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Indoor Air Pollution: A Public Health Perspective

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Abstract

In developed and developing countries, indoor air pollution is gaining increasing prominence as a public health problem. Time-activity studies and exposure surveys have shown the dominant contributions of indoor environments to population exposures for many pollutants. Mounting epidemiological evidence documents adverse health effects of indoor pollutants and risk assessments indicate that indoor carcinogens may contribute substantially to the population's burden of lung and other cancers. Unacceptable indoor air quality has also been identified as a common cause of symptoms. This paper addresses the public health problem posed by indoor air pollution, offering a schema for categorizing adverse health effects of indoor air pollution, and considers the complexity of estimating the full scope of the problem.

KEY WORDS:
Air pollution – indoor, Air pollution/ae (adverse effects), Environmental health, Risk factors, Disease susceptibility

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Introduction

Although indoor environments have long been known to be associated with clinically evident adverse health effects, only recently has indoor air pollution received widespread recognition as a public health problem (National Research Council, 1981; Sigerist, 1943; Spengler and Sexton, 1983). Earlier concerns about specific clinical entities such as carbon monoxide poisoning and tuberculosis have been supplanted by a broader and unifying view of indoor air pollution as a single threat to public health. The emergence of this conceptualization of indoor air pollution as a single public health problem followed the reduction of outdoor air pollutant concentrations in many developed countries and the recognition of the dominant contributions of indoor exposures to total personal exposures for many pollutants (National Research Council, 1981; Spengler and Sexton, 1983). Beginning in the 1970s, evidence from epidemiological studies provided affirmation that indoor air pollutants cause adverse health effects (Samet and Spengler, 1991).

The continuance and growth of the International Conferences on Indoor Air Quality and Climate reflect the emergence of public health concern about indoor air pollution. These and similar conferences have facilitated interactions among the diverse disciplines involved in indoor air quality issues: engineers, architects, clinically-oriented physicians, epidemiologists, persons with expertise in exposure assessment and risk assessment, and persons from regulatory and public health agencies and from the many industries with interest in indoor air quality. In the 15 years that have passed from the First International Indoor Climate Symposium to the most recent conference, Indoor Air '93, there have been substantial advances in our knowledge of the adverse health effects of indoor air pollution. We are beginning to gain understanding of the broad array of health concerns associated with indoor air pollution. The progression of our knowledge can be readily demonstrated by a review of the proceedings of earlier conferences in this series. The 47 papers

at the first conference in 1978 included only a few titles suggesting a focus on health, with thermal comfort and odor receiving the greatest emphasis (Fanger and Valbjørn, 1979). By contrast, health was the pervasive theme in the hundreds of presentations at the Fifth International Conference held in Toronto in 1990 (Walkinshaw, 1990).

This overview highlights the now extensive literature on the adverse health effects of indoor air pollution and offers a classification scheme for these effects. My purpose is to systematize a seemingly endless listing of health effects that range from decreased comfort and irritation and possibly neurotic concerns about the quality of indoor air to death from asphyxiation. Initially, I consider concepts of health and adverse health effects that are fundamental to any classification system. Subsequently, a classification system and examples are provided. The scope is limited to the problem of indoor air pollution in developed countries. Smith and others have addressed the public health consequences of indoor air pollution in developing countries (Smith, 1987; Chen et al., 1990).

Concepts of Health and Adverse Health Effects

Concepts of health have been variable across history, changing in response to societal expectations and understanding of the nature and causes of disease (Last, 1983; Sigerist, 1943). The World Health Organization has described health as "a state of complete physical, mental, and social well-being, and not merely the absence of disease or infirmity". Last (1983) has defined health as "a state characterized by anatomic integrity, ability to perform personally valued family, work, and community roles; ability to deal with physical, biologic, and social stress; a feeling of well-being; and freedom from the risk of disease and untimely death". If the broad construct of health explicit in these definitions is accepted, then adverse effects of air pollution include not only clinically evident disease but more subtle symptomatic and physiological responses *and* compromise of well-being and an increased risk of disease.

