

CHAPTER 9

INDOOR ENVIRONMENTAL HEALTH

<i>Terminology</i>	9.1	<i>Bioaerosols</i>	9.6
<i>Scope</i>	9.1	<i>Gaseous Contaminants</i>	9.7
<i>DESCRIPTIONS OF SELECTED HEALTH SCIENCES</i>	9.1	<i>Gaseous Contaminants in Industrial Environments</i>	9.8
<i>Epidemiology and Biostatistics</i>	9.1	<i>Gaseous Contaminants in NonIndustrial</i>	
<i>Toxicology</i>	9.2	<i>Environments</i>	9.8
<i>Molecular Biology</i>	9.2	<i>PHYSICAL AGENTS</i>	9.11
<i>Cellular Biology</i>	9.2	<i>Thermal Environment</i>	9.11
<i>Genetics</i>	9.2	<i>Electrical Hazards</i>	9.13
<i>Industrial Hygiene</i>	9.2	<i>Mechanical Energies</i>	9.13
<i>AIR CONTAMINANTS</i>	9.4	<i>Electromagnetic Radiation</i>	9.15
<i>Particles</i>	9.4	<i>Ergonomics</i>	9.17

INTRODUCTION

THIS CHAPTER INTRODUCES the field of environmental health as it pertains to the indoor environment of buildings, briefly covering problem identification and control and regulations. The role of HVAC designers in delivering clean, appropriately conditioned air and removing contaminants is vital, both in industrial and nonindustrial environments. Some knowledge of indoor environmental health will assist in that delivery. In many cases, architectural, structural, cleaning, maintenance, materials use, and other activities that affect the environment are outside the control of that individual. Nevertheless, whenever possible, the HVAC designer should encourage features and decisions that create a healthy building environment.

TERMINOLOGY

The most clearly defined area of indoor environmental health is occupational health, particularly as it pertains to workplace air contaminants. Poisoning incidents as well as human and animal laboratory studies have generated reasonable consensus on safe and unsafe workplace exposures to about one thousand chemicals and dusts. Consequently, many countries regulate exposures of workers. However, contaminant concentrations meeting occupational health criteria usually exceed levels found acceptable to occupants in nonindustrial spaces such as offices, schools, and residences, where exposures are often longer in duration and may involve mixtures of many contaminants and a less robust population (NAS 1981).

Operational definitions of health, disease, and discomfort are controversial (Cain et al. 1995). The World Health Organization (WHO) defines health as “a state of complete physical, mental, and social well-being and not merely the absence of disease or disability.” Last (1983) defines health as “a state characterized by anatomic integrity, ability to perform personally valued family, work, and social roles; ability to deal with physical, biologic, and social stress; a feeling of well-being; and freedom from the risk of disease and untimely death.” Higgins (1983) defines an adverse health effect as a biological change that reduces the level of well-being or functional capacity. These definitions indicate that good health is a function of freedom from active ill health or disease (i.e., short- and long-term disability or impairment, freedom from risk of disease in the future resulting from current exposures, and current subjective well-being).

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

Definitions of comfort are also varying. Traditionally comfort refers to immediate satisfaction. It encompasses perception of the environment (hot, cold, noisy, etc.) and a value rating of affective implications (too hot, too cold, etc.). Rohles et al. (1989) noted that acceptability may represent a more useful concept as it allows progression toward a concrete goal. This serves as the foundation of a number of standards including thermal comfort, acoustics, etc. Nevertheless, acceptability may change over time (secular drift) as expectations change.

SCOPE

This chapter covers indoor environmental health issues such as effects of air contaminants, thermal comfort, acoustics, exposure to radiation, ergonomics, and electrical safety. It presents information on the mechanisms by which the environment affects the human body, gives guidance on recognizing problems, and discusses standards and guidelines that provide protection. Since the number of air contaminants is very large, the introductory material on types, their characteristics, typical levels, and measurement methods is given in [Chapter 12](#). [Chapter 13](#) covers odors.

This chapter also covers indoor air quality (IAQ). The field of indoor air quality is distinct from that of occupational health in several ways, including locations of concern, which are mainly nonindustrial and include offices, schools, public and commercial buildings, and residences; existence of both comfort and health issues; presence of multiple contaminants at low concentrations rather than single ones at high concentration; and impacts of noncontaminant-related issues such as thermal comfort.

More is known about industrial exposures to air contaminants than is known about residential and light commercial exposures. Therefore, most of this chapter focusses on industrial environments.

DESCRIPTIONS OF SELECTED HEALTH SCIENCES

Normal interactions of the human body with its surrounding environment occur in predictable fashions. Light, heat, cold, and sound at extremes of the exposure range can result in such diseases as frostbite, burns, and noise-induced hearing loss. Some transitions between normal and disease states are more difficult to delineate. Pain from bright light, erythema from heat, and nausea from vibration represent reversible effects but are interpreted by health professionals as abnormal. Study of these effects includes a number of scientific disciplines.

EPIDEMIOLOGY AND BIOSTATISTICS

Epidemiology is the study of distributions and determinants of disease. It represents the application of quantitative methods to evaluate diseases or conditions of interest. The subjects may be humans, animals, or even buildings. Epidemiology is traditionally subdivided into observational and analytical components. It may be primarily descriptive, or it may attempt to identify causal associations. Some classical criteria for determining causal relationships in epidemiology are consistency, temporality, plausibility, specificity, strength of the association, and dose-response relationships.

Observational studies are generally performed by defining some 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 factor of interest. Observations conducted at one point in time are considered cross-sectional studies. On the other hand, a group may be defined by some criteria at a specific time, for example, all employees who worked in a certain building for at least one month in 1982. They may then be followed over time, leading to an observational cohort study.

Analytical studies may be either experimental or case-control studies. In experimental studies, individuals are selectively exposed to specific agents or conditions. Such studies are generally performed with the consent of the participants, unless the conditions are part of their usual working conditions and known to be harmless. Sometimes exposures cannot be controlled on an individual basis, and the intervention must be applied to entire groups. Control groups must be observed in parallel. Case-control studies are conducted by identifying individuals with the condition of interest and comparing risk factors between these people and individuals without that condition.

All factors of interest must be measured in an unbiased fashion to avoid a subconscious influence of the investigator. The method of measurement should be repeatable and the technique meaningful. Statistical methods for data analysis follow standard procedures. Tests of hypotheses are performed at a specific probability level. They must have adequate power; that is, if a sample size or a measurement difference between the factors or groups is too small, a statistically significant association may not be found even if one is present.

Results obtained in a specific situation (i.e., in a sample of exposed individuals) may be generalized to others only if they share the same characteristics. For example, it may not be legitimate to assume that all individuals have the same tolerance of thermal conditions irrespective of their heritage. Therefore, the results of studies and groups must be evaluated as they apply to a specific situation.

TOXICOLOGY

Toxicology is the study of the influence of poisons on health. Most researchers hold that essentially all substances may function as poisons and that only smaller doses prevent them from becoming harmful. Of fundamental importance is defining which component of the chemical structure predicts the harmful effect. The second issue is defining the dose-response relationships. The definition of dose may refer to delivered dose (exposure that is presented to the lungs) or absorbed dose (the dose that is actually absorbed through the lungs into the body and available for metabolism). Measures of exposure may be quite distinct from measures of effect because of internal dose modifiers (e.g., the delayed metabolism of some poisons because of lack of enzymes to degrade 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 humans often cannot be exposed in experimental conditions, most toxicological literature is based on animal studies. Recent studies suggest that it is not easy to extrapolate between dose level effects from animals to man. Isolated animal systems, such as

homogenized rat livers, purified enzyme systems, or other isolated living tissues, may be used to study the impact of chemicals.

MOLECULAR BIOLOGY

Molecular biology is the branch of science that studies the chemical and physical structure of biological macromolecules. (DNA, proteins, carbohydrates, etc.). It is interested in processes on a molecular level, identifying actual mechanisms of effect or toxicity, rather on the level of cells.

CELLULAR BIOLOGY

Cellular biology is the branch of science that studies cellular organelles, activities, and processes. Little indoor air quality related research has been done at this mechanistic level, but it offers the final evidence in postulated cause-and-effect relationships.

GENETICS

Genetics is a branch of science that examines heredity and variation among organisms at population, individual, and chromosomal levels. Newer studies in genetics appear to indicate that some individuals are more susceptible to or are at greater risk from certain exposures than the rest of the population. This susceptibility would explain why not all individuals react the same way to the same exposure or lifestyle.

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 included under these principles are (1) identification of toxic chemicals; (2) evaluation of the importance of the physical state and size of airborne particle to absorption by the lungs; (3) evaluation of the importance of airborne particle size to absorption by the lungs and the physical state of individuals; (4) evaluation of the importance of skin absorption and ingestion to exposure and absorption; (5) identification of chemicals to be collected and analyzed; (6) determination of methods for collection of air samples; (7) identification of analytic methods to be used or collaboration with a chemist to develop methods to be used; (8) evaluation of results of measurements; (9) identification of physical stressors, including noise, heat stress, ionizing radiation, nonionizing radiation, ergonomics, and illumination; and (10) development of control measures. In addition to examining the environment, interpreting collected data, and implementing control measures, the industrial hygienist has responsibilities related to creating regulatory standards for the work environment, preparing programs to comply with regulations, and collaborating in epidemiologic studies to document exposures and potential exposures to help determine occupation-related illness.

Hazard Recognition

Occupational hazards generally fall into one of four classes of environmental stressors: chemical, biological, physical, and ergonomic.

Chemical Hazards. Airborne chemical hazards exist as concentrations of mists, vapors, gases, fumes, or solids. Some are toxic through inhalation, some can irritate the skin on contact, some can be toxic by absorption through the skin or through ingestion, and some are corrosive to living tissue. The degree of risk from exposure to any given substance depends on the nature and potency of the toxic effects and the magnitude and duration of exposure. Air contaminants represent a very important subclass of chemical hazards due to their mobility and the ease of exposure through the lungs when they are inhaled. Air contaminants are commonly classified as either particulate or gaseous. Common particulate contam-

Table 1 Diseases Related to Buildings

Disease	History and Physical Examination	Laboratory Testing	Linkage	Causes
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	Direct occupational exposures; molds in the workplace; specific occupational factors (laser toners, carbonless copy paper, cleaning agents), secondary occupational exposures; pet (e.g., cat) danders brought from home
Asthma	Coughing, wheezing, episodic dyspnea, wheezing on examination, chest tightness, temporal pattern at work	Spirometry before and after work on Monday, peak expiratory flow diary, methacholine challenge	Immunologic: skin prick or RAST testing Physiologic: related to work*	See rhinitis/sinusitis
Hypersensitivity pneumonitis	Cough, dyspnea, myalgia, weakness, rales, clubbing, feverishness	DLCO, FVC, TLC, CXR, lung biopsy	Immunologic: IgG ab to agents present, challenge testing Physiologic (in acute forms): spirometry, DLCO	Molds, moisture
Organic dust toxic syndrome	Cough, dyspnea, chest tightness, feverishness	DLCO, TLC	Temporal pattern related to work	Gram-negative bacteria
Contact dermatitis (allergic)	Dry skin, itching, scaling skin	Scaling rash, eczema biopsy	Patch testing	Molds, carbonless copy paper, laser toners
Contact urticaria	Hives	Inspection biopsy	Provocative testing	Office products (carbonless copy paper)
Eye irritation	Eye itching, irritation, dryness	Tear-film break-up time, conjunctival staining (fluorescein)	Temporal pattern	Low relative humidity, volatile organic compounds (allergic conjunctivitis), particles
Nasal irritation	Stuffy, congested nose, rhinitis	Acoustic rhinometry, posterior and anterior rhinomanometry, nasal lavage, nasal biopsy	Temporal pattern	Low relative humidity, volatile organic compounds (allergic conjunctivitis), particles
Central nervous system symptoms	Headache, fatigue, irritability, difficulty concentrating	Neuropsychological testing	Temporal pattern (epidemiology)	Volatile organic compounds, noise, lighting, work stress, carbon monoxide, cytokines from bioaerosol exposure
Legionnaires' disease	Pneumonic illness	History, <i>Legionella</i> culture from biopsy fluids	1) Organism isolated from patient and source, 2) immunologic watch	Aerosols from contaminated water sources, shower heads, water faucet aerators, humidifiers at home and at work, potable water sources (hot water heaters, etc.)

