CHAPTER 10

INDOOR ENVIRONMENTAL HEALTH

INDOOR environmental health comprises those aspects of human health and disease that are determined by factors in the indoor environment. It also refers to the theory and practice of assessing and controlling factors in the indoor environment that can potentially affect health. The practice of indoor environmental health requires consideration of chemical, biological, physical and ergonomic hazards.

It is essential for engineers to understand the fundamentals of indoor environmental health because the design, operation, and maintenance of buildings and their HVAC systems significantly affect the health of building occupants. In many cases, buildings and systems can be designed and operated to reduce the exposure of occupants to potential hazards. Unfortunately, neglecting to consider indoor environmental health can lead to conditions that create or worsen those hazards.

The first three sections of this chapter provide general background information, describe relevant health sciences, and introduce important concepts of hazard recognition, analysis and control. The remainder of the chapter is devoted to presenting information on specific hazards, and describes sources of exposure to each hazard, potential health effects, relevant exposure standards and guidelines, and methods to control exposure.

This chapter is introductory in nature, and indoor environmental health is a very broad and dynamic field. Thus, descriptions of potential hazards (and especially their controls) presented are not a comprehensive, state-of-the-art review. Additional detail is available on many important topics in other ASHRAE Handbook chapters, including

- Chapter 9, Thermal Comfort, of this volume
- Chapter 11, Air Contaminants, of this volume
- Chapter 12, Odors, of this volume
- Chapter 16, Ventilation and Infiltration, of this volume
- Chapter 28, Air Cleaners for Particulate Contaminants, of the 2008 *ASHRAE Handbook—HVAC Systems and Equipment*
- Chapter 29, Ventilation of the Industrial Environment, of the 2007 *ASHRAE Handbook—HVAC Applications*
- Chapter 45, Control of Gaseous Indoor Air Contaminants, of the 2007 *ASHRAE Handbook—HVAC Applications*

Other important sources of information from ASHRAE include the building ventilation and related requirements in *Standards* 62.1 and 62.2. Additional details are available from governmental and private sources, including the U.S. Center for Disease Control, U.S. Environmental Protection Agency, Occupational Safety and Health Administration, World Health Organization, American Conference of Governmental Industrial Hygienists, and National Institute for Occupational Safety and Health.

The preparation of this chapter is assigned to the Environmental Health Committee.

BACKGROUND

The most clearly defined area of indoor environmental health is occupational health, particularly as it pertains to workplace airborne contaminants. Evaluation of exposure incidents and laboratory studies with humans and animals have generated reasonable consensus on safe and unsafe workplace exposures for about 1000 chemicals and particles. Consequently, many countries regulate exposures of workers to these agents. However, chemical and dust contaminant concentrations that meet occupational health criteria usually exceed levels found acceptable to occupants in nonindustrial spaces such as offices, schools, and residences, where exposures often last longer and may involve mixtures of many contaminants and a less robust population (e.g., infants, the elderly, and the infirm) (NAS 1981).

Operational definitions of health, disease, and discomfort are controversial (Cain et al. 1995). However, the most generally accepted definition is that in the constitution of the World Health Organization (WHO): "Health is a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity."

Definitions of comfort also vary. Comfort encompasses perception of the environment (e.g., hot/cold, humid/dry, noisy/quiet, bright/dark) and a value rating of affective implications (e.g., too hot, too cold). Rohles et al. (1989) noted that acceptability may represent a more useful concept of evaluating occupant response, because it allows progression toward a concrete goal. Acceptability is the foundation of a number of standards covering thermal comfort and acoustics. Nevertheless, acceptability varies between climatic regions and cultures, and may change over time as expectations change.

Concern about the health effects associated with indoor air dates back several hundred years, and has increased dramatically in recent decades. This attention was partially the result of increased reporting by building occupants of complaints about poor health associated with exposure to indoor air. Since then, two types of diseases associated with exposure to indoor air have been identified: **sick building syndrome (SBS)** and **building-related illness (BRI)**.

SBS describes a number of adverse health symptoms related to occupancy in a "sick" building, including mucosal irritation, fatigue, headache, and, occasionally, lower respiratory symptoms and nausea. There is no widespread agreement on an operational definition of SBS. Some authors define it as acute discomfort (e.g., eye, nose, or throat irritation; sore throat; headache; fatigue; skin irritation; mild neurotoxic symptoms; nausea; building odors) that persists for more than two weeks at frequencies significantly greater than 20%; with a substantial percentage of complainants reporting almost immediate relief upon exiting the building.

The increased prevalence of health complaints among office workers is typical of sick building syndrome (Burge et al. 1987;

Table 1 Illnesses Related to Exposure in Buildings

Illness	Physical Examination	Laboratory Testing	Linkage	Causes/Exposures
Allergic rhinitis, Sinusitis	Stuffy/runny nose, post-nasal drip, pale or erythematous mucosa	Anterior and posterior rhinomanometry, acoustic rhinometry, nasal lavage, biopsy, rhinoscopy, RAST or skin prick testing	Immunologic skin prick or RAST testing, bracketed physiology	Mold, pollen, and dust mites are common examples
Building-related asthma	Coughing, wheezing, episodic dyspnea, wheezing on examination, chest tightness, temporal pattern at work	Spirometry before and after work at start of week, peak expiratory flow diary, methacholine challenge	Immunology testing: skin prick or RAST; physiology testing*	Pet dander, mold, environmental tobacco smoke, and dust mites are common examples
Organic dust toxic syndrome	Cough, dyspnea, chest tightness, feverishness	DLCO, TLC	Temporal pattern related to work	Gram-negative bacteria or endotoxin
Hypersensitivity pneumonitis	Cough, dyspnea, myalgia, weakness, rales, clubbing, feverishness	DLCO, FVC, TLC, CXR, lung biopsy	Immunology testing: IgG antibody to agents present, challenge testing, physiology testing (in acute forms): spirometry, DLCO	Causative agents include thermophilic actinomycetes; molds; mixed amoebae, fungi, and bacteria; avian proteins; certain metals and chemicals
Contact dermatitis	Dry skin, itching, scaling skin	Scaling, rash, eczema, biopsy	Patch testing; allergy testing	Range of microorganisms, chemicals
Urticaria (hives)	Multiple swollen raised itchy areas of skin	Inspection, biopsy	Provocation testing	Skin irritation, foods, heat/cold, direct pressure, sunlight, drugs
Eye irritation	Eye itching, irritation, dryness	Tear-film break-up time, conjunctival staining (fluorescein)	Temporal pattern	Low relative humidity, VOCs, and particulate matter are common examples
Nasal irritation	Stuffy, congested nose, rhinitis	Acoustic rhinometry, posterior and anterior rhinomanometry, nasal lavage, nasal biopsy	Temporal pattern	Low relative humidity, VOCs, and particulate matter are common examples
Central nervous system symptoms	Headache, fatigue, irritability, difficulty concentrating	Neuropsychological testing	Temporal pattern (epidemiology)	Organic compounds, noise, lighting, work stress, and carbon monoxide are common examples
Legionnaires' disease, Aspergillosis, *Pseudomonas* infection	Pneumonia, high fever, organ dysfunction	Environmental surveillance (water system monitoring), *Legionella pneumophila* identification from patient	Organism isolated from patient and source; immunology testing	*Legionella* (and other microorganism)-contaminated aerosols from water sources
Pontiac fever	Non-pneumonic flulike illness	Environmental surveillance (water system monitoring)		*Legionella*-contaminated aerosols from water sources

*(1) 10% decrement in FEV_1 across workday,
(2) peak flow changes suggestive of work relatedness
(3) methacholine reactivity resolving after six weeks away from exposure
RAST = radio allergen sorbent test
DLCO = single breath carbon monoxide diffusing capacity
FVC = forced vital capacity
TLC = total lung capacity
CXR = chest X-ray
IgG = class G immune globulins
FEV_1 = forced expiratory volume in the first second

Skov and Valbjorn 1987). Widespread occurrence of these symptoms has prompted the World Health Organization to classify SBS into several categories (Morey et al. 1984):

- Sensory irritation in the eyes, nose, or throat
- Skin irritation
- Neurotoxic symptoms
- Odor and taste complaints

Sick building syndrome is characterized by an absence of routine physical signs and clinical laboratory abnormalities. The term *nonspecific* is sometimes used to imply that the pattern of symptoms reported by afflicted building occupants is not consistent with that for a particular disease. Additional symptoms can include nosebleeds, chest tightness, and fever.

Some investigations have sought to correlate SBS symptoms with reduced neurological and physiological performance. In controlled studies, SBS symptoms can reduce performance in susceptible individuals (Mølhave et al. 1986).

Building-related illnesses, in contrast, have a known origin, may have a different set of symptoms, and are often accompanied by physical signs and abnormalities that can be clinically identified with laboratory measurements. For example, hypersensitivity illnesses, including hypersensitivity pneumonitis, humidifier fever, asthma, and allergic rhinitis, are caused by individual sensitization to bioaerosols.

Illnesses associated with exposure in indoor environments are listed in Table 1. Laboratory testing and development of linkages should be performed under direction of a qualified health care professional.

DESCRIPTIONS OF SELECTED HEALTH SCIENCES

The study of health effects in indoor environments includes a number of scientific disciplines. A few are briefly described here to further the engineer's understanding of which health sciences may be applicable to a given environmental health problem.

Epidemiology and Biostatistics

Epidemiology studies the cause, distribution, and control of disease in human and animal populations. It represents the application of quantitative methods to evaluate health-related events and effects. Epidemiology is traditionally subdivided into observational and analytical components; the focus may be descriptive, or may attempt to identify causal relationships. Some classical criteria for determining causal relationships in epidemiology are consistency, temporality, plausibility, specificity, strength of association, and dose/response.

Observational epidemiology studies are generally performed with a defined group of interest because of a specific exposure or

risk factor. A control group is selected on the basis of similar criteria, but without the exposure or risk factor present. A prospective study (cohort study) consists of observations of a specific group.

Examples of epidemiological investigations are cross-sectional, experimental, and case-control studies. Observations conducted at one point in time are considered cross-sectional studies. In experimental studies, individuals are selectively exposed to a specific agent or condition. These studies are performed with the consent of the participants unless the condition is part of the usual working condition and it is known to be harmless. Control groups must be observed in parallel. Case-control studies are conducted by identifying individuals with the condition of interest and comparing factors of interest in individuals without that condition.

Industrial Hygiene

Industrial hygiene is the science of anticipating, recognizing, evaluating, and controlling workplace conditions that may cause worker illness or injury. Important aspects of industrial hygiene include identifying toxic exposures and physical stressors, determining methods for collecting and analyzing contaminant samples, evaluating measurement results, and developing control measures. Industrial hygienists also create regulatory standards for the work environment, prepare programs to comply with regulations, and collaborate with epidemiologists in studies to document exposures and potential exposures to help determine occupation-related illness.

Microbiology and Mycology

Microbiology studies microorganisms, including bacteria, viruses, fungi, and parasites; mycology is a subspecialty that focuses on fungi. Environmental microbiologists and mycologists investigate the growth, activity, and effect of microorganisms found in nature, many of which can colonize and grow in buildings and building systems. Important aspects of environmental microbiology and mycology are identification of populations of contaminant microorganisms in buildings; determination of methods of collection of air, water, and surface samples; and evaluation of results of microbiological measurements. Effective, practical, and safe disinfection practices are usually developed and validated by microbiologists and mycologists.

Aerobiology is the study of airborne microorganisms or other biologically produced particles, and the effects of these aerosols (bioaerosols) on other living organisms. The section on Bioaerosols has more information on these contaminants.

Toxicology

Toxicology studies the influence of chemicals on health. All chemical substances may function as toxins, but low concentrations prevent many of them from being harmful. Defining which component of the structure of a chemical predicts the harmful effect is of fundamental importance in toxicology. A second issue is defining the dose/response relationships of a chemical and the exposed population. Dose may refer to delivered dose (exposure presented to the target tissue) or absorbed dose (the dose actually absorbed by the body and available for metabolism). Measures of exposure may be quite distinct from measures of effect because of internal dose modifiers (e.g., delayed metabolism of some toxins because of a lack of enzymes to transform or deactivate them). In addition, the mathematical characteristics of a dose may vary, depending on whether a peak dose, a geometric or arithmetic mean dose, or an integral under the dose curve is used.

Because permission to conduct exposure of human subjects in experimental conditions is difficult to obtain, most toxicological literature is based on animal studies. Isolated animal systems (e.g., homogenized rat livers, purified enzyme systems, or other isolated living tissues) are used to study the effects of chemicals, but extrapolation between dose level effects from animals to humans is problematic.

HAZARD RECOGNITION, ANALYSIS, AND CONTROL

Hazard recognition and analysis are conducted to determine the presence of hazardous materials or conditions as sources of potential problems. Research, inspection, and analysis determine how a particular hazard affects occupant health. Exposure assessment, an element of hazard recognition, relies on qualitative, semiquantitative, or quantitative approaches. In many situations, air sampling can determine whether a hazardous material is present. An appropriate sampling strategy must be used to ensure validity of collected samples, determining worst-case (for compliance) or usual (average) exposures. Air sampling can be conducted to determine **time-weighted average (TWA)** exposures, which cover a defined period of time, or **short-term exposures**, which determine the magnitude of exposures to materials that are acutely hazardous. Samples may be collected for a single substance or a multicomponent mixture. Hazard analysis also characterizes the potential skin absorption or ingestion hazards of an indoor environment. Analyses of bulk material samples and surface wipe samples are also used to determine whether hazardous conditions exist. Physical agent characterization may require direct-reading sampling methods. After collection and analysis, the results must be interpreted and an appropriate control strategy developed to control, reduce, or eliminate the hazard.

Hazards are generally grouped into one of the following four classes of environmental stressors:

- **Chemical hazards.** Routes of exposure to airborne chemicals are inhalation (aspiration), dermal (skin) contact, dermal absorption, and ingestion. The degree of risk from exposure depends on the nature and potency of the toxic effects, susceptibility of the person exposed, and magnitude and duration of exposure. Airborne contaminants are very important because of their ease of dispersal from sources and the risk of exposure through the lungs when they are inhaled. Airborne chemical hazards can be gaseous (vapors or gases) or particulate (e.g., dusts, fumes, mists, aerosols, fibers). For more information, see Chapter 11.
- **Biological hazards.** Bacteria, viruses, fungi, and other living or nonliving organisms that can cause acute and chronic illness in workers and building occupants are classified as biological hazards in indoor environments. Routes of exposure are inhalation, dermal (skin) contact, and ingestion. The degree of risk from exposure depends on the nature and potency of the biological hazard, susceptibility of the person exposed, and magnitude and duration of exposure.
- **Physical hazards.** These include excessive levels of ionizing and nonionizing electromagnetic radiation, noise, vibration, illumination, temperature, and force.
- **Ergonomic hazards.** Tasks that involve repetitive motions, require excessive force, or must be carried out in awkward postures can damage muscles, nerves, and joints.

Hazard Control

Strategies for controlling exposures in indoor environment are substitution (removal of the hazardous substance), isolation, disinfection, ventilation, and air cleaning. Not all measures may be applicable to all types of hazards, but all hazards can be controlled by using one of them. Personal protective equipment and engineering, work practice, and administrative controls are used to apply these methods. Source removal or substitution, customarily the most effective measure, is not always feasible. Engineering controls (e.g., ventilation, air cleaning) may be effective for a range of hazards. Local exhaust ventilation is more effective for controlling point-source contaminants than is general dilution ventilation, such as with a building HVAC system.

Hazard Analysis and Control Processes. The goal of hazard analysis and control processes is to prevent harm to people from

hazards associated with buildings. Quantitative hazard analysis and control processes are practical and cost-effective. Preventing disease from hazards requires facility managers and owners to answer three simple, site-specific questions:

- What is the hazard?
- How can it be prevented from harming people?
- How can it be verified that the hazard has been prevented from harming people?

Seven principles comprise effective hazard analysis and control:

- Use process flow diagrams to perform systematic hazard analysis
- Identify critical control points (process steps at which the hazard can be eliminated or prevented from harming people)
- Establish hazard control critical limits at each critical control point
- Establish a hazard control monitoring plan for critical limits at critical control points
- Establish hazard control corrective actions for each critical limit
- Establish procedures to document all activities and results
- Establish procedures to confirm that the plan (1) actually works under operating conditions (**validation**), (2) is being implemented properly (**verification**), and (3) is periodically reassessed

AIRBORNE CONTAMINANTS

Many of the same airborne contaminants cause problems in both industrial and nonindustrial indoor environments. These include nonbiological particles [e.g., synthetic vitreous fibers, asbestos, environmental tobacco smoke (ETS), combustion nuclei, nuisance dust], bioaerosols, and chemical gases and vapors. Airborne contaminants may be brought in from the outdoors or released indoors by industrial processes, building materials, furnishings, equipment, or occupant activities. In industrial environments, airborne contaminants are usually associated with the type of process that occurs in a specific setting, and exposures may be determined relatively easily by air sampling. Airborne contaminants in nonindustrial environments may result from emissions and/or shedding of building materials and systems; originate in outside air; or result from building operating and maintenance programs, procedures, or conditions. In general, compared to industrial settings, nonindustrial environments include many more contaminants that may contribute to health-related problems. These contaminants are usually present in lower concentrations and often are more difficult to identify. More information on contaminant types, characteristics, typical levels, and measurement methods is presented in Chapter 11.

PARTICLES

Particulate matter can be solid or liquid; typical examples include dust, smoke, fumes, and mists. Dusts are solid particles that range in size from 0.1 to 100 μm, whereas smoke particles are typically 0.25 μm and fumes are usually less than 0.1 μm in diameter (Zenz 1988). In contrast, mists are fine droplets of liquid in the air. Fibers are solid particles with length several times greater than their diameter, such as asbestos, manufactured mineral fibers, synthetic vitreous fibers, and refractory ceramic fibers. Bioaerosols of concern to human health range from 0.5 to 30 μm in diameter, but generally bacterial and fungal aerosols range from 2 to 8 μm in diameter because of agglomeration or rafting of cells or spores (Lighthart 1994).

