low-income households of the EPA proposal to reduce leaded gasoline use. Oak Ridge, TN: U. S. Department of Energy, Office of Minority Economic Impact, Research and Education Division; Report no. ANL/EES-TM-289.

Samfield, M. (1985) Indoor air particulate matter: a literature survey. Research Triangle Park, NC: U. S. Environmental Protection Agency; HATB report 85-1 [unpublished]; p. 3.

Sandia National Laboratories. (1982) Indoor air quality handbook for designers, builders, and users of energy efficient residences; report no. SAND82-1773.

Schenker, M. B.; Samet, J. M.; Speizer, F. E. (1983) Risk factors for childhood respiratory disease: the effect of host factors and home environmental exposures. Am. Rev. Respir. Dis. 128: 1038-1043.

Selikoff, I. J. (1981) Household risks with inorganic fibers. In: Symposium on health aspects of indoor air pollution; May; New York, NY. Bull. N. Y. Acad. Med. 57: 947-961.

Sexton, K. (1985) Indoor air quality: an overview of policy and regulatory issues. Presented at: 78th annual meeting of the Air Pollution Control Association; June; Detroit, MI. Pittsburgh, PA: Air Pollution Control Association; paper no. 85-46.1.

Sexton, K. (1986) Indoor air quality: an overview of policy and regulatory issues. Sci. Technol. Human Values 11(1): 53-67.

Sexton, K.; Repetto, R. (1982) Indoor air pollution and public policy. Environ. Int. 8: 5-10.

Sherrill, R. (1971) Why Uncle Sam is pulling back his dollars from healthful housing programs for the poor. Today's Health, July:17-19.

Solomon, W. R. (1975) Assessing fungus prevalence in domestic interiors. J. Allergy Clin. Immunol. 56:235-242.

Speizer, F. E.; Ferris, B.; Bishop, Y. M. M.; Spengler, J. (1980) Respiratory disease rates and pulmonary function in children associated with NO2 exposure. Am. Rev. Respir. Dis. 121:3-10.

Spengler, J. D.; Dockery, D. W.; Turner, W. A.; Wolfson, J. M.; Ferris, B. G. (1981) Long-term measurements of respirable sulfates and particles inside and outside homes. Atmos. Environ. 15:23-30.

Spengler, J. D.; Sexton, K. (1983) Indoor air pollution: a public health perspective. Science (Washington, DC) 221: 9-17.

Sterling, T. D.; Kobayashi, D. (1981) Use of gas ranges for cooking and heating in urban dwellings. J. Air Pollut. Control Assoc. 31:162-165.

Sterling, T. D.; Sterling, E. (1979) Carbon monoxide levels in kitchens and homes with gas cookers. J. Air Pollut. Control Assoc. 29: 238-241.

Surgeon General of the United States. (1986) The health consequences of involuntary smoking: a report of the Surgeon General. Rockville, MD: U. S. Department of Human Services, Office on Smoking and Health.

Tennessee Valley Authority. (1985) Indoor air quality study, phase II. Chattanooga, TN: Tennessee Valley Authority, Division of conservation and Energy Management.

Tennessee Valley Authority. (1986) TVA/BPA indoor air quality study, v. 1. Chattanooge, TN: Tennessee Valley Authority, Division of Conservation and Energy Management.

Ter Haar, G.; Aronow, R. (1974) New information on lead in dirt and dust as related to the childhood lead problem. EHP Environ. Health Perspect. 7: 83-89.

Tokiwa, H.; Nakagawa, R.; Horikawa, K. (1985) Mutagenic/carcinogenic agents in indoor pollutants: the dinitropyrenes generated by kerosene heaters and fuel gas and liquefied petroleum gas burners. Mutat. Res. 157: 39-47.

Tosteson, T. D.; Spengler, J. D.; Weker, R. A. (1982) Atomic absorption analysis in a personal monitoring study. Boston, MA: Harvard University School of Public Health.

Traynor, G. W.; Allen, J. R.; Apte, M. G.; Girman, J. R.; Hollowell, C. D. (1983) Pollutant emissions from portable kerosene-fired space heaters. Environ. Sci. Technol. 17: 369-371.