* (1) 10% decrement in FEV₁ across workday,
 (2) peak flow changes suggestive of work relatedness, and
 (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
 FEV₁ = forced expiratory volume in the first second

inants include dusts, fumes, mists, aerosols, and fibers. Gaseous contaminants exist as vapors or gases. More background information on these is given in [Chapter 12](#). Air contaminants are of concern in both industrial and nonindustrial environments. [Table 1](#) summarizes diseases that have been associated with specific aspects of indoor environments, mostly in nonindustrial environments. For these diseases, diagnostic criteria may be used to distinguish between presence or absence of disease. These diseases come about because of the presence of an exposure, a susceptible host, and a vector of transmission.

Biological Hazards. These include bacteria, viruses, fungi, and other living or nonliving organisms that can cause acute and chronic infections by entering the body either directly or through breaks in the skin. In addition, some biological agents can cause allergic reactions or be toxic. Wastes, body parts, etc., of these organisms can also cause illness and allergic reactions.

Physical Hazards. These include excessive levels of ionizing and nonionizing electromagnetic radiation, noise, vibration, illumination, temperature, and force.

Ergonomic Hazards. These include tasks that involve repetitive motions, require excessive force, or must be carried out in awkward

postures, all of which can result in damage to muscles, nerves, and joints.

Hazard Evaluation

Hazard evaluation determines the sources of potential problems. Safety and health professionals research, inspect, and analyze how the particular hazard affects worker health. Assessment of such exposures relies on qualitative, semiquantitative, or quantitative approaches. In many situations, air sampling will determine whether a hazardous condition exists. An appropriate sampling strategy must be used to ensure the 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 would cover an entire work shift; or **short-term exposures**, which would determine the magnitude of exposures to materials that are acutely hazardous. Samples may be collected for a single substance or a multicomponent mixture. Hazard evaluation also characterizes the workplace with respect to potential skin absorption or ingestion hazards. Analysis of bulk material samples and surface wipe samples could determine whether hazardous conditions exist. Physical agent characterization may require direct-

reading sampling methods. After collection and analysis, the industrial hygienist must interpret the results and determine appropriate control strategies.

Hazard Control

The principles for controlling the occupational environment are substitution, isolation, ventilation, and air cleaning. Not all of these principles may be applied to all types of hazards, but all hazards can be controlled by using one of these principles. Engineering controls, work practices controls, administrative controls, and personal protective equipment are used to apply these principles. Source removal or substitution are customarily the most effective measure, but are not always feasible. Engineering controls such as ventilation and air cleaning may be effective for a range of hazards, but usually consume energy. Local exhaust ventilation is more effective for controlling point-source contaminants than is general ventilation. A building HVAC system is an example of a general ventilation system.

AIR CONTAMINANTS

Many of the same air contaminants cause problems in industrial and nonindustrial indoor environments. These include nonbiological particles (synthetic vitreous fibers, combustion nuclei, nuisance dust, and others); bioaerosols; and gases and vapors that may be generated due to industrial processes, by building materials, furnishings, and equipment, by occupants and their activities in a space, or brought in from the outdoors. In industrial environments, contaminants are usually known from the type of process, and exposures may be determined relatively easily by air sampling. Airborne contaminants in nonindustrial environments may (1) occur from emissions and/or shedding of building materials and systems, (2) originate in outside air, and/or (3) be from building operating and maintenance programs and procedures and conditions that may foster growth of biological organisms. In general, in nonindustrial environments, there are many more contaminants that may contribute to problems, they are more difficult to identify, and they are usually present in much smaller concentrations. More information on contaminant types, characteristics, typical levels, and measurement methods can be found in [Chapter 12](#).

PARTICLES

Particulate matter includes airborne solid or liquid particles. Typical examples of particles include dust, smoke, fumes, and mists. Dusts range in size from 0.1 to 25 μm , smoke particles are typically around 0.25 μm , and fumes are usually less than 0.1 μm in diameter (Zenz 1988). Bioaerosols and particles produced from abrasions are usually smaller than 1.0 μm .

Units of Measurement

The quantity of particles in the air is frequently reported as the mass or the 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 ($\mu\text{g}/\text{m}^3$). $1 \text{ mg}/\text{m}^3 = 1000 \mu\text{g}/\text{m}^3$. Mass units are widely used in industrial environments as these units are used to express occupational exposure limits.

Particle counts are usually quoted for 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 useful in less contaminated environments such as office buildings and industrial clean rooms.

Particles in Industrial Environments

Particles found in the work environment are generated as a result of work-related activities (i.e., 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 in order to appropriately address exposure concerns.

The engineer or industrial hygienist determining worker exposure should assess particulate release by the activity, local air movement caused by makeup air and exhaust, and worker procedures for a complete evaluation (Burton 2000).

Health Effects of Exposure. The health effects of airborne particulate matter depend on several factors that include particle dimension, durability, dose, and toxicity of the materials in the particle. A particle must first be inhalable to be hazardous. Respirable particles vary in size from <1 to 10 μm (Alpaugh and Hogan 1988) depending on the source of the particle. Methods for measuring particles are discussed in [Chapter 12](#). Durability, or how long the particle can exist in the biological system before it dissolves, can determine relative toxicity. Lastly, the dose or the amount of exposure encountered by the worker must be considered. In some instances, very small exposures can cause adverse health effects (hazardous exposures) and in others, seemingly large exposures may not cause any adverse health effects (nuisance exposures).

Safety and health professionals are primarily concerned with particles smaller than 2 μm , since this is the range of sizes most likely to be retained in the lungs (Morrow 1964). 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. Submicrometre particles penetrate deeper into the lungs, but many do not deposit and are exhaled.

Dusts

Dusts are coarse solid particles generated by handling, crushing, or grinding. Their size range is typically 1 to 100 μm . They may become airborne during the generation process or through handling. Any industrial process that produces dust fine enough to remain in the air long enough to be inhaled or ingested (size about 10 μm) should be regarded as hazardous until proven otherwise.

Fibers are solid particles whose length is several times greater than their diameter, such as asbestos, man-made mineral fibers, synthetic vitreous fibers, and refractory ceramic fibers.

Mechanisms of Health Effects. A disease associated with the inhalation of particles in industrial settings is pneumoconiosis, a fibrous hardening of the lungs caused by the irritation created from the inhalation of dust. The most commonly known pneumoconioses are asbestosis, silicosis, and coal worker's pneumoconiosis.

Asbestosis results from the inhalation of asbestos fibers (chrysotile, crocidolite, amosite, actinolite, anthophyllite, and tremolite) found in the work environment. The onset of symptomatic asbestosis is uncommon under exposures encountered in the last 45 years before at least 20 to 30 years of exposure (Selikoff et al. 1965, Smith 1955). The asbestos fibers cause fibrosis (scarring) of the lung tissue, which clinically manifests itself as dyspnea (shortness of breath) and a nonproductive, irritating cough. Asbestos fiber is both dimensionally respirable as well as durable in the respiratory system.

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

Coal worker's pneumoconiosis (CWP) results from the 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). Due to the confined underground work environment, exposures have the potential to be

Table 2 OSHA Permissible Exposure Limits (PELs) for Particles (29 CFR 1910.1000, 29 CFR 1926.1101)

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
Respirable dust	Nuisance	5.0 mg/m ³

very high at times, thus creating very high doses. Data meanwhile show that workers may develop CWP at exposure below the current dust standard of 1 mg/m³.

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

The most effective means of controlling exposures to a particle is to totally eliminate it from the work environment. The best dust-control method is a total enclosure of the dust-producing process. A negative pressure is maintained inside the entire enclosure by exhaust ventilation (Alpaugh and Hogan 1988). This control strategy is typically found in manufacturing operations.

Local exhaust ventilation as an exposure control strategy is most frequently used where particles are generated either at large volumes or with high velocities (i.e., lathe and grinding operations). In this situation, high-velocity air movement captures the particle and removes it from the work environment. A number of recent studies show that push/pull methodology enhances the capture efficiency, but requires care in not “pushing” contaminants into the work environment. Counter airflow situations in source capture applications should be avoided.

General ventilation control of the work environment is defined as a dilution approach to reducing exposures. This type of ventilation is used when particulate sources are numerous and widely distributed over a large area. In this control strategy, the work environment is exhausted outside and resupplied with fresh air, thus diluting the work environment. Unfortunately this strategy is the least effective means of control and very costly because conditioned (warm or cold) air is exhausted and nonconditioned air is introduced.

Recirculation of indoor air through filters can be an effective method of reducing indoor particle concentrations. Filtration costs are often lower than costs of general ventilation.

The least desirable strategy used to control exposures is the use of personal protective equipment—a respirator. Respirators are appropriate as a primary control during intermittent maintenance or cleaning activities when fixed engineering (local or general ventilation) controls may not be feasible. Respirators can also be used as a supplement to good engineering and work practice controls to increase employee protection and comfort (Alpaugh and Hogan 1988).

Exposure Standards and Criteria. In the United States, the Occupational Safety and Health Administration (OSHA) has established Permissible Exposure Limits (PELs), which are published in the Code of Federal Regulations (CFR 1989a,b) under the authority of the Department of Labor. [Table 2](#) lists PELs for several particles commonly encountered in the workplace.

Synthetic Vitreous Fibers

Exposures and Exposure Sources. A fiber can be defined as a slender, elongated structure with substantially parallel sides. These

parameters distinguish this form of particulate matter from a dust, which is more spherical. Synthetic vitreous fibers (SVFs) comprise a large number of important manufactured products, such as textile fibers; insulation and ceiling tile wool, including glass fibers, slag, and rock wool fibers; refractory ceramic fibers; and certain specialty glass fibers.

Exposures to SVFs primarily occur during manufacture, fabrication and installation, and demolition. Simultaneous exposures to other dusts (asbestos during manufacture, demolition products and bioaerosols during demolition) may be important as well. Facilities generally manufacture only one form. Generally, only spun glass and refractory ceramic fibers are in the respirable range. Manufacturing operations are most easily designed to assure a clean work environment, while product application operations are more difficult to control. Data on exposure likely to occur in buildings show that background levels are almost uniformly below 0.0001 fibers/cm³.