Any definition of an "adverse health effect" is also made in the context of prevalent societal values. The need to define this context has been made clear in interpreting the language of the Clean Air Act in the United States. While the Clean Air Act uses the term "adverse health effect," it does not offer an explicit definition, and the boundary between "adverse" and "non-adverse" has been a subject of controversy. Both individual researchers (see Ferris, 1978 and Higgins, 1983 for example) and a committee of the American Thoracic Society (1985) have offered conceptual frameworks for defining adverse health effects. In a 1978 review of the health effects of exposure to regulated air pollutants, Ferris (1978) began by considering the definition of "health effects" and "how does one categorize adverse?" He acknowledged the difficulty of interpreting effects at low levels of exposure and the considerable judgmental element in any determination of adversity. In a 1983 paper, Higgins (1983) offered the following: "An adverse health effect may be defined as a biological change that reduces the level of well-being or functional capacity". This definition shares emphasis on well-being with the prevailing definitions of health.

A committee of the American Thoracic Society (1985) offered guidelines on what constitutes an adverse respiratory health effect in a 1985 report. The committee defined adverse respiratory health effects as "medically significant physiologic or pathologic changes generally evidenced by one or more of the following: 1. interference with the normal activity of the affected person or persons, 2. episodic respiratory illness, 3. incapacitating illness, 4. permanent respiratory injury, and/or 5. progressive respiratory dysfunction". The committee emphasized the interpretation of epidemiological data and implicitly focused on outdoor air pollution. The committee noted that all changes are not adverse and described a spectrum of response extending from pollution exposure through mortality. In this continuum, the boundary between adverse and non-adverse effects was placed between "physiologic changes of uncertain significance" and "pathophysiologic changes". An editorial commenting on these guidelines pointed to the potentially variable interpretation of the language and the inherent difficulty of establishing criteria for "medical significance" (Samet, 1985).

The U.S. Environmental Protection Agency has not directly defined an adverse health effect. In considering the evidence on the health effects of ozone in the 1989 Office of Air Quality Planning and Standards (OAQPS) Staff Paper, Agency staff acknowledged the need for a framework for categorizing lung function responses to ozone as either adverse or non-adverse (U.S. Environmental Protection Agency, 1989). Normal individuals exposed to

ozone during exercise respond with a reduction of lung function as assessed by spirometry and respiratory symptoms questionnaires. Responses placed into a "mild" category according to Agency criteria were not considered adverse. Recent studies of ozone toxicity in humans further illustrate the difficulty of classifying responses as adverse or non-adverse. Abnormalities indicative of injury in bronchoalveolar lavage fluid can be found in volunteer subjects exposed to ozone concentrations near the current U.S. National Primary Ambient Air Quality Standard (Devlin et al., 1991); does evidence of any response, i.e., a marker of injury, constitute an adverse health effect?

Increased risk for cancer (or other diseases) estimated by risk assessment methods has not been addressed in these definitions. The increased risks for cancer estimated for individuals exposed to carcinogens have no detectable correlates at present and the ultimate manifestation of the adverse health effect is discrete (i.e., the development of malignancy). We still lack biological markers indicative of a particular cancer being caused by a particular environmental agent, and there is no universally recognized level of increased risk that is deemed unacceptable. For example, at present within the U.S. Environmental Protection Agency, risk levels at which regulatory responses are invoked vary, and no effect has yet been made to establish a worldwide standard for the level of unacceptable risk. Furthermore, risk levels are dependent on the assumptions made in performing risk assessments, and changes in the assumptions can have a substantial impact on the risks assigned to carcinogens.