U. S. Bureau of the Census. (1985) Statistical Abstract of the United States 1986. 106th ed. U. S. Department of Commerce.

U. S. Department of Health, Education, and Welfare. (1968) Basic housing inspection. Public Health Service, Cincinnati, OH.

U. S. Department of Health, Education, and Welfare. (1970) "NEEDS" Neighborhood environmental evaluation and decision system. U. S. Department of Health, Education, and Welfare, Public Health Service, Bureau of Community Environmental Management.

U. S. Department of Health and Human Services. (1985) Fourth annual report on carcinogens. NTP 85-002.

U. S. Environmental Protection Agency. (1979) Air quality criteria for carbon monoxide. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-79-022. Available from: NTIS, Springfield, VA; PB81-244840.

U. S. Environmental Protection Agency. (1982a) Air quality criteria for oxides of nitrogen: final report. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-82-026F. Available from: NTIS, Springfield, VA; PB83-163337.

U. S. Environmental Protection Agency. (1982b) Air quality criteria for particulate matter and sulfur oxides: volume III. Research Triangle Park, NC: Environmental Critera and Assessment Office; EPA report no. EPA-600/8-82-029c. Available from: NTIS, Springfield, VA; PB84-120427.

U. S. Environmental Protection Agency. (1982c) Air quality criteria for particulate matter and sulfur oxides, volume I. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-82-029a. Available from: NTIS, Springfield, VA; PB84-120401.

U. S. Environmental Protection Agency. (1984a) Health and environmental effects profile for diazinon. Cincinnati, OH: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; draft no. ECAO-Cin-P074.

U. S. Environmental Protection Agency. (1984b) Revised evaluation of health effects associated with carbon monoxide exposure: an addendum to the 1979 air quality criteria document for carbon monoxide. Washington, DC: Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-83-033F. Available from: NTIS, Springfield, VA; PB85-103471.

U. S. Environmental Protection Agency. (1985) Costs and benefits of reducing lead in gasoline. Final regulatory impact analysis. Washington, DC: Office of Policy Analysis; EPA report no. EPA-230-05-85-006.

U. S. Environmental Protection Agency. (1986a) A citizen's guide to radon. What it is and what to do about it; report no. OPA-86-004.

U. S. Environmental Protection Agency. (1986b) Air quality criteria for lead: volume II of four. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-83/028bF.

U. S. Environmental Protection Agency. (1986c) Second addendum to air quality criteria for particulate matter and sulfur oxides (1982): assessment of newly available health effects information. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA-600/8-86-020A. Available from: NTIS, Springfield, VA; PB86-221249.

U. S. Environmental Protection Agency. (1987) EPA indoor air quality implementation plan: Appendix A. preliminary indoor air pollution information assessment; EPA report no. EPA/600/8-87/014.

U. S. Environmental Protection Agency (1988) Summary review of health effects associated with organic phosphorus compounds: health issue assessment. Research Triangle Park, NC: Environmental Criteria and Assessment Office; EPA report no. ECAO-R-199A (unpublished).

Urban America, Inc. (1975) The ill-housed. A compendium of recent writings and reports on national housing policy.

Walsh, P. J.; Dudney, C. S.; Copenhaver, E. D., eds. (1984) Indoor air quality. Boca Raton, FL: CRC Press, Inc.

Wang, H. H.; MacMahon, B. (1979) Mortality of workers employed in the manufacture of chlordane and heptachlor. J. Occup. Med. 21:745-748.

Williams, D. T.; Shewchuk, C.; Lebel, G. L.; Muir, N. (1987) Diazinon levels in indoor air after periodic application for insect control. Am. Ind. Hyg. Assoc. J. 48: 780-785.

World Health Organization. (1973) Chlorpyrifos. In: 1972 evaluations of some pesticide residues in food. Geneva, Switzerland: Food and Agriculture Organization of the United Nations; pp. 149-163.

World Health Organization. (1987) Improving environmental health conditions in low-income settlements. World Health Organization, Switzerland; report no. ISBN 92 4 170100 5.

Wyngaarden, J. B. (1988) Adverse effects of low-level lead exposure on infant development. JAMA J. Amer. Med. Assoc. 259:2524.