Building-Related Illness

ICD-10 T75.8

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Workers with building-related symptoms generally have either nonspecific symptoms of unclear etiology or specific clinical diagnoses with objective findings. Sick building syndrome consists of nonspecific symptoms of headache, mucous membrane irritation, and difficulty concentrating that occur in close temporal association with being in an implicated building. In contrast, building-related illnesses, such as asthma and hypersensitivity pneumonitis, have symptoms that may be exacerbated by building occupancy, but generally persist away from the building. (See Table 1.)

In addition, building-related illnesses exist where symptoms have no temporal relation to building occupancy, but are due to building-related exposures. Examples are infectious diseases, such as Legionnaires' disease and tuberculosis, when epidemiologically linked with a building. There is some evidence that transmission of common communicable respiratory infections may be increased in relation to relatively low ventilation rates. The spector of bioterrorism from bacterial diseases, such as anthrax, and from viral diseases, such as smallpox, has increased interest in the role of ventilation and resuspension of particles from indoor surfaces in transmission of infections.

Rarely, specific toxic exposures lead to serious illness that is associated with a building environment. Examples include carbon monoxide poisoning from unvented combustion products and lung cancer from radon gas infiltrating from soil. However, public health authorities usually solve acute toxic problems rapidly, and unsuspected exposures rarely present to clinicians as building-related complaints, so these will not be considered further here.

Occurrence

The EPA has supported cross-sectional studies of occupants from 115 buildings without a history of publicized indoor air quality complaints, and found that more than 40% of workers experienced frequent work-related symptoms consistent with sick building syndrome. On the basis on this estimate, 35 to 60 million indoor workers in the United States are suspected to have at least one weekly building-related symptom. Prevalence rates in publicized "sick buildings" are sometimes higher. The proportion of the building stock in which occupants commonly have sick building syndrome (however defined) is unknown. No national surveillance system exists for nonspecific building-related symptoms. However, public health authorities at local, state, and federal levels commonly receive building-related complaints from office

Table 1. Building-Related Conditions

Condition	Etiologic Agent(s)	Basis of Diagnosis
Sick building syndrome	Unknown	Clinical history in setting of similar complaints by co-workers
Asthma	Bioaerosols associated with water-damaged building materials; latex antigen in health care settings	Clinical history, work-related airflow limitation or airways hyperresponsiveness
Hypersensitivity pneumonitis	Fungi contaminating HVAC systems and humidifiers; bioaerosols associated with water-damaged building materials	Clinical history, clinical responses to removal from exposure, epidemiologic pattern surrounding sentinel cases, objective tests for restrictive and granulomatous lung disease
Respiratory infection	Legionella species, *Mycobacterium tuberculosis*, respiratory viruses, anthrax	Culture, appropriate stains, serology
Carbon monoxide intoxication	Carbon monoxide from combustion sources, vehicle exhaust	Elevated carboxyhemoglobin level

workers and school personnel. Indoor air quality complaints constituted 37% of the requests for health hazard evaluations received by NIOSH in the 1999-2002 period.

Although no national data are available with which to estimate incidence or prevalence of building-related asthma, increasing proportions of occupational asthma are attributed to the nonindustrial built environment or indoor air quality. Building-related asthma accounted for 28% of all work-related asthma identified in the 1993-1999 period by four state health departments engaged in work-related asthma surveillance. AOEC reports an increase from about 6% of occupational asthma cases being building-related in 1991-1992 to 30% in 1995-1996. Finally, NIOSH has observed an increasing proportion of asthma health hazard evaluation (HHE) requests from the public in which the exposure listed in the request is an indoor air quality concern. None were reported in the 1970-1978 period, but the proportion increased relentlessly by subsequent 4-year intervals to 75% in 1999-2002. Among requests for HHEs for indoor air quality, however, only a minority mention asthma or chest symptoms. In the second half of the 1990s, only about 20% of indoor air quality requests mentioned asthma, which is consistent with the observation that respiratory symptoms are not typical of sick building syndrome alone. Collectively, these physician-diagnosis and public-request databases suggest that asthma in relation to the indoor environment is increasingly recognized. (See Asthma.)