Units of Measurement. The quantity of particles in the air is frequently reported as the mass or particle count in a given volume of air. Mass units are milligrams per cubic metre of air sampled (mg/m^3) or micrograms per cubic metre of air sampled ($\mu g/m^3$). For conversion, 1 mg/m^3 = 1000 $\mu g/m^3$. Mass units are widely used in industrial environments because these units are used to express occupational exposure limits.

Particle counts are usually expressed in volumes of 1 cubic foot, 1 litre, or 1 cubic metre and are specified for a given range of particle diameter. Particle count measurements are generally used in environments such as office buildings and industrial cleanrooms.

General Health Effects of Exposure. Health effects of airborne particulate matter depend on several factors, including particle dimension, durability, dose, and toxicity of materials in the particle. Respirable particles vary in size from <1 to 10 μm (Alpaugh and Hogan 1988). Methods for measuring airborne particles are discussed in Chapter 11. **Durability** (how long the particle can exist in the biological system before it dissolves or is transported from the system) and **dose** (amount of exposure encountered by the worker) both affect relative toxicity. In some instances, very low exposures can cause adverse health effects (hazardous exposures), and in others, seemingly high exposures may not cause any adverse health effects (nuisance exposures).

Safety and health professionals are primarily concerned with particles smaller than 2 μm. Particles larger than 8 to 10 μm in aerodynamic diameter are primarily separated and retained by the upper respiratory tract. Intermediate sizes are deposited mainly in the conducting airways of the lungs, from which they are rapidly cleared and swallowed or coughed out. About 50% or less of the particles in inhaled air settle in the respiratory tract. Submicron particles penetrate deeper into the lungs, but many do not deposit and are exhaled.

Industrial Environments

Exposures and Exposure Sources. In industrial environments, airborne particles are generated by work-related activities (e.g., adding batch ingredients for a manufacturing process, applying asphalt in a roofing operation, or drilling an ore deposit in preparation for blasting). The engineer must recognize sources of particle generation to appropriately address exposure concerns. Dusts are generated by handling, crushing, or grinding, and may become airborne during generation or during handling. Any industrial process that produces dust fine enough (about 10 μm) to remain in the air long enough to be inhaled or ingested should be regarded as potentially hazardous. In determining worker exposure, the nature of particles released by the activity, local air movement caused by makeup air and exhaust, and worker procedures should be assessed for a complete evaluation (Burton 2000).

Health Effects of Industrial Exposures. Pneumoconiosis is a fibrous hardening of the lungs caused by irritation from inhaling dust in industrial settings. The most commonly known pneumoconioses are asbestosis, silicosis, and coal worker's pneumoconiosis.

Asbestosis results from inhalation of asbestos fibers found in the work environment. The U.S. Department of Health and Human Services (ATSDR 2001) characterizes the toxicological and adverse health effects of asbestos and indicates that asbestos-induced respiratory disease can generally take 10 to 20 years to develop, although there is evidence that early cases of asbestosis can develop in five to six years when fiber concentrations are very high. Asbestos fibers cause fibrosis (scarring) of lung tissue, which clinically manifests itself as dyspnea (shortness of breath) and a nonproductive, irritating cough. Asbestos fiber is both dimensionally respirable and durable in the respiratory system.

Silicosis, probably the most common of all industrial occupational lung diseases, is caused by inhalation of silica dust. Workers with silicosis usually are asymptomatic, even in the early stages of massive fibrosis (Leathart 1972). It is not considered a problem in nonindustrial indoor environments.

Coal worker's pneumoconiosis (**CWP**, also known as "black lung") results from inhalation of dust generated in coal-mining operations. The dust is composed of a combination of carbon and varying percentages of silica (usually <10%) (Alpaugh and Hogan 1988). Because of the confined underground work environment, exposures can be very high at times, thus creating very high doses.

Table 2 OSHA Permissible Exposure Limits (PELs) for Particles (29CFR1910.1000, 29CFR1926.1101)

Substance	CAS* #	PEL
Cadmium	7440-43-9	0.05 mg/m^3
Manganese fume	7439-96-5	1.0 mg/m^3
Plaster of Paris	Nuisance	10.0 mg/m^3
Emery	Nuisance	10.0 mg/m^3
Grain dust	Nuisance	10.0 mg/m^3
Crystalline silica (as quartz)	14808-60-7	0.1 mg/m^3
Asbestos	1332-21-4	0.1 fibers/cm^3
Total dust	Nuisance	15.0 mg/m^3

*Chemical Abstract Survey

Data show that workers may develop CWP at exposures below the current dust standard of 1 mg/m^3).

Exposure Standards and Criteria. In the United States, the Occupational Safety and Health Administration (OSHA) has established permissible exposure limits (PELs) for many airborne particles. PELs are published in the Code of Federal Regulations (CFR 1989a, 1989b) under the authority of the Department of Labor. Table 2 lists PELs for several common workplace particles.

Exposure Control Strategies. Particulate or dust control strategies include source elimination or enclosure, local exhaust, general dilution ventilation, wetting, filtration, and use of personal protective devices such as respirators.

The most effective way to control exposures to particles is to totally eliminate them from the work environment. The best dust control method is total enclosure of the dust-producing process, with negative pressure maintained inside the entire enclosure by exhaust ventilation (Alpaugh and Hogan 1988).

Local exhaust ventilation as an exposure control strategy is most frequently used where particles are generated either in large volumes or with high velocities (e.g., lathe and grinding operations). High-velocity air movement captures the particles and removes them from the work environment.

General dilution ventilation in the work environment reduces particulate exposure. This type of ventilation is used when particulate sources are numerous and widely distributed over a large area. This strategy is often the least effective means of control, and may be very costly if conditioned (warm or cold) air is exhausted and unconditioned air is introduced without benefit of airside energy recovery. Ventilation and local exhaust for industrial environments are discussed more thoroughly in Chapters 29 and 30 of the 2007 *ASHRAE Handbook—HVAC Applications*.

Filtration can be an effective control strategy and may be less expensive than general ventilation, although increased pressure drop across a filter adds to fan horsepower requirements, and maintenance adds to system operating cost.

Using personal protective equipment (e.g., a respirator) is appropriate as a primary control during intermittent maintenance or cleaning activities when other controls are not feasible. Respirators can also supplement good engineering and work practice controls to increase employee protection and comfort (Alpaugh and Hogan 1988). Consultation with an industrial hygienist or other qualified health professional is needed to ensure proper selection, fit, and use of respirators.

Synthetic Vitreous Fibers

Exposures and Exposure Sources. Fibers are defined as slender, elongated structures with substantially parallel sides (as distinguished from a dust, which is more spherical). Synthetic vitreous fibers (SVFs) are inorganic fibrous materials such as glass wool, mineral wool (also known as rock and slag wool), textile glass fibers, and refractory ceramic fibers. These fibers are used primarily in thermal and acoustical insulation products, but are also used for filtration, fireproofing, and other applications. Human exposure to SVFs occurs mostly during manufacture, fabrication and installation, and demolition of those products, because the installed products do not result in airborne fiber levels that could produce significant consumer exposure. Simultaneous exposure to other dusts (e.g., asbestos during manufacture, demolition products and bioaerosols during demolition) is also important.

Health Effects of Exposure. Possible effects of SVFs on health include the following.

Cancer. In October 2001, an international review by the International Agency for Research on Cancer (IARC) reevaluated the 1988 IARC assessment of SVFs and insulation glass wool and rock wool. This resulted in a downgrading of the classification of these fibers from Group 2B (possible carcinogen) to Group 3 (not classifiable as to the carcinogenicity in humans). IARC noted specifically that "Epidemiologic studies published during the 15 years since the previous IARC Monographs review of these fibers in 1988 provide no evidence of increased risks of lung cancer or mesothelioma (cancer of the lining of the body cavities) from occupational exposures during manufacture of these materials, and inadequate evidence of any overall cancer risk." IARC retained the Group 2B classification for special-purpose glass fibers and refractory ceramic fibers, but its review indicated that many of the previous studies need to be updated and reevaluated, because they did not include the National Toxicology Program's Report on Carcinogens and the State of California's listing of substances known to cause cancer.

Dermatitis. SVFs may cause an irritant contact dermatitis with dermal contact and embedding in the skin, or local inflammation of the conjunctiva when fibers contact the eye. Resin binders sometimes used to tie fibers together have, on rare occasions, been associated with allergic contact dermatitis.

Exposure Standards and Criteria. OSHA has not adopted specific occupational exposure standards for SVFs. A voluntary workplace health and safety program has been established with fibrous glass and rock and slag wool insulation industries under OSHA oversight. This Health and Safety Partnership Program established an 8 h, time-weighted average permissible exposure limit of 1 fiber per cubic centimetre for respirable SVFs.

Exposure Control Strategies. As with other particles, SVF exposure control strategies include engineering controls, work practices, and use of personal protective devices. Appropriate intervention strategies focus on source control.

Combustion Nuclei

Exposures and Sources. Combustion products include water vapor, carbon dioxide, heat, oxides of carbon and nitrogen, and combustion nuclei. Combustion nuclei, defined in this chapter as particulate products of combustion, can be hazardous in many situations. They may contain potential carcinogens such as polycyclic aromatic hydrocarbons (PAHs).

Polycyclic aromatic compounds (PACs) are the nitrogen-, sulfur-, and oxygen-heterocyclic analogs of PAHs and other related PAH derivatives. Depending on their relative molecular mass and vapor pressure, PACs are distributed between vapor and particle phases. In general, combustion particles are smaller than mechanically generated dusts.

Typical sources of combustion nuclei are tobacco smoke, fossil-fuel-based heating devices (e.g., unvented space heaters and gas ranges), and flue gas from improperly vented gas- or oil-fired furnaces and wood-burning fireplaces or stoves. Infiltration of outdoor combustion contaminants can also be a significant source of these contaminants in indoor air. Therefore, combustion nuclei are important in both industrial and nonindustrial settings.

Exposure Standards and Criteria. OSHA established exposure limits for several of the carcinogens categorized as combustion nuclei [i.e., benzo(a)pyrene, cadmium, nickel, benzene, *n*-nitrosodimethylamine]. These limits are established for

industrial work environments and are not directly applicable to general indoor air situations. Underlying atherosclerotic heart disease may be exacerbated by carbon monoxide (CO) exposures.

Exposure Control Strategies. Exposure control strategies for combustion nuclei are similar in many ways to those for other particles. For combustion nuclei derived from space heating, air contamination can be avoided by proper installation and venting of equipment to ensure that these contaminants cannot enter the work or personal environment. Proper equipment maintenance is also essential to minimize exposures to combustion nuclei.

Particles in Nonindustrial Environments

Exposures and Sources. In the nonindustrial indoor environment, particle concentrations are greatly affected by the outdoor environment. Diesel engines emit large quantities of fine particulate matter. Indoor particle sources may include cleaning, resuspension of particles from carpets and other surfaces, construction and renovation debris, paper dust, deteriorated insulation, office equipment, and combustion processes (including cooking stoves, fires, and environmental tobacco smoke).

Although **asbestos** is commonly found in buildings constructed before the 1970s, it generally does not represent a respiratory hazard except to individuals who actively disturb it during maintenance and construction. School custodians, therefore, are recognized to be at risk for asbestos-related changes. Anderson et al. (1991) and Lilienfeld (1991) raise questions about risk to teachers.

An important source of particulates, **environmental tobacco smoke (ETS)** consists of exhaled mainstream smoke from the smoker and sidestream smoke emitted from the smoldering tobacco. Approximately 70 to 90% of ETS results from sidestream smoke, which has a chemical composition somewhat different from mainstream smoke. More than 4700 compounds have been identified in laboratory-based studies, including known human toxic and carcinogenic compounds such as carbon monoxide, ammonia, formaldehyde, nicotine, tobacco-specific nitrosamines, benzo(a)pyrene, benzene, cadmium, nickel, and aromatic amines. Many of these constituents are more concentrated in sidestream smoke than in mainstream smoke (Glantz and Parmley 1991). In studies conducted in residences and office buildings with tobacco smoking permitted, ETS was a substantial source of many gaseous and particulate PACs (Offermann et al. 1991).

Health Effects of Exposure. The health effects of exposure to combustion nuclei depend on many factors, including concentration, toxicity, and individual susceptibility or sensitivity to the particular substance. Combustion-generated PACs include many PAHs and nitro-PAHs that have been shown to be carcinogenic in animals (NAS 1983). Other PAHs are biologically active as tumor promoters and/or cocarcinogens. Mumford et al. (l987) reported high exposures to PAH and aza-arenes for a population in China with very high lung cancer rates.

According to the U.S. EPA (2005) fine particulate matter (particles less than 2.5 μm in diameter) is associated with lung disease, asthma, and other respiratory problems. Short-term exposure may cause shortness of breath, eye and lung irritation, nausea, lightheadedness, and possible allergy aggravations.

ETS has been shown to be causally associated with lung cancer in adults and respiratory infections, asthma exacerbations, middle ear effusion (DHHS 1986; NRC 1986), and low birth weight in children (Martin and Bracken 1986). The U.S. Environmental Protection Agency classifies ETS as a known human carcinogen (EPA 1992). Health effects can also include heart disease, headache, and irritation. ETS is also a cause of sensory irritation and annoyance (odors and eye irritation).

Exposure Standards. There are no established exposure guidelines for particles in nonindustrial indoor environments. The EPA National Ambient Air Quality standard (NAAQS) is 150 $\mu g/m^3$ for a 24 h average for particles smaller than 10 μm in diameter, and 35 $\mu g/m^3$ for a 24 h average for particles smaller than 2.5 μm in diameter.

Exposure Control Strategies. Particulate or dust control strategies for the nonindustrial environment include source elimination or reduction, good housekeeping, general dilution ventilation, and upgraded filtration. In general, source control is preferred. Combustion appliances must be properly vented and maintained. If a dust problem exists, identify the type of dust to develop an appropriate intervention strategy. Damp dusting and high-efficiency vacuum cleaners may be considered. Building spaces under construction or renovation should be properly isolated from occupied spaces to limit transport of dust and other contaminants. Minimizing idling of diesel-powered vehicles near buildings can reduce entry of fine particulate matter.

Control of ETS has been accomplished primarily through regulatory mandates on the practice of tobacco smoking indoors. Most U.S. states and E.U. member states have passed laws to control tobacco smoking in at least some public places, including public buildings, restaurants, and workplaces, and the FAA (2000) has prohibited smoking on all flights to and from the United States, as have many airlines throughout the world. Where tobacco smoking is permitted, appropriate local and general dilution ventilation can be used for control; however, the efficacy of ventilation is unproven (Repace 1984). Some studies indicate that extremely high ventilation rates may be needed to dilute secondhand smoke to minimal risk levels (Repace and Lowrey 1985, 1993). Although subsequently withdrawn (OSHA 2001), the Occupational Safety and Health Administration proposed (OSHA 1994) that tobacco smoke in indoor environments be controlled by using separately ventilated and exhausted smoking lounges, in which no work activities would occur concurrent with smoking. These lounges were to be kept under negative pressure relative to all adjacent and communicating indoor spaces, with smoking allowed only when the exhaust ventilation system was working properly.

Bioaerosols

Bioaerosols are airborne biological particles derived from viruses, bacteria, fungi, protozoa, algae, mites, plants, insects, and their by-products and cell mass components. Bioaerosols are present in both indoor and outdoor environments. For the indoor environment, locations that provide appropriate temperature and humidity conditions and a food source for biological growth may become problematic.

In microbiology, **reservoirs** allow microorganisms to survive, **amplifiers** allow them to proliferate, and **disseminators** effectively distribute bioaerosols. Building components and systems may have only one factor, or all three; for instance, a cooling tower is an ideal location for growth and dispersal of microbial contaminants and can be the reservoir, amplifier, and disseminator for *Legionella* (harboring microorganisms in scale, allowing them to proliferate, and generating an aerosol).

Both the physical and biological properties of bioaerosols need to be understood. For a microorganism to cause illness in building occupants, it must be transported in sufficient dose to a susceptible occupant. Airborne infectious particles behave physically in the same way as any other aerosol-containing particles with similar size, density, and electrostatic charge. The major difference is that bioaerosols may cause disease by several mechanisms (infection, allergic disease, toxicosis), depending on the organism, dose, and susceptibility of the exposed population. Although microorganisms exist normally in indoor environments, the presence of abundant moisture and nutrients in interior spaces results in the growth of fungi, bacteria, protozoa, algae, or even nematodes (Arnow et al. 1978; Morey and Jenkins 1989; Morey et al. 1986; Strindehag et al. 1988). Thus, humidifiers, water spray systems, and wet porous surfaces can be reservoirs and sites for growth. Excessive air moisture (Burge 1995) and floods (Hodgson et al. 1985) can also result in

proliferation of these microorganisms indoors. Turbulence associated with the start-up of air-handling unit plenums may also elevate concentrations of bacteria and fungi in occupied spaces (Buttner and Stetzenbach 1999; Yoshizawa et al. 1987).

Building Surface and Material Sources. Floors and floor coverings can be reservoirs for organisms that are subsequently resuspended into the air. Routine activity, including walking and vacuuming (Buttner et al. 2002), may even promote resuspension (Cox 1987). Some viruses may persist up to eight weeks on nonporous surfaces (Mbithi et al. 1991).

Building Water System Sources. Although potable water is usually delivered to buildings free of biological hazards, once the water enters the facility it becomes the responsibility of facility managers and owners to ensure that its microbial and chemical quality does not degrade. In fact, biological hazards associated with processes in building water systems cause considerable disease. Most cases of legionellosis, for example, result from exposure to potable water in buildings (McCoy 2005; WHO 2007).

Nonpotable water is a well-known source of infective agents, even by aerosolization. Baylor et al. (1977) demonstrated the sequestering of small particles by foam and their subsequent dispersal through bubble bursting. This dispersal may take place in surf, river sprays, or artificial sources such as whirlpools.

Building Occupant Sources. People are an important source of bacteria and viruses in indoor air. Infected humans can release virulent agents from skin lesions or disperse them by coughing, sneezing, or talking. Other means for direct release include sprays of saliva and respiratory secretions during dental and respiratory therapy procedures. Blood sprays during dental and surgical procedures are of potential concern for aerosol transmission of bloodborne diseases, including HIV and hepatitis. Large droplets can transmit infectious particles to those close to the disseminator, and smaller particles can remain airborne for short or very long distances (Moser et al. 1979).

Health Effects. The presence of microorganisms in indoor environments may cause infective and/or allergic building-related illnesses (Burge 1989; Morey and Feeley 1988). Some microorganisms under certain conditions may produce volatile chemicals (Hyppel 1984) that are malodorous. Microorganisms must remain viable to cause infection, although nonviable particles may promote an allergic disease, which is an immunological response. An organism that does not remain virulent in the airborne state cannot cause infection, regardless of how many units of organisms are deposited in the human respiratory tract. Virulence depends on factors such as relative humidity, temperature, oxygen, pollutants, ozone, and ultraviolet light (Burge 1995), each of which can affect survival and virulence differently for different microorganisms. Harmful chemicals produced by microorganisms can also cause irritant responses or toxicosis.

A wide variety of bacteria, fungi, and protozoa are prevalent in health care building water systems and can cause disease by transmission through water and air. Clinically important microorganisms known to cause disease in health care facilities include the bacteria *Legionella*, *Pseudomonas*, and *Mycobacteria*; the fungi *Aspergillus* and *Fusarium*; and the protozoa *Cryptosporidium*, *Giardia*, and *Acanthamoeba*.

Fungal Pathogens. Exposure to airborne fungal spores, hyphal fragments, or metabolites can cause respiratory problems ranging from allergic diseases (e.g., allergic rhinitis, asthma, hypersensitivity pneumonitis) to infectious diseases such as histoplasmosis, blastomycosis, and aspergillosis. In addition, acute toxicosis and cancer have been ascribed to respiratory exposure to mycotoxins (Levetin 1995). A large body of literature supports an association between moisture indicators in the home and symptoms of coughing and wheezing (Miller and Day 1997; Spengler et al. 1992).

Many fungal genera are widely distributed in nature and are common in the soil and on decaying vegetation, dust, and other organic debris (Levetin 1995). Fungi that have a filamentous structure are called **molds**, and reproduce by spores. Mold spores are small (2 to 10 μm in diameter), readily dispersed by water splash and air currents, and may remain airborne for long periods of time (Lighthart and Stetzenbach 1994; Streifel et al. 1989). *Aspergillus fumigatus* is one of the few molds that can cause infections in humans, and is the most frequent cause of **aspergillosis**, a lung infection that has been extensively researched. Aspergillosis in hospital patients has been caused by environmental sources, especially during renovation or nearby construction activity. **Histoplasmosis** is an infection caused by *Histoplasma capsulatum*, which has been reported to cause building-related illness among workers removing bat or bird droppings in abandoned buildings (Bartlett et al. 1982) or cleaning chicken coops. Presumably, asexual spores from this fungus were inhaled by workers who removed the droppings without adequate respiratory protection.

When moisture problems result in mold growth, building occupants may begin to report odors and a variety of health problems, such as headaches, breathing difficulties, skin irritation, allergic diseases, and aggravation of asthma symptoms, all of which may be associated with mold exposure. All molds have the potential to cause health effects. Molds produce allergens, irritants, and, in some cases, toxins that may cause reactions in humans. The types and severity of the symptoms depend, in part, on the types of mold present, extent of an individual's exposure, ages of exposed individuals, and their existing sensitivities or allergies (EPA 2001).

Symptoms of irritant responses and toxigenic reactions from exposure to molds in indoor environments range from mild to severe. Irritant reactions, including conjunctivitis, vasomotor or irritant rhinitis, rhinosinusitis, and asthma exacerbation, are poorly understood but are thought to be a nonspecific reaction to bioaerosol particles rather than a specific allergic response to a particular protein. The specific toxigenic effects of mycotoxins remain controversial, but are under investigation. More than 300 toxins are produced by molds (mycotoxins); *Stachybotrys chartarum* is often cited as a toxigenic mold, but all fungal genera have the potential to produce chemicals that could be harmful to humans. Ingestion of contaminated grain is the most common route of exposure to mycotoxins, but inhalation and dermal contact have also resulted in toxicoses in building occupants and agricultural workers. Mycotoxins are associated with actively growing colonies and spores; they are not gaseous, unlike microbial volatile organic compounds (MVOCs). MVOCs are natural by-products of microbial metabolism produced by actively growing organisms, and they have been alleged to cause headache, nausea, and malaise in building occupants.

Bacterial Pathogens. Diseases produced by the bacterial genus ***Legionella*** are collectively called legionelloses. More than 45 species have been identified, with over 20 isolated from both environmental and clinical sources. Conditions favorable for *Legionellae* growth include water temperatures of 77 to 108°F; stagnant conditions; presence of scale, sediment, and biofilms; and the presence of amoebas (Geary 2000). Diseases produced by *Legionella pneumophila* include Legionnaires' disease (pneumonia form) and Pontiac fever (flulike form). *L. pneumophila* serogroup 1 is the most frequently isolated from nature and most frequently associated with disease, but characteristics of the exposed individual (e.g., tobacco smoking, excessive weight, age) and viability of the bacterium affect the virulence. Legionellosis is not rare, but it is rarely diagnosed, and is severely underreported, often lost among other causes of pneumonia. McCoy (2006) estimated that, every day in the United States, an average of about 11 people die from legionellosis, and another 57 are infected but recover, often with lifelong debilitation.

In a review of waterborne infections from building water systems, it was estimated that 1400 deaths occur each year in the United States from *Pseudomonas aeruginosa*, another waterborne bacteria commonly found in building water systems (Anaissie et al. 2002).

Viral Pathogens. Outbreaks of infection in indoor air may also be caused by **viruses**. Viruses are readily disseminated from infected

individuals, but cannot reproduce outside a host cell. Therefore, they do not reproduce in building structures or air-handling components, but can be distributed throughout buildings through duct systems and on air currents. Human-to-human dispersal is common. In one example, most of the passengers in an airline cabin developed influenza following exposure to one acutely ill person (Moser et al. 1979). In this case, the plane had been parked on a runway for several hours with the ventilation system turned off. Severe acute respiratory syndrome (SARS), caused by a corona virus similar to the common cold, was assumed to result from large droplet transmission; however, in an outbreak in a high-rise apartment, airborne transmission was the primary mode of disease spread, likely through dissemination from a bathroom drain (Yu et al. 2004).

Infectious diseases are transmitted through three primary routes: (1) direct contact and fomites (i.e., inanimate objects that transport infectious organisms from one individual to another), (2) large droplets [generally with a mass median aerodynamic diameter (MMAD) > 10 µm], and (3) fine particles, sometimes called *droplet nuclei* (MMAD < 10 µm) (Mandell et al. 1999). Additional transmission routes, such as through blood transfusions, intravenous injections, or injuries, are not of concern here. Table 3 lists infections considered transmissible by air.

Nonviable Biological Substances. **Allergic reactions** are an immunological response to foreign protein. Allergies may develop after dermal contact or inhalation of particles containing microorganisms, microbial fragments or by-products, and other biological components (e.g., enzymes, mite and cockroach excreta, pet dander). Cases of allergic respiratory illness (e.g., humidifier fever, hypersensitivity pneumonitis) manifest acute symptoms such as malaise, fever, chills, shortness of breath, and coughing (Edwards 1980; Morey 1988). In buildings, these illnesses may occur as a response to microbiological contaminants from HVAC system components, such as humidifiers and water spray systems, or other mechanical components that have been damaged by chronic water exposure (Hodgson et al. 1985, 1987). The severity of immunological reactions to bioaerosols can vary dramatically, from discomfort (allergic rhinitis and sinusitis) to life-threatening asthma. Allergy testing may be helpful in identifying an offending agent, but often is not. In cases of more severe illness, it may be necessary to remove an affected individual from exposure, even after appropriate abatement and exposure control methods have been instituted within the building.

Crandall and Sieber (1996) demonstrated that 47 of 104 problem buildings evaluated had water damage in occupied areas. Other studies concluded that unusual populations and high concentrations of microorganisms in indoor air may increase occupants' health complaints (Brundage et al. 1988; Burge 1995; Burge et al. 1987).

Exposure Guidelines for Bioaerosols. At present, numerical guidelines for bioaerosol exposure in indoor environments are not available for the following reasons (Morey 1990):

- Incomplete data on background concentrations and types of microorganisms indoors, especially as affected by geographical, seasonal, and building parameters
- Incomplete understanding of and ability to measure routes of exposure, internal dose, and intermediate and ultimate clinical effects
- Absence of epidemiological data relating bioaerosol exposure indoors to illness
- Enormous variability in types of microbial particles, including viable cells, dead spores, toxins, antigens, MVOCs, and viruses
- Large variation in human susceptibility to microbial particles, making estimates of health risk difficult

However, even without numerical guidelines, bioaerosol sampling data can be interpreted based on factors such as

- Rank order assessment of the kinds (genera/species) of microorganisms present in complainant and control locations (ACGIH 1999)
- Medical or laboratory evidence that illness is caused by a microorganism (ACGIH 1999)
- Indoor/outdoor concentration ratios for various microbial agents (ACGIH 1999; Morey and Jenkins 1989)

Table 3 Diseases Spread by Droplet or Airborne Transmission

Disease	Organism	Clinical Manifestations
Adenovirus	Adenovirus	Rhinitis, pharyngitis, malaise, rash, cough
Influenza*	Influenza virus	Fever, chills, malaise, headache cough, coryza, myalgias
Measles (rubeola)*	Rubeola virus	Fever, rash, malaise, coryza, conjunctivitis, Koplik's spots, adenopathy, CNS complications
Meningococcal disease	*Neisseria meningitides*	Fever, headache, vomiting, confusion, convulsions, petechial rash, neck stiffness
Mumps*	Mumps virus	Painful/swollen salivary glands, orchitis, meningoencephalitis
Pertussis (whooping cough)	*Bordetella pertussis*	Malaise, cough, coryza, lymphocytosis
Parvovirus B19	Parvovirus B19	Rash, aplastic anemia, arthritis, myalgias
Respiratory syncytial virus	RSV	Often asymptomatic; respiratory symptoms
Rubella	Rubella virus	Fever, malaise, coryza, rash
Tuberculosis*	*Mycobacterium* species	Fever, weight loss, fatigue, night sweats, pulmonary disease, extra pulmonary involvement including lymphatic, genitourinary, bone, meningeal, peritoneal, miliary
Varicella	Human herpes virus 3	Chickenpox or zoster presentation

Source: Adapted from Russi et al. (2008).
*Airborne transmission is reasonably certain, although it may not be primary mode.

Exposure Control Strategies. Because of the wide variety of pathogens and sources, a range of bioaerosol exposure control strategies may be required. Typically, these strategies should focus on source control (including good housekeeping and proper HVAC system operation and maintenance), but dilution ventilation, local exhaust ventilation, disinfection procedures, space pressure control, and filtration may also be considered.

Moisture control is the key to mold control. Molds need both food and water to survive; because molds can digest most things, water is the key factor that limits mold growth. Molds often grow in damp or wet areas indoors, including bathroom tiles, basement walls, areas around windows where moisture condenses, and near leaky water fountains or sinks. Uncontrolled humidity can also be a source of moisture leading to mold growth, particularly in hot, humid climates. For this reason, manage indoor relative humidity to minimize dew-point conditions that can result in moisture accumulation in building materials and contents (EPA 2001). More detailed information may be found in Harriman et al. (2001) and ASHRAE's (2003) *Mold and Moisture Management in Buildings*.

ASHRAE *Guideline* 12 provides environmental and operational guidance for safe operation of building water systems to minimize the risk of Legionnaires' disease. It is in the process of being upgraded to a standard by ASHRAE special project committee SPC 188.

GASEOUS CONTAMINANTS

Gaseous contaminants include both true gases (which have boiling points less than room temperature) and vapors of liquids with boiling points above normal indoor temperatures. It also includes both volatile organic compounds and inorganic air contaminants.

Volatile organic compounds (VOCs) include 4- to 16-carbon alkanes, chlorinated hydrocarbons, alcohols, aldehydes, ketones, esters, terpenes, ethers, aromatic hydrocarbons (such as benzene and toluene), and heterocyclic hydrocarbons. Also included are chlorofluorocarbons (CFCs) and hydrochlorofluorocarbons (HCFCs), which are still commonly used as refrigerants in existing installations, although production and importation have been phased out for environmental protection (Calm and Domanski 2004). More information on classifications, characteristics, and measurement methods can be found in Chapter 11.

Inorganic gaseous air contaminants include ammonia, nitrogen oxides, ozone, sulfur dioxide, carbon monoxide, and carbon dioxide. Although the last two contain carbon, they are by tradition regarded as inorganic chemicals.

The most common units of measurement for gaseous contaminants are parts per million by volume (ppm) and milligrams per cubic metre (mg/m^3). For smaller quantities, parts per billion (ppb) and micrograms per cubic metre (µg/m^3) are used. The relationship between these units of measure is also described in Chapter 11.

Industrial Environments

Exposures and Sources. In the industrial environment, a wide variety of gaseous contaminants may be emitted as process by-products (e.g., paints, solvents, and welding fumes) or as accidental spills and releases.

Health Effects of Industrial Exposures. Given that tens of thousands of contaminants are regularly used by industry, possible health effects can range from mild skin or eye irritation and headaches, to failure of major organs or systems and death. Exposure standards and specific health effects for various industrial contaminants are discussed in the following section.

Exposure Standards. The U.S. Occupational Safety and Health Administration (OSHA) sets permissible exposure limits (PELs) for toxic and hazardous substances, which are enforceable workplace regulatory standards. These are published yearly in the *Code of Federal Regulations* (29CFR1910, Subpart Z) and intermittently in the *Federal Register*. Most of the regulatory levels were derived from those recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) and American National Standards Institute (ANSI). The health effects on which these standards were based can be found in their publications. ACGIH reviews data on a regular basis and publishes annual revisions to their Threshold Limit Values (TLVs®).

The National Institute for Occupational Safety and Health (NIOSH), a research agency of the U.S. Department of Health and Human Service, conducts research and makes recommendations to prevent work-related illness and injury. NIOSH publishes the *Registry of Toxic Effects and Chemical Substances* (RTECS), as well as numerous criteria on recommended standards for occupational exposures. Some compounds not listed by OSHA are covered by NIOSH, and their recommended exposure limits (RELs) are sometimes lower than the legal requirements set by OSHA. The NIOSH *Pocket Guide to Chemical Hazards* (NIOSH 1997) condenses these references and is a convenient reference for engineering purposes.

The harmful effects of gaseous pollutants depend on both short-term peak concentrations and the time-integrated exposures received by the person. OSHA defined three periods for concentration averaging and assigned allowable levels that may exist in these categories in workplaces for over 490 compounds, mostly gaseous contaminants. Abbreviations for concentrations for the three averaging periods are

AMP = acceptable maximum peak (for a short exposure)

ACC = acceptable ceiling concentration (not to be exceeded during an 8 h shift, except for periods where an AMP applies)

TWA8 = time-weighted average (not to be exceeded in any 8 h shift of a 40 h week)

The respective levels are presented in Tables Z-1, Z-2 and Z-3 of 29CFR1910.1000, *Occupational safety and health standards: Air contaminants*.

In non-OSHA literature, the AMP is sometimes called a short-term exposure limit (STEL), and a TWA8 is sometimes called a threshold limit value (TLV). NIOSH (1997) also lists values for the toxic limit that is immediately dangerous to life and health (IDLH).

Standards differ for industrial and nonindustrial environments (EHD 1987). A Canadian National Task Force developed guideline criteria for residential indoor environments, and the World Health Organization (WHO) published indoor air quality guidelines for Europe (WHO 2000). Table 4 compares these guidelines with occupational criteria for selected contaminants.

The National Primary Drinking Water Standards (EPA 2003) are legally enforceable standards that apply to public water systems. Primary standards protect public health by limiting the levels of contaminants in drinking water.

Exposure Control Strategies. Gaseous contaminant control strategies include eliminating or reducing sources, local exhaust, general dilution ventilation, and using personal protective devices such as respirators. The most effective control strategy is source control. If source control is not possible, local exhaust ventilation can often be the most cost-effective method of controlling airborne contaminants. General dilution ventilation is often the least effective means of control. Ventilation and local exhaust for industrial environments are discussed more thoroughly in Chapters 29 and 30 of the 2007 *ASHRAE Handbook—HVAC Applications*.

Nonindustrial Environments

Gaseous contaminants of concern in nonindustrial environments include volatile organic compounds, refrigerants, and inorganic gases.

Volatile Organic Compounds.

Sources. Indoor sources of VOCs include building materials, furnishings, cleaning products, office and HVAC equipment, ETS, people and their personal care products, and outdoor air. The California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA 2008) maintains a list of VOCs and other chemicals known to the state to cause cancer or reproductive toxicity.

Health Effects. Potential adverse health effects of VOCs in nonindustrial indoor environments are not well understood, but may include (1) irritant effects, including perception of unpleasant odors, mucous membrane irritation, and exacerbation of asthma; (2) systemic effects, such as fatigue and difficulty concentrating; and (3) toxic, chronic effects, such as carcinogenicity (Girman 1989).

Chronic adverse health effects from VOC exposure are of concern because some VOCs commonly found in indoor air are human (benzene) or animal (chloroform, trichloroethylene, carbon tetrachloride, p-dichlorobenzene) carcinogens. Some other VOCs are also genotoxic. Theoretical risk assessment studies suggest that risk from chronic VOC exposures in residential indoor air is greater than that associated with exposure to VOCs in the outdoor air or in drinking water (McCann et al. 1987; Tancrede et al. 1987).

A biological model for acute human response to low levels of VOCs indoors is based on three mechanisms: sensory perception of the environment, weak inflammatory reactions, and environmental stress reaction (Mølhave 1991). A growing body of literature summarizes measurement techniques for the effects of VOCs on nasal (Koren 1990; Koren et al. 1992; Meggs 1994; Mølhave et al. 1993; Ohm et al. 1992) and ocular (Franck et al. 1993; Kjaergaard 1992; Kjaergaard et al. 1991) mucosa. It is not well known how different sensory receptions to VOCs are combined into perceived comfort and the sensation of air quality. This perception is apparently related

Table 4 Comparison of Indoor Environment Standards and Guidelines

	Canadian[c]	WHO/Europe	NAAQS/EPA[f]	NIOSH REL (TWA)[h]	OSHA (TWA)[h]	ACGIH (TWA)[h]	MAK[g] (TWA)[h]
Acrolein	0.02 ppm[a]			0.1 ppm 0.3 ppm (15 min)	0.1 ppm	C 0.1 ppm, A4	
Acetaldehyde	5.0 ppm			Ca: ALARA[b]	200 ppm	C 25 ppm	50 ppm 100 ppm (5 min)
Formaldehyde	0.1 ppm (1 h) 0.04 ppm (8 h)	0.081 ppm (30 min)		0.016 ppm 0.1 ppm (15 min) Ca	0.75 ppm 2 ppm (15 min) Ca	C 0.3 ppm, A2	0.3 ppm 1.0 ppm (5 min)
Carbon dioxide	3500 ppm			5000 ppm 30,000 ppm (15 min)	5000 ppm	5000 ppm 30,000 ppm (15 min)	5000 ppm 10,000 ppm (60 min)
Carbon monoxide	11 ppm (8 h) 25 ppm (1 h)	8.6 ppm (8 h) 25 ppm (1 h) 51 ppm (30 min) 86 ppm (15 min)	9 ppm (8 h) 35 ppm (1 h)	35 ppm C 200 ppm	50 ppm	25 ppm	30 ppm 60 ppm (30 min)
Nitrogen dioxide	0.05 ppm 0.25 ppm (1 h)	0.02 ppm (1 yr) 0.1 ppm (1 h)	0.053 ppm (1 yr)	1 ppm (15 min)	C 5 ppm	3 ppm 5 ppm (15 min), A4	5 ppm 10 ppm (5 min)
Ozone	0.12 ppm (1 h); Insufficient data for long-term level	0.06 ppm (8 h)	0.12 ppm (1 h) 0.085 ppm (8 h)	C 0.1 ppm	0.1 ppm	0.05 ppm, A4 (for heavy work) 0.2 ppm (2 h) (light, moderate, or heavy work)	
Particles <2.5 MMAD[d]	40 μg/m^3 (8 h) 100 μg/m^3 (1 h)		15 μg/m^3 (1 yr) 35 μg/m^3 (24 h)		5 mg/m^3 (respirable fraction)	3 mg/m^3 (8 h) (no asbestos, <1% crystalline silica, with median cut point of 4.0 μm)	1.5 mg/m^3 (for less than 4 μm)
Sulfur dioxide	0.019 ppm 0.38 ppm (5 min)	0.047 ppm (24 h) 0.019 ppm (1 yr)	0.03 ppm (1 yr) 0.14 ppm (24 h)	2 ppm (8 h) 5 ppm (15 min)	5 ppm	2 ppm 5 ppm (15 min)	0.5 ppm 1.0 ppm (5 min)
Radon	800 Bq/m^3[e]		4 pCi/1				

( ) Numbers in parentheses represent averaging periods
C = ceiling limit
Ca = carcinogen
A4 = not classifiable as human carcinogen per ACGIH

[a]Parts per million (10^6)
[b]As low as reasonably achievable
[c]Health Canada *Exposure Guidelines for Residential Indoor Air Quality*
[d]Mass median aerodynamic diameter
[e]Mean in normal living areas
[f]U.S. EPA National Ambient Air Quality Standards
[g]German Maximale Arbeitsplatz Konzentrationen
[h]Value for 8-h TWA, unless otherwise noted
[i]WHO Air Quality Guidelines for Europe

to stimulation of the olfactory sense in the nasal cavity, the gustatory sense on the tongue, and the common chemical sense (Cain 1989; Mølhave 1991).

Cometto-Muñiz and Cain (1994a, 1994b) addressed the independent contribution of the trigeminal and olfactory nerves to the detection of airborne chemicals. Smell is experienced through olfactory nerve receptors in the nose. Nasal pungency, described as common chemical sensations such as prickling, irritation, tingling, freshness, stinging, and burning, is experienced through nonspecialized receptors of the trigeminal nerve in the face. Odor and pungency thresholds follow different patterns related to chemical concentration. Odor is often detected at much lower levels. A linear correlation between pungency thresholds of homologous series (of alcohols, acetates, ketones, and alkylbenzenes, all relatively nonreactive agents) suggests that nasal pungency relies on a physicochemical interaction with a susceptible biophase within the cell membrane. Through this nonspecific mechanism, low, subthreshold levels of a wide variety of VOCs, as found in many polluted indoor environments, may be additive in sensory impact to produce noticeable sensory irritation.

Exposure Standards. Few standards exist for exposure to VOCs in nonindustrial indoor environments. NIOSH, OSHA, and ACGIH have regulatory standards or recommended limits for industrial occupational exposures [ACGIH (annual); NIOSH 1992]. With few exceptions, concentrations observed in nonindustrial indoor environments fall well below (100 to 1000 times lower) published pollutant-specific occupational exposure limits. The California Office of Environmental Health Hazard Assessment (OEHHA 2007) established chronic reference exposure limits (cRELs) for inhalation exposure to 80 compounds, including many VOCs found in indoor air, which can be used as guidelines for establishing appropriate IAQ criteria regarding specific VOCs of interest.

Total VOC (TVOC) concentrations have been suggested as an indicator of the ability of combined VOC exposures to produce adverse health effects. This approach is no longer supported, because the irritant potential and toxicity of individual VOCs vary widely, and measured concentrations are highly dependent on the sampling and analytical methods used (Hodgson 1995). In controlled exposure experiments, odors become significant at roughly 3 mg/m^3. At 5 mg/m^3, objective effects were seen, in addition to subjective reports of irritation. Exposures for 50 min to 8 mg/m^3 of synthetic mixtures of 20 VOCs led to significant irritation of mucous membranes in the eyes, nose, and throat.

Exposure Control Strategies. VOC control strategies include source elimination or reduction, local exhaust, air cleaning, and general dilution ventilation. Ventilation requirements and other means of control of gaseous contaminants are discussed more thoroughly in Chapter 16 of this volume and Chapter 45 of the 2007 *ASHRAE Handbook—HVAC Applications*.

Refrigerants.

Sources. The primary sources of exposure to refrigerants are leaks from refrigeration and HVAC equipment and refrigerant storage containers. Exposure may also result from poor practice when servicing refrigeration equipment.

Health Effects. ASHRAE *Standard* 34 assigns refrigerants to one of two toxicity classes (A or B) based on allowable exposure.

Fatalities have been reported following acute exposure to fluorocarbon refrigerants. Chronic, low-level inhalation exposures to refrigerants can cause cardiotoxicity. Some are thought to be cardiac sensitizers to epinephrine and put occupants at risk for arrhythmias. Central nervous system (CNS) depression and asphyxia have been noted with exposures to very high concentrations. Hathaway et al. (1991) found that volunteers exposed to 200,000 ppm of R-12 experienced significant eye irritation and CNS effects. Chronic exposure to 1000 ppm for 8 h per day for up to 17 days caused no subjective symptoms or changes in pulmonary function.

A significant hazard exists when chlorinated hydrocarbons (R-11, for example) are used near open flame or heated surfaces. Phosgene gas (carbonyl chloride, an extreme irritant to the lungs) and halogen acids may be generated when chlorinated or fluorinated solvents or gases decompose in the presence of heat.

Exposure Standards. ASHRAE *Standard* 15 discusses safety for refrigeration systems, and *Standard* 34 classifies refrigerants by safety levels.

Exposure Control Strategies. Refrigerant-containing systems may only be serviced by certified technicians. Controls for preventing exposures include selection and use of appropriate fittings and valves, and ensuring that compressed gas cylinders are secured during use, transport, and storage. When repairs are made to leaking or defective HVAC equipment, adequate dilution ventilation should be provided to the work area. ASHRAE *Standard* 15 establishes specific requirements for designing, installing, operating, and servicing mechanical refrigeration equipment.

Inorganic Gases.

Sources. Inorganic gases in the nonindustrial environment may come from a combination of outdoor air and indoor sources, including occupants (e.g., respiration, toiletries), processes (e.g., combustion, office equipment), and indoor air chemistry (e.g., reaction between ozone and alkenes).

Health Effects. **Carbon monoxide** is a chemical asphyxiant. Inhalation of CO causes a throbbing headache because hemoglobin has a greater affinity for CO than for oxygen (about 240 times greater), and because of a detrimental shift in the oxygen dissociation curve. Carbon monoxide inhibits oxygen transport in the blood by forming carboxyhemoglobin and inhibiting cytochrome oxidase at the cellular level. Cobb and Etzel (1991) suggested that CO poisoning at home represented a major preventable disease. Moolenaar et al. (1995) had similar findings, and suggested that motor vehicles and home furnaces were primary causes of mortality. Girman et al. (1998) identified both fatal outcomes and "episodes." Respectively, 35.9% and 30.6% of fatal outcomes and episodes resulted from motor vehicles, 34.8% and 39.9% from appliance combustion, 4.5% and 5.2% from small appliances, 2.2% and 2.3% from camping equipment, 5.6% and 5.0% from fires, 13.4% and 13.3% from grills and hibachis, and the remainder were unknown. In a review of CO exposures in the United States from 2001 to 2003, the Centers for Disease Control (CDC 2005) found that nearly 500 people died and over 15,000 were treated in emergency departments each year after unintentional, non-fire-related CO exposures. Of cases with known sources, the most common source of CO was furnaces (18.5%), followed by motor vehicles (9.1%). Inappropriate use of portable generators, a growing problem, resulted in around 50 deaths per year from 2002 to 2005 (CPSC 2006).

Carbon dioxide can become dangerous not as a toxic agent but as a simple asphyxiant. When concentrations exceed 35,000 ppm, central breathing receptors are triggered and cause the sensation of shortness of breath. At progressively higher concentrations, central nervous system dysfunction begins because of simple displacement of oxygen. Concentrations of CO_2 in the nonindustrial environment are often measured in the range of 400 to 1200 ppm, depending on occupant density and ventilation quantity and effectiveness.

Inhalation of **nitric oxide (NO)** causes methemoglobin formation, which adversely affects the body by interfering with oxygen transport at the cellular level. NO exposures of 3 ppm have been compared to carbon monoxide exposures of 10 to 15 ppm (Case et al. 1979, in EPA 1991).

Nitrogen dioxide (NO_2) is a corrosive gas with a pungent odor, with a reported odor threshold between 0.11 and 0.22 ppm. NO_2 has low water solubility, and is therefore inhaled into the deep lung, where it causes a delayed inflammatory response. Increased airway resistance has been reported at 1.5 to 2 ppm (Bascom 1996). NO_2 is reported to be a potential carcinogen through free radical production (Burgess and Crutchfield 1995). At high concentrations, NO_2 causes lung damage directly by its oxidant properties, and may cause health effects indirectly by increasing host susceptibility to respiratory infections. Health effects from exposures to ambient outdoor concentrations or in residential situations are inconsistent, especially in studies relating to exposures from gas cooking stoves (Samet et al. 1987). Indoor concentrations of NO_2 often exceed ambient concentrations because of the presence of strong indoor sources and a trend toward more energy-efficient (tighter) homes. Acute toxicity is seldom seen from NO_2 produced by unvented indoor combustion, because insufficient quantities of NO_2 are produced. Chronic pulmonary effects from exposure to combinations of low-level combustion pollutants are possible, however (Bascom et al. 1996).

Sulfur dioxide (SO_2) is a colorless gas with a pungent odor detected at about 0.5 ppm (EPA 1991). Because SO_2 is quite soluble in water, it readily reacts with moisture in the respiratory tract to irritate the upper respiratory mucosa. Concomitant exposure to fine particles, an individual's depth and rate of breathing, and preexisting disease can influence the degree of response to SO_2 exposure.

Ozone (O_3) is a pulmonary irritant and alters human pulmonary function at concentrations of approximately 0.12 ppm (Bates 1989). Exposure to ozone at 60 to 80 ppb causes inflammation, bronchoconstriction, and increased airway responsiveness. The EPA's BASE study of over 100 randomly selected typical U.S. office buildings (Apte et al. 2007) found a clear statistical relationship between ambient ozone concentrations and building-related health symptoms, despite the fact that only one building had a workday average ambient ozone concentration greater than the 8 h national ambient air quality standards (NAAQS; EPA 2008a) level of 80 ppb.

Ozone reacts with many organic chemicals and airborne particulate matter commonly found indoors. Weschler (2006) summarizes current knowledge of these reactions and their products, which include both stable reaction products that may be more irritating than their chemical precursors (Mølhave et al. 2005; Tamas et al. 2006; Weschler and Shields 2000) and relatively short-lived products that are highly irritating and may also have chronic toxicity or carcinogenicity (Destaillats et al. 2006; Nazaroff et al. 2006; Weschler 2000; Wilkins et al. 2001; Wolkoff et al. 2000).

Inhalation exposures to gaseous oxides of nitrogen (NO_x), sulfur (SO_2), and ozone (O_3) occur in residential and commercial buildings. These air pollutants are of considerable concern because of the potential for acute and chronic respiratory tract health effects in exposed individuals, particularly individuals with preexisting pulmonary disease.

Exposure Standards and Guidelines. Currently, there are no specific U.S. government standards for nonindustrial occupational exposures to air contaminants. Occupational exposure criteria are health-based; that is, they consider only healthy workers, and not necessarily individuals who may be unusually responsive to the effects of chemical exposures. The U.S. EPA's (2008a) NAAQS are also health-based standards designed to protect the general public from the effects of hazardous airborne pollutants (see Chapter 11); however, there is debate as to whether these standards truly represent health-based thresholds, because two (ozone and carbon monoxide) of the six criteria involve toxicologically based research for standard development.

Table 5 Inorganic Gas Comparative Criteria

Contaminant	OSHA TWA[a]	U.S. EPA NAAQS[b]
Nitric oxide	25 ppm (30 mg/m^3)	None
Nitrogen dioxide	Ceiling[c] 5 ppm (9 mg/m^3)	0.053 ppm (100 µg/m^3)
Sulfur dioxide	5 ppm (13 mg/m^3)	0.03 ppm (80 µg/m^3) 24 h: 0.14 ppm (365 µg/m^3)[d]
Ozone	0.1 ppm (0.2 mg/m^3)	0.08 ppm (8 h in specified form)[e]

[a]TWA: 8 h time-weighted average
[b]Values are annual arithmetic mean unless otherwise specified
[c]Ceiling value, not to be exceeded during any part of working exposure
[d]Not to be exceeded more than once per year
[e]Per revision July 1997; see Final Rule at *Federal Register* 62(138):38856

Table 5 is not meant as a health-based guideline for evaluating indoor exposures to inorganic gases; rather, it is intended for comparison and consideration by investigators of the indoor environment. These criteria may not be completely protective for all workers.

Exposure Control Strategies. Inorganic gas contaminant control strategies include source elimination or reduction, local exhaust, space pressure control, and general dilution ventilation. Ventilation requirements and other means of control of gaseous contaminants are discussed more thoroughly in Chapter 16 of this volume and Chapter 45 of the 2007 *ASHRAE Handbook—HVAC Applications*.

The by-products of indoor air chemistry can be limited by using carbon-based filters in locations where outdoor ozone concentrations commonly approach or exceed the NAAQS.

PHYSICAL AGENTS

Physical factors in the indoor environment include thermal conditions (temperature, moisture, air velocity, and radiant energy); mechanical energy (noise and vibration); and electromagnetic radiation, including ionizing (radon) and nonionizing [light, radiofrequency, and extremely low frequency (ELF)] magnetic and electric fields. Physical agents can act directly on building occupants, interact with indoor air quality factors, or affect human responses to the indoor environment. Though not categorized as indoor air quality factors, physical agents often affect perceptions of indoor air quality.

THERMAL ENVIRONMENT

The thermal environment affects human health in that it affects body temperature regulation and heat exchange with the environment. A normal, healthy, resting adult's internal or core body temperatures are very stable, with variations seldom exceeding 1°F. The internal temperature of a resting adult, measured orally, averages about 98.6°F; measured rectally, it is about 1°F higher. Core temperature is carefully modulated by an elaborate physiological control system. In contrast, skin temperature is basically unregulated and can (depending on environmental temperature) vary from about 88 to 96.8°F in normal environments and activities. It also varies between different parts of the skin, with the greatest range of variation in the hands and feet.

Range of Healthy Living Conditions

Environmental conditions for good thermal comfort minimize effort of the physiological control system. The control system regulates internal body temperature by varying the amount of blood flowing to different skin areas, thus increasing or decreasing heat loss to the environment. Additional physiological response includes secreting sweat, which can evaporate from the skin in warm or hot environments, or increasing the body's rate of metabolic heat production by shivering in the cold. For a resting person wearing trousers and a long-sleeved shirt, thermal comfort in a steady state is experienced in a still-air environment at 75°F. A zone of comfort extends about 3°F above and below this optimum level (Fanger 1970).

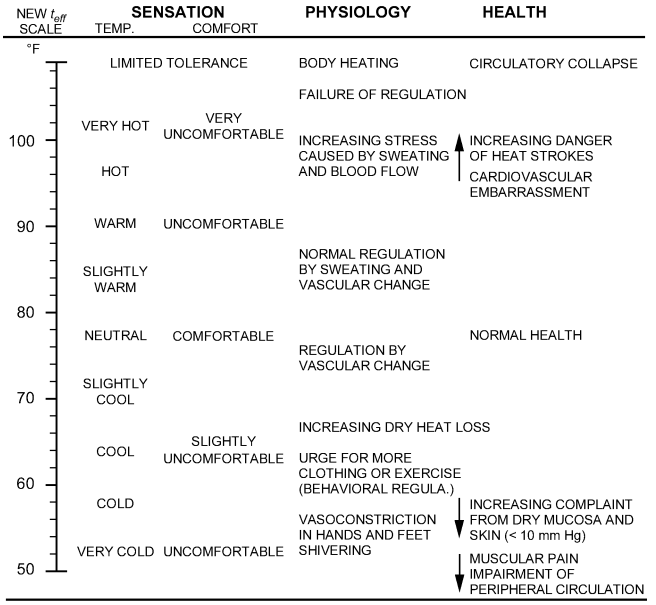

Fig. 1 Related Human Sensory, Physiological, and Health Responses for Prolonged Exposure

An individual can minimize the need for physiological (involuntary) responses to the thermal environment, which generally are perceived as uncomfortable, in various ways. In a cool or cold environment, these responses include increased clothing, increased activity, or seeking or creating an environment that is warmer. In a warm or hot environment, the amount of clothing or level of physical activity can be reduced, or an environment that is more conducive to increased heat loss can be created. Some human responses to the thermal environment are shown in Figure 1.

Cardiovascular and other diseases and aging can reduce the capacity or ability of physiological processes to maintain internal body temperature through balancing heat gains and losses. Thus, some persons are less able to deal with thermal challenges and deviations from comfortable conditions. Metabolic heat production tends to decrease with age, as a result of decreasing basal metabolism together with decreased physical activity. Metabolic heat production at age 80 is about 20% less than that at 20 years of age, for comparable size and mass. Persons in their eighties, therefore, may prefer an environmental temperature about 3°F warmer than persons in their twenties. Older people may have reduced capacity to secrete sweat and to increase their skin blood flow, and are therefore more likely to experience greater strain in warm and hot conditions, as well as in cool and cold conditions. However, the effect of age on metabolism and other factors related to thermal response varies considerably from person to person, and care should be exercised in applying these generalizations to specific individuals.

Hypothermia

Hypothermia is defined as a core body temperature of less than 95°F. Hypothermia can result from environmental cold exposure, but may also be induced by other conditions, such as metabolic disorders and drug use. Occupational hypothermia occurs in workers in a cold environment when heat balance cannot be met while maintaining work performance. Elderly persons sitting inactive in a cool room may become hypothermic, because they often fail to observe a slow fall in body temperature (Nordic Conference on Cold 1991).

Deleterious effects of cold on work performance derive from peripheral vasoconstriction and cooling, which slows down the rate

of nerve conduction and muscle contraction, and increases stiffness in tendons and connective tissues. This induces clumsiness and increases risk for injury (e.g., in occupational settings). Direct effects of cold include injuries from frostbite (skin freezes at 32 to 35.5°F) and a condition called **immersion foot**, in which the feet are exposed to wetness and temperatures of 34 to 50°F for more than 12 h, and vasoconstriction and low oxygen supply lead to edema and tissue damage.

Hyperthermia

In hyperthermia, body temperatures are above normal. A deep-body temperature increase of 4°F above normal does not generally impair body function. For example, it is not unusual for runners to have rectal temperatures of 104°F after a long race. An elevated body temperature increases metabolism. However, when body temperature increases above normal for reasons other than exercise, heat illness may develop. Heat illness represents a number of disorders from mild to fatal, which do not depend only on the hyperthermia in itself. In heat stroke, the most severe condition, the heat balance regulation system collapses, resulting in a rapid rise in body temperature. Central nervous system function deteriorates at deep body temperatures above 106 to 108°F. Convulsions may occur above such temperatures, and cells may be damaged. This condition is particularly dangerous for the brain, because lost neurons are not replaced. Thermoregulatory functions of sweating and peripheral vasodilation cease at about 110°F, after which body temperatures tend to rise rapidly if external cooling is not imposed (Blatteis 1998; Hales et al. 1996).

Seasonal Patterns

Ordinary seasonal changes in temperate climates are temporally associated with illness. Many acute and several chronic diseases vary in frequency or severity with time of year, and some are present only in certain seasons. Most countries report increased mortality from cardiovascular disease during colder winter months. Minor respiratory infections, such as colds and sore throats, occur mainly in fall and winter. More serious infections, such as pneumonia, have a somewhat shorter season in winter. Intestinal infections, such as dysentery and typhoid fever, are more prevalent in summer. Diseases transmitted by insects, such as encephalitis and endemic typhus, are limited to summer, because insects are active in warm temperatures only.

Hryhorczuk et al. (1992), Martinez et al. (1989), and others describe a correlation between weather and seasonal illnesses, but correlations do not necessarily establish a causal relationship. Daily or weekly mortality and heat stress in heat waves have a strong physiological basis directly linked to outdoor temperature. In indoor environments, which are well controlled with respect to temperature and humidity, such temperature extremes and the possible adverse effects on health are strongly attenuated.

Increased Deaths in Heat Waves

The role of ambient temperature extremes produced by weather conditions in producing discomfort, incapacity, and death has been studied extensively (Katayama and Momiyana-Sakamoto 1970). Military personnel, deep-mine workers, and other workers occupationally exposed to extremes of high and low temperature have been studied, but the importance of thermal stress affecting both the sick and healthy general population is not sufficiently appreciated. Collins and Lehmann (1953) studied weekly deaths over many years in large U.S. cities and demonstrated the effect of heat waves in producing conspicuous periods of excess mortality. Excess mortality caused by heat waves was of the same amplitude as that from influenza epidemics, but tended to last one week instead of the 4 to 6 weeks of influenza epidemics.

Ellis (1972) reviewed heat wave-related excess mortality in the United States. Mortality increases of 30% over background are commonly seen, especially in heat waves early in the summer. Much of the increase occurs in the population over age 65, more of it in women than in men, and many deaths are from cardiovascular, cerebrovascular, or respiratory causes (often exacerbated preexisting conditions). Oeschli and Buechley (1970) studied heat-related deaths in Los Angeles heat waves of 1939, 1955, and 1963. Kilbourne et al. (1982) suggested that the same risk factors (i.e., age, low income, and African-American derivation) persist in more recent heat death epidemics. In Paris, about 3000 persons died during the heat wave in the summer of 2003.

Among the most notable lethal heat waves in Europe are Athens in 1987 and 1988 (Giles et al. 1990), Seville in 1988 (Diaz et al. 2002), Valencia in 1991 and 1993 (Ballester et al. 1997), London in 1995 (Hajat et al. 2002), the Netherlands between 1979 and 1991 (Kunst et al. 1993), and Paris in 2003 (Thirion et al. 2005).

The temperature/mortality relation varies greatly by latitude and climatic zone (McMichael et al. 2006). Occupants of hotter cities are more affected by colder temperatures, and occupants of colder cities are more affected by warmer temperatures. People living in urban environments are at greater risk than those in nonurban regions. Thermally inefficient housing and the so-called urban heat island effect amplify and extend the rise in temperatures (especially overnight).

Hardy (1971) showed the relationship of health data to comfort on a psychrometric diagram (Figure 2). The diagram contains ASHRAE effective temperature (ET*) lines and lines of constant skin moisture level or skin wettedness. Skin wettedness is defined as that fraction of the skin covered with water to account for the observed evaporation rate. The ET* lines are loci of constant physiological strain, and also correspond to constant levels of physiological discomfort (i.e., slightly uncomfortable, comfortable, and very comfortable) (Gonzalez et al. 1978). Skin wettedness, as an indicator of strain (Berglund and Cunningham 1986; Berglund and Gonzalez 1977) and the fraction of the skin wet with perspiration, is fairly constant along an ET* line. Numerically, ET* is the equivalent temperature at 50% rh that produces the strain and discomfort of the actual condition. The summer comfort range is between an ET* of 73 and 79°F. In this region, skin wettedness is less than 0.2. Heat strokes occur generally when ET* exceeds 93°F (Bridger and Helfand 1968). Thus, the ET* line of 95°F is generally considered dangerous. At this point, skin wettedness will be 0.4 or higher.

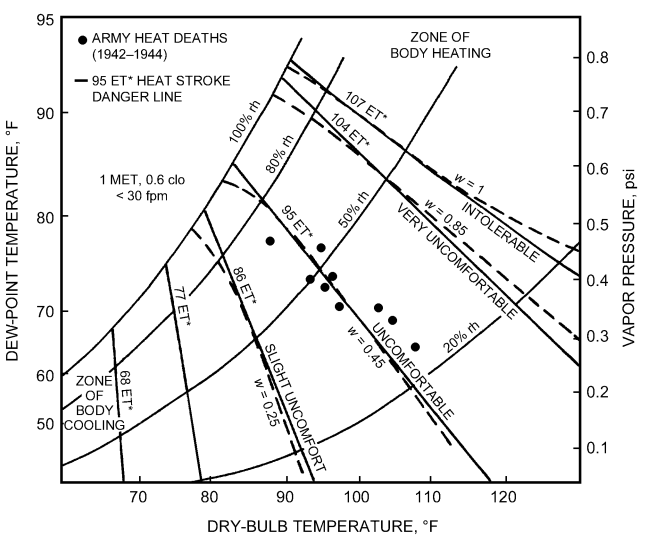

Fig. 2 Isotherms for Comfort, Discomfort, Physiological Strain, Effective Temperature (ET*), and Heat Stroke Danger Threshold

The black dots in Figure 2 correspond to heat stroke deaths of healthy male U.S. soldiers assigned to sedentary duties in midwestern army camp offices (Shickele 1947). Older people can be expected to respond less well to thermal challenges than do healthy soldiers. This was apparently the case in the Illinois heat wave study (Bridger and Helfand 1968), where the first wave with a 33% increase in death rate and an ET* of 85°F affected mainly the over-65-year-old group. The studies suggest that the "danger line" represents a threshold of significant risk for young healthy people, and that the danger tends to move to lower values of ET* with increasing age.

Effects of Thermal Environment on Specific Diseases

Cardiovascular diseases are largely responsible for excess mortality during heat waves. For example, Burch and DePasquale (1962) found that heart disease patients with decompensation (i.e., inadequate circulation) were extremely sensitive to high temperatures, and particularly to moist heat. However, both cold and hot temperature extremes have been associated with increased coronary heart disease deaths and anginal symptoms (Teng and Heyer 1955).

Both acute and chronic respiratory diseases often increase in frequency and severity during extreme cold weather. No increase in these diseases has been noted in extreme heat. Additional studies of hospital admissions for acute respiratory illness show a negative correlation with temperature after removal of seasonal trends (Holland 1961). Symptoms of chronic respiratory disease (bronchitis, emphysema) increase in cold weather, probably because reflex constriction of the bronchi adds to the obstruction already present. Greenberg (1964) found evidence of cold sensitivity in asthmatics: emergency room treatments for asthma increased abruptly in local hospitals with early and severe autumn cold spells. Later cold waves with even lower temperatures produced no such effects, and years without early extreme cold had no asthma epidemics of this type. Patients with cystic fibrosis are extremely sensitive to heat because their reduced sweat gland function greatly diminishes their ability to cope with increased temperature (Kessler and Anderson 1951).

Itching and chapping of the skin are influenced by (1) atmospheric factors, particularly cold and dry air; (2) frequent washing or wetting of skin; and (3) low indoor humidities. Although skin itching is usually a winter cold-climate illness in the general population, it can be caused by excessive summer air conditioning (Gaul and Underwood 1952; Susskind and Ishihara 1965).

People suffering from chronic illness (e.g., heart disease) or serious acute illnesses that require hospitalization often manage to avoid serious thermal stress. Katayama and Momiyana-Sakamoto (1970) found that countries with the most carefully regulated indoor climates (e.g., Scandinavian countries, the United States) have only small seasonal fluctuations in mortality, whereas countries with less space heating and cooling exhibit greater seasonal swings in mortality. For example, mandatory air conditioning in retirement and assisted living homes in the southwest United States has virtually eliminated previously observed mortality increases during heat waves.

Injury from Hot and Cold Surfaces

The skin has cold, warm, and pain sensors to feed back thermal information about surface contacts. When the skin temperature rises above 113°F or falls below about 59°F, sensations from the skin's warm and cold receptors are replaced by those from pain receptors to warn of imminent thermal injury to tissue. The rate of change of skin temperature and not just the actual skin temperature may also be important in pain perception. Skin temperature and its rate of change depend on the temperature of the contact surface, its conductivity, and contact time. Table 6 gives approximate temperature limits to avoid pain and injury when contacting three classes of conductors for various contact times (ISO 2006).

Table 6 Approximate Surface Temperature Limits to Avoid Pain and Injury

	Contact Time				
Material	**1 s**	**10 s**	**1 min**	**10 min**	**8 h**
Metal, water	149°F	133°F	124°F	118°F	109°F
Glass, concrete	176°F	151°F	129°F	118°F	109°F
Wood	248°F	190°F	140°F	118°F	109°F

Source: ISO *Standard* 13732-1:2006.

ELECTRICAL HAZARDS

Electrical current can cause burns, neural disturbances, and cardiac fibrillation (Billings 1975). The threshold of perception is about 5 mA for direct current, with a feeling of warmth at the contact site. The threshold is 1 mA for alternating current, which causes a tingling sensation.

Resistance of the current pathway through the body is a combination of core and skin resistance. The core is basically a saline volume conductor with very little resistance; therefore, the skin provides the largest component of the resistance. Skin resistance decreases with moisture. If the skin is moist, voltages as low as 2 V (ac) or 5 V (dc) are sufficient to be detected, and voltages as low as 20 V (ac) or 100 V (dc) can cause a 50% loss in muscular control.

The dangerous aspect of alternating electrical current is its ability to cause cardiac arrest by ventricular fibrillation. If a weak alternating current (100 mA for 2 s) passes through the heart (as it would in going from hand to foot), the current can force the heart muscle to fibrillate and lose the rhythmic contractions of the ventricles necessary to pump blood. Unconsciousness and death will soon follow if medical aid cannot rapidly restore normal rhythm.

MECHANICAL ENERGIES

Vibration

Vibration in a building originates from both outside and inside the building. Sources outside a building include blasting operations, road traffic, overhead aircraft, underground railways, earth movements, and weather conditions. Sources inside a building include doors closing, foot traffic, moving machinery, elevators, HVAC systems, and other building services. Vibration is an omnipresent, integral part of the built environment. The effects of vibration on building occupants depend on whether it is perceived by those persons and on factors related to the building, building location, activities of occupants in the building, and perceived source and magnitude of vibration. Factors influencing the acceptability of building vibration are presented in Figure 3.

The combination of hearing, seeing, or feeling vibration determines human response. Components concerned with hearing and seeing are part of the visual environment of a room and can be assessed as such. The perception of mechanical vibration by feeling is generally through the cutaneous and kinesthetic senses at high frequencies, and through the vestibular and visceral senses at low frequencies. Because of this and the nature of vibration sources and building responses, building vibration may be conveniently considered in two categories: low-frequency vibrations less than 1 Hz and high-frequency vibrations of 1 to 80 Hz.

Measurement and Assessment. Human response to vibration depends on vibration of the body. The main vibrational characteristics are vibration level, frequency, axis (and area of the body), and exposure time. A root-mean-square (RMS) averaging procedure (over the time of interest) is often used to represent vibration acceleration ($ft/s^2 \cdot RMS$). Vibration frequency is measured in cycles per second (Hz), and the vibration axis is usually considered in three orthogonal, human-centered translational directions (up-and-down, side-to-side, and fore-and-aft). Although the coordinate system is centered inside the body, in practice, vibration is measured at the

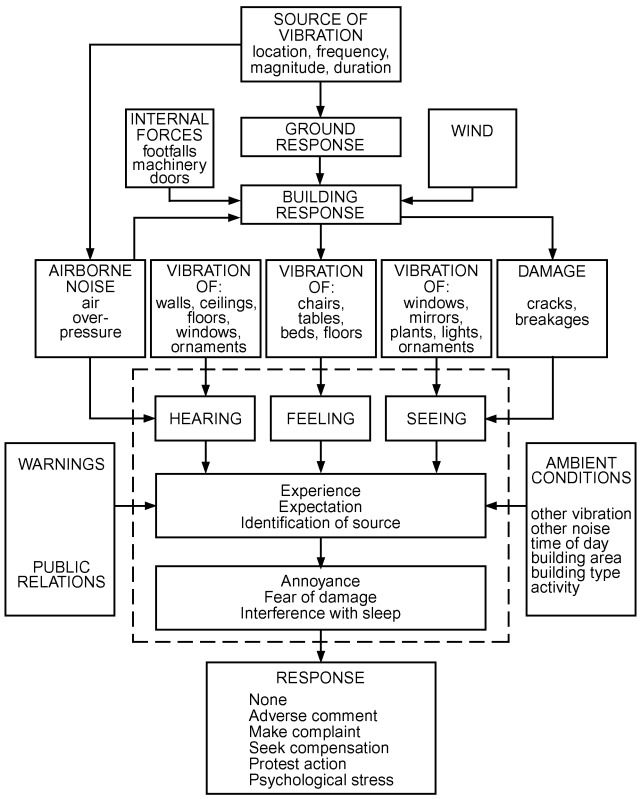

Fig. 3 Factors Affecting Acceptability of Building Vibration

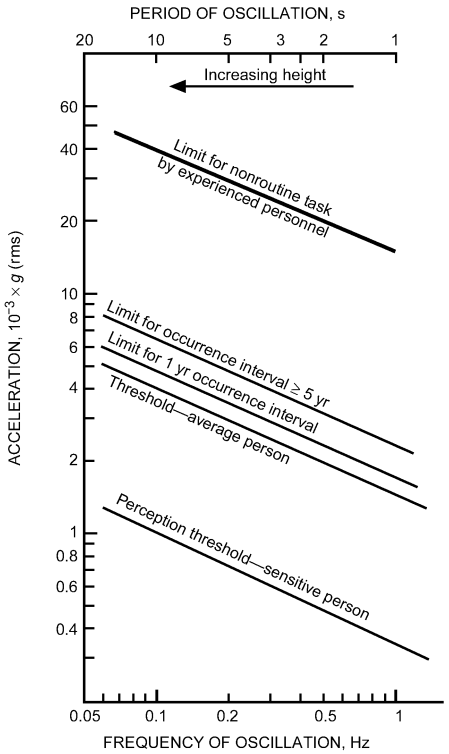

Fig. 4 Acceleration Perception Thresholds and Acceptability Limits for Horizontal Oscillations

human surface, and measurements are directly compared with relevant limit values or other data concerning human response.

Rotational motions of a building in roll, pitch, and yaw are usually about an axis of rotation some distance from the building occupants. For most purposes, these motions can be considered as the translational motions of the person. For example, a roll motion in a building about an axis of rotation some distance from a seated person has a similar effect as side-to-side translational motions of that person, etc.

Most methods assess building vibrations with RMS averaging and frequency analysis. However, human response is related to the time-varying characteristics of vibration as well. For example, many stimuli are transient, such as those caused by a train passing a building. The vibration event builds to a peak, followed by a decay in level over a total period of about 10 s. The nature of the time-varying event and the number of occasions it occurs during a day are important factors that might be overlooked if data are treated as steady-state and continuous.

Standard Limits

Low-Frequency Motion (1 Hz). The most commonly experienced form of slow vibration in buildings is building sway. This motion can be alarming to occupants if there is fear of building damage or injury. Whereas occupants of two-story wood frame houses accept occasional creaks and motion from wind storms or a passing heavy vehicle, such events are not as accepted by occupants of high-rise buildings. Detected motion in tall buildings can cause discomfort and alarm. The perception thresholds of normal, sensitive humans to low-frequency horizontal motion are given in Figure 4 (Chen and Robertson 1972; ISO 1984). The frequency range is from 0.06 to 1 Hz or, conversely, for oscillations with periods of 1 to 17 s. The natural frequency of sway of the Empire State Building in New York City, for example, has a period of 8.3 s (Davenport 1988). The thresholds are expressed in terms of relative acceleration, which is the actual acceleration divided by the standard acceleration of gravity g (32.2 ft/s^2). The perception threshold to sway in terms of building accelerations decreases with increasing frequency and ranges from 0.16 to 0.06 ft/s^2.

For tall buildings, the highest horizontal accelerations generally occur near the top at the building's natural frequency of oscillation. Other parts of the building may have high accelerations at multiples of the natural frequency. Tall buildings always oscillate at their natural frequency, but the deflection is small and the motion undetectable. In general, short buildings have a higher natural frequency of vibration than taller ones. However, strong wind forces energize the oscillation and increase the horizontal deflection, speed, and accelerations of the structure.

ISO (1984) states that building motions should not produce alarm and adverse comment from more than 2% of the building's occupants. The level of alarm depends on the interval between events. If noticeable building sway occurs for at least 10 min at intervals of 5 years or more, the acceptable acceleration limit is higher than if this sway occurs annually (Figure 4). For annual intervals, the acceptable limit is only slightly above the normal person's threshold of perception. Motion at the 5-year limit level is estimated to cause 12% to complain if it occurred annually. The recommended limits are for purely horizontal motion; rotational oscillations, wind noise, and/or visual cues of the building's motion exaggerate the sensation of motion, and, for such factors, the acceleration limit is lower.

The upper line in Figure 4 is intended for offshore fixed structures such as oil drilling platforms. The line indicates the level of horizontal acceleration above which routine tasks by experienced personnel would be difficult to accomplish on the structure. Because they are routinely in motion in three dimensions, Figure 4 does not apply to transportation vehicles.

High-Frequency Motion (1 to 80 Hz). Higher-frequency vibrations in buildings are caused by machinery, elevators, foot traffic,

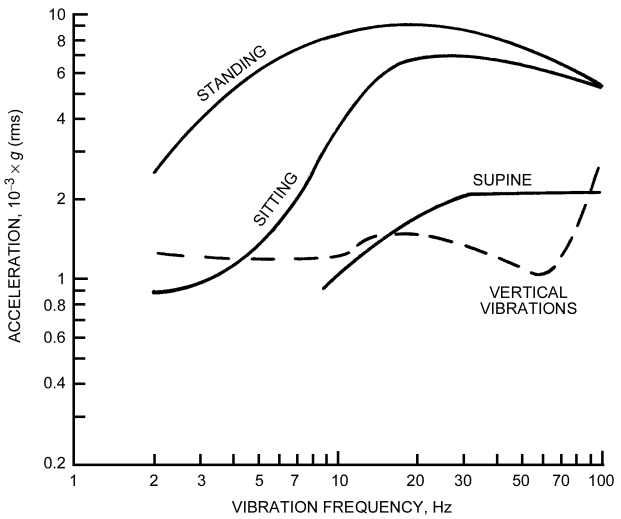

Fig. 5 Median Perception Thresholds to Horizontal (Solid Lines) and Vertical (Dashed Line) Vibrations

Table 7 Ratios of Acceptable to Threshold Vibration Levels

Place	Time	Continuous or Intermittent Vibration	Impulse or Transient Vibration Several Times per Day
Critical work areas	Day or night	1	1
Residential	Day/night	2 to 4/1.4	30 to 90/1.4 to 20
Office	Day or night	4	60 to 128
Workshop	Day or night	8	90 to 128

Note: Ratios for continuous or intermittent vibration and repeated impulse shock range from 0.7 to 1.0 for hospital operating theaters (room) and critical working areas. In other situations, impulse shock can generally be much higher than when vibration is more continuous.

fans, pumps, and HVAC equipment. Further, the steel structures of modern buildings are good transmitters of high-frequency vibrations. The sensitivity to these higher frequency vibrations is indicated in Figure 5 (Parsons and Griffin 1988). Displayed are median perception thresholds to vertical and horizontal vibrations in the 2 to 100 Hz frequency range. The average perception threshold for vibrations of this type is from 0.03 to 0.3 ft/s^2, depending on frequency and on whether the person is standing, sitting, or lying down.

People detect horizontal vibrations at lower acceleration levels when lying down than when standing. However, a soft bed decouples and isolates a person fairly well from vibrations of the structure. The threshold to vertical vibrations is nearly constant at approximately 0.04 ft/s^2 for both sitting and standing positions from 2 to 100 Hz. This agrees with earlier observations by Reiher and Meister (1931).

Many building spaces with critical work areas (surgery, precision laboratory work) are considered unacceptable if vibration is perceived by the occupants. In other situations and activities, perceived vibration may be acceptable. Parsons and Griffin (1988) found that accelerations twice the threshold level were unacceptable to occupants in their homes. A method of assessing acceptability in buildings is to compare the vibration with perception threshold values (Table 7).

Sound and Noise

In general terms, sound transmitted through air consists of oscillations in pressure above and below ambient atmospheric pressure. A vibrating object causes high- and low-pressure areas to be formed; these areas propagate away from the source. The entire

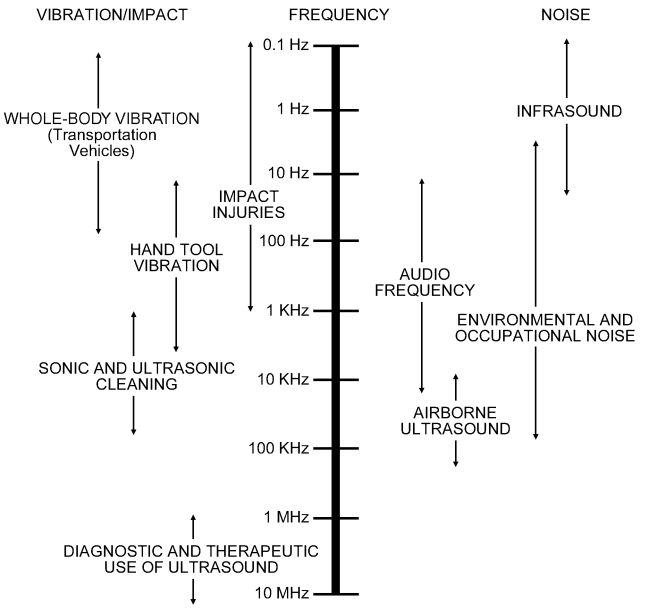

Fig. 6 Mechanical Energy Spectrum

mechanical energy spectrum includes infrasound and ultrasound as well as audible sound (Figure 6).

Health Effects. Hearing loss is generally considered the most undesirable effect of noise exposure, although there are other effects. **Tinnitus**, a ringing in the ears, is really the hearing of sounds that do not exist. It often accompanies hearing loss. **Paracusis** is a disorder where a sound is heard incorrectly; that is, a tone is heard, but has an inappropriate pitch. **Speech misperception** occurs when an individual mistakenly hears one sound for another (e.g., when the sound for *t* is heard as a *p*).

Hearing loss can be categorized as conductive, sensory, or neural. **Conductive** hearing loss results from a general decrease in the amount of sound transmitted to the inner ear. Excessive ear wax, a ruptured eardrum, fluid in the middle ear, or missing elements of bone structures in the middle ear are all associated with conductive hearing loss. These are generally not occupationally related and are generally reversible by medical or surgical means. **Sensory** hearing losses are associated with irreversible damage to the inner ear. Sensory hearing loss is further classified as (1) presbycusis, loss caused as the result of aging; (2) noise-induced hearing loss (industrial hearing loss and sociacusis, which is caused by noise in everyday life); and (3) nosoacusis, losses attributed to all other causes. **Neural** deficits are related to damage to higher centers of the auditory system.

Noise-induced hearing loss is believed to occur in the most sensitive individuals among those exposed for 8 h per day over a working lifetime at levels of 75 dBA, and for most people similarly exposed to 85 dBA.

ELECTROMAGNETIC RADIATION

Radiation energy is emitted, transmitted, or absorbed in wave or particulate form. This energy consists of electric and magnetic forces, which, when disturbed in some manner, produce electromagnetic radiation. Electromagnetic radiation is grouped into a spectrum arranged by frequency and/or wavelength. The product of frequency and wavelength is the speed of light (3×10^8 m/s). The spectrum includes ionizing, ultraviolet, visible, infrared, microwave, radio, and extremely low frequency (ELF) (Figure 7). Table 8 presents these electromagnetic radiations by their range of energies, frequencies, and wavelengths. The regions are not sharply delineated from

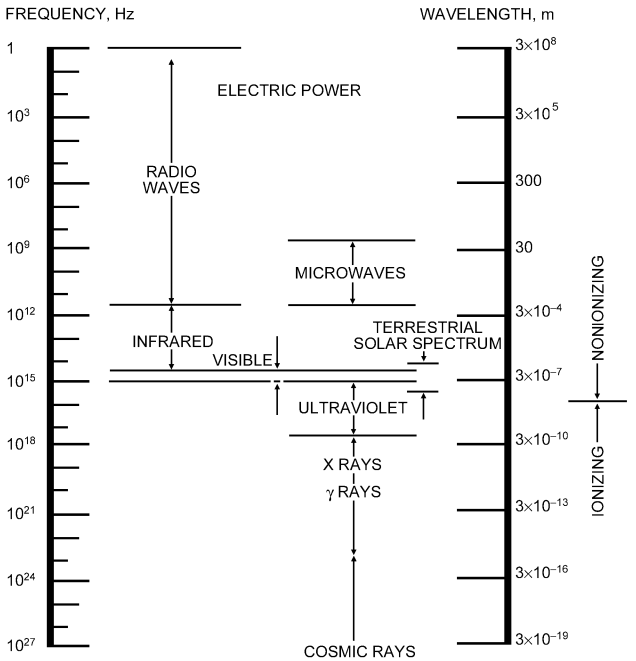

Fig. 7 Electromagnetic Spectrum

Table 8 Energy, Wavelength, and Frequency Ranges for Electromagnetic Radiation

Radiation Type	Energy Range	Wavelength Range	Frequency Range
Ionizing	>12.4 eV	<100 nm	>3.00 PHz
Ultraviolet (UV)	12.40 to 3.10 eV	100 to 400 nm	3.00 to 0.75 PHz
Visible	3.10 to 1.63 eV	400 to 760 nm	750 to 395 THz
Infrared (IR)	1.63 to 1.24 meV	760 nm to 1 mm	395 to 0.30 THz
Microwave (MW)	1.24 meV to 1.24 eV	1 mm to 1 m	300 GHz to 300 MHz
Radio-frequency (RF)	1.24 eV to 1.24 peV	1 m to 1 Mm	300 MHz to 300 Hz
Extremely low frequency (ELF)	<1.24 peV	>1 Mm	<300 Hz

each other and often overlap. It is convenient to divide these regions as listed in Table 8, because of the nature of the physical and biological effects.

Ionizing Radiation

Ionizing radiation is the part of the electromagnetic spectrum with very short wavelengths and high frequencies, and it has the ability to ionize matter. These ionizations tend to be very damaging to living matter. Background radiation that occurs naturally in the environment is from cosmic rays and naturally occurring radionuclides. It has not been established whether exposure at the low dose rate of average background levels is harmful to humans.

The basic standards for permissible air concentrations of radioactive materials are those of the National Committee on Radiation Protection, published by the National Bureau of Standards as *Handbook* No. 69. Industries operating under licenses from the U.S. Nuclear Regulatory Commission or state licensing agencies must meet requirements of the *Code of Federal Regulations*, Title 10, Part 20. Some states have additional requirements.

Table 9 Action Levels for Radon Concentration Indoors

Country/Agency	Action Level Bq/m³	Action Level pCi/L
Australia	200	5.4
Austria	400	10.8
Belgium	400	10.8
CEC	400	10.8
Canada	800	21.6
Czech Republic	400	10.8
P.R. China	200	5.4
Finland	400	10.8
Germany	250	6.7
ICRP	200	5.4
Ireland	200	5.4
Italy	400	10.8
Norway	400	10.8
Sweden	400	10.8
United Kingdom	200	5.4
United States	148	4.0
World Health Organization	200	5.4

Source: Tansey and Fliermans (1978).

An important naturally occurring radionuclide is radon (^{222}Rn), a decay product of uranium in the soil (^{238}U). Radon, denoted by the symbol Rn, is chemically inert. Details of units of measurement, typical radon levels, measurement methods and control strategies can be found in Chapter 11.

Health Effects of Radon. Radon is the leading cause of lung cancer among nonsmokers, according to EPA (2008b) estimates. Most information about radon's health risks comes from studies of workers in uranium and other underground mines. The radioactive decay of radon produces a series of radioactive isotopes of polonium, bismuth, and lead. Unlike their chemically inert radon parent, these progeny are chemically active and can attach to airborne particles that subsequently deposit in the lung, or deposit directly in the lung without attachment to particles. Some of these progeny, like radon, are alpha-particle emitters, which can cause cellular changes that may initiate lung cancer when they pass through lung cells (Samet 1989). Thus, adverse health effects associated with radon are caused by exposures to radon decay products, and the amount of risk is assumed to be directly related to the total exposure. Even though it is the radon progeny that present the possibility of adverse health risks, radon itself is usually measured and used as a surrogate for progeny measurements because of the expense involved in accurate measurements of radon progeny.

Exposure Standards. Many countries have established standards for exposure to radon. International action levels are listed in Table 9.

About 6% of U.S. homes (i.e., 5.8 million homes) have annual average radon concentrations exceeding 148 Bq/m³ (4 pCi/L), the action level set by the U.S. Environmental Protection Agency (Marcinowski et al. 1994). Because there is no known safe level of exposure to radon, the EPA (2008b) also recommends that all homes should be tested for radon, regardless of geographic location, and consideration should be given to remedial measures in homes with radon levels between 2 and 4 pCi/L.

Nonionizing Radiation

Ultraviolet radiation, visible light, and infrared radiation are components of sunlight and of all artificial light sources. Microwave and radio-frequency radiation are essential in a wide range of communication technologies and are also in widespread use for heating as in microwave ovens and heat sealers, and for heat treatments of various products. Power frequency fields are an essential and unavoidable consequence of the generation, transmission, distribution, and use of electrical power.

Optical Radiation. Ultraviolet (UV), visible, and infrared (IR) radiation compose the optical radiation region of the electromagnetic spectrum. The wavelengths range from 100 nm in the UV to 1 mm in the IR, with 100 nm generally considered to be the boundary between ionizing and nonionizing. The UV region wavelengths range from 100 to 400 nm, the visible region from 400 to 760 nm, and the IR from 760 nm to 1 mm.

Optical radiation can interact with a medium by reflection, absorption, or transmission. The skin and eyes are the organs at risk in humans. Optical radiation from any spectral region can cause acute and/or chronic biologic effects given appropriate energy characteristics and exposure. These effects include tanning, burning (erythema), premature "aging," and skin cancer; and dryness, irritation, cataracts, and blindness in the eyes.

The region of the electromagnetic spectrum visible to humans is known as light. There can be biological, behavioral, psychological, and health effects from exposure to light. Assessment of these effects depends on the purpose and application of the illumination. Individual susceptibility varies, with other environmental factors (air quality, noise, chemical exposures, and diet) acting as modifiers. It is difficult, therefore, to generalize potential hazards. **Light pollution** is the presence of unwanted light.

Light penetrating the retina not only allows the exterior world to be seen, but, like food and water, it is used in a variety of metabolic processes. Light stimulates the pineal gland to secrete melatonin, which regulates the human biological clock. This, in turn, influences reproductive cycles, sleeping, eating patterns, activity levels, and moods. The color of light affects the way the objects appear. Distortion of color rendition may result in disorientation, headache, dizziness, nausea, and fatigue.

As the daylight shortens, the human body may experience a gradual slowing down, loss of energy, and a need for more sleep. It becomes harder to get to work, and depression or even withdrawal may take place. This type of seasonal depression, brought on by changes in light duration and intensity, is called **seasonal affective disorder (SAD)**. Sufferers also complain of anxiety, irritability, headache, weight gain, and lack of concentration and motivation. Treatment of this problem is through manipulation of environmental lighting (exposure to full-spectrum lighting for extended periods, 12 h/day).

Radio-Frequency Radiation. Just as the body absorbs infrared and light energy, which can affect thermal balance, it can also absorb other longer wavelength electromagnetic radiation. For comparison, visible light has wavelengths in the range 0.4 to 0.7 μm and infrared from 0.7 to 10 μm, whereas the wavelength of K and X band radar is 12 and 28.6 mm. The wavelength of radiation in a typical microwave oven is 120 mm. Infrared is absorbed within 1 mm of the surface (Murray 1995).

The heat of absorbed radiation raises skin temperature and, if sufficient, is detected by the skin's thermoreceptors, warning the person of possible thermal danger. With increasing wavelength, radiation penetrates deeper into the body. Energy can thus be deposited well beneath the skin's thermoreceptors, making the person less able or slower to detect and be warned of the radiation (Justesen et al. 1982). Physiologically, these longer waves only heat the tissue and, because the heat may be deeper and less detectable, the maximum power density of such waves in occupied areas is regulated (ANSI 1991) (Figure 8). Maximum permitted power densities are less than half of sensory threshold values.

ERGONOMICS

Ergonomics is the scientific study of the relationship between humans and their work environments to achieve optimum adjustment in terms of efficiency, health, and well-being. Ergonomic designs of tools, chairs, etc., help workers interact more comfortably and efficiently with their environment. In jobs that were ergonomically designed, productivity typically increases and the worker enjoys a healthier working experience. More recently, researchers have distinguished intrinsic ergonomics from extrinsic, or traditional, ergonomics. Intrinsic ergonomics considers how the interface between an individual and the environment affects and relies on specific body parts (i.e., muscles, tendons, and bones) and work practices such as force of application, relaxation intervals, styles, and strength reserves that are not adequately considered in simple analyses of the physical environment.

The goals of ergonomic programs range from making work safe and humane, to increasing human efficiency, to creating human well-being. The successful application of ergonomic factors is measured by improved productivity, efficiency, safety, and acceptance of the resultant system design. The design engineer uses not only engineering skills, but also the principles of anatomy, orthopedics, physiology, medicine, psychology, and sociology to apply ergonomics to a design.

Implementing ergonomic principles in the workplace helps minimize on-the-job stress and strain, and prevents cumulative trauma

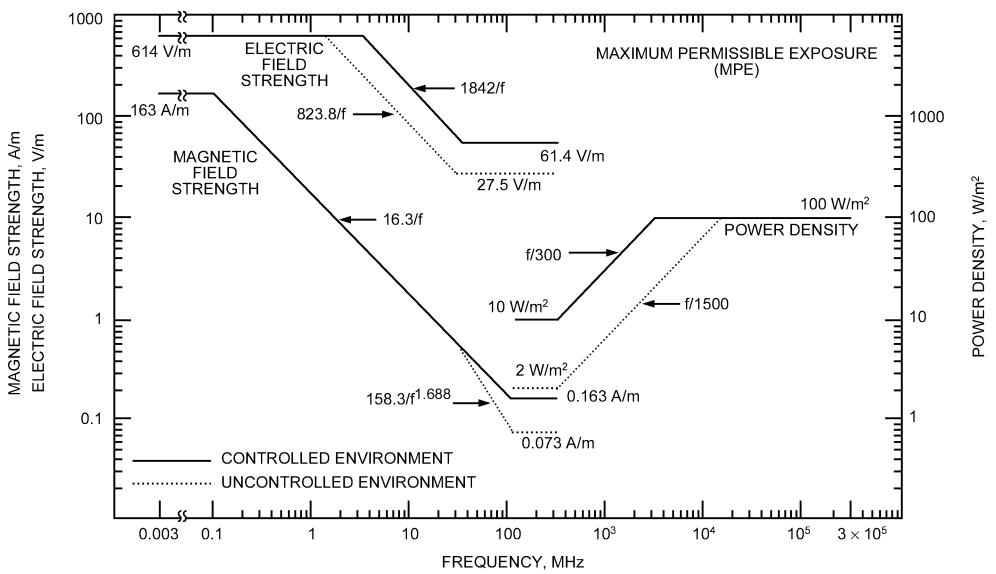

Fig. 8 Maximum Permissible Levels of Radio Frequency Radiation for Human Exposure

disorders (CTDs). These disorders are subtle injuries that can affect the muscles, tendons, and nerves at body joints, especially the hands, wrists, elbows, shoulders, neck, back, and knees. Carpal tunnel syndrome is an example of a CTD. CTDs most frequently occur as a result of strain from performing the same task on a continuous or repetitive basis. This strain can slowly build over time, until the worker experiences pain and difficulty using the injured part of the body. Higher risks of developing CTDs are encountered when the work task requires repetitive motions, excessive force, or awkward postures. The ergonomics engineer addresses these risk factors by analyzing the task thoroughly and minimizing the repetitive motion, excessive force, and awkward posture.

Poor space ergonomics (Hartkopf and Loftness 1999) and consequent occupant interventions may also directly affect indoor conditions. For example, inappropriate use of cabinets, closets, furniture, partitions, room equipment or other obstructions may block air supply or exhaust vents, reduce airflow rates and temperature or humidity regulation, and disturb airflow (Lee and Awbi 2004). These kinds of problems are usually attributed to poor space layout and ventilation design, but usually originate from lack of space availability, such as small room dimensions and high occupancies. Reduced ventilation rates deteriorate conditions for indoor environmental health, working, and comfort. They may be encountered in overstaffed offices (Mahdavi and Unzeitig 2005) or in demanding environments such as hospital operating theatres (Balaras et al. 2006).

REFERENCES

ACGIH. 1999. *Bioaerosols: Assessment and control*. American Conference of Government Industrial Hygienists, Cincinnati, OH.

ACGIH. Annual. *TLVs® and BEIs®*. American Conference of Government Industrial Hygienists, Cincinnati, OH.

ACSM. 1996. American College of Sports Medicine position stand on heat and cold illnesses during distance running. *Medicine & Science in Sports & Exercise* 28(12):1.

Alpaugh, E.L. and T.J. Hogan. 1988. *Fundamentals of industrial hygiene*, 3rd ed. National Safety Council, Itasca, IL.

Anaissie, E.J., S.R. Penzak, and M.C. Dignani. 2002. The hospital water supply as a source of nosocomial infections: A plea for action. *Archives of Internal Medicine* 162(13):1483-1492.

Anderson, H.A., D. Higgins, L.P. Hanrahan, P. Sarow, and J. Schirmer. 1991. Mesothelioma among employees with likely contact with in-place asbestos-containing building materials. *Annals of the New York Academy of Sciences* 643:550-572.

ANSI. 2005. Safety levels with respect to human exposure to radio frequency electromagnetic radiation, 3 kHz to 300 GHz. *Standard* C95.1-2005. American National Standards Institute, New York.

Apte, M.G., I.S.H. Buchanan, and M.J. Mendell. 2007. Outdoor ozone and building related symptoms in the BASE study. *Report* LBNL-62419. Lawrence Berkeley National Laboratory, Berkeley, CA.

Arnow, P.M., J.N. Fink, D.P. Schlueter, J.J. Barboriak, G. Mallison, S.I. Said, S. Martin, G.F. Unger, G.T. Scanlon, and V.P. Kurup. 1978. Early detection of hypersensitivity pneumonitis in office workers. *American Journal of Medicine* 64(2):237-242.

ASHRAE. 2000. Minimizing the risk of legionellosis associated with building water systems. *Guideline* 12-2000.

ASHRAE. 2003. M*old and moisture management in buildings*.

ASHRAE. 2004. Safety standard for refrigeration systems. *Standard* 15-2004.

ASHRAE. 2007. Designation and safety classification of refrigerants. ANSI/ASHRAE *Standard* 34-2007.

ATSDR. 2001. *Toxicological profile for asbestos*. Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services, Washington, D.C.

Balaras, C.A., E. Dascalaki, and A. Gaglia. 2006. HVAC and indoor thermal conditions in hospital operating rooms, *Energy and Buildings* 39(4): 454-470.

Ballester, F., D. Corella, S. Perez-Hoyos, M. Saez, and A. Hervas. 1997. Mortality as a function of temperature: A study in Valencia, Spain, 1991–1993. *International Journal of Epidemiology* 26(3):551-561.

Bartlett, P.C., L.A. Vonbehren, R.P. Tewari, R.J. Martin, L. Eagleton, M.J. Isaac, and P.S. Kulkarni. 1982. Bats in the belfry: An outbreak of histoplasmosis. *American Journal of Public Health* 72(12):1369-1372.

Bascom, R.A. 1996. Environmental factors and respiratory hypersensitivity: The Americas. *Toxicology Letters* 86:115-130.

Bascom, R., P. Bromberg, D.A. Costa, R. Devlin, D.W. Dockery, M.W. Frampton, W. Lambert, J.M. Samet, F.E. Speizer, and M. Utell. 1996. Health effects of outdoor pollution, parts I and II. *American Journal of Respiratory and Critical Care Medicine* 153:3-50, 477-489.

Bates, D.V. 1989. Ozone—Myth and reality. *Environmental Research* 50: 230-237.

Baylor, E.R., V. Peters, and M.B. Baylor. 1977. Water-to-air transfer of virus. *Science* 252:763.

Berglund, L.G. and D. Cunningham. 1986. Parameters of human discomfort in warm environments. *ASHRAE Transactions* 92(2).

Berglund, L.G. and R.R. Gonzalez. 1977. Evaporation of sweat from sedentary man in humid environments. *Journal of Applied Physiology, Respiratory, Environmental and Exercise Physiology* 42(5):767-772.

Billings, C.E. 1975. Electrical shock. In *Textbook of medicine*. Saunders, Philadelphia.

Blatteis, C.M., ed. 1998. *Physiology and pathophysiology of temperature regulation*. World Scientific, Singapore.

Bridger, C.A. and L.A. Helfand. 1968. Mortality from heat during July 1966 in Illinois. *International Journal of Biometeorology* 12:51.

Brundage, J.F., R.M. Scott, W.M. Lednar, D.W. Smith, and R.N. Miller. 1988. Building-associated risk of febrile acute respiratory diseases in Army trainees. *Journal of the American Medical Association* 259:2108.

Burch, G.E. and N.P. DePasquale. 1962. *Hot climates, man and his heart.* Charles C. Thomas, Springfield, IL.

Burge, H.A. 1989. In *Occupational medicine: State of the art reviews*, vol. 4, *Problem buildings: Building-associated illness and the sick building syndrome*, pp. 713-721. J.E. Cone and M.J. Hodgson, eds. Hanley and Belfus, Philadelphia.

Burge, H.A. 1995. *Bioaerosols*. Lewis, Chelsea, MI.

Burge, S., A. Hedge, S. Wilson, J.H. Bass, and A. Robertson. 1987. Sick building syndrome: A study of 4373 office workers. *Annals of Occupational Hygiene* 31:493-504.

Burgess, J.L. and C.D. Crutchfield. 1995. Quantitative respirator fit tests of Tucson fire fighters and measures of negative pressure excursions during exertion. *Applied Occupational and Environmental Hygiene* 10(1): 29-36.

Burton, D.J. 2000. *Industrial ventilation: A self-directed learning workbook*, 4th ed. Carr, North Bountiful, UT.

Buttner, M.P. and L.D. Stetzenbach. 1999. Dispersal of fungal spores from three types of air handling system duct material. *Aerobiologia* 15:1-8.

Buttner, M.P., P. Cruz-Perez, L.D. Stetzenbach, P.J. Garrett, and A.E. Luedtke. 2002. Measurement of airborne fungal spore dispersal from three types of flooring materials. *Aerobiologia* 18:1-11.

Cain, W.S. 1989. *Perceptual characteristics of nasal irritation*. National Danish Institute on Occupational Health, Copenhagen.

Cain, W.S., J.M. Samet, and M.J. Hodgson. 1995. The quest for negligible health risk from indoor air. *ASHRAE Journal* 37(7):38.

Calm, J.M. and P.A. Domanski. 2004. R-22 replacement status. *ASHRAE Journal* 46(8):29-39.

CDC. 2005. Unintentional non-fire-related carbon monoxide exposures—United States, 2001-2003. *Morbidity and Mortality Weekly Report* 54(2): 36-39.

CEN. *Surface temperatures of touchable parts, a draft proposal*. TC 114 N 122 D/E. European Standards Group.

CFR. 2001a. *Occupational safety and health standards: Air contaminants.* 29CFR1910.1000. U.S. Government Printing Office, Washington, D.C.

CFR. 2001b. *Occupational safety and health standards: Asbestos.* 29CFR1926.1101. U.S. Government Printing Office, Washington, D.C.

Chen, P.W. and L.E. Robertson. 1972. Human perception thresholds of horizontal motion. *ASCE Journal*, Structure Division, August.

Cobb, N. and R.A. Etzel. 1991. Unintentional carbon monoxide-related deaths in the United States, 1979 through 1988. *Journal of the American Medical Association* 266:659-663.

Collins, S.D. and J. Lehmann. 1953. Excess deaths from influenza and pneumonia and from important chronic diseases during epidemic periods, 1918-51. *Public Health Monograph* 20.10, U.S. Public Health Service *Publication* 213.

Cometto-Muñiz, J.E. and W.S. Cain. 1994a. Sensory reactions of nasal pungency and odor to volatile organic compounds: The alkylbenzenes. *American Industrial Hygiene Association Journal* 55(9):811-817.

Cometto-Muñiz, J.E. and W.S. Cain. 1994b. Perception of odor and nasal pungency from homologous series of volatile organic compounds. *Indoor Air* 4:140-145.

Cox, C.S. 1987. *The aerobiological pathway of microorganisms*. John Wiley & Sons, New York.

CPSC. 2006. *Portable generators: Legal memorandum and staff briefing package for ANPR*. Consumer Product Safety Commission, Washington, D.C.

Crandall, M.S. and W.K. Sieber. 1996. The NIOSH indoor environmental evaluation experience: Part one, building evaluations. *Applied Occupational and Environmental Hygiene*.

Davenport, A.G. 1988. The response of supertall buildings to wind. In *Second century of the skyscraper*, pp. 705-726. L. Beedle, ed. Van Nostrand Reinhold, New York.

Destaillats, H., M.M. Lunden, B.C. Singer, B.K. Coleman, A.T. Hodgson, C.J. Weschler, and W.W. Nazaroff. 2006. Indoor secondary pollutants from household product emissions in the presence of ozone: A bench-scale chamber study. *Environmental Science & Technology* 40:4421-4428.

DHHS. 1986. The health consequences of involuntary smoking. A report of the surgeon general. DHHS *Publication* (PHS) 87-8398. U.S. Department of Health and Human Services, Public Health Services, Office of the Assistant Secretary for Health, Office of Smoking and Health.

Diaz, J., R. Garcia, F. Velazquez de Castro, E. Hernandez, C. Lopez, and A. Otero. 2002. Effects of extremely hot days on people older than 65 years in Seville (Spain) from 1986 to 1997. *International Journal of Biometeorology* 46(3):145–149.

Edwards, J.H. 1980. Microbial and immunological investigations and remedial action after an outbreak of humidifier fever. *British Journal of Industrial Medicine* 37:55-62.

EHD. 1987. *Exposure guidelines for residential indoor air quality.* EHD-TR-156. Environmental Health Directorate, Health Protection Branch. Ottawa, ON.

Ellis, F.P. 1972. Mortality from heat illness and heat aggravated illness in the United States. *Environmental Research* 5.

EPA. 1991. Introduction to indoor air quality, a reference manual. *Report* EPA 400/3-91/003.

EPA. 1992. *Respiratory health effects of passive smoking: Lung cancer and other disorders, review draft*. EPA/600-6-90/006F. Office of Research and Development, Washington, D.C.

EPA. 2001. Mold remediation in schools and commercial buildings. *Report* 402-K-01-001. U.S. Environmental Protection Agency, Washington, D.C.

EPA. 2003. National primary drinking water standards. *Standard* 816-F-03-016. U.S. Environmental Protection Agency, Washington, D.C. Available at http://www.epa.gov/safewater/consumer/pdf/mcl.pdf.

EPA. 2005. IAQ tools for schools—IAQ reference guide. *Report* 402-K-95-001. U.S. Environmental Protection Agency, Washington, D.C.

EPA. 2008a. *National ambient air quality standards (NAAQS)*. U.S. Environmental Protection Agency, Washington, D.C. Available at http://www. epa.gov/air/criteria.html.

EPA. 2008b. *Radon: Health risks*. U.S. Environmental Protection Agency, Washington, D.C. Available at www.epa.gov/radon/healthrisks.html.

FAA. 2000. Prohibition of smoking on scheduled passenger flights: Final rules. 14CFR121, 129, and 135. *Federal Register* 65(112):36, 776-36, 780.

Fanger, P.O. 1970. *Thermal comfort*. Teknisk Forlag, Copenhagen.

Franck, C., P. Skov, and O. Bach. 1993. Prevalence of objective eye manifestations in people working in office buildings with different prevalences of the sick building syndrome compared with the general population. *International Archives of Occupational and Environmental Health* 65:65-69.

Gaul, L.E. and G.B. Underwood. 1952. Relation of dew point and barometric pressure to chapping of normal skin. *Journal of Investigative Dermatology* 19:9.

Geary, D.F. 2000. New guidelines on *Legionella*. *ASHRAE Journal* 44(9): 44-49.

Giles, B., C. Balafoutis, and P. Maheras. 1990. Too hot for comfort: The heatwaves in Greece in 1987 and 1988. *International Journal of Biometeorology* 34:98-104.

Girman, J.R. 1989. Volatile organic compounds and building bake-out. In *Occupational medicine: State of the art reviews*, vol. 4, *Problem buildings: Building-associated illness and the sick building syndrome*, pp. 695-712. J.E. Cone and M.J. Hodgson, eds. Hanley and Belfus, Philadelphia.

Girman, J., Y.-L. Chang, S.B. Hayward, and K.S. Liu. 1998. Causes of unintentional deaths from carbon monoxide poisonings in California. *Western Journal of Medicine* 168(3):158-165.

Glantz, S.A. and W.W. Parmley. 1991. Passive smoking and heart disease epidemiology, physiology, and biochemistry. *Circulation* 83:633-642.

Gonzalez, R.R., L.G. Berglund, and A.P. Gagge. 1978. Indices of thermoregulatory strain for moderate exercise. *Journal of Applied Physiology: Respiratory Environmental and Exercise Physiology* 44(6):889-899.

Greenberg, L. 1964. Asthma and temperature change. *Archives of Environmental Health* 8:642.

Hajat, S., R.S. Kovats, R.W. Atkinson, and A. Haines. 2002. Impact of hot temperatures on death in London: A time series approach. *Journal of Epidemiology and Community Health* 56(5):367-372.

Hales, J.B.S., R.W. Hubbard, and S.L. Graffin. 1996. Limits of heat tolerance. Chapter 15 in *Handbook of physiology*, sect. 4, *Environmental physiology.* M.J. Fregly and C.M. Blatteis, eds. *American Physiological. Society,* Bethesda, MD.

Hardy, J.D. 1971. Thermal comfort and health. *ASHRAE Journal* 13:43.

Hartkopf, V. and V. Loftness. 1999. Global relevance of total building performance. *Automation in Construction* 8(4):377-393.

Harriman, L., G. Brundrett, and R. Kittler. 2001. *Humidity control design guide for commercial and institutional buildings*. ASHRAE.

Hathaway, G.J., N.H. Proctor, J.P. Hughes, and M.L. Fischman, eds. 1991. *Proctor and Hughes' chemical hazards in the workplace*, 3rd ed. Van Nostrand Reinhold, New York.

HC. 2006. *Residential indoor air quality guideline: Formaldehyde*. Health Canada.

Hodgson, A.T. 1995. A review and a limited comparison of methods for measuring total volatile organic compounds in indoor air. *Indoor Air* 5(4):247.

Hodgson, M.J., P.R. Morey, M. Attfield, W. Sorenson, J.N. Fink, W.W. Rhodes, and G.S. Visvesvara. 1985. *Archives of Environmental Health* 40:96.

Hodgson, M.J., P.R. Morey, J.S. Simon, T.D. Waters, and J.N. Fink. 1987. An outbreak of recurrent acute and chronic hypersensitivity pneumonitis in office workers. *American Journal of Epidemiology* 125:631-638.

Holland, W.W. 1961. Influence of the weather on respiratory and heart disease. *Lancet* 2:338.

Hryhorczuk, D.O., L.J. Frateschi, J.W. Lipscomb, and R. Zhang. 1992. Use of the scan statistic to detect temporal clustering of poisonings. *Journal of Toxicology—Clinical Toxicology* 30:459-465.

Hyppel, A. 1984. Fingerprint of a mould odor. *Proceedings of the 3rd International Conference on Indoor Air Quality and Climate*, Stockholm, Sweden, vol. 3, pp. 443-447. B. Berglund, T. Lindvall, and J. Sundell, eds.

ISO. 1984. Guidelines for the evaluation of the response of occupants of fixed structures, especially buildings and off-shore structures, to low-frequency horizontal motion (0.063 to 1 Hz). ISO *Standard* 6897. International Organization for Standardization, Geneva.

ISO. 2006. Ergonomics of the thermal environment—Methods for the assessment of human responses to contact with surfaces—Part 1: Hot surfaces. *Standard* 13732-1:2006. International Organization for Standardization, Geneva.

Justesen, D.R., E.R. Adair, J.C. Stevens, and V. Bruce-Wolfe. 1982. A comparative study of human sensory thresholds: 2450 MHz microwaves vs. far-infrared radiation. *Bioelectromagnetics* 3:117-125.

Katayama, K. and M. Momiyana-Sakamoto. 1970. A biometeorological study of mortality from stroke and heart diseases. *Meteorological Geophysics* 21:127.

Kessler, W.R. and W.R. Anderson. 1951. Heat prostration in fibrocystic disease of pancreas and other conditions. *Pediatrics* 8:648.

Kilbourne, E.M., T.S. Jones, K. Choi, and S.B. Thacker. 1982. Risk factors for heatstroke: A case-control study. *Journal of the American Medical Association* 247(24):3332-3336.

Kjaergaard, S. 1992. Assessment methods and causes of eye irritation in humans in indoor environments. In *Chemical, microbiological, health, and comfort aspects of indoor air quality*, H. Knoeppel and P. Wolkoff, eds., pp. 115-127. Energy Cost Savings Council, European Economic Community, and European Atomic Energy Council, Brussels.

Kjaergaard, S., L. Molhave, and O.F. Pedersen. 1991. Human reactions to a mixture of indoor pollutants. *Atmospheric Environment* 25:1417-1426.

Koren, H. 1990. The inflammatory response of the human upper airways to volatile organic compounds. *Proceedings of Indoor Air '90*, vol. 1, pp. 325-330.

Koren, H., D.E. Graham, and R.B. Devlin. 1992. Exposure of humans to a volatile organic mixture III: Inflammatory response. *Archives of Environmental Health* 47:39-44.

Kunst, A.E., C.W. Looman, and J.P. Mackenbach. 1993. Outdoor air temperature and mortality in The Netherlands: A time-series analysis. *American Journal of Epidemiology* 137(3):331–341.

Leathart, G.L. 1972. Clinical aspects of respiratory disease due to mining. In *Medicine in the mining industry*, J.M. Rogan, ed. Heinemann Medical, London.

Lee, H., and H. B. Awbi. 2004. Effect of internal partitioning on indoor air quality of rooms with mixing ventilation—Basic study. *Building and Environment* 39(2):127-141.

Levetin, E. 1995. Fungi. In *Bioaerosols*, H.A. Burge, ed. CRC Press, Lewis Publishers, Boca Raton, FL.

Lighthart, B. 1994. Physics of bioaerosols. In *Atmospheric microbial aerosols: Theory and applications*, pp. 5-27. B. Lighthart and J. Mohr, eds. Chapman and Hall, New York.

Lighthart, B. and L.D. Stetzenbach. 1994. Distribution of microbial aerosol. In *Atmospheric microbial aerosols: Theory and applications*, pp. 68-98. B. Lighthart and J. Mohr, eds. Chapman and Hall, New York.

Lilienfeld, D.E. 1991. Asbestos-associated pleural mesothelioma in school teachers: A discussion of four cases. *Annals of the New York Academy of Sciences* 643:454-486.

Liu, K.S., J. Wesolowski, F.Y. Huang, K. Sexton, and S.B. Hayward. 1991. Irritant effects of formaldehyde exposure in mobile homes. *Environmental Health Perspectives* 94:91-94.

Mahdavi, A. and U. Unzeitig. 2005. Occupancy implications of spatial, indoor-environmental, and organizational features of office spaces. *Building and Environment* 40(1):113-123.

Mandell, G.L., J.E. Bennett, and R. Dolin, eds. 1999. *Principles and practice of infectious disease*. G. Churchill Livingstone, New York.

Marcinowski, F., R.M. Lucas, and W.M. Yeager. 1994. National and regional distributions of airborne radon concentrations in U.S. homes. *Health Physics* 66(6):699-706.

Martin, T.R. and M.B. Bracken. 1986. Association of low birth weight with passive smoke exposure in pregnancy. *American Journal of Epidemiology* 124(4):633-642.

Martinez, B.F., M.L. Kirk, J.L. Annest, K.J. Lui, E.M. Kilbourne, and S.M. Smith. 1989. Geographic distribution of heat-related deaths among elderly persons: Use of county-level dot maps for injury surveillance and epidemiologic research. *Journal of the American Medical Association* 262:2246-2250.

Mbithi, J.N., V.S. Springthorpe, and S.A. Sattar. 1991. Effect of relative humidity and air temperature on survival of hepatitis A virus on environmental surfaces. *Applied and Environmental Microbiology* 57(5):1394-1399.

McCann, J., L. Horn, J. Girman, and A.V. Nero. 1987. *Short-term bioassays in the analysis of complex mixtures V*, pp. 325-354. Plenum Press, New York.

McCoy, W.F. 2005. *Preventing legionellosis*. International Water Association Publishing, London.

McCoy, W.F. 2006. Legionellosis: Why the problem continues. *ASHRAE Journal* 45(1):24-27.

McMichael, A.J., R.E. Woodruff, and S. Hales. 2006. Climate change and human health: Present and future risks. *The Lancet* 367(9513):859-869.

Meggs, W.J. 1994. RADS and RUDS—The toxic induction of asthma and rhinitis. *Journal of Toxicology—Clinical Toxicology.* 32:487-501.

Miller, J.D. and J. Day. 1997. Indoor mold exposure: Epidemiology, consequences and immunotherapy. *Journal of the Canadian Society of Allergy and Clinical Immunology* 2(1):25-32.

Mølhave, L., R. Bach, and O.F. Pederson. 1986. Human reactions to low concentrations of volatile organic compounds. *Environment International* 12:167-175.

Mølhave, L. 1991. Volatile organic compounds, indoor air quality and health. *Indoor Air* 1(4):357-376.

Mølhave, L., Z. Liu, A.H. Jorgensen, O.F. Pederson, and S. Kjaergard. 1993. Sensory and physiologic effects on humans of combined exposures to air temperatures and volatile organic compounds. *Indoor Air* 3:155-169.

Mølhave, L., S.K. Kjaergaard, T. Sigsgaard, and M. Lebowitz. 2005. Interaction between ozone and airborne particulate matter in office air. *Indoor Air* 15:383-392.

Moolenaar, R.L., R.A. Etzel, and R.G. Parrish. 1995. Unintentional deaths from carbon monoxide poisoning in New Mexico, 1980 to 1988: A comparison of medical examiner and national mortality data. *Western Journal of Medicine* 163(5):431-434.

Morey, P.R. 1988. Experience on the contribution of structure to environmental pollution. In *Architectural design and indoor microbial pollution*, pp. 40-80. R.B. Kundsin, ed. Oxford University Press, New York.

Morey, P.R. 1990. The practitioner's approach to indoor air quality investigations. *Proceedings of the Indoor Air Quality International Symposium*, American Industrial Hygiene Association, Akron, OH.

Morey, P.R. and J.C. Feeley. 1988. *ASTM Standardization News* 16:54.

Morey, P.R. and B.A. Jenkins. 1989. What are typical concentrations of fungi, total volatile organic compounds, and nitrogen dioxide in an office environment? *Proceedings of IAQ '89, The Human Equation: Health and Comfort*, pp. 67-71.

Morey, P.R., M.J. Hodgson, W.G. Sorenson, G.J. Kullman, W.W. Rhodes, and G.S. Visvesvara. 1986. Environmental studies in moldy office buildings. *ASHRAE Transactions* 93(1B):399-419.

Moser, M.R., T.R. Bender, H.S. Margolis, G.R. Noble. A.P. Kendal, and D.G. Ritter. 1979. An outbreak of influenza aboard a commercial airliner. *American Journal of Epidemiology* 110:1-6.

Mumford, J.L., X.Z. He, R.S. Chapman, S.R. Cao, D.B. Harris, K.M. Li, Y.L. Xian, W.Z. Jiang, C.W. Xu, J.C. Chang, W.E. Wilson, and M. Cooke. 1987. Lung cancer and indoor air pollution in Xuan Wei, China. *Science* 235:217-220.

Murray, W. 1995. Nonionizing electromagnetic energies. Chapter 14 in *Patty's industrial hygiene and toxicology*, vol. 3B, pp. 623-727. R.L. Harris, L.J. Cralley, and L.V. Cralley, eds. John Wiley & Sons, Hoboken, NJ.

NAS. 1981. *Indoor pollutants*. National Research Council/National Academy of Sciences, Committee on Indoor Pollutants. National Academy of Sciences Press, Washington, D.C.

NAS. 1983. *Polycyclic aromatic hydrocarbons: Evaluation of sources and effects*. National Academy Press, Washington, D.C.

Nazaroff, W.W., B.K. Coleman, H. Destaillats, A. Hodgson, D.T.L. Liu, M.M. Lunden, B.C. Singer, and C.J. Weschler. 2006. Indoor air chemistry: Cleaning agents, ozone and toxic air contaminants. *Final Report*, ARB Contract 01-336. California Environmental Protection Agency, Air Resources Board, Sacramento.

NIOSH. 1992. NIOSH recommendations for occupational safety and health compendium of policy documents and statements. DHHS (NIOSH) *Publication* 92-100. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Atlanta.

NIOSH. 2007. NIOSH pocket guide to chemical hazards. DHHS (NIOSH) *Publication* 2005-149. U.S. Department of Labor, Occupational Safety and Health Administration, Washington, D.C. Available at www.cdc.gov/niosh/npg/pdfs/2005-249.pdf.

NIOSH. *Annual registry of toxic effects of chemical substances*. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Washington, D.C.

Nordic Conference on Cold. 1991. Cold physiology and cold injuries. *Arctic Medical Research* (now *International Journal of Circumpolar Health*) 50(6).

NRC. 1986. *Environmental tobacco smoke: Measuring exposures and assessing health effects*. National Research Council. National Academy Press, Washington, D.C.

OEHHA. 2007. Air toxicology and epidemiology: All chronic reference exposure levels (cRELs). California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Sacramento. Available at http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html.

OEHHA. 2008. *Chemicals known to the state to cause cancer or reproductive toxicity.* California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Sacramento. Available at http://www.oehha.ca.gov/prop65/prop65_list/files/032108list.pdf.

Oeschli, F.W. and R.W. Buechley. 1970. Excess mortality associated with three Los Angeles September hot spells. *Environmental Research* 3:277.

Ohm, M., J.E. Juto, and K. Andersson. 1992. Nasal hyper-reactivity and sick building syndrome. *IAQ '92: Environments for People*. ASHRAE.

Offermann, F.J., S.A. Loiselle, A.T. Hodgson, L.A. Gundel, and J.M. Daisey. 1991. A pilot study to measure indoor concentrations and emission rates of polycyclic aromatic hydrocarbons. *Indoor Air* 4:497-512.

OSHA. 1994. Proposed rulemaking: Indoor air quality. 29CFR1910, 1915, 1926, and 1928. *Federal Register* 59:15,968-16,039.

OSHA. 2001. Withdrawal of proposal: Indoor air quality. *Federal Register* 66(24).

Parsons, K.C. and M.J. Griffin. 1988. Whole-body vibration perception thresholds. *Journal of Sound and Vibration* 121(2):237-258.

Reiher, H. and F.J. Meister. 1931. The sensitivities of the human body to vibrations. *Forschung* VDI 2:381-386. 1946 translation of *Report* Fts616RE. Headquarters Air Material Command, Wright Field, Dayton, OH.

Repace, J.L. 1984. Effect of ventilation on passive smoking in a model workplace. *Proceedings of an Engineering Foundation Conference on Management of Atmospheres in Tightly Enclosed Spaces*, Santa Barbara.

Repace, J.L. and A.H. Lowrey. 1985. An indoor air quality standard for ambient tobacco smoke based on carcinogenic risk. *New York State Journal of Medicine* 85:381-383.

Repace, J.L. and A.H. Lowrey. 1993. An enforceable indoor air quality standard for environmental tobacco smoke in the workplace. *Risk Analysis* 13(4).

Rohles, F.H., J.A. Woods, and P.R. Morey. 1989. Indoor environmental acceptability: Development of a rating scale. *ASHRAE Transactions* 95(1):23-27.

Russi, M., W. Buchta, M. Swift, L. Budnick, M. Hodgson, D. Berube, and G. Kelefant. 2008. *Guidance for occupational health services in medical centers*. American College of Occupational and Environmental Medicine, Elk Grove Village, IL. Available at www.acoem.org/uploadedFiles/Policies_And_Position_Statements/Guidelines/Guidelines/MCOH Guidance.pdf.

Samet, J.M. 1989. Radon and lung cancer. *Journal of the National Cancer Institute* 81:145.

Samet, J.M., M.C. Marbury, and J.D. Spengler. 1987. Health effects and sources of indoor air pollution. *American Review of Respiratory Disease* 136:1486-1508.

Schulman, J.H. and E.M. Kilbourne. 1962. Airborne transmission of influenza virus infection in mice. *Nature* 195:1129.

Shickele, E. 1947. Environment and fatal heat stroke. *Military Surgeon* 100:235.

Skov, P. and O. Valbjorn. 1987. Danish indoor climate, study group: The sick building syndrome in the office environment: The Danish town hall study. *Environment International* 13:339-349.

Spengler, J.D., H.A. Burge, and H.J. Su. 1992. Biological agents and the home environment. *Bugs, Mold and Rot (I): Proceedings of the Moisture Control Workshop*, E. Bales and W.B. Rose, eds., pp. 11-18. Building Thermal Envelope Council, National Institute of Building Sciences, Washington, D.C.

Strindehag, O., I. Josefsson, and E. Hennington. 1988. *Healthy Buildings '88,* Stockholm, vol. 3, pp. 611-620.

Streifel, A.J., D. Vesley, F.S. Rhame, and B. Murray. 1989. Control of airborne fungal spores in a university hospital. *Environment International* 15:221.

Susskind, R.R. and M. Ishihara. 1965. The effects of wetting on cutaneous vulnerability. *Archives of Environmental Health* 11:529.

Tamas, G., C.J. Weschler, J. Toftum, and P.O. Fanger. 2006. Influence of ozone-limonene reactions on perceived air quality. *Indoor Air* 16: 168-178.

Tancrede, M., R. Wilson, L. Ziese, and E.A.C. Crouch. 1987. *Atmospheric Environment* 21:2187.

Tansey, M.R. and C.B. Fliermans. 1978. Pathogenic species of thermophilic and thermotolerant fungi in reactor effluents of the Savannah River Plant. *DOE Symposium Series CONF-77114: Energy and Environmental Stress in Aquatic Systems Symposium*, pp. 663-690. J.H. Thorpe and J.W. Gibbons, eds.

Teng, H.C. and H.E. Heyer, eds. 1955. The relationship between sudden changes in the weather and acute myocardial infarction. *American Heart Journal* 49:9.

Thirion, X., D. Debensason, J.C. Delaroziere, and J.L. San Marco. 2005. August 2003: Reflections on a French summer disaster. *Journal of Contingencies and Crisis Management* 13(4):153-158.

Weschler, C.J. 2000. Ozone in indoor environments: Concentration and chemistry. *Indoor Air* 10:269.

Weschler, C.J. 2006. Ozone's impact on public health: Contributions from indoor exposures to ozone and products of ozone-initiated chemistry. *Environmental Health Perspectives* 114:1489-1496.

Weschler, C.J. and H.C. Shields. 2000. The influence of ventilation on reactions among indoor pollutants: Modeling and experimental observations. *Indoor Air* 10:92-100.

WHO. 2000. Air quality guidelines for Europe, 2nd ed. *European Series* 91. World Health Organization, Copenhagen.

WHO. 2007. *Legionella and the prevention of Legionellosis.* World Health Organization, Geneva.

Wilkins, C.K., P.A. Clausen, P. Wolkoff, S.T. Larsen, M. Hammer, K. Larsen, V. Hansen, and G.D. Nielsen. 2001. Formation of strong airway irritants in mixtures of isoprene/ozone and isoprene/ozone/nitrogen dioxide. *Environmental Health Perspectives* 109:937-941.

Wolkoff, P., P.A. Clausen, C.K. Wilkins, and G.D. Nielsen. 2000. Formation of strong airway irritants in terpene/ozone mixtures. *Indoor Air* 10: 82-91.

Yoshizawa, S., F. Surgawa, S. Ozawo, Y. Kohsaka, and A. Matsumae. 1987. *Proceedings of the 4th International Conference on Indoor Air Quality and Climate*, Berlin, vol. 1, pp. 627-631.

Yu I.T., Y. Li, T.W. Wong, W. Tam, A.T. Chan, J.H. Lee, D.Y. Leung, and T. Ho. 2004. Evidence of airborne transmission of the severe acute respiratory syndrome virus. *New England Journal of Medicine* 350(17):1731-1739.

Zenz, C., ed. 1988. *Occupational safety in industry, occupational medicine, principles and practical applications*. Year Book Medical Publishers, Chicago.