Health Effects of Exposure. The possible effects of SVFs on health include the following:

Cancer. Respirable SVFs are considered to have the potential to cause carcinogenic and noncarcinogenic health effects. Although implantation studies have suggested the potential for carcinogenesis, this route of exposure is generally not pertinent for humans. Therefore, although SVFs are often classified as potential human carcinogens by regulatory and professional agencies and organizations, reviews of epidemiology studies generally fail to find convincing evidence that they are associated with excess rates of human cancer. Some mortality studies have identified mild excesses of respiratory cancer. These have been attributed to concurrent asbestos exposure and to smoking. Only refractory ceramic fibers are currently considered likely to represent true human carcinogens, although other very hard fibers are likely to have similar effects.

Nonmalignant respiratory disease. Cross-sectional surveys have suggested that few measurable adverse health effects are attributable to SVFs alone. The strongest evidence suggests that SVFs may exacerbate smoking-induced obstructive lung disease; some authors consider fiberglass, no different than any other dust, to cause excess rates of chest symptoms.

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

Exposure Control Strategies. As with other particles, SVF control strategies include source exclusion or enclosure, local exhaust, and the use of personal protective devices such as respirators. In indoor environments, SVFs may be identified in surface wipe samples. Appropriate intervention strategies focus on source control.

Exposure Standards and Criteria. At present, SVFs are regulated by OSHA as a “nuisance dust” with an 8-hour time-weighted average of 15 mg/m³ for total dust and 5 mg/m³ for respirable dust.

Combustion Nuclei

Exposures and Exposure Sources. Combustion nuclei can be defined as the particulate products of the combustion process. Combustion products from a material include water vapor, carbon dioxide, heat, oxides of carbon and nitrogen, and particles known as combustion nuclei. In many situations combustion nuclei can be hazardous. They may contain potential carcinogens such as polycyclic aromatic hydrocarbons (PAHs).

Polycyclic aromatic compounds (PACs) are the nitrogen-, sulfur-, and oxygen-heterocyclic analogs of PAH and other related PAH derivatives. Depending on their relative molecular mass and vapor pressure, PACs are distributed between vapor and particulate phases. In

general, combustion particles are smaller than dusts generated by mechanical means.

Typical sources of combustion nuclei are tobacco smoke (cigarettes, pipes, and cigars), fossil-fuel-based heating devices such as 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 such contaminants in indoor air. Combustion nuclei are thus important in both industrial and non-industrial settings.

Exposure Standards and Criteria. OSHA has established exposure limits for several of the carcinogens categorized as combustion nuclei (benzo(a)pyrene, cadmium, nickel, benzene, *n*-nitrosodimethylamine). These limits are established for industrial work environments and are not directly applicable to indoor air situations. Underlying atherosclerotic heart disease in individuals may be exacerbated by carbon monoxide (CO) exposures.

Exposure Control Strategies. Exposure control strategies for combustion nuclei are in many ways similar to those applied for other particles. For combustion nuclei derived from heating spaces, air contamination can be avoided by proper installation and ventilation 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. Changing makeup air availability, through the addition of enclosures, may be equally important.

Particles in Nonindustrial Environments

In the nonindustrial indoor environment, the indoor aerosol will be affected greatly by the outdoor particle environment. Indoor particulate 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 and fires and environmental tobacco smoke. In general, source control is the preferred method. If a dust problem is identified, characterization of the nature of the dust will allow the development of an appropriate intervention strategy.

Although asbestos is encountered in insulation in many buildings, it generally does not represent a respiratory hazard except to individuals who actively disturb it in the course of 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.

The combustion nuclei of environmental tobacco smoke (ETS) consists of exhaled mainstream smoke from the smoker and sidestream smoke that is emitted from the smoldering tobacco. ETS consists of between 70 and 90% sidestream smoke and has a somewhat different chemical composition 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 toxic constituents are more concentrated in sidestream than in mainstream smoke (Glantz and Parmley 1991). In studies conducted in residences and office buildings with tobacco smoking, ETS was a substantial source of many gas 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. Polycyclic aromatic compounds generated by combustion processes include many PAHs and nitro-PAHs that have been shown to be carcinogenic in animals (NAS 1983). Other PACs are biologically active as tumor promoters and/or cocarcinogens. Mumford et al. (1987) reported high exposures to PAH and

aza-arenes for a population in China with very high lung cancer rates.

ETS has been shown to be causally associated with lung cancer in adults (NRC 1986, DHHS 1986), and respiratory infections, asthma exacerbations, middle ear effusion (NRC 1986, DHHS 1986), and low birth mass 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 headache and irritation. ETS is also a cause of sensory irritation and annoyance (odors and eye irritation).

Control of ETS is somewhat different in that it has been done primarily through regulatory mandates controlling the practice of tobacco smoking. Most states in the United States have passed laws to control tobacco smoking in public places such as restaurants and workplaces, and airlines have prohibited tobacco smoking on flights lasting 6 h or less. Where tobacco smoking is allowed, appropriate local and general ventilation can be used for control. OSHA has proposed that tobacco smoke in indoor environments be controlled through the use of separately ventilated and exhausted smoking lounges. These lounges are kept under negative pressure relative to all adjacent and communicating indoor spaces.

BIOAEROSOLS

Aerobiology is the study of airborne microorganisms or other biologically produced particles and the effects of these aerosols on other living organisms (people, animals, vegetation, etc.). Bioaerosols are airborne microbiological particulate matter derived from viruses, bacteria, fungi, protozoa, algae, mites, plants, insects, and their cellular or cell mass components. Bioaerosols are present in both indoor and outdoor environments. For the indoor environment, locations that provide appropriate temperature and humidity conditions for reproduction and a food source to support growth may become problematic.

Sources

Floors and floor coverings in hospitals can be reservoirs for organisms that are subsequently resuspended into the air. Carpet cleaning may even promote resuspension (Cox 1987). Some viruses may persist up to 8 weeks on nonporous surfaces (Mbithi et al. 1991).

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 a bubble burst phenomenon. Such dispersal may take place in surf, river sprays, or man-made sources such as whirlpools.

People are an important source of bacteria and viruses in indoor air. Contagious diseases occur when living organisms overcome the defense of the host and establish an infection in the host that may in turn infect another human. Infected humans are the primary sources for contagious disease and primary disseminators as well. Virulent agents can also be released from human skin when disease produces skin lesions, or dispersed from respiratory tract infection during coughing, sneezing, or talking. Other means for release directly from infected humans include sprays of saliva and other respiratory secretions during dental and respiratory therapy procedures. Blood sprays that occur during dental and surgical procedures are of potential concern for aerosol transmission of bloodborne diseases, including HIV and hepatitis viruses. Large droplets can transmit infectious particles to those close to the disseminator, while smaller particles can remain airborne for short or very long distances (Moser et al. 1979).

Both the physical and biological properties of the bioaerosols need to be understood. For a microorganism to cause a building-related illness, it must be transported in sufficient dose to the breathing zone of a susceptible occupant. Airborne infectious par-

ticles behave physically in the same way as any other aerosol-containing particles with similar size, density, electrostatic charge, etc. The major difference with bioaerosols is that they must remain viable to cause infection, although nonviable particles may promote 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 such factors as relative humidity, temperature, oxygen, pollutants, ozone, and ultraviolet light (Burge 1995). The effect of any one factor on survival and virulence can be different for different organisms.

Although microorganisms are normally present in indoor environments, the presence of abundant moisture and nutrients in interior niches amplifies the growth of some microbial agents. Thus certain types of humidifiers, water spray systems, and wet porous surfaces can be reservoirs and sites for growth of fungi, bacteria, protozoa, algae, or even nematodes (Strindehag et al. 1988, Arnow et al. 1978, Morey et al. 1986, Morey and Jenkins 1989). Excessive air moisture (Burge 1995) and floods (Hodgson et al. 1985) may cause the proliferation of microorganisms indoors. The turbulence associated with the start-up of air-handling unit plenums may also elevate concentrations of bacteria and fungi in occupied spaces (Yoshizawa et al. 1987).

Health Effects

Exposure to airborne fungal spores, hyphal fragments, or metabolites can cause a variety of respiratory diseases. These range from allergic diseases including allergic rhinitis, asthma, and 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 (Spengler et al. 1992, Miller and Day 1997).

The presence of microorganisms in indoor environments may cause infective and/or allergic building-related illnesses (Morey and Feeley 1988, Burge 1989). Some microorganisms under certain conditions may produce volatile chemicals (Hyppel 1984) that are malodorous.

The diseases produced by the *Legionella* genus of bacteria are collectively called legionellosis. Presently more than 34 species of the *Legionella* family have been identified, of which over 20 have been isolated from both environmental and clinical sources. The diseases produced by *Legionella pneumophila* include the pneumonia form, Legionnaires' disease, and the flu-like form, Pontiac fever. *L. pneumophila* serogroup 1 is the most frequently isolated from nature and most frequently associated with disease. It has also been suggested that the host relationship affects the virulence of *Legionella* spp.

The fungal genus *Aspergillus* is widely distributed and is common in the soil and on decaying vegetation, dust, and other organic debris (Levetin 1995). The small spores are buoyant and remain airborne for long periods (Streifel et al. 1989). Most opportunistic fungal infections are caused by *Aspergillus fumigatus*. The literature on aspergillosis is extensive, particularly for hospitals, and in many cases the environmental source of the infection has been identified.

Histoplasmosis, an infective illness caused by the fungus *Histoplasma capsulatum*, has occurred (rarely) as a building-related illness among individuals involved in the removal of bat or bird droppings in abandoned buildings (Bartlett et al. 1982) and among chicken coop cleaners. Presumably asexual spores (conidia) from this fungus were inhaled by workers who removed the droppings without adequate respiratory protection.

Outbreaks of infective illness in the indoor air may be caused by other types of microorganisms, such as viruses. For 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.

Allergic respiratory illness may develop due to inhalation of particles containing microorganisms or their components, such as spores, enzymes, mite excreta, and cell wall fragments. Numerous cases of allergic respiratory illness (humidifier fever, hypersensitivity pneumonitis) report affected people manifesting 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 originating 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). Affected individuals usually experience relief only after having left the building for an extended period in contrast to occupants with sick building syndrome, where relief is relatively rapid.

Crandall and Sieber (1996) showed that 47 of 104 problem buildings evaluated had water damage in occupied building areas. Other studies suggest that microorganisms in indoor air are important (Burge et al. 1987, Brundage et al. 1988, Burge 1995).

Guidelines

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

- Incomplete data on concentrations and types of microbial particles indoors, especially as affected by geographical, seasonal, and type-of-building parameters
- Absence of data relating bioaerosol exposure to building-related illness
- Enormous variability in kinds of microbial particles including viable cells, dead spores, toxins, antigens, and viruses
- Large variation in human susceptibility to microbial particles, making estimates of health risk difficult

However, even in the absence of numerical guidelines, bioaerosol sampling data can be interpreted based on such factors as

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

For a microorganism to cause a building-related illness, it must be transported in sufficient dose to the breathing zone of a susceptible occupant. Thus, the concepts of reservoir, amplifier, and disseminator need to be considered in interpreting data. Reservoirs allow microorganisms to survive, amplifiers allow microorganisms to proliferate, and disseminators effectively distribute bioaerosols. Some factors and systems may be all or only one of these. A cooling tower is all three for *Legionella*; that is, a cooling tower can harbor microorganisms in scale, allow them to proliferate, and generate an aerosol.