Varying susceptibility to indoor pollutants further complicates any schema for classifying adverse health effects. Responses to indoor air pollutants are not uniform within populations. The term "susceptible" has most often been applied to groups of people who share one or more characteristics that place them at increased risk compared to people without these characteristics. Even within a susceptible class of persons, a range of susceptibility can be assumed for many determinants of susceptibility and environmental agents. Many susceptibility factors are potentially relevant to indoor air pollution; for example: the underlying degree of airways responsiveness or the presence of asthma; the presence of cardiac or vascular disease; abnormal lung function and the presence of chronic obstructive pulmonary disease; the presence of atopy; and inherently increased risk for respiratory cancer.

In regulating outdoor air quality, the U.S. Environmental Protection Agency has explicitly attempted to set standards to protect the health of all susceptible groups within the population (except those requiring life-support systems) (Frank, 1988). Approaches to controlling indoor air pollution need to recognize the heterogeneity of populations. It may not be practicable to assure protection in indoor environments for the most susceptible persons. For example, some severe asthmatics may respond adversely to levels of biological pollutants or particulate matter commonly found in indoor environments. The recent emergence of persons who identify themselves as sensitive to contaminants in indoor environments merits consideration; regardless of underlying pathogenetic mechanism(s), persons with "multiple chemical sensitivity" are a growing and vocal group who are asking for uncontaminated air in indoor environments. Persons who are immunocompromised by acquired immune deficiency syndrome (AIDS) or other diseases, or from therapeutic agents, represent another growing susceptible population at risk for infections transmitted indoors. Guidelines for indoor air quality have acknowledged heterogeneity of response in regard to the perception of indoor air quality as "acceptable". Standards of both the American Society of Heating, Refrigerating and Air-Conditioning Engineers and of the Commission of the European Communities define indoor air quality as acceptable on the basis of acceptance by 80% of the members of a panel (ASHRAE, 1989; Commission of the European Communities, 1992).

Lacking a clear definition of an "adverse health effect", any schema for classifying adverse health effects must be based on an assumption of prevailing societal views and is, therefore, subject to review and criticism. Even the designation of selected groups within the population as susceptible requires such assumptions. Nevertheless, proposal of a classification scheme is a starting point for the move toward better public health protection.

A Classification of Adverse Health Effects of Indoor Air Pollution

Introduction

As with outdoor air pollution, a spectrum of health responses to indoor air pollution can be identified (Table 1). Table 1 provides a classification of these responses including categories for disease, impairment, symptoms, increased risk, and perceptions.

Table 1 A classification of the adverse effects of indoor air pollution.

Clinically evident diseases: Diseases for which the usual methods of clinical evaluation can establish a causal link to an indoor air pollutant.
Exacerbation of disease: The clinical status of already established disease is exacerbated by indoor air pollution.
Increased risk for diseases: Diseases for which epidemiological or other evidence establishes increased risk in exposed individuals. However, the usual clinical methods indicative of injury typically cannot establish the causal link in an individual patient.
Physiological impairment: Transient or persistent effects on a measure of physiological functioning which are of insufficient magnitude to cause clinical disease.
Symptom responses: Subjectively reported responses which can be linked to indoor pollutants or are attributed to indoor pollutants.
Perception of unacceptable indoor air quality: Sensing of indoor air quality as uncomfortable to an unacceptable degree.
Perception of exposure to indoor air pollutants: Awareness of exposure to one or more pollutants with an unacceptable level of concern about exposure.

Table 2 Selected examples of clinically evident disease linked to indoor air pollution.

Carbon monoxide poisoning
Hemorrhagic pneumonitis from high levels of NO_2
Hypersensitivity pneumonitis and humidifier fever
Legionella pneumonia
Cat- and mite-induced asthma

Each category is treated below and examples provided.

Clinically Evident Disease

While exposures to indoor air pollutants are universal, clinically evident cases of pollution-related disease appear to be relatively infrequent. In the case of such a clinically evident disease, a link can be established to an indoor pollutant by specific diagnostic tests (Table 2). For example, an appropriate clinical picture and an elevated serum precipitin titer are sufficient to document hypersensitivity pneumonitis due to thermophilic actinomycetes contaminating an air-conditioning system (Weissman and Schuyler, 1991). The level of carbon monoxide bound to hemoglobin (carboxyhemoglobin) provides a marker of exposure to concentrations of carbon monoxide associated with carbon monoxide poisoning. Skin tests and serologic tests can provide evidence of sensitization to antigens that produce disease through immediate hypersensitivity responses.

In classifying illnesses associated with public and commercial building environments, this category of adverse effects, e.g., hypersensitivity pneumonitis, has been referred to as specific building-related illnesses (American Thoracic Society, 1990; Marbury and Woods, 1991). However, the distinction between specific building-related illnesses and the non-specific syndrome referred to as sick (or tight) building syndrome rests on the capability of establishing a clinical diagnosis and this group of adverse effects is better recognized as unified on this basis. The occurrence of the entities included in this category is also not limited to commercial environments.

The public health burden posed by diseases in this category is potentially estimable by using hospital discharge and other medical care data bases along with population survey techniques. To date, however, estimates have not been published.

Exacerbation of Established Disease

Conditions that may be exacerbated by indoor air pollution are common in the population. Asthma, a chronic respiratory disease characterized by hyperresponsiveness of the lung's airways to environmental factors, affects approximately 5 to 10% of children and adults. Indoor exposures to animal danders, molds, and allergens from house dust mites and other insects may both cause and worsen the clinical status of persons with asthma. Environmental tobacco smoke may increase the non-specific responsiveness of the lung to environmental stimuli and even trigger attacks of asthma (Samet, 1991); the U.S. Environmental Protection Agency has recently estimated that exposure to environmental tobacco smoke exacerbates symptoms in about 20% of the 2 million to 5 million U.S. children with asthma and is a "major" exacerbating factor in approximately 10% (U.S. Environmental Protection Agency, 1992a).

Increased Risk for Disease

Many pollutants in indoor air are associated with increased risk for a variety of malignant and nonmalignant diseases (Table 3). The evidence supporting the relationships between exposures to these agents and increased risk comes from epidemiological studies, short-term exposures of volunteer subjects, animal studies, and *in vitro* toxicological studies. The population burden of disease attributable to such agents is often estimated using quantitative risk assessment, a technique for integrating information on the population pattern of exposure

Table 3 Selected examples of exposure-disease associations for indoor air pollutants.

Radon: Lung Cancer
Environmental Tobacco Smoke: Lung cancer, increased lower respiratory illness in infants
Benzene: Leukemia
Asbestos: Lung cancer and mesothelioma
Formaldehyde: Nasal cancer

Table 4 Prevalence of selected symptoms in office workers by type of ventilation system.*

Hazard identification: The determination of whether an agent is causally linked to the health effect of concern
Dose-response assessment: The determination of the relation between level of exposure and risk of the health effect
Exposure Assessment: Description of the extent of human exposure
Risk characterization: Description of the human risk, including uncertainties

* Source: Based on data from the National Research Council, 1983.

and the exposure-response relationship to obtain an estimate of the attributable number of disease cases or deaths associated with the exposure. The U.S. National Research Council (1983) has characterized risk assessment as a four-step process (Table 4). The results of risk assessment guide policy development and implementation in the separate activities of risk management. The risk assessment approach also supplies a framework for characterizing uncertainties in the risk estimates and identifying research needs. To date, risk assessment has been most widely applied to carcinogens, although the methodology could be applied to noncarcinogens as well.

The problem of indoor radon and lung cancer is illustrative. Radon, a ubiquitous contaminant of indoor air in homes, has been causally linked to lung cancer by epidemiological studies of underground miners and confirmatory animal investigations (Samet, 1989). To date, there is little evidence that lung cancers in radon-exposed miners differ from lung cancers in the general population, and lung cancers associated with radon cannot be separated directly from those caused by other factors; moreover, studies of underground miners suggest that cigarette smoking and radon exposure synergistically increase lung cancer risk (National Research Council, 1988). Thus, lung cancers in individuals cannot be designated as caused by radon or by cigarette smoking or another factor. In fact, the majority of radon-associated cases occur in cigarette smokers. However, risk assessment methods can be used to describe the population's increased risk of lung cancer from radon and even to apportion this risk between radon and the combined effect of radon and smoking (National Research Council, 1988; U.S. Environmental Protection Agency, 1992b). For radon, risk estimates are made by applying the exposure-response relationship observed in the miners to the distribution of exposures received by the general population (Samet, 1992). Adjustment can be made for physical differences between mines and residences and for physiological differences between the two circumstances of exposure (National Research Council, 1991). Key uncertainties include the extrapolation from higher exposures and exposure rates in mines to generally lower exposures and exposure rates in residences and the pattern of the combined effect of smoking and radon.

For some agents in this category, risk assessments provide estimates of the numbers of attributable cases of disease or death in selected populations. For example, the current distribution of indoor exposures to radon in the United States is estimated to cause approximately 14,000 lung cancer deaths annually (U.S. Environmental Protection Agency, 1992b). Exposure-response relationships can also be used to describe the risks associated with specific exposures and thereby project the risks for individuals. However, an individual case of lung cancer in the general population could not be attributed to indoor radon with a high degree of certainty; the best estimate of the probability of causation in an individual would be the attributable risk for a population of similar individuals. Risk assessments have also been published for environmental tobacco smoke and lung cancer (Repace and Lowrey, 1990).

Physiological Impairment

Exposures to indoor pollutants can impair physiological functioning, although not to a degree necessarily associated with disability or disease. For example, exposure to environmental tobacco smoke during childhood reduces the rate of lung growth and the maximum level of lung function achieved; the average estimated effect is not anticipated to be clinically detectable nor to be associated with reduced functional capacity (Samet and Spengler, 1991). Similarly, low levels of carbon monoxide exposure transiently impair oxygen delivery to tissues; however, the impact on exercise capacity is limited and likely to be manifest only during maximal activity (Coultas and Lambert, 1991). On the other hand,

reduced oxygen transport in the carbon-monoxide-exposed individual with coronary artery disease may increase the likelihood of clinically significant myocardial ischemia. The public health relevance of this category of adverse effects of indoor air pollution has received little consideration to date.

Symptom Responses

Epidemiological evidence links specific indoor air pollutants to a variety of symptoms. Environmental tobacco smoke exposure is associated with increased risk of respiratory symptoms in children (U.S. Department of Health and Human Services, 1986). The sick-building syndrome is a non-specific constellation of symptoms characteristically affecting multiple occupants of a building (American Thoracic Society, 1990). In some outbreaks of sick-building syndrome, specific pollutants are found to cause the symptoms, but in the majority only general etiological factors reflecting building operation and maintenance can be identified (American Thoracic Society, 1990; Samet et al., 1988).

Estimates of the burden of symptoms associated with indoor air pollutants have not been made; however, this burden is likely to be substantial because of the high prevalence rates of exposure to agents associated with symptoms. Surveys of the prevalence of work-related symptoms, although not conducted in random samples of buildings, indicate high prevalence rates for symptoms. Burge and coworkers (1987) described symptom rates in 4,373 office workers in 42 different buildings. Symptoms were considered work-related if they occurred more than twice during the previous 12 months and improved on days away from the office. Using this definition, the mean number of work-related symptoms varied across the sample of buildings from approximately 1.5 to 5. Symptoms of eye and upper airway irritation and headaches were common (Table 5).

Perception of Unacceptable Indoor Air Quality

The perception that indoor air quality is unacceptable should be considered as distinct from the symptoms caused by indoor air pollutants. To the extent that unacceptable indoor air quality reduces well-being, the perception of indoor air quality as unacceptable should be classified as an adverse health effect in the context of current concepts of health. Judgements as to the acceptability of indoor air quality presumably integrate multiple characteristics of the air, including the presence of odor and irritants, humidity, air movement, and temperature (Berglund and Lindvall, 1990; Spengler and Samet, 1991). Undoubtedly, there is a range of responses and expectations across the population. Physical and psychological aspects of the environment not directly related to indoor air quality may also influence judgments as to the acceptability of indoor air quality.

The findings of a nationwide survey of U.S. office workers suggest that dissatisfaction with the air quality in offices is common (Woods et al., 1987). Of 600 workers surveyed by telephone in 1984, 20% perceived that their work performance was affected "often" or "sometimes". Aspects of indoor air quality that were found to be "very serious" or "serious" by at least 50% of the affected respondents included lack of air movement (67%), being too hot in summer (61%), stagnant or still air (55%), cigarette smoke (54%), being too cold in winter (53%), and being too humid in summer (50%).

Perception of Exposure to Indoor Air Pollutants

The perception of exposure to indoor pollutants should also be regarded as an adverse health effect if the perception reduces well-being. The range of responses to the perception of exposure is broad, extending from annoyance because of an odor to the sometimes disabling symptom complex now frequently referred to as "multiple chemical sensi-

Table 5 Prevalence of selected symptoms in office workers by type of ventilation system.*

Ventilation type	Symptoms			
	Dry eyes	Blocked nose	Dry throat	Headache
Natural	18 (%)	40 (%)	36 (%)	39 (%)
Mechanical	20	32	33	33
Local induction/fan coil	34	58	56	52
Central induction/fan coil	31	57	54	47
All air	31	45	46	43
Whole group	27	47	46	43

* Source: Data from Table 5 in Burge et al., 1987.

tivity". While the pathogenetic mechanisms underlying multiple chemical sensitivity remain unknown and may be multiple, some cases are likely to represent somatization triggered by awareness of exposure to environmental pollutants (Miller, 1992). The numbers of persons who are adversely affected by the perception of exposure cannot presently be estimated.

The Public Health Burden of Indoor Air Pollution

This broad classification scheme makes clear the potential scope of the public health problem posed by indoor air pollution and the difficulty of fully estimating the magnitudes of adverse effects in each of the six categories (Table 1). Within this schema, some adverse effects have clear definitions (e.g., death from carbon monoxide poisoning) while others are defined on the basis of subjective responses, and any criteria for placing responses into a particular category require the assumption of a societal framework for separating adverse from non-adverse responses. Risk assessement has been used to quantify the hazard associated with some carcinogens of current concern and a few exposures associated with non-malignant respiratory effects. However, symptom and perceptual responses to indoor air pollutants can only be addressed by directly investigating exposed populations. To date, few studies have addressed these responses in population-based samples, and we thus lack any comprehensive and population-based assessment of the full scope of the public health consequences of indoor air pollution.

We also lack societal guidelines for describing the magnitude of the problem. In the United States, for example, public policy has evolved for single pollutants without broader consideration of more fundamental principles. Even for single agents, e.g., radon, conflicting views among involved regulators, the Congress, and scientists have led to persistent controversy (Cole, 1993). Without any firm guidelines, the scope of the problem of indoor air pollution can be readily manipulated, as underlying assumptions in risk assessments are varied. A process is needed for establishing a conceptual framework for indoor air pollution (Nero, 1993; Spengler and Samet, 1991).

Would the goal of public health protection be better achieved by more complete information on the health effects of indoor air pollution? My answer to this question is affirmative. The emergence of indoor air pollution as a unified public health concern has fostered the interdisciplinary interactions that are needed to find solutions. If the consequences of indoor pollution exposures are fragmented into a series of seemingly unrelated problems (e.g., combustion products, hypersensitivity pneumonitis, comfort, and radon), the imperative to achieve solutions through actions needing coordination of a wide range of professionals and organizations is diminished. More information on the public health burden posed by indoor air pollution is central to maintaining research on sources, exposures, health effects, and control measures.

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