In comparison to asthma, hypersensitivity pneumonitis is uncommon in the general population. Many occupational causes are known in diverse industries with bioaerosol exposure. A case of hypersensitivity pneumonitis linked to a nonindustrial work environment represents a sentinel event for risk among co-workers. Most well-documented outbreaks of hypersensitivity pneumonitis have been traced to microbially contaminated ventilation system components. However, current cases often occur in the setting of water-damaged buildings from roof leaks, other building envelope water intrusions, or below-grade moisture. As with building-related asthma, no surveillance data exist with which to estimate its prevalence or incidence. (See Hypersensitivity Pneumonitis.)

National incidence data are available for some infections that may be building-related. In 2002, a total of 1 163 cases of Legionella pneumonia in the United States were reported to the CDC, which estimated an incidence of 8 000 to 18 000 cases per year, most being sporadic. National surveillance data for tuberculosis exist, but nonoccupational transmission among family members and close contacts accounts for a large portion of tuberculosis. The proportion of all tuberculosis that could be considered to be occupational, for example, to health care workers and prison staff, is unknown. (See Tuberculosis.)

Recent work on sick leave among office workers, largely due to common communicable respiratory infections (colds and influenza), suggests that office ventilation with outdoor air has a substantial effect. Several studies have shown that some characteristic of a building or indoor environment has been associated with a 50% or greater difference in a metric of illness, such as rates of febrile respiratory disease requiring infirmary care. Since most workers suffer from a cold or influenza every year (0.58 cases per person per year in the working age population), even small effects of building characteristics on infection rates provide opportunities for prevention of sickness and for increasing productivity. Of the approximately 52 million cases of influenza or the common cold occurring annually among indoor workers in the United States, it has been estimated that 10% to 14% could probably be prevented with changes in indoor environments.

Causes

The cause of sick building syndrome remains unknown. Historically, many complaints were attributed to mass psychogenic illness. However, hysteria does not explain the tight temporal association with building occupancy, nature of the symptoms, lack of visual chain of transmission, and endemic nature of building-related complaints. European investigators showed two decades ago that the prevalence of building-related complaints was associated with building characteristics, such as air conditioning. Sick building syndrome came to public health attention in the late 1970s, following an energy crisis and change in consensus building ventilation standards that limited recommended outdoor air intake into mechanically ventilated buildings.

In the 1960s, building construction practices changed significantly. New construction emphasized the use of prefabricated exterior sections mounted on a steel frame, creating a much tighter building envelope. Windows were made inoperable, and mechanical ventilation, often under centralized control, replaced natural ventilation under individual control. Often, changing use of spaces after the original design leads to inadequate ventilation. Inadequate maintenance of building heating, ventilation, and air-conditioning (HVAC) systems may also threaten indoor air quality. At the same time that ventilation has decreased, the number and variety of potentially toxic agents in the office environment have increased. The sources are diverse (Table 2). Outdoor pollutants may enter through improperly placed ventilation intakes. Bacteria, fungi, and other biological agents may grow on moist surfaces in the building and in air conditioners, ducts, filters, and humidifiers. Building materials and furnishings may release formaldehyde and other volatile organic compounds; sources include building insulation, adhesives, tiles, vinyl wall coverings, rugs, carpets, copying machines, and furniture. Cleaning products and pesticides may also contaminate the air. Ozone from office machines may transform volatile organic chemicals indoors into short-lived irritants, such as aldehydes. Cigarette smoke is a significant source of noxious gases and respirable particles. Human bioeffluents, such as butyric acid and body odor, may also contribute to an unsatisfactory work environment.

Decades of research have pursued hypothetical causes of sick building syndrome, including volatile organic compounds, bioaerosols, and inadequate outdoor air ventilation, which limits dilution of indoor concentrations of suspected agents. Some evidence exists that outdoor air ventilation rates are related to sick building syndrome prevalence, particularly for ventilation rates below 30 cubic feet per person per minute. Since ventilation is difficult to measure, indoor air quality consultants commonly measure carbon dioxide levels in buildings with complaints. Carbon dioxide concentrations reflect ventilation effectiveness in relation to human occupancy (which is not likely the cause), and they are not predictive of sick building syndrome.

Although no measurement can assure occupants that they are not at risk of sick building syndrome, epidemiologic studies of occupants of buildings selected without regard to known indoor air quality complaints have contributed information about causes. The variation of complaint prevalence among buildings suggests remediable causes. Air conditioning is associated with higher building-related complaint prevalences than natural ventilation

Table 2. Common Indoor Pollutants and Their Sources in Commercial Buildings

Source	Pollutants
Tobacco smoke	Carbon monoxide, particles, organics
Gas boilers, furnaces, cookers	Carbon monoxide, nitrogen dioxide, particles, organics
People	Carbon dioxide, organics (bioeffluents, perfumes, fragances), bacteria, viruses
Standing water, water damage	Biological agents
Furnishings, building materials	Formaldehyde and other organics, fibers
Computers, copiers, correction fluids, typesetting equipment, cleaning agents	Organics, particles, ozone
Air from garages and loading docks	Carbon monoxide, particles, organics
Outdoor air	Carbon monoxide, nitrogen dioxide, ozone, sulfur dioxide, particles, organics, pollen, fungi
Soil gas	Radon, biocides, organics

or mechanical ventilation that does not alter air temperature or humidity. Studies of air quality acceptability have shown that the ventilation system itself may be the origin of poor indoor air quality. Other environmental correlates of sick building syndrome include carpeting, high occupancy, and video display terminal use. Personal factors associated with symptoms include female gender, job stress or dissatisfaction, and allergies. The perception of comfort may depend also on the movement of air, temperature, lighting, and humidity. Workers' responses to indoor air quality problems may also be affected by the quality of labor-management relations.

Building-related respiratory diseases of asthma and hypersensitivity pneumonitis are commonly associated with building dampness. Many epidemiologic studies have demonstrated an association of respiratory symptoms and asthma to residential dampness, and this work is now being extended to the nonindustrial work environment. The presumed causes are bioaerosols. The specific microbial cause is seldom evident in specific case patients or outbreaks. Rather, indices of bioaerosols, such as evident moisture, visible mold, moldy odor, and water stains, correlate better with respiratory disease risk than counts of viable colony forming units, for example. Most available bioaerosol measurements are limited in repeatability by short sampling times and wide fluctuations in concentration, making exposure classification suspect. In some outbreaks of hypersensitivity pneumonitis, the illness cannot be linked to a specific biological agent, but the affected people may have precipitating antibodies to extracts of the biological mixture contaminating an implicated humidifier or HVAC system. Research continues on more promising bioaerosol measurement techniques addressing biomass indices, such as endotoxin and fungal wall constituents, rather than culturable microorganisms.

Bacteria and viruses that are disseminated in the indoor environment can cause infections. In the case of bacteria, the source of amplification may be an environmental reservoir, as in Legionella growth in cooling towers or in hot water systems. Alternatively, the source of communicable infections, such as tuberculosis, is infected human building occupants. The marker for risk to some communicable respiratory infections may well be carbon dioxide concentration, which reflects the potential to rebreathe other occupants' exhaled breath. Viral and bacterial agents of infection vary in transmissibility by the airborne route.

Pathophysiology

Sick building syndrome mucous membrane symptoms reflect irritation of short duration. Efforts to establish pathophysiologic correlates of irritation have been meager. Objective evidence has been documented in tear film breakup time, which correlates with eye symptoms that are building-related. Some work has been initiated on nasal resistance measurements. The pathophysiologies of headache and difficulty concentrating in implicated buildings remain unexplored. However, no evidence exists that sick building syndrome leads to a serious or chronic condition that persists after employees leave an implicated building.

Asthma symptoms in relation to building occupancy reflect both exacerbation of pre-existing asthma and new-onset asthma. Exacerbation of asthma might reflect inflammation from irritant exposure or allergen-mediated inflammation, but the phenomenon has not been studied. Similarly, the mechanism of new-onset asthma with work-related patterns of symptoms and medication use has not been studied. Asthma is diagnosed by establishing the existence of bronchial hyperresponsiveness in the presence of asthma symptoms. Bronchial hyperresponsiveness is reflected by reversible airflow limitation, response to bronchodilator, or an abnormal methacholine bronchial provocation test.

Hypersensitivity pneumonitis is a lymphocyte-mediated immune system response to an inhaled antigen to which a patient has become sensitized. In acute hypersensitivity pneumonitis, symptom onset is delayed 4 to 12 hours after onset of exposure, characteristic of a cell-mediated reaction. Macrophages presenting antigen to sensitized lymphocytes participate in formation of giant cells characteristic of granulomatous inflammation in the lung. Lymphocytic alveolitis is characteristic of hypersensitivity pneumonitis, and can be demonstrated in bronchoalveolar lavage cell proportions. Unrecognized hypersensitivity pneumonitis can progress to permanent lung fibrosis and accompanying permanent impairment.

The pathophysiology of building-related infections is as diverse as the number of microbial agents and immune system responses to infection.

Prevention

Primary prevention of building-related complaints and illness requires implementing health protective practices that are known but often not implemented. These include attention to indoor air quality in architectural design and inspection of completed construction; choice of building materials and furnishings; control of pollution generated by occupant activities, cleaning products, and office machines; and design and maintenance of the HVAC system. Redesign of interior spaces needs to consider ventilation requirements for new occupants, the impact of modular office design, and the load of lights, computers, and equipment.

Building-related complaints and illnesses result when primary prevention has failed. The class of complaint should guide the investigation of building-related problems. Multidisciplinary "building diagnostics" teams, including physicians or epidemiologists, industrial hygienists, and ventilation engineers, can be an advantage in resolving the problems prompting health complaints.

Sick building syndrome does not include lower respiratory symptoms. Physicians can establish whether respiratory symptoms exist that may reflect asthma or hypersensitivity pneumonitis. These more serious medical conditions require attempts to identify the symptomatic workers for medical evaluation and consideration of work restriction from the implicated environment. Early recognition and diagnosis can provide a more favorable prognosis and even cure by removing the affected persons from continuing exposure to an environment in which there are antigens to which they react. The corollary environmental investigation should focus on identifying and remediating sources of bioaerosol amplification and dissemination. For at least hypersensitivity pneumonitis cases, remediation sometimes fails to prevent disease recurrence when affected workers return to the implicated environment.

Some infections, such as Legionella pneumonia, prompt examination of water reservoirs which disseminate aerosols. Cooling towers, whose mists sometimes are entrained in fresh air ventilation systems, are a classic source of Legionella organisms. Other infections, such as fungal infections in hospitalized immune-deficient patients, may arise in transplant units which have water-damaged building components that become sources of microbial amplification. Risk of transmissible infections, such as tuberculosis, may be influenced by negative pressure rooms, upper air ultraviolet irradiation, and room ventilation rate. Research is beginning on ways to control risk of common airborne respiratory infections that are not usually considered building-related, but may be influenced by the built environment.

Remediation of buildings with occupants having sick building syndrome is challenging because there is no environmental measure to establish its likelihood or to assure its resolution. The complaints are commonly contested or trivialized by management, resulting in workplace conflict and distrust. A multidisciplinary team can assist by (1) establishing the existence of complaints in a substantial portion of the workforce; (2) evaluating the heating, ventilation, and air-conditioning system design, performance, and maintenance; (3) checking the building envelope for integrity against water damage; and (4) reviewing housekeeping measures.

Further Reading

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Burn Injury

ICD-10 T20-T32

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Thermal burns are classified according to the depth of the wounds and the percent of the body surface area affected. First-degree burns, the least severe category, affect only the epidermis and are characterized by redness and swelling of the affected areas. Sunburns exemplify this depth of burning. Second-degree burns affect not only the epidermis but also the dermis and

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