GASEOUS CONTAMINANTS

This category of indoor contaminants includes 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 (VOCs) and inorganic air contaminants.

Volatile organic compounds 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) used as refrigerants. More information on classifications, characteristics and measurement methods can be found in [Chapter 12](#).

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/m³). For smaller quantities, parts per billion (ppb) and micrograms per cubic metre (µg/m³) are used. The relationship between all of these is explained in [Chapter 12](#).

GASEOUS CONTAMINANTS IN INDUSTRIAL ENVIRONMENTS

The Occupational Safety and Health Administration (OSHA) sets Permissible Exposure Limits (PELs), which are the only workplace regulatory standards in the United States. These are published yearly in the *Code of Federal Regulations* (29 CFR 1900, Part 1900.1000 ff) and intermittently in the *Federal Register*. Most of the levels were recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) and the American National Standards Institute (ANSI). The health effects on which these standards were based can be found in their publications (ACGIH 1989). ACGIH reviews data on a regular basis and publishes Threshold Limit Values (TLVs) yearly.

The National Institute for Occupational Safety and Health (NIOSH) is charged with researching toxicity problems, and it influences the legally required levels. NIOSH publishes the *Registry of Toxic Effects and Chemical Substances* (RTECS) as well as numerous Criteria for Recommended Standard for Occupational Exposure to (compound). Some compounds not in the OSHA list are covered by NIOSH literature, and their Recommended Exposure Limits (RELs) are sometimes lower than the legal requirements set by OSHA. The NIOSH Pocket Guide to Chemical Hazards (NIOSH 1990) is a condensation of these references and is convenient for engineering purposes.

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

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

In non-OSHA literature, AMP is sometimes called STEL (short-term exposure limit), and TWA8 is sometimes called TLV (threshold limit value.) NIOSH (1990) also lists values for the toxic limit IDLH—immediately dangerous to life and health.

[Table 3](#) lists values of the various exposure limits defined above for a selection of common gaseous industrial contaminants. Other countries have also issued standards for industrial exposure.

It is of interest to compare standards for industrial and nonindustrial environments. A Canadian National Task Force developed guideline criteria for residential indoor environments. Similarly, the World Health Organization has published indoor

Table 3 Characteristics of Selected Gaseous Air Pollutants

Pollutant	Allowable Concentration, mg/m ³			
	IDLH ^a	AMP ^a	ACC ^a	TWA8 ^a
Acetaldehyde	18 000			360
Acetone	4 800		3 200	2 400
Acetonitrile	7 000	105		70
Acrolein	13		0.75	0.25
Acrylonitrile	10			45
Allyl chloride	810		9	3
Ammonia	350		35	38
Benzene	10 000		25	5
Benzyl chloride	50			5
2-Butanone (MEK)	8 850			590
Carbon dioxide	90 000		54 000	9 000
Carbon monoxide	1 650		220	55
Carbon disulfide	1 500	300	90	60
Carbon tetrachloride	1 800	1 200	150	60
Chlorine	75		1.5	3
Chloroform	4 800		9.6	240
Chloroprene	1 440		3.6	90
<i>p</i> -Cresol	1 100			22
Dichlorodifluoromethane	250 000			4 950
Dioxane	720			360
Ethylene dibromide	3 110	271	233	155
Ethylene dichloride	4 100	818	410	205
Ethylene oxide	1 400		135	90
Formaldehyde	124	12	6	4
<i>n</i> -Heptane	17 000			2 000
Hydrogen chloride	140		7	7
Hydrogen cyanide	55			11
Hydrogen fluoride	13		5	2
Hydrogen sulfide	420	70	28	30
Mercury	28			0.1
Methane	ASPHY ^b			
Methanol	32 500			260
Methyl chloride	59 500		1 783	1 189
Methylene chloride	7 500		3 480	1 740
Nitric acid	250			5
Nitric oxide	120	45		30
Nitrogen dioxide	90		1.8	9
Ozone	20			2
Phenol	380		60	19
Phosgene	8		0.8	0.4
Propane	36 000			
Sulfur dioxide	260			13
Sulfuric acid	80			1
Tetrachloroethane	1 050			35
Tetrachloroethylene	3 430	2 060	1 372	686
<i>o</i> -Toluidene	440			22
Toluene	7 600	1 900	1 140	760
Toluene diisocyanate	70		0.14	0.14
1,1,1-Trichloroethane	2 250			45
Trichloroethylene	5 410	1 620	1 080	541
Vinyl chloride monomer			0.014	0.003
Xylene	43 500		870	435

^aIDLH, AMP, ACC, and TWA8 are defined in the section on Gaseous Contaminants in Industrial Environments.

^bASPHY = Simple asphyxiant; causes breathing problems when concentration reaches about 1/3 atmospheric pressure.

air quality guidelines for Europe. [Table 4](#) compares these guidelines with occupational criteria for selected contaminants.

GASEOUS CONTAMINANTS IN NONINDUSTRIAL ENVIRONMENTS

The gaseous contaminants that are of concern in nonindustrial environments are volatile organic compounds and inorganic gases.

Table 4 Comparison of Standards Pertinent to Indoor Environments

	Canadian	WHO/Europe	NAAQS/EPA ^f	NIOSH REL	OSHA	ACGIH	MAK ^g
Aldehydes							
Acrolein	0.02 ppm ^a			0.1 ppm 0.25 ppm (15 min)	0.1 ppm 0.3 ppm (15 min)	0.1 ppm 0.3 ppm (15 min)	0.1 ppm 0.2 ppm (15 min)
Acetaldehyde	5.0 ppm			ALARA ^b	100 ppm 150 ppm (15 min)	100 ppm 150 ppm (15 min)	50 ppm
Formaldehyde	0.1 ppm ^c	0.081 ppm		0.016 ppm 0.1 ppm (15 min)	0.75 ppm 2 ppm (15 min)	0.3 ppm	0.3 ppm
Carbon dioxide	3500 ppm			5000 ppm 30,000 ppm (15 min)	10,000 ppm 30,000 ppm (15 min)	5000 ppm 9000 ppm (15 min)	5000 ppm 9000 ppm (15 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 (8 h) 200 ppm (15 min)	35 ppm (8 h) 200 ppm (15 min)	25 ppm (8 h)	30 ppm
Nitrogen dioxide	0.05 ppm 0.25 ppm (1 h)	0.08 ppm (24 h) 0.2 ppm (1 h)	0.053 ppm (1 yr)		1 ppm (15 min)	3 ppm 5 ppm (15 min)	5 ppm
Ozone	0.12 ppm (1 h) no long-term level	0.08 ppm (8 h) 0.1 ppm (1 h)	0.12 ppm (1 h) 0.085 ppm (8 h)	0.1 ppm (15 min)	0.1 ppm (8 h) 0.3 ppm (15 min)	0.05 ppm (8 h) 0.2 ppm (15 min)	0.1 ppm
Particle < 2.5 MMAD ^d	40 µg/m ³ (8 h) 100 µg/m ³ (1 h)		50 g/m ³ (1 yr)		5 mg/m ³ (8 h) (respirable dust)	3 mg/m ³ (8 h) (no asbestos, <1% crystalline silica)	
Sulfur dioxide	0.019 ppm 0.38 ppm (5 min)			2 ppm (8 h) 5 ppm (15 min)	2 ppm (8 h) 5 ppm (15 min)	2 ppm (8 h) 5 ppm (15 min)	2 ppm
Radon	800 Bq/m ^{3e}						
Relative humidity	30-80% (summer) 30-55% (winter)						

() Numbers in parentheses represent averaging periods

^aParts per million (10⁶)^bAs low as reasonably achievable^cTarget level of 0.05 ppm because of its carcinogenic effects^dMass median aerodynamic diameter^eMean in normal living areas^fU.S. EPA National Ambient Air Quality Standards^gGerman Maximale Arbeitsplatz Konzentrationen

The alternative Indoor Air Quality Procedure specified in ASHRAE *Standard* 62 sets limits for concentrations of several contaminants in nonindustrial environments and cautions that contaminants whose toxicity is well-known should be kept at or below one-tenth of the threshold limit value (TLV) specified by ACGIH (annual). When outdoor air quantities are reduced, the actual gaseous contaminant concentrations must be measured to ensure that *Standard* 62 is met.

Health Effects of Volatile Organic Compounds

Adverse health effects potentially caused by 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 et al. 1989).

The chronic adverse health effects due to 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 chronic exposure risk due to VOCs 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).

Carbon tetrachloride (CCl₄) causes central nervous system (CNS) depression and significant liver and kidney damage. CCl₄ has also been shown to be an animal carcinogen and is classified as a potential human carcinogen.

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 (Molhave 1991). A growing body of literature summarizes measurement techniques for the effects of VOCs on nasal (Koren 1990, Koren et al. 1992, Meggs 1994, Ohm et al. 1992, Molhave et al. 1993) and ocular (Kjaergard et al. 1991, Kjaergard 1992, Franck et al. 1993) 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 inter-related to stimulation of the olfactory sense in the nasal cavity, to the gustatory sense on the tongue, and the common chemical sense (Molhave 1991, Cain 1989).

Cometto-Muñiz and Cain (1994a,b) addressed the independent contribution of the trigeminal and olfactory nerves to the detection of airborne chemicals. The sense of smell is experienced through receptors in the nose of the olfactory nerve. Nasal pungency, described as common chemical sensations including prickling, irritation, tingling, freshness, stinging, and burning among others, is experienced through the 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, relatively nonreactive agents—suggests that nasal pungency relies on a physicochemical interaction with a susceptible biophase within the cell membrane. It is postulated that through this nonspecific mechanism, low, subthreshold levels of a wide variety of VOCs—as found in many polluted indoor environments—can be additive in their sensory impact to produce noticeable sensory irritation.

Formaldehyde is a very reactive small molecule that requires different analytical techniques than those usually employed in VOC assessment. Primary sources include urea-formaldehyde resin-based particle and chipboard products used in indoor spaces. It is frequently encountered in indoor spaces in concentrations between 0.04 ppm, a frequently encountered lower limit of detection, and 0.1 ppm (Liu et al. 1991, Ritchie and Lehnen 1987). Many studies have demonstrated its ability to trigger mucous membrane irritation at levels below the ACGIH TLV, and even at levels below 0.1 ppm.

Standards for Volatile Organic Compounds

No standards for exposure to VOCs relevant to nonindustrial indoor environments are in place. NIOSH, OSHA, and the ACGIH have published regulatory standards or recommended limits for industrial occupational exposures (NIOSH 1992, ACGIH annual). With few exceptions, concentrations observed in nonindustrial indoor environments fall well below (100 to 1000 times lower) these published pollutant-specific occupational standards or recommended exposure limits. However, standards for the industrial workplace are higher than would be appropriate for the general population, which includes the elderly, children, and people who are more sensitive to VOCs than the average industrial worker.

Total VOC (TVOC) concentration has previously been used as an indicator of the potency of VOCs to cause health effects. This approach is no longer recommended since the toxicities of individual VOCs vary very widely, and concentrations differ depending on the measurement method used (Hodgson 1995). In controlled exposure experiments, odors are significant at 3 mg/m³. At 5 mg/m³, objective effects were seen in addition to the subjective irritation. Exposures for 50 min to 8 mg/m³ of synthetic mixtures of 20 VOCs lead to significant irritation of mucous membranes in the eyes, nose, and throat.

Both OSHA and the ACGIH have set 8 h standards for formaldehyde as a ceiling level. California issued a residential air quality guideline of 0.1 ppm. In the setting of occupant complaints, the target guideline is 0.05 ppm.

Health Effects of Refrigerants

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. Inhalation exposures to CFCs can cause cardiotoxicity at chronic, low-level exposures. Some are thought to be cardiac sensitizers to epinephrine and put occupants at risk for arrhythmias. Central nervous system (CNS) depression has been found at very high concentrations along with asphyxia. Proctor and Hughes (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 in the vicinity of 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.

CFC-containing systems may only be serviced by certified technicians. Controls for preventing exposures include selection and use of appropriate fittings and valves and insuring that compressed gas cylinders are secured when in use, in transport, and in storage. When repairs are made to leaking or defective components in HVAC equipment, adequate dilution ventilation should be provided to the work area. CFCs should never be used in the vicinity of open flame or heated materials due to the potential for the formation of phosgene gas. ASHRAE *Standard* 15 establishes specific requirements for designing, installing, operating, and servicing mechanical refrigeration equipment.

Health Effects of Inorganic Gases

Carbon monoxide is a chemical asphyxiant. Inhalation of CO causes a throbbing headache brought about because CO has a competitive preference for hemoglobin (about 240 times that of oxygen) and also a shift in the oxygen dissociation curve. Carbon monoxide inhibits oxygen transport in the blood through the formation of carboxyhemoglobin and inhibition of cytochrome oxidase at the cellular level. Deaths and adverse health effects from overexposures are attributed primarily to motor vehicles. Cobb and Etzel (1991) suggested that CO poisoning at home represented a major preventable disease. Moolenaar et al. (1995) subsequently identified similar data and suggested that motor vehicles and home furnaces were primary causes of mortality. Girman et al. (1996) identified both fatal outcomes and episodes. Respectively, 35.9% and 30.6% 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.

Carbon dioxide can become dangerous not as a toxic agent but as a secondary 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 due to simple displacement of oxygen. Concentrations of CO₂ in the nonindustrial environment (office buildings and schools) are often measured in the range of 400 to 1500 depending on occupant density, ventilation distribution, and amount of outside air supplied to the occupied spaces.

Inhalation of **nitric oxide** (NO) causes the formation of methemoglobin, 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 (NO₂) is a corrosive gas with a pungent odor, the odor threshold of which is reported to be between 0.11 and 0.22 ppm (WHO 1987). NO₂ has a low water solubility and therefore can be 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). NO₂ is reported to be a potential carcinogen by way of free radical production (Burgess and Crutchfield 1995). At high concentrations, NO₂ 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 show inconsistency, especially studies relating to exposures from gas cooking stoves (Samet et al. 1987). Indoor concentrations of NO₂ often exceed ambient concentrations due to the presence of strong indoor sources and a trend toward more energy efficient (tighter) homes. Acute toxicity is seldom seen from NO₂ produced by unvented indoor combustion because of the insufficient quantities of NO₂ produced. Chronic pulmonary effects from exposure to combinations of low-level combustion pollutants are possible, however (Bascom et al. 1996).

Sulfur dioxide (SO₂) is a colorless gas with a pungent odor detected at about 0.5 ppm (EPA 1991). Because SO₂ is quite soluble in water, it can react with moisture in the upper respiratory tract to produce irritant effects on the upper respiratory mucosa. Concomitant exposure to fine particles, an individual's depth and rate of breathing, and the presence of preexisting disease can influence the degree of SO₂ toxicity.

Ozone is a pulmonary irritant and causes changes in 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.

Table 5 Inorganic Gas Comparative Criteria

Contaminant	OSHA/NIOSH TWA ^a	EPA NAAQS 1 Std.
Nitric oxide	1 h 2 ppm (5 mg/m ³) 24 h 25 ppm (30 mg/m ³)	None
Nitrogen dioxide	1 h 5 ppm (9 mg/m ³) 24 h 1 ppm (1.8 mg/m ³)	0.053 ppm (100 µg/m ³)
Sulfur dioxide	1 h 5 ppm (13 mg/m ³) 24 h 2 ppm (5 mg/m ³)	0.014 ppm (365 µg/m ³)
Ozone	1 h 0.1 ppm (0.2 mg/m ³) 24 h 0.1 ppm (0.2 mg/m ³)	0.12 ppm (235 µg/m ³)

^aThe values listed are the annual arithmetic mean unless otherwise listed. The first value listed is the 24 h average and the second value is the maximum 1 h average. (TWA = time weighted average)

Inhalation exposures to the gaseous oxides of nitrogen, sulfur (NO_x and SO₂) and ozone (O₃) can and do occur in residential and commercial buildings. These air pollutants are of considerable concern due to the potential for acute and chronic respiratory tract health effects in exposed individuals, particularly individuals with preexisting pulmonary disease.

Standards for Inorganic Gases

Currently, there are no specific United States government standards relative to nonindustrial occupational exposures to air contaminants. Occupational exposure criteria are health-based; that is, they consider healthy workers in an environment, and not necessarily individuals who may be unusually responsive to the effects of chemical exposures. The EPA National Ambient Air Quality Standards (NAAQS) (see [Chapter 12](#)) are also health-based standards designed to protect the general public health from the effects of hazardous airborne pollutants. However, there is a debate as to whether these standards truly represent health-based thresholds. Two of the six criteria, ozone and carbon monoxide, involve toxicologically based research for the development of the standards. The criteria ([Table 5](#)) are not meant to be health-based guidelines for the evaluation of exposures to inorganic gasses in the indoor environment. [Table 5](#) is included for comparative use and consideration by investigators of the indoor environment with the understanding that these criteria may not be completely protective to all industrial workers.

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 [ionizing (radon) and nonionizing (light, radio-frequency, and extremely low frequency magnetic and electric fields)]. Physical agents can act directly upon building occupants, interact with indoor air quality factors, or affect the way humans respond to the indoor environment. Physical agents, while not categorized as indoor air quality factors, often affect human perception of the quality of indoor air.

THERMAL ENVIRONMENT

The thermal environment affects human health in that it affects body temperatures, both internally and externally (of the skin). In the normal, healthy, resting adult, internal or core body temperatures are very stable, with variations seldom exceeding 0.5 K. The internal temperature of a resting adult, measured orally, averages about 37°C; measured rectally, it is about 0.5 K higher (Guyton 1972). The temperature of the core is carefully modulated by an elaborate physiological control system. In contrast, the temperature of the skin is basically unregulated and can vary from about 31 to 36°C in normal environments and activities and also varies over different parts of the skin, with the greatest variation in the hands and feet.

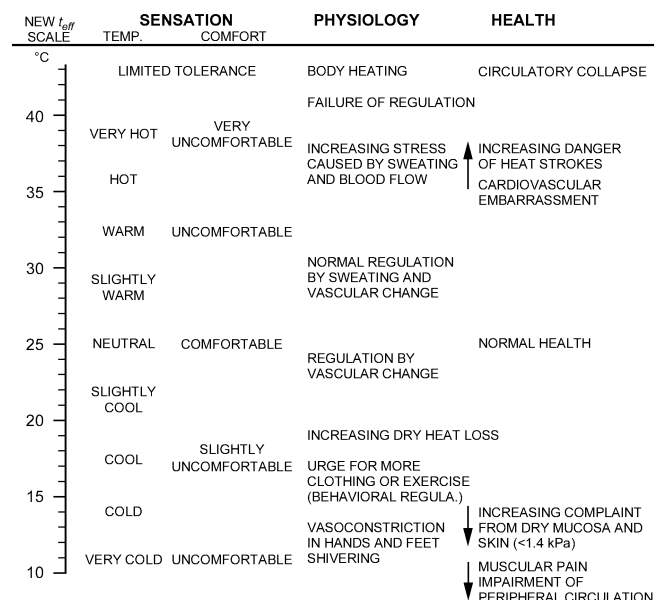


Fig. 1 Related Human Sensory, Physiological, and Health Responses for Prolonged Exposure

Range of Healthy Living Conditions

The environmental conditions for thermal comfort are those that minimize the effort of the physiological control systems to maintain the internal temperature. The control system regulates the internal body temperature by varying the amount of blood flowing to different skin areas, thus increasing or decreasing heat lost to the environment, by secreting and evaporating sweat from the skin in warm or hot environments, and by increasing the metabolic heat production by shivering in the cold. For a resting person wearing trousers and a long-sleeved shirt, thermal comfort is experienced in a still air environment at 24°C. A zone of comfort extends about 1.5 K above and below this optimum level. An individual can minimize the need for physiological (involuntary) responses to the thermal environment that are generally perceived as uncomfortable, by a variety of behavioral responses. In a cool or cold environment, such 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 can be reduced, the level of physical activity can be reduced, or an environment that is more conducive to increased heat loss can be created. Some of the human responses to the thermal environment are shown in [Figure 1](#).

Cardiovascular and other diseases and the inevitable processes of aging can reduce the capacity or ability of physiological processes to maintain internal body temperature through the balancing of heat gains and heat losses. Thus, some persons are less able to deal with thermal challenges and deviations from comfortable thermally neutral 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 old, for comparable size and mass. Persons in their eighties, therefore, prefer an environmental temperature about 1.5 K warmer than persons in their twenties. In any given environment near thermally neutral temperature, an older person is likely to have a lower core and skin temperature. Older people may have reduced capacity to secrete and evaporate 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.

Hyperthermia

Hyperthermia refers to the condition where body temperatures are above normal. A deep body temperature increase of 2 K above the normal range does not generally impair body function. For example, it is not unusual for runners to have rectal temperatures of 40°C after a long race. An elevated body temperature increases metabolism. Central nervous system function deteriorates at deep body temperatures above 41 to 42°C. 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 43°C, after which body temperatures tend to rise rapidly if external cooling is not imposed.

Seasonal Patterns

Ordinary season changes in temperate climates are temporally associated with the prevalence of illness. Many acute and several chronic diseases vary in frequency or severity with time of year, and some are present only in certain seasons. 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 since insects are active in warm temperatures only.

Martinez et al. (1989), Hryhorczuk et al. (1992), and others describe a correlation between weather and seasonal illnesses; but such 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 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 is not sufficiently appreciated. Collins and Lehmann (1953) studied weekly deaths over many years in large cities in the United States and demonstrated the impact of heat waves in producing conspicuous periods of excess mortality. Excess mortality due to heat waves was of the same amplitude as that due to influenza epidemics, but tended to last one week instead of the 4 to 6 weeks duration 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 that occur 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 due to cardiovascular and cerebrovascular causes. 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 the heat death epidemics that continue to occur.

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 w . 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—slightly uncomfortable, comfortable, and very

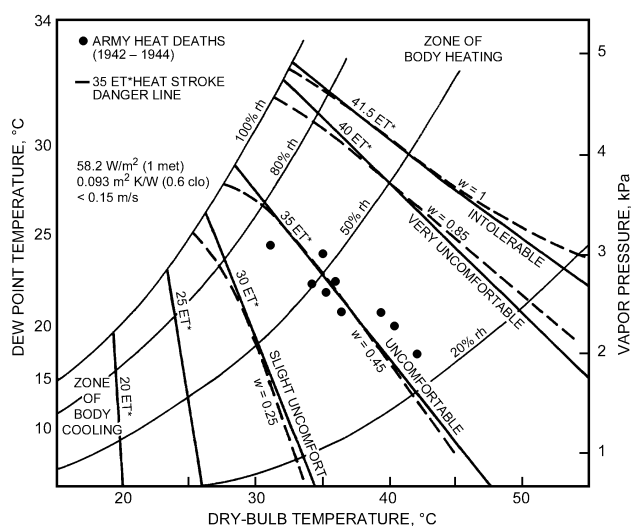


Fig. 2 Isotherms for Comfort, Discomfort, Physiological Strain, Effective Temperature (ET^*), and Heat Stroke Danger Threshold

comfortable (Gonzalez et al. 1978). Skin wettedness, as an indicator of strain (Berglund and Gonzalez 1977, Berglund and Cunningham 1986) 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 23 and 26°C. In this region, skin wettedness is less than 0.2. Heat strokes occur generally when ET^* exceeds 34°C (Bridger and Helfand 1968). Thus, the ET^* line of 35°C is generally considered dangerous. At this point, skin wettedness will be 0.4 or higher.

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). It is to be expected that older persons respond less well to thermal challenges than do healthy soldiers. This was apparently the case in the Illinois heat wave study mentioned earlier, where the first wave with a 33% increase in death rate and an ET^* of 29.5°C 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 the heart disease cases in whom decompensation is present are extremely sensitive to high temperatures and particularly to moist heat. However, both cold and hot temperature extremes are 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). The symptoms of patients with chronic respiratory disease (bronchitis, emphysema) increase in cold weather. This is thought to be due to reflex constriction of the bronchi, adding to the obstruction already present. Greenberg (1964) revealed 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 diminished sweat gland function greatly diminishes their ability to cope with increased temperature (Kessler and Anderson 1951).

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

People suffering from chronic illness (heart disease) or serious acute illnesses that require hospitalization often manage to avoid serious thermal stress. Katayama et al. (1970) found that countries with the most carefully regulated indoor climates (such as the Scandinavian countries and the United States) have had only small seasonal fluctuations in mortality in recent decades, while countries with less space heating and cooling exhibit greater seasonal swings in seasonal mortality. For example, mandatory air conditioning in homes for the aged in the southwest United States has virtually eliminated previously observed mortality increases during heat waves.

Summer cooling reduces heat stress by removing both sensible and latent heat from the occupied space, but winter heating has a mixed effect. It reduces cold stress, but it usually does not increase the low water vapor pressure that occurs outdoors during the winter. This results in very low relative humidity in the heated space, which can contribute to dehydration and discomfort and cause injury to skin, eyes, nose, throat, and mucous membranes. These dry tissues may be less resistant to infection. Animal experiments also show that infection rates increase with low levels of either ventilation or relative humidity (Schulman and Kilbourne 1962).

In various tests conducted under identical conditions except humidity level, mechanical humidification raised the relative humidity in one space above that in the matched space; no humidified room was higher than 50% rh (Green 1979 and 1982, Gelperin 1973, Serati and Wuthrich 1969). In each investigation, the humidified rooms showed a reduction in absenteeism and upper respiratory infection—49% reduction in kindergarten children, 6% and 18% in office workers, and 8% and 18% in army recruits. Since occupants in each pair of spaces were subject to the same outdoor conditions and the same indoor air temperature, reductions were attributed to differences in humidity or a related factor (e.g., reduced dust levels and coughing). Therefore, while low humidity does not have a direct pathological effect, it is a factor contributing to disease. A more direct effect has been indicated among users of contact lenses on long airline flights in cabins at low humidity. Here, dehydration of the eyes has been blamed for causing irritation and corneal edema or even ulceration of the corneal epithelium (Laviana et al. 1988).

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 45°C or falls below about 15°C, sensations from the skin's warm and cold receptors are replaced by those from the pain receptors to warn of thermal injury to the tissue (Guyton 1968). The temperature of the skin depends on the temperature of the contact surface, its conductivity, and the contact time. [Table 6](#)

Table 6 Approximate Surface Temperature Limits to Avoid Pain and Injury

Material	Contact Time				
	1 s	10 s	1 min	10 min	8 h
Metal, water	65°C	56°C	51°C	48°C	43°C
Glass, concrete	80°C	66°C	54°C	48°C	43°C
Wood	120°C	88°C	60°C	48°C	43°C

gives approximate temperature limits to avoid pain and injury when contacting three classes of conductors for various contact times (CEN).

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.

The 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 resistance provides the largest component of the resistance. The 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 the vibration on building occupants depend on whether it is perceived by those persons and on factors related to the building, the location of the building, the activities of the occupants in the building, and the perceived source and magnitude of the 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 the 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 (m/s^2 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 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

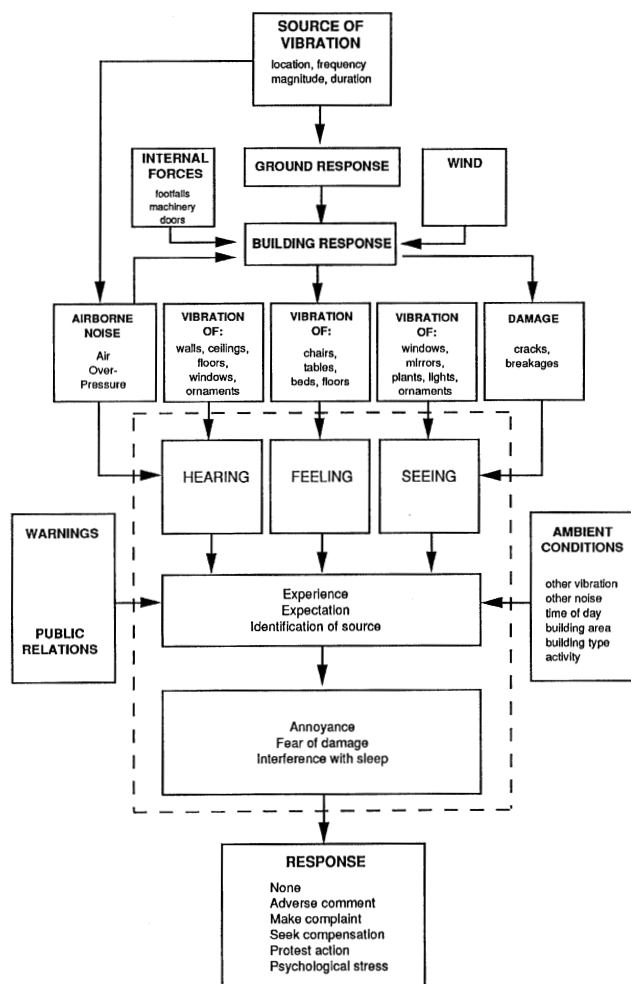


Fig. 3 Factors Affecting Acceptability of Building Vibration

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 will have 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. While 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

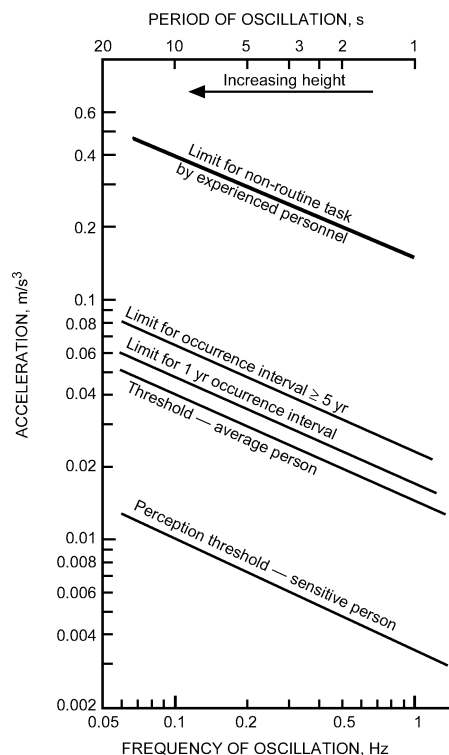


Fig. 4 Acceleration Perception Thresholds and Acceptability Limits for Horizontal Oscillations

(ISO 1984, Chen and Robertson 1972). 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 ($g = 9.8 \text{ m/s}^2$). The perception threshold to sway in terms of building accelerations decreases with increasing frequency and ranges from 50 to 20 mm^2/s .

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 are not to 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 would be 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.

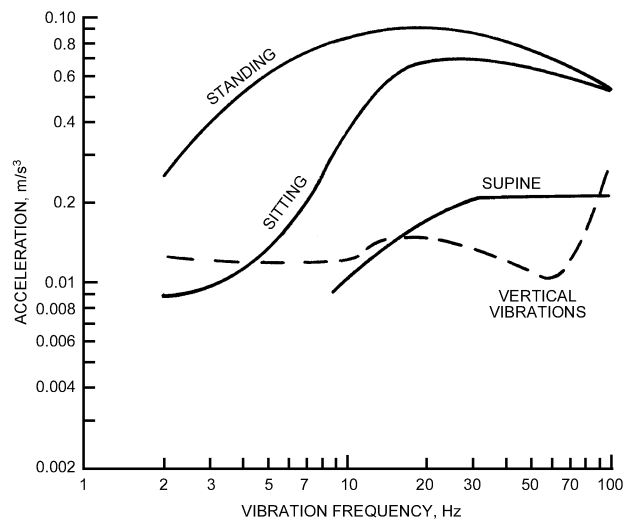


Fig. 5 Median Perception Thresholds to Horizontal (Solid Lines) and Vertical (Dashed Line) Vibrations

Table 7 Acceptable to Threshold Vibration Level Ratios

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: The ratios for continuous or intermittent vibration and repeated impulse shock are in the range of 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 the vibration is more continuous.

High-Frequency Motion (1 to 80 Hz). Higher frequency vibrations in buildings are caused by machinery, elevators, foot traffic, 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 the 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 10 to 90 mm/s², depending on the 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 the vibrations of the structure. The threshold to vertical vibrations is nearly constant at approximately 12 mm/s² 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 would be unacceptable to occupants in their homes. A method of assessing vibrational acceptability in buildings is to compare the vibration with perception threshold values ([Table 7](#)).

Sound and Noise

When the vibration of an object is transmitted to air particles, making them vibrate, a variation in normal atmospheric pressure is

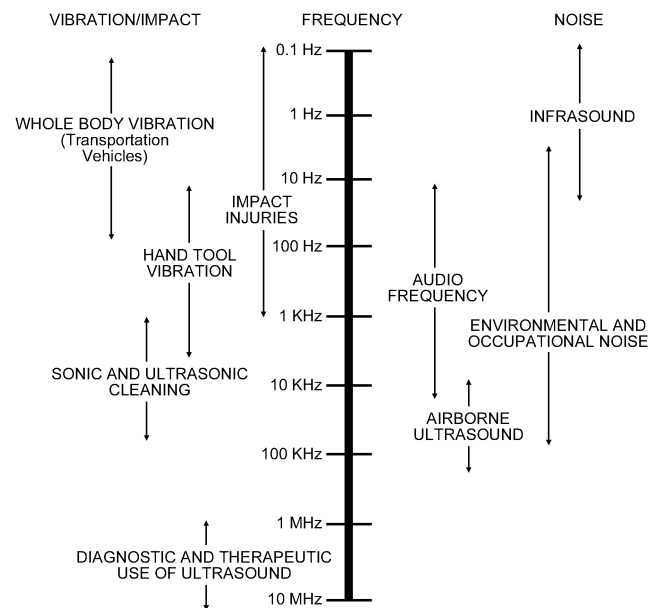


Fig. 6 Mechanical Energy Spectrum

created. When this disturbance spreads to the eardrum, it is vibrated, and this vibration is translated into the sensation called “sound.” In general terms, sound in the physical sense is the vibration of particles in a gas, a liquid, or a solid. The entire 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; for example, 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 ear drum, fluid in the middle ear, or missing elements of the 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) nosacusis, 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, in 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

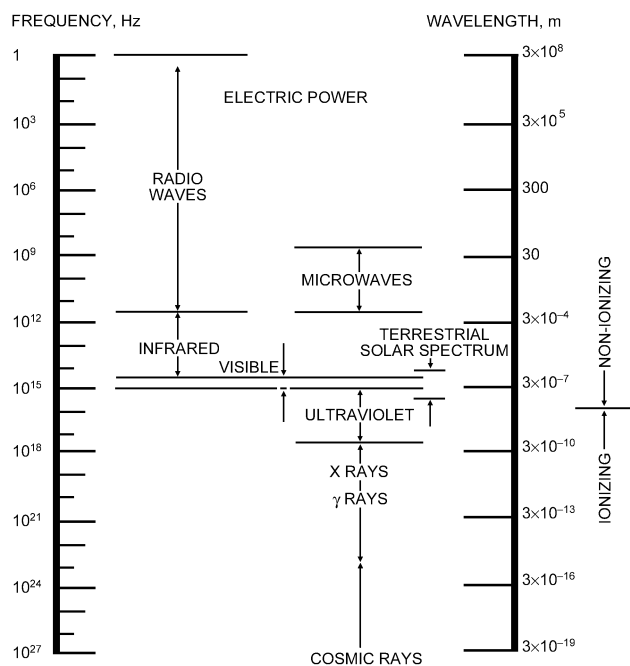


Fig. 7 Electromagnetic Spectrum

spectrum includes ionizing, ultraviolet, visible, infrared, microwave, radio-frequency, and extremely low frequency (Figure 7). Table 8 presents these electromagnetic radiations by their range of energies, frequencies, and wavelengths. The regions are not sharply delineated from each other and, in fact, often overlap. It is convenient to divide these regions as listed in Table 8, due to the nature of the physical and biological effects.

Ionizing Radiation

Ionizing radiation is that part of the electromagnetic spectrum that has very short wavelengths and high frequencies, and it has the ability to ionize matter. Such 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.

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 12.

Health Effects of Radon. Studies of workers in uranium and other underground mines form the principal basis for knowledge about health risks due to radon. 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. They can attach to airborne particles that subsequently deposit in the lung or deposit directly in the lung without prior attachment to particles. Some of these progeny, like radon, are alpha-particle emitters, and the passage of these alpha particles through lung cells can lead to cellular changes that may initiate lung cancer (Samet 1989). Thus, adverse health effects

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 – 3.10 eV	100 – 400 nm	3.00 PHz – 0.75 PHz
Visible	3.10 – 1.63 eV	400 – 760 nm	750 THz – 395 THz
Infrared (IR)	1.63 – 1.24 meV	760 nm – 1 mm	395 THz – 0.30 THz
Microwave (MW)	1.24 meV – 1.24 eV	1 mm – 1 m	300 GHz – 300 MHz
Radio-frequency (RF)	1.24 eV – 1.24 peV	1 m – 1 Mm	300 MHz – 300 Hz
Extremely low frequency (ELF)	< 1.24 peV	> 1 Mm	< 300 Hz

Table 9 Action Levels for Radon Concentration Indoors

Country/Agency	Action Level	
	Bq/m ³	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: DOE (1995).

associated with radon are due to 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.

Standards. Many countries besides the United States have established standards for exposure to radon. International action levels are listed in Table 9.

About 6% of homes in the United States (5.8 million homes) have annual average radon concentrations exceeding the action level of 148 Bq/m³ (4 pCi/L) set by the U.S. Environmental Protection Agency (Marcinowski et al. 1994).

Nonionizing Radiation

Ultraviolet radiation, visible light, and infrared radiation are components of sunlight and of all artificial light sources. Microwave radiation 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 a variety of 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 of the spectral regions can cause acute and/or chronic biologic effects given appropriate energy characteristics and exposure. These effects include tanning, burning (erythema), premature “aging,” and cancer of the skin; 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 results from 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. The 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 of this syndrome also complain of anxiety, irritability, headache, weight gain, and lack of concentration

and motivation. Treatment of this problem is through the 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 , while 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 the absorbed radiation raises the skin temperature and, if sufficient, is detected by the skin’s thermoreceptors, warning the person of the possible thermal danger. With increasing wavelength, the radiation penetrates deeper into the body. The 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). The maximum permitted power densities are less than half of sensory threshold values.

ERGONOMICS

Ergonomics may be defined as the scientific study of the relationship between man and his work environment 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 increased and the worker enjoyed 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 goal of ergonomic programs ranges from making work safe and humane, to increasing human efficiency, to creating human

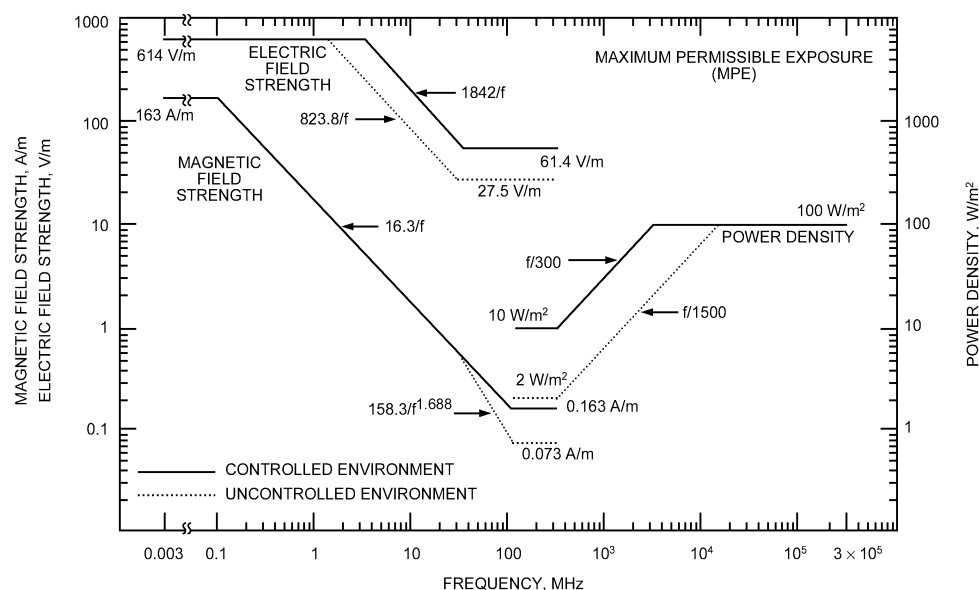


Fig. 8 Maximum Permissible Levels of Radio Frequency Radiation for Human Exposure

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 sciences and 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 disorders or 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.

REFERENCES

- ACGIH. 1989. Guidelines for the assessment of bioaerosols in the indoor environment. American Conference of Government Industrial Hygienists, Cincinnati, OH.
- ACGIH. Annual. Threshold Limit Values and Biological Exposure Indices for 1995-1996.
- Alpaugh, E.L. and T.J. Hogan. 1988. *Fundamentals of industrial hygiene: Particulates*, 3rd Edition. National Safety Council.
- 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. *Ann. N.Y. Acad. Sci.* 643:550-72.
- ANSI. 1991. Safety level with respect to human exposure to radio frequency electromagnetic radiation of 3KH-300GH *Standard C95.1-1991*. American National Standards Institute, New York.
- 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. *American Journal of Medicine* 64:237.
- Bartlett, P.C., L.A. Vonbehren, R.P. Tewari, R.J. Martin, L. Eagleton, M.J. Isaac, and P.S. Kulkarni. 1982. *American Journal of Public Health* 72: 1369.
- Bascom, R.A. 1996. Environmental factors and respiratory hypersensitivity: The Americas. *Toxicol. Lett.* 86:115-30.
- Bascom, R., P. Bromberg, D.L. Costa, et al. 1996. Health effects of outdoor pollution, Parts I and II. *American Journal Respir. Crit. Care Med.* 153:3-50, 477-89.
- Bates, D.V. 1989. Ozone—Myth and reality. *Environmental Research* 50: 230-37.
- Baylor, E.R., V. Peters, and M.B. Baylor. 1977. Water-to-air transfer of virus. *Science*. 197:763.
- 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):767-72.
- Billings, C.E. 1975. Electrical shock. In *Textbook of medicine*. Saunders, Philadelphia, 72-73.
- 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. Lodnar, D.W. Smith, and R.N. Miller. 1988. *JAMA* 259:2108.
- Burch, G.E. and N.P. DePasquale. 1962. *Hot climates, man and his heart*. Charles C. Thomas, Springfield, IL.
- Burge, H.A. 1995. *Bioaerosols*. Lewis Publishers, Chelsea, MI.
- Burge, H.A. 1989. *Occupational medicine: State of the art reviews*. J.E. Cone and M.J. Hodgson, eds. 4:713-21. Hanley and Belfus, Philadelphia.
- Burge, S., A. Hedge, S. Wilson, J.H. Bass, and A. Robertson. 1987. *Annals of Occupational Hygiene* 31:493.
- Burgess, J.L. and C.D. Crutchfield. 1995. Quantitative respirator fit tests of Tucson fire fighters and measures of negative pressure excursions during exertion. *Appl. Occup. Env. Hyg.* 10(1):29-36.
- Burton, D.J. 2000. Industrial ventilation, a self-directed learning workbook. Carr Printing.
- 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.
- CEN. Surface temperatures of touchable parts, a draft proposal. TC 114 N 122 D/E. European Standards Group.
- CFR. 1989a. 29 CFR 1910.1000. U.S. Government Printing Office, Washington, DC.
- CFR. 1989b. 29 CFR 1926.1101.
- 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. *JAMA* 266:659-63.
- 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 No. 213.
- Cometto-Muñiz, J.E. and W.S. Cain. 1994a. Sensory reactions of nasal pungency and odor to volatile organic compounds: The alkylbenzenes. *Am. Ind. Hyg. Assoc. J.* 55(9):811-17.
- Cometto-Muñiz, J.E. and W.S. Cain. 1994b. Perception of odor and nasal pungency from homologous series of volatile organic compounds. *Indoor Air* 4:140-45.
- Cox, C.S. 1987. *The aerobiological pathway of microorganisms*. John Wiley and Sons, New York.
- 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*. Van Nostrand Reinhold, New York, 705-26.
- DHHS. 1986. The health consequences of involuntary smoking. A report of the surgeon general. *DHHS Publication No. (PHS) 87-8398*. U.S. Department of Health and Human Services, Public Health Services, Office of the Asst. Secretary for Health, Office of Smoking and Health.
- DOE. 1995. *Symposium Series CONF-77114*, pp. 663-90. U.S. Dept. of Energy.
- 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.
- 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/600-6-90/006B. Office of Research and Development, Washington, DC.
- 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. *Int. Arch. Occup. Environ. 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.
- Gelperin, A. 1973. Humidification and upper respiratory infection incidence. *Heating, Piping and Air Conditioning* 45:77.
- Girman, J.R. 1989. Volatile organic compounds and building bake-out. In *Occupational medicine: State of the art reviews—Problem buildings*. J.E. Cone and M.J. Hodgson, eds. Hanley and Belfus, Philadelphia. 4(4):695-712.
- Girman, J., Y.-L. Chang, S.B. Hayward, and K.S. Liu. 1996. Causes of unintentional deaths from carbon monoxide poisonings in California. Unpublished.
- Glantz, S.A. and W.W. Parmley. 1991. Passive smoking and heart disease epidemiology, physiology, and biochemistry. *Circulation* 83:633-42.
- Gonzalez, R.R., L.G. Berglund, and A.P. Gage. 1978. Indices of thermoregulatory strain for moderate exercise. *Journal of Applied Physiology: Respiratory Environmental and Exercise Physiology* 44(6):889-99.
- Green, G.H. 1979. The effect of indoor relative humidity on colds. *ASHRAE Transactions* 85(1).
- Green, G.H. 1982. The positive and negative effects of building humidification. *ASHRAE Transactions* 88(1):1049.

- Greenberg, L. 1964. Asthma and temperature change. *Archives of Environmental Health* 8:642.
- Guyton, A.C. 1968. *Textbook of medical physiology*. Saunders, Philadelphia.
- Hardy, J.D. 1971. Thermal comfort and health. *ASHRAE Journal* 13:43.
- Higgins, I.T.T. 1983. What is an adverse health effect? *APCA Journal* 33:661-63.
- 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, J.S. Simon, T.D. Waters, and J.N. Fink. 1987. *American Journal of Epidemiology* 125:631.
- 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.
- 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. *J. Toxicological Toxicology* 30:459-65.
- Hypel, A. 1984. Proceedings of the 3rd International Conference on Indoor Air Quality and Climate, Stockholm, Sweden, 3:443-47.
- ISO. 1984. Guideline to the evaluation of responses of occupants of fixed structures, especially buildings and off-shore structures, to low frequency horizontal motion, 0.063 to 1 Hz. ISO 6897. 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* 3:117-25.
- 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. *JAMA* 247(24):3332-36.
- Kjaergard, S., L. Molhave, and O.F. Pedersen. 1991. Human reactions to a mixture of indoor pollutants. *Atmos. Environ.* 25:1417-26.
- Kjaergard, S. 1992. Assessment methods and causes of eye irritation in humans in indoor environments. H. Knoepfel and P. Wolkoff, eds. *Chemical, Microbiological, Health, and Comfort Aspects of Indoor Air Quality*. ECSC, EEC, EAEC, Brussels 115-27.
- Koren, H. 1990. The inflammatory response of the human upper airways to volatile organic compounds. In: *Proceedings of Indoor Air 90* 1:325-330.
- Koren, H., D.E. Graham, and R.B. Devlin. 1992. Exposure of humans to a volatile organic mixture III: Inflammatory response. *Arch. Environ. Health* 47:39-44.
- Last, J.M. 1983. *Public health and human ecology*. Appleton & Lange, New York.
- Laviana, J.E., F.H. Rohles, and P.E. Bullock. 1988. Humidity, comfort and contact lenses. *ASHRAE Transactions* 94(1).
- Leathart, G.L. 1972. Clinical Aspects of Respiratory Disease Due to Mining. *Medicine in the Mining Industry*.
- Levetin, E. 1995. Fungi. In: Burge, H.A. 1995. *Bioaerosols*. CRC Press. Lewis Publishers. Boca Raton, FL.
- Lilienfeld, D.E. 1991. Asbestos-associated pleural mesothelioma in school teachers: A discussion of four cases. *Ann. N.Y. Acad. Sci.* 643:454-86.
- Liu, K.S., J. Wesolowski, F.Y. Huang, K. Sexton, and S.B. Hayward. 1991. Irritant effects of formaldehyde exposure in mobile homes. *Environ. Health Perspect.* 94:91-94.
- 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. Braken. 1986. Association of low birth weight passive smoke and exposure in pregnancy. *American Journal of Epidemiology* 124:633-42.
- 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. *JAMA* 262:2246-50.
- 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. *Appl. Environ. Microbiol.* 57:1394.
- McCann, J., L. Horn, J. Girman, and A.V. Nero. 1987. *Short-term bioassays in the analysis of complex mixtures*. Plenum Press, New York 5:325-54.
- Meggs, W.J. 1994. RADS and RUDS—The toxic induction of asthma and rhinitis. *J. Toxicol. Clin. Toxicol.* 32:487-501.
- Miller, J.D. and J. Day. 1997. Indoor mold exposure: epidemiology, consequences and immunotherapy. *Can. J. Allergy and Clinical Immunology* 2(1):25-32.
- Molhave, L. 1991. Volatile organic compounds, indoor air quality and health. *Indoor Air* 1(4):357-76.
- Molhave, 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* 3:155-69.
- 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):431-4.
- Morey, P.R. 1988. Architectural design and indoor microbial pollution. Oxford University Press, New York, 40-80.
- 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., 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.
- 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. ASHRAE, Atlanta, 67-71.
- Morrow, P.E. 1964. Evaluation of inhalation hazards based upon the respirable dust concept and the philosophy and application of selective sampling. *AIHA Journal* 25:213.
- Moser, M.R., T. R. Bender, H.S. Margolis, et al. 1979. An outbreak of influenza aboard a commercial airliner. *Am. J. Epidemiol.* 110:1.
- 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* 235:217-20.
- Murray, W. 1995. Non-ionizing electromagnetic energies (Chapter 14). *Patty's Industrial Hygiene and Toxicology* 3B:623-727 (644).
- NAS. 1981. *Indoor Pollutants*. National Research Council/National Academy of Sciences, Committee on Indoor Pollutants. National Academy of Sciences Press, Washington, D.C., Oct.
- NAS. 1983. Polycyclic aromatic hydrocarbons: Evaluation of sources and effects. National Academy Press, Washington, DC.
- NIOSH. 1990. NIOSH/OSHA pocket guide to chemical hazards. DHHS (NIOSH) Publication 90-117. U.S. Department of Labor, OSHA, Washington, DC. Users should read Cohen (1993) before using this reference.
- 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 DC.
- NIOSH. 1992. NIOSH recommendations for occupational safety and health compendium of policy documents and statements. DHHS (NIOSH) Publication 92-100. U.S. Dept. of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Atlanta.
- NRC. 1986. Environmental tobacco smoke: Measuring exposures and assessing health effects. National Research Council. National Academy Press, Washington, DC.
- Oeschli, F.W. and R.W. Beuchley. 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 hyperreactivity and sick building syndrome. *IAQ 92: Environments for People*. ASHRAE, Atlanta.
- Offermann, F.J., S.A. Loiselle, A.T. Hodgson, L.A. Gundel, and J.M. Dai-sey. 1991. A pilot study to measure indoor concentrations and emission rates of polycyclic aromatic hydrocarbons. *Indoor Air* 4:497-512.
- Parsons, K.C. and M.J. Griffin. 1988. Whole-body vibration perception thresholds. *Journal of Sound and Vibration* 121(2):237-58.
- Proctor and Hughes' chemical hazards in the workplace, 3rd ed. 1991. G.J. Hathaway et al. eds. Van Nostrand Reinhold, New York.
- Reiher, H. and F.J. Meister. 1931. Translation of Report Fts616RE 1946. *Forschung VDI* 2, 381-86, "The sensitivities of the human body to vibrations." Headquarters Air Material Command, Wright Field, Dayton, OH.
- Ritchie, I.M. and R.E. Lehnen. 1987. Irritation from formaldehyde in mobile and regular homes. *Am J. Public Health* 77:323-28.

- 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.
- Samet, J.M., M.C. Marbury, and J.D. Spengler. 1987. Health effects and sources of indoor air pollution. *Am. Rev. Respir. Disease* 136:1486-1508.
- Samet, J.M. 1989. Radon and lung cancer. *J. Natl. Cancer Institute* 81:145.
- Schulman, J.H. and E.M. Kilbourne. 1962. Airborne transmission of influenza virus infection in mice. *Nature* 195:1129.
- Selikoff, I.J., J. Churg, and E.C. Hammond. 1965. The occurrence of asbestosis among insulation workers in the United States. *Ann. Rpt. NY Academy of Science*.
- Serati, A. and M. Wuthrich. 1969. Luftfeuchtigkeit und saison Krankheit. *Schweizerische Medizinische Wochenschrift* 99:46.
- Shickele, E. 1947. Environment and fatal heat stroke. *Military Surgeon* 100:235.
- Smith, K.W. 1955. Pulmonary disability in asbestos workers. *Archives of Industrial Health*.
- Spengler, J.D., H.A. Burge, and H.J. Su. 1992. Biological agents and the home environment. In: *Bugs, Mold and Rot (I): Proceedings of the Moisture Control Workshop*, E. Bales and W.B. Rose (eds). 11-18. Building Thermal Envelope Council, National Institute of Building Sciences, Washington, DC.
- Strindehag, O., I. Josefsson, and E. Hennington. 1988. Healthy Buildings '88, 3:611-20. Stockholm, Sweden.
- Streifel, A.J., D. Vesley, F.S. Rhame, and B. Murray. 1989. Control of air-borne fungal spores in a university hospital. *Environ. Int.* 15:221.
- Susskind, R.R. and M. Ishihara. 1965. The effects of wetting on cutaneous vulnerability. *Archives of Environmental Health* 11:529.
- Tancrede, M., R. Wilson, L. Ziese, and E.A.C. Crouch. 1987. *Atmospheric Environment* 21:2187.
- 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.
- WHO. 1987. Air Quality Guidelines for Europe. European Series No. 23. WHO, Copenhagen.
- Yoshizawa, S., F. Surgawa, S. Ozawo, Y. Kohsaka, and A. Matsumae. 1987. *Proceedings 4th International Conference on Indoor Air Quality and Climate*, Berlin 1:627-31.
- Zenz, C. 1988. Occupational safety in industry, occupational medicine, principles and practical